

Saturated fat has been unfairly demonised

YES

Reading the evidence, there does not appear to be unanimous agreement regarding the role of saturated fatty acids (SFA) in coronary heart disease (CHD) and cardiovascular disease (CVD). The champions who support this hypothesis argue that the evidence is unequivocal. However, the fact that this dogma is not universally accepted suggests that either such affairs of the heart are not entirely correct, or that the issue is more complex than it first seems. Despite the fact that there is widespread conviction that SFA are responsible for a large proportion of the coronary disease burden, current evidence, including systematic reviews and meta-analyses, seem to dispute this.

Common high-SFA foods are butter, coconut cream and some meats, each with their own ratios of various SFA, which in turn have a diverse range of biological functions, with multiple effects on circulating lipids and lipoproteins. Humans adapted to consume milk into adulthood around 10 000 years ago, with the ability to digest lactase beyond infancy arising several times independently; our ancestors traditionally sought out the fattiest organ meats in preference to leaner cuts. Perhaps the reason that not all

roads lead to the proverbial Rome is because SFA have a complex role in human health and accurately capturing this is difficult, not just because of other dietary confounders, but also within individual variability.

Roads that do not lead to Rome—reliance on biomarkers and ecological fallacies

The assumption has been that diets high in SFA lead to high total cholesterol and therefore increase the risk of cardiovascular disease. A 2003 meta-analysis evaluated 60 controlled trials testing the effect of dietary fatty acids on serum lipids and lipoproteins, and concluded that replacing saturated fat with carbohydrate has no effect on total serum cholesterol, cautioning against using cholesterol alone as a marker of disease.¹ By 2010 a review highlighted more questions than solutions about the effect of SFA on serum lipids, particularly raising the risks of their replacement with carbohydrate which can lead to atherogenic dyslipidaemia.²

More directly, people who eat more SFA should also have more CVD than people who follow low fat diets; however, studies looking at dairy

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While evidence can help inform best practice, it needs to be placed in context. There may be no evidence available or applicable for a specific patient with his or her own set of conditions, capabilities, beliefs, expectations and social circumstances. There are areas of uncertainty, ethics and aspects of care for which there is no one right answer. General practice is an art as well as a science. Quality of care also lies with the nature of the clinical relationship, with communication and with truly informed decision-making. The **BACK TO BACK** section stimulates debate, with two professionals presenting their opposing views regarding a clinical, ethical or political issue.



Murray Skeaff



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BACK TO BACK this issue:

consumption and associations have returned positive, neutral and negative results. A 2011 meta-analysis on milk and dairy consumption and CVD and all-cause mortality indicated that milk intake is not associated with these outcomes and may be inversely associated with overall CVD risk. Whether or not the milk and dairy products were low fat did not appear to make a difference.³

Conclusions from the 2008 Joint FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition concluded that:

- replacing SFA with polyunsaturated fats (PUFA) decreased LDL and total/HDL concentration
- replacement with carbohydrate decreased both LDL and HDL but did not change HDL/total cholesterol ratio.

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

In 2011, the revised Cochrane review on the matter found a small but potentially important reduction in cardiovascular risk, but not mortality, by replacing SFA with some plant oils but not reduction of total fat. Replacing SFA with carbohydrate was not beneficial.

Perhaps more importantly, the report also concluded that replacing SFA with PUFA decreases the risk of CHD; however, replacement with carbohydrate probably increases CHD and contributes to metabolic syndrome development. It found insufficient evidence relating to SFA and CHD when monounsaturated fat (MUFA) or largely whole grain carbohydrates are the replacement and insufficient evidence that SFA affects the indices related to the metabolic syndrome.⁴

In 2009 a systematic review of the evidence supporting a causal link between dietary factors and CHD, published in the *Archives of Internal Medicine*, summarised the current knowledge of dietary factors and their relationship with CHD in the four points below.⁵

1. A higher intake of trans-fatty acids and a higher intake of foods of high glycaemic index

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.⁵

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".⁶ 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.⁷

The 2009 findings were also echoed by a 2010 Harvard review of the effects of SFA consumption on CVD risk which concluded that "Public health emphasis on reducing SFA

consumption without considering the replacement nutrient or, more importantly, the many other food-based risk factors for cardiometabolic disease is unlikely to produce substantial intended benefits".⁸

A 2010 meta-analysis of prospective cohort studies found no further significant evidence for concluding that SFA are associated with an increased risk of CVD⁹ 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.¹⁰

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What is now clear is if SFA are replaced by something, it very much depends what that replacement is. Replacing them with PUFA appears to confer health benefits, while replacing them with MUFA is uncertain and carbohydrate appears neutral or even detrimental.^{2,8,11}

For the past 60 years, public health obsessions with dietary fat have resulted in fat being generally and indiscriminately viewed as 'bad'. Low fat is seen as low calorie and therefore healthy, and there is little regard for the very real adverse effects of replacing fats, particularly saturated fats, with refined carbohydrate. Amongst this, SFA appear somewhat victimised. A booming private market in processed 'low fat', high carbohydrate foods and spiralling obesity prevalence may be some of the legacy of such messages.

The very recent comparative meta-analysis by Mente⁵ highlights that the statistical evidence supports modifying your carbohydrate intake, by means of a Mediterranean or a low glycaemic-index diet (vegetables, legumes, fruit, nuts, cheese, fish and olive oil), is more likely to help you avoid a visit to coronary care than focusing merely on reducing your saturated fat intake.

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