

# Monounsaturates in the diet

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## Summary

Monounsaturates (MUFA) are not essential components of the diet as the body can synthesise all it needs to function properly. However, they are present in many foods, such as meat, dairy products and nuts, and contribute to approximately 15% of the total fatty acids in the UK diet. The most common MUFA is oleic acid, which accounts for 92% of dietary MUFA; 60–80% of this is derived from olive oil. There are no formal recommendations for MUFAs in the diet in the UK. However, a population average of 13% arises by difference when recommendations for saturates (SFA) and polyunsaturates are taken into account. Average population total fat intakes in the UK are around the recommended 35% of food energy; however, currently intake of SFAs is a little higher than recommended. Scope to address this problem exists by way of a shift from food sources of SFAs to those higher in MUFAs.

Detecting associations between diet and chronic diseases such as cardiovascular disease and cancer is not an easy task. However, there is accumulating evidence suggestive of a protective effect of a Mediterranean-style diet (*i.e.* a diet low in SFAs and rich in MUFAs) against coronary heart disease, certain cancers and type 2 diabetes. The Mediterranean diet is a complex dietary exposure and intakes of fruits, vegetables and wholegrains are also higher. However, further investigation has identified MUFAs as having independent health benefits, protecting against coronary heart disease, diabetes and cancer as well as favourably affecting a number of risk factors for these diseases including hypertension, insulin sensitivity, plasma lipoprotein concentrations and factors related to blood clotting.

To address the current imbalance of fatty acids in the UK diet, sources of SFAs could be replaced by foods containing MUFAs. This will have a twofold benefit in chronic disease outcomes, reducing cardiovascular disease risk by replacing dietary SFAs, as well as affording a degree of protection against other diseases. The extent of dietary change required is not actually that great; the substitution of 12 g of SFA with 12 g of MUFA (possible by a modest, albeit significant change in dietary patterns) in a 2200 kcal diet will increase MUFA from 14% to 19% of energy (SFA will decrease from 13% to 8%). One approach to achieve an improvement in the fatty acid profile of the diet is to alter the nutrient composition of foods that are commonly consumed so that they are 'healthier'. This way, people do not have to make radical changes to their lives and they can continue eating the foods that they

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are used to. Such approaches include the use of oils with a high oleic acid content. These have been produced by conventional breeding methods and can be easily used in the food-manufacturing industry.

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## What are monounsaturates?

Fatty acids are the building blocks from which fats (lipids) are made. Fatty acids found in foods and fats stored in the body are mainly present in the form of triacylglycerols; a glycerol molecule backbone to which three, often different, fatty acids are attached. Monounsaturated fatty acids, or monounsaturates (MUFA) are fatty acid molecules of varying chain lengths that contain a single carbon-carbon double bond. MUFAs are key components of membrane structural lipids, particularly nervous tissue myelin. Unlike some groups of fatty acids, the body is capable of synthesising all the MUFAs it requires in a process catalysed by the enzyme complex, fatty acid synthase. Thus, they are not essential components in the diet. However, MUFAs are present in many animal- and plant-derived foods and they contribute to roughly 15% of the fatty acids in the diet (see below). The main MUFAs in the diet are listed in Table 1; oleic acid 18:1(*n*-9) accounts for 92% of dietary MUFA (FNB 2005).

## Food sources of monounsaturates

Fat is found in most food groups and foods containing fat generally provide a range of different fatty acids, both saturated and unsaturated. Table 2 outlines the main sources of MUFAs in the British diet. As mentioned above, 92% of the MUFA present in foods is

oleic acid and of this, it has been estimated that 60–80% comes from olive oil. However, based on their favourable fatty acid profile, other food sources could be useful contributors to the MUFA content of the diet, especially snacks, *e.g.* nuts.

## Dietary recommendations on fat

Fat is the most energy-dense macronutrient so it is therefore important that guidelines are set to ensure that the population consumes adequate amounts of the individual fatty acids without encouraging people to consume more energy than they require. Opportunities also exist to take the health effects of the various types of fatty acids into consideration and aim to achieve a more optimal fatty acid profile. Current UK recommendations (Table 3), which were derived in 1991 by the Committee on Medical Aspects of Nutrition Policy, state that the population average for total fat consumption should not exceed 35% of daily food energy intake (or 33% of daily total energy intake *i.e.* including energy consumed as alcohol). Population averages for the different types of fatty acids are: saturated fatty acids (SFA) should not exceed 11% of daily food energy and *cis*-polyunsaturated fatty acids (PUFA) approximately 6.5%. In order to avoid the atherogenic effects of the *trans*-PUFAs, the population average should not exceed 2% of daily food energy. Thus MUFAs should make up approximately 13% of daily food energy, this value

**Table 1** Main monounsaturated fatty acids present in food

Trivial name	Systematic name	Rich dietary sources
Myristoleic (14:1 <i>n</i> -7)	<i>cis</i> -9-tetradecenoic acid	Whale blubber and dairy fats
Palmitoleic (16:1 <i>n</i> -7)	<i>cis</i> -9-hexadecenoic	Fish oil
Oleic (18:1 <i>n</i> -9)	<i>cis</i> -9-octadecenoic	Olive oil, rapeseed oil, palm oil
Elaidic (18:1 <i>n</i> -9 <i>trans</i> )	<i>trans</i> -9-octadecenoic	Partially hydrogenated fat
<i>Trans</i> -vaccenic (18:1 <i>n</i> -7 <i>trans</i> )	<i>trans</i> -11-octadecenoic	Ruminant fats
<i>Cis</i> -vaccenic (18:1 <i>n</i> -7)	<i>cis</i> -11-octadecenoic	Ruminant fats
Gondoic (20:1 <i>n</i> -9)	<i>cis</i> -11-eicosenoic	Rapeseed oil, mustard seed oil
Erucic (22:1 <i>n</i> -9)	<i>cis</i> -13-docosenoic	Mustard seed oil

Source: Sanders and Emery (2003).

**Table 2** Fat in the diet (g fat/100 g food)

	Total fat g/100 g	MUFA g/100 g	SFA g/100 g		Total fat g/100 g	MUFA g/100 g	SFA g/100 g
Olive oil	99.9	73.0	14.3	Peanuts	46.0	22.0	8.7
Sunflower oil	99.9	20.5	12.0	Hazelnuts	63.5	49.2	4.6
HO sunflower oil	99.9	85.4 <sup>†</sup>	9.4	Almonds	55.8	38.2	4.4
Safflower oil	99.9	11.9	9.7	Potato crisps	34.2	13.5	14.0
Corn oil	99.9	29.9	14.5	Lard	99.9	43.4	40.6
Soybean oil	99.9	21.2	15.6	Butter	82.2	20.9	52.1
Rapeseed oil	99.9	57.2	6.6	Beef fat	45.0	18.9	19.8
HO rapeseed oil	99.9	77.3 <sup>‡</sup>	7.0	Chicken skin	46.1	22.0	13.4
Peanut oil	99.9	44.4	20.0	Pork fat	48.6	20.9	16.4
Hazelnut oil	99.9	76.5	7.8	Lamb fat	56.3	17.5	25.5
Mayonnaise	75.6	17.9	11.4	Beef (mince)	16.2	6.9	6.9
Whole milk	4.0	0.9	2.5	Chicken (leg)	10.9	5.0	2.9
Margarine	80.0	38.9	23.4	Pork (chop)	15.7	6.4	5.5
Avocado	19.3	14.5	2.3	Lamb (chop)	11.2	3.5	4.6
Olives	11.0	7.7	1.5				

Source: FSA (2002); <sup>†</sup>Sunola oil (Allman-Farinelli *et al.* 2005); <sup>‡</sup>Natreon (Minihane & Harland 2007).  
HO, high oleic acid; SFA, saturate; MUFA, monounsaturate.

**Table 3** Adult UK dietary reference values for fat expressed as a percentage of daily food energy intake (total energy in brackets)

	Individual minimum	Population average	Individual maximum
SFA		11 (10)	
cis-PUFA		6.5 (6)	10
<i>n</i> -3	0.2		
<i>n</i> -6	1.0		
MUFA		13 (12)	
trans-PUFA		2 (2)	
Total fat		35 (33)	

Source: DH (1991).  
SFA, saturate; PUFA, polyunsaturate; MUFA, monounsaturate.

being derived 'by difference' once all other recommendations are considered. In 1991, recommendations for specific fatty acids were made only for the essential fatty acids, linoleic acid (an *n*-6 or omega-6 PUFA) and alpha linolenic acid (an *n*-3 or omega-3 PUFA), on the basis of prevention of deficiency. These fatty acids should contribute at least 1% and at least 0.2% of daily food energy respectively (DH 1991). At the time these guidelines were set, fat contributed approximately 40% of daily food energy so in order to achieve these targets, dietary modification was required (Gregory *et al.* 1990).

### Monounsaturates in the UK diet

The average daily intakes of fatty acids as reported in the National Diet and Nutrition Survey of adults aged

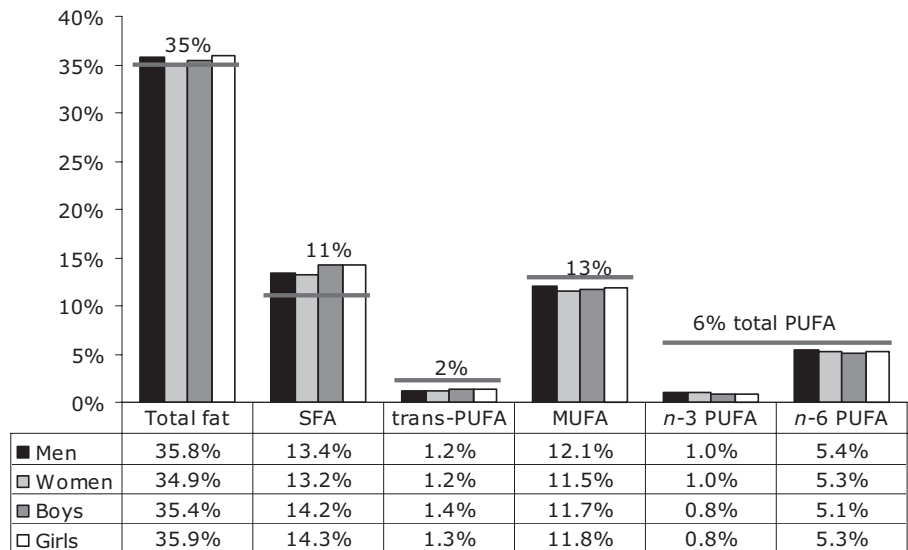
**Table 4** Total daily amount of fat (g) in the diets of adult men and women (amount as percentage of food energy in brackets)

	Men <sup>†</sup>	Women <sup>†</sup>	Boys <sup>‡</sup>	Girls <sup>‡</sup>
Total fat	86.5 (35.8)	61.4 (34.9)	74.4 (35.4)	63.1 (35.9)
SFA	32.5 (13.4)	23.3 (13.2)	29.8 (14.2)	25.2 (14.3)
trans-PUFA	2.9 (1.2)	2.0 (1.2)	2.9 (1.4)	2.4 (1.3)
MUFA	29.1 (12.1)	20.2 (11.5)	24.6 (11.7)	20.6 (11.8)
<i>n</i> -3 PUFA	2.3 (1)	1.7 (1)	1.8 (0.8)	1.5 (0.8)
<i>n</i> -6 PUFA	12.9 (5.4)	9.4 (5.3)	10.7 (5.1)	9.3 (5.3)

Source: <sup>‡</sup>Gregory and Lowe (2000); <sup>†</sup>Henderson *et al.* (2003).  
SFA, saturate; PUFA, polyunsaturate; MUFA, monounsaturate.

19–64 years (Henderson *et al.* 2003) and children aged 4–18 years (Gregory & Lowe 2000) are shown in Table 4. These figures represent the most recent data available on the average daily intake of fatty acids in the UK population. Currently, 43% of men, 50% of women and 42% of children obtain less than 35% of their total food energy from fat which is indicative of an improvement in diets since the 1980s when fat contributed to approximately 42% of total food energy. As is clear from Figure 1, of greatest concern is the higher average intake of SFAs in the population (current recommendations are that the population average should be 11% of food energy). Scope to address this problem exists by way of a shift from food sources of SFAs towards those higher in MUFAs as currently intake is only approximately 11% of food energy.

**Figure 1** UK Population intakes for total fat and individual fatty acids UK population (Gregory & Lowe 2000; Henderson *et al.* 2002). SFA, saturate; PUFA, polyunsaturate; MUFA, monounsaturate. Line indicates population average recommendation (percentage food energy).



**Table 5** Percentage contribution of the main sources of dietary fat in the UK adult diet

	Total fat				SFA				MUFA			
	M†	F†	B‡	G‡	M†	F†	B‡	G‡	M†	F†	B‡	G‡
Cereals & cereal products	19	20	23	22	17	19	22	22	17	18	19	21
Milk & milk products	14	15	16	16	23	25	24	24	9	11	12	14
Fat spreads	12	11	9	10	12	11	8	8	12	11	8	8
Meat & meat products	25	20	20	18	25	19	18	17	30	24	24	20
Fish & fish dishes	3	4	2	2	2	2	1	1	3	4	2	2
Potatoes & savoury snacks	10	10	18	20	7	7	13	15	11	12	21	22
Other	17	20	22	12	14	35	14	13	18	20	14	13

Source: †Henderson *et al.* (2002); ‡Gregory and Lowe (2000).  
SFA, saturate; MUFA, monounsaturate; M, male; F, female; B, boys; G, girls.

### Major contributors to intake of monounsaturates in the UK diet

Animal products (*i.e.* meat and meat products, eggs, and milk and milk products) contribute to a substantial proportion of the fat in the diet, owing in part to their make-up, combined with the popularity of these foods in the UK (Table 5). In recent years, animal husbandry techniques have been modified to produce meat with a more favourable fatty acid profile. Initially, the aim was to reduce the SFA content, but now scientists are developing feeds that will increase the concentrations of MUFAs and the long chain *n*-3 PUFAs in meat and ultimately enhance human health (see Nugent 2005).

### The Mediterranean diet, olive oil and other dietary sources of MUFA

The traditional dietary pattern of countries bordering the Mediterranean is very different from a traditional Western diet. A working definition of the traditional Mediterranean diet is characterised by: ‘... an abundance of plant foods such as bread, pasta, vegetables, salad, legumes, fruit, nuts; olive oil as the principal source of fat; low to moderate amounts of fish, poultry, dairy products and eggs; only little amounts of red meat; low to moderate amounts of wine, normally consumed with meals. This diet is low in saturated fatty acids, rich in carbohydrate and fibre, and has a high content of monounsaturated fatty acids. These are primarily

derived from olive oil' (see Willett 2006). Although the diets of Mediterranean countries can vary, MUFAs typically account for 16–29% of energy intake (Kris-Etherton 1999). Indeed, there is much research on the anecdotal health benefits of olive oil consumption and a clear pattern is emerging of reductions in the risk of cardiovascular disease (CVD) when a Mediterranean-type dietary pattern is followed (see Alonso *et al.* 2006; Covas 2007). Olive oil is also a very good source of so-called antioxidant components, mainly in the form of hydrophilic phenolic compounds and vitamin E which may also play a role in determining an individual's disease risk (see Perez-Jimenez *et al.* 2005; Wahle *et al.* 2004). However, the Mediterranean diet is different from a traditional Western diet for more reasons than solely its olive oil/MUFA content. Intakes of fruits, vegetables and wholegrains are significantly higher in the countries bordering the Mediterranean and thus there might be other components of the diet or even the lifestyle that are bringing about the observed health benefits.

In the last 20 years, novel oils such as high oleic rapeseed, safflower and sunflower oils that have resulted from traditional breeding techniques have appeared on the market. These oils offer higher stability during processing and storage because of lower fatty acid unsaturation making them less prone to oxidation and off-flavour development. Consequently, they are becoming increasingly popular for use by the food industry as a replacement for oils high in undesirable saturated or *trans* fatty acids.

### Monounsaturates and health

Detecting associations between nutrition and chronic diseases such as CVD and cancer is not an easy task. Consequently, a number of different approaches have been taken by researchers attempting to link dietary factors with disease states. There are four main types of research: mechanistic, epidemiological, biomarker trials and clinical trials. Each approach has its own strengths and weaknesses, and individuals tackling research in this area often have to make compromises when selecting which method to use.

The published studies on MUFAs and health have used various approaches, so it is very important to take into account the relative importance of each of the different types of evidence when generating a consensus opinion on the health effects of diet. In general, randomised controlled trials with well-defined, clinical endpoints are considered to be the 'gold standard', but in reality are very difficult to conduct. Nutrition research

does not especially lend itself to this form of study, as diet as an exposure is difficult to randomise in an effective way particularly throughout the long periods of time required to investigate effects on disease. (Supplement trials are more easily randomised, but their effects are not always equivalent to those observed when a whole diet is consumed.) Instead, high-quality epidemiological studies, involving large cohorts of healthy individuals followed over a long period of time, provide some of the strongest evidence of any interaction, although they can, by their very nature, have many confounding factors, not least measurement bias. This is not to say that the other forms of study do not offer valuable evidence, it is just that their findings must be interpreted with caution, especially when extrapolating from mechanistic studies in the laboratory or in animal models to humans.

The World Health Organization reviewed the evidence relating dietary fat to the risk of developing a number of chronic diseases in 2003. Their findings are summarised in Table 6. As yet there are no definitive answers and it is clear that more research is required in order to fully understand the complex relationship that fat and the individual fatty acids have in influencing the risk of developing various diseases.

### Cardiovascular disease

Cardiovascular disease, including coronary heart disease (CHD), stroke and diseases of other arteries, is a major cause of early death and disability (see Frayn & Stanner 2005). Because of the large variation in risk of mortality from CHD and stroke, it is likely that behavioural risk factors play an important role in the aetiology of these diseases. It is now recognised that diet, as well as other lifestyle factors (*e.g.* alcohol consumption, smoking and physical activity), is strongly related to several of the main risk factors for CVD. The fatty acid profile of the diet has been demonstrated to be particularly important.

#### *Coronary heart disease*

In 1986, publication of the findings from the Seven Countries Study demonstrated a protective effect against CHD of a relatively high-fat diet (33–40% total energy) rich in MUFAs and low in SFAs (7–8% total energy) (Keys *et al.* 1986). Indeed, the mortality rate from CHD is very low in Mediterranean countries where olive oil is commonly consumed which is further suggestive of an inverse association between MUFA intake and death from CHD. In the Lyon Diet Heart Study, patients ran-

**Table 6** Strength of the evidence linking dietary fat and risk of chronic disease

	Decreased risk	No relationship	Increased risk
CVD			
Convincing	EPA/DHA; LA		Myristic and palmitic acids (SFA)
Probable	ALNA; oleic acid	Stearic acid	
Possible			Lauric acid (SFA)
Type 2 diabetes			
Probable			SFA
Possible	<i>n</i> -3 PUFAs		Total fat
Cancer			
Possible	<i>n</i> -3 PUFAs		Animal fats

Source: WHO/FAO (2003).

SFA, saturate; PUFA, polyunsaturate; ALNA, alpha linolenic acid; LA, linoleic acid; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; CVD, cardiovascular disease.

### Box 1 The relationship between plasma cholesterol and CHD risk

LDL-cholesterol: major, causality established; 1% increase → 1-2% increase in CHD risk

HDL-cholesterol: most probably causal, independent risk predictor; 1% increase → 3% decrease in CHD risk

Total/HDL ratio: the strongest single risk predictor; 1% increase → 1.5-3% increase in CHD risk

Source: Zock (2006).

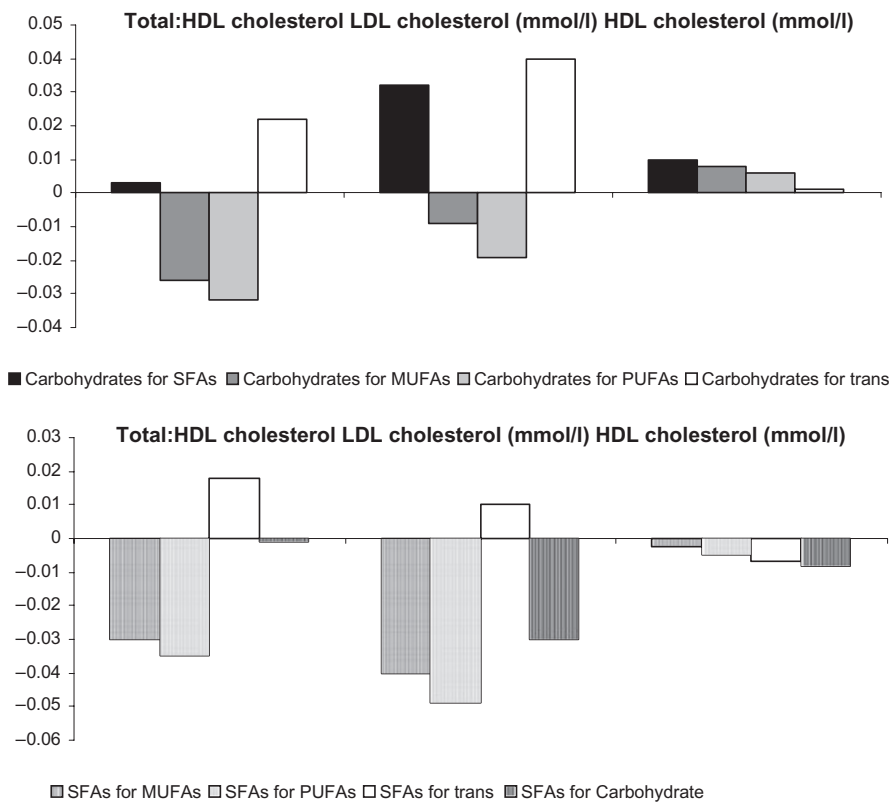
LDL, low-density lipoprotein; HDL, high-density lipoprotein; CHD, coronary heart disease.

domised to receive a Mediterranean-type diet including high MUFA had a significant reduction in the risk of death from cardiovascular causes or non-fatal acute myocardial infarction (73%), as well as in cardiac mortality (76%) and total mortality (70%) (de Lorgeril *et al.* 1994). This was much higher than was observed in studies using hypolipaeamic drugs or other dietary regimens. Similarly, the Nurses' Health Study reported that a 5% increase in energy derived from MUFA to be associated with a significant 19% reduction in risk of developing CHD in women with no prior history of CHD (Hu *et al.* 1997). However, there are a number of studies which refute an association, including the Framingham Heart Study (Posner *et al.* 1991). CHD is a complex, multifactorial disease and in an attempt to elucidate the specific effect of MUFAs on the risk of developing CHD, much research has been carried out on the major risk factors. The effects of high-MUFA diets on blood lipoproteins, endothelial function and the development of the atherosclerotic plaque are also discussed in the following sections.

**Blood lipoproteins** Raised blood cholesterol, particularly low-density lipoprotein (LDL) cholesterol, is a well-established risk factor for CHD; high-density lipoprotein (HDL) cholesterol concentrations are inversely

associated with risk, and the total/HDL ratio is the strongest single risk predictor (see Box 1). Meta-analyses report that both MUFAs and, to a greater extent, total PUFAs (*i.e.* *n*-3 and *n*-6 fatty acids together), when substituted for SFAs in the diet, reduce both total cholesterol and LDL-cholesterol and increase HDL-cholesterol (Mensink *et al.* 2003). An isocaloric substitution of MUFA and total PUFAs for carbohydrate does not have any influence on fasting TAG concentrations (Mensink & Katan 1992).

Overall, there is little to differentiate between the effects of MUFAs and PUFAs on the total/HDL-cholesterol ratio and thus the best advice with respect to CHD protection is to replace SFAs in the diet with a mixture of MUFAs and PUFAs. Work has been carried out to investigate so-called qualitative changes to the LDL particles namely oxidation. Oxidised LDLs are readily taken up by monocyte-derived macrophages. This results in the formation of foam cells which is an early event in the formation of the atherosclerotic plaque. PUFAs are particularly susceptible to oxidation by virtue of the presence of multiple carbon-carbon double bonds. It has been shown that increasing the MUFA content of the diet, at the expense of PUFAs, results in LDL particles



**Figure 2** Changes in the plasma concentration of lipoproteins in the context of dietary macronutrient substitutions (Mensink *et al.* 2003). LDL, low-density lipoprotein; HDL, high-density lipoprotein; SFA, saturate; PUFA, polyunsaturate; MUFA, monounsaturate.

that are less susceptible to oxidation *in vitro* (Reaven 1995). However, it has not yet been established whether this is related to CHD risk (Kris-Etherton 1999). Few studies have assessed the effects of high-carbohydrate diets *vs.* high-MUFA diets with respect to oxidation of LDL. Most show that the high-MUFA diet results in either a reduced susceptibility of LDL to oxidation or no effect, although the antioxidant effect of MUFA-rich foods and oils may have contributed to this effect (*e.g.* Perez-Jimenez *et al.* 1999) (Fig. 2).

**Endothelial function** There has been much interest in the role of the endothelium (the layer of cells lining the blood vessels) in CVD over recent years (see Frayn & Stanner 2005). The endothelium is a selective barrier between the blood and the underlying tissue, which nutrients, solutes, hormones, macromolecules and leucocytes (white blood cells) can cross. The endothelium has many functions including: control of vascular homeostasis; maintenance of blood flow through vessels in response to blood borne and locally produced chemical and physical stimuli; regulation of vessel tone and haemostasis; and regulation of coagulation and fibrinolysis and thrombosis.

MUFAs have beneficial effects on endothelium-dependent dilatation as determined by the measurement

of markers of endothelial function *in vitro* (see Sattar & Ferns 2005). Few studies have investigated their influence *in vivo*. A MUFA-rich meal has been shown to impair short-term endothelial function (3 hours after test meal) in healthy subjects (Ong *et al.* 1999). However, in the longer-term, beneficial effects of a MUFA-rich diet have been reported. Fuentes and colleagues reported an improvement in endothelial function in individuals with hypercholesterolaemia receiving a MUFA-rich Mediterranean diet compared with a diet high in saturates (Fuentes *et al.* 2001). Similarly, Esposito *et al.* (2004) reported long-term beneficial effects of a MUFA-rich Mediterranean diet in individuals with metabolic syndrome.

**Haemostasis** The haemostatic system maintains blood in the fluid state, controls bleeding by coagulation and plays a role in tissue repair. The system involves a cascade of events leading to the formation of a fibrin clot. The effect of the different dietary fatty acids on the haemostatic system is complex, and more research is needed before there is a clear picture. Studies investigating the chronic and acute effects of MUFAs on haemostasis are few in number and inconsistent in findings (see Miller & Bruckdorfer 2005). Some of the main findings are summarised in Table 7 but, again, there is a discrepancy

**Table 7** Effects of a high monounsaturate intake in humans on haemostatic markers

Haemostatic parameter	Expected changes following a high MUFA diet
Platelet aggregation	↓ aggregation; ↓ thromboxane production (especially marked when olive oil consumed; Karantonis <i>et al.</i> 2002)
Coagulation	↓ Factor VII, XI and X plasma concentrations (Oakley <i>et al.</i> 1998; Sanders <i>et al.</i> 1999)
Fibrinolysis	↓ plasminogen activator inhibitor (again, especially marked when olive oil consumed; Avellone <i>et al.</i> 1997)

Source: Lopez-Miranda *et al.* (2006).

MUFA, monounsaturate.

### Box 2 The relationship between blood pressure and CVD risk

For people aged 40–69 years:

Systolic BP: each increase of 20 mmHg is associated with a two-fold difference in the stroke death rate, and with two-fold differences in the death rates from IHD and from other vascular causes of death

Diastolic BP: each increase of 10 mmHg is associated with a two-fold difference in the stroke death rate, and with two-fold differences in the death rates from IHD and from other vascular causes of death

Source: Lewington and Clarke (2005).

between studies where the MUFA source is high oleic acid seed oils compared with studies in Mediterranean populations, where olive oil is the main MUFA source.

**Postprandial lipaemia** Postprandial lipaemia is defined as the extent and duration of the rise in plasma triacylglycerides (TAG) after a fatty meal. During this period, the body's ability to metabolise TAG is challenged, and if the body is incapable of processing them efficiently, circulating TAG-rich lipoproteins can make a significant contribution to the development of atherosclerosis (see Karpe 2005).

Some postprandial studies have suggested that meals containing large amounts of MUFAs cause exaggerated lipaemia (Sanders *et al.* 1999) although adverse effects have not been seen when subjects habitually consume a diet high in olive oil, *e.g.* those consuming a Mediterranean diet (Roche *et al.* 1998). In general, there is a lower, less attenuated late postprandial lipaemic response. Therefore, although the postprandial profiles are different, MUFA-rich diets generally give a more favourable response (Silva *et al.* 2003).

### Stroke

Despite the wealth of scientific literature on the influence of unsaturated fatty acids on CHD, relatively few studies have investigated their influence on stroke risk. Even fewer studies have investigated the influence of unsaturated fatty acids on the different types of stroke: ischaemic stroke and haemorrhagic stroke.

The measurement of different classes of unsaturated fatty acids in serum, plasma or red blood cells of ischaemic stroke cases and of controls suggests that higher proportions of MUFAs are associated with a reduced risk. However, the numbers of cases identified in some of these studies have been small (Ricci *et al.* 1997).

**Blood pressure** Hypertension (elevated blood pressure) is an established risk factor for CVD, strongly predicting risk of stroke (see Box 2). As blood pressure increases, so does CVD risk; in the Seven Countries Study for every 10 mmHg rise in systolic blood pressure and every 5 mmHg rise in diastolic blood pressure the risk of all cause mortality rose by 28% in both normotensive and hypertensive individuals (van den Hoogen *et al.* 2000). Epidemiological studies report an inverse association between MUFA intake and blood pressure (see Table 8). However, the association is stronger when the source of the MUFAs is olive oil.

A recent meta-analysis has assessed the effectiveness of the substitution of SFAs in the diet with either carbohydrates or MUFAs on blood pressure (Shah *et al.* 2007). Based on the findings of 10 intervention trials, the authors concluded that diets rich in carbohydrates may be associated with slightly higher blood pressure [+2.6 (95% CI 0.4–4.7) mmHg systolic, 1.8 (95% CI 0.01–3.6) mmHg diastolic] than diets rich in MUFAs. However, they concluded that the magnitude of the difference may not be enough to justify making recommendations to alter the carbohydrate and MUFA content of the diet to manage blood pressure. Indeed,

**Table 8** Epidemiological studies assessing the association between monounsaturate intake, olive oil consumption and hypertension

Study	Country	Study design	Participants	Main findings
Nurses' Health Study (1996)	USA	Cohort	58218 women 34–59 years	No effect on hypertension
Health Professionals' Follow-up Study (1992)	USA	Cohort	30681 men 40–75 years	No effect on hypertension
Chicago Western-Electric Company Study (1987)	USA	Cohort	1714 men 40–55 years	MUFA intake associated with increased BP
MRFIT (1996)	USA	Cohort	11342 men 35–57 years	No effect on BP
Williams <i>et al.</i> (1987)	USA	Cross-sectional	76 men 30–55 years	MUFA intake inversely associated with BP
Nine Communities Study (1990)	Italy	Cross-sectional	4903 men & women 20–59 years	Olive oil consumption inversely associated with BP
EPIC-Greece (2004)	Greece	Cross-sectional	20343 men & women 20–86 years	Olive oil consumption inversely associated with BP
SUN Study (2004)	Spain	Cohort	6863 men & women 20–80 years	Olive oil consumption inversely associated with hypertension in men

Source: Alonso *et al.* (2006).

MUFA, monounsaturate; BP, blood pressure.

**Table 9** Net change in plasma lipid concentrations in individuals with type 2 diabetes consuming a high monounsaturated fatty acid diet (>25% energy) compared with a high carbohydrate diet

	No. subjects (studies)	Change (mmol/l)	% change
Total cholesterol	133 (9)	-0.15 (-0.24, -0.06)	-3.0
TAG	133 (9)	-0.36 (-0.43, -0.29)	-19.0
LDL-cholesterol	105 (6)	-0.01 (-0.10, 0.08)	0
HDL-cholesterol	133 (9)	0.05 (0.03, 0.07)	4.0

Source: Garg (1998a).

LDL, low-density lipoprotein; HDL, high-density lipoprotein; TAG, triacylglyceride.

the authors of an early intervention study remarked that in healthy normotensive individuals, increasing the proportion of MUFAs in the diet will have only minor effects, if indeed any, on blood pressure (Mensink *et al.* 1988).

### Type 2 diabetes and glycaemic control

There is now much interest in the fatty acid profile of the diet, the effect that this can have on the risk of type 2 diabetes and its management as well as improvements in insulin sensitivity and glycaemic control.

#### Type 2 diabetes

Observational studies support the hypothesis that unsaturated fatty acids are protective with respect to

type 2 diabetes (Salmerón *et al.* 2001; Louheranta *et al.* 2002). However, once diagnosed, diet can still play a role in the management of the condition. Current dietary advice is very similar to that for the general population. With respect to fat, a low-fat diet is recommended with a restriction on the SFA content of the diet. The rationale behind such recommendations is based on the observed increased risk of CVD experienced by individuals with type 2 diabetes. However, as shown in Table 9, in many studies, high-fat diets enriched with MUFAs have shown to improve anthropometric and metabolic parameters, especially in relation to blood lipid concentrations, in individuals with type 2 diabetes. Such diets have consequently been suggested as an alternative to high-carbohydrate diets with a high-fibre content and low glycaemic index (see Garg 1998a).

Additionally, there is other evidence to support the use of low-fat diets for the management of type 2 diabetes. It has been demonstrated that a low-fat, high complex carbohydrate diet caused weight loss in individuals with type 2 diabetes, which was not observed in individuals fed with a high-MUFA diet (Gerhard *et al.* 2004). The low-fat diet did not cause the plasma TAG concentrations to increase and did not worsen glycaemic control. Thus, the researchers concluded that such diets may be very useful in the dietary management of type 2 diabetes owing to the improvement in metabolic control known to occur when excess weight is lost (Gerhard *et al.* 2004). Again it is clear that more research is needed in this area to determine whether the ratio of unsaturated fatty acids is an important consideration when recommending low-fat diets to individuals with diabetes.

#### *Insulin sensitivity*

Few studies have attempted to address the issue of insulin resistance with respect to fat quality, and many that have reported are observational studies focusing on plasma fatty acid concentrations and insulin action. Only two out of 10 short-term studies comparing low-fat and high-MUFA diets in individuals with type 2 diabetes have measured insulin sensitivity, and these have demonstrated no marked effect (Garg 1998b). However, improvements have been observed in other studies where MUFAs have been included at the expense of SFAs (Vessby *et al.* 2001). Overall, there is currently insufficient evidence to draw conclusions about the MUFA content of the diet and insulin sensitivity, although it is an area of considerable research interest.

#### *Glycaemic control*

A low-fat diet (21% energy from fat) and a high-MUFA diet (35% energy from fat) appear to afford similar glycaemic control (*e.g.* Luscombe *et al.* 1999). This was assessed by fasting glucose concentrations and glycosylated haemoglobin (a marker of longer-term glycaemic control). In light of similar findings reported by other researchers, it has been suggested that provided the amount of SFAs in the diet is low, a high-MUFA diet has a similar effect on glycaemic control as a high-carbohydrate/low-fat diet and may confer some additional improvement in CHD risk factors (see Ros 2003).

#### **Weight control**

Overweight and obesity are perhaps the most pressing public health issues facing the Western world today and

hence much research effort is being invested in identifying lifestyle patterns that can: (i) reduce the risk of becoming overweight, (ii) facilitate weight loss and (iii) improve the metabolic profile of overweight and obese individuals. The overriding theme when looking at any work that is being conducted in this area is to remember the importance of balancing energy intake with regular exercise, as a dietary pattern alone will never be able to fully address the metabolic disturbances often associated with excess weight.

In common with new approaches to the treatment and management of other chronic diseases, fat quality is viewed as being as important, if not more than, fat quantity. Fat is the most energy-dense macronutrient, contributing 9 kcal/g. However, despite an obvious reduction in energy intake when low-fat diets are followed, there is an argument that a reduced energy/moderate fat/high-MUFA diet brings about weight loss, decreases CVD risk and facilitates long-term adherence, minimising weight fluctuations which adversely affect CVD risk (Gumbiner *et al.* 1998; Yu-Poth *et al.* 1999; Pelkman *et al.* 2004). This could be because the higher fat diet is more satiating although more work needs to be done in this area to elucidate any possible mechanisms.

#### **Cancer**

The work of Ancel Keys established that the major explanatory factor for the differences in cancer rates seen in the regions bordering the Mediterranean Sea was the plant-based Mediterranean diet (Keys *et al.* 1986). Trichopoulou *et al.* (2000) estimated that up to 25% of new colorectal cancer cases, 15% of new breast cancer cases and 10% of new prostate cancer cases could be prevented if 'Western' countries adopted a Mediterranean-style diet; that is a diet rich in fruits, vegetables, fish, legumes, wholegrains and, most notably, oleic acid-rich olive oil. Because of this, it was thought that MUFAs might have a protective effect against the development of cancers. *In vitro* studies have suggested that MUFAs reduce the occurrence of molecular biomarkers typically associated with carcinogenesis, *e.g.* lipid peroxidation damage or oxidative stress.

The 1997 report of the World Cancer Research Fund, entitled *Food, Nutrition and the Prevention of Cancer: A Global Perspective*, concluded that there was a small amount of evidence that MUFAs might influence cancer risk based on the strength of the evidence from epidemiological, intervention and experimental studies (WCRF 1997).

## Optimising intakes of unsaturated fatty acids

Improving the fatty acid make-up of the UK diet requires a shift in the proportions of the fatty acids that constitute our diet occurring alongside a decrease in total fat consumption. Given the evidence presented in this report, along with other known health benefits of unsaturated fatty acids (see Lunn & Theobald 2006), MUFAs, *n*-6 and *n*-3 PUFAs should be substitutes for dietary SFAs in our diets, rather than consumed in addition. Based on current data, intakes of PUFAs are within recommended levels (1–10% of total energy). Furthermore, there is no upper limit for intake of MUFAs and no evidence of adverse effects from consuming a high (>25% energy) MUFA diet. Regardless of the nature of the ‘good fats’ used to substitute the SFAs in the diet, it is important that they are not promoted in such a way that people may actually increase their total fat intakes, while believing that they are making positive dietary changes for their future health.

## Impact of increasing monounsaturates in the diet at the expense of saturates

In the UK diet, average intakes of SFAs are higher than those recommended by health professionals for a healthy diet (see Fig. 1). In agreement with evidence from Keys *et al.* SFAs increases risk of CHD by approximately 17% (RR 1.17; 95% CI 0.97–1.41) for every 5% increment in energy from SFAs; as do *trans* fatty acids, almost doubling CHD risk (RR 1.93; 95% CI 1.43–2.61) for every 2% increment in energy from *trans* fatty acids when compared with carbohydrate (Key *et al.* 1986). Thus by minimising the amounts of these fatty

acids in the diet, one could expect this to have an impact on the incidence of CHD in the population. (Although it is important to note that CHD is a complex disorder with many other factors contributing to an individual’s risk of developing the disease.) Conversely, MUFAs reduce CHD risk (Kris-Etherton 1999) and evidence is accumulating that they can reduce the risk of other diseases such as type 2 diabetes and perhaps even some types of cancer (Table 10). Thus substituting SFAs with MUFAs will have a twofold benefit in chronic disease outcomes, reducing risk by replacing dietary SFAs, as well as affording a degree of protection against other diseases in their own right. The extent of dietary change required is not actually that great; the substitution of 12 g of SFAs with 12 g of MUFAs (possible by a modest, albeit significant change in dietary patterns) in a 2200 kcal diet will increase MUFAs from 14% to 19% of energy (SFAs will decrease from 13% to 8%).

One approach to achieve an improvement in the fatty acid profile of the diet is to alter the nutrient composition of foods that are commonly consumed so that they are ‘healthier’. In this way, people do not have to make radical changes to their lives and they can continue eating the foods that they are used to. There are a number of ways that this can be achieved. It is possible to alter the fatty acid profile of foods such as meat or milk by changing the composition of the animal feed. In this way, the amount of SFAs is reduced, and the amount of MUFAs and PUFAs, which are better for heart health and other chronic diseases, is increased. Similarly, conventional breeding methods have resulted in the production of oils with a high oleic acid content which can be used in the food manufacturing industry. Use of these oils, along with other reformulation strategies in common products including spreads and mayonnaise,

**Table 10** Expected health effects with the replacement of dietary saturates by monounsaturates

Level of evidence	Type of effect
Demonstrated by dietary intervention trials in different populations	<ol style="list-style-type: none"> <li>1. A more favourable lipid profile, with a decrease in LDL-cholesterol plasma concentrations. Moreover, HDL-cholesterol is higher than with replacement by carbohydrate.</li> <li>2. Reduction in <i>in vitro</i> oxidation of LDL-cholesterol.</li> <li>3. Improvement of glucose metabolism in normal subjects and those with type 2 diabetes. MUFAs result in a lower insulin requirement and plasma glucose concentration compared with replacement by carbohydrate.</li> </ol>
Suggested by few dietary intervention trials or with <i>in vitro</i> experiments	<ol style="list-style-type: none"> <li>1. Reduced activation of monocytes by oxidised LDL-cholesterol.</li> <li>2. 3–10% reduction in systolic and diastolic BP in normotensive and hypertensive subjects.</li> <li>3. Changes in arterial wall components.</li> <li>4. The promotion of a less prothrombotic environment influencing different thrombogenic factors.</li> </ol>

LDL, low-density lipoprotein; HDL, high-density lipoprotein; BP, blood pressure; MUFAs, monounsaturates.

Source: Lopez-Miranda *et al.* (2006).

has the potential to incorporate more favourable fats in many commercial foods such as snacks and pre-prepared meals.

More research needs to be carried out to gain a more complete understanding of the public health impact of increasing MUFAs in the UK diet. In the meantime, researchers are working to better understand the potential impact on intakes and health outcomes, of switching to a diet with a healthier fatty acid profile. Recent estimates predicted how modest changes in the oil used to fry foods, such as crisps, could impact on average intakes of SFAs and MUFAs in the UK population (Minihane & Harland 2007). The authors of this paper conclude that the use of alternative novel oils, such as the high oleic acid sunflower or rapeseed oils, in place of traditional oils used in food manufacturing, provides a realistic means of reducing population SFA and *trans* fatty acid intake.

## Conclusion

There is strong epidemiological evidence that dietary MUFAs have a beneficial effect on the risk of CHD, diabetes and possibly even cancer. Moreover, evidence from controlled clinical studies has shown that MUFAs favourably affect a number of risk factors for CHD, including plasma lipids and lipoproteins, factors related to thrombogenesis, LDL oxidative susceptibility (compared with PUFA), and insulin sensitivity. Compared with SFAs, MUFAs lower total and LDL-cholesterol levels, and relative to carbohydrate, they increase HDL-cholesterol levels and decrease plasma TAG levels. Similarly, a diet high in MUFA (*vs.* a high-carbohydrate diet) improves glycaemic control in individuals with type 2 diabetes and there may be benefits for certain individuals with diabetes from following a high-MUFA diet.

In America, health professionals have recommended a diet that provides as much as 15% of energy from MUFAs, approximately 7% from PUFAs and 8% from SFAs as an option for those who wish to reduce their risk of developing CHD. Currently, in the UK, there are no specific recommendations about MUFAs and public health messages are focused on the need to reduce total fat as well as the SFAs in the diet. Based on the evidence presented in this report, increasing the amounts of MUFAs in the diet could help to improve the fatty acid profile of the UK diet and most likely have an impact on the incidence of chronic diet-related diseases. Indeed, recent innovations in the food industry, such as the use of high oleic acid oils, may represent an opportunity to bring about such improvements without consumers having to make substantial changes to their diets.

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