

REFERENCES

A1 – A2 Beta-Casein Milk

1. Allison, A. J. and A. J. Clarke. 2006. "Further Research for Consideration in 'the A2 Milk Case'." *European Journal of Clinical Nutrition* 60:921-4.

Abstract: The recently published review considers some of the scientific literature relating to the proposed relationship between the A1 and A2 variants of beta casein ('A1' and 'A2', respectively) and Type 1 diabetes mellitus (DM-1) and coronary heart disease (CHD). It extends also to discuss briefly the putative link between the beta casein variants and neurological conditions. The authors of this letter wish to introduce literature and data omitted by the said review believed to be relevant to the considerations presented and the conclusions drawn there from.

2. Bartley, Jim and Susan McGlashan. 2010. "Does Milk Increase Mucus Production?"

Medical Hypotheses 74:732-4

Abstract: Excessive milk consumption has a long association with increased respiratory tract mucus production and asthma. Such an association cannot be explained using a conventional allergic paradigm and there is limited medical evidence showing causality. In the human colon, β -casomorphin-7 (β -CM-7), an exorphin derived from the breakdown of A1 milk, stimulates mucus production from gut MUC5AC glands. In the presence of inflammation similar mucus overproduction from respiratory tract MUC5AC glands characterizes many respiratory tract diseases. β -CM-7 from the blood stream could stimulate the production and secretion of mucus production from these respiratory glands. Such a hypothesis could be tested in vitro using quantitative RT-PCR to show that the addition of β -CM-7 into an incubation medium of respiratory goblet cells elicits an increase in MUC5AC mRNA and by identifying β -CM-7 in the blood of asthmatic patients. This association may not necessarily be simply cause and effect as the person has to be consuming A1 milk, β -CM-7 must pass into the systemic circulation and the tissues have to be actively inflamed. These prerequisites could explain why only a subgroup of the population, who have increased respiratory tract mucus production, find that many of their symptoms, including asthma, improve on a dairy elimination diet.

3. Beales, P., R. Elliot, H. Flohe, J. Hill, H. Kolb, P. Pozzilli, G. S. Wang, H. Wasmuth and F. Scott. 2002. "A Multi-Centre, Blinded International Trial of the Effect of A1 and A2 Beta-Casein Variants on Diabetes Incidence in Two Rodent Models of Spontaneous Type 1 Diabetes." *Diabetologia* 45:1240-6.

Abstract / Aims / Hypothesis. The diabetes-inducing potential of cows' milk is still debated and there is no consensus on the diabetogenicity of individual milk proteins. A1- β -casein has been associated with increased diabetes frequency in ecological studies and in NOD mice. Our aim was to ascertain whether A1- β -casein was more diabetogenic than A2 and to test the diabetogenicity of a milk-free diet in animals representing different forms of spontaneous Type I (insulin-dependent) diabetes mellitus. Methods. Defined diets were coded and shipped to laboratories in New Zealand (NOD/NZ), Canada (BB) and the UK (NOD/Ba). Base diets were Pregestimil (PG) and ProSobee (PS). Purified fractions of whole casein (WC), A1 or A2- β -casein were added at 10%. A milk-free, wheat-predominant, NTP-2000 diet was the control. Animals were fed from weaning up to 150 or 250 days, and insulinitis, diabetes frequency and expression of pancreatic cytokines were assessed. Results. Diabetes incidence was highest in three locations in animals fed NTP-2000. PG and PS diets were protective except for NOD/Ba mice fed PG+WC where incidence was similar to NTP-2000. A1 and A2 diets were protective in both models, but A1 β -casein was

slightly more diabetogenic in PS-fed BB rats. The New Zealand study was confounded by an infection. Conclusion / interpretation: A milk-free, wheat-pre-dominant diet was highly diabetogenic in three widely separate locations in both animal models. A previous result that A1 β -casein was more diabetogenic than A2 β -casein in NOD mice was not confirmed; both β -casein variants were protective in BB rats and NOD mice. Whole Casein promoted diabetes in NOD/Ba but protected BB showing that unique diabetes haplotypes react differently to dietary proteins. A1- was more diabetogenic than A2- β -casein only in PS-fed BB rats. Neither the analysis of insulin nor of pancreatic cytokine gene expression showed a difference between A1 or A2 β -casein fed animals. Milk caseins are unlikely to be exclusive promoters of Type I diabetes, but could enhance the outcome of diabetes in some cases. Other diet components such as wheat could be more important promoters of Type I diabetes.

4. Bell, Stacey, G. Grochoski and A. J. Clarke. 2006. "Health Implications of Milk Containing β -Casein with the A 2 Genetic Variant." *Critical Reviews in Food Science and Nutrition* 46:93-100.

Abstract: Milk from dairy cows has long provided a high quality source of protein and selected micronutrients such as calcium to most populations. Recently, a relationship between disease risk and consumption of a specific bovine β -casein fraction either A1 or A 2 genetic variants has been identified. Populations, which consume milk containing high levels of β -casein A 2 variant, have a lower incidence of cardiovascular disease and type 1 diabetes. Furthermore, consumption of milk with the A 2 variant may be associated with less severe symptoms of autism and schizophrenia. The mechanism of action focuses on β -casein A 1 and related forms preferentially that are able to produce a bioactive opioid peptide, β -casomorphin-7 (B-CM-7) during digestion. Infants may absorb B-CM-7 due to an immature gastrointestinal tract. Adults, on the other hand, appear to reap the biological activity locally on the intestinal brush boarder. B-CM-7 can potentially affect numerous opioid receptors in the nervous, endocrine, and immune systems. Whether there is a definite health benefit to milk containing the A 2 genetic variant is unknown and requires further investigation.

5. Boland, Mike, Alastair MacGibbon and Jeremy Hill. 2001. "Designer Milks for the New Millennium." *Livestock Production Science* 72:99-109.

Abstract: Milk is an inherently expensive raw material for use as a food. To compete in the new millennium, dairy products will need to be based on special values that can only come from milk. These include traditional dairy products and health-giving products. Designer milks will be needed to give new, enhanced products and to improve the quality and value of traditional products. The use of milk for traditional products is likely to continue to be strong in western cultures. For these products, key issues are naturalness of supply, with "organic" milk being an important issue; and low-fat products, which may imply a need for lower fat milk. Health products are the most exciting new area for milk-based products. A number of components in milk are being recognised as conferring health benefits. These include minerals (calcium), peptides derived from milk proteins (ACE inhibitor peptide) and lipid components (conjugated linoleic acid). A number of harmful effects have been attributed to milk, often by groups with a vested interest, and often based on dubious data. We have investigated claims relating to diabetes, ischaemic heart disease and hypercholesterolaemia and been unable to substantiate any harmful effect. Designer milks that are improved raw materials can be approached through various combinations of genetics (including traditional genetics, marker-assisted selection and genetic modification of dairy cattle) and by farm and feed management. Examples are presented.

6. Caroli, A. M., S. Chessa and G. J. Erhardt. 2009. "Invited Review: Milk Protein Polymorphisms in Cattle: Effect on Animal Breeding and Human Nutrition." *Journal of Dairy Science* 92:5335-52 10.3168/jds.2009-2461.

[http://www.journalofdairyscience.org/article/S0022-0302\(09\)70865-3/fulltext](http://www.journalofdairyscience.org/article/S0022-0302(09)70865-3/fulltext).

7. Cattell, M.B., Nelsson, A.J. "[Beta-Casomorphin-7 and A1, A2 Milk "The Devil's in the Details."](#)" Presentation.
8. Chin-Dusting, Jaye, Jane Shennan, Emma Jones, Carolyn Williams, Bronwyn Kingwell and Anthony Dart. 2006. "Effect of Dietary Supplementation with B-Casein A1 Or A2 on Markers of Disease Development in Individuals at High Risk of Cardiovascular Disease." *British Journal of Nutrition* 95:136-44.

Abstract: The present study is the first to examine the hypothesis that dietary supplementation with \hat{I}^2 -casein A1 promotes an increased risk relative to supplementation with \hat{I}^2 -casein A2 in patients traditionally at high risk of developing CVD. The study was conducted in fifteen asymptomatic participants (six male; nine female) at high risk of developing CVD. A doubleblind cross-over study design was used with a total duration of 24 weeks. Dietary intervention was a daily supplementation (25g) of either casein A1 or A2 (for 12 weeks each). Surrogate measures of cardioprotection studied included the examination of vascular (endothelium and arterial) function, resting blood pressure, plasma lipids and biochemical markers of inflammation. Total plasma cholesterol levels were significantly lower following 12 weeks of both casein A1 and A2 interventions but the decrease was not different between intervention. Plasma insulin, homocysteine, C-reactive protein, fibrinogen, protein C and S and von Willebrand factor levels were not different between the two casein supplements. Endothelium function, measured as a vascular response using venous occlusion plethysmography to intra-arterial infusions of the endotheliumdependent agonist acetylcholine, were not different between the two casein interventions. Similarly, neither blood pressure nor measures of large artery stiffness were affected by differing the casein variant. We therefore conclude that there is no evidence from the present study that supplementation with casein A1 has any cardiovascular health disadvantage over consumption of casein A2.

9. De Noni, Ivan. FitzGerald, Richard J. Hannu J. T. Korhonen, Le Rouxm Y. , Chris T. 2009. "[SCIENTIFIC REPORT OF EFSA - Review of the Potential Health Impact of \$\beta\$ -Casomorphins and Related Peptides 1 Report of the DATEX Working Group on \$\beta\$ -Casomorphins.](#)" *European Food Safety Authority Scientific Report (231) 1 - 107*
10. Elliott, R. B., D. P. Harris, J. P. Hill, N. J. Bibby and H. E. Wasmuth. 1999. "Type I (Insulin-Dependent) Diabetes Mellitus and Cow Milk: Casein Variant Consumption." *Diabetologia* 42:292,296; 296.

Abstract: Previously published Type I (insulin-dependent) diabetes mellitus incidence in 0 to 14-year-old children from 10 countries or areas was compared with the national annual cow milk protein consumption. Countries which were selected for study had appropriate milk protein polymorphism studies, herd breed composition information and low dairy imports from other countries. Total protein consumption did not correlate with diabetes incidence ($r = + 0.402$), but consumption of the \hat{I}^2 -casein A1 variant did ($r = + 0.726$). Even more pronounced was the relation between \hat{I}^2 -casein (A1 + B) consumption and diabetes ($r = + 0.982$). These latter two cow caseins yield a bioactive peptide \hat{I}^2 -casomorphin-7 after in vitro digestion with intestinal enzymes whereas the common A2 variant or the corresponding human or goat caseins do not. \hat{I}^2 -casomorphin-7 has opioid properties including immunosuppression, which could account for the specificity of the relation between the consumption of some but not all \hat{I}^2 -casein variants and diabetes incidence.

11. Kaminski, S., A. Cieslinska and E. Kostyra. 2007. "Polymorphism of Bovine Beta-Casein and its Potential Effect on Human Health." *Journal of Applied Genetics* 48:189-98.

Abstract: Proteins in bovine milk are a common source of bioactive peptides. The peptides are released by the digestion of caseins and whey proteins. In vitro the bioactive peptide beta-casomorphin 7 (BCM-7) is yielded by the successive gastrointestinal proteolytic digestion of bovine beta-casein variants A1 and B, but this was not seen in variant A2. In hydrolysed milk with variant A1 of beta-casein, BCM-7 level is 4-fold higher than in A2 milk. Variants A1 and A2 of beta-casein are common among many dairy cattle breeds. A1 is the most frequent in Holstein-Friesian (0.310-0.660), Ayrshire (0.4320.720) and Red (0.710) cattle. In contrast, a high frequency of A2 is observed in Guernsey (0.880-0.970) and Jersey (0.490-0.721) cattle. BCM-7 may play a role in the aetiology of human diseases. Epidemiological evidence from New Zealand claims that consumption of beta-casein A1 is associated with higher national mortality rates from ischaemic heart disease. It seems that the populations that consume milk containing high levels of beta-casein A2 have a lower incidence of cardiovascular disease and type 1 diabetes. BCM-7 has also been suggested as a possible cause of sudden infant death syndrome. In addition, neurological disorders, such as autism and schizophrenia, seem to be associated with milk consumption and a higher level of BCM-7. Therefore, careful attention should be paid to that protein polymorphism, and deeper research is needed to verify the range and nature of its interactions with the human gastrointestinal tract and whole organism.

12. Knivsber, A. M., K. L. Reichelt and M. Nodland. 2001. "Reports on Dietary Intervention in Autistic Disorders." *Nutritional Neuroscience* 4:25-37.

Abstract: Autism is a developmental disorder for which no cure currently exists. Gluten and/or casein free diet has been implemented to reduce autistic behaviour, in addition to special education, since early in the eighties. Over the last twelve years various studies on this dietary intervention have been published in addition to anecdotal, parental reports. The scientific studies include both groups of participants as well as single cases, and beneficial results are reported in all, but one study. While some studies are based on urinary peptide abnormalities, others are not. The reported results are, however, more or less identical; reduction of autistic behaviour, increased social and communicative skills, and reappearance of autistic traits after the diet has been broken.

13. Kost, N. V., O. Y. Sokolov, O. B. Kurasova, A. D. Dmitriev, J. N. Tarakanova, M. V. Gabaeva, Y. A. Zolotarev, A. K. Dadayan, S. A. Grachev, E. V. Korneeva, I. G. Mikheeva and A. A. Zozulya. 2009. "Beta-Casomorphins-7 in Infants on Different Type of Feeding and Different Levels of Psychomotor Development" *Peptides* 30:1854-60.

Abstract: Casomorphins are the most important during the first year of life, when postnatal formation is most active and milk is the main source of both nutritive and biologically active material for infants. This study was conducted on a total of 90 infants, of which 37 were fed with breast milk and 53 were fed with formula containing cow milk. The study has firstly indicated substances with immunoreactivity of human (irHCM) and bovine (irBCM) beta-casomorphins-7 in blood plasma of naturally and artificially fed infants, respectively. irHCM and irBCM were detected both in the morning before feeding (basal level), and 3h after feeding. Elevation of irHCM and irBCM levels after feeding was detected mainly in infants in the first 3 months of life. Chromatographic characterization of the material with irBCM has demonstrated that it has the same molecular mass and polarity as synthetic bovine beta-casomorphin-7. The highest basal irHCM was observed in breast-fed infants with normal psychomotor development and muscle tone. In contrast, elevated basal irBCM was found in

formula-fed infants showing delay in psychomotor development and heightened muscle tone. Among formula-fed infants with normal development, the rate of this parameter directly correlated to basal irBCM. The data indicate that breast feeding has an advantage over artificial feeding for infants' development during the first year of life and support the hypothesis for deterioration of bovine casomorphin elimination as a risk factor for delay in psychomotor development and other diseases such as autism.

14. Luff, B. "[A2 – A Dilemma for the Guernsey Breed](#)". The World Guernsey Cattle Federation
15. Merriman, Tony R. 2009. "Type 1 Diabetes, the A1 Milk Hypothesis and Vitamin D Deficiency." *Diabetes Research and Clinical Practice* 83:149-56.

Abstract: The 'A1' genetic variant of β -casein in milk has been linked to type 1 diabetes (T1D). The keystone piece of supporting evidence is an ecological study positively correlating the incidence of T1D with amount of A1 β -casein consumption per capita. Of relevance, A1 β -casein consumption is also positively correlated with latitude, itself implicated in T1D through vitamin D deficiency. Ecological and biological evidence convincingly implicate vitamin D deficiency in T1D. Latitude is a confounder of the ecological data that underpin the hypothesis that A1 β -casein in cow's milk is a causative factor in T1D.

16. Michalski, Marie-Caroline. 2007. "On the Supposed Influence of Milk Homogenization on the Risk of CVD, Diabetes and Allergy." *British Journal of Nutrition* 97:598-610.

Abstract: Commercial milk is homogenized for the purpose of physical stability, thereby reducing fat droplet size and including caseins and some whey proteins at the droplet interface. This seems to result in a better digestibility than untreated milk. Various casein peptides and milk fat globule membrane (MFGM) proteins are reported to present either harmful (e.g. atherogenic) or beneficial bioactivity (e.g. hypotensive, anticarcinogenic and others). Homogenization might enhance either of these effects, but this remains controversial. The effect of homogenization has not been studied regarding the link between early cow's milk consumption and occurrence of type I diabetes in children prone to the disease and no link appears in the general population. Homogenization does not influence milk allergy and intolerance in allergic children and lactose-intolerant or milk-hypersensitive adults. The impact of homogenization, as well as heating and other treatments such as cheesemaking processes, on the health properties of milk and dairy products remains to be fully elucidated.

17. Muntoni, S. and S. Muntoni. 2006. "Epidemiological Association between some Dietary Habits and the Increasing Incidence of Type 1 Diabetes Worldwide." *Annals of Nutrition & Metabolism* 50:11-9.

Abstract BACKGROUND/AIMS: The variation in incidence of type 1 diabetes (T1D) worldwide is genetically based. However, its increasing incidence is environmentally determined. Our aim was to describe the role of nutritional habits and of gene-nutrient interactions in the rising incidence of T1D. METHODS: We did an ecological study in the 37 world areas where a 3% yearly increase of T1D incidence had been reported, and we calculated through the FAO's Food Balance Sheets the per caput daily supply of milk, meat and cereals from 1961 to 2000 and its correlation with the T1D incidence. RESULTS: The supply of milk and cereals remained almost unchanged, whereas that of meat increased by over 31%. The absolute mean T1D increase (number of cases per 100,000 per year) was + 0.32. A significant positive correlation with supply of milk was present from 1961 to 2000, while that with meat and cereals became significant in 1983 and 2000. CONCLUSION: Our ecological analysis indicates that nutritional factors, and in particular meat consumption, play a role in the incidence of T1D and its

increase worldwide. Further experimental and case-control studies are warranted in order to assess the gene-nutrient interactions.

18. Newswanger, J. 2009. "What to do with A2? A Review of the A2 Milk Controversy for Dexter Owners." <http://hoperefugefarm.com/archives/243>
19. Noni, Ivano. 2008. "Release of [Beta]-Casomorphins 5 and 7 during Simulated Gastro-Intestinal Digestion of Bovine [Beta]-Casein Variants and Milk-Based Infant Formulas." *Food Chemistry* 110:897-903.

Abstract The release of [beta]-casomorphin-5 (BCM5) and [beta]-casomorphin-7 (BCM7) was investigated during simulated gastro-intestinal digestion (SGID) of bovine [beta]-casein variants (n = 3), commercial milkbased infant formulas (n = 6) and experimental infant formulas (n = 3). SGID included pepsin digestion at pH 2.0, 3.0 and 4.0 and further hydrolysis with Corolase PP(TM). [beta]-Casein ([beta]-CN) variants were extracted from raw milks coming from cows of Holstein-Friesian and Jersey breeds. Genomic DNA was isolated from milk and the [beta]-CN genotype was determined by a PCR-based method. Phenotype at protein level was determined by capillary zone electrophoresis in order to ascertain the level of gene expression. Recognition and quantification of BCMs involved HPLC coupled to tandem MS. Regardless of the pH, BCM7 generated from variants A1 and B of [beta]-CN (5-176 mmol/mol casein) the highest amount being released during SGID of form B. As expected, the peptide was not released from variant A2 at any steps of SGID. BCM5 was not formed in hydrolysates irrespective of either the genetic variant or the pH value during SGID. Variants A1, A2 and B of [beta]-CN were present in all the commercial infant formulae (IFs) submitted to SGID. Accordingly, 16-297 nmol BCM7 were released from 800 ml IF, i.e. the daily recommended intake for infant. Industrial indirect-UHT treatments (156 °C × 6-9 s) did not modify release of BCM7 and, during SGID, comparable peptide amounts formed in raw formulation and final heat-treated IFs.

20. Scott, F. W. and H. Kolb. 2003. "A1 Beta-Casein Milk and Type 1 Diabetes: Causal Relationship Probed in Animal Models." *The New Zealand Medical Journal* 116:U368.
21. Smith, W. B., Deryn Thompson, M. Kummerow, Patrick Quinn and M. S. Gold. 2004. "A2 Milk is Allergenic." *Medical Journal of Australia* 181 / 10:574.

Abstract: Compared skin prick testing and Wheal diameter test reaction on children with milk allergies. Found no significant differences in reaction of children with A2/A2 or A1/A2 milk.

22. Sun, Zhongjie, Zhong Zhang, Xiuqing Wang, Robert Cade, Zaher Elmir and Melvin Fregly. 2003. "Relation of β -Casomorphin to Apnea in Sudden Infant Death Syndrome." *Peptides* 24:937-43.

Abstract: Sudden infant death syndrome (SIDS) is the most common cause of death in infants and its pathogenesis is complex and multifactorial. The aim of this review is to summarize recent novel findings regarding the possible association of β -casomorphin (β -CM) to apnea in SIDS, which has not been widely appreciated by pediatricians and scientists. β -CM is an exogenous bioactive peptide derived from casein, a major protein in milk and milk products, which has opioid activity. Mechanistically, circulation of this peptide into the infant's immature central nervous system might inhibit the respiratory center in the brainstem leading to apnea and death. This paper will review the possible relationship between β -CM and SIDS in the context of passage of β -CM

through the gastrointestinal tract and the blood-brain barrier (BBB), permeability of the BBB to peptides in infants, and characterization of the casomorphin system in the brain.

23. Swinburn, Boyd. 2004. "[Beta Casein A1 and A2 in Milk and Human Health: Report to New Zealand Food Safety Authority.](#)" *New Zealand Food Safety Authority*
24. Thorsdottir, Inga, Bryndis Birgisdottir, Inga Johannsdottir, Paul Harris, Jeremy Hill, Laufey Steingrimsdottir and Arni Thorsson. 1999. "Different Beta -Casein Fractions in Icelandic Versus Scandinavian Cow's Milk may Influence Diabetogenicity of Cow's Milk in Infancy and Explain Low Incidence of Insulin-Dependent Diabetes Mellitus in Iceland." *Pediatrics* 106:719-24.
25. Truswell, A. S. 2005. "The A2 Milk Case: A Critical Review." *European Journal of Clinical Nutrition* 59:623-31.

Abstract: This review outlines a hypothesis that A1 one of the common variants of β -casein, a major protein in cows milk could facilitate the immunological processes that lead to type I diabetes (DM-I). It was subsequently suggested that A1b-casein may also be a risk factor for coronary heart disease (CHD), based on between-country correlations of CHD mortality with estimated national consumption of A1b-casein in a selected number of developed countries. A company, A2 Corporation was set up in New Zealand in the late 1990s to test cows and market milk in several countries with only the A2 variant of β -casein, which appeared not to have the disadvantages of A1b-casein. The second part of this review is a critique of the A1/A2 hypothesis. For both DM-I and CHD, the between-country correlation method is shown to be unreliable and negated by recalculation with more countries and by prospective studies in individuals. The animal experiments with diabetes-prone rodents that supported the hypothesis about diabetes were not confirmed by larger, better standardised multicentre experiments. The single animal experiment supporting an A1b-casein and CHD link was small, short, in an unsuitable animal model and had other design weaknesses. The A1/A2 milk hypothesis was ingenious. If the scientific evidence had worked out it would have required huge adjustments in the world's dairy industries. This review concludes, however, that there is no convincing or even probable evidence that the A1 β -casein of cow milk has any adverse effect in humans. This review has been independent of examination of evidence related to A1 and A2 milk by the Australian and New Zealand food standard and food safety authorities, which have not published the evidence they have examined and the analysis of it. They stated in 2003 that no relationship has been established between A1 or A2 milk and diabetes, CHD or other diseases.

26. -----2006. "Reply: The A2 Milk Case: A Critical Review." *European Journal of Clinical Nutrition* 60:924-5.
27. Venn, B. J., C. M. Skeaff, R. Brown, J. I. Mann and T. J. Green. 2006. "A Comparison of the Effects of A1 and A2 β -Casein Protein Variants on Blood Cholesterol Concentrations in New Zealand Adults." *Atherosclerosis* 188:175-8.

Abstract: Casein is a cow's milk protein that occurs predominantly in two forms, A1 and A2. Epidemiological evidence suggests that per capita consumption of β -casein A1 is associated with national mortality rates from ischaemic heart disease. A biological mechanism was proposed after rabbits fed diets containing β -casein A2 had lower serum cholesterol concentrations and less aortic intimal thickening than rabbits fed β -casein A1. We tested whether β -casein A1 and A2 variants differentially affect plasma cholesterol concentrations in humans. In a randomised crossover trial of two four-and-a-half week periods without washout, 62 participants replaced all dairy

products in their diet with 500mL of low-fat milk and 28g of full-fat cheese that differed in the proportion of \hat{I}^2 -casein A1 and A2 variants. Duplicate blood samples were taken on non-consecutive days at the end of each treatment period from 55 people who completed the study. Mean (S.D.) plasma total, low-density and high-density lipoprotein cholesterol concentrations were 5.60 (0.77), 3.73 (0.70) and 1.26 (0.34)mmol/L after the A1 diet and 5.63 (0.81), 3.75 (0.75) and 1.27 (0.37)mmol/L after the A2 diets. We found no evidence that dairy products containing \hat{I}^2 -casein A1 or A2 exerted differential effects ($P>0.05$) on plasma cholesterol concentrations in humans.

28. Woodford, Keith. 2008. "A1 Beta-Casein, Type 1 Diabetes and Links to Other Modern Illnesses." *International Diabetes Federation Western Pacific Congress* <http://hdl.handle.net/10182/484> .
 29. ----- . "[Devil in the Milk: Illness, Health and Politics of A1 and A2 Milk](#)" Chelsea Green.
 30. Woodford, K. B. 2006. "A Critique of Truswell's A2 Milk Review." *European Journal of Clinical Nutrition* 60:437-9.
- List of more additional research on Beta-Caseins (some repetition). www.betacasein.net