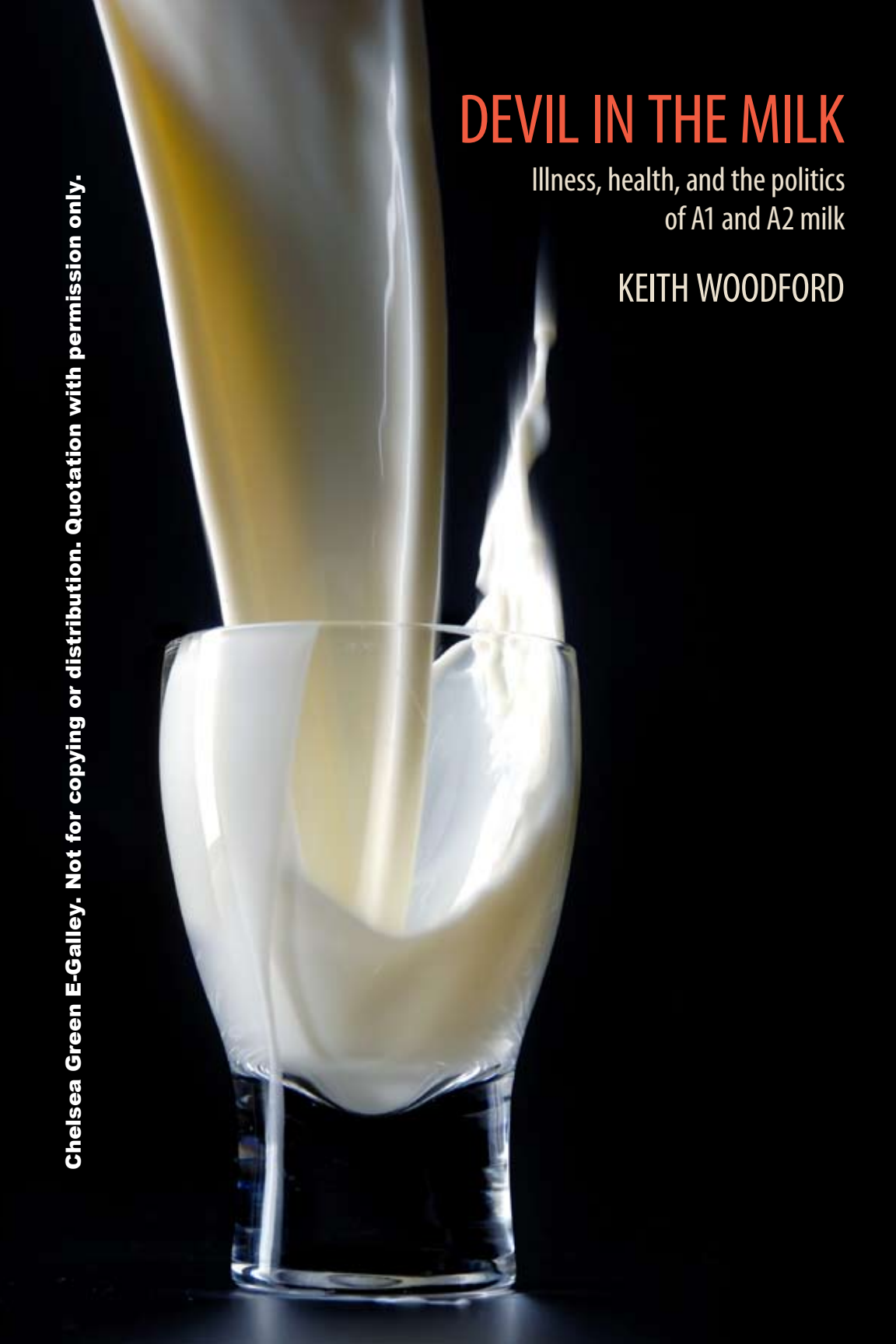

DEVIL IN THE MILK

Illness, health, and the politics of A1 and A2 milk

KEITH WOODFORD

–First North American Edition–
(Originally published in New Zealand)
Now with a new postscript from the author.

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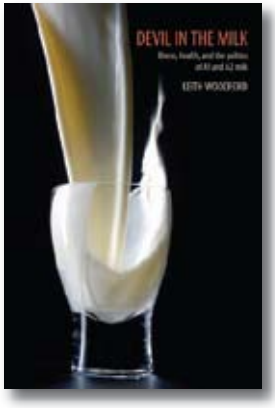
Keith Woodford



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- National Media
- U.S. Radio Tour

Evidence shows cows' milk is a link to solving many medical mysteries, from diabetes to autism.

This groundbreaking work is the first internationally published book to examine the link between a protein in the milk we drink and a range of serious illnesses, including heart disease, Type 1 diabetes, autism, and schizophrenia.

These health problems are linked to a tiny protein fragment that is formed when we digest A1 beta-casein, a milk protein produced by many cows in the United States and northern European countries. Milk that contains A1 beta-casein is commonly known as A1 milk; milk that does not is called A2. All milk was once A2, until a genetic mutation occurred some thousands of years ago in some European cattle. A2 milk remains high in herds in much of Asia, Africa, and parts of Southern Europe. A1 milk is common in the United States, New Zealand, Australia, and Europe.

In *Devil in the Milk*, Keith Woodford brings together the evidence published in more than 100 scientific papers. He examines the population studies that look at the link between consumption of A1 milk and the incidence of heart disease and Type 1 diabetes; he explains the science that underpins the A1/A2 hypothesis; and he examines the research undertaken with animals and humans. The evidence is compelling: We should be switching to A2 milk.

A2 milk from selected cows is now marketed in parts of the U.S., and it is possible to convert a herd of cows producing A1 milk to cows producing A2 milk.

This is an amazing story, one that is not just about the health issues surrounding A1 milk, but also about how scientific evidence can be molded and withheld by vested interests, and how consumer choices are influenced by the interests of corporate business.

Keith Woodford is Professor of Farm Management and Agribusiness at Lincoln University in New Zealand. A regular commentator in the news media, he was previously at the University of Queensland (Australia) for 20 years. He lives with his family in Christchurch, New Zealand.

"*Devil in the Milk* is potentially as significant as Carson's *Silent Spring* or Nader's *Unsafe at Any Speed*. . . . *Devil in the Milk* is soundly researched. . . . It should be read by every dairy farmer and consumer." —Alan Robb, *The New Zealand Farmer's Weekly*

"[A] shattering exposé of the health problems caused by milk and the efforts of elements in the dairy industry and government to cover them up." —Jacqueline Steincamp, *Healthy Options*

"I believe this is an important book. Critics should think carefully and avoid knee-jerk reactions." —Professor Sir John Scott, Professor Emeritus of Medicine, University of Auckland

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DEVIL IN THE MILK

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Illness, health, and the politics of A1 and A2 milk

KEITH WOODFORD

Chelsea Green Publishing
White River Junction, Vermont

AUTHOR'S NOTE

Where scientists play a major role in the milk devil story, and where their titles are known (either Professor or Doctor) I use those titles when first mentioning a person. I also use the given name. However, for some scientists, particularly those mentioned primarily as authors, neither title nor given name is evident from their publications. Most medical scientists will hold the degree of PhD (or its European equivalent) and have the title of Doctor. However, some scientists have different qualifications that may or may not provide the title of Doctor. I apologise to any scientist mentioned in this book without use of title who considers that a title should have been used. On occasions there may also be inconsistency in the use of given names. This can arise when scientists from non-English–speaking countries use both anglicised and non-anglicised versions. Where names are used widely within the same section, for brevity I usually repeat only the surname.

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PREFACE TO THE NORTH AMERICAN EDITION

When I wrote the first edition of this book, I wrote for a New Zealand and Australian audience. This was because much of the early work on A1 beta-casein and its health effects took place in New Zealand and to a lesser extent Australia. Also, these are my 'home audiences'. But the issue of A1 beta casein and its health effects is a matter of huge importance throughout much of the world, particularly those countries that have 'black and white' cows of European origin. The USA, Canada, Britain, and much of Scandinavia come into this category.

At the moment, hardly anyone in North America knows anything about A1 beta-casein or the alternative milk known as A2 milk. A2 milk is available in seven Midwest states but only in small quantities. However, I am told that the main industry players in the USA do have a large file on the topic and are watching closely.

International trade in dairy products is dominated by my home country, New Zealand. This includes large quantities of casein that are exported to North America by our dominant milk-marketing cooperative, Fonterra. Most American readers of this book will never have previously heard of Fonterra, but if they have ever purchased a muesli bar, there is a fair chance that the material that binds it all together is casein from Fonterra. Some of the infant formula consumed in North America will also be made from milk powder that comes from Fonterra in New Zealand.

Americans should not be concerned that some of their casein comes from New Zealand's Fonterra. In fact, New Zealand is quietly converting its herds to A2 without telling the rest of the world. What North Americans should be concerned about is that North American milk is very high in A1 beta-casein, and no one is doing anything about it.

ACKNOWLEDGEMENTS

Researching and writing this book, largely at nights and weekends and over a period of some three years, has been a solitary experience. I therefore acknowledge foremost my family, and particularly my wife Annette, who has had to put up with my pre-occupation. My family has also been an important sounding board at critical times.

Dr Andrew Clarke was particularly helpful in leading me to some of the relevant literature. I admire the way that Andrew combines a commitment to the cause of A2 milk with a stance of scientific caution, and a steadfast belief that arguments must be evidence-based.

Mike Bradstock worked closely with me as editor. His own scientific background and questioning attitude were invaluable. Mike also made a major stylistic contribution.

Julian Bateson and Don Cameron both read early drafts and helped provide me with confidence that a complex story did indeed hang together.

Robbie Burton and his team at Craig Potton Publishing have been enthusiastic and professional. It takes a brave publisher to commit to a book such as this. I like the commitment at CPP to publishing books which they believe are important.

Of course this book could never have been written without some outstanding work by many scientists who made the discoveries on which this book is based. Some of them I have met and corresponded with; others I know only by the scientific evidence that they have produced.

One always hopes that a book such as this is free of scientific error. But science is seldom free of error. Indeed the way that scientific knowledge progresses is by refutation of theories that conventional wisdom says are correct. If there are errors in this book then they are my responsibility.

PROLOGUE

This book is about the effects on human health of a tiny protein fragment called beta-casomorphin-7, or BCM7 for short.

BCM7 is unquestionably a powerful opioid and hence a narcotic. It is also an oxidant. It is formed by digestion of a particular type of milk protein produced by some cows. This milk protein is called A1 beta-casein.

The BCM7 that is released from A1 beta-casein has been implicated in many illnesses, including heart disease, Type 1 diabetes and autism. And there is increasing evidence that it is associated with milk intolerance and an additional range of auto-immune diseases. Metaphorically, it is 'the devil in the milk'.

The 'milk devil' story is built upon more than a hundred scientific papers published in international journals, and on documents from milk-marketing companies. It is a story that has never been brought together before.

There is strong evidence that the milk devil is produced only from the milk of cows that are of European origin, and then from only some of these cows. Asian and African breeds of cows are free of it (unless they have some hidden European ancestry). So are goats. And so (with a very minor but fascinating qualification) is human milk.

No-one can tell by looking at a cow whether or not she is a source of the milk devil. However, genetic testing is possible, and it is also possible to test the milk. Farmers can breed cows that are free of the problematic protein by using appropriately tested bulls and semen.

Anyone who buys ordinary milk at the supermarket can be sure that it will contain milk from many cows and therefore there will be lots of A1 beta-casein in it. However, the level varies between countries, and even between regions. Some countries such as Australia, New Zealand,

Finland, the United States and Great Britain have milk with high levels of this protein. The milk in others, such as Iceland, France and the island of Guernsey, has much lower levels.

We don't have to stop drinking cows' milk to avoid this devil. But we do have to drink milk from cows that have been tested and found to be free of what is called the A1 variant (or 'allele') of the beta-casein gene. Milk that is free of A1 beta-casein is known as A2 milk. All milk used to be A2 milk until a natural mutation affected some European cows a long time ago.

A2 milk is available in more than 1000 Australian supermarkets and stores, although with a low market profile. It is also available in a very limited number of stores in New Zealand. In 2007 it became available for the first time in seven mid-western states of the USA. The reasons why A2 milk has had such a low market profile are themselves a major part of the story.

Throughout this book I often refer to A1 milk. This is essentially a shorthand for milk that contains some A1 beta-casein, the source of the milk devil.

The story of A1 versus A2 milk may sound stranger than fiction. Indeed, it is an amazing story. It is a story of how science works and doesn't work. It is also a story of how the forces of big business and the so-called 'health industry' work, and of how wishful thinking can get in the way of truth.

INTRODUCTION

I am often asked why I have such an interest in A2 milk, and why I have taken to speaking in public about the issue. It is a valid question, because we live in a world where people are often driven by hidden motives. There is also another question, sometimes left unstated, as to whether I have the competency to talk about such matters.

I prefer to address these issues head-on, and so will explain something of my own background. But in the final analysis, my arguments and perspectives should stand or fall on the quality of the evidence. What I try to do is to provide a balanced perspective of that evidence, including the arguments of those who do not believe in what is sometimes called 'the A2 hypothesis'.

My personal intention has been to read everything in the scientific literature that seems relevant to the issue, and to treat all evidence with scepticism. This is not an easy task. For a start, the relevant literature spans research into diabetes, heart disease, autism and schizophrenia. It also includes biochemistry, pharmacology and genetics (human and bovine). Scientific journals in each of these fields have their own specialist language. Making judgements about the A2 hypothesis also requires an understanding of biometrics (the testing and interpretation of biological data) and an understanding of research processes and philosophies.

It has been interesting for me to find that there are very few people who have read widely across the literature in relation to A1 and A2 milk. This deficiency is at least in part because we live in a reductionist world where specialisation requires people to put boundaries around issues. Back in the days of Darwin, scientists from different disciplines read each other's work, but today in general that no longer occurs.

My early formal training was a four-year degree in agricultural science from Lincoln College, at that time part of Canterbury University,

in New Zealand. Subsequently I studied for a Master of Agricultural Science, specialising in agricultural systems and management. Much later I undertook a PhD degree at University of Queensland in Australia, focusing on the bio-economics of industry development. Much of my professional life has been spent in and around universities. I have lived mainly in New Zealand and Australia, but also worked on development projects in more than 20 Asian and Pacific countries such as Papua New Guinea, Fiji, Cambodia and Vietnam. I have also squeezed in quite a lot of mountaineering, particularly but not exclusively in my younger days, and this has taken me around the world including Antarctica, South America and the Himalayas.

Currently I am Professor of Farm Management and Agribusiness within the Division of Agriculture and Life Sciences at Lincoln University in New Zealand. My professional interests relate to agriculture as a field of study that crosses the boundaries of science, economics, management and commerce. In the final analysis, agriculture is about people and the decisions they make, at least as much as it is about biology. At heart, I would characterise myself as an agriculturalist with particular interests in farming systems, farming decisions, and the linkages within the overall value chain from consumers back to the farm. These interests force me to read across the disciplines. Because the A2 milk issue crosses all of the normal disciplinary boundaries, it is exactly the sort of thing I enjoy getting my teeth into.

No-one can know everything in relation to the science of BCM7. There is always more to know. Biology and medicine are seldom simple. They are like a big jigsaw puzzle. In the case of BCM7, more and more of the pieces of that puzzle have been coming together. Occasionally a piece may get wrongly placed, and there can be disputes about these individual pieces and where they fit.

It has not been my job to construct the individual pieces of the puzzle. That has been the task of many specialist scientists. Given the range of disciplines involved, it would be impossible for any one person, or indeed a group of people, to construct more than a few individual pieces. My task has been to help bring together the available pieces of scientific information to illuminate the big picture. All of the key scientific information comes from published scientific papers. In this book I share this evidence, including both what we do and do not know, with you, the reader. It seems to me that this big picture has been getting progressively clearer, but you can draw your own conclusions.

As a professor of farm management and agribusiness I am interested

in assessing risks to our agricultural industries and then working on strategies that farmers and downstream agribusinesses can use to minimise those risks. In relation to A2 milk it is not particularly difficult to convert a herd of cows so that they produce only A2 milk, but it typically takes about 10 years (roughly two generations of cows) to make the change. Therefore, if the issue of A1 versus A2 milk becomes important to a lot of consumers, farmers will have needed to start acting before the market demands that they do so. Also, during the period of transition there are marketing issues as to how a company can position both A1 and A2 milk in the marketplace. In a professional context, these are the types of issues that I have to address. I therefore find myself talking to farmers in New Zealand, Australia, the Americas and Europe about the business risks of moving to produce A2 milk when subsequent events may prove it was not necessary, versus the risks of not making the move and then finding that the marketplace demands that milk be the A2 variant.

Prior to November 2003 my knowledge about A1 and A2 milk was minimal. I was vaguely aware that there was a company called A2 Corporation and that it was claiming to have milk that was healthier than so-called 'normal' milk. This milk had recently gone on sale in both Australia and New Zealand. I was also aware that Fonterra, the major milk-processing and marketing co-operative in New Zealand, and also a major force within the Australian industry, was disputing the claims of A2 Corporation. (Fonterra is also by far the world's largest international trader of dairy products.) I had assumed that if Fonterra said the claims were not valid, then that was probably true.

My perspective changed totally as the result of a casual inquiry of a colleague. At the time I was a member of the Telford Rural Polytechnic Council, which meets regularly in its governance role. The Chair of the Council at that time was Dr Jock Allison, a well-known agricultural scientist who in 2003 won Lincoln University's Bledisloe Medal for the alumnus who had contributed the most to New Zealand agriculture.

One Friday morning in November 2003 I flew from Christchurch, where I live, to Dunedin, and then drove to Balclutha for one of our regular Council meetings. I had recently become aware that Jock had become a director of A2 Corporation. During a break in the meeting I asked him why he had got involved with such a company, given the questions as to the validity of the A2 Corporation's claims. Jock's response was to pull out some papers from his briefcase and tell me to read them.

On returning to Christchurch that night I was sufficiently intrigued

to begin a computer search for more information. I ended up spending most of the weekend reading more and more about it. By the end of the weekend I was persuaded that the A2 hypothesis 'had legs'. I was convinced that it was going to become a really big issue and not going to go away in a hurry. I knew that I would have to do a lot more reading to get my mind around some very complex issues, and that there would be many twists and turns, with evidence and counter-evidence, argument and counter-argument, before the final truth would emerge.

Subsequent to this, but before I had a clear intention of writing extensively about A1 beta-casein and BCM7, some of my family, but not me personally, purchased a minor shareholding in A2 Corporation. These shares were purchased on the New Zealand Stock Exchange in the same way and at the same price that any citizen could obtain them. It could therefore be argued that I was no longer completely independent in relation to these matters. On the other hand, when I first started to tell my colleagues about the A2 hypothesis, and put forward the view that it seemed to have considerable merit, one of them told me that it would be much more convincing if I 'put my money where my mouth was'. What I have learned long ago is that it is not possible to keep everyone happy. In regard to a controversy such as A1 and A2 there will be detractors whatever stance one takes. Accordingly, in articles that I wrote thereafter, I disclosed my interest, such as it was, and left people to make their own judgements. Most publishers printed the disclosure, but others chose not to, presumably on the grounds that they deemed it of no significance.

More recently, it became apparent that some people, struggling to find flaws in the evidence I presented, were indeed going to use this share ownership issue to argue that I was not independent and was writing articles for ulterior motives. It was a distraction that I did not need. Accordingly, following family discussions, the Woodford family has sold those shares. I therefore advise that neither I nor my family have any financial interest in either A2 Corporation or any franchisee thereof. I also advise that I have undertaken no consultancies for A2 Corporation, or for any joint venture company or franchisee associated with A2 Corporation.

I make the observation that disclosure of interest, and the potential for conflict of interest, can be a very tricky issue. The reality is that most people involved with the issue of A2 milk have potential conflicts

of interest. That includes all dairy farmers, the dairy marketing companies, and also the scientists who depend on industry funding. I will talk more about those issues throughout this book. What we all have to endeavour to do is to recognise and disclose our interests and act with integrity in the search for truth.

I have been fascinated by the number of inherently good people who say to me 'but we must not do anything that damages the dairy industry'. When I respond that this sounds very much like the historical attitudes within the tobacco industry they are shocked by the comparison. Integrity requires that we go wherever the path of evidence takes us.

The A2 story is complex and sorting out the wheat from the chaff has not been particularly easy.

My personal assessment of the evidence is that the issue of A1 and A2 milk is a major health issue. It is also my assessment that some people have acted, either purposefully or accidentally, in a way that has obscured the truth. It is very easy to ignore unpleasant evidence that threatens an existing stance. It is a human trait. After reading this book you can make your own judgements on these matters.

Despite being a proponent of A2 milk, I do not wish to make any suggestion regarding investment in A2 Corporation. Although I am very confident that in fifty years' time, and hopefully much sooner, we will all be drinking milk that is free of A1 beta-casein, I have no clear view whether or not A2 Corporation is a good investment. Nevertheless, I do believe that A2 Corporation is very important: without it the message about A1 beta-casein and BCM7 would probably have been buried, or at least taken a great deal longer to emerge. But it is another matter whether or not A2 Corporation can prosper from its patents and trademarks. Capitalising on intellectual property is not always easy. There are lots of pitfalls, as readers of this book will become aware.

I also want to make a statement about the freedom of speech that goes with being an academic. The considered opinions and judgements that I make in this book are entirely my own. When academics speak about an issue they are representing themselves and not the university that employs them. Speaking about issues is part of our role. As academics we do not seek permission to speak on any particular issue, and we should never imply that the stance we take is the university's position. This is quite different from the situation of scientists who work for commercial organisations or for non-university government research organisations.

It is an important distinction. My employer, Lincoln University, holds no position either for or against the putative role played by what I call the milk devil.

So why have I written this book? Some of my friends have suggested to me that from a career perspective it is not a very smart move. They may be correct. But I have now got to a stage in life where some things are more important than others. I believe the A2 story is one that needs to be told.

CHAPTER ONE

BEGINNINGS

The A2 story starts in 1993 with Professor Bob Elliott from Auckland University in New Zealand. Elliott was Professor (now Professor Emeritus) of Child Health, and as part of his work had been looking at the incidence of Type 1 diabetes among Samoan children. Type 1 diabetes is an immune-response disease where the pancreas loses its ability to produce insulin. Insulin is a natural hormone required for the transport of glucose into the cells, where the glucose provides energy. The disease usually strikes either in childhood or early adulthood, but only a small proportion of people seems to be susceptible. People with Type 1 diabetes need regular insulin injections for the rest of their life. The incidence of Type 1 diabetes has been steadily rising throughout the world and it has been a real puzzle as to why this is happening. Another puzzle is why the incidence of the disease varies greatly (as much as 300-fold) between countries.

There are two types of diabetes, Type 1 and Type 2. Type 1 usually develops in childhood or young adulthood, while Type 2 is mainly a disease of older people. Both diseases relate to an inability to metabolise glucose, and both are linked to insulin, but they are also fundamentally different. Type 1 diabetics do not produce the insulin they need because of damage to the insulin-producing cells in the pancreas. In contrast, Type 2 diabetics still produce at least some insulin but their body is 'insulin resistant'. This means that the insulin, although present, cannot do a good job of getting glucose into the cells where it is needed. The way to prevent or greatly reduce the risk of Type 2 diabetes is through exercise and weight control. In contrast, there are no generally accepted health strategies for avoiding Type 1 diabetes. Our interest in this book is with Type 1 diabetes.

Bob Elliott was aware that Samoan children living in New Zealand were very susceptible to Type 1 diabetes, whereas Samoan children living

in Samoa had an extremely low incidence. The tenfold difference could be explained only by an environmental or dietary factor. Elliott suspected that at least part of the answer related to the consumption of milk, which was much lower in Samoa. But he also knew that the complete answer was unlikely to be anywhere near as simple as that.

Accordingly, some time in 1993 Elliott telephoned the New Zealand Dairy Research Institute (NZDRI) and asked to speak to someone who knew about cows and milk-protein biochemistry. Dr Jeremy Hill took the call. His advice was that it could be worth looking at the beta-casein proteins, although it would be a long shot.

Hill would have known that in cattle there are essentially two major types of beta-casein protein, known as A1 and A2. There are also some other minor variants within the A1 and A2 families, but at this stage of the story they can be ignored.

The beta-casein proteins found in cattle comprise 209 amino acids in a fixed sequence and making up a convoluted string. The difference between the A1 and A2 variants is just one of these 209 amino acids. Whereas A1 milk has the amino acid histidine at position 67, the A2 milk contains proline at the same position. Back in 1993 the significance of this minor difference was not understood, although it had been known to milk biochemists for about 25 years.

The prevalence of the A1 and A2 beta-casein protein varies from one herd of cows to another, and also between countries. However, the A1 version of the gene is found only among cattle in the western world, all of which belong to the subspecies *Bos taurus*. Asian cattle are of the subspecies *Bos indicus* and do not produce A1 beta-casein.[1] African cattle, although mainly *Bos taurus*, also do not produce A1 beta-casein. However, a qualification needs to be made in that many supposedly 'pure' Asian and African cattle contain genes that can only have come from breeding with European cattle at some time in the last few thousand years, and hence may produce some A1 beta-casein. Scientists think that a mutation occurred about 8000 years ago, such that the proline at position 67 was replaced by histidine. The genetic evidence for this mutation is very clear, although there could be an error of some thousands of years as to when it occurred.[2] This mutation has subsequently been spread widely throughout herds in the western world. However, there is considerable difference in the prevalence of the A1 gene between breeds, countries, and in some cases, provinces.

So the hypothesis that Bob Elliott set out to investigate was that the

risk of getting Type 1 diabetes would depend on the amount of milk that was drunk and the proportion of A1 protein in that milk. The key risk factor would be the volume of milk multiplied by its A1 content.

Undertaking human trials to investigate such issues is very difficult. The subjects of the trial would need to be identified as babies and then put on either A1 or A2 formula milk once breastfeeding ceased. The trials would probably need to go on for many years, and the children prevented from eating any 'ordinary' dairy products. The parents of each child would need to give permission and be actively involved, but could not be permitted to know whether their beautiful and initially healthy baby was getting the A1 or A2 formula. This is called a 'blind trial' and it is a very important element of experimental design. Indeed to have a high level of scientific validity the trial should be 'double blind', where none of the scientists dealing with the babies, nor their parents, nor the investigators doing the blood analyses, would know which baby was receiving which treatment. Someone totally separate would hold the codes. (These, and other principles of scientific investigation, are discussed in more detail in Appendix 1.)

It is therefore not surprising that scientists often seek out easier methods of getting answers than by working directly with human patients. One such approach is epidemiology: looking at what happens to populations of people over time in regard to disease incidence, or alternatively looking at different populations at a point in time. (In medical terminology, 'incidence' measures the number of new cases per year; 'prevalence' measures the total number of cases, both new and old, in a population.) Another approach is to use animals as surrogates for people, in the hope that animals will react in the same way as humans.

Bob Elliott decided to look at the problem both ways. The epidemiological approach was to compare the incidence of disease against the intake of A1 and A2 milk for each country. For the animal work he decided to use mice that had been specially bred for susceptibility to diabetes. One of his co-workers in both projects was Dr Jeremy Hill.

The initial results with the mice were exciting. Elliott found that there was indeed a difference in the diabetes incidence between those fed the A1 beta-casein and those fed A2 beta-casein. In fact none of the mice fed A2 beta-casein got diabetes, whereas 47% of those fed A1 beta-casein were diabetic after 250 days. He also found that feeding naloxone with the A1 beta-casein nullified the effect. Naloxone is an opioid antagonist. In other words, it blocks the narcotic effects of opioids. Elliott would

have known that if there were a difference between the digested A1 and A2 beta-caseins, it would almost certainly be related to the release of BCM7. He would also have known that BCM7 was a powerful opioid, as this had been published back in 1985.[3] These results therefore suggested very strongly that the effects of the A1 beta-casein were indeed linked to the opioid characteristics of BCM7, although the mechanism by which this might be occurring was not apparent.

Some people might argue that this initial work is best considered as preliminary, in that it was not a 'blind' experiment. In other words, the investigators who did the analyses knew which group of mice was getting which feed. Also, it was not published in the normal scientific literature, but instead as a paper in a special 1997 publication of the International Dairy Federation called *Milk Protein Polymorphism*.[4] Within the scientific community such publications are considered less weighty than international journals. But it did get things started. It provided an empirical (data-based) underpinning of the hypothesis. And it certainly made some people in the dairy industry sit up and take notice.

The other thread of Bob Elliott's work was epidemiology. It was initially thwarted by difficulties in getting data on the A1 and A2 milk composition in different countries. But good fortune intervened at this stage through the chance involvement of Dr Corran McLachlan. Dr McLachlan had been working on processes to manufacture low-cholesterol and cholesterol-free foodstuffs, and was asked by the New Zealand Child Health Research Foundation to review Elliott's 1994 work programme. The Child Health Research Foundation was a Rotary charity set up to fund child-health research. McLachlan was startled when looking at the incidence of Type 1 diabetes to see that the incidence correlated very strongly with data on heart disease with which he was already very familiar (Figure 1).

In a letter to the *New Zealand Medical Journal* in March 2003, in which this graph was published, Corran McLachlan wrote,[5] 'Considering IDDM [Type 1 diabetes] is thought to be a disease of immune stimulation and IHD [ischaemic heart disease] is a disease associated with immune compromisation, the parallels are remarkable. This similarity raises questions with respect to commonality of the source of damage, as well as the time of primary damage.'

In referring to 'the time of primary damage', McLachlan was suggesting that the causal agents of heart disease might be doing their damage early in life, a point which I will come back to in Chapter 3. But the really

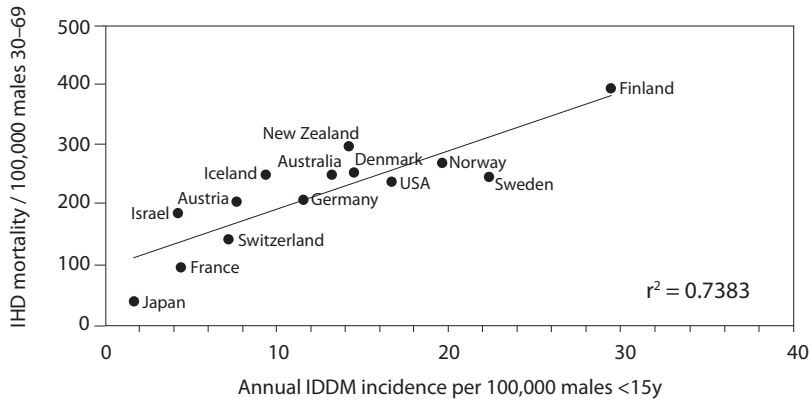

Fig. 1. IHD death rate 1985 for males aged 30–69 vs IDDM incidence for males aged <15. (Reproduced with permission from the *New Zealand Medical Journal* 116 (1170).)

important message was the strong implication that there was a common factor causing both heart disease and Type 1 diabetes. Populations that had high levels of heart disease (mainly among their old people) were the same populations that had high levels of Type 1 diabetes (mainly developing in young people). Statistical theory (which I will turn to in more detail later) tells us that it is highly unlikely that a correlation as strong as this would be simply due to chance. In other words, there is a very high probability of one or more common causal factors.

Determining that countries with high Type 1 diabetes also had high rates of heart disease was a defining moment in the life of Corran McLachlan. It caused him to redirect his own research work. From 1994 until his tragic death from melanoma in August 2003 he was driven by the belief that A1 beta-casein, and the BCM7 fragment that derives from it, were a huge public health issue affecting both heart disease and Type 1 diabetes. It became the focus of both his professional and personal life.

Among the key figures in the story of A2 milk, Corran McLachlan and Bob Elliott stand pre-eminent. So who was Corran McLachlan? The following description is taken from an obituary that was for several years on the A2 Corporation website.

> Corrie McLachlan was born in 1944 to an old established New Zealand family and raised on the family farm in Masterton with his two

brothers and his sister. His country upbringing engendered a love of natural history that remained with him throughout his life.

He was educated at Wairarapa College where he developed his interest in physics and chemistry and also became the head boy and senior athletics champion. In 1962 he went on to Canterbury University where he gained a first class honours degree in Chemical Engineering. This was followed by a move to Cambridge University in England, where he completed a PhD thesis on the Reactions of CO_2 in Alkaline Solutions under Professor de Danckwerts in 1969.

During this time, he met and married his wife, Ulrike, who was working in Cambridge as an au pair. He also began to collect rare books on natural history, later specializing in the natural history of New Zealand.

Dr McLachlan returned to New Zealand in 1970 to take up a position in the Chemistry Division of the DSIR, doing biotechnology research on polymers, agricultural processes, and the dairy industry. He was the recipient of the first United Development Corporation Inventor's Prize in 1974.

After two years as a Visiting Research Fellow at the Engler-Bunte Institute, University of Karlsruhe between 1975–77, Dr McLachlan returned to the DSIR in 1978 as Group Leader of the Industrial Research Division, investigating biotechnology, food processing, industrial chemistry, and chemical engineering.

In 1981 he joined the Energy Section of the NZ Treasury, where he conducted appraisals of the Electricity Division, NZ Power Planning, and NZ Steel.

Dr McLachlan then joined Kupe Group in 1985 as the General Manager, New Investments; and also became an Executive Director of Duncan & Davies Nurseries Ltd, responsible for Operations and Management in New Zealand, as well as Chairman of their UK subsidiary.

Three years later, he set up a venture research company with the Morrinsville-Thames Valley Dairy Co-operative to manufacture a cholesterol-free butter and low-fat meat products through the use of novel extraction technology. He remained as the Managing Director of Tenon Developments Ltd until his death.

Dr McLachlan was made an honorary Senior Research Fellow of the School of Biological Sciences, Auckland University, in 1995. He authored 29 scientific papers and confidential reports and holds 11 patents.