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Reply: The A2 milk case: a critical review

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The three paragraphs under 'Epidemiology' in KB Woodford's long letter discuss which of several between-country studies are likely to be more reliable. He favours those published by originators of the A1/A2 hypothesis. I favour others that have included more countries (including the Netherlands and Ireland) and FAO food consumption data. As I wrote 'There is more uncertainty with national figures for A1 beta casein than for total milk casein and more uncertainties for these than for average milk consumption.' For infants consumption (considered with type 1 diabetes) there are the special problems that infant formulas usually contain increased whey and reduced casein and the milk protein used for their manufacture does not always come from within the country where the formulas are consumed.

Correlation studies of national food intake against chronic diseases have been notoriously unreliable in nutrition research (and I gave some classic examples).

Answering the next paragraph Milk chemistry & pharmacology, there are, as I wrote, reports that BCM-7 can be released from cows milk A1 beta casein by *IN VITRO* digestion with three enzymes. But, I have not yet seen clear evidence that this peptide is released and active in humans *in vivo*. See for example Svedberg *et al*. (1985) found peptides in the human small intestine after 11 of bovine milk that reacted immunologically as if beta-casomorphin 7 but it did not show opioid activity or behave chromatographically as authentic beta-casomorphin-7. Effects in animals, injecting

pure BCM-7 does not establish that this would happen when humans drink milk.

Answering the next paragraph Animal studies, first, the single, rabbit experiment was not a realistic model for human atherosclerosis, as I wrote. 'The experiment was of very short duration, diet groups were very small, the diets were very far from a rabbit's natural diet, early fatty streaks are different from human atherosclerosis and the lesions were not read blind as to the diet group.'

Then with the rats (BB) and mice (NOD) genetically liable to develop diabetes, any reader of the literature must surely take the findings of experienced researchers in Ottawa, London (England) and Auckland as the latest (perhaps the final) word on the subject. In Ottawa and London there was no significant difference in diabetes between the A1 and A2 milk groups.

If milk containing A1 beta casein has adverse effects on coronary heart disease, since milks in most developed countries contain substantial amounts of the beta-casein variant, it would be expected that people who drink more milk would be more likely to experience coronary heart disease. Elwood's collection of published prospective studies (including their own) shows that this has not happened.

AJ Allison and AJ Clarke's letter from A2 corporation makes many of the same points as Woodford. They also discuss hypothetical links between A1 consumption and autism and schizophrenia. I have seen the paper they quote of a trial of (combined) casein- and gluten-free diet in autistic children. The result did not seem to be clear cut and this is not, of course, direct evidence about A2 beta-casein. My review was not about autism or schizophrenia. I think the evidence relating either of these to bovine A1 or A2 beta-casein is even more unsubstantial than that for type 1 diabetes or coronary heart disease.

Professor Swinburn's review (referred to in Woodford's letter) was completed after I submitted my review to the EJCN. It was less complete than mine and has fewer references. Nevertheless Swinburn's bottom line, as I read it, is that there is insufficient overall evidence that either A1 or A2 milk has benefits over the other. Food Standards Australia and New Zealand on their website state that they do not believe the available information warrants any amendment to their Food Standards Code.

As this reply was originally drafted a paper has been published by Chin-Dusting *et al*. (2006), from the Baker Institute, Melbourne reporting double blind crossover human experiments with 25 g/day A1 beta casein compared with the same dose of A2 beta casein for 12 weeks each way. A large number of biochemical measurement and endothelial function tests and large artery property measurements were made in 24 subjects. None showed any significant difference between the two beta caseins and the authors conclude that there is no evidence from this large study that supplementation with casein A1 has any cardiovascular health disadvantage over consumption of casein A2.

Another piece of research by Venn *et al*. (2006) in New Zealand finds no difference in plasma cholesterols of 55 people between periods taking ordinary New Zealand milk and A2 milk.

In yet another recent paper Muntoni and Muntoni (2006) review changes in type 1 diabetes from 1961 to 2000 in 37 different populations. While the supply of milk has, they estimate, remained almost unchanged there has been a large increase in this type of diabetes (+3% per annum). Ordinary milk in these 28 different countries contains A1-beta-casein.

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