

Invited Commentary

Dairy, dairy, quite contrary: further evidence to support a role for calcium in counteracting the cholesterol-raising effect of SFA in dairy foods

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Dietary saturated fat and CVD are inextricably linked through the positive influence of SFA, with a chain length of between twelve and sixteen carbons, on serum LDL-cholesterol concentration. While the association between saturated fat and CVD has recently been called into question by meta-analysis⁽¹⁾, the cholesterol paradigm continues to provide a supporting rationale for restricting the intake of foods with a high content of saturated fat, primarily for the purpose of reducing CVD risk. However, since foods are complex mixtures of nutrients that are consumed within a varied diet, it is perhaps not surprising that a high content of saturated fat is no guarantee that a food will exert an adverse effect on serum LDL-cholesterol. Likewise, the health benefits associated with a specific nutrient in one type of food may not necessarily translate into equivalent efficacy in another. In both cases, the pattern of food consumption, accompanying diet and composition, and nature of the food matrix may all exert effects that modify the biological response to a bioactive component.

In 2009, the UK Food Standards Agency launched its 'saturated fat and energy campaign' (www.food.gov.uk/news/pressrelease/2009/feb/launchsatfatcampaign) that aimed to reduce the contribution of saturated fat to the average intake of food energy from 13.4 to 11% or below, by 2010, a target, which if met, was predicted to save an impressive 3500 lives a year. There are two important recommendations for achieving this target: (1) increasing consumer understanding and awareness of the impact of eating SFA on CVD and (2) the need for industry to reformulate foods to reduce their SFA content. These recommendations signified major implications for the dairy industry, not least because of the widely held view that dairy foods exert unfavourable effects on blood cholesterol and thus cardiovascular health, and the reality that milk and milk products, including cheese and butter, contribute approximately 30% of SFA to the UK diet⁽²⁾. In practice, advice to restrict dairy foods might be all well and good, if it were not for the lack of evidence to link dairy foods with CVD. There is actually no clear evidence from prospective cohort studies for a consistent association between dairy foods and CVD^(3,4), and a greater tendency for a protective, inverse association with CVD, and favourable effects of certain dairy foods on blood pressure and serum LDL- and HDL-cholesterol concentrations. Furthermore,

there are credible data to support mechanisms to explain how these effects occur^(5,6). These include the anti-hypertensive effects of bioactive peptides in milk that act as inhibitors of angiotensin-1-converting enzyme, and the binding and sequestration of SFA and bile acids in the gut by Ca.

The study by Lorenzen & Astrup in this issue of the *British Journal of Nutrition*⁽⁷⁾ revisits the idea that high Ca in dairy can attenuate the LDL-raising effects of SFA by increasing the excretion of SFA and bile acids. It tested the effects of four experimental diets, based on milk, that contained combinations of high Ca or low Ca and high and low dairy fat (SFA), in nine normal healthy males for 10 d in a randomised cross-over design. Its principal finding was a significant increase in serum LDL-cholesterol in response to the high fat/low Ca that was diminished with the addition of high Ca to the high-fat diet. This finding was accompanied by an increase in the faecal excretion of SFA and bile acids in high-Ca groups. The authors acknowledge the limitations of the study, in that the sample size was small and the dietary interventions were of short duration, and the need for longer-term studies to confirm their findings. Nevertheless, the outcome, in terms of the pattern and magnitude of lipid responses and increased excretion of SFA and bile acids, was consistent with their original hypothesis, and a role for Ca in attenuating the effects of dairy fat on LDL-cholesterol through the binding of SFA and bile acids. The sequestration of bile acids is of particular note, in that this represents the mode of action of the first generation of cholesterol-lowering drugs based on anion exchange resins.

The results from this study build on a previous trial by the same authors in which they intervened with a mixture of dairy products⁽⁸⁾, and a meta-analysis of the effects of Ca from dairy and supplements on faecal fat excretion⁽⁹⁾. This earlier work provided evidence for a greater impact of cheese and yogurt on the excretion of faecal fat, and ascribed this to the significantly higher content and greater bioavailability and interaction of Ca in these foods with fat in the gut.

While it is important to interpret the favourable findings from this and other studies in relation to the absolute risk associated with serum LDL-cholesterol, the potential benefits of certain dairy foods on blood pressure and HDL are encouraging, and may have wider implications for the modification of cardiometabolic risk found in obesity, the metabolic

syndrome and diabetes^(6,10). In future, it would seem prudent to proceed with caution in restricting dairy foods simply on the grounds that they increase serum LDL-cholesterol, at least until we have more information on how dairy's garden grows!

Conflicts of interest

B. A. G. has acted as a scientific consultant for the British Dairy Council, The FAT PANEL, HEART UK, and as a contractor for the UK Food Standards Agency.

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