

Opioids in Milk and Beef



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Submission: June 21, 2017; **Published:** September 06, 2017

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Abstract

It appears possible that the type of gene mutation that produces the opioid effect in milk and meat may also produce genetic abnormalities in the human genome leading to diseases such as autism, Alzheimer's, heart disease and other chronic debilities. The identification, isolation and subsequent modification of these genes would appear to be a step toward the eradication of the diseases associated with them. Most are familiar with organic milk as a healthier alternative to industrial (non-organic) milk that has been accused of being laden with antibiotics and stress hormones. Organic milk is essentially antibiotic and hormone free and healthier because the cows are fed grass or organically cultivated fodder. Still, it appears that the breed of cow may matter more than its feed. All milk is a great source of calcium and protein, but in this paper I'm going to write about A1 beta-casein and A2 beta-casein cow milk and what difference each makes. Our concern is an opioid, beta-casomorphine-7 (BCM-7) present in A1 cow milk. Cows producing A1 milk include breeds such as Holstein, Friesian, British Shorthorn and Ayrshire that originated in northern Europe. Cows producing A2 milk include such dairy breeds as Guernsey, Jersey and beef cattle breeds, Charolais and Limousin that developed in the Channel Islands and Southern France.

The percentage of A1 and A2 beta-casein protein varies between herds of cattle and also between countries. Between 46 percent and 70 percent of Holsteins and Ayrshires, most commonly found in the US, Australia, New Zealand, and Europe (excluding France) produce the A1 type beta-casein protein in their milk. A2 cow milk comes from the older cattle breeds such as desi Indian cows or African cows that produce protein in their milk with an amino acid called proline. In hybrid breeds, the proline amino acid mutated to histidine due to gene alteration thousands of years ago as cattle were being taken north into Europe. The proline at position 69 was replaced by histidine, with the mutation subsequently spreading widely throughout herds in the Western world through interbreeding. African and Asian cattle continue to produce primarily A2 beta-casein milk, and, on average, more than 70 percent of Guernsey and Jersey cows produce milk with predominantly A2 protein.

A1 and A2 Milk: What is the Difference?

Most industrial milk contains both A1 and A2 beta-casein, but A2 milk contains only A2 beta-casein. When A1 beta-casein is digested, it releases a peptide (protein fragment of a short chain of amino acids) called beta-casomorphin-7 (BCM-7), with seven amino acids at position 69 in its 209 peptide sequence that is an opioid found in a high percentage of milk produced by type A1 cows. BCM-7 is not active in A2 beta-casein because proline (an α -amino acid) is strongly bonded to the small protein fragment BCM-7, preventing it from being released into the milk produced by A2 cows [1]. On the other hand, histidine in A1 cows holds a weak bond with BCM-7, so it is easily released in the GI tract of animals and can enter the human body upon consumption of A1 milk and interact with the digestive system and circulation. There appears to be a high degree of correlation between A1 beta-casein and heart disease and diabetes, which has raised the possibility that the type of casein in the fresh milk supply could possibly be a risk factor. This is under investigation [2]. Interest in the distinction between A1 and A2 beta-casein proteins began in the early 1990s via epidemiological research and animal

studies. Initially conducted by scientists in New Zealand, they found correlations between the prevalence of milk with A1 beta-casein proteins in some countries and the prevalence of various chronic diseases in those countries [3]. The research generated interest in the media, among some in the scientific community and entrepreneurs. If it is indeed true that BCM-7 could harm humans, it would be an important public health issue as well as a commercial opportunity.

What Does All This Mean?

An emerging body of research suggests that many of the one in four Americans who exhibit symptoms of lactose intolerance could instead be unable to digest A1 beta-casein, most often found in milk from high-producing Holstein cows favored by American and some European industrial dairies [4]. A number of observations indicate that many people who cannot digest A1 milk are able to digest A2 milk. Surveys of A1 beta-casein consumption confirm the possibility that intensive dairy cattle breeding may have favored a genetic variant in milk with adverse effects in humans. Further animal research and clinical trials will

be needed to compare disease risks of A1-free versus ordinary milk. More than 100 studies suggest links between the A1 protein and a range of health conditions—from heart disease to diabetes to autism—though evidence to date is far from conclusive. Two observational studies have linked the consumption of A1 milk with an increased risk of heart disease [5]. One experiment in rabbits showed that consuming A1 beta-casein promoted fat buildup in injured blood vessels. This buildup was much lower when the rabbits consumed A2 beta-casein. Some theorize that peptides such as BCM-7 might play a role in the development of autism. One study of infants found higher levels of BCM-7 in those who were fed cow's milk, compared to those who were breastfed. However, studies do not support all of the proposed mechanisms. BCM-7 was strongly associated with an impaired ability to plan and perform actions, and another study suggested that drinking cow's milk could worsen behavioral symptoms in autistic children. Despite the suggested possibility, there is no conclusive evidence about the effects of A1 milk on autism, and the issue needs to be studied further.

Concern among medical ethicists and investors about such research seems unjustified. Review of published research in this current study reveals that there are no conclusive claims made about milk and health risks. All statements are conditioned by the caveat that “the evidence to date is far from conclusive.” Several commercial dairymen and distributors were questioned about the issue of A1 and A2 milk, and it was discovered that financing of almost all dairy associations are, like the financing of medical associations and legal associations, non-profit and solely for promotion of interests of the industry. No evidence of federal funding was found. What started my research in genetic mutations in cows leading to different types of milk was my interest and research in diseases possibly caused by or related to genetic chromosomal mutations. My first involvement in this type of research was in 1965, 66 and 67 when I was researching my book “Ophthalmic Eponyms: An Encyclopedia of Named Signs, Syndromes and Diseases in Ophthalmology”, published in 1967 by Aesculapius Publishing Company in Birmingham, Alabama. In this book I described more than 200 syndromes and diseases that were familial and possibly the result of genetic mutations. Examples include Adies Syndrome, Albers-Schönberg Disease, Albright's Syndrome, Alport's Syndrome, Alström-Hallgren Syndrome, Alzheimer's Disease, Amalric's Syndrome, Andogski's Syndrome, Angelvecci's Syndrome, Apert's Syndrome, Aubineau-Lenoble Syndrome, Axenfeld-Schüremberg Syndrome, Berdet-Biedl Syndrome, Barlenwerfer's Syndrome, Basedow's Disease, Bassen-Kornzweig Syndrome, Batten-Mayor Syndrome, Behr's Disease, Benjamin's Syndrome, Best's Macular Degeneration, Biber-Haab-Dimmer Corneal Dystrophy,

Bielschowsky-Jansky Syndrome, Bieman's Syndrome, and many more from A to Z. My current study began as a search for possible genetic links for cures to diseases such as Alzheimer's, Autism, diabetes, heart disease and other diseases of aging, and to identify diseases and syndromes that, with today's advanced technology, might possibly respond to deliberate alteration in genes, providing a cure or amelioration [6]. What I discovered was a number of studies linking, not only genetic connections to abnormal conditions in humans, but also to genetic alterations in other animals such as cattle. I discovered articles on genetic alterations in proteins in milk, serum, hormones and enzymes. Some of these appear to have the potential of affecting brain function, reproduction, and resistance to disease. The evidence suggests the possibility that diseases such as Alzheimer's, type 2 diabetes and other degenerative conditions may be traced to mutations in genes. It would follow that with current science one could identify the abnormal gene characteristic of conditions such as Alzheimer's and replace it with normal genes, effecting cure or amelioration [7]. Tobacco and cancer research and its opposition by the tobacco industry over the past six or seven decades seem similar to the current dairy industry reaction to this current research.

Conclusion

The facts will survive, and research will lead to cures, and the public health will be better served as a result. With the recognition of the potential for gene modification the potential for familial disease prevention may be realized.

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DOI: [10.19080/JOJO.2017.04.555638](https://doi.org/10.19080/JOJO.2017.04.555638)

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