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Milk-Derived Proteins and Digestive Wellness

Rita Sandhu[1] and *Balraj Sandhu[2]
[1]Assistant Professor, Department of Dairy Chemistry, CDST, RAJUVAS, Bikaner, Rajasthan, India
[2]M.Tech, Geotechnical Engineering, Bikaner Technical University, Bikaner (Rajasthan)
***Corresponding Author's email: balrajsandhu421@gmail.com**

Milk is a cornerstone of human nutrition, supplying not only energy but also high-quality proteins, essential amino acids, and bioactive compounds that exert physiological effects beyond basic nourishment. Proteins make up approximately 3–3.5% of bovine milk and are broadly divided into two categories: caseins (≈80%) and whey proteins (≈20%) (Farrell et al., 2004). Both fractions contribute to nutrition and health but differ in structure, functionality, and digestive fate. Caseins, particularly β-casein, play a central role in micelle formation, mineral binding, and calcium–phosphate transport. During gastrointestinal digestion, caseins are hydrolyzed into a wide range of peptides, some of which possess biological activities relevant to gut function. Whey proteins, including β-lactoglobulin, α-lactalbumin, lactoferrin, and immunoglobulins, are structurally more globular and soluble. Their enzymatic breakdown generates peptides with antimicrobial, antioxidant, immunomodulatory, and even antihypertensive properties (Korhonen & Pihlanto, 2006).

Among caseins, β-casein is especially notable due to its genetic polymorphisms. Over a dozen β-casein variants are known, but the A1 and A2 types dominate scientific and commercial interest (Kaminski et al., 2007). The structural difference between these variants lies in a single amino acid substitution: histidine at position 67 in A1 β-casein replaces proline in A2. This seemingly minor change significantly alters proteolysis and leads to differential release of bioactive peptides. Specifically, A1 β-casein is more susceptible to cleavage, producing β-casomorphin-7 (BCM-7), an opioid-like peptide (Jinsmaa & Yoshikawa, 1999). BCM-7 has been shown to bind μ-opioid receptors in the gastrointestinal tract, where it can influence gut motility, mucus secretion, epithelial permeability, and immune signaling (Sienkiewicz-Szłapka et al., 2009). Clinical evidence suggests that the release of BCM-7 may help explain why some individuals experience gastrointestinal symptoms after consuming A1 milk but not A2. Controlled trials report that A1 milk consumption is associated with abdominal pain, bloating, delayed transit, and inflammatory responses, whereas A2 milk is generally better tolerated (Ho et al., 2014; Jianqin et al., 2016; He et al., 2017). Strikingly, individuals with self-reported lactose intolerance also report fewer symptoms when consuming A2 milk, despite identical lactose content. This observation points toward the role of protein-derived peptides—rather than lactose alone—in driving digestive discomfort.

Beyond the A1/A2 distinction, milk proteins exert broader influences on gut microbial ecology. The digestion of caseins and whey proteins yields peptides and amino acids that serve as substrates for microbial metabolism. These substrates can promote the growth of beneficial bacteria such as Bifidobacterium and Lactobacillus, enhancing mucosal immunity and producing health-promoting short-chain fatty acids (SCFAs) like butyrate (Davila et al., 2013). However, under certain conditions, excessive proteolysis may favor proteolytic or pathogenic bacteria, generating ammonia, phenolic compounds, and other metabolites linked to intestinal inflammation. Thus, milk proteins are not passive nutrients but active modulators

of gastrointestinal health. Their effects are mediated through structural polymorphisms, peptide release, and interactions with both the host and the gut microbiota. Understanding these processes has direct dietary and public health relevance, particularly in distinguishing lactose intolerance from protein sensitivity and in guiding the development of functional dairy products tailored for gut health.

Milk Proteins and β-Casein Variants: Molecular Basis and Distribution

Milk proteins, encompassing caseins and whey proteins, form the cornerstone of milk's nutritional and functional properties. Together, they not only provide a complete source of essential amino acids but also contribute to numerous physiological processes through their digestion products. Caseins constitute about 80% of total milk protein, with β-casein alone representing nearly one-third of this fraction. Caseins are characterized by their open, flexible structure and their ability to self-associate into micelles, which serve as vehicles for calcium and phosphate transport (Farrell et al., 2004). In addition to these structural and nutritional roles, caseins are a major source of bioactive peptides that exert antimicrobial, immunomodulatory, antioxidant, and gut-regulatory effects following proteolysis.

Molecular Basis of β-Casein Variants

Among the casein family, β-casein is notable for its polymorphism. The β-casein gene (CSN2) exhibits high allelic diversity, with over a dozen genetic variants described to date (Farrell et al., 2004). Of these, the A1 and A2 variants have attracted the greatest attention due to their distinct physiological implications. The difference arises from a single nucleotide polymorphism leading to the substitution of histidine in A1 for proline in A2 at position 67 of the amino acid chain (Kaminski et al., 2007). While seemingly subtle, this variation has major consequences for the peptide bonds' stability during gastrointestinal digestion.

The proline residue in A2 β-casein confers conformational rigidity, which restricts proteolytic cleavage at the 66–67 bond. By contrast, histidine in A1 reduces steric hindrance, making the peptide bond more susceptible to enzymatic hydrolysis. As a result, A1 β-casein is more likely to release β-casomorphin-7 (BCM-7), an opioid peptide fragment, during digestion (Jinsmaa & Yoshikawa, 1999). This difference highlights the profound effect that a single amino acid substitution can have on digestion outcomes and, subsequently, on host physiology.

Functional Implications of BCM-7 Release

BCM-7 possesses affinity for μ-opioid receptors expressed throughout the gastrointestinal tract. Its binding has been implicated in modulation of intestinal motility, mucin secretion, and epithelial barrier integrity (Sienkiewicz-Szłapka et al., 2009). Furthermore, BCM-7 may activate pro-inflammatory pathways, influencing cytokine production and gut immune responses. Although the extent of BCM-7 release in vivo depends on factors such as enzyme activity and gut transit time, evidence from animal models and human studies suggests that A1 β-casein consumption is associated with gastrointestinal discomfort, delayed transit, and mild inflammatory changes. Conversely, A2 β-casein, which releases little to no BCM-7, is often better tolerated (Ho et al., 2014; Jianqin et al., 2016; He et al., 2017).

Distribution of β-Casein Variants in Dairy Cattle

The occurrence of A1 and A2 β-casein alleles is unevenly distributed across cattle breeds, reflecting both evolutionary divergence and selective breeding practices. Holstein-Friesian cattle, which dominate commercial dairy herds in North America and Europe, typically carry a mixture of A1 and A2 alleles, resulting in milk with variable proportions of the two variants. In contrast, traditional breeds such as Jersey and Guernsey are enriched in the A2 allele, often producing predominantly A2 milk (Truswell, 2017). Similarly, Asian Zebu breeds, including Gir and Sahiwal cattle, are natural sources of A2-type milk, which has contributed to the interest in their conservation and selective breeding in regions such as India.

β-Casein in Non-Bovine Species

Interestingly, the A2 variant is not unique to certain cattle breeds but is also characteristic of milk from non-bovine species. Goats, sheep, and camels naturally produce β-casein proteins that closely resemble the A2 structure, lacking the histidine substitution associated with A1 (Lonnerdal, 2016). This structural similarity may explain the long-standing perception that milk from these species is more easily digestible and less likely to cause discomfort in sensitive individuals. Camel milk, in particular, is increasingly marketed for its digestibility and potential therapeutic benefits, partly attributable to its A2-like β-casein profile and distinct whey protein composition.

Implications for Human Health and Dairy Production

The distribution of β-casein variants carries implications for both dairy production and public health. From a production standpoint, the rising consumer demand for A2 milk has led to genetic testing and selective breeding programs aimed at increasing the prevalence of A2 allele carriers in dairy herds. This has not only commercial implications but also potential nutritional benefits, especially in populations with high prevalence of self-reported lactose intolerance, where A2 milk often provides better tolerance despite identical lactose content.

From a health perspective, the A1/A2 distinction underscores the importance of protein structure in shaping digestive outcomes. While lactose has long been blamed for post-dairy gastrointestinal symptoms, emerging evidence suggests that milk protein polymorphisms also play a significant role. Understanding the molecular basis and breed distribution of β-casein variants is therefore essential for developing evidence-based dietary guidelines and for designing functional dairy products tailored to support gut health.

Digestion of Milk Proteins and Release of Bioactive Peptides

Milk proteins undergo extensive proteolysis during gastrointestinal digestion, giving rise to a wide range of bioactive peptides that can influence gut function and overall health. Caseins are particularly susceptible to enzymatic cleavage by gastric and pancreatic enzymes, producing peptides with antimicrobial, immunomodulatory, antioxidant, and opioid-like activities (Korhonen & Pihlanto, 2006). Whey proteins, such as β-lactoglobulin and α-lactalbumin, also yield peptides with demonstrated roles in gut immunity and microbial balance. Among these peptides, β-casomorphins have attracted significant attention because of their interaction with the gastrointestinal tract. The most studied is β-casomorphin-7 (BCM-7), an opioid peptide released primarily from the A1 variant of β-casein. The substitution of histidine at position 67 in A1 increases susceptibility to enzymatic cleavage at the 66–67 bond, promoting BCM-7 release (Jinsmaa & Yoshikawa, 1999). By contrast, the proline residue in A2 β-casein reduces this cleavage, resulting in minimal BCM-7 formation (De Noni & Cattaneo, 2010).

Once generated, BCM-7 can bind to μ-opioid receptors on enteric neurons and epithelial cells, modulating gut motility, mucin secretion, epithelial barrier integrity, and immune activity (Sienkiewicz-Szłapka et al., 2009). Normally, brush-border enzymes such as dipeptidyl peptidase-IV (DPP-IV) degrade BCM-7 into inactive fragments. However, individual differences in enzyme activity, gut transit time, and microbiota composition may affect the extent of BCM-7 breakdown (Ul Haq et al., 2014).

Experimental evidence supports the biological activity of milk-derived peptides in the gut. Animal studies demonstrate that BCM-7 and A1 β-casein ingestion can delay intestinal transit and induce mild mucosal inflammation (Barnett et al., 2014). In vitro studies suggest that BCM-7 activates NF-κB–mediated pro-inflammatory pathways, elevating cytokines such as IL-4, IL-6, and TNF-α (Sienkiewicz-Szłapka et al., 2009). Beyond β-casomorphins, other casein- and whey-derived peptides have been shown to modulate microbial growth, enhance mucosal defense, and influence gut immunity. Together, these findings illustrate how milk protein digestion is not merely a process of nutrient release but a key driver of gut physiology. The generation of bioactive peptides, particularly BCM-7 from A1 β-casein,

highlights the potential for differential gastrointestinal effects depending on milk protein variants, enzyme activity, and host–microbiota interactions.

Gastrointestinal Symptoms: Evidence from Human Studies

The clinical evidence on A1 versus A2 β-casein primarily centers on their differential effects on gastrointestinal tolerance. Several randomized controlled trials (RCTs) and cross-over studies have assessed symptoms such as abdominal pain, bloating, stool consistency, and bowel transit time in individuals consuming A1- or A2-dominant milk. A landmark study in Chinese adults with self-reported milk intolerance demonstrated that participants consuming A1-containing milk reported significantly higher levels of abdominal pain, bloating, and stool irregularity compared to those who consumed A2-only milk (Jianqin et al., 2016). Importantly, these effects were observed even though lactose content was identical in both test milks, suggesting that β-casein variants, rather than lactose, contributed to the differential response.

Ho et al. (2014) reported that healthy adults consuming A1 milk experienced slower gastrointestinal transit and increased stool firmness compared to when they consumed A2 milk. He et al. (2017) further supported these findings, showing that A1 milk consumption was associated with higher levels of gastrointestinal inflammation markers, including faecal calprotectin and myeloperoxidase, along with self-reported discomfort. Meta-analyses remain limited due to small sample sizes and heterogeneity in study designs, but overall, clinical evidence suggests that A1 milk may exacerbate gastrointestinal symptoms in sensitive populations, while A2 milk tends to be better tolerated (Brooke-Taylor et al., 2017). These effects are especially relevant for individuals who experience “milk intolerance” but test negative for lactose malabsorption, as protein variant sensitivity may play a role.

Distinguishing Lactose Intolerance from A1/A2 Sensitivity

A critical issue in the A1/A2 debate is the overlap of symptoms between lactose intolerance and protein variant sensitivity. Both conditions can present with bloating, abdominal pain, flatulence, and diarrhea, which complicates clinical differentiation. However, the pathophysiological mechanisms are distinct. Lactose intolerance arises from insufficient activity of the enzyme lactase-phlorizin hydrolase, resulting in undigested lactose passing into the colon where it is fermented by microbiota, producing gas and osmotic diarrhea (Misselwitz et al., 2019). In contrast, A1 β-casein sensitivity is related to the proteolytic release of BCM-7 and its interaction with opioid receptors, which may slow transit, alter barrier integrity, and trigger immune activation (Ul Haq et al., 2014). Several clinical trials have highlighted this distinction. Jianqin et al. (2016) showed that individuals with confirmed lactose maldigestion experienced fewer symptoms when consuming A2 milk compared to A1 milk, despite identical lactose loads. Similarly, Chao et al. (2024) noted that self-reported lactose-intolerant individuals could tolerate A2 milk significantly better, indicating that β-casein polymorphisms may contribute to symptom expression independent of lactose digestion.

From a practical perspective, this means that some individuals who believe they are lactose intolerant may, in fact, be sensitive to A1 β-casein. This distinction is important for dietary management, as it opens alternatives beyond lactose-free milk, including the use of A2-only milk or non-bovine milks (goat, sheep, camel) that predominantly contain A2-like β-casein.

Gut Microbiota Modulation by A1 and A2 Milk

Emerging research suggests that the impact of A1 and A2 β-casein extends beyond digestion and immediate gastrointestinal symptoms, influencing the gut microbiota composition and activity. The gut microbiome is central to host health, regulating immunity, nutrient metabolism, and intestinal barrier integrity. Dietary proteins, including milk proteins, serve as substrates for microbial fermentation, and the peptides released during digestion can shape microbial communities (Davila et al., 2013). Animal studies provide important insights.

Barnett et al. (2014) reported that A1 β-casein consumption in rodents was associated with increased intestinal inflammation and altered microbial populations, including a reduction in beneficial Bifidobacteria. In contrast, A2 β-casein diets preserved microbiota balance and maintained mucosal integrity. In pigs, A1 milk intake resulted in changes to short-chain fatty acid (SCFA) profiles, particularly a reduction in butyrate, a metabolite critical for colonocyte health and anti-inflammatory signaling (Chao et al., 2019).

Human evidence is more limited but promising. He et al. (2017) observed that subjects consuming A1 milk exhibited elevated levels of pro-inflammatory gut metabolites and microbial dysbiosis markers compared to those consuming A2 milk. Moreover, differences in gut transit time between A1 and A2 consumers (Ho et al., 2014) may indirectly shape microbial fermentation patterns by altering substrate availability. Overall, while more longitudinal human studies are required, current evidence suggests that A2 milk may promote a healthier gut microbiota environment, whereas A1 milk could predispose individuals to dysbiosis and gut inflammation.

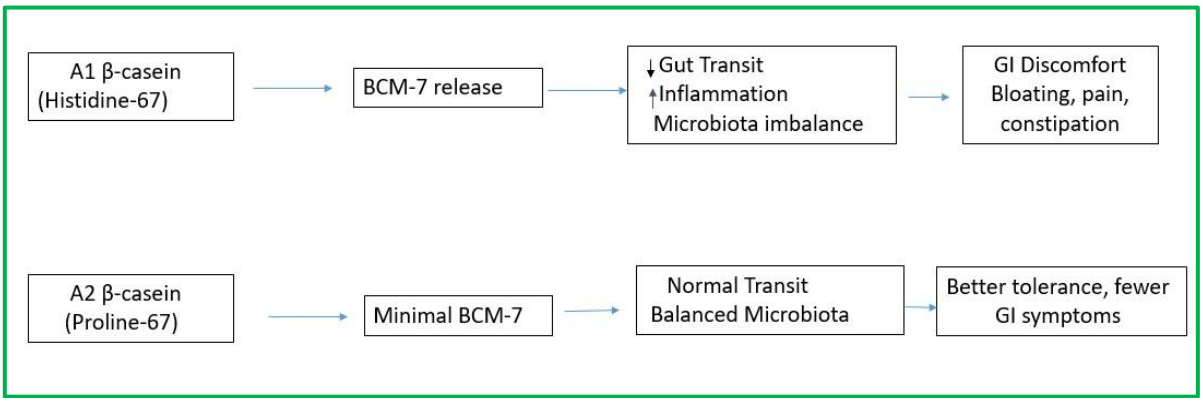

Diagram: A1 vs A2 milk digestion and gut effects

Population and Species Differences (Cow, Goat, Sheep, Camel Milk)

The health implications of A1 and A2 milk are also shaped by population genetics, dietary habits, and species-specific milk composition. Globally, the distribution of A1 and A2 alleles in cattle varies. European Holsteins and Friesians typically produce mixed A1/A2 milk, while Jersey and Guernsey breeds have a higher proportion of A2 alleles (Truswell, 2017).Indigenous Asian and African Zebu cattle predominantly produce A2 milk, which aligns with anecdotal reports of better digestibility of milk in these populations (Lonnerdal, 2016).

Beyond bovine milk, non-bovine species naturally produce A2-like β-casein. Goat and sheep milk contain proline at position 67, resembling sA2, and are often perceived as gentler on digestion, especially in children and elderly populations (Park et al., 2007). Camel milk, which also has A2-type β-casein, is gaining attention due to reports of improved tolerance in lactose-intolerant individuals and additional therapeutic properties such as anti-diabetic and immunomodulatory effects (Al-Saleh et al., 2013). From a population health perspective, the prevalence of lactose intolerance is high in Asia and Africa, but sensitivity to A1 β-casein may represent an additional layer of dietary intolerance. Thus, distinguishing between lactose malabsorption and A1 sensitivity could have important implications for nutritional strategies in these regions.

Feature	A1 β-casein	A2 β-casein
Amino acid at position 67	Histidine	Proline
Susceptibility to proteolytic cleavage	High (favours BCM-7 release)	Low (resistant to BCM-7 release)
Major bioactive peptide generated	β-casomorphin-7 (BCM-7)	Minimal BCM-7
BCM-7 activity	Opioid activity, delayed transit, increased mucus, pro-inflammatory signaling	Negligible

Gastrointestinal effects	Slower gut transit, firmer stools, abdominal pain, bloating in sensitive individuals	Better tolerance, fewer symptoms
Microbiota impact	Potential dysbiosis, reduced Bifidobacteria, altered SCFA profile	Preserves microbial balance
Population relevance	Predominant in Holstein-Friesian milk, common in Western diets	Common in Jerseys, Guernsey, Zebu, and all non-bovine species (goat, sheep, camel)

Controversies and Public Health Perspectives

The debate over A1 and A2 milk is not without controversy. On one hand, advocates argue that A2 milk offers improved digestibility and may reduce gastrointestinal discomfort in sensitive populations. On the other hand, critics highlight the limited scope and small sample sizes of existing clinical studies, cautioning against broad generalizations. Regulatory bodies such as the European Food Safety Authority (EFSA) and the Food Standards Australia New Zealand (FSANZ) have reviewed the evidence and concluded that, while there is some mechanistic plausibility, current data are insufficient to confirm direct health risks associated with A1 milk (EFSA, 2009; Truswell, 2017). Commercial and marketing dynamics further complicate the issue. The rise of A2-only branded milk has created economic opportunities for farmers and dairy industries, particularly in regions where consumer demand for "digestive-friendly" milk is growing. However, questions remain about whether these benefits extend beyond niche populations with gut sensitivity. Moreover, conflating lactose intolerance with A1 sensitivity in public communication risks consumer confusion.

From a public health standpoint, the nutritional value of milk remains high regardless of variant. Both A1 and A2 milks are rich in proteins, calcium, and micronutrients. However, for individuals with gastrointestinal discomfort, A2 milk or non-bovine alternatives (goat, sheep, camel) may serve as practical dietary substitutes without the need to completely exclude dairy.

Future Directions and Research Needs

Despite growing consumer interest and emerging evidence, several gaps remain in our understanding of A1 and A2 milk. First, large-scale, long-term clinical trials are needed to confirm the effects of β-casein variants on gut health and systemic outcomes. Existing studies are often short-term and limited to self-reported symptoms. Second, the role of gut microbiota as a mediator of A1/A2 effects requires deeper investigation using high-throughput sequencing and metabolomics.

Third, research should extend beyond gastrointestinal outcomes to explore potential associations between A1 β-casein consumption and neurological or autoimmune conditions, where BCM-7's opioid and immunomodulatory properties may be relevant (Bell et al., 2006). However, such links remain speculative and controversial.

Finally, breeding strategies and dairy industry practices may evolve as consumer demand for A2 milk rises. Understanding the genetic distribution of A1 and A2 alleles in different cattle populations and their impact on milk yield and processing properties will be critical for sustainable production.

Conclusion

The A1 versus A2 β-casein debate underscores the complexity of milk as more than a simple nutrient source. The substitution of a single amino acid in β-casein has far-reaching implications for protein digestion, peptide release, and gastrointestinal physiology. Evidence from mechanistic studies, animal experiments, and human clinical trials suggests that A1 milk, via BCM-7 release, may slow intestinal transit, promote inflammation, and contribute to gut discomfort, while A2 milk is generally better tolerated.

Although not universally accepted, the possibility that some cases of "milk intolerance" may be due to A1 protein sensitivity rather than lactose malabsorption has important dietary and clinical implications. A2 milk and non-bovine milks may therefore

represent suitable alternatives for individuals with gastrointestinal symptoms. Continued research is required to resolve controversies, clarify mechanisms, and inform public health recommendations. Until then, the A1/A2 distinction highlights the intricate interplay between food genetics, digestion, and human health, offering both challenges and opportunities for nutrition science and the dairy industry.

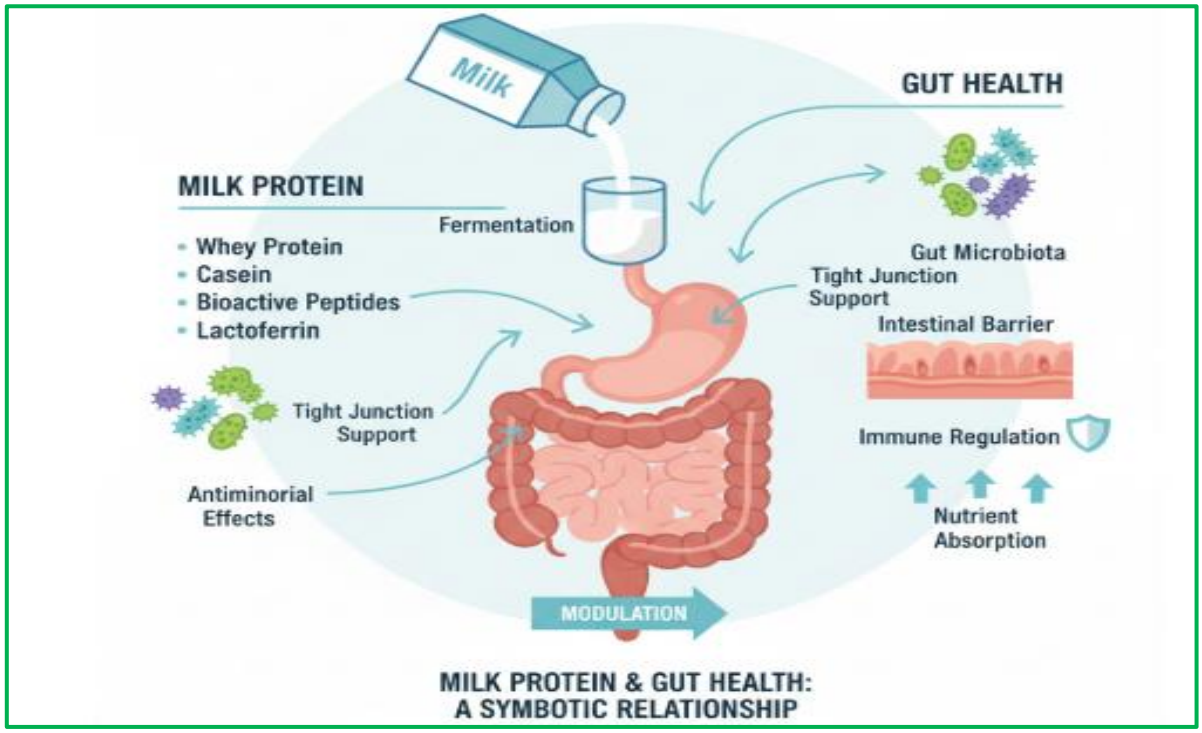

Figure 1: Milk protein and gut health

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