Milk and coronary heart disease mortality

The self completed questionnaire study by Ness et al found that men who reported drinking more than a third of a pint of milk each day were not at increased risk of death from coronary heart disease (CHD) in comparison with those drinking less than this or none.

The study inappropriately equates daily milk consumption with the answers the participants gave to the single question that asked how many pints of milk the men usually drank each day. This was in 1970 or 1973, when in Britain milk was normally delivered to households in pint bottles. The meaning of the question lacks precision, and may have caused some of the participants to exclude milk taken out of the home in hot beverages and in milk drinks such as milk shakes. The authors note that the question did not ask about milk used in food preparation, so some answers could have excluded regular milk intake in porridge, with cereals, and as milk puddings or custard. Milk in milk chocolate and milk or constituents in ready made foods would certainly have been excluded. It is feasible that some participants who drank less than a third of a pint of milk as such consumed more milk from these other sources than did those who reported drinking more than this amount.

Five years, on average, after the initial screening, about half of the participants returned for a second screening, which apparently showed “a reasonable level of stability in reported milk consumption.” This does not necessarily translate into the same relation with CHD mortality of the reported milk drinking of the whole cohort at the initial screening and of the half at the second screening. The latter results should therefore have been included in the published paper. The follow up period took place against a background of falling milk consumption in the United Kingdom. The significance of relating levels of milk intake in 1970 and 1973 to CHD mortality up to 25 years later is therefore questionable, especially as lifestyle changes that include restriction of milk to one cup daily can bring about regression of coronary atherosclerosis in one year. In the discussion, Ness et al state that the Caerphilly cohort study showed a striking inverse association between milk consumption and CHD risk, but they fail to refer to the final report on this study, which shows that none of the nutrients considered was significantly associated with major CHD events when standardised for energy. The authors refer to the possible CHD protective effect of dietary calcium, but not to an experimental dyslipidaemic effect of defatted skimmed milk, which provided a high calcium intake. This apparent paradox can be explained by the enhancing effect of lactose on the absorption of calcium, which reduces the availability of intestinal calcium for forming insoluble calcium salts of fatty and bile acids. This action of lactose supports ecological evidence that a high intake of this disaccharide could be the dietary risk factor for CHD in milk.

Elwood in his commentary considers that the avoidance of cow’s milk in adults would be a formidable undertaking for a randomised clinical trial. However, a RCT for secondary prevention of CHD to compare usual dietary advice with this plus the exclusion of lactose (and galactose) should not present undue difficulties.

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LETTERS TO THE EDITOR

Table 1 Relative rates of mortality by milk consumption on two screenings in 2686 men from the Collaborative study in the 21 year follow up period after the second screening

<table>
<thead>
<tr>
<th>Milk consumption (1st/2nd screening)</th>
<th>Some/Some</th>
<th>Some/None</th>
<th>None/Some</th>
<th>None/None</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number of men</strong></td>
<td>979</td>
<td>481</td>
<td>337</td>
<td>889</td>
</tr>
<tr>
<td>All cause</td>
<td>366</td>
<td>176</td>
<td>158</td>
<td>395</td>
</tr>
<tr>
<td>No of deaths</td>
<td>1</td>
<td>1.00 (0.84 to 1.20)</td>
<td>1.22 (1.01 to 1.47)</td>
<td>1.14 (0.99 to 1.31)</td>
</tr>
<tr>
<td>Age adjusted relative rate*</td>
<td>1</td>
<td>0.98 (0.74 to 1.06)</td>
<td>1.17 (0.97 to 1.41)</td>
<td>1.00 (0.87 to 1.16)</td>
</tr>
<tr>
<td>Fully adjusted relative rate*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD</td>
<td>143</td>
<td>75</td>
<td>60</td>
<td>152</td>
</tr>
<tr>
<td>Age adjusted relative rate</td>
<td>1</td>
<td>1.09 (0.82 to 1.44)</td>
<td>1.19 (0.88 to 1.61)</td>
<td>1.14 (0.90 to 1.43)</td>
</tr>
<tr>
<td>Fully adjusted relative rate</td>
<td>1</td>
<td>0.90 (0.68 to 1.19)</td>
<td>1.17 (0.86 to 1.58)</td>
<td>0.99 (0.78 to 1.24)</td>
</tr>
</tbody>
</table>

*Adjusted for age, smoking, diastolic blood pressure, cholesterol, body mass index, adjusted FEV1, social class, father’s social class, education, deprivation category, siblings, car user, angina, ECG ischaemia, bronchitis, and alcohol consumption.

Authors’ reply

We accept, as we discussed in our paper, that our assessment of milk consumption was crude and may have affected our ability to detect associations. Even so, the available epidemiological evidence, in our opinion, does not lend support to Segall’s view that milk (by virtue of its lactose content) consumed in moderation is harmful to health. We did not refer to the 1993 report of the Caerphilly study as this did not include any data on milk consumption and coronary heart disease. We did look at the associations between milk consumption measured on two

References


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occasions and mortality and included it in our original submission but were advised by one of the referees to remove it. We are happy to publish it with this letter (table 1). The men who reported some milk consumption on both occasions experienced the lowest mortality, although differences were small and not statistically robust.

We agree that trials could be carried out to confirm whether these associations are causal. Based on experience with previous food based secondary prevention trials, however, we believe that such trials would have to be large, intensive and run for some years to provide a definitive answer.

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References
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