Milk, coronary heart disease and mortality

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Abstract

Study objective—To study the association between reported milk consumption and cardiovascular and all cause mortality.

Design—A prospective study of 5765 men aged 35–64 at the time of examination.


Participants—Men who completed a health and lifestyle questionnaire, which asked about daily milk consumption, and who attended for a medical examination.

Main results—150 (2.6%) men reported drinking more than one and a third pints a day, Some 2977 (51.6%) reported drinking between a third and one and a third pints a day and 2638 (45.8%) reported drinking less than a third of a pint a day. There were a total of 2350 deaths over the 25 year follow up period, of which 892 deaths were attributed to coronary heart disease. The relative risk, adjusted for socioeconomic position, health behaviours and health status for deaths from all causes for men who drank one third to one and a third pints a day versus those who drank less than a third of a pint was 0.90 (95% CI 0.83, 0.97). The adjusted relative risk for deaths attributed to coronary heart disease for men who drank one third to one and a third pints a day versus those who drank less than one third of a pint was 0.92 (95% CI 0.81, 1.06).

Conclusions—No evidence was found that men who consumed milk each day, at a time when most milk consumed was full fat milk, were at increased risk of death from all causes or death from coronary heart disease.

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The effects on population health of increased intake of milk are uncertain. In the first half of this century increased milk production and consumption was encouraged, particularly in children, as it was believed to improve vitality and shown to increase growth in children.1 After the second world war the epidemic of coronary heart disease (CHD) and the accumulating evidence that increased saturated fat intake, by increasing blood cholesterol levels, increased risk of coronary disease,2,3 led to a review of policy. Increasingly, in recent years, the public has been encouraged both to reduce their milk intake and to switch to low fat milk products in order to reduce their fat consumption.4 Recent studies, however, have suggested that increased fat intake may be protective for stroke.5

Ecological analyses have shown high correlations between milk intake and coronary mortality rates6,7 (which tend to be at least as strong as those observed for saturated fat). The focus of many observational studies on dietary constituents rather than food items means that the individual based data available on milk and cardiovascular risk are limited. Given the relative lack of prospective data on the association between milk intake, CHD and other diseases we have analysed the association between milk intake and mortality in a large cohort of men in the west of Scotland on whom we have detailed information regarding important potential confounding factors.

Methods

This analysis is based on a cohort of 5765 men aged 35–64 at the time of examination recruited from workplaces in the west of Scotland between 1970 and 1973. Full details of the present study population (The Collaborative Study) and the data collection methods have been described previously.8,9

The information collected at baseline included: sociodemographic data, health status measures, information on health related behaviours and measurements recorded at a physical examination. Data are available on the subject’s age, their father’s occupation and their occupation at the time of screening (coded to social class). Data are also available on the subject’s age at leaving full time education, their number of siblings and whether they were regular car drivers. In addition, the home address at the time of screening was retrospectively allocated a postcode, enabling an area-based deprivation category at the time of the 1981 census to be ascertained. Deprivation category varies from 1 (least deprived) to 7 (most deprived). Details of self reported angina from the Rose questionnaire (with angina defined as definite)10 and self reported respiratory symptoms from the Medical Research Council questionnaire11 are available. Detailed information on smoking and alcohol consumption12 was recorded. At examination height, weight, blood pressure and lung function were measured, an electrocardiogram performed and blood drawn for estimation of plasma cholesterol and triglycerides. Forced expiratory volume in one second, FEV1, expressed as a percentage of the expected FEV1, for age and height, was calculated from the subset of healthy participants.7

Men were sent a self completed questionnaire. One question asked men how many pints of milk they usually drank each day. The answers to this question were checked when the men attended for screening. The responses to this question were used to allocate men into three groups. Men who drank less than a third
Health Service Central Registry in Edinburgh, which also provides death certificates coded according to the ninth revision of the International Classification of Diseases (ICD).

Continuous variables have been age standardised using PROC GLM, in the SAS system, with tests for trend for age adjusted means being obtained through PROC REG. Categorical variables have been age standardised by the direct method and tests for trend performed in PROC LOGIST. Proportional hazards coefficients and their standard errors were calculated using PROC PHREG with adjustment for age and other risk factors by including terms for these in the proportional hazards models. Exponentiated hazards coefficients are taken as indicators of relative rates of mortality. For missing values of variables a dummy variable was created and used in the adjustments. Only for father’s social class was there a considerable number of missing values. Cronbach’s α was calculated to compare the reliability of responses to the same question on frequency of milk consumption five years apart.

Results

Some 45.8% of the men reported drinking less than a third of a pint a day and 51.6% reported drinking more than a third of a pint a day and less than one and a third pints day. Only 2.6% reported drinking over one and a third pints per day. The characteristics of different groups of milk drinkers are shown in table 1. Non-milk drinkers tended to be shorter, older and of lower socioeconomic position and were more likely to be current cigarette smokers, to drink more alcohol and to have worse lung function than milk drinkers. There was little difference in blood pressure, cholesterol concentration, triglyceride concentration, body mass index and prevalence of ECG ischaemia between the milk consumption groups.

Over the 25 year follow up period 2350 men died, 892 of CHD, 196 of stroke, 266 of lung cancer, 448 of other cancers and 548 of other causes. The association between reported milk consumption and mortality is shown in table 2. Milk consumption was inversely associated with all cause, cardiovascular, coronary heart disease, non-lung cancer and other mortality. As milk consumption was associated with a more favourable profile of socioeconomic and behavioural risk factors for mortality the relative rates were adjusted for a comprehensive range of relevant potential confounding factors. These adjustments attenuated slightly the apparent protective effect of milk consumption but the associations with all cause mortality and cardiovascular mortality remained statistically significant at conventional levels. The analyses were repeated splitting the follow up period into deaths in the first 10 years and deaths in the next 15 years. The results were essentially unchanged for all cause, cardiovascular and coronary mortality (data not shown).

Cronbach’s α was calculated for the 2686 men who were screened twice, on average five
years apart. This yielded a value of 0.59, representing a reasonable level of stability in reported milk consumption.

Discussion
In this prospective study of Scottish working men we have found no evidence that regular consumers of milk are at increased risk of death from heart disease or death from all causes. In fact the age adjusted risk of mortality from all causes and mortality from cardiovascular disease was lower in those drinking between one third and one and a third pints a day. Though adjustment attenuated the relative risk slightly it remained statistically significant for deaths from all causes. The risk of death from stroke was also reduced in regular milk drinkers, though this reduction was not statistically significant and the confidence intervals were wide.

It is likely that there is measurement error in our assessment of usual milk consumption, which was based on a single question about milk drunk. The question did not ask men about milk used in food preparation. Neither did it ask men about intake of skimmed or semi-skimmed milk though data from the British National Food Survey suggest that consumption of reduced fat milks was unusual in the 1970s. The reasonable agreement between repeat measures some years apart suggests that this question was reproducible. Any measurement error associated with our measure of milk consumption will have reduced our ability to detect any real milk mortality associations. Such measurement error is random and though it might explain a failure to find an adverse association with coronary death or death from all causes in regular milk drinkers it is unlikely to explain the apparent protective association observed.

Confounding is a possible alternative explanation for the associations we have observed as men who consume milk regularly may be healthier or live healthier lives. The observations that regular milk consumers have better risk profiles and that regular milk consumption is associated with reduced risk of death from a number of different causes (both coronary heart disease and cancers unrelated to smoking) support this notion. However, adjustment for a large number of potential confounding variables results in only modest attenuation of the observed associations. Though we cannot discount confounding as an explanation for these findings it seems unlikely that residual confounding is masking a markedly increased risk of coronary death, or death from all causes, in regular milk drinkers.

If regular milk consumption is not associated with increased risk of death and particularly coronary death (as predicted by the classic diet-heart hypothesis) why might this be? One possible explanation is that milk does not raise serum cholesterol (despite its high saturated fat content). Observations that the Maasai (a semi-nomadic people living in East Africa) who consume large quantities of milk have low serum cholesterol concentrations led to speculation that milk might contain a cholesterol lowering factor or factors. Further studies in the Maasai, however, suggest that their calorie intake may be lower and their milk consumption higher than in industrialized western societies. It is possible that their consumption of milk has been high for many years and that it is the effect of children consuming milk in childhood that is protective. The studies of the Caerphilly cohort and those of the Seventh Day Adventists both involved higher milk consumption at an earlier age than in the UK. This is consistent with data from the British National Food Survey which suggest that a milk diet was encouraged more frequently in the UK in the 1970s. The reasonable agreement between repeat measures some years apart suggests that this question was reproducible. Some studies of increased milk intake in free living humans have reported reduced serum cholesterol concentrations, but controlled feeding studies, despite their small size, seem to confirm that increased milk intake does indeed increase serum cholesterol concentrations. Though milk contains other factors that may increase coronary risk, such as lactose it also contains some that may reduce coronary heart disease risk, such as calcium. Also if milk consumption in adulthood is associated with milk consumption in childhood, this might explain the association. Increased milk in childhood leads to increased growth and thus increased height. And increased leg length in childhood and height in adulthood is associated with reduced coronary and all cause mortality.

A necropsy study showed a high prevalence of coronary heart disease in peptic ulcer patients treated with a milk diet in the UK and the USA. In the UK arm of the study 18% of necropsied ulcer patients on a milk diet had evidence of myocardial infarction compared with 3% of necropsied ulcer patients not given a milk diet (p<0.01). A case-control study of women in Italy reported a modest (non-significant) reduction in risk of myocardial infarction with increased milk consumption—an odds ratio of 0.9 in women drinking milk daily compared with those drinking milk weekly.

We are aware of seven prospective studies that have examined the association between milk consumption and coronary, cerebrovascular or all cause mortality. In a cohort study of over 25 000 Seventh Day Adventists the relative risk of coronary death was 0.94 (p<0.05) in men drinking two glasses a day versus none but for women the relative risk was 1.11 (and not statistically significant). In the Caerphilly cohort study there was a striking inverse association between milk consumption and coronary heart disease risk. In the 2818 men the unadjusted relative risk of major ischaemic disease was 0.12 (95% CI 0.03 to 0.53) for men drinking one or more pints a day compared with those drinking none. Data from the British Regional Heart study suggested that the apparent protective effect observed in the Caerphilly study might be a

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result of men with existing coronary heart disease reducing their milk intake because of their illness, thus generating an association between milk intake and future coronary events. It was also suggested that confounding by socioeconomic position, cigarette smoking and other coronary heart disease risk factors might have accentuated the association between milk consumption and coronary heart disease incidence. Nevertheless, even after adjustment the relative risk of fatal and non-fatal cardiovascular disease among the 7735 men in the British Regional Heart Study was 0.88 for men who drank milk and had milk on their cereal compared with those who did not. The Basel study of 2974 male employees of three pharmaceutical companies after eight years reported that: “Milk consumption and coronary atherosclerosis at autopsy (1980 mortality follow-up) shows a trend for an inverse correlation (p=0.12).” In the Honolulu Heart Study milk consumption was associated with reduced risk of thromboembolic stroke—in 3150 men of Japanese ancestry who reported drinking 16 oz of milk per day had half the stroke risk of non-milk drinkers. Temporal analyses from Japan have also shown that increased milk consumption is associated with decreased stroke mortality. In a study of over 10 000 UK vegetarians the death rate ratio for deaths from all causes was reduced in those who drank more than half a pint a day compared with those who drank less than half a pint a day: 87 (95% CI 68 to 1113). And in those that drank half a pint a day the death rate ratio compared with those that drank less than half a pint a day was 70 (95% CI 55 to 88). The death rate ratio for ischaemic heart disease was 150 (95% CI 81 to 278) in those drinking more than half a pint a day but 76 (95% CI 40 to 143) for those drinking half a pint a day. Finally, in a cohort of over 34 000 middle aged Iowa women the adjusted risk of coronary death in those in the top quartile of consumption of dairy products (excluding butter) versus the lowest quartile was 0.94 (95% CI 0.66 to 1.35, p for trend =0.64).

So in summary we have found no evidence that men who reported regular consumption of milk were at increased risk of death from coronary heart disease, or death from all causes. While the modest protective associations we observed for deaths from all causes and deaths from coronary heart disease could be explained by residual confounding by dietary or non-dietary exposures our data do not support the notion that regular consumption of milk is hazardous to health.

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