

Updating 'Devil in the Milk'

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Since 'Devil in the Milk' was first published in September 2007, the story has moved on considerably. The purpose of this update is to summarise the main events, recognising that it will continue to be an ongoing story, and only time will allow some events to be seen in their appropriate context. Essentially, there are three parts to the ongoing story. The first is about the politics of milk and health, how information is communicated, and market responses. The second is about what is happening 'behind the scenes' to Australian and New Zealand dairy herds. The third is about the new science. All are important to an overall understanding.

This update is meant as an accompaniment to the NZ and Australian edition of the book. It is not a stand alone document. Some components of this update were incorporated in the American edition of 'Devil in the Milk'.

Health, Politics, and Commerce

Publication of 'Devil in the Milk' was certainly controversial. When the New Zealand and Australian edition of the book came out in September 2007 (the American edition from Chelsea Green was published in 2009) there was an immediate media reaction. I found myself doing more than 40 radio and television interviews within the first week in both New Zealand and Australia. However, the response varied in each country.

In Australia, I was interviewed by Helen Wellings on Channel Seven's program 'Today Tonight'. This program was particularly important in bringing the 'A2 issue' to the attention of a major segment of the population. It was only a brief segment of about six minutes, and so it could not explore many of the issues, but it was seen by millions. I also gave a six-minute commentary for ABC Radio National in Australia, which was played at prime time in the evening and again in the morning. There was no interviewer; the producer simply gave me six minutes in which to say what I wanted. According to an A2 Corporation information release to the Stock Exchange, the impact of my book and the publicity that it generated was closely linked to more than doubling of the sales of A2 milk across Australia within a very short time. Sales have continued to grow, and have more than doubled again. By mid 2009, A2 milk was available in almost all Coles and Woolworths supermarkets, plus many independents, right across Australia. Also, as of April 2010, A2 yoghurt became available under the Jalna brand. However, A2 milk in Australia remains a niche product, albeit now profitable, selling at a significant premium over other branded milks.

The initial response at the time of publication from Fonterra, headquartered in New Zealand, was to say nothing. This was exactly what I expected. Three weeks before publication, the Fonterra CEO had asked me in a telephone conversation to delay the release of the book for six months so they could undertake an internal review, but by that late stage I was neither able nor willing to accommodate them. (The reason they knew the book was coming was because I had told them.) So the internal advice of their public relations people was to say nothing and hope the controversy would blow over.

In contrast, the New Zealand Food Safety Authority (NZFSA) came out with all guns blazing. They claimed that there was nothing new in the book, but then had to admit that they had not had time to read it. On NZ National television (Channel 1), NZFSA spokesperson Carole Inkster and I

were interviewed together in a live interview, Inkster from Wellington and me from Christchurch. Inkster claimed that if there had been anything new since the Swinburn review, Professor Swinburn would have advised them. That was easy to refute. I said that I had rung Professor Swinburn in Australia some three days earlier, and he had confirmed to me that he had not been working in this field for three years. Inkster also repeated the line that the Swinburn review had found that all milk was safe. I pointed out that nowhere in his report had Professor Swinburn said that all milk was safe.

At that time the television producer was unable to make contact with Professor Swinburn, who was by then on a working trip to Samoa, but Radio New Zealand National did manage to interview him from Samoa two days later. Professor Swinburn confirmed that he had never used those words, and also that he was very frustrated with the way that NZFSA had managed the release of his report. He made it clear that there were important health issues involved. He also defended my own integrity, which was nice to hear.

The NZFSA was unable to argue against the substance of what I wrote, but they were embarrassed by what I had exposed. So instead they attacked me personally (my qualifications to write on such matters), the format of the book (paperback), and also my publisher (non-scientific). Subsequently, the Minister of Food Safety at that time, Lianne Dalziel, apologised to me in writing for the manner of the attack by the NZFSA bureaucrats. She also repeated that apology when we met more recently at a social event.

There was also an attack in the media from a group of scientists from the University of Otago, led by Professor Jim Mann. Professor Mann is mentioned in several places in 'Devil in the Milk' and at various times he was an adviser to Fonterra. Professor Mann did his credibility little good by criticising my book but then admitting that he too had not read it and was too busy. "I haven't read his book and I'm not going to. I have better things to do with my life. I have got too much to do".¹ However, he had found the time to check my publication record in relation to medical science, which he had found wanting.

In Chapters 3, 6, 11, and 13 of this book I make extensive mention of Professor Stewart Truswell from Sydney University. Professor Truswell confirmed in writing in the *New Zealand Dairy Exporter* (December 2007) that he had been a paid consultant of Fonterra in relation to A1 and A2 beta-casein. There is, of course, nothing wrong with being a consultant for Fonterra. However, some of us would have liked this disclosure made a lot earlier.

Another scientist from Otago who criticised the book in the general media (print and radio) was Dr Tony Merriman. Dr Merriman is a researcher investigating the genetic aspects of Type 1 diabetes. His criticism related specifically to the epidemiological link between Type 1 diabetes and intake of A1 beta-casein. He put forward the alternative hypothesis that the between-country differences in Type 1 diabetes can be explained by latitudinal effects influencing exposure to UV light and subsequent impact on Vitamin D synthesis. It is indeed true that there is a cross-correlation between latitude and intake of A1 beta-casein, and this was discussed in Chapter 5. This is because many of the countries with high intake of A1 beta-casein are also high-latitude countries. But there are plenty of exceptions. And the Laugesen and Elliott evidence shows that the explanatory power of latitude in relation to Type 1 diabetes is only half that of A1 beta-casein.² In the case of sunlight there was no meaningful relationship at all (M. Laugesen, personal communication). So it is possible that the modest latitude correlation is being dragged along by its association with A1 beta-casein, but the evidence does not support the converse notion that the strong A1 beta-casein relationship can be explained by the modest latitudinal relationship.

What I do accept is that Vitamin D may well be part of the overall story on Type 1 diabetes. I made very clear in the concluding paragraphs of Chapter 7 that causation of Type 1 diabetes is almost certainly multi-factorial. It is only when a number of factors line up together that the disease manifests itself. There is evidence that Type 1 diabetes typically reaches the clinical stage

during winter, when UV radiation is lowest (although the development of the disease occurs over a much longer period), and there is also emerging evidence that those with Type 1 diabetes may have lower circulating Vitamin D levels (although there are very interesting issues about what may be causing the low Vitamin D levels). Indeed, low blood levels of Vitamin D may be associated with a broad range of health conditions including parathyroid disease, kidney disease, intestinal cancers and prostate cancers. Personally, I find the evidence for this to be strong. But what I do say very strongly is that neither latitude nor sunlight exposure can be used to explain away the relationship that exists between Type 1 diabetes incidence and intake of A1 beta-casein.

Some weeks following publication of my book, I released further information obtained under the Official Information Act as to how NZFSA dealt with the Swinburn review.³ It had taken me some time to obtain this information (NZFSA had delayed releasing it to me), and so it was not in the book. Despite the omission of key information in the released documentation that was 'whited out' on the grounds of confidentiality, the material was sufficiently embarrassing that NZFSA had to do something. So they announced that they would be calling in an external consultant to review NZFSA risk-management procedures, including specific consideration as to how these procedures were applied to the issue of A1 and A2 milk. Amazingly, the NZFSA CEO, Andrew McKenzie, said on Radio New Zealand National that the aim was 'to bury the issue once and for all' and that the key issue was to demonstrate the integrity of the NZFSA. Mmmm!

The review was undertaken by Dr Stuart Slorach from Sweden. Although external to the NZFSA, his investigations could hardly be called independent. He visited New Zealand and made a hurried visit to me in Christchurch (he could spare less than one hour) and also to Auckland. In Christchurch he was accompanied by the Chief Scientist for NZFSA and in Auckland by the CEO Andrew McKenzie. When his report came out in May 2008, it suggested many ways in which NZFSA could be improved. But the release of the report and the associated media conference were carefully stage-managed by NZFSA. The key statements in regard to A1 beta-casein were deeply buried on page 41 of the report.

"The assertion that *'there is no safety issue with either type of milk'* can be interpreted in different ways. If it is interpreted, as some do, as meaning that there is no scientific debate about possible negative health effects of A1 milk, it is not correct and is also contradicted by the quotes from Swinburn's report given lower down in the same media release. According to NZFSA, the phrase *'there is no safety issue with either type of milk'* was intended to provide the public with assurance that their choice to use either (A1 or A2) milk product was not going to result in the safety issues that are otherwise associated with unsafe food, such as sickness or hospitalisation."

What a remarkable statement! Suddenly NZFSA were telling Dr Slorach that they were never referring to the negative health effects discussed in my book, but to other issues associated with unsafe food! But of course this contradicts much of what they had been saying in public.

Ironically, over the next two years, if any readers were to seek out information on this issue on the NZFSA Web site (www.nzfsa.govt.nz), they would easily find information critical of me in NZFSA's own press releases. They would easily get to this information very quickly from the home page by clicking on 'A1 and A2 milk'. But they would have struggled to find Dr Slorach's report except by scrolling down and eventually finding it at the bottom via a 'related link'. Originally, Dr Slorach's report could not be found there at all, but following my remonstrations to the Food Safety Minister Lianne Dalziel, NZFSA did make it available, even if deeply buried. The message I took from this was that bureaucrats have many ways to defend their public reputations.

More recently, the NZFSA website has been re-organised, but a site search using my name will quickly lead to critical material about me, and a search on 'Slorach' will lead to the Slorach Report.

Since the publication of my book I have spoken to many medical and scientific groups both in New Zealand and Australia. One of the most interesting requests was to present the closing plenary paper to the International Diabetes Federation (IDF) Western Pacific Congress.⁴ This was set up as a forum, with Professors Boyd Swinburn and Bob Elliott as commentators on my paper. Both Swinburn and Elliott were strongly supportive, but the co-chair, Professor Len Harrison from Australia, gave a summing up that was much more cautious, perhaps even negative. The original plan had been to have commentators who would give both positive and negative commentary, but those asked to speak in the negative had pulled out. So it was a bit disappointing when the co-chair took on that role. Professor Harrison had taken a similar stance when interviewed for the Four Corners 'White Mischief' program on Australian TV some years previously.⁵

Although some aspects of the IDF Diabetes presentation were frustrating, there was some good that came out of it. The informal discussions that were held led to Professor Swinburn, whom NZFSA had falsely claimed as supporting their 'all milk is safe' stance, now taking a stronger public position. He sent an open letter to the New Zealand media, and addressed to all farmers, stating that the time to shift their herds to A2 was 'right now'. He clarified his position by saying that, although in his opinion there was still no final proof, the potential benefits to public health were sufficiently strong, and the costs so small, that it should be done.

In some ways Professor Swinburn's position was not all that different to back in 2004 when, in private correspondence with NZFSA (which I obtained through the Official Information Act) and in remonstrating with them as to how they were handling his report, he had said:

. . . if I had a child with Type 1 diabetes and was due to have another and I could easily obtain and afford A2 milk or formula, I would certainly use it for the next child because the cost/benefit is low because of the potentially very large benefit of preventing Type 1 diabetes.

Some people have asked me why Professor Swinburn did not go public a lot earlier. My response is that he was in a difficult position. He had undertaken the study for NZFSA under contract, and therefore they owned the report. He chose initially to remonstrate with them in private rather than in public. I admire him greatly for subsequently going public.

Given my professional position within agribusiness, I regularly come into contact with various Fonterra directors and senior management. However, I have been unable to convince them to engage with the issue. They continue to take the advice of Fonterra's Chief Scientist Jeremy Hill, who features so prominently within my book, that the issue has no substance. It seems to me that none of the directors or top level management are willing to engage on the issue because they lack confidence in their own ability to read and understand the science. So, they simply rely on Fonterra's scientific leader. That in itself is a fundamental flaw within Fonterra's governance and management.

In terms of industry politics and the 'PR game', the publication in January 2009 of a European Food Safety (EFSA) Scientific Report titled 'Review of the potential health impact on β -casomorphins and related peptides' was a major win for those opposing the A2 issue. The background to this report was that NZFSA, under instructions from the NZ Government, and in addition to commissioning Dr Slorach to look at their procedures, had asked EFSA to review the substantive scientific issues around the A1/A2 milk debate. This led to EFSA deciding to undertake its own review, which was completed on 29 January 2009.⁶ They concluded:

"Based on the present review of available scientific literature, a cause-effect relationship between the oral intake of BCM7 or related peptides and aetiology or course of any suggested non-communicable diseases cannot be established."

At a political level, this outcome was a major blow to the A2 cause. It has been used to good effect by those who want to bury the issue (i.e. the mainstream dairy industry). But the finding was an inevitable outcome of how EFSA defined the evidential requirement. To determine cause-

effect using their criteria, there would have needed to be human clinical trials which showed a clear quantitative relationship between the intake of BCM7 and the risk for individuals. That information does not exist. And if it did exist it would almost certainly vary for different individuals, given the apparent link with 'leaky gut'. Of course there is strong evidence at the population level, but EFSA chose for its own reasons to not place weight on that evidence.

On reading the EFSA report I was puzzled by the negativity. In particular, I found myself reading in the report the same arguments made by the mainstream dairy industry that I have already presented in 'Devil in the Milk'. I found that they were questioning rabbits as a suitable model (I discussed that in Chapter 13). I also found they were using the FAD trial (discussed in detail in Chapter 6) without any acknowledgement of the contamination issue. But clearly at least some people in EFSA should have known of the issue, given that Food Safety Minister Lianne Dalziel told me that she received a 'yes' to her explicit question as to whether they had my book and were aware of the arguments therein. They were also using the strongly flawed Caerphilly cohort data (discussed in Chapter 3) as contradictory information to the A2 hypothesis. They essentially ignored the Laugesen and Elliot epidemiology on the grounds that such studies prove nothing. They used the elementary argument that ecological (i.e. between-country) studies are liable to find false associations on account of lifestyle factors. But they ignored the fact that the A1/A2 epidemiology is restricted to developed country comparisons and hence to developed country lifestyles. They also ignored the painstaking but unsuccessful search by Laugesen and Elliott for alternative factors that could have been confounding. It seemed to me that EFSA knew the answers they wanted from the outset.

So I set to work to find out a little more about the eight authors. On searching databases I found that five could be classed as dairy scientists with strengths in biochemistry. Another two were trained in veterinary faculties and now specialise in toxicity and pharmacology. The remaining one is a human nutrition professor from Iceland (Professor Thorsdottir) who is listed in 'Devil in the Milk' as a co-author of papers suggesting that A1 beta casein is indeed a risk factor in Type 1 diabetes. Where were the human health experts in heart disease, diabetes and autism? Where were the experts on food intolerances and leaky gut? Where were the medical experts in population health studies and epidemiology? I was no longer quite so puzzled as to the content and tone of the report.

Indeed the EFSA outcome was exactly what some people had been warning me. If the EFSA report had found against A1 beta-casein, even as something that was uncertain, then the worldwide implications for the milk industry could have been both enormous and unfortunate. Parts of the media would inevitably have interpreted it as a finding against all milk rather than a component that can be easily bred out of our dairy herds. And a positive finding would have led to a formal investigation to determine maximum safe intakes. So perhaps the outcome was predestined.

In the days following the release of the report, the media sought Professor Boyd Swinburn's latest opinion. He stated that in his view A2 remained the safe option, and that none of the science was refuted. He also said that the evidential barrier was very high and the terms of reference narrow. Given those narrow terms of reference, he said that the conclusions might be defensible but not helpful. That also summed up my perspective.

Nevertheless, the EFSA report did provide a very valuable crutch for the dairy industry in both New Zealand and Australia. I know of no-one apart from one scientist who has actually read the full report, but in terms of letting industry off the hook, at least in the short term, it has been very effective. Both Dairy Australia and Fonterra have used it to good effect as a publicity weapon. Dairy Australia have even gone further by making claims such as "There is no good scientific evidence that A2 milk is any different to A1 milk".⁷

In April 2010 there was further publicity in Australia as a result of a program on the ABC 7.30 report for which I was interviewed. I considered the program to be disappointing. Important and impressive interview material by Professor Boyd Swinburn was cut at the last moment, although the extended version with Swinburn's comments included is still currently available on the ABC website.⁸ Also, a number of incorrect statements from the opposing side went unchallenged. The mainstream industry tried to portray the 'A2 issue' as a marketing gimmick that lacked scientific evidence. Nevertheless, the publicity did lead to a further boost to A2 milk sales across Australia.

Outside of Australia, progress has been slower. In New Zealand, A2 milk is available through many Countdown, Progressive and Fresh Choice supermarkets. But unlike Australia, there is almost no publicity. In the United States, A2 milk was available for a period in Hy-Vee supermarkets in seven Midwest States. However, in December 2008 the A2 Milk Company announced they were withdrawing product from sale pending a re-branding and re-launch on a broader scale across the US. To date that has not occurred. The evidence is clear that it is hard to market a product where the issues are complex, where there are constraints on the promotion of that message, and where consumers remain ignorant or confused about the issues.

Converting the National Herds

There is a lot of ongoing confusion as to exactly what is meant by 'A2 milk'. In terms of the commercial product, it is the product labelled and trademarked as 'a2 Milk™'. This milk comes from herds which are comprised totally of cows that produce only A2 beta-casein and not A1 beta-casein. However, all other Australian and New Zealand herds contain some cows that produce A2 beta-casein ('A2 cows'), plus other cows that produce a mix of A1 and A2 beta-casein, plus other cows that produce only the A1 variant of beta-casein. From a public health perspective, progress can be made by increasing the number of A2 cows in the national herds, and this can be done, over time, simply by using A2 bulls.

To some extent this is occurring, particularly in New Zealand. In part this is because some farmers are purposely using only A2 semen. (There has been sufficient publicity that almost every dairy farmer in New Zealand must be aware of the issue.) It is also because a particularly high proportion of the top New Zealand bulls are homozygous A2 (i.e. carry two copies of the A2 variant of the beta-casein gene). Given that almost all of the bulls in New Zealand are genetically tested for A2 status, this information is publicly available. However, I have more work to do, using data from the breeding companies as to the number of artificial inseminations from each bull, to document exactly what is happening. At this stage my best estimate is that the proportion of the beta-casein in New Zealand milk that is A2 has been increasing for about eight years at 1.5% to 2% per annum. Whereas in the late 1990s it would have been about 50% A1 and 50% A2, I believe it is now closer to 35% A1 and 65% A2. I am confident that the A2 level is going to continue to rise at between 2% and 3% each year for at least the next two years, given the lag between time of mating and subsequent arrival of the progeny in the milking herd. So it may not be too long before New Zealand is up to the levels that are found in some of the southern European countries.

In Australia there is probably a similar but smaller drift occurring. However, many farmers in Australia are less aware of the issue than New Zealand farmers. They have been fed information by Dairy Australia that it is a non-issue. But perusal of the bull semen catalogues indicates a similar preponderance of A2 bulls and so some serendipity will be occurring.

Many people continue to tell me that this unannounced drift to A2 in New Zealand, and perhaps the smaller drift in Australia, has to be a conspiracy, but I can see no evidence for that. It is genuine serendipity caused by A2 bulls ranking highly for productive traits. But there is a huge irony. Fonterra continues to tell the world dairy community that A1 beta-casein is a non-issue, and I keep hearing this from dairy companies overseas as far afield as Mexico to Sweden. So no countries apart from New Zealand, and to a lesser extent Australia, are doing anything about it.

New Science

First of all, I need to explain what I now regard as an omission from 'Devil in the Milk'. I should have written about the enzyme dipeptidyl peptidase 4, commonly abbreviated to DPP4. It is this enzyme which breaks down BCM7 in the digestive system. However, this enzyme is only found attached to epithelial cells in the lining of the stomach and intestines. On the one hand this can be used to explain why, for people with properly functioning digestive systems, the BCM7 should not get through into the blood. But it also provides an explanation why, for those who have an impaired digestive system with damaged epithelial cells (a 'leaky gut'), the BCM7 can sneak through. That potentially includes all those people with undiagnosed or untreated stomach ulcers, ulcerative colitis, Crohns, and untreated coeliacs. It can also include those who have been on antibiotics, or subject to various stress factors. So this would seem to be one more piece of the jigsaw.

Much of the information about DPP4 has been known for quite some time. However, increased understanding is now developing about how BCM7 crosses what is known as the caco-2 monolayer (between the digestive system and the circulatory system) in the presence of DPP4. This work is coming out of Poland, with Malgorzata Iwan the lead author of a 2008 paper in the journal *Peptides*. Indeed the group to which Iwan belongs has been particularly active in investigating the biochemistry of both human and bovine BCM7 and also linking it to allergies. Their work can be found readily by searching the Pubmed public access electronic database (www.ncbi.nlm.nih.gov/sites/entrez) and searching on the word 'casomorphin'.

The most exciting recent work has come from a Russian group of 12 scientists from four leading research institutions, and funded by the Russian Foundation for Basic Research. (In scientific circles, 'basic' means 'fundamental' or 'non commercial'). It is published in the international journal *Peptides*, with Natalya Kost as the lead author.⁹ The Russians have been steadily building up knowledge on BCM7 for quite some years, but it is only now that they have started publishing in English.

Natalya Kost and colleagues have made three complementary breakthroughs. First, they have successfully developed tests for measuring BCM7 in the blood. This might seem very simple, but blood is a complex substance, and until now it has only been possible to measure BCM7 in urine and digestive fluids. Being able to test for BCM7 in the blood will be a huge step forward for subsequent research. Second, the Russians have shown that babies fed formula milk do indeed absorb BCM7 into their blood. This absorption is exactly what would be expected on theoretical grounds, given the permeability of babies' digestive systems, but it is a huge step to go from theory to empirical evidence.

However, the Russians have gone much further than that. They have shown that some of the babies can eliminate the BCM7 rapidly from their systems (either through metabolising or excreting it), but that other babies retain it in the bloodstream. Then comes the final blow. **Those babies who are unable to rapidly breakdown and excrete the BCM7 from their systems are at very high risk of delayed psychomotor development.** The Russians found in their study that 30% of the babies fed formula had developmental delay whereas only 3% of breast fed babies were in that category.

There is a lot more of importance in the Russian paper. For example, they have shown that the human form of BCM7 (which is actually considerably different in its biochemical structure to the bovine form found in A1 milk), and which is found only in breast milk, is actually a good casomorphin, that enhances psychomotor development and works best in those children who don't break it down quickly. It is only the bovine form, released in large quantities from A1 cows, that is the 'devil'.

When history looks back on the saga of A1 beta-casein and the 'milk devil' I think the verdict will be that this Kost *et al* paper is the most significant breakthrough for at least five years. It is not only the results themselves, but that the Russians have given major new insights which others can now follow up. For example, the insight that it is not just a case of whether the BCM7 is absorbed, but also whether or not the individual has the ability to rapidly metabolise and excrete the little devil, will open the door to new research pathways.

Of course much of the mainstream industry is attempting to downplay this research. Dairy Australia, when it became aware of this research, was quick to emphasise the earlier and greatly flawed EFSA report which attempted to hose down the A1 milk issue. The New Zealand Food Safety Authority said that the Russian research was just one paper and that the results needed confirmation. Others have tried to downplay it because it has come from Russia. But the bell is now tolling. This is not just a report by some mad scientist with a 'bee in his bonnet'. It is a peer reviewed paper in an international journal by 12 scientists from four leading research institutions, with funding from the Russian Foundation for Basic Research. All praise to the Russians.

Another stream of new research has emerged from the Czech Republic. For more than 10 years, Alexandra Steinerova and colleagues have been untangling the causes of oxidative stress in infants. It is a fascinating story of how one investigation leads to another; of how BCM7 and A1 beta-casein were subsequently implicated; and then how causation has been subsequently demonstrated.

The research is important because oxidised LDL (caused by oxidative stress) and antibodies thereto, are key risk factors and indicators of heart disease. They are also important indicators of Alzheimer's disease. Most people associate heart disease with easily measured cholesterol. But amongst researchers, oxidised LDL is much more important because it is this substance that makes arteries sticky and leads to formation of plaque.

The initial work of Steinerova and colleagues showed that, during the first few months of age, some babies have increasing antibodies to oxidised LDL, whereas others have declining values relative to levels at birth. In their first paper, in 1999, Steinerova and colleagues were unable to explain the findings.

Then in 2001, they reported that further work had shown that it was the babies on milk formula who had the increasing levels, and the breast-fed babies were the ones with declining levels. In fact, by three months of age the formula fed babies had almost 50 times the antibodies to oxidised LDL of the breast-fed babies. They offered no hypothesis as to which component(s) within milk formula might be causing this.

In 2004, Steinerova and her colleagues reported in the journal *Atherosclerosis* that they were by then hypothesising that it was caused by BCM7 from A1 beta-casein. I mentioned this in 'Devil in the Milk', but at that stage it seemed to me that it was still just a hypothesis, although supported by earlier work by French scientists Torreilles and Guerin who had shown in the test tube that BCM7 does indeed oxidise LDL. Accordingly, at that time I wrote: "this is an evolving story, with quite a lot known, but still much more to be discovered".

Steinerova and colleagues published more evidence in 2006, but this was in a Czech medical journal (*CS.Pediatric*)¹⁰ and was not widely available to English-speaking people. It was only when Steinerova and colleagues presented a paper in the United States at the XV International Symposium on Atherosclerosis in 2009 that the strength of their evidence became apparent. They now have new data not only confirming her original results (although this time the difference in oxidised LDL in formula-fed infants was only 18 times that in the breast-fed babies), but also now showing that the formula-fed babies had very high antibodies to BCM7 and A1 beta-casein, whereas breast-fed did not. Equally important, the antibodies to A2 beta-casein were much lower.

Arguably, there was still a weakness in the argument. These results were based on what is called epidemiology. This epidemiology clearly showed there was an association between A1 beta-casein and BCM7 on the one hand, and high levels of oxidative stress. And the statistical analysis showed that this was highly unlikely to be due to chance. But for those people who want proof rather than just exceptionally strong evidence, there was still no clinical trial. Where was the direct trial comparing A1 and A2 beta-casein?

In fact, Steinerova and colleagues do have an answer to that. In their 2006 paper in the journal *CS.Pediatrics* they reported that piglets fed A1 beta-casein had much higher and statistically significant levels of antibodies to oxidised LDL than did those fed A2 beta-casein. **So yes, they have established what is known as 'cause and effect'.**

So, are there any remaining weaknesses in this line of work? Those who wish to deny the links between BCM7 and health (and there are plenty of those people associated with the international milk industry) will make three claims. The first is that the trial was with pigs and not with humans. That is true. However, taking the combination of the human epidemiology and the animal results points only in one direction. The second criticism is that Steinerova has not published her recent work in top quality western journals, but instead she has published in a peer reviewed Czech language medical journal. Yes, that too is true. And until she does publish all of this work in English it will not receive the full attention that it deserves. The third criticism will probably be that the pigs when slaughtered at six months of age did not have visible heart disease. That too is true; but that is not surprising, for heart disease takes a long time to build up.

The bottom line is that Steinerova's work provides chilling support to the prior body of heart disease evidence, undertaken by researchers such as McLachlan, Laugesen and Elliott, Briggs, and Campbell, which I reported in 'Devil in the Milk'. It also dovetails nicely with the new research from Natalya Kost and colleagues showing that children fed formula not only absorb BCM7, but those who cannot then rapidly break it down or excrete it are likely to suffer developmental delay.

In the long run, Steinerova's work may prove to be much more important for adults than babies. After all, babies do not get heart disease, and it may well be that they outgrow the oxidative stress (although that is currently unknown, and it may well be a precursor of adult heart disease). However, if BCM7 causes oxidative stress and oxidised LDL in formula-fed babies (which Steinerova has clearly shown it does), then logic says it must also cause oxidative LDL in those adults who absorb BCM7 into the blood stream, i.e. those who have a leaky gut. So, here we now have a solid mechanism capable of explaining the link between BCM7 and heart disease. It ties in perfectly with the epidemiology, the rabbit work, and the evidence of Briggs about heart disease in ulcer sufferers on high milk diets.

Another field of research on which more data is emerging is the effect of casein-free gluten-free diets in relation to autism. In the past this work has been held back by the difficulty of implementing long term blind trials. In April 2010 Paul Whitely and colleagues have published data in the journal *Nutritional Neuroscience*, from what they call the ScanBrit study, of children on this diet for up to 2 years, where the investigators taking the measurements were blinded as to which children were on the diet and which were controls. (Not surprisingly, the parents could not be blinded.)¹¹ The evidence from the first year met predetermined statistical differences in outcomes such that the children on the control diet were, on ethical grounds, also offered the treatment in the second year. The evidence shows that the diet has benefits for some but not all children. And the trial design could not determine separate effects from gluten and casein. In my judgement, this data is generally supportive of the case against A1 beta-casein, but it is not by itself going to be a 'debate changer' in the way that the Russian and Czech work has potential to be.

Another area where information continues to build up is in observational evidence, which some people dismissingly call 'anecdotal'. At one of my Australian talks to medical groups, Dr Merv Garrett, a specialist in food allergies and intolerances from the Gold Coast stood up and said that he had successfully treated about 20 people with food-intolerance problems by shifting them to A2 milk. He said he had a colleague in New South Wales who had successfully treated even more people than he had, but also had a few failures. Well, none of us have ever suggested that A1 milk is the only food that causes food-intolerance problems. In the last three years I have been approached by many people who say they can drink A2 milk after a lifetime of problems with ordinary milk. I have also had people tell me that they no longer have mucus problems that they had previously associated with milk. This is consistent with the link between casomorphins and mucins (the proteins in mucus) discussed in Chapter 9. It is difficult to be precise with numbers, but the evidence I see points to well over half the people who have previously been unable to tolerate 'ordinary' milk, because of conditions such as nausea, bloating and eczema, now finding that they are able to digest A2 milk. This is consistent with the notion that many people have been assumed to be lactose intolerant whereas it is really A1 beta-casein to which they are intolerant.

A Final Comment

If there is one thing I have had reinforced since writing 'Devil in the Milk', it is that the path of knowledge, and how that knowledge is communicated, is long and tortuous. Information, misinformation, and vested interests get inextricably intertwined. Intellectual property rights to patents and trademarks, and how these might be interpreted in different jurisdictions, adds a further complication. In health and medical matters, truth will always win out in the long run, whatever that truth may be, but the journey can be very long. The beta-casein journey is far from over.

I plan to keep writing about further developments relating to A1 beta casein, A2 milk, and BCM7 at <http://keithwoodford.wordpress.com>

Keith Woodford confirms that he receives no financial benefits from consulting in relation to beta-casein or A2 milk, nor does he have a financial interest in any A2 products.

*Keith Woodford
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¹ The Press. October 11 2007. www.stuff.co.nz/4233104a11.html

² See Laugesen and Elliott (2003a) in Diabetes section of Bibliography

³ See Woodford, K (2007). 'The role of the NZFSA in investigating health issues concerning A1 and A2 milk'. Available at www.lincoln.ac.nz/story1057.html

⁴ The paper is available at www.lincoln.ac.nz/diabetes or from my own internet site at <http://keithwoodford.wordpress.com>

⁵ Australian Broadcasting Corporation. 2003. *White Mischief*. Available at www.abc.net.au/4corners/content/2003/transcripts/s820943.htm

⁶ Scientific Report of EFSA prepared by a DATEX Working group on the potential health impact of B-casomorphins and related peptides. EFSA Scientific Report (2009) 231,1-107. Available at http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_home.htm

⁷ See http://www.weeklytimesnow.com.au/article/2010/04/27/179131_dairy.htm

⁸ Go to http://www.abc.net.au/reslib/201004/r544499_3184147.aspx

⁹ Kost NV, *et al*. 2009. B-casomorphins-7 in infants on different types of feeding and different levels of psychomotor development. *Peptides* 2009 Oct; 30(10):1854-60.)

¹⁰ Steinerova, A, *et al* 2006. Does artificial suckling nutrition pose a risk of atherosclerosis at adult age? *Cesko Slovenska Pediatria* 61(9): 519-523)

¹¹ Whitely, P *et al* 2010. The ScanBrit randomised, controlled, single-blind study of a gluten- and casein-free dietary intervention for children with autism spectrum disorders. *Nutritional Neuroscience* 13(2):87-100.