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# Is dairy product consumption associated with the incidence of CHD?

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## Abstract

*Objective:* Studies examining the association of dairy consumption with incident CHD have yielded inconsistent results. The current prospective study examined the association between dairy consumption and CHD in a population-based sample of older community-dwelling adults.

*Design:* Baseline CHD risk factors were assessed and an FFQ was self-administered. Participants were followed for morbidity and mortality with periodic clinic visits and annual mailed questionnaires for an average of 16·2 years, with a 96 % follow-up rate for fatal and non-fatal CHD.

*Setting:* Community.

*Subjects:* Participants were 751 men and 1008 women aged 50–93 years who attended a clinic visit in 1984–1987.

*Results:* At baseline the mean age was 70·6 (SD 9·8) years for men and 70·1 (SD 9·3) years for women. Participants who developed CHD during follow-up were significantly older ($P < 0·001$), had higher BMI ($P = 0·035$) and higher total cholesterol ($P = 0·050$), and were more likely to be male ($P < 0·001$), diabetic ($P = 0·011$) and hypertensive ($P < 0·001$), than those who did not develop CHD. Multivariate regression analyses adjusting for age, BMI, diabetes, hypertension, LDL-cholesterol and oestrogen use (in women) indicated that women who consumed low-fat cheese 'sometimes/often' and women who consumed non-fat milk 'sometimes/often' had an increased risk of incident CHD (hazard ratio = 2·32; 95 % CI 1·57, 3·41) and CHD (hazard ratio = 1·48; 95 % CI 1·02, 2·16) compared with women who 'never/rarely' ate these dairy products.

*Conclusions:* Woman with higher intake of low-fat cheese and non-fat milk seem to have a higher risk of incident CHD. This needs further investigation considering recent evidence of cardiovascular benefits from certain dairy fat.

**Keywords**
CHD
CHD risk factors
Dairy consumption

Milk consumption has been considered a risk factor for atherosclerosis and CHD due to its relatively high cholesterol, SFA and lactose content[1]. However, many studies examining the association of milk products with CHD have yielded inconsistent results[2–41]. Concern about an adverse effect of milk consumption on vascular disease risk is supported by reports showing that diets high in cholesterol and saturated fat increase the risk for CVD[2–5]. However, more recent studies indicate that Ca and protein in dairy products may be associated with reduced blood pressure and risk of stroke[42,43]. The Dietary Approaches to Stop Hypertension (DASH) study has shown substantial blood pressure reductions achieved by simply adding low-fat dairy products to a healthy food and vegetable diet[44]. Furthermore, studies have suggested that dairy or Ca contributes to maintaining a healthy body weight[45].

Hu *et al.* reported that consumption of high-fat dairy products in the Nurses' Health Study was associated with an increase in CHD risk[6]. With the exception of the Nurses' Health Study, however, most cohort studies do not support an association between dairy consumption and increased risk for CHD and suggest, instead, that dairy products may help prevent CHD[10–13,30,33–35]. In a meta-analysis of twenty studies on milk or dairy consumption and incidence of vascular disease conducted in 2008, Elwood *et al.* reported a reduction of 10–15 % in the incidence of heart disease in the men and women who reported drinking the most milk[14]. However, a more recent meta-analysis (2011) of prospective cohort studies found no consistent association between milk and CHD risk[15]. The evidence for and against milk and CHD risk was also debated in a series of commentaries which concluded that there was no clear support for a harmful

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or protective effect of milk on CHD[16,17]. The Collaborative Study, a prospective cohort study, conducted in an era when most people consumed full-fat milk, found no evidence that men who consumed milk each day were at increased risk of death from all causes or CHD[18]. More recently, three cohort studies have found that individuals who consumed low-fat dairy compared with high-fat dairy were less likely to develop CHD[29,40,41].

The purpose of the present study was to examine the association of dairy product consumption (milk, non-fat milk, cream, ice cream, yoghurt, cheese, low-fat cheese, cream cheese, cottage cheese, butter, hot chocolate and milk chocolate) with CHD in a well-characterized cohort of men and women who provided detailed information on diet at a time before milk and dairy restrictions were commonly recommended, and who were followed for up to 20 years for CHD morbidity and mortality.

## Experimental methods

Between 1972 and 1974, 82% of all adult residents of a southern California community, Rancho Bernardo, participated in a study of heart disease risk factors. All were Caucasian, middle class and community dwelling. They have been followed since initial enrolment with periodic clinic visits and annual mailed questionnaires. Between 1984 and 1987, 82% of local survivors (*n* 2837) aged 40 years or older at baseline participated in a follow-up clinic visit. After excluding eighty-nine participants less than 50 years of age at baseline, four using aspirin, thirty-three premenopausal women, 115 with C-reactive protein >10 mg/l and 498 who had known CHD, there remained 751 men and 1008 postmenopausal women who form the basis of the present report. The study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by the University of California, San Diego Human Research Protections Program. Written consent was obtained from all individuals prior to participation.

### *Procedures*

During the 1984–1987 visits a standardized interview was used to obtain medical history and information on lifestyle variables including: alcohol consumption (number of drinks per week), smoking (never/past/current) and exercise three or more times per week (no/yes). Information on dairy and other food intake was obtained using a 128-item self-administered FFQ that contained questions regarding portion size and consumption frequency (never, 1–11 times/year, 1–3 times/month, 1–3 times/week, 4–6 times/week and daily) of common food items, including, but not limited to: milk, non-fat milk, cream, ice cream, yoghurt, cheese, low-fat cheese, cream cheese, cottage cheese, butter, hot chocolate and milk chocolate.

Blood samples were obtained by venepuncture between 07.00 and 11.00 hours after a requested fast for 12–16 h. Serum and plasma were separated and stored at −70°C until they were analysed for blood lipids and C-reactive protein. Height and weight were measured with participants wearing light clothing and no shoes. BMI, calculated as weight (in kilograms) divided by the square of height (in metres), was used as an estimate of obesity. Systolic blood pressure and diastolic blood pressure were measured by a nurse trained in the Hypertension Detection and Follow-up Program Protocol[46] after the participant had been seated quietly for 5 min; the average of two measurements was used. Current medication use was validated with prescriptions and containers brought to the clinic for that purpose.

Lipids and lipoproteins were assayed using fresh plasma in a Lipid Research Clinics laboratory at the University of California, San Diego (San Diego, CA, USA). Total cholesterol and TAG levels were measured by enzymatic techniques using an ABA-200 biochromatic analyser (Abbott Laboratories, Irving, TX, USA). HDL-cholesterol was measured by precipitating other circulating lipoproteins with heparin and manganese chloride according to the standard Lipid Research Clinics protocol[47]. LDL-cholesterol was estimated using the Friedewald formula[48]. Plasma glucose levels were measured by the glucose oxidase method.

Medical history and incident CHD information were obtained using standard questionnaires at research clinic visits approximately every 4 years thereafter and from annual mailed surveys. Follow-up continued over 20 years through 2007. Death certificates were obtained for 90% of all decedents and coded by a certified nosologist using the International Classification of Diseases 9th revision (ICD-9) criteria; decedents whose death certificates were not yet available were excluded from this analysis.

### *Statistical analysis*

Prevalent CHD was defined as doctor-diagnosed myocardial infarction, angina or coronary artery bypass grafting in 1984–1987. Study participants were classified as incident CHD cases if at any time during the follow-up period they had a fatal or non-fatal myocardial infarction, coronary artery bypass grafting or angina (grades 1 or 2 by Rose criteria)[49]. Myocardial infarction was diagnosed based on self-reported history of physician-diagnosed myocardial infarction. Vital status was known for 96% of participants. Fatal CHD included deaths assigned ICD-9 codes 410–414. Participants who were alive and event-free were censored at the end of follow-up (31 December 2007) or at the date of their last follow-up for the 4% for whom vital status was unknown. Individuals who died of non-CHD causes were censored at time of death.

To achieve greater statistical power, we collapsed dairy intake categories into two categories: 'never/rarely' (defined as 0–11 times/year) and 'sometimes/often'

(defined as 1–3 times/month, 1–3 times/week, 4–6 times/week and daily). Alcohol consumption was not normally distributed and was log transformed for analysis. Age was examined as a continuous variable and also categorically by decade (50–59, 60–69, 70–79 and 80–89 years). Hypertension was defined as systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg or use of antihypertensive medication[50]. Type 2 diabetes was defined using the 1999 WHO diabetes criteria, defined as fasting plasma glucose ≥126 mg/dl, post-challenge glucose ≥200 mg/dl, a history of diabetes diagnosed by a physician or use of diabetes-specific medication[51,52].

Means and rates were calculated for continuous and categorical demographic and dairy variables. Comparisons of individuals with and without incident CHD were performed with $t$ tests and $\chi^2$ analyses for continuous and categorical variables, respectively. Age-adjusted comparisons of those with and without incident CHD on demographic and lifestyle variables, death outcome and dairy consumption were performed. Exploratory model analyses were completed to assess multicollinearity, and then simultaneous regression analyses were performed to examine the association between each dairy product with incident CHD after adjusting for potentially confounding covariates. Stratified analyses and interaction terms were included to examine the sex-specific association between dairy consumption and incident CHD. No significant interactions were found between dairy consumption and sex; however, sex-specific analyses are presented for incident CHD to allow comparisons with results of other studies.

Multivariate Cox regression models were used to examine the association between consumption of each dairy product and incident CHD. All models met the proportional hazards assumption; none of the regression results were significantly influenced by outliers. The proportionality assumption for significant factors was tested graphically and by including time-dependent covariates in the model. Follow-up duration was calculated from date of research clinic visit until death, outcome, loss to follow-up or 31 December 2007, whichever came first. Results were expressed as hazard ratios (HR) with 95% confidence intervals. Unadjusted univariate associations of each covariate as predictors of CHD events were examined and included as covariates in later models. Model 1 adjusted for age and BMI; model 2 adjusted for age, BMI, diabetes, hypertension, LDL-cholesterol and current oestrogen use in women. All $P$ values are two-sided; $P \leq 0{\cdot}05$ was considered statistically significant. All data manipulation and analyses were conducted using the SAS statistical software package version 9·1·3 (2006).

## Results

As shown in Table 1, average age of participants was 70·6 (SD 9·8) years in men and 70·1 (SD 9·3) years in women. In the total population, few participants were current smokers (13%) and 82% of participants reported physical exercise, usually walking, three or more times weekly; hypertension was common (71%) and 40% of women were currently using oestrogen therapy. Of the 1759 older adults included in the study, 451 (222 men and 229 women) reported their first CHD event during the 20 years of follow-up. Table 1 also shows the proportion of men and women who reported 'never/rarely' or 'sometimes/often' consuming the following dairy products: cheese, ice cream, milk chocolate, butter, cream, non-fat milk, yoghurt, cream cheese, cottage cheese, hot cocoa and low-fat cheese. Cheese consumption was reported as 'sometimes/often' consumed by 92% and 90% of men and women, respectively. Ice cream, whole milk and cottage cheese were reported as 'sometimes/often' consumed by 75%, 70% and 56% of men, respectively, and 66%, 61% and 65% of women, respectively.

Comparisons of baseline characteristics by the presence of a first CHD event are shown in Table 2. Participants who developed CHD were significantly older ($P < 0{\cdot}001$), had higher BMI ($P = 0{\cdot}035$) and higher total cholesterol ($P = 0{\cdot}050$), and were more likely to be male ($P < 0{\cdot}001$), diabetic ($P = 0{\cdot}011$) and hypertensive ($P < 0{\cdot}001$), than those who did not develop CHD. No significant differences between CHD cases and non-cases were observed for alcohol consumption, TAG, HDL-cholesterol, smoking, exercise or current oestrogen use (in women). There were no differences in baseline dairy consumption between participants with or without incident CHD (all $P > 0{\cdot}10$; Table 3).

Results of Cox proportional hazard models examining the time-to-event association of dairy product intake with incident CHD after adjustment for CHD risk factors, including age, BMI, diabetes, hypertension, LDL-cholesterol and current oestrogen use (in women), are shown in Table 4 for both men and women. In partially adjusted and fully adjusted models, there were no significant associations between CHD and dairy product consumption in men (see Table 4).

In unadjusted models (not shown), women had an increased risk of CHD if they 'sometimes/often' consumed non-fat milk as compared with 'never/rarely' consuming non-fat milk (HR = 1·49; 95% CI 1·10, 2·03) and if they 'sometimes/often' consumed low-fat cheese as compared with 'never/rarely' consuming low-fat cheese (HR = 1·68; 95% CI 1·21, 2·35). After adjusting for age and BMI, women who 'sometimes/often' as compared with 'never/rarely' consumed non-fat milk had a significantly increased risk of CHD (HR = 1·55; 95% CI 1·14, 2·12) that was slightly attenuated but stayed significant after adjusting for all other CHD risk factors (HR = 1·48; 95% CI 1·02, 2·16; Table 4). Likewise, women who consumed low-fat cheese 'sometimes/often' as compared with 'never/rarely' had a significantly increased risk of CHD (HR = 2·32; 95% CI 1·57, 3·41) in the multivariate analyses adjusting for all CHD risk factors. The survival curves of these associations are plotted in Figs 1 and 2,

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**Table 1** Demographic characteristics of Rancho Bernardo participants, 1984–1987 (*n* 1759)

| | Men (*n* 751) | | Women (*n* 1008) | |
|---|---|---|---|---|
| Variable | Mean | SD | Mean | SD |
| Age (years) | 70·6 | 9·8 | 70·1 | 9·3 |
| BMI (kg/m$^2$) | 25·9 | 3·3 | 24·1 | 3·7 |
| Total cholesterol (mg/dl) | 212·4 | 54·9 | 234·8 | 78·3 |
| HDL-cholesterol (mg/dl) | 54·2 | 14·6 | 70·1 | 18·7 |
| LDL-cholesterol (mg/dl) | 134·6 | 56·1 | 144·1 | 85·1 |
| TAG (mg/dl) | 122·3 | 88·5 | 110·3 | 63·8 |
| | Median | IQR | Median | IQR |
| Alcohol (g/week) | 4·8 | 4·1–5·3 | 4·6 | 3·9–5·1 |
| | *n* | % | *n* | % |
| Diabetes | 116 | 15·5 | 97 | 9·6 |
| Hypertension | 544 | 72·4 | 700 | 69·4 |
| Smoking | | | | |
| Current | 80 | 10·7 | 151 | 15·0 |
| Former | 444 | 59·1 | 371 | 36·8 |
| Never | 227 | 30·2 | 486 | 48·2 |
| Strenuous exercise ≥ 3 times/week | 640 | 85·2 | 798 | 79·2 |
| Current oestrogen user* | – | 0·0 | 402 | 39·9 |
| Dairy product consumption | Sometimes/often† (%) | Never/rarely‡ (%) | Sometimes/often† (%) | Never/rarely‡ (%) |
| Cheese | 92·4 | 7·6 | 90·1 | 9·9 |
| Ice cream | 75·4 | 24·6 | 65·9 | 34·1 |
| Whole milk | 69·8 | 30·2 | 61·4 | 38·6 |
| Cottage cheese | 55·7 | 44·3 | 65·1 | 34·9 |
| Milk chocolate | 45·1 | 54·9 | 43·6 | 56·4 |
| Butter | 38·8 | 61·2 | 33·6 | 66·4 |
| Cream | 32·4 | 67·6 | 33·9 | 66·1 |
| Non-fat milk | 32·2 | 67·8 | 40·0 | 60·0 |
| Yoghurt | 24·8 | 75·2 | 40·0 | 60·0 |
| Cream cheese | 21·9 | 78·1 | 25·2 | 74·8 |
| Hot cocoa | 16·4 | 83·6 | 14·7 | 85·3 |
| Low-fat cheese | 15·7 | 84·3 | 23·6 | 76·4 |

IQR, interquartile range.
Due to rounding, percentages may not add up to 100.
*Assessed only in women, *n* 721.
†Sometimes/often includes 1–3 times/month, 1–3 times/week, 4–6 times/week and daily.
‡Never/rarely includes never and 1–11 times/year.

respectively. As shown in Figs 1 and 2, there is clear separation of survival risk for women according to high or low intake of non-fat milk and low-fat cheese.

## Discussion

In the present study consumption of dairy products, in general, was not related to incident CHD. However, we did find that women who 'sometimes/often' consumed non-fat milk and women who 'sometimes/often' consumed low-fat cheese had a significantly increased risk of incident CHD compared with those who 'never/rarely' consumed these dairy products, after including the known covariates of CHD mortality in the model.

We found no association between intake of the majority of the dairy products and CHD, a finding consistent with previous literature[15,17,20,23,32,36,37]. For example, in a case–control study of 378 men and 129 women aged 25–79 years, Tavani *et al.* concluded that neither total milk, whole milk, semi-skimmed milk, yoghurt nor cheese was associated with increased CHD risk in men and women[1]. Likewise, no significant association was found for milk and butter consumption among the 2818 men in the prospective British Regional Heart Study[38]. In a more recent case–control study conducted in Costa Rican adults, dairy product intake (butter, buttermilk, cheeses, cream, ice cream, lactocrema, milk and yoghurt), as assessed by adipose tissue and by FFQ, was not associated with an increase in the risk of myocardial infarction[28].

The results of the present study by gender are in contrast to several other studies that report dairy intake increases CHD risk. In a recent population-based cohort study of rural Swedish men with 12 years of follow-up, men who consumed low-fat dairy compared with high-fat dairy were 1·43 times more likely to have CHD[33]. The Nurses' Health Study found that the ratio of high-fat to low-fat dairy food consumption was positively associated with an

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**Table 2** Age-adjusted comparison characteristics in participants with and without CHD, Rancho Bernardo, 1984–1987

| | Non-CHD cases (*n* 1308) | | CHD cases (*n* 451) | | |
|---|---|---|---|---|---|
| Variable | Mean | SD | Mean | SD | *P** |
| Age (years) | 69·6 | 9·7 | 72·3 | 8·5 | 0·001 |
| BMI (kg/m$^2$) | 24·7 | 3·6 | 25·1 | 3·9 | 0·035 |
| Total cholesterol (mg/dl) | 223·9 | 68·7 | 229·1 | 74·0 | 0·050 |
| HDL-cholesterol (mg/dl) | 64·3 | 18·9 | 60·3 | 18·3 | 0·407 |
| LDL-cholesterol (mg/dl) | 138·1 | 72·6 | 145·5 | 78·6 | 0·038 |
| TAG (mg/dl) | 112·8 | 74·4 | 123·0 | 78·3 | 0·184 |
| | Median | IQR | Median | IQR | |
| Alcohol (g/week) | 4·6 | 4·0–5·3 | 4·7 | 3·9–5·3 | 0·389 |
| | *n* | % | *n* | % | |
| Men | 529 | 40·4 | 222 | 49·2 | 0·001 |
| Diabetes | 142 | 11·0 | 69 | 15·5 | 0·011 |
| Hypertension | 897 | 68·6 | 346 | 76·7 | 0·001 |
| Smoking | | | | | 0·401 |
| Current | 173 | 13·2 | 58 | 12·9 | |
| Former | 594 | 45·4 | 221 | 49·0 | |
| Never | 541 | 41·4 | 172 | 38·1 | |
| Strenuous exercise ≥ 3 times/week | 1075 | 82·2 | 363 | 80·5 | 0·421 |
| Current oestrogen user† | 220 | 41·3 | 61 | 35·5 | 0·176 |

IQR, interquartile range.
Due to rounding, percentages may not add up to 100.
Alcohol was log transformed.
*$P$ value from $t$ test for continuous variables and from $\chi^2$ test for categorical variables.
†Assessed only in women.

increased risk of CHD among women[6]. More recently, the Nurses' Health Study found that a high-fat dairy diet in combination with a higher intake of red meat was significantly associated with elevated risk of CHD[29]. A case–control study showed that only butter (but not cheese, yoghurt, low-fat dairy or high-fat milk) was significantly positively associated with the risk of first myocardial infarction[53].

The null results of the present study for dairy product consumption and CHD observed in men and the positive associations of dairy product consumption with CHD in women are somewhat comparable to a Dutch study examining dairy consumption and 10-year total and cardiovascular death. In that study of 16 136 men and women, researchers found low-fat cheese intake in men was associated with a small increased risk of all-cause mortality[30]. The CARDIO2000 multicentre case–control study found that consumption of milk, cheese and yoghurt in 700 and 148 Greek men and women, respectively, was significantly associated with lower odds of having acute coronary events, specifically acute myocardial infarction and unstable angina (OR = 0·88; 95% CI 0·83, 0·93)[34]. One other study did find a similar association between cheese consumption and CHD in women. Sonestedt *et al.* explored the association between the intake of specific dairy products and incidence of CHD among 26 445 middle-aged men and women in the Swedish Malmö Diet and Cancer (MDC) cohort. Cheese intake was significantly associated with decreased CHD risk in women but not in men[40]. The absence of a clear consistent association between dairy and CHD could be due to inconsistent study designs. While ecological studies were the first to report an association between dairy consumption and CHD, ecological studies are typically regarded as weaker studies because they are susceptible to the ecological fallacy. Case–control studies are used to identify factors that may contribute to a medical condition by comparing individuals who have a disease with individuals who do not have the disease but are otherwise similar. A limitation to case–control studies is that they rely on memory and are subject to recall bias (people with a condition will be more motivated to recall risk factors). Case–control studies can only assess association, not causation. Cohort studies follow a population over time to determine which risk factors are associated with the development of a disease or outcome. Since information on risk factors is ascertained before development of disease, cohort studies can infer causation. Meta-analyses have the highest level on the hierarchy of evidence. A meta-analysis is a method for systematically combining pertinent qualitative and quantitative study data from several selected studies to develop a single conclusion that has greater statistical power. This conclusion is statistically stronger than the analysis of any single study, due to increased numbers of subjects, greater diversity among subjects, and/or accumulated effects and results. Previous small-sample studies, and/or relatively short follow-up, may be insufficient to assess mortality. Some studies did not include both sexes and/or included younger participants who are at low risk for CHD. Additionally, most previous studies failed to report whole and non-fat dairy separately and excluded

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**Table 3** Age-adjusted comparisons of participants with and without incident CHD on dairy characteristics, Rancho Bernardo, 1984–1987

| Variable | Non-CHD | CHD | *P** |
|---|---|---|---|
| Whole milk | | | |
| Sometimes/often† (%) | 64·5 | 65·9 | 0·59 |
| Never/rarely‡ (%) | 35·5 | 34·1 | |
| Non-fat milk | | | |
| Sometimes/often† (%) | 36·0 | 37·8 | 0·49 |
| Never/rarely‡ (%) | 64·0 | 62·2 | |
| Cheese | | | |
| Sometimes/often† (%) | 91·6 | 89·4 | 0·16 |
| Never/rarely‡ (%) | 8·4 | 10·6 | |
| Butter | | | |
| Sometimes/often† (%) | 36·9 | 33·6 | 0·22 |
| Never/rarely‡ (%) | 63·1 | 66·4 | |
| Ice cream | | | |
| Sometimes/often† (%) | 69·4 | 71·0 | 0·55 |
| Never/rarely‡ (%) | 30·6 | 29·0 | |
| Cottage cheese | | | |
| Sometimes/often† (%) | 60·6 | 61·1 | 0·85 |
| Never/rarely‡ (%) | 39·5 | 38·9 | |
| Yoghurt | | | |
| Sometimes/often† (%) | 33·5 | 32·3 | 0·63 |
| Never/rarely‡ (%) | 66·5 | 67·7 | |
| Cream | | | |
| Sometimes/often† (%) | 33·3 | 33·6 | 0·90 |
| Never/rarely‡ (%) | 66·7 | 66·4 | |
| Milk chocolate | | | |
| Sometimes/often† (%) | 44·9 | 41·7 | 0·24 |
| Never/rarely‡ (%) | 55·1 | 58·3 | |
| Low-fat cheese | | | |
| Sometimes/often† (%) | 19·4 | 21·0 | 0·47 |
| Never/rarely‡ (%) | 80·6 | 79·0 | |
| Cream cheese | | | |
| Sometimes/often† (%) | 23·1 | 24·9 | 0·45 |
| Never/rarely‡ (%) | 76·9 | 75·1 | |
| Hot chocolate | | | |
| Sometimes/often† (%) | 15·4 | 14·1 | 0·51 |
| Never/rarely‡ (%) | 84·6 | 85·9 | |

Due to rounding, percentages may not add up to 100.
*$P$ value from $\chi^2$ test.
†Sometimes/often includes daily, 4–6 times/week, 1–3 times/week and 1–3 times/month.
‡Rarely/never includes never and 1–11 times/year.

information on consumption of other types of dairy products (cream, cheese, butter). A double-blind randomized controlled study by Malpuech-Brugere *et al.*, which examined the impact of milk fats on CVD risk factors, found that consumption of dairy fat with naturally present ruminant total *trans*-fatty acids (different from the industrially manufactured harmful *trans*-fatty acids) improved some CVD risk factors (decrease in total cholesterol, decrease in LDL-cholesterol and decrease in LDL-cholesterol:HDL-cholesterol) for healthy volunteers in comparison to a dairy fat diet with low total *trans*-fatty acids[31]. The levels of ruminant *trans*-fatty acids in dairy fats is influenced by the type of diet the cows feed on[54]. Another study by Mozaffarian *et al.* reported on plasma *trans*-palmitoleic acid (one of the above-mentioned dairy *trans*-fatty acids), which was found to be highly correlated with intake of whole-fat dairy products and to predict lower incidence of type 2 diabetes and a favourable metabolic profile[55]. These recent studies may explain the negative association of dairy products and risk of CVD, and support the higher risk of CVD among women who consumed low-fat cheese found in our study.

The present study is the first to report an increase in CHD mortality among women due to non-fat milk and low-fat cheese consumption. Although not commonly advocated at the time, women with high levels of cholesterol at baseline in 1984–1987 may have been told to limit dairy to non-fat forms. Our results may also be due to having a family history of CHD; we could not verify these reports in the present study. It should also be noted that the association observed between 'sometimes/often' and 'never/rarely' consumed low-fat cheese in women includes a small number of individuals in this category. Several limitations of the study should be noted. This sample of

**Table 4** Crude and adjusted associations of dairy consumption with risk of incident CHD morbidity and mortality, Rancho Bernardo, 1984–1987 and 2007

| | Men | | | | Women | | | |
|---|---|---|---|---|---|---|---|---|
| | Model 1* | | Model 2† | | Model 1‡ | | Model 2§ | |
| Variable | HR | 95 % CI | HR | 95 % CI | HR | 95 % CI | HR | 95 % CI |
| Non-fat milk | 1·05 | 0·76, 1·45 | 1·08 | 0·78, 1·49 | **1·55** | **1·14, 2·12** | **1·48** | **1·02, 2·16** |
| Yoghurt | 1·16 | 0·83, 1·63 | 1·20 | 0·85, 1·68 | 1·12 | 0·81, 1·54 | 1·32 | 0·90, 1·92 |
| Ice cream | 1·25 | 0·88, 1·78 | 1·25 | 0·88, 1·79 | 0·93 | 0·67, 1·30 | 0·96 | 0·64, 1·45 |
| Low-fat cheese | 0·96 | 0·64, 1·44 | 0·98 | 0·65, 1·46 | **1·72** | **1·23, 2·41** | **2·32** | **1·57, 3·41** |
| Cheese | 1·20 | 0·68, 2·11 | 1·23 | 0·70, 2·18 | 0·81 | 0·52, 1·26 | 0·71 | 0·43, 1·20 |
| Cottage cheese | 1·19 | 0·87, 1·61 | 1·21 | 0·89, 1·66 | 0·84 | 0·61, 1·16 | 1·04 | 0·71, 1·53 |
| Cream | 0·94 | 0·68, 1·31 | 0·94 | 0·68, 1·31 | 1·15 | 0·83, 1·60 | 1·10 | 0·74, 1·62 |
| Cream cheese | 1·00 | 0·70, 1·43 | 1·03 | 0·72, 1·48 | 1·29 | 0·92, 1·80 | 1·33 | 0·89, 1·99 |
| Whole milk | 1·00 | 0·72, 1·39 | 0·99 | 0·71, 1.38 | 1·05 | 0·76, 1·45 | 1·01 | 0·68, 1·49 |
| Milk chocolate | 0·88 | 0·65, 1·20 | 0·88 | 0·64, 1·20 | 0·99 | 0·72, 1·35 | 1·06 | 0·73, 1·53 |
| Butter | 1·04 | 0·76, 1·42 | 1·06 | 0·77, 1·46 | 0·86 | 0·61, 1·22 | 0·88 | 0·58, 1·33 |
| Hot chocolate | 0·91 | 0·58, 1·43 | 0·96 | 0·61, 1·51 | 0·92 | 0·59, 1·45 | 0·91 | 0·53, 1·54 |

HR, hazard ratio.
Significant associations are shown in **bold** font.
*Adjusted for age and BMI.
†Adjusted for age, BMI, diabetes, hypertension and LDL-cholesterol.
‡Adjusted for age and BMI.
§Adjusted for age, BMI, diabetes, hypertension, LDL-cholesterol and current oestrogen use.

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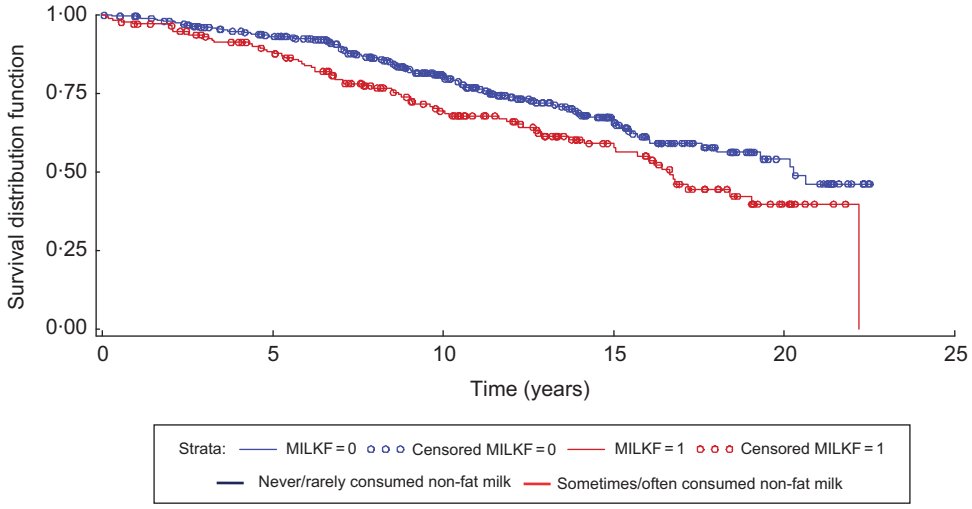


**Fig. 1** (colour online) Hazard curve of non-fat milk (MLKF) consumption in Rancho Bernardo women with risk of incident CHD morbidity and mortality, 1984–1987 and 2007

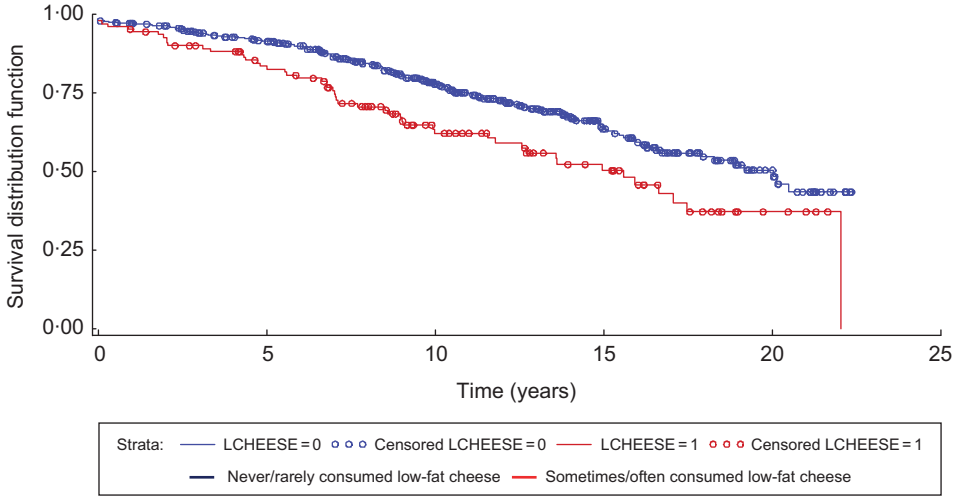


**Fig. 2** (colour online) Hazard curve of low-fat cheese (LCHEESE) consumption in Rancho Bernardo women with risk of incident CHD morbidity and mortality, 1984–1987 and 2007

community-dwelling, older, Caucasian, middle-class adults living in southern California may not be representative of the general US population, thus limiting the generalizability of the results. However, this homogeneity means that associations are less confounded by socio-economic status and ethnicity. Additionally, exercise, smoking, alcohol consumption and dairy consumption were all self-reported. However, the high educational level of this cohort makes it more likely that self-reports are valid. In Rancho Bernardo participants, self-reported alcohol intake was indirectly validated by parallel increases in HDL-cholesterol with increasing alcohol intake, a well-known consequence of alcohol consumption[56,57]. Participation bias – whereby those who were ill may not have participated – cannot be excluded. A limitation of exposure assessment was the need to combine categories of dairy and milk intake due to small sample size within the original categories of frequency of intake. Although this could have attenuated the association, we kept the participants who rarely used these products as a well-defined group and compared them with all others. Dairy consumption was measured with a validated FFQ, which was based on self-reported intake. This may have introduced misclassification of dairy intake, which could have attenuated the associations. However, an advantage of the FFQ is that it measures food consumption over a year, which is relevant for chronic diseases[58]. The positive associations observed in the present study could be a chance finding due to multiple comparisons, and further studies with larger sample size need to specifically address these associations. However, recent evidence does provide biological plausibility of these associations[6,33].

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The present study also has a number of strengths. Its prospective design allows for investigation of the risk of CHD mortality associated with dairy consumption over a 20-year follow-up period and avoids recall bias. We obtained robust hazard estimates and plots of adjusted cumulative probability of mortality over time while controlling for confounding and adjusting for minimal loss to follow-up. The uniquely characterized population is at an increased risk of CHD because of their much older age than most previous study populations, therefore increasing the potential for finding any true association with dairy intake.

## Conclusions

Dairy product consumption assessed in a sample of older men and women was not statistically associated with CHD, providing assurance to elderly who need dairy Ca sources to maintain healthy bones. Non-fat milk and low-fat cheese intake among women were related to CHD, an association that requires further investigation in other cohort studies that utilize measures of different dairy fats.

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