Fat and Cholesterol Are Not Your Enemy

Summary

Conventional wisdom's diet-heart disease hypothesis (that consumption of saturated fat and cholesterol foods raises blood cholesterol and triggers the heart disease process) oversimplifies and grossly distorts the true causes and mechanisms that have led to today's heart disease epidemic. The true risk factors for heart disease are oxidation and inflammation in the bloodstream, a state that is caused by poor dietary and lifestyle habits.

There is no correlation between the consumption of foods high in saturated fat or cholesterol and the risk of heart disease. Saturated fat has been the preferred fuel source for humans throughout evolution. The familiar villain in conventional wisdom's diet-heart hypothesis--LDL cholesterol--is only potentially problematic in the context of a high insulin-producing diet or other risk factors that compromise the effective clearance of oxidized LDL from the bloodstream.

The actual heart disease process occurs as follows:

● excess consumption of processed carbohydrates (grains and sugars) promotes excess insulin production and high triglycerides in the bloodstream.

● Along with consumption of easily oxidized polyunsaturated oils and a lifestyle of excessive stress (not enough sleep, not enough sun, chronic exercise), this dietary pattern promotes a state of oxidation and inflammation in the bloodstream.
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- Under these circumstances, cholesterol in the bloodstream can then turn dangerous, with small, dense LDL molecules becoming lodged on artery walls and sustaining oxidative damage.
- This elicits an immune system response that leads to further inflammation, the formation of plaque on the artery walls, and an eventual heart attack or stroke.

Saturated fat is an excellent source of energy and supports healthy cellular function, since our cell membranes are comprised mainly of saturated fat and many hormonal and metabolic processes utilize saturated fat. There has never been any scientific evidence presented to confirm that consuming saturated fat by itself is unhealthy. Saturated fat, primarily from animal products, has been a dominant source of calories for humans throughout evolution.

Conventional wisdom has promoted a false association between high levels of fat in the diet, and high levels of fat in the blood (aka, high triglycerides). High triglycerides in a fasted state are confirmed to be unhealthy but are associated with excess carbohydrate intake, not fat intake.

LDL converts from VLDL into either small, dense LDL or large, fluffy LDL. Small, dense LDL is more inclined to be dangerous, because it can lodge in the artery wall and become oxidized and inflamed. Traditional blood readings for total LDL are of little to no value when assessing heart disease risk. Surprisingly, the majority of heart attack victims have a safe LDL value.

HDL cholesterol protects against heart disease by cleansing the bloodstream of potentially damaging small, dense LDL molecules. Consuming saturated fat helps elevate HDL levels, since HDL is used to digest fat. A sensible exercise program has also been shown to help elevate HDL.

Statins have minimal impact on the most important heart disease risk factors, and have numerous problematic side effects that can compromise health. Dietary and lifestyle modification can quickly and dramatically reduce heart disease risk factors, as revealed in blood values for HDL, triglycerides, assorted inflammation markers like C-reactive protein, and small, dense LDL.
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The true catalysts for heart disease, oxidation and inflammation, occur from adverse dietary habits and lifestyle practices that are unrelated to saturated fat or cholesterol intake, but closely related to excess carbohydrate intake/excess insulin production and excess intake of easily oxidized polyunsaturated fats.

Some other lifestyle practices that promote oxidation and inflammation in the bloodstream are smoking, poor stress management, insufficient sleep, and chronic, overly stressful exercise patterns.

Facts

Cholesterol:

Cholesterol is a waxy molecule comprised of lipid (fat). Cholesterol is contained in the membrane of every cell in the body, as well as in blood plasma. It is critical to building and maintaining cell membranes, metabolizing fat-soluble vitamins, producing bile to help digest fat, and synthesizing many hormones, including sex hormones. The liver manufactures ample cholesterol to provide for healthy bodily functions and varies its production level in accordance with dietary intake. Since cholesterol is oil-based and the bloodstream is water-based, cholesterol and other fats in the bloodstream are packaged together with tiny protein-covered particles called lipoproteins (lipid + protein), which avails easy transportation through the bloodstream. Thus, lipoproteins are comprised of cholesterol, triglyceride, phospholipid (another type of fat), and, of course, protein.

The notion that consuming foods high in cholesterol will promote heart disease is a grossly inaccurate soundbite that disregards the critical role of cholesterol in metabolism and the manufacture of steroid hormones. Cutting back on dietary cholesterol will result in the liver producing more cholesterol. In fact, virtually all the cells in the body can manufacture the cholesterol they need because the protein molecules in the cell will direct the production of more cholesterol (or pulling in more LDL particles circulating in the bloodstream) if insufficient levels are detected in the cell. Cholesterol-lowering statin drugs override this elegant balancing process at the cellular level. They effectively lower cholesterol levels in the body, but with negative side effects discussed in detail shortly. Therefore, moderating dietary cholesterol in an effort to regulate blood cholesterol is not only a fruitless effort, but can also unnecessarily restrict your intake of highly nutritious foods.

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There are several varieties of lipoproteins, all with different transporting functions, that comprise the total amount of cholesterol in the body. In order of size, from largest to smallest, they are: chylomicrons, VLDLs, IDLs, LDLs, and HDLs (as well as subfractions of each). The three that are the most prominent in the heart disease saga are VLDLs, LDLs, and HDLs (very low-density, low-density, and high-density lipoproteins, respectively). Each of these lipoproteins carries a certain percentage of cholesterol, triglycerides, and other minor fats.

HDLs (High-density lipoproteins):

HDLs take oxidized cholesterol from the bloodstream back to the liver for excretion or recycling into useful service. About 30 percent of the total cholesterol in a healthy body is composed of HDL. HDLs are known as the good cholesterol or nature's garbage trucks for their ability to cleanse the arteries and bloodstream of oxidized cholesterol. High levels of HDL help reduce the risk of heart disease.

VLDLs (Very-low-density lipoproteins):

These cholesterol complexes are manufactured in the liver to transport triglyceride and cholesterol to target fat or muscle cells. Initially, VLDLs are composed of about 80 percent triglyceride. After VLDLs deliver their cargo to various target cells in the body, they (containing mostly cholesterol and minimal triglyceride) shrink substantially in size and transform into either large, fluffy LDLs or small, dense LDLs.

Large, fluffy LDL:

Also known as buoyant LDL, this type of cholesterol is formed from VLDL when blood levels of triglycerides and insulin are low. These molecules are generally harmless, even at high concentrations (a condition that is often associated with genetics). However, in the presence of other risk factors (high insulin-producing diet, systemic inflammation, abdominal obesity, family history, smoking, etc.), even large, fluffy LDL can make a contribution to heart disease if the particle count is high enough and it's not cleared from the blood quickly enough.

With other risk factors and adverse lifestyle practices present, large, fluffy LDL can circulate in the blood for too long and become oxidized. One such risk factor is low
HDL values, since HDL is so critical to clearing oxidized cholesterol from the bloodstream. Another risk factor is poor thyroid function, which down-regulates LDL receptors and causes LDL to circulate for longer periods in the bloodstream with insufficient antioxidant protection. These risk factors are relevant to people with familial hypercholesterolemia, a genetic condition in which LDL values are extremely high, and almost entirely of the large, fluffy variety.

Small, dense LDL:

VLDL converts to small, dense LDL when triglyceride and insulin levels are elevated in the bloodstream. Small and dense, they can become lodged onto the endothelial cell layer (ECL) the cells that line the walls of your arteries. Since oxygen is constantly flowing through arteries, the small, dense LDL that end up trapped in the ECL sustain oxidative damage and trigger an immune response that progresses toward an eventual heart attack. While it's confirmed that small, dense LDL are prone to oxidation, we have a classic chicken-or-the-egg issue here. The act of being oxidized decreases LDL size, so it's possible that small, dense LDL are already partially oxidized, and then become further oxidized when they attach to the ECL.

Hence, it's noteworthy that high-carbohydrate eating patterns are not the only pathway to the accumulation of dangerous small, dense LDL. For example, some who convert to primal/paleo eating develop sluggish thyroid function (possibly due to hormone alterations from the radically new diet, consuming too many goitrogenic vegetables, or getting insufficient iodine or selenium). The thyroid problems trigger an elevation in small, dense LDL because LDL is being cleared from the blood slower than usual. Often this unique population of thyroid sufferers can tweak their eating habits (e.g., adding back more high-nutrient value carbs), and regain optimal thyroid function and clearance of oxidized cholesterol.

Understanding the difference between LDL particles makes it apparent how misleading a total LDL cholesterol value can be in evaluating heart disease risk. The total LDL reading on a blood test indicates the mass, or total amount, of cholesterol carried by your LDL particles, but it does not reveal what size the particles are. For example, a total LDL cholesterol reading of 100 could be comprised of many small, dense particles (which are more dense and more potentially problematic), and fewer large, fluffy particles (which are lighter and less apt to lodge into tight spaces between the endothelial cells that line the walls of your arteries). Hence, heart attacks can and do
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occur in individuals who have a relatively low total, and seemingly safe, LDL reading. This can be the case if a high percentage of the total LDL is small, dense LDL, and if systemic inflammation and oxidative damage are present due to adverse diet and lifestyle practices.

Busses or Mini Coopers?:

Particle size is a critically important distinction to understand when it comes to LDL cholesterol and disease risk. LDL-C is the term for total amount of cholesterol someone is carrying, while LDL-P is the number of LDL particles in the blood. Think of it like cars on a freeway: LDL-C represents the total number of passengers on the road (total inside all the vehicles), while LDL-P represents the number of vehicles on the road. Although LDL-C is often used to estimate the amount of LDL someone's blood contains (with the logic that having more passengers to transport will usually mean there are more vehicles zooming to and fro), it can be misleading when we take particle size into account.

For instance, say you have 1 deciliter of blood, and the LDL-C the total amount of cholesterol (passengers) in all the LDL particles (vehicles) captured in that sample is 130 mg. This 130 can be from a bunch of tiny, dense LDLs that carry relatively little cholesterol in each particle, or it can be 130 from a smaller number of large, fluffy LDLs that carry lots of cholesterol in each particle.

Going with our car analogy, the former case (lots of tiny, dense LDLs) is like a high-traffic road crammed with motorcycles and solo-driver Mini Coopers zooming around, while the latter example (large, fluffy LDLs with lots of cholesterol in each particle) would be a wide open freeway with plenty of space between the occasional large, full bus or passenger van cruising along. The number of traveling passengers (LDL-C) might be 130 in both situations, but because there are more vehicles (LDL-P) in the first scenario, the chance of an accident goes up due to sheer probability.

All else being equal, it's a numbers game: the more vehicles there are, the more likely it is for some to crash and cause damage (like hitting the endothelium) or end up in gridlock and stay on the road too long (i.e., staying in the bloodstream too long and becoming oxidized as a result). For that reason, it's much more useful to measure the number of LDL particles someone has (the vehicles, or LDL-P) instead of estimating LDL levels by measuring cholesterol content (the passengers, or LDL-C). This is

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especially important for people eating paleo or primal diets high in natural saturated fats, which might increase the total amount of cholesterol in the bloodstream, but typically in the form of large, fluffy LDL. Seeing one's LDL-C increase as a consequence of eating primally might elicit a knee-jerk negative reaction, until the individual receives confirmation from a particle size test that the increase is mostly large, fluffy LDL and generally harmless.

Realizing how the various forms of cholesterol have a different impact on the body, and how cholesterol only becomes problematic in the arteries when combined with other circumstances, it's clear that obsessing over a single blood reading for total LDL value is an ineffective way to evaluate heart disease risk. In South Asian Health Solution, author Dr. Ronesh Sinha references a UCLA meta-study (analysis of many independent studies) showing that 75 percent of patients hospitalized for a heart attack had an LDL of less than 130 mg/dl (widely accepted as safe), and that half of the victims had an LDL under 100, widely considered ideal. Unfortunately, as Dr. Sinha explains, instead of seeing the big picture of how all risk factors interplay, many physicians and drug companies are interpreting the results of the UCLA meta-study to mean we should set our LDL targets even lower.

Heart disease process:

1. Small, dense LDL molecules loaded with triglyceride (excess insulin = excess triglycerides = a pattern of fat storage instead of fat burning) lodge in the endothelial cell layer (ECL).
2. The delicate and sensitive ECL has become damaged due to systemic inflammation, making it easier for small, dense LDL particles to become lodged in this gap junction between ECL cells.
   a. As Dr. Sinha explains in The South Asian Health Solution, Your ECL is in constant contact with your blood and can detect even the subtlest chemical disturbances arising from poor nutrition or negative emotions; it responds to any damage or perceived threats by sounding the inflammation alarm.
   b. In a healthy body, the inflammation crew is remarkably effective at making repairs and keeping the ECL in great condition.
   c. However, in an unhealthy body, this pro-inflammatory emergency response system often does more damage than good.
3. Interestingly, in a healthy body, cholesterol molecules can be used as a temporary band-aid to cover lesions in the ECL. When the state of inflammation...
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subsides, the cholesterol leaves the now-healed lesion and is recycled back to the liver by HDL.

4. When inflammation is chronic, problems can breed with small, dense LDL on the ECL as described. They remain in place for too long, inflammation is exacerbated by the aforementioned macrophage immune response, and the molecules become oxidized by the constant exposure to hemoglobin on the arterial wall. It's likely that the originally flawed association between cholesterol and heart disease came about from the simple observation of cholesterol molecules forming plaque on the arterial walls of deceased heart attack victims. In other words, blame was placed on the band-aid (cholesterol attached to the ECL) rather than examining the original cause of the wound, and why it became infected (inflamed and oxidized) and eventually fatal.

The delicate endothelial cell layer (ECL) is sensitive to environmental disturbances from poor nutrition to negative emotions.

When small, dense LDL molecules become lodged in the ECL, they are exposed to oxygenated blood (hemoglobin) passing through the arteries. This constant oxidative exposure causes the area to become inflamed and to form the familiar plaque on arterial walls that characterizes atherosclerosis.

The immune system, reacting to this undesirable plaque formation, sends macrophages scavenging white blood cells designed to engulf and digest cellular debris to the area in an effort to mitigate the damage. The macrophages rush to the scene and attempt to gobble up the oxidized small, dense LDL. While well intentioned to begin with, the macrophages become overwhelmed by the major effort and expand in size to become what are known as foam cells. These foam cells produce a chemical called myeloperoxidase, which further oxidizes LDL particles. Foam cells also release chemicals called cytokines into the bloodstream, which attract more macrophages to the area to cause further inflammatory damage. It boils down to the following sequence:

1. Small, dense LDL cholesterol molecules hang out in the bloodstream for far too long, and are oxidized due to the constant flow of hemoglobin.
2. These oxidized LDL molecules then lodge onto damaged (and thus more receptive) ECL, only to become oxidized even further.
3. Inflammation is caused by the immune response of the macrophages turning into foam cells.
4. Coagulation (clot formation) eventually causes a significant obstruction, a fat-filled tumor known commonly as plaque on the ECL.
5. Macrophages convert into foam cells.
6. Clots form and plaque accumulates
7. As oxidation and inflammation continue, the plaque becomes stiff and calcified and more susceptible to rupture.
8. Once a rupture occurs, it can block the artery on the spot, or more commonly detach from the wall, drift into circulation and eventually cause a stroke (blockage of an artery heading to the brain), or a heart attack (aka myocardial infarction blockage of a coronary artery).

Hyperinsulinemia:
A dietary pattern of excess carbohydrate intake results in chronically elevated insulin levels, a condition known as hyperinsulinemia. Hyperinsulinemia promotes systemic inflammation in the body. In the cardiovascular system, this causes damage to the delicate ECL by reducing levels of nitric oxide (a compound that keeps the ECL relaxed) and promoting platelet adhesiveness (sticky platelets that clot more readily). These conditions increase the sheer force of blood flowing against the ECL (high blood pressure), and facilitate the oxidation of small, dense LDL on the ECL.

High triglycerides:
When carbs are ingested, they are broken down into glucose in the digestive tract and either burned quickly in the bloodstream, transported by insulin into fat cells (where the glycerol molecule in glucose joins up with excess free fatty acid molecules in the bloodstream to form triglycerides the storage form of fat), or transported to the liver for conversion into glycogen or triglyceride. About 60 percent of ingested glucose is transported to the liver in a healthy person. The liver converts glucose into glycogen for storage in muscle tissue and the liver. When liver glycogen stores are full, the liver then converts excess glucose into triglyceride. Triglyceride is then transported by VLDL into cells throughout the body.

Chronically elevated insulin levels prevent triglyceride from being mobilized into free fatty acids for use as energy. Instead, you are stuck in a fat storage pattern instead of a
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balance between energy mobilization (burning of fatty acids) and energy storage (storing ingested fats or ingested carbs converted into fats). Elevated levels of triglyceride in the bloodstream drive VLDL conversion into small, dense LDL instead of large, fluffy LDL.

Excess cortisol:
This stress hormone promotes inflammation as a component of the fight-or-flight response in the body. Chronically elevated cortisol levels produced by poor eating habits, chronic exercise habits, insufficient sleep, and other forms of life stress that overstimulate the fight-or-flight response can result in chronic, or systemic, inflammation. The overstress condition characterized by chronically elevated cortisol is believed to be a driving factor in assorted health problems, cancers, and heart disease. Chronic stress is pro-inflammatory.

Insufficient HDL:
Insufficient levels of HDL due to poor diet and exercise habits, and even the use of statins (which lower HDL as well as LDL), can significantly increase heart disease risk. Conversely, optimal HDL levels can efficiently cleanse the bloodstream of damaged cells, including potentially dangerous small, dense LDL molecules that promote heart disease.

Poor diet:
Besides excess carb intake and insulin production, other elements of the Standard American Diet can elevate disease risk, among them: excess consumption of PUFA oils (which are prone to oxidation); any consumption of chemically altered trans or partially hydrogenated fats (which are highly oxidized and pro-inflammatory); insufficient intake of antioxidants (which can help prevent oxidation in the first place); insufficient intake of saturated fat (eating saturated fat helps elevate HDL and makes LDL particles more resistant to oxidation); and poor LDL clearance (with diet likely contributing to poor thyroid function that inhibits LDL clearance).

Blood tests:
The mainstream medical community emphasizes total LDL cholesterol reading and commonly dispenses prescription statin medication when a patient’s total LDL levels and/or total cholesterol levels exceed a certain threshold. Statins effectively reduce the
level of all forms of cholesterol in the bloodstream; an indicator that pharmaceutical and medical experts believe will reduce heart disease risk.

Triglycerides-to-HDL ratio:
Dr. Cate Shanahan, author of Deep Nutrition and Food Rules suggests that the most important blood metric to assess heart disease risk is not total LDL but the triglyceride-to-HDL ratio. A ratio of 3.5:1 or below is desirable, while a ratio of 1:1 is considered superior. Remember, excessive triglycerides are driven by excess carbohydrate intake, excess insulin production, and the conversion of excess ingested carbohydrates into triglycerides in the liver. High triglycerides indicate the body's cholesterol processing system is overwhelmed. On the other side of the ratio, high levels of HDL can effectively remove potentially dangerous small, dense cholesterol molecules from the bloodstream. Hence, elevating HDL and reducing triglycerides is the most worthwhile tandem goal to reduce heart disease risks. Other recommended blood tests to assess the big picture of heart disease risk include:

Blood pressure: Hypertension puts excessive stress on the cardiovascular system and increases disease risk. High blood pressure is a reliable indicator of unhealthy dietary, exercise, and lifestyle habits. Experts agree that a safe blood pressure threshold is a systolic/diastolic reading of 120/80 or lower (for each value).

Vitamin D:
This essential nutrient supports healthy cell division and thus cancer prevention. Vitamin D is obtained primarily through sun exposure during the months, and times of day, of peak solar intensity at your particular latitude. Even an optimal primal-aligned diet contributes only minimally to your requirements. Supplementing can be effective during the winter months for those who are sun challenged or those who live at a latitude incongruent with skin pigment/their ancestor's latitude.

Long-standing government recommendations for both dietary vitamin D intake (600 I.U. per day) and sun exposure are very low and in sharp contrast to the recommendations dispensed by vitamin D experts. While mainstream resources like the Vitamin D Council and the Institute of Medicine have been gradually raising their daily intake recommendations, they still effectively promote insufficient vitamin D levels and also reference blood ranges that are much lower than vitamin D advocates believe is
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optimal. For example, the Institute of Medicine and Endocrine Society defines vitamin D deficiency as less than 20 ng/mL, and vitamin D insufficiency as under 30 ng/mL. These values are derived from the preferred vitamin D blood test known as 25(OH)D.

In contrast, vitamin D experts reference much higher ranges. In 2007, a group of vitamin D and nutrition researchers published an article in the American Journal of Clinical Nutrition stating that the optimal vitamin level was 75 ng/mL. Vitamin D advocates agree on an optimal blood value range of 50-70 ng/mL, and a range of 70-100 ng/mL when treating heart disease or cancer. Furthermore, any value below 50 ng/mL is categorized as deficient.

Recently, the “more is better” vitamin D message has been tempered a bit. While much less likely than a vitamin D deficiency, it’s possible to develop problems with excessive vitamin D levels, especially if you are deficient in vitamin A. Potassium and vitamin K also work synergistically with vitamin D. If you are making an effort to increase your sun exposure and/or are taking vitamin D supplements, it is imperative that you also take steps to ensure your vitamin A, vitamin K, and potassium levels are adequate. Primal eating patterns will ensure you are good for vitamin K and potassium. The best source of vitamin A is animal products, especially liver. Also, heavy sun exposure can deplete vitamin A, so if you are going hog-wild for vitamin D health but not eating animals, you may want to supplement with cod liver oil to boost vitamin A levels and minimize the risks of vitamin D toxicity driven by low vitamin A levels.

Vitamin D toxicity is most likely to happen in unique, sensitive individuals when they too many supplements over a long period of time. It’s impossible to become vitamin D toxic from sun exposure, since the body automatically regulates vitamin D production by tanning the skin (and consequently shutting down further vitamin D production) when you’ve had enough. Regarding diet, even a deliberately high vitamin D diet won’t provide nearly enough to put you into the excess zone. The concentrated dose that supplements provide make it necessary to exercise some caution against indiscriminate pill popping. The best idea is to get your levels tested before beginning a supplementation program, and then retest frequently.

There is believed to be some variation in optimal vitamin D levels based on ethnicity, with those of non-White ancestry able to thrive in lower ranges than light-skinned folks. Chris Kresser, leading expert on ancestral and paleo health and nutrition, argues that
there is no good evidence that levels above 50 ng/mL deliver any health benefits, citing a review of over 1,000 studies conducted by the Institute of Medicine. However, Kresser agrees that levels up to 60 ng/mL are still healthy for most people. Toxicity concerns start to arise if you are in the 100-150 ng/mL range.

Fasting blood insulin:
Levels outside the standard recommended zone specified on blood reports indicate chronic overproduction of insulin.

HbA1C:
Known as the estimated average glucose test, HbA1C measures how much glucose is attached to a hemoglobin molecule, a reliable marker for a state of elevated blood glucose levels over an extended time period. This is a superior test to the more common instantaneous blood glucose readings (something commonly tracked by diabetics with a portable machine) that vary throughout the day and are strongly influenced by meals.

Triglycerides:
Under 150 mg/dL is essential to reduce disease risk, while under 100 is optimal. A reading over 150 suggests an excess of oxidized LDL and thus elevated disease risk.

High sensitivity C-reactive protein (hs-CRP):
In the absence of other acute infections, a high level of hs-CRP in your blood is a reliable sign of inflammation in the blood vessels. Elevated C-reactive protein is associated with an increased risk of heart attack, stroke, and sudden cardiac death. Normal CRP levels are supposedly 10 mg/L. If you have an infection or acute stressors, you might experience temporarily elevated CRP. For example, drawing blood from marathon runners at the finish line will reveal high CRP levels (and other markers of cardiac and musculoskeletal stress) as a consequence of the trauma sustained from running 26 miles, but these levels will typically return to normal in 72 hours.

Under normal circumstances or over the long-term, ideal CRP levels are well under 1 mg/L. Between 10-40 mg/L (and perhaps even 1-9 mg/L) indicates systemic inflammation (or pregnancy), while anything above that is associated with a serious acute condition (e.g., heart attack, major infection, post-marathon, or Ironman
Excess belly fat is often an indicator of elevated C-reactive protein and systemic inflammation.

**IL-6, or Interleukin-6:**

T cells (a type of white blood cell that plays a huge role in the immune response) and macrophages (cells that engulf and digest also known as phagocytosing stray tissue and pathogens) both secrete IL-6 as part of the inflammatory response, so elevated IL-6 can indicate systemic inflammation.

**Homocysteine:**

This is an amino acid that when elevated can increase risk of heart attacks, strokes, and blood clots. Vitamin and mineral deficiencies can lead to elevated levels too, making homocysteine a good general health marker to track.

**Creatine phosphokinase (CPK):**

Enzyme found in brain and lungs (CPK-1) heart (CPK-2), and skeletal muscle (CPK-3). Elevated levels indicate damaged tissues from a medical incident or extreme athletic stress.

**Lipoprotein(a):**

Lipoprotein(a) promotes coagulation (clotting), making it a heart disease risk marker. Elevated values are strongly associated with early and aggressive forms of heart disease.

**LpA2:**

LpA2 is an enzyme that promotes oxidation of lipoproteins, making it a heart disease risk marker.

**Apolipoprotein B (ApoB):**

Measures a protein residing in all LDL particles. High levels indicate elevated heart disease risk. Request this test from your physician and discuss your readings in the context of normal ranges. Keep in mind that ApoB readings can fluctuate from day to day and provide only a snapshot of the health status of your cardiovascular system over time.
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LDL particle size/advanced lipid profile testing:
Provides values for large, fluffy LDL (also known as Type A LDL) and small, dense LDL (aka, Type B LDL). High triglyceride levels suggest that Type B values may be elevated, because blood triglyceride levels determine whether VLDL converts into large, fluffy or small, dense molecules after delivering cargo to target organs and cells. Examples of advanced tests are the Berkeley Heart Lab test, The VAP test (Vertical Auto Profile), and NMR (Nuclear Magnetic Resonance) test.

Coronary calcium testing:
Measures calcium buildup in the arteries via CT scan, a strong indicator of atherosclerosis. Values are assigned an Agatson score (after Dr. Agatson, South Beach Diet) correlating with a cardiac mortality risk percent value.

Getting the advanced blood tests mentioned can help track the true causes of heart disease: oxidation, inflammation, and coagulation. The abbreviations in the graphic give further detail about some of the other agents involved in the heart disease process. CETP = cholesteryl ester transfer protein. It is believed to transfer cholesterol molecules from HDL over to LDL, and thus contribute to heart disease risk. IL-6 = Interleukin-6, a cytokine that has a proinflammatory effect on the oxidation and inflammation process of heart disease. TNF = tumor necrosis factors a group of cytokines that can cause cell death. PAI-1 = plasminogen activator inhibitor 1 believed to interfere with healthy blood clotting and also facilitate further growth of malignant tumors.

Statins:
The evolutionary health movement asserts that a blanket strategy of reducing total cholesterol levels through potent statin medication is problematic for several reasons.

#1 LDL particle size:
Critical patient care decisions are commonly made based on the total LDL reading instead of testing and considering the levels of each fraction. It is often the obligation of the patient to request blood tests with separate readings for LDL particle size. Evaluating the big picture of heart disease risk factors as discussed with triglycerides, LDL particle sizes, HDL, systemic inflammation markers, and other advanced blood tests is a more effective approach than the current trend of prescribing statins to
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millions of patients based on total cholesterol readings or total LDL cholesterol readings.

#2 Oxidation and inflammation:
Reducing heart disease risk to a single number on a blood test might assuage fears and anxieties harbored by both patient and doctor (take this pill, lower your LDL, and be home free), but predominant general risk factors can be overlooked in the process. For example, even relatively low levels of small, dense LDL can still be problematic when other risk factors are present, such as insufficient HDL levels and high stress, proinflammatory lifestyle practices. The highly publicized death by heart attack of television journalist Tim Russert brought the issue to light, as he had an extremely low level of total cholesterol in the bloodstream (and was taking statin medication), yet still succumbed to a coronary incident at the age of 57.

#3 Side effects:
Statins suppress the production of cholesterol in the liver, producing lower blood values of all forms of lipoproteins, including beneficial HDL. Since cholesterol is critical to many important functions in the body, artificially suppressing cholesterol production through pharmaceutical means can interfere with healthy serotonin balance (less energy and alertness; potential mood consequences too); hampered vitamin D synthesis, disturbances in regulating blood sugar, disturbances in controlling inflammation, and disturbances in an assortment of critical hormonal processes.

Statin use has been shown to cause inflammation in the liver and also have a tendency to deplete cells of a critical energy-producing nutrient known as coenzyme Q-10 (CoQ10). CoQ10 helps mitochondria produce energy in cells. Depletion of this critical coenzyme can result in fatigue, muscle pain, and other indicators of muscle dysfunction. Furthermore, CoQ10 depletion can hamper the ability of all cells to fight free radicals and moderate inflammation. This state of cellular depletion can lead to adverse lifestyle practices such as insufficient exercise (prompted by the lack of energy and muscle pain side effects) and elevated oxidative damage in cells whose normal energy production mechanisms are compromised.

Statin use comes with serious side effects and fails to address the proximate causes of heart disease: oxidation and inflammation.
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Furthermore, statins do not affect triglyceride (blood fat) levels or LDL (the so-called bad cholesterol) particle size, nor do they decrease risk of death in any women, in men over 65, or in men under 65 who have not had a heart attack. That said, statins have been shown to reduce risk of heart attack in specific high-risk populations, and have been shown to generate some incidental benefits outside their intended effect of lowering total cholesterol levels. These benefits include an anti-inflammatory effect, a blood thinning effect (helping reduce plaque formation in those susceptible), and a plaque-stabilizing effect (reducing the risk of rupture in those susceptible). Obviously, these side benefits, or pleiotropic effects, of statin use are not enough to warrant their widespread use as the first line of defense against heart disease risk in the broad population. As Dr. Sinha explains, I prescribe statins and other lipid-lowering drugs to very high-risk patients who cannot carry out even a minimum level of recommended lifestyle changes. Numerous studies, including a long-term study published three times in the New England Journal of Medicine, showed that people taking multiple cardiac medications have a 40 percent higher risk of mortality after four years than those taking no medication.

**Epidemiological studies:**

The diet-heart hypothesis of heart disease is finally being rejected on the basis of compelling evidence from some of the world's largest and most respected epidemiological studies (the study of how diet and lifestyle practices affect health). In 1997, Ancel Keys, the originator of the flawed hypothesis decades before, conceded that there's no connection whatsoever between cholesterol in food and cholesterol in the blood. A meta-analysis of 21 studies and 350,000 people published in 2010 in the American Journal of Clinical Nutrition revealed that there was insufficient evidence to associate dietary saturated fat intake with stroke or heart disease. Other respected studies such as the Women's Health Initiative, The Health Professional's Study, The Nurses Health Study (the largest epidemiological study of women in history, leading to the publication of 265 scientific papers in acclaimed journals), and the Framingham Heart Study (the largest and most comprehensive epidemiological study in medical history, tracking the dietary and lifestyle habits of over 15,000 residents of Framingham, MA since 1948, leading to the publication of over 1,200 research articles in respected medical journals), also showed no correlation between dietary fat intake or cholesterol intake and heart disease. A 2014 study from Ohio State University and Dr. Jeff Volek (co-author of The Art and Science of Low Carbohydrate Eating) revealed that even when subjects doubled, or...
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even nearly tripled, dietary saturated fat intake, blood levels of fat did not increase. However, when carbohydrate intake increased, blood levels of a critical marker for unhealthy fat storage and metabolic syndrome, palmitoleic acid, increased in close correlation with increases in carbohydrate intake. Volek commented, People believe 'you are what you eat,' but in reality, you are what you save from what you eat. The point is you don't necessarily save the saturated fat that you eat. And the primary regulator of what you save in terms of fat is the carbohydrate in your diet. Since more than half of Americans show some signs of carb intolerance, it makes more sense to focus on carb restriction than fat restriction.

Conventional wisdom fat story:

Dietary fat has been maligned as unhealthy for decades based upon flawed science, propaganda, and manipulative marketing and advertising. As we have already discussed healthy and unhealthy dietary fats in detail in Module 3, this section will cover three ways that dietary fat has been inaccurately maligned as unhealthy. Let's first address the failure to distinguish between healthy fats and unhealthy fats. Second, we'll discuss the negative influence a high-carbohydrate diet has on dietary fat. Finally, we will highlight the flawed science and manipulative marketing that generated a widespread shift in dietary habits several decades ago.

The original USDA Food Guide Pyramid, introduced in 1992

As we learned in Module 3, chemically altered trans and partially hydrogenated fats, found in many processed snacks, treats, and frozen meals, are extremely destructive to your health, as they are easily oxidized and reactive upon ingestion. Chemically altered fats create destruction at the cellular level by promoting systemic inflammation, interfering with hormone signaling, compromising immune function, and damaging various organs and systems such as the nervous system, cardiovascular system, endocrine system, and even the brain (since these fats are able to cross the blood-brain barrier). Experts estimate that consumption of trans and partially hydrogenated fats are directly responsible for 300,000 cancer deaths annually.

While polyunsaturated fats do offer some health benefits, the high omega-6 varieties are consumed in excess in the modern diet, particularly in light of commonly insufficient intake of omega-3 fatty acids. The most offensive sources of polyunsaturated fats are bottled vegetable oils (canola, corn, soybean, safflower, etc.), which are easily oxidized
by light, heat, or oxygen and so disrupt healthy hormone and immune function upon ingestion.

It is universally agreed that omega-3 fats are health promoting, due to their anti-inflammatory effect. Omega-3s, found in highest concentration in oily, coldwater fish, are lauded for enhancing cardiovascular, brain, skin, and immune function. Monounsaturated fats (found in macadamsias, avocados, olives and extra-virgin olive oil) are also universally regarded as healthy as they enhance cardiovascular and immune function and help protect against heart disease.

There is disparate thinking about saturated fat in the diet. Primal/paleo/evolutionary health enthusiasts tout saturated fat as a dietary centerpiece, referencing the fact that the consumption of high-fat animal products has been a key factor in human evolution and the complete lack of evidence that saturated fat by itself is unhealthy to consume. Indeed, saturated fat comprises about half of our cell membranes, contributes to a variety of healthy metabolic and hormonal processes in the body, is an abundant source of important vitamins like K2, A and D, and is easily burned as free fatty acids in the bloodstream.

Unfortunately, conventional wisdom makes an inaccurate association between saturated fat consumption and heart disease. While saturated fat and cholesterol are indeed the agents that clog your arteries and cause heart attacks and strokes, the process by which saturated fat and cholesterol turn dangerous is completely independent of their intake in the diet. While science proves this unequivocally, it might be considered counterintuitive on the surface. Eating fat makes you fat, or eating cholesterol clogs your arteries with cholesterol, are more easily accepted as simple and logical assumptions. This is especially so when trusted sources such as the US government, medical doctors, and certified nutritionists toe the party line, and when billions of dollars of advertising drive these points home to the public over decades.

As we learned in the preceding cholesterol section, the pro-inflammatory effects of excess carbohydrate consumption and excess insulin production, and the oxidative damage caused by excess intake of polyunsaturated oils and also chronic elevation of stress hormones, are the factors that set the stage for fat and cholesterol to turn troublesome in the bloodstream. In the absence of high carbohydrate intake and other stress factors, saturated fat is an efficient, clean-burning energy source for the body.
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Bad influence of high-carbohydrate diet:

Fat is calorically dense at nine calories per gram. Consuming a high-carbohydrate diet and producing excessive insulin locks you into a fat-storage pattern as we learned previously. Hence, consuming calorically dense fatty foods can indeed lead to weight gain thanks to the high insulin production (from a grain-based diet) that drives ingested calories into fat storage. When fat is consumed in the context of a high-carbohydrate diet, the excess insulin production promotes a state of oxidation and inflammation in the bloodstream, along with high triglycerides. These are the key risk factors for atherosclerosis. When carbohydrate intake is minimal, however, dietary fat is easily mobilized as a stable and long-lasting energy source. Its high satiety factor promotes high levels of dietary satisfaction and stable appetite and caloric intake levels.

Unfortunately, conventional wisdom distorts the literal truth of calories in = calories out by failing to account for other metabolic variables that dramatically influence outcomes when attempting to regulate caloric intake and body composition. Since fat has over twice as many calories per gram as carbs or protein, the recommendation to avoid fat is made in hopes of reducing overall caloric intake and thereby regulating body composition. The suggestion to avoid saturated fat negates the high satiety factor of saturated fat, which helps regulate appetite and total caloric intake naturally, instead of via ill-fated willpower or portion control efforts.

Propaganda and flawed science:

American scientist Ancel Keys was a key instigator in the promotion of the diet-heart hypothesis of heart disease in the 1960s, which drove unhealthy dietary transitions in Western society namely the shift to a low-fat, high-carbohydrate diet, and the promotion of polyunsaturated fats in favor of saturated fats.

Using observational data, Keys presented research to lawmakers and government authorities suggesting that consumption of foods high in saturated fat and cholesterol contributes to atherosclerosis. He was later accused of cherry picking data to make his hypothesis seem stronger than it really was, and some of his contemporaries challenged his work on the grounds that other diet and lifestyle variables such as sugar consumption, national affluence, and even TV ownership correlated more tightly with heart disease than saturated fat did.
Mary Enig, Ph.D., a renowned nutritionist from the University of Maryland, lipid biochemistry expert, and author of Know Your Fats, spent her career battling conventional wisdom's take on fat intake and heart disease. In the 1970s, she was a central figure in challenging the corruption and misinformation dispensed by the USDA and the U.S. Senate's McGovern Committee (headed by former presidential candidate George McGovern). Influenced by heart disease trends from war-rationed Europe and highly questionable, lobbyist-tainted testimony, the committee published its report directing Americans to replace saturated fat with PUFAs and to limit fat intake in general (which was then disastrously replaced with excessive carbohydrate intake).

The influence of special interest propaganda and flawed science is evident in the saga of the USDA Food Pyramid. Just prior to the scheduled 1991 release of the updated Eating Right Pyramid food guide (an update to the long-standing Basic Four food groups the government had been promoting for decades), the USDA halted the rollout on the grounds that it would be confusing to children, according to Secretary of Agriculture Edward R. Madigan. This aroused great suspicion among skeptics, who believed the USDA was bowing to intense pressure from the cattle and dairy lobbying groups, who were upset that their foods were listed near the use sparingly section of the pyramid.

According to Marion Nestle, healthy food advocate and author of numerous books including Why Calories Count: From Science to Politics and Food Politics: How the Food Industry Influences Nutrition and Health, The pyramid controversy focuses attention on the conflict between federal protection of the rights of food lobbyists to act in their own self-interest, and federal responsibility to promote the nutritional health of the public... and the inherent conflict of interest in the Department of Agriculture's dual mandates to promote U.S. agricultural products and to advise the public about healthy food choices. Informed observers lamenting the obvious special interest influence have long criticized USDA guidelines, yet these same guidelines have succeeded in becoming ingrained into conventional wisdom for the general public as well as for medical and nutrition professionals. One of the worst examples of unsound science and manipulative special interests emerged in the 1994 publication of an article titled How to Put the Food Guide Pyramid into Practice in the Journal of the American Dietetic Association. The intent was to categorize assorted foods that didn't conveniently fit the broad categories of the food pyramid (grains, fruits, vegetables,
meat, dairy). Suddenly, foods such as cake, cheese balls, and fried corn chips could fit snugly into the grains section of the pyramid, indulgences in frozen yogurt could slot right into the dairy category, and, of course, potato chips could count toward one's daily recommended intake of vegetables. Obviously, this Hard-to-Place Foods message implicitly gave consumers a free pass to consume assorted highly processed, nutrient-deficient foods.

While critics predictably slammed the message, the USDA's perseverance prevailed and conventional wisdom about healthy eating was molded by the iconic pyramid concept. The USDA Food Pyramid underwent another comprehensive update in 2005, but the most disturbing elements the recommendation to follow a high-carbohydrate, grain-based diet, and the lack of distinction between healthy and unhealthy types of foods in the various categories remained unchanged.

The updated MyPyramid, introduced in 2005

Ensuing government-funded research was essentially mandated to fall in line with the original recommendations of the McGovern Committee, and the notion that fats were bad took hold and flourished into conventional wisdom for decades (and is still going strong!). The pyramid concept was finally phased out in 2011 with the introduction of the US government's ChooseMyPlate imagery.

ChooseMyPlate.gov, introduced in 2011

Some observers complimented the new device, praising it for being an easier image to understand, especially for children; for presenting a positive image of what foods to choose instead of what foods to avoid; and for devoting half the plate to fruits and vegetables. Unfortunately, the importance of high quality dietary fats is completely absent, and the continued emphasis on grains and dairy reveals little progress from the untoward influences of food industry lobbyists from prior decades. Furthermore, the section labeled protein can be seen as misleading, since obtaining adequate protein is virtually a non-issue when it comes to healthy eating goals for Americans.

Reconciling opposing beliefs:
The extreme disparity between dietary recommendations from various experts is disturbing to informed consumers, but some reconciliation can be achieved by realizing that anyone who departs from the disastrous norms of the Standard American Diet (SAD) will succeed at least in the short term with health and weight loss goals. For
example, popular author Dr. Dean Ornish touts a 4 Elements approach to healthy living, consisting of the pillars of nutrition, fitness, stress management, and love and support. The nutrition element is decidedly low fat, and emphasizes nutrient-dense whole foods such as vegetables, fruits, whole grain products, legumes, lean protein sources, herbs and spices, and omega-3 oils.

While primal/paleo/ancestral health advocates will take issue with the aversion to fat and the recommendation of whole grains and so forth, the described diet is vastly superior to the massive intake of sugar, chemically altered fats, and heavily processed foods in the SAD diet. Similarly, the elements of vegetarian eating, zone eating, blood type eating and the like all represent a positive departure from the Standard American Diet, albeit in disparate directions. Primal/paleo/ancestral health advocates will point out the long-term challenges with adhering to a strict low-fat or vegan/vegetarian diet.

- First, these diets exclude many rich and satisfying foods, making satiety difficult to achieve.
- Second, their emphasis on carbohydrates promotes energy, appetite, and mood swings due to excess insulin production.
- Third, their emphasis on grains, even whole grains, delivers less nutrient density per calorie than a Spartan Method type eating pattern featuring liberal intake of nutrient-dense meats, fish, fowl, eggs, vegetables, fruits, nuts, and seeds.

Review

Cholesterol Affirmations:

Cholesterol is contained in the membrane of every cell in the body and plays a critical role in metabolism and hormone synthesis. The conventional wisdom notion that LDL cholesterol levels are an accurate predictor of heart disease risk is disastrously flawed and oversimplified. LDL start out as VLDL and, after delivering triglycerides to assorted cells in the body, they convert into either large, fluffy LDL (mostly harmless) or small, dense LDL (potentially dangerous). The true catalysts for heart disease are oxidation and inflammation of small, dense LDL lodged onto the endothelial cell layer (the highly sensitive cells that line the walls of the arteries). The state of oxidation and inflammation in the bloodstream is driven by chronically excessive insulin production and overly stressful lifestyle habits.

The heart disease process is thus:
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- Small, dense LDL molecules predominate due to excess insulin/high triglyceride levels, and even large, fluffy LDL can become problematic due to poor clearance and the oxidation process.
- Small, dense LDL particles (likely already oxidized) lodge onto inflamed ECL and are further oxidized by the constant flow of hemoglobin in the artery.
- The immune system sends macrophages to the inflamed area; macrophages become overwhelmed and convert to foam cells. Clots form, plaque accumulates, and then ruptures.
- Artery clogs or plaque breaks away and causes a stroke or heart attack.

The real heart disease catalysts are:

#1 Excess insulin:
Promotes inflammation, stiff arteries, sticky platelets, and oxidized small, dense LDL (from a high-carbohydrate diet)

#2 High triglycerides:
Promotes conversion of VLDL into small, dense LDL instead of large, fluffy LDL, and locking into fat-storage pattern (from a high-carbohydrate diet).

#3 Excess cortisol:
Chronic stress promotes an inflammatory environment in the bloodstream and damage to the sensitive ECL (from poor diet, chronic exercise, insufficient sleep, emotional stress).

#4 Insufficient HDL:
Body is less efficient at clearing oxidized cholesterol from bloodstream (from poor diet, statin use, chronic exercise).

LDL cholesterol readings are minimally relevant to heart disease risk, and in fact many heart attack victims have LDL values considered within the safe range. HDL are nature's garbage trucks, cleansing the arteries and bloodstream of potentially problematic oxidized cholesterol. More relevant blood tests to track disease risk are:

- triglyceride-to-HDL ratio (3.5:1 or better imperative; 1:1 or better ideal);
- blood pressure (high pressure = poor ECL function);
- Vitamin D;
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- HbA1C (average glucose long-term);
- fasted glucose;
- LDL particle count;
- inflammatory markers like C-reactive protein, homocysteine, and CPK;
- and calcium scan to reveal arterial damage.

Statins lower all types of cholesterol, including HDL, and deplete the critical nutrient CoQ10, which helps the mitochondria produce energy. They fail to address the true cause of heart disease and deliver disturbing side effects. Decades-long meta-studies involving hundreds of thousands of subjects are finally encouraging mainstream appreciation for the idea that dietary saturated fat and cholesterol are not the proximate causes of heart disease.

Fat Affirmations:

Due to the influence of flawed science and government policy, and propaganda and manipulative advertising from BigAgra, conventional wisdom has made an inaccurate association between dietary fat and fat in the blood (triglycerides). It is now becoming more widely accepted that excess carbohydrate consumption drives high triglycerides in the blood, and that consuming saturated fat long maligned as a heart disease catalyst is entirely healthy and in fact a centerpiece of the human diet for millions of years. Three main reasons that fat has been inaccurately maligned by conventional wisdom are:

1. Failure to distinguish between healthy and unhealthy fats: PUFA and trans fat = bad; saturated, monounsaturated, omega-3 = good.
2. Deleterious influence of a high-carbohydrate diet: Excess carb intake drives excess insulin drives high triglycerides and a fat-storage pattern. Fat and cholesterol consumed in an oxidative, pro-inflammatory environment exacerbate high-risk health conditions (weight gain, heart disease risk).
3. Propaganda and flawed science: Ancel Keys used questionable correlative studies to condemn saturated fat, helped drive government policy to transition away from butter to vegetable oil, and ultimately vilified fat-rich animal products. The USDA food pyramid and food plate imagery are graphic examples of the disastrously flawed, special interest-influenced government policies (e.g., grouping all fats together to consume sparingly; or to designate hard to place junk foods in certain pyramid categories as implicit recommendations to
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consume). The increased rates of obesity, heart disease, and cancer, despite the most advanced health care in the world, are proof that the Standard American Diet has been a dismal failure.
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