Milk A1 and A2 Peptides and Diabetes

Roger A. Clemens

Nearly two decades ago, some food-derived peptides, specifically those derived from milk, were suggested to present adverse effects on health by increasing the risk of insulin-dependent diabetes. This position was based on the apparent relationship of T1D and the consumption of variants A1 and B β-casein from cow’s milk. Researchers noted that β-casomorphin (BCM-7) from β-casein may function as an immunosuppressant and impair tolerance to dietary antigens in the gut immune system, which may contribute to the onset of T1D. However, over the past decade numerous international clinical studies have not supported that association.

There are 13 genetic variants of β-casein in dairy cattle. Among those variants are A1, A2, and B, which are also found in human milk. The amino acid sequences of β-casomorphins in these bovine variants and those found in human milk are similar, often differing only by a single amino acid. In in vitro studies, BCM-7 could be produced from A1 and B during typical digestive processes; however, BCM-7 is not a product of A2 digestion. These in vitro observations were inconsistent following digestion in the gut of humans, and quantification of these hydrolytic products has not been reported. However, depending on the proteolytic systems applied in milk fermentation and cheese production, BCM-7 can be produced [1].

Four prospective studies indicated that the consumption of low-fat dairy products was associated with a decreased risk of type 2 diabetes, an adult-onset disease. These conclusions were supported by the most recent review by the European Food Safety Authority (EFSA) [2]. Upon examination of an array of environmental and genetic factors that contribute to diabetes and immunological responses to cow’s milk proteins, particularly β-casomorphin 7, the EFSA stated that the suggested milk protein and T1D link remains unclear, and the implications of the variants difficult to interpret.
Additional evidence against the role of milk proteins, even breastfeeding, and the development of T1D was provided through epidemiological studies and animal models [3]. Other reports argue the A1 variant of β-casein is diabetogenic [4]. These reports advance biological data indicating A1 β-casein antibody titers were greater among T1D patients vs. A2 β-casein titers, and the differences were significant when compared to controls and parents. However, it appears that the antibody titers were not specific to T1D. On the other hand, ecological data, primarily based on A1/A2 variations among livestock breeds, do not demonstrate causation, even in the countries where there is considerable dairy consumption.

Many hypotheses have been made regarding the etiology and apparent increase in T1D, particularly in developed countries. Our diets contain a broad range of biologically active peptides. Dairy products provide an array of nutrients necessary for normal growth and development of children and adolescents. Yet, the dairy debate continues with respect to an apparent T1D and BCM-7 association.

References