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The role of animal nutrition in improving the nutritive value of animal-derived foods in relation to chronic disease

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Foods derived from animals are an important source of nutrients in the diet; for example, milk and meat together provide about 60 and 55% of the dietary intake of Ca and protein respectively in the UK. However, certain aspects of some animal-derived foods, particularly their fat and saturated fatty acid (SFA) contents, have led to concerns that these foods substantially contribute to the risk of CVD, the metabolic syndrome and other chronic diseases. In most parts of Europe dairy products are the greatest single dietary source of SFA. The fatty acid composition of various animal-derived foods is, however, not constant and can, in many cases, be enhanced by animal nutrition. In particular, milk fat with reduced concentrations of the $C_{12–16}$ SFA and an increased concentration of 18:1 MUFA is achievable, although enrichment with very-long-chain *n*-3 PUFA is much less efficient. However, there is now evidence that some animal-derived foods (notably milk products) contain compounds that may actively promote long-term health, and research is urgently required to fully characterise the benefits associated with the consumption of these compounds and to understand how the levels in natural foods can be enhanced. It is also vital that the beneficial effects are not inadvertently destroyed in the process of reducing the concentrations of SFA. In the future the role of animal nutrition in creating foods closer to the optimum composition for long-term human health is likely to become increasingly important, but production of such foods on a scale that will substantially affect national diets will require political and financial incentives and great changes in the animal production industry.

Chronic disease: Foods from animals: Animal nutrition

The burden of chronic disease is rapidly increasing worldwide. Recent data (World Health Organization/Food and Agriculture Organization, 2003) suggest that in 2001 chronic diseases were responsible for about 60% of the 56·5 million deaths reported worldwide and about 46% of the global burden of disease. It has been projected that by 2020 chronic diseases will account for approximately 75% of all deaths worldwide. Approximately half the total deaths from chronic disease are attributable to CVD, but the rapid increase in the obesity–type 2 diabetes syndrome is particularly worrying, not only because it already affects a large proportion of the population worldwide, but also because it is now starting to affect individuals earlier in life. Also, contrary to popular belief, developing countries are increasingly being affected by chronic disease (World Health Organization, 2002).

It has been known for many years that diet plays a key role as a risk factor for chronic disease. At a global level it is clear that in the second half of the twentieth century there were very major changes to diet in the developed world and, more recently, in developing areas. Often largely plant-based diets have been replaced by an increased consumption of animal products, with a consequent increase in fat content and hence energy density. It is mainly because of the composition of many animal fats that their increased consumption has been associated with increased chronic disease. The present paper will examine the opportunities to improve the composition of animal fats through animal nutrition and will also discuss how these improvements need to be rationalised against other evidence that points to benefits from the consumption of animal-derived foods.

Abbreviations: CLA, conjugated linoleic acid; SFA, saturated fatty acids.
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Table 1. Trends in consumption of animal products (from World Health Organization/Food and Agriculture Organization, 2003)

	Meat (kg/*per capita* per year)			Milk (kg/*per capita* per year)		
Region	1964–6	1977–99	2030*	1964–6	1977–99	2030*
World	24·2	36·4	45·3	73·9	78·1	89·5
Developing countries	10·2	25·5	36·7	28·0	44·6	65·8
Transition countries	42·5	46·2	60·7	156·7	159·1	178·7
Industrialised countries	61·5	88·2	100·1	185·5	212·2	221·0

*Projected values.

Contribution by animal products to diets

The demand for animal products is growing globally at a substantial rate, driven by a combination of population growth, urbanisation and rising income. Table 1 shows the trends in meat and milk consumption over the past 40 years for various parts of the world. For large parts of the population animal products represent a source of high-quality protein, but high intakes of particular animal products can lead to excessive fat intakes. From the late 1960s to the late 1990s fat intake *per capita* rose from 53 to 73 g/d worldwide and from 117 to 148 g/d within the EU (World Health Organization/Food and Agriculture Organization, 2003). Interestingly, data from the UK National Food Survey (Department for Environment Food and Rural Affairs, 2001) show that over the same period, intakes of total fat and saturated fatty acids (SFA) respectively fell from 120 and 57 g *per capita* per d in 1968 to 74 and 29 g *per capita* per d in 2000.

Foods of animal origin make a major contribution to the UK diet. Although in the UK the consumption of whole milk and meat from ruminant animals has declined during recent years, the amounts of skimmed milk and poultry meat in the typical diet have substantially increased (Department for Environment Food and Rural Affairs, 2001). The contribution of various animal-derived foods to key aspects of the UK diet in 2000 (expressed as a percentage of the total dietary intake) is shown in Table 2. These data clearly show that these food sources make a major contribution to protein and Ca intake and a sizeable contribution to the intake of Fe. As the amount of nutrients derived from eggs and fish and marine products are relatively small, these food sources will not be considered.

Table 2. Contribution (%) of animal products to mean daily adult intake of energy, protein, fat, calcium and iron in the UK*

Nutrient	Milk and milk products	Meat and meat products	Eggs	Fish and fish and marine products	Total excluding fish
Energy	12	15	2	3	29
Protein	16	36	3	7	55
Fat	18	23	4	3	45
Ca	54	5	1	0·4	60
Fe	1·3	16	2·6	0·5	20

*Data derived from a combination of the National Food Survey (Department for Environment Food and Rural Affairs, 2001) and Food Standards Agency (2003).

Table 2 also indicates that while the supplies of energy from milk and its products and those from meat are approximately equal, milk is a major source of Ca and meat (predominantly red meat) makes an important contribution to protein and Fe in the diet. Overall, animal-derived foods contribute about 30% of the total energy intake, but a high proportion (0·51) of this energy is derived from fat. Since the lipids in milk and dairy products, and to a lesser extent those in the meat of ruminant animals, contain relatively large amounts of SFA (Table 3), these products make a major contribution to SFA intake. A study of fatty acid intake across Europe (Hulshof *et al*. 1999) has shown that milk and milk products (including cheese and butter) and meat and meat

Table 3. Typical fatty acid composition (g/100 g total fatty acids) of fats in animal products (adapted from McCance & Widdowson, 1998)

Fatty acid	Cow's milk	Beef	Chicken meat (white)
4:0	3·88	0	0
6:0	2·49	0	0
8:0	1·39	0	0
10:0	3·05	0	0
10:1	0·28	0	0
11:0	1·39	0	0
12:0	4·16	0	0
14:0	11·4	2·70	0·99
14:1 (*n*-5)	1·11	0·54	0·00
15:0	1·11	0·54	1·98
16:0	29·4	26·2	21·8
16:1 (*n*-9)	1·94	4·05	3·96
17:0	0·55	1·08	0·99
17:1	0·28	1·08	0·99
18:0	11·4	16·0	6·93
18:1 (*n*-9)	21·9	39·7	39·6
18:1 *trans* (*n*-7)	0·28	3·51	2·97
18:2 (*n*-6)	1·94	2·97	15·8
18:3 (*n*-3)	0·55	0·81	1·98
20:0	0·00	0·00	0·00
20:5 (*n*-3)	0·00	0·27	0·99
22:5 (*n*-3)	0·83	0·54	0·99
Summary			
Total SFA	70·1	46·5	32·7
Total MUFA	25·8	48·9	47·5
Total PUFA	3·32	4·59	19·8
n-6 PUFA	1·94	2·97	15·8
n-3 PUFA	1·39	1·62	3·96

SFA, saturated fatty acids.

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products are major sources of SFA in all countries. Dairy products are consistently the greatest source, with the highest intakes being found in Germany and France where approximately 60% of the SFA intake is derived from this source (in the UK the contribution is approximately 40%). Interestingly, the contribution of butter to SFA intake varies widely; in Greece, Spain, The Netherlands and Norway butter provides >5% of the SFA intake, in France and Germany the contribution is high (30 and 39% respectively) and in the UK it is intermediate (10%).

Dietary fatty acids and chronic disease

The relationship between dietary fat type and intake and CVD (particularly CHD) has been extensively investigated, and strong and consistent associations have been reported from a wide body of data (Kris-Etherton *et al*. 2001). Although SFA generally raise total cholesterol and LDL-cholesterol, individual fatty acids in this family have markedly different effects. In particular, myristic acid (14:0) and palmitic acid (16:0) have been associated with elevated plasma LDL-cholesterol concentrations in human subjects (for example, see Katan *et al*. 1995; Temme *et al*. 1996), while the other major SFA in foods, stearic acid (18:0), has been shown to be essentially neutral (Bonanome & Grundy, 1988). Some studies suggest that lauric acid (12:0) and myristic acid exert more potent effects on plasma cholesterol than palmitic acid (Zock *et al*. 1994), but other research has indicated that myristic acid and palmitic acid are more potent than lauric acid (Denke & Grundy, 1992). However, palmitic acid is quantitatively the most important SFA in milk and meat fat (Table 3). Most of the lauric acid and myristic acid in the human diet is derived from milk fat (Gunstone *et al*. 1994), and the consumption of whole milk and dairy products would therefore be expected to have adverse effects on plasma cholesterol levels. The results from a large longitudinal cohort study (Steffen & Jacobs, 2003) of 2778 black and white men and women, initially aged 18–30 years old, appear to support this concept. In this study diet was assessed over a 7-year period and the various plasma cholesterol fractions measured. Plasma LDL-cholesterol concentrations were found to increase by 0·078 mmol/l across all quintiles of high-fat dairy intake ($P < 0·05$), although it was suggested that after correction for within-subject errors in dietary assessment the true mean increase is likely to be three to six times greater (0·26–0·47 mmol/l). Furthermore, it was also observed that consumers of low-fat milk have lower HDL-cholesterol levels and consumers of cream and butter have higher HDL-cholesterol levels.

When substituting for SFA in metabolic studies both MUFA and PUFA lower plasma total cholesterol and LDL-cholesterol. Although the cholesterol-lowering response to PUFA is greater than that of MUFA, there has been some caution in recommending high-PUFA diets because of potentially-adverse health effects of their lipoperoxidation products (Williams, 2000). Three important *n*-3 PUFA are EPA (20:5*n*-3) and DHA (22:6*n*-3) and α-linolenic acid (18:3*n*-3). There is evidence from a range of epidemiological data and from intervention studies in populations at risk of CHD that indicate that EPA and DHA have substantial cardio-protective actions. These data have led to a widespread belief that there should be small increases in *n*-3 PUFA intake (for details, see Williams, 2000). In general, fats in animal-derived foods are very poor sources of *n*-3 PUFA.

Most of the interest in high intakes of SFA has been focused on the increase in plasma LDL-cholesterol levels and associated increases in CVD risk. However, there is now evidence that high intakes of SFA may also be related to reduced insulin sensitivity, which is a key factor in the development of the metabolic syndrome (Nugent, 2004). In epidemiological studies high intakes of saturated fats have been associated with a higher risk of impaired glucose tolerance and higher fasting plasma glucose and insulin concentrations (Feskens & Kromhout, 1990; Parker *et al*. 1993; Feskens *et al*. 1995). Notably, in a recent 3-month intervention study involving 162 healthy subjects (Vessby *et al*. 2001) given diets rich in SFA (from butter and margarine) or MUFA (from high-oleic sunflower oil) the subjects on the SFA diet were found to have a markedly impaired insulin sensitivity (−10%), while that of the subjects on the MUFA diet was unchanged (Table 4). Furthermore, additional dietary inclusion of *n*-3 fatty acids from fish oil was shown to have no effect on insulin

Table 4. Effect of challenging with saturated fatty acids (SFA) or MUFA on insulin variables, plasma glucose and serum lipids in healthy men and women (from Vessby *et al*. 2001)

	SFA diet			MUFA diet		
Measurement	Change*	Change (%)	*P*	Change*	Change (%)	*P*
Insulin sensitivity index†	−4·2	−10·3	0·032	+0·10	+12·1	0·518
Serum insulin (mU/l)	+0·25	+3·5	0·466	−0·35	−5·8	0·049
First-phase insulin response‡ (mU/l)	+3·3	+9·0	0·029	+3·8	+10·1	0·139
Plasma glucose (mmol/l)	0·00	0	0·995	−0·03	−0·60	0·413
Total cholesterol (mmol/l)	+0·14	+2·5	0·018	−0·15	−2·7	0·012
LDL-cholesterol (mmol/l)	+0·15	+4·1	0·006	−0·19	−5·2	0·006

*Mean change during treatment expressed as least square mean.
†One of the four indices produced by the MINMOD model that analyses insulin kinetics following a frequently-sampled intravenous glucose tolerance test (Pacini & Bergman, 1986).
‡The mean of the insulin concentrations at 2, 4 and 8 min after intravenous glucose dose minus the fasting insulin values (Vessby *et al*. 2001).

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sensitivity or insulin secretion, and the favourable effects of the MUFA diet were not seen in individuals with a high fat intake (>37% energy intake).

The evidence clearly indicates that SFA intakes need to be reduced and that replacing them with MUFA and PUFA has potential benefits. Given the current contribution that ruminant animal products make to the consumption of SFA, it seems inescapable that the current situation is unsustainable.

Manipulating the fatty acid composition of animal-derived foods

It is not always recognised that the fatty acid composition of animal-derived foods is not fixed and can vary considerably in response to changes in the diet of productive animals. It follows, therefore, that animal and human nutrition are intimately linked, and current research is actively investigating ways in which milk and meat containing lower concentrations of SFA and enhanced amounts of MUFA and PUFA can be produced, whilst not diminishing the inherent and widely-acclaimed nutritional benefits of these foods.

Manipulating the fatty acid composition of milk

Milk fat typically contains (g/100 g) 70–75 SFA, 20–25 MUFA and small amounts (2–5) of PUFA (Table 3; Lock & Shingfield, 2004). Fatty acids secreted in milk originate from two sources, either by direct incorporation from the peripheral circulation or from *de novo* synthesis in the mammary gland using short-chain (2:0 and 4:0) precursors. Mammary *de novo* synthesis accounts for all 4:0–12:0, most of the 14:0 and typically about half the 16:0 secreted in milk, while all C_{18} and longer-chain fatty acids are derived entirely from circulating plasma lipids (Hawke & Taylor, 1995). A distinctive feature of the bovine mammary gland is its ability to release fatty acids from the synthetase complex at various stages, resulting in the secretion of a wide range of short- and medium-chain fatty acids (Table 3). As a result of extensive biohydrogenation of dietary unsaturated fatty acids by rumen bacteria, under most conditions 18:0 is the predominant long-chain fatty acid available for absorption. However, increasing the supply of long-chain fatty acids (of chain length ≥ 18) to the mammary gland inhibits the synthesis of short- and medium-chain SFA (Chilliard *et al*. 2000). Unlike other ruminant tissues, the mammary gland cannot convert 16:0 to 18:0 by chain elongation (Chilliard *et al*. 2000). However, it is also notable that the secretion of oleic acid (*cis*-9-18:1) in milk exceeds its uptake by the mammary gland as a result of the activity of stearoyl-CoA (Δ^9) desaturase in mammary secretory cells that converts 18:0 to *cis*-9-18:1 (Kinsella, 1972). Conversion of 18:0 to *cis*-9-18:1 is the predominant precursor-to-product process of the Δ^9-desaturase, and about 40% of the 18:0 taken up by the mammary gland is desaturated (Chilliard *et al*. 2000).

As a result of the complex process by which milk fatty acids originate, several nutritional approaches can be used to manipulate milk fatty acid composition. Both the amount and source of dietary lipid affect the extent and type of change that can be achieved. A summary of the effects of including various lipids in the diets of dairy cows are reported in Table 5. In general, supplements of plant oils or oilseeds reduce the levels of short- and medium-chain fatty acids in milk but increase those of long-chain fatty acids, resulting in an overall shift towards 18:0 at the expense of 16:0 as a result of decreased *de novo* synthesis and/or reduced mammary uptake of absorbed 16:0. Reductions in milk SFA content are characterised by small increases in the concentration of the predominant fatty acid in the lipid supplement. In all cases feeding plant oils increases milk fat 18:0, *cis*-9-18:1 and *trans*-18:1 contents because of extensive rumen metabolism of long-chain fatty acids, leading to an increased supply of biohydrogenation intermediates (both *trans*-18:1 and 18:2) and 18:0 to the mammary gland. Table 6 shows the marked decreases in SFA and increases in MUFA observed (Givens *et al*. 2003) as a result of supplementing dairy cows with cracked whole rapeseed, the oil of which is rich in oleic acid.

PUFA are not synthesised in any appreciable quantities in ruminant tissues, and concentrations in milk are, therefore, essentially a reflection of the amount leaving the rumen. Consequently, oils rich in linoleic acid (18:2*n*-6)

Table 5. Typical milk fatty acid responses in dairy cows to dietary lipid supplementation (from Givens & Shingfield, 2003)

	Mean response*									
Lipid source	14:0	16:0	18:0	*cis*-9-18:1	*trans*-18:1	18:2 (*n*-6)	18:3 (*n*-3)	Total SFA	MUFA	PUFA
Rapeseed oil	−0·03	−0·20	0·55	0·39	0·30	−0·09	0·11	−0·07	0·30	−0·04
Rapeseed oil	−0·19	−0·33	0·51	0·67	2·00	−0·04	0·60	−0·18	0·60	0·15
Soyabean oil	−0·29	−0·28	0·45	0·23	4·61	0·33	–	−0·19	0·53	0·33
Sunflower oil†	−0·32	−0·32	0·14	0·27	3·07	−0·05	−0·24	−0·22	0·53	0·12
Sunflower oil	−0·23	−0·26	0·15	0·11	2·79	0·07	−0·21	−0·17	0·36	0·21
Linseed oil	−0·11	−0·15	0·27	0·26	0·94	−0·14	0·17	−0·07	0·23	−0·06
Fish oil	0·04	−0·01	−0·45	−0·25	8·08	0·24	0·03	−0·09	0·24	0·29
Fish oil	0·30	0·35	−0·77	−0·73	2·19	0·39	0·07	−0·05	−0·10	2·17
Tallow	–	0·06	0·04	0·22	0·19	−0·37	−0·37	−0·06	0·19	−0·36

SFA, saturated fatty acids.
*Responses calculated as proportionate differences between treatment controls and lipid-supplemented diets.
†Sunflower oil rich in *cis*-9 18:1.

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Table 6. Effect on milk fatty acid composition (g/100 g total fatty acids) of feeding dairy cows diets containing zero (ZR), medium (MR) and high (HR) inclusion rates of whole cracked rapeseed (from Givens *et al.* 2003)

	Treatment*		
Fatty acid	ZR	MR	HR
4:0	4·99	3·16	2·72
6:0	2·26	1·13	0·96
8:0	1·25	0·55	0·43
10:0	3·07	1·3	0·98
10:1	0·37	0·13	0·08
11:0	0·11	0·05	0·05
12:0	3·95	1·89	1·44
14:0	11·6	7·85	6·0
14:1 (*n*-5)	1·4	1·15	0·75
15:0	2·28	1·43	0·99
16:0	30·7	19·8	18·0
16:1 (*n*-9)	0·18	0·23	0·26
16:1 (*n*-7)	1·92	1·83	1·58
17:0	1·63	1·16	0·89
17:1	0·31	0·22	0·18
18:0	8·3	14·1	15·8
18:1 (*n*-9)	18·1	34·7	39·3
18:1 *trans* (*n*-7)	1·97	2·58	2·03
18:2 (*n*-6)	2·08	2·37	2·76
18:2 (conjugated)	0·60	1·02	0·74
18:3 (*n*-3)	0·45	0·48	0·60
20:0	0·13	0·27	0·30
Others	0·74	1·13	1·29
Summary			
Total SFA	70·6	52·8	48·6
Total MUFA	24·2	40·8	44·2
Total PUFA	3·13	3·87	4·1

SFA, saturated fatty acids.
All treatment effects were significant: $^{*}P < 0{\cdot}05$.

and α-linolenic acid (18:3*n*-3) have been used to increase the concentrations of these PUFA in milk or ruminant meat. In addition, the potential to enhance the concentrations of the long-chain *n*-3 PUFA, EPA (20:5*n*-3) and DHA (22:6*n*-3), in milk (and beef) have been examined, typically using fish oil as a source of EPA and DHA. However, the transfer efficiency of EPA and DHA from the diet into milk is very low because of extensive biohydrogenation in the rumen and subsequent transport of absorbed *n*-3 PUFA in phospholipid and cholesteryl ester fractions of plasma that are poorly utilised by the mammary gland (Rymer *et al*. 2003). In addition, inclusion of fish oil in the diets of dairy cows reduces levels of 18:0 and markedly increases the amounts of *trans*-18:1 and *trans*-18:2 in milk (Shingfield *et al*. 2003). The effects of reducing the extent of saturation of fatty acids in dairy products have been demonstrated in an 8-week study (Noakes *et al*. 1996) of thirty-three men and women in which the effects of fatty acid-modified and normal dairy products were compared. The modified products were found to result in a marked reduction in total cholesterol (0·28 mmol/l) and LDL-cholesterol (0·24 mmol/l), with HDL-cholesterol being unaffected. The authors suggest that if these changes were to be applied to Western populations they would represent a potential strategy for lowering the risk of CHD without the need to change normal eating patterns.

There has also been much recent research aimed at increasing the conjugated linoleic acid (CLA) concentration in milk fat because of its potentially powerful anti-cancer effect. CLA is a generic term used to describe a mixture of geometric and positional isomers of 18:2 that contain a conjugated double bond. There is now increasing evidence, based on studies in rodent models, that *cis*-9, *trans*-11-CLA exhibits anti-carcinogenic properties (Parodi, 2003), although there is currently no convincing evidence from human studies. Dairy products are the major source of CLA in the human diet (Lawson *et al*. 2001), and the *cis*-9, *trans*-11-isomer is the most abundant isomer in ruminant-derived foods. A number of nutritional strategies involving extended grazing or lipid supplementation have been developed to enhance CLA concentrations in milk, and it has been shown that concentrations of CLA are higher in milk fat from cows offered fresh forages as compared with conserved forages, and they can also be enhanced using whole oilseeds or oil supplements in the cow's diet (Griinari & Bauman, 1999). Fish oil has been shown to increase the CLA content of milk fat more effectively than plant oils (Chilliard *et al*. 2000), and these responses can be further enhanced when fish oil is fed in combination with 18:2-rich supplements (for a fuller discussion on CLA enrichment, see Givens & Shingfield, 2004).

Manipulating the fatty acid composition of meat

In general, fat in meats derived from ruminant animals is composed of (g/100 g) approximately 45–55 SFA, 45–50 MUFA and relatively minor amounts of PUFA (Table 3; Mir *et al*. 2003). Much of the effort in manipulating the fatty acid composition of beef, and to a lesser extent lamb, has been directed towards increasing PUFA:SFA and enhancing *n*-3 PUFA:*n*-6 PUFA. The most effective means of manipulating the fatty acid composition of beef is through nutrition, using high-forage-based diets or oil supplements rich in PUFA. In northern Europe an important feed for beef cattle is fresh grass, which has a number of benefits because the major fatty acid in grass is α-linolenic acid (Dewhurst *et al*. 2003). Even though most of the α-linolenic acid is hydrogenated in the rumen, a small proportion of it can escape and, once absorbed, is available for incorporation into tissue lipids.

In contrast, the fatty acid profile of non-ruminant meat is essentially a reflection of that in the diet, since limited transformation of dietary fatty acids occurs during digestion and absorption. Accordingly, it is theoretically easier to enhance long-chain *n*-3 PUFA levels in non-ruminant tissue lipids using dietary sources. Since little transformation of dietary α-linolenic acid to EPA and DHA occurs in pig and poultry tissues, enrichment in EPA and DHA has therefore relied heavily on dietary supplements of fish oil (Table 7). Whilst this nutritional strategy is relatively effective it can result in the production of meat with a metallic taint, fish-like flavour and reduced shelf-life (Leskanich & Noble, 1997). One of the key challenges ahead is to identify novel alternative sources of EPA and DHA.

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Table 7. The effect of including fish oil in the diet of broilers on selected fatty acids (g/100 g total carcass lipids) in the thigh meat (from Lopez-Ferrer *et al.* 2001)

	Dietary inclusion rate of fish oil (g/kg)		
Fatty acid	0	20	40
14:0	0·77	1·26	1·47
16:0	33·8	32·1	29·0
18:0	8·54	9·42	8·54
18:1*n*-9	34·7	31·8	29·3
18:2*n*-6	11·7	11·4	12·9
18:3*n*-3	1·63	2·46	2·98
20:5*n*-3	0·20	0·77	1·33
22:6*n*-3	0·10	1·03	2·42
Total SFA	43·8	43·6	39·8
Total MUFA	41·3	38·7	37·6
Total PUFA	14·9	17·5	22·2
n-6:*n*-3	6·11	2·50	1·73

SFA, saturated fatty acids.

Other considerations

Whilst there is now much evidence to show that it may be possible to improve the lipid profile of animal-derived foods, there are also some serious concerns. A recent prospective study (Ness *et al.* 2001) over a period of 25 years has indicated that increased consumption of milk is associated with a substantially-reduced risk of death from CVD, particularly CHD (Fig. 1), and the authors refer to a number of similar studies that support their findings. A number of factors have been proposed for the apparent protective effect of milk. For example, although 12:0, 14:0 and 16:0 fatty acids increase serum LDL-cholesterol when substituted for 18:1 (or carbohydrate), they also increase the concentration of HDL-cholesterol, with 14:0 possibly producing the greatest increase (for example, see Temme *et al.* 1996). This finding, together with the view of some scientists that serum HDL-cholesterol better discriminates between subjects at risk of CHD than total cholesterol or LDL-cholesterol (Rubins *et al.* 1995), gives rise to doubts about the benefits of reducing the SFA content of foods. Furthermore, Samuelson *et al.* (2001) have reported an unexpected inverse association between dietary SFA and serum cholesterol in adolescents whose dietary source of SFA is mainly milk fat, and have suggested that milk fat may contain or be associated with some component that counterbalances the expected positive relationship between SFA and plasma lipids. These findings are essentially in agreement with those of Smedman *et al.* (1999) that in 70-year-old men there are negative relationships between the intake of milk products and a number of variables, including LDL-cholesterol:HDL-cholesterol, BMI and waist circumference, but positive relationships with HDL-cholesterol and apoA-I.

Furthermore, a US study (Pereira *et al.* 2002) involving a total of 3157 young adults has shown that increased consumption of dairy products over a 10-year period appears to protect overweight individuals (BMI $\geq 25\,kg/m^2$) from the development of obesity and insulin resistance (Fig. 2). The authors have proposed a number of possible reasons for the observations, including a protective effect of milk Ca and the low glycaemic index associated with dairy products. Although this study was observational, it is of great interest. More recently, Wijga *et al.* (2003), in a study involving 2978 preschool children, have shown that frequent consumption of whole milk is associated with a reduced prevalence of asthma and daily consumption of butter produces an even lower prevalence.

There has also been much speculation about the possibility that consumption of dairy products can give protection from various cancers. A review of the evidence (Norat & Riboli, 2003) indicates that data from cohort studies support the hypothesis of a protective effect of total dairy product consumption and milk against colo-rectal cancer. It has been suggested that milk fat contains potential anti-cancer agents, including CLA, sphingolipids and butyrate (for review, see Parodi, 2003). It is notable that a number of studies in animal models have shown that milk-derived sphingomyelin reduces colon tumour development (Schmelz *et al.* 2000). Overall, it seems that milk fat contains, or is associated with, components that counterbalance the expected negative effects of SFA. All these issues need investigation at the mechanistic level.

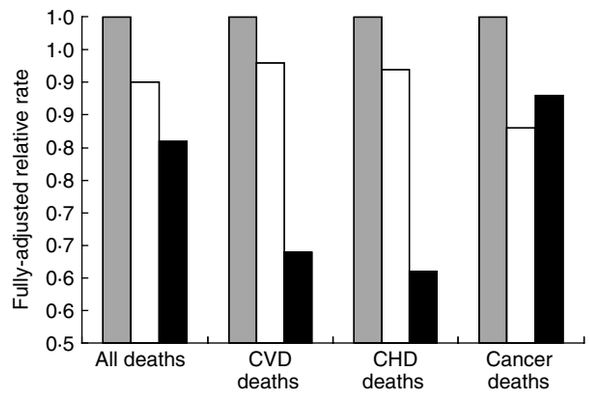

Fig. 1. Relative mortality rate (25 years) in 5765 men according to level of milk consumption: (▨), very low; (□), moderate; (■), high. (From Ness *et al.* 2001.)

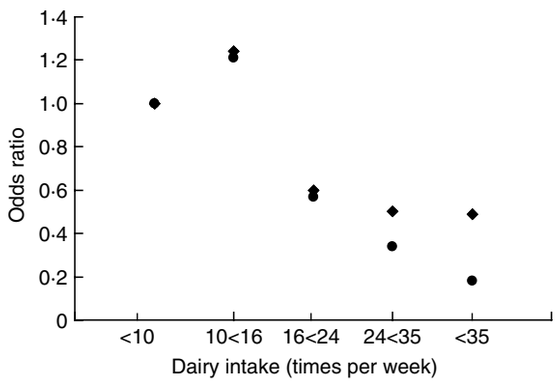

Fig. 2. Adjusted odds ratio for the incidence of insulin resistance relative to intake of dairy products. (◆), Men; (●), women. (From Pereira *et al.* 2002.)

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It is also known that milk proteins are potentially very good sources of bioactive peptides. These peptides have a range of activities, including angiotensin-converting enzyme inhibition and hence a blood pressure-reducing effect (Korhonen & Pihlanto, 2003). All these potentially very powerful opportunities must be explored.

Conclusions

Foods derived from animals are an important source of nutrients in the diet. However, certain aspects of some animal-derived foods, particularly SFA, have led to concerns about the contribution of these foods to increased risk of CVD and the metabolic syndrome. The fatty acid composition of various animal-derived foods is not constant and can, in many cases, be enhanced by animal nutrition. In the future, the role of animal nutrition in creating foods closer to the optimum composition for long-term human health will become increasingly important. However, certain animal-derived foods contain compounds that may actively promote long-term health, and research is urgently required to fully characterise the benefits associated with the consumption of these compounds and to understand how the levels in natural foods can be enhanced. It is also vital that the beneficial effects are not inadvertently destroyed in the process of reducing the concentrations of SFA. Although SFA have clearly been shown to increase LDL-cholesterol, the effects of consumption of milk fat and dairy products on the risk of CVD and other chronic diseases are clearly complex. Research is urgently needed to resolve this issue. It should also be noted that the production of improved animal-derived foods on a scale that will substantially affect national diets will require both substantial political and financial incentives and great changes in the animal production industry.

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