

# Dairy Products in the Food Chain: Their Impact on Health

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Annu. Rev. Food Sci. Technol. 2011. 2:21–36

First published online as a Review in Advance on September 14, 2010

The *Annual Review of Food Science and Technology* is online at food.annualreviews.org

This article's doi:  
10.1146/annurev-food-022510-133734

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1941-1413/11/0410-0021\$20.00

## Keywords

milk, dairy, cardiovascular disease, cancer, fatty acids

## Abstract

Milk is a complex and complete food containing an array of essential nutrients that contribute toward a healthy, balanced diet. Numerous epidemiological studies have revealed that high consumption of milk and dairy products may have protective effects against coronary heart disease (CHD), stroke, diabetes, certain cancers (such as colorectal and bladder cancers), and dementia, although the mechanisms of action are unclear. Despite this epidemiological evidence, milk fatty acid profiles often lead to a negative perception of milk and dairy products. However, altering the fatty acid profile of milk by changing the dairy cow diet is a successful strategy, and intervention studies have shown that this approach may lead to further benefits of milk/dairy consumption. Overall, evidence suggests individuals who consume a greater amount of milk and dairy products have a slightly better health advantage than those who do not consume milk and dairy products.

## THE CONTRIBUTION OF MILK AND DAIRY PRODUCTS TO THE HUMAN DIET

**SFA:** saturated fatty acid(s)

### Introduction

Milk is a unique and complex food of great interest, intended to be a complete food for young mammals. The important role of cow's milk in the human diet as a supplier of energy, protein, and other key nutrients, including calcium, is well known. Milk is essentially a complex colloidal system comprising globules of milk fat suspended in an aqueous medium containing lactose, a range of proteins, mineral salts, and water soluble vitamins. Milk from modern Holstein/Friesian cows will typically contain about 40, 36, and 45 g kg<sup>-1</sup> of fat, protein, and lactose, respectively, and have an energy content of approximately 2.8 MJ kg<sup>-1</sup>. The fat and protein contents of milk vary considerably due to the breed and nutrition of the cow. The effect of breed is particularly noticeable in milk from Channel Island breeds, which typically have a fat content of approximately 65 g kg<sup>-1</sup>. Although milk is widely consumed, there has recently been increased concern that a high proportion (>50%) of the energy in milk is derived from fat, approximately 70% of which is made up of saturated fatty acids (SFA).

### Trends in Milk and Dairy Product Consumption

Globally, the demand for animal-derived foods in general is growing rapidly, driven by a combination of population growth, urbanization, and rising income. **Table 1** shows the trends in milk consumption over the past 40 years for various regions of the world. Although the historical and projected trend is upward, in the United Kingdom and other Western countries consumption has shown considerable change over recent decades. The decline in whole milk consumption in the United Kingdom and the increase in lower-fat milk consumption (Givens & Kliem 2009) reflects a general trend in a number of other developed countries (**Figure 1**). Denmark, France, the United States, and Canada also show similar trends since the 1970s. Germany, however, has shown a less-marked decline in whole milk consumption, and there has been little change in consumption of lower-fat milk up to 1993, although data for recent years were not available. The consumption of liquid milk in Italy has always been much lower than in northern Europe, but between 1977 and 1990 this remained relatively constant for both whole and lower-fat milk (**Figures 1 and 2**).

**Figure 3** illustrates the changes in cheese consumption. Of the selected countries, France has the highest consumption followed by Italy. The United Kingdom has always had the lowest. Most countries show a gradual increase in cheese consumption, although that of Canada and Denmark did fluctuate during the 1990s, and that of the United Kingdom post-1980s remained relatively constant. Butter consumption appears to have followed several different trends (**Figure 4**). Consumption in Germany, Italy, and the United States appears to

**Table 1** Trends in consumption of milk (from WHO/FAO 2003)

Region	Milk (kg/person/year)		
	1964–66	1977–99	2030 <sup>a</sup>
World	73.9	78.1	89.5
Developing countries	28.0	44.6	65.8
Transition countries	156.7	159.1	178.7
Industrialized countries	185.5	212.2	221.0

<sup>a</sup>projected.

have remained constant, whereas that in the United Kingdom and Denmark has declined, and that of France and Canada has fluctuated.

## Nutrients Provided by Milk and Dairy Products

Milk and dairy-derived foods are available in the retail market in many forms. Based on food intakes assessed by the U.K. National Diet and Nutrition Survey (Henderson et al. 2003a,b) over the period July 2000 to June 2001, the contribution of the major dairy-derived food types to energy and nutrient intakes of the United Kingdom male population (aged 19 to 64 years) is shown in **Table 2**.

Milk and dairy food products are clearly important sources of protein, calcium, phosphorus, iodine, riboflavin, and vitamins A and B<sub>12</sub>. Indeed, milk and dairy products alone usually provide

**Table 2** Energy and selected nutrients provided by milk and dairy products to men's diets in the UK (derived from Henderson et al. 2003a,b)

Energy/ nutrient	Contribution from milk and dairy products	Liquid whole milk	Semi- and skimmed milk	Cheese	Other dairy products	Butter	Total dairy
Energy	Intake (MJ/d)	0.49	nas <sup>a</sup>	0.29	0.10	0.10	0.97
	% of EAR <sup>b</sup>	5	nas	3	1	1	9
Protein	Intake (g/d)	1.8	5.3	4.4	1.8	0	13.2
	% of RNI <sup>c</sup>	3	10	8	3	0	24
Fat	Intake (g/d)	1.7	2.6	5.2	2.6	2.6	14.7
	% of ADI <sup>d</sup>	2	3	6	3	3	17
Calcium	Intake (mg/d)	61	203	112	41	nas	417
	% of RNI	9	29	16	5	nas	60
Phosphorus	Intake (mg/d)	45	165	90	30	nas	330
	% of RNI	8	30	16	6	nas	60
Magnesium	Intake (mg/d)	6.2	19	nas	6.2	nas	31
	% of RNI	2	6	nas	2	nas	10
Zinc	Intake (mg/d)	0.21	0.60	0.64	0.21	nas	1.7
	% of RNI	2	7	7	2	nas	18
Iodine	Intake (μg/d)	15	46	4.4	11	nas	77
	% of RNI	11	33	3	8	nas	55
Vitamin A <sup>e</sup>	Intake (μg/d)	20	31	61	31	41	183
	% of RNI	3	4	9	4	6	26
Riboflavin	Intake (mg/d)	0.12	0.40	0.07	0.09	nas	0.68
	% of RNI	9	31	5	7	nas	52
Vitamin B <sub>12</sub>	Intake (μg/d)	0.41	1.4	0.34	0.14	nas	2.2
	% of RNI	27	91	23	9	nas	150
Folate	Intake (μg/d)	25	nas	nas	nas	nas	25
	% of RNI	13	nas	nas	nas	nas	13

<sup>a</sup>nas, not available separately, included in total.

<sup>b</sup>EAR, estimated average requirement.

<sup>c</sup>RNI, reference nutrient intake.

<sup>d</sup>ADI, average daily intake.

<sup>e</sup>Retinol equivalents.

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**MUFA:**

monounsaturated fatty acid

**PUFA:**

polyunsaturated fatty acid

**Prospective cohort**

**study:** follows a group of individuals over a length of time, to detect how factors being studied affect health outcome

**Case-control study:**

compares individuals with a defined condition with individuals who do not have the condition but are otherwise similar

**CHD:** coronary heart disease

**RR:** relative risk

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more than the daily recommended intake of vitamin B<sub>12</sub>. The current importance of semi-skimmed and skimmed milk in the diet is clear and notably, some 70% of liquid milk is consumed as semi-skimmed (Milk Development Council 2004). Although milk and dairy products only provide about 13% of the recommended folate intake of 200 µg day<sup>-1</sup> (Henderson et al. 2003b), there is evidence that the presence of milk in the diet can increase overall folate bioavailability compared with diets containing no milk (Wigertz et al. 1997). Also, Smith et al. (1985) proposed that folate present in milk is more available than folates from other foods, at least for infants. It is probable that these properties of milk are due to the fact that it uniquely contains folate-binding proteins. Although their exact role is not fully understood (de Jong et al. 2005), it is possible that these proteins increase the availability of folate in other foods consumed and make milk a good candidate for fortification with folate.

Milk and dairy products, including butter, contribute almost 20% of the total fat consumed, but because the lipids in these products are rich in SFA they make a major contribution to SFA intake. In the United Kingdom, the National Diet and Nutrition Survey (Henderson et al. 2003a) estimated the contribution as 30% of total SFA intake, although this excluded milk fats in manufactured foods such as cakes, biscuits, etc. A study on fatty acid intake across Europe (Hulshof et al. 1999) suggested a higher figure of 40% for the United Kingdom, and milk and dairy foods were consistently the largest source of SFA, with the greatest contribution being observed in Germany and France, where some 60% of SFA were from these foods (Hulshof et al. 1999). Contribution of milk and dairy products to *cis*-monounsaturated fatty acid (MUFA) consumption averaged 18% across the 13 European countries included in the study of Hulshof et al. (1999). However, milk and dairy products are not a major contributor to *cis*-polyunsaturated fatty acid (PUFA) consumption (Hulshof et al. 1999).

Milk and milk-derived foods contribute to most of the *trans* fatty acids consumed (Hulshof et al. 1999). The contribution in Germany, Italy, and France is particularly high at approximately 72%, 62%, and 61%, respectively, although in the United Kingdom this is lower (25%). The high contributions in Germany arise mainly from butter consumption, whereas in Italy cheese is probably the main source. In addition, milk and dairy products are major contributors to the consumption of conjugated linoleic acid isomers (67% total consumption) (Lawson et al. 2001), which are mainly found in ruminant products.

## EFFECTS OF MILK AND DAIRY PRODUCT CONSUMPTION ON CHRONIC DISEASE RISK

Epidemiological studies presenting outcomes in terms of disease incidence have major advantages over the use of risk factors as predictors of disease. Disadvantages of such studies include the long periods of time required to measure disease outcomes and the large numbers of participants required. Nonetheless, there are many published prospective cohort and case-control studies that have attempted to investigate relationships between dairy product consumption and risk of chronic disease. In general, prospective cohort studies provide stronger evidence than case-control studies (Elwood et al. 2010).

### Coronary Heart Disease

Coronary heart disease (CHD) refers to the failure of the coronary circulation to supply adequate blood flow to cardiac and surrounding tissue. Risk factors for CHD include hypertension, diabetes, and hyperlipidemia. Several case-control studies have demonstrated an overall reduced relative risk (RR) (0.83) of myocardial infarction in patients consuming higher amounts of milk and/or

dairy products (Elwood et al. 2008). A number of prospective cohort studies (with follow-up periods of between 8 and 28 years) reported differences in RR of CHD related to milk and dairy consumption. For example, Mann et al. (1997) reported an increase in RR (1.50) of CHD events in high milk consumers ( $n = 10,802$ ), whereas a significant risk reduction (0.88) of CHD events was observed by Shaper et al. (1991) in high milk consumers ( $n = 7,735$ ). The Nurses' Health Study specified variations in response to milk of different fat levels in terms of RR of CHD events, with high whole milk consumers having increased RR (1.67) compared with high low-fat milk consumers (0.78) (Hu et al. 1999). A meta-analysis of fifteen prospective cohort studies (Elwood et al. 2008) revealed that overall, high milk consumption did not result in an increase of RR of CHD (0.91 when including whole milk consumption reported by Hu et al. 1999; 0.84 when including low-fat milk consumption reported by Hu et al. 1999). More recent, large meta-analyses of both prospective cohort and clinical studies concluded that milk consumption displayed weak evidence (RR = 0.94) of being the cause of CHD (Mente et al. 2009), and the study of Elwood et al. (2010) involving 4.3 million person-years and 16,212 CHD events concluded that high intakes of milk are suggestive of a reduced risk of CHD [RR = 0.92, 95% confidence interval (CI) 0.80–0.99].

The mechanism of action of milk and dairy products on risk of CHD is complicated. Some studies have reported that increased dairy consumption increases circulating total lipoprotein concentrations and low density lipoprotein (LDL) concentrations (Thompson et al. 1982, Steinmetz et al. 1994). However, there is limited evidence of effect of dairy product consumption on circulating high density lipoprotein (HDL) cholesterol levels, increases in which are known to exert a protective effect against CHD. A prospective case-control study that used plasma phospholipid 15:0 and 17:0 concentrations as markers of milk fat intake found there was an inverse relationship between the sum of 15:0 and 17:0 in phospholipids and total cholesterol and plasma triacylglycerols (Warensjö et al. 2004). Interestingly, different dairy products may exert varying effects on plasma lipoprotein levels. Plasma total and LDL cholesterol were significantly higher in volunteers consuming 40 g of dairy fat as butter per day compared with the same amount of cheese (Nestel et al. 2005). It was suggested that fermentation products in cheese or the physical state of cheese fat globules compared with butter may explain differences between the two dairy fat types.

## Stroke

A meta-analysis of eleven cohort studies (with follow-up periods ranging from 12.9 to 68 years) with ischemic stroke as an outcome reported that overall, the risk of stroke in subjects with the highest dairy intake compared to that of subjects with the lowest intake was reduced (RR = 0.79, 95% CI 0.68–0.91), although there was significant heterogeneity between studies (Elwood et al. 2010). The same authors attempted to differentiate between cohort studies reporting hemorrhagic strokes and subarachnoid bleeds (Elwood et al. 2010), although there were fewer published data with these events as an outcome and again significant heterogeneity. The maximum follow-up period was 16 years. Overall, high dairy consumers displayed a lower RR for hemorrhagic (0.75) and subarachnoid (0.65) strokes. However, this meta-analysis should be considered with caution due to the low number of studies included.

Individual cohort studies have suggested possible mechanisms behind the apparent protective effect of high dairy intake against stroke incidence. The Honolulu study (a cohort of initially 8,006 men) reported that over 22 years, men who did not drink milk were twice as likely to experience a stroke event as those who regularly consumed two glasses or more of milk per day (Abbott et al. 1996). More detailed analysis of intake data revealed that calcium intake from nondairy sources

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**Meta-analysis:** combines the results of several studies that address a set of related research hypotheses

**CI:** confidence interval

**LDL:** low density lipoprotein

**HDL:** high density lipoprotein

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was not related to stroke incidence, suggesting that other constituents of milk may also have an effect, which will be discussed later.

Hypertension is itself a risk factor for stroke (and CHD). A prospective cohort of 28,886 women (with a 10-year follow up) reported that a high intake of low fat dairy products was associated with an 11% reduced RR of hypertension (Wang et al. 2008). A smaller study involving 2,245 older participants ( $\geq 55$  years) demonstrated an inverse relationship between dairy product consumption and hypertension (Engberink et al. 2009). When analyzed in greater detail, it was apparent that this reduced risk of hypertension was specific to milk consumption and not that of cheese or fermented dairy products. The effect of dairy products on hypertension is thought to be mainly related to calcium intake because dietary calcium suppresses 1,25 dihydroxyvitamin D production, which among other things reduces the cellular influx of calcium into smooth muscle cells, thus reducing contraction and vascular resistance of smooth muscle cells (Zemel 2001). Comparisons of dairy and nondairy sources of calcium have shown that dairy calcium may exert a greater effect on hypertension (Griffith et al. 1999), which may reflect the balanced composition of milk in terms of calcium, potassium, and magnesium content. The review of Álvarez-León et al. (2006) concluded that a diet balanced in these minerals is recommended to prevent hypertension. Also, the presence of bioactive peptides within the casein and whey fractions of milk may also contribute to reduced vascular resistance in high dairy product consumers. These have been found to inhibit angiotensin-I-converting enzyme, thus modulating endothelial function and leading to vasodilation (Fitzgerald & Meisel 2000).

## Diabetes

Type II diabetes incidence is increasing worldwide, and the combination of diabetic characteristics such as dysfunctional glucose-insulin homeostasis, dyslipidaemia, and proinflammatory states leads to diabetes increasing the risk of cardiovascular disease. There are relatively few cohort studies analyzing the relationship between milk and dairy consumption and diabetes incidence. The longest cohort study to date was followed up over 25 years and showed that a higher milk intake resulted in a reduced RR (0.57) of diabetes than low milk intakes in 640 males (Elwood et al. 2007). Four other cohort studies (with follow-up periods ranging from 6.9 to 12 years) resulted in similar outcomes for high dairy consumers. A meta-analysis concluded that overall, the RR of diabetes incidence from high versus low milk and dairy consumption was 0.85 (95% CI, 0.75–0.96) (Elwood et al. 2010). Another recent review and meta-analysis reached the same conclusion (Pittas et al. 2007) and suggested that incidence of type II diabetes was inversely related to total vitamin D and calcium status.

## Selected Cancers

**Table 3** presents relevant information from the recent report of the World Cancer Research Fund (2007). These data suggest that increased milk consumption appears to reduce the RR of colorectal cancers, and possibly bladder cancer, although data from both the cohort and case-control studies displayed moderate to high heterogeneity. However, the data also suggest a high milk and dairy intake may increase risk of prostate cancer.

**Colorectal cancer.** Cho et al. (2004) conducted a pooled analysis of ten prospective cohort studies, which involved detailed investigation into different types of foods consumed. The follow-up periods ranged from 6 to 16 years, and over 500,000 subjects (men and women) were included. The authors showed that milk consumption ( $>250$  g day<sup>-1</sup> compared with  $<70$  g day<sup>-1</sup>) was

**Table 3 Relationship between milk and dairy product consumption and cancer (data taken from the World Cancer Research Fund (2007))**

Cancer	Predictor	Number of studies	Pooled relative risk	Heterogeneity
Colorectal	Milk	4 cohorts	0.94 (0.85–1.03)	Low
	Milk	10 cohorts	0.78 (0.69–0.88)	Not reported
Prostate	Milk	8 cohorts	1.05 (0.98–1.14)	Low
	Milk	6 case-control	1.08 (0.98–1.19)	Moderate
	Milk and dairy	8 cohorts	1.06 (1.01–1.11)	Moderate
	Milk and dairy	5 case-control	1.03 (0.99–1.07)	Low
Bladder	Milk	4 cohorts	0.82 (0.67–0.99)	Moderate
	Milk	3 case-control	1.00 (0.87–1.14)	High

inversely related to colorectal cancer incidence (RR 0.85). The same authors also reported that there was no significant evidence that cheese or yogurt affected RR. It was concluded that both milk and calcium intake were associated with a reduction in risk of colorectal cancer.

**Prostate cancer.** A meta-analysis of case-control studies revealed that high milk consumers tended to show an increased risk (as measured by an odds ratio of 1.68) of prostate cancer compared with low milk consumers (Qin et al. 2004). However, these authors pointed out that eight of the eleven studies involved hospital-based controls, which are more prone to bias. A larger study (whereby thirty-seven prospective cohort and four intervention studies were reviewed) reported that consumption of milk and dairy products was either positively associated or had no effect on prostate cancer risk (Dagnelie et al. 2004). Further investigation revealed that calcium intakes of between 1330 mg day<sup>-1</sup> and 1840 mg day<sup>-1</sup> showed no association with prostate cancer risk (Shuurman et al. 1999 and Chan et al. 2000, respectively), and yet calcium intakes of greater than 2000 mg day<sup>-1</sup> were associated with increased risk (Giovannucci et al. 1997, Rodriguez et al. 2003). Elwood et al. (2008) calculated from the results of pooled cohort studies that overall, milk and dairy products increased RR to prostate cancer (RR 1.06). However, Huncharek et al. (2008) carried out a meta-analysis of pooled data from 45 observational studies and concluded that cohort studies showed no evidence of a relationship between dairy products (RR 1.06; 95% CI 0.92, 1.02) or milk consumption (RR 1.06; 95% CI 0.91, 1.23) and risk of prostate cancer, and this conclusion was supported by data from case-control studies. Similarly, Parodi (2009) concluded that there was not any plausible biological explanation for an association between dairy product consumption and prostate cancer; however, he conditioned this by saying that a combination of factors, as yet undetermined, cannot be excluded. Overall, the current evidence indicates very little, if any, evidence that high consumers of milk have greater risk of prostate cancer than low consumers.

**Bladder cancer.** The results from the World Cancer Research Fund (2007) (Table 3) highlight the heterogeneity of data from cohort studies measuring the effects of dairy consumption on risk of bladder cancer. Results of a more recent cohort study involving 120,852 men and women, and with a follow-up period of 16.3 years, demonstrated little effect of dairy intake on bladder cancer risk (Keszei et al. 2010). However, when individual dairy products were analyzed it was reported that fermented dairy product intake appeared to have an inverse relationship with bladder cancer risk (hazard ratio 0.71), whereas butter intake by women was positively associated with bladder cancer (hazard ratio 1.61). A similar effect with fermented dairy products was observed by Larsson et al. (2008) over a 9.4 year follow up, with no effect of total dairy consumption or milk and cheese.



**Figure 5** illustrates the number of deaths in England and Wales in 2005 from various causes, together with pooled RRs for these diseases in subjects with the highest milk/dairy consumption relative to risk in subjects with the lowest consumption. RR estimates for all cancers are those listed in **Table 3**. These data suggest that consumers of high intakes of dairy products appear to have a survival advantage compared with consumers of low intakes of dairy products (Elwood et al. 2008).

## Dementia

Partly because of the increased life expectancy, there is growing concern about the trend in the prevalence of dementia, with the numbers in the United Kingdom projected to double between 2001 and 2040 (Jagger et al. 2009). There are few studies that have examined any relationship between milk consumption and risk of dementia. One study that did look at this was the Adult Health Study, a prospective cohort of 1,774 subjects in Hiroshima, Japan born before September 1932 (Yamada et al. 2003). Between 1992 and 1997, 1,660 were shown to have no dementia and 114 had dementia (51 with Alzheimer's disease, 38 with vascular dementia). Vascular dementia prevalence increased significantly with age, with higher systolic blood pressure and, crucially, with lower milk intake. The odds ratios of vascular dementia for age (in five-year increments), systolic blood pressure (10 mm Hg increments), and milk intake (almost daily versus less than four times a week) were 1.29, 1.33, and 0.35, respectively. Yamada et al. (2003) concluded that increased blood pressure and low milk intake in midlife were associated with vascular dementia detected 25 to 30 years later. The mechanism whereby milk provided protection cannot be stated with certainty but given other evidence linking milk consumption with lowered blood pressure, it would seem probable that this mechanism was involved. Clearly, much further research is needed in this area.

## IMPROVING THE NUTRITIONAL QUALITY OF MILK USING ANIMAL NUTRITION

Manipulating milk constituent content is a difficult task, not least due to the complex nature of milk and its production by lactating mammals. The majority of milk consumed in Western diets is of bovine origin, so the following discussions relate to experimental studies involving dairy cows. The previous sections of this chapter have suggested that the apparent protective or detrimental effects of milk and dairy products against certain chronic diseases can be partially attributed to the high calcium content of milk or the presence of a high concentration of SFA (as a proportion of total fatty acids). Calcium excretion in milk is highly complex with calcium taking a variety of forms, including casein-bound calcium, colloidal calcium phosphate, calcium citrate, and free ionized calcium, with the majority being within casein micelles (Neville et al. 1995). Casein content of milk is independent of nutrition (Coulon et al. 1998), so changing milk calcium content indirectly by manipulating casein content by nutrition is unrealistic. However, a recent study has shown a moderate heritability for milk calcium concentration, which would lead to good prospects for selective breeding (van Hulzen et al. 2009).

Including a wide range of lipid-rich supplements in the diet of the dairy cow has been the most common means for manipulating milk fatty acid composition. However, both the type and source of the lipid and basal diet influences the extent of changes that can be achieved. Attempts to increase the concentration of one or more fatty acids may cause changes in other fatty acids, which may reduce potential beneficial effects. For example, feeding diets to enrich milk fat with *cis*-9 C18:1, C20:5 *n*-3 or C22:6 *n*-3 content will usually also result in an increase in *trans* C18:1



concentrations. Although likely to have few negative effects on health, such changes are generally perceived negatively by consumers and some health professionals.

The following sections summarize the possibilities of using animal nutrition to alter the fatty acid profile of milk, which, if adopted on a wide scale, may offer a potential means of reducing cardiovascular disease at a population level.

## Reducing the Saturated Fatty Acid Content of Bovine Milk

Supplements of plant oils or oilseeds rich in unsaturated C18 fatty acids can be used to reduce the proportion of short- and medium-chain fatty acids (C6:0-C16:0) and increase the concentrations of long-chain fatty acids in milk (Grummer 1991, Doreau et al. 1999). These changes are primarily due to long-chain fatty acids (C16 and above) inhibiting *de novo* fatty acid synthesis in the mammary gland and because lipid supplements increase the amount of circulating long-chain fatty acids available for incorporation into milk fat. In general, feeding plant lipids to cows (other than palm oil rich in C16:0) has no effect on milk fat content of C4:0 or long-chain saturates (C16 and above), but consistently increases C18:0 concentrations at the expense of C16:0 (Palmquist et al. 1993, Chilliard et al. 2000). Furthermore, comparison of milk fatty acid responses when oils are fed in the diet compared with rumen-protected sources or duodenal infusions of these lipids indicates that the proportion of C6 and C8 fatty acids are lowered when dietary fats are exposed to ruminal metabolism, whereas the increase in milk C18 content during early lactation or in response to duodenal infusion is associated with a reduction in C10-C16 content (Chilliard et al. 2000). In all cases, inclusion of plant oils and oilseeds in the diet results in an unavoidable increase in milk *trans* C18:1 content in milk due to extensive lipolysis and biohydrogenation of C18 PUFA in the rumen. A number of studies on the effects of plant lipids on milk SFA were summarized by Givens & Kliem (2009).

An example of the effects of reducing the degree of saturation of fatty acids in dairy products was shown by the human dietary intervention study of Noakes et al. (1996) who studied 33 men and women for eight weeks, comparing the effects of fatty acid modified (51% SFA, 39% *cis*-MUFA) dairy products with normal dairy (70% SFA, 28% *cis*-MUFA). The modified products resulted in a significant reduction in total (0.28 mmol/l) and LDL cholesterol (0.24 mmol/l), with HDL cholesterol being unaffected. The authors suggest that if these changes were applied to Western populations, they would represent a potential strategy to lower the risk of CHD by about 10% without the need to change normal eating patterns.

## Increasing the *cis* Monounsaturated Fatty Acid Content of Milk

Although C18:0 is the predominant long-chain fatty acid available for incorporation into milk fat, secretion of *cis*-9 C18:1 in milk exceeds mammary C18:0 uptake due to the activity of stearoyl CoA ( $\Delta$ -9) desaturase activity in mammary secretory cells. Conversion of C18:0 to *cis*-9 C18:1 is the predominant precursor-product of the  $\Delta$ -9 desaturase, transforming about 40% of C18:0 uptake by the mammary gland (Chilliard et al. 2000). It is therefore theoretically possible to exploit the ability of the mammary gland to enhance milk fat *cis*-9 C18:1 by supplementing diets with lipids rich in C18:0, such as tallow or hydrogenated oils. However, this approach does not change the *cis*-9 C18:1:C18:0 ratio in milk fat, and the feeding of tallow to dairy cows is not permitted within the European Union (Chilliard et al. 2000). Feeding plant oils or oilseeds rich in *cis*-9 C18:1 can be used to enhance milk fat *cis*-9 C18:1 content, but unless these sources are effectively protected from ruminal metabolism, this strategy will also increase the concentrations of *trans* C18:1 in milk. Supplements of *cis*-9 C18:1 acyl amides (Jenkins 1998, Looij et al. 2002) or high levels of

rapeseeds or rapeseed oil in the diet (Murphy et al. 1987, Givens et al. 2003, Ryhänen et al. 2005) have been shown to substantially increase milk fat *cis*-9 C18:1 content (Givens & Kliem 2009), but both approaches can cause significant reductions in feed intake that can result in lowered milk production. Recent studies in our laboratory have however shown that rapeseed prepared by careful milling with wheat has substantial potential to be used in dairy cow diets to produce milk with lower saturates and higher *cis*-MUFA in a sustainable way (Givens et al. 2009, Kliem et al. 2009).

### Effect of Increasing Monounsaturated Fatty Acids in Milk on *Trans* Fatty Acids

Including plant oils rich in C18:2 *n*-6 and C18:3 *n*-3 and fish oils in dairy cow diets causes *trans*-11 C18:1 to accumulate in the rumen, and this subsequently leads to increased concentrations in milk. While *trans*-11 C18:1 normally represents the major *trans* fatty acid, increases in it are usually accompanied by increases in other *trans* fatty acids.

There has been concern for many years about the negative health effects of dietary *trans* fatty acids because high intakes have been associated with a substantially increased risk of CHD (Willett et al. 1993, Kromhout et al. 1995, Ascherio et al. 1999), and the more recent meta-analysis of Mensink et al. (2003) indicated that *trans* fatty acids represented a greater risk to CHD than SFA. Early studies in the United Kingdom identified an association between consumption of hydrogenated vegetable and marine oils and deaths from ischemic heart disease (Thomas et al. 1983, Thomas 1992), and by the mid 1990s it was clear that not only did epidemiological data highlight the increased CHD risk, but unique adverse effects on blood lipids were also evident (Ascherio et al. 1999).

Despite the concern about dietary *trans* fatty acids, it seems that the metabolic response to different *trans* fatty acids can be variable, and it is of note that the profile of *trans* fatty acids in milk fat is quite different to that in industrially hydrogenated foods (Shingfield et al. 2008). Four prospective epidemiological studies have examined the relationship between the intake of *trans* fatty acids from ruminant derived foods and the risk of CHD (Willett et al. 1993, Pietinen et al. 1997, Oomen et al. 2001, Jakobsen et al. 2006). None of these studies found a significant positive relationship, and indeed in three of the studies there was a nonsignificant trend toward a negative relationship. These findings are supported by the overarching evidence discussed earlier, that increased milk consumption is associated with a reduction in the risk of ischemic heart disease (Elwood et al. 2004). Mozaffarian et al. (2006) proposed that lack of an increased risk of CHD associated with the intake of *trans* fatty acids from ruminant-derived foods relative to the substantially increased risk from industrially produced *trans* fatty acids may be a result of lower intakes, different bioactivities, or the fact that dairy and meat products contain some other factors that negate any negative effects that the *trans* fatty acids present cause. It is noteworthy, however, that because comparisons of ruminant and industrial *trans* fatty acids have been based on few studies using relative intake data (e.g., quintiles of intakes), the review of Weggemans et al. (2004) examined the relationship between absolute intake (i.e., g consumed per day) of ruminant and industrial *trans* fatty acids and risk of CHD. They reported that where direct comparison was possible, there was no difference in risk between total, ruminant, and industrial *trans* fatty acids for daily intakes up to 2.5 g. At higher daily intakes (>3 g) total and industrial *trans* fatty acids are associated with an increased risk of CHD, but there were insufficient data available on ruminant *trans* fatty acids at this level of intake. Weggemans et al. (2004) therefore concluded that based on the small amount of data available there was no case to discriminate between ruminant and industrial *trans* fatty acids in dietary recommendations or legislation, although subsequently Lock et al. (2005) challenged this interpretation.

Because of the small amount of data available and given that most human intervention studies have evaluated monounsaturated *trans* fatty acids from industrial sources, a study (TRANSFACT) was designed to directly compare the effects of *trans* fatty acids from milk and industrial sources on risk factors for cardiovascular disease in healthy humans (Chardigny et al. 2006). The first output from the TRANSFACT study showed that industrial and ruminant *trans* fatty acids have different effects on CHD risk factors. Only industrial *trans* fatty acids lowered HDL cholesterol, although the responses were greater in women than in men (Chardigny et al. 2008). Another clinical comparison at two intake levels revealed that both sources increased total:HDL cholesterol similarly at a higher intake level (3.7% EI), but at a lower intake level (1.5% EI) there was little difference between a diet containing ruminant-derived *trans* fatty acids and the control diet (Motard-Bélanger et al. 2008). A recent review of 39 clinical studies concluded that all fatty acids with a double bond in the *trans* configuration can raise the ratio of LDL:HDL cholesterol (Brouwer et al. 2010). However, it should be remembered that clinical studies tend to be quite short in duration, and many involve higher intakes in *trans* fatty acids than that which is normally observed. Overall, there appears to be little if any increased risk of CHD from consumption of ruminant *trans* fatty acids, at least at current intakes.

## CONCLUSIONS

Although it is generally accepted that milk and dairy products are foods that provide the diet with important amounts of key nutrients, there has been concern that high levels of consumption would increase the risk of cardiovascular disease and other chronic health problems. This concern has mainly been the result of the fact that milk-derived foods are often the largest single source of dietary SFA. Epidemiological evidence does not support this view, and indeed there are indications that milk may provide some protective effects against vascular disease and other conditions. Despite this, there is intervention evidence that further benefits may be had from consumption of milk/dairy products that have had some of the SFA replaced by *cis*-monounsaturates. This, however, needs further evaluation in more powerful intervention studies.

## SUMMARY POINTS

1. Consumption of whole milk is on the decline in many Western countries; however, since the early 1980s consumption of lower fat milk appears to be increasing. Cheese and butter consumption trends have varied depending on country.
2. Milk and dairy products are important sources of many essential nutrients (such as vitamin B<sub>12</sub> and riboflavin) and have been shown to contribute toward around 60% of the recommended adult calcium and phosphorus intakes.
3. Prospective cohort and clinical studies have revealed that, overall, consumers of greater amounts of milk and dairy products appear not to have an increased risk of coronary heart disease, stroke, or hypertension compared with individuals with lower dairy consumption. Some studies have reported a possible protective effect of dairy products against cardiovascular disease risk.
4. The relative risk of type II diabetes incidence has been reported to be reduced for consumers of high amounts of dairy products compared with low dairy consumption.

5. A high milk and dairy intake may reduce the relative risk of colorectal and, possibly, bladder cancers. However, a meta-analysis of prospective cohort and case-control studies has reported inconclusive results for risk of prostate cancer.
6. Many of the protective effects of milk and dairy consumption are thought to be related to milk calcium content. However, milk also contains other essential nutrients, such as bioactive peptides, that may contribute.
7. Despite epidemiological evidence suggesting protective effects of milk on coronary heart disease, the saturated fat content of milk fat has led to recommendations to reduce milk and dairy product consumption. Studies have shown that changing the fatty acid profile of milk by reducing saturates and increasing *cis*-monounsaturates by altering the dairy cow diet is one strategy for further improving the beneficial effects of milk consumption.

## FUTURE ISSUES

1. Epidemiological evidence relates mainly to full fat milk, and there are few data relating to fat-reduced milk, cheese, butter, cream, etc. Given the trends in dairy product consumption, this needs attention, such that any differential effects can be confirmed.
2. Epidemiology does not ascribe causes or mechanisms. There is a need to follow up the key epidemiological outcomes with well-powered intervention and mechanistic studies.
3. Attention should focus on the effects of milk/dairy foods on vascular health. Newer functional measures, such as endothelial function, should be explored.
4. Intervention evidence is needed to confirm the benefits from consumption of milk/dairy products that have had some of the saturated fatty acids replaced by *cis*-monounsaturates.

## DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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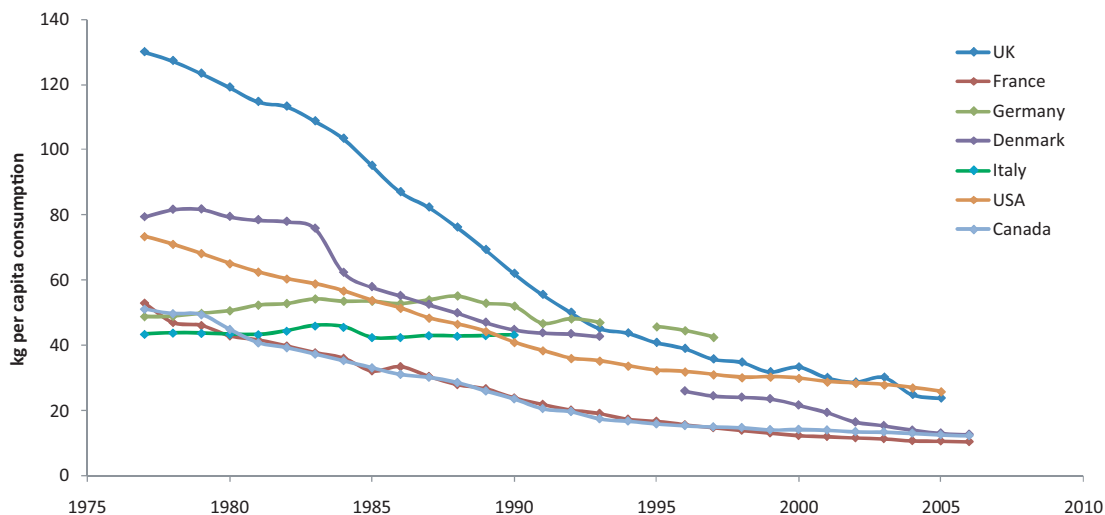
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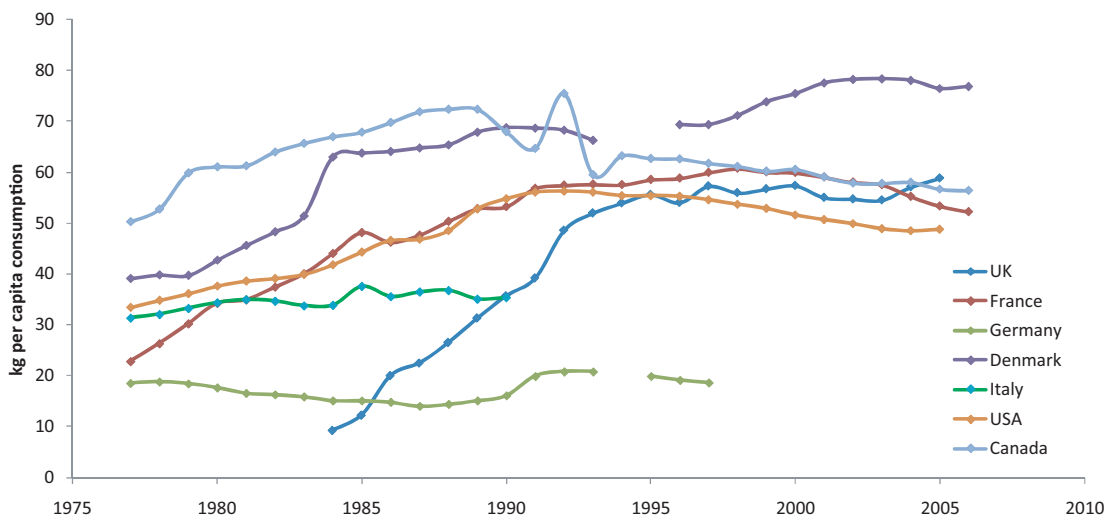


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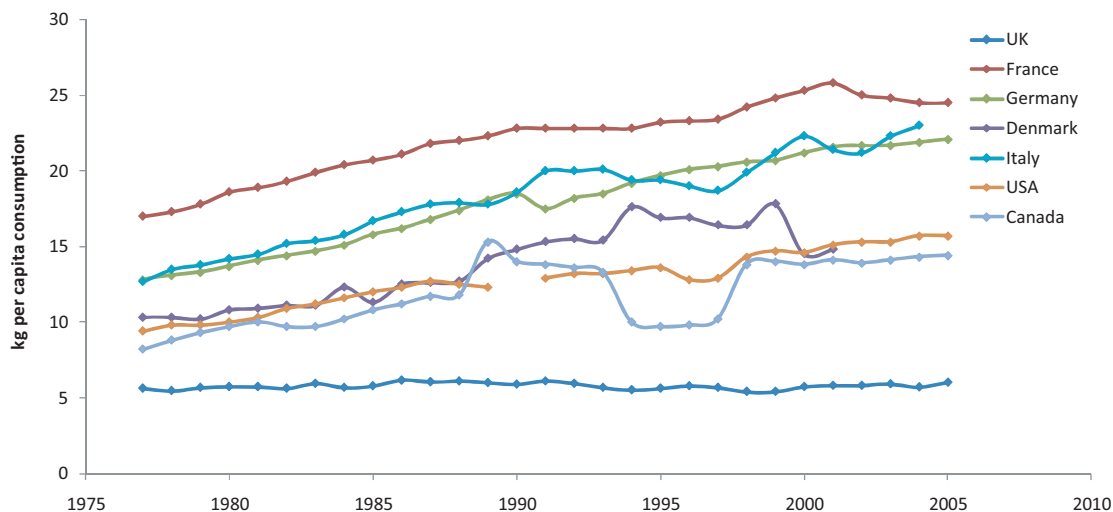
**Figure 1**

Consumption of whole milk in selected countries, 1977–2006. Sources: IDF, personal communication; DEFRA (2001, 2005); CNIEL (2007); ZMP (2007); USDA (2007); Agriculture and Agri-Food Canada (2007); Danish Dairy Board (2007).



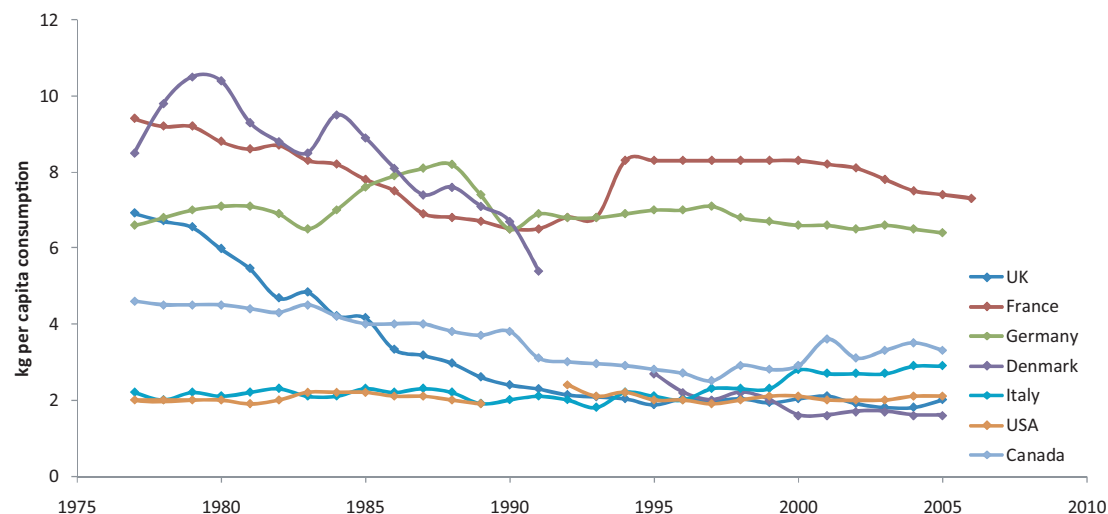
**Figure 2**

Consumption of lower fat milk in selected countries, 1977–2006. Sources: IDF, personal communication; DEFRA (2001, 2005); CNIEL (2007); ZMP (2007); USDA (2007); Agriculture and Agri-Food Canada (2007); Danish Dairy Board (2007).



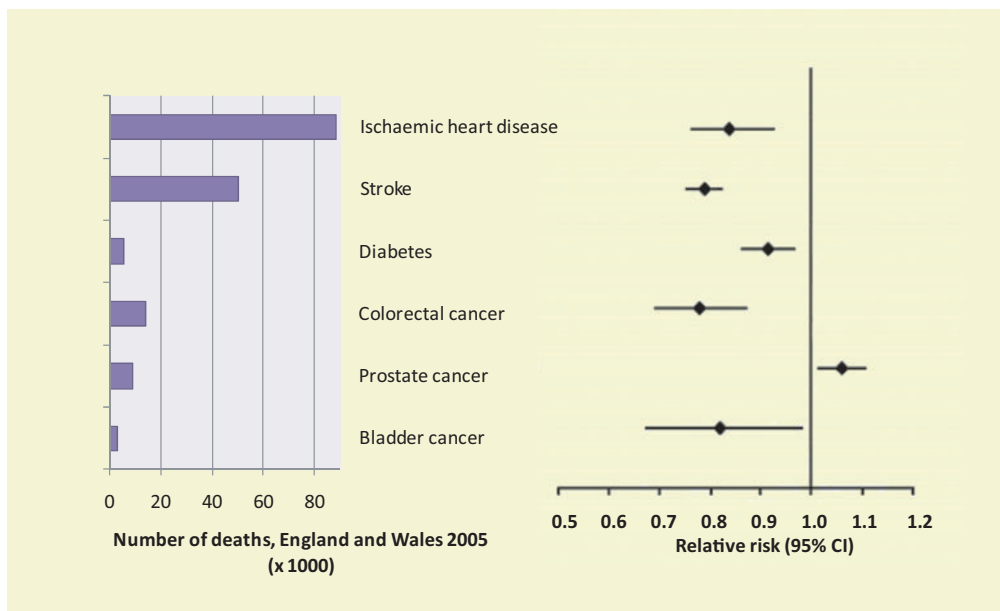
**Figure 3**

Consumption of cheese in selected countries, 1977–2006. Sources: IDF, personal communication; DEFRA (2001 & 2005); CNIEL (2007); ZMP (2007); USDA (2007); Agriculture and Agri-Food Canada (2007); Danish Dairy Board (2007).



**Figure 4**

Consumption of butter in selected countries, 1977–2006. Sources: IDF, personal communication; DEFRA (2001 & 2005); CNIEL (2007); ZMP (2007); USDA (2007); Agriculture and Agri-Food Canada (2007); Danish Dairy Board (2007).



**Figure 5**

Deaths in England and Wales (2005) from various causes versus relative risk for these causes in subjects with highest dairy consumption relative to subjects with lowest dairy consumption (adapted from Elwood et al. 2008).



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## Errata

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