Essay on Health

Refined foods & degenerative disease

Jonathan Stuart Christie – jonty@ix.netcom.com

I'm not a medical doctor and this is not medical advice. Your mileage may vary.

Contents

Despite every effort, medicine can neither prevent nor cure heart disease. The same goes for cancer, diabetes, dementia and even arthritis. As a result, we suffer horribly as we age. Medicine pays lip service to nutrition, yet public policy allows foods depleted of essential nutrients to reach the marketplace. Examples include *trans*-fats, refined vegetable oils and sugar. Societies which do not eat these foods do not deteriorate as we do! Ironically, the healthy people in these societies have almost no knowledge of nutrition. While we have a great deal, we don't use what we know because the food industry and the pharmaceutical companies *with the complicity of government* distort this knowledge for their own purposes.

I have insulin-dependent diabetes, a disease notorious for accelerating heart disease. When I was diagnosed at age 37, I saw the devastation wreaked by the disease in my doctor's waiting room and was driven by sheer terror to search for a solution. For years, my wife and I experimented with diets. We learned that raw vegetables are kinder to the insulin-dependent diabetic than cooked vegetables, but I wasn't out of the woods. In fact, I was at my wit's end because I couldn't keep my blood sugar in the normal range, and the harder I tried, the worse the hypoglycemic episodes I suffered. Then Dr Richard Bernstein published his low-carbohydrate approach, which is the *opposite* of the medical prescription. I found that the combination of my raw vegetables and his approach of avoiding starchy foods like bread and potatoes worked like a charm. I eat meat and fish with (mostly) raw vegetables and almost no refined foods, and eating this way keeps my blood sugar stable and in the normal range. I never gave a thought to heart disease, yet after fifteen years of eating this way, my wife and I *both* have calcium scores in the

aorta of *zero*, which is unusual and implies a low risk of heart disease (Pletcher 2004). I've researched the phenomenon and I'm convinced I've found a reason why: people who have no heart disease eat unrefined foods. Explore the evidence with me and conclude for yourself if this is coincidence.

Kitava Islanders have *no* Coronary Heart Disease

Near Papua, New Guinea, there is an island called Kitava among the Trobriand Islands, which were made famous by Margaret Mead in her 1928 book about their freewheeling sexual practices, *Coming of Age in Samoa*. Later researchers have suggested that the islanders were pulling her leg, and that fear of sorcery is actually far more widespread than promiscuity. However, there is a truly remarkable thing about Kitava: there is today a *complete absence* of heart disease, and also of stroke, high blood pressure, cancer and dementia besides! Whole foods are eaten almost exclusively, and this suggests to me that a benefit of whole-food nutrition really is zero risk of CHD.

The Kitavans are by all accounts a charming people with a rich culture. They have a long history of sailing their ornate, hand-carved outrigger canoes to neighboring islands and trading necklaces and carved shells in an intricate, competitive exchange of gifts. The anthropologist Bronislaw Malinowski was exiled to the island during WWI, and published his

ground-breaking cultural investigation into this so-called Kula exchange in his book, *Argonauts of the Western Pacific.*

In spite of an abundance of food, the Kitavan Islanders are lean and their blood pressure does not rise with age. The common causes of death are infections, accidents, complications of pregnancy and *senescence*. Senescence means old age: *the Kitavans die of old age.* You can hear the cholesterol chorus chime in with "It's their genes!", but a visiting emigrant islander who had lived on a Western diet for many years gave the lie to this notion. He had the typical Western health pattern of high blood pressure and abdominal obesity. And besides, the researchers noted that "compared with [Westerners] … Pacific Islanders seem more prone, not less, to develop diabetes after adopting a Western lifestyle" (Lindeberg 1999).

The Kitavans eat taro, sweet potatoes, yams, fruit, fish and coconuts, and eat very little Western food such as sugar, alcohol, grains, refined vegetable oils, and *trans*-fats. Almost all of them smoke (78%!), all chew the betel nut, and they exercise only at the level of a moderately active Westerner. Their cholesterol levels are said to be "unfavorable." The reason for this is that 60-year-old males average a "bad" LDL-cholesterol level of 120, and the average 60-year-old female scores even higher at 148, while a "desirable" level is less than 100. The researchers thought this was "probably due to a high intake of saturated fat from coconut", albeit in a diet with only a low 21% calories from fat. This seems to me to be a truly awesome failure of the imagination: whatever their cholesterol picture, it must be entirely favorable since they have no heart disease whatsoever! And this is not a fluke. Interestingly, the Tokelau Islanders (among the Cook Islands in the South Pacific) eat no less than 47% of their calories as saturated (coconut) fat, and males aged 55 to 64 have cholesterol levels averaging 245, yet they, too, enjoy robust vascular health (Prior 1981).

So far, the Kitavans have lots of supposed Western risk factors *but no heart disease*. This suggests that cholesterol is not actually a cause of heart disease, and that saturated fat is not, in itself, dangerous! It cannot be their *low-fat* diet is protecting them, because this is inconsistent with the experience of the Tokelau islanders, and with that of traditional-living Eskimos who were free of heart disease while eating the most fat of any diet ever investigated. What the diets of the Kitava Islanders, the Tokelau Islanders and the Eskimo have in common is a very small amount of refined food, unlike the Western diet.

The surviving hunter-gatherer tribes are also without heart disease:

> Field studies of twentieth century hunter-gathers (HG) showed them to be generally free of the signs and symptoms of cardiovascular disease (CVD). … In this review we have analyzed the 13 known quantitative dietary studies of HG and demonstrate that animal food actually provided the dominant (65%) energy source, while gathered plant foods comprised the remainder (35%). … and a lower omega-6/omega-3 fatty acid ratio, would have served to inhibit the development of CVD. Other dietary characteristics including high intakes of antioxidants, fiber, vitamins and phytochemicals along with a low salt intake may have operated synergistically with lifestyle characteristics (more exercise, less stress and no smoking) to further deter the development of CVD. (Cordain 2002)

Polynesian horticulturalists, East African nomads, Eskimos and Cretans have also been studied and found to have negligible heart disease. All ate predominantly unrefined foods, and some (especially the Cretans) had characteristics such as smoking and "high" cholesterol which, were they Westerners, would put them in the high-risk category.

Significantly, the insulin levels of the Kitava Islanders are *half* those of Swedes living in Sweden (Lindeberg 1999), meaning that the Kitavans are not insulin-resistant and do not develop insulin resistance with age as Westerners do.

The Kitavans demonstrate that our conception of the cause of the diseases which kill us is mistaken.

US Healthcare: Caveat emptor!

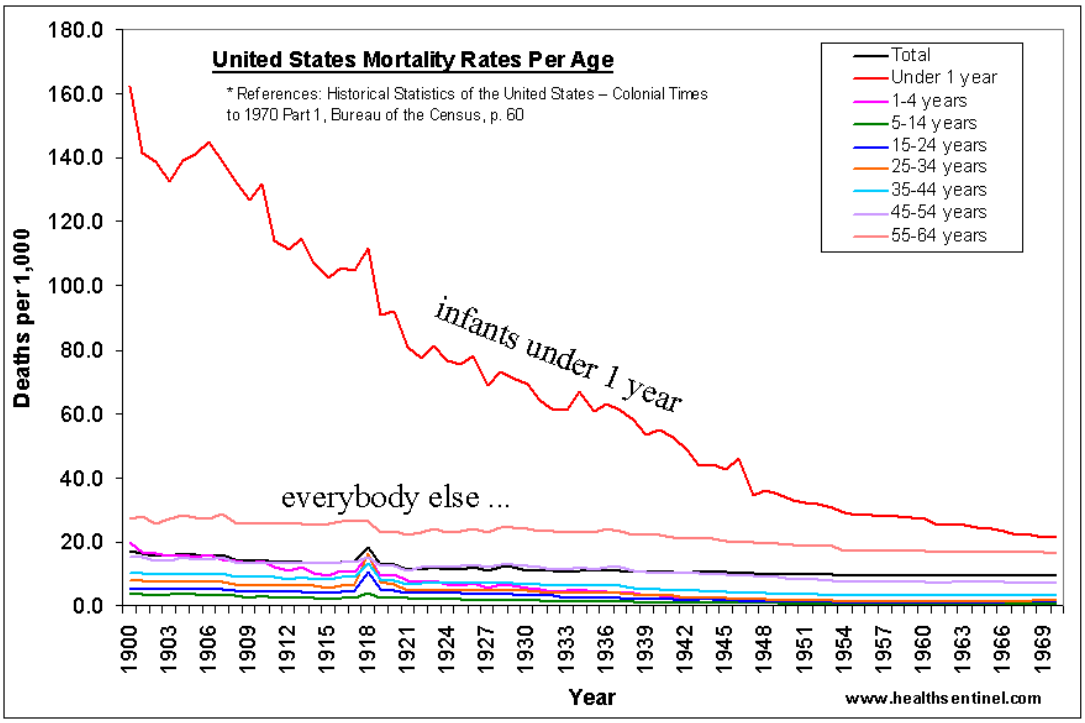
I'm a psychotherapist. Nutrition is not my field, but I started out as an engineer so I applied my engineering skills to this new problem when I developed diabetes at 37 years of age. I knew I had to make careful study of how to avoid the complications this malady. I gained a PhD in Health Principles, and eventually published a book, *Food for Vitality*, on essential fatty acid disturbances in disease. The *low*-carbohydrate solution I found through my researches turned out to be amazingly simple and effective, yet it is diametrically *opposite* to the prescription of the diabetes healthcare system. The price of the diabetologists' bad advice is complications which eventually kill *most* of the diabetic population. And yet the conventional diabetes diet was adopted directly from the American Heart Association diet, on the principle that since diabetics suffer accelerated heart disease, they need a heart-healthy diet. *Unfortunately, the flaws in this diet accelerate both diabetic complications and heart disease.*

When the measures my doctor prescribed made things worse, I researched the question and what I discovered from the scientific literature left me shocked and appalled. For example, I learned that the commonly-accepted idea that cholesterol causes heart disease must be wrong because lowering cholesterol fails to prevent heart disease. This is crystal clear. However, cholesterol is very good business for food manufacturers, drug makers, cholesterol testers, cardiologists, surgeons, hospitals and so on. I also learned that nutritional measures which do alleviate heart disease have been discovered, and rediscovered, over the years, but are simply ignored if they contradict the cholesterol idea. The healthcare system neglects nutrition, and therefore fails those who depend on it.

Most of us believe that America has the most advanced healthcare system in the world, and that we have the best care money can buy. In the narrow arena of the repair of coronary arteries and the removal of cancerous tumors, advances are nothing short of miraculous but elsewhere there is much cause for concern.

Let us look at what we spend, and what we actually get. *Mirabile dictu* – incredible to relate – America spent 1.5 *trillion* dollars on healthcare in 2001. That's $4,887 on each man, woman and child in America, which adds up to 14% of the Gross National Product. To put this figure in perspective, consider that it's by far the highest in the 10 industrialized nations, yet our life expectancy is near the bottom, our infant mortality is the worst, and we have the fewest "quality-adjusted life-years." This means that while the life expectancy of a 65-year-old increased by about 3% in the decade, the expected years of life with "core activity restriction" increased by 34% for women, and by 51% for men. *We're getting sicker younger with each passing generation, in spite of spending more on healthcare than anyone else on Earth.*

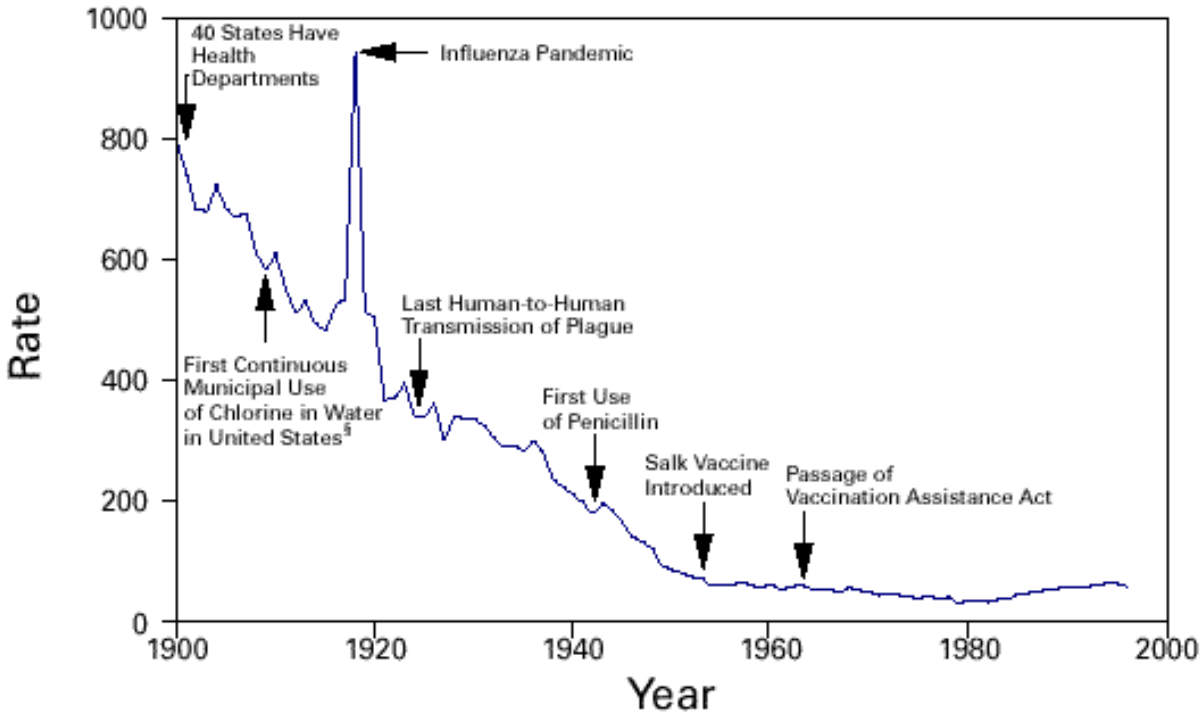
The popular perception that we're living a great deal longer than we did a century ago is quite wrong, a figment of statistics. It is true to say, *statistically speaking*, that life expectancy at birth was 47 years in 1900 and 75 years in the year 2000, but the telling statistic is that the life expectancy of a 65 year old male in 1900 was 12 years (i.e. he'd live to 77), and the life expectancy of a 65 year old male in 2000 was 16 years, an increase of but four years. The key to the puzzle is that infant mortality was 216 per 1,000 births in 1900, and only 6.3 in 2000:



Infant mortality has fallen. (Click on images for large view)

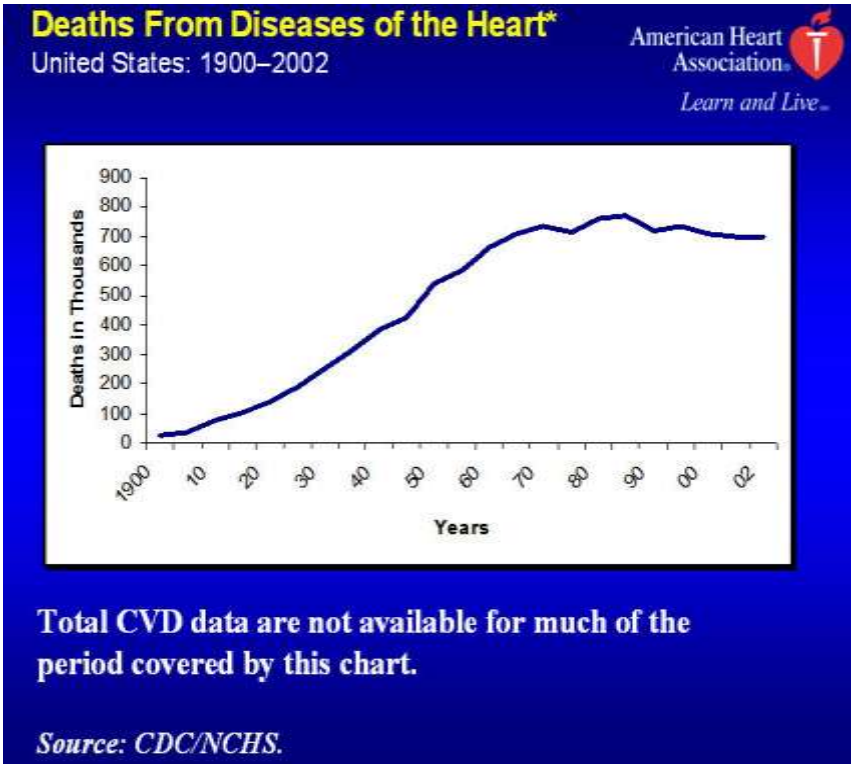
Running water, indoor plumbing and refrigeration controlled the infectious diseases and permitted more babies to survive, but medical advances have improved our circumstances towards the ends of our lives hardly at all. We used to die of infectious diseases, and now we die of heart disease and cancer, so it's really only the *manner* of our passing that has changed:

FIGURE 1. Crude death rate* for infectious diseases — United States, 1900–1996†



*Per 100,000 population per year.
†Adapted from Armstrong GL, Conn LA, Pinner RW. Trends in infectious disease mortality in the United States during the 20th century. JAMA 1999:281;61–6.
§American Water Works Association. Water chlorination principles and practices: AWWA manual M20. Denver, Colorado: American Water Works Association, 1973.

Deaths from infections disease have fallen dramatically. Interestingly, the introduction of antibiotics and vaccines *did not steepen the fall*: in other words, measures such as the chlorination of water, refrigeration and improved sanitation did far more than medicine.



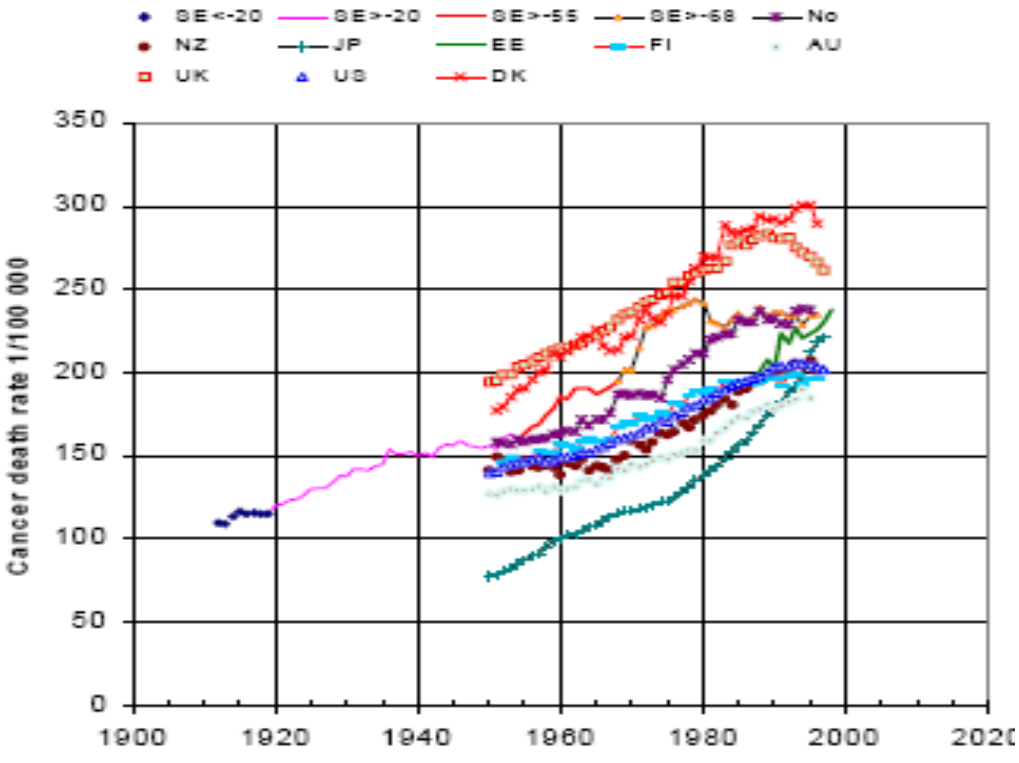


Figure 26. Cancer mortality development in several countries since 1950.

As deaths from infectious diseases fell, deaths from heart disease and cancer have risen. Mortality from heart disease is deceptively low because the figures are *age-adjusted* to the 1940 population, in which there were far fewer older people. There were 1.7 times the number of people aged 65-84 years in 2000 than there were in 1940, which means that the heart-death rate in 2000 was 1.7 times what the graph shows (because most heart deaths take place in this age group.) The 1940 point is accurate at about 300,000 deaths, and the 2000 point

would be about 1,400,000 were the graph not age-adjusted. That there has actually been an *increase* in the incidence of angina corroborates this: angina almost doubled between 1978 and 1995 in a sample of British men aged 55-59 (Lampe 2005).

We know that heart disease is not caused by a cholesterol-lowering drug deficiency, and that cancer is not caused by neglecting to take chemotherapeutic agents. But the truth is that we don't really know what causes heart disease or cancer, and today's ideas don't suggest preventive strategies or effective treatments. *Healthcare is virtually helpless to prevent these epidemics.*

Medical care *itself* is estimated to have caused 783,936 deaths in 2001. Among other things, there were adverse reactions to drugs (106,000), medical errors (98,000), infections acquired in hospitals (88,000), botched surgeries (32,000), unnecessary procedures (37,136), even malnutrition (108,800) and, amazingly, bedsores (115,000), making the healthcare industry itself the *third leading cause of death in America.*

To add insult to injury, according to a Harvard University study of 2001 data, half of all personal bankruptcies in the U.S. were caused at least in part by medical bills, and most of these people had health insurance!

Yet in more "primitive" cultures eating their traditional diets, people who survive childhood infections and accidents die of old age with their faculties intact at pretty much the same age we do. The health surveys are unequivocal, the facts inarguable. Perhaps their immunity from our degenerative diseases is because of the higher nutrient content of their traditional, unrefined diets. This seems to me to be self-evident, but are we using this information to improve our health? We don't use this information to improve our health because we mostly don't know about it, and, what's worse, we don't know we don't know!

There are known knowns ... but there are also unknown unknowns.
The ones we don't know we don't know.
Donald Rumsfeld

The dissemination of this knowledge is hindered at every turn by vested interests, media bias and medical prejudice, and this is made possible by our trusting naïveté.

The doctor of the future will give no medicine, he will interest his patients in the care of the human frame, in diet and the cause and prevention of disease
Thomas Edison

But this promised tomorrow has not come to be, and I see that this is because the forces of misinformation are ascendant. The price is poor health and premature death for millions, a staggering and unimaginable toll of human misery, with chronic pain and minds lost to dementia instead of the productive old age in strong extended families seen in traditional societies.

So the Conventional Wisdom fails

John Kenneth Galbraith coined the term in his 1958 book *The Affluent Society*. According to him, conventional wisdoms are beliefs which "serve the ego", meaning they make us feel good by giving us a sense of belonging to the conservative orthodoxy, confirming we are correctly oriented with regard to the issues of the day. These beliefs are articulated at all levels of sophistication from blue-collar to university professor. With cholesterol, clever men put forward an idea which everybody now clings to as if to a life-belt.

For what a man had rather were true he more readily believes
Francis Bacon

Galbraith wrote that minor disagreements with the conventional wisdom are "much

cherished" and "the very vigor of minor debate makes it possible to exclude as irrelevant, and without seeming to be unscientific or parochial, any challenge to the framework itself", and "… the conventional wisdom often makes vigorous advocacy of originality a substitute for originality itself." But when a serious challenge emerges, "… men react, not infrequently with something akin to religious passion, to the defense of what they have so laboriously learned." In other words, the conventional wisdom is comforting and saves us the effort of thinking for ourselves, but is not necessarily true. "The enemy of the conventional wisdom is not ideas but the march of events … the fatal blow to the conventional wisdom comes when the conventional ideas fail signally to deal with some contingency."

In this case, we see that the Kitavan experience disproves the cholesterol-causes-heart disease idea: they have lousy cholesterol levels and no heart disease whatsoever. The "march of events" in this case is that more than half of us die of heart disease in spite of changing our diets in accordance with the cholesterol idea. One of the attractive features of the cholesterol theory is that everybody thinks they understand it – that LDL-cholesterol is "bad" is self-explanatory. However:

A stupid man's report of what a clever man says can never be accurate, because he unconsciously translates what he hears into something he can understand.
Bertrand Russell

If the cholesterol idea is *not* true, then understanding it is indeed unfortunate for people of any intellectual level. And if the clever men who came up with the cholesterol idea were not entirely pure of motive, then what we may actually be seeing is the greatest marketing coup in the history of Western civilization.

My healthcare misfortunes

When I got back trouble, my doctor prescribed traction in hospital. He wouldn't discuss the chiropractic alternative, and I was surprised at how angry he seemed at the very idea. I'd never seen him angry. In fact, a chiropractor quickly solved my problem, and some time later the American Medical Association was found guilty of violating the Sherman Antitrust Laws by conspiring to destroy the chiropractic profession. In the Permanent Injunction, Judge Getzendanner made it clear that the American Medical Association knew of a study showing chiropractic to be twice as effective at half the cost of medical treatment, and that she felt it necessary to order the Injunction sent to every member of the Association because she had no confidence that the AMA would change its ways unless forced to do so. *My doctor, a good, kind man, was deceived by his own Medical Association in its attempt to eliminate competition*.

(The American Medical Association is) … just another mean trust
Harry S Truman

Later, I got chronic fatigue syndrome. I'd had it before (I was diagnosed with infectious mononucleosis) when I was studying Mechanical Engineering at London University. I spent six weeks in Coppett's Wood Isolation Hospital (to protect me from further infection) because there was (and still is) no known medical treatment. I was exhausted beyond belief for six months and almost lost my place at the University. But by now, I'd read Irwin Stone's *The Healing Factor: Vitamin C Against Disease*, so I asked my doctor about Dr Robert Cathcart's regimen of massive vitamin C intake for infectious diseases. Again, he seemed disappointed, as though the diagnosis had unhinged me and I'd fallen into quackery. I got the sense that dealing with this kind of question was, for him, the most disagreeable part of practicing medicine.

I went ahead anyway because I knew the risk was negligible, and *anything* that might help the aching, grinding exhaustion (seemingly worsened by sleep) was worth trying. I took "mixed mineral ascorbates" because they're easier on the stomach, but I eventually learned that the minerals themselves were important and contributed to my improvement. After three weeks

of very high doses of vitamin C – up to 140 *grams* per day – I was on the road to recovery. I felt it was a miracle. Since then, I've encouraged three other chronic fatigue sufferers to treat themselves, and two of them recovered as I did. (The third person had a normal bowel tolerance for vitamin C of about 10 grams, so her chronic fatigue hadn't been caused by a bug).

But how can it be that my doctor didn't know about this effective, natural anti-viral treatment? After all, Dr Fred Klenner published his findings that intravenous vitamin C cured polio when sufficiently large doses are used as long ago as 1949! (Klenner FR, The Treatment of Poliomylitis and Other Virus Diseases with Vitamin C, *Southern Medicine and Surgery,* 1949; 111(7):209-14). Instead, when two-time Nobel Prize winner Dr Linus Pauling published studies indicating that intravenous vitamin C is an effective anti-cancer agent, Dr Charles G Moertel of the Mayo Clinic refuted his claim with two of the most bizarre studies in the medical literature, which have been interpreted as bunk designed to protect the incredible profits from the so-called chemotherapy concession: "oncologists purchase prescription chemotherapy drugs from their manufacturers and wholesalers, administer the drugs to patients … Government audits have found that profit margins for doctors of 80 to 90 percent are not uncommon in these transactions."

The vast majority of research published since then has supported Pauling's approach, most recently demonstrating the likely mechanism by which intravenous vitamin C is effective against cancer cells (Chen 2005), and providing reports of three cases (Padayatty 1006). It may be no coincidence that Dr Moertel died of cancer aged 66 years, while Dr Pauling took 18 grams of vitamin C per day and died (also of cancer) at 93 years of age.

There are striking examples of facts that have been ignored because the cultural climate was not ready to incorporate them into a consistent theme.
Ilya Prigonine

And then there are facts which are commercially inconvenient. It seems that pharmaceutical companies advance their interests by using advertising dollars to encourage (or the withdrawal of advertising dollars to coerce) medical journals such as the *Journal of the American Medical Association* to de-emphasize research on real results and natural remedies like vitamin C:

Medical journals are no more than an extension of the marketing arm of the pharmaceutical companies.
Dr Richard Smith, editor, British Medical Journal

There seems to be no study too fragmented, no hypothesis too trivial, no literature citation too biased or too egotistical, no [trial] design too warped, no methodology too bungled, no presentation of results too self-serving, no argument too circular, no conclusions too trifling or too unjustified … for a paper to end up in print.
Dr Durmond Rennie, editor, Journal of the American Medical Association

Medical schools are renowned for the paucity of their nutrition courses, and renowned also for their massive dependence on drug company money – let alone the billion dollars a year that the drug companies invest in the continuing education of doctors. As a consequence, my doctor is ignorant and distrustful of natural remedies and unknowingly acts against the interests of his patients. *He is deceived, once again for commercial reasons, but this time by the pharmaceutical industry.*

Then I got insulin-dependent diabetes. This was a big shock, and the more I learned about diabetic complications the more scared I became. The long-time diabetics I met in my new, specialist doctor's waiting room had a smorgasbord of rampant heart disease (in spite of the supposedly heart-healthy diet), kidney failure, fading eyesight and even "salami amputations" made necessary by galloping atherosclerosis in their legs. A study of insulin-dependent diabetics in Pittsburgh found their risk of dying in any particular year was 2%, more than *20 times* the risk of a non-diabetic (Dorman 1984).

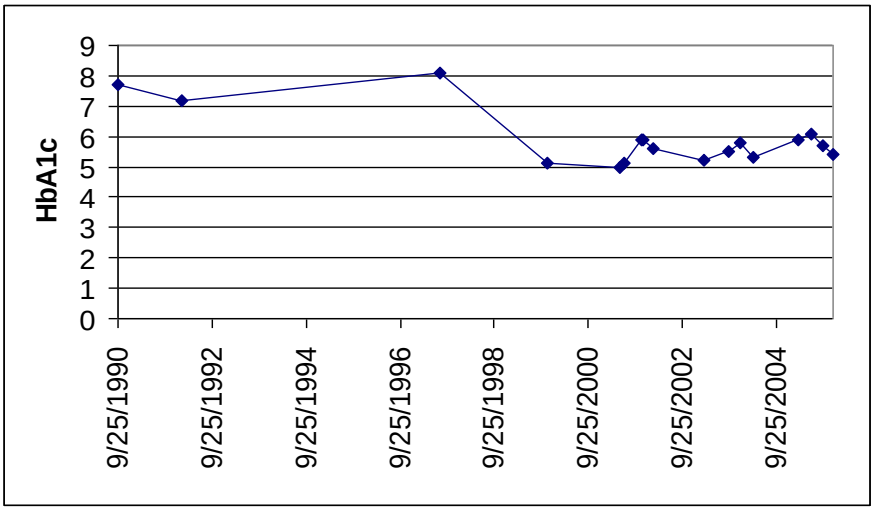
Neither a man, nor a crowd, nor a nation can be trusted to act humanely
or to think sanely under the influence of a great fear.
Bertrand Russell

I learned to inject insulin and, in my fear, I accepted unquestioningly the prescribed (at the time) low-fat high-carbohydrate American Diabetes Association "heart-healthy" diet. I quickly began to suffer brutal hypoglycemic episodes. No matter how often I tested my blood sugar, it would get away from me and I lived in fear that I'd wake up in the emergency room. My HbA_{1c} (*glycosylated hemoglobin*) score, which indicates the risk of diabetic complications, remained obstinately high no matter how hard I worked at the diet. Eventually, I was shocked to realize that my doctor believed the diet wasn't working *because I was cheating*. He was wrong. I was never more serious about anything in my life.

Eventually, I learned of Dr Richard Bernstein. This brilliant diabetic engineer learned by self-experimentation that eating a high-carbohydrate diet is the height of folly when one has to depend on injected insulin because carbohydrates quickly elevate glucose in the bloodstream which insulin acts slowly. Dr Berstein pioneered the idea that good blood-sugar control controls diabetic complications, an idea since validated by the Diabetic Control and Complications clinical trial (DCCT/EDIC). He reversed his own diabetic complications, and he had most of them, by normalizing his blood sugar with a low-carbohydrate diet. Amazingly, he couldn't get the medical profession to listen to him so he qualified as a doctor himself at the age of 49. He now has a diabetes practice in Mamaroneck, New York, and although the mainstream medical profession *still* doesn't listen to him, he has established a strategy for surviving diabetes without complications which he has laid out in his books *Dr Bernstein's Diabetes Solution* and *The Diabetes Diet*.

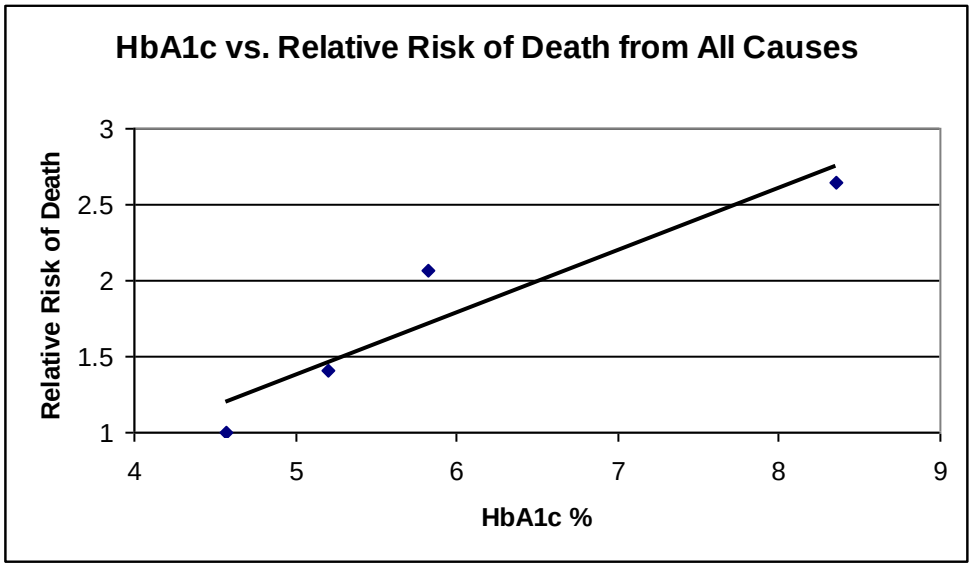
I quickly found that the *low-carbohydrate* diet restored my "hypoglycemic awareness" (insulin-using diabetics often don't know when their blood sugar is falling to dangerous levels),

and put an end to my hypoglycemic episodes. In addition, the new regime and dramatically improved my HbA_{1c} score. The American Diabetes Association's low-fat, high-carbohydrate diet had caused sudden, violent blood-sugar excursions, which I tried to control with slow-acting insulin injections. I might as well have tried to nail jelly to the ceiling.



A graph of my $HbA1_c$ scores before and after I adopted a low-carbohydrate diet

The HbA_{1c} score predicts the likelihood of diabetic complications: "patients … will accrue substantial benefit from almost-normal glycemic control. In patients with later onset, moderate glycemic control prevents most end-stage complications caused by microvascular disease" (Vijan 1997). However, in the Diabetes Control and Complications Trial, the "Intensive Treatment" group ended the trial with an average HbA_{1c} of 7.4%, while the "Conventional Care" group's average was 9.1% (DCCT/EDIC). You can see my HbA_{1c} score was in the Intensive Treatment range, and fell 2 full percentage points after I began to avoid starches. When the HbA_{1c} scores of 4,662 men aged from 45 to 79 years were measured, those who died during the two-to-four years of follow-up were found to have had the highest scores. The researchers calculated that for each 0.1% reduction in HbA_{1c} risk of death fell by 5%, so my low-carbohydrate diet has therefore likely reduced my risk of death by 20% (Khaw 2001):



Recently, the National Institutes of Health funded a ground-breaking study in which a low-carbohydrate diet was shown to normalize the blood sugars of Type 2 Diabetics after just two short weeks. The HbA_{1c} score of the participants averaged 7.3% at the start, and was projected to end at a normal 5.6% had the experiment continued for two months (Boden 2005). This is just as Dr Bernstein suggests in his book, and exactly what I experienced. There's no room left for doubt: the conventional dietary treatment of diabetes guarantees that diabetics, especially insulin-dependent diabetics, will contract life-threatening complications, no matter how hard they try to control their blood sugar.

So, anyway, it came as no surprise to me that key American Diabetes Association donors included General Mills, Kraft Foods, Nestlé, Coca Cola, Hershey Foods and Frito-Lay. The biggest sugar and refined-carbohydrate purveyors on the planet have contrived to encourage those who are least able to handle their products to consume them! My doctor is thrice deceived, this time by food manufacturers. *With the drug companies, the food manufacturers have made my doctor their cat's paw, a person used by another as a dupe or tool.*

I thought I was out of the woods: by avoiding refined foods and starchy vegetables, I

could keep my blood sugar from wandering into dangerous territory, and, in fact, my blood tests were better than ever before with my LDL-cholesterol down to 111. But my doctor pointed out that the American Heart Association guidelines on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults suggests that diabetes be treated aggressively, and that the lower my LDL-cholesterol level, the better. He suggested I adopt the low-fat American Heart Association "Therapeutic Lifestyle Changes" (TLC) Diet to lower my LDL-cholesterol level; and if the diet hasn't achieved this after three months, that I should take a cholesterol-lowering statin drug.

Now I was as frightened of heart disease as I had been of diabetic complications, so frightened that I actually read the entire 284-page report! I was startled to learn that "Modification of blood pressure and lipids in people with diabetes, however, does not reduce CHD risk" (p. II -15). But I quickly recognized the TLC diet as a retread of my old nemesis, the American Diabetes Association diet, with a few minor differences (such as just 10% of calories from refined vegetable oil instead of 22%). This time, as I researched it as a tool to lower my LDL-cholesterol, I could find almost no benefit *except for the manufacturers of the foods and drugs involved.* Furthermore, it became clear from the studies I read that cholesterol isn't even a primary cause of heart disease! But first of all, what is this coronary heart disease of which I'm so at risk?

CHD: Coronary Heart Disease

Coronary heart disease is a frequent complication of diabetes, and diabetics have about 4 times the risk; a male who contracts diabetes early in life may lose up to twelve years of life. A condition which often precedes it is *atherosclerosis* (from the Greek for *porridge* and *stone*), which is found to a greater or lesser extent in the arteries of people all over the world. It may start as early as infancy, with *fatty streaks* forming at points in the arteries subject to the greatest mechanical stresses from turbulent blood flow, or from distension from the blood-pressure pulses. Although some 35% of people have "clinically significant" atherosclerosis, others are entirely free of it (Enriquez-Sarano 1996).

Atherosclerosis was found in Egyptian mummies dating from 100BC, and was first described at autopsy by Leonardo da Vinci in about 1520. The 17th Century English physician Thomas Sydenham wrote that "A man is as old as his arteries" because atherosclerosis eventually constricts the coronary arteries so that the blood supply to the heart muscle is inadequate, causing the agonizing pain of *angina*. However, according to the AHA, "only 20% of coronary attacks are preceded by long-standing angina." Interestingly, the first *heart attack* to be described in the medical literature was in 1912. In a heart attack, a *blood clot* may block an artery narrowed by atherosclerosis, cutting off the blood supply to the heart muscle and resulting in *myocardial infarction* (which is a heart attack). *Unstable plaque* (inflamed atherosclerotic plaque) is often found in those who eat a Western diet, and should it rupture, the plaque contents cause a massive clot and an immediate heart attack (Forrester 2002).

Interestingly, coronary heart disease doesn't always involve atherosclerosis. One in five heart victims have clean arteries, and these deaths are thought to be caused by *spasms of the coronary artery* or some form of *arrhythmia*, in which the heartbeat becomes uncoordinated and the heart fails to pump blood. So "*Coronary Heart Disease*" includes angina, heart attack and sudden heart death, and is included in the more general term "*Cardiovascular Disease"* which itself includes high blood pressure, *cardiomyopathy* (in which the heart swells and pumps inefficiently), stroke and various other circulatory problems. The incidence of heart disease increases with age, and is truly epidemic: by 75 years of age, about 78% of men and 86% of women have cardiovascular disease, and 17% of men and 10% of women have CHD.

Clearly, several disease processes at work. For starters, there's whatever causes fatty streaks and *atheroma* (porridge before it turns to stone), then there's whatever causes calcium to enter the atheroma to cause atherosclerosis, whatever causes the blood to clot, whatever causes arrhythmias, and whatever causes arterial spasms. As we shall see, nutritional factors are strong modifiers of all these disease processes.

However, it is generally believed that high blood cholesterol is bad for the heart, LDL-cholesterol particularly so. We are told, and we believe, that we are helping ourselves by lowering cholesterol and saturated fat in what we eat. The American Heart Association has made the Therapeutic Lifestyle Changes Diet and statin cholesterol-lowering drugs cornerstones of their strategy for lessening risk of CHD by lowering "bad" LDL-cholesterol. The TLC diet lowers cholesterol and saturated fat in the diet in order to lower cholesterol and LDL-cholesterol in the bloodstream. But I quickly found that almost every tenet of the Therapeutic Lifestyle Changes Diet is speculative. First of all, cholesterol in the diet has little to do with cholesterol in the bloodstream!

Lowering dietary cholesterol hardly affects cholesterol in the bloodstream

Even Dr Ancel Keys, who wrote the mammoth *Seven Countries Study* which

investigated heart disease risk factors in seven countries and started the whole cholesterol scare, didn't believe dietary cholesterol was important. He wrote:

> In the adult man the serum cholesterol level is essentially independent of the cholesterol intake over the whole range of human diets (1956). There's no connection whatsoever between cholesterol in food and cholesterol in blood. And we've known that all along. Cholesterol in the diet doesn't matter at all unless you happen to be a chicken or a rabbit (1997). I've come to think cholesterol is not as important as we used to think it was (1987).

Few know that the diet-cholesterol question was studied in the Framingham Heart Study before the cholesterol spin-doctors took over. At 22 years, the results showed that the average blood levels of cholesterol were essentially the same in men and women consuming less than and more than the average intake. This means, in the words of the researchers, that "There is considerable range of cholesterol levels within the Framingham study group. Something explains this inter-individual variation, but it is not diet":

		Blood cholesterol (mg/dL)	in those consuming:-
	Average Cholesterol Intake, mg/day	Less than the average:	More than the average:
Men	704	240	240
Women	492	248	244

(Kannel, William B, Gordon, Tavia, The Framingham Diet Study: Diet and the regulation of serum cholesterol.
In *The Framingham Study: An Epidemiological Investigation of Cardiovascular Disease. Section 24*. US Government Printing Office, Washington, D.C., 1970)

Interestingly, data from the same source compared cholesterol intakes of healthy Framingham subjects and those with CHD:

Cholesterol intake (mg/day) of:	Healthy subjects	Patients with CHD
Men	716	708
Women	477	520

Their conclusion was the only conclusion possible from this data: "There is, in short, no

suggestion of any relation between diet and the subsequent development of CHD in the study group."

Lowering dietary cholesterol hardly affects "bad" LDL-cholesterol in the bloodstream

We know this because it took a *meta-analysis* of locked-ward metabolic studies to show that cholesterol in the diet raises "bad" LDL-cholesterol because the effect is small:

> Dietary cholesterol causes marked hypercholesterolemia in many laboratory animals, including non-human primates. High intakes of cholesterol in humans, however, do not cause such a marked increase in serum cholesterol. Nonetheless, controlled metabolic studies in humans indicate that high cholesterol intakes raise LDL cholesterol. The degree of rise varies from person to person as is true for all nutrients. ***Meta-analysis of studies done in controlled settings confirm the LDL-raising action of dietary cholesterol.*** (652, 653). (page V-9 of the 3rd Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults)

In fact, although there seems to be a relationship when different countries are compared, the effect has not been found in within-population studies. As is so typical in the cholesterol-heart disease field, this finding was blamed on confounding factors, and dismissed (Jacobs 1979). Meta-analyses are used when effects are so small that huge numbers of subjects are necessary for a study to achieve statistical significance. The first study says that if I replace 60% of saturated fats with other fats, and avoid 60% of dietary cholesterol, I may lower my LDL-cholesterol by 8 to 12% (Clarke 1997). This is a draconian, near-vegetarian prescription for a small benefit! The second study concludes that "People desiring maximum reduction of serum cholesterol by dietary means may have to reduce their dietary cholesterol to minimal levels (less than 100-150mg/day) to observe modest serum cholesterol reductions …." (Hopkins 1992).

I see that the National Cholesterol Education Committee has told a half-truth. I can expect little further lowering of my already-low LDL-cholesterol by restricting my cholesterol intake because the TLC diet advocates only about half of the reduction in cholesterol which was

found to be effective in the locked-ward studies.

Lowering saturated fat hardly affects cholesterol in the bloodstream either

In the Tecumsheh study of 957 free-living adults in Tecumseh, Michigan, those with the lowest cholesterol levels and those with the highest cholesterol levels were found to eat the same amount of saturated fat, so the TLC diet with its lesser amount of saturated fat will likely not lower my blood cholesterol by much.

Most importantly, lowering cholesterol in the bloodstream doesn't lower CHD!

Incredibly, an analysis of 22 cholesterol-lowering studies prior to the advent of statins, in which 114,000 people participated, showed almost no change! There were only 0.3% fewer nonfatal heart attacks, the same number of fatal heart attacks, and, worryingly, 0.3% *more* deaths from all causes in the treatment groups whose cholesterol was lowered:

	Treatment groups	Control groups	Change
Nonfatal heart attacks	2.8%	3.1%	-0.3%
Fatal heart attacks	2.9%	2.9%	0
Total deaths	6.1%	5.8%	***+0.3%***

(Ravnskov 1992)

Cholesterol is a lousy risk factor

And cholesterol in the bloodstream is a very poor predictor of heart risk. An analysis of the Framingham results published in 1980 showed that the 10% of the population with the highest risk according to the cholesterol measures experienced *less than 25% of the CHD* (Orchard 1980). If cholesterol was as dangerous as it has been represented to be, this percentage would be far higher. The curves for the distribution of cholesterol in those with and without CHD overlap so closely that it is obvious that cholesterol's predictive power is weak (Ebrahim 1998):

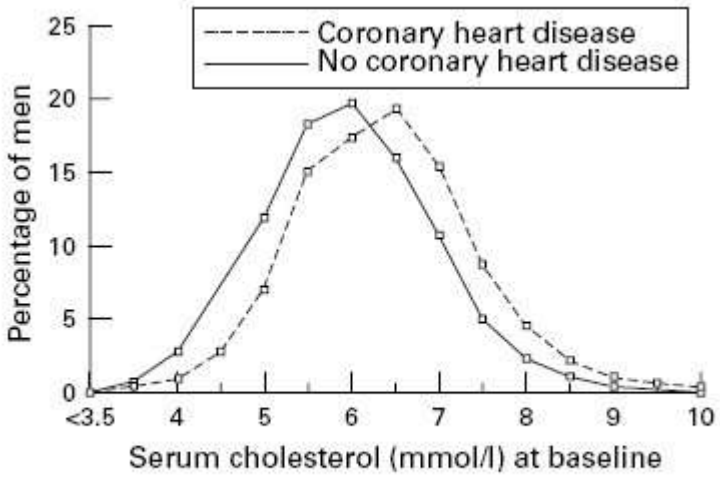


Figure 2 Blood cholesterol in British men aged 40–59 and coronary heart disease events.

In fact, only 42% of these British men who had a blood cholesterol level above 6.5 mmol/l (253 mg/dl) went on to have heart trouble in the next 15 years. They might as well have tossed a coin – in fact, tossing a coin would have "predicted" 50% of the men at risk!

Lowering "bad" LDL-cholesterol doesn't necessarily lower heart risk

Suppose the TLC Diet did work and lowered my LDL-cholesterol, will it lower my risk of CHD? A study of an aging Italian population has shown that risk of both heart death and all-cause mortality actually *increased* dramatically as LDL-cholesterol fell:

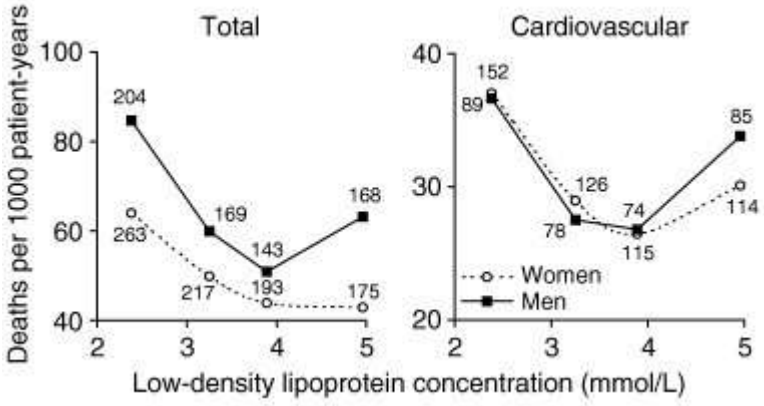


Figure 1. Sex-specific and age-adjusted rates of total and cardiovascular mortality by quartiles of serum low-density lipoprotein cholesterol at baseline. The number of deaths is given for each quartile. Conversion factor to conventional units is 38.6 (Tikhonoff 2005).

Apparently, an LDL-cholesterol level of about 154mg% (4mmol/L in the units of the figure) conferred a considerable survival advantage over the supposedly more desirable under-100mg% (2.6mmol/L) level. This seems to fly in face of conventional wisdom, but when Dr Uffe Ravsnkov (Ravnskov 2000) actually read the references of the National Research Council's massive Diet and Health: Implications for Reducing Chronic Disease Risk report, which established LDL-cholesterol as a risk factor for heart disease, he found the voluminous literature relies, at bottom, on a single study of the Framingham population which concluded that: "Under age 50 years, [LDL-]cholesterol levels are directly related with 30-year overall and CVD mortality … after age 50 years there is no increased overall mortality with either high or low serum cholesterol levels (Kannel 1979). But I'm 60 years of age, so my LDL-cholesterol level is beside the point!

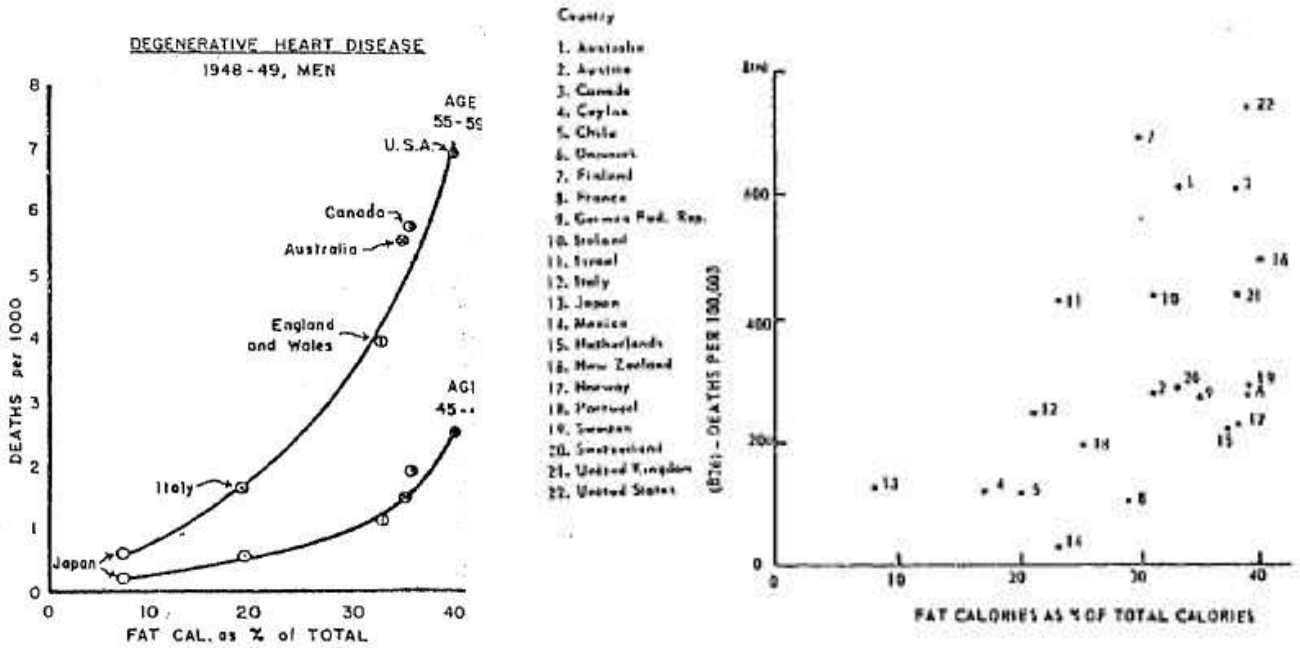
So the extraordinary conclusion is forced upon me:

Cholesterol doesn't cause heart disease!

For every complicated problem there is a solution that is simple, direct, understandable, and wrong
HL Menken

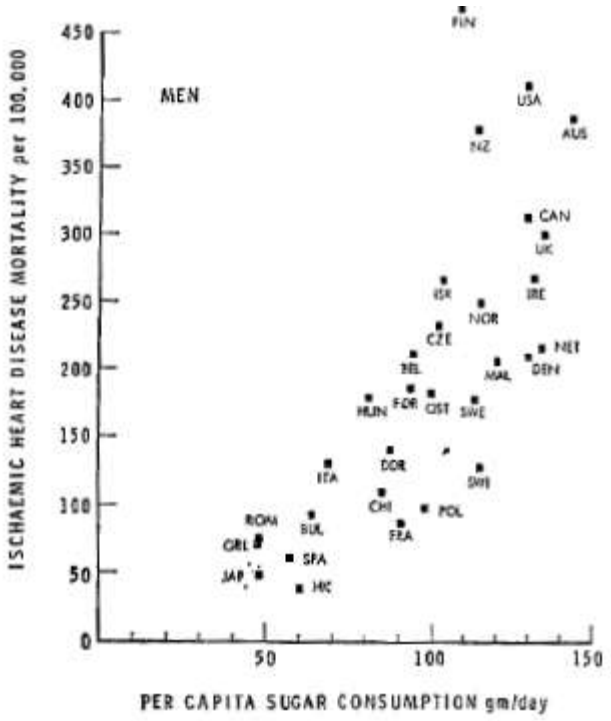
There is no nonsense too arrant to become policy with sufficient interference by the government
Bertrand Russel

I was so intrigued that I researched the matter further, and found that the low-fat preoccupation was started by Dr Ancel Keys. Keys was a prolific researcher, and we have him to thank for K-rations, the first scientifically-formulated provisions for the American fighting man. In researching the causes of heart disease, Keys demonized all fats at first, then saturated fat. His research was incredibly sloppy. For example, he cherry-picked among the available data to make a convincing graph, which needless to say is a deeply bogus practice:



The left graph is from Keys, showing data from 6 countries which promise a close relationship between fat in the diet and heart deaths. The right graph is from contemporary data for 22 countries, and shows that Keys cherry-picked his data to make his graph more convincing (Yerushalmy 1957). You don't need statistics to see the Key's persuasive relationship disappears when the data for all 22 countries are plotted, or that picking (say) Japan, Ceylon, Chile and France would seem to show that fat protects against heart death.

Keys almost certainly knew from his own investigations that although between-population studies show an association between fat and CHD mortality, within-population studies generally do not (Khor 2004). This implies that it's not fat that causes CHD mortality, but rather some fellow-traveler such as, for example, the degree of refinement of the diets. In fact, sugar consumption correlates very closely indeed with fat consumption in 30 different countries, and the correlation between heart deaths and sugar consumption has actually been found to be stronger than that between fat and heart deaths. The authors of the study from which the graph below of sugar consumption vs. heart deaths concluded "These results suggest that associations identified in this type of investigation should be interpreted with great caution and need not necessarily reflect causal relationships, but rather suggest avenues along which further research might proceed" (Armstrong 1975):



Sugar consumption in 30 countries plotted against the heart-attack death rates. The relationship is obviously stronger than that between fat and heart death (compare Key's graph above), yet Keys dismissed it with scorn: " … the correlation between sugar and CHD does not remotely approach significance" (Keys 1973).

It is long yet vigorous, like the penis of a jackass
Sydney Smith

The Reverend Sydney Smith was talking about the quality of his sermon, but the description fits Key's writings: one does not get the chance to be won over by the persuasiveness of the argument, but is rather browbeaten into submission. Few know that Keys was on the American Heart Association nutrition advisory committee, or that grants from the American Heart Association Minnesota Affiliate paid for much of his research. Dr Key's low-fat, low-cholesterol dietary prescription became the official AHA dietary guidelines in 1961. I imagine that the farmers of Minnesota and the refined food manufacturers chortled with glee, for patterns of consumption changed dramatically in their favor. People began to avoid fat in favor of refined carbohydrates:

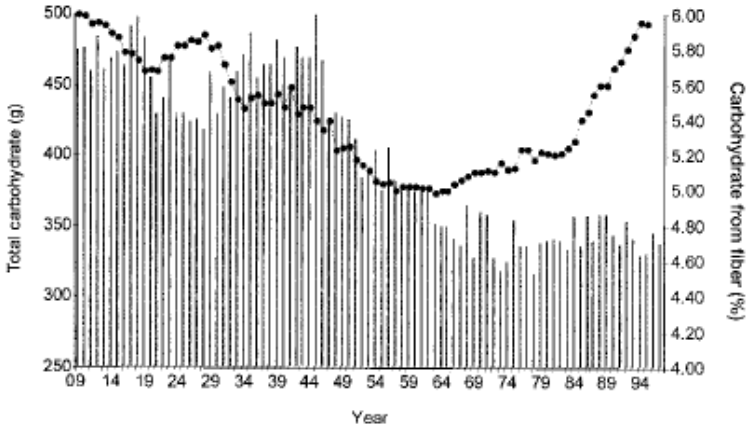


FIGURE 1. Change in total carbohydrate consumption (●) and the percentage of carbohydrate from fiber (vertical bars) in the United States between 1909 and 1997 (17).

Since 1963, the consumption of carbohydrates steadily increased back to 500 g/d; however, fiber consumption did not increase proportionately. This finding reflects an increased consumption of refined carbohydrates over this time period (Gross 2004).

My conclusion? The Therapeutic Lifestyle Changes Diet would be useless for me. I believe it would likely be useless for anybody else, unless they ate nothing but fast foods. This is so reminiscent of my attempt to use the very similar American Diabetes Association diet to help my diabetic control: it *worsened* my blood sugar control because it had too many carbohydrates. Substituting *fat* for the starchy vegetables and whole grains in my diet returned my HbA_{1c} test result to the normal range, indicating my risk for diabetic complications is at a minimum. And as an insulin-dependent diabetic, I simply don't have a choice about eating fat; the calories have to come from somewhere, so I can't be both low-fat and low-carbohydrate! Since carbohydrates send my blood sugar into orbit, I eat eggs and snack on almonds and Brazil nuts instead, and the consequence is that my cholesterol risk factors are better than they have ever been in my life. My experience utterly refutes the conventional prescription in diabetes management, and the scientific literature suggests that the cholesterol hypothesis is a porky.

But why don't I use refined vegetable oils and avoid saturated fats? Because studies show that this approach has risks of its own. In the UCLA Veterans Administration trial, saturated fat was replaced by polyunsaturated soybean oil in a 40% calories-from-fat diet and there was a

small decrease in the number of deaths from heart disease, *but this decrease was completely offset by a robust, 15% increase in deaths from cancer in the treatment group* (Dayton 1969). This is not a rogue result, for an analysis of 8 trials employing this strategy (including some low-fat diet trials) shows that *it carries with it a 0.1% absolute increased risk of death,* meaning that it is at least ineffectual, and probably dangerous (Ravnskov 2003). And, amazingly:

Saturated fat is protective!

Even in Key's time, there was data which suggested that saturated fat rendered some benefits. Before dismissing saturated fat as a dietary fiend with no redeeming features, it's necessary to explain the high rate of stroke in Japan, where the saturated fat consumption is among the lowest in the world:

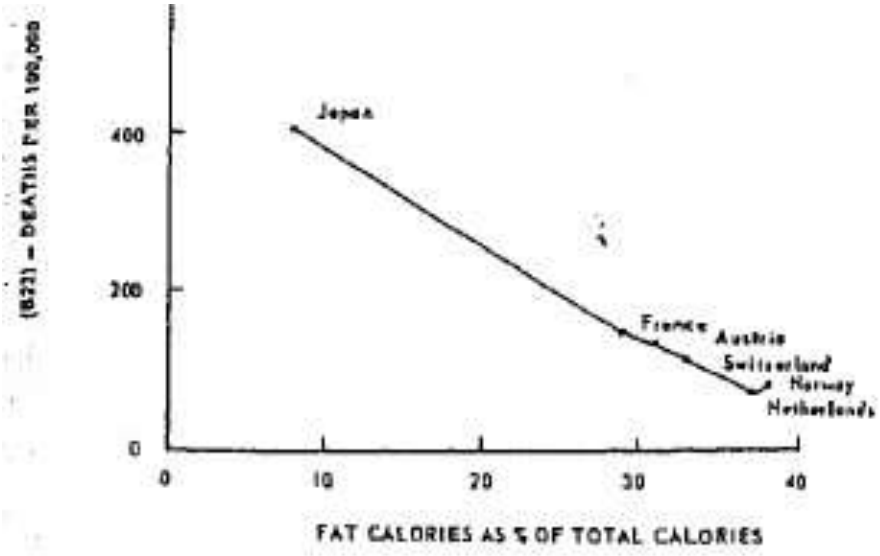


FIG. 2. Mortality from vascular lesions affecting the central nervous system (B-22) and fat calories as per cent of total calories in males fifty-five to fifty-nine

Saturated fat (or something else) protects the peoples of France, Austria, Switzerland, Norway and the Netherlands from stroke, while Japan eats very little and has a high rate of stroke (Yerushalmy 1957).

The relative risk of stroke between the highest and lowest quintiles of saturated fat intake in a 20-year follow-up of the male Framingham population was 0.43, a "robust association" according to the researchers (Gillman 1997). The increment of relative risk of heart problems

between the highest and lowest quintiles of saturated fat intake was only 1.03 in this study. The jargon means that those who ate the most saturated fat in Framingham were at 57% less risk of stroke and had a 3% increased risk of heart problems.

Men of Japanese descent living in Hawaii experienced similar outcomes and the researchers concluded with this ringing endorsement of the lipid-heart hypothesis: "This increased risk [of eating a diet low in fat], due to an excess risk of death from stroke and cancer, indicates that there is no overall beneficial effect from a low fat diet in this cohort" (McGee 1985).

More recently, saturated fat in the diet was found to protect post-menopausal women against progression of their atherosclerosis. The study concluded that "In postmenopausal women with relatively low total fat intake, a greater saturated fat intake is associated with *less progression of coronary atherosclerosis*, whereas *carbohydrate intake is associated with a greater progression*" (Mozaffarian 2004). In this study, *sugars* (carbohydrates) were found to be dangerous, and *saturated fats* were found to be protective!

This raises doubt in my mind about the population studies in which saturated fat intake appears to elevate the risk of heart disease, for most were not controlled for carbohydrate intake. In one such oft-quoted investigation of saturated-fat intake in the Nurses Health Study, fiber intake (which is lowered by sugar intake, and raised by fruit and vegetable intake) was 17 grams per day in the lowest-risk group and 10 grams in the highest-risk group (Hu 1999). But in this study, no distinction is made between "carbohydrates" and "refined carbohydrates", and low fiber implies a high intake of refined carbohydrates which are low in fiber. So was it the saturated fat that raised their risk of CHD, or was it sugar? Another study which compared the diets of lean and obese individuals concluded that "Obesity is maintained primarily by a diet that is high in fat and added sugar and relatively low in fiber" (Miller 1994).

There is, in fact, an immense body of evidence to suggest that saturated fat in the diet is,

at the least, not harmful:

To date, some 26 long-term follow-up studies, ranging in length from 4 to 23 years, have examined the relationship between saturated fat intake and cardiovascular disease.[1-26] We are constantly told that saturated fat is a toxic "artery-clogger", yet only four of these studies have managed to detect even desperately weak statistical associations between saturated fat and CHD/CVD mortality. One study observed a protective relationship, while all the rest found no association at all.

Even more importantly, controlled clinical trials – which represent far more reliable evidence than confounder-prone epidemiological studies – have completely failed to show any CVD or total mortality benefit for individuals randomized to saturated fat restricted diets (Anthony Colpo, TheOmnivore.com 2005).

In the reference supporting the last assertion, Dr MF Oliver wrote that "The commonly-held belief that the best diet for prevention of coronary heart disease is a low-saturated fat, low-cholesterol diet is not supported by the available evidence from clinical trials. In primary prevention, such diets do not reduce the risk of myocardial infarction or coronary or all-cause mortality (Oliver 1997).

Worse still, I learn that:

Low blood cholesterol is *itself* not without risk

Other than that, Mrs. Lincoln, how did you enjoy the play?

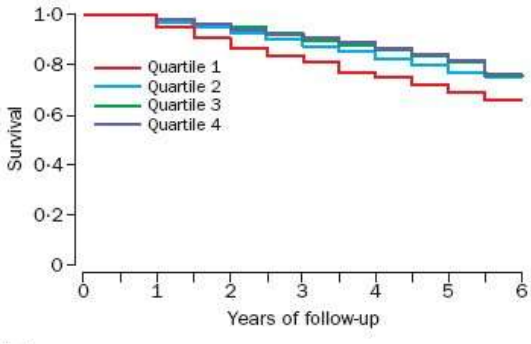
Low cholesterol is associated with a much higher risk of violent death and suicide, so much so that six studies in which cholesterol was lowered and deaths from heart disease reduced showed no change in the death rate from all causes between the treatment and control groups because of increased death by violence and suicide. The study concluded that "The association between reduction of cholesterol concentrations and deaths not related to illness warrants further investigation. Additionally, the failure of cholesterol lowering to affect overall survival justifies a more cautious appraisal of the probable benefits of reducing cholesterol concentrations in the

general population" (Muldoon 1990).

Even more disquieting is the outcome of a huge study of 149,650 men and women which concluded that:

In men, across the entire age range, although of borderline significance under the age of 50, and in women from the age of 50 onward only, *low* cholesterol was significantly associated with all-cause mortality, showing significant associations with death through cancer, liver diseases, and mental diseases. (Ulmer 2004)

Studies suggesting a link between low cholesterol and all-cause mortality are thick on the ground. In New Zealand Maoris, low cholesterol predicted death, raising the relative risk of death by 2.3 in men and 1.9 in women (Beaglehole 1980), and in Korean men, "The cholesterol level associated with the lowest mortality ranged from 211 to 251 mg/100ml …" (Song 2000), well above the American Heart Association's upper limit of normal, which is 200mg%. After age 72, the Honolulu Heart Program study suggests that low cholesterol is associated with *increased* risk of death from all causes (Schatz 2001):



Quartile 1	1	0·95	0·91	0·87	0·84	0·81	0·77	0·75	0·72	0·69	0·66	0·66
Quartile 2	1	0·97	0·95	0·93	0·90	0·88	0·85	0·82	0·80	0·77	0·76	0·76
Quartile 3	1	0·98	0·96	0·95	0·93	0·90	0·88	0·86	0·84	0·81	0·75	0·75
Quartile 4	1	0·98	0·96	0·94	0·92	0·91	0·89	0·87	0·84	0·82	0·76	0·76

Figure 2: **Probability of mortality by quartiles of serum cholesterol**
Kaplan-Meier survival curves for 5-year all-cause mortality in association with quartiles of serum cholesterol at examination 4 (1991–93). Wilcoxon Log-Rank test p<0·0001.

Quartile 1 had the *lowest* ave
and the *highest* mortality:
Quartile 1: 149mg%
Quartile 2: 178mg%
Quartile 3: 199mg%
Quartile 4: 232mg%
And Quartile 4 had the *highe*
and the *lowest* mortality! The dose-r
relationship is present in each quarti
which suggests that the findings are

The red line represents the group with the lowest average cholesterol, 149mg%. This group had the lowest probability of survival at all times during the 6 years of follow-up, indicating that, after age 72, *higher* cholesterol is protective!

One reason may be that low cholesterol is associated with low immunity. This is a

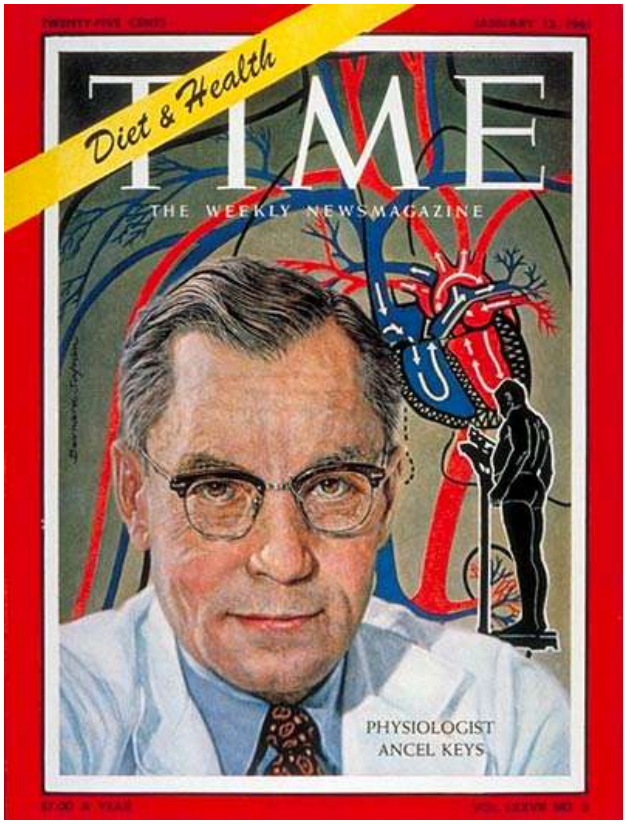
robust effect which has been found in any number of studies, although it is usually dismissed as an artifact:

Men occasionally stumble over the truth, but most of them pick themselves up and hurry off as if nothing ever happened.
Winston Churchill

For example, a 15-year Kaiser Permanente study of 61,827 patients found "an inverse association … between total cholesterol and incidence of infections either requiring hospitalization or acquired in the hospital" (Irribarren 1998).

Triglycerides and VLDL-cholesterol: the plot thickens

In the confusing maze of blood fats, there *is* a pattern which is associated with increased risk of heart disease: elevated *triglycerides* raise the relative risk of CHD considerably. "… an 88 mg/dl (1.0 mmol/L) increase in plasma triglyceride levels significantly increased the relative risk of cardiovascular disease by approximately 30% in men and 75% in women" (Cullen 2001). It is by now generally accepted that triglycerides are elevated by refined carbohydrates in the diet (Parks 2000), and that fasting triglycerides are a risk factor for heart disease. Ancel Keys, wrong in this as in so much else, heatedly dismissed these finding when they first appeared (Keys 1963). Right for the wrong reasons, he adopted the Cretan diet, retired to Tuscany and died in 2004 at 100 years of age (VanItallie 2005). Irritatingly, his flawed ideas live on, causing misery and premature death the world over.



High triglycerides combined with low HDL-cholesterol were found in heart-attack survivors, showing that "the ratio of triglycerides to HDL was a strong predictor of myocardial infarction … RR in the highest compared with the lowest quartile=16.0 …" (Gaziano 1997). Dr Gaziano is saying that our relative risk of heart attack is 16 times greater if we have both high triglycerides *and* low HDL-cholesterol. This pattern of blood fats is associated with insulin resistance.

It may help to understand how these different blood fats are related:

Total cholesterol = LDL-cholesterol + HDL-cholesterol + (triglycerides ÷ 5)

Triglycerides are themselves one-fifth cholesterol! LDL-cholesterol is actually a triglyceride- and cholesterol-delivery system, and LDL-cholesterol is what remains after *very*-low-density cholesterol, VLDL-cholesterol which is made in the liver, releases most of its triglycerides. This confusing naming system complicates matters until one is ready to throw up

one's hands – but to do this is to trust one's health to people who don't have our best interests at heart, so to speak. The point here is that elevated triglycerides, especially when combined with low HDL-cholesterol, pose an exceedingly serious threat to heart health, apparently considerably greater that any form of cholesterol, assuming that cholesterol is, in truth, any threat at all.

A further threat, one which is increased considerably by low-fat diets containing refined carbohydrates, is posed by *remnant lipoproteins,* which are what's left after the triglyceride part of the lipoprotein has been taken up by cells (Jialal 2002). These so-called "small, dense" LDL remnants are elevated by high carbohydrate, low fat diets:

> These results indicate that the effects of low-fat diets on lipoprotein metabolism are not limited to higher fasting plasma triglyceride and lower HDL cholesterol concentrations, but also include a persistent elevation in "remnant lipoproteins". Given the atherogenic potential of these changes in lipoprotein metabolism, it seems appropriate to question the wisdom of recommending that all Americans should replace dietary saturated fat with CHO [carbohydrates] (Abbasi 2000).

It's worth repeating: low-fat diets containing refined carbohydrates worsen atherogenic remnant lipoproteins. Dr Abbasi's study compared low-fat diets (like the American Heart Association Therapeutic Lifestyle Changes Diet, which is prescribed to lower high cholesterol) with low-carbohydrate diets (like the Atkins diet). The main finding was that the low-fat diet actually *elevated* risk of heart disease. Oddly, we don't hear much about this pattern of increased heart risk from low-fat diets, although, as we shall see, it is a consistent finding in studies which use refined foods among their carbohydrate sources. Remnant lipoproteins are violently atherogenic (Koba 2006) unless quickly removed from the circulation by the liver, and what's important about this study is that it points out that carbohydrate in the diet causes a *persistent elevation* of these small, dense atherogenic remnant lipoproteins.

There is a profoundly important distinction here between *simple* carbohydrates, which are sugars, and *starchy* (complex) carbohydrates from, for example, whole grains. Starches are

actually long chains of glucose molecules, and most starches are quite resistant to digestion so that their glucose enters the bloodstream more slowly than sugars. It's quite clear that simple carbohydrates elevate triglycerides and small, dense LDL-cholesterol and lower HDL-cholesterol (Parks 2000), and that this pattern of fat in the blood is a virulent risk factor for heart disease (Wilson 2005). Starchy carbohydrates simply do not cause these blood-lipid changes at any level of consumption, no doubt in part because they come with the full complement of nutrients such as magnesium which are well-known to be protective.

It looks like "three strikes" for sugars. It's worth repeating here, to underline the point, that a prospective study has found that the pattern of high triglycerides and low HDL-cholesterol (which can be caused by eating refined carbohydrates) confers a remarkable *16 times* greater risk of heart attack (Abbasi 2000)! Interestingly, one way a sugary diet worsens this disturbance in the blood fats is via alterations of mineral levels within cells, which are also associated with insulin resistance. Now this is a worthwhile observation, for, as we shall see, there are simple dietary remedies. But I'm getting ahead of myself.

What the literature *really* shows

In the first actual clinical test of the low-fat diet (the Therapeutic Lifestyle Changes Diet is a low-fat diet) ever performed in its thirty-plus years, *no benefit was found.* This $415,000,000 study (funded with tax dollars) revealed that, after eight years:

> The intervention was associated with increased risk in the 3.4% of women with baseline CVD [cardiovascular disease] … In conclusion, this long-term dietary intervention in postmenopausal women, intended to reduce fat intake and increase intake of vegetables, fruits, and grains, achieved an 8.2% of energy decrease in total fat intake but only a 2.9% of energy decrease in saturated fat intake and only modest increases in intakes of vegetables, fruits, and grains. ***The intervention did not reduce risk of CHD or stroke*** (Howard 2006).

In other words, the low-fat diet my doctor has prescribed for me neither increased nor decreased the risk of heart disease among most of the study participants, but for the 3.4 percent

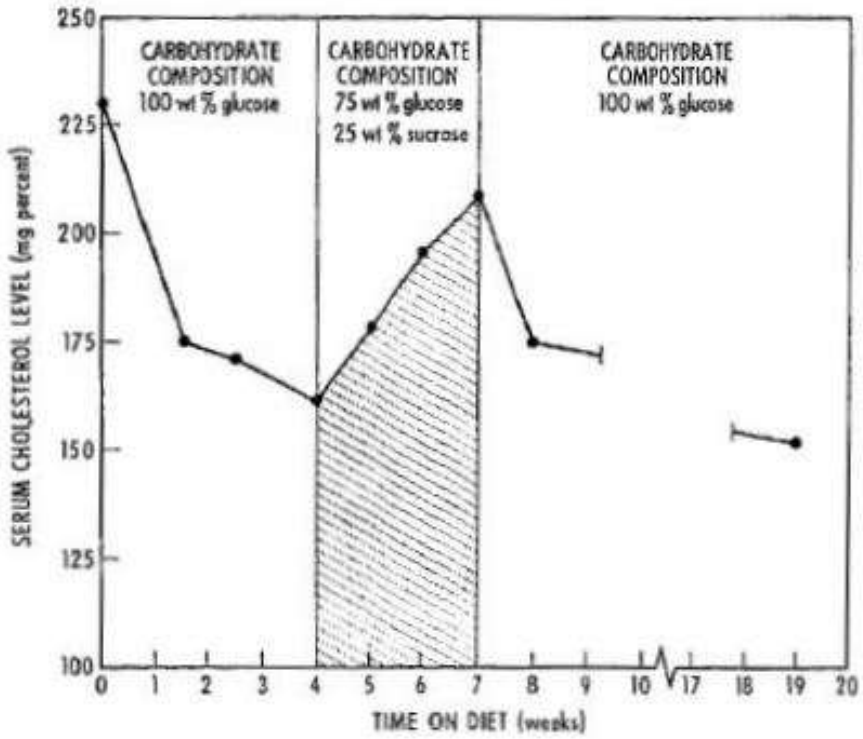
of trial participants with pre-existing cardiovascular disease, the relative risk of non-fatal and fatal CHD was actually *increased* by 26%!

Despite of the slick PR of the American Heart Association, there are many in the scientific community who simply do not subscribe to the cholesterol hypothesis. One such is Dr Uffe Ravnskov, an independent researcher who wrote *The Cholesterol Myths*. How is it that the world believes that cholesterol kills? Because the American Heart Association and the National Heart Lung and Blood Institute (one of the National Institutes of Health) between them fund over 90% of heart research, and they fund cholesterol research almost exclusively. Instead of dying a natural death, as defective hypotheses must for science to progress, cholesterol and its researchers are propped up on the life-support of practically unlimited funds! Science is very much like evolution in the sense that science depends on the survival of the fittest hypothesis. If defective hypotheses are favored for commercial reasons over fitter hypotheses, some may achieve commercial success but provide little in the way of useful strategies for preventing heart disease. This is to say that the cholesterol hypothesis is 100% wrong, but it lives because it sells a phenomenal amount of sugar-fortified low-fat foods and statin drugs.

What raises cholesterol?

Studies through the years have revealed what really causes high cholesterol: sugar, hypothyroidism, stress, and nutrient deficiencies.

A study funded by NASA to discover the best diet for astronauts found that sugar (but not glucose) profoundly elevated serum cholesterol:



Twenty-four felons aged from 24 to 43 years in the California prison system at Vacaville were fed a low-fat, 14% protein, chemically-defined diet containing carbohydrate calories either from 100% glucose, or 75% glucose with 25% sucrose. The decreases in serum cholesterol levels at the end of the first 4 weeks on the 100% glucose diet ranged from 9 to 49% of the baseline values, an average of 76mg%, and a significant change in the opposite direction occurred when the sucrose-containing diet resumed. There was very little fat in the diet, so that the experiment "unequivocally demonstrates an important relationship between the nature of the dietary carbohydrate and serum cholesterol levels" (Winitz 1970, Winitz 1964). This sugar-raises-cholesterol effect was confirmed in a more recent study which used an American-style diet containing 42% fat – cholesterol rose with time in a dose-dependent fashion when sugar was fed, but remained at baseline levels when carbohydrate was given as starch (Reiser 1979). A low glycemic index diet (meaning low in simple sugars (Wolever 1994) lowered cholesterol by 15% in six healthy male volunteers (Jenkins 1989).

Stress raised the cholesterol of tax accountants by an average of 20% around April 15th (Friedman 1958), and all manner of emotionally-arousing events, from race-car driving to examinations provoke the same reaction (Dimsdale 1982). Correcting sub clinical hypothyroidism lowered LDL-cholesterol by an average of 8% in one study (Kahaly 2000). "Chronic magnesium supplementation produced a significant reduction of plasma cholesterol and LDL cholesterol, and an increase of HDL cholesterol" in Type 2 diabetics (Corica 1994). Taking vitamin C varied an experimenter's cholesterol level between 230 and 140 mg% (Spittle 1971), and chromium given with niacin lowered LDL-cholesterol by 27% (Gordon 1991).

In summary, for 24 California felons, replacing sugar with starch lowered cholesterol, on average, by 76mg%. That this is likely an important determinant of our cholesterol levels because we eat, on average, 160 pounds of sugars annually – most of us eat our own weight in sugars each year! And since between 10% and 40% of us are sub clinically hypothyroid, most of us feel stressed, 56% of us eat diets with insufficient magnesium (USDA 2005), 30% suffer vitamin C-depletion (Hampl 2004) and almost all of us are depleted of chromium (because sugar causes both chromium and magnesium to be lost in the urine), it's really not a surprise that many of us have high cholesterol. The *really* surprising thing is that we are not advised to abstain from sugar, neither are we tested for thyroid deficiency, nor are we prescribed vitamin C, magnesium, chromium, niacin or even prescribed security blankets for our stress when we consult doctors because of high cholesterol.

So does cholesterol really matter?

Dr William Castelli, director of the Framingham study, revealed in a moment of candor what he'd really found in the Framingham population. The Framingham study followed most of the residents of Framingham, Massachusetts, for many years, and revealed much of what we know, or think we know, about heart-disease risk factors. In 1992, Dr Castelli wrote to an obscure journal in response to a study (Fraser 1992) which had found raw nuts to be powerfully cardio-protective. The tone of the letter is tongue in cheek, and a careful reading of the letter itself suggests to me that he is bemoaning the tendency of epidemiological studies to throw up results which fly in the face of reason, such as, in his mind, findings which contradict the cholesterol-causes-heart-disease notion (Castelli 1992):

> In Framingham, Massachusetts, the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower people's serum cholesterol ... 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. ... In view of this, this study fails to describe a relationship of those traditional dietary constituents, saturated fat and cholesterol, known to have an adverse effect on blood lipids, and thereby, on the subsequent development of coronary disease end points.

Dr Castelli actually found no relation whatsoever between cholesterol or saturated fat and heart disease in the Framingham study, but he has the conviction of a religious zealot that the relationships exist! You can see the danger of funding research projects headed by a guy like this: No matter what he finds, cholesterol is the cause. It seems the entire cholesterol preoccupation and the dietary advice based upon it are nothing more than a marketing scheme employing the supposedly-disinterested American Heart Association as a mouthpiece to plant an erroneous belief so deeply in the minds of a generation that it has become the conventional wisdom.

Marketing disguised as science?

The fact is that some form of the worthless low-fat Therapeutic Lifestyle Changes Diet is the medical prescription for weight loss (66% of Americans are overweight), diabetes (7% have diabetes, and a further 40% have "pre-diabetes") and heart disease (24%), and it is even suggested to healthy people for the prevention of disease! For example, the new, 2005 USDA Food Pyramid strongly promotes the low-fat diet to the public, but few of us have read the USDA mission statement: … *a strategic plan [for] expanding markets for agricultural products [and] … further developing alternative markets for agricultural products.* It is bizarre that the USDA should also be given responsibility for advising the nation on what to eat when it is also responsible for subsidizing corn, wheat, soybeans and cotton crops to the tune of $20bn per year and finding markets for all this bounty!

In the Food Pyramid fine print, I learn that if I follow this diet, I have 217 "discretionary" calories left over after I have achieved all the Recommended Daily Allowances for vitamins, minerals and the essential fatty acids. Discretionary calories are, according to the USDA, from refined foods, and 217 calories is equivalent to about 12 teaspoons of sugar which is the amount in one can of Coca Cola. Vanishingly few people in America eat as little sugar as

this, for the *average* American eats about 50 teaspoons of sugar daily. But Heaven forbid that we should restrict a market for agricultural products! The USDA Food Pyramid fails us because it doesn't explicitly condemn refined carbohydrates; it only advises that half one's grain intake should be unrefined. And we are not warned that eating more than trivial amounts of sugar negates the benefit of the diet by *guaranteeing* nutritional deficiencies.

The low-fat Therapeutic Lifestyle Changes Diet has too many carbohydrates to work for insulin-dependent diabetes, or Type 2 Diabetes. And such diets do not work very well for weight loss: "within the United States, a substantial decline in the percentage of energy from fat consumed during the past two decades has corresponded with a massive increase in obesity" (Willett 1998), although *low*-carbohydrate diets probably do: "consumption of high-GI carbohydrates may increase hunger and promote overeating" (Roberts 2000). Low-fat diets provoke too much insulin secretion to be healthy in heart disease or, in fact, for anybody at any time. But what a clever way to market refined grain products, sugar and refined vegetable oils to the overweight, diabetics, heart victims and in fact the entire population: low-fat diets are prescribed for the very problems they cause! And, in a truly diabolical twist, the money that the food refiners have given to the various disease Associations to establish this useless diet as the standard of care is tax-deductible to them as a charitable contribution!

Nutrients: The missed clue

Dr Ancel Key's original paper on total fat consumption and mortality from coronary heart disease cherry-picked among the countries for which data was available, apparently because this misrepresentation fit his graph more convincingly. Tragically, it is apparent from a plot of all the countries for which data was available that something other than fat consumption was affecting mortality. The countries with the lowest mortality were Ceylon, Japan, Mexico, Chile, Portugal and France, and their percentages of calories from fat ranged from 7% in Japan to 30% in France. What do these countries have in common? There are two island nations and Portugal (which has a very long coastline relative to its area) in which fish are prominent in the

diet, four relatively undeveloped nations with largely unrefined diets, and one Western nation which prefers butter over refined vegetable oils. It begins to look as thought fish and unrefined foods could be important in the prevention of heart disease. The countries with the highest mortality were the United States, Finland, Australia, Canada, New Zealand and Israel; that these countries are among those with the most refined diets in the world (check out their sugar consumption) lends strength to the idea that refined foods contribute to heart disease.

The true tragedy of the entire cholesterol fiasco is that, buried in Key's *Seven Countries Study* (in which Keys blamed saturated fat rather than total fat), there is the fascinating information that the heart death rate in Corfu is five times that of Crete. These neighboring Greek islands had diets which were identical according to laboratory assessments of the time. A similar disparity existed in neighboring counties in Finland, where the heart death rate in North Karelia was much higher than that of counties to the south which have the same demographic and diet. Keys obscured this clue by choosing the high death rate of North Karelia to represent Finland, and the low death rate of Crete to represent Greece. Had this intriguing hint been followed up, millions of lives might have been saved. It is quite certain that all this was not lost on Keys for he adopted the Cretan diet, as related in his book *Eat Well and Stay Well* (Doubleday 1959), and lived to be 100 years old.

Studies which have passed unnoticed since then reveal that the diet of Crete, but not that of Corfu, contains abundant *purslane*, a salad leaf which is rich in cardio-protective omega-3 fatty acids. And in North Karelia, the blood lipoproteins which transport cholesterol contain less vitamin E than those of the inhabitants of the southern counties, so that their LDL-cholesterol is at greater risk of oxidation. And the water supply of North Karelia is softer, meaning that it contains less cardio-protective magnesium than the water in the south. It remains to be seen whether these *nutrient* differences explain the difference in heart deaths. But I'm getting ahead of myself again.

More on Cholesterol …

My doctor's plan for me was that I should take a statin drug if a three-month trial of the Therapeutic Lifestyle Changes Diet didn't lower my LDL-cholesterol. My LDL cholesterol is 111. My doctor offers a statin drug because the Cholesterol Assessment and Treatment guidelines say that LDL-cholesterol should be treated aggressively in diabetics, even though they acknowledge that "Modification of blood pressure and lipids in people with diabetes, however, does not reduce CHD risk" (p. II -15). My cholesterol is 165, my triglycerides are 63, my ratios are good, my pulse barely gets off the peg on the treadmill ... should be on a statin drug? I knew little about statins except that the people I knew who were on statins complained of their side-effects, particularly fatigue and confusion. When I looked into them, what I learned convinced me that statins are only ever appropriate for secondary prevention, meaning after a heart attack when the danger of another heart attack is very high.

The trouble with statins

It's clear that statins work, to the extent they do work, by lowering inflammation because studies have shown that they are effective long before the cholesterol level drops (Ridker 2005, Nissen 2005). But my C-reactive protein level, an index of the amount of inflammation I suffer, is already so low as to be near the limits of measurement. And how much do statins actually help? Figures are usually quoted as *relative risk reductions.* For example, in the West of Scotland primary prevention trial in men with an average cholesterol level of 272, there was a 31% relative risk reduction for "coronary events" (meaning heart attack or death from CHD) in the statin group (Shepherd 1995). The study was of primary prevention, designed to see if a statin drug would prevent heart trouble among healthy men (like me, although I do have diabetes) who had high cholesterol (which I don't have); in other words, the study was as close to my circumstances as I could find.

The outcome sounds very encouraging: my chance of heart trouble is cut by almost a

third if I take the statin for five years, and moreover cholesterol fell by 20% and LDL-cholesterol fell by an even more impressive 26%. But the *absolute* risk of a coronary event was 1.32% in the statin group and 1.88% in the placebo group, giving an *absolute* risk reduction of 0.56%, or 0.11% for each year of the study. Put another way, *one* heart attack would be prevented if 874 men took the statin for a year! This "number needed to treat" figure shows that encouraging *relative* risk reductions are scarily misleading unless the *absolute* risk reduction is also given, and that this statin treatment cuts the risk of heart trouble in men like me by only a derisory amount.

Lies, damned lies and statistics
Benjamin Disraeli

But much more troubling to me is the all-cause mortality was unchanged. In the WOSCOPS trial, the change in risk of death from all causes was statistically insignificant! This means that for every person whose life was saved by the drug treatment, another died of something else. I wouldn't last long at the track if I bet these odds – what the hell is my doctor thinking? In fact, this has proved to be the case in several statin trials, in which the only statistically-significant results were fewer non-fatal CHD events (Ravnskov 2000):

	WOSCOPS Pravastatin	CARE Pravastatin	AFCAPS/TexCAPS Lovastatin
	Healthy people; High cholesterol	CHD patients; Normal cholesterol	Healthy people; Normal cholesterol
Non-fatal CHD events: Drug Group/Control Group	143/204	135/173	116/183
Relative Risk of Non-fatal CHD	-22%	-22%	-38%
	Significant	Significant	Significant
Deaths from CHD: Drug Group/Control Group	38/52	96/119	11/15
Relative Risk of CHD death	-27%	-19%	-27%
	Not significant	Not significant	Not significant
Deaths from all causes: Drug Group/Control Group	106/135	180/196	80/77
Change in **Absolute Risk** of	-0.9%	-0.77%	+0.09%

Death (%)			
	Not significant	**Not Significant**	**Not Significant**

A possible interpretation is that a side-effect of statin drugs is death! The *Therapeutics Newsletter* from the University of British Columbia came to the same conclusion after analyzing the outcomes of five statin trials: "If cardiovascular serious adverse events are viewed in isolation, 71 primary prevention patients with cardiovascular risk factors have to be treated with a statin for 3 to 5 years to prevent one myocardial infarction or stroke. This cardiovascular benefit is not reflected in 2 measures of overall health impact, total mortality and total serious adverse events. Therefore, statins have not been shown to provide an overall health benefit in primary prevention trials" (Do Statins have a Role in Primary Prevention? 2003).

And are there side-effects? A widely-quoted study suggests that the risk of side-effects is between 3% (Atorvastatin) and 4% (other statins), *but the study itself says the risk is 22%*. Incredibly, in the body of the review, Dr Newman (who was working for Pfizer, the manufacturer of Atorvastatin) arbitrarily reclassified most side-effects as not "treatment-associated" to achieve the low incidence of side-effects in her conclusion (Newman 2003):

TABLE 3. All Completed Studies Data Grouping: Overview of Safety, Comparing Atorvastatin With Other Statins and Placebo

	Placebo (n = 1,789)	Atorvastatin (all doses) (n = 9,416)	Other Statins (n = 5,290)
Patients experiencing ≥1 adverse event			
All	45%	65%	67%
Treatment-associated	15%	18%	19%
Withdrawals due to adverse events (no. of patients (%))			
All	25 (1%)	398 (4%)	297 (6%)
Treatment-associated	16 (1%)	241 (3%)	188 (4%)
Serious, nonfatal adverse events (no. of patients (%))			
All	137 (8%)	963 (10%)	590 (11%)
Treatment-associated	114 (6%)	19 (<1%)	6 (<1%)
Deaths (no. of patients (%))	12 (1%)	66 (1%)	30 (1%)

It's clear that 45% of patients experienced one or more adverse events in the placebo

group, compared with 67% in the "Other statins" group, so that the incidence of side-effects is actually 22%. There's no explanation of what side-effects are not "treatment-associated", so we are asked to accept the 3-4% side-effects conclusion as an act of faith! Muscle pain and weakness, numbness and tingling, congestive heart failure, cognitive impairment and depression have been reported (Enig, accessed 2006), but were obviously classified as not "treatment-associated" by Dr Newman. In the real world, it is evident that unpleasant side effects are widespread: "… approximately 50% of patients placed on a lipid-lowering drug quit taking the drug in 1 year and only 25% still take the drug 2 years after it was started" (Roberts 1996).

He who takes medicine is ill-informed
Leonardo da Vinci

Ominously, animal studies suggest statin treatment causes cancer, and human trials hint that this effect is real. In the PROSPER trial, elderly individuals in the statin group had a relative risk of cancer of 1.25 compared to the placebo group (Shepherd 2002), and women in the Cholesterol and Recurrent Events trial had an *absolute* increased risk of breast cancer of 4.2%, a relative risk increase of no less than 15 (Sacks 1996). Further, statin therapy does not stop the progression of calcification of the coronary arteries, despite lowering LDL-cholesterol (Houslay 2006), and progression of coronary artery calcification is a powerful predictor of heart death (Wayhs 2002). So, in spite of my doctor's enthusiasm, I can't help but conclude that statins are at once both perilous and futile. Statin drug treatment of cholesterol for heart disease seems to me to belong in the book *Extraordinary Popular Delusions and the Madness of Crowds* (Three Rivers Press, 1995):

Why do otherwise intelligent individuals form seething masses of idiocy when they engage in collective action? Why do financially sensible people jump lemming-like into hare-brained speculative frenzies – only to jump broker-like out of windows when their fantasies dissolve? We may think that the Great Crash of 1929, junk bonds of the '80s, and over-valued high-tech stocks of the '90s are peculiarly 20th century aberrations, but Mackay's classic – first published in 1841 – shows that the madness and confusion of crowds knows no limits, and has no temporal bounds. These are extraordinarily illuminating, and, unfortunately, entertaining tales of chicanery, greed and naïveté (from a review at Amazon.com).

So how on Earth did we end up with such lousy advice? The American Heart Association cholesterol guidelines are a thinly-disguised sales pitch for statin drugs. They were actually written by an Expert Panel of the National Cholesterol Education Program. This organization was founded by the National Heart Lung and Blood Institute (one of the government's National Institutes of Health) in 1985, and consists of 41 associations like the American Diabetes Association, American Medical Association, American Heart Association etc, plus *"media and industry representatives also participate in the program."* Since the various associations are largely funded by the food and pharmaceutical industries, perhaps it is not surprising that their bias towards statin drug treatment is so clear:

> Of the nine [Expert] panel members, six had each received research grants, speaking honoraria or consulting fees from at least three and in some cases all five of the manufacturers of statins. If all the members with conflicts had recused themselves, only two would have been left. (Detroit News editorial 8/8/2004)

The pharmaceutical industry has cleverly put their statin advertising into the mouth of the government! We have set the fox to guard the chickens, with predictable results.

So if it's not cholesterol, what is it?

Whole, unrefined foods support health very successfully. In the 1930s, the dentist Dr Weston Price documented a very low rate of dental decay amongst twelve diverse peoples, from Alaskan Eskimos to New Zealand Maoris, but only so long as they remained on their traditional, unrefined diets. In his 1939 book *Nutrition and Physical Degeneration* (Keats Publishing, 15th edition, 2003), he described well-developed skeletal structure, strong immune systems, almost no cancer or heart disease and a striking absence of mental illness. He went on to document the precipitate decline in health consequent on these peoples adopting refined foods: "These primitives with their fine bodies, homogeneous reproduction, emotional stability and freedom

from degenerative ills stand forth in sharp contrast to those subsisting on the impoverished foods of civilization – sugar, white flour, pasteurized milk and convenience foods filled with extenders and additives." (WestonAPrice.org)

But almost no one knows these things because the food refining process is so profitable. There is a massive cash-flow available to buy PR to drown out these truths, because refined food is more than delicious, it is addictive, and because the high rate of disease caused by refined food supports both the pharmaceutical industry and the medical profession. Furthermore, once a position is taken by researchers, public health officials and the medical profession, then careers and reputations are on the line and the defense of the indefensible begins.

What I have learned

The diet-heart hypothesis [which is, in essence, that fat in the diet causes heart disease] has been repeatedly shown to be wrong, and yet, for complicated reasons of pride, profit and prejudice, the hypothesis continues to be exploited by scientists, fund-raising enterprises, food companies and even governmental agencies. The public is being deceived by the greatest health scam of the century." George V Mann MD, Ed., *Coronary Heart Disease: The Dietary Sense and Nonsense*, Janus, London UK, 1993; Dr Mann was one of the original Framingham investigators.

"Anyone who questions cholesterol usually finds his funding cut off." Paul Rosch, MD

Almost all heart disease research funding comes from the American Heart Association and the National Heart Lung and Blood Institute, and these institutions fund cholesterol studies almost exclusively. Consider further that the American Heart Association's biggest contributors are General Mills, Heinz, Schering Plough, Merck, Bristol Meyers Squibb, Pfizer, GlaxoSmithKline, Novartis, Astra Zenica, Aventis and Bayer – food refiners and pharmaceutical houses who benefit from the sale of refined, low-cholesterol and fat-free foods and from the sale of cholesterol-lowering drugs. Research depends on funding, so if funding is lost when the focus of the research veers away from cholesterol, it's cholesterol that will be researched!

So cholesterol's a sham, kept alive to sell refined foods, and sell drugs. In his book *The Cholesterol Myths* (New Trends, Washington DC, 2000), Dr Uffe Ravnskov, wrote:

> Our ancestors did not know better because they had only the naked eye and lacked the technology needed to discover the truth. But the proponents of the diet-heart idea ought to know. Instead, their cocksure writings demonstrate that for them the idea has become a fact, the cholesterol Earth is flat. Or is it only a game? Those of you who read this book will realize that scientists who support the diet-heart idea and who are honest must be ignorant, either because they have failed to understand what they have read or else, by blindly following the authorities, they have failed to check the accuracy of the studies written by those authorities. But some scientists must surely have realized that the diet-heart idea is impossible and yet, for various reasons, have chosen to keep the idea alive.

Cardiologist Dr Arthur Blumenfeld surveyed 100 eminent medical scientists in the '70s in an attempt to garner support for his preferred heart disease treatment, which was the low-fat approach:

> The final question in the survey asked their choice of one of two diets they considered more anti-coronary. … Over 90 of the 100 expressed a preference for a low-carbohydrate diet … In opposition to this apparent overwhelming majority opinion are the conclusions of the American Heart Association … whose diet of choice for coronary prevention is low-fat rather than low-carbohydrate. Obviously there is a basic conflict concerning rational and therapeutic diets (Blumenfeld 1974).

It's clear that most medical scientists disagreed completely with the American Heart Association's prescription – but we do not hear from them.

The world is a dangerous place, not because of those who do evil,
but because of those who look on and do nothing
Albert Einstein

Yet there is dissent. The prestigious magazine Science published an article by journalist Gary Taubes entitled The Soft Science of Dietary Fat in 2001, and the *Journal of American Physicians and Surgeons* published Anthony Colpo's LDL Cholesterol: "Bad" Cholesterol, or Bad Science? in 2005. The articles question the science behind current dietary recommendations and outline their commercial motivations, and indeed question the motives of the organizations promulgating the recommendations. Although these are scientific journals, these men are not researchers and therefore do not rely on the research funding sources which, in effect, gag researchers who must obtain future funding. It seems that scientists must let others speak for them if they wish to stay in the game.

I think statins and the Therapeutic Lifestyle Changes Diet are dangerously stupid. But I'm intrigued by the Kitava Islanders freedom from heart disease, and with Dr Weston Price's observation that diverse healthy populations lived on wildly different diets having in common only that each was composed of whole foods. The industrial revolution started a migration to the cities, which, in turn, created a demand for foods with "shelf-life", foods which can be transported and stored without spoilage. Whole foods, fresh from the fields or the sea, were replaced by processed and preserved foods of lesser nutritional quality: refined foods. What happens to a food when it's refined?

Food refining causes nutrient losses

Since at least 20% of the average citizen's calories come from sugar, with perhaps another 30% from refined flour and alcohol which have much or all of their vitamins and minerals refined out of them, few of us actually consume a balanced diet. The amounts of 21 nutrients lost in the refining of the 1200 calories of sugar and refined flour in the average citizen's diet are easily calculated to lie between 95% (magnesium) and 58% (selenium). We've thrown away the wrong parts of the food!

Comparing the National Center for Health Statistics dietary data to the government's suggested Dietary Reference Intakes suggests that many of us don't achieve even the Recommended Dietary Allowance of calcium, folic acid, fiber, or magnesium. *One in three* of those surveyed had vitamin C deficiency or depletion, and between 20% and 90% (depending which study you favor) don't take in sufficient vitamin B6. Refining removes twice the RDA for magnesium, and fully *56%* of us don't get even the RDA from our diets. *Five times* the RDA of folate is lost (the "enrichment" of the flour does not replace all that is taken out), and the elevated homocysteine level of *half* the population tells us they don't get enough folate, even if we're *technically* not deficient in it. The list goes on.

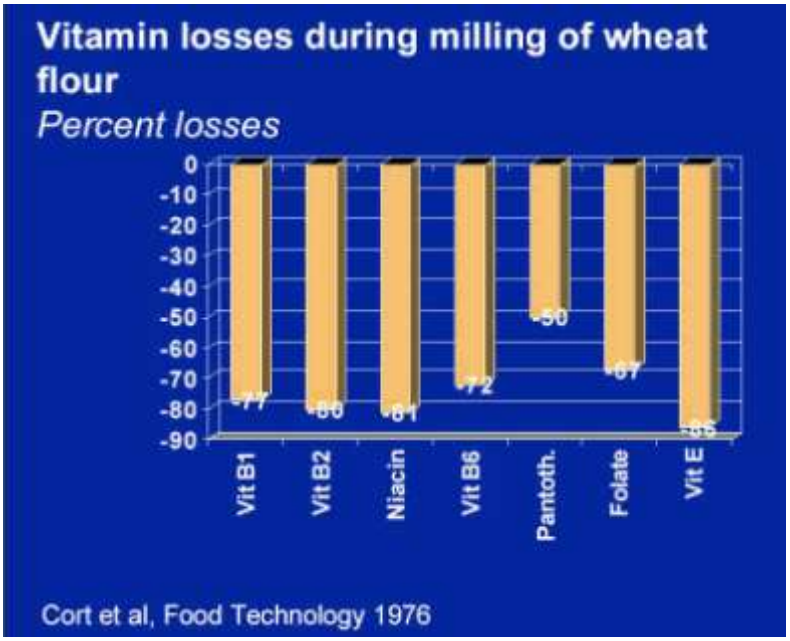


TABLE 2— Percentages of Vitamin C Deficiency, Depletion, and Normal Serum Values Among US Males and Females

Gender and Age, y	Serum Vitamin C Value		
	<11 µmol/L, %	11–28 µmol/L, %	>28 µmol/L, %
Male			
12–17 (n = 975)	6	17	77
18–24 (n = 1011)	13	22	65
25–44 (n = 2649)	17	23	60
45–64 (n = 1765)	17	20	63
65–74 (n = 955)	11	15	74
Overall	14	20	66
Female			
12–17 (n = 1133)	5	15	80
18–24 (n = 1186)	11	19	70
25–44 (n = 3212)	12	20	68
45–64 (n = 1916)	10	15	75
65–74 (n = 967)	6	13	81
Overall	10	17	73

Left: Nutrient loss during flour refining; Right: One in three of us are low in vitamin C! (Figure from Hampl)

Incredibly, this is not news. Although it seems scandalous to me, I did not learn of it on CNN. It is not even new, for it was remarked on in the ***1939*** US Department of Agriculture Yearbook:

The chief fault of many American diets is that they provide too little of the essential minerals and vitamins. This fault is due in large measure to the fact that refined foods are consumed in such amounts that the intake of mineral and vitamin rich natural foods is lower than it should be (*Food and Life*, USDA Yearbook, 1939)

What effect does this have on us? If whole foods carry with them the nutrients needed for their metabolism and refined foods do not, common sense suggests Dr Weston Price was right in his belief that it's the lack of the missing nutrients that causes disease. With this organizing principle in mind, let us look to the scientific literature. This is really easy to do this, by the way, thanks to your tax dollars at work. I simply enter the query in the National Library of Medicine database and it returns the abstract of the article. If I want to read the whole article, I can order it through the delightfully-named Loansome Doc service.

I particularly like Harvard epidemiologist Dr Walter C Willett's population studies because they look at what people are eating (or not eating), and if they get, say, heart disease in the following years. Of course, such studies cannot prove that dietary deficiencies *cause* heart disease, but they can show if they are fellow travelers with some unknown cause. So I enter "Willett WC, vitamin E CHD" (or whatever the current nutrient of interest may be) and discover a wealth of information.

Often, the results of these studies are presented as "*relative risk*" of heart disease, which is the ratio of the incidence of whatever's being investigated in the groups taking in the highest and lowest levels of a nutrient (Barratt 2004). Thus, in Dr Eric Rimm's study (in which Dr Willett participated) of "Vitamin E Consumption and the Risk of Coronary Heart Disease in Men", the study participants were divided into five groups. Over the four years of the study, the quintile taking in the least vitamin E developed coronary heart disease at the rate of 19.4 per 1000 people, while the incidence among those taking the most was 14.4 per 1000 people. The *relative risk* is the ratio of 14.4 ÷ 19.4, which is 0.74 (which may also be stated as a *relative risk reduction* of 26%).

Individuals poorly nourished in E developed CHD at the rate of 0.49%, close to the 0.5% rate found by the American Heart Association for a 60-year-old male, which is perhaps unsurprising since 93% of the population consumes less than the RDA for vitamin E (USDA 2005). The *absolute risk reduction* was that 5 fewer people per 1000 developed CHD over the four years of the study in the high intake group compared with the low intake group; thus, individuals well-nourished in vitamin E had an absolute risk reduction of (5 ÷ 4) or 0.13% per year. This absolute risk reduction of 0.13% therefore represents about one third of the risk of CHD for a 60-year-old male, a considerable proportion.

Thus, the absolute risk says whether the problem is clinically significant in the study population, and the relative risk indicates how effective the intervention is. The relative risk reduction of 26% looks impressive, but is essentially meaningless without the absolute risk figure of 0.49% which means being well-nourished in vitamin E halves the risk of CHD. Together, they answer the question: is the effect of vitamin E large enough to be clinically important? When the absolute risk reduction is small, as in the statin studies, the relative risk reduction numbers have little clinical significance, however large they may be. But when the absolute risk is large, the relative risk reduction is meaningful and provides guidance on how effective the intervention is.

I have listed a number of such studies in order of their clinical relevance. Why these particular Rag, Tag and Bobtail studies? Mainstream studies play down the risks and exaggerate the benefits of drugs, advocating almost useless cholesterol treatments on study outcomes which failed to reach significance; drugs which do not offer a remedy for what ails me. Mainstream nutritional recommendations have clearly done more harm than good; but, by contrast, I believe these few studies actually show which way the wind blows.

Nutrients lower Cardiovascular Disease risk

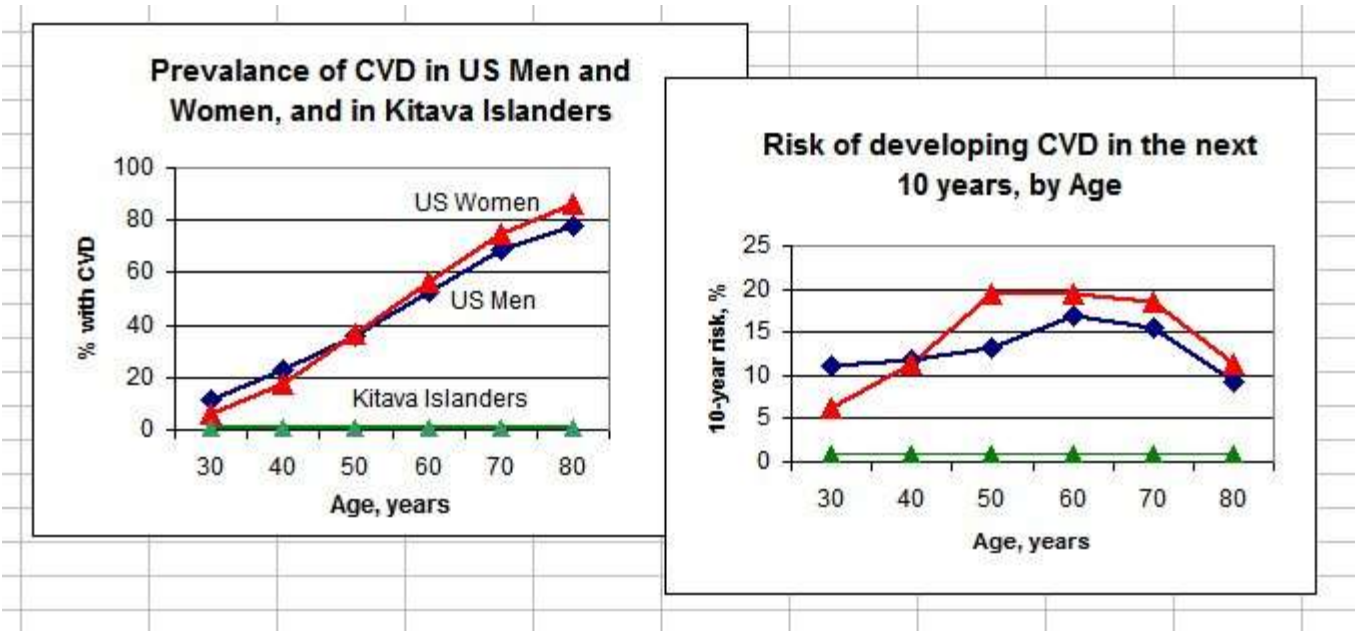
Nutrient	How many have "low nutrient status"?	Amount, Study population; Study duration	Absolute Risk high-intake group (hi)/year	Absolute Risk low-intake group (lo)/year	Relative Risk (hi/lo)	Absolute Risk Reduction/year (hi – lo)/duration
Carotenoids (Gaziano 1995)	44% (USDA 2005)	<0.8βcarotene foods>2.05/svgs/day Elderly subjects; 4.75 years	2.23%	4.53%	0.54 (0.49)	2.3% CVD death
Magnesium (Singh 1990)	67% of adults (USDA 2005)	1142mg vs. 418mg High-risk adults; 10 years	0.93%	1.6%	0.6 (0.6)	0.63% CHD death
Vitamin C (Enstrom 1992)	40%(M) 32%(F) (Hampl 2004)	>800 vs. <50mg Adults; 10 years	0.91%	1.3%	0.65(M) (0.69%)	0.42% CVD death
Omega-3 fish oils (Mozaffarian 2003)	US: 0.01- 0.02g/day Desirable: 0.65g/day (Kris-Etherton 2000)	≥3/wk Fish meals <1/mo (not fried or fish sandwich) Subjects ≥ 65 years; 9.3 years	0.35%	1.1%	0.28 (0.32)	0.76% Sudden CHD death
Sodium (Tuomilehto 2001)	90% > UL* of 2.3g (FNB 2004)	<3.64 vs. >6g/day in urine Finnish men; 10 years	0.72%**	0.5%**	1.38 (1.43)	0.22%** CVD death
Potassium (Bazzano 2001)	>97% (USDA 2005)	>2.67 vs. <1.37 g/day Adults; 19 years (AI=4.7 g)	0.38%	0.62%	0.72 (0.61)	0.27% Stroke
Vitamin E (Rimm 1993)	93% (USDA 2005)	>60IU vs. <7.5IU (M) Adult men; 4 years	0.36%	0.49%	0.64(M), (0.74)	0.13% New CHD
Sedentary lifestyle (Tanasescu 2002)	88% are sedentary (Reeves 2005)	Most vs. least exercise Adult men; 12 years	0.25%	0.41%	1.72(M) (1.62)	0.17% New CHD
trans-fats (Ascherio 1996)	US 5.8g/day (USDA 2006)	4.3 vs. 1.5g/day (M) Adults; 6 years	0.38%	0.25%	1.43(M) (1.5)	0.13% New CHD
Hypothyroidism (Barnes 1972)	10-40% (Barnes 1972)	1-4gr vs. 0gr Armour thyroid vs. Framingham pop.; 20 years	0.01%	0.22%	0.06	0.9% New CHD
Glycemic Load (Liu 2000)	US sugar: 146lb/yr (USDA 2004)	206 vs. 117 Adult women; 10 years	0.09%	0.13%	1.98(F) (1.37)	0.04% New CHD
Folate (Rimm 1998)	88% (Subar 1998)	696 vs. 158µgm Adult women; 14 years	0.06%	0.11%	0.69(F) (0.58)	0.05% New CHD
Folate *and* B6 (Rimm 1998)	High folate *and* B6 give lowest RR	Folate 696 vs. 158µgm + B6 4.6 vs. 1.1mg/day	?	?	0.55(F)	? New CHD
Vitamin B6 (Rimm 1998)	71%(M) 90%(F) (Kant 1990)	4.6 vs. 1.1mg/day Adult women; 14	0.06%	0.09%	0.67(F) (0.62)	0.04% New CHD

		years				
						6.1%

Relative Risks are controlled for age, smoking and other risk factors in most studies and may therefore differ from "(hi/lo)". Absolute Risks are from the raw data, the number of deaths or new cases in each group.
*Tolerable Upper Intake Level. **Estimated assuming a linear dose-response relationship.

The most striking thing about this table is that nutritional deficiencies are so widespread as to constitute a public health scandal. My nutritional status simply hasn't come up in my medical examinations, yet these studies imply that if I am well-nourished in these eight essential nutrients and avoid unhealthful practices, my risk of the various troubles subsumed under the rubric of cardiovascular disease may be reduced by 6.1% per year.

Carotenoids from vegetables together with magnesium and vitamins B, C and E found in whole foods, along with avoiding *hypothyroidism* and *eating fish*, are associated with lowered rates of cardiovascular disease. Of course, the iodine which helps prevent hypothyroidism comes with the package if ocean fish are eaten. *Sugar* and *trans*-fats (the man-made fats in margarines and refined vegetable oils), and pursuing a sedentary lifestyle add to the risk of heart disease.



The American Heart Association 2005 Statistical Update shows that the ten-year risk of developing cardiovascular disease (which includes coronary heart disease) for a male aged 60 years is 15.6%, about 1.56% per year. This figure is simply dwarfed by the 6.1% risk reduction afforded by attending to just eight of the 44 essential nutrients, exercising and avoiding junk food. And in the study of folate and vitamin B6, being replete in both nutrients improved the relative risk of developing CHD from 0.67 to 0.55, so synergy is at work here too: the more nutrients we are replete in, the better our chances.

Furthermore, other essential nutrients certainly contribute to CVD risk; vitamin D, for example, is insufficient in about half the population, and low levels are associated with high blood pressure, impaired fasting glucose and overweight (Martins 2007). Nobody has yet performed a prospective population study to discover the strength of its association with heart risk, but there is no question that it exists. Thus, the annual absolute risk reduction from an unrefined diet and healthy lifestyle is *at least* 6.1%. Can it really this simple? Is this the "secret" of the Kitava Islanders robust good health?

Once you eliminate the impossible, whatever remains, no matter how improbable, must be the truth
Sherlock Holmes

A Note on Causation

These epidemiological studies suggest that attending to nutrition and avoiding refined foods will drastically reduce the risk of CVD, but they cannot prove that poor nutrition *causes* CVD. In science, a causal relationship is established if there is a plausible mechanism, a dose-response relationship, consistency across populations, and verification by clinical trial. This is a summary of Dr Bradford Hill's criteria for causation, which were used, for example, by Sir Richard Doll when he established that smoking causes lung cancer.

The studies in the table above support dose-response relationships between nutrients and susceptibility to CVD. Interestingly, the dose-response relationship between impoverished Western food and degenerative disease is relentlessly detailed in the World Health Organization report Diet, Nutrition and the Prevention of Chronic Diseases, which also establishes consistency across populations. As refined foods enter the diets of undeveloped countries, so too does CVD and rest of the Western degenerative diseases. Papua, New Guinea, is an example, with heart disease well established in the capital, Port Moresby (although it was unknown there until 1964), and just beginning to be found in outlying areas. The observations corroborate and extend Dr Weston Price's 1930s studies referred to earlier.

The great natural experiment of the Kitava Islanders might be said to satisfy the requirement for verification by clinical trial. Here is a population which has not been exposed to Western foods, and which is free of cardiovascular disease and free also from the cancers and dementias which afflict so many in the West.

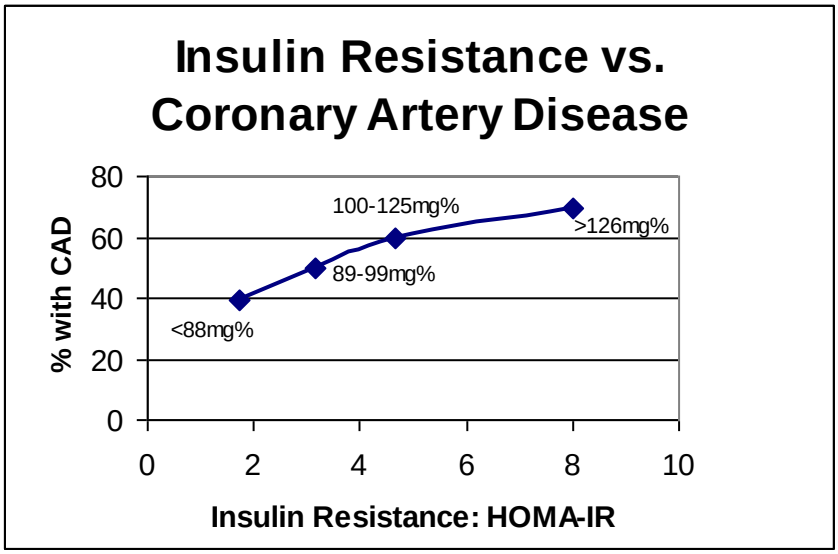
The final requirement: plausible mechanisms. Many plausible mechanisms are already established. After all, essential nutrients are those we can't live without. As I read with increasing fascination the obscure research which suggests nutrients may be more effective than drugs, I learned of many plausible mechanisms I'd never dreamed of. Just as pulling a woolen thread can unravel the entire sweater, I found that, far from being the implausible imaginings of wild eccentrics, there is consistent, solid science underlying the links between lousy food and degenerative disease. One such link is *insulin resistance* in which the muscles, fatty tissue and the liver lose their capacity to take in glucose from the blood when stimulated by insulin. To preserve blood sugar levels in the normal range, the amount of insulin in the circulation rises so that a state of *hyperinsulinism* follows. The link between heart disease and insulin resistance is that some two thirds of heart patients are insulin resistant

Almost all individuals with Type 2 Diabetes and most with hypertension, cardiovascular disease, and obesity are insulin resistant, and, ominously, more than 40% of the remaining supposedly healthy individuals have it by age 70 (Ford 2002). This means that insulin resistance is a chronic progressive condition that almost all of us are incubating. Worse, the associated high insulin level is a "very good predictor of the development of CHD" (Moller 1995), conferring a relative risk of 2.2 for the development of coronary heart disease (Feskens 1994).

Insulin Resistance

I had always thought that nutritional deficiencies impaired the immune system (say), or crippled the antioxidant defense system, or that they elevated homocysteine that damaged the arterial system – that they affected some bodily system.

However, these various dietary faults all worsen insulin resistance at the level of the cell, the fundamental unit of all bodily systems. Insulin resistance contributes to heart disease: people with Coronary Heart Disease were found to have 64% worse insulin resistance than normal subjects. People with impaired glucose tolerance *and* Coronary Artery Disease had 98% worse insulin resistance than people with Impaired Glucose Tolerance but no Coronary Artery Disease – the "data suggest that in patients with CAD, insulin-mediated glucose metabolism is significantly impaired, and a significant correlation was noted between insulin resistance and severity of CAD" (Shinozaki 1996). In a study of 154 people referred for coronary angiography, the most insulin-resistant were nearly twice as likely to have CAD as those who retained their sensitivity to insulin (Quadros 2007):



The fasting blood sugar figures on the graph show a continuous relationship between the severity of CAD in this symptomatic population from low-normal (<88mg%) via Impaired Glucose Tolerance (100-125mg%) to diabetic (>126mg%); insulin resistance increases the incidence of CAD.

Moreover, insulin resistance predisposes to the other Western degenerative diseases: hypertension, obesity, diabetes and cancer. Remarkably, pieces of this puzzle rarely come together so the true picture can be seen: degenerative disease usually starts with insulin resistance!

On Kitava, remember, insulin levels actually fall with age. In the figure below, the hatched area shows how insulin rises with age in Swedes while the line shows that the Kitava Islanders' insulin resistance falls with age. Unlike the Swedes, the Kitava Islanders retain their sensitivity to insulin with age, and have no degenerative disease (Lindeberg 1999):

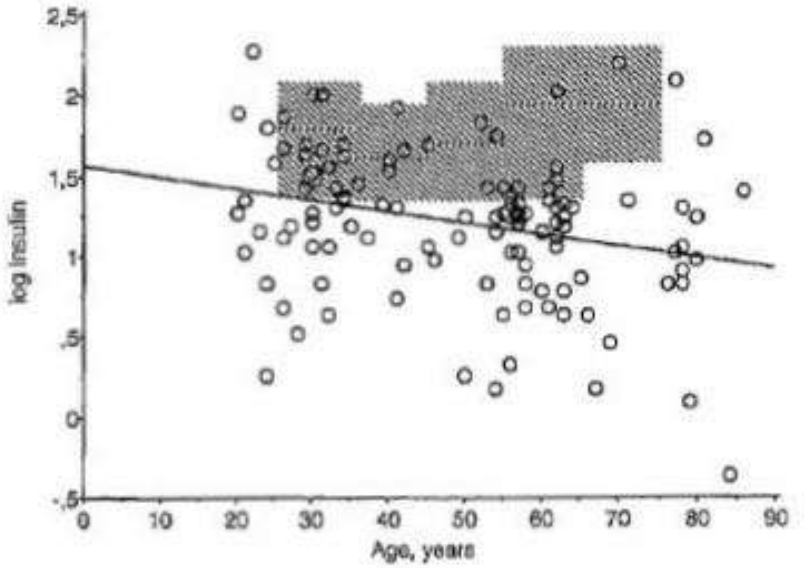


Fig 1. Fasting serum insulin in Kitavan males (▨) Reference intervals for healthy Swedish subjects, whose lower and upper limits represent the 10th and 90th percentiles, respectively. (---) Swedish medians.

Another clue is that healthy Western centenarians retain childhood's high sensitivity to insulin (Barbieri 2001, Marigliano 1992); they may have high or low cholesterol, exercise or be sedentary, eat commonplace foods or follow unusual diets – but all have low insulin levels. This is particularly impressive because only about one in 10,000 of us see our 100th birthday.

For most of us, insulin levels rise dramatically with age, reflecting a chronic, progressive increase in insulin resistance. Dr Vladimir Dilman gave glucose tolerance tests to Westerners at all stages of life, and demonstrated this graphically. Peak insulin levels after glucose is taken rise higher by the decade, which is to say that we develop insulin resistance with age (VRP):

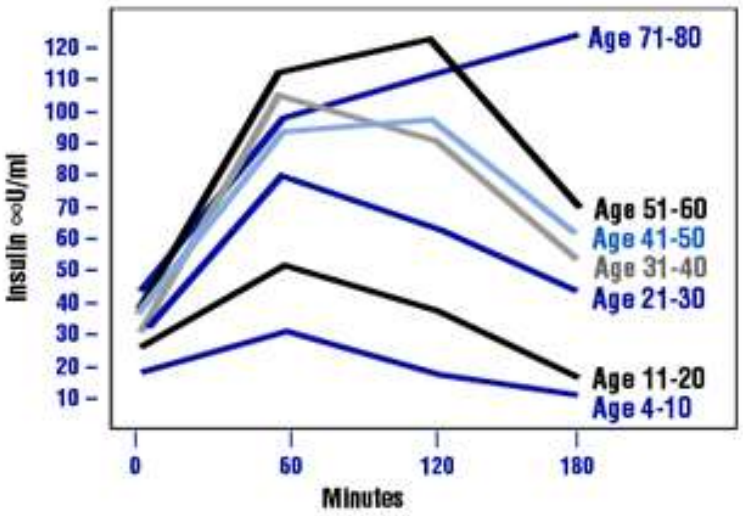
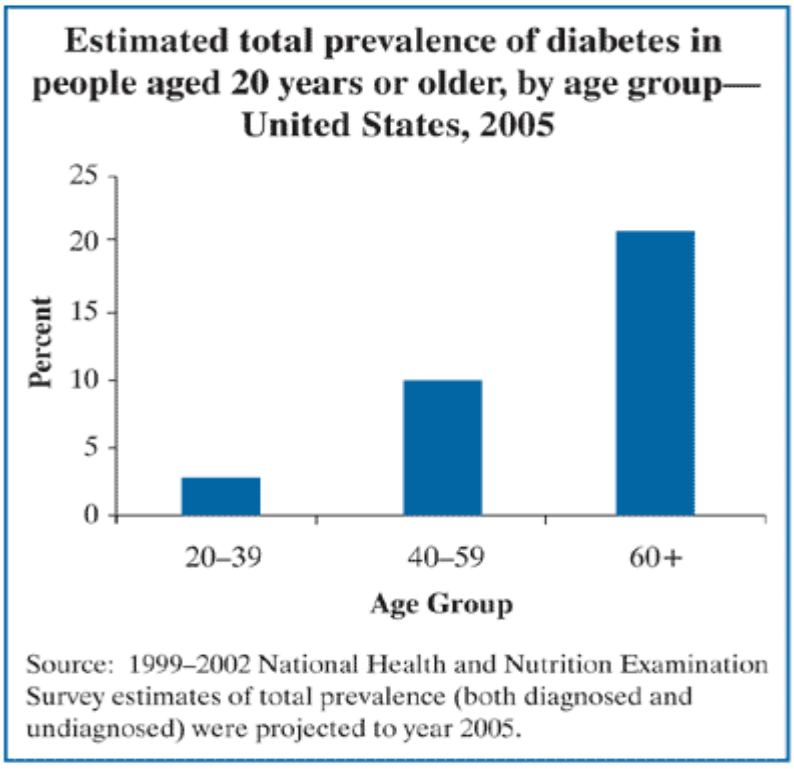


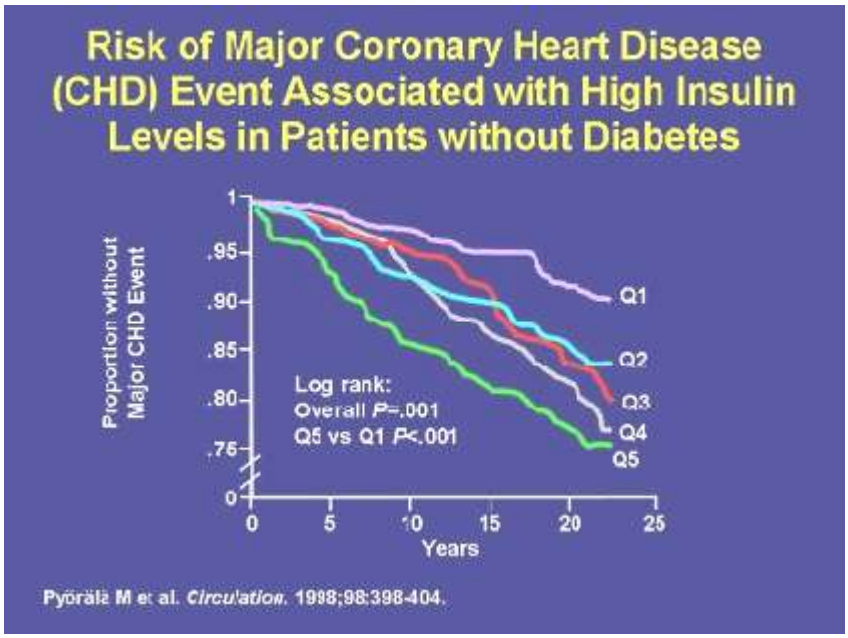
Fig. 6. Age-related changes in insulin in response to glucose. This shows that as we get older, insulin levels tend to rise higher and stay elevated longer, due to "insulin resistance" (loss of insulin receptor sensitivity). This results in obesity, diabetes, hypertension, cancer, and other diseases of aging.

As insulin resistance increases, the blood sugar stays more or less normal because the pancreas secretes more insulin unless the system breaks down into diabetes, when it spirals out of control:



Although only 8% of the whole population has diabetes, the prevalence is 20% of the population over 60 years of age.

The price of keeping the blood sugar in the normal range is, therefore, higher levels of circulating insulin; but insulin has many functions beyond blood sugar control. For one thing, insulin stimulates cells to make fat while inhibiting the breakdown of existing fat. Only when insulin is low does the pancreas secrete *glucagon*; glucagon is the hormone that encourages the breakdown of fat to provide energy. Thus, the elevated insulin level caused by insulin resistance promotes weight gain. Furthermore, elevated insulin levels are strongly associated with increased risk of heart attacks (Lipids online):



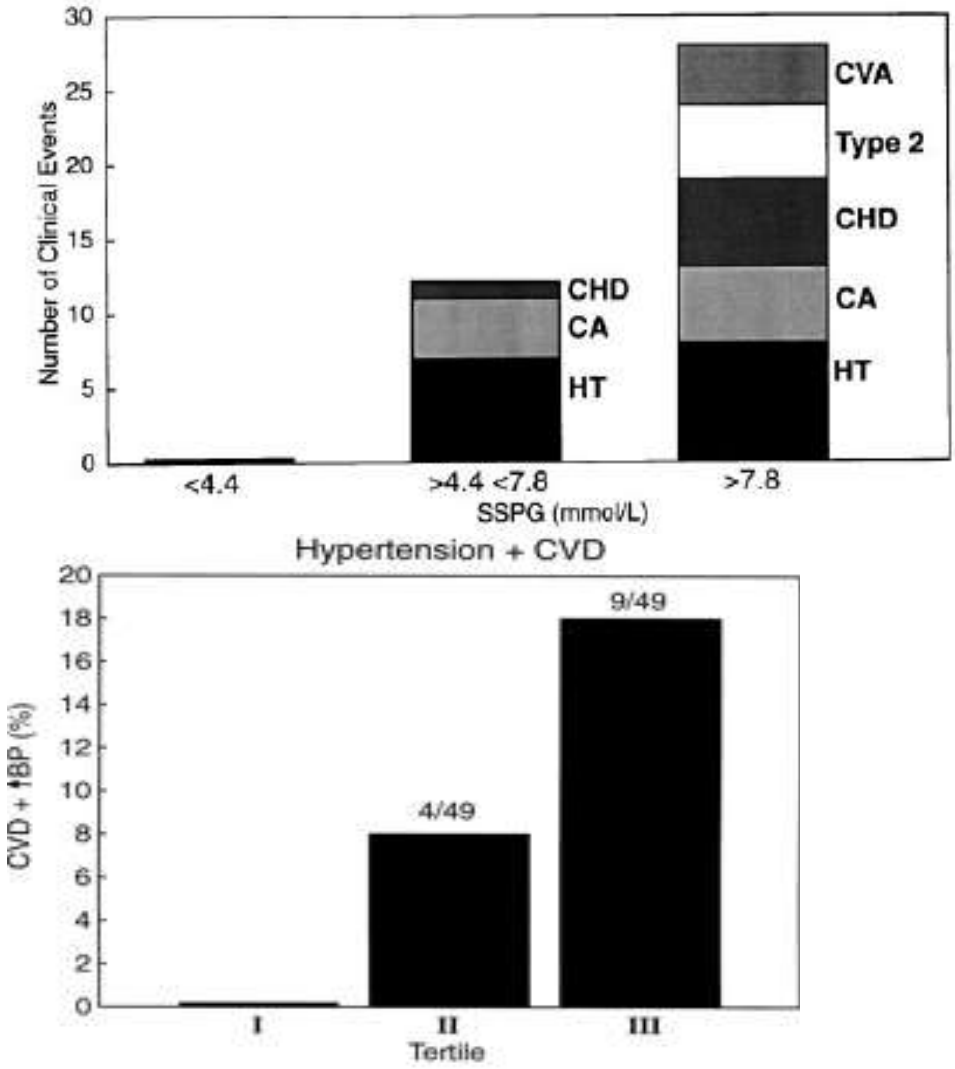
This is a 25-year follow up from the Helsinki Policemen's Study in which they showed a dose-dependent decrease in people who were free from vascular disease in relationship to how high their insulin concentrations were.

Insulin's primary purpose seems to be to help store resources against a rainy day. Caloric restriction lowers insulin and causes weight loss, and, remarkably, it extends the lifespan by 30 to 200%, depending on the species. If food is plentiful, most creatures grow fast and reproduce quickly. In lean times, their life cycles slow. Their lifespan depend on their insulin levels, which depend on the food supply.

Western refined foods are associated with insulin resistance, which is associated with CHD, diabetes, cancer and the autoimmune diseases. The Kitavan Islanders, however, not only retain their youthful sensitivity to insulin, they also remain lean despite an abundance of calories and are immune from the degenerative diseases that kill almost all of us. Is this association causative?

Insulin Resistance predisposes to degenerative disease

Two prospective studies at Stanford University involved 355 healthy people aged about 50 years. Dr Gerald Reaven measured their sensitivity to insulin, and found that *only* the insulin-resistant among them developed degenerative diseases over the 5 to 6 years of follow-up:



Left: The number of clinical events observed, as a function of insulin resistance tertile [third] at baseline. CA, Cancer; Type 2, type 2 diabetes. There were 28 events in the highest tertile (SSPG › 7.8mM), 12 in the intermediate tertile (SSPG › 4.4 ‹ 7.8mM), and none in the most insulin-sensitive tertile (SSPG ‹ 4.4mM) [average 6.4 years of follow-up] (Facchini 2001). Right: … neither hypertension nor CVD developed in the one third of the population that was most insulin sensitive (tertile I). In contrast, almost one of every five individuals in the most insulin-resistant tertile (tertile III) developed hypertension or CVD during the period of observation [average 5 years] (Yip 1998). A study of Japanese people with coronary artery disease documented by angiography had previously found the SSPG of the patient group averaged 7.9 vs 4.8 mmol/L in the CAD-free control group (Shinozaki 1996).

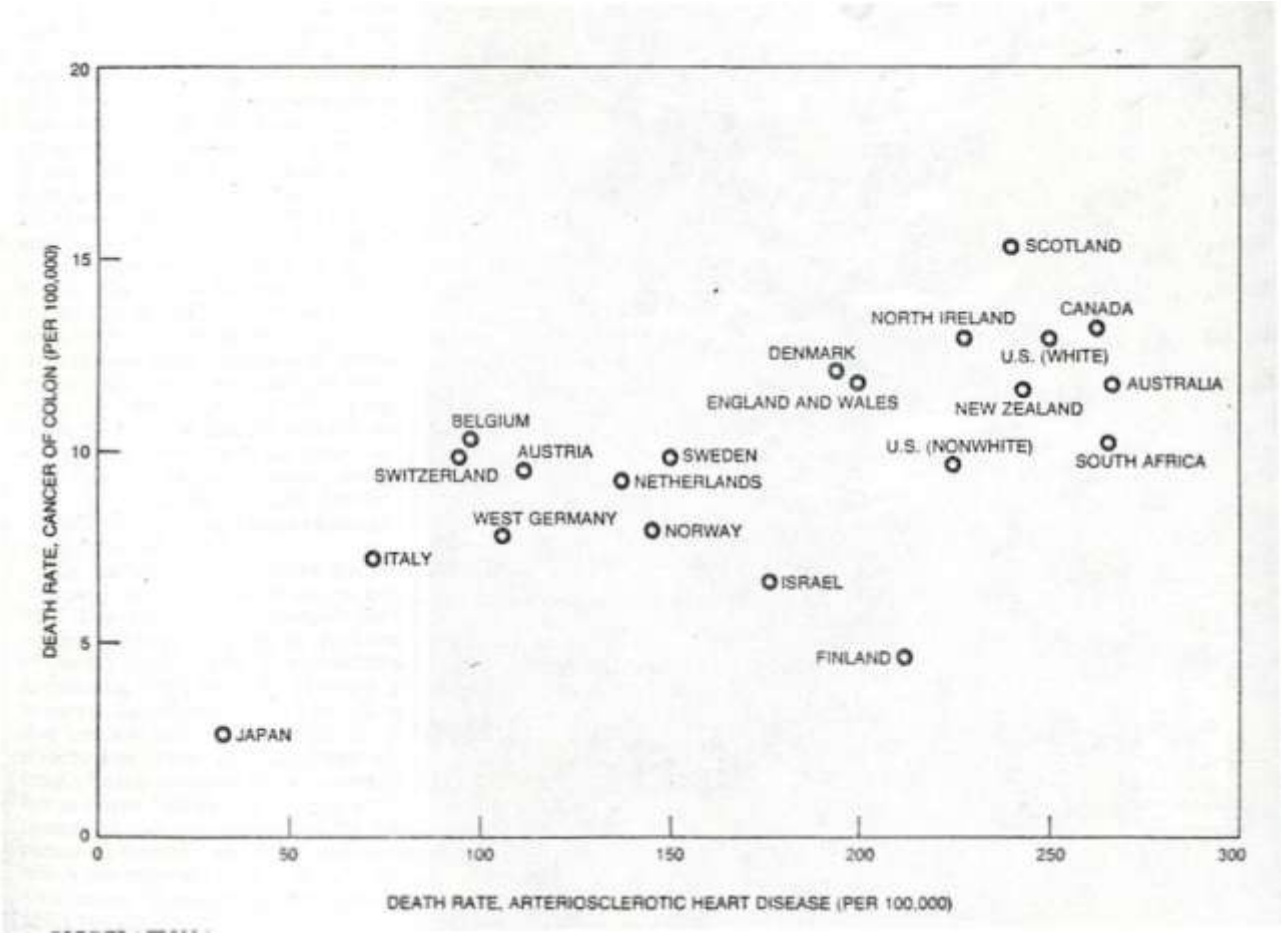
Every case of cancer, high blood pressure, coronary heart disease, Type 2 diabetes and stroke occurred in the two-thirds of people who were more insulin-resistant, as shown by their *Steady State Plasma Glucose* (SSPG) levels. SSPG is measured by giving *somatostatin* (an inhibitory hormone that suppresses pancreatic insulin release), while giving insulin and glucose intravenously. Everybody ends up with the same amount of insulin, so that the glucose level becomes a measure of how sensitive or resistant they are to insulin's effect without any potentially confounding changes in β-cell performance; thus, it is a pure measure of insulin resistance. The results correlate closely with values obtained with the better-known insulin-clamp technique (Greenfield 1981), but these research methods haven't filtered down to clinical practice, where at best the fasting blood sugar and fasting insulin level may be entered into the Homeostasis Model Calculator to estimate insulin resistance and β-cell performance. The more cumbersome glucose tolerance test is rarely employed, and, instead, insulin resistance is usually inferred from the symptoms it causes.

What's astounding about Dr Reaven's studies is that, firstly, *no* insulin-sensitive subject developed a degenerative disease, and secondly, that two-thirds of them – us – are sufficiently insulin-resistant to be at risk of degenerative disease. Apparently, insulin sensitivity is by far the most predictive physiological parameter for degenerative disease, far outperforming cholesterol, and two-thirds of us are sufficiently insulin-resistant to be at risk of degenerative disease by the age of 50 years. It's worth repeating: *no* degenerative disease appeared in the insulin-sensitive third! Such unequivocal results are vanishingly rare in medical research, where it is more usual to ballyhoo some trivial improvement in the death rate and suggest that statin drugs be made available to everybody. The silence which greeted Dr Reaven's results is surely because very few *drugs* address insulin resistance, and none of these are sufficiently effective to lower insulin resistance into the safe zone.

How many are insulin resistant? Another study by Dr Gerald Reaven found some 25% of healthy people are at least as insulin-resistant as people with Impaired Glucose Tolerance or Type 2 diabetes (Hollenbeck 1987); since about 47% of the population has either pre-diabetes or Type 2 diabetes, the problem is widespread. There is about a three-fold difference in sensitivity to insulin between the most insulin-resistant and the most insulin-sensitive people with normal glucose tolerance (Golay 1986). Moreover, the situation is getting worse. "Using nationally representative samples, we show for the first time that the prevalence of hyperinsulinemia increased greatly in the U.S. during the 1990s. From 1988–1994 to 1999–2002 among nondiabetic adults aged ≥20 years in the U.S., the mean fasting insulin concentrations increased by ~5.0%. Meanwhile, the prevalence of hyperinsulinemia increased by 35.1% overall (38.3% among men and 32.1% among women)" (Li 2007). Dr Chaoyang Li found that 20% of white women and 50% of Mexican American women were hyperinsulinemic in 1999-2002.

Cancer and Insulin Resistance

Studies suggest that cancer and heart disease have a common cause in insulin resistance; prospective studies have found metabolic syndrome increases risk of prostate cancer (Holme 2006) and colorectal cancer (Ahmed 2006). This would explain why mortality from cancer and heart disease are so closely related in different countries (Benditt E, Scientific American 2/1977):



Current thinking is that:

… little doubt exists about the involvement of insulin in tumoural processes. Indeed, increased insulin production (either directly by the tumour in an ectopic fashion, or indirectly by stimulation of the pancreatic secretion) is a common phenomenon during cancer development. Paradoxically, the increased production and circulating levels of the hormone are associated with a decrease in sensitivity which leads to insulin resistance in the non-tumoural tissues, resulting in hyperlipaemia and profound alterations in carbohydrate and lipid metabolism. In addition to these effects on the host, insulin can actually increase the incidence of neoplasias and promote tumour growth (Argiles 2001).

Further, "Evidence is growing that the metabolic syndrome may be a marker for a physiologic milieu of growth that encourages tumor initiation, promotion, and/or progression" (Ahmed 2006). Dr Bruce Ames of the University of California pointed out that (Ames 1998):

Approximately 40 micronutrients are required in the human diet. Deficiency of vitamins B12, folic acid, B6, niacin, C, or E, or iron, or zinc, appears to mimic radiation in damaging DNA by causing single- and double-strand breaks, oxidative lesions, or both. The percentage of the US population that has a low intake (< 50% of the RDA) for each of these eight micronutrients ranges from 2% to ≥ 20%; half of the population may be deficient in at least one of these micronutrients. Folate deficiency occurs in approximately 10% of the US population, and in a much higher percentage of the poor. Folate deficiency causes extensive incorporation of uracil into human DNA (4 million/cell), leading to chromosomal breaks. This mechanism is the likely cause of the increased cancer risk, and perhaps the cognitive defects associated with low folate intake.

Later in the abstract of the same paper, he notes that two widely available nutritional supplements, N-acetyl-carnitine and alpha lipoic acid, roll back the clock for aging rats:

In old rats mitochondrial membrane potential, cardiolipin [a vital component of mitochondrial membranes] levels, respiratory control ratio, and overall cellular O_2 consumption are lower than in young rats, and the level of oxidants (per unit O_2) is higher. The level of mutagenic aldehydes from lipid peroxidation is also increased. Ambulatory activity declines markedly in old rats. Feeding old rats the normal mitochondrial metabolites acetyl carnitine and lipoic acid for a few weeks restores mitochondrial function, lowers oxidants to the level of a young rat, and increases ambulatory activity.

In addition, the aged rats' memories improved almost to the level of young rats. Dr Ames started a company to market this combination of nutrients in a pill called Juvenon for human consumption.

The Västerbotten Intervention Project revealed the strong link between blood sugar and cancer. Fasting and post-glucose load blood sugars were measured in 64,597 people, who were followed for ten years. The relative risks between the highest and the lowest quartiles of blood sugar were 2.49 for cancer of the pancreas, 2.16 for malignant melanoma, 1.86 for endometrial cancer and 1.69 for cancer of the urinary tract (Stattin 2007).

Insulin Resistance and Syndrome X, the Metabolic Syndrome

What happens when you're insulin resistant? Dr Reaven pointed out that everybody gets higher insulin levels, some people get a fat tummy, some get high blood pressure or risky changes in their blood fats (high triglycerides, low HDL-cholesterol), and a few get elevated glucose levels. He called this cluster of symptoms Syndrome X (Reaven 1988). The Syndrome X cluster of symptoms are each associated with increased risk of heart disease, and the National Cholesterol Education Program has recognized this and moved to claim it as its own by renaming it Metabolic Syndrome (Reaven 2004):

Clinical Identification of the Metabolic Syndrome – Any 3 of the Following:

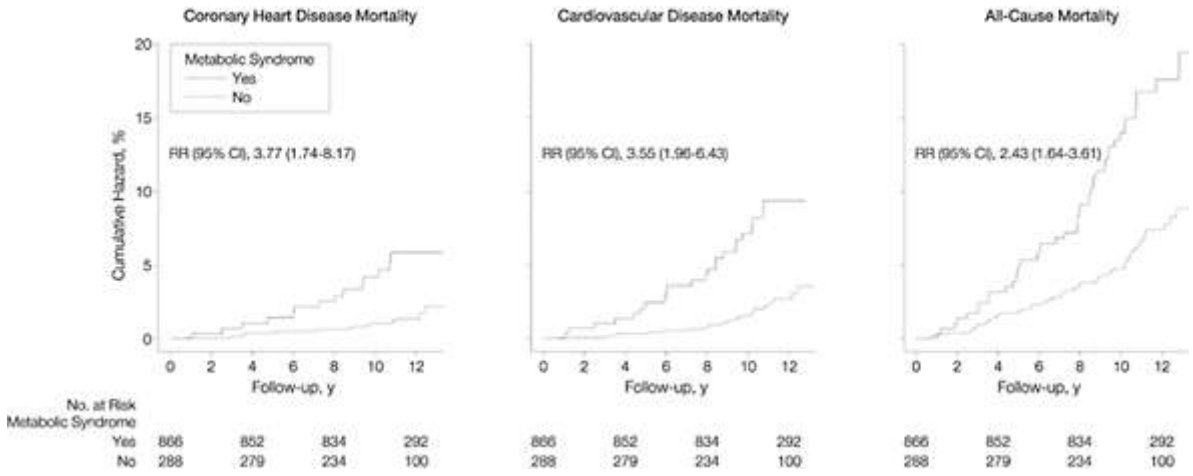
Risk Factor	Defining Level
Abdominal obesity*	Waist circumference†
Men	>102 cm (>40 in)
Women	>88 cm (>35 in)
Triglycerides	≥150 mg/dL
HDL cholesterol	
Men	<40 mg/dL
Women	<50 mg/dL
Blood pressure	≥130/≥85 mmHg
Fasting glucose	≥110 mg/dL

ATP III Guidelines At-A-Glance

Having any three of these means Metabolic Syndrome: "The probability of developing Cardiovascular Disease or Diabetes II over 20 years increased from 11.9% in those with no abnormalities to 31.2% in those with 3 abnormalities to 40.8% in those with 4 or 5 abnormalities" (Wannamethee 2005). Another study found the relative risk of developing Type 2 Diabetes over the 8 years of the study was 6.92 compared to people who did not have the Syndrome (Wilson 2005). The incidence of Metabolic Syndrome is more than 40% by the age of 70 years in America (Ford 2002).

Other conditions associated with insulin resistance include abnormalities of fibrinolysis (clots do not readily dissolve), early menarche, acanthosis nigricans (velvety patches of skin associated with tumors of the gut), myopia, tallness, skin tags, acne, polycystic ovarian disease and male-pattern balding (Cordain 2003). Further, hypercoagulability (increased clotting) of the blood and vascular inflammation can be added to the list (Gogia 2006).

Is the Metabolic Syndrome important? Just how important may be gleaned from the fact that 50% of men with CHD had *none* of the conventional risk factors (Futterman 1998). Further, only 34% of men with CHD had hyperlipidemia, and even *high*-dose statin therapy is associated with a 25% recurrent event rate after just 2.5 years (Ridker 2005). In other words, the usual medical checkups miss half of those at risk of CHD, and the gold standard of preventive care following a heart attack fails quickly in one of every four patients. Pathetic, really. A diagnosis of Metabolic Syndrome certainly predicts shortened survival (Lakka 2002):



The right hand graph shows that the risk of dying 12 years after diagnosis was 20%. This prospective study of 1209 Finnish men found that one in five were dead 12 years after their diagnosis with Metabolic Syndrome, compared with only one in 14 without it. Heart deaths and deaths from stroke and hypertension claimed 6% and 8% of them, respectively, and those with metabolic syndrome were between 3.3 and 4.2 times more likely to die of CHD. This risk was independent of conventional cardiovascular risk factors: LDL-cholesterol, smoking, or a family history of CHD.

Dr Reaven has pointed out that some of the National Cholesterol Education Program's risk factors aren't very good at identifying insulin resistance. He divided 403 subjects into three groups based on their SSPGs (the measure of insulin resistance explained above), defining the third with the highest values insulin-resistant. BMI (which is closely related to abdominal obesity), HDL-cholesterol and fasting glucose identified only 43% of the most insulin-resistant third; and while glucose plus other risk factors found 100% of insulin-resistant subjects, there were only three of them in the study group. The various combinations of BMI, blood pressure, triglycerides and HDL-cholesterol were most effective, finding 65% to 78% of the most insulin-resistant third of subjects, but I dare say they likely identified virtually 100% of the top *two*-thirds of insulin-resistant subjects in danger of degenerative disease.

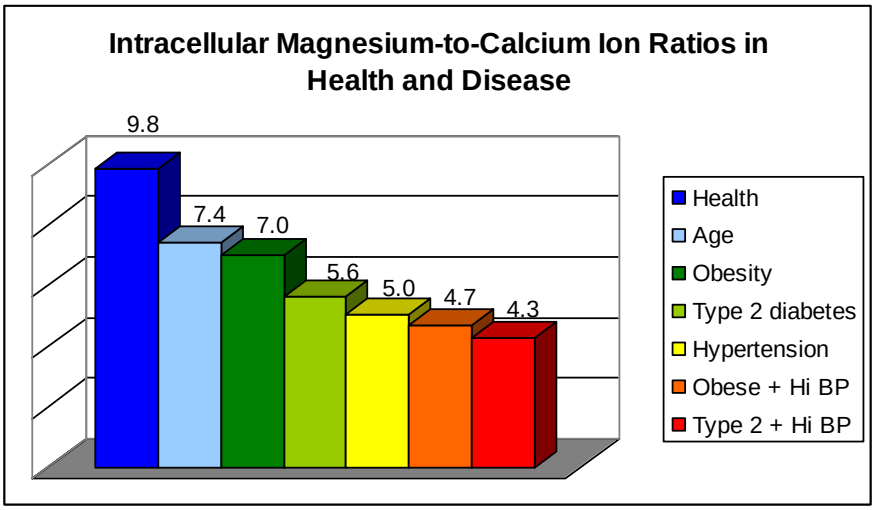
Nevertheless, Dr Reaven blessed the initiative, writing that "the greatest benefit from the introduction of the ATP III criteria may be emphasizing the fact that there are important CVD risk factors beyond hypercholesterolemia" (Cheal 2004). This is heartening news to those among us who do not believe hypercholesterolemia has much predictive value.

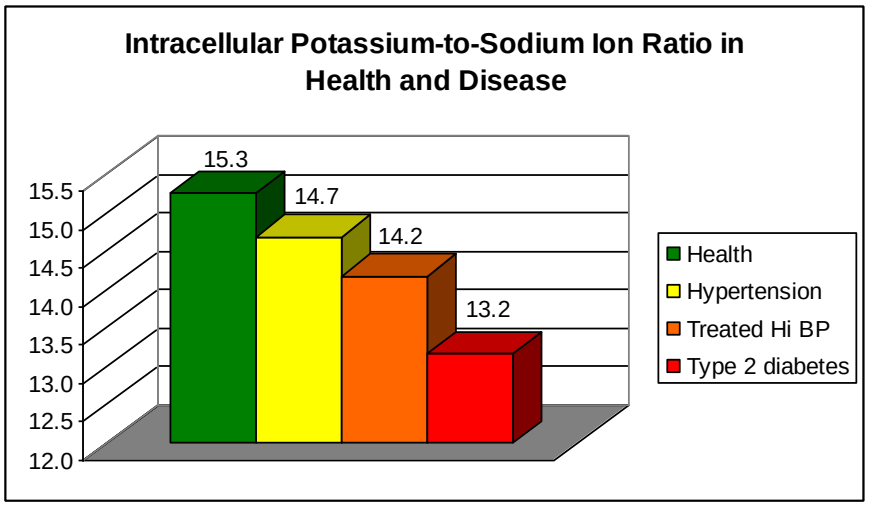
The causes of Insulin Resistance

When magnesium levels fall and calcium accumulates in cells, they become insulin resistant. Cells work hard to pump calcium out, and calcium entering the cell is usually the signal for the cell to do its thing – muscle cells contract, nerve cells conduct, the β-cells of the pancreas release insulin, and so forth. Then the cell pumps the calcium out and the muscle cell relaxes, the nerve cell readies itself to fire again and the β-cells prepare more insulin. As cells accumulate calcium for whatever reason, they become more resistant to insulin's action. Further, muscles cells no longer relax completely, which leads to high blood pressure if the muscle cells are in the muscular wall of the arterial system. Nerve cells become more excitable, which we may experience as increased aggression, anxiety and/or insomnia. β-cells over-secrete insulin, contributing to "compensatory hyperinsulinemia", the higher insulin levels necessary to get glucose into the cells in the face of insulin resistance. This is Dr Lawrence Resnick's "ionic hypothesis", here presented in relation to hypertension:

> Two central concepts of human hypertensive disease remain poorly understood: (1) elevated blood pressure as merely one component of an underlying systemic condition, characterized by multiple defects in diverse tissues (eg, "Syndrome X"), and (2) the heterogeneity of hypertension, in which different and even opposite clinical responses to different dietary and drug therapies are routinely observed among equally hypertensive subjects. To help explain these clinical phenomena, a unifying "Ionic hypothesis" is proposed, in which steady-state elevations of cytosolic [the fluid inside the cell] free calcium and suppressed intracellular free magnesium levels, characteristic features of all hypertension, concomitantly alter the function of many tissues. In blood vessels this causes vasoconstriction, arterial stiffness, and/or hypertension; in the heart, cardiac hypertrophy; in platelets, increased aggregation and thrombosis; in fat and skeletal muscle, insulin resistance; in pancreatic beta cells, other endocrine tissues, and sympathetic neurons, potentiated stimulus-secretion coupling resulting in hyperinsulinemia, increased sympathetic nerve activity, and so on (Resnick 1999).

Dr Resnick and his team used Nuclear Magnetic Resonance spectroscopy techniques to demonstrate these imbalances in the red blood cells of people with Type 2 diabetes (1993 i), obesity (1991), insulin resistance (1993 ii), and in aging, healthy people (2000). This is amazing – cells from healthy people contain little calcium and sodium, and plentiful magnesium and potassium, but cells from people with the Syndrome X cluster of symptoms are physiologically older than their years, or, to put it another way, Syndrome X is a disease of accelerated aging:

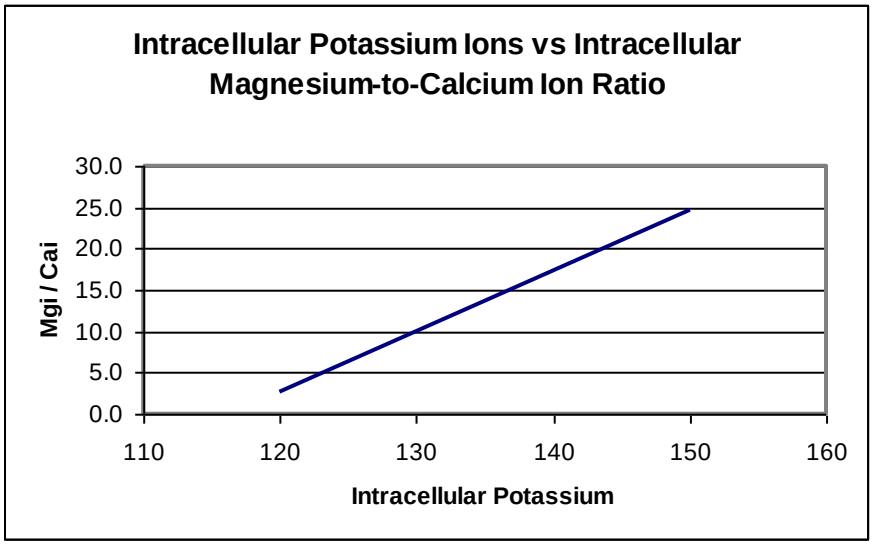




Left: Cells from healthy controls contained more magnesium and less calcium than cells from age-matched obese people, diabetics and hypertensives; obese and diabetic hyptertensives had even lower ratios. In other words, the more of the Syndrome X cluster, the lower the ratio of intracellular magnesium and calcium (Resnick 1991).

Right: Cells from healthy controls contained more potassium and less sodium than cells from age-matched diabetics and hypertensives; hypertensives treated with potassium-wasting diuretics were in worse shape than untreated hypertensives.

Furthermore, the disturbances in the magnesium-to-calcium ratio and the concentration of potassium ions within the cells were directly related, which suggests that cellular potassium ion content may modulate the cell magnesium-to-calcium ratio. (Resnick 2001):



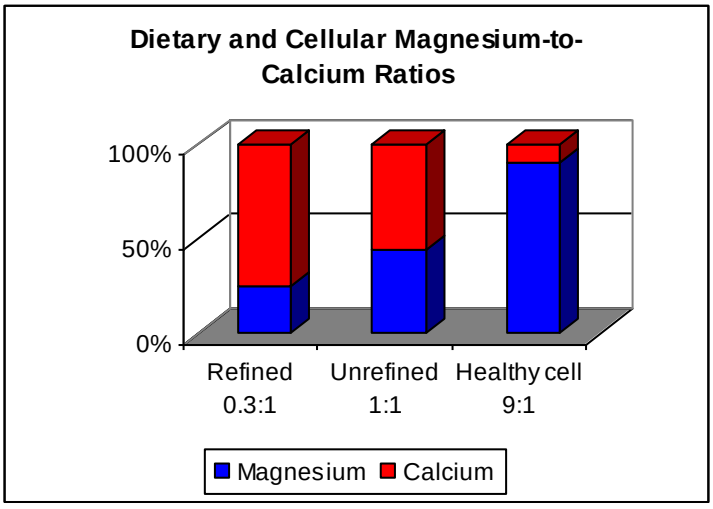
Dr Resnick made the experiment of infusing saline into diabetics and hypertensives (who had lower cellular potassium before the infusions than the normal control subjects did), and measuring the ion concentrations within their cells directly afterwards using nuclear magnetic resonance techniques. He found that saline infusion elevated sodium and lowered potassium and magnesium, while calcium increased. "We conclude that (1) potassium depletion is a common feature of essential hypertension and Type 2 Diabetes, (2) treatment of hypertension [by drugs] at least partially restores potassium levels toward normal, and (3) fasting steady-state potassium levels are closely linked to calcium and magnesium homeostasis" (Resnick 2001).

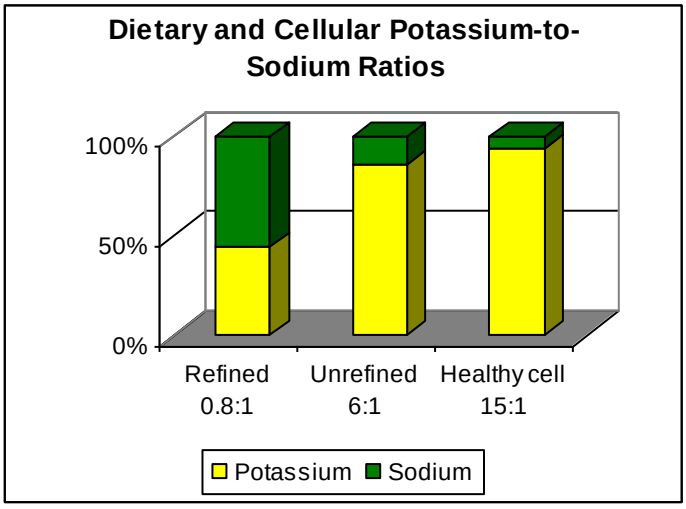
So the high-sodium Western diet raises sodium and calcium within our cells and lowers potassium and magnesium. In consequence:

> Under different genetic and/or environmental influences, hypertension or diabetes may predominate clinically, each associated with a predisposition towards the other on an intracellular ionic basis ... Thus, hypertension, peripheral insulin resistance, and hyperinsulinemia may be different clinical manifestations of a common underlying cellular defect in divalent ion metabolism (Resnick 1989).

Sadly, Dr Resnick died of pancreatic cancer in 2005, at the tender age of 55 years. Nevertheless, his elegant notion weaves together the seemingly unrelated threads between our diet and the degenerative diseases so many of us develop. Unfortunately, the remedy of lowering the amount of refined foods involves no drugs, making it unattractive to the usual sources of research funding.

Interestingly, the mineral ratios in *un*refined diets are much closer to that within the healthy cell than the ratios in refined diets:





This means that the cellular mechanisms that maintain the ion gradients across the cell wall have far less work to do if the diet is unrefined. Since cells expend a great deal of energy in this endeavor, this is likely important.

Dr Resnick concluded that "the depletion of cellular K_i [intracellular potassium ions] or Mg_i [magnesium ions], and/or the Ca_i [calcium ion] excess can each directly produce or predispose to vascular smooth muscle contraction, increased constrictor tone, increased blood pressure, insulin resistance, and abnormalities of glucose and insulin metabolism. … Although the causal mechanisms of these ionic changes have yet to be defined, the observation here of K_i depletion in hypertension and diabetes and its strong linkage with Mg_i and Ca_i levels further supports the notion of increased vasoconstrictor tone and insulin resistance as different tissue manifestations of a common cellular ionic defect" (Resnick 2001). A low-*potassium* diet slows the sodium pump because it cannot cycle until its potassium receptors are filled which takes longer if potassium is in short supply. Slowing of the sodium pump was found in a study of obese subjects, and the number of sodium pumps on each cell was reduced in direct proportion to the subject's degree of obesity (De Luise 1980). Worse, the number of sodium pumps drops by up to 80% if potassium is low (Hsu 1990), and, incredibly, less than 3% of Americans get an Adequate Intake of potassium (USDA 2005).

Further, Dr Resnick established that the higher the intracellular calcium and the lower the magnesium, the less cells responded when called upon to do their thing. Thus, the aged muscle cell is more contracted at rest (contributing to high blood pressure when the muscle cell affected is in the arterial wall) and easier to trigger, but its calcium level rises less and the magnesium level hardly at all when the cell is called upon to contract by calcium infusion from the nerve that innervates it. Consequently, its contraction is weaker, which is to say we weaken with age. But consider the implications: the weakness of age may be prevented if calcium is not permitted to accumulate within the muscle cells.

That tissues accumulate calcium with age in the West is well-established (VRP):

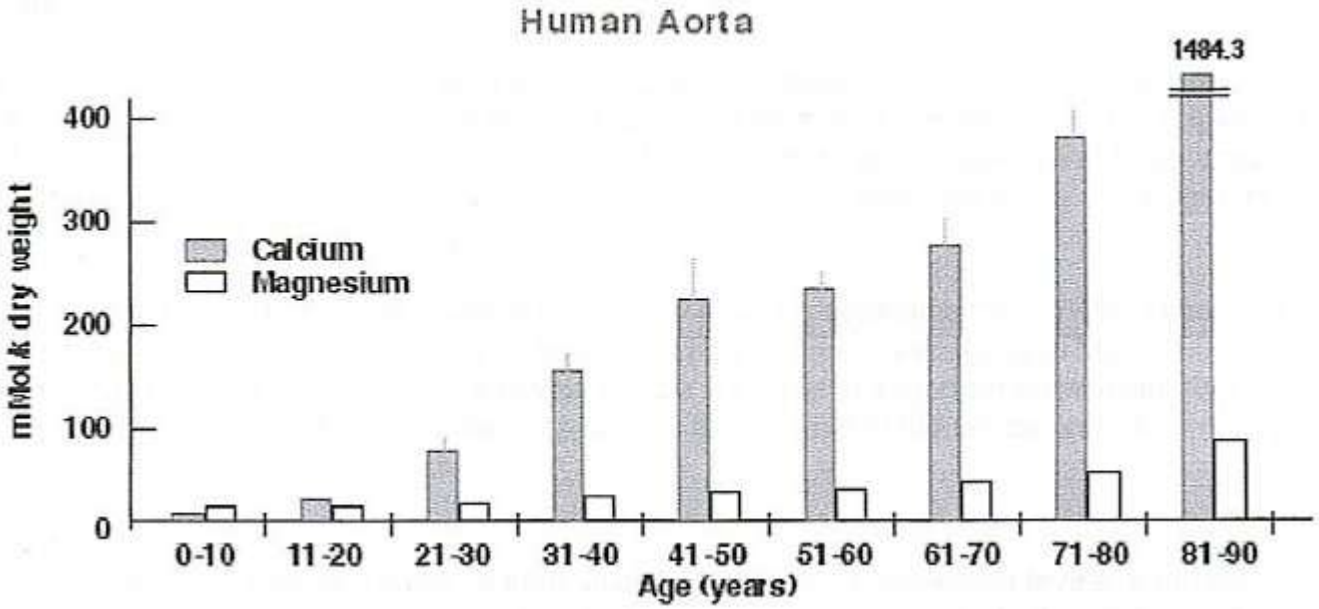


Fig 3. Effects of age on tissue levels of calcium and magnesium in human aorta (Fleckenstein 1983)

Dr Fleckenstein (who created this remarkable graph) developed "calcium channel blocker" drugs for the pharmaceutical industry. He wrote:

The formation of conventional human coronary artery plaques is characterized from the very early lesion onwards by a progressive local uptake of calcium, finally leading to lethal consequences. Conversely, the analysis of the mural [meaning "in the arterial wall"] cholesterol does not allow to discriminate arteriosclerotic from normal coronary artery segments. Thereby, conventional human coronary plaques typically represent a calcium-dominated type of human arteriosclerosis … Moreover, the antiarteriosclerotic effects of calcium antagonists are demonstrated to be based – in various types of experimental arteriosclerosis – on the inhibition of intra- and extracellular calcium overload of arterial walls evoked by various risk factors (vitamin D3 intoxication, hypertension, nicotine, diabetes) (Fleckenstein-Grun 1991).

Dr Fleckenstein believes that calcium accumulation is more important than cholesterol in atherosclerosis (Fleckenstein 1992), which certainly accords with my understanding of cholesterol's importance in atherosclerosis. And, interestingly, reversing calcium accumulation increases sensitivity to insulin. For example, Dr Rhian Touyz tested two calcium channel blockers for 12 weeks in hypertensive diabetics. Both drugs lowered blood pressure, and "Both calcium channel blockers equally decreased fasting serum insulin levels (from 138 pmol/L before therapy to 106 pmol/L after therapy)" (Touyz 1995). In other words, calcium channel blockers lowered calcium within the cells and increased insulin sensitivity by 21%, so calcium accumulation within the cell is likely *causally* related to insulin resistance. This is an important distinction – cholesterol's non-causal relationship with heart disease has derailed research efforts for the last fifty years.

Dietary faults and Insulin Resistance

So what causes calcium to enter the cells and not be pumped out? Unsurprisingly, the very same dietary faults that are associated with insulin resistance!

First, the low calcium and magnesium content of the America diet makes a direct contribution: low magnesium lets calcium "leak" into the cell, and a low calcium intake causes calcium to be released from bone by a mechanism that also lets calcium into the cell.

Second, the high sugar content of the diet not only increases calcium and magnesium losses, it also causes chaos within cells. The fructose found in High Fructose Corn Syrup and table sugar has no "rate-limiting step" in its metabolic pathways and does not need insulin's help in entering the cell. The cell is asked to metabolize all fructose at once but it can't do it, so toxic intermediate products build up when necessary molecules such as phosphate run out; and a shortage of phosphate ions slows the *sodium pump*. These cell-wall molecules pump sodium out of and potassium into the cell, using the energy of the cellular "gasoline" molecule, ATP; they use between 25% (for a muscle cell) and 75% (nerve cells) of all the cells' energy. They are the cells largest single energy expense, and they maintain a potassium concentration within the cell 30-40 that of the serum, and a sodium concentration 14 times less than the serum; and this "sodium gradient" powers a pump which pumps calcium into compartments within the cell, and others which pump it out of the cell. Together with ATP-powered calcium pumps, a healthy cell achieves a 10,000 times concentration gradient between its interior and the serum outside its wall.

Thirdly, the fat content of a mixed meal pours triglycerides into the bloodstream from the gut just as the cells process fructose into triglycerides, leading to high triglycerides with only one place to go: fat cells. It is easy to over-consume our refined diet, but with the unrefined diets of Kitava, citizens remain slim despite an abundance of calories. Consequently, sugars together with fat are strongly associated with dyslipidemia and overweight. We have declared war on fat but not on sugar, and it is slowly becoming clear that we picked the innocent bystander.

Finally, the elevated sodium and depressed potassium content of the diet directly impede the activity of the cells' sodium pumps. The low potassium-to-sodium ratio of the Western diet seems especially strongly linked to the development of high blood pressure.

Low calcium

Counter-intuitively, a *low*-calcium diet increases intracellular calcium *via* the action of parathyroid hormone which mobilizes supplies from bone, and correcting this dietary fault with "a short period (8 weeks) of high calcium intake (2 g/d) reduces intra-platelet free calcium concentration and positively affects glucose metabolism, increasing insulin sensitivity and decreasing fasting plasma insulin" (Sánchez 1997). Accordingly, it was "recently demonstrated … that low calcium diets … stimulate Ca^{2++} influx in human adipocytes [fat cells] and thereby promote adiposity" (Zemel 2002). And a double-blind, placebo-controlled, randomized trial (the best kind) of calcium supplementation in 780 women showed "Significant negative associations between calcium intake and weight were found for all three age groups, and the odds ratio for being overweight (body mass index >26) was 2.25 for young women in the lower half of the calcium intakes of their respective study groups ($P< 0.02$) (Davies 2000). Importantly, intracellular calcium falls during weight loss (Jacobs 1993), which probably accounts for part of the salutary effect of weight loss on insulin resistance and blood pressure.

Research has further shown that low dietary calcium exaggerates the effect of a poor potassium-to-sodium dietary intake (Hollenberg):

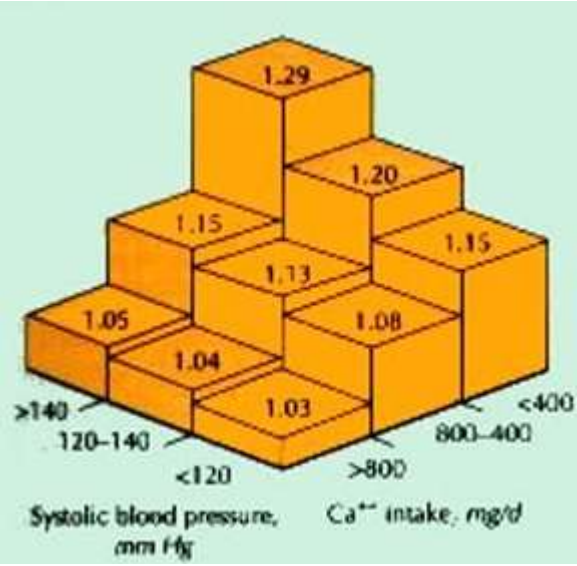


Figure 1-35: Dietary factors may interact to promote hypertension. The effect of dietary sodium and potassium on blood pressure may be conditioned by the contemporaneous intake of calcium. In this survey, continuous and graded relationships between blood pressure and dietary calcium and the ratio of dietary sodium to potassium intake [numbers inside each bar] were found. Low calcium intake and an increased ratio of sodium to potassium intake were both associated with higher systolic blood pressure; the combination of both dietary habits was associated with the highest systolic blood pressure. Note that expressed as Potassium:Sodium ratio, the values fall between .97 and .77, while Dr Moore suggests a value of 2 or more for protection against hypertension. Values are adjusted for age, body mass index and alcohol intake. Adapted from Gruchow 1988, who was at pains to point out that "Together, the correlates of blood pressure measured in this study including Na:K, accounted for ~one-fourth of the total variances in blood pressure." Which leaves plenty of room for magnesium, omega-3 fatty acids, sugar, phosphate and so on.

In America, only 12% of adult women and 37% of men get the Adequate Intake of calcium from their diets; worse, only 5% of women over 50 achieve the Adequate Intake (USDA 2005).

Low magnesium

A low-*magnesium* diet allows calcium into the cells because magnesium is a little larger than calcium and blocks the calcium channel. If magnesium is in short supply, the cell membrane "leaks" more calcium into the cell: "This calcium antagonism acts centrally on both muscle and nerve cells: the magnesium ion occupies the calcium binding sites on muscle cells, thereby displacing calcium …" (EXAtest). Magnesium is so effective in this role that it has been called "Nature's physiologic calcium blocker" (Iseri 1984), and its effects compared directly and favorably with calcium-channel blocker drugs (Altura 1984). I believe this effect is responsible for my aorta calcium score of zero, as determined by Computerized Axial Tomography:

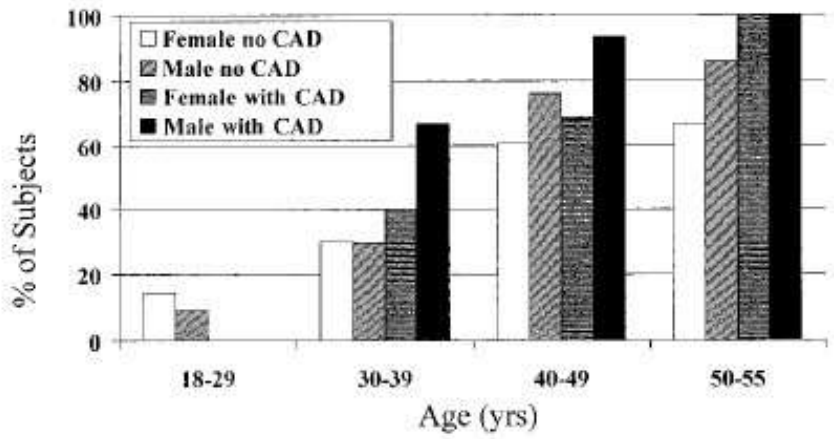


FIG. 1. Prevalence of CAC by age, sex, and clinical CAD status.

The prevalence of any Coronary Artery Calcium among 302 insulin-dependant diabetics increased with age from 11% before age 30 years to 88% in individuals aged 50–55 years (Fig. 1). In other words, CAC is almost universal in insulin-dependant diabetics aged over 60, like me. Calcification was more common in subjects with clinical Coronary Artery Disease (77%) than in those without CAD (39%; $P < 0.0001$). CAC was detected in all subjects with CAD aged ≥50 years. In subjects with the same CAD status, there was little sex difference in CAC prevalence at every age (Olson 2000).

This study suggests that coronary artery calcium is almost universal in insulin-dependant diabetics aged over 60, like me, so my lack of aortic calcium was a surprise to my doctor who said "We think it's genetic!" However, my wife's aorta calcium score was also zero, but her family history of heart disease makes a genetic invulnerability unlikely. The thing we have in common is 20 years of a high-magnesium, whole food diet.

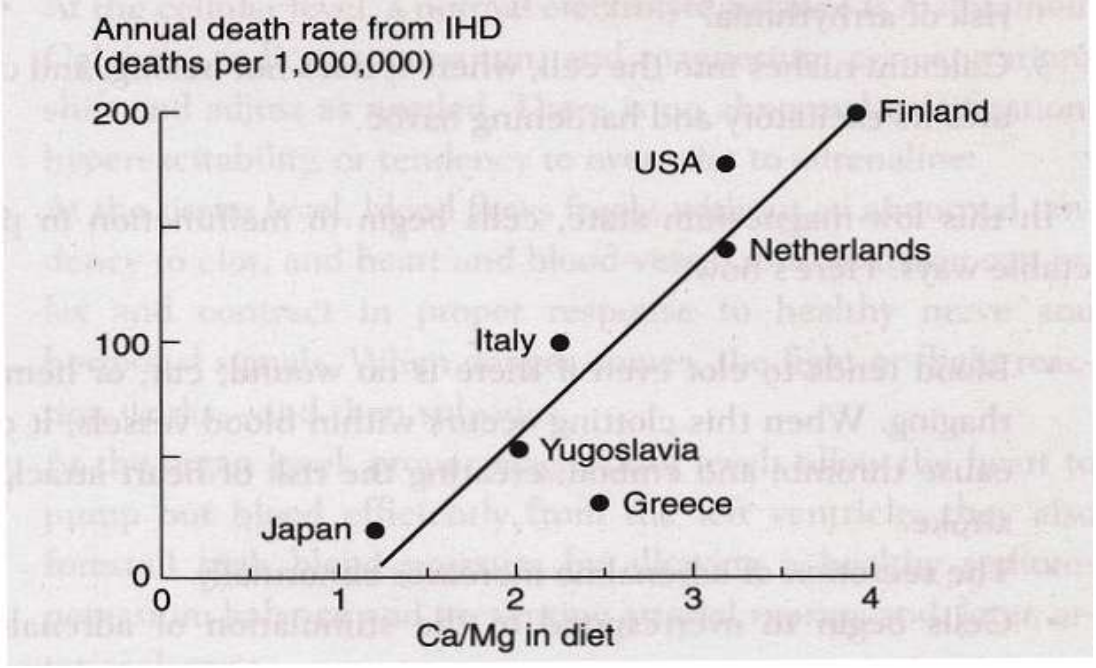
Low magnesium diets are associated with more 21% more Type 2 diabetes in Black women (van Dam 2006), and 40% more CHD in men (Al-Delaimy 2004), yet 67% of American adults eat diets containing insufficient magnesium (USDA 2005). A study in which eight elderly diabetics were supplemented with magnesium suggests the relationship is causative, for their insulin resistance fell by almost twenty percent:

… magnesium supplementation to diet versus placebo produced 1st a significant increase in plasma (0.83 vs. 0.78 mM, P<.05) and erythrocyte (2.03 vs. 1.88 mM, P<.01) magnesium levels, 2nd an increase in acute insulin response (AIR) (4.0 vs. -1.6 mU/L, P<.05) to glucose pulse, and 3rd an increase in glucose infusion rate [a measure of insulin resistance] (3.6 vs. 2.9 mg.kg-1.min-1, P<.025) calculated in the last 60 min of a euglycemic-hyperinsulinemic (100 mU.m2.min-1 during 180 min) glucose clamp (Paolisso 1989).

Nevertheless, magnesium just isn't very interesting to the research establishment. The 10th Edition of the Institute of Medicine's RDAs said of magnesium that "Although dietary surveys indicate that magnesium intakes of some segments of the population are lower than current recommendations, there is no unequivocal evidence that magnesium deficiency is a problem among healthy persons in this country" (1989). Not much has changed since.

Notwithstanding, it's clear that not only are the levels of calcium and magnesium in the diet are important, the ratio of calcium to magnesium in the diet appears to have a direct and compelling relationship with the rate of heart disease in different countries. The strength of this association is remarkable:

Figure 1.4 Ischemic Heart Disease Rates Correlated with Dietary Calcium/Magnesium Ratios



This figure (from Karppanen H et al, *Adv Cardio* 1978; 25(19/8):9-24) shows a direct relationship between the incidence of death from ischemic heart disease and the ratio of calcium to magnesium in the average

diet [note the correlation is very high at 0.90]. It is interesting to note that Finland, which was the highest of all countries in heart-disease deaths in 1977, is now down to tenth in heart-disease deaths. What did they do? They undertook two important public health measures. First, they worked to educate the population about the importance of reducing dietary salt and fat and eating more vegetables and fruit – foods rich in magnesium and potassium. At the same time, they made available a magnesium-containing salt product that contained less sodium than standard table salt but still had a desirable salty taste, and promoted the use of this product. This high-magnesium salt was also sold to producers of sausage and other processed foods as a replacement for standard salt. Finns in general like salty food, and they accepted foods labeled as containing the new salt in preference to foods labeled low-salt (Mildred Seelig, *The Magnesium Factor*).

Historically, measuring levels of ionized magnesium in the body was very difficult. Less than 1% of the body's magnesium is in the bloodstream, and the body regulates this serum magnesium very tightly. Sixty percent of magnesium in the body is sequestered within bone, and the 39% in cells could, until recently, only be measured directly with expensive Nuclear Magnetic Resonance techniques. The "magnesium loading test" involves giving a magnesium supplement and then measuring the amount excreted in a 24-hour urine sample, and is understandably little-used. A new test, the EXA Test, uses skin cells scraped from the floor of the mouth which are bombarded with X-rays and the spectrum examined to discover the amounts of minerals present within them; the main thrust of applications is "cardiovascular conditions, arrhythmias, heart failure, myocardial infarction, and bypass surgery" (Silver 2004). In the guide to interpreting the results, the following appears (EXA Interpretation):

MAGNESIUM: Desirable Intracellular Reference Range: 33.9-40 mEq/l

Magnesium modulates tissue transport of calcium and potassium ions and participates in hundreds of enzyme systems including formation of high-energy compounds such as ATP. All physiological activity, secretion, bone formation, cardiac and neuromuscular activity is affected by magnesium in tissues. Optimal tissue levels of magnesium prevent cardiac irregularities and tend to maintain lower blood pressure. Magnesium concentrations in optimal ranges indicate possible lowered risk factor for hypertension, angina, arrhythmias and vascular spasm. … LOW magnesium has been associated with EKG and cardiac abnormalities, fibrillation, vascular and muscle spasms. Correlations with migraine headaches, asthma, eclampsia, PMS, and chronic fatigue syndrome are abundant in the medical literature. Low magnesium is seen in cardiac failure and prolonged QT syndrome. Neurological disorders, panic attacks and nerve irritability have been associated with low tissue magnesium levels.

CALCIUM: Desirable Intracellular Reference Range: 3.2-5.0 mEq/l

Calcium is involved in secretory functions of tissues at the cellular level. Neurotransmission and neuromuscular transmission require regulated calcium movements. Structure of the supportive tissues, bone and cartilage, involves normal calcium metabolism. Serum calcium is narrowly regulated by endocrine secretions. Intracellular calcium may vary much more than serum levels and tissue imbalances can cause a variety of syndromes affecting bone/tooth formation, blood clotting, heart rhythm, and permeability of cell membranes. Elevated tissue calcium may be a sign of mobilization of bone calcium into soft tissues signaling early signs of developing osteoporosis. … HIGH intracellular calcium interferes with ATP formation, muscle contraction, relaxation, enzyme activity, and neuromuscular transmission. Increased cellular calcium predisposes to spasm of peripheral arterioles leading to increased blood pressure. Calcium may also be a factor in plaque formation, angina, hypertension and athero-arteriosclerosis. Calcium channel blockers as well as magnesium affects movement of calcium into the soft tissues and heart muscle.

Intracellular Electrolyte Ratios: MAGNESIUM/CALCIUM
Desirable Intracellular Reference Range 8-10.6

As this ratio lowers, cardiac risk factors increase and ATP production decreases. Serious pathology such as calcinosis, atherosclerosis, vascular occlusion, acute myocardial vasospasm or infarction with related arrhythmias is believed to be related to such imbalances.

Intracellular Electrolyte Ratios: POTASSIUM/SODIUM
Desirable Intracellular Reference Range: 15-63

Active transport of K and Na produces major energy processes, normal cell volume, and is vital to ion transport, as well as producing the membrane potentials for all secretory functions, neurotransmission, and neuromuscular activity. Serum potassium levels are not good indicators of tissue levels. This ratio is vital to establishment of homeostasis for normal function of intracellular biochemical events.

The desirable range of the Magnesium/Calcium ratio, calculated from the desirable Intracellular Electrolyte Ranges, lies between 8 and 10.6, which agrees with the value of 9.8 found for healthy control subjects in Dr Lawrence Resnick's NMR determinations. It seems that determinations of intracellular ions in epithelial cells and red blood cells agree, and other studies suggest they predict levels in liver, muscle and fat cells.

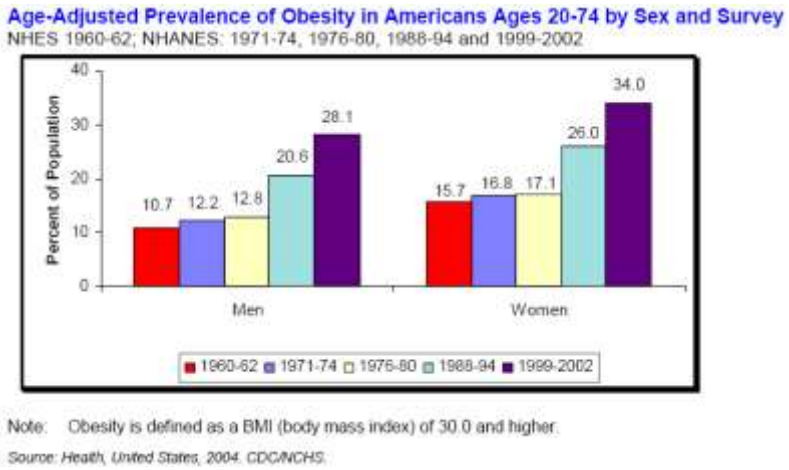
Sugar wastes magnesium

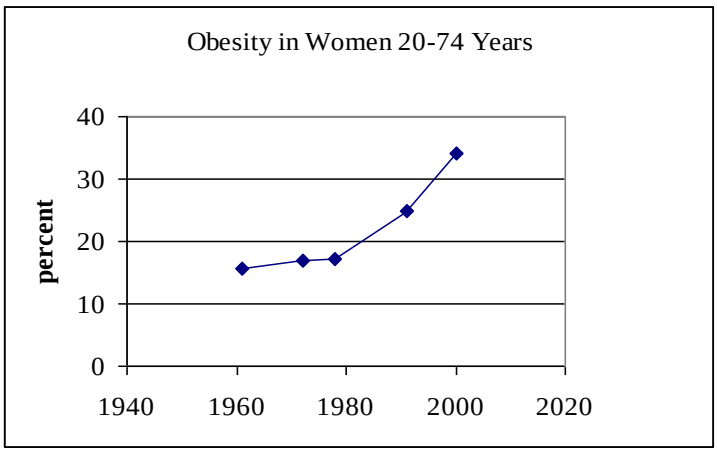
Interestingly, sugar intake causes magnesium and calcium to be lost in the urine. Sugar is well-known to cause approximately equal amounts of magnesium and calcium to be lost in the urine (Ericsson 1990), so much so that in a study of sugar intake and calcium excretion, those who ate little sugar were in negative calcium balance by 40mg per day, and those who ate the most lost 100mg per day (Thom 1978). Further, alcohol "acts acutely as a Mg [magnesium] diuretic, causing a prompt, vigorous increase in the urinary excretion of this metal along with that of certain other electrolytes [including calcium]" (Rivlin 1994), and the chronic hyperglycemia experienced by diabetics also causes the loss of magnesium in the urine (Mather 1979). Given that only 32% of Americans eat a diet adequate in magnesium and the American diet contains an average of 187 grams of sugars per day, the number of Americans actually getting the magnesium their metabolisms require to work properly must be very low indeed.

Thus, a high sugar intake exacerbates the low calcium and magnesium intake found in the already over-refined American diet. Consequently, sugars have deleterious effects on the metabolism: where there's sugar, there's obesity and diabetes.

Sugar is associated with obesity, impaired glucose tolerance and disturbed blood fats

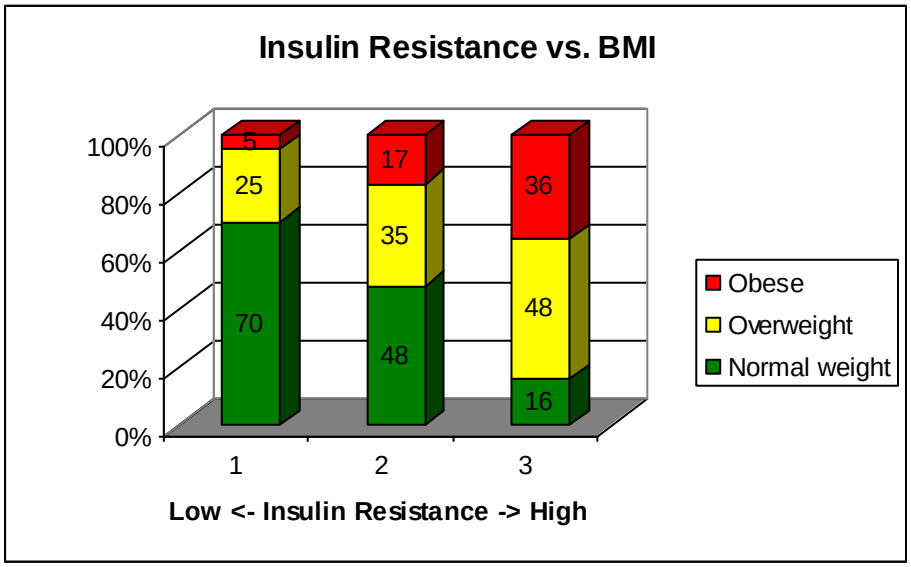
The prevalence of obesity has exploded in the past decades:





The figure on the left is from the Center for Disease Control analysis of the National Health and Nutrition Evaluation Surveys, and this data is re-plotted at right to show more clearly that the explosive rise in obesity in women began about 1980.

Insulin resistance was clearly linked to obesity in a study by Dr Gerald Reaven of 465 healthy volunteers. He found that only 16% of the most insulin-resistant third of the subjects were of normal weight (meaning a Body Mass Index under 25), compared to 70% of the most insulin-sensitive:



… [although] increased BMI is more prevalent in insulin-resistant individuals, not all overweight/obese persons are insulin resistant. Furthermore, because CVD risk factors were accentuated in association with increased degrees of insulin resistance, independently of BMI or age, it is the subset of overweight, obese individuals, who are also insulin resistant, who are at greatest CVD risk. Indeed, we have recently demonstrated that elevated levels of C-reactive protein are only increased in insulin-resistant, overweight individuals (versus insulin-sensitive, equally-overweight individuals Mclauglhin 2002) … Thus, we believe that these results provide support for the view that the most intensive efforts to decrease CVD risk should be directed towards those overweight individuals who are also insulin resistant (McLaughlin 2004).

These studies showed that the higher the insulin resistance, the higher the triglycerides, while the fatter the subjects, the higher their LDL-cholesterol, which means that insulin resistance and overweight make separate but additive contributions to heart risk. Interestingly, obesity raises the risk of death to such an extent that the usual Syndrome X prevalence-rises-with-age graph changes so that the prevalence seems to fall with age; but this is because of all the premature deaths among the obese (CDC):

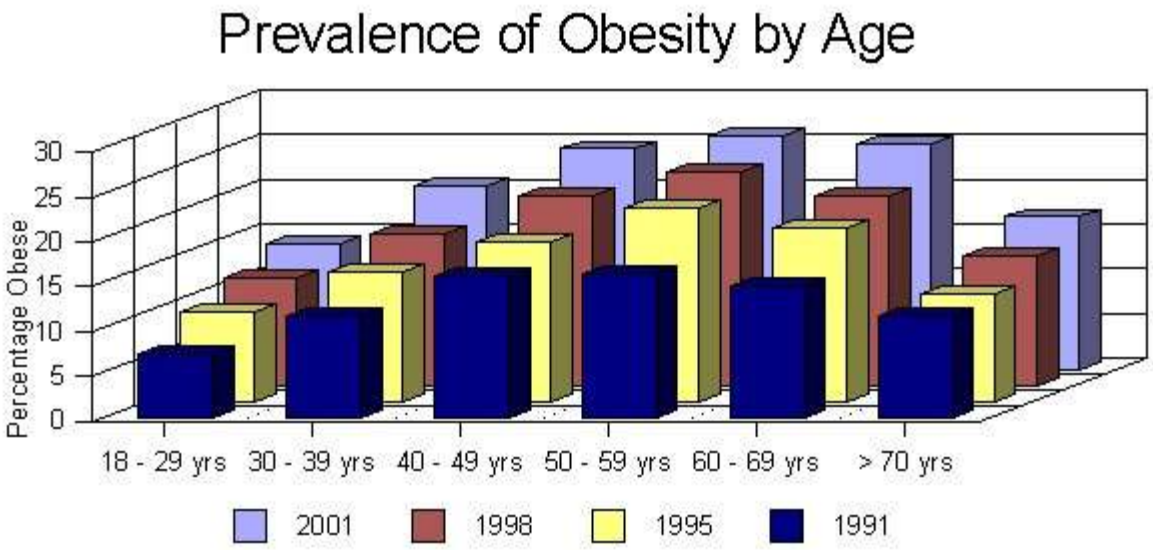


Figure1. Source of data: 1991-2001 Prevalence of Obesity among US Adults, by Characteristic; Behavioral Risk Factor Surveillance System; Self-reported data.

Estimates of the number of years of life lost as a result of overweight and obesity range as high as 20 years for certain age and racial groups. For example, a 20-year-old white male could realize a 17 percent reduction in life expectancy due to obesity (Fontaine 2003). Ominously, research published in the *New England Journal of Medicine* suggests, "From our analysis of the effect of obesity on longevity, we conclude that the steady rise in life expectancy during the past two centuries may soon come to an end" (Olshansky 2005).

It is quite clear that the problem is not dietary fat. As America's premier epidemiologist Dr Walter Willett put it, "within the United State, a substantial decline in the percentage of energy from fat during the last two decades has corresponded with a massive increase in the prevalence of obesity. Diets high in fat do not appear to be the primary cause of the high prevalence of excess body fat in our society, and reductions in fat will not be a solution" (Willett 2002). The National Health and Nutrition Survey found that "Among food groups, 'Sweets, desserts' contributed the most to energy intake. Three nutrient-poor food groups, 'Sweets, desserts', 'Soft drinks' and 'Alcoholic beverages' contributed almost 25% of all the energy consumed in the US population. Efforts to reduce obesity should focus on … actions to reduce

the importance of nutrient-poor foods in the US diet" (Block 2004).

So it's sugar then

Higher blood sugars mean more *glycation*, which is to say more sugar molecules reacting with proteins in the body. The HbA_{1c} glycosylated hemoglobin test for diabetic control uses this phenomenon: the more sugar that has reacted with hemoglobin molecules in the bloodstream, the worse the blood-sugar control over the past months. Interestingly, antioxidants lessen this free-radical process. My HbA_{1c} score is reduced by 0.4% when I take 6 grams of the antioxidant vitamin C per day, and this phenomenon has achieved scientific respectability: "We studied GHb [glycosylated hemoglobin] in subjects supplementing up to 20 g AA [vitamin C] daily and found that for each 30 µmol/L increase in plasma AA, GHb was reduced by approximately 0.1%." The researchers were concerned that this effect might "lead to a clinically relevant underestimation of average blood sugar" but acknowledged "the possibility of AA-mediated inhibition of glycation in all proteins and the [beneficial] implications for aging." (Krone 2004). Taking a gram of vitamin C every few hours should raise my plasma AA by about 120 µmol/L (Padayatti 2004), and should therefore reduce my HbA_{1c} by 0.4%, which is in remarkably good agreement. Similarly, a study in Finland found 600mg of vitamin C per day lowered the average HbA_{1c} in 56 Type 2 diabetics from 9.3% to 8.5% (Eriksson 1995).

Unfortunately, glycation negates the biological usefulness of the glycated molecule, and worse, glycated molecules stick together to form Advanced Glycation End products. The immune system has trouble removing these, so they build up to become, among other things, the amyloid plaque found in the brains of Alzheimer's victims. In the skin, cross-linked collagen molecules show up as wrinkles and age spots, and they stiffen the walls of the arteries and contribute to high blood pressure. Thus, insulin resistance accelerates aging. However, this is old news. In 1989, the *New Scientist* published an article entitled "Why sugar is bad for you" which clearly explains the biochemistry and points out how insulin resistance rises with age so that both insulin and blood sugar rises on the Western diet, and how this contributes to the development of cataracts. The conclusion? "Perhaps the best advice is to keep off sugary snacks ..."

Following this advice may prevent depression. A 2002 study of sugar consumption and the prevalence of depression in Western nations found that as sugar increases in the diet, so does depression (Westover 2002):

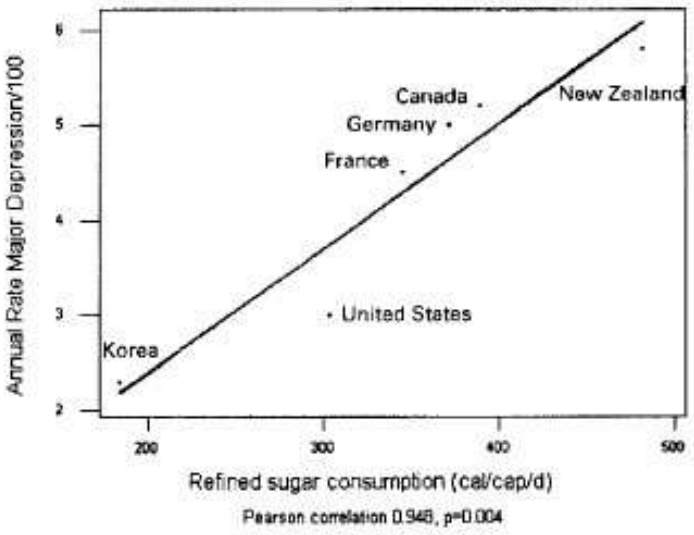


Figure 1. Refixed sugar consumption and prevalence of major depression.

The relationship is remarkably strong, although this type of study can only point out an association.

Nevertheless, studies that point up the dangers of sugar are drowned out by reviews funded by sugar trade groups that trivialize its effects. For example, a study supported by the UK Sugar Bureau concluded "No group of researchers has yet shown a convincing negative or positive effect of sucrose on insulin sensitivity *by using dynamic insulin sensitivity assessment*" (Daly 2003). In other words, more research is needed, in spite of the staggering weight of existing evidence! Moreover, amazingly, the FDA itself gave sugar a clean bill of health in a 1986 report, *Evaluation of Health Aspects of Sugars* (Glinsmann 1986). The references betray the strategy of "overlooking" studies that found fault with sugar. For example, Dr Joseph Egger's double blind, placebo-controlled study of hyperactive children found that 16% of them became worse when given sugar, and was published in *The Lancet* (Egger 1985). The study was not to be found in the reference section. The segment on "Behavior" concluded that "There is no conclusive evidence that sugar consumption causes significant changes in the behavior of children or adults."

This conclusion is tripe. Dr Stephen Schoenthaler analyzed the academic performance of the New York School System before and after foods were restricted to a maximum of 11% of calories as sugar, and some preservatives and food colorings eliminated. Before the changes, the School System's national percentile rank was about 11% below the national average, and after the changes, about 5% above the national average! This 16% change is far greater than the usual year-to-year perturbations of about 2%. Interestingly, before the changes, the more children ate in the cafeterias of the School System, the worse they performed; after the changes, the more the children ate in the cafeterias, the better they performed (Schoenthaler J et al, The Impact of a Low Food Additive and Sucrose Diet on Academic Performance in 803 New York City Public Schools. *Int. J Biosocial Research*, 1986; 8(2):185-195). This is a consistent finding. For example, a recent study of 10th grade students in Oslo found "High consumption levels of sugar-containing soft drinks [4 or more glasses per day] were associated with mental health problems [hyperactivity, mental distress, conduct problems] among adolescents even after adjustment for possible confounders" (Lien 2006).

Similarly, prison studies have shown that less sugar and better nutrition reduces violence. Sixty-two violent juveniles among a population of a "mid-western state's youthful 'chronic offenders', the 6% of the population that commits more than half the murders, rapes, robberies and assaults," were divided into two groups and counseled on healthy diet practices. One group received vitamin and mineral supplements, and violent infractions dropped 26% among them. The difference would have been far larger, but 6 of the control subjects used the counseling they received to repair their own diets, replacing calories from sugar and saturated fat with fruits and vegetables. Violent acts among them dropped dramatically, from 131 during the baseline period to 11 during the intervention period. The rate of violent acts did not change among the other 10 control subjects, whose blood-nutrient levels stayed low throughout the study (Schoenthaler 1997). Similarly, a recent British study of 231 young adult prisoners demonstrated that that supplements of vitamins, minerals and essential fatty acids reduced violence by 37% (Gesch 2002). At the start of this study, some offenders were taking up to 47% of their calories as sugar!

Therefore, it is clear that nutrient deficiencies and/or hypoglycemia caused or exacerbated by sugar and refined foods interfere with learning and promote violence. You would think such a simple interventions as cutting sugar intake to improve learning in schools and reduce violence in the prison system might be welcome, but such is not the case. Instead, the American Medical Association, the Nutrition Foundation and the International Life Sciences Institute sponsored a symposium on diet and behavior in 1984. Incredibly, major researchers such as Dr Stephen Schoenthaler who had actually conducted studies on the relationship between diet and behavior were excluded from the panel (Schoenthaler S, *Int J Biosocial Research*, 1987; 9(2):161-81). Further, the journal publishing the panel's conclusion that there was no relationship and further research was unwarranted allowed no rebuttal to be published (Pease 1986, Gray 1986)! Thus, this industry-sponsored symposium promulgated a completely bogus "consensus" opinion that diet is unrelated to behavior in the scientific literature, almost certainly to prevent the loss of schools and prisons as markets for sugar.

In addition, sugar producers have enormous political pull, as detailed in a Wall Street Journal Online article; a Washington Post article referred to the "sugar racket." A UK Guardian article detailed how the American Sugar Association pressured the World Health Organization to drop a suggestion that less than 10% of calories should come from sugar in a healthy diet in the WHO report Diet, Nutrition, and the Prevention of Chronic Diseases. In a move the Guardian deemed "worse than any pressure exerted by the tobacco lobby", the Sugar Association threatened to "exercise every avenue available to expose the dubious nature of the report", demanding that Congress end funding of the WHO. Truly,

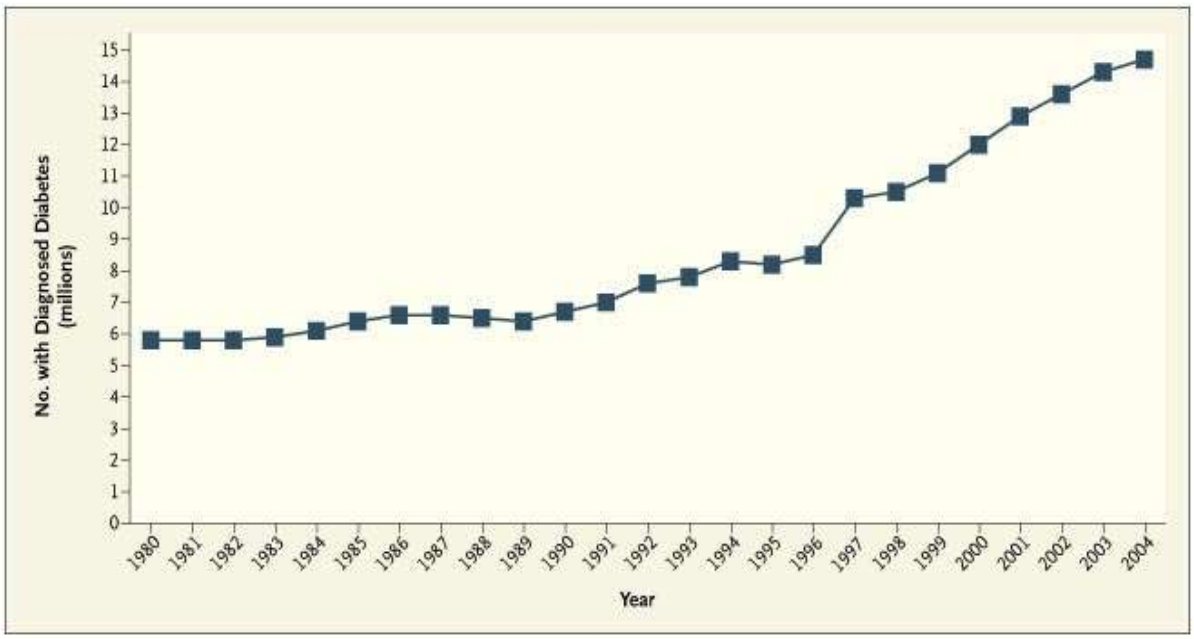
Hell hath no fury like a vested interest masquerading as a moral principle
Judge Barber Conable

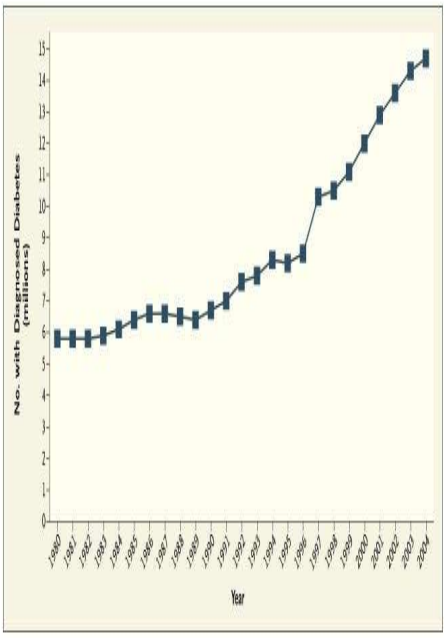
The sugar magnates' influence on the US government reaches all the way to the top. According to the National Review of Medicine, President Bill Clinton famously took a phone call from Alfy Fanjul, CEO of the sugar producer Florida Crystals, while White House intern Monica Lewinsky was entertaining him. Florida Crystals is based in Florida, a state run by governor Jeb Bush, President George W Bush's brother. Big Sugar raised millions of dollars for President Bush's reelection campaign, and U.S. taxpayers send enormous checks to U.S. sugar growers in the form of federally approved subsidies (Herald 2004). The sugar growers can easily afford the political contribution "cost of doing business." The "Sugar Program" protects domestic growers with tariffs and price guarantees, and cost American consumers about two billion dollars in 1999 (in addition to the $532 million cost of the program itself), according to a US General Accounting Office report to Congress (GAO 2000). The one hand washes the other.

Groups like the National Soft Drink Association argue that 25% added sugar in the diet is not harmful (despite the USDA Food Pyramid implied maximum of 10%, above which level there will be nutrient deficiencies). The "25% added sugar is OK" view is backed by the prestigious-sounding International Life Sciences Institute (ILSI). However, the ILSI is actually a joint creation of Coca-Cola, PepsiCo, General Foods, Proctor and Gamble, and Kraft. ILSI published a ludicrous pamphlet in 1998 called *Carbohydrates and Weight Management*. The authors couldn't resist quoting their own research: "… sucrose-containing drinks (Rolls 1990) and desserts (Rolls 1989) given either before or with lunch were associated with increased energy intake when compared with aspartame-sweetened versions of the drinks or desserts" and they point out that "… small amounts of additional energy from any nutrient source may increase daily intake and could, over time, be important in the etiology of obesity." Yet their conclusion holds sugar blameless and faults the obese for overeating and exercising too little! I believe this sort of relentless century-long "hidden persuasion" of the sugar industry has distorted our perception of sugar. This has blinded us to the idiocy of treating pre-diabetes and diabetes with *high*-carbohydrate diets, which might make some sense if the diet emulated that of the Kitava Islanders, but makes no sense whatsoever if it includes refined carbohydrates.

Impaired Glucose Tolerance and Diabetes

As with obesity, so it goes insulin resistance and impaired glucose tolerance. Type 2 diabetes is the diagnosis when the pancreas cannot keep up with the extra demand for insulin consequent on insulin resistance, and the blood sugar rises above the normal range. Not only is diabetes the sixth most prevalent cause of death in America, it is increasing at an epidemic rate: "Worldwide increases in obesity and diabetes have aroused concern that increased morbidity and mortality will follow … During this decade, all-cause and cause-specific mortality rates declined, with the striking exception of diabetes, which increased 61% and 52% for men and women" (Fang 2006). Type 2 diabetes used to be called Adult-Onset Diabetes, but it's incidence in childhood has increased 30-fold in the past decades, forcing the name change (Rosenbloom 1999). About 7% of the population has diabetes. In addition, at least 40% of people between 40 and 74 years of age have pre-diabetes, meaning a fasting blood sugar of 100 to 126mg/dl, and/or Impaired Glucose Tolerance in which the blood glucose level is 140 to 199 mg/dl after a 2-hour oral glucose tolerance test (National Diabetes Statistics 2003). In addition, people who have it generally go on to develop Type 2 diabetes; however, this is "not inevitable" if they lose weight and exercise (Lindstrom 2006).





Number of Persons with Diagnosed Diabetes in the United States, 1980–2004 (Steinbrook 2006). The same graph compressed at right shows the prevalence of diabetes began to increase rapidly in about 1989.

Obesity and diabetes are closely linked, since about 80% of diabetics are overweight at diagnosis; and a graph of diabetes by age shows the same form as that for obesity, with the prevalence appearing to fall with age because of excess deaths (Harris 1998):

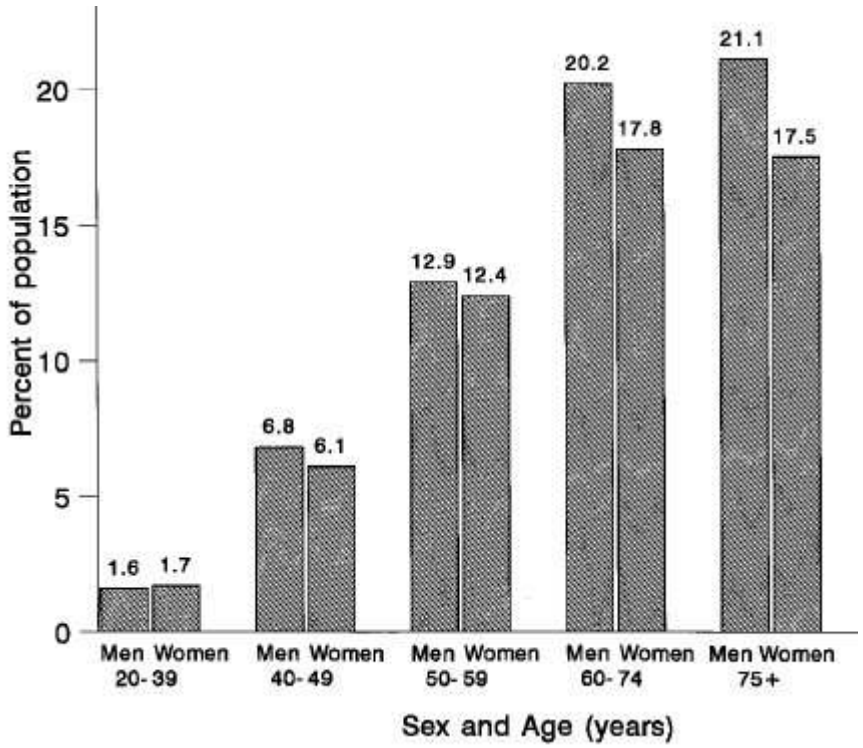
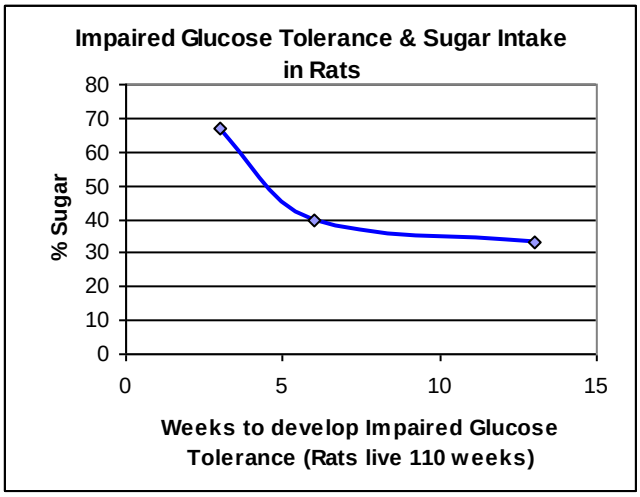


Figure 1 – Prevalence of diabetes in men and women in the US population … based on NHANES III. Diabetes includes previously diagnosed and undiagnosed diabetes defined by fasting plasma glucose >126 mg/dl.

Interestingly, epidemiological studies have shown that there is a 20-year lag between the introduction of refined carbohydrates and an increase in the incidence of diabetes in populations, an observation made by TL Cleave and discussed in his book *The Saccharine Disease*. The graph of diabetes prevalence by year shows that this diabetes epidemic took off in about 1989, which is consistent with Dr Cleave's "rule of 20 years", since America increased its intake of carbohydrates on the advice of the public health authorities in the mid-sixties. The public health authorities naively thought the public would understand that they meant *unrefined* carbohydrates.

Insulin resistance has been linked to sugar consumption in animal experiments. When Dr Aharon Cohen fed starch to rats, they lived out their lives with normal insulin sensitivity. However, when he fed them sugar, he found that the more he fed them, the shorter the time it took for the vulnerable among them to develop impaired glucose tolerance (Cohen 1964):



The rats recovered their sensitivity to insulin within a few weeks of returning to a starch diet, but lost it once more within days on the sugar diet, making it clear that there was a permanent injury to their insulin systems. Dr Cohen went on to breed the sugar-vulnerable rats in such a way as to develop a strain that is prone to diabetes, the Cohen Diabetic Rat, which is still in use today as an animal model of Type 2 diabetes. Dr Jim Barnard of UCLA performed similar experiments, confirming that a fatty diet high in sugar promotes insulin resistance and syndrome X in rats (Barnard 1995). Some humans are like these rats, exquisitely sensitive to simple carbohydrates. The Pima Indians of Arizona, for example, have a staggering 50% incidence of Type 2 Diabetes, and at least two genes which confer such sensitivity have been identified (Gerich 1998).

Dr Cohen went on to demonstrate the phenomenon in humans. He studied the incidence of diabetes among groups in Israel, where a great deal of sugar is eaten: There were almost no cases of diabetes among Yemenite newcomers, whose diet in the Yemen included almost no sugar, whereas 2.5% of Yemenites who had lived in Israel for more than 25 years had diabetes (Cohen 1971). He concluded that a high sugar intake exposes any genetic predisposition to develop diabetes: No sugar, no diabetes.

In 1961, a Dr Cruz infused dogs with insulin through a needle into a vein in the leg (Cruz 1961). Within three months, there was rampant atheroma downstream of the needle, while there was none in the vein of the other leg: apparently, insulin *itself* is toxic to the endothelium, the specialized cells lining the arteries and veins (Hsueh 2004). It is generally accepted that injury to the endothelial cells is the first step in the development of atherosclerosis. Furthermore, insulin so resembles a growth-factor hormone that it encourages the reproduction of the smooth muscle cells of the blood-vessel wall. These muscle cells are found in atherosclerotic plaque.

Sugar has addictive qualities

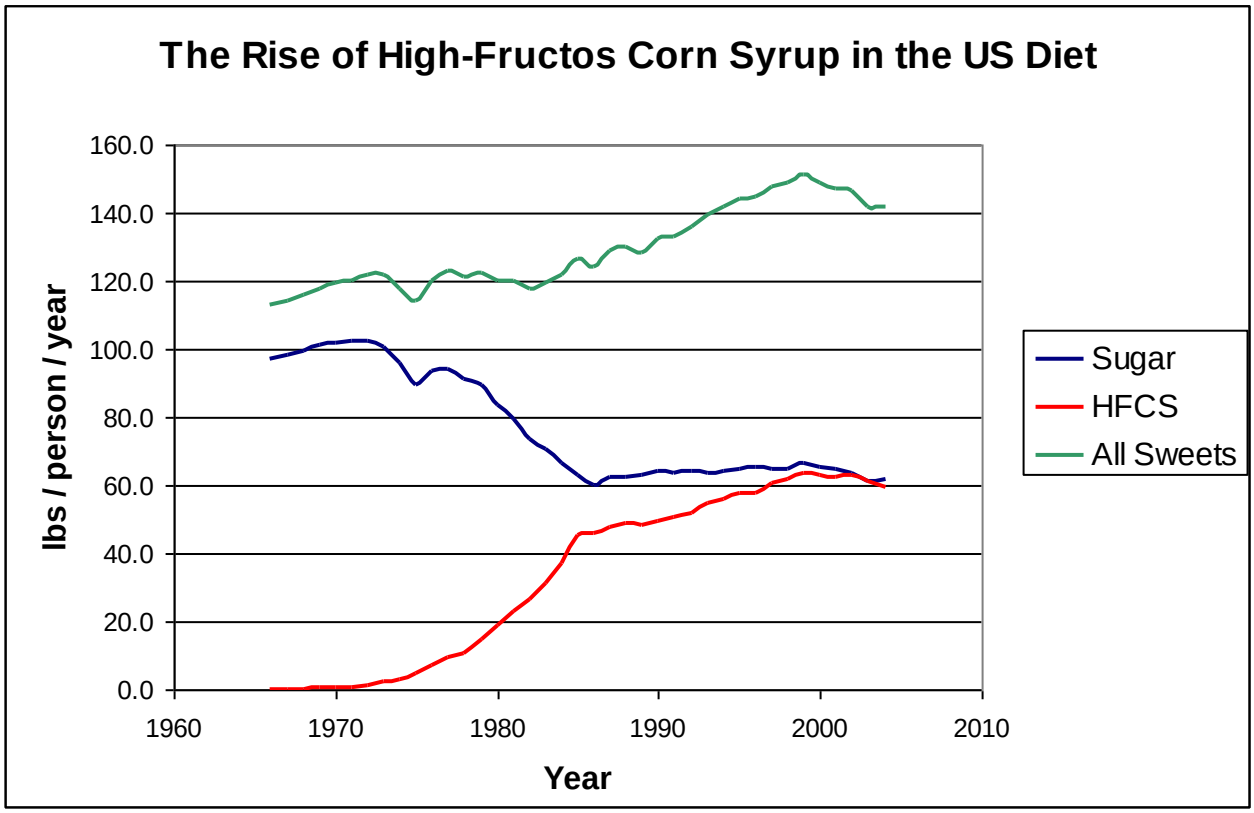
> … sustained consumption of sugars and fats may have additional metabolic consequences; among these are neurochemical changes in brain sites involved in feeding and reward, some of which are also affected by drugs of abuse … [and] may contribute to the observed increase in the body weight of populations (Levine 2003).

> [in rats] with a concentrated sucrose solution to drink, an opiod dependence developed with 1st increased consumption of sucrose 2nd abstinence symptoms with no sucrose and 3rd anxiety with an opiate blocker (Erlanson-Albertsson 2005)

The seductive nature of refined foods is evident from Dr Weston Price's investigations in the '30s and '40s of twelve racial groups in dozens of civilizations; all, without exception, adopted refined foods when they became available to them in spite of their evident drawbacks:

> … the cause of tooth decay … was established quite readily as being controlled directly by nutrition, it rapidly became apparent that a chain of disturbances developed in these primitive racial stocks starting even in the first generation after the adoption of the modernized diet and rapidly increased in severity with expressions quite constantly like the characteristic degenerative processes of our modern civilization of America and Europe (Price 1945).

Surprisingly few commentators connect the dots. In 1976, the McGovern committee published its Dietary Goals for the United States, which called for reduced fat and for the replacement of saturated fats with unsaturated vegetable oils. Agriculture and the food industry were ready with margarines and low-fat, low-cholesterol food products in which fat calories were replaced with calories from refined carbohydrates, accelerating the trend begun by Ancel Keys in 1961. Obesity began to rise after a few years, and the incidence of diabetes began to increase about ten years later. Simultaneity doesn't prove causality, of course, but it raises legitimate question which should prompt research to prove, or disprove, causation. Sugars have increased dramatically in the US diet, and there have been changes in the *type* of sugar (USDA):



The epidemic of obesity and diabetes really took off when *high-fructose corn syrup* (HFCS) began to replace table sugar in "foods" such soft drinks (because it's so much cheaper), which changed the type of fructose in the national diet. HCFS contains more fructose (55-75%) than table sugar (50%), so the 15% increase in sweetener intake from 1970 to the present actually means about 20% more fructose. Whereas table sugar contains fructose bound to glucose, HFCS contains free fructose and free glucose. Some, like Harvard's Dr Walter Willett, dismiss this change as a cause of health troubles because "fructose is fructose", but the free fructose and free glucose in HFCS skip the digestive step in which the sugar molecule is broken apart, hastening their appearance in the bloodstream. Moreover, the introduction of HFCS coincided with a reduction of calories from fat and a fall in fiber intake, both of which slow the absorption of sugars. Less fiber is a consequence of more refined foods in the diet, which means a lowered intake of the vitamins and minerals that are lost with the fiber during refining, and which are necessary for the metabolism of sugars. For the reasons set out below, I believe these factors have worked together synergistically to turn the century-long pandemic of degenerative diseases such as heart disease into the current epidemic of Syndrome X and the dementias.

The studies that follow support this idea. Culled from hundreds on the subject, they cut through the confusing mass of claims in this area:

There is a "remarkable difference" in results from industry-funded and non-industry-funded studies on soft drink consumption and health outcomes "with the industry-funded studies much more likely to find the results favorable to industry."
Dr Kelly Brownell

Apparently, sugars are so immensely profitable that its purveyors can afford to fund junk research to confuse the issue; these studies funded by the sugar interests generally contain little of substance and conclude "More study is needed!"

Importantly, fructose does not cause *satiety*, the feeling of having eaten (Anderson 2002), so fructose calories *add to*, rather than *replace*, calories from the rest of the diet. This effect is seen most clearly in three well-designed studies of humans. In each study, the group receiving fructose gained a statistically-significant amount of weight. When soft drinks sweetened with either fructose or aspartame were given to 30 subjects of normal weight for three weeks, all subjects gained weight only during the fructose period (Tordoff 1990). Diabetics given fructose also gained weight (Anderson 1989). In addition, overweight subjects allowed free access to either sugar-sweetened (sugar is half fructose) or artificially sweetened drinks gained weight only during the sugar-sweetened drink phase of the study (Astrupp 2002). A meta-analysis of 30 studies spanning the years 1966 to 2005 which concluded that "The weight of … evidence indicates that a greater consumption of sugar-sweetened beverages [which contain HFCS] is associated with weight gain and obesity" (Malik 2006). What we see here is that because fructose does not cause satiety, calories from soft drinks and the like do not reduce the calories taken in at the mealtimes, so that weight gain ensues. Incredibly, one out of every four children obtains more than 25% of calories from sugars (which, even more incredibly, is the maximum level "recommended" by the American Dietetic Association and the Institute of Medicine), so the epidemic of childhood obesity is almost certainly due to the rise in fructose intake.

When the progression of the obesity epidemic in America is plotted against free fructose intake (from HFCS), the association jumps off the page (Bray 2004):

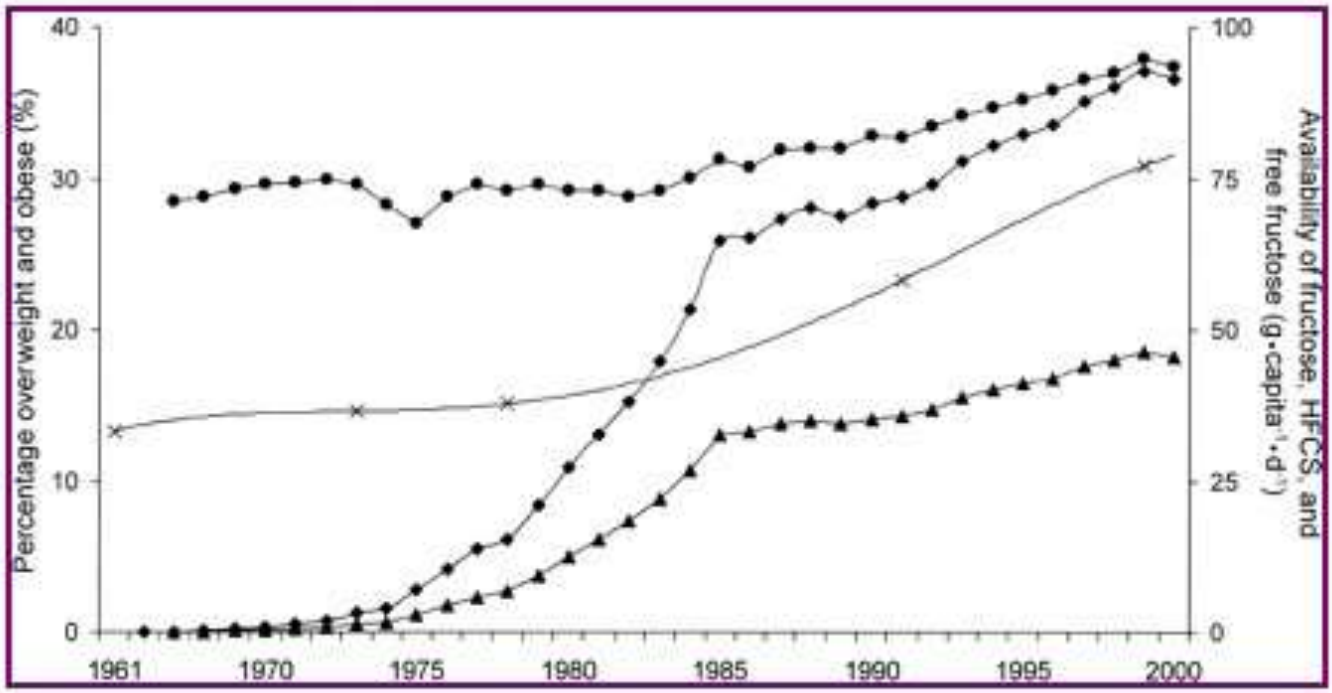


FIGURE 1. Availability of total fructose (•), high-fructose corn syrup (HFCS;♦), and free fructose (▲) in relation to obesity prevalence (x) in the United States. Data are from references 7 and 35.

A role of fiber?

The Insulin Resistance and Atherosclerosis Study showed that, statistically speaking, both fiber and magnesium contribute to insulin sensitivity: "fiber and magnesium … account for some of the effect of whole grains on [insulin sensitivity] (Liese 2003). Dr Lee Gross performed an analysis: "Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States." He also found statistically-significant relationships between refined carbohydrate intake and the incidence of Type 2 diabetes, and between increased HFCS consumption and the prevalence of diabetes (with one chance in 26 that the result is a fluke); and fiber intake was negatively associated with the epidemic (with just one chance in 100). The graph below at left shows how carbohydrates have increased even as fiber has fallen because of increased refined carbohydrate in the diet, while the graph at right shows the prevalence of Type 2 diabetes during these changes (Gross 2004):

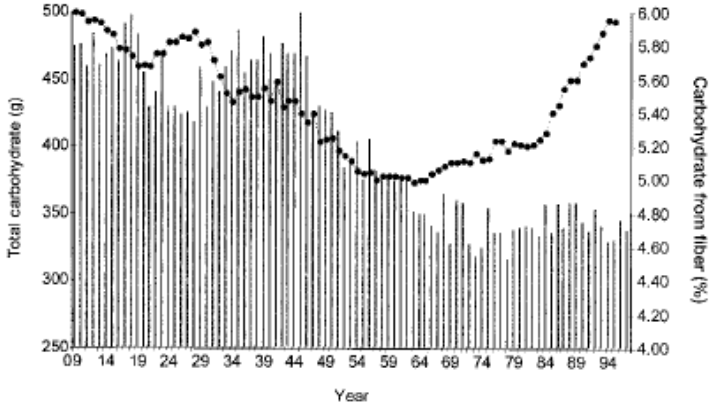


FIGURE 1. Change in total carbohydrate consumption (●) and the percentage of carbohydrate from fiber (vertical bars) in the United States between 1909 and 1997 (17).

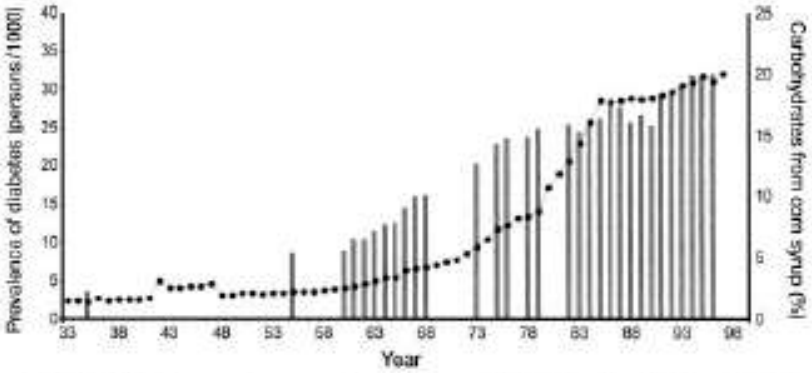


FIGURE 3. Increasing prevalence of type 2 diabetes (vertical bars) in the United States between 1933 and 1997 with increasing per capita percentage of carbohydrate intake from corn syrup (●) (14, 17).

Left: Dietary carbohydrate steadily decreased from 500 g/d in 1909 to 374 g/d in 1963, largely because of a decrease in the consumption of whole grains. Simultaneously, dietary fiber decreased at a greater rate — by nearly 40%. Since 1963, the consumption of carbohydrates steadily increased back to 500 g/d; however, fiber consumption did not increase proportionately. This finding reflects an increased consumption of refined carbohydrates over this time period.

Low fiber has been associated with a raised risk of Type 2 diabetes in other epidemiological studies. For example, in the Nurses Health Study, women with the lowest intake of cereal fiber and the highest glycemic-load diet had a relative risk of Type 2 diabetes of 2.43 (Meyer 2000); similarly, in the Health Professionals Follow-Up Study, men who ate this way had a relative risk of developing Type 2 diabetes of 2.17 (Salmeron 1997). There is definitely a strong association, but is it causative?

The studies of Drs Gross and Liese mentioned above suggest, on the face of it, that fiber and magnesium promote insulin sensitivity. However, a meticulous experiment which used two diets identical in every respect but for the use of white flour in one study group and whole-wheat flour in another found that while the whole-wheat flour diet improved insulin sensitivity in overweight people, the white flour diet did not (Pereirra 2002). This raises the interesting possibility that cereal fiber slows the absorption of glucose from the food – that unrefined foods with their lower glycemic index protect the pancreas.

But the glycemic index for whole-wheat bread products is practically the same as that of bread made from white flour! For example, the GI of Wonder Bread is 73 (on the scale where glucose is 100), and that of a 100% barley flour bread was 67, a trivial difference. Compare these numbers with General Mills porridge oats at 71 and a baked potato at 85 (Foster-Powell 2002) and it becomes clear that "complex carbohydrates" such as whole-grain flour are absorbed almost as fast as glucose.

In important experiment, Dr David Jenkins (who pioneered the Glycemic Index) supplemented the diets of Type 2 diabetics with cereal fiber, and found that their "markers of glycemic control" (and therefore their insulin resistance) remained unchanged. This removes all doubt: cereal fiber, *per se,* does not increase sensitivity to insulin, and the association between cereal fiber and insulin resistance is not causative. Dr Jenkins concluded that "cereal fiber in the diet may be a marker for another component of whole grains that impart health advantages" (Jenkins 2002). Dr Jenkins is saying that the statistical analyses are picking up the fact that the presence of fiber is an indication that the grain is unrefined and has its full complement of nutrients, and it is to these nutrients we must look to discover the basis for whole grains' insulin-sensitizing properties.

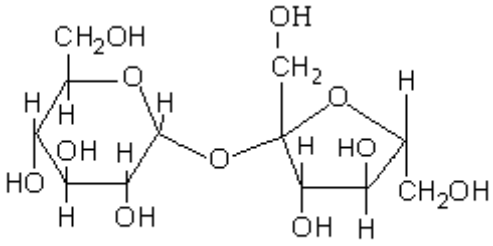
Fructose worsens every feature of Syndrome X

The damaging effects of fructose are easily demonstrated in rats, but feeding fructose to people has not always shown the same harmful effects. The research effort may be confounded because human studies are often short, and generally use young, fit subjects who can get away with almost any dietary indiscretion. A second reason may be that most (but not all) studies are brief, and start with an overnight fast, that is, with a low insulin level. When insulin is low, much less triglyceride is made by the liver; and if triglycerides are low when the fructose is given, the rise in triglycerides is less, and lasts for a shorter time. When insulin is kept high by frequent sugary snacks (sugar is added to the rats' drinking water in most rat studies), the triglyceride pathway turns on and stays on (Abraha 1998).

However, a single can of HFCS-sweetened soda per day contributes enough calories to cause a 15-pound weight gain over a year. Because fructose calories do not contribute to satiety, people do not compensate for them by eating fewer calories at the next meal (Appovian 2004). "… the long-term consumption of diets high in fat and fructose is likely to lead to increased energy intake, weight gain, and obesity. The potential for weight gain from increased fructose consumption may only represent one aspect of its metabolic consequences" (Elliott 2002).

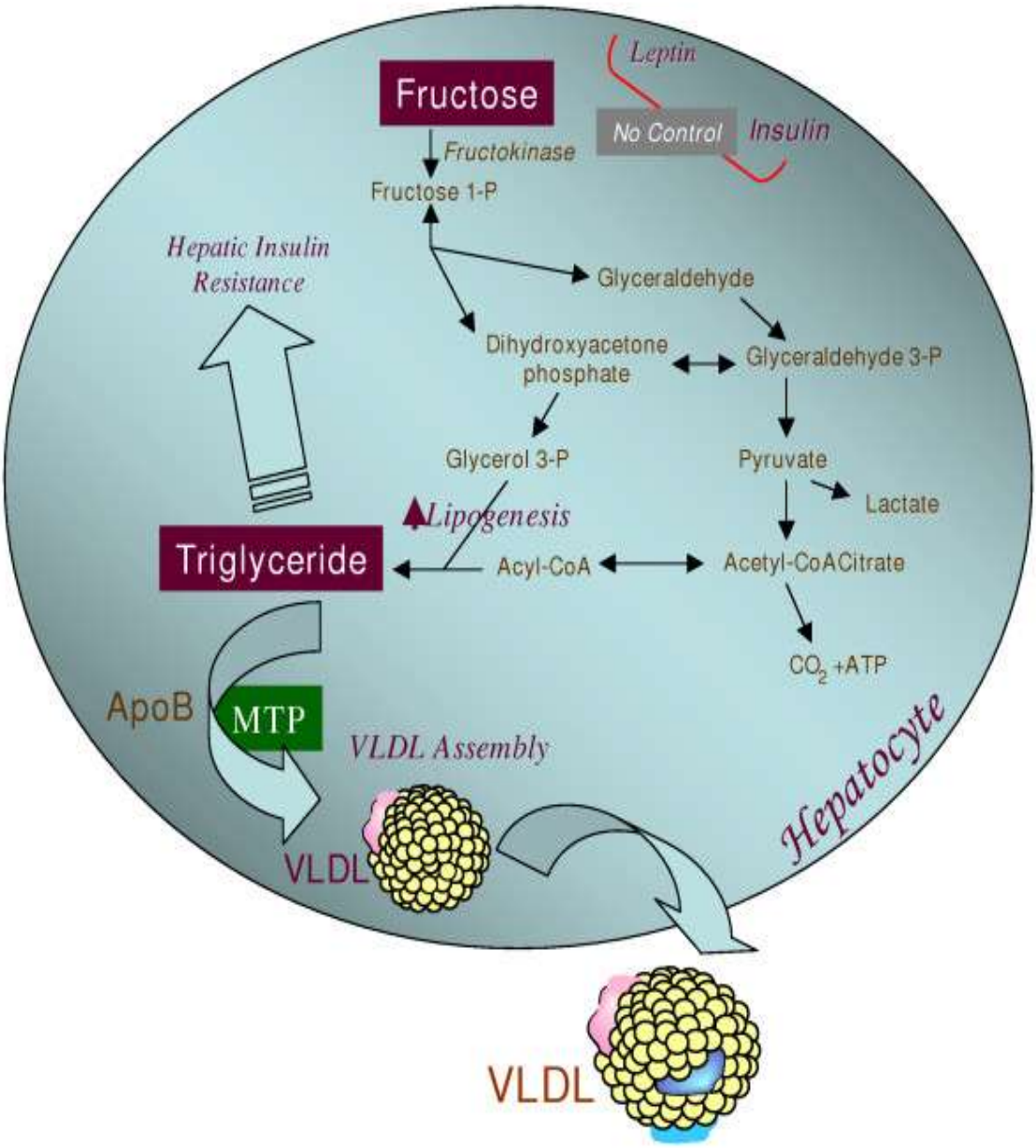
It's not just weight – apparently, fructose worsens every aspect of the metabolic syndrome. In a study of 32 healthy subjects, there was a significant correlation between fructose and lowered HDL-cholesterol (Slyper 2005), and fructose is renowned for raising triglycerides (Le 2006). The research community is worried: "… emerging evidence from recent epidemiological and biochemical studies clearly suggests that the high dietary intake of fructose has rapidly become an important causative factor in the development of the metabolic syndrome. There is an urgent need for increased public awareness of the risks associated with high fructose consumption" (Basciano 2005). Amidst the dispassionate writings of science, this is like shouting "Fire!"

So what is fructose and why is it so dangerous? Fructose and glucose are sugars composed of the same atoms ($C_6H_{12}O_6$) but in different arrangements, and sugar – sucrose – is made up of one molecule of fructose and one molecule of glucose:



Glucose (left) and fructose (right) in sucrose (table sugar)

Digestive enzymes break the bond between glucose and fructose, and the two sugars pursue different pathways in the metabolism. Glucose enters the bloodstream, triggering the release of insulin which causes most of it to be taken into muscle and fat cells. Fructose, on the other hand, does not trigger the release of insulin but is rather taken up by the liver and made into fat – triglycerides – that are then packaged into *very low-density lipoproteins* (VLDL) and released into the bloodstream. The triglycerides are taken up by muscle and fat cells and the remaining lipoprotein becomes "bad" LDL-cholesterol, especially the "small, dense" LDL particles known to increases the risk of CHD. In short, fructose causes high triglycerides, which is one of the hallmarks of Syndrome X (Basciano 2005):



Hepatic [meaning liver] fructose metabolism is a highly lipogenic [fat-forming] pathway. Fructose is readily absorbed from the diet and rapidly metabolized principally in the liver. Fructose can provide carbon atoms for both the glycerol and other portions of triglyceride. Fructose is thus a highly efficient inducer of *de novo* lipogenesis. High concentrations of fructose can serve as a relatively unregulated source of acetyl CoA [acetyl co-enzyme A is the precursor of many compounds, including cholesterol, bile salts, sex hormones, and cortisol]. In contrast to glucose, dietary fructose does NOT stimulate insulin or leptin (which are both important regulators of energy intake and body adiposity). Stimulated triglyceride synthesis is likely to lead to hepatic accumulation of triglyceride, which has been shown to reduce hepatic insulin sensitivity, as well as increased formation of VLDL-cholesterol.

Meanwhile, glucose enters muscle and fat cells with insulin's help. It may be made into the body's storage carbohydrate, glycogen, if glycogen is in short supply. Otherwise, the *glycolytic* (glucose-splitting) enzymes begin work on it until a rate-limiting enzyme is reached: plentiful ATP (cells use ATP for energy) and/or citrate (which is the raw material of Krebs cycle, which makes ATP in the mitochondria) inhibit this enzyme so that glucose is not processed further. However, the fructose pathway has no such rate-limiting step, so the cell must do its best to process all available fructose into citrate or triglycerides. This may be because fructose in the bloodstream reacts with – glycosylates – proteins seven times faster than glucose (Levi 1998), and these glycosylates form Advanced Glycosylation Endproducts (AGEs) up to ten times faster than those containing glucose (McPherson 1988). AGEs are both a marker for, and a cause of ageing. They bind to receptors on endothelial cells, smooth muscle or cells of the immune system and contribute to age- and diabetes-related chronic inflammatory diseases such as atherosclerosis, asthma, arthritis, myocardial infarction, nephropathy, retinopathy or neuropathy (Wells-Knecht 1995). Fructose in the bloodstream is bad news and the body wants it out of the bloodstream as soon as possible. This is not a problem with physiological quantities such as the amount in a piece of fruit, but we run into trouble with the supra-physiological quantities in, say, a soda.

Fructose wastes phosphate

The cell needs phosphate ions to process fructose, for the first step in its processing within the cell is "phosphorylation." Humans have a limited ability to metabolize fructose (fruit sugar). Fructose is metabolized differently from other sugars. A fructose load leads to accumulation of fructose-1-phosphate ['Fructose 1-P' in the diagram above] in cells, which may partially deplete intracellular ATP levels (Milne 2000).

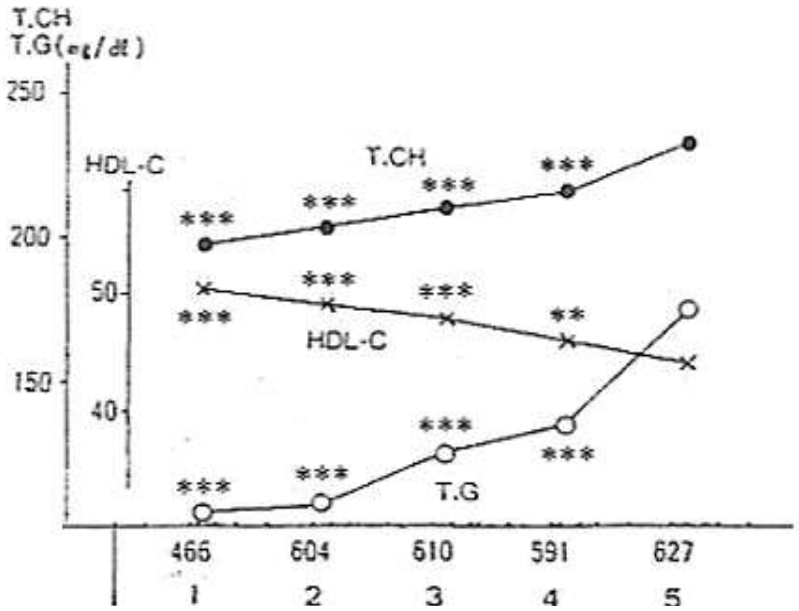
Available phosphate molecules in the energy molecule of the cell, ATP, are vital because phosphorylation reactions are everywhere within the cell. The insulin receptor "auto-phosphorylates" when insulin binds to it, and then catalyzes the phosphorylation of a whole series of proteins called "insulin receptor substrates." This causes glucose transporters to be expressed on the cell membrane, and the first thing that happens to the glucose on entering the cell is that it is itself phosphorylated to become glucose-6-phosphate, just as the first thing that happens to fructose is that it is phosphorylated to fructose-1-phosphate. And while the cell has control of how much glucose it allows to enter, it has no control over fructose. Even more fundamentally, ATP itself cannot be reconstituted if phosphate ions are in short supply, so that the cell runs short of power and the sodium pump (which is an obligate consumer of ATP) slows, allowing calcium to build up within the cell.

Thus, if the cell runs short of phosphate ions, then it should become insulin resistant – and this is, indeed, the case. Diabetics are commonly depleted of phosphate, and the degree of their insensitivity to insulin is proportional to their degree of phosphate depletion (DeFronzo 1980). Further, the insulin sensitivity of non-diabetics is associated with their serum phosphate levels (Haap 2006). Severe hypophosphatemia occurs in diabetic keto-acidosis (Ravenscroft 1999), but is most often found in primary hyperparathyroidism, which is also associated with insulin resistance and a higher incidence of diabetes (Kautzky-Willer 1992); the American diet is adequate in phosphorus, and deficiencies usually involve losses in the urine or difficulties of its distribution. In a study of eleven healthy men, a high-fructose low-magnesium diet for 42 days induced negative phosphate balance: "It is likely that long periods of negative phosphorus balance could lead to … hypophosphatemia … This trend seems to be the most dramatic in children in the US who are consuming large amounts of soft drinks containing HCFS at the expense of foods containing adequate amounts of magnesium and calcium" (Milne 2000). Moreover, patients with four or more features of the metabolic syndrome had low serum phosphate (and low magnesium) compared to controls who had three or fewer metabolic syndrome symptoms (Kalaitzidis 2005); this means that low serum phosphate (and low magnesium) are closely associated with the metabolic syndrome and probably contribute to its cause.

Further, intracellular calcium acts as a co-enzyme for three of the reactions of Krebs cycle, so that the elevated calcium levels of those with features of the Metabolic Syndrome will increase the reaction rate of many of the steps in the cycle, and therefore increases flux throughout the pathway; this means more of the aforementioned intermediate compounds will build up. One such intermediary compound which builds up is an *aldehyde* ('Glyceraldehyde 3-P' in the diagram above). An aldehyde that may be familiar is acetaldehyde, made from alcohol and which contributes to hangovers. "Excess aldehydes can bind sulfhydryl groups of membrane proteins, altering membrane calcium channels, increasing cytostolic free calcium, peripheral vascular resistance and blood pressure. The presence of reactive aldehydes can also lead to oxidative stress" (Vasdev 2003). Fructose raises the concentration of calcium ions within the cells that causes the muscles in the capillary wall to contract, raising the blood pressure; and fructose causes oxidative stress.

Insulin resistance elevates cholesterol!

Insulin resistance explains a mystery posed earlier: how it is that in Framingham, high cholesterol levels doubled the relative risk of developing CHD, yet lowering cholesterol lowered the risk of CHD by only 0.3% and actually increased all-cause mortality by the same amount. The answer to this conundrum is that the insulin-resistant have higher cholesterol levels! In a 1985 study, 2,927 Japanese people living in Japan, Hawaii or Los Angeles were divided into five groups according to their F-IRI (fasting insulin) level, which is proportional to their insulin resistance, and their cholesterol, HDL-cholesterol and triglyceride levels were measured (Nagasaki 1986):



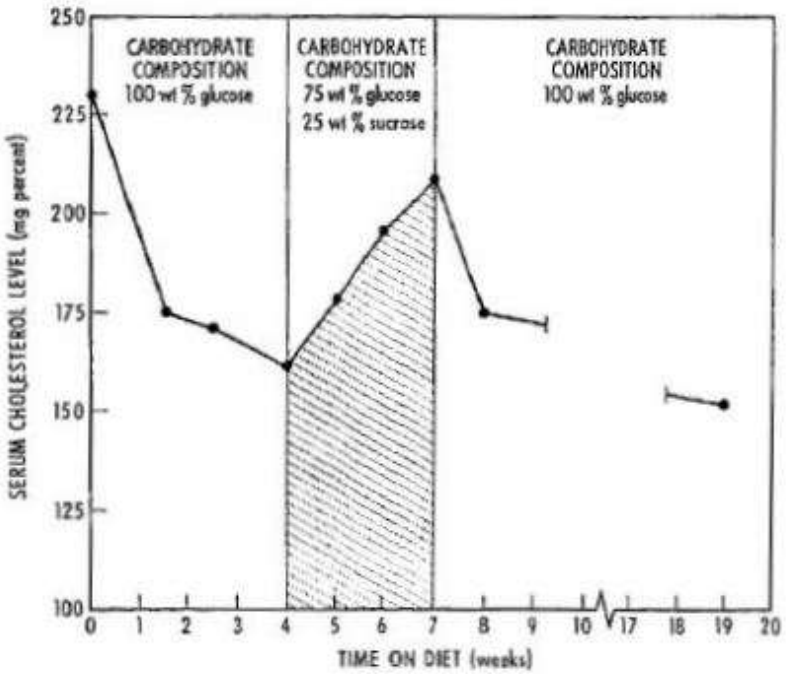
Quintiles 1 – 5 plotted according to their F-IRI [insulin resistance] score show that the more insulin resistant the quintile, the higher their average cholesterol (T. CH) and triglycerides (T.G.), and the lower their HDL-cholesterol (HDL-C) levels. The asterisks indicate that these results are highly statistically significant: ** P<0.01, *** P<0.001.

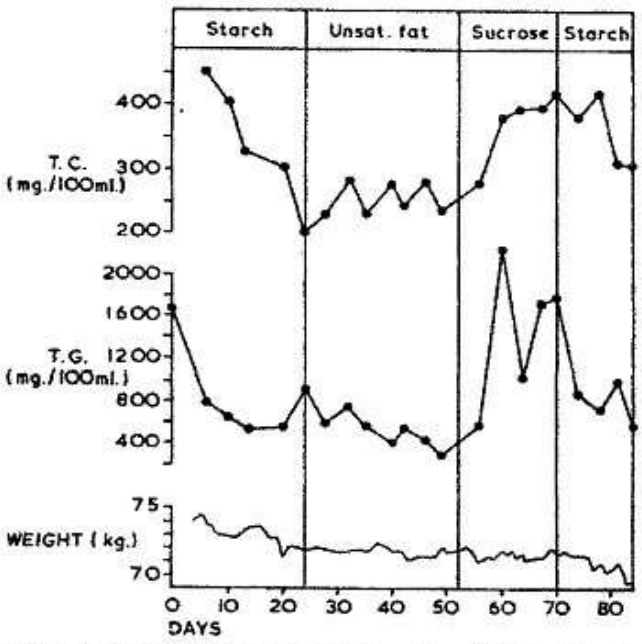
We see that cholesterol is an *association* without a *cause* where heart disease is concerned, the James Dean of physiological measurements. Insulin resistance increases cholesterol levels, which explains the association between high cholesterol levels and heart disease in the Framingham Study. As detailed earlier, the research shows unequivocally that manipulating cholesterol has no effect on heart disease; and I have to believe the research even though it runs counter to the conventional wisdom. The research is clear, yet the cholesterol interests have spun it into the opposite conclusion! It is awe-inspiring what a few billion dollars of drug revenue can accomplish. However, lowering insulin resistance has a long and successful history in lessening the virulence of disease: exercise and weight loss, which both lower insulin resistance, both lower heart risk. Dr Reaven's researches suggest that if your cholesterol is high, it would behoove you to look to your insulin resistance. The link to heart disease may be that high-fructose low-magnesium diets are associated with high oxidant stress, which is itself associated with inflammation; and inflammation is far more strongly linked to endothelial dysfunction, atherosclerosis and heart disease than elevated cholesterol, as will be discussed in the upcoming segment "Insulin Resistance causes CHD via inflammation?"

Weight loss also lowers C-reactive protein, the marker for inflammation (Selvin 2007), perhaps in part because of the "enhanced efficiency of ATP production" remarked upon by Dr Semenkovich (Semenkovich 2006). Here is yet another link between the insulin resistant state and inflammation.

How fructose elevates cholesterol

Acetate made in the *glycolysis* of sugars is fuel used to make ATP in the Krebs cycle, or, if there is an excess, it enters the pathway for the synthesis of either cholesterol or triglycerides, meaning fat. Acetate ('Acyl-CoA' in the diagram above) builds up when too much fructose is processed, leading eventually to elevated cholesterol and triglyceride levels; acetate is made from citrate in the aforementioned Krebs cycle. This solves the mystery of how the fructose in table sugar and HFCS elevates cholesterol. It further explains why cholesterol fell so precipitously when sugar was replaced by glucose in Dr Winitz' low-fat feeding trial for NASA mentioned earlier (Winitz 1970, Winitz 1964, left), and in Dr Aharon Cohen's similar experiment in a diet with a more normal fat content (Cohen 1966, right):



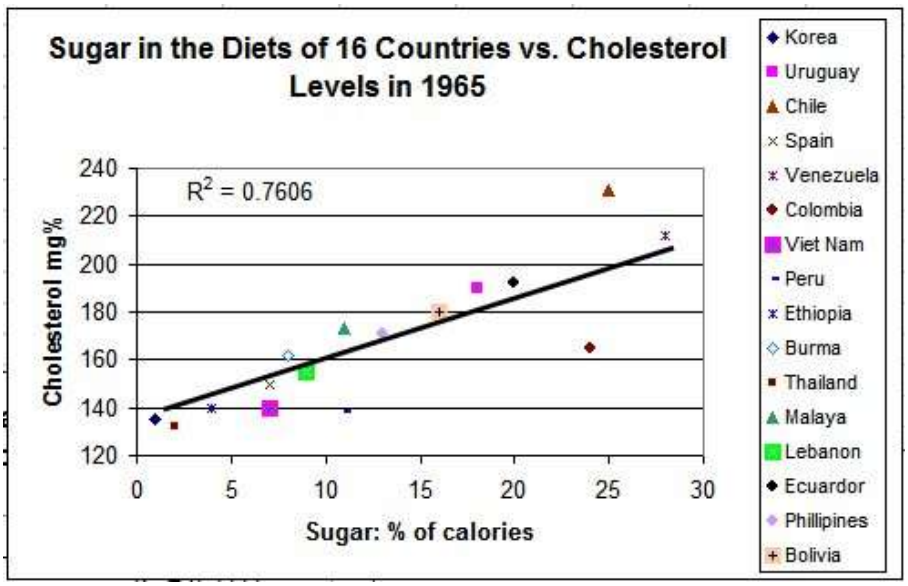


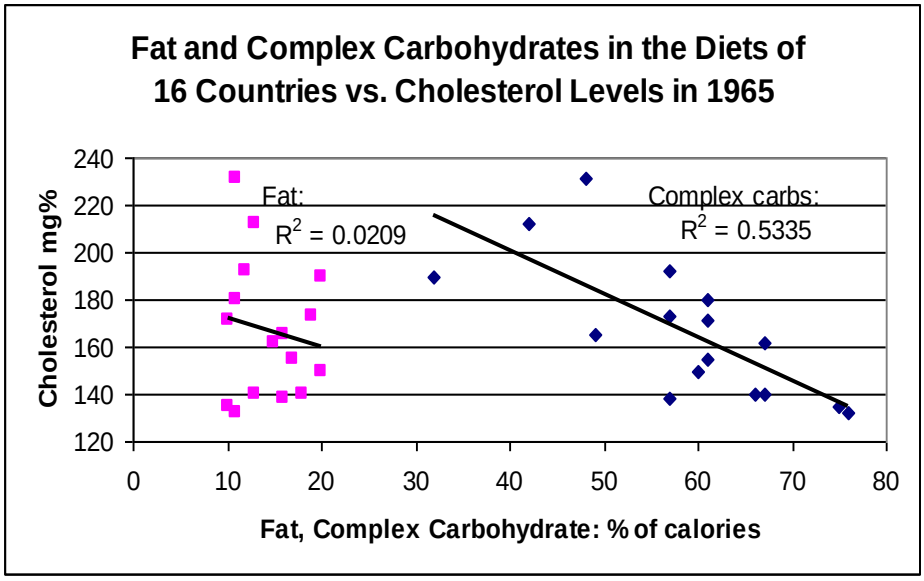
Effect of various diets on serum triglycerides (T.G.) and total cholesterol (T.C.). Composition of diets given in Table.

Left: Twenty-four felons aged from 24 to 43 years in the California prison system at Vacaville were fed a low-fat, 14% protein, chemically-defined diet containing carbohydrate calories either from 100% glucose, or 75% glucose with 25% sucrose. The decreases in serum cholesterol levels at the end of the first 4 weeks on the 100% glucose diet ranged from 9 to 49% of the baseline values, an average of 76mg%, and a significant change in the opposite direction occurred when the sucrose-containing diet resumed. There was very little fat in the diet, so that the experiment "unequivocally demonstrates an important relationship between the nature of the dietary carbohydrate and serum cholesterol levels" (Winitz 1970, Winitz 1964). This sugar-raises-cholesterol effect was confirmed in a more recent study which used an American-style diet containing 42% fat – cholesterol rose with time in a dose-dependent fashion when sugar was fed, but remained at baseline levels when carbohydrate was given as starch (Reiser 1979). A low glycemic index diet (oddly, "Diet GI is inversely associated with simple sugar intake because foods rich in simple sugars, such as milk and fruit, have relatively low GI values" Wolever 1994) lowered cholesterol by 15% in six healthy male volunteers (Jenkins 1989).

Right: Dr Cohen fed a 50-year-old Rumanian woman with very high cholesterol and triglycerides a series of diets; starch caused a precipitous drop in both parameters. Triglycerides fell even further on a high-monounsaturated, fat diet. Returning to a high-sugar diet reversed these improvements.

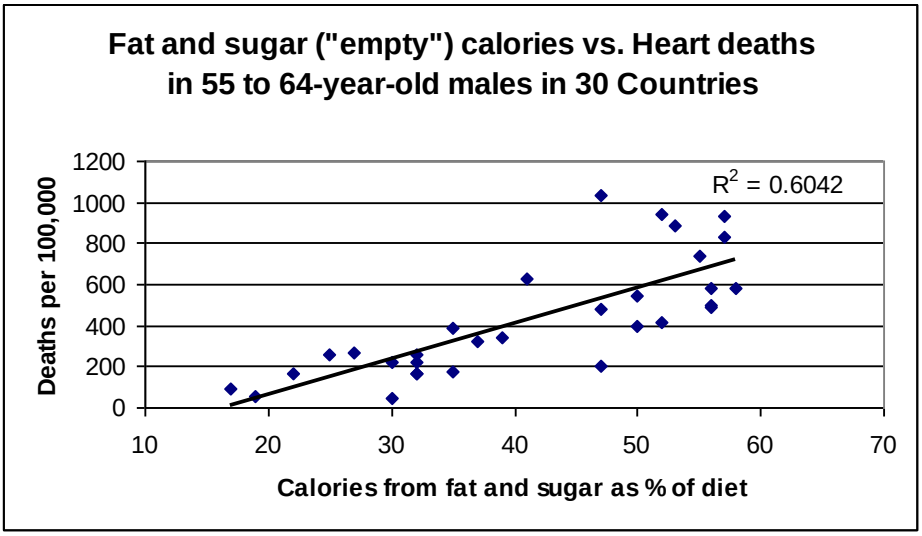
This relationship between sugar intake and cholesterol level exists in the real world. Dr Alfredo Lopez tabulated food disappearance data and cholesterol levels in 16 populations (Lopez 1966):

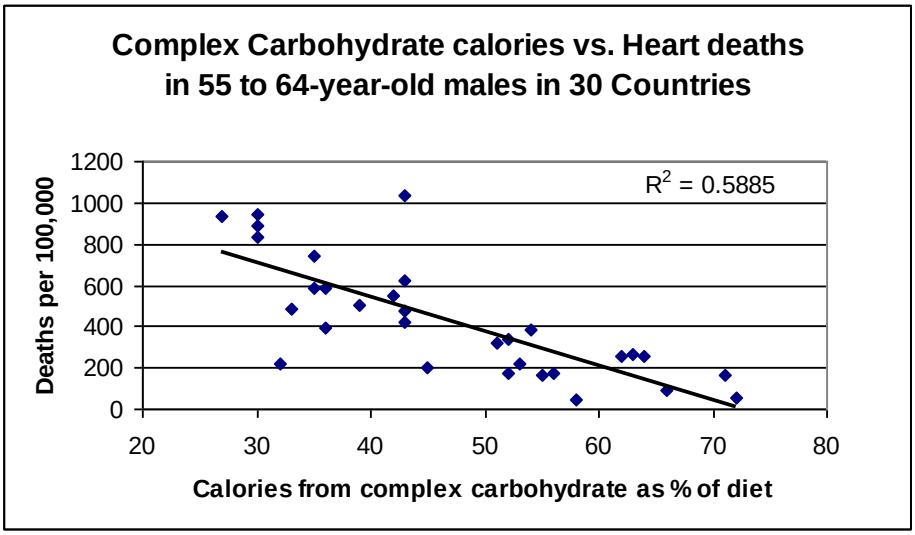




Left: Sugar intake is strongly correlated with cholesterol levels in 13 populations. Right: Complex carbohydrate intake is associated with *lower* cholesterol levels, while fat intake had little effect in these populations with relatively low-fat diets.

Complex carbohydrates lower cholesterol – how does this work? Complex carbohydrates carry with them more micronutrients and fiber: more complex carbohydrates, more micronutrients. If micronutrients are protective, then complex carbohydrates might be expected to lower the death rate from heart disease, and this was found to be the case in a 1970 study (which included populations with higher fat intakes) published by the World Health Organization (Masironi 1970):





Left: With increasing prosperity, people eat more fat and sugar and consequently experience a reduced intake of micronutrients. Countries with the highest death rates (Finland, Australia, USA, New Zealand and Canada) ate the most "empty" calories, and countries with the lowest death rates (Jordan, El Salvador, Taiwan, Philippines and Guatemala) ate the least. This suggests micronutrients are far more important than has been thought.

Right: The more complex carbohydrates in the diet, the lower the heart death rate. Complex carbohydrates in this study were carbohydrates not from sugar or simple carbohydrates (sugars found in milk, fruit etc.). Data on sugar intake were available for 30 countries; cholesterol levels were not reported.

Sugar *and* fat

In a review entitled *Sugar and Fats: The Neurobiology of Preference*, Dr Allen Levine pointed out that "Fat and sugars are highly preferred … as mixtures in foods" (Levine 2003). The reason is likely that sugar makes fat palatable, since both must be present for cake, say, to be delicious (Emmett 1995).

Studies have implicated the characteristic Western dietary combination of sugar *together with fat* in the genesis of insulin resistance. "… we have shown in short-term animal studies that hyperinsulinemia can be induced independent of obesity by placing them on a high-fat, high-sucrose diet" (Barnard 1992). High-quality USDA studies have shown that triglycerides rise in humans when sugar or fructose is eaten in a 42%-fat diet, but not when the sugar is replaced by starch (Hallfrisch 1979, Cohen 1988). The significance of this is that starch contains glucose, but no fructose: triglycerides rise in humans when there is *fructose* in their diets. A further analysis of this data showed that sugar elevates both fasting insulin and fasting glucose, which is to say that it causes insulin resistance (Reiser 1979). Moreover, fructose (but not starch) in a diet high in saturated fat elevated triglycerides and lowered HDL-cholesterol, and these effects were stronger in insulin-resistant men (Reiser 1989).

Recall that "the ratio of triglycerides to HDL was a strong predictor of myocardial infarction … RR in the highest compared with the lowest quartile=16.0 …" (Gaziano 1997):

Table 5. Relative Risk of Myocardial Infarction Quartile of Log Triglyceride Level/HDL level

Risk Factor Adjusted[1] RR (n=599) (95% CI)	1.00 Referent	4.1 (2.0-8.5)	5.8 (2.8-12.1)	16.0 (7.7-33.1)	P<.001

Risk factors-adjusted multivariate model includes control for sex, age (10 year categories), history of treatment for hypertension, history of diabetes mellitus, body mass index, type A personality, family history of premature myocardial infarction, alcohol consumption, physical activity, smoking (never, past, current -<1 pack, 1-<2 packs, 2+ packs), caloric intake, percent of caloric intake as saturated fat.

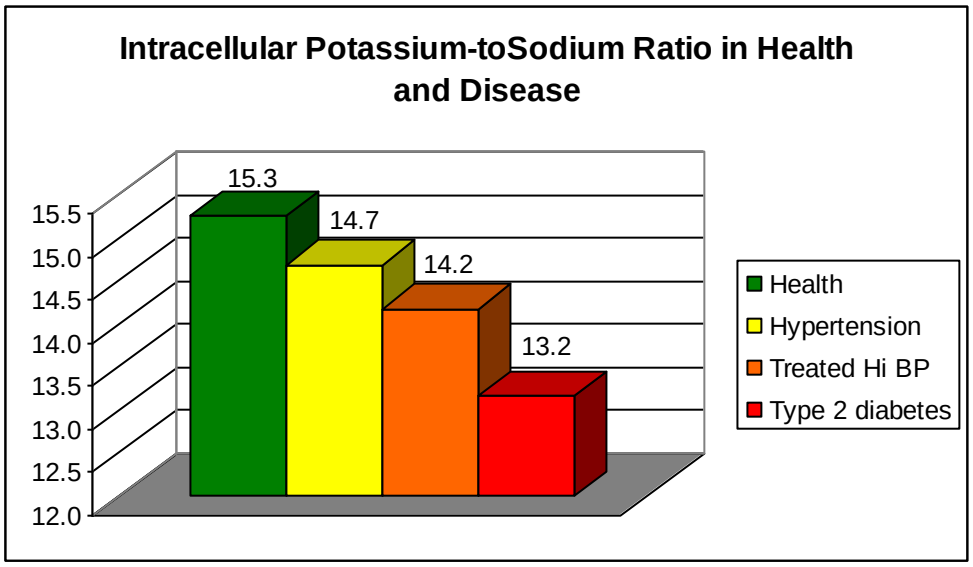
What this table means is that the ratio of triglycerides to HDL-cholesterol predicted a 16 times elevated risk of heart attack *independent* of the effects of high blood pressure, diabetes, overweight, exercise, a family history of heart trouble, smoking and even sex! These studies involved giving fructose to humans in diets with typically American levels of fat and sugar, and they showed that blood fats changes in a direction that increases the risk of heart attack by up to 16 times. Parenthetically, the title of another study describes succinctly why this should be so: "A high-fat, refined carbohydrate diet induces endothelial dysfunction [meaning the arterial lining stops controlling the blood pressure and clotting tendency] and [induces] oxidant/antioxidant imbalance [promotes inflammation] and depresses NOS protein expression" Roberts 2005). In other words, the combination of sugar and fat shuts down the endothelial cells' all-important nitric oxide production that is essential for proper blood vessel function, and, even worse, shuts down the production line for the NOS-producing proteins.

Over-nutrition leads to weight gain

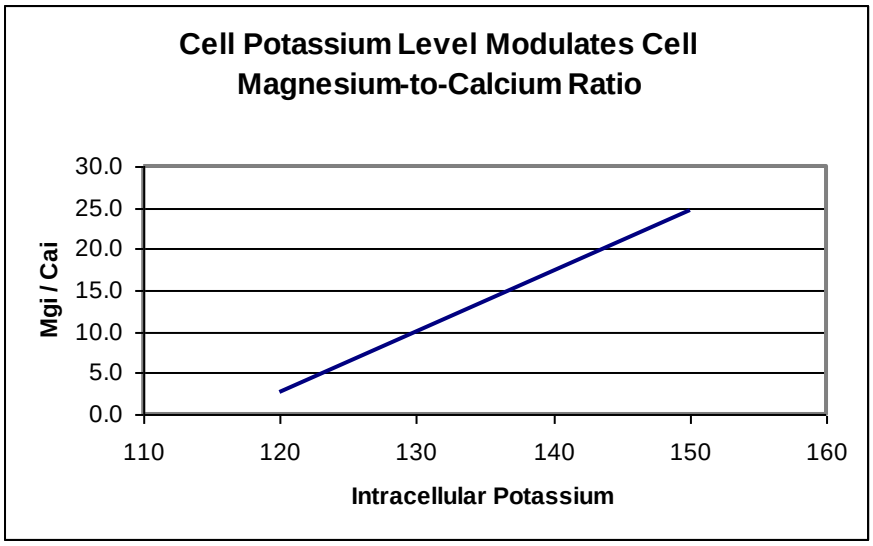
Problems ensue when we consume too much food at once. Consider that the sugars from a soda reach the bloodstream in moments, while the equivalent amount of sugars contained in several apples is absorbed over a period of hours. The fructose *must* be processed (because the liver cells have no rate-limiting step in the fructose pathway) and made into triglycerides since the amount far exceeds the cells immediate requirement for ATP and glycogen. Meanwhile, the glucose raises the insulin level just as the glucose pathway is blocked by phosphate deficiency (the phosphate ions are all doing their duty in the fructose pathway). But phosphate is required for the synthesis of ATP which fuels all cellular processes, so that, paradoxically, too much food at once causes the cell to run low in energy. This will slow the sodium pump, which will slow the sodium-calcium exchange pump and allow calcium to build up in the cell, causing insulin resistance. If, say, a cheese Danish is eaten with the soda, triglycerides made by the gut will be flood into the bloodstream at the same time as the liver is trying to dispose of the triglycerides made from the fructose, causing metabolic chaos in the fat cells as well. Frequent sugar-and-fat snacks, so much a part of the Western way of life, not only keep the triglyceride pathway turned on and cause high triglycerides and LDL-cholesterol, but they also force the development of insulin resistance.

High-salt diets low in vegetables slow the sodium pump and cause high blood pressure

Potassium ions play a part within the cell in both hypertension and diabetes. Potassium is low in the American diet (2,723mg IOM) compared to the RDA (4,700mg CDC) because we eat so few vegetables and fruit: "Less than 11 percent meet the current USDA guidelines for both fruits and vegetables" (Casagrande 2007). Furthermore, low dietary magnesium makes things worse: "Lack of magnesium is a major factor in the loss of potassium", since "magnesium is absolutely needed for potassium transport into tissues" (EXA Interpretation). Interestingly, Dr Lawrence Resnick found that "the depletion of cellular K_i [intracellular potassium ions] or Mg_i [magnesium ions] and/or Ca_i excess [calcium ions] can each directly produce or predispose to vascular smooth muscle contraction, increased constrictor tone, [and hence] increased blood pressure, insulin resistance and abnormalities of glucose and insulin metabolism". In an NMR study, he found potassium ions to be lower in the cells of hypertensives than in those of healthy controls; hypertensives treated with potassium-wasting diuretics and diabetics had even lower levels (Resnick 2001):



These changes parallel the changes in the ratio of intracellular magnesium-to-calcium ions shown above. "Although the causal mechanisms of these ionic changes have yet to be defined, the observation here of K_i [intracellular potassium] depletion in hypertension and diabetes and its strong linkage with Mg_i {intracellular magnesium] and Ca_i [intracellular calcium] levels further supports the notion of increased vasoconstrictor tone and insulin resistance as different tissue manifestations of a common cellular ionic defect" (Resnick 2001):



Alarmingly, the body potassium pool of Westerners shrinks with age while the bodies of animals maintain a youthful level of potassium into old age (Henningsen 1985):

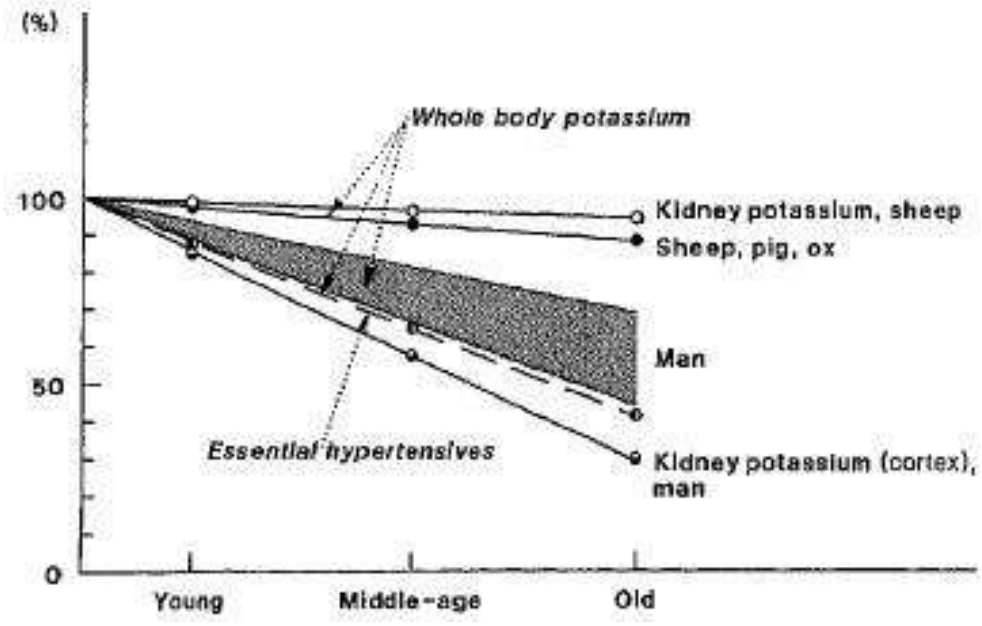
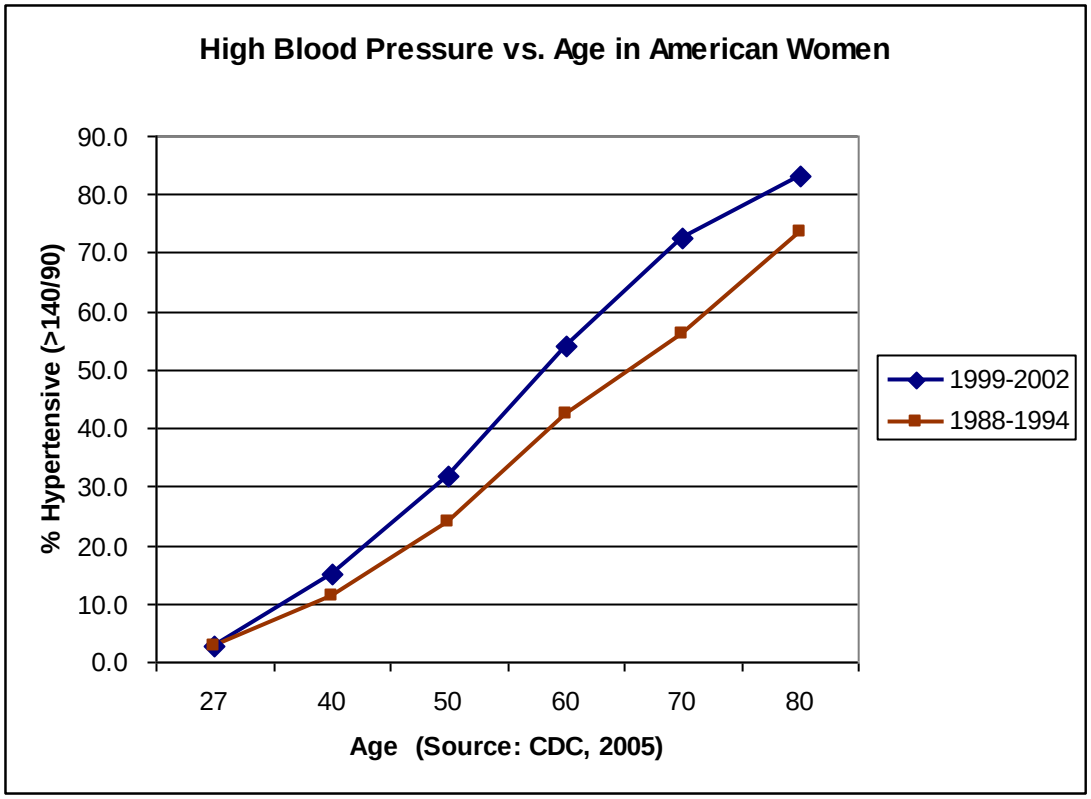


Fig. 2. In modern, "normal" man the whole body potassium decreases with age considerably (shaded area) [1, 2, 3], this tendency is less or non-existing in animals [4, 5]. Patients with essential hypertension but also patients with adult diabetes, obesity and alcohol over-consumption tend to have lower tissue stores of potassium than "normal" man [2, 21, 22]. The kidney seems to be the most prominent organ in man, in which potassium and phosphor depletion with increasing age is found [3]

Dr Henningsen, who drew this graph, pointed out as early as 1985 that the amount of sodium pump activity is directly related to energy production within the cell, and that:

There is a growing evidence for that in modern societies the function of the cellular sodium-potassium pump … in several tissues in man cannot respond adequately to demands. This is not seen in any other free-living vertebrates on this earth. The clearly unphysiological very high intake of sodium-chloride (salt) and also alcohol is definitely playing an important role in the development of the common degenerating metabolic aberrations, e.g. essential hypertension, diabetes II and severe overweight, in man. … There is a considerable overlap between essential hypertension, diabetes II and overweight in our populations [22] and it has to be considered that some very common life-style factors, e.g. high intake of alcohol, a high sodium/potassium ratio in food and a relatively low intake of magnesium and phosphate, however slow acting, are responsible. It is more and more evident that sodium restriction together with potassium supplementation or a high intake of fresh food is effective in the treatment of hypertension [57] in diabetes II [47, 58] and overweight [59] (Henningsen 1985).

Interestingly, the prevalence of the Syndrome X cluster of conditions – hypertension, dyslipidemia, obesity and impaired glucose tolerance – characteristically parallel potassium depletion and increase with age. High blood pressure is a physiological marker for aging in the West:

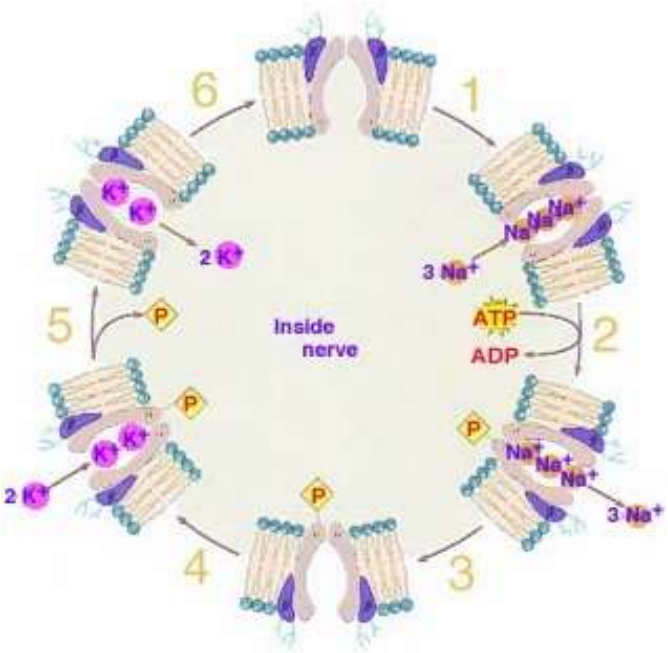


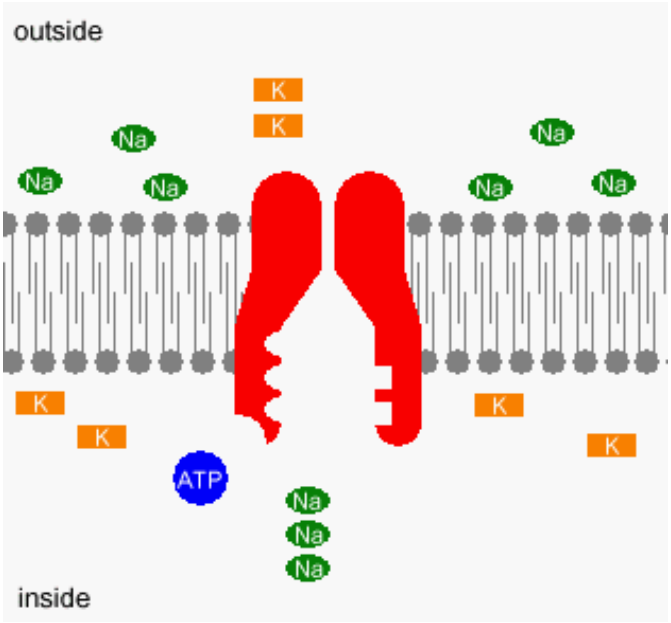
By 80 years of age, 83% of American women have high blood pressure! Furthermore, the graph shows that the situation is getting worse: over the last decade, the proportion of 70-year-old women with high blood pressure has risen by 26%. Compare the Kitava Islanders, *none* of whom has high blood pressure at any age; their experience would consist of a horizontal line across the bottom of this graph. According to the researches of biophysicist Dr Richard Moore, blood pressure is sensitive to the ratio potassium to sodium in the diet.

Only scientists who have spent their lives looking at these systems can truly appreciate what a fantastically interconnected system the cell actually is. A spider's web is the best analogy I can think of. If you touch one part of the web, every other part moves also. But whereas a spider's web is a two-dimensional network, the living cell is a multi-dimensional network. Where the different regions in a spider's web move a bit when one part is touched, different regions of the living cell go through complete transformations as a result of an initial change in just one particular part.

Dr Richard Moore

Dr Richard Moore explains the mechanism by which these alterations in cellular minerals alter the function of the cells in his book, The High Blood Pressure Solution (Healing Arts Press, Rochester VT, 2001). Cells expend between a quarter (muscle cells) and three-quarters (nerve cells) of all their energy driving *sodium pumps*. Sodium pumps are mechanisms in the cell wall which drive sodium out of the cell and pump potassium in; this takes power, and the result is a voltage across the cell membrane which powers other pumps in the cell membrane. This animation shows a sodium pump using the energy of an ATP molecule to pump 3 sodium ions (Na) out of a cell and import 2 potassium ions (K) into it, maintaining concentrations of about 14 times more sodium outside the cell, and 35 times more potassium within it (Biochemistry 4 and BioPix4U):





Left: 1. Three Sodium ions (Na+) are attracted into a cavity inside the protein embedded in the cell membrane by carbonyl functional groups lining the cavity. 2. Following the bonding of the three Na+, a section of the protein inside the cell reacts with an ATP molecule, resulting in one of the phosphates bonding to the protein, bringing with it much of the energy stored previously in its bond to ATP. The influence of the phosphate's oxygen and energy causes the cavity to twist, narrowing the channel's inner neck while widening the outer passage. 3. The change in protein shape perhaps enlarges the cavity reducing the ability of functional groups lining the cavity to attract the Na+. The ions, now finding stronger attraction to water in the exterior environment, depart from the cavity leaving it empty. 4. The cavity is now attractive to two, 25% larger Potassium ions, K+. 5. The presence of the K+ bonded to the protein's functional groups is apparently sufficiently different from the situation with attached Na+ to result in the phosphate group being released which results in the outer cavity opening closing and the inner one reopening. 6. Much as the change in cavity shape encouraged the earlier release of Na+, the now probably smaller cavity size when open inward may crowd the K+ enough to encourage them to abandon attraction to the carbonyl groups in exchange for being surrounding by water inside the cell. With the cavity again empty, the protein pump is ready to repeat the process of pumping Sodium out and Potassium into the cell.

Right: The animation shows an artist's conception of the pump at work.

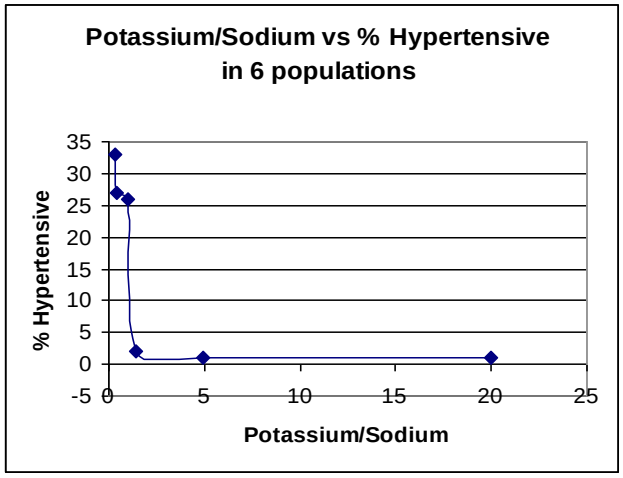
That the Western diet has too much salt and insufficient potassium- and magnesium-containing fruits and vegetables is widely acknowledged: "only 11% met the USDA guidelines for both fruit and vegetables" (Casagrande 2007). This means too much sodium and/or too little potassium outside the cell for the sodium pumps to do their job. As sodium begins rise, the cell *must* excrete an equal number of potassium ions for the cell to stay in osmotic equilibrium with its surroundings, or water would be drawn into the cell leading to edema and ultimately cell death. This is because sodium and potassium constitute the vast majority of ions in the cell, so their sum controls the osmotic pressure across the cell wall: if the cell did not expel potassium ions, it would blow up like an over-inflated balloon.

Other pumps depend on the sodium gradient across the cell wall for energy. Rising sodium lessens the efficiency of the *sodium-hydrogen exchange pumps*, cell-wall mechanisms that push hydrogen ions (also known as protons) out of the cell in exchange for sodium ions. More hydrogen ions within the cell cause the cell contents to become more acid, and therefore more prone to reproduce (which is only appropriate in the young of the species), meaning more cancer. In addition, rising sodium with the cell lessens the efficiency of the *sodium calcium exchangers*, a cell-wall mechanism that imports magnesium and exports calcium to maintain the desirable 10,000-to-1 gradient of calcium concentration across the cell wall. Low magnesium outside the cell renders the cell membrane more permeable to calcium, and this phenomenon is so well established that magnesium has been called "Nature's physiologic calcium-channel blocker" (Iseri 1984). In short, a high salt intake causes higher calcium concentrations within the cell, and, if magnesium is in short supply, the calcium concentration inside the cell will rise still further.

These changes affect each and every cell in the body, and have far-reaching effects. For example, in fat and muscle, increased intracellular calcium (Draznin 1988) and decreased magnesium (Takaya 2004) cause insulin resistance, which the pancreas compensates for by secreting more insulin so that insulin levels become elevated. Then higher insulin levels cause the kidneys to retain more sodium, making the imbalance of sodium and potassium worse. This vicious circle further slows the sodium pump and lets even more calcium enter the cell. Higher insulin levels influence the liver to make more VLDL particles that release triglycerides and become LDL-cholesterol in the circulation, so that both LDL-cholesterol and triglycerides tend to rise.

Rising calcium causes muscle cells to contract. It is the rise of calcium in the muscle cells that causes rigor mortis after death. When calcium rises in the muscle cells in the walls of small arteries, they contract and increase resistance to blood flow which raises the blood pressure. Higher calcium in the cells of the sympathetic nervous system cause them to secrete more noradrenalin, which both makes us anxious and further raises the tone of the musculature of the wall of the arterioles. Higher calcium levels within the pancreatic β-cells cause them to over-secrete insulin, further elevating insulin levels. High insulin acts as a growth factor, causing proliferation of the smooth muscle of the arterial wall, which explains why the arterioles of hypertensives become "muscle bound." It also explains why muscle cells in the middle layer of the arterial wall proliferate in atherosclerotic plaque. The elevated acidity within the smooth muscle cells of the arterial wall (recall that high calcium interferes with the cell-wall sodium-hydrogen exchange pumps leading to greater acidity) causes them to produce more collagen, which appears as scar tissue and stiffens the walls of the small arteries of long-time hypertensives. These changes all work to raise the blood pressure, a chain of events elegantly corroborated in a 5-year study in which high sodium within the cells predicted the development of hypertension, while no one with normal sodium levels developed it (Ambrosioni 1986).

Plotting the potassium-to-sodium ratio against the percentage of the population with hypertension reveals that the critical dietary ratio is about 2:1, and populations whose diets are below this level suffer a very high incidence of hypertension (data from page 96 of *The High Blood Pressure Solution*):



Correcting the balance of potassium and sodium in the Finnish diet while adding magnesium led to a 60% decline in death from both stroke and heart attacks in the North Karelia project (although these results were questioned by a lead author of the study!):

> … the population adherence to recommendations to decrease the intakes of sodium and saturated fats, and to reduce the sodium-to-potassium ratio and the saturated-to-unsaturated fat ratio, has been good. These dietary changes appear to account for a major part of the fall of blood pressure and the decrease in the cardiovascular diseases. Currently a rapid further population-wide decrease in the dietary sodium-to-potassium ratio is taking place, due to a decrease in the use of salt and replacement of common salt by a novel sodium-reduced, potassium-, magnesium-, and l-lysine HCI-enriched salt, both in home kitchens and in the food industry (Karppanen 1996).

The salt used was Pansalt, and the product was not only heavily promoted by the Finnish government as a healthful salt substitute but also made available to manufacturers of processed foods who incorporated it into many of the Finns' favorite foods like salami and canned fish; Pansalt is marketed as Cardia Salt in America. Pansalt contains magnesium, which makes the correction of dietary potassium and sodium much more effective because magnesium is a natural calcium-channel blocker. Interestingly, a recent study found simply switching to a conventional potassium-enriched salt lowered the heart death rate among veterans from 20.5 to 13.1 per 1000 (Chang 2006).

Dr Moore emphasizes that to bring down elevated blood pressure, the potassium-to-sodium ratio or (as he calls it) the K factor of the diet must be at least 4:1 (four parts potassium to one part sodium) and this level must be maintained for a month to see any change and up to *six years* for the most lowering possible. Refined foods have very low ratios – about 0.1:1 for Saltines and corn flakes – while fruits and nuts have very high ratios: bananas at 260:1, for example, and almonds at 440:1. Nuts not only have a high potassium-to-sodium ratio, they also contain plentiful magnesium that may contribute to their heart-healthy effects: "It has been estimated that 1oz of daily nut ingestion may reduce the risk of fatal CHD by 45%" (Strahan 2004). Some food for thought:

Best Potassium to Sodium ratio …	Most magnesium …	Best in both …
Beans (black)	Walnuts	Almonds
Pecans	Spinach	Avocado
Banana	Shrimp	Banana
Orange	Rice (brown)	Beans (black)
Walnuts	Pecans	Pecans
Almonds	Pumpkin seeds	Rice (brown)
Avocado	Beans (black)	Walnuts

Salt-restriction for lowering blood pressure has a spotty history. Gary Taubes wrote a scathing article in *Science* about the controversy, pointing out that neither side held strong cards yet they each claimed the same evidence supported their contention (Taubes 2000). However, a meticulous study published in the *New England Journal of Medicine* established that correcting the sodium intake of the diet really does lower blood pressure, and that the blood pressure is lowered further if potassium is increased:

> … the combined effects on blood pressure of a low sodium intake and the [high potassium] DASH diet were greater than the effects of either intervention alone and were substantial. In participants with hypertension, the effects were equal to or greater than those of single-drug therapy. … the combination of the two interventions achieved the greatest effect on blood pressure, and therefore, both — not just one or the other — merit recommendation. … Our results should be applicable to most people in the United States. Approximately 50 percent of the adult population of the United States and 80 percent of those 50 years of age or older have a blood pressure of at least 120/80 mm Hg, which is the upper limit of optimal blood pressure and which was the lower limit of the eligibility requirements for blood pressure for our trial. Furthermore, epidemiologic studies suggest that diets low in sodium and high in potassium blunt the rise in blood pressure that normally occurs with age. The intervention periods in our trial were, of necessity, brief — just 30 days (Sacks 2001).

The "low sodium intake" in this study was one third of our national average intake of 9 grams of salt per day, and systolic blood pressure dropped by about 12 points in hypertensive subjects. Interestingly, lowering blood pressure with drugs does little to reduce heart and stroke risk:

> It is widely believed that randomised trials have proved that lowering blood pressure is beneficial. Actually, that is not true. All antihypertensive drugs have profound effects on the cardiovascular system, aside from their haemodynamic (blood pressure lowering) effect. How much, if any, of the observed risk reduction can be ascribed to the reduction in pressure and how much to the direct action of the drug on the cardiovascular system? Motivated by the belief in the linear relationship of risk to pressure, many automatically attribute the risk reduction to the pressure reduction, ignoring the direct action of the drugs on the target outcomes. But results of a multitude of clinical trials make it clear that such a simplistic view cannot be true. In fact, evidence is mounting (especially from the newer trials) that it is the direct effects that are producing most, if not all, of the benefit and that the accompanying blood pressure reduction may be just an inconsequential side effect (Port 2000).

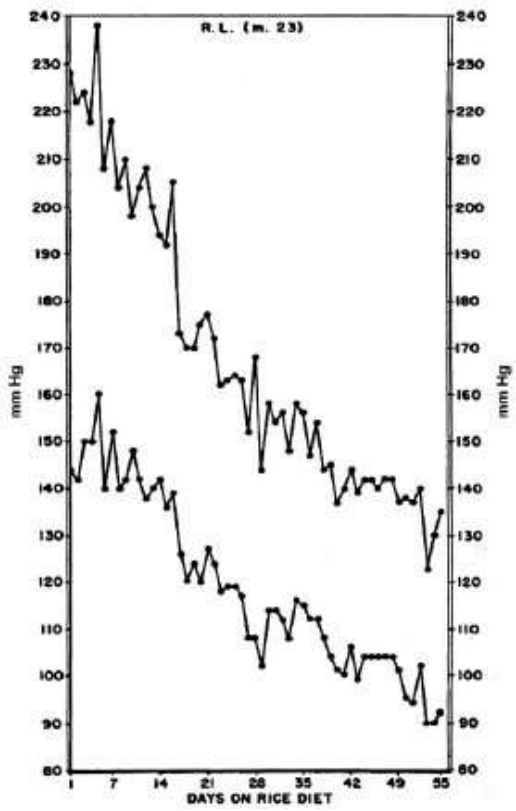
In the Medical Research Council trial, drug-treated hypertensives have fewer strokes – but *total* cardiovascular mortality was unchanged because the treatment caused a 25% *increase* in the death rate for women:

> … these results provide clear evidence that active treatment was associated with a reduction in the stroke rate in this mildly hypertensive population and shows no clear overall effect on the incidence of coronary events. Active treatment had no evident effect on the overall all cause mortality, but there was a beneficial effect in men and an adverse effect in women … this trial has shown that if 850 mildly hypertensive patients are given active antihypertensive drugs for about one year, about one stroke will be prevented. … Treatment did not appear to save lives or substantially alter the overall risk of coronary heart disease. (MRC Trial 2001)

If, on the other hand, treatment is by correcting the diet, the outlook is markedly improved. Correcting the potassium-to-sodium ratio in the diet not only lowers stroke and overall CHD mortality, it lessens the damage from strokes which do occur. Dr Moore explains that much of the brain damage due to strokes is caused by high calcium within the neurons which allows more sodium to enter, which, in a vicious cycle, allows more calcium to enter. This eventually triggers the release of the neurotransmitter glutamate which makes the membrane even more permeable to calcium, until the calcium level is such that digestive enzymes are released from the lysosomes ("sacs") within the cells, which kills them. But if potassium is plentiful, this vicious cycle does not occur until oxygen levels are much lower, so that more brain tissue survives.

Further, it is rare for vegetarians to become hypertensive, develop heart disease or have strokes. Between 1% and 2% have hypertension, compared with between 30% and 40% of non-vegetarians. Vegetarian groups studied include Trappist monks in Holland and Belgium, Seventh-Day Adventists in Australia, and vegetarian communities in Tel Aviv, Israel, and in Boston, Massachusetts. Vegetarian diets are higher in potassium and magnesium, and the blood pressure seems proportional to the dietary potassium-to-sodium ratio. A review of the National Health and Nutrition Examination Survey data showed that a "dietary pattern low in mineral intake, specifically calcium, potassium and magnesium was associated with hypertension in American adults", and this pattern was present in data from NHANES III and IV." The review concluded that "dietary management of hypertension may be more effective if the focus is on the overall nutritional profile rather than single-nutrient intake" (Townsend 2005).

Yet lowering salt intake was for years the treatment of last resort for hypertension, obesity, kidney failure and heart disease. Dr Walter Kempner used a rice and fruit diet for years at Duke University, with remarkable success. In 2,000 calories, the diet contains 5g or less of fat, about 20g of protein, and not more than 150mg of sodium giving a potassium-sodium ratio near 300:1 (Shaldon 2002) :



Left: The blood pressure of patient ML, a male aged 23, suffering from chronic renal failure, fell from 230/145 to 135/90 over 8 weeks on the rice-and-fruit diet; Right: Dr Walter Kempner *circa* 1940.

Poor Dr Walter Kempner is not well remembered. Gary Taubes mistakenly called him "Wallace", writing that "Wallace Kempner's regimen was also extraordinarily low in calories and fat and high in potassium, factors that themselves are now known to lower blood pressure" – but it is not clear to me how the emergence of newly-discovered protective factors invalidates the approach. Similarly, Dr Walter Gratzer twice called him "Kampner" in his otherwise entertaining book *Terrors of the Table*, and derided the rice-fruit diet as "gruesome … tasted like damp Kleenex … administered to more than 18,000 obese patients at extortionate cost … his intimidating presence … cowed his patients into persisting." I might consider these valuable motivating factors if I had malignant hypertension.

Yet Dr Kempner's rice-fruit diet lowered blood pressure very effectively, and what's more, when salt was added to it, blood pressure rose once again (Dole 1950):

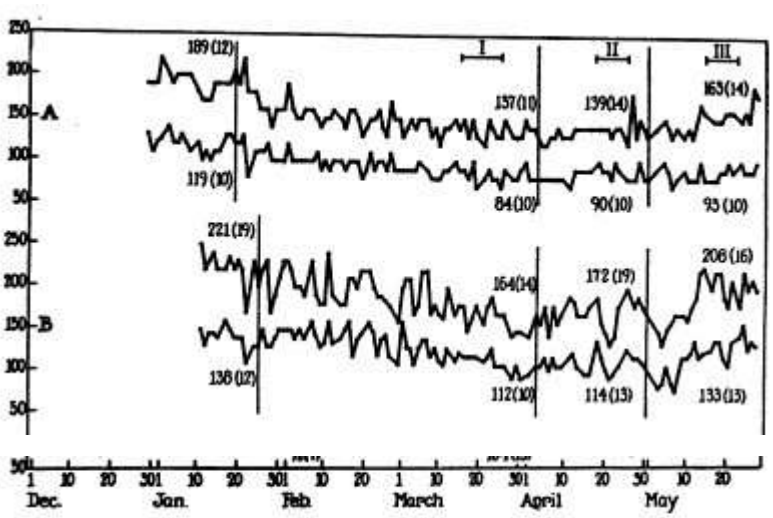
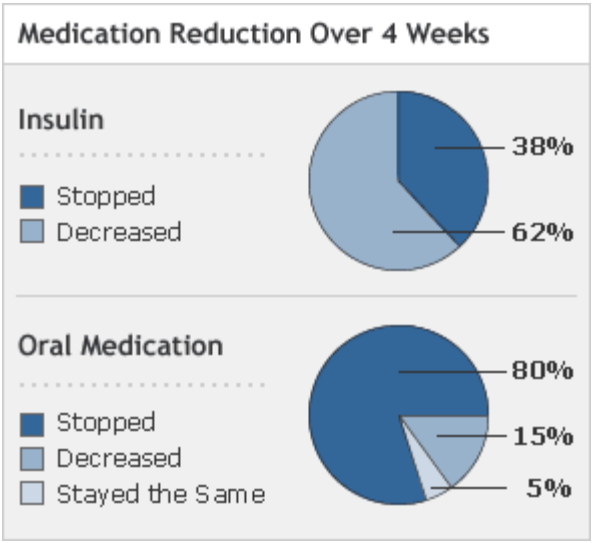


FIG. 1. MORNING BLOOD PRESSURE VALUES

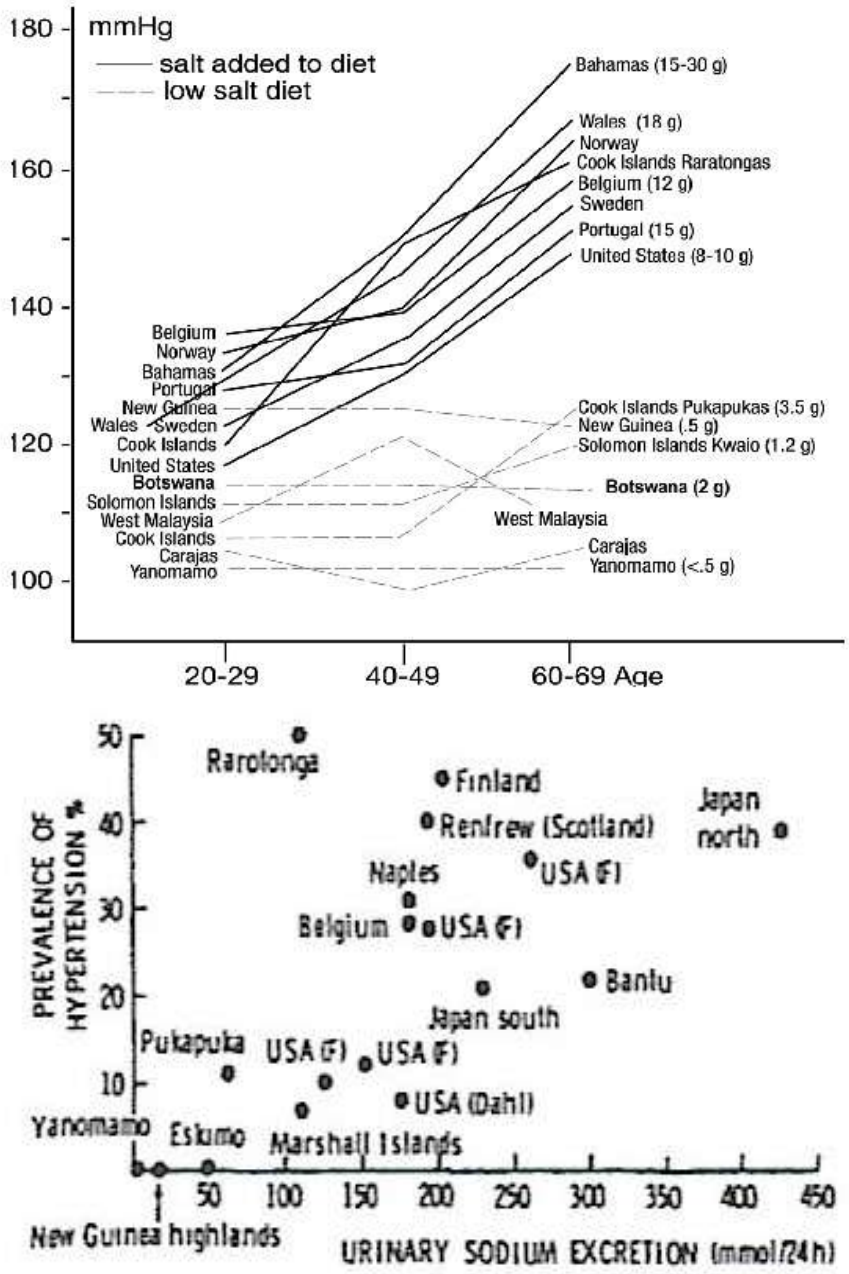
The graph shows the blood pressures of patients A and B were 189/119 and 221/138 before treatment. Over six weeks of the rice-fruit diet with its potassium-to-sodium ratio of 12:1, their blood pressure dropped to 137/84 and 164/112 (Period I). Adding 2 grams of salt changed the potassium-to-sodium ratio to 0.74:1 and sent their pressures back up to 163/93 and 208/133 (Period III). It is difficult to imagine confounding factors which would explain away the direct and immediate contribution of salt to high blood pressure in this study.

Interestingly, Dr Walter Kempner's rice and fruit diet not only lowered blood pressure, but also reduced the need for insulin and oral medications in diabetics over 30 days of in-clinic dieting (The Rice Diet Clinic):



This is extraordinary because the rice and fruit diet is perforce a very high carbohydrate diet, so that conventional thinking would be that such a diet would *increase* the need for insulin. However, the diet evidently reduces insulin resistance and/or restores β-cell function. Exercise is a part of the program. Dr Kempner died in 1997, but his diet is still available at The Rice Diet Clinic in Durham, North Carolina.

But back to salt. Then there are the fascinating relationship between salt intake and the development of hypertension with age, and salt output and the prevalence of hypertension (MacGregor 1985):



Left: Populations who do not add salt to their food do not develop high blood pressure with age. This association is rarely challenged, but has been attacked because factors other than salt (such as high vegetable intake and low refined-food intake) have not been ruled out.

Right: The amount of sodium excreted in the urine is a measure of sodium intake, and is strongly associated with the prevalence of hypertension in different countries. This association has been attacked on the grounds that urinary sodium excretion is an unreliable measure of sodium intake – silly stuff.

Only populations who salt their food heavily develop high blood pressure with age! Gary Taubes points out that the numerous potential confounders, including stress, potassium and magnesium intake, and fruit and vegetable intake all favor the Third World, low-salt groups. Nevertheless, Taubes conviction that the scientific evidence does not support salt restriction as a public health policy is unconvincing: the bottom line on the virtue of added salt is that it's a bonanza for the food refiners. Foods absorb up to 20% more water when salt is added. Thus, we swell the bottom line of the food industry by 20% when we purchase heavily-salted foods such as bacon, say, or soup; small wonder the industry mouthpiece, the Salt Institute, defends the practice so vigorously. No one has ever convincingly shown that avoiding salt is harmful, or that adding salt benefits health one iota; even the notoriously conservative American Dietetic Association is against the stuff, going so far as to offer an interesting Continuing Professional Education course on the subject: Diet, Hypertension and Salt Toxicity. This article is far more persuasive than that of Taubes. Therefore, in my mind, salt joins the American Diabetes Association's promotion of carbohydrates for diabetics as something that is of considerable benefit, but not to the consumer.

Insulin Resistance causes CHD *via* inflammation

There are interesting links between low magnesium intake, oxidative stress and heart risk. The low-magnesium group in a study of rats had markedly higher triglycerides and oxidative stress as well as lower HDL-cholesterol, the pattern of changes shown to increase risk of coronary heart disease (Hans 2002). Triglyceride-rich lipoproteins from rats on low-magnesium diets were more susceptible to oxidation, and promoted the "proliferation of vascular smooth muscle cells" found in the plaque-formation of atherosclerosis (Bussiere 1995). Another group of low-magnesium rats showed the risky pattern of excessive very low density lipoproteins, which were, moreover, very prone to becoming oxidized; oxidized LDLs are well-known to be virulently atherogenic (Rayssiguier 1993). In rats fed a very high starch diet, the effects of low magnesium intake were exaggerated: higher triglycerides, insulin resistance, lower HDL-cholesterol and greater oxidative stress; and when the carbohydrate was changed from starch to

sugar, the effect was even stronger (Rayssiguier 1981).

Importantly, the *insulin-resistant* obese subjects in Dr Reaven's study suffered far more *inflammation*, evidenced by their higher C-reactive protein levels: "we have recently demonstrated that elevated levels of C-reactive protein are only increased in *insulin-resistant*, overweight individuals (versus insulin-sensitive, equally-overweight individuals) (Mclauglhin 2002). A recent study found that insulin-resistant Korean children of normal weight had low antioxidant status (Shin 2006); another showed that "reactive oxygen species" (meaning free radicals) caused insulin resistance in mice (Houstis 2006), so it seems that insulin resistance is associated with oxidant stress and inflammation.

The National Health and Nutrition Examination Survey 1999-2000 revealed that 68% of citizens consumed less that the RDA for magnesium, and, incredibly, 19% got less than half the RDA. Those who got less that the RDA were between 1.48 and 1.75 times more likely to have high CRP (above 3), while those who got less than half the RDA were 2.24 times more likely to have high CRP (King 2005). Interestingly, lower magnesium was linked to higher C-reactive protein levels among 1,653 humans, some of whom had metabolic syndrome (Bo 2006). So it may be that a refined diet, rich in fructose and poor in magnesium, contributes to both metabolic syndrome and inflammation. This may account for the failure of the antioxidant trials, in which supplements of vitamin C, vitamin E and/or beta-carotene failed to arrest coronary heart disease: without restoring magnesium to the diet and restricting sugar, such a strategy is doomed to failure. Sugar itself elevated inflammatory markers in overweight humans (Sørensen 2005), and causes approximately equal amounts of magnesium and calcium to be lost in the urine (Ericsson 1990). The RDA for magnesium was established using "typical diets" and considered the effects of alcohol on magnesium excretion, but not that of sugar (Institute of Medicine).

Numerous rat studies have shown that a low magnesium intake elevates undesirable blood fats, makes these blood fats more vulnerable to oxidation, and promotes oxidative stress; and that

this oxidative stress oxidizes the undesirable blood fats, making them dangerous to the blood vessels. It is not that magnesium is an antioxidant exactly; it is more that low magnesium is pro-oxidant. Among the 300 or so enzymes which magnesium works with, the enzymes of the "respiratory chain" figure prominently; when there aren't enough magnesium ions available to activate these enzymes, electrons escape the chain and electrically unbalance other molecules, creating free radicals, which is to say oxidative stress. When healthy people were compared to people with metabolic syndrome, researchers found that "The interaction of inflammation and oxidative stress is related and increases the risk for MetS [metabolic syndrome], whereas serum magnesium levels and MetS are independently associated" (Guerrero-Romero 2006). Another study of people with metabolic syndrome showed that their oxidative stress was 3.7 times higher than normal controls, and they had "dysfunctional dense high-density lipoprotein particles displaying impaired antioxidant activity"; in normal people, HDL is the "good" protective form of cholesterol (Hansel 2004).

A second, related source of oxidant stress is the white cells of the immune system which use "respiratory bursts" of free radicals to kill invading bacteria. Low magnesium elevates this source of oxidant stress also because when magnesium is low, intracellular calcium levels rise and the white cells become more *primed* or *activated*, which is to say more inflamed and over-reactive. Interestingly, two months treatment of hypertensives with the calcium channel blocker Lercanidipine lowered CRP by lowering the number of circulating leukocytes (a white blood cell) while lessening the superoxide (a free radical) output of the remainder; insulin resistance fell also (Shurtz-Swirski 2006). Similarly, the endothelial cells which line the arterial system become activated when irritated by, for example, low vitamin C levels or high homocysteine levels. In a study of human endothelial cells, adding magnesium to the culture medium lowered intracellular calcium and increased their output of prostacyclin I2, an extremely valuable prostaglandin which both thins the blood and acts as an anti-inflammatory. The output of PGI2 falls precipitously when endothelial cells are "activated", meaning inflamed (Satake 2004). On the other hand, feeding a magnesium-deficient diet to human volunteers lowered magnesium in red

blood cells, causing insulin resistance and elevated thromboxane, a particularly undesirable inflammatory prostaglandin (Nadler 1993).

Low magnesium worsens homocysteine damage

Dr Mildred Seelig wrote in *The Magnesium Factor* (Avery, New York 2003):

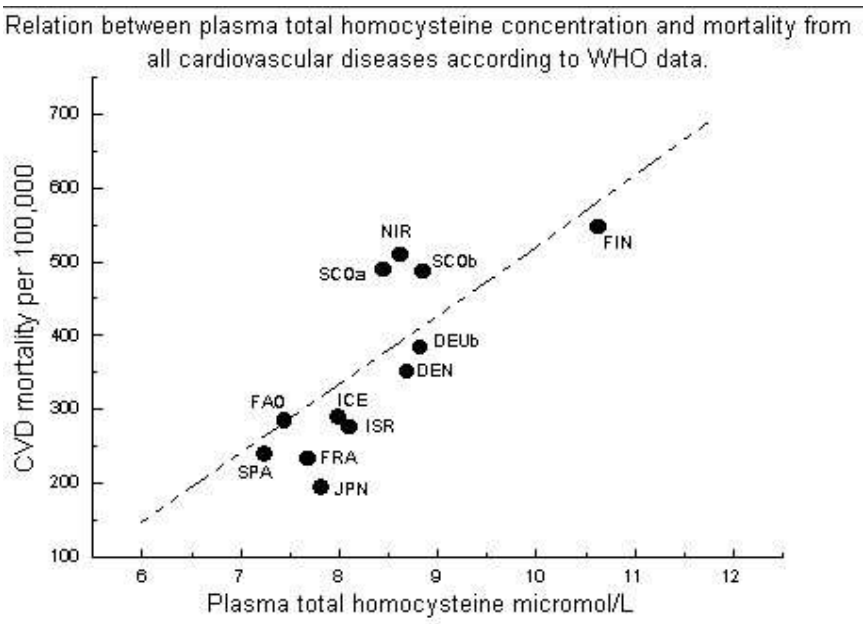
High Homocysteine and Low Cellular Magnesium

If there is too much homocysteine in a cell's environment, cellular magnesium levels drop, especially if the cell's magnesium level is low to begin with. It does not alter the calcium level. The B vitamins that lower homocysteine in the blood – folic acid, vitamin B_{12}, and vitamin B_6 – stop homocysteine from lowering the level of magnesium in the cells. But all three of these B vitamins have to be together for this to happen, and the cells' magnesium level must be adequate. If the cellular magnesium is too low to begin with, these vitamins, even if together, cannot stop the drop in cellular magnesium that homocysteine evokes.

Studies on metabolic syndrome X tell us that low cellular magnesium causes abnormal functions that lead to atherosclerosis and associated conditions. We see these same conditions in people with elevated homocysteine, including thrombosis, platelet aggregation, and endothelial damage, as well as early-onset coronary artery disease and heart attacks at young ages. It seems plausible that high homocysteine levels produce these atherosclerotic conditions by lowering the cells' magnesium content.

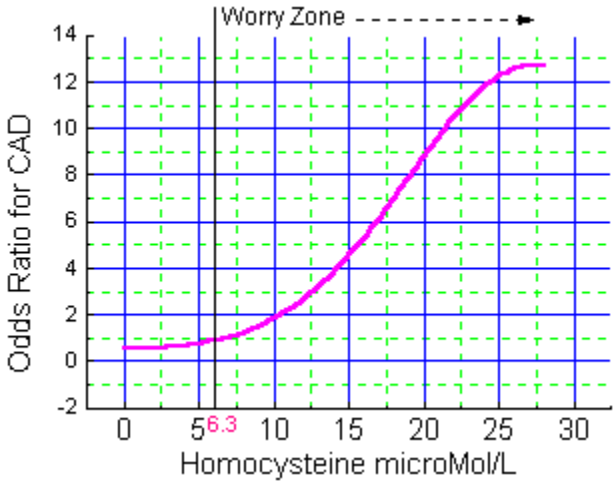
Homocysteine: Risk Factor for Heart Disease and Marker for B-Vitamin Deficiency

Homocysteine is an established risk factor for heart disease whose predictive power is considerably greater than that of cholesterol in any form, and high homocysteine is found even among people who have normal cholesterol levels. The World Health Organization has established that homocysteine levels in various countries are closely linked to their rate of cardiovascular death:



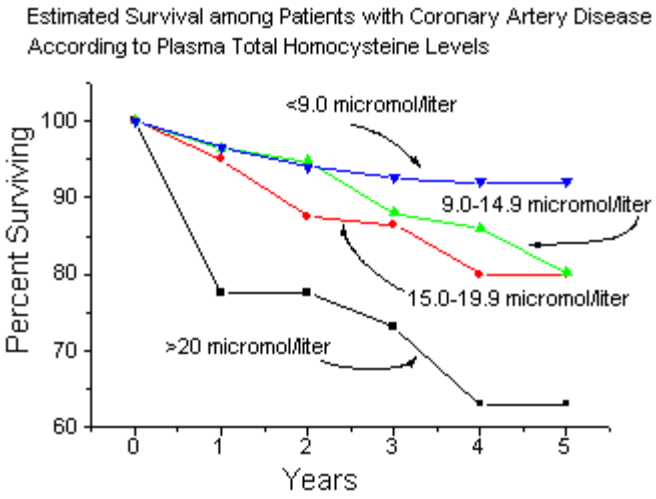
(Alfthan 1997)

The strength of the correlation between homocysteine levels and heart death rate was 0.71, which means that homocysteine levels correlate closely with the heart-death rate; it is a strong predictor. The correlation between cholesterol and heart death rate, by contrast, is considerably weaker. Further, studies have found that a more-or-less "normal" plasma homocysteine level of 14 µMol/L *doubled* the chance of Alzheimer's in the Framingham study, and has been strongly associated with the other dementias. High homocysteine multiplied the chance of coronary artery disease (CAD) by 4.8 times, compared to those with the lowest level of homocysteine:



Data from Robinson et al, Hyperhomocysteinemia and low pyridoxal phosphate. Common and independent reversible risk factors for coronary artery disease. (Robinson 1995 redrawn by Dr Glenn Tisman)

A 1997 study estimated the chances of surviving a heart attack at *three times* worse if the homocysteine is high. And high homocysteine levels *are* widespread: in this study, "more than 50% of our older patients were hyperhomocysteinemic."



Data from Nygard et al, Plasma Homocysteine Levels and Mortality in Patients with Coronary Artery Disease. (Nygard 1997, redrawn by Dr Glenn Tisman)

The B vitamins folic acid, vitamins B6 and B12 lower homocysteine. Unfortunately, "Vitamin B12 deficiency is surprisingly common among patients with vascular disease …" (Robertson 2005), and the amount of folic acid needed to lower elevated homocysteine levels to normal has actually been shown to be a great deal more that the RDA of 0.4 mg/day:

A dosage of folic acid of 0.8 mg/day appears necessary to achieve the maximum reduction in serum homocysteine level across the range of homocysteine levels in the population. Current US food fortification levels will achieve only a small proportion of the achievable homocysteine reduction (Wald 2001).

Several studies point to the dangers of elevated homocysteine associated with low dietary intakes of B vitamins:

Nutrient	How many of us have "low nutrient status"?	Amount	Relative Risk of Coronary Heart Disease
Vitamin B6 (high intake)	71%(M) 90%(F)	50 to 300mg	0.27
Vitamin B6 (low intake)	71%(M) 90%(F)	4.6 vs. 1.1mg/day	0.67(F)
Folate	88%	696 vs. 158micro-grams	0.69(F)

In the first study, Dr John Ellis prescribed high doses of vitamin B6 to patients with any sign of carpal tunnel syndrome in rural Titus County, Texas. In this retrospective investigation performed with Dr Kilmer McCulley, about one of these patients died of CHD for every four patients who died of CHD in other doctors' practices (Ellis 1995).

Furthermore, high intake of vitamin B6 lowers *xanthurenic acid*, a metabolite of the amino acid tryptophan which causes diabetes in experimental animals, and which is found in the urine of almost all diabetics. It is bad news, promoting cell death, cataracts, atherosclerosis and diabetes, among other things.

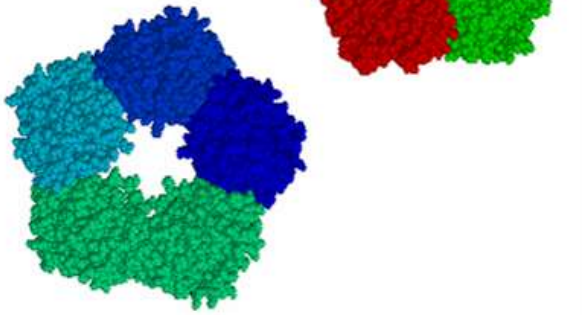
The second study, by Dr Walter Willett and others, shows that folate (which is part of the B-complex) and B6 each lessen CHD by about one third; the women who had the highest levels of both folate *and* B6 halved their risk of CHD (Rimm 1998). Vitamin B6, vitamin B12 and folate lower *homocysteine*, a cardio-toxic metabolite of the amino acid methionine, found in red meat. If we are short of these critical B vitamins, we have trouble breaking homocysteine down into non-toxic cysteine, or converting it back into methionine.

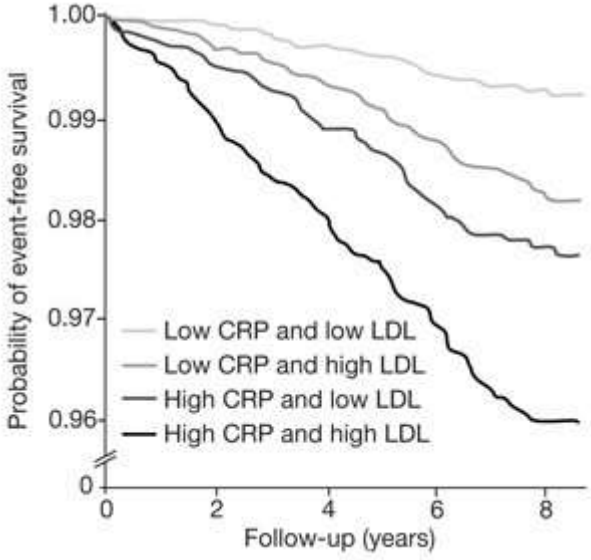
The RDA for folic acid is obviously too low because people with so-called "normal" intakes have high homocysteine. If these vitamins are taken in amounts that lower homocysteine levels to normal, the risk of heart death is cut at least by half. A further benefit is that the risk of losing your mind is also halved – and study after study finds the aged to be especially depleted.

A low magnesium intake predicts inflammation: C-Reactive Protein

Evidence from animal studies suggests that magnesium is associated with inflammatory processes. A study of the data from the National Health and Nutrition Examination Survey 1999–2000 found that the 68% of adults who consumed less than the RDA of magnesium were 1.48–1.75 times more likely to have elevated CRP than adults who consumed more than the RDA. "Most Americans consume magnesium at levels below the RDA. Individuals with intakes below the RDA are more likely to have elevated CRP, which may contribute to cardiovascular disease risk" (King 2005). Research has revealed strong links between low magnesium and high blood pressure, and the complications of diabetes (Rude 1992). Disquietingly, elevated C-reactive protein levels were found to triple the risk of Alzheimer's disease in the Honolulu Aging Study (Schmidt 2002), and were associated with almost three times the sudden cardiac death rate in men (Albert 2002).

Inflammation anywhere in the body causes the liver to create C-reactive protein, a molecule that can be easily measured from a blood sample and is widely used as a marker for inflammation:

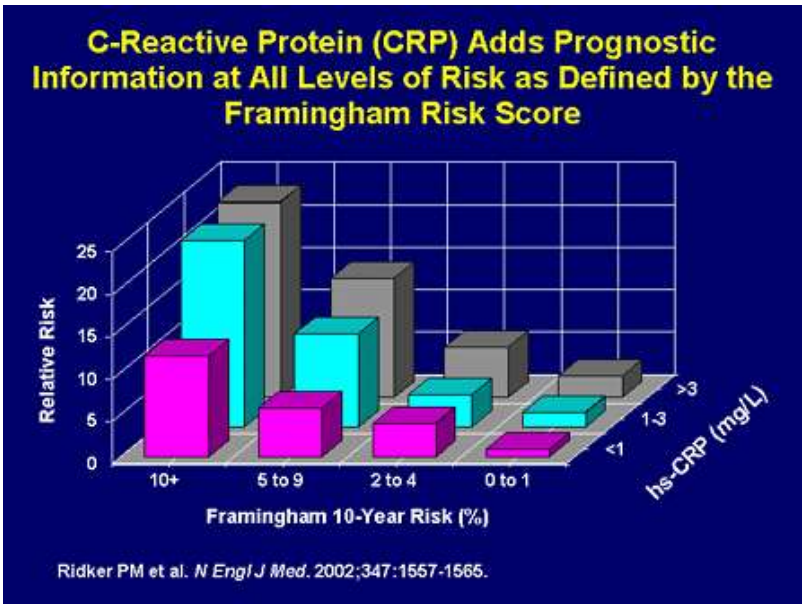




Left: "CRP is a member of the class of acute phase reactants as its levels rise dramatically during inflammatory processes occurring in the body. It is thought to assist in complement binding to foreign and damaged cells and affect the humoral response to disease. It is also believed to play an important role in innate immunity, as an early defense system against infections" (Search.com)

Right: "Predictive value of high-sensitivity C-reactive protein … Cardiovascular event-free survival among apparently healthy individuals based on high-sensitivity CRP and LDL-cholesterol levels" (Ridker 2002).

C-reactive protein is a strong independent predictor of cardiovascular disease. Dr Paul Ridker calculated that the relative risk of heart attack in women divided into five groups according to their CRP scores was 1.4, 1.6, 2.0, and 2.3 times the risk for the women in the lowest quintile:



(Lipids online)

... the relative risks of first cardiovascular events according to increasing quintiles of C-reactive protein, as compared to the women in the lowest quintile, were 1.4, 1.6, 2.0, and 2.3 (P<0.001) … (Ridker 2002)

Their relative risks according to their LDL-cholesterol levels were, by contrast, 0.9, 1.1, 1.3 and 1.5, so their CRP scores were considerably stronger risk marker than LDL-cholesterol (Ridker 2002). It appears that CRP elevates insulin resistance. Critically-ill patients (who have high CRP levels) often have very high blood sugars, regardless of their diabetic status. Insulin "reduces morbidity and mortality in critically ill populations" when used in intensive care units to bring their blood sugar down to normal (Lewis 2004). Among 360 children admitted to a pediatric ICU, 52% had elevated blood sugar, and 47% had a low magnesium level (Ruiz Magro 1999).

Interestingly, CRP is more than just a marker, for it actually reacts with oxidized LDLs

(recall that easily oxidized LDLs form when magnesium is low) to form compounds which are incorporated in atherosclerotic plaque (Tabuchi 2007). Another interesting association: literally hundreds of studies have found higher CRP in the metabolic syndrome conditions. For example, C-reactive protein was almost 5 times more likely to be elevated in obese women (Visser 1999), and nearly 4 times more likely in CHD or Type 2 diabetes (Bahceci 2005). In a study of 5,502 Chinese people in Shanghai, investigators found that "Compared to those in the lowest quartile, men in the highest quartile [of CRP] had increased relative risk of hyperglycemia (3.8 times), central obesity (5.5 times), hypertension (2.8 times), hyper-triglyceride (1.3 times), low HDL-c (1.5 times), and metabolic syndrome (10 times) (Bao 2006). Similarly, a study of 3,692 Japanese men found that the more components of the metabolic syndrome were present, the higher the CRP. "Compared with men who had no such components of the MS, those who had one, two, three, four, and five or more components were, respectively, 1.48, 1.84, 1.92, 3.42, and 4.17 times more likely to have mildly [or moderately] elevated CRP levels (trend $P<0.001$) … These results indicate that a variety of components of the Metabolic Syndrome are associated with elevated CRP levels in a systemic low-grade inflammatory state" (Tamakoshi 2003).

It may be that elevated C-reactive protein itself causes insulin resistance. A recent study established that CRP causes the "phosphorylation of insulin-receptor substrate-1 … thereby impairing the insulin-signaling pathway that promotes glucose transport" into the cell (D'Alessandris 2007). Two interesting interactions between CRP and VLDL provide food for thought. Firstly, the initial CRP level of men fed high-carbohydrate diets predicted the increase of VLDL during the study: those with high CRP at the beginning of the study produced a great deal more VLDL when given plentiful carbohydrates (Desroches 2006). Secondly, CRP actually binds VLDL (Rowe 1984), and the combination "could have a pathogenic role in disseminating the process of intravascular coagulation" (Dennis 2004). We see that fructose causes insulin resistance, which causes inflammation that not only worsens the consequent VLDL-cholesterol secretion but also reacts with it in such a way as to increase the likelihood of blood clots.

Interestingly, trials show antioxidants lower CRP. In other words, antioxidants lower oxidant stress, which lessens inflammation. In one such trial, 1200IU of vitamin E was given to diabetic patients with and without micro-vascular complications: "Alpha tocopherol [vitamin E] supplementation significantly lowered levels of C-reactive protein … Alpha Tocopherol therapy decreases inflammation in diabetic patients and controls and could be an adjunctive therapy in the prevention of atherosclerosis" (Devaraj 2000).

In a second trial, 515mg of vitamin C given to subjects exposed to cigarette smoke: "Vitamin C supplementation yielded a 24.0% reduction … in plasma CRP … vitamin C, should be investigated further to confirm their CRP-lowering and anti-inflammatory effects" (Block 2004).

A further trial concluded that "Plasma vitamin C, fruit intake, and dietary vitamin C intake were significantly and inversely associated with mean concentrations of C-reactive protein … even after adjustment for confounders … Plasma (but not dietary) vitamin C also showed inverse associations with both fibrinogen concentrations and blood viscosity" (Wannamethee 2006).

Apparently, the key to lowering inflammation and CRP lies in obtaining plentiful magnesium and antioxidants. We see that a low magnesium intake predisposes to elevated CRP, and that vitamin E, vitamin C and fruits (which have a high antioxidant content) all lower C-reactive protein. This cuts the risk of heart disease and Alzheimer's disease, and lessen the risk of heart attack by modulating the clotting tendency of the blood.

Obesity and CRP revisited

Dr Gerald Reaven pointed out that not all obese people have insulin resistance. It's the ones who have insulin resistance and elevated C-reactive protein who are at the greatest risk:

… [although] increased BMI is more prevalent in insulin-resistant individuals, not all overweight/obese persons are insulin resistant. Furthermore, because CVD risk factors were accentuated in association with increased degrees of insulin resistance, independently of BMI or age, it is the subset of overweight, obese individuals, who are also insulin resistant, who are at greatest CVD risk. Indeed, we have recently demonstrated that elevated levels of C-reactive protein are only increased in insulin-resistant, overweight individuals (versus insulin-sensitive, equally-overweight individuals Mclauglhin 2002) … Thus, we believe that these results provide support for the view that the most intensive efforts to decrease CVD risk should be directed towards those overweight individuals who are also insulin resistant (McLaughlin 2004).

Inflammation causes heart attacks

Current thinking is that acute coronary syndromes – unstable angina, myocardial infarction, and sudden cardiac death – are often caused by unstable coronary plaques. Compared to stable plaques, these have a lipid core up to five times larger, contain up to eight times the macrophages (inflammatory cells), and have a fibrous cap one third the thickness. The process is thought to start with "endothelial activation" (inflammation) caused by oxidant stresses from immune system activity, smoking, homocysteine, inadequate antioxidant and/or magnesium intake and so forth. Activated endothelial cells express "adhesion molecules" which attract inflammatory immune cells. Oxidized LDL-cholesterol particles pass freely into the plaque, where they are taken up by immune cells adding to the pool of "foam cells" in the lipid core. The activated immune cells express enzymes that degrade the collagen of the fibrous cap, causing it to become thinner. This process, in which every cell type in and around the plaque is activated, is strikingly similar to other chronic inflammatory conditions such as rheumatoid arthritis (Forrester 2002).

Statin drugs inhibit an enzyme in the pathway between acetate and cholesterol, "HMG co-enzyme A reductase." This led Brown and Goldstein, winners of the Nobel Prize in medicine for their discovery of the LDL-cholesterol receptor, to predict with remarkable hubris that the availability of statins would lead to the disappearance of coronary artery disease as a public health problem by the year 2000 (Brown 1996). However, the statins small effect on heart death correlates much better with the inhibition of inflammation than with lowered cholesterol levels. Statins reduce C-reactive protein, a marker for inflammation which has been called an "emerging

risk factor" for CHD. Two studies in the *New England Journal of Medicine* suggest the benefit of statins must come from this effect:

> "Patients who have low CRP levels after statin therapy have better clinical outcomes than those with higher CRP levels, regardless of the resultant level of LDL cholesterol. Strategies to lower cardiovascular risk with statins should include monitoring CRP as well as cholesterol" (Ridker 2005).

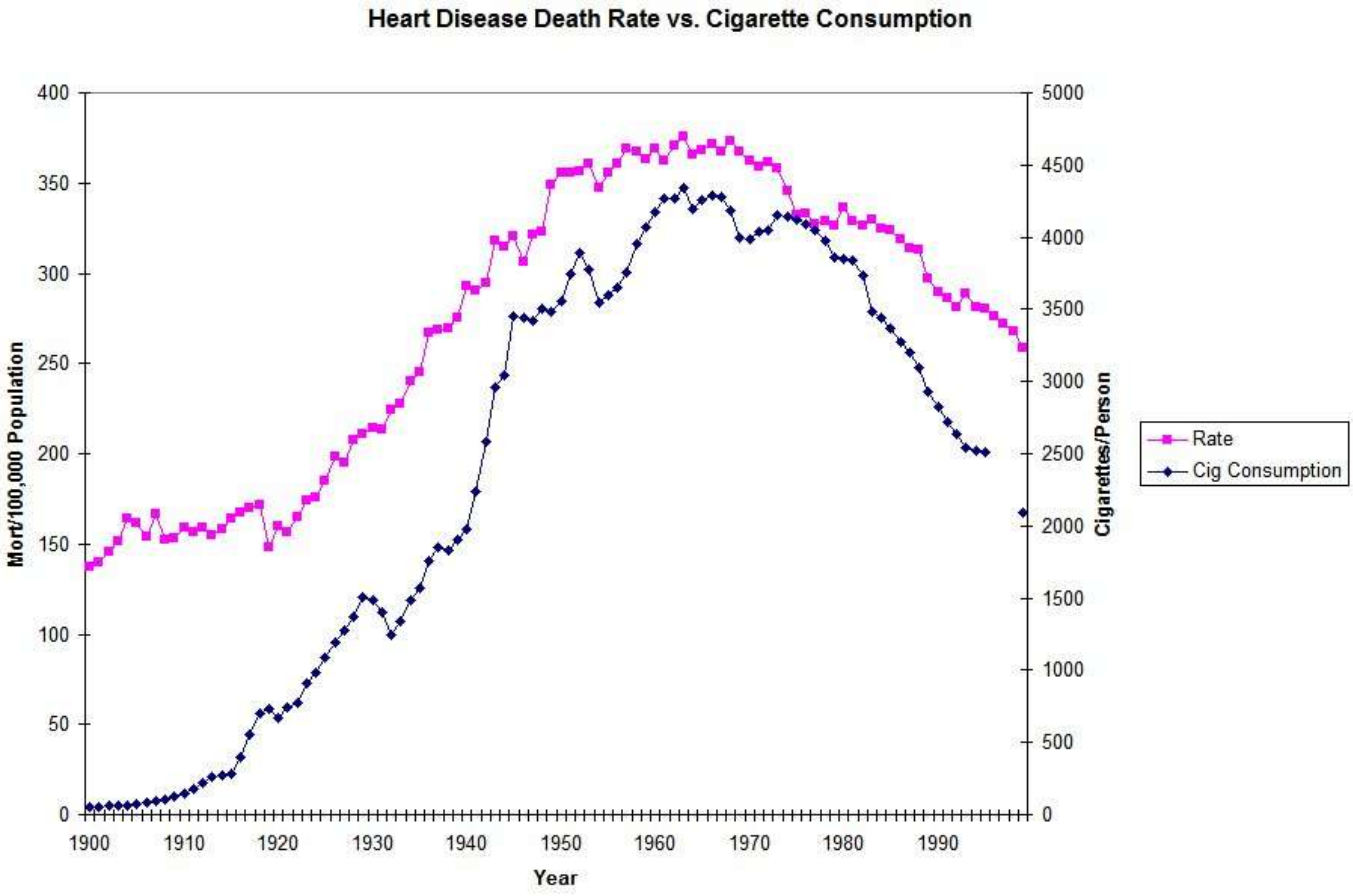
> "For patients with coronary artery disease, the reduced rate of progression of atherosclerosis associated with intensive statin treatment, as compared with moderate statin treatment, is significantly related to greater reductions in the levels of both atherogenic lipoproteins and CRP" (Nissen 2005).

These authors emphasize that in the studies showing a protective effect for statins, the effect kicks in long before the cholesterol and LDL-cholesterol levels change; and those with normal levels of LDL-cholesterol also benefit. Together, these reviews make it plain that the statins' protective effect cannot stem from their effect on cholesterol or LDL-cholesterol, but rather comes from lowered C-reactive protein, meaning their control of inflammation.

Inflammation and Heart Attacks

Chlorination of the nation's water supplies began in 1908, and was credited with eradicating typhoid fever by 1950 (WA Health). Interestingly, chlorine catalyses the oxidation of LDL-cholesterol (Hazen 1997), and oxidized LDL is powerfully atherogenic (Hamilton 1997). Iron also catalyses this reaction, but iron is lowered in women by menstruation, which may explain the lesser incidence of atherosclerosis in pre-menopausal women. Thus, chlorination may have elevated already higher levels of oxidized LDL in men, and so fulfills the requirement for the cause to affect men more than women. Dr Joseph Price showed that chlorinated water caused atherosclerosis in chickens, related in his book Coronaries Cholesterol Chlorine. The rise of bottled drinking water could explain some of the fall in deaths from atherosclerosis.

Research by Dr Dennis B Waddell on the prevalence of cigarette smoking and heart attack incidence supports the conjecture that cigarette smoking elevates heart attack risk via inflammation:



Cigarette consumption data: USDA and CDC; Heart mortality data: CDC

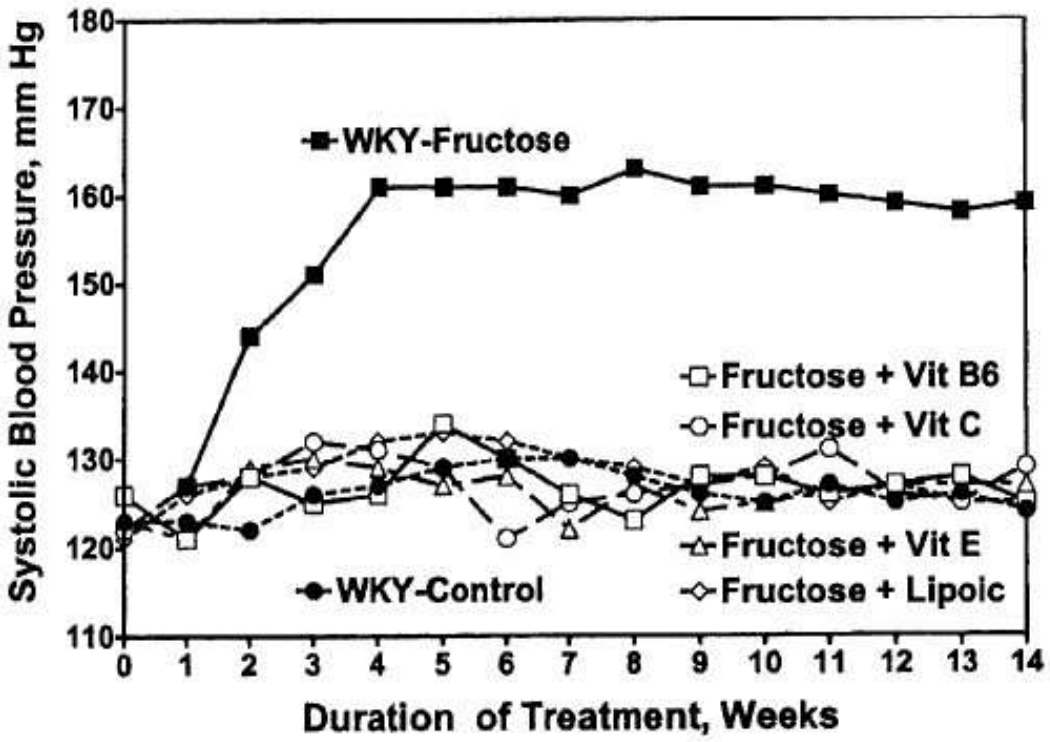
The correlation between cigarette consumption and heart deaths is strong at 0.98. Dr Waddell points out "This goes against conventional theory as there is no time lag in the correlation and it is thought that the effect of smoking on CHD takes several years to manifest." However, contrast the experience of the Kitava Islanders: 80% of them smoke, and none develop cardiovascular disease. A prominent difference between Kitava and America is the exclusive use of unrefined foods; with unrefined levels of magnesium and the antioxidants, their oxidant stress is apparently so low that they can accommodate the oxidant stress of cigarette smoking without precipitating endothelial dysfunction and atherosclerosis.

I am at a loss for other "confounders." We have more pesticides and pollution today than we did then, yet heart deaths are falling. Factors that impoverish the diet such as the synthetic fertilizers of modern agriculture are themselves a part of the proposition that a refined diet causes degenerative diseases. Proof, then, depends on fulfilling the criteria for cause and effect.

How plentiful magnesium and antioxidants prevent and treat Syndrome X

Supplementary magnesium not only protected fructose-fed rats against the development of high blood pressure (Balon 1994) but also "improved insulin sensitivity, hyperglycemia, hyperlipidemia and reduced lipid-peroxidation [i.e., lower oxidative stress] in fructose-fed rats" (Olatunji 2006). Interestingly, Dr Sudesh Vasdev of the Memorial University of Newfoundland protected fructose-fed rats from developing high blood pressure by giving antioxidants (Vasdev 2004):

Elevating Glutathione Prevents Hypertension in Fructose-Fed Rats



In these rats bred to develop high blood pressure when fed fructose, giving N-acetyl cysteine or the antioxidants vitamin C, vitamin E, vitamin B6 or lipoic acid prevented the development of high blood pressure. These substances all help in the making of *glutathione* or have a glutathione-sparing effect, and all would be present in an unrefined diet. Glutathione is a versatile antioxidant, which is also used by the liver for detoxification. Many toxic molecules are no longer dangerous once joined with glutathione, and the new compounds are easily be excreted. The toxic molecule acetaminophen (the active ingredient in Tylenol) is dealt with in this way. *N-acetyl cysteine* contains the amino acid cysteine and has the property of raising glutathione levels, and is the treatment given when people present at emergency rooms with Tylenol poisoning. "N-acetyl cysteine normalizes elevated blood pressure in SHRs [spontaneously hypertensive rats] by binding excess endogenous aldehydes" (Vasdev 2001); aldehydes are intermediary compounds which build up when cells must metabolize an excess of fructose. In rats that become insulin resistant when fed fructose, magnesium added to their chow provided protection: "Blood pressure and fasting insulin levels were also lower in the magnesium-supplemented group. These results suggest that magnesium deficiency and not fructose *per se* leads to insulin insensitivity … and changes in blood pressure" (Balon 1994). Dr Bella Altura fed supplemental magnesium to rabbits on cholesterol-rich diets, and found that it lowered their elevated blood cholesterol and triglyceride levels to normal (Altura 1990). Magnesium supplements protected diabetic rats from the vascular complications of diabetes (Soltani 2005), and returned blood pressure and blood sugar to normal levels in another group of diabetic rats (Kesharvarz 2005). Finally, rats that develop high blood pressure and insulin resistance were protected when fish oil was added to their chow (Huang 1997).

These few obscure animal studies have momentous implications. They suggest diets composed of refined foods and containing few vegetables contribute to both metabolic syndrome and inflammation because they are poor in magnesium and rich in fructose. The nutrients refined out of these diets, especially magnesium and the antioxidants, are indispensable for the cells to metabolize the foods, especially the fructose. Consequently, such diets give rise to high cholesterol and triglycerides, which are, then, nothing more than markers for failing cell metabolism; and they give rise to oxidative stress, which is probably the real culprit behind heart disease. If this is the case, then plentiful magnesium and antioxidants should both prevent the development of the Syndrome X cluster of conditions, and should vitiate them if they have already developed, always keeping in mind that "once high blood pressure [for example] is established, removing the cause does not necessarily lower the blood pressure to normal levels" (MacGregor 1983).

Human studies support this view!

Recall that in population studies, low magnesium diets are associated with more 21% more Type 2 diabetes in Black women (van Dam 2006) and 40% more CHD in men (Al-Delaimy 2004), while the highest quintile of magnesium intake protected both men (RR=0.66) and women (RR=.67) against Type 2 diabetes (Lopez-Ridaura 2004). Lower magnesium was associated with higher C-reactive protein levels, meaning inflammation, among 1,653 humans, some of whom had metabolic syndrome (Bo 2006). In 179 healthy young Blacks, the insulin-resistant subjects ate 20% less magnesium than those who were insulin-sensitive (Humphries 1999). Dietary magnesium protected against hypertension in the Nurses Health Study: "women in the highest quintile (median 434 mg/day) had a decreased risk for hypertension (relative risk 0.87 … $p < 0.0001$) compared with those in the lowest quintile (256 mg/day) (Song 2006). Among 9,887 women in the Women's Health Study, the highest quintile of magnesium consumption had CRP levels 12% lower than the lowest quintile, and a 27% lower risk of having the metabolic syndrome (Song 2005). A prospective study followed 4,637 young adults for 15 years and found the quartile eating the most magnesium had 0.69 of the risk of developing Metabolic Syndrome as the quartile eating the least (He 2006). Elevated fasting insulin correlated with low magnesium intake, high sugar intake and low exercise capacity in 5,115 healthy 18-30 year-olds (Manolio 1991). Magnesium intake predicted, "improved insulin homeostasis" in the Framingham Offspring Study (Rumawas 2006). In Dr Pereira's study of diets identical but for the use of whole wheat and white flour, the whole-grain diet contained 387mg of magnesium compared to 259mg in the white-flour diet. This 50% increase in magnesium intake correlated with improved sensitivity to insulin (Pereirra 2002).

Second, low magnesium levels are actually found in people with Metabolic Syndrome, insulin resistance, CHD, obesity, hypertension and Type 2 diabetes. Both dietary and serum magnesium were lower in 24 obese children compared to lean controls, and their degree of insulin resistance correlated directly with their intake of magnesium (Huerta 2005). Individuals with low serum magnesium levels were 6.8 more likely to have metabolic syndrome, and this association was independent of the systemic inflammation and oxidative stress found in cardiovascular disease (Guerrero-Romero 2002). Feeding a magnesium-deficient diet to human volunteers lowered magnesium as measured by Nuclear Magnetic Resonance in red blood cells, and all subjects' insulin resistance worsened (Nadler 1993). Using the "magnesium loading test" (in which 24-hour urine samples from before and after a dose of magnesium are compared), all 38 heart patients in one study were shown to be deficient in magnesium compared with healthy controls, with the worst deficiencies found in the patients who had had the condition the longest. The researchers felt that "patients with IHD [ischemic heart disease] may be severely magnesium deficient; [and] … long-term diuretic treatment contributes to this deficiency" (Rasmussen 1988). A review goes so far as to state that "Type 2 diabetes is *characterized* by cellular and extra-cellular Mg [magnesium] depletion" (Barbagallo 2007).

Thirdly, giving antioxidants and magnesium is helpful, just as they were in the animal studies. Vitamin E improved insulin action by 31% in 20 elderly subjects of normal glucose tolerance (Paolisso 1994), and in a group of Type 2 diabetics (Paolisso 1993). Giving 40 Type 2 diabetics one gram of vitamin C per day lowered their fasting insulin by 19%, and decreased their blood cholesterol, LDL-cholesterol and triglycerides (Paolisso 1995). Infusions of the antioxidant glutathione improved insulin resistance (by enhancing glucagon production) in both normal and diabetic people (Paolisso 1992). A case-control study of 742 people demonstrated a "significant inverse relationship between PLP [the active form of vitamin B6] and both hs-CRP [inflammation] and fibrinogen [clotting tendency]." The C-reactive protein level of the quartile with the lowest PLP averaged <0.81, and that of the highest quartile was >4.18 mg/L, and the CAD patients had lower PLP levels (Friso 2004). A large infusion of lipoic acid lowered insulin resistance in Type 2 diabetics by 50% (Jacob 1996), and half that amount given to 20 Type 2 diabetics for 10 days improved their insulin sensitivity by 30% (Jacob 1999).

Magnesium supplements given to 47 heart patients lowered their triglycerides and VLDLs by 27%, and lowered LDL-cholesterol by 15% in a double blind, placebo controlled study (Rasmussen 1989). Magnesium supplementation improved insulin resistance by 43% in 32 non-diabetic, insulin-resistant people with low serum magnesium (Guerrero-Romero 2004). "Chronic magnesium supplementation produced a significant reduction of plasma cholesterol and LDL-cholesterol, and an increase of HDL-cholesterol" in Type 2 diabetics" (Corica 1994). Magnesium supplementation lowered triglycerides by 27% in patients who had had heart attacks (Rasmussen 1989). Dr Guiseppe Paolisso found that dietary magnesium supplements improve the pancreatic insulin-producing β-cells' response to glucose in elderly Italian diabetics (Paolisso 1989). In another study in the same year, he confirmed that the "acute insulin response" improves with by magnesium supplementation, and that insulin resistance lessened (Paolisso 1989). This is the so-called Phase I insulin response which is lost in Type 2 diabetes. Dr Mario Barbagallo of Milan explains:

A poor intracellular Mg concentration, as found in non-insulin-dependent diabetes mellitus (NIDDM) and in hypertensive patients, may result in a defective tyrosine-kinase activity [this is insulin's "second messenger" which passes on insulin's message within the cell] at the insulin receptor level, and exaggerated intracellular calcium concentration. Both events are responsible for the impairment in insulin action and a worsening of insulin resistance in non-insulin dependent diabetic and hypertensive patients. By contrast, in NIDDM patients daily Mg [magnesium] administration, restoring a more appropriate intracellular Mg concentration, contributes to improve insulin-mediated glucose uptake. … We further suggest that a reduced intracellular Mg concentration might be the missing link helping to explain the epidemiological association between NIDDM and hypertension (Barbagallo 2003)

Dr PJ Lefebvre found the same thing in Belgian diabetics:

The interrelationships between magnesium and carbohydrate metabolism have regained considerable interest over the last few years. Insulin secretion requires magnesium: magnesium deficiency results in impaired insulin secretion while magnesium replacement restores insulin secretion. Furthermore, experimental magnesium deficiency reduces the tissues' sensitivity to insulin. Subclinical magnesium deficiency is common in diabetes. It results from both insufficient magnesium intakes and increased magnesium losses, particularly in the urine. In Type 2, or non-insulin-dependent, diabetes mellitus, magnesium deficiency seems to be associated with insulin resistance. … Administration of magnesium salts to patients with Type 2 Diabetes tends to reduce insulin resistance (Lefebvre 1994).

Replenishing magnesium and the antioxidants does not cure insulin resistance, but then retrieving the baseball does not cure the broken window either; damage has been done. However, given the foregoing, magnesium is palliative of insulin resistance, which supports the notion that the relation between magnesium and insulin resistance is causative. This has interesting implications. For one thing, it is not the high glycemic index or load of foods that causes insulin resistance; it is that refined carbohydrates arrive without the micronutrients necessary for their metabolism. Thus, any preventive or therapeutic intervention based upon the glycemic index or the glycemic load of foods is doomed to failure unless unrefined foods are used.

The *pièce de résistance* is a study that demonstrates the inter-relatedness of oxidant stress, intracellular magnesium and insulin sensitivity: a four-week, double-blind controlled study of 24 hypertensives showed that 600 mg of vitamin E per day increased sensitivity to insulin by an average of 24%. This improvement in insulin sensitivity was significantly related to a 50% increase in the ratio of glutathione to oxidized glutathione, and to a 16% increase in intracellular magnesium; and the blood pressure of the group fell from 158/90 to 132/82 (Barbagallo 1999):

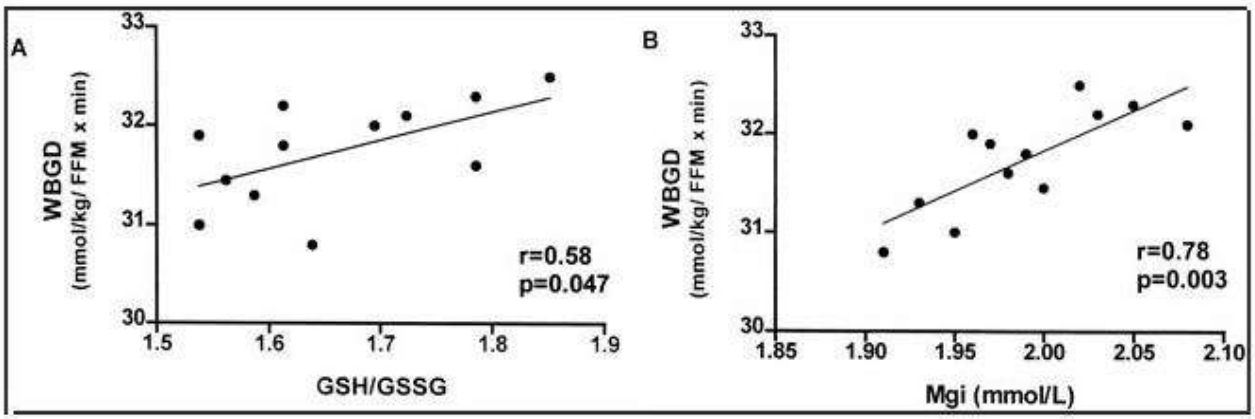


Figure 2. Relation of WGBD [Whole-Body Glucose Disposal, a measure of insulin resistance] to GSH/GSSG [glutathione / oxidized glutathione] ratio (A) and to total red blood cell intracellular magnesium (B) in vitamin E-treated patients

Supplemental vitamin E apparently lessened oxidant stress, which made available more glutathione so that cells were able to neutralize more noxious aldehyde intermediates. This eased the metabolic chaos caused by over-nutrition and restored ATP levels, so that the cells were able to pump out calcium and take in more magnesium; this caused an improvement in their sensitivity to insulin consonant with their increased ability to process the glucose it would cause to enter the cell. Taken altogether, these studies suggest that refined foods cause over-nutrition because they do not trigger the satiety clues put in place during evolution. Over-eating refined foods causes oxidant stress and insulin resistance because refined foods lack (at least) the magnesium and antioxidants necessary for proper metabolism; and this causes the Western degenerative diseases.

The remedial diets

Recall that an implication of Dr Resnick's ionic hypothesis is that insulin resistance should be lowered by a diet containing less salt and more potassium and magnesium. Interestingly, there is research in this important area. Dr Moore offers an anecdote about a colleague who normalized a 400 mg% blood sugar level by giving up soy sauce, and there is a 1984 study of Australian aborigines who went walkabout for seven weeks and "greatly improved or completely normalized" the major metabolic abnormalities of Type 2 Diabetes, namely insulin resistance and with it high triglycerides and high VLDL levels (O'Dea 1984). Their traditional, unrefined diet was low in fat (13%), with a high 64% of energy intake from animal foods, and supplied only 1200 calories; and although their traditional lifestyle involved more exercise than their urban existence, "There was no correlation between level of physical activity and improvement in any of the physiological parameters" in this investigation. Exercise, weight loss and low-fat diets are all known to increase sensitivity to insulin, but could there be a contribution from the fact that their traditional diet was unsalted and high in potassium and magnesium?

The Gerson Therapy for cancer treatment also involves draconian salt restriction with massive potassium intake from supplements and vegetable juices, and routinely has a positive effect on diabetes *without* exercise:

> In one particularly dramatic case, we saw a diabetic patient, aged 46, with sugar levels in his bloodstream of over 200, that were uncontrollable with insulin and drugs. This problem completely cleared in five weeks. At that time, he was able to take all the juices of the full Gerson Therapy (carrot juices contain large amounts of complex carbohydrates) without any further need of insulin. His blood sugar level returned to a normal 120, and remained static. (Gerson Healing Newsletter 3/1995; 10(2):1) (Gerson.org)

These anecdotal accounts are interesting and supportive, but prove nothing scientifically. However, the Pritikin Program has been scientifically investigated and the studies published. The Pritikin Program consists of exercise and a salt-restricted diet of whole grains (wheat, oats, rye, brown rice, barley, millet), starchy vegetables (potatoes, yams, winter squash), chestnuts, beans and peas; raw or cooked vegetables, and fruit. The diet is based on that of the Tarahumara Indians of the western Sierra Madre in Mexico, who are renowned for their physical endurance and very low rate of CHD; interestingly, the Pima Indians north of the border are their genetic twins and have developed the worst case of syndrome X in the Western world. In three weeks on this program, "insulin levels fell 46% in men." (Christian 2002). Among "652 diabetics, 70% on oral agents left Pritikin free of such medications; 39% on insulin left insulin-free; and participants who continued the Pritikin Program stayed off the medications" (Barnard 1994). In addition, it helps with all aspects of syndrome X: "the Pritikin Program controlled this syndrome in just three weeks in a majority of 72 people studied. Fasting insulin was reduced by 30-40%. … normalization of weight is not a requisite for a reduction or normalization of other risk factors … we have shown in short-term animal studies that hyperinsulinemia can be induced independent of obesity by placing rats on a high-sucrose diet … these data suggest that attention should be focused on the quality of the food consumed …" (Barnard 1992).

These high-quality studies were published in peer-reviewed medical journals, and they say, in essence, that unrefined diets rich in potassium and magnesium while low in sodium, with or without exercise, and with or without weight-loss, lessen insulin resistance and quickly repair syndrome X. Dr Lawrence Resnick's minerals account for some of these results, while restoring the natural balance between the essential fatty acids likely also contribute benefit. The Pritikin experiment showed that removing as much fat as possible from the diet is an effective intervention in those at immediate risk of heart attack, but that this is not a sustainable strategy. Long-time Pritikin devotees developed allergies and other symptoms of essential fatty acid deficiencies. Ann Louise Gittleman, who was Pritikin's head of nutrition, wrote *Beyond Pritikin* about the problems the program attendees experienced and the importance of providing sufficient essential fats in the diet.

Therefore, it seems that whole foods restore sensitivity to insulin and correct the signs of syndrome X. It is also apparent that the danger of sugar is not only its very high glycemic index but also *because it is entirely denuded of minerals.* In a fascinating series of experiments with rats, Dr Jim Barnard of UCLA has shown that a high fat, high sugar diet (modeled on the American diet), causes insulin resistance and the rat equivalent of syndrome X (Barnard 1995), and degrades the function of the all-important arterial epithelium (Roberts 2004). A low fat, high starch diet like the Kitavan diet had no such bad effects, and the rats retained their health. He has replicated in rats the American dietary experiment, and compared it with an unrefined diet like that of the Kitava Islanders, and his results entirely support the thesis that refined foods cause our degenerative diseases. Many others have chronicled sugar's damaging effects – see Sugar ...

Reversing Insulin Resistance

Two ways insulin sensitivity can be increased are exercise and weight loss. Each improves insulin sensitivity by up to 25%, and the cause of the remaining 50% of insulin resistance is undetermined (An Interview with Gerald Reaven). Exercise uses up calories, and weight loss occurs if the diet supplies fewer calories than are used in the business of life: *insulin sensitivity improves when the cells actually need the calories available to them in the bloodstream.* Interestingly, intracellular calcium falls during weight loss (Jacobs 1993), which provides a plausible mechanism for the insulin-sensitizing effects of weight loss. A recent review, *Insulin Resistance and Atherosclerosis*, pointed out that "Caloric restriction, which improves features of insulin resistance, increases mitochondrial biogenesis [more mitochondria means a higher capacity for "burning" glucose] and, surprisingly, enhances the efficiency of ATP production (95, S9) … insulin resistance and atherosclerosis could represent independent and ultimately maladaptive responses to the disruption of cellular homeostasis caused by *excess delivery of fuel*" (Semenkovich 2006).

However, it is hardly news that caloric excess causes insulin resistance; Dr Rabinowitch of Montreal wrote as early as 1930 that a high-carbohydrate, low-*calorie* diet increased the sensitivity to insulin in man, and that *under-nutrition* was the key to health for diabetics (Rabinowitch 1930). In 50 patients, the average insulin dose dropped from 25 to 11 units (57%) after five years on his high carbohydrate, low-calorie diet: in other words, these people were getting better! (Rabinowitch IM, Ca Med Assn J, 8/1935:136-44).

This principle was the foundation of diabetes treatment before the advent of insulin. First, there was a fast until blood sugars were brought down into the normal range. Then, carbohydrates (as low-carbohydrate green vegetables) were increased until sugar once again appeared in the urine. This established the amount of carbohydrate that kept the urine sugar-free, and this amount formed the basis of the diet: 15% of protein plus the balance of calories as fat completed the diet. In other words, no more carbohydrates were given than the diabetic's damaged system could handle, and patients left the hospital with diet plans employing, on average, 70% calories from fat, 10% from carbohydrate and 20% from protein, some with the recommendation of a day's fast per week. Dr Elliot Joslin was a proponent of such diets, and they prevailed until insulin became available in 1922 (Westman 2006).

This doesn't happen on the American Diabetes Association regime, which mentions weight loss only in passing (ADA FAQ) and fails to point out that if you're not losing weight on their low-fat diet (which does not prohibit sugar), it *will* worsen insulin resistance and raise the risk of heart disease and diabetic complications. Type 2 diabetics who follow the ADA dietary recommendations deteriorate, routinely developing β-cell failure and requiring insulin with the passage of time. The Belfast Diet Study followed 432 Type 2 diabetics from diagnosis for 10 years: "Secondary failure of plasma glucose control following initial successful response to diet therapy may be due to … progression of the intrinsic diabetic condition … the ongoing fall in beta-cell function assessed by HOMA modeling closely mirrored the progressive rise in fast blood glucose" (Levy 1998). Diabetics die from heart disease and the complications of diabetes as a direct consequence of their elevated HbA_{1c} levels, which simply cannot be controlled on a high-carbohydrate, high calorie diet. Could this be because diabetics develop "cognitive dysfunction" from the poor blood sugar control inevitable on high carbohydrate diets *unless calories are restricted*? Research published in the ADA journal *Diabetes Care* confirms a high level of depression, and both "functional" and "cognitive" dysfunction in older subjects who had had diabetes for about 14 years (Munshi 2006).

Giving sugars in isolation, as in, for example, drinking a can of soda, probably causes incremental damage to the cell machinery because magnesium levels fall abruptly just as the cells' requirements peak. Interestingly, rats excrete magnesium when given sugars just like humans (Roy 1981), but supplemental dietary magnesium prevented fructose-induced insulin resistance in rats (Balon 1994). Enriching sugared foods with magnesium might lessen their deleterious effects, but as one commentator put it, "only a modest or inconsistent response has been reported ..." to such measures and efforts may be more effective when "the focus is on the overall nutritional profile rather than the single nutrient intake" (Bo 2006). Dr Simona Bo was commenting on the hopelessly inconsistent results of nutritional interventions on hypertension (Townsend 2005).

Diabetics are at greater risk of magnesium depletion because hyperglycemia causes urinary losses, which confounds most studies of magnesium supplementation in diabetics. However, no study has found any harm from magnesium supplementation, and most demonstrate improvements; one review of studies found benefits in insulin sensitivity, blood pressure, HDL-cholesterol and triglycerides, and/or HbA_{1c} (Guerrero-Romero 2005). But another review gave the HbA_{1c} levels of subjects given magnesium supplements in four trials: they ranged from 7.3% to 8.7% (O'Connell 2001). These HbA_{1c} levels correspond to average blood sugars of 159 mg/dl and 201mg% over the preceding three months, a normal blood sugar being 100mg%. Their hyperglycemic state guarantees that after-meal blood sugars will exceed the kidney threshold of 175mg%, at which point glucose appears in the urine and magnesium is lost. Magnesium supplementation is then like pouring water into a bucket with a hole in it. That this is important is shown by a study which showed that improved blood sugar control in insulin-dependent diabetics resulted in higher intracellular magnesium levels, and the concomitant benefits of 4% more HDL-cholesterol and a 10% decrease in triglycerides show that their insulin resistance had lessened (Djurhuus 1999).

That magnesium supplementation improves insulin sensitivity is true in my experience. Hyperglycemia causes insulin resistance in diabetics so reliably that insulin-dependent diabetics are taught a rule of thumb: if the blood sugar is over 200mg%, then inject 150% of the insulin needed when the blood sugar is near the target level of 100mg%. However, a study of magnesium supplementation in Type II diabetics helped by "restoring a more appropriate intracellular Mg concentration, [which] contributes to improve insulin-mediated glucose uptake" (Barbagallo 2003). And indeed, when I took a magnesium supplement, I found that I no longer needed extra insulin when my blood sugar escaped the usual range. With plentiful magnesium, one unit of insulin lowers my blood sugar 20mg%, no matter what my blood sugar level. Magnesium abolishes the insulin resistance of hyperglycemia! You'd think the American Diabetes Association would be all over this, but, inexplicably, they list magnesium as an "unproven remedy" which should only be given in clinical trials (ADA).

So what lowers inflammation and increases sensitivity to insulin?

In various studies, CRP was lowered by fruits and vegetables (Esmaillzadeh 2006), dietary fiber, both soluble and insoluble (Ma 2006), a low dietary glycemic index (Liu 2002), a low dietary glycemic load (Pittas 2006), and a lowered caloric intake (Kasim-Karakas 2006). In short, unrefined foods and caloric restriction lower inflammation.

Fish oils are protective

An essential fatty acid which is in short supply in the Western diet is docosahexanoic acid (DHA). DHA is an omega-3 fatty acid found in fish oils, or made in-house from shorter omega-3 fatty acids such as the α-linolenic acid found in walnuts, flax seed oil and other foods. It can exist in many different shapes, called "molecular conformations." Apparently, rapid changes between conformations facilitates chemical reactions in the cell wall: "The … simulations present an image of DHA thrashing about … in the core of a … membrane … which may possibly explain why high levels of DHA are associated with increased speed in many processes occurring across the membrane" (Turner 2003). In this case, more DHA in the cell membrane means the sodium pump can work better. The average Western intake of DHA is about one-tenth the desirable intake (Kris-Etherton 2002), which compromises the performance of our sodium pumps and contributes to our ills.

Another function of DHA involves the making of prostaglandins, which are short-lived hormones for things like inhibiting blood clotting and instructing the arteries to relax and lower blood pressure. When DHA is in short supply, the same enzymes, which would make these beneficial prostaglandins, act instead on arachidonic acid (AA), an omega-6 fatty acid that is as plentiful in the Western diet as DHA is scarce:

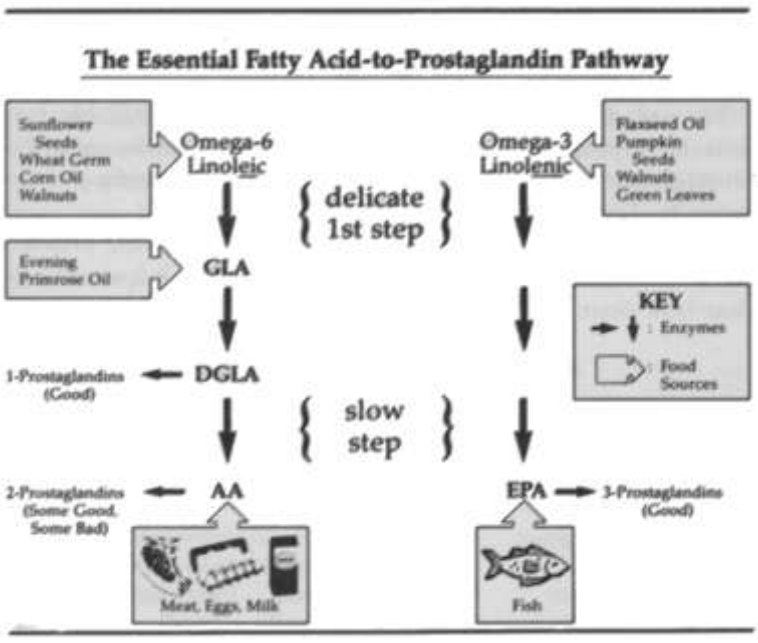
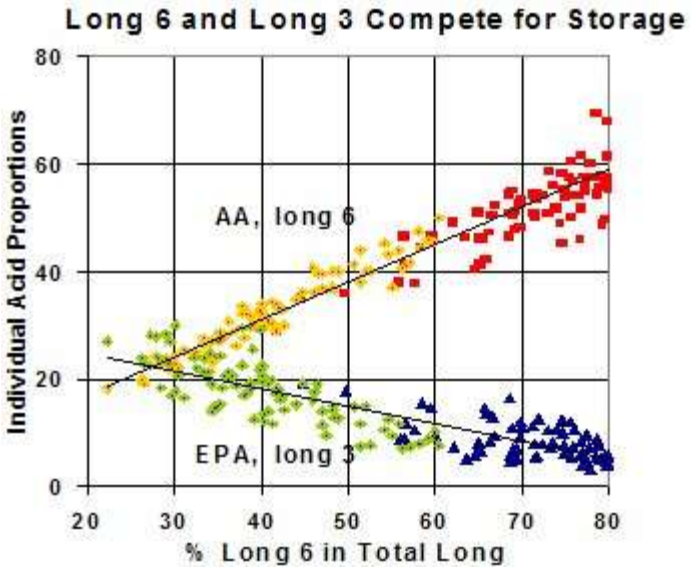
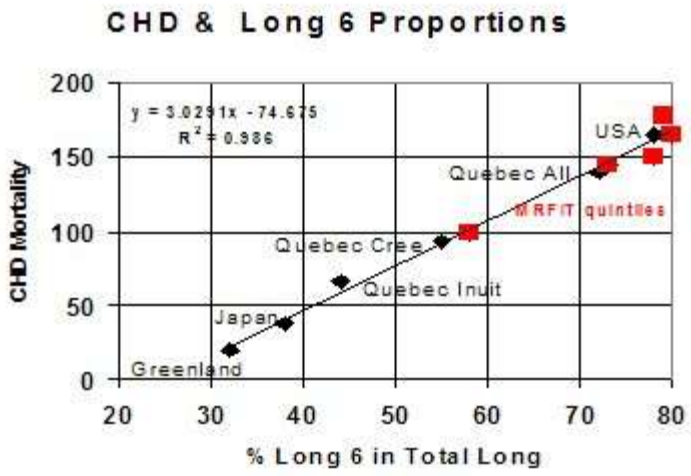


Figure 1 The Essential Fatty Acid-to-Prostaglandin Pathway. The metabolic pathways of the essential fatty acids are well known, and since we can neither manufacture them nor convert one into the other, both are vital for good health. Only tiny fractions of the EFAs we consume are made into prostaglandins, the messenger molecules that control local conditions all over the body, but without them there would be biological chaos. The PGls and PG3s are 'good' because they promote healing, lower blood pressure and inhibit the PG2s. The PG2s are 'bad' because their "prepare for fight or flight" messages (Raise the blood pressure! Get ready to clot the blood!) are usually false alarms caused by the deficiencies of our Western diet. (From *Food for Vitality*, Bantam, UK 1992)

Unfortunately, the prostaglandins made from AA (which come from omega-6 fatty acids predominant in refined vegetable oils) tend to clot the blood and constrict the arteries, and have been implicated in all phases of the development of atherosclerosis. In a fascinating study, these omega-6 and omega-3 fatty acids were found in the cell membranes according to their availability in the diet (National Institutes of Health's Office of Dietary Supplements):



The green and yellow points are from Japanese people, while the blue and red points are from Americans. EPA is an omega-3 fatty acid (a close relative of DHA), and it is clear that the more EPA the cell membrane contains, the less AA is present. The Japanese ate far more omega-3s than the Americans did, so the prostaglandins made by the Japanese are far more anti-heart disease that those made by the Americans. In other words, what we eat in terms of essential fatty acids directly and immediately affects our risk of heart disease:



This graph (from the same source) shows that the more of the omega-6 family were contained in the cell membranes, the greater the risk of dying from coronary heart disease. The relationship is very strong, and the figure indicates that Japanese have on average 32% AA versus 78% for Americans. Perhaps in consequence, the Japanese rate of heart death is about 40 per 100,000 while the American rate is near 160 per 100,000 people.

Returning to the effect of DHA on the sodium pump, it has been shown that fish oil prevents insulin resistance induced by high-fat feeding in rats (Storlien 1987), perhaps because the rate of sodium-pump activity is increased by greater membrane fluidity from its greater DHA content. A study in humans found that insulin sensitivity was associated with the concentration of polyunsaturated fatty acids in muscle cell membranes (Borkman 1993), and a review found that "a high n-6/n-3 ratio appears deleterious" to insulin sensitivity in man (Storlien 1996).

Importantly, "Membranes remain relatively constant in their saturated (SFA) and monounsaturated (MUFA) fatty acid levels over a wide range of dietary variation for these fatty acids. Membrane composition was found to be more responsive to n-6 and n-3 polyunsaturated fatty acid (PUFA) levels in the diet and most sensitive to n-3 PUFA and to the n-3/n-6 ratio" (Hulbert 2005). And insulin sensitivity was found to be related to the fatty-acid composition of serum lipids and skeletal muscle phospholipids (especially their content of DHA) in 70-year-old men, which is to say that what one eats directly and almost immediately affects heart risk (Vessby 1994). The mechanism for this rapid effect is not controversial: fish oils lower the tendency to arrhythmias and coronary spasm. This is likely because they improve sodium-pump performance, which raises protective magnesium and lowers excitatory calcium within the cells of the coronary arteries and the heart's pacemaker circuitry.

These studies suggest that a good ratio of omega-3 to omega-6 fatty acids in the diet, along with a good ratio of these essential fatty acids to saturated fat, is essential for insulin sensitivity. These ratios are bad to very bad in the Western diet, and are even worse in anyone who follows the Therapeutic Lifestyle Changes Diet recommendation to replace saturated fat with refined vegetable oil, which contains a high percentage of omega-6s and no omega-3s:

The information presented herein shows clearly that with respect to n23 fatty acids. EPA and DHA intakes are significantly below amounts that have been recommended by different countries and agencies, as well as by nutritionists in the United States. At the present time, increasing fish consumption by 4-fold is one strategy that will facilitate meeting the recommendations that have been made for intake of EPA and DHA (Kris-Etherton 2000).

All the remedial diets reviewed above return towards hunter-gatherer ratios (Simopoulos 1999):

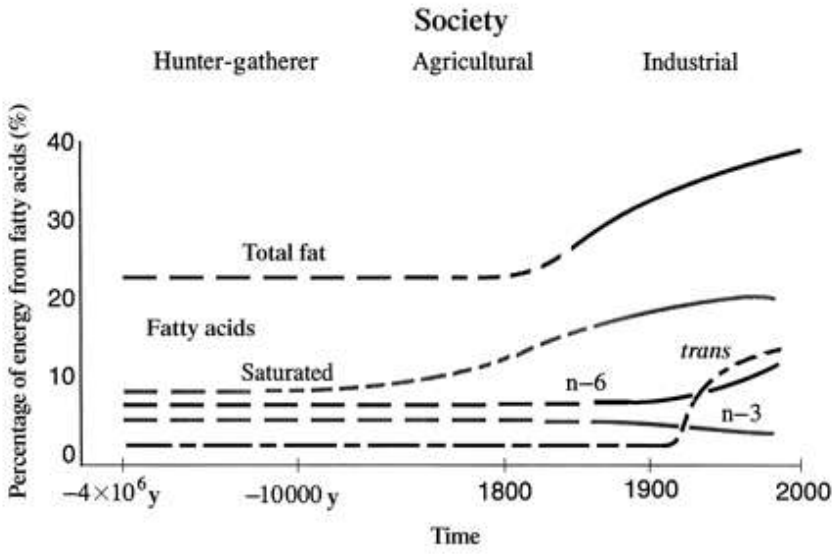


FIGURE 1. Hypothetical scheme of the relative percentages of fat and different fatty acid families in human nutrition as extrapolated from cross-sectional analyses of contemporary hunter-gatherer populations and from longitudinal observations and their putative changes during the preceding 100 y. *trans*-fatty acids, the result of the hydrogenation process, have increased dramatically in the food supply during this century.

Fish oils improve matters by preventing the fructose-caused rise in triglycerides (TAG in the figure), although LDL-cholesterol still goes up (Roche 2000):

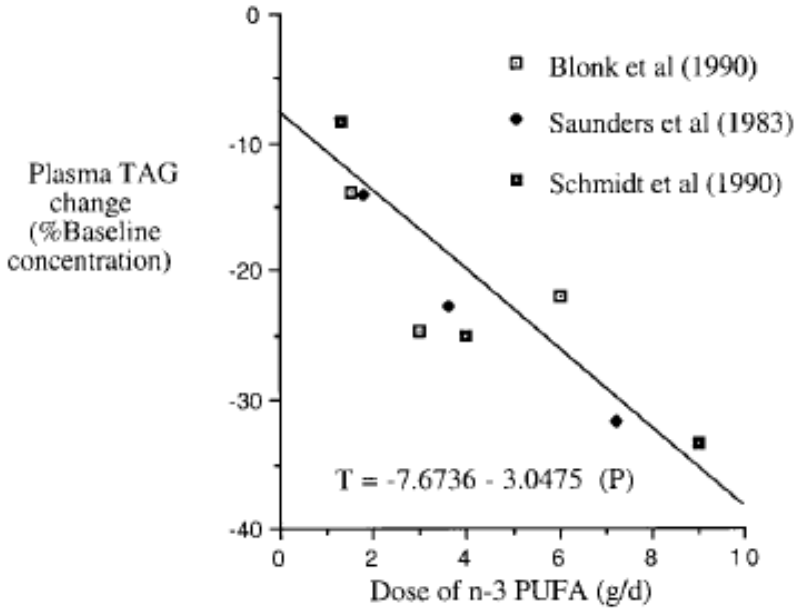


FIGURE 3. The dose-dependent hypotriacylglycerolemic effect of n−3

This effect is sufficiently robust that triglycerides fell even when the fish oil was given with omega-6 fatty acids from vegetable oil: "After the 6-wk period of fish oil supplementation, fasting and postprandial plasma triacylglycerol [triglyceride] concentrations decreased significantly" (Brady 2004). In addition, "Fish oils, but not atorvastatin [the statin drug Lipitor], influence [improve] HDL metabolism chiefly by decreasing both the catabolism and production of HDL … in insulin-resistant obese men. Addition of atorvastatin to treatment with fish oils had no additional effect on HDL kinetics compared with fish oils alone" (Chan 2006). The study shows that fish oils raise HDL (good) cholesterol better than the statin drug Lipitor. This is especially true for obese and insulin-resistant men.

Caloric restriction

Caloric restriction extends the lives of organisms from bacteria to humans by lowering insulin and insulin resistance, and by lowering inflammation. Interestingly, caloric restriction lessens oxidative stress by a counter-intuitive mechanism. Less food causes the expression of more mitochondria, which burn the available fuel more efficiently, thereby generating fewer free radicals. This interesting adaptation provides the same amount of energy from a reduced intake of food (Civitarese 2007).

Caloric restriction proved to be astonishingly effective in reducing the risk factors for heart disease in 18 subjects who limited calories for between three and 15 years (Fontana 2004):

Table 2. Risk factors for atherosclerosis

Parameter	Value		
	CR ($n = 18$)	Controls ($n = 18$)	P value
Tchol, mg/dl	158 ± 39	205 ± 40	0.001
LDL-C, mg/dl	86 ± 28	127 ± 35	0.0001
HDL-C, mg/dl	63 ± 19	48 ± 11	0.006
Tchol/HDL-C ratio	2.6 ± 0.5	4.5 ± 1.3	0.0001
TG, mg/dl	48 ± 15	147 ± 89	0.0001
TG/HDL-C ratio	0.8 ± 0.3	3.5 ± 2.8	0.0001
Systolic BP, mmHg	99 ± 10	129 ± 13	0.0001
Diastolic BP, mmHg	61 ± 6	79 ± 7	0.0001
Fasting glucose, mg/dl	81 ± 7	95 ± 8	0.0001
Fasting insulin, mIU/ml	1.4 ± 0.8	5.1 ± 2	0.0001
Hs-CRP, μg/ml	0.3 ± 0.2	1.6 ± 2.2	0.001

Values are means ± SD. IU, international unit; Hs-CRP, high-sensitivity CRP; 1 mmHg = 133 Pa.

Note particularly the last lines of the Table: caloric restriction produced an average blood pressure of 99/61, a fasting blood sugar of 81 despite an extremely low fasting insulin level of 1.4 which denotes very low insulin resistance, and a very low CRP of 0.3! They have risk factors which are so low that they are simply off the radar. Recall that the Kitava Islanders remain lean in spite of an abundance of (whole-food) calories, and, in fact, preserve their insulin sensitivity and actually lose weight with age. The Kitavan Islanders unknowingly practice caloric restriction, but without suffering the privations of hunger and discontent I imagine must accompany such a practice.

The "secret" to painless caloric restriction apparently lies in whole foods, which are more satiating than refined foods and engender less oxidative stress because of their full complement of nutrients, including magnesium. Most diet studies are at pains to maintain weight on the principle that there should be but one variable so that, for example, the effect of low-fat and low-carbohydrate diets can be directly compared. However, when a group of researchers compared a weight-maintaining low-fat diet with an unlimited low-fat diet, they found to their surprise that the unlimited-intake group ate less food, lost weight and experienced less inflammation (Kasim-Karakas 2006). A similar result was obtained using a low-carbohydrate diet: "The mechanism for

the rapid weight loss with the low-carbohydrate diet is a suppressed appetite, 1st through the high protein content of the diet, 2nd through the ketogenic nature of the diet with satiety signals for fat being active and 3rd through the absence of hunger-promoting carbohydrate components like sucrose and/or fructose" (Erlanson-Albertsson 2005).

The folly of the American Diabetes Association's dietary recommendations

An eminent diabetologist, Dr Ralph DeFronzo, described the deterioration into diabetes in the Lilley Lecture of 1987, the pre-eminent forum of accumulated medical wisdom and insight:

> … there are two primary defects responsible for the pathogenesis of non-insulin-dependent diabetes mellitus (NIDDM). In some NIDDM patients the primary defect starts at the level of the β-cell and manifests itself as an impairment in insulin secretion; these individuals are represented by the lean diabetic patient. In other NIDDM patients the primary defect starts as an impairment in tissue (muscle and liver) sensitivity to insulin; these individuals are represented by the obese diabetic. However, whichever defect – diminished insulin secretion or insulin resistance – initiates the development of NIDDM, it will subsequently lead to the emergence of the second abnormality (DeFronzo 1987).

Dr DeFronzo's first category includes insulin-dependent diabetics who have no β-cells, and are generally lean and sensitive to insulin, at least initially. But what he describes is startling: whether there is diminished insulin secretion or insulin resistance, the second abnormality will emerge. The insulin-deficient become insulin-resistant and the insulin-resistant become insulin-deficient! Then, in both cases, high blood sugar becomes an agent of further destruction and it's startling to learn that high blood sugar is almost universal in the diabetic population receiving medical care:

… there appears to be abundant evidence from both animal and human studies that sustained chronic hyper-glycemia can lead to the development of defects in both insulin secretion and insulin action. Furthermore, it appears that these deleterious effects of hyperglycemia are mediated by downregulation of the glucose transport system. Recognition of the important pathogenetic role of hyperglycemia *per se* in the evolution of NIDDM has important therapeutic implications. Over the last decade, we have performed oral glucose (100-g) tolerance tests (OGTT) in many normal-weight NIDDM subjects. All subjects had fasting hyperglycemia (mean ± SE 148 ± 8). After glucose ingestion, the plasma glucose concentrations were >200 mg/dl at all time points … (DeFronzo 1987).

Even more extraordinary is that this widely acknowledged and accepted:

The secondary component of insulin resistance in NIDDM is most likely a result of hyperglycemia, which causes a decrease in insulin receptor kinase function, further exacerbating the insulin-resistant state. This defect is completely reversible by any therapeutic intervention that ameliorates the hyperglycemia (Olefsky 1885)

Yet, inexplicably, only completely out-of-control diabetics are treated so as to achieve normal blood sugars:

Thus, in the poorly controlled diabetic with fasting plasma glucose levels >200 mg/dl it seems reasonable to institute a short course of intensive insulin therapy (4-6 wk) or severe caloric restriction (10-14 days under close supervision by a physician) to restore normoglycemia and reverse the deleterious effects of hyperglycemia on cell function (DeFronzo 1987).

The treatment suggested to all diabetics is the low-fat American Diabetes Association diet, yet this diet causes the hyperglycemia which eventually makes all diabetics both insulin-dependent and insulin resistant! There is no doubt that this is still the case today, in spite of treatment with insulin-stimulating sulfonylurea drugs and the insulin-sensitizing drug metformin. The findings of the UK Prospective Diabetes Study included that:

The study showed an initial increase in β-cell function (from 46 to 78%) at 1 year in subjects on a sulfonylurea, followed by a steady decline in function to 52% at 6 years. Subjects on diet only (n = 486) exhibited a gradual decline in β-cell function of ~ 4% per year. Insulin sensitivity only changed in subjects on metformin (n ~ 159), increasing from 51 to 62% at 1 year and remaining at 62% at 6 years (Wallace 2004).

In other words, the American Diabetes Association diet alone destroys β-cells at the rate of about 4% per year, and the diet plus a sulfonylurea drug improves β-cell performance initially but then destroys them at a rate near 5% per year. At these rates, diet alone will destroy all β-cells in 11 years, while diet plus a sulfonylurea drug will destroy them in 16 years.

Furthermore, physicians are slow to treat high HbA_{1c} levels in the way that Dr DeFronzo suggested way back in 1987. At the 2006 Scientific Sessions of the American Diabetes Association, we learned that:

> The majority of patients with Type 2 diabetes have very high HbA1c levels, and it takes physicians months before they intensify oral antidiabetic therapy … a study of 9,416 patients … [showed that] Mean HbA1c was 8.4% at baseline when therapy was initiated. Only 33% had levels at or below the ADA goal of 7%, and 67% had A1c levels approaching 10% (Kerr 2006).

That Dr DeFronzo's prescription of caloric restriction is effective was demonstrated in a study of obese, hyperglycemic diabetics. "Severe" caloric restriction quickly lowered their fasting blood sugar from 326mg% (!) to 150mg%, and their β-cells showed significant recovery (Stanik 1980). However, a similar study showed that subjects with diabetes of less than two years duration responded far better than subjects who'd had diabetes for more than five years: caloric restriction brought the less-than-two-year diabetics down to 119mg%, but the more-than-five-year group averaged 175mg% (Nagulesparan 1981), for the reason that chronic hyperglycemia had killed more β-cells in the diabetics of long standing.

β-cells are not the only cell type to be damaged by high blood sugar, and the Diabetes Control and Complications Study showed that this damage causes the diabetic complications of blindness, cataracts, heart attack, stroke and the amputation of limbs. An exacting study has described the relationship between high blood sugar and the risk of various complications (Stratton 2000):

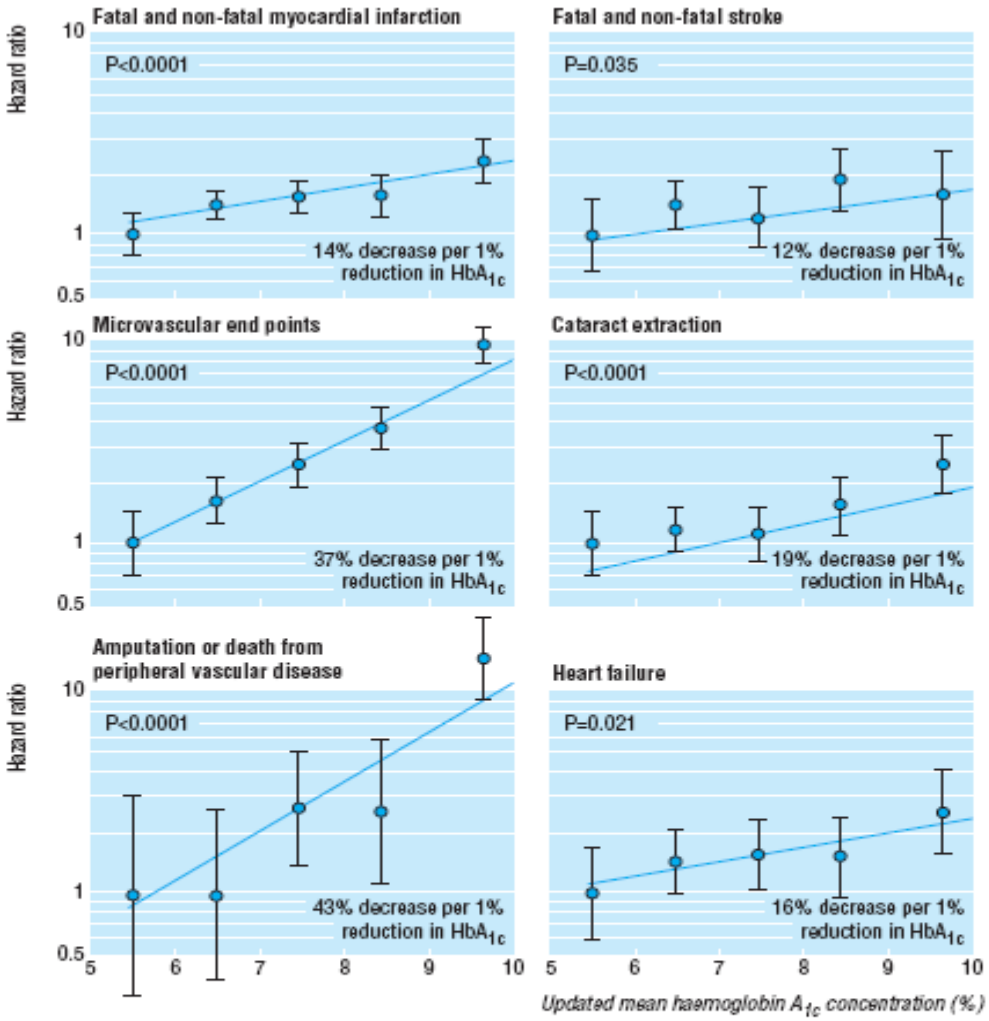


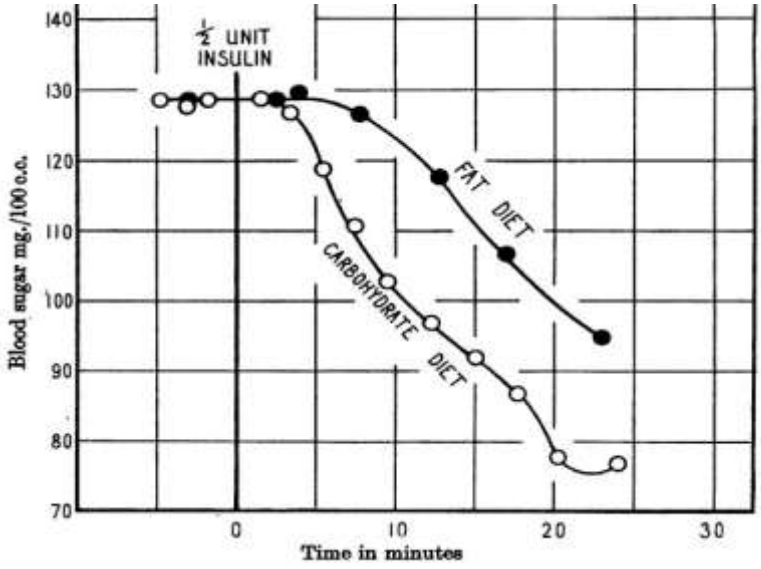
Fig 4 Hazard ratios, with 95% confidence intervals as floating absolute risks, as estimate of association between category of updated mean haemoglobin A_{1c} concentration and myocardial infarction, stroke, microvascular end points, cataract extraction, lower extremity amputation or fatal peripheral vascular disease, and heart failure. Reference category (hazard ratio 1.0) is haemoglobin A_{1c} <6% with log linear scales. P value reflects contribution of glycaemia to multivariate model. Data adjusted for age at diagnosis of diabetes, sex, ethnic group, smoking, presence of albuminuria, systolic blood pressure, high and low density lipoprotein cholesterol, and triglycerides

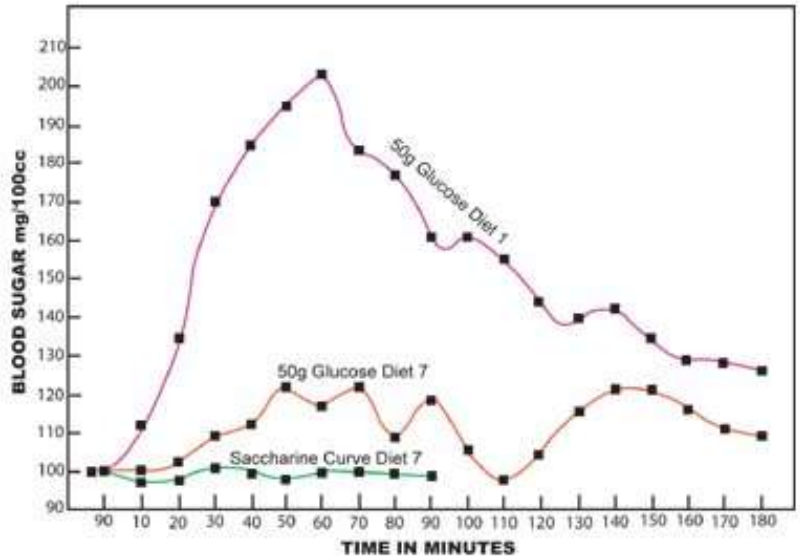
The graph at bottom left shows that risk of amputation or death from peripheral vascular disease doubles between an HbA_{1c} of 5.6% (like mine) and the low end of "tight control" at 7%. But recall that "67% [of Type 2 diabetics] had HbA_{1c} levels approaching 10%" (Kerr 2006), and we see that the risk for these people is more than 10 times higher. But anyone who's prepared to avoid sugars, starches and starchy vegetables can maintain an HbA1c of 5.6% like mine.

So why does the ADA advocate a low-fat diet?

It is a mystery.

Ancient studies showed insulin sensitivity increases with increase of carbohydrate in the diet, and more recent studies have shown that cells remodel themselves to burn the proportion of carbohydrate and fat in the diet. Thus, insulin sensitivity will inevitably be "worse" in cells adapted to burning fat. Dr Harold Himsworth noticed in 1935 that giving carbohydrates increased insulin sensitivity in rabbits, which is to say that more carbohydrates can be tolerated on a high-carbohydrate diet, and confirmed the effect in humans:





Left: Two insulin depression curves from the same rabbit, one (●) obtained on the fat diet, the other (○) obtained on the carbohydrate diet (Himsworth 1935).

Right: Diet 1 is high in fat and low in carbohydrates. It produces pseudo-carbohydrate intolerance with administration of 50g of glucose. Diet 7 is high in carbohydrate and low in fat. Now, this same patient does not show carbohydrate intolerance. These studies demonstrated the importance of providing substantial amounts of carbohydrate in the diet before testing for glucose intolerance (Himsworth HH. Clinical Science 2:67-94, Sept 1935).

Dr Himsworth didn't look at the effects of different types of carbohydrates, but Dr Aharon Cohen showed that replacing sugar with bread made a considerable improvement in the glucose tolerance test curves of 16 healthy individuals (Cohen 1966):

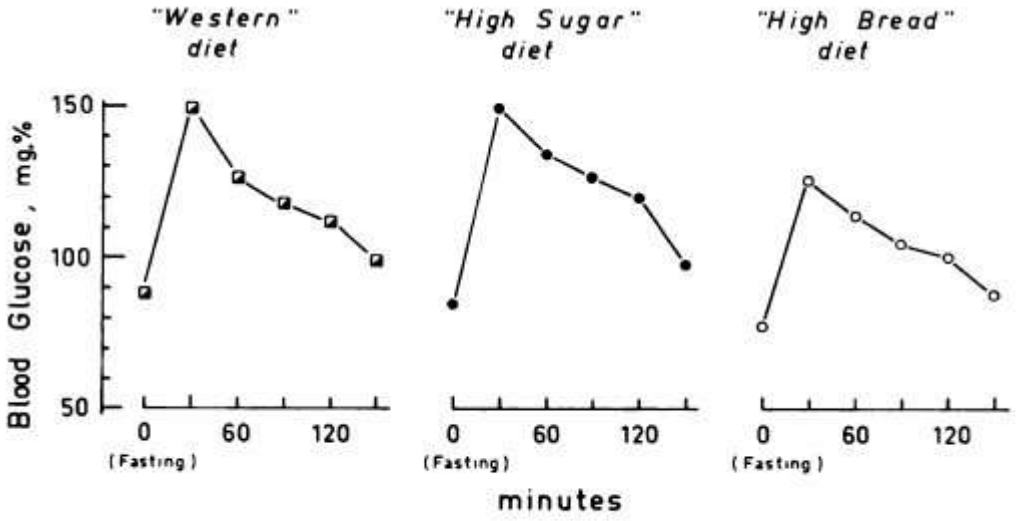
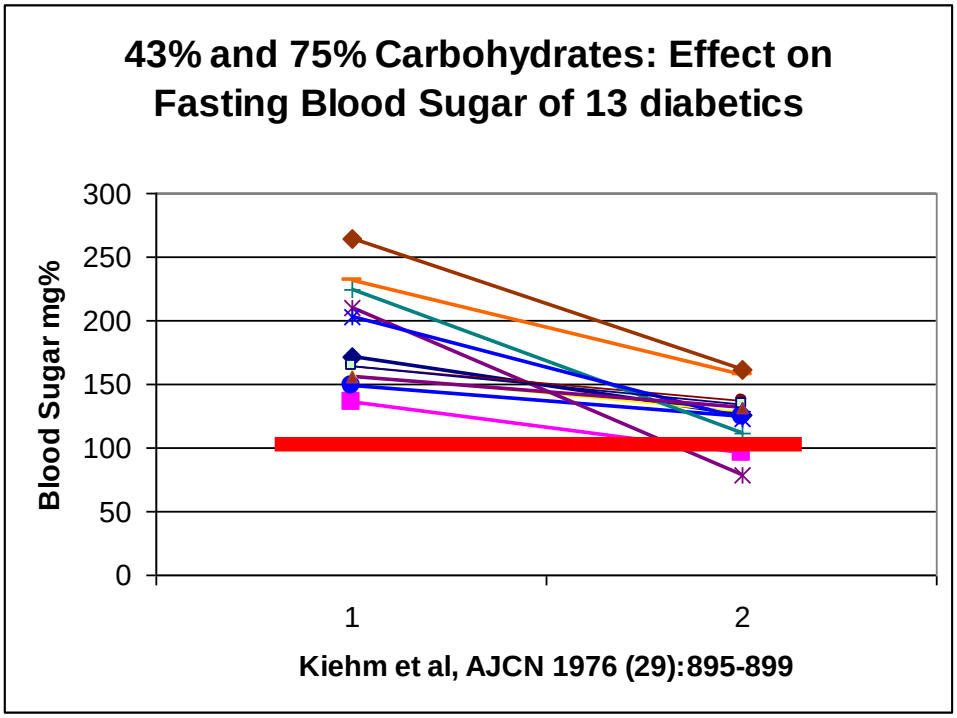


FIG. 3. Average glucose tolerance curves of all subjects at the end of "western," "sucrose" and "bread" diet periods.

The "Western diet" was commonly eaten in Israel; the "High Sugar diet" was the Western diet with all carbohydrate taken as sugar, and the "High Bread diet" was the traditional diet of the Yemen, a high-bread diet containing no sugar. The peak blood-glucose concentration and the "area under the curve", which is a measure of the health of the carbohydrate metabolism, are both considerably lower or the "High Bread diet." This study suggests that sugar erodes glucose tolerance.

Generations of researchers have misinterpreted this to mean that high-carbohydrate diets will restore insulin sensitivity to diabetics and that fat should be avoided; but this simply is not true for diabetics: the UKPDS has shown that the consequent elevated blood sugars cause deterioration of diabetes because of progressive β-cell loss. The ADA raised the percentage of carbohydrates from 45% to 70% in response to studies showing better glycemic control in diabetics on higher carbohydrate diets, which was probably due in part to the Himsworth effect (Kiehm 1976):



Only two of the 13 subjects achieved a normal β-cell-preserving blood sugar level (below the red bar), and one of these success stories was among the two who could not tolerate the diet; two others had high triglycerides. Three insulin-dependent diabetics did not respond well to the diet, and the researchers concluded "… improvement of glucose metabolism on high-carbohydrate diets may require the availability of endogenous insulin." However, this did not stop the ADA generalizing the results to insulin-dependent diabetics. All but two of the subjects lost weight during the study, and even though weight loss is well known to improve glucose tolerance, benefits were ascribed entirely to the increased carbohydrate. It is flimsy evidence at best, and an extraordinary failure of imagination to investigate only the addition of carbohydrates, rather than also testing the effect of their removal!

Dr Peter Bisschop set out to compare the effects of diets with fat contents of 0%, 44% and 85% on peripheral and liver sensitivity to insulin. He found that "high fat, low carbohydrate diets do not unequivocally affect the action of insulin on glucose disposal and tend to enhance the action of insulin on non-oxidative glucose disposal [meaning more glucose is made into glucagon]. … Remarkably, in the context of diabetes risk, two aspects of glucose homeostasis actually improved … decreased basal endogenous glucose production and improved insulin-stimulated non-oxidative glucose disposal" (Bisschop 2001). This effect is actually seen in low-carbohydrate diet studies, so long as insulin sensitivity is not assessed by glucose tolerance test. For example, a six-month trial of a low-carbohydrate compared to a high-carbohydrate diet in 132 obese subjects found "Insulin sensitivity, measured only in subjects without diabetes, also improved more among subjects on the low-carbohydrate diet (6% vs. 3%, P=0.01) (Samaha 2003).

The hoary old unthinking fear that fats promote heart disease must play a part. This fear, implicit and unquestioned in the preamble of every low-fat diet study, is, in fact, irrelevant. Diabetic *complications* rob the diabetic of vitality, the enjoyment of life, and eventually of life itself; it is the "macrovascular complication" of heart disease which kills most diabetics. Hyperglycemia accelerates the development of macrovascular complications, so prescribing a diet which guarantees hyperglycemia in the interests of avoiding heart disease is a protracted oxymoron – heart disease and stroke account for about 65% of deaths in people with diabetes, and adults with diabetes have heart disease death rates about 2 to 4 times higher than adults without diabetes. This deadly error *causes* the complications of diabetes, which are, then, iatrogenic diseases, caused by the American Diabetes Association "Medical Nutrition Therapy" prescribed to treat it. The lesson is crystal clear and unequivocal: avoid carbohydrates, avoid hyperglycemia.

Numerous studies in the medical literature agree that treating insulin-resistant conditions with a low-fat, high-carbohydrate diet worsens features of metabolic syndrome (Gerhard 2004); and the Insulin Resistance Atherosclerosis study established that 92% of Type 2 diabetics are insulin resistant (Haffner 1999). The prestigious American Association of Clinical Endocrinologists (AACE) says: "Of greatest importance is the avoidance of low fat-high carbohydrate diets *unless weight loss is also occurring*. The more insulin resistant an individual is, the more insulin they must secrete in order to maintain normal glucose homeostasis, as a consequence, in the absence of weight loss, manifestations of the Insulin Resistance Syndrome will be accentuated when insulin resistant persons increase the amount of carbohydrates in their diet (50)" (AACE on IRS).

Even insulin-sensitive people don't do that well on such diets. Dr Gerald Reaven fed 10 healthy post-menopausal women either 60% or 40% carbohydrate diets. The higher carbohydrate level worsened insulin resistance and elevated unhealthy blood fats: "Because these changes would increase risk of ischemic heart disease in postmenopausal women, it seems reasonable to question the wisdom of recommending that postmenopausal women consume low-fat, high-carbohydrate diets" (Jeppesen 1997). And these were healthy women.

When Dr Reaven made a similar experiment in Type 2 diabetics, he found the same adverse changes. He compared the 20% fat, 60% carbohydrate ADA-pattern diet which included 10% of calories as sugar, as the ADA diet allows, with a diet containing 40% fat and 40% carbohydrate, and found that:

> Although plasma fasting glucose and insulin concentrations were similar with both diets, incremental glucose and insulin responses from 8 a.m. to 4 p.m. were higher ($p<0.01$), and mean … 24-hour urine glucose excretion was significantly greater (55 … versus 26 … g/24 hours $p<0.02$) in response to the low-fat, high-carbohydrate diet. In addition, fasting and postprandial triglyceride levels were increased ($p < 0.001$ and $p < 0.05$, respectively) and high-density lipoprotein (HDL) cholesterol concentrations were reduced ($p<0.02$) when patients with NIDDM ate the low-fat, high-carbohydrate diet … These results document that low-fat, high-carbohydrate diets, containing moderate amounts of sucrose, similar in composition to the recommendations of the ADA, have deleterious metabolic effects when consumed by patients with NIDDM for 15 days. Until it can be shown that these untoward effects are evanescent, and that long-term ingestion of similar diets will result in beneficial metabolic changes, it seems prudent to avoid the use of low-fat, high-carbohydrate diets containing moderate amounts of sucrose in patients with NIDDM (Coulston 1987).

In other words, they did better on the lower carbohydrate diet and got worse on the ADA-pattern diet; but both diets provoked "glycosurea", the spilling of sugar in the urine, which means that both diets elevated blood sugars far beyond the normal range into the territory where kidney deterioration and progression of the other diabetic complications is inevitable.

Similarly, Dr Arbhimanyu Garg performed a similar study in 42 Type 2 diabetics and found that, "In NIDDM patients, high-carbohydrate [55% carbohydrate, 30% fat] diets compared with high-monounsaturated fat [40% carbohydrate, 45% fat] diets cause persistent deterioration of glycemic control, as well as increased plasma triglyceride and very low density lipoprotein levels, which may not be desirable" (Garg 1994).

There are dozens of such studies, and their results are consistent: higher carbohydrate diets cause deterioration of diabetic control and elevation of heart risk factors, while lower levels of carbohydrates improve all aspects of the condition and lower risk factors for heart disease. Yet the ADA advocates a low fat, high-carbohydrate diet for Type 2 diabetics.

The ADA's rationale for a high-carbohydrate diet

The ADA advice for diabetics goes like this:

> The message today: Eat more whole grains! Whole grains and starches are good for you because they have very little fat, saturated fat, or cholesterol. They are packed with vitamins, minerals, and fiber. Yes, foods with carbohydrate -- starches, vegetables, fruits, and dairy products -- will raise your blood glucose more quickly than meats and fats, but they are the healthiest foods for you. Your doctor may need to adjust your medications when you eat more carbohydrates. You may need to increase your activity level or try spacing carbohydrates throughout the day.
>
> On average Americans eat around 40-45% of our calories as carbohydrate. This is a moderate amount of carbohydrate, not high. Currently some controversy about carbohydrates is raging due to a few new diet books. These books encourage a low carbohydrate, high protein and moderate fat intake. These diets are not in synch with the American Diabetes Association nutrition recommendations, which are based on years of research and clinical experience. In addition, these trendy diets are hard to follow year after year.
>
> A way to see how carbohydrates affect your blood glucose is to monitor your blood 1&1/2 to 2 hours after meals. Checking your blood glucose at this point tells you how high your blood glucose went from the carbohydrates you ate. For good diabetes control, keep your after-meal blood glucose levels at 180 or below (ADA Diabetes Food Pyramid FAQ on 4/8/07).

In other words, forget the Zone (40% carbohydrate) and South Beach (45% carbohydrate) diets, go for maximum carbohydrates! But there are troubling questions about the effectiveness of this approach for weight loss: "overweight and obese women assigned to follow the Atkins diet, which had the lowest carbohydrate intake, lost more weight and experienced more favorable overall metabolic effects at 12 months than women assigned to follow the Zone, Ornish, or LEARN [very high carbohydrate] diets" (Gardner 2007). More troubling questions arise over the effects of the high-carbohydrate diets on lipid profiles:

Table. Changes From Baseline at 12 Months*

Outcome	Atkins	Zone	LEARN	Ornish
Weight, kg	-4.7	-1.6	-2.6	-2.2
LDL, mg/dL	0.8	0.0	0.16	-3.8
HDL, mg/dL	4.9	2.2	2.8	0.0
Triglycerides	-29.3	-4.2	-14.6	-14.9
Systolic BP	-7.6	-3.3	-3.1	-1.9

*LDL, low-density lipoprotein cholesterol; HDL, high-density lipoprotein cholesterol; and BP, blood pressure.
Source: *JAMA*. 2007;297:969-977.

In an interview, researcher Dr Christopher Gardner remarked that "After all those [statistical] adjustments... everything that was still significant favored Atkins, more weight loss than Zone, better triglycerides than Zone, better HDL than Ornish, better blood pressure than all three … there is something really interesting, I think, about carbohydrates and the emphasis really seems to be on cutting back those simple carbs" (Lie 2007).

The science behind the recommendation …

In the ADA's most recent summary of the research underlying their nutritional recommendations is their *Evidence-Based Nutrition Principles and Recommendations for the Treatment and Prevention of Diabetes and Related Complications* (Franz 2002), there is not a shred of high-quality research evidence cited in support of their *opinion* that "Carbohydrate and monounsaturated fat should together provide 60–70% of energy intake", certainly no randomized controlled clinical trials which are the gold standard among research studies. It is the consensus opinion of the ADA Expert Panel. The only high-quality evidence cited relates to how destructive very high carbohydrate diets are!

In their current update, *Nutrition Recommendations and Interventions for Diabetics* (ADA 2006), they add their opinion that "Low-carbohydrate diets (restricting total carbohydrate to <130g/day) [about 25% of calories in a 2000 calorie diet] are not recommended in the treatment of overweight/obesity … [or] diabetes", and cite the Institute of Medicine's *Dietary Reference Intakes: Carbohydrate, Fiber, Fat etc.* (IOM DRIs), in which it is made clear that the amount of glucose necessary to power the obligate glucose-consuming tissues like the brain and blood cells is about 130 grams. But this is a specious rationale! By definition, RDAs are for healthy people, being "the average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all (97 to 98%) *healthy individuals* in a particular life stage and gender group" (p 22). Diabetics are not healthy people; carbohydrates are a metabolic poison for diabetics. One hundred and thirty grams per day is far too much carbohydrate for a diabetic; my diet contains nearer 30 grams per day, about 6% of calories.

A second difficulty with this citation is that the Institute of Medicine is at pains to point out that the absolute requirement for carbohydrates in human nutrition is actually *zero*, "provided that adequate amounts of protein and fat are consumed." They point out that "There are traditional populations that ingested a high fat, high protein diet containing only a minimal amount of carbohydrate for extended periods of time (Masai), and in some cases for a lifetime after infancy (Alaska and Greenland Natives, Inuits, and Pampas indigenous people) (Du Bois, 1928; Heinbecker, 1928). There was no apparent effect on health or longevity." However, the Food and Nutrition Board wish to err on the side of caution because although various populations thrive on very low carbohydrate diets, "a comparison with populations ingesting the majority of food energy as carbohydrate has never been done" (p 275). This is not scientific evidence, it's an opinion tendered because of the absence of evidence. We think that you should eat lots of carbohydrates, and the scientific evidence is, er, others chaps think the same thing. Now we are really down the rabbit hole! It is a different world, in which there is resort to Aristotelian logic: we reason what should be, and then assert it. It is as though Francis Bacon had never formulated the scientific method, and where, as Bacon put it: "For what a man had rather were true, he more readily believes." We see the ADA's enthusiasm for carbohydrates is no more than an article of faith.

Faith … an illogical belief in the occurrence of the improbable
HL Menken

A close reading of the material intended for medical professionals gives implicit approval to a 25% carbohydrate diet, but the material intended for the public says, "Go all-out for carbohydrates!" The material intended for medical professionals acknowledges the destructive potential of high carbohydrate diets for diabetics, but the material intended for the public says no such thing. How can these positions cannot be reconciled: (1) eat a high carbohydrate diet; but (2) high carbohydrate diets may well harm the 92% of diabetics who are insulin-resistant? Does the ADA feel that the public will not make the "right" decision because the issues are too complex?

"No, not really. Except this: we think they're stupid"
Dominic Lawson on the Jewish equivalent of anti-Semitism

Or is it because the enthusiasm of major contributors such as General Mills and Cadbury-Schweppes might cool?

It is difficult to get a man to understand something when his salary depends upon his not understanding it
Upton Sinclair

The ADA go on to justify the presence of carbohydrates in the diabetic's diet: "An important reason for not recommending low-carbohydrate diets is that they eliminate many foods that are important sources of energy, fiber, vitamins, and minerals and are important in dietary palatability." About 80% of diabetics are overweight or obese at diagnosis, so the ADA's concern about energy intake is puzzling. As to palatability, *de gustibus non est disputandum* – matters of taste cannot be debated. However, what is truly unpalatable to me is that I am not to trusted with the real choice here: eat freely of mashed potatoes, develop complications and lose 12 years of life, or to skip the carbohydrates and live out my full span of years *without* developing diabetic complications.

To evaluate the importance of starchy foods as sources of fiber, vitamins and minerals, I used the USDA National Nutrient Database. I looked up amounts of nutrients in a representative group of starchy foods – potatoes, peas and carrots, oatmeal and un-enriched whole-grain bread (less 10% for the sugar in the ADA diet) – and an equal number of calories from low-starch foods I have replace these foods with in my diet: almonds, eggplant, asparagus and bell peppers. There was 35% more fiber in the low-starch group. Minerals were between 3.94 (calcium) and 1.33 (copper) times higher, while sodium (0.07), iron (0.68) and selenium (0.2) were lower; but lower is better where sodium is concerned, and copious amounts of iron and selenium are supplied by red meat and salmon, respectively. Vitamins were between 34 (vitamin E) and 1.4 (vitamin B1) times higher in the low-starch group, but vitamin B6 (0.7) and vitamin A (0.5) were lower; again, salmon is a rich source of vitamin B6, and eggs supply abundant vitamin A. The claim that we may suffer deficiencies without high-carbohydrate foods is patently absurd.

That this prescription makes high blood sugars inevitable is implicit in the advice from the ADA FAQ: "For good diabetes control, keep your after-meal blood glucose levels at 180 or below." A glucose level of 180mg% is not only egregiously high, it's the renal threshold where glucose begins to appear in the urine. But even this admonition is honored mainly in the breach. A typical study gave the mean HbA_{1c} of 17 diabetics in the experimental groups at 9.9%, and that of the control group at 10% (Georgopoulos 1995); an HbA_{1c} of 10% corresponds to an average blood sugar level of 247mg%, which guarantees chronic glycosurea. In Dr Gerald Reaven's diet comparison excerpted above, "24-hour urine glucose excretion was significantly greater (55 versus 26g/24 hours $p<0.02$) in response to the low-fat, high-carbohydrate [ADA] diet" (Coulston 1987). Dr Lawrence Resnick apparently felt an HbA_{1c} of 8.52% was an acceptable outcome after a yearlong trial of a prepared meal plan in 41 diabetics (Metz 2000). The literature is rife with such studies (Gumbiner 1998, Parker 2002,), performed by researchers who are sanguine to the point of complacency about their experimental subjects' failed glycemic control: *any* sugar in the urine means a diabetic is dangerously out of control. Where were the Ethics Committees? How can a literature search be so bungled that low-carbohydrate, blood sugar-normalizing diet studies are overlooked?

This is where the mystery deepens. Diabetic complications are a clear and present danger to the health of diabetics, and these complications are caused by elevated blood sugars, as graphically illustrated above. The ADA dietary recommendations are based upon the hypothetical dangers of high-fat diets causing heart disease. However, the ADA diet causes such high blood sugars that sugar appears in the urine (even when monounsaturated fats replace some carbohydrate), and it is widely accepted that these high blood sugars *accelerate the development of heart disease!* As well as making complications inevitable, Dr DeFronzo has pointed out that hyperglycemia causes both increased insulin resistance and the progressive loss of β-cell function which eventually renders insulin necessary. In other words, the hyperglycemia attendant upon the ADA dietary prescription *causes* the progression of diabetes. The ADA is silent on this consequence of their recommendations, and the researchers in the field are seemingly oblivious of the danger.

Yet the low-carbohydrate diet which renders my blood sugar normal is "not in synch" with the ADA recommendations: "Currently some controversy about carbohydrates is raging due to a few new diet books. These books encourage a low carbohydrate, high protein and moderate fat intake. These diets are not in synch with the American Diabetes Association nutrition recommendations, which are based on years of research and clinical experience. In addition, these trendy diets are hard to follow year after year." Nevertheless, I have followed a far more stringent diet for nine years with considerable pleasure, my blood sugar is usually below 120 mg% after meals, I do not experience glycosurea and my HbA_{1c} is in the normal range at 5.6%. My blood fats actually improved with carbohydrate restriction, to the point where I am at very low risk by the usual yardsticks.

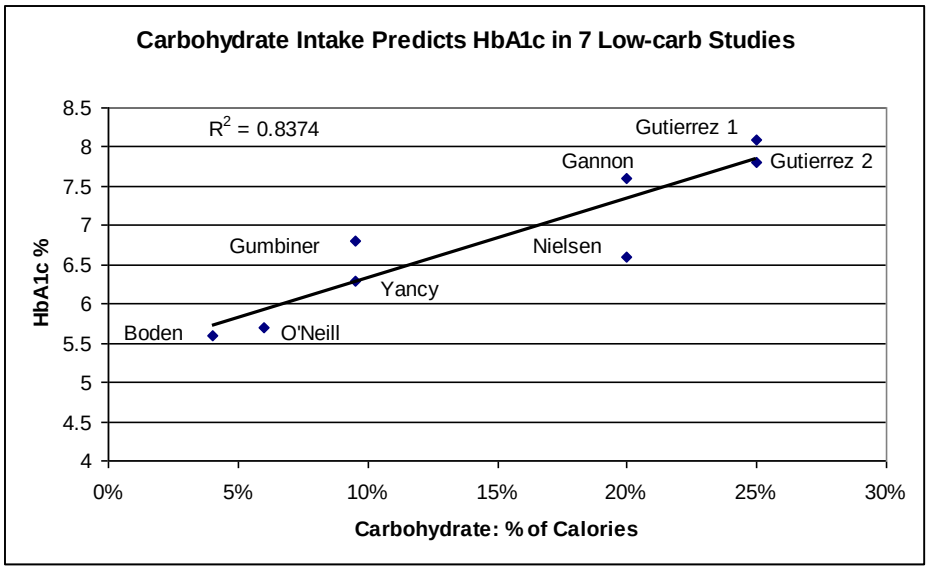
The ADA protests that low-carbohydrate diet studies are short-term and that their safety has not been proven. But this is disingenuous, as the area has actually been quite thoroughly researched. For example "The ketogenic diet is a low-carbohydrate, adequate-protein, high-fat diet that biochemically mimics the fasting state and has been used to successfully treat seizures for 85 years" (Huffman 2006). A long-term follow-up study from Johns Hopkins University showed that "Three to 6 years after initiation, the ketogenic diet had proven to be effective in the control of difficult-to-control seizures in children. The diet often allows decrease or discontinuation of medication. It is more effective than many of the newer anticonvulsants and is well-tolerated when it is effective" (Hemingway 2001). A yearlong Korean study concluded "The KD [Ketogenic Diet] is a safe and effective alternative therapy for intractable childhood epilepsy" (Kang 2005). Several studies a year or more in length establish that less extreme low-carbohydrate diets, used for glycemic control in diabetics or for weight loss in both diabetics and healthy people, are well-tolerated and without side-effects. For example, Dr Jørgen Nielsen found a 20% carbohydrate diet lowered the average HbA_{1c} of 16 obese diabetics from 8% to 6.6% in six months, and it was still low at 6.9% after 22 months (Nielsen 2006). A definitive study followed 66 obese people, some with high cholesterol, on a diet containing less than 20 grams of carbohydrate for 56 weeks to determine the truth of the conventional wisdom that such a diet would cause deterioration of the lipid profile. Everybody lost weight and found their cholesterol, LDL-cholesterol and triglycerides fell, while HDL-cholesterol increased, and the study concluded that low-carbohydrate diets are safe to use "for a longer period of time" (Dashti 2006). There is, in fact, a great deal of long-term research into very low-carbohydrate diets which find them safe and effective; and the diet I follow results in only trace amounts of ketones in my urine.

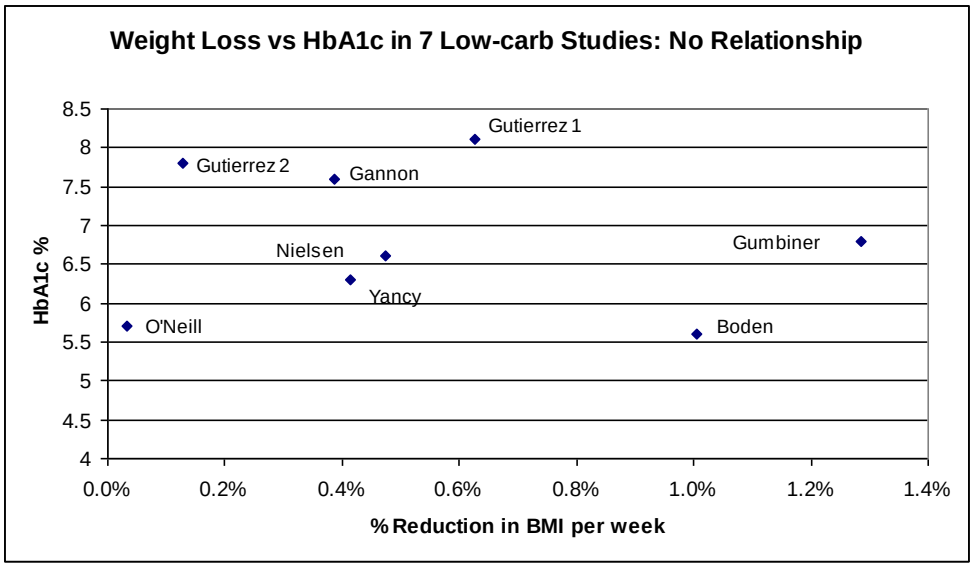
A claim that low-carbohydrate diets are ineffective for glycemic control would be mendacious, but the ADA has sidestepped this necessity with impressive cunning by claiming that low-carbohydrate diets cannot be recommended. However, numerous studies demonstrate that lower-carbohydrate diets are very effective at lowering HbA_{1c}:

Effect of Carbohydrate Intake on HbA1c in Seven Low-Carbohydrate Diet Studies

	Number	Type	Duration wks	BMI pre	BMI post	ΔBMI%/wk	Carbs pre	Carbs post	HbA1c pre	HbA1c post
Boden 2005	10	2	2	39.8	39.0	1.0%	40%	4%	7.3	
Gannon 2004	8	2	5	31.0	30.4	0.4%	55%	20%	9.8	
Gumbiner 1998	9	2	6	36.3	33.5	1.3%	55%	10%	9.6	
Gutierrez 1998	19	2, no OHAs*	8	27.9	26.5	0.6%	55%	25%	9.9	
Gutierrez 1998	9	2, OHAs*	8	29.6	29.3	0.1%	55%	25%	9.2	
O'Neill 2003	30	20x2, 10x1	86	28.7	27.9	0.0%	55%	6%	7.9	
Nielsen 2006	16	2	24	36.1	32.0	0.5%	55%	20%	8	
Yancy 2005	21	2	16	42.2	39.4	0.4%	40%	10%	7.5	

*OHAs: prior treatment with Oral Hypoglycemic Agents

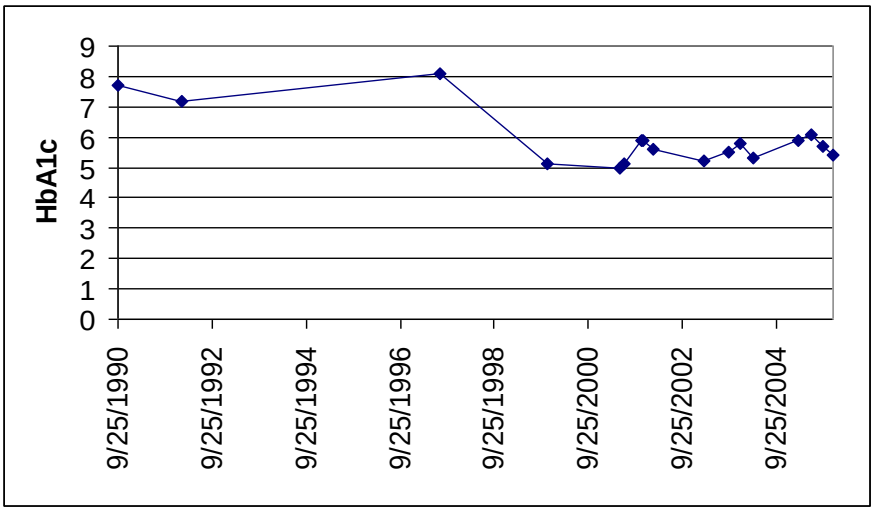




Left: Interestingly, these seven low-carbohydrate (<25% of calories) studies show that there is a strong correlation between the percentage of calories as carbohydrate and HbA_{1c} at the end of the study; "$R^2 = 0.8374$" means that, statistically speaking, the differences in the percentage of carbohydrate calories accounts for about 84% of the change in the values of HbA1c. The rest of the variation likely comes from duration of diabetes and the degree of obesity.

Right: Weight loss no doubt contributes to the lowering of HbA_{1c} for weight loss occurred in all 7 studies, but the absolute intake of carbohydrate was far more important. A review of very low-calorie diet therapy for obese diabetics points out that "Metabolic benefits occur quickly with only modest weight reduction, suggesting that calorie restriction plays a more critical role [than the absolute amount of weight lost]" (Henry 1991). It seems that, in low-carbohydrate diets, caloric restriction is a necessary but not sufficient condition for improvement of the diabetic state, and that it is the degree of restriction of carbohydrate which predicts the degree of benefit which will accrue. *Only* the diets with less than 8% of carbohydrate achieved an HbA_{1c} of less than 6%.

This is in good agreement with my experience. My diet contains about 6% carbohydrate, like Dr Richard Bernstein's diet upon which it is based, and my HbA_{1c} is 5.6%. Before Dr Bernstein published his book in 1998, I ate about 25% of calories as carbohydrate and my HbA_{1c} wandered between 7 and 8%:



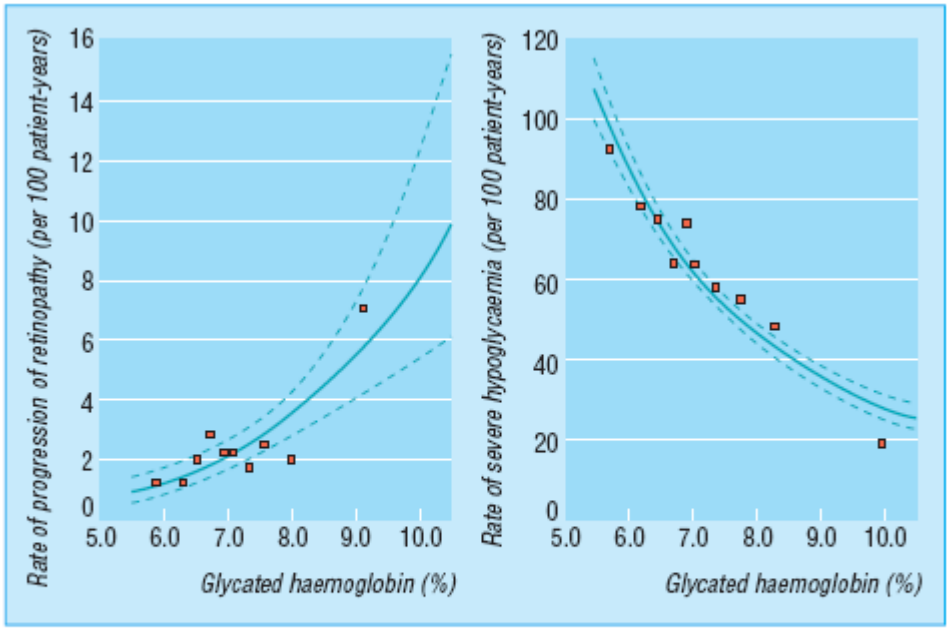
A graph of my $HbA1_c$ scores before and after I adopted a low-carbohydrate diet

Conversely, high-carbohydrate diets improve HbA_{1c} only if they are calorie-restricted, and this effect is barely strong enough to be clinically useful for Type 2 diabetics. For example, when Dr Leonie Heilbronn restricted calories by 30% in two diets containing 60% carbohydrate which were made up of either of low or high Glycemic Index foods and fed them to two groups of diabetics for 8 weeks, she found that "Urinary glucose excretion was not significantly changed … in either group" (Heilbronn 1999). There was a trivial improvement in the low Glycemic Index group, but *any* urinary glucose excretion means microalbinurea (the loss of protein through the kidneys) is not far behind, which will be followed by macroalbinurea and eventually by kidney failure.

Parenthetically, most of the foods I eat mostly do not *have* a glycemic index! "Foods containing little or no carbohydrate (such as meat, fish, eggs, avocado, wine, beer, spirits, *most* vegetables) cannot have a GI value. No carbohydrates = no GI" (The GI Database).

And sometimes, we just don't see the forest for the trees. Dr Cecilia Low compared calorie-restricted diets with 10% and 70% carbohydrate content and found clear advantages for the low-carbohydrate diet over the 6-week study period in obese Type 2 diabetics, yet she dismissed the entire approach: "it is impractical to consume weight-maintaining diets composed of 70% fat" (Low 1996). Never mind that the low-carbohydrate group had just completed four weeks of "re-feeding" doing precisely that, albeit on a liquid-formula diet! Never mind that there is a whole population of free-living diabetics who approach this level routinely, and consequently have normal blood sugars. Extraordinarily, even proponents of low carbohydrate diets draw the line well above the level of carbohydrates shown to be effective. Dr Surender Arora wrote in a review *The case for low-carbohydrate diets in diabetes management* that "Some form of low carbohydrate diet in combination with exercise is a viable option for patients with diabetes. However, the extreme reduction of carbohydrates of popular diets (<30 g/day) cannot be recommended for a diabetic population at this time without further study" (Arora 2005).

On the ADA high-carbohydrate way of eating, tight blood sugar control lowers complications but markedly increases hypoglycemic episodes in insulin-dependent diabetics (Donelly 2000):



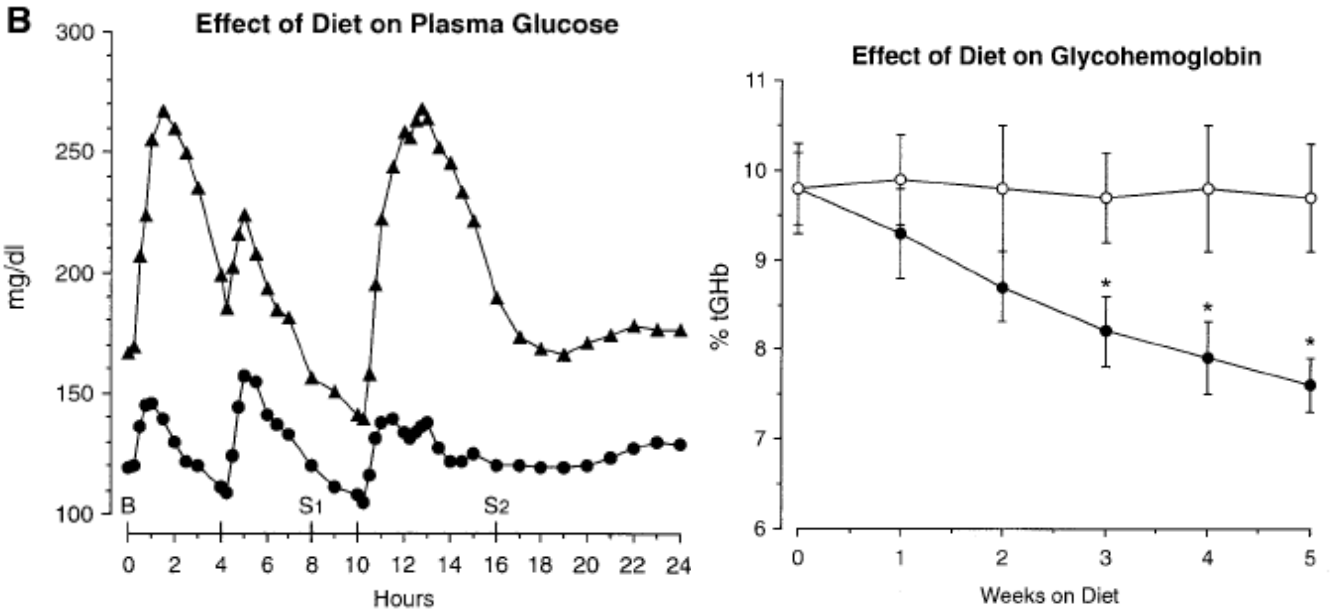
Relation between glycaemic control (Hb_{A1c}) and risk of progression of microvascular complications (retinopathy) and severe hypoglycaemia in patients with type 1 diabetes. Data from the diabetes control and complications trial. Dotted lines represent 95% confidence intervals

The graphs show that the rate of progression of retinopathy is lowest at an HbA_{1c} of 5.5%, but the rate of severe hypoglycemia is highest, about one episode per year. Severe hypoglycemia is very risky because you may make catastrophic errors of judgment, lose control of your car, have a seizure or worse. However, diabetic complications consequent on high blood sugars cause blindness, limb amputation, kidney failure and accelerated heart disease. Insulin-dependent diabetics constitute only 10% of the diabetic population, yet the argument that tight control is too risky because of the attendant hypoglycemic episodes is somehow generalized to all diabetics, implicit in the ADA's target HbA_{1c} of 7% for good control. Yet I take insulin, maintain an HbA_{1c} of 5.6%, and do not have severe hypoglycemic episodes. Nor do the patients of Dr Richard Bernstein, who wrote:

In the 20 plus years that I have been in practice, only five of my patients have had severe hypoglycemia causing loss of consciousness. Two of these people were eating excessive amounts of carbohydrate and three made major mistakes such as taking the wrong type of insulin. I'm sure this is a far cry from the incidence of severe hypoglycemia among patients of high carbohydrate practitioners (Bernstein Interview).

Severe hypoglycemia is vanishingly rare on low-carbohydrate diets. So what's the secret? For one thing, there's ketosis, in which two- to four-carbon fragments of fats are formed in the liver when the diet is low in carbohydrates – these "ketones" can directly nourish many tissues, including some brain structures which would otherwise burn only glucose, so that when the blood sugar drops too low, the brain runs mostly on ketones (Johnson 1978). Ketones have been found to lower the excitability of a brain-stem structure that makes the neurotransmitter dopamine, the *substantia nigra*, by promoting potassium accumulation within its cells (Ma 2007); and this protects against seizures. Ketones have also been shown to protect the mitochondria of this structure against toxins and may therefore protect against Parkinson's disease; further, they similarly protect the mitochondria of the hippocampus and so likely protect against the dementias (Veech 2000). The widespread prejudice against ketones and ketosis stems from the widespread misunderstanding that ketones and ketosis only occur in diabetic ketoacidosis, an often-deadly consequence of too little insulin in insulin-dependent diabetes. The fact is that low-carbohydrate diets protect against hypoglycemia.

Interestingly, it is the ADA's own much-vaunted research, used so much to justify their low-fat diet, which has demonstrated that a low-carbohydrate diet lowered HbA_{1c} to normal in 8 overweight Type 2 diabetics. The study was funded by the ADA with help from the beef industry and published in the ADA journal *Diabetes* in 2004. A diet containing 20% carbohydrate, 30% protein, and 50% fat was compared with American Heart Association 55%-carbohydrate, 15% protein and 30% fat diet for 5 weeks, and demonstrated such improved control that the conclusion of the study was that the diet "could be a patient-empowering way to ameliorate hyperglycemia without pharmacological intervention." Blood sugars plummeted to almost-normal levels, serum insulin fell dramatically, triglycerides dropped to a normal 150 from Syndrome X-territory near 250, and HbA_{1c} fell two full percentage points in just five weeks; had the diet continued for three months, the researchers projected that HbA_{1c} would have fallen to 5.6% (Gannon 2004):



Left: Mean plasma glucose concentration before (▲) and after (●) 5 weeks on the LoBAG [low-biologically-available-glucose] diet. Right: Mean % tGHb [HbA_{1c}] response during the 5 weeks of the control (○) or LoBAG diet (●). *The tGHb on the test diet was significantly lower at weeks 3, 4, and 5 vs. the control diet ($P < 0.05$).

In another study of a 20% carbohydrate diet, this time in obese diabetics, "Patients spontaneously reduced their mean energy intake to approximately 2200 kcal/day [a reduction of 30%], which is approximately the caloric intake of normal-weight individuals with the same height as our patients … Mean plasma leptin levels were lower …" (Boden 2005). Leptin is a hormone with helps control appetite, and sensitivity to leptin parallels sensitivity to insulin. These diabetics lost their excessive appetites in spite of lower leptin levels, so this 20% carbohydrate diet increased their sensitivity to insulin (and therefore leptin) dramatically, perhaps in part by controlling their hyperglycemia. Spontaneous caloric restriction is unheard of among Type II diabetics, 80% of whom are overweight at diagnosis. Most diabetics gain weight over time, whatever advice they may be given on weight-loss, and a study has found that fully 59% of them do not exercise – in fact, this study showed that the higher their HbA_{1c}, the greater the likelihood that the diabetic would be sedentary (Morrato 2007). Any diabetic knows that the worse the blood-sugar control, the lower one's energy level. But it is the complacency with which this staggering lack of compliance with a common-sense protective prescription is viewed by the medical profession that is truly surprising.

While mainstream medicine seems unable to think outside the box, articles such as "Can Diabetics Have Normal Blood Sugars with Diet Alone?" by Regina Wilshire summarizes some of the evidence and concludes that essentially normal glycemic control is possible for diabetics. Similarly, Men's Health magazine published an article by Adam Campbell entitled "The Cure for Diabetes" which points out that diabetics treated by Dr Mary Vernon with the Atkins low-carbohydrate diet are "cured" in the sense that they no longer have a blood sugar problem so long as they stick to the diet. Anthony Colpo ably summarized the issues in his excellent, heavily-referenced articles Why Diabetics Should Avoid High Carbohydrate Diets and Why the Low-Fat Diet is Stupid and Potentially Dangerous.

The ADA's much-vaunted "clinical research" has scrupulously neglected to test this way of eating since it was proposed by Dr Robert Atkins in 1972 until very recently. Nevertheless, could it be that, after more than 30 years, they are surreptitiously maneuvering towards a bid to make it their own? Perhaps there now begins that medical procedure requiring the utmost skill and delicacy: distancing oneself from the defective medical advice that was once the standard of care but which has been found to maim and kill! But there is precedent. Dr Walter Willett acknowledged the folly of promoting margarine over butter:

> Unfortunately, as a physician back in the 1980s, I was telling people that they should replace butter with margarine because it was cholesterol free, and professional organizations like the American Heart Association were telling us as physicians that we should be promoting this. In reality, there was never any evidence that these margarines, that were high in trans fat, were any better than butter, and as it turned out, they were actually far worse than butter (Dr Walter Willett interview).

Similarly, Dr. Thomas Stamey of Stanford, who was one of the first to link PSA with prostate cancer, now believes that the test is overused. He recently published a study that said that PSA levels between 2 and 9 are "clinically useless" for determining tumor size (Stamey 2002). He regrets his previous advocacy of the test: "I've removed a few hundred prostates that I wish I hadn't" (PSA Testing).

If diabetics were not rendered so compliant by their condition, the "lucky" ones who were merely maimed might band together in a class-action suit and prevail upon the ADA to accelerate their glacial pace of change. However, if the American Diabetes Association was to change its stance, and diabetes became a benign chronic condition requiring only a low-carbohydrate high-vegetable diet, consider what would happen. The forty-seven percent of the country that already has diabetes or pre-diabetes would be prescribed this new low-carbohydrate diet, halving the profits of the food industry overnight. Then the new, redesigned USDA food pyramid would advise against sugar and refined food, and the markets for genetically-modified corn (high-fructose corn syrup) and soybeans (the oil in most processed foods) would dwindle, forcing painful changes in agriculture. Similarly, the falling demand for diabetic drugs and impedimenta would cause squeals of pain from the pharmaceutical industry, which wealds so much financial clout that it actually elects Presidents sympathetic to its needs. And if a revolution in favor of avoiding carbohydrates were to occur, the first casualty of this new world order? The American Diabetes Association! Since the food industry and the pharmaceutical companies would no longer have the means or desire to support them, their continued existence depends on their aligning their dietary advice with the interests of their contributors.

This has not gone altogether un-remarked. Australian physician Dr Jay Wortman wrote:

> The epidemics of obesity, metabolic syndrome and type 2 diabetes have worsened over the past decades … these conditions [may be] inter-related and may be caused by a single underlying factor related to the carbohydrate content of diet … Aboriginal people suffer more acutely from the epidemics in question and their dietary history suggests that a sudden increase in carbohydrates is to blame. Recent studies … demonstrate that carbohydrate consumption can drive appetite and over-eating while carbohydrate restriction leads to weight loss and improvement in the markers for metabolic syndrome and type 2 diabetes. The growing evidence in support of low-carbohydrate diets will encounter resistance from economic interests threatened by changes in consumption patterns (Wortman 2006).

His prescience stems in part from solving his own brush with diabetes:

"… diabetes is a disorder of blood sugar, your blood sugar is too high -- my immediate instinctive response was to stop eating any food that causes your blood sugar to rise. So I basically right away eliminated carbohydrates from my diet … In four weeks, I lost 18 pounds. My blood sugars normalized, my blood pressure became normal, and I felt much better," Wortman says. "I don't know if you're ever not diabetic, but I think for me, I've been able to reverse the effects of diabetes through diet" (Wortman Interview).

There's a pattern here. Any who have experience of resolving unhealthy blood sugar excursions by eliminating carbohydrates so far as is possible never look back. Perhaps the American Diabetes Association is nobly holding the line for evidence-based medicine. Or (my favorite theory) these are the guys Max Planck was thinking of when he wrote:

A new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it
Max Planck

Whatever the reason, the ADA behaves as a cat's paw for its pharmaceutical and food industry contributors, and its high-carbohydrate diet remains the standard of care while "more research" is called for. Yet they are obtuse to the point of misrepresentation in their coverage of low-carbohydrate diet studies. For example, researchers found that a low-carbohydrate diet dramatically reduced HbA_{1c} and other important disturbances typical of the diabetic metabolism and concluded "The lack of negative effects, improved glucose control, and a positive nitrogen balance suggest beneficial effects for subjects with type 2 diabetes mellitus …" (Nuttall 2006). However, the ADA summary read "The amount of fat and carbohydrates in a person's diet does not appear to have an effect on metabolism."

Further, the ADA is adamantly opposed to any and all vitamin or mineral supplements. This is awfully hard to reconcile with the staggering weight of evidence in favor of, for example, chromium, biotin and magnesium for lowering blood sugar, or the published research on deficiencies actually found in diabetics. Spectracell Inc, who market a test for the adequacy of nutrition determined by the health or otherwise of cells in nutrient solutions deficient in a single nutrient – if the cells thrive, their owner is likely adequate in the missing nutrient – summarize the research on deficiencies found in diabetics:

> [Type II diabetes is a] widespread metabolic disease [which] has been associated with several nutrient deficiencies, some of which have been linked to progression of clinical disorders that can occur secondary to chronic diabetes. Magnesium deficiency, in particular, has been associated with type 1 and type 2 diabetes as well as with gestational diabetes. Supplementation of type 2 diabetics with magnesium was found to improve both insulin secretion and insulin sensitivity. Experimental studies in animals and cross-sectional studies in humans have suggested that low serum magnesium levels might actually contribute to the development of diabetes. A recent prospective study revealed a graded inverse relationship between serum magnesium levels and the development of diabetes. Magnesium deficiency has also been linked to progression of clinical disorders related to chronic diabetes.
>
> Deficiencies of several vitamins have also been identified in diabetic individuals. Vitamin B6 levels were found to be lower in diabetic animals than in normal controls, and sub-clinical vitamin B1 deficiency was prevalent in pregnant women with gestational diabetes.
>
> Deficiencies of antioxidant vitamins have also been associated with diabetes. Several studies have found that diabetic patients had at least a 30% lower level of plasma ascorbic acid than non-diabetic subjects, and a strong independent association was found between low plasma vitamin E levels and an increased risk of developing diabetes. Subjects with clinical nephropathy had lower mean plasma ascorbic acid levels and higher mean renal clearance of ascorbic acid than patients having only microalbuminuria. Thus, SpectraCell's antioxidant panel and functional determinations of B vitamins and minerals can help to detect diabetes-related nutrient deficiencies before they contribute to the progression of the disease or to the development and progression of its complications (Spectracell).

A controlled study in which diabetics were given a multivitamin showed that their risk of illness was reduced by 82% (Barringer 2003).

The ADA is against supplementation with magnesium, even though low magnesium has been linked to the progression of diabetic complications such as retinopathy and kidney failure! All this renders the ADA's dietary advice antithetical to the interests of any diabetic who wants to live out his or her span: their prescription costs the average diabetic 12 years of life, and the risk of death doubles at any age for a person with diabetes. Consequently, Dr Lois Jovanovic of the Sansum Diabetes Clinic in Santa Barbara has called their prescription "Malpractice!", and Dr Richard Bernstein wrote with admirable restraint "the guidelines given by the American Diabetes Association have proven unhelpful … in managing to maintain a healthy weight and normal blood sugars." Dr Bernstein developed insulin-dependent diabetes in 1946 when he was 12 years old; he wrote that "For many years I accepted the medical orthodoxy and it nearly killed me" (The Diabetes Diet)"

In summary, diabetes and caloric excess coexist in those at the greatest risk of complications and death. Either avoiding carbohydrates so far as is possible, or restricting calories in a high-carbohydrate diet lessens insulin resistance and lowers this risk. But carbohydrates do not of themselves cause weight gain and insulin resistance, for the Kitava Islanders remain lean and insulin-sensitive throughout their lives on an unrestricted, *unrefined* carbohydrate diet. It is therefore refined carbohydrates which must be at fault, probably both because they do not trigger satiety signals and because they no longer contain nutrients essential for their metabolism; so we over-feed and under-nourish our metabolisms until they fail. A key part of any remedy, then, is to avoid refined carbohydrates altogether, and to avoid nutrient-poor carbohydrates like potatoes to a degree commensurate with the extent of the damage. This is the polar opposite of the Medical Nutrition Therapy prescription of the ADA, but, in contrast, it has been shown to be effective.

I personally bitterly resent the ADA's dietary position because I believe that following their advice has shortened my life. It cost me fifteen years of my HbA_{1c} score elevated by two percentage points to learn how wrong they are. To the extent they believe what they say, I believe they are tragically mistaken. To the extent that they know the low-carbohydrate approach saves lives, but put their own interests above the interests of the diabetics who turn to them for guidance, I am at a loss for words.

Summary

Drs Moore, Reaven, Willett, Resnick and the others cited herein are truly prophets without honor in their own land, for they have not only elucidated the cause of the epidemic that plagues us, they have demonstrated a solution. They used epidemiology to prompt hypotheses, and confirmed them by interventional studies: the very highest quality of scientific evidence supports the notion that refined foods cause insulin resistance and its *sequelae*, the degenerative diseases suffered by Western cultures.

Nevertheless, we have put our faith in the false prophets of the cholesterol hypothesis, and those who advocate high-carbohydrate diets for weight loss, diabetes and the treatment of heart disease. When you actually look at the evidence, it requires an act of faith to believe the flimsy results of the flawed trials offered in support their conjectures; trials moreover which were conceived and carried out by commercial interests with a colossal financial interest in their outcome. As Mark Twain put it, "Many a small thing has been made large by advertising."

Conclusion

In facing my own health crisis, I bet the farm on a little-known dietary strategy for controlling my diabetes because the conventional medical prescription is certain death. Not only did it succeed, it has apparently protected my wife and me from heart disease as well, even from the small degree of calcification of the aorta which is considered a "normal" part of aging. I have come to believe that fresh, nutrient-dense foods are protective, so that people like the Kitava Islanders who eat such foods simply do not suffer from Western degenerative diseases.

Dr Lawrence Resnick has provided a *plausible mechanism* that explains why correction of the potassium-to-sodium ratio and the calcium-to-magnesium ratio to their values in an unrefined diet normalizes insulin sensitivity, high blood pressure, diabetes and syndrome X, thereby decreasing the tendency towards CHD and cancer. Similarly, restoring the levels of the B vitamins lowers homocysteine, an irritant of the blood vessel lining which contributes to cardiovascular disease and Alzheimer's. And adding back the antioxidant minerals and vitamins resolves the inflammation which robs the blood-vessel lining of its protective functions, and lessens the atherogenic oxidation of LDL-cholesterol which sustains the growth of atherosclerotic plaque. Refined food *causes* the ills that plague us.

It is worth spelling out the implications here because they run so counter to the conventional wisdom that it is easy to miss them. The American Heart Association has chronicled the massive increase in health conditions characterized by underlying insulin resistance: "There's been an explosive increase in the prevalence of obesity and Type 2 Diabetes. Their related complications – hypertension, hyperlipidemia and atherosclerotic vascular disease – also have increased." More than six of every ten of us is overweight, eight in ten of those over 50 have high blood pressure (Sacks 2001), one in four has diabetes or pre-diabetes, and one in four has syndrome X. Although there is overlap in these figures (for example, eight of 10 Type 2 diabetics are overweight at diagnosis), it is clear that almost all of us are incubating an insulin resistance-related disease because, by the age of 75 years, 90% of men and 80% of women have cardiovascular disease! This is entirely self-inflicted: we are digging our graves with our teeth, at least with those teeth that remain to us since the sugar which we eat our own weight of each year has been proven to cause tooth decay. No Kitava Islander has heart disease or diabetes.

Another implication is that the healthfulness of a diet does not depend on whether it is low in carbohydrate or fat. The conclusion of the seemingly endless debate over the merits of low-carbohydrate and low-fat diets is simply that *either* diet work to eliminate heart risk, obesity and diabetes *if* magnesium is plentiful and the potassium-sodium ratio is high, and there are abundant vitamins, minerals and omega-3 fatty acids. My experience is that the low-starch diet which controls my blood sugar is healthful even though most of its calories come from fat. My risk factors at 60 years of age are remarkably good after almost 25 years of insulin-dependent diabetes, and, in fact, for anybody at any age. My most recent tests show my blood pressure to be 110/76, HbA_{1c} at 5.6%, triglycerides are 64, cholesterol is 164 with LDL-cholesterol at 111 and HDL-cholesterol at 42; and my calcium score in the aorta is zero. Apparently, Dr Dean Ornish, Dr Robert Atkins, Nathan Pritikin and so many others among us have been as the blind men who described the Elephant: each solved a part of the puzzle and thought they had deciphered the mystery. It does not much matter what is in the diet as long as the food is unrefined. Imagine! No more fiddling about with the ratios of fat, carbohydrate and protein in search of the elusive healthful combination, no more futile anxiety over cholesterol levels, no need for ever-more-expensive drugs.

So what have we got here? Nothing less than the chance of living out our span! All that is required of us is that we eschew refined foods and table salt. This may be hard, given the addictive nature of sugar and our taste for salt, but we *can* use sweeteners and salt substitutes, and relieve our indigestion with Milk of Magnesia instead of sodium bicarbonate. Of course, we can also choose *not* to do this. As Robert Frost put it, "I hold it to be the inalienable right of anybody to go to hell in his own way." It is the American way! But should health failure stiffen our resolve, we have the choice. Health failure was a wake-up call for me that had an almost miraculous effect. This has happened to others. The National Weight Loss Registry reported that nearly 77% of the sample reported that a "triggering event" had preceded their successful weight loss.

For more than a century, the food refiners have encouraged us to eat freely of refined cereal products, assuring us of their safety with junky studies funded through their donations to the disease Associations. Physicians have been lulled into a false sense of security along with the rest of us.

It is so hard to get anything out of the dead hand of medical tradition
Oliver Wendell Holmes

So many of medicines sacred cows, so much of the conventional wisdom, turn out to be irrelevant. Cholesterol has little to do with heart disease, and statin drugs lessen risk by a trivial amount. Lowering blood pressure with drugs does little to mitigate the danger of heart disease and stroke, and changing the proportions of carbohydrate, fat and protein in the diet is like rearranging the deck chairs on the *Titanic*: utterly ineffectual. It is the *quality* of food that counts, and whole foods do more than anything in the pharmacy does: they *cure* the underlying imbalances that lead to the diseases that afflict us. The primary fault of our Western diet is that it is refined almost beyond recognition.

Yet a diet high in refined carbohydrate is *implied* to be healthy by the USDA Food Pyramid, and by the complacency of the Nutrition Research Council who set the RDAs for nutrients and have the power to, but do not suggest, fortification. The government we have elected to protect us is asleep at the switch.

If people were told of their dangers, they would consent to make the necessary sacrifice …
only the facts can tell the tale, and the public ought to have them.
Winston Churchill

We have the example of the Kitava Islanders, who simply prepare and eat what they gather and do not get sick as we do. We are not members of a primitive society, but we can choose to "gather" selectively in our supermarkets, electing *not* to gather Dr Weston Price's

"impoverished foods of civilization." In this way, we can likely both protect our health *and* prevent the dementias that rob so many of us of our golden years.