

SATURATED FAT – FRIEND OR FOE?

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For many people, the words ‘saturated fat’ evoke images of clogged arteries, greasy food and gut rolls. Health authorities have been advising people to reduce their saturated fat intake for decades and the message that ‘saturated fat is bad for you’ has been imprinted in most people’s nutritional knowledge. In the past 5 years a significant body of published research has questioned this longstanding belief. In light of these new pieces to the puzzle, world experts are hotly debating saturated fat’s role in our health. While the answers are not completely clear, there is certainly a new understanding of this macromolecule emerging.

To understand the situation, it is important to recall the basics of lipid biochemistry. All fats and oils, whether of vegetable or animal origin, are some combination of saturated fatty acids, monounsaturated fatty acids (one carbon to carbon double bond) and polyunsaturated fats such as linoleic acid and linolenic acid. In general, animal fats such as butter, lard and tallow contain about 40-60% saturated fat and are solid at room temperature. Vegetable and seed oils from northern climates contain a preponderance of polyunsaturated fatty acids and are liquid at room temperature, while vegetable oils from the tropics are highly saturated. Coconut oil, for example, is 92% saturated. The reason the oils in plants are more saturated in hot climates is because the increased saturation helps maintain stiffness in plant leaves.

Fatty acids are not only classified according to their degree of saturation, but also by their molecular length. As with the degree of saturation of a fatty acid, the length of the fatty acid gives it unique biochemical and physiological properties. Long chain fatty acids make up the vast majority of fatty acids consumed by humans in a typical Western diet. Short and medium chain fatty acids, 4-12 carbon atoms in length, behave quite differently in the body to long chain fatty acids. They are absorbed quickly into the bloodstream, transported without the need of lipoproteins and provide a rapid source of cellular energy. They also exert powerful antimicrobial properties.

When we look at saturated fats in general, they have many vital roles in the human body, including:

- Saturated fatty acids constitute at least 50% of cell membranes, providing necessary ‘stiffness’ and integrity.
- Saturated fats are required for effective calcium incorporation into bones.¹
- They lower Lp(a)²
- They help protect the liver from alcohol and other toxins, such as paracetamol.³
- They enhance the immune system.⁴
- Elongated omega-3 fatty acids are better retained in the tissues when the diet is rich in saturated fats.⁵
- Short- and medium-chain saturated fatty acids have important antimicrobial properties, particularly in the digestive tract.

WHEN DID SATURATED FATS BECOME ‘BAD’?

To understand better why saturated fat, which is clearly essential for human life, came to be considered bad for our health, it is helpful to look back in history a little.

In the 1850s, a German pathologist, Dr. Virchow (of the famous Virchow's triad) proposed that accumulation of lipids in blood vessels caused atherosclerosis, which led to heart disease. This suggestion certainly seems reasonable when you see an atherosclerotic plaque, as they are very fatty (although the fat composition is usually around 25% saturated and 75% unsaturated). Virchow developed a 'Lipid Hypothesis', that proposed was a *link between blood cholesterol levels and the development of cardiovascular disease*.

By the middle of the 20th century, cardiovascular disease was becoming an increasingly common cause of death, and scientific interest in the lipid hypothesis was renewed. A timely publication of population research by Ancel Keys suggested a link between high saturated fat diets and heart disease. Keys' research received a great deal of attention in the United States, and became known as 'the Seven Countries Study'. While Keys' research was initially refuted but scientists, Keys' appointment onto the American Heart Association's (AHA) advisory board soon changed things. The AHA issued advice to the American people that a diet which included large amounts of butter, lard, eggs and beef would lead to coronary heart disease. This resulted in the American government recommending that people adopt a low saturated fat diet in order to prevent heart disease, spawning the low fat revolution. With time and the 'dumbing down' of public health advice, saturated fat soon became considered not only bad for heart health, but also bad for human health.

WHY IS THE ROLE OF SATURATED FAT SO DIFFICULT TO WORK OUT?

Fuelled by the increasing burden of obesity, diabetes and degenerative diseases in Western societies, a great deal of research has been undertaken over recent decades to try and answer the questions regarding fat and our health. In particular, the role of saturated fat has continued to be questioned.

At first glance, it may seem ridiculous that the quest to understand the role of saturated fat in human health has spanned over six decades. When you consider that there was about 70 years between the first air flight and man reaching the moon, it makes you realize how complex the relationship between lipid consumption and cardiovascular health must be.

There are several reasons why it has taken researchers so long to get useful information regarding saturated fat. Firstly, saturated fat cannot be examined in isolation from other components of the diet, because when saturated fat is removed from the diet it must be replaced with another macronutrient such as protein, carbohydrate, or other types of fat.

In addition, high quality research studies, such as randomized, double blind, placebo-controlled trials, are extremely difficult to achieve for any reasonable length of time. Some research has come from metabolic ward studies and prospective control studies using veterans in long-term hospital-level care. Overall, however, research on saturated fat has taken the form of population studies and cohort studies. While these study types are useful to suggest hypotheses, they are not powerful enough to identify causality.

In addition to dietary confounders and study design difficulties, genetic variation has increasingly been understood to be a significant factor in an individual's response to dietary lipids. ⁶

Another significant cause of confusion regarding the role of saturated fat in heart disease has been the well-supported association between blood cholesterol and heart disease. The Prospective Cohort Collaboration, published in the Lancet in 2007⁷, was a meta-analysis of individual participant data from 61 prospective studies with 55 000 vascular deaths. The researchers showed that a 1 mmol/L lower total cholesterol was associated with a halving of CHD risk in both sexes at ages 40–49 years, a 33% reduction at ages 50–69, and a 17% reduction at ages 70–89 years. It is not difficult, when blood cholesterol is causally linked with heart disease, to consider that saturated fat, which is normally associated with cholesterol -containing foods, has a part to play in heart disease. (Cholesterol is only found in animals, therefore no saturated fat of plant origin occurs with cholesterol.)

SO IS SATURATED FAT A PROBLEM?

While research supporting saturated fat's association with heart disease continue to be published, over the past five years a significant body of published research has thrown this association into question. These large, well-designed papers have concluded that the association between saturated fat and heart disease either doesn't exist or is not as significant as was previously thought. Reading the studies feels a little like watching a dusty row of dominoes falling.

WHAT THE SCIENTISTS ARE SAYING

In 2009 a systematic review of the evidence supporting a causal link between dietary factors and CHD, published in the Archives of Internal Medicine,⁸ summarised the current knowledge of dietary factors and their relationship with CHD in the four points below.

1. A higher intake of trans-fatty acids and a higher intake of foods of high glycaemic index or high glycaemic load are associated with an **INCREASED** risk of heart disease.
2. Increased consumption of alcohol, dietary beta-carotene, fibre, fish, omega-3, folate, fruit and vegetables, nut, monounsaturated fat, vitamin C, E, wholegrain and adherence to 'Mediterranean' dietary pattern are significantly associated with **REDUCED** risk of heart disease.
3. Factors with the most evidence for effect on heart health were Mediterranean diet (protective), vegetable consumption (protective), nut consumption (protective), trans-fatty acid consumption (detrimental) and consumption of foods of high GI or GL (detrimental).
4. There was no evidence found to support overall reduction of saturated fatty acids and concern was raised about making recommendations without the benefit of RCTs. There was also concern that limiting dietary fat may result in increased consumption of carbohydrates that may lead to increased incidence of CHD.

Another 2009 review of the evidence for the contribution of dietary fatty acids to CHD concluded that, "Intake of SFA was not significantly associated with CHD mortality" and "not significantly associated with CHD events".⁹ Consistent with this conclusion

was a pooled analysis of cohort studies in the same year which supported benefits of PUFA but not MUFA or carbohydrate in replacing SFA. ¹⁰

A 2010 meta-analysis of prospective cohort studies found no further significant evidence for concluding that SFA are associated with an increased risk of CVD ¹¹ and a meta-analysis of RCTs reiterated the value of PUFA but also concluded that, given the modest plausible benefit, policies should focus substantially on other risk factors such as low seafood, fruit and vegetable consumption. ¹²

2011 saw further support for saturated fat with the Cochrane review ¹³. Taking things one step further the Cochrane report on dietary fat and cardiovascular disease concluded that saturated fat is not causally linked to cardiovascular disease, or *any* other disease for that matter. In fact, the Cochrane report stated there is no significant effect of any dietary fat intervention, compared to usual or control diet, on cardiovascular mortality. The Cochrane report further states that, while the available evidence suggests increasing MUFA and/or PUFA and reducing saturated fat may reduce cardiovascular events, this effect has only been found in men!

PUT SIMPLY....

Comprehensive reviews of all research to date are showing that altering dietary fat (either by reducing it or increasing unsaturated fats and reducing saturated fat) on average, does not significantly alter any of the following:

1. total or cardiovascular mortality
2. Individual cardiovascular events: infarctions, stroke
3. Cancer deaths or cancer diagnosis
4. Non-fatal myocardial infarction diagnosis
5. Quality of life
6. HDL
7. Systolic and diastolic blood pressure.

^{8 9 10 11 12 13 14}

PRACTICAL IMPLICATIONS

While epidemiological research is a lynch-pin in our understanding of human health and disease, it does not necessarily recommend the best treatment for the patient sitting in front of you. It is becoming better understood that genetic variability plays a significant role in cardiovascular disease, as well as an individual's biochemical response to nutrients. With regard to saturated fat, review of the current evidence suggests focusing on this macronutrient may not produce the intended benefits. ¹²

The emphasis on cardiovascular disease by epidemiologists and research scientists can cloud the bigger picture required to practice clinical medicine. After all, there is more to a human than just a heart. Similarly, scientific evidence on individual macromolecules does not equate to clinical advice on foods. It is now clear that the effect of particular foods on cardiovascular disease cannot be predicted solely by their content of total SFA. ^{9 10 12} This is understood to be because saturated fats are a diverse group of fats with variable cardiovascular effects. Additionally, major SFA food sources contain other constituents that appear to influence cardiovascular disease risk.

The misguided focus on SFA intake and heart disease by public health authorities can also lead lay people to believe SFA are 'artery clogging' and unhealthy, when it is well established that SFA consumption does not increase the risk of stroke, and in

fact some studies suggest a protective effect⁸. Furthermore, saturated fats are essential for normal growth, development and health in humans.

CONCLUSION

There is no doubt the relationship between SFA and cardiovascular disease is complex. In 2012 the evidence is certainly mounting that the real demons for cardiovascular health are trans –fats and refined carbohydrates.^{10, 13, 14} Perhaps it is time we leave naturally occurring saturated fat in foods to do the essential structural, and biochemical actions we have evolved to depend upon, and concentrate on reducing the consumption of highly refined and processed foods.

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