

ORIGINAL COMMUNICATION

Milk drinking, ischaemic heart disease and ischaemic stroke II. Evidence from cohort studies

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Objective: Milk consumption is considered a risk factor for vascular disease on the basis of relevant biological mechanisms and data from ecological studies. The aim was to identify published prospective studies of milk drinking and vascular disease, and conduct an overview.

Design: The literature was searched for cohort studies, in which an estimate of the consumption of milk, or the intake of calcium from dairy sources, has been related to incident vascular disease.

Main outcome measures: Ischaemic heart disease and ischaemic stroke.

Results: In total, 10 studies were identified. Their results show a high degree of consistency in the reported risk for heart disease and stroke, all but one study suggesting a relative risk of less than one in subjects with the highest intakes of milk. A pooled estimate of relative odds in these subjects, relative to the risk in subjects with the lowest consumption, is 0.87 (95% CI 0.74–1.03) for ischaemic heart disease and 0.83 (0.77–0.90) for ischaemic stroke. The odds ratio for any vascular event is 0.84 (0.78–0.90).

Conclusions: Cohort studies provide no convincing evidence that milk is harmful. While there still could be residual confounding from unidentified factors, the studies, taken together, suggest that milk drinking may be associated with a small but worthwhile reduction in heart disease and stroke risk.

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Introduction

Milk has long been considered an important factor in coronary heart disease because of the contribution it makes to the dietary intake of saturated fats. Expert groups have, therefore, advised that milk consumption should be limited, and that fat-reduced milk should be preferred (Nutritional Aspects of Cardiovascular Disease, 1994; Naidoo & Willis, 1994).

There is a large number of ecological studies in which the total supply of various food items in different countries is related to the mortality from vascular or other diseases in the same countries. Commencing with Knox (1973) many authors have demonstrated a relatively strong association

between the *per capita* supply of milk or some constituent of milk and heart disease mortality (Segall, 1977; Seely, 1981; Grant, 1998). In some studies, changes in gross milk production over a period of years and changes in mortality have been shown to correlate (Moss & Freed, 2003; Segall, 1994).

Although a few of these studies take some account of possible confounding and, for example, adjust for differences in overall average smoking rates, or average *per capita* alcohol consumption in the different countries, only very limited attention can be given to confounding. In addition, there are very marked trends in vascular disease incidence across Europe (East to West and North to South) the origins of which are largely speculative, yet the effects of these will also confound the relationships derived from ecological data. Furthermore, it has been pointed out that countrywide food data take no account of food wastage (Grant, 1998).

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Other studies, both observational and experimental, have reported on relationships between milk consumption and risk factors for vascular disease. Milk intake is probably positively related to blood lipid levels (Steinmetz *et al*, 1994; Nagaya *et al*, 1996), but the effect shown in many studies is either trivial or absent (Shaper *et al*, 1991; Jacobsen & Stensvold, 1992; D'Avanzo *et al*, 1995; Abbott *et al*, 1996; Onning *et al*, 1998; Barr *et al*, 2000; Ness *et al*, 2001; Elwood *et al*, 2003). In fact, in some studies a milk supplement led to a decrease in blood lipids (Buonapane *et al*, 1992; St Onge *et al*, 2000; Samuelson *et al*, 2001), leading again to the suggestion that milk may contain substances that counterbalance any expected positive effect on lipid levels (Pfeuffer & Schrezenmeir, 2000). Then an inverse relationship between calcium intake and blood pressure has been consistently reported (Kromhout *et al*, 1985; Jorde & Bonna, 2001). Another vascular risk factor, homocysteine, is raised by animal proteins, including milk (Oshaug *et al*, 1998) and it has been suggested that milk may be of relatively greater importance in this effect than meat because it has a low content of the B group vitamins necessary for its metabolism (Grant, 1998). No relationship with homocysteine was, however, detected with milk intake in the cohort study of Elwood *et al* (2004). Owing to the inconsistencies in the magnitude and the direction of these various associations, it would clearly be unwise to attempt to predict the likely overall relevance of milk consumption on vascular disease risk.

For these reasons, we believe that prospective cohort studies, based on data for individual subjects within a single community, with detailed attention given to confounding, give a much better basis for the examination of independent associations between food items and disease incidence than studies based on either ecological data or relationships with risk factors. We, therefore, report here the results of a systematic search and overview of 10 prospective cohort studies in which incident vascular disease events were related to estimates of milk intake at baseline. We also include data from two retrospective case-control studies.

Methods

The literature was searched with MEDLINE, using the keywords: milk, dairy and dietary calcium, and a range of words appropriate to vascular disease. References to the papers thus identified were examined for further relevant reports. Enquiries were also made with the authors of several reports about possible publications. The original aim was to identify population-based prospective cohort studies, in which milk consumption, or a surrogate for milk consumption, is related to vascular disease incidence and 10 such studies were identified. Two case-control studies were also identified (Gramenzi *et al*, 1990; Tavani *et al*, 2002). A systematic search for case-control studies was not

undertaken and limited use is made of these two in what follows.

The definition of milk intake presented some difficulties. In all the reports, subjects had been divided into three or four subgroups and in most, the basis of the division was an estimate of total milk consumption. Two studies grouped subjects by their intakes of 'milk products' (Bostic *et al*, 1999; Iso *et al*, 1999), while another used calcium from dairy sources (Abbott *et al*, 1996). Yet another defined the group with the highest milk intake as taking milk on cereals and drinking milk (Shaper *et al*, 1991). A further study (Vijver *et al*, 1992) in which the analyses presented are based on estimates of total dietary calcium has been included on the grounds that milk and milk products are not only responsible for a relatively high proportion of the total calcium in the diets of most people, but also because milk and milk products are the most variable source of calcium from person to person. All these have been accepted as reasonable definitions of subjects whose milk consumption is high.

Similar difficulties arose with subjects who had been judged to have the lowest consumption of milk. Most reports refer to subjects drinking less than a defined amount of milk per day, but other authors had grouped subjects under the term 'none'. The group within each study with the lowest milk intake, however this had been defined, was accepted as identifying subjects with intakes of milk that differ greatly from those in the groups with the highest intakes.

There are differences between the studies in the factors for which adjustments are made for possible confounding. These are listed against each study in Table 1. An important confounding factor is total energy intake and the three studies in which it is stated that correction had been made for this are considered separately in the final pooling process.

For most of the studies, the estimates of relative risk of either, or both, ischaemic heart disease and ischaemic stroke are those that are given in the published paper, and no attempt is made to distinguish between 'relative risk', 'hazard ratio', etc. Most reports include 95% confidence intervals of the risk ratio, and for some of the others these have been estimated from data presented in the original report. In order to achieve consistency between these, we have inverted the subgroups upon which the relative risk, as given by some of the authors, had been based. That is, in this overview, the risk of a vascular disease event in the group with the lowest milk intakes is set in all the studies at 1.0 and the risk in the subjects with the highest intakes is shown relative to this.

Pooled estimates of the log-odds for all the cohort studies were obtained by weighting the individual values by the reciprocal of their sampling variances. A χ^2 test was performed to confirm that there was no significant heterogeneity and the variance of the pooled estimates was obtained from the reciprocal of the sum of the weights. Data from the two case-control studies were not included in the pooled estimates.

Table 1 Studies of milk drinking and vascular outcomes

Study	Country	Year of set up	Duration of follow-up	No. of subjects followed	Estimate of milk intake based on
Bostic <i>et al</i> (1999)	USA (Iowa)	1986	8 y	34 486 women aged 55–69 y	Intake of milk products
Shaper <i>et al</i> (1991)	UK	1981	9.5 y	7735 men aged 40–59 y	Milk drunk and taken on cereals
Mann <i>et al</i> (1997)	UK	1980	13 y	10 802 vegetarians aged 16–79 y	Milk drunk
Elwood (2003)	UK (Wales)	1979	22 y	2512 men aged 45–59 y	Milk drunk
Iso <i>et al</i> (1999)	USA (Boston)	1976	14 y	85 764 female nurses aged 34–59 y	Calcium from milk and milk products
Ness <i>et al</i> (2001)	UK (Scotland)	1970	25 y	5765 men aged 35–64 y	Milk drunk
Kinjo <i>et al</i> (1999)	Japan	1965	25 y	223 170 men and women	Times a week milk drunk
Abbott <i>et al</i> (1996)	USA (Hawaii)	1965	22 y	3150 men aged 55–68 y	Calcium from dairy sources
Snowdon <i>et al</i> (1984)	USA (California)	1960	20 y	8724 men, 15 048 women aged 45–64 y	Milk drunk
Vijver <i>et al</i> (1992)	Netherlands	1953	28 y	1340 men, 1265 women aged 40–65 y	Total calcium intake
Gramenzi <i>et al</i> (1990)	Italy	1985	Retrospective	936 female patients aged 22–69 y	Milk drunk
Tavani <i>et al</i> (2002)	Italy	1995	Retrospective	985 patients aged 25–79 y	Milk drunk

Listed in order by the year they were set up.

Results

A short summary of each study follows and further details are given in Tables 1–3. Throughout what follows, the studies are listed in order according to the date in which they were set up and observations commenced.

Data from a prospective study of 35 000 postmenopausal women set up in 1986 were reported by Bostic *et al* (1999). Total calcium, and that from milk products was estimated. The risk for heart disease in the quarter of women with the highest total calcium intake was 0.67 (0.47–0.94) relative to the risk in the quarter of women with the lowest intakes. Subdividing the women by their consumption of milk and milk products gave a relative risk of 0.94 (0.66–1.35) in the quarter with the highest milk intakes.

In the British Regional Heart Study (Shaper *et al*, 1991), 7000 middle-aged men were asked about milk intake in an examination around 1981 and those who both drank milk and took it on cereals were assumed to have the highest intakes. After 9 y, incident heart disease events were related to milk intake. The authors comment that while men who had the highest milk intake at baseline had a lower rate of heart attack than men who drank no milk, there were many differences between these groups in factors of likely relevance to heart disease risk. On adjusting for these factors, the relative risk of heart disease in the men who both drank milk and took it on cereals, compared to those who took no milk, was 0.88 (0.55–1.40).

Mann *et al* (1997) enlisted almost 11 000 male and female members of a vegetarian society, together with their friends and relatives. After 13 y, later these were followed up and 383 deaths identified. The ratio of deaths from all causes in subjects who drank more than half a pint of milk per day, compared with those who drank less than half a pint, was 0.87 (0.68–1.13). Surprisingly, only 63 (16%) of the deaths had been certified as due to ischaemic heart disease and the

ratio in these was 1.50 (0.81–2.78) for the subjects in the highest milk consumption group, relative to the low-consumption group.

In the Caerphilly Prospective Study (Elwood *et al*, 2003), set up in 1979–83, 2500 men completed a food frequency questionnaire. During the following 21 y, the relative risk in men who drank more than a pint per day, compared with the risk in men who drank no milk, was 0.66 (0.24–1.81) for ischaemic heart disease and 0.84 (0.40–1.26) for ischaemic stroke.

The US Nurses Health Study (Iso *et al*, 1999) commenced in 1976. Calcium intakes from dairy sources were estimated for 85 764 women, none of whom had evidence of coronary heart disease. An inverse association was found with the risk of ischaemic stroke during the following 14 y, and this was strengthened when embolic infarctions were omitted. In the fifth of women with the highest dairy calcium intakes, compared with the fifth with the lowest intakes, the adjusted relative risk for ischaemic stroke was 0.70 (0.51–0.97). The authors comment that the inverse association observed with dairy calcium was not restricted to milk, but was also observed for yoghurt, for hard cheese and for ice cream.

In a study set up in Scotland in 1970–73 (Ness *et al*, 2001), almost 6000 men were followed for 25 y. Men who drank more than one and a third pints of milk per day had an adjusted risk of death from coronary heart disease, relative to the men who drank less than a third of a pint per day of 0.90 (0.83–0.97) and their risk of death from stroke was 0.84 (0.31–2.30).

In 1981, over 223 000 middle-aged Japanese men and women were questioned and then followed for 25 y in order to examine associations between dietary intakes and stroke mortality (Kinjo *et al*, 1999). There were 3084 deaths due to cerebral embolism and thrombosis. Risk ratios, adjusted for confounding, were inversely associated with milk

consumption. The relative risk in subjects who drank milk more than four times a week compared with those who drank milk less than once a week was 0.85 (0.77–0.92).

The milk intake of 3000 Hawaiian men was recorded in 1965 (Abbott *et al*, 1996). During the following 22 y, the incidence of stroke in the men who consumed the highest amounts of milk (3.7%) was less than the incidence in men with the lowest milk intakes (7.9%). The authors comment, however, that the decline in stroke risk with increased consumption was modest with intakes of less than 16 oz (0.46l) milk/day. They also noted that during the early years the milk consumed was 'largely' whole milk, but later, 5% of the milk consumed was skimmed.

In 1960 (Snowdon *et al*, 1984), the consumption of certain food items in 25 000 subjects was recorded in California, about half of whom were lacto-vegetarians. Milk drinking was quantified simply as two glasses or more per day. In subjects with this intake, the relative risk of coronary death during the following 20 years, compared with those who drank no milk, was 0.94 in men and 1.11 in women. It is not possible to estimate confidence limits for these ratios.

In 1953–54 (Vijver *et al*, 1992), the dietary calcium intake of 2605 Dutch civil servants was recorded and cardiovascular disease events were identified during the following 28 y. Total dietary calcium intake was derived from the consumption of a wide range of foods, including milk, cheese, eggs and yoghurt. Data for milk consumption alone are not given. Adjusted odds ratios for cardiovascular disease in the fifth of subjects with the highest calcium intakes, compared with the lowest, were 0.77 (0.53–1.11) and 0.91 (0.55–1.50) for male and female members, respectively.

In a retrospective case–control study (Gramenzi *et al*, 1990), 287 women with acute myocardial infarction and 649 control women with other acute disorders who were admitted to hospital during 1983–89 were questioned about the frequency and amount of various foods consumed prior to the onset of symptoms of infarction. The odds ratio for the risk of myocardial infarction in the third of women with the highest consumption of milk, compared with the third with the lowest consumption, was 0.9.

In another case–control study (Tavani *et al*, 2002), 507 patients admitted to hospital with an acute myocardial infarction, and 478 patients admitted with other acute conditions were questioned about their prior consumption of milk. The odds ratios for infarction in patients who had been drinking seven or more cups of milk per week, compared to patients who had drunk no milk, were 0.78 (0.54–1.12) after adjustment for numerous possible confounding factors.

Using the methods described above, a pooled estimate of the relative odds for ischaemic heart disease in the subjects with the highest milk intakes, compared to those with the lowest intakes, in 10 prospective studies is 0.87 (0.74–1.03), and for stroke the pooled estimate is 0.83 (0.77–0.90). A pooled estimate of the odds for a vascular event in the 10 prospective studies, either heart disease or stroke, is 0.84

(0.78–0.90). Total energy intake is an important confounding factor in this context and so a separate pooling was done with the three studies in which adjustment had been made for this (Vijver *et al*, 1992; Bostic *et al*, 1999; Elwood *et al*, 2003). This gave an almost identical estimate, namely 0.85 (0.70–1.03).

Discussion

The most reasonable conclusion from the 10 prospectives is that milk consumption is not associated with any increased risk of heart disease or stroke. The results of only one study (Mann *et al*, 1997) disagree with this conclusion, but the numbers of vascular events in that study are very small and they constitute an unusually small proportion of total deaths.

It could be that differences between high and low milk consumers, in life-style and other factors relevant to vascular disease, and for which no adjustments have been made, could invalidate any conclusions drawn from the studies, either alone or in an overview. Certainly, there are differences between the subgroups of subjects defined in the various studies, but these are small and adjustments are made for possible confounding in different combinations. While residual confounding is therefore possible, it does seem unlikely that a harmful effect of milk on vascular disease could have been missed in all these studies simply because of some important, but as yet unknown, confounding factor(s).

In fact, an examination of the consistency shown by the various studies, together with our estimates of the pooled risk across all the studies (0.84 (0.78–0.90) for ischaemic heart disease and/or ischaemic stroke), would seem to suggest that milk drinking may be associated with a small reduction in vascular disease risk. Furthermore, it could be argued that a degree of confidence is given to this conclusion by the relative consistency in the estimates of risk in the various studies, despite differences in population samples, in design, in definitions and in other aspects.

An issue that cannot be answered on present evidence is whether or not similar results would have been shown had the milk drunk throughout the studies been fat-reduced. In one of the studies (Abbott *et al*, 1996), it is stated that the milk was 'largely' whole milk, and in another (Elwood *et al*, 2003) information obtained retrospectively make it seem likely that most of the subjects had consumed whole milk for most of the period of follow-up. One of the case–control studies (Tavani *et al*, 2002) is helpful on this, in that a comparison of the past diets of the patients gives an odds ratio for infarction in 59 patients who had had a high intake of full-fat milk, relative to 330 who had taken no milk as 0.89 (0.57–1.38) while 145 patients who had had a high intake of semiskimmed milk was virtually identical at 0.83 (0.59–1.16).

Table 2 Milk drinking and heart disease

Study	Definition of group with the highest milk intake	No. of events/deaths	Adjustments for possible confounding	Estimate of risk of IHD ^a
Bostic <i>et al</i> (1999)	Women in top quartile of intake of 'milk products' vs lowest quartile	387 IHD deaths	Age, energy, BMI, waist-hip ratio, diabetes, smoking, Vit. E, saturated fat, oestrogen, alcohol, education, activity	0.94 (0.66–1.35)
Shaper <i>et al</i> (1991)	Men who drank milk and took milk on cereals vs men who took 'none'	608 events	Age, social class, smoking, cholesterol, blood pressure and diabetes	0.88 (0.55–1.40) ^b
Mann <i>et al</i> (1997)	Vegetarians who drank more than $\frac{1}{2}$ pint milk per day vs less than $\frac{1}{2}$ pint	63 IHD deaths	Age, sex, smoking, social class	1.50 (0.81–2.78)
Elwood <i>et al</i> (2004)	Men who drank more than one pint per day vs none or under a third of a pint	440 IHD events	Age, smoking, social class, IHD, BMI, energy, alcohol, fasting cholesterol, HDL cholesterol and triglycerides	0.71 (0.40–1.26)
Ness <i>et al</i> (2001)	Men who drank more than one and a third of a pint per day vs less than one-third	892 IHD deaths	Age, social class, health behaviour and health status	0.68 (0.40–1.13)
Snowdon <i>et al</i> (1984)	Two glasses of milk drunk/day vs none	758 male fatal IHD, 841 female fatal IHD	Age, smoking and other food items, weight, marital status	0.94, 1.11
Vijver <i>et al</i> (1992)	Subjects in top fifth of total calcium intake vs bottom fifth	366 male IHD deaths, 178 female IHD deaths	Age, smoking, BMI, systolic BP, cholesterol, energy, alcohol	0.77 (0.53–1.11) ^b , 0.91 (0.55–1.50) ^b
Gramenzi <i>et al</i> (1990)	Women in top third of 'portions' of milk drunk per week vs lower third	287 events	Age, education, smoking hyperlipidaemia, diabetes, hypertension and BMI	0.90
Tavani <i>et al</i> (2002)	Subjects who had drunk seven or more cups of milk vs those who had drunk none	Not stated	Age, sex, education, BMI, serum cholesterol, smoking, coffee, alcohol, vegetables etc. (see text)	0.78 (0.62–1.34)

Listed in order by the year they were set up.

^aIHD=ischaemic heart disease.

^b95% confidence limits estimated by us for this report.

Table 3 Milk drinking and stroke

Study	Definition of group with the highest milk intake	No. of events/deaths	Adjustments for possible confounding	Estimate of risk (95% CI)
Elwood <i>et al</i> (2004)	Men who drank one or more pint per day vs none	173 ischaemic strokes	Age, smoking, social class, IHD, BMI, energy, alcohol, fasting cholesterol, HDL cholesterol and triglycerides	0.66 (0.24–1.81)
Iso <i>et al</i> (1999)	Women in top 1/5 dairy Ca intake vs lowest 1/5	347 nonthrombo embolic strokes	Age, smoking, time interval, BMI, alcohol, menopause, hormone use, exercise, multivitamins, fatty acid intake, history of hypertension, diabetes and cholesterol	0.70 (0.51–0.97)
Ness <i>et al</i> (2001)	Men who drank more than one and a third pint per day vs less than one-third	196 stroke deaths	Social class, health behaviour and health status	0.84 (0.31–2.30)
Kinjo (1999)	Milk four times+/week vs less than once/week	3085 embolic and thrombotic strokes	Sex, age, area, smoking, alcohol, occupation	0.85 (0.77–0.92)
Abbott <i>et al</i> (1996)	Men who consumed 16 oz/day vs nondrinkers of milk	229 thromboembolic strokes	Age, dietary K and Na, alcohol, smoking, activity, cholesterol and glucose, uric acid and haematocrit	0.67 (0.45–1.00) ^a

Listed in order by the year they were set up.

^a95% confidence limits estimated by us for this report.

All the cohort studies in the present review had, however, been set up at times when reduced-fat milks were unavailable, or scarce. Towards the end of the observation period in most of the studies, skimmed or semiskimmed milk may well have been used by an increasing proportion of the surviving subjects, but evidence on this is inadequate to enable a comparison of early and late disease events within the studies. Only two studies give evidence on the type of milk consumed (Abbott *et al*, 1996; Elwood *et al*, 2003), but it would seem reasonable to assume that the risk estimates obtained relate largely to the consumption of whole milk.

Milk makes a contribution to total dietary fat consumption, and to the intake of saturated fat of most subjects, but in most subjects and in most communities, this contribution is likely to be relatively small. A study of foodstuffs purchased by 224 women in UK of different ethnic origin, led to the judgement that full-fat milk contributed around 5% of the fat in their total food purchases (Lip *et al*, 1995).

Conclusion

In total 10 cohort studies that examine the association between milk consumption and vascular disease risk have been identified in the literature. Except for one, with a small number of outcome events, none gives convincing evidence of any increased risk of ischaemic heart disease or ischaemic stroke in subjects who have the highest milk consumption. While chance or residual confounding cannot be ruled out with certainty, the pooled estimate of the risk of an incident vascular disease event in such subjects, relative to subjects with a low milk intake, is around 0.87 (0.74–1.03) for ischaemic heart disease, 0.70 (0.55–0.88) for ischaemic stroke and 0.84 (0.78–0.90) for either event.

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References

Abbott RD, Curb JD, Rodriguez BL, Sharp DS, Birchfield CM & Yano K (1996): Effect of dietary calcium & milk consumption on risk of thrombo-embolic stroke in older middle aged men: The Honolulu Heart Program. *Stroke* **27**, 813–818.

Barr SI, McCarron DA, Heaney RP, Dawson-Hughes B, Berga SL, Stern JS & Oparil S (2000): Effects if increased consumption of fluid milk on energy and nutrient intake, body weight, and cardiovascular risk factors in healthy older adults. *J. Am. Diet. Assoc.* **100**, 810–817.

Bostic RM, Kushi LH, Wu Y, Meyer KA, Sellers TA & Folsom AR (1999): Relation of calcium, vitamin D, and dairy food intake to ischaemic heart disease mortality among postmenopausal women. *Am. J. Epidemiol.* **149**, 151–161.

Buonapane GJ, Kilara A, Smith JS & McCarthy RD (1992): Effect of skim milk supplementation on blood cholesterol concentration, blood pressure and triglycerides in a free-living human population. *J. Am. Coll. Nutr.* **11**, 56–67.

D'Avanzo B, Negri E, Nobili A & Vecchia C (1995): Frequency of consumption of selected indicator foods and serum cholesterol. GISSI-EFRIM investigators. *Eur. J. Epidemiol.* **11**, 269–274.

Elwood PC, Pickering JE, Fehily AM, Hughes J & Ness AR (2004): Milk drinking, ischaemic heart disease and ischaemic stroke. I. Evidence from the Caerphilly Cohort. *Eur. J. Clin. Nutr.* **58**, 711–717.

Gramenzi A, Gentile A, Fasoli M, Negri E, Parazzini F & Vecchi CL (1990): Association between certain foods and risk of acute myocardial infarction in women. *BMJ* **300**, 771–773.

Grant WB (1998): Milk and other dietary influences on coronary heart disease. *Altern. Med. Rev.* **3**, 281–294.

Iso H, Stampfer MJ, Manson JE, Rexrode K, Hennekens CH, Colditz GA, Speizer FE & Willett WC (1999): Prospective study of calcium, potassium and magnesium intake and risk of stroke in women. *Stroke* **30**, 1772–1779.

Jacobsen BK & Stensvold I (1992): Milk — a better drink? Relationships with total serum cholesterol in a cross sectional survey. The Nordland Health Study. *Scand. J. Soc. Med.* **20**, 204–208.

Jorde R & Bonna KH (2001): Calcium from dairy products, vitamin D intake and blood pressure: the Tromso Study. *Am. J. Clin. Nutr.* **73**, 659–660.

Kinjo Y, Beral V, Akiba S, Key T, Mizuno S, Appleby P, Yamaguchi N, Watanabe S & Doll R (1999): Possible protective effect of milk and fish for cerebrovascular disease mortality in Japan. *J. Epidemiol.* **9**, 268–274.

Knox EG (1973): Ischaemic heart disease mortality and dietary intake of calcium. *Lancet* **1**, 1465–1467.

Kromhout D, Bosschieter EB & Coulanderr CD (1985): Potassium, calcium, alcohol intake and blood pressure: The Zutphen Study. *Am. J. Clin. Nutr.* **45**, 1299–1304.

Lip GY, Malik I, Luscombe C, McCarron M & Beevers G (1995): Dietary fat purchasing habits in whites, blacks and Asian peoples in England — implications for heart disease prevention. *Int. J. Cardiol.* **48**, 287–293.

Mann JI, Appleby PN, Key TJ & Thorogood M (1997): Dietary determinants of ischaemic heart disease in health conscious individuals. *Heart* **78**, 450–455.

Moss M & Freed D (2003): The cow and the coronary, biochemistry and immunology. *Internal J. Cardiol.* **87**, 203–216.

Nagaya T, Yoshida H, Hayashi T, Takahashi H, Kawai M & Matsuda Y (1996): Serum lipid profile in relations to milk consumption in a Japanese population. *J. Am. Coll. Nutr.* **15**, 625–629.

Naidoo J & Willis J (1994): *Health Promotion: Foundation for Practice*. London: Bailliere Tindall.

Ness AR, Davey Smith G & Hart C (2001): Milk, coronary heart disease and mortality. *J. Epidemiol. Community Health* **55**, 379–382.

Nutritional Aspects of Cardiovascular Disease (1994): *Report of the Cardiovascular Review Group: the Committee on Medical Aspects of Food Policy*, London: HMSO.

Onning G, Akesson B, Oste R & Lundquist I (1998): Effects of consumption of oat milk, soya milk, or cow's milk on plasma lipids and antioxidant capacity in healthy subjects. *Ann. Nutr. Metab.* **42**, 211–220.

Oshaug A, Bugge KH & Refsum H (1998): Diet, an independent determinant for plasma total homocysteine. *Eur. J. Clin. Nutr.* **52**, 7–11.

Pfeuffer M & Schrezenmeir J (2000): Bioactive substances in milk with properties decreasing risk of cardiovascular diseases. *Br. J. Nutr.* **85**, S155–S159.

Samuelson G, Bratteby LE, Mohsen R & Vessby B (2001): Dietary fat intake in healthy adolescents: inverse relationships between the estimated intake of saturated fatty acids and serum cholesterol. *Br. J. Nutr.* **85**, 333–341.

- Seely S (1981): Diet and coronary disease: a survey of mortality rates and food consumption statistics of 24 countries. *Med. Hypotheses* **7**, 907–918.
- Segall JJ (1994): Dietary lactose as a possible risk factor for ischaemic heart disease. *Int. J. Cardiol.* **46**, 197–207.
- Segall JJ (1977): Is milk a coronary health hazard? *Br. J. Prev. Soc. Med.* **31**, 81–85.
- Shaper AG, Wannamethee G & Walker M (1991): Milk, butter and heart disease. *BMJ* **302**, 786–787.
- Snowdon DA, Phillips RL & Frazer GE (1984): Meat consumption and fatal ischaemic heart disease. *Prev. Med.* **13**, 490–500.
- Steinmetz KA, Childs MT, Stimson C, Kushi LH, McGovern PG, Potter JD & Yamanaka WK (1994): Effect of consumption of whole milk and skim milk on blood lipid profiles in healthy men. *Am. J. Clin. Nutr.* **59**, 612–618.
- St Onge MP, Farnworth ER & Jones PJH (2000): Consumption of fermented and non-fermented dairy products: effects on cholesterol concentrations and metabolism. *Am. J. Clin. Nutr.* **71**, 674–681.
- Tavani A, Gallus S, Negri E, Al & Vecchia C (2002): Milk, dairy products and coronary heart disease. *J. Epidemiol. Community Health* **56**, 471–472.
- Vijver L Van der, Wall M Van der, Wetterings KGC, Dekker JM, Schouten EG & Kok FJ (1992): Calcium intake and 28-year cardiovascular and coronary heart disease mortality in Dutch civil servants. *Int. J. Epidemiol.* **21**, 36–39.