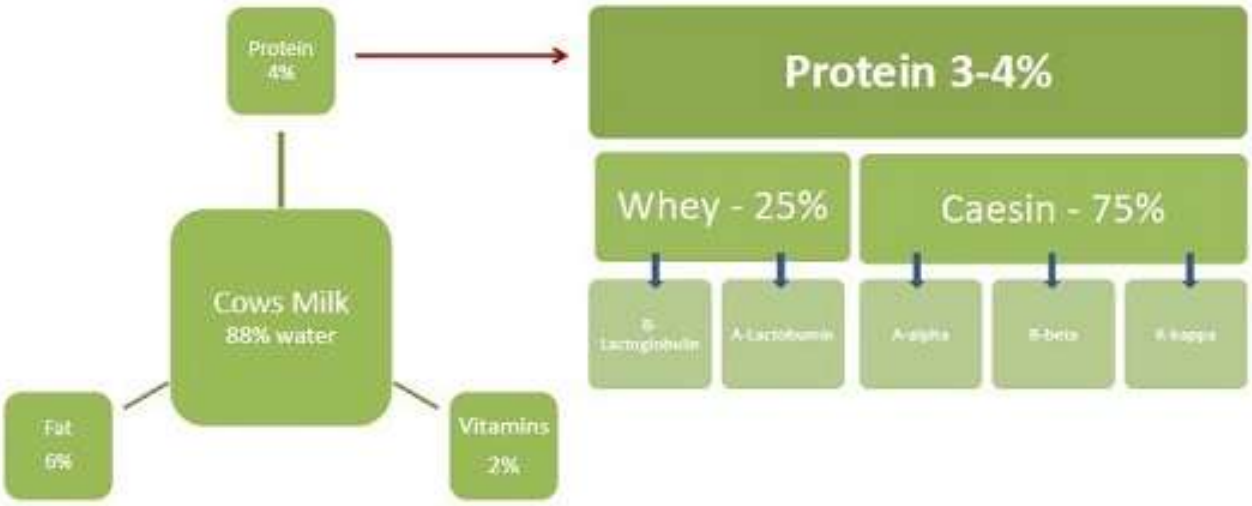
Milk- This traditional dietary staple is now a problem for many. What is science saying about it; a review by Martin Murray.

'Drink your milk, it's good for you' was a phrase we heard frequently as children. As CAM Providers over the last few decades, we have been finding that ingestion of milk or dairy products is troublesome to many and the majority of people put it down to lactose intolerance. Many scientists have examined milk and its impact on health or ill-health, depending on how you want to view it. In this review I am endeavouring to record some of the more important aspects of the scientific evidence that has been compiled, which indeed will show that milk can be a problem. But there is also a positive side which could restore milk to its preeminent position in the hearts of people as an important element in their diet.

MILK - What's in it?

Let's look at what cow's milk is composed of. The biggest component, 88%, is water and the remainder are milk solids made up of fat, protein, lactose (milk sugar) and other minerals. Protein accounts for approximately 3-4% of which 75% is casein and 25% whey. This varies from breed to breed. Depending on the type of milk, the fat content which is so important can vary from 3% to 6%. Milk contains all 8 essential amino acids required by humans. Milk also contains water soluble B vitamins and vitamin C, together with vitamins A, D, E and K in small amounts.[1]



The whey protein consists of B-lactoglobulin and A-lactobumin and some other less prevalent molecules like blood serum, albumin, lactoferrin, transferrin and other minor proteins and enzymes. The casein family consists of a number of types of casein - A-alpha, B-Beta and K-kappa. The phosphate content which is high, associates with calcium and this combination makes milk a good source of calcium for humans. The beta casein proteins found in milk are made up of a long string comprised of 209 amino acids. The caseins in milk reside in the milk-solid portion of the cow's milk

and is not present in the fat portion which means it will not be present in butter.[2] That is good news. There is debate amongst experts as to whether the nutrients in milk are lost during pasteurisation but that is not the subject of this review.

Different milks:

Scientific reports show that milk is increasingly becoming a problem for many people and contributing to such health conditions as heart disease, diabetes (Type 1), schizophrenia, autism, digestive disorders and other problems. The molecule in milk giving rise to those health issues has been identified as a protein in casein, **Beta-casomorphine-7 (BCM-7).**[3] This protein was identified by scientists at position 67 in the long string of 209 amino acids already mentioned. At position 67, histidine occurs in milk from certain breeds whereas proline occurs in that position from other breeds. As a result of that discovery, milk is categorised as either A1 (with histidine at position 67) or A2 (with proline at position 67). Proline, like other amino acids, cluster and bind together whereas the histidine did not cluster but fell apart into 7 separate molecules. Hence the name BCM-7. It had been established by German scientists that BCM-7 is an opioid, which means a narcotic. (Brantl and Techemacher 1979). Casein is used in the production of a variety of foods such as baked goods, infant formula, sports drinks, pastas and soups, so all of these are potential carriers of the opioid peptide.

A1 and A2 beta-casein protein varies from one herd of cows to another and from one country to another. Scientists believe that originally all cows will have been A2. As civilisation moved North and West from Asia to Europe, a mutation occurred whereby position 67 changed from proline to histidine. This may have occurred as far back as 5,000 years ago or maybe earlier. The A1 version of the gene is found in cattle mostly in the Western world, which belong to the sub-species *Bos Taurus*, Asian cattle of the sub-species *Bos Indigus* do not produce the A1 version of the gene. Today Asian cattle largely remain A2 but some cross-breeding with cattle from Europe has occurred.

The breed of cow is an important determining factor and essentially the black and white herds across Northern Europe could be described as producers of A1 beta-casein and Mediterranean herds are typically A2 milk producers.[4] A report by EFSA (European Food Safety Authority) lists many of the best known breeds differentiating between those that are carriers of the A1-A2 allele (gene variation). Holstein Friesian, which are predominantly found in Northern Europe, could be described as higher level carriers of the A1 allele. Jersey and Guernsey cows on the other hand are predominantly carriers of the A2 gene and French herds are predominantly A2. A sample of Guernsey cows in the USA recorded a 98% A2 allele. (Aschaffenburg 1963). Large batch sampling of Simmental was conducted in Denmark and Croatia where results showed A2 allele at 67% and 62% respectively. (Baryani et al 1993) and (Curik et al 1997).

Of local interest, we had mixed results for Kerry cows whereby one sample did not differentiate between A1/A2 (Murphy and Downy 1969). Another sample indicated the A2 gene was present in 76% of Kerry cows tested.[4] (O'Hara 1995) Sampling of Friesian cows in Ireland indicated a high level of the A1 gene present at 72% (O' Hara 1995).

The EFSA report indicated that cross-country breeding has been on-going and is extensive as many farmers sought to increase farm productivity and milk output. The Holstein Friesian breed was the important breed of choice for expansion because of their of milk output levels. One exception on inter-country swapping of breeds was Iceland which banned the import of animals for breeding and remained with their traditional Icelandic (Norse) cow. The Norse cow scored highly with 67% carriers of the A2 allele (Lien et al 1999).

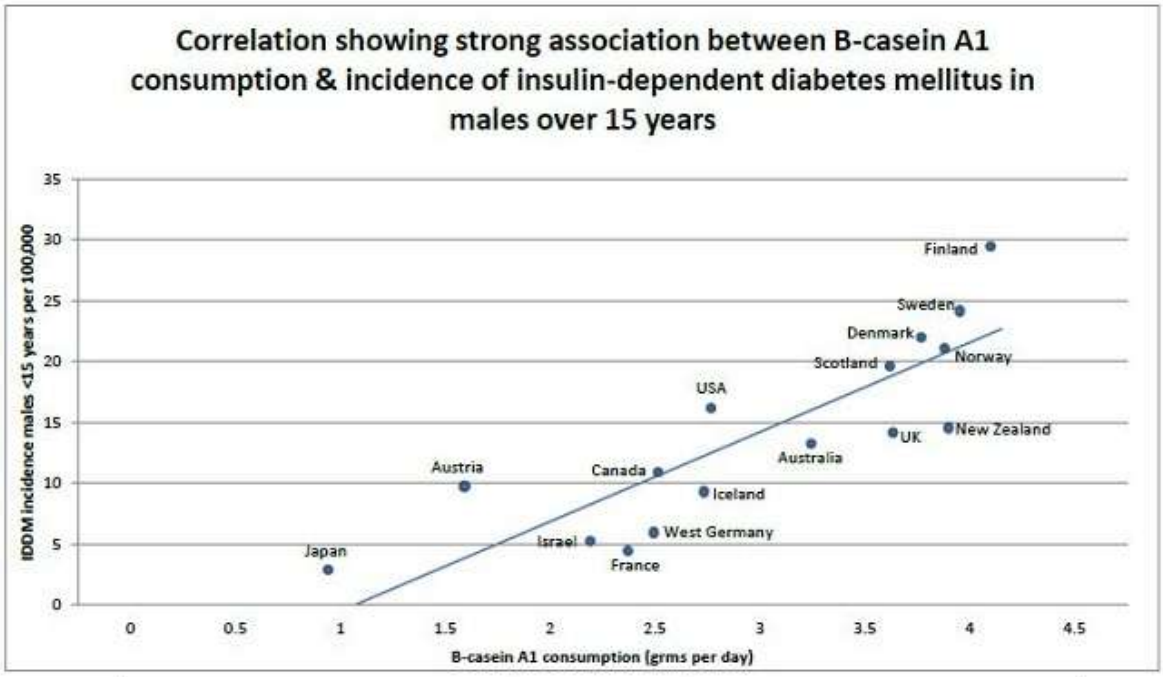
Other milk-producing animals - Goats, Sheep, (majority) Buffalo and Yak all carry the A2 allele. Papers published to date indicate that human milk does not contain B-casein A1.[4] (Dev et al 1994, Steinerova 2004)

Heart Disease and Diabetes

Scientific discovery on milk, and in particular A1 versus A2 milk, started in the early 1990s through the work of Bob Elliott, Professor of Child Health at the University of Auckland, New Zealand. Elliott was aware that Samoan children living in New Zealand had a high level of Type 1 Diabetes compared to children living in Samoa. When he found that the differential was tenfold, he immediately concluded that the causative factor must be either dietary or environmental. Finding that the consumption of milk per head of population was much lower in Samoa that NZ, he focused on milk. This led him to make enquiries with the NZ Dairy Research Institute (DRI) where he spoke to senior scientist Jeremy Hill. Mr. Hill was very familiar with milk chemistry including the BCM-7 aspect. Elliott conducted his first experiment on laboratory mice using A1 and A2 milk supplied by the NZ DRI. All of the mice fed A1 milk developed diabetes. None of the mice fed A2 milk developed diabetes. He had a starting point and detailed research got underway.

Scientific momentum continued with the introduction of Corran McLachlan. Dr. McLachlan was a well-known food scientist, innovator and entrepreneur and he was asked by a charity to peer review Elliott's work, a regular practice when funding is sought. McLachlan was immediately taken aback with some of the findings as they coincided with information he was already aware of. However, the direct link between A1 beta-casein and adverse health conditions was new to him. One noteworthy statistic (of many) taken from the WHO (World Health Organisation) Monica study was that death levels from ISD (Ischemic Heart Disease) were 3.5 times higher in Belfast than in Toulouse, despite the fact that there was no appreciable difference in lifestyle factors or nutrient intake for either city.[5]

When he compiled the data on the annual incidence of Type 1 Diabetes and the death levels from IHD, the resulting graph was striking; two health conditions which were clearly far apart. Type 1 Diabetes occurs usually in the early stages of life but death from heart disease occurs late in life. Was there a link between the two? Was one causative of or contributory to the other? Alternatively was there a third element which independently contributed to both? Enquiry on those issues formed the basis of what became McLachlan's hypotheses. His paper was published in the International Journal of Medical Hypotheses and received a considerable level of attention.



Ref: McLachlan CNS. Beta-Casein A1 ischaemic heart disease mortality, and other illnesses. Med Hypotheses 2001; 56: 262-72

Meanwhile Elliott combined with Murray Laugesen, a former Department of Health scientist, who was internationally recognised and decorated for his contribution to public health, in particular his campaign for curtailment of tobacco use.

Laugesen and Elliott conducted epidemiological research based on data from 20 developed countries. They included only countries where the standard of living and lifestyle generally matched and where public health systems were up to international standards. They concluded that A1 Beta-casein from cow's milk was significantly and positively correlated with IHD, and Type 1 Diabetes.[6] Data from their report showed much higher levels of ISD in Northern Europe compared to Southern Europe. Overall statistics of death from IHD in the 20 developed countries showed France had Europe's lowest level with 33 deaths per 100,000 people and Ireland ranked highest with 131 deaths.

On diabetes, this study confirmed Elliott's earlier conclusions that the incidence of diabetes in 12 countries correlated strongly with A1 beta-casein consumption. There were no other ready or available explanations why Iceland, with a high level of milk consumption, had lower levels of Type 1 Diabetes than other genetically-related Nordic countries such as Denmark, Sweden and Finland. New incidences per year of diabetes were 12 times higher in Finland than France per 100,000 people.[5]

Another researcher, Julie Cambell, and colleagues from the University of Queensland conducted research on laboratory rabbits. One group were fed a specific diet, 20% of which consisted of A1 milk. A similar diet containing 20% A2 milk was used for the other group. The animals which were fed the A1 diet developed fatty plaque lesions in their aortas which were larger and thicker than those noted in the A2 diet group.[7]

Autism, Schizophrenia and Cot Death

Diet had featured for a long time in the work of several medical scientists, particularly in studies on mental health conditions such as autism and schizophrenia. Dr. Robert Cade of the University of Florida was a long-standing researcher on autism and other mental health issues. Cade and colleagues published an article linking autism and schizophrenia with intestinal disorders in 2000.[8] He had already recommended by the late 90s the elimination of milk from the diet of autistic children.[9] At that stage he was not to know that there were different types of milk. Medical scientists don't tend to read the work of dairy scientists.

Learning of the new information from NZ, one member of Cade's team set up an experiment with laboratory rats.[10] Each group were injected with a preparation of A1 or A2 milk. The animals injected with the A1 preparation changed behaviour within 30 minutes. They became withdrawn, unsociable and at times aggressive with their cage mate. They ignored different sounds even when a special bell sounded over their cage. The animals administered the A2 preparation displayed no change in their behaviour. Cade observed that the behaviour of the animals treated with the A1 preparation closely resembled the normal day-to-day behaviour of many children with autism and the wider autistic spectrum disorder.

The research conducted by Cade was mirrored by the work of other scientists such as Dr. Calle Reichtt at the University of Oslo, Ann Marie Knivsberg, Paul Shattock and Paul Whiteley at the University of Sunderland. Reichelt and Knivsberg reported their findings of higher levels of opioid peptides in the urine of autistic children compared to the absence of such peptides in non-autistic children could be a causative factor or contributor to autism.[11] The severity of ASD (autistic spectrum disorder) was found to relate to the level of peptide found in the urine of children; the higher the

level of peptide the greater the severity. Low levels in the urine were associated with mild autistic complaints.[12]

Whilst all those scientists conducted extensive lab work, it was not all in the lab. They also developed a number of biomedical interventions for the treatment of autism and related conditions. They worked on elimination diets, in particular gluten-free and casein-free (GFCF). All reported reductions in symptom levels and in many cases entire relief from symptoms. Many thousands of people have benefitted through improved quality of life.

David Niebuhr and colleagues conducted schizophrenia research among US military personnel. From a group of 855 selected cases against 1165 healthy controls, they found an 18% increased hazard risk resulting from ingestion of beta-casein.[13]

As the level of research continued to grow, the focus was on to the opioid called BCM-7. Was this peptide the devil in the milk? The area of research widened to other conditions like allergies and SIDS (sudden infant death syndrome or cot death) and Retts Syndrome. Were there links with ingestion of casein? Animal studies indicated that beta-casomorphine could induce apnoea and irregular heartbeat in new born rabbits. (Hedner and Hedner 1987). Ramabadran and Bansinath developed a hypotheses that opioid peptides could be a possible cause of SIDS.[14] Sun et al, have put forward possible explanations as to how opioid peptides circulating in the infants' immature central nervous system might inhibit respiration and could lead to SIDS.[15]

Reichelt and Skjeldal found antibodies to casein in girls already diagnosed with Retts Syndrome which they described as significant. Retts is a neurodegenerative condition mostly affecting young women. The researchers are of the opinion that the opioid peptide from milk may pass through the gut wall and via the blood to the brain and exasperate the condition.[16]

Parkinson's is another neurodegenerative condition which is not well understood but has undergone extensive research for causative factors. Milk is now under review by Dr. Albert Aschiro and colleagues at Harvard Medical School. However they have not indicated if they believe BCM-7 is a factor.[17]

Dr. Natasha Campbell McBride, in an open letter to parents of autistic children, gives a detailed outline of the history of autism and related conditions over the past 20 years and the inadequacy of conventional medicine to discover possible causes or find solutions. Her conclusions are presented in an excellent book which suggests that many of those health conditions have their origin in diet.[18]

What's the mechanism of action?

With research now concentrating on the different milks, the question arises, what's the mechanism of action? Experiments by Cade and colleagues injecting preparations (both A1 and A2) were clear as the opioid in A1 preparation entered the blood stream and reached the brain quickly. In a normal healthy individual, we could expect a healthy immune system and a healthy digestive system and expect the body could handle different proteins and peptides from food in the normal way. During digestion, proteins are broken up into different fragments called peptides. Many people suffer from gut permeability, a weaker gut which is often described as leaky gut. Other associated conditions are Crohn's, coilits and irritable bowel. Could the intestinal health of such people permit those unwanted proteins/peptides to pass into the bloodstream and from there cross the blood/brain barrier, thereby giving rise to unhealthy mental conditions?

DPP4 (dipepeptidyl peptidase 4) is the enzyme that breaks down the casein in the digestive system.[3] This enzyme is attached to epithelial cells in the lining of the stomach and normally should prevent

fragments of undigested protein passing through the gut wall. However in cases of digestive problems described above, it may explain why infiltration of the blood occurs. Cade and Sun (Peptides 2003) argued that BCM-7 acted in a cascade fashion affecting the brain in 45 locations, whereas peptides from gluten only impacted on the brain in 3 regions.[3] These are issues that scientists are working on and with time we might have more clarity or proof on whether A1 milk gives rise and contributes to the problems referred to in the nutritional press as the gut-brain connection. A group of Polish scientists led by Iwan Malgorzata are working on BCM-7, how it crosses the blood/brain barrier and its connection with other health conditions such as allergies.[19]

Science, Business and Politics

Those three branches of civil society work hand in hand when the need arises. The publication of the Laugesen and Elliott paper attracted considerable media attention, particularly in New Zealand. The NZ Food Safety Authority (NZFSA) commissioned the services of a consultant to investigate the A1/ A2 milk controversy. Prof. Boyd Swinburn of Deakon University in Australia was chosen for the task. Swinburn later presented his report and included therein what he called a lay summary. Clearly he was of the view that he should summarise for public use his findings whilst at the same time providing standard technical details for the scientific community. NZFSA later published the report but interestingly they omitted the lay summary. Swinburn was not pleased and made his views known. PR-wise it went badly wrong for FSA officials as they were forced to acknowledge their omission and then publish the lay summary. Swinburn, in recommending more research, described the A1/A2 hypothesis as intriguing and potentially very important for public health if it was proved correct.[3]

Corran McLachlan, the innovator, was not just content writing reports. He reached into his bank account and established a new Biotech company named A2 Corporation. This company's mission was to develop test systems and technology for the genotyping of animals with the intention of marketing this service internationally. A natural follow-on was the production of A2 milk.

Competition always attracts attention as big corporates don't like new entrants on their patch. Soon after the creation of A2 Corporation, the A2 hypothesis got more coverage in the media but the core message was missed and the theory was essentially denigrated. Nothing new in that.

The controversy raged on mostly in Australia and with that the EFSA commenced a review and assessment of the A1 / A2 issue. The EFSA Report was eventually published in 2009 and concluded with the recommendation; "*based on the present review of available scientific literature, a cause/effect relationship between oral intake of BCM-7 or related peptides and etiology or course of any suggested non-communicable disease cannot be established. Consequently, a formal EFSA risk assessment of food-derived peptides is not recommended "*

In their review, ESFA reported on many scientific papers presented by different experts, some of which linked casein consumption to adverse health conditions whereas other authors reached opposite conclusions. In summary, the review concluded that inadequate evidence existed. As regards to the role of CAM Therapies, and particularly exclusion diets, it states the evidence is poor. On infant milk formula a reader could draw the conclusion that no evidence exists of the presence of A1 beta-casein in those products.

The EFSA report gave a level of comfort to the opponents of A2 milk, particularly in Australia. They used it to great effect. In PR and publicity materials they focused on the statement - A1 or A2 milk; there is no difference, there is no issue. Unfortunately it had the effect of slowing down the very desirable objective of the founding scientists i.e. the conversion of national herds. However there is

an irony in everything. Every occasion when there was publicity or denial of a safety issue in the media, producers reported that demand for A2 milk surged. In addition, scientific enquiry started to grow.

Converting the national herd

A possible genetic solution. CAM supporters will say its nature's way of providing solutions. It is rather disappointing that the dairy industry has gone to such lengths to protect their patch. The reality is that a relatively low-cost test system has been developed for the genotyping of animals so as to convert national cow herds to be A2 milk producers. This could increase demand for milk in the long run, from which the dairy industry would be financially rewarded.

A test procedure was developed by A2 Corporation (on which they received a patent) which can identify whether or not the animal carries the A1 or A2 allele (variant). If an A2 cow is mated with an A2 bull, the progeny will be an authentic A2 animal and the female will produce A2 milk.[3] As a result of historic crises in Europe such as BSE, all bovine animals are now tagged and recorded and their genotype could easily be tracked. Experts in the field report that a farmer could convert his/her herd in a period of about 10-15 years through a combination of farm breeding and use of the AI (artificial insemination) Service. Groups of progressive farmers could share breeding resources and heifer calf exchange so as to increase output of A2 milk, a premium product for which people would be prepared to pay a higher price. Probably the national herd could be converted if the authorities were to recognise the benefits and incentivise such a program in the interests of both public health and expanded export markets.

Epidemiology vs Clinical Trials.

Readers might ask that with all of this scientific data available why don't we have more information on the safety or otherwise of milk. Epidemiology is the science (a science itself) of data collection based upon findings. For example, in the case of the subject under review, France has a national herd with a high proportion of A2 cows. France also has the lowest level of death in Europe from coronary heart disease. It raises the question of a strong connection between A1 milk and CHD. But it is not proof. Epidemiology is a strong indicator of proof but science requires absolute proof.

Medicinal products could be viewed as the best example of how science works for the establishment of proof. Double-blind controlled trials are the methods used to establish the required proof. These work on the basis that a group of people with a particular condition are selected to whom the specific product is administered, whilst another group, similar in number are administered a placebo (a sugar pill of similar appearance). Neither party know whether they are getting the product under investigation or the placebo. Furthermore, the researcher does not know which individual gets the product or the placebo nor do they know what group the individual belongs to. At the end of the trial an independent statistician collates the data so as to provide an assessment of the trial. Whilst this is desirable for the assessment of medicinal products (high risk chemical compounds), it is much more difficult to apply in the case of a food product such as milk, maybe even impossible. Would a parent permit their three month old child be a subject in a double blind trial where their child could ingest a substance which could cause developmental problems or an adverse health condition later.

New Science continues to flow.

Ivano De Noni, from the University of Milan, has presented a number of valuable papers which have been published in the International Journal, Food Chemistry. His paper in 2008 stated that BCM-7 could not be found in commercial samples of milk or milk products.[20] However, in 2010, following laboratory testing replicating human digestive processes, he discovered that that BCM-7 has been found in commercial infant formula and is released from the digestive system.[21] This finding has subsequently been supported by a group of American scientists who found BCM-7 in the digestive tract of healthy individuals.[22] Furthermore, De Noni found that it was released from specially selected A1 samples but not from A2 samples. That's in keeping with the findings of the EFSA Review.

Another significant finding from De Noni is that BCM-7 is released from the digestion of cheese and yogurt. In earlier research, the NZ scientists excluded processed dairy products (cheese and yogurt) from their calculations on the basis that the processing might have released the BCM-7 and it would not therefore have reached the digestive system in a manner that would be harmful. This is now important new information from De Noni as there are many people who do not drink milk but do eat cheese, so there is greater population exposure to BCM-7. Furthermore, yogurt is given to children as a health food. Whilst Di Noni has been categorical on some important issues he raises an important question; is the amount of BCM-7 released sufficient to cause harm? However the issue of exposure has already been addressed, higher urine excretion levels reflect higher symptom levels and vice versa.[11 12]

Alexandra Steirenova is a researcher in the Czech Republic who has studied milk and BCM-7 for more than 10 years. Her work has centred on oxidative LDL (low density lipoproteins, the bad cholestrol) and anti-bodies thereto. LDL is an important indicator of heart disease and maybe Alzheimer's also. Amongst researchers, oxidised LDL is considered very important as it's the sticky substance that builds up as plaque in the arteries. Earlier work by French researchers had shown the casein peptides can promote human LDL oxidation (Torreilles and Guerin 1995**). Steirenova** and her team have shown that some babies have increasing anti-bodies to oxidised LDL whereas others have declining levels relative to levels at birth. The key element in her paper was that the babies with increasing levels were fed on milk formula whereas breastfed babies had the declining levels and furthermore the anti-bodies were directly related to the BCM-7.[23]

Natalya Kost leads a group of scientists in her home country of Russia and that group have been studying BCM-7 for some years and their latest work has been published in the International Journal of Peptides. The research is funded by the Russian Foundation for Basic Research and therefore is not connected to any commercial organisation. Findings by Kost have probably been the most important and relevant to date. Firstly the Russian group have identified the BCM-7 peptide in blood and have developed a blood test for it. This is important as the test will now be available to other researchers who have had to rely on urine or other body fluids for their tests in the past.

Kost and colleagues, in their latest research, have analysed the results obtained from two groups of children under one year. One group were breastfed and the other fed on commercial infant formula. Tests have shown that some babies can eliminate BCM-7 from their system either through metabolising it or excreting it, but that other babies retain it in the bloodstream. Babies who were breast fed had normal psychomotor development and muscle tone whereas babies who are unable to rapidly break down and excrete the BCM-7 have been found to have delayed psychomotor development. The researchers express the view that those children are at risk of developing diseases such as autism.[24]

Conclusion, Recommendations and **Acknowledgment**.

This milk debate is now running for more than 20 years. I believe it will run for some time yet and I have the feeling there is more information in the pipeline. As new scientific data emerges it is pointing in the one direction; A1 milk is a risk. Practitioners who are aware of the issue report improvement or disappearance of symptoms when clients change from A1 to A2 milk. (Cross 2015, Woodford 2010)

However, I feel we will need more direct human trials of A1 and A2 products for direct comparison of outcomes. This will be costly. Classical scientists are difficult to convince. We need more information for different sub-groups such as pregnancy. Could the A1 peptide be harmful to the foetus? Could mothers who drink cow's milk pass the BCM-7 to her baby through breastfeeding?

EFSA is the mothership so far as European food safety is concerned and it is improbable that National Food Safety Authorities will intervene. I have spoken to members of the scientific team at Teagasc, which is the body responsible for Research and Food Science in Ireland. They are fully aware of the debate on different milks and have confirmed that Ireland has the capacity and the tools for herd conversion. A2 cows in Ireland make up 40% of the national herd. [25] That means that the higher proportion of milk available is in the higher risk category. However, Teagasc is not actively promoting herd conversion but equally they are not discouraging conversion either. At this time they consider that A2 remains a hypothesis and until such time as it is proven scientifically they feel they should not engage at the business level.[26] Not an unreasonable position.

As we in the CAM community are well aware, responsibility for personal health rests with oneself. We need to create awareness and develop consciousness around this issue. We are well aware that where any health condition is preventable, clients don't want ambiguity, they want facts. On that note I recommend that:

- All users of baby formula should write to the producers and ask them if their formula is made from A1 or A2 milk.
- Consumers should be advised to ask their milk provider if their milk and dairy products come from A2 herds.
- Practitioners should write to their TD/MP and advise him/her of the role that milk plays in the health of their clients and the importance of informing and developing national policy on the role of food in public health.

My thanks to Prof. Keith Woodford for his excellent book, 'Devil in the Milk', which was a key source of information for the preparation of this review. Keith Woodford is Professor of Farm Management and Agribusiness at Lincoln University in NZ. His motto is, "follow the scientific path wherever it leads you."

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About the Author

Martin Murray is Managing Director of New Vistas Healthcare Ltd. He is a member of the Advisory Board of University of Limerick, Kemmy Business School and is former President of the Irish Health Trade Association. He can be contacted at: martin@newvistashealthcare.com