Dietary fats in the management of diabetes

Trudi Deakin

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Article points

1. A healthy diet is an essential part of the management of diabetes, but many healthcare professionals are confused about what advice they should be giving.
2. Processed food is often wrongly classified as saturated fat when, in reality, it is a mix of refined carbohydrates, refined vegetable oils and trans-fatty acids.
3. Advising people with diabetes to consume a "real food" diet that contains nutrient-dense and satiating foods, which have been minimally processed, will benefit their health more than focusing on the amount and type of dietary fat.

Key words

- Diabetes
- Dietary fats

Author

Trudi Deakin is Chief Executive of X-PERT Health and Consultant Research Dietitian

We all know that people with diabetes benefit from a healthy lifestyle and many education programmes aim to educate people about the benefits of a healthy diet. However, there continues to be much debate about what a healthy diet consists of and many healthcare professionals report feeling confused about the advice they should be providing. This article discusses the debate surrounding the low-fat diet and explains some of the research into dietary fats, diabetes and cardiovascular disease.

The field of diet therapy in diabetes has undergone a series of transformations during the years. Prior to the discovery of insulin, starvation therapy was the only dietary treatment for diabetes (Mazur, 2011). People with diabetes were then encouraged to adhere to a strict diet high in fat and low in carbohydrate (Newburgh and Marsh, 1921) in recognition of the fact that carbohydrate is the only nutrient that has a direct impact on blood glucose levels (Sheard, 2004). With the increasing prevalence of cardiovascular disease (CVD) and the diet-heart hypothesis (Keys, 1997), guidelines for people with diabetes were updated in the 1980s to recommend a low-fat and high-carbohydrate diet in line with the diet recommended to the general population (National Advisory Committee on Nutritional Education, 1983).

However, the low-fat diet was introduced in the absence of randomised controlled trial (RCT) evidence (Harcombe et al, 2015) and a recent meta-analysis does not support low-fat diets over other dietary interventions (Tobias et al, 2015).

Although UK dietary guidelines for diabetes still advocate a low-fat diet (NICE, 2015), Diabetes UK nutrition guidelines (Dyson et al, 2011) acknowledge the following:

- Nutrition management has shifted from a prescriptive one-size-fits-all approach to a person-centred approach.
- It is unclear what ideal proportion of macronutrients to recommend for optimal glycaemic control.
- Encouraging the individual to adopt their diet of choice may well improve outcomes. It is the degree of adherence that will predict outcomes rather than type of dietary strategy. It is intuitive that a diet an individual enjoys and finds acceptable is more likely to succeed.

Fats explained

Fats (dietary and human) are usually in the form of triglyceride. A triglyceride is three fatty acids connected with a glycerol backbone. There are three classes of fatty acids: saturated (SFA), monounsaturated (MUFA) and polyunsaturated (PUFA). In SFA, all the carbons are saturated with hydrogen, MUFAs have one double bond and PUFAs have two or more double bonds (*Figure 1*).

No foods contain just one type of fat. Animal products tend to contain mainly MUFA and SFA. For example, lard contains 43% MUFA, 40% SFA and 10% PUFA; olive and rapeseed

oils contain mainly MUFA; seeded vegetable oils, such as sunflower oil, contain mainly PUFA. There are 36 types of SFA, differing in chain length from three to 38 carbons. There are eight types of MUFA (16 to 24 carbons with one double bond) and 10 types of PUFA (18 to 22 carbons with 2–6 double bonds).

Food contains a mixture of triglycerides, with a different mix of the 54 fatty acids. Nutrition information tables on the back of food packaging often separate total fat into SFA, MUFA and PUFA. Although this demonstrates that no food contains just one type of fat (*Table 1*), it is an over simplification as it does not acknowledge that within these categories, the fatty acids can be very different (Orsavova et al, 2015).

Fatty acids have different properties based on number of double bonds, chain length and odd or even number of carbons (*Table 2*, overleaf). Foods containing more saturated fat tend to be hard at room temperature whereas foods containing more polyunsaturated fat tend to be liquid.

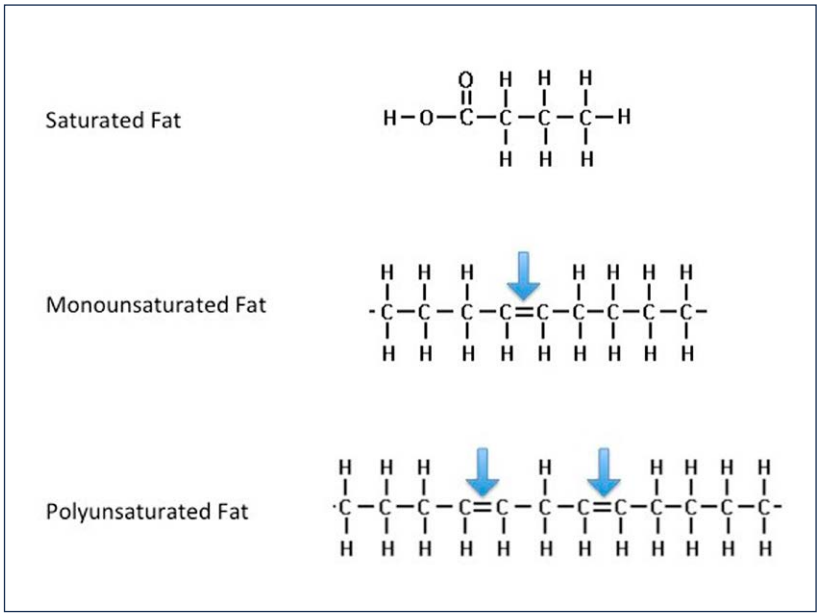

Figure 1. Molecular structure of different types of fat.

Why we are not what we eat

Human adipose tissue contains around 45–50% MUFA, 25–35% SFA and 15–20% PUFA, whereas human plasma contains 46% SFAs (Hodson et al, 2008). Cattle are fed a diet of grass and grain, but store large amounts of SFA. People with type 2 diabetes can still store excess body fat, even if they consume a low-fat diet.

Samples have been taken from adipose tissue and plasma to see if we can use them as biomarkers of dietary intake. There is a good correlation between what we eat and stored levels of omega-6 PUFA, omega-3 PUFA and odd-chain fatty acid, but no correlation for MUFA, short-chain fatty acids (SCFA), medium-chain fatty acids (MCFA) and even-chain fatty acids (ECFA) (Hodson et al, 2008).

To put this into context, the dietary intake of the essential fatty acids (PUFA omega-6 and omega-3) and the odd-chain fatty acids (OCFAs;

Table 1. Total fat and SFA, MUFA and PUFA content of "real" unprocessed foods (Roe et al, 2015).

Foods per 100g	Total fat	SFA	MUFA	PUFA
Eggs (boiled)	9.6%	2.7%	3.7%	1.5%
Beef steak (fat/lean)	9.6%	3.7%	4.2%	0.9%
Full fat yogurt	10.2%	6.8%	2.5%	0.3%
Cheddar cheese	34.9%	21.7%	9.4%	1.1%
Mackerel	22.4%	5.1%	8.3%	5.4%
Brazil nuts	68.2%	17.4%	22.4%	25.4%
Butter	82.2%	52.1%	20.9%	2.8%
Extra-virgin olive oil	99.9%	14.3%	73.0%	8.2%

SFA=saturated fatty acid; MUFA=monounsaturated fatty acid; PUFA=polyunsaturated fatty acid.

Page points

1. Processed food is often wrongly classified as saturated fat when, in reality, it is a mix of refined carbohydrates, refined vegetable oils and trans-fatty acids.
2. There is no conclusive evidence that saturated fat causes CVD. If people consume wholesome, natural foods in replacement of processed foods, their body is much more likely to obtain all the nutrients it requires.
3. Discourage the use of refined vegetable oils. Vegetable oils are not made from vegetables and there is hardly anything natural left in them by the end of the processing.

from milk and dairy foods) can be reflected in human fat stores, but other fats cannot (*Table 2*).

Is dietary saturated fat bad for us?

Processed food is often wrongly classified as saturated fat when, in reality, it is a mix of refined carbohydrates, refined vegetable oils and trans-fatty acids. A medium-sized meal from a well-known fast-food chain contains almost 1000 calories, 31g fat, but only 10% (3.1g) SFA. The same applies for snack food; the ingredients for a brand of potato crisps are "dehydrated potatoes, vegetable oils (sunflower, corn), rice flour, wheat starch, emulsifier (E471), maltodextrin, salt", resulting in only 3.1g/32g fat being SFA (10%) and the remaining largely being PUFA.

There has been some debate over whether dietary saturated fat causes CVD (Deakin and Garden, 2015). Nutritional research tends to start with observational cohort studies, but these can only show a correlation. To prove causation, we need a clinical trial. The best level of evidence is where a meta-analysis combines data from several RCTs that have met the quality criteria. A Cochrane Review is classed as the gold standard and is most often used to assess the clinical effectiveness of healthcare interventions.

There is one Cochrane Systematic Review for the "Reduction of saturated fat intake for cardiovascular disease" (Hooper et al, 2015). It is a quality review because the primary and secondary outcomes were hard endpoints (deaths and events), rather than risk factors that have not yet been proven, such as LDL-cholesterol. Fifteen RCTs met the quality criteria, involving around 59 000 individuals. The review demonstrated that reducing SFA in the diet does not reduce total mortality or CVD mortality, but suggests a relative risk reduction of 17% for CVD events. However, secondary outcomes demonstrated that reducing dietary SFA does not reduce fatal myocardial infarctions (MIs), non-fatal MIs, strokes, coronary heart disease (CHD) mortality, CHD events or diagnosis of diabetes. The 17% risk reduction for CVD events disappeared when only clinical trials that actually reduced dietary saturated fat (>52 000 individuals) were included in a sensitivity analysis. This is an excellent review, but unfortunately the conclusions do not accurately report the findings that there is no evidence that cutting down on saturated fat reduces CVD. A plausible explanation for this finding is that:

1) Many recommendations that demonise saturated fat are actually referring to processed foods that are high in PUFA and refined carbohydrates.
2) SFA is present in similar amounts in foods that are promoted and restricted (for example, a mackerel fillet, which is recommended, has 7% SFA, but a steak fillet, which is generally not recommended has only 5% SFA).
3) SFA dietary intake is not correlated with SFA content in human plasma and tissue.
4) SCFA, MCFA, OCFA appear to be protective against type 2 diabetes and CVD (Alexander et al, 2016; Praagman et al, 2016).
5) Even-chain SFA and MUFA created from *de novo* lipogenesis (DNL) have been correlated with insulin resistance and incidence of type 2 diabetes (Bigornia et al, 2016).
6) CVD is caused by inflammation, endothelium damage, clotting and repair. Oxidised LDL particles are involved in this process, but future research needs to focus on nutritional strategies that can prevent the cascade of events that result in cholesterol, the innocent backseat passenger of the LDL transporter, being caught up in atheroma.

Practical advice for people with diabetes

1. Encourage "real foods". All the foods listed in *Table 1* fall into this category. Processed foods, such as low-fat spreads, snack foods and convenience meals should be avoided. Real foods are nutrient dense and promote satiety, resulting in less snacking between meals.
2. Do not demonise saturated fat. There is no conclusive evidence that SFA causes CVD. If people consume wholesome, natural foods in replacement of processed foods, their body is much more likely to obtain all the nutrients it requires.
3. Discourage the use of refined vegetable oils. Vegetable oils are not made from vegetables and there is hardly anything natural left in

Table 2. Is there a correlation between dietary intake and fatty acids stored in adipose tissue and plasma?

Fatty acids	Properties	Sources	Correlation (Yes/No) and reason
Polyunsaturated fatty acids (PUFA): Omega-6 and omega-3 long-chain fatty acids (LCFA)	These fatty acids cannot be made in the body and are therefore called essential fatty acids. Contain 2+ double bonds. Double bonds are weak spots, easily attacked by oxygen and less stable with a lower melting point. Increases the production of highly reactive oxygen species "free radicals" that stimulate cell damage and systematic inflammation (Nair et al, 2007). LCFAs have a chain length greater than 16 carbons and these fatty acids are more likely to be stored in the adipose tissue.	Omega-6 fatty acids are typically found in seeded vegetable oils and omega-3 fatty acids in flaxseed, nuts, fatty fish and fish oils.	Yes. Cannot be made by the body. Stored in adipose tissue and plasma.
Odd-chain fatty acids (OCFA)	OCFAs have been shown to be protective against incidence of diabetes (Forouhi et al, 2014).	Can only be obtained from the diet, mainly from dairy foods.	Yes. Cannot be made by the body. Stored in adipose tissue and plasma.
Monounsaturated fatty acids (MUFA)	MUFA have one double bond and are therefore more stable than PUFA, but not as stable as SFA.	Animal fats, and olive and rapeseed oils. Also obtained via *de novo* lipogenesis (DNL)*.	No. Can be made by the body via DNL.
Short-chain fatty acids (SCFAs)	SCFAs contain less than 8 carbons and these are more likely to be used for fuel for the gut (den Besten et al, 2013).	SCFA arise from fermentation in the gut and from consumption of dairy foods.	No. Oxidised as fuel in the gut and less likely to be stored in adipose tissue and plasma
Medium-chain fatty acids (MCFAs)	MCFAs contain 8–14 carbons. Rapidly absorbed from the gut and transported to the liver where they are metabolised as fuel, promoting fat oxidation and reduced food intake (St-Onge and Jones, 2002)	Coconut oil, cheese, butter, milk and yogurt.	No. Oxidised as fuel in the liver and less likely to be stored in adipose tissue and plasma.
Even-chain fatty acids (ECFAs)	ECFAs have been strongly correlated to incidence of diabetes (Forouhi et al, 2014).	Can either be obtained through the diet or via DNL.	No. Can be made by the body via DNL.

**De novo* lipogenesis (DNL) is where fat is produced within the body usually from glucose and fructose. This mainly occurs in the liver but it can occur in every cell (Volek et al, 2012; Volk et al, 2014). Therefore, plasma levels of SFA and MUFA are more dependent on the level of carbohydrate in the diet then the level or type of fat (Sanders and Griffin, 2015).

them by the end of the processing. Many studies have raised serious concerns about these oils (Rose et al, 1965; Katragadda et al, 2010; Ramsden et al, 2010; 2013). The processing method involves pressing, heating, various industrial chemicals and highly toxic solvents (such as hexane). When heated, vegetable oil produces aldehydes at levels 20 times higher than recommended by the World Health Organization (Katragadda et al, 2010). Extra-virgin olive oil, cold-pressed rapeseed oil, butter and goose fat produce far fewer aldehydes because they are richer in MUFA and SFA, and therefore much more stable when heated. Consuming excess omega-6 PUFA from vegetable oils without also increasing omega-3 PUFA has been shown to increase the risk of CHD and death. This may be due to their pro-

"People need to understand which foods contain carbohydrate and develop the tools to easily assess foods and, if needed, adjust the amount of carbohydrate they are consuming."

inflammatory nature (Ramsden et al, 2013).

4. Promote dairy foods. These are excellent sources of SCFAs, MCFAs and OCFAs, and there is emerging evidence suggesting that higher consumption from dairy food may reduce risk of CVD and type 2 diabetes (Alexander et al, 2016; Praagman et al, 2016). However, cohort studies can only show a correlation and not prove causation. Therefore, clinical trials are required to confirm these observations.
5. Deliver carbohydrate awareness education. It is likely that people with type 2 diabetes have a different tolerance level for dietary carbohydrate. Hyperinsulinaemia and insulin resistance promote DNL (the conversion of glucose and fructose to SFA and MUFA; Bigornia et al, 2016), resulting in lipogenesis, weight gain and dyslipidaemia. People need to understand which foods contain carbohydrate and develop the tools to easily assess foods and, if needed, adjust the amount of carbohydrate they are consuming.

Conclusion

People purchase foods, not nutrients. Demonising or promoting dietary fat based on the three categories (SFA, MUFA, PUFA) is too simplistic for the following reasons:

- There are 54 types of fatty acids with different properties.
- Stored human fat in adipose tissue and plasma do not reflect dietary intake of fats.

Advising people with diabetes to consume a "real food" diet that contains nutrient-dense and satiating foods, which have been minimally processed, will benefit their health more than focusing on the amount and type of dietary fat. It is time to revise the dietary guidelines that advocate a low-fat and low-saturated fat diet. A Public Health Collaboration (https://phcuk.org) has recently been launched to address this issue. ■

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