

A2 Milk, Farmer Decisions, and Risk Management

Keith Woodford
Professor of Farm Management and Agribusiness
Lincoln University
New Zealand
woodfork@lincoln.ac.nz

Published in the Proceedings of the 16th International Farm Management Association Congress, Peer Reviewed Papers Vol2, pp 641-648. University College, Cork, July 2007. (Eds S. O'Reilly, M. Keane, P. Enright. ISBN:978-92-990038-3-1)

Abstract

Approximately 500 New Zealand (NZ) dairy farmers are converting their herds to eliminate production of A1 beta-casein within the milk. The alternative casein is A2 beta-casein, and the associated milk is known as A2 milk. A2 milk can be considered the original milk before a mutation affected some antecedents of modern European breeds. A1 beta-casein and its derivative beta-casomorphin7 (BCM7) have been implicated in numerous health issues including Type 1 diabetes, heart disease and autism. There are now more than 100 relevant papers in peer reviewed journals. The broader NZ herd is also drifting away from A1 beta-casein production due to a serendipitous association between genetic merit as measured in NZ and A2 beta-casein. There is no evidence of this occurring in other countries. The farmer decisions can be structured using concepts of risk management and decision theory. However analysis is complicated by uncertainty as to future premiums/discounts associated with A2/A1 milk. Outside of NZ most farmers know nothing about the issue.

Introduction

Approximately 500 New Zealand dairy farmers are currently converting their herds so as to eliminate the production of A1 beta-casein. Milk that is free of the A1 beta-casein is called A2 milk.

Originally all cow milk was of the A2 type. However, a genetic mutation, probably between 5000 and 10,000 years ago, has resulted in a proportion of cows of European breeds producing a casein variant called A1 beta-casein. The proportion of A1 beta-casein is higher in the black and white breeds compared to

the yellow and brown breeds. A1 beta-casein is absent in the milk of pure Asian and African cattle (Ng-Kwai-Hang and Grosclaude 2002).

Farmers are converting to A2 milk production because of concern that A1 beta-casein may be associated with a range of human health issues. However, the decisions are complex because of uncertainties about future premiums for A2 milk (or discounts for A1 milk) combined with long lag times between breeding decisions and herd outcomes.

The Scientific Evidence

There are at least eight strands to the evidence, with more than 100 relevant papers in the peer reviewed medical and science literature.

The first strand is remarkable epidemiological evidence that countries with high intakes of A1 beta-casein are the same countries that have high levels of Type 1 diabetes (Fig. 1) and heart disease (Fig. 2) (Laugesen and Elliott 2003, McLachlan 2001). Type 1 diabetes is the form that typically develops in childhood and requires insulin injections. The statistical associations are extremely strong such that it is highly unlikely to be due to random factors. In terms of alternative possibilities that have been put forward there are none that explain the statistical association.

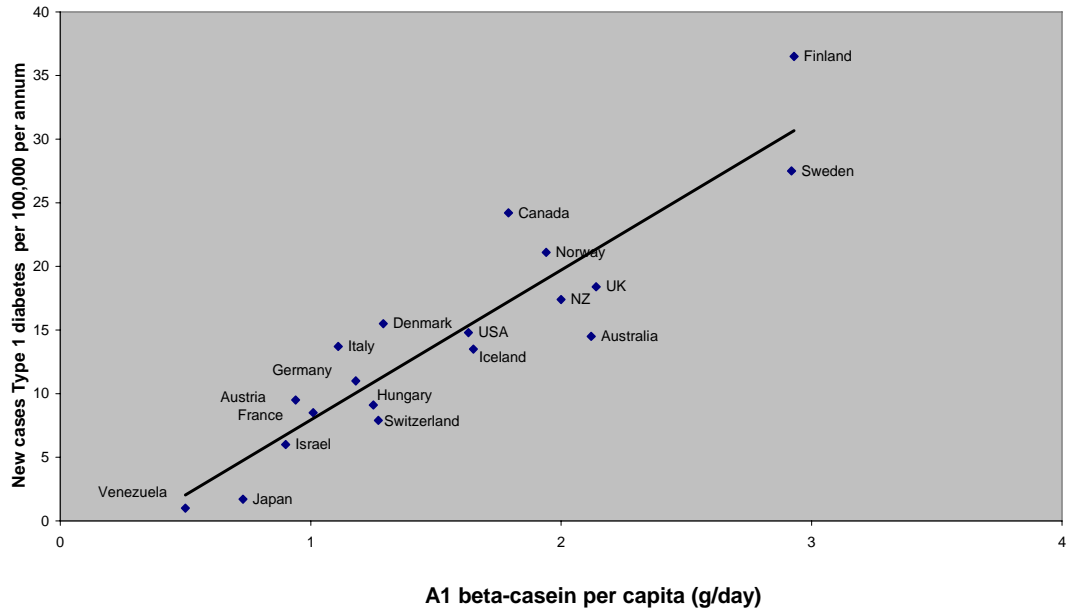
The second strand is the biochemical knowledge that A1 and A2 beta-casein digest differently. Empirical evidence from at least three laboratories confirms that, *in vitro* and in the presence of digestive enzymes, A1 beta-casein releases large amounts of beta-casomorphin7 (BCM7) whereas A2 milk does not (Hartwig *et al.* 1997, Jinsmaa and Yoshikawa 1999).

The third strand is that BCM7 is known with certainty to be a powerful opioid. This has been known for many years from laboratory work (Brantl and Teschemacher 1994). The effects have also been clearly demonstrated when BCM7 is injected into rats (Sun *et al* 1999). The effects can be counteracted by the use of naloxone which is an opioid antagonist.

The fourth strand is that the incidence of Type 1 diabetes is higher in some genotypes of rats and mice when they are fed A1 beta-casein than A2 beta-casein (Elliott *et al* 1997). However, in one trial this effect was masked through non-disclosed diet confounding (Beales *et al* 2002, Woodford 2006).

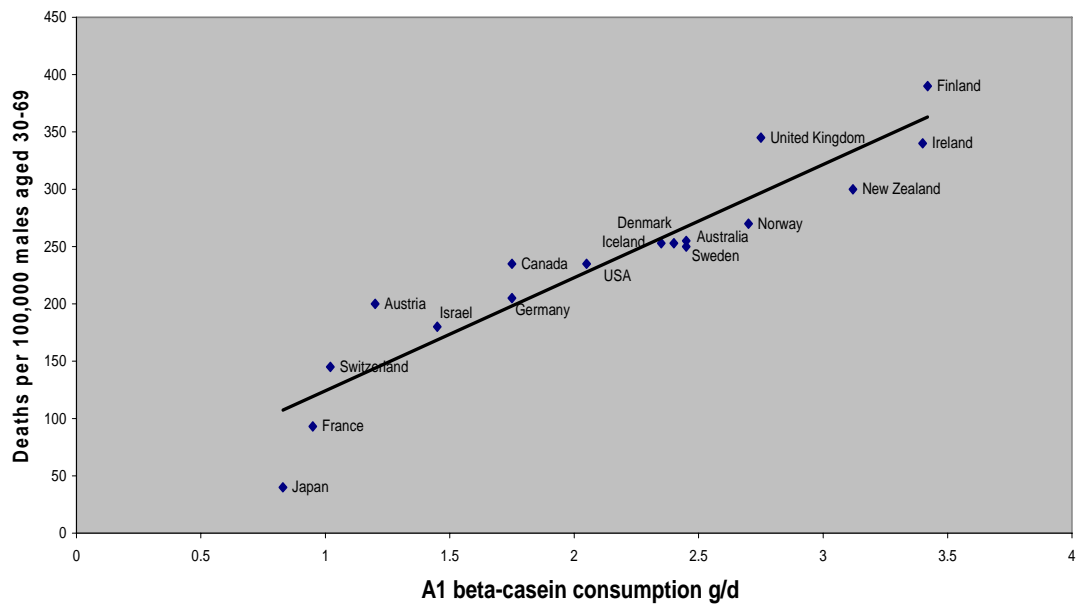
The fifth strand is that it has been demonstrated that rabbits fed A1 beta-casein develop considerably more plaque on their aorta than do similarly treated rabbits fed A2 beta-casein (Tailford *et al* 2003). This happened over a period of just a few weeks.

Figure 1. Incidence of Type 1 diabetes and A1 beta-casein intake



(Data from Laugesen and Elliott 2003)

Figure 2. A1 beta-casein consumption excluding cheese, and deaths from ischaemic heart disease



(Data from McLachlan 2001)

The sixth strand is that BCM7 is known to be an oxidant of LDL (Torreilles and Guerin 1995), and oxidation of LDL is believed to be important in formation of arterial plaque.

The seventh strand is a broad range of evidence from American and European investigations that autistic and schizophrenic persons typically excrete large quantities of BCM7 in their urine (Cade *et al* 2000, Knivsberg *et al* 2001). The only known source of this peptide is casein. When these people are placed on a milk and gluten free diet the excretion of the peptide declines to almost nothing, and there is typically a reduction in the autistic and schizophrenic symptoms.

The eighth strand is anecdotal and observational evidence from a considerable number of consumers that whereas they suffer intolerance to normal milk (such as bloating, diarrhoea, and nausea) they are able to tolerate A2 milk.

Issues surrounding so-called A1 and A2 milk remain controversial. The science is complex and it is apparent that only some people are susceptible to the effects of A1 beta-casein. It would seem that the majority of people excrete the BCM7 from their digestive system, and that a minority absorb the peptide through into the bloodstream. However, this minority is substantial. It seems likely that the issues are much more important in milk than in cheese. It is possible that heat treatment (including method of pasteurisation) may be relevant (McLachlan 2001).

There are complex commercial and intellectual property issues surrounding A2 milk. Both A2 Corporation and Fonterra (New Zealand's dominant milk processor and marketer) have property rights relating to patents for testing milk and genetic testing of cows. A2 Corporation also has trade marks relating to the words 'A2 milk'. Although these international trademarks would probably preclude other companies from marketing A2 milk using 'A2 milk' nomenclature, there are no restraints preventing anyone marketing and advertising this milk as 'free from A1 beta-casein'.

Currently, the mainstream dairy industry in New Zealand has chosen to argue against the so-called A2 hypothesis. With one notable exception (Truswell 2005), this has tended to be in industry journals rather than peer reviewed health journals. Outside of the NZ industry there remains a low level of awareness.

In 2003 the New Zealand Food Safety Authority commissioned an external review of the evidence relating to A1 beta casein and human health which was undertaken by Professor Boyd Swinburn from Deakin University, Australia (Swinburn 2004). This report focused primarily on human health trials (which are difficult to undertake and hence limited) and did not explore the underlying science. Nor did it identify most of the available evidence linking BCM7 to autism and schizophrenia. Despite these limitations, Professor Swinburn concluded in the Lay Summary of his report that:

“The A1/A2 hypothesis is both intriguing and potentially very important for public health if it is proved correct. It should be taken seriously...”

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.”

When the report was subsequently released by the NZFSA (August 2004), the Lay Summary which included the above statements was excised. However, the Lay Summary was subsequently obtained by this author under freedom of information legislation, and the NZFSA subsequently agreed following a question in Parliament to place it on their website together with the main technical report. Subsequently the Swinburn Report has been used by both supporters and detractors of the A2 hypothesis to buttress their arguments.

Some Basic Genetics

The A1/A2 status of a cow is determined by a pair of genes on the sixth chromosome. There are two major alleles (or variants) of the gene. These are called the A1 and A2 beta-casein alleles.

Because a cow carries two copies of the beta-casein gene, she can carry either two copies of the A2 allele, or one copy of each of the A1 and A2 alleles, or two copies of the A1 allele. The three states are referred to as being homozygous A2A2, heterozygous A1A2, or homozygous A1A1.

Neither allele is dominant over the other. Instead they are co-dominant, i.e. additive in their effect. Therefore an A1A2 cow will produce A1 and A2 beta-casein in equal amounts. An A2A2 cow will only produce A2 beta-casein and an A1A1 cow will only produce A1 beta-casein.

As a very rough generalisation, herds based on the Northern European black and white breeds such as the Friesian Holstein typically carry the A1 and A2 allele at about equal levels. The Southern European breeds and the Jersey are likely to carry the A1 allele at about 35%. There are plenty of exceptions. For example

the Guernsey breed appears to carry the A1 allele at less than 10% and the Scottish Ayrshire breed appears to be well over 50%. In addition, individual herds may carry the allele at levels that are quite different to the average for the breed.

If a cow is A2A2 then she is guaranteed to pass on the A2 allele to her progeny. Similarly, an A1 cow is guaranteed to pass on the A1 allele. For an A1A2 cow there is a 50% chance of passing on either allele.

Breeding for A2A2 cows is based on using semen for bulls that have been tested as being A2A2. The breeding process can be speeded up by selective culling of A1A1 and A1A2 cows and by selective retention of A2A2 calves.

The speed at which a herd will be converted to A2 milk production depends on whether the strategy relies solely on use of A2 semen (the passive approach) or a more active strategy that requires testing all cows together with selective culling and calf retention.

If farmers rely on the passive approach, and assuming that cows start milk production at two years of age, it will take 2.75 years subsequent to mating decisions before there is any impact in the milk vat. Thereafter, and assuming an initial A1:A2 ratio of 50:50, then the A2 proportion will increase each year by about 5 percentage units (e.g. to 55:45 once the first cohort of specially bred heifers enters the milking herd approximately three years after conception). However, the rate of improvement gradually slows down (the relationship is asymptotic) and a herd will never reach 100% A2 without testing of cows. For farmers who start with A2 semen and then complete the process by testing cows and selecting only A2A2 replacements, the total process is likely to take about two cow generations, i.e. about 10 years.

Farmer Decisions

The estimate of 500 farmers currently converting to A2 milk is based on year 2005 data from an unpublished Lincoln University survey of 2000 randomly selected farmers, supervised by this author, that showed 4% were using exclusively A2 semen, and then applying this figure to the national herd. In addition, one of the New Zealand animal breeding companies, which has a market share for semen approaching 30%, has estimated (also to this author) that about 300 (or 10%) of their clients use exclusively A2 semen. However, this company is probably not representative of the total market.

It is believed that almost all of the NZ farmers currently breeding for A2 have been taking a passive approach based on exclusive use of A2 semen but without testing the cows. This is a simple procedure given that both of the major animal breeding companies in New Zealand routinely test the A1/A2 status of their bulls and this is recorded in the catalogues.

In addition, most other New Zealand dairy herds are currently drifting towards higher A2 status because of an apparently chance association between genetic merit and A2 status in the results of the national sire testing programme. Quite simply, the most efficient producers of milk protein (the key selection criteria in New Zealand) tend to be A2A2 (Morris *et al* 2005). Consequently, the A2 status is much higher in the semen (about 70%) than it is in the national cow herd (probably about 50% A2 allele based on an assumption of about 25% of cows being A2A2 with another 50% A1A2, and 25% A1A1).

Given that outside of NZ there is no routine testing of bulls for A1/A2 status, it is unknown whether or not there is a similar drift factor in national herd status as is occurring in New Zealand. Given that the New Zealand national sire testing system uses different selection criteria (very high weighting on efficiency of protein production with discounts for high liquid volume and large animal size) than are used in most other countries, there is probably no justification for assuming that a similar drift factor might be occurring in these other countries.

Most consumers world-wide, and also most dairy farmers world-wide, remain unaware of the issues surrounding so-called A1 and A2 milk. This is because consumer laws prevent A2 Corporation and its franchisees from making negative health statements about A1 milk. In contrast, within New Zealand, almost all dairy farmers would be aware of the so-called A1/A2 issue, although few would be aware of the detailed evidence.

Currently, the major consumer market for A2 milk is in Australia where the product is available in some 800 supermarkets and 200 convenience stores. However, overall market share is probably less than 1% because of limited publicity. A2 Corporation has also announced its intention to enter the American market in 2007 (A2 Corporation 2006). This will initially be in just a few states. Within New Zealand A2, milk is difficult to procure outside the upper half of the North Island.

Apart from a very small number of farmers who are supplying pure A2 milk to specialist A2 marketers in Australia, the USA and New Zealand, there are currently no premiums or discounts at the farm gate associated with A1/A2 content.

The problem facing farmers can be characterised as having two alternative decisions and two possible states of nature, giving four market outcomes (Table 1). A more complex representation would allow for a range of premiums, each with an associated probability. The matrix can also be cast in terms of discounts for A1 rather than premiums for A2. In theory, if we can quantify the probabilities for the outcomes then it is possible to calculate which strategy has the greatest expected payoff and which strategy minimises risk. These two criteria (payoff and risk) may lead to different preferred strategies.

Table 1 *Decisions and outcomes matrix*

	Outcomes	
Farmer Decision	Premium for A2 milk (discount for A1 milk) (probability =x)	No premium for A2 milk (no discount for A1 milk) (probability =1-x)
Convert to A2	Box 1: Farmer receives A2 premium (alternatively avoids A1 discount) but has cost of conversion.	Box 3: Farmer receives no A2 premium (alternatively receives no A1 discount) but has cost of conversion.
Don't convert to A2	Box 2: Farmer does not receive A2 premium (alternatively does receive A1 discount) and has no conversion costs.	Box 4: Farmer receives no A2 premium (alternatively receives no A1 discount) and has no conversion costs.

In practice, it is unlikely that many farmers construct an outcomes matrix, but it is essentially this process that they go through in an intuitive way. Discussions with a number of farmers who have started the conversion process indicate that they have typically identified that ending up in Box 2 could threaten their business through discounts for A1 milk, whereas the loss associated with Box 3 is minor, particularly if they implement a passive approach based solely on semen selection. This places them in a strong position to rapidly complete the process by shifting to an active strategy, that includes testing all animals with associated selection and culling, if a premium for A2 (discount for A1) does eventuate. In essence, the adopting farmers perceive moving to A2 as a potential business opportunity, but staying with A1 as a potential threat. In contrast, farmers who choose not to convert are placing a low probability on there being a discount associated with A1 milk. The extent to which this is currently an informed or uninformed decision as to either the threat or the opportunity can be debated.

Conclusion

The issue of A2 milk is a real life decision issue of relevance to all dairy farmers throughout the world who farm European breeds of cattle. The problem can be structured using concepts of strategic management, risk management and decision theory. The key difficulty arises in placing probabilities on the alternative market outcomes (i.e. milk and milk product prices). A lack of awareness of the issues means that most dairy farmers, apart from in New Zealand, have yet to factor the A1/A2 issue into their decision making. Within New Zealand, a serendipitous association between genetic merit and A2 status, together with a higher level of existing knowledge about the issue, gives the New Zealand industry a potential early mover advantage relative to other countries with high A1 beta-casein status in avoiding the threat and turning it into an opportunity.

References

- A2 Corporation 2006. 2006 Annual Report. Available at www.a2corporation.com
- Beales PE, Elliott RB, Flohé S, Hill JP, Kolb H, Pozzilli P, Wang GS, Wasmuth H, Scott FW. 2002. A multi-centre, blinded international trial of the effect of A1 and A2 beta casein variants on diabetes incidence in two rodent models of spontaneous Type 1 diabetes. *Diabetologia* 45:1240-1246.
- Brantl V, Teschemacher L. 1994. Beta casomorphins and related peptides. VCH Weinheim.
- Cade R, Privette M, Fregly M, Rowland N, Sun Z, Zele V, Wagemaker H, Edelstein C. 2000. Autism and schizophrenia: intestinal disorders. *Nutritional Neuroscience* 3:57-72.
- Elliott R, Wasmuth H, Bibby N, Hill J. 1997. The role of beta-casein variants in the induction of insulin-dependent diabetes in the non-obese diabetic mouse and humans. In: *Milk Protein Polymorphism*. Brussels: International Dairy Federation, Special Issue 9702:445-453.
- Hartwig A, Teschemacher H, Lehmann W, Gaulty M, Erhardt G. 1997. Influence of genetic polymorphisms in bovine milk on the occurrence of bioactive peptides. In: *Milk Protein Polymorphism*, International Dairy Federation Special Publication 9702., Brussels, Belgium, pp459-460.
- Jinsmaa Y, Yoshikawa M. 1999. Enzymatic release of neocasomorphin and beta-casomorphin from bovine beta-casein. *Peptides* 20(8):957-962.
- Knivsberg A-M, Reichelt KL, Høien T, Nodland M. 2002. A randomized, controlled study of dietary intervention in autistic syndromes. *Nutritional Neuroscience* 5:251-261.
- Laugesen M, Elliott R. 2003. Ischaemic heart disease, Type 1 diabetes, and cow milk A1 beta-casein. *New Zealand Medical Journal* 116(1168).
- McLachlan CNS. 2001. Beta-casein A1, ischaemic heart disease mortality, and other illnesses. *Medical Hypotheses* 56:262-72.
- Morris CA, Hickey SM, Cullen NG, Prosser CG, Anderson RM, Tate ML. 2005. Associations between beta-casein genotype and milk yield and composition in grazing dairy cows. *New Zealand Journal of Agricultural Research* 48:441-450.

Ng-Kwai-Hang KF, Grosclaude F 2002. Genetic polymorphism of milk proteins. In Fox PF and McSweeney PLH (eds), *Advanced Dairy Chemistry*, Chapter 16, pp737-814, Kluwer Academic/Plenum Publishers, New York.

Swinburn B. 2004 Beta casein A1 and A2 in milk and human health. Report to New Zealand Food Safety Authority. Available at www.nzfsa.govt.nz

Sun Z, Cade JR, Fregly MJ, Privette RM. 1999. Beta casein induces Fos-like immunoreactivity in discrete brain regions relevant to schizophrenia and autism. *Autism* 3(1):67-83

Sun Z, Cade RJ. 1999. A peptide found in schizophrenia and autism causes behavioural changes in rats. *Autism* 3(1)85-95.

Tailford, Kristy A, Berry, Celia L, Thomas, Anita C and Campbell, Julie H. 2003. A casein variant in cow's milk is atherogenic. *Atherosclerosis* 170:13-19.

Torreilles J, Guerin MC. 1995. Casein-derived peptides promote peroxidase-dependent oxidation of human blood low-density lipoproteins. *C Seances Soc Biol Fil* 189:933-942. [In French].

Truswell AS. 2005. The A2 milk case: a critical review. *European Journal of Clinical Nutrition* 59:623-631.

Woodford KB. 2006. A critique of Truswell's A2 milk review. *European Journal of Clinical Nutrition*. 60(3):437-439.