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Can the controversial relationship between dietary calcium and body weight be mechanistically explained by alterations in appetite and food intake?

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Abstract

The prevalence of obesity has reached epidemic proportions worldwide and the incidence of overweight and obesity continues to rise. Diet plays a significant role in the modulation of body weight and there is some evidence to suggest that calcium or dairy intake may modulate body weight and body fat mass. Several mechanisms through which calcium or dairy products may affect body weight or fat have been suggested, including a possible effect on appetite and food intake. A recent study investigated to what extent people could compensate for increased energy intake from dairy products and found that a 7-day increase in dairy intake had no effect on appetite and no evidence of complete compensation for the raised energy intake. In another study, the effects of altered calcium content of a dairy-based test meal was evaluated in obese subjects; the findings indicated that although a higher calcium content of the meal reduced the extent of post-prandial chylomicron-associated triglyceridemia, there was no effect on appetite-related hormones (CCK, ghrelin, GLP-1, or PYY) or on energy intake from a subsequent *ad libitum* test meal. Thus, this new evidence does not support the hypothesis that high calcium or dairy intake reduces appetite or food intake.

Keywords

appetite; body weight; dairy; dietary calcium; fat mass; food intake

INTRODUCTION

The regulation of food intake is important for maintaining a healthy body weight and reducing the risk of disease. The incidence of obesity continues to grow worldwide. Efforts to identify the macronutrient composition of diets effective in reducing overall energy intake and fat mass is an active area of research and public interest. In addition to the role of the levels of protein, fat, and carbohydrate in the diet on weight gain and body composition, there has been interest in the possibility that the level of dietary calcium can influence body weight and fat mass; consequent efforts have been made to understand the mechanism of these dietary effects. One suggested hypothesis is that the level of calcium in the diet may affect appetite and food intake.

The concept that non-energy-supplying nutrients, such as calcium, may regulate food intake or appetite seems counterintuitive. One suggestion is that mammals may have evolved to respond to specific dietary components that act as indicators of dietary availability, so that, when food is plentiful, a mineral present in the diet (e.g., calcium) acts as the signal to reduce body fat mass accumulation. On the other hand, when food is scarce, the lower level of calcium in the diet may serve as an indicator to promote fat mass accumulation, protecting the body against the scarcity of food. Therefore, teleologically, dietary calcium content may function as a marker for energy nutrient availability and thus regulate physiological processes and food intake to appropriately respond to the energy availability in the environment.

The mechanism by which dietary intake of calcium may regulate appetite and food intake is unknown. Calcium may participate in eliciting hormonal responses that impact these physiological functions. It could, therefore, be instructive to measure the response of mediators of food intake, e.g., ghrelin, cholecystokinin (CCK), peptide YY (PYY), and glucagon-like peptide-1 (GLP-1), to calcium intake. Obviously, the regulation of food intake is a complex interaction of not only hormonal and neuronal controls, but also of learned behaviors and environmental influences that may override the natural cues controlling food intake.

IMPACT OF CALCIUM INTAKE ON APPETITE AND FOOD INTAKE

Dairy versus non-dairy sources of calcium - a complicating factor—A

complicating factor that makes the interpretation of many of these studies difficult in the critical analysis of calcium consumption and body composition is that the primary source of calcium is dairy products. Independent from its calcium content, dairy protein (a macronutrient having high satiety properties¹) and other dairy components (e.g., caseinomacropptide or CMP)² have been implicated in regulating body composition and food intake or affecting mediators of food intake.

Calcium, body weight, and fat mass—In addition to the well-established role of calcium in bone and mineral metabolism, recent studies suggest that high calcium intake may lead to reduced body fat accumulation or enhance fat mass loss during energy restriction.³ Some,^{4–8} but not all,⁹ evidence supports an inverse relationship between calcium (and/or dairy product) intake and body fat mass. Thus, this area of research remains somewhat controversial.

Recently, our laboratory conducted a 1-year dairy product intervention trial¹⁰ that investigated the effect of dairy food consumption on changes in fat accumulation in healthy young women and found no evidence of a dairy group effect. However, a 6-month follow-up study on these trial subjects found a significant effect of high dietary calcium in reducing body fat accumulation.⁸ The effect of high calcium and/or dairy intake on body fat accumulation may likely be quite small, but it could be significant over the long term.

Another research interest in the area of calcium and body composition has been whether calcium and/or dairy intake influences the extent of fat loss during energy restriction. The evidence for calcium and/or dairy intake enhancing fat-mass loss during energy restriction is also contradictory.^{11–16} The discrepancies in findings among studies, along with the small treatment effect observed in various epidemiological and intervention studies, suggests a greater understanding of the underlying mechanism(s) contributing to the calcium and/or dairy effect on body weight and body composition is needed in order to determine the specific circumstances under which an impact might be seen. Several biologic mechanisms have been proposed as potentially contributing to the overall impact of calcium and/or dairy on body weight or body fat mass: 1) increased energy loss through increased expression of uncoupling proteins or increased lipid oxidation;^{17–19} 2) decreased availability of energy by the formation of fecal calcium: fatty acid soaps accompanied by reduced fat absorption;²⁰ and 3) reduced food intake caused by decreased appetite.

Does increased dairy consumption lead to reduced total energy intake?—An important practical question is whether the addition of more sources of dairy foods to the diet to increase calcium intake will result in an at least reciprocal compensation in total energy intake through decreased intake of non-dairy foods. Barr et al.⁹ found that elderly people compensated well for the energy contained in three additional daily servings of milk by reducing food intake from other sources. Indeed, since the satiety value of dairy products, yogurt, and cheese has been empirically determined to be higher than that of many similar foods,²¹ increased consumption of dairy foods could result in lower total energy intake and promote weight loss. However, Almiron-Roig et al.²² found that increased intake of milk was not associated with compensation for the energy contained in the milk consumed in young adults.

This question was recently addressed in another study that investigated the impact of a 7-day dairy product intervention on food intake.²³ Participants (18–50 years, BMI 25–32 kg/m², *n* = 60, equal numbers of men and women) with either habitual low or high intake of dairy were enrolled. Subjects were instructed to consume either a low-dairy diet (one serving of dairy product/day) or a high-dairy diet (three portions of dairy products/day) for 7 days with a 7-day washout between test periods in a randomized crossover design. No other requirements for intake were given. Thus, this study tested the ability of the participants to compensate for the addition of dairy products to their usual intakes. Estimated dietary intakes were assessed daily and appetitive measurements were taken by a computerized system throughout the day.

This study found that increased intake of dairy food did not lead to complete compensation of the added energy intake. Male participants consumed significantly more calories with the high-dairy intervention diet (approximately 200 kcal/day) compared to the low-dairy diet. Men with high habitual dairy intake reduced their intake of energy from other sources by only 31%. In fact, men with low habitual dairy intake did not compensate for the extra dairy intake at all; they increased their energy intake by 12% more than would be predicted by the addition of two additional dairy products to the diet. Female participants also did not fully compensate for the additional energy intake from the high-dairy-product intervention. Women with high habitual calcium intake reduced their non-dairy energy intake by 72% of the extra dairy energy load. Women with low habitual intake reduced their non-dairy energy intake by 50% of the extra dairy energy load. In addition, there were no significant differences in ratings of hunger, fullness, desire to eat, or preoccupation with foods between intervention groups. The findings of this study suggest that, in contrast to the hypothesis that dairy products may lead to a reduction in food intake, there is incomplete compensation for the increased energy intake from the dairy products, or, potentially, a higher intake of energy with a low habitual intake of calcium in men than would be expected. However, this study did not specifically test the effect of calcium alone on dietary energy intake; rather, it tested the ability to compensate for added dietary calories from dairy products (a rich source of calcium). The negative findings from this study support the null hypothesis that there is not complete compensation for added dietary energy intake from dairy, thus suggesting that added calcium intake in the form of a dairy source does not cause a net reduction in energy intake. In addition, these examples do not support the position that dairy products and/or dairy calcium has a net beneficial effect on appetite and satiety or feelings of fullness and subsequent compensation in energy intake.

Calcium and food intake: potential mechanisms—Various peptide hormones (e.g., ghrelin, CCK, PYY, GLP-1) have been implicated as key players in the control of appetite, satiety, and energy intake. Ghrelin has been shown to increase food intake and decrease fat utilization.²⁴ Consistent with this observation, levels of ghrelin are increased before meals and decreased in the postprandial state.^{25–29} CCK is the first gut hormone found to have effects on appetite.³⁰ CCK mediates meal termination and, possibly, early-phase satiety, at least in part through its effects on gastric emptying.³⁰ GLP-1 inhibits acid secretion and gastric

emptying in the stomach; it also increases satiety, leading to decreased food intake.³¹ Long-term (5 days) administration of GLP-1 has been shown to cause weight loss in obese, though otherwise healthy, adults.³² PYY has been shown to reduce appetite and food intake.³⁰ Notably, obese subjects secrete less PYY than normal-weight subjects and reduce food intake in response to administered PYY.³³ These collective data provide a reasonable basis for examining the response of satiety regulators to different levels of dietary calcium intake as a means of gaining a better understanding of the potential effects of calcium and dairy products on appetite and subsequent food intake in humans.

The effects of dairy product intake on CCK was investigated several years ago in a randomized crossover-design study conducted by Schneeman et al.³⁴ The study found that the intake of meals containing dairy products increased CCK levels within a 6-hour period more than the intake of non-dairy meals in women but not in men. However, when a subjective satiety response to the test meals was assessed using visual analog scales (VAS) over the 6-hour period following meal consumption, the results suggested that the non-dairy-product meals suppressed hunger more strongly than the dairy product meals, which was in contrast with the CCK response. The subjects reported that the “desire to eat” was lower and “fullness” was higher following consumption of the non-dairy-product meals compared to the meals containing dairy products. Thus, according to the VAS analysis, the non-dairy meals were more satiating than the dairy product. Again, it is important to note that this study investigates the impact of dairy product, not calcium specifically, on CCK and appetitive responses.

Does the calcium content of a meal affect food intake?—Few studies have assessed the direct impact of calcium consumption on food intake. Recently, Lorenzen et al.²⁰ investigated the acute impact of calcium from various levels of dairy product supplementation in a randomized, controlled, crossover design study in 18 overweight male subjects. The four isocaloric test meals consisted of three with dairy products as the calcium source: low calcium (68 mg), medium calcium (350 mg), and high calcium (793 mg). The fourth meal consisted of a calcium supplement with a meal containing whey, casein, and lactose; thus, all four meals contained equivalent levels of dairy protein. Blood was drawn at intervals for 7 hours following meal consumption; thereafter, subjects were served an *ad libitum* meal with instructions to eat until “comfortable satisfaction”, and energy intake from this meal was estimated. A 3-week washout period occurred between test meals.

The study findings demonstrated that, consistent with an increase in fecal fat loss noted previously by these authors,³⁵ the area-under-the-curve (AUC) for serum chylomicron-associated triglycerides was reduced after intake of the medium- and high-calcium meals compared to the low-calcium meal. However, there were no significant differences between the test meals in the AUC for CCK, ghrelin, glucose, GLP-1, PYY, or insulin measured post consumption. This observed lack of response of CCK to dietary calcium differs from findings in the study of Schneeman et al.³⁴ mentioned above, where the product tested was non-dairy compared to dairy. In contrast, the test meals in the study of Lorenzen et al.²⁰ were modified by the calcium content with equivalent levels of dairy components. Thus, the CCK response observed in the study of Schneeman et al.³⁴ may be due to the dairy product components rather than the extra calcium delivered.

In the current study,²⁰ the high-calcium meal was more palatable than the low-calcium or supplement meal, and the subsequent *ad libitum* meal smelled better and was more visually appealing to the subjects following the high-calcium meal compared to the low-calcium meal. However, there was no significant difference in energy intake from the *ad libitum* meal following consumption of the test meals. In theory, the increase in palatability could promote an increase in food intake or lack of compensation, similar to that noted in the study by Hollis and Mattes.²³ Cumulatively, these results are consistent with studies in rodents, which

demonstrate no changes in food intake with low or high calcium intakes.^{36,37} Thus, these findings suggest that calcium intake does not influence food intake and are thus consistent with studies in experimental animals.

CONCLUSION

The observations from studies investigating the impact of calcium intake on food intake are limited and controversial. None of the studies performed to date directly show that dietary calcium alone alters food intake. The suggestion that calcium intake reduces CCK or other appetite-regulating hormones has little support. Cumulatively, these observations suggest that calcium intake is not likely to have a significant impact on food intake. In conclusion, the size effects of calcium on weight or fat mass accumulation are, if anything, small (less than 100 kcal/day)³; therefore, it may be difficult or impossible to measure such small effects of calcium on food intake, appetite, or hormonal changes.

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