Milk Proteins in the Regulation of Bodyweight, Satiety, Food Intake and Glycemia

G. Harvey Anderson, Bohdan Luhovyy, Tina Akhavan and Shirin Panahi

Several epidemiological studies of adults have reported an inverse association between frequent dairy intake and adiposity [1, 2]. Dietary patterns characterized by higher dairy consumption have also been associated with lower prevalence of insulin resistance, risk of type 2 diabetes and cardiovascular disease among overweight adults [2–4]. Although the reasons for the observed benefits of increased dairy consumption and lower prevalence of obesity and chronic diseases remain unclear, the physiologic actions of their proteins, casein and whey, beyond providing essential amino acids for protein synthesis, have been offered as an explanation [1–3].

Dairy products and their milk proteins increase satiety and reduce food intake and blood glucose response when consumed alone or with carbohydrate. Milk proteins are also more satiating than either carbohydrate or fat [1]. This can be explained by the effects of the multiple interactions between milk proteins and their products during digestion on regulatory systems controlling food intake and metabolism [5]. These effects arise from the combined actions of the intact protein, encrypted peptides and amino acids, and can be achieved within the range of usual consumption of dairy products.

There is a long history of the study of intake regulation based on amino acid-sensing systems in the brain. In addition, a role for bioactive peptides (BAPs) has been identified in the reduction of food intake associated with protein ingestion. BAPs stimulate satiety hormone receptors in the gut in rats, while milk proteins potentiate release of many satiety hormones. In addition, mixtures of whey protein-derived amino acids fail to reproduce the effects of intact whey protein on metabolism and satiety hormones [1, 4, 5].
Although proteins are known to be insulinotropic [1–3], recent research shows that metabolic control is not solely dependent on their insulinotropic effects [5]. Small amounts of protein, as low as 10 g [5], or 2 cups of milk [unpubl.] consumed shortly before a meal, reduced the glycemic response to a mixed meal containing carbohydrate, protein and fat. With increasing doses of whey protein, a corresponding decrease in blood glucose response occurred after a fixed size meal. This occurred in the presence of a lower, not higher,
postmeal insulin area under the curve (AUC) and with similar cumulative (0–170 min) insulin AUC (table 1). When the cumulative AUC for blood glucose was divided by the cumulative AUC for insulin to evaluate the efficacy of insulin action, the ratio was decreased, in a dose-dependent manner, to 50% of the control after premeal consumption of intact whey protein of 40 g (table 2). In contrast to 10 g of intact protein, 10 g of whey protein hydrolysate did not result in a lower cumulative blood glucose than the control, even though it increased the postmeal and cumulative insulin AUC similarly, suggesting that noninsulinotropic mechanisms require stimulation arising from the digestion of intact proteins, perhaps due to the release of encrypted BAPs.

Thus, it can be suggested that physiological responses to dairy protein ingestion support the epidemiologic observations and suggest that milk products and proteins have application in strategies aimed at controlling the metabolic consequences of obesity.

### References


