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## THE GAMMA SERIES

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### Adversity's Sweet Milk

***It would seem that all milk is not created equal, and that's even before humans start "adding value" by reducing fat content or adding vitamins, minerals or flavourings. A genetic variation among cattle could be affecting the milk we drink and causing health problems for a few people with particular types of health predispositions.***

In September 2007, Professor Keith Woodford from Lincoln University released his book, "*Devil in the milk: Illness, health and politics, A1 and A2 milk*", claiming that a particular type of milk protein is causing some people health problems and that the dairy industry is doing little to change the situation or to alert consumers of the potential problems. This issue is one that has existed in scientific circles for decades, but Professor Woodford's book has finally thrust the issue into the public arena. The subsequent media attention has many consumers concerned about the milk they drink. But what are the scientific facts surrounding Professor Woodford's claims.

There are two main types of dairy cows in this world, those which produce milk with the protein known as A1 beta-casein and those that produce milk with A2 beta-casein. The difference between these two proteins is small. In a sequence of 209 amino acids, only one of these amino acids is different, that at position 67 on the chain. In this position, A1 type milk has the amino acid histidene, while the A2 type milk has proline. The beta-casein protein only constitutes between 25 to 30% of the protein contained in milk, other proteins include whey and alpha and kappa-caseins. Milk itself is about 88% water and 12% solids. The solids include fat, protein, lactose and minerals.

Although seemingly small, this tiny difference in A1 milk is claimed to implicate the product with a range of health problems including Type 1 diabetes, heart disease, autism and schizophrenia.

However, it is important to stress, that for most people milk is a healthy and nutritious food important

for bone growth. In addition, the two milk types have been with us throughout human history, with the A1 type (called a variant) believed to be caused by a mutation some 5000 years ago in Europe. It is clear from our long history of milk drinking that most people are not adversely affected by either type of milk protein; however, for those few with a predisposition to certain conditions the news might be different. In addition, Type 1 diabetes (the insulin dependent type normally occurring in childhood) has been slowly increasing throughout the world with no clear reason as to the cause.

It is possible to identify which cows produce which milk with a DNA testing process (developed and patented in New Zealand), however, at present little testing has been done in New Zealand and only a few independent milk companies separate the different types of milk. The New Zealand developed test is being used in Australia and in the United States, to separate A2 herds for emerging A2 milk markets. It is possible to slowly change New Zealand's milk herds to the A2 type with selective breeding and artificial insemination. Although the New Zealand dairy industry (predominantly represented by Fonterra) claim that this is not necessary as they believe that the balance of the science does not yet present a plausible case for this being an issue. Professor Woodford claims that the industry has been quietly converting herds to A2 milk producers through artificial insemination. However, an industry spokesperson says that it is coincidental that 60% of bulls currently used for artificial insemination are A2 type.

The proportions of A1 and A2 type milks differ from country to country, with countries such as Iceland with almost entirely A2 type herds. The native cows of Africa and Asia are almost entirely A2 types. In New Zealand, we have an approximately 60% A1 type dairy cows, and there's no way for the consumer to tell the proportion of the two milk protein types in any given bottle of generic milk. Milk marketed by A2 Corporation is identifiable by label and price.

The two different genes are linked to specific breeds of cattle, however, both genes can be found in all breeds. The Guernsey breed of cattle has the highest frequency of the A2 gene, followed by Jersey cows. Holstein and Friesian breeds carry the A1 and A2 genes in roughly equal proportions. Some cows are a mix of both A1 and A2 and there are also a few minor subvariants of the A1 gene, usually called B, C and F.

This difference between milk types is undisputed in the scientific community. There is also scientific evidence from several sources that shows that these two proteins behave differently in our digestive systems. Laboratory research has shown that under conditions of in vitro digestion, A1 beta-casein breaks down to form the peptide beta-casomorphin-7 (BCM7). The same breakdown does not occur with A2 beta-casein.

After this the scientific waters become somewhat more muddled. A glance at the papers published in medical journals (available from the National Centre for Biotechnology Information (NCBI) at <http://www.ncbi.nlm.nih.gov>) reveals hundreds of papers and letters on the potential adverse affects of milk, with dozens of papers examining the differences in proteins – with a number pointing the finger at A1 beta-casein, especially in relation to type1 diabetes. However, in the process of healthy scientific debate, there are also several reviews which dispute the findings of other researchers or conclude there is not enough scientific evidence to conclude a link between A1 beta-casein and the various conditions.

A correlation between consumption of cows' milk protein and Type 1 diabetes was first proposed in 1984 by Professor Bob Elliott and others. The link between milk consumption and heart disease was proposed as early as the 1970s, although this early research failed to identify the vector involved.

In 1993, Professor Elliot at Auckland University's Child Health department, decided to investigate why

Samoan children living in Samoa did not often get Type 1 diabetes, while Samoan children living in New Zealand did. As the genetics between the two groups was essentially the same he based his research on the idea of a differing environmental or dietary factor causing the noted difference in disease between the two groups.

Again, Professor Elliot suspected dairy consumption might be a contributing factor, but that the actual component or components of dairy responsible might be harder to identify. He contacted the New Zealand Dairy Research Institute (now the Fonterra Research Centre) and was advised by Dr Jeremy Hill that the beta-casein proteins in milk might be worth investigating. The pair worked together on the research identified through inter-country comparisons that the incidence of type 1 diabetes was higher in countries or areas which consumed predominantly A1 milk, compared to places which consumed predominantly A2 milk. This was possible because there are many geographical areas which have predominantly either A1 or A2 herds.

The second phase of Professor Elliot's research involved observing what happened to two separate groups of diabetes-prone mice. One group was fed A1 milk and the other A2 milk. The mice that were fed A1 milk developed a high incidence of diabetes whereas the mice fed A2 milk did not develop the disease at all.

Dr Corran McLachlan reviewed the epidemiology of the research for the NZ Child Health Foundation. He was struck by the similarity between Type 1 diabetes incidence rates and the ischaemic heart disease (IHD) mortality data that he had encountered in his work on cholesterol-free foodstuffs. He not only agreed with the spatial correlation between A1 milk and Type 1 diabetes, but over the next 15 months collected herd data for A1 and A2 milk and, like Elliott, compared it with disease data, only this time for heart disease. Dr McLachlan was the inventor of a cholesterol free butter but had often openly stated that there was more to heart disease than the intake of cholesterol.

Although it is important to note that statistical correlation is not, in itself, proof of cause and effect, Dr McLachlan believed that the correlation between A1 beta-casein distribution and heart disease was too obvious to ignore and that A1 beta-casein, along with cholesterol, was at least part of a multifaceted equation which contributed to heart disease. His report showed that countries such as Iceland, where the milk is mainly A2 have much lower heart disease

rates than countries of similar ethnicity such as Finland which has a high intake of A1 milk.

Meanwhile, in unrelated research into autism and schizophrenia, Professor Robert Cade from the University of Florida published a paper in *Nutritional Neuroscience* in 2000 stating that schizophrenics have extremely high levels of BCM7 (up to 100 times normal) in their urine. BCM7 is the byproduct from the breakdown of the A1 beta-casein protein chain. Professor Cade noted that when his test subjects were placed on a gluten and casein free diet for several weeks that not only did the BCM7 disappear from the urine, but there is also a marked reduction in disease symptoms. If the diet was continued indefinitely, disease symptoms continued to improve for up to a 12 month period and then stabilized, by which time the obvious affects of the disease were often greatly reduced. Professor Cade and his team also experimented on rats by injecting BCM7. They observed that when BCM7 entered the brains of the rats it often caused symptoms that are similar to those of autism and schizophrenia.

If the BCM7 peptide is indeed the culprit in the equation, then theoretically, it should be possible for schizophrenic suffers to consume A2 milk without detrimental side affects.

In 2003, a specialist in vascular heart disease, Professor Julie Campbell from the University of Queensland, published her team's research on feeding A1 milk and A2 milk to rabbits in the international medical journal 'Atherosclerosis'. The paper concluded that the rabbits fed A1 milk had higher cholesterol levels than the rabbits fed A2 milk. They also observed that the A1 milk-fed rabbits developed lesions and thickening on the artery walls. Professor Campbell observed that the lesions in the arteries were similar to 'juvenile fatty streaks' in humans – a precursor of heart disease. Although most concerned scientists seemed to agree that Professor Campbell's work was well conducted, she was later criticized for extrapolating from rabbits to humans.

Meanwhile, the New Zealand Food Safety Authority asked former New Zealand Heart Foundation Medical Director, Professor Boyd Swinburn, to

undertake a literature review on research surrounding the A1/A2 debate. This report was finally released in August 2004 after being held for nearly a year for peer review. A copy of the report and a lay summary are available from the Food Safety Authority's website at <http://www.nzfsa.govt.nz/policy-law/projects/a1-a2-milk/a1-a2-report.pdf> and <http://www.nzfsa.govt.nz/policy-law/projects/a1-a2-milk/lay-summary.htm>. His report recommends that more research is needed. In his lay summary, Professor Swinburn concludes:

“The A1/A2 hypothesis is both intriguing and potentially very important for population health if it is proved correct. It should be taken seriously and further research is needed. In addition, the appropriate government agencies have a responsibility to communicate the current state of evidence to the public, including the uncertainty about the evidence. Further public health actions, such as changing dietary advice or requiring labelling of milk products, are not considered to be warranted at this stage. Monitoring is also required to ensure that any claims made for A2 milk fall within the regulations for food claims.”

“Changing the dairy herds to more A2 producing cows is an option for the dairy and associated industries and these decisions will undoubtedly be made on a commercial basis. Changing dairy herds to more A2 producing cows may significantly improve public health, if the A1/A2 hypothesis is proved correct, and it is highly unlikely to do harm.”

“As a matter of individual choice, people may wish to reduce or remove A1  $\beta$ -casein from their diet (or their children's diet) as a precautionary measure. This may be particularly relevant for those individuals who have or are at risk of the diseases mentioned (Type 1 diabetes, coronary heart disease, autism and schizophrenia). However, they should do so knowing that there is substantial uncertainty about the benefits of such an approach.”

However, there are few places to buy A2 milk in New Zealand and those that do sell it charge a premium price. A2 milk however, is widely available in Australian supermarkets (supplied by a New Zealand company – the A2 Corporation).

## References and further reading

(those marked with an '\*' are suitable for senior secondary school students, other texts are university level)

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