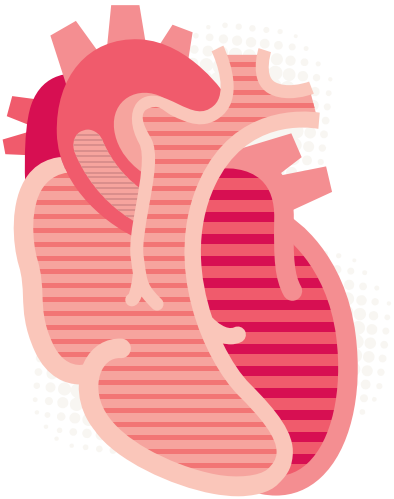
Center for Creative Leadership®

The Care and Feeding of the Leader's Heart



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Video clips of **Yoga** and **Dynaband Workouts** and **Recipes** are available at: www.sharonlarsen.org

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"He who does not know food, how can he understand the diseases of man?"

~Hippocrates

Some who read the title of this paper might well ask: Do executives even have hearts? The stereotype of top level CEOs, or at least one that is often portrayed in movies (think Gordon Gecko), is that executives are cold, calculating, politically savvy and heartless creatures who will sacrifice all for the bottom line. Our experience, however, is quite the opposite. Leaders, with few exceptions, have big hearts and care deeply for their work, the people that work for them and their families. Yes, they are driven. But they are driven because they want to be the best leaders they can in order to best serve their organizations and the people who work within those organizations.

But there is one area where most leaders are guilty; they neglect to take physical care of their hearts. From data collected on thousands of executives we know that, while most leaders are not the proverbial heart attacks waiting to happen, the vast majority have cholesterol levels that are above the ideal, don't exercise regularly and don't eat a particularly healthy diet, or at least a diet that has been shown to prevent and reverse heart disease.

We all know the statistics. Heart disease or, more aptly, coronary arterial disease (from now on referred to as CAD) is the leading cause of death in western countries. And, recently, it became the leading cause of death worldwide.

Arterial Plaque is Ubiquitous as Is Getting a Heart Attack

The lifetime odds of having a heart attack or stroke are quite high. If you put one bullet into a three chambered gun, my guess is that you wouldn't be willing to put the gun to your head for a quick game of Russian roulette. Yet those are the same odds of dying from a heart attack. Ischemic strokes are closely related and are not too far behind heart disease as a leading cause of death. Cardiovascular diseases, in fact, kill more people than all cancers combined.

But more troubling were the results of a pediatric study where researchers autopsied and studied the arteries of 3000 young people who had died in suicides, automobile accidents and homicides. What they discovered was that nearly all had evidence of plaque in their arteries.[1,2] Even more concerning was, and I quote, that "almost all persons have aortic fatty streaks by age 10".[3] Thus if you are reading this and are over the age of 10 chances are that you don't need to prevent this disease, you need to reverse the disease you already have.

Another landmark study that alerted us as to how widespread this disease is (and to the fact that it starts early in life, even years before there are clinical manifestations of the disease) was a study where 300 autopsies were

performed on US soldiers who died in the Korean War.[4,5] The average age of these soldiers was 22. Of these 300, 77% showed gross evidence (i.e. visible to the naked eye) of plaque. Some even had arteries that were already close to 90% blocked.

So what (or who) can we blame for this epidemic?

Mostly, we love to blame our parents. As the ads for cholesterol lowering medications say, "when diet and exercise aren't enough..." the implication being that genetics must be the primary culprit, and thus we badly need lipid lowering drugs. However, we are not dying from this disease because we are suffering from a drug deficiency.

This is not to say that genetics doesn't ever play a role. But to quote Dr. Clifford Roberts, the long-time editor-in-chief of the American Journal of Cardiology, someone who has had his finger on the pulse of cardiac research for many years, "Heart disease is infrequently genetic in origin.[6] Most of us get heart disease because we eat too much fat, cholesterol and calories." He goes on to say that only 1 in 500 people have heterozygous familial hypercholestremia. These people have cholesterol levels in the 300-400 range and tend to have heart attacks at 31 to 50 years of age. One in one million people have homozygous familial hypercholestremia and their cholesterol levels often exceed 800. These people tend to have heart attacks in their 20s or younger.
His point being that for the vast majority of people, parents are NOT to blame.[7] CAD is a food borne illness. It is preventable and reversible.

But we are getting ahead of ourselves. Let's start by looking at populations where heart disease was very rare.

Examples of Populations Where Heart Disease Was Very Rare

There are a number of populations around the world where autopsied and pathological verified studies have historically found CAD to be very rare or even non-existent.

Dutch East Indies 1916-1922: De Langen, a Dutch doctor working in Java, was probably the first to report a relationship between diet, serum cholesterol and CAD. He observed that native Javanese did not experience heart disease and atherosclerosis. This was in stark contrast to the Dutch immigrants. He also observed that their diets were very different from those of the Dutch in that they ate a mostly vegetarian, rice based diet. He also measured their serum cholesterol levels and found it to be much lower in the Javanese compared to the Europeans. The Javanese working as stewards on Dutch ships, however, who ate the same foods as the Dutch, had levels closer to those of the Dutch. He then took a group of Javanese and fed them a diet rich in eggs, butter and meat for three months. Their mean cholesterol rose 28%.[8]

Okinawa circa 1949: In one study in which 150 autopsies were performed, only 7 were found to have slight evidence of fatty streaks. All seven were over the age of 66.[9] Only one had evidence of slight calcification. Subsequent studies showed similar results based on over 200 autopsies.[10] Dietary analysis revealed that their fat and protein intake was very low, 6% and 9% respectively. Most of their calories (69%) came from vegetables, mostly in the form of sweet potatoes. They also ate green and yellow vegetables, soy-based foods and medicinal plants.[11] Rice, wheat and other grains made up 19% of their calories. Only 2% of their total

calories came from fish, milk, eggs and meat. Pork was eaten on special occasions; however, it was boiled and the saturated fat carefully removed.[12] Not only were cardiac diseases rare, but other diseases, such as various cancers (breast and prostate, for example), were rare as well.[13] Additionally, these are among the longest lived people on the planet. Not only did they live long lives, but they also exhibited a high degree of functionality in their later years.

Africa circa 1950s: A paper published in 1959 by researchers at the famed Groote Skuur hospital in Cape Town, South Africa, reported that "There is striking freedom among the Bantu and Negro populations of Africa from ischaemic heart disease. This freedom is evident from clinical, electrocardiographic and pathological evidence. It is real and not attributable to lessor age expectation".[14] In another study from this region, evidence of ischemic heart disease (as confirmed by autopsies) was around 7% in the Bantu group and over 50% in the age-matched European group. When dietary comparisons were made, the authors commented that "The Bantu diet is comparable to that of the White with respect to total caloric consumption, but there the resemblance ends." The Bantu diet consisted mostly of maize, sorghum, beans and pumpkins. Meat was only eaten on special occasions. The authors went on to say "The Bantu diet is deficient by western standards in animal protein (although total protein intake may be high). It has very low total fat, but it is rich in carbohydrates and food fibre." The White populations on the other hand ate a typical European meat heavy, high fat (43%) diet.[15]

Uganda circa 1960s: In a paper published in 2012 it reviewed clinical and dietary data of native and immigrant populations in Uganda. The leading paragraph of the paper said this, "In the African population of Uganda coronary heart disease is almost non-existent. This statement is confirmed by adequate necropsy evidence."[16] In another paper published in 1960 on this population it was reported that out of 1427 autopsies only 1 showed slight evidence of a myocardial infarction. Another study on this group in which 632 autopsies were performed, found only one person who showed evidence of a myocardial infarction vs. 136 (21%) in an age matched group in the US. Again the diets differed markedly with respect to fat, plant food intake and meat intake.[17] Staple foods for the Africans were plantain, sweet potatoes, yams, cassava, maize, millet, groundnuts, leafy greens and other vegetables. Among the Africans, meat was only eaten about once per month or at most 1x week.

Papau, New Guinea circa 1970s: Of interest is that this group was known for its heavy tobacco use (most smoked) yet autopsies on 724 individuals (as well as subsequent studies) found no evidence of heart attack incidence and only one case where there was evidence of a slight narrowing of the coronary arteries.[i] In 1973 results of another survey were published about this group and reported that there was no evidence of heart disease, diabetes, gout, Parkinson's disease or stroke.[18] They were also reported to have the lowest rates of hip fractures in the world and mortality from colorectal cancer was also very low. When the diets of these people were analyzed, it was found that, while their caloric intake was more than adequate, carbohydrates accounted for 94.6% of their total energy intake (the highest in the world) and protein was only 3% of their calories. Estimated grams were 25g for men and 20g for women (Atkins would turn over in his grave at this one). In spite of this low intake there was no evidence of dietary induced protein deficiency or anemia. They were described as being "muscular, lean, physically fit and in a good nutritional state".

China, Guizhou county, circa 1960s: Out of 246,000 men under the age of 64, who were followed for three years, there were no coronary disease deaths. None, zero. Once again, in-depth analysis (i.e. researchers went into homes and recorded dietary practices) showed that their diets were low in animal foods and rich in plant foods.[220]

i. The McDougal Newsletter, February 2013. An independent critique of low-carb diets: the diet wars continue-Part 3. Vol 12, Issue 2

The common dietary thread among these populations is that they all consumed a whole foods, plant centered diet. Animal and processed foods made up a very small percentage of their diets.But these populations were also highly active. Or maybe it was the lack of processed foods? Could that have been what made the difference?

Studies on an Asiatic people at the opposite end of the dietary spectrum--the nomadic Kirghuz plainsmen who were also highly active, and ate no processed foods, yet who consumed unusually large amounts of milk and meat (all grass-fed)--showed a high incidence of premature extensive atherosclerosis, poor kidney function, apoplexy and they died young. In contrast their genetic cousins, who ate mostly potatoes and wheat, did not exhibit such severe vascular disease or other chronic conditions. They were described as being supremely healthy and they lived well into their 70s.[19]

But what about other primitive populations where heart disease was purportedly low, yet who eat lots of animal based foods like meat, animal fat (blubber), blood and milk? The Inuit and Masai are often cited as examples of meat eating populations that were apparently free of atherosclerosis, heart attacks and strokes.[20]

Populations Where Meat and Fat Consumption is High and CAD is Low.
A Paradox?

Tales of populations who do eat high amounts of meat and dairy and who supposedly don't have heart disease are just that, a tall tale. An in-depth review of the research does not support this apparent paradox. For a detailed critique and review of the research on this topic, I refer you to: www.plantpositive.com and his videos on the Masai, Eskimos and the Aboriginals of Australia.

But here are some of the highlights:

One study published in 1960 about the Alaskan natives said this, "Qualitatively, the pathological findings within the native Alaskan population do not appear to be significantly different from those found in the rest of the US."[21] In other words, their rates of disease were not found to be that different compared to the US at a time when deaths from heart disease in the US had peaked. Another study published in 1990 by Danish researchers reported that Greenlanders who ate traditional diets (fish and blubber) had almost the same degree and extent of atherosclerosis in the carotid and femoral arteries as the Danes.[22] Studies of mummified Eskimos from pre-historic times have also shown evidence of substantial arterial plaque.[23]

Rates of osteoporosis among this population are also reported to be amongst the highest in the world.[24] Parasitic infections are also widespread; no doubt a result of eating raw and undercooked fish.[25] Their toxic burden is also

very high, again from their high consumption of fish.[26] Certain types of cancers are also reportedly high (even in mummified corpses).[27,28] I could go on, but suffice it to say, it is hard to understand why low-carb advocates hold these up to be the epitome of the ideal dietary model.

As for the Masai, early reports[29] of them having low rates of CAD were later disputed by subsequent autopsies. When bodies of Masai were autopsied, their arteries were found to be as thick and hardened as old men in the US.[30] However, in spite of this, the diameter of their blood vessels was found to be quite large. This was attributed to their high levels of physical activity. Another fact worth noting is that while whole milk consumption was relatively high, meat consumption was likely not as high as some would suggest. Their cattle, quite frankly, are more valuable to them alive and as a means of providing them with milk. They also eat a variety of grains, medicinal plants and vegetables. Their blood cholesterol levels are, paradoxically, quite low. There are a number of explanations for this. First, it has been reported that their caloric intake was low relative to their energy expenditure. A number of studies have reported that their energy intakes were 65-80% of their estimated needs.[31] When calorie intake is low, cholesterol levels also tend to be low, regardless of the make-up of the diet. Finally, they also have parasites, no doubt from consuming raw blood. Parasitic infections result in lower serum cholesterol levels.[32] Yet, in spite of low cholesterol levels, their arteries still exhibited evidence of plaque deposition. Again, it is hard to fathom why low-carb advocates hold up this population as a model of nutritional excellence.

A modern day population that is often held up as an example of a population who eats lots of fatty foods yet who have low rates of heart disease are the French. This is called the French paradox. Here again, an examination of the data shows otherwise. A French statistician, Pierre Ducimetiere exposed the real truth of this paradox: It simply does not exist.[33] What was happening was that heart disease was not always identified as the real cause of death. Many heart attack deaths were in fact labeled under so called “junk codes”.[34] This has since been rectified and, low and behold, the French now have comparable heart disease rates to other Western countries.

But population data are observational in nature and only show associations. However, they are a good start and can provide clues as to what factors contribute to this disease. More importantly, what are the biologically plausible mechanisms which might explain these associations? Let’s take a closer look at this disease, how it happens and the factors which have been shown to prevent, arrest, reverse or promote it.

Understanding CAD and How Heart Attacks Happen

Before a discussion of how to prevent heart disease from progressing or how to reverse established disease, it is helpful to understand those factors which contribute to its formation. As previously mentioned, this is a disease of accumulation (as are most of our chronic diseases). It can start as early as childhood but takes years to develop into full blown atherosclerosis and ultimately a heart attack. This is why it is difficult to make the connection between what we eat and getting this disease. We don't eat a burger one day and suffer a heart attack the next.

The traditional paradigm of CAD was that it was simply a plumbing problem, that fatty, cholesterol deposits built up in the arteries to the point where it would completely plug up the artery. Many of these deposits were also hardened, hence the term "hardening of the arteries" or atherosclerosis (sclerosis means hard). This is due, in part, to the calcification of the plaque. However, most sudden heart attacks don't occur as a result of these fatty deposits building up in our arteries to the point where it shuts off the blood supply. Most sudden heart attacks are the result of ruptured plaque and endothelial erosion. A smaller fraction of heart attacks do occur as a result of extensive occlusion of coronary arteries by plaque build-up (i.e. it is a plumbing problem).

Plaque Rupture and Endothelial Function

Our arteries are lined with cells called endothelial cells. These cells are critical for maintaining arterial health. If these become damaged and dysfunctional, it sets the stage for the progression of the disease. The good news is that damaged endothelial cells can be replaced by progenitor endothelial cells which are produced in the bone marrow. Dietary factors can inhibit or promote the production of these cells (but more on that later); thus, given the right diet and lifestyle, damaged endothelial cells can be replaced and repaired.

Endothelial cells secrete a gas called nitric oxide that keeps the arteries healthy. Nitric oxide is a potent vasodilator. If our ability to produce nitric oxide becomes compromised, the arteries stiffen and won't expand in response to increased blood flow. When there is excess LDL cholesterol in circulation, LDL can migrate into the wall (or intima) of the artery. The lesion or plaque is preceded by a fatty streak which is an accumulation of lipid-laden cells beneath the endothelial layer. Over time, this build up will create fatty deposits in the artery.[35] Remember this can occur systemically, not just in the arteries to the heart, (i.e. it can occur in the arteries to the lower back and penis, creating a different set of problems). This build up will also often occur in areas where there is "hemodynamic stress", or increased pressure from blood flow, such as where there are bends in the artery. This is one reason why having lower blood pressure is really important.

This fatty core is covered by a delicate cap of smooth-muscle cells and a collagen-rich matrix. Various factors can cause the plaque to rupture which exposes the plaque material to the blood. A thrombus or clot may result and occlude the artery, resulting in a sudden heart attack or stroke, depending on where it gets lodged. Plaque rupture is evident in 60-70 percent of heart attack cases.[36] This rupture and subsequent clot formation can happen very suddenly with little prior warning and in over **40% of cases, the first symptom is death.**

But what causes the plaque to rupture? A number of mechanisms have been proposed.

First, uptake of LDL particles into the arterial wall can trigger an inflammatory response. This inflammation may lead to increased blood levels of inflammatory cytokines and other markers of inflammation which may erode the delicate cap. The most commonly measured marker of inflammation is C-reactive protein (or CRP). However, other markers include interleukin-6, fibrinogen, interleukin-7, and interleukin-8. A moderately elevated CRP may indicate smoldering inflammation and has been shown to be an independent risk factor for CAD.[37]

However, recently researchers at Michigan State proposed a different mechanism, cholesterol crystals. They "hypothesized that soft atheromatous plaque with large deposits become supersaturated with cholesterol that can undergo physical transformation from a liquid to a solid crystal state. The growing crystals can then expand within the confined space of the plaque and damage the fibrous cap."[38] The crystal tips have sharp jagged needles and it can cause the delicate cap to rupture. This idea was confirmed by human autopsies which showed "cholesterol crystals perforating the cap and intimal surface was made in the plaques of patients who died with acute coronary syndrome."[39] In fact this was found to be true of ALL the patients who had died of a heart attack whereas those who had severe plaque buildup, but died of other causes, had no evidence of crystal induced perforation of the plaque.[40]

As the plaque progresses from foam cells to larger plaque, calcium, smooth muscle cells and connective tissue can migrate into the site. This causes the plaque to harden and to stabilize; however it can eventually occlude the artery and shut off blood supply to the heart.

So what do we need to pay attention to, to prevent this from occurring?

After a careful review of the research it is clear that we need to pay attention to a number of factors, a) serum cholesterol levels and specifically oxidized **LDL cholesterol**, b) **endothelial function**, c) certain toxins like **TMAO** and **AGEs**, and d) **inflammation**. Lifestyle and dietary factors play a contributing role in all of these.

Serum Cholesterol

Cholesterol is the most decorated molecule in biology. Over 13 Noble prizes have been awarded to those who have spent the better part of their lives studying it. According to Brown and Goldstein, themselves Noble prize winners, "The potential for errant cholesterol deposition is aggravated by its dangerous tendency to exchange

passively between blood lipoproteins and cell membranes. If cholesterol is to be transported safely in blood its concentration must be kept low, and its tendency to escape from the bloodstream must be controlled."[41] The operative statement here is that levels must be kept low.

Cholesterol is produced in your liver and plays an important role in steroid hormone, bile, and vitamin D production. It is also an essential structural component of cell membranes. However, excess cholesterol in the blood stream for extended periods of time can increase your risk for arterial disease, erectile dysfunction[42], dementia[43], quality of life in later years[44], lower back pain and disc degeneration.[45]

In light of this, serum cholesterol levels are something we need to pay attention to.

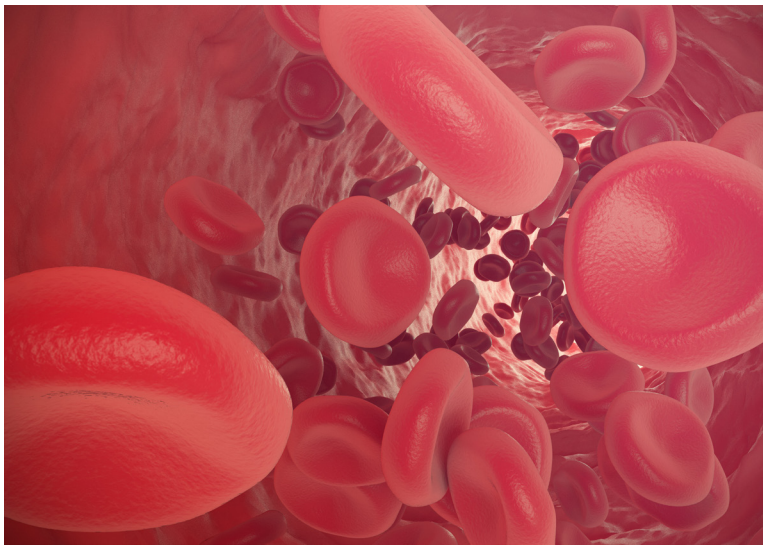
Cholesterol Subfractions

The major classes or subfractions of cholesterol were delineated in the 1950s and 1960s and are: a) very low density lipoprotein **(VLDL)**, b) intermediate density lipoprotein **(IDL)**, c) low density lipoprotein **(LDL)** and d) high density lipoprotein **(HDL)**.

A typical blood test will report total, LDL and HDL cholesterol, and triglycerides. A risk ratio is also reported, which is either the ratio of total cholesterol to HDL or of LDL to HDL cholesterol. Occasionally, VLDL and IDL levels are also reported. After a period of time in circulation, VLDL will become LDL, thus you want to reduce your levels of both LDL and VLDL; otherwise known as the non-HDL cholesterol fraction. LDL is often reported as two values, a calculated LDL and a measured LDL. The measured LDL is the more accurate value.

LDL Cholesterol

LDL is the most abundant cholesterol-carrying lipoprotein in human plasma. It is essentially an oily core of cholesterol that is shielded from the watery plasma by phospholipids. LDL is the delivery mechanism; it is the stuff that plaque is made of.



Recent trials on cholesterol lowering found that the lower the LDL cholesterol, the greater the regression of arterial plaque. By low, they found that getting LDL cholesterol to <80 mg/dL (2.08 mmol/L) showed a reversal of disease in most patients, whereas lowering LDL to 110 mg/dL (2.86 mmol/L) did not. In fact, those who lowered it to only 110 showed a worsening of their atherosclerosis.[46] In another study patients with severe arterial disease were able to reverse their plaque size after lowering total cholesterol levels from an average of 243 (6.36 mmol/L) to 148 (3.88 mmol/L) by switching to a plant-based diet.[47]

Lipid lowering recommendations have changed over the years (and will likely keep changing). Yet original guidelines were only concerned with slowing progression of the disease, not with preventing or reversing it. To quote Dr Roberts, "current cholesterol guidelines have to do only with decreasing atherosclerotic events. They do not concern themselves with preventing atherosclerotic plaques in the first place or reversing existing plaque." He goes on to say that in his view, "in order to prevent

atherosclerotic plaques, serum LDL cholesterol needs to be <70mg/dL (<1.8 mmol), and/or total cholesterol <150 mg/dL (<3.88 mmol/L)."[6] However, LDL levels around 100 are acceptable if you eat a healthy diet, exercise and don't smoke. Thus, the context is important as well.

Dr. Roberts, however, would recommend using statins to do the trick, mostly because he believes that most people would prefer this to changing their diets to that of a "strict vegetarian" (to use his term). Yet it is questionable as to how beneficial lipid lowering drugs actually are. A 2010 meta-analysis which analyzed eleven randomized controlled trials involving 65,229 people found no reduction in all-cause mortality with the use of statins in patients with high cholesterol.[48] Thus not all ways of lowering cholesterol are created equal; likely because this is a multi-faceted disease and just lowering one element, while important, will not confer all the necessary protection.

Another thing to keep in mind is that risk is a function of both elevated LDL levels, numerous other lifestyle factors (like smoking, obesity, fitness, diet, stress) and the length of time that LDL cholesterol is elevated. Having years of elevated LDL is what poses the greatest risk. Thus, dietary strategies to reduce LDL should be implemented as young as possible, even in childhood. Dietary factors also impact the oxidation of LDL. Oxidized LDL is actually the most dangerous and is what primarily gets incorporated into vulnerable plaque. Factors like smoking, low intakes of antioxidant rich foods, obesity and eating cholesterol laden foods like meat and dairy will promote oxidation of LDL particles. **This is why two people with the same LDL level can be at different risk for heart disease.**

How Low Can You Go?

Newborns have LDL levels of around 30. Humans on low-fat, plant based diets are in the 50-80 range. But how much do we really need? Those on the opposite end of the genetic spectrum, i.e. those that have familial hypobetalipoproteinemia or lifelong genetically low levels of LDL (<15) were found to have normal growth and development and an increased lifespan.[49] Various researchers and experts on cholesterol have found that LDL levels as low as 10-20 mg/dL are perfectly healthy. There are no upsides to having more LDL than you need.

Thus, all things being equal, getting LDL as low as possible is important, but via dietary means, not drugs. According to Dr. Esselstyn, who has successfully reversed CAD in his patients via diet, "lowering lipid levels also lowers concentrations of harmful foam cells within plaques and reduces the quantity of proteolytic enzymes." As a result, plaques may shrink, their caps stabilize and they become much less likely to rupture."[50]

Eating a healthy diet will confer greater protection as it will also help with weight loss, improve glucose control, lower inflammation and blood pressure, prevent oxidation of the LDL particles and protect the endothelium.

LDL Subfractions: Large vs. Small Particle

Recently it has become popular to measure subfractions of LDL in an effort to further define one's risk. These subfractions are generally referred to as small dense or large particle LDL. Large LDLs carry more cholesterol (which is why they are large). The cholesterol content of LDL can thus vary as much as 2-fold depending on its size.

Another way to measure LDL is to measure apoB (the protein on the surface of the LDL molecule) which represents the number of LDL and VLDL particles in the blood stream. The association of apoB to risk is actually greater than it is for LDL cholesterol, likely because it better reflects the total number of LDL particles.

Are small dense LDL particles more atherogenic?

It has been suggested that it is only the small dense LDL that are problematic while the large LDLs are benign, i.e. they are too large to get into the arterial wall. However, this is not the case. Particles as large as 70 nm can enter the arterial wall. The largest LDL particles are around 27 nanometers (nm), well below the 70 nm. Thus both small and large LDL particles are atherogenic. To quote one paper, "incorrect conclusions may have been drawn regarding the potential importance of certain "novel" risk factors....The former observation led to the belief that small LDL particles are inherently more atherogenic than large ones, a conclusion not supported by recent analyses."[51] In a large scale women's study, elevations of large LDL particles increased risk for CVD by 44% while the small particle LDL increased risk by 63%.[52] For men, risk for CVD was increased 44% with small LDL and 31% with large LDL.[53] All this to say that identifying LDL particle size does not appreciably improve risk prediction above and beyond what total LDL cholesterol does.[54] Both types of LDL phenotypes (large and small alike) are atherogenic and are associated with increased risk.[55]

Thus the number that you need to pay the most attention to is the LDL level and/or the non-HDL portion. If it is elevated; all things being equal, you are at greater risk of arterial disease.

HDL-Cholesterol (HDL-C)

HDL is usually referred to as the "good" cholesterol as it is involved in reverse transport, i.e. it helps transport cholesterol back to the liver. Think of it as being the garbage men; if you have more garbage, you need more garbage men. It can also play a role in reducing inflammation, preventing and correcting endothelial dysfunction, and promoting vasorelaxation. In addition HDL affects coagulation, fibrynolisis, and platelet adhesion and can exert antioxidant activity.[56] Thus HDL is protective in a number of ways.

Does increased HDL mean lower risk? Not always—it depends

There is some debate as to how protective elevated HDL values really are. In cross sectional population studies, higher HDL is correlated with lower risk, while in Mendelian randomization studies (i.e. studies where they look at people with genetically high HDL) higher levels were not associated with lower risk, while elevated LDL values did confer increased risk.[57] A meta-analysis of 108 randomized controlled trials found that while lowering LDL cholesterol significantly decreased the risk of coronary disease and all-cause mortality, modifying HDL had little appreciable benefit.[58] The results showed a 7% relative risk reduction in coronary heart disease events for every 10 mg/dl reduction in LDL cholesterol.

Of interest is that countries with the highest levels of HDL also have higher rates of heart disease.

Given the positive, arterial health promoting functions of HDL, why is HDL sometimes protective but sometimes not?

We are finding that the blood level of HDL cholesterol doesn't necessarily correspond with its capacity to perform reverse cholesterol transport or anti-inflammatory functions.[59] It is not so much the amount of HDL that is important, but rather its ability to exert its protective benefits, i.e. not all HDL is created equal, the HDL you do have needs to be effective.[60] A recent (2014) study highlighted the fact that HDL can be functionally impaired due to oxidative modification. They found that, similar to oxidized LDL (which promotes atherogenesis), oxidized HDL also induced significant oxidative stress, and release of pro-inflammatory cytokines, albeit it was somewhat less potent than LDL.[61] Oxidized HDL is essentially HDL gone bad, and, as it turns out, might also be bad for your arteries.

Research suggests that dietary factors can play a role in how effective (or protective) your HDL is. For example, the typical western diet may oxidatively injure apo-A1 (the major protein of the HDL molecule) which renders HDL incapable of anti-inflammatory functions. When HDL becomes oxidized it becomes a pro-inflammatory agent, which along with oxidized LDL can promote injury.[62] People eating antioxidant rich diets (i.e. plants) may reduce apo-A1 oxidation thus protecting HDL and preserving its protective functions.[63]

What this suggests is that higher levels of HDL are only protective when accompanied by an antioxidant rich and anti-inflammatory diet (i.e. a diet that is the opposite of a western diet). Those with high levels of HDL who consume a western diet might actually have an increased risk as compared to someone who has a moderate to low HDL and who eats a healthy diet.

Thus, the focus should be to lower LDL via dietary means if possible, eat an anti-oxidant rich diet (one that is rich in colorful fruits and vegetables, legumes, seeds and intact grains) and to increase your HDL via non-pharmacological means, such as regular exercise.

So what is the best diet for lowering cholesterol?

How to Lower LDL via Dietary Means

Atherosclerosis (or plaque) is easily produced in herbivores by feeding them a diet that contains cholesterol and saturated fat (and, in some studies animal protein). In fact atherosclerosis is one of the easiest diseases to produce experimentally and has been produced in hundreds of different species including mammalian[64], avian and fish species[65] as well as herbivores[66], omnivores and even carnivores.[67] However, the animals most susceptible to atherosclerosis are herbivores. When herbivores are fed a high fat/cholesterol diet the disease doesn't just happen in some of the animals; it happens in ALL of the animals. The atherosclerotic lesions induced by cholesterol feeding in animals have been shown to closely resemble the disease in humans. Thus, the arteries of virtually every animal species are susceptible to this disease if the blood cholesterol level can be raised high enough for a long enough period of time.

Population studies show markedly strong positive correlations of serum cholesterol levels to saturated fat intake and levels of heart disease. One study comparing diets and cholesterol levels in 20 different countries found that as saturated fat intake went up so did serum total cholesterol.[68] Those who ate low-fat, high carbohydrate diets had the lowest levels of cholesterol. However, population studies have had mixed results when it comes to showing a strong association of saturated fat intake and arterial disease. This has led to intense debate as to the role of saturated fat in elevating cholesterol and causing arterial disease (more on this to come).

Metabolic ward feeding studies make a stronger case, however. These are studies where people are fed various diets under very controlled conditions. Unequivocally such studies have shown increases in LDL cholesterol with intakes of certain types of saturated fats. One study compared intakes of butter fat, beef fat, cocoa butter and olive oil on their ability to raise cholesterol. Butter fat was found to be the worst, followed by beef fat, cocoa butter and olive oil.[69] A meta-analysis of almost 395 metabolic ward feeding studies published in the British Medical Journal concluded that "In typical British diets replacing 60% of saturated fats by other fats or carbohydrates and avoiding 60% of dietary cholesterol would reduce blood total cholesterol by about 0.8 mmol/l, with four fifths of this reduction being in low density lipoprotein cholesterol."[70] Incidentally, 0.8 mmol/l is around 28 mg/dl.

Foods to Eat Less of or Eliminate

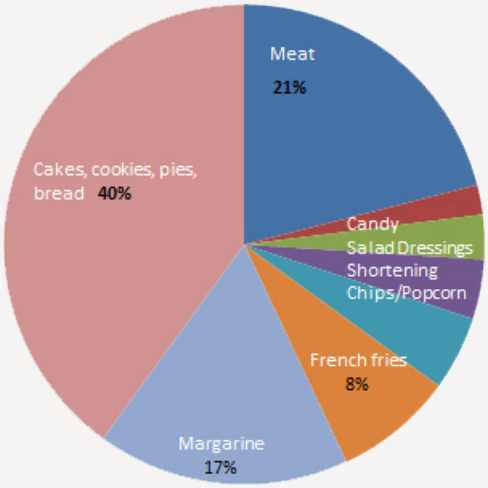
What these studies show is that if you want to lower your LDL cholesterol, you need to severely reduce or perhaps even eliminate animal foods (dairy and meat), foods that contain trans-fats (dairy, beef, processed foods) and foods that are Calorie Rich And Processed (CRAP foods). Why?

These foods are the biggest sources of saturated fat, cholesterol, trans fats, and animal protein (yes animal protein, independent of fat, is a contributing factor to heart disease and will also increase cholesterol levels). Additionally, these foods also contain no fiber and few nutrients (like antioxidants).

The National Academy of Sciences (NAS), in a recent statement, concluded that they could not set upper limits for trans-fat, saturated fat or cholesterol intake, as any intakes of these fats above zero increases LDL.[71,72] In a

report published by the WHO on diet and chronic disease, they stated that "The relationship between dietary fats and CVD, especially coronary heart disease, has been extensively investigated, with strong and consistent associations merging from a wide body of evidence accrued from animal experiments, as well as observational studies, clinical trials and metabolic studies conducted in diverse human populations."[73]

Sources of Trans Fat, Saturated Fat and Cholesterol



USFDA Department of Health and Human Services

	SATURATED FAT	CHOLESTEROL
1.	Cheese	Eggs, egg dishes
2.	Dairy desserts (ice cream)	Chicken
3.	Pizza	Beef
4.	Chicken & mixed dishes	Burgers
5.	Desserts (cakes, cookies, donuts)	Cheese
6.	Sausage, franks, bacon, ribs	Sausage, franks, bacon, ribs
7.	Reduced fat milk	Fish, fish dishes
8.	Burgers	Desserts (cakes, cookies, donuts)
9.	Mexican dishes	Dairy desserts
10.	Pasta dishes	Cheesy pasta dishes

Bonus: How Saturated Fats Increase Serum Cholesterol

Saturated fats are used as fuel as well as in cell membranes. Problems arise when there are too many saturated fats in the cell membranes. When this occurs, cell membranes become hardened and less flexible. This hardening restricts the necessary removal of cholesterol from the blood, likely because there are fewer receptors for cholesterol in over-saturated cell membranes restricting the movement of cholesterol from the blood into the cells. Saturated fats from our diet can also increase our own production of saturated fatty acids, which further contributes to excess blood cholesterol.[74] Suppression of liver LDL receptors also occurs when saturated fat intake is high. This limits uptake of LDL by the liver and further elevates serum LDL.[75]

Saturated fats also increase secretion of VLDL, which as previously mentioned eventually becomes LDL cholesterol. Saturated fat increases the **persistence** of VLDL and LDL in the bloodstream (i.e. they stay in circulation for longer periods of time). The longer LDL stays in circulation, the more likely it will become oxidized. More oxidized LDL results in more damage to the endothelium which in turn can increase risk of plaque formation in the artery.[76]

But not all saturated fat is created equal. Of all the saturated fatty acids, lauric acid, myristic acid and palmitic acid are known to raise blood cholesterol. Which foods have these three types of fatty acids? Beef, cheese, chicken and hamburgers are major contributors of palmitic and myristic acid. Dairy products like butter and cheese are the major contributors of myristic acid and coconut and palm kernel oil (often found in processed foods) are major contributors of lauric acid.[77]

Saturated Fats: Not So Bad After All?

But what about recent media reports of studies suggesting that saturated fats (and cholesterol) aren't the bad fats that we once thought they were? This has been a disturbing recent trend. Two meta-analyses of population based studies were recently published suggesting that saturated fat intake is not associated with increased risk of heart disease. Given that this cast doubt on conventional wisdom made for sensational headlines and no doubt increased magazine sales. These make for popular reading as people like to hear good news about their bad habits. Additionally the leap has been made that saturated fat is now actually good for you and can be eaten with impunity. The studies never actually came to that conclusion (that was all hype); all they suggested is that saturated fat maybe isn't as bad as once believed.

The meta-analysis in question were published by Siri-Tarino and Chowdhury.[78,79] A meta-analysis is a useful tool when analyzing findings on a particular question or research topic. It combines the results of different studies by different researchers. Yet, the conclusions of the meta-analysis are only as valid as the papers included in the analysis. The papers used in this analysis were a **weak** subset of thousands of studies which have looked at the relationship between saturated fat intake, diet and CD risk. Yet, from the author's conclusion and the media commentary, a naive public is led to believe that this paper provided a definitive, all-encompassing review of the research. Alas, this was not the case.

For a more complete review of the papers I refer you to my website: www.sharonlarsen.org/Fit_to_Lead/Fit_to_Lead_News and the article Saturated Fat: Friend or Foe? But here are some highlights as to why these studies were the exception to the rule and how they were seriously flawed in their analysis.

1 **Over adjustment.** A number of the studies used in both the Chowdhury and Siri-Tarino analysis adjusted for serum total and LDL cholesterol levels. As saturated fat has been shown in both human and animal feeding studies to increase serum cholesterol, this is problematic and would serve to dilute the correlation of saturated fat intake and CD risk. This is a bit like looking at the impact of obesity on the risk of diabetes and then adjusting for waist circumference.

2 **Lack of meaningful differences between dietary groups.** Many of the studies used in the analysis were done on western populations where diets tend to be relatively homogeneous, i.e. there were only small differences between those eating a diet "low" in saturated fat vs. those eating a diet high in saturated fat. It is virtually impossible to have a "low" saturated fat group in a typical western population as average intakes are high to begin with. This is akin to looking at mortality rates of cars crashing at 80 mph vs 100 mph. The outcomes of crashing at either of those speeds probably won't be that different, you will likely die. Without meaningful differences in saturated fat content, there is less of a chance of finding any relationship to CD risk.

3 **Measures of food intake.** A number of the studies (if not most) used a 24 hr. dietary recall or a food frequency questionnaire to determine saturated fat intake. They then correlated this with disease outcomes 5-20 years later without further analyzing the diets. The reliability of this method is questionable for obvious reasons. To quote one researcher in a letter to the editor, "Such methods [24 hr. dietary recalls] cannot reliably rank individuals by their long-term intake, especially within populations with a uniformly high saturated fat intake."[80] In the massive Cornell-China study (where saturated fat intake, and in particular animal-sourced fat, was strongly associated with increased risk of CD) researchers went into people's homes and recorded dietary habits, weighed food and took food samples.[81] This is considered the gold standard when doing diet-disease research at the population level. Additionally, many of these studies were started in the 80s, where there was a subsequent reduction in smoking rates and statin drugs were introduced, further diluting any associations.

There are other reasons which make these studies suspect, not the least of which is the fact that a number of the authors received funding from the National Dairy Association, the National Beef Council and Atkins Nutritionals. The dairy and beef industries are losing market share and have taken a page from the tobacco industry: Pay scientists to publish studies which sew doubt that their products are not that harmful. They don't pretend to try to prove that saturated fat is healthful, just create a measure of doubt and one can see how successful this approach can be.

As a side note: I am not a fan of this reductionist approach to nutrition, i.e. blaming an isolated element of food for our poor health outcomes. The primary reason being that we eat foods, not isolated elements of those foods, how the fat comes packaged is also important. Examining the types of foods consumed in relation to disease outcomes is a better approach. Secondly, focusing just on saturated fat detracts from the real problem and that is our high consumption of animal based foods.[82] *This sole focus has led to confusion and unintended consequences, not the least of which is that we have now created "low-fat" foods (think skim milk or lean meat) and replaced butter with olive oil. This has in no way solved the problem. Reductionism is only helpful when trying to tease out mechanisms to help explain what it is about meat and dairy that is potentially harmful.*

Trans Fat and Cholesterol

Trans fats start out as normal fatty acids and then are altered, either by hydrogenation or by bacteria in the guts of cows, sheep and goats. Trans fats can also be created during the process of extracting vegetable oils even without hydrogenation. These altered fats adversely affect health and can increase LDL cholesterol more than saturated fatty acids (although our diets contain about 1/10th the amount of trans fat). They can also raise triglyceride levels and lower HDL levels.[83]

Thus dietary sources of trans fat are oils, the hydrogenation of oils and meat and dairy products from ruminant animals. A recent prospective study found that trans fats irrespective of source, (i.e. from meat, dairy or hydrogenated oils) increases risk for CVD.[84]

Dietary Cholesterol: Eggs, Not What They Are Cracked Up to Be

Eggs contribute the most dietary cholesterol in western diets. Much of the debate has been around whether egg yolk consumption increases cholesterol levels. When eggs are eaten by a population who already eat a high cholesterol diet, serum cholesterol levels don't go up. However, adding 400 mg of cholesterol to a cholesterol-free diet will raise serum total cholesterol about 20-30 mg/dl. Higher amounts of dietary cholesterol do not appear to further increase serum cholesterol.[85]

There are also genetic differences in how people's serum cholesterol responds to intakes of dietary cholesterol. In one study, 6 egg yolks (1500 mg of cholesterol) were added to the diets of 37 healthy medical students. Mean serum cholesterol rose by an average of 29 mg/100 ml. However, there was a wide range of individual responses; for some their cholesterol dropped by 6 points and for others it increased by 75 points. Subsequently, they repeated the experiment, but this time only added 3 eggs to the diet. Average serum cholesterol levels rose by about the same amount, confirming the idea that beyond a certain intake, serum cholesterol levels do not continue to increase.[86]

Researchers in a different study had healthy subjects consume 2 eggs per day with meals for 3 weeks. This resulted in minor elevations in glucose levels and an 11% increase in LDL levels. HDL also decreased by 11%. However, more concerning was that the susceptibility of LDL to oxidation was increased by 34%. They concluded that egg consumption, in addition to its hypercholesterolemic effect, increased both plasma and LDL oxidation which has been "shown to enhance the progression of atherosclerosis."[87]

A 2012 study found that carotid plaque increased exponentially with years of egg consumption. Egg consumption remained significant after adjusting for other coronary risk factors. They reported that the effect size of eating eggs to be approximately 2/3 that of smoking.[88] The bottom line is that egg yolk consumption can increase our risk of disease regardless of how it impacts our cholesterol levels.

Dietary Cholesterol Increases Oxidization of LDL: The Bacon and Egg Effect

Of greater concern is that in the hours after you eat it, "cholesterol increases the susceptibility of LDL-C to oxidation (the most dangerous type of LDL), vascular inflammation, oxidative stress, and postprandial heyperlipidemia and potentiates the harmful effects of saturated fat, impairs endothelial function and increases cardiovascular events".[89] This aforementioned duo of saturated fat and cholesterol is known as the bacon and egg effect.

What About Coffee?

Diterpenes found in coffee may elevate cholesterol.

Caffeine itself has not been proven to increase risk for CAD. However, early studies on coffee showed mixed results; some studies showed increased risk while others showed no risk. Part of the reason for this could be explained by the different methods of preparing coffee. Two types of fats which are present in coffee, cafestol and kahweol have been shown to elevate LDL.[90] Cafestol may act on the liver to raise LDL but it hasn't been completely elucidated as to how. Even though amounts in coffee are low, these appear to be potent cholesterol raising substances. Filtering coffee results in lower levels of diterpenes, while modest levels are found in espresso and the highest levels in boiled coffees, like French press or Turkish coffee.[91] If you are struggling to lower your LDL-C or are at high risk of CAD, it would be best to drink filtered coffee or better yet, drink green tea, which has been shown to help lower LDL-C!

However, the link between coffee consumption was recently examined relative to those with genetic differences in their ability to metabolize caffeine. Some people are carriers of a CYP1A2*1F gene and are slow caffeine metabolizers, whereas others have the CYP1A2*1A gene and are rapid metabolizers. What they found was that the slow metabolizers were at greater risk of myocardial infarction than the fast metabolizers. Carriers of the CYP1A2*1F have impaired ability to metabolize caffeine, thus the researchers concluded that it was likely the caffeine that was the problem, not the coffee per se.[92]

Foods to Eat More of

While reducing your intake of trans, saturated and cholesterol laden foods, such as processed foods, (including oils), eggs, meat and dairy, is a good start, there are also foods you should **eat more of** which can help to lower cholesterol. These are: a) foods high in **viscous fiber**, b) foods that contain **phytosterols** and c) foods **low in complete proteins** (i.e. plant protein sources vs. animal protein sources).

Fiber works its magic in two ways; 1) it binds to the cholesterol in our gut (released by the bile duct) preventing it from being reabsorbed and 2) when our gut bacteria work to digest the fiber they produce something called **propionate** which inhibits cholesterol and fatty acid synthesis (it is also hypophagic—meaning it promotes satiation).[93]

Good sources of **soluble fiber** are oats, barley, fruits and all legumes (or pulses).

A good recommendation is to eat one or more cups of beans every day. Beans are packed with resistant-starch (in addition to soluble fiber) which also helps to lower cholesterol. A pooled analysis of 10 trials found that regular consumption of beans significantly reduced total and LDL cholesterol.[94] Another study found that people who ate beans 4 or more times per week reduced their risk of heart disease by 22% compared to those who ate beans less than once per week.[95]

In addition to eating beans, **try to eat one ounce or a handful of nuts and seeds per day**. These contain phytosterols which are the plant-kingdom's version of cholesterol. Basically they are "cholesterol look-alikes" and they compete with real cholesterol for absorption in the gut.[96] Thus the body will absorb the plant kind (which is good) and not the animal kind (you ending up pooping more of the bad stuff down the toilet).

A 2009 meta-analysis of forty human trials found that plant sterol supplements can safely reduce LDL levels by up to 15% (although results can vary depending on your genetic makeup).[97] If you have difficulty getting your LDL cholesterol levels into the favorable range, then plant sterol supplements might be a safe and effective choice. For the most part, however, it is best to get your sterols from foods. Foods rich in phytosterols are nuts (almonds, peanuts and walnuts), seeds (sesame, flax, pistachios, sunflower seeds, pumpkin seeds), wheat germ, split peas, and kidney beans. This may explain why eating a handful of nuts and/or seeds a day (or even 3x weekly) may lower mortality from heart disease. Nut consumption is also associated with reduced risk of diabetes, certain cancers and longer life.[98]

Other specific foods shown to lower cholesterol are dried apples[99], kiwi fruit[100], chia and flax seeds[101], green tea[102] and legumes such as soy beans and lentils (likely due to their fiber content). Substituting soy protein (or other plant protein sources) for animal protein seems to be particularly effective for those who have higher levels of cholesterol.[103]

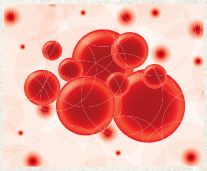
Replace animal protein with plant proteins.

Plant proteins inhibit liver cholesterol synthesis and also increase the liver LDL receptor uptake of cholesterol.[104] Plant proteins are higher in non-essential amino acids than animal-derived food proteins, and as a result preferentially favor glucagon production. Acting on liver cells, glucagon promotes mechanisms that down-regulate cholesterol promoting enzymes and cholesterol synthesis, while up-regulating liver LDL receptors (all this to say that plant proteins may help to reduce cholesterol production by the liver).[105] In a 2016 paper, it was proposed that plant proteins stimulate increased activity

of the enzyme GCN2, which boosts the liver's production of FGF21, a factor "which favorably affects serum lipids and metabolic syndrome" and which reduces cholesterol synthesis.[106]

Human studies show a strong association between animal protein consumption, heart disease and risk of premature mortality, independent of fat content. In a study on healthy middle-aged men, there was an increase in risk of ischemic heart disease with higher intakes of animal protein but no increase in risk for plant protein.[107] A large scale study on Swedish women found a 60 percent increased risk of heart disease in women adhering to a low-carb, high-protein diet. The results showed a gradual and consistent increased risk as the consumption of animal protein went up.[108] A 2014 study, which followed patients who had suffered from a myocardial infarction, found that a low-carb diet which was high in animal protein increased subsequent mortality rates whereas a similar low-carb diet high in plant protein was not associated with increased risk.[109] A very recent study (released August of 2016) showed that among two very large American study populations, those that consumed the most animal protein compared to plant protein had a higher risk of premature mortality, particularly from cardiovascular disease. They found that replacing 3% of calories from animal protein (red meat) with 3% plant protein was linked to a 34% lower risk of death. This was after controlling for different types of fat, total energy intake, glycemic index, whole grain intake, fiber intake, smoking and a host of other lifestyle factors.[110]

In combination, eating more of these foods can be powerful. A recent cross-over study compared a low-fat diet to which these foods were added (i.e. foods high in fiber and phytosterols and where soy protein replaced animal protein) to a low-fat diet where just a statin drug was added (and not the foods). They found that the diet produced similar reductions in LDL cholesterol (about a 30% reduction, on average) as did the drug.[111] Another study by the same author compared a very-high-fiber (55g/1,000 kcal) diet to a low-fat contemporary therapeutic diet. The beauty of this study was that it was designed to be weight maintaining. This is important as cholesterol will go down during periods of caloric restriction. The average drop in LDL on the high-fiber diet was 33%.[112]



Endothelial Function

As mentioned previously, the key to healthy arteries are the endothelial cells which line all of our blood vessels and which secrete **Nitric Oxide (NO)**. NO was the molecule of the year in 1992 and three US scientists were awarded the Nobel Prize for their work on the role of NO in arterial health.[113]

NO has a number of important functions. It is a) a potent vasodilator, i.e. it opens up the arteries in response to increased blood flow, b) it keeps the arteries from getting sticky and c) it inhibits the production of foam cells (an early step in the formation of plaque). Thus when these cells become dysfunctional and can no longer produce NO, this sets the stage for plaque deposition.

To quote one paper published in the European Heart Journal, "evidence suggests that endothelial dysfunction is on the causal pathway for both disease evolution and destabilization of established plaques".[114] A 2013 review article said this, "Endothelial dysfunction is the initial step in the pathogenesis of PAD [peripheral arterial disease], cardiovascular diseases, stroke, chronic kidney failure, cancer, and infectious diseases."[115]

Thus, it is important to pay attention to those factors which are damaging to endothelial cells independent of their ability to elevate LDL cholesterol. For example, the toxins in cigarette smoke can damage the endothelium while not elevating LDL cholesterol.[116] Smoking one cigarette, for example, has been shown to significantly impair and reduce FMD.[117] Even second hand smoke can have a negative effect. This is one reason why smoking, while not a cause of heart disease per se, will accelerate its progression.

One way that researchers study factors that affect endothelial health is to measure something called flow-mediated dilation (FMD) or the ability of the vessel to dilate in response to increased blood flow. Reduced FMD is evidence of endothelial dysfunction. A recent meta-analysis of observational studies found that a 1% decrease in FMD is associated with a 13% increase in the risk of future cardiovascular events.[118]

Diet can have a significant impact on endothelial function and FMD—both positively and negatively.

Negative Factors

Researchers in Singapore had people eat a high fat meal from a popular fast food eatery consisting of eggs, sausages and hash brown patties.[119] They found marked decreases in FMD in response to this meal (2-6 hours later), i.e. dilation went from 6% down to -2.8%. Following the isocaloric low-fat control meal (frosted cornflakes) there was no decrease. Fat in the diet, and in particular, saturated fat, trans fat and fats that have been re-used in cooking, are the worst offenders when it comes to reduced FMD.[120]

Vogel et al. reported that even a single meal high in saturated fat may be associated with a transient impairment of vascular function. In the study, flow-mediated dilation was impaired by 50% 4 hours after the consumption of a high-fat meal.[121] Combine a stressful event (stress will also impair endothelial function) and a high-fat meal and the effect is heightened even more.[122]

A 2011 study compared the effects of a low-fat and high-fat diet on FMD after 6 weeks in matched obese individuals. This was a well-controlled study in that meals were provided to participants. Weight loss can improve FMD, so both groups reduced caloric intake by 25% in order to lose weight. Thus both groups lost weight, but total weight loss was slightly greater in the high-fat group. FMD was found to improve in the low-fat group and was impaired in the high-fat group. Thus while both groups lost weight, it was only the low-fat group that showed improved endothelial function.[123] A subsequent pooled analysis of eight studies looking at low-fat vs high-fat diets also found significant impairment of FMD for those eating high-fat diets.[124]

Oils, like olive oil, traditionally believed to be "healthy" have also been shown to have adverse effects on the endothelium.[125] However, if you add olive oil to a salad, the negative effects are mitigated. This is likely due to the antioxidants in the salad. Fats rich in omega-3 fatty acids, such as canola oil, do not appear to impair endothelial function as much.[126] Bear in mind, however, that oils, like white flour, are a processed food and are nutrient poor and very calorie dense, so there really is no such thing as "healthy" oil.

However, fish oil, in spite of having omega-3 fatty acids, has not been found to improve endothelial function.[127] In fact, one study found that women eating the most fish (more than 2x per week) had significantly impaired endothelial function compared to those who never or only rarely ate fish.[128]

High salt intake has also been shown to impair endothelial function. Take people off of their usual salt diet and put them on a low-salt diet and their endothelial function improves.[129,130] This is true for those whose blood pressure is unresponsive to higher salt intakes.[131] Dietary sodium intake has even been shown to impair microvascular function, or function of the tiny vessels.[132] Why? Salt can depress the activity of superoxide dismutase, a powerful antioxidant that protects our endothelium (among other things) from oxidative damage helping to preserve its function.[127] Potassium, found in fruits and vegetables, on the other hand, helps to soften arteries by increasing nitric oxide release which helps our arteries relax.[133]

Positive Factors

Foods that are rich in antioxidants have been shown to improve endothelial function. Italian researchers put this to the test when they switched people to an isocaloric antioxidant rich diet. FMD increased significantly in response to the change (in fact it doubled).[134] The negative effects of olive oil, for example, can be mitigated when eaten with an antioxidant rich salad.

Other antioxidant rich foods, like walnuts, berries and cocoa powder, have also been shown to positively affect endothelial function.[135,136] (Note: Some research suggests that adding dairy to antioxidant rich foods can interfere with the absorption of antioxidants.[137] This may be why adding milk to these foods negates some of the benefits)

Vegetarian diets have indeed been shown to "have a direct beneficial effect on vascular endothelial and smooth muscle function...."[138] This study found that vegetarian arteries had FMDs that were four times better than matched omnivores. These results were independent of non-dietary risk factors like smoking, diabetes, hypertension, aging or even high cholesterol. And the longer they ate vegetarian, the better their endothelial function.

This study was cross sectional in nature, thus an intervention trial was needed to help establish cause and effect. Such a trial was done by Dr. Dean Ornish who did indeed show improved endothelial function when patients switched to a plant-based diet that contained no meat and very limited dairy.[139]

Exercise can also improve endothelial function. This was shown to be true for regular moderate physical activity which "promotes an antioxidant state and preserves endothelial function".[140]

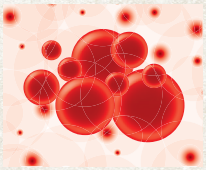
In population studies, fruit and vegetable intake has been shown to decrease risk for coronary heart disease. One study found that for every 1-serving per day increase in intake of fruits or vegetables there was a 4% lower risk for coronary heart disease. Green leafy vegetables and vitamin C-rich fruits and vegetables contributed most to the protective effect.[141] In another study, cruciferous vegetables showed a dose-dependent inverse response to risk of total mortality and cardiovascular disease mortality.[142]

Progenitor Cells – Restoring Endothelial Function

Endothelial progenitor cells are produced in the bone marrow. These are the endothelial precursor cells which replenish and replace old and damaged endothelial cells. Herein lies the good news, that given the right dietary conditions, we can heal damaged cells and arteries. However, a poor diet can also inhibit production of these progenitor cells.

Foo et. al. fed three diets to three groups of mice. The control diet was a low fat healthy chow. The other was a diet based on the typical western diet, moderate amounts of fat, protein and carbs. The third was a high protein, low carb, moderate fat diet (the fat content was the same as for the western style diet). Upon autopsy, those on the low fat healthy chow had no plaque and progenitor production was normal. Those on the western diet had moderate amounts of plaque disposition. The group on the high protein diet had almost double the plaque of the western diet and they had compromised progenitor cell production.[143] Incidently the protein used in the study was casein, the predominant protein found in dairy. Compromised progenitor cell production is significant as it can promote atherosclerosis by compromising the ability to restore endothelial function.

In a different study, researchers fed antioxidant rich vegetables (the food was delivered to them) to a group of Okinawan women (women who already ate a relatively healthy diet). Compared to a matched control group, endothelial progenitor cells increased significantly in those eating the vegetables.[144]



Trimethylamine Oxide (TMAO)

Two additional factors that can cause harm to our arteries are **choline** and **carnitine**. In 2013 researchers reported that certain gut bacteria metabolize carnitine and choline into a toxic substance called trimethylamine (or TMA).[145] TMA gets oxidized in the liver to TMAO which then circulates throughout the blood stream. TMAO is nasty stuff in that it contributes to the build-up of cholesterol and inflammatory cells in arterial plaque, increasing risk for heart attack, stroke and death.[146] It appears to do this even if serum LDL and total cholesterol levels are relatively low. TMAO can also inhibit the reverse transport activities of HDL.

Where do carnitine and choline come from? We make carnitine in our livers, as do other animals. Thus when we eat animals, which also have livers, we are getting a good dose of the stuff. It is especially concentrated in red meat, so when you eat a steak, TMAO levels shoot up. However, if you feed vegans a steak, no TMA is produced, likely due to the fact that the gut flora of vegans is quite different than the meat eaters. Choline on the other hand is found mostly in **eggs, dairy, poultry, fish and shell fish**. Thus these foods are also culprits in TMAO production. Carnitine supplements (found in some energy drinks) and lecithin supplements (source of choline) may also foster TMAO and TMA producing bacteria in the gut, but it appears that it is worse when sourced from meat. Incidentally, high egg consumption and high dietary choline consumption has also been strongly linked to greater risk of prostate and other cancers.[147]

Inflammation

Animal and processed foods promote inflammation in a number of ways. First animal foods contain pre-formed arachidonic acid (AA). AA results in the production of pro-inflammatory eicosanoids, which promotes inflammation.[148] Omega-6 fatty acids, found in high amounts in most processed oils, are converted to AA and thus, these oils can promote inflammation as well. Processed oils have also been shown to hurt endothelial (or arterial function), as previously mentioned.[149,150,151,152]

Secondly, animal foods promote inflammation via endotoxins. Investigators have discovered that after a meaty meal one's bloodstream becomes soiled with bacterial toxins known as endotoxins. Endotoxins come from bacteria in the meat. These get absorbed into the blood stream inducing an inflammatory response.

British researchers looked at 27 foodstuffs and found that foods like pork, poultry, dairy, cheese (in particular hard cheeses) and eggs produce a significant bacterial load and subsequent increases in inflammatory markers within hours of consuming these foods.[153] Among the more infamous inflammatory markers measured was tumor necrosis factor (TNF). TNF is known for the role it plays in autoimmune attacks like inflammatory bowel disease (there are even TNF blocking drugs on the market).[154] The inflammatory response held even after boiling the meat for two hours or dipping it in an acid bath. The concern is that these foods could **"promote transient, mild, systemic inflammatory episodes that predispose subjects to the development of atherosclerosis and insulin resistance".** Saturated fats also play a role as they may assist endotoxins to cross the gastrointestinal wall.[155]

Carbohydrates, and especially wheat, have often been blamed as playing a causative role in producing inflammation. However, when put to the test, people fed a high-fat meal and then fed a high-carb meal, show significantly more postprandial inflammation following the high-fat meal, while the high carb meal showed much less inflammation.[156,157] A recent study which compared consumption of red meat and whole-grain bread found that whole-grain bread was related to lower levels of inflammatory markers while red meat was associated with higher markers.[158]

If you want to reduce inflammation, eat a plant-based diet. To quote one German researcher, "...there are also data from human intervention studies suggesting an anti-inflammatory potential of these plant foods [fruit, vegetables and whole wheat]...primarily carotenoids and flavonoids seem to modulate inflammatory as well as immunological processes. By means of anti-inflammatory activities a plant-based diet may contribute to the lower risk of cardiovascular diseases and cancer."[159]

Specific plant foods that have been shown to reduce inflammation are nuts[160], berries[161], mushrooms[162], beans[163], purple potatoes[164], and spices such as cloves, ginger, rosemary and turmeric.[165] This may be why studies done on those eating a vegetarian or vegan diet show significantly less inflammation in their bodies than omnivores.[166]

The question is does inflammation cause heart disease, as some would like to suggest. Having chronic levels of inflammation is not a good thing for a number of reasons and probably doesn't help the process of healing and reviving our arteries. But to say it plays a causative role would be stretch. To quote Dr. Roberts, "I am unconvinced that inflammation or infection actually play a role in the production of atherosclerotic plaques."[167] Inflammation occurs as a result of injury, in this case injury to our arteries. So could just be a marker for arterial damage. However, it could play a role in degrading the cap which covers arterial fatty deposits which in turn can provoke formation of a thrombus and a sudden heart attack. Chronic inflammation is also not good for joint tissues and it hurts brain function. So it is something to pay attention to.

Homocysteine

A short discussion about homocysteine is worth, well, discussing. Homocysteine (tHcy)[ii] is a sulfur-containing metabolite of methionine. It can be measured in a blood test; and, higher levels (>10uml/L) appear to damage the endothelium and are associated with higher risk of CAD.[168] Vitamin B6, folate and B12 are all involved in pathways that metabolize and lower homocysteine. Elevated plasma tHcy concentrations are found in those who eat **methionine rich-diets**, whose diets are low in B6 and B12, males, smokers, those with impaired renal function and in those with genetic defects of enzymes involved in homocysteine metabolism.[156,169]

Animal protein, and especially fish, chicken and eggs are highest in methionine, thus eating these foods will raise homocysteine.[170]

For those eating lots of green leafy vegetables, seeds, legumes, fruits and other vegetables, it is easy to get enough B6 and folate (note: folic acid is the supplement form, folate is the form found in food and comes from the word foliage).

B12 on the other hand, is not made by plants but by bacteria, some of which line the guts of animals. Thus, those who eat meat do get some B12 (although they can also run low). But considering the packaging that meat comes with, it is probably not the best way to get your daily dose of B12. Our gut bacteria also make B12, but it is too far downstream to be absorbed. Alcohol consumption, some prescription drugs (like lipid lowering drugs), and poor gut health can also play a role in B12 deficiencies.

Low B12 status in vegans and vegetarians can undermine the cardiovascular benefits of eating a plant based diet. As one paper put it, "In order to reap the full benefits of cardiovascular disease prevention in plant-based eating styles of vegan diets, individuals should maintain adequate vitamin B12 status".[171]

ii. There are three forms of homocysteine: the dimer homocysteine, the mixed homocysteine-cysteine disulphide and free homocysteine. General lab tests measure total homocysteine (tHcy) which is a measure of all three of these forms combined.

What to do?

The good news is we don't need that much and some is stored in the liver and other body tissues. Fermented foods such as tempeh, kombucha, kimchi and sauerkraut, some fermented teas, and some types of seaweed can have strains of bacteria that produce B12.[172] Foods like barley, spinach and certain types of radishes can also absorb B12 from the soil. Certain types of mushrooms (chanterelle, black trumpet mushrooms, and dried shiitake, for example) also contain B12. However, I do think it prudent (even for meat eaters) to take a sublingual B12 supplement. So far, getting too much B12 has not proven to have any ill effects as we will pee out any excess. So play it safe and take a B12 supplement.

Other Risk Factors Worth Talking About

Triglycerides

The vast majority of fat in the human body is stored in the form of triglycerides. It is the fat we eat and the fat we wear. It is stored subcutaneously and in skeletal muscle. A small proportion is found in the blood and is measured as serum triglycerides. It is a very different type of fat than is cholesterol and is burned during exercise.

Immediately after a fatty meal, serum levels will rise; however, an exaggerated response is seen in those who are insulin resistant and following meals high in dietary cholesterol.[173] Severe hypertriglycemia is defined as >1000 ml/dL. This is quite rare and is usually the result of a genetic defect.

Normal fasting levels are defined as being below 150 mg/dl (1.74mmol/l), 150-199 mg/dl are considered borderline high, 200-499 are considered high and anything above 500 mg/dl (5.81 mmol/l) is defined as very high. The American Heart Association considers <100 mg/dl (1.16 mmol/L) to be optimal.[174]

There are significant associations of high triglycerides to increased risk of CAD; however, this is somewhat controversial. The question is, does lowering triglycerides result in decreased risk, and indications are that it may. Very elevated levels (>500) can lead to pancreatitis.

Elevated triglycerides in the blood along with elevated fasting blood glucose levels can be indicative of insulin resistance or metabolic syndrome. Elevated triglycerides can cause arteries to become sticky and red blood cells to clump together (known as RBC aggregability). Red blood cells normally repel each other, which is a good thing. However, if there is fat in the blood stream (as occurs after eating a meal rich in saturated fat and cholesterol) they tend to lose this ability and they start sticking together.[175] Overeating of fatty foods, foods high in fats and sugar (yes fat and sugar most often go together), and foods composed of refined carbohydrates can raise triglyceride levels (again these usually come with fat as well; there are not too many people who eat dry white bread). Additional factors such as physical inactivity, cigarette smoking, excessive alcohol intake and obesity contribute to hypertriglycemia.[174]

The best way to control triglyceride levels is to eat slowly digesting starchy carbohydrates as found in whole plant foods. Foods like vegetables, sweet potatoes, beans and whole grains are good examples. Daily exercise will also help lower triglycerides, as will decreasing alcohol intake. Weight loss will also help. It is important to understand, however, that chronically elevated triglycerides in an obese, insulin resistant person pose a much higher risk than does elevated triglycerides in a healthy, normal weight person with normal glucose levels.[176]

The Deadly Quartet

The deadly quartet is elevated glucose, triglycerides, blood pressure and excess abdominal fat (as measured by waist circumference).[177] Having excess abdominal fat plus elevated triglycerides, glucose, and/or high blood pressure, is known as metabolic syndrome and is indicative of metabolic dysfunction, or the inability to properly metabolize fats and sugars. The presence of these factors puts one at serious risk for cardiovascular disease as well as other diseases such as stroke, cancer and diabetes.

High fasting glucose (>125mg/dL or 6.94 mml/L) by itself is a marker for diabetes (Type 2), which can be indicative of insulin resistance.

Insulin is the key which acts on cell receptors to get sugar into the cells (where it is needed and used for energy). If insulin cannot do its job, sugar is unable to get inside of the cells. This is known as insulin resistance and is characterized by above normal blood glucose levels. Chronically elevated glucose can result in glucose binding irreversibly to proteins (such as protein in the eyes) causing damage to arteries and other organs. When glucose is uncontrolled this can lead to blindness and loss of limbs, for example.

Causes of Insulin Insensitivity

What causes insulin insensitivity? Obesity, high fat diets (in particular animal fats), meat (in particular processed meats), lack of exercise and weight gain can all contribute to insulin insensitivity.[178] Diets high in fat can lead to a buildup of fat in the cells known as intramyocellular fat. Intramyocellular fat interferes with insulin cell receptors, inhibiting the ability of insulin to do its work (it gums up the lock so to speak).[179] Inflammatory cytokines that often accompany excess abdominal fat can also interfere with receptors from the outside of the cell. Thus insulin is inhibited from doing its work from factors both internal and external to the cell.[180] This is one reason why excess abdominal fat is a risk factor and why weight loss can be helpful.

The EPIC study (a large scale European study which follows thousands of people) concluded that meat, and especially processed meat (bacon and lunch meats) is significantly associated with the development of type 2 diabetes.[181] The Adventist Health Study (a study of a homogeneous group of people with regard to lifestyle but

who have varied eating patterns) found that the incidence of diabetes was very low in those who ate no animal foods (vegans) and the incidence increased in a dose dependent manner as animal food consumption went up, independent of obesity level.[182]

There are a number of mechanisms as to why increased meat consumption is associated with increased risk of type 2 diabetes. The pro-oxidative effect of the heme-iron found in meat (which affects the pancreas's ability to secrete insulin), advanced glycation end-products (produced when meat is cooked at high temperatures), the cholesterol and saturated fat content, the inflammation promoting properties of meat, the endocrine disrupting contaminants, have all been shown to play a contributing role.[183,184,185,186,187]

Studies Showing Reversal of Diabetes with Diet

Numerous interventional studies have shown a reversal of diabetes when patients are placed on a relatively low-fat, high fiber, plant-based diet. One study which put diabetics on a high fiber, high starch diet found that not only was glucose normalized, but that most went off of (or severely reduced) their diabetes medication after only three weeks! The best part of this study was that if subjects started losing weight they were made to eat more food. Thus the benefits and reversal of their diabetes occurred in the absence of weight loss. This is important as sometimes weight loss by any means can help with diabetes.[188]

In another study published in the European Journal of Clinical Nutrition, those eating a low-fat plant-based diet for 18 weeks showed significant improvements in weight, LDL cholesterol, and HbA1c (a measure of glycemic control) compared to a matched control group.[189] In another study, insulin therapy was discontinued in 11 out of 11 patients with type 2 diabetes after just 3-5 weeks of switching to a high-carbohydrate (70%), high fiber (65 grams), low fat diet (11%).[190]

Even more compelling were the results of another study where twenty-five type 1 and type 2 **lean** diabetics were switched to a high-fiber, plant-based, whole foods diet. After three weeks the type 1 diabetics reduced their insulin medication by 40%. Of the twenty-five type 2 diabetics, **all but one discontinued** their insulin medication (i.e. 24/25 discontinued all medication), this after only 3 weeks.[191] Of interest is that this was not mediated by weight loss as none of the subjects lost weight during the trial. As with heart disease, the combination of a high-fiber, low-fat, plant based diet along with regular exercise and adequate sleep can go a long way towards preventing and even reversing this syndrome.

Blood Pressure

Blood pressure is a measure of the pressure exerted on the arteries when the heart is ejecting and filling with blood. It is expressed as two numbers, the systolic blood pressure (SBP) and the diastolic blood pressure (DBP). The SBP is when the heart is ejecting blood and the blood is forced through the arteries increasing the pressure, thus it is the higher of the two values. Recent evidence suggests that SBP may be a more important indicator of high blood pressure in middle-aged and older adults. A substantial body of evidence has shown that an elevated SBP carries a high risk of heart attack

and stroke.[192] It has been estimated that high blood pressure contributes to about half of the heart disease and stroke deaths worldwide.[193]

Ideally you want to have your systolic pressure below 110 and your diastolic below 70 (or lower). People who start out with a so-called normal pressure of 120/80 appear to benefit when they get down to 110/70.[194] Increased BP with age does not have to be the norm as indicated by a study from rural Kenya. In the 1920s researchers measured the BP of a thousand Kenyans who ate a low-sodium, plant based diet (whole grains, leafy greens, beans, fruit and other vegetables). What was remarkable was that as the Kenyans aged, average BPs actually declined to 110/70. This was compared to age-matched Westerners whose average BP went up to greater than 140/90 by age 60.[195]

The best way to control or reduce blood pressure is to take a multifaceted approach—exercise regularly, lose weight if needed, stop smoking, reduce alcohol intake and eat a low-fat, low-salt, nutrient rich, plant-based diet. Taking medications for hypertension do nothing to treat the causes of high blood pressure; they only deal with the symptoms.

Eat Nitrate - Containing Foods:

Foods that are high in nitrates[3] can help to lower blood pressure. Why? Plant sources of nitrates result in increased production of nitric oxide (which helps to improve blood flow). In one study, when healthy volunteers were fed beetroot juice, blood pressure was substantially reduced (-10.4/8 mm Hg); an effect that correlated with peak increases in plasma nitrate concentration. Their conclusion, *"We advocate consumption of a diet high in nitrate (i.e., a "natural" strategy) to treat hypertension.... These findings suggest that dietary nitrate underlies the beneficial effects of a vegetable rich diet and highlights the potential of a natural low cost approach for the treatment of cardiovascular disease"*.[196] Foods high in nitrates are leafy greens (butter leaf lettuce, basil, Swiss chard, beet greens, cilantro), rhubarb and beet juice. The food with the highest source, however, is arugula.[197]

A 2014 meta-analysis published in JAMA reported that consumption of vegetarian diets (compared to omnivore diets) was associated with lower blood pressure and that such diets could be a "useful nonpharmacologic means for reducing BP".[198] This could be due, in part, to the fact that vegetarians tend to eat more anti-oxidant rich diets, and, as previously mentioned, have less inflammation. Oxidative stress and inflammation have also been implicated in the development of hypertension.[199] These foods are also high in potassium which, as previously mentioned, helps to improve endothelial function and increase production of nitric oxide.[200]

3. This is not to be confused with nitrites found in processed meats. These are converted to nitrosamines which are potent carcinogens.

Eat a Low Salt Diet:

The evidence that salt contributes to higher BPs is pretty clear, as much as the salt industry tries to protest to the contrary. Put people on a low sodium diet their BP will drop. Secretly give them a placebo or salt tablets and the BP for those on the salt tablets will go up while those on the placebo won't.[201,202]

About 75% of our sodium comes from processed foods. Salt is also often added to meats as it draws in water and so adds weight, which is an easy way to increase profits. So avoid meats, and especially processed meats, processed foods and not adding salt while cooking (add some afterwards if need be as our salt taste buds are on the tips of the tongue). The tough part is eating restaurant food, which can be especially high in salt. Ordering dishes with LOTS of vegetables will help offset some of the downsides to eating out.

Of interest is that those with low BP who eat a lot of salt can damage their endothelium.[203] Sodium appears to impair arterial function by suppressing superoxide dismutase, a potent antioxidant we make internally, which is important for maintaining endothelial health.[204]

Reversing Atherosclerosis

Ever since a Russian scientist induced atherosclerosis in rabbits back in the 1800s, the question as to whether induced atherosclerotic lesions could be reversed in humans has been a question of strong debate; that is until recently. The traditional and commonly accepted view was that the clinical manifestations and the lesions of atherosclerosis worsened with time and that, while progression could perhaps be slowed, these lesions, once formed, could not be reversed.

Early evidence that atherosclerosis could be reversed was provided by Armstrong et. al. in a series of elegant experiments on primates back in 1970.[205] Forty Rhesus monkeys were used in a long-term feeding study, 10 of which were used as controls (fed a regular monkey chow). The monkeys were fed a diet high in fat and cholesterol (40% fat diet, mostly from egg yolks) after which half the monkeys were then fed a diet low in cholesterol and saturated fat for three years. Upon autopsy, there was 61% arterial occlusion in the monkeys following the high fat diet. After three years of low fat feeding, however, the arteries regressed to only 20% narrowing. Thus the reduction was on average 61% occlusion to only 20% occlusion, and chemically the lesions lost much of the cholesterol and cholesteryl esters. Of additional interest is that the lesions which developed in the monkeys strongly resembled human lesions, with similar accumulations of fat and cholesterol, fibrosis and resultant narrowing of the arterial lumen.

Monkeys however are not humans and there are some differences in how rapidly these lesions form over time. Until better imaging techniques arrived on the scene, it was debatable as to whether similar regressions could be shown to occur in humans.

Enter Doctor Dean Ornish.

As a young physician, Ornish became frustrated with traditional medical approaches to CAD. No sooner were patients given stints and by-pass surgeries, they were back getting more procedures and surgeries. Medicine was only treating symptoms, not causes; the disease was not being halted.

To make a long story short, via a series of intervention studies on patients with severe coronary disease, he showed that when patients were placed on a regimen of smoking cessation, exercise, stress management and eating a low-fat, plant-based diet, not only did the disease halt, but in many patients it regressed. Moreover, those patients who were most compliant showed the greatest amounts of regression, whereas those who were only moderately compliant showed slowed progression. Those who did not adhere to the diet showed significant progression of the plaque.[206]

Of interest is that after only 24 days of eating this way, angina patients achieved, on average, a 44% increase in exercise duration and a remarkable 91% reduction in angina symptoms.[207] Most patients treated with this lifestyle intervention were able to avoid coronary bypass surgery or angioplasty due to the rapidity and magnitude of symptomatic benefits.[208] These benefits appeared long before regression of plaque had occurred and were most likely due to improved endothelial function and other structural effects.[209]

Quitting smoking, starting to exercise and dramatically changing eating habits could be considered tough changes to make, drastic even. So drastic in fact, it could be argued that few would stick with the program. A previous study, for example, found that within three years of having a heart attack, fewer than 10% had made any permanent changes to their eating and lifestyle habits.[210] Given that these patients were literally at death's door, it is amazing that so few were able to make any significant changes to their lifestyles!

In a separate study, it was found that only 30% of those prescribed a drug to lower their cholesterol, were still taking the drug a year after it was prescribed (this is a drug which patients are supposed to take for the rest of their lives, unless of course they change what they eat).[211] What could be easier than taking a pill?

Yet what Ornish found was that given the right amount of instruction, support and inspiration, patients can make BIG changes. At the end of three years, a whopping 77% had stayed the course.

What could explain this? According to Ornish, "When people who have had so much chest pain that they can't work, or make love, or even walk across the street without intense suffering find they can do all of these things without pain... they often say, these are choices worth making."[210]

Taking a pill is a daily reminder of having a chronic disease, and, neither does it change how one feels. Changing lifestyle habits changes how people feel; and the bigger the changes, the bigger the benefits. When big changes are made, big benefits are experienced and patients are more motivated to continue with the changes.

But which aspect of Ornish's program elicited these dramatic results? Exercise, not smoking, eating a plant-based diet and exercise will all likely contribute to regression of this disease. But which is the most important?

Enter Dr. Caldwell Esselstyn.

Doctor Esselstyn was a physician at the Cleveland Clinic in Ohio. After reviewing the research, he became convinced that diet played a significant role in the etiology of the various chronic diseases that we suffer from, namely cancer and heart disease. He was initially interested in studying breast cancer, but decided that doing a dietary intervention with heart patients might be easier, quicker and more dramatic than one with breast cancer patients. He recruited 23 patients to be a part of his initial dietary intervention study. Between them, these 23 patients had experienced 49 cardiac events in the eight years prior to starting the study (stints, by-pass surgeries and heart attacks). They were, he discovered, pretty bad off. A number had been told by their doctors that no more could be done and for them and they should put their affairs in order.

All 23 patients received intensive counseling and instruction on how to eat a low-fat, mostly vegan diet (initially he allowed some low-fat dairy, but later removed even that). He then followed up with them at 5 and 12 years.

At the 5-year follow up those who adhered to the diet experienced no new coronary events, and angiography showed that their disease had stabilized and in some cases had reversed (8 of the 11 tested showed regression).[212] Six of the patients dropped out of the program within 12-18 months and they returned to traditional medical care

(and eating a standard diet). Twelve years later, these six had experienced 13 new cardiac events. Adherent patients, however, had no coronary events and no surgical interventions 12 years later.[213]

Imaging techniques also showed that many had experienced marked regression of their plaque. All of them, within a few months of starting the program showed enhanced blood flow to the heart, no doubt due to improved endothelial function.

However, this was a small sample size. A larger dietary study was needed. In 2014 Esselstyn published a study on 198 patients who were all instructed on how to eat a low-fat, plant based diet.[214] They were then followed for four years.

What they found was that four years after being instructed on how to eat differently, 89% had stuck with the changes. More importantly, less than 0.6% (i.e. one person) experienced a cardiac event (he experienced a small stroke, purportedly because he had travelled to Asia and his consumption of salt went through the roof). Of those who didn't stick with the program, 69% experienced an adverse cardiac event during the same time period! Another physician who practices lifestyle medicine and who uses a nutrient-dense, plant-based dietary intervention to treat his patients is Dr. Joel Fuhrman. He has had wonderful success in treating his diseased patients, to the point where many of his patients have claimed it to be a miracle. In his book "The End of Heart Disease" (which I highly recommend) he states, "Over the past twenty-five years, I have counseled thousands of patients who've had advanced heart disease, many of whom experienced angina (chest pain) or were told they needed urgent bypass surgery or angioplasty. Not one of the individuals who followed my nutritional recommendations ever had a heart attack or died from heart disease." He has published a paper which evaluated more than one thousand individuals following his dietary recommendations which provides compelling results documenting the effectiveness of this approach (a plant-based diet) to reverse and prevent the disease.[215]

These are exciting and compelling data, yet many physicians still resist making these types of dietary recommendations; either they don't believe the data, aren't aware of the data, don't believe patients will follow their advice, or cite that there is "still insufficient proof". Given what we know, it seems that this should be the default diet against which all other diets are tested! I also think that patients should at least be informed of this option.

Taken together, there are some important lessons we can learn from these studies (as well as all the previous information presented in this paper): a) progression of atherosclerosis can be halted with the right dietary changes, b) regression can also occur in most patients when appropriate dietary changes are made, c) regression doesn't have to occur to reduce events and relieve symptoms, eating a nutrient rich diet free of animal products can result in rapid improvements in endothelial function and reduction of symptoms such as chest pain and shortness of breath d) dying from heart disease is preventable for the vast majority of people, e) big changes bring about big results; moderate changes may or may not reduce or reverse arterial disease, e) while stress management and exercise can play an important role, switching to a whole-foods, plant based diet is one of the most important changes you can make to prevent and reverse this disease.

The Role of Exercise and Fitness

But speaking of exercise, what role can being fit play in reducing risk for this disease?
While dietary factors are the biggest contributor to arterial disease, regular exercise and good levels of cardiorespiratory fitness can also play a protective role. But first let's define what we mean by cardiovascular exercise and fitness.

Exercise is the behavior, fitness is the outcome.

Cardiorespiratory exercise (commonly referred to as "cardio" or "aerobic" exercise) by definition, involves the repeated use of large muscle groups, and preferably use of the same large muscle groups. Thus activities such as running, walking, stair climbing, hiking, cycling, swimming, cross-country skiing or the sports that require running like soccer, basketball, or tennis are good examples of cardiorespiratory exercise. Activities in which different muscle groups are used repeatedly, yet which elevate the heart rate, tend to have less of an impact on cardiorespiratory fitness (like circuit weight training).

Aerobic Capacity versus Aerobic Endurance

Fitness is something that we can measure in a lab using standardized, graded exercise tests. The test goes something like this: You get on a treadmill at an easy grade and speed. The speed and/or the grade is gradually increased until you get to a maximal effort (or close to your maximal effort) and you can no longer continue. The longer you last on the test, the fitter you are. A more sophisticated test will measure your ability to use and consume oxygen at your maximal effort. This is known as VO2max or aerobic capacity.

Another type of aerobic fitness is the ability to go for longer periods of time, or aerobic endurance. Aerobic endurance is more about the ability of the mitochondria within the working muscles to convert fat and sugar into useable energy than your maximal oxygen consumption rate.

Developing both types of fitness is important.

Researchers who assess the impact of exercise and fitness on risk for CVD will either ask patients how much they exercise or directly measure fitness levels, i.e. their aerobic capacity using the protocol described above. For obvious reasons, a more direct measure of fitness is more reliable than having participants self-report what they do for exercise.

The aerobics longitudinal study out of the Cooper clinic is one in which they measured fitness directly, rather than relying on self-reported levels of exercise. For this study they performed treadmill tests on thousands of people, followed them over time and looked at who died and who got heart disease. They then adjusted for confounding factors like smoking and age.

Exercise, like Charity, Covers a Multitude of Sins: or Does it?

What they found was that high levels of fitness (and we are not talking marathon running type of levels here) were associated with lower rates of all-cause mortality and risk of heart disease and stroke, even in the presence of other risk factors.[216,217,218] As running guru Dr. George Sheehan liked to say, "exercise is like charity, it covers a multitude of sins".

However, high fitness levels don't necessarily guarantee that you won't get a heart attack.

Remember the story of Jim Fix.

Jim Fix was an overweight, two pack-a-day smoker and a stressed out executive. Then he discovered running. Or at least he woke up one day, looked in the mirror, didn't like what he saw and started running. Soon he was running marathons. His weight dropped from 240 lbs to under 180. He wrote a bestselling book called the Complete Book of Running. His lean muscular legs were pictured on the front. This is a great example of the power of exercise to improve health and to cause a remarkable turnaround in an overweight, out of shape, stressed out body. It is actually pretty amazing what exercise can do for you.

But the one thing Jim didn't change was how he ate. He continued his diet of eggs and bacon for breakfast and steak and burgers for dinner. He even debated with Nathan Pritikin, who at the time was pioneering the notion that heart disease could be reversed and prevented by eating a mostly plant based, low-fat diet. Jim believed that anyone who could run a marathon in under four hours was immune to getting a heart attack and that diet was incidental to the problem (especially if you ran marathons). Alas, however, soon after this debate, Jim died of a massive heart attack while out running. Upon autopsy, given how clogged up his arteries were, the doctors were amazed that he hadn't had a heart attack much sooner! This was no doubt due to his prodigious levels of exercise which had helped. But sadly his exercise only delayed the inevitable.

The moral of this story is you can't exercise your way out of a bad diet.

A recent study which illustrates this quite nicely was one in which researchers looked at intima-media thickness in the carotid artery of vegans, runners and those eating a standard American diet (SAD). Carotid intima-media thickness is a non-invasive test and a pretty good indicator of arterial health and risk of future cardiac events.[219]

Both the vegans and those eating the SAD were relatively sedentary.

Not surprisingly, the vegans had much less intima thickening than those eating the SAD. However, the vegans on average were 38 pounds skinnier. So this could've explained their better arterial health. Enter the runners. Now these weren't your "run of the mill" (no pun intended) recreational runners. These were serious distance runners who had been running an average of 40-50 miles per week for 20 plus years. The runners also ate a SAD diet but, because of all that running, were as slim as the non-exercising vegans (lazy vegans). So how did they fare? Their intima thickness was somewhere between that of the vegans and the sedentary, overweight, SAD eating group.

Thus, when it came to reducing intima thickness, eating a plant based diet was the most beneficial, but high levels of exercise also had some benefit.

In addition to reducing risk for CAD, there are countless other benefits of exercise that should be elucidated. It is mood enhancing. It can mitigate the negative effects of stress. It reduces anxiety and increases optimism and self-efficacy. It improves metabolic function, bone strength, blood flow, and brain function. It improves immune function (if not done to the extreme). It reduces risk for 11 kinds of cancer. It helps with weight management. It can even improve creativity. And that is the short list.

In Conclusion

Eating a plant based diet is the only diet that has been shown to both stop the progression and even reverse arterial disease. Combine this dietary approach with not smoking, stress management, regular exercise and, in particular exercise that results in higher levels of aerobic fitness, and you have powerful protection from the scourge of heart disease. It has the power to make you “heart attack proof”. Additionally, this way of living and eating will also protect you from a host of other chronic diseases like cancer, Alzheimer’s and diabetes. Finally, eating well and exercising will boost energy levels, improve sleep, and negate jet-lag and ultimately leadership performance.

I recently received an email from a three star general who had attended our Leadership at the Peak program. He started the email by saying, “I will start by saying, you were absolutely right”. I of course immediately cut and pasted that line and sent it to my husband. Needless to say it wasn’t greeted with much enthusiasm!

His email went on to say, “Other than a bite of camel in the United Arab Emirates, a mouthful of squid in Egypt, and a piece of shrimp in Jordan, all for diplomatic reasons . . . I've been a 100% plant-based consumer for nearly six months. Carol is sticking with it too. My body has adjusted, inside and outside. Jet lag in multiple time zone travels is not an issue. Thirty one pounds of weight melted away. Cholesterols dropped from continuously elevated to continuously normal - more than 25 points down in LDL. The last time I had numbers like these was more than 26 years ago. I am now an advocate, as many notice the visible change and ask for the reason. I have shared some of your research material contained in "Fit to Lead" to help them move ahead.”

Enough said.

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