



# Health

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[www.healthnz.co.nz/milk.pdf](http://www.healthnz.co.nz/milk.pdf)

## **BCM7 in the milk we drink**

Woodford, professor of farm management at Lincoln University, has reviewed a hundred scientific papers on the peptide betacasomorphin BCM7 in A1 milk, raising concerns about possible health effects. Research in 2003 by Laugesen and Elliott (see below) is featured, but recommendations by Professors Beaglehole, Jackson and Swinburn in 2003 for more research have yet to be implemented. The issue is not about whether people should drink milk but about whether people should be able to buy A1-free milk. A2 milk sales have increased in the North Island, but South Islanders cannot yet buy A2 milk at a reasonable price.

*Devil in the Milk Illness, health and politics A1 and A2 milk.* Professor Keith Woodford, Craig Potton Publishing ISBN 978-1-877333-70-5 – Oct. 07)

### **Data on BCM7 in New Zealand dairy products**

Neither Fonterra nor A2 Corporation has published data on the BCM7 content of their milk products. The NZ Food Safety Authority repeats that milk is safe, but offers no test results to show that infant formula, for example, is free of BCM7.

Woodford's book offers more than enough evidence to apply the precautionary principle, and he urges dairy farmers, at no extra cost, to use pure A2 semen from now on. For whatever one's position on the science, once dairy farmers decide to inseminate their cows with pure A2 semen rather than with A1, the A1 content of New Zealand milk will decline to near zero (Guernsey Island levels) within 10 years. If Fonterra offered a slight premium at the farm gate for A2 milk, this goal would be reached much sooner.

### **Milk – is it safe?**

The NZ Food Safety Authority's re-iteration that milk is safe, is a generalization. Great care is taken to pasteurize it and keep it safe from communicable disease. With respect to non-communicable disease, milk, like most food additives and flavours, is Generally Regarded As Safe (GRAS). It is not, however, completely safe. For example, milk including A2 milk, can cause serious milk allergy.<sup>1</sup>

1. Smith WB, Thompson D, Kummerow M et al. Letter to MJA 2004 181 (10) 574.)

## **Research to reduce heart disease and diabetes**

Laugesen and Elliott's 2003 research paper confirms a high degree of correlation between A1 beta casein and heart disease and diabetes, at population level. This has raised the possibility that the type of casein in the fresh milk

supply could be a risk factor. But proof of this concept is elusive. As Beaglehole and Jackson said in the accompanying NZMJ editorial, further research is recommended.

A1 but not A2 milk breaks down to form the peptide casomorphin-7. Much more needs to be known as to the final fate in the body of this peptide, known to be bioactive.

Health New Zealand's research paper:  
[www.nzma.org.nz/journal/116-1168/](http://www.nzma.org.nz/journal/116-1168/)

NZ MJ editorial

[www.nzma.org.nz/journal/116-1168/](http://www.nzma.org.nz/journal/116-1168/)

Fonterra's comment:

[www.nzma.org.nz/journal/116-1169/](http://www.nzma.org.nz/journal/116-1169/)

The authors' reply:

[www.nzma.org.nz/journal/116-1170/](http://www.nzma.org.nz/journal/116-1170/)



Heart disease and diabetes type 1 are commoner in North Europe, and one possible explanation may lie in the genetics of the cow and type of milk consumed. A1 milk differs very slightly from A2 milk in the composition of one of its main proteins, beta casein. A1 is a genetic variant of A2 milk.

A1 milk was commoner from black and white (Holstein-Friesian herds) or red and white herds, as found in Northern Europe, and A2 more in brown herds as in Southern Europe and the Channel Island breeds. These associations with skin colour have become blurred in recent decades by widespread artificial insemination from Holstein bulls. The proportion of A1 milk in the town supply still varies considerably across countries and somewhat over the years.

### Further thoughts on A1 and A2 milk

Laugesen and Elliott found that while differences in A1 milk consumption can explain differences in heart disease and diabetes type 1 between countries, they do not explain why diabetes type 1 is increasing in almost all countries.

Casomorphin 7 in A1 milk if glycosylated can be absorbed orally and have adverse immune effects. Recent data support the hypothesis that non-enzymatic pathways (glycation and oxidation) are involved in the pathogenesis of tissue damage in diabetes mellitus. Infant formula is high in advanced glycation end products (AGEs), which can cause diabetes in mice. A diet low in AGEs is protective in mice.

See Elliott RB. Diabetes – a man-made disease. *Med Hypotheses*. 2006;67(2):388-91. Epub 2006 Mar 10.

[http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list\\_uids=16530335&query\\_hl=1&itool=pubmed\\_docsum](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16530335&query_hl=1&itool=pubmed_docsum)

Health New Zealand Ltd is interested in sponsors for further research on this topic.

### A1 and A2 milk – what is the difference?

Health New Zealand on its own initiative, carried out research on the relation of A1 milk to heart disease, and type 1 diabetes, to prove or disprove the existence of correlations noted by Professor Bob Elliott and the late Dr Corran McLaughlin, later founder of A2 Corporation [www.A2corporation.com](http://www.A2corporation.com) which promotes A2 milk. If this correlation was true, it had importance for investors, for New Zealand, for public health and disease prevention. If not, then the sooner it was disproved the better. Fonterra's Research Institute put its library at our disposal. Preliminary work showed the presence of strong correlations. The work was then completed with the assistance of A2 Corporation. The country-level correlations are not proof of concept, for which individual-level data were needed. This we acknowledge in our paper.

Publication of the Laugesen and Elliott paper in January 2003 resulted in several papers by Fonterra staff (Hill, Crawford) and nutritionists Mann (Otago) and Trusswell (Sydney), all critical of the A1/A2 concept. Cardiovascular epidemiologists, Beaglehole and Jackson, who wrote the editorial accompanying the paper, and cardiovascular nutritionist Swinburn who reviewed research to date on the issue, took the view that although correlations have their pitfalls, this correlation was of potentially great importance and deserving of further (commercially-funded) research to prove or disprove it.

When the paper below was published in 2003, A2 milk was not available in New Zealand, and even today it is only available in certain supermarkets. To a limited extent then, this research has secured greater consumer choice. As most of the top bulls are now pure A2, (whether by accident or design), the A2 content of the town milk supply will gradually increase.

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NZ Med J 24 January 2003; vol 116, no. 1168. Full text at [www.nzma.org.nz/journal](http://www.nzma.org.nz/journal) under Archived

## Ischaemic heart disease, Type 1 diabetes, and cow milk A1 $\beta$ -casein

Murray Laugesen and Robert Elliott

### Abstract

#### Aim

To test the correlation of per capita A1  $\beta$ -casein (A1/capita) and milk protein with: 1) ischaemic heart disease (IHD) mortality; 2) Type 1 (insulin-dependent) diabetes mellitus (DM-1) incidence.

#### Methods

A1/capita was estimated as the product of per capita cow milk and cream supply and its A1  $\beta$ -casein content (A1/ $\beta$ ) (calculated from herd tests and breed distribution, or from tests of commercial milk), then tested for correlation with: 1) IHD five years later in 1980, 1985, 1990 and 1995, in 20 countries which spent at least US \$1000 (purchasing power parities) per capita in 1995 on healthcare; 2) DM-1 at age 0–14 years in 1990–4 (51 were surveyed by WHO DiaMond Project; 19 had A1 data). For comparison, we also correlated 77 food, and 110 nutritive supply FAO (Food and Agriculture Organization)-based measures, against IHD and DM-1.

#### Results

For IHD, cow milk proteins (A1/capita,  $r = 0.76$ ,  $p < 0.001$ ; A1/capita including cheese,  $r = 0.66$ ; milk protein  $r = 0.60$ ,  $p = 0.005$ ) had stronger positive correlations with IHD five years later, than fat supply variables, such as the atherogenic index ( $r = 0.50$ ), and myristic, the 14-carbon saturated fat ( $r = 0.48$ ,  $p < 0.05$ ). The Hegsted scores for estimating serum cholesterol ( $r = 0.42$ ); saturated fat ( $r = 0.37$ ); and total dairy fat ( $r = 0.31$ ) were not significant for IHD in 1995. Across the 20 countries, a 1% change in A1/capita in 1990 was associated with a 0.57% change in IHD in 1995.

A1/capita correlations were stronger for male than female mortality. On multiple regression of A1/capita and other food supply variables in 1990, only A1/capita was significantly correlated with IHD in 1995.

DM-1 was correlated with supply of: A1/capita in milk and cream ( $r = 0.92$ ,  $p < 0.00001$ ); milk and cream protein excluding cheese ( $r = 0.68$ ,  $p < 0.0001$ ); and with A1/ $\beta$  in milk and cream ( $r = 0.47$ ,  $p < 0.05$ ). Correlations were not significant for A2, B or C variants of milk  $\beta$ -casein. DM-1 incidence at 0–4, 5–9 and 10–14 years was equally correlated ( $r = 0.80$ ,  $0.81$ ,  $0.81$  respectively) with milk protein supply. A 1% change in A1/capita was associated with a 1.3% change in DM-1 in the same direction.

#### Conclusions

Cow A1  $\beta$ -casein per capita supply in milk and cream (A1/capita) was significantly and positively correlated with IHD in 20 affluent countries five years later over a 20-year period – providing an alternative hypothesis to explain the high IHD mortality rates in northern compared to southern Europe.

For DM-1, this study confirms Elliott's 1999 correlation on 10 countries for A1/capita,<sup>1</sup> but not for B  $\beta$ -casein/capita. Surveys of A1  $\beta$ -casein consumption in two-year-old Nordic children, and some casein animal feeding experiments, confirm the A1/capita and milk protein/capita correlations. They raise the possibility that intensive dairy cattle breeding may have emphasised a genetic variant in milk with adverse effects in humans. Further animal research and clinical trials would be needed to compare disease risks of A1-free versus 'ordinary' milk.