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Effect of a dairy and calcium rich diet on weight loss and appetite during energy restriction in overweight and obese adults: a randomized trial

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Abstract

Background/Objectives—A diet rich in dairy and calcium (Ca) has been variably associated with improvements in body composition and decreased risk of type 2 diabetes. Our objective was to determine if a dietary pattern high in dairy and Ca improves weight loss and subjective appetite to a greater extent than a low dairy/Ca diet during energy restriction in overweight and obese adults with metabolic syndrome.

Subjects/Methods—49 participants were randomized to one of two treatment groups: CONTROL [low dairy, ~700 mg/day Ca, −500 kcal/d] or DAIRY/CA [high dairy, ~1400 mg/day Ca, −500 kcal/d] for 12wk. Body composition, subjective ratings of appetite, food intake, plasma satiety hormones, glycemic response and inflammatory cytokines were measured.

Results—CONTROL (−2.2±0.5 kg) and DAIRY/CA (−3.3±0.6 kg) had similar weight loss. Based on self-reported energy intake, the percent of expected weight loss achieved was higher with DAIRY/CA (82.1±19.4%) than CONTROL (32.2±7.7%)(P=0.03). Subjects in the DAIRY/CA group reported feeling more satisfied (P=0.01) and had lower dietary fat intake

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Supplementary Information
Supplementary information is available at EJCN's website.

RAR, ALE, and PKDB designed research; KWJ and JAP conducted research; RAR and LKE analyzed data and wrote the paper; RAR had primary responsibility for final content. All authors read and approved the final manuscript.

Conflict of Interest
KWJ, JAP, LKE, PKDB, and ALE declare no conflict of interest. RAR previously held research funding from the Dairy Farmers of Canada for work distinct from this study.

(*P*=0.02) over 12wk compared to CONTROL. Compared to CONTROL, DAIRY/CA had higher plasma levels of peptide tyrosine tyrosine (PYY, *P*=0.01) during the meal tolerance test at wk12. Monocyte chemoattractant protein-1 was reduced at 30 min with DAIRY/CA compared to CONTROL (*P*=0.04).

Conclusions—In conclusion, a dairy and Ca rich diet was not associated with greater weight loss than control. Modest increases in plasma PYY concentrations with increased dairy/Ca intake, however, may contribute to enhanced sensations of satisfaction and reduced dietary fat intake during energy restriction. Registered Trial: ClinicalTrials.gov (NCT00564551).

Keywords

Calcium; dairy; peptide YY; clinical trial; satiety

Introduction

The consumption of dairy foods and calcium (Ca) has been variably linked to regulation of body weight and risk of type 2 diabetes.[1] Part of the variability stems from the examination of low-fat dairy foods and/or Ca intake within two distinct contexts, one of which is energy balance and weight maintenance and the other of which is energy restriction and weight loss.[1–4] Furthermore, discrepancies between studies may be due to variation in the type of dairy foods (yogurt versus milk), in total Ca intake, or source of Ca. A threshold of Ca intake of ~600–800 mg/day has been proposed for the beneficial effects on weight regulation.[1] Moreover, whether elemental Ca supplementation in combination with increased dairy food intake is effective for weight management in humans requires clarification.

The individual dairy proteins (whey and casein) may enhance satiety via increases in circulating appetite regulating hormones including glucagon-like peptide-1 (GLP-1).[5,6] A recent 6 month study found an attenuation in desire to eat and hunger during weight loss when participants consumed milk[7], although the mechanism remains unclear as there were no changes in ghrelin or leptin. Similarly, a single meal study found no effect of dairy foods on GLP-1, ghrelin, peptide tyrosine tyrosine (PYY), and CCK.[8] Ca may affect energy intake as seen in a 15wk study where Ca plus vitamin D supplementation reduced spontaneous fat intake, although the effect was only seen in a small subpopulation with very low Ca intake.[9] In addition to regulating appetite, dairy and/or Ca may influence metabolic health via upregulation of genes associated with metabolic rate[10]; enhanced fecal fat excretion[11]; and by mediating the inflammatory response.[12]

As reviewed by Teegarden and Gunther[2], evidence in support of the hypothesis that dairy foods and/or dietary Ca influence appetite control and food intake remains inconclusive. Our primary objective was to determine if a dietary pattern high in dairy and Ca, derived from both dairy foods and a Ca supplement, would improve weight loss and appetite regulation during energy restriction (−500 kcal/d). Specifically, we examined plasma GIP, GLP-1, ghrelin, leptin, and PYY concentrations and subjective appetite ratings in overweight and obese adults with metabolic syndrome. Glycemic, insulinemic and inflammatory cytokine responses were also examined.

Subjects and methods

Subjects

Forty-nine men and women (BMI 27–37 kg/m^2), 20 to 60 y with metabolic syndrome were recruited from Calgary, AB, Canada. Twenty-three were randomized to CONTROL and twenty-six to DAIRY/CA (Supplemental Figure 1). The National Cholesterol Education Program Adult Treatment Panel III guidelines were used to identify metabolic syndrome[13]. Exclusion criteria included: type 1 diabetes; type 2 diabetes treated with oral hypoglycemic agents or insulin therapy; hemoglobin A1c (HbA1c) >8%; liver or pancreas disease; major gastrointestinal surgeries; pregnancy or lactation; cardiovascular disease; alcohol or drug dependence; milk allergy or lactose intolerance; use of a diet, supplement or exercise regime designed for weight loss; body mass >159kg; fibrate or statin use; chronic use of laxatives, antacids, Ca, or vitamin D; or high habitual Ca intake. A registered dietitian assessed typical Ca intake using verbal recall. All enrolled participants had self-reported low dairy and Ca (<700 mg/d)[7,9] intake at baseline. All participants provided written informed consent. Ethical approval was provided by the Calgary Conjoint Health Research Ethics Board. This study was registered at ClinicalTrials.gov (NCT00564551). A power calculation with an α of .05 and power of .80 indicated a minimum of 18 participants per group would be required.

Intervention

Prior to the start of the intervention, participants attended an orientation in which motivational interviewing was used to encourage adherence.[14] Instructions regarding use of a food scale, meal plans, and 3-d food records were provided. Participants were randomized (random number generator; stratified according to BMI and sex) to either CONTROL or DAIRY/CA and provided with an individualized meal plan that prescribed a 500 kcal/d energy deficit. CONTROL meal-plans included 1 serving of dairy (non-fat or 1% milk or yogurt) with total Ca ~700 mg/day. DAIRY/CA meal-plans prescribed 3–4 servings of dairy (non-fat or 1% milk or yogurt) and included a daily 350 mg Ca supplement (Cal-Chews™, Jamieson Laboratories Ltd., Windsor, ON, Canada) with total Ca ~1400 mg/day.

An initial 3-d food record was completed by participants prior to the first meal tolerance test (MTT) to gain a baseline estimate of energy requirements.[15] This estimate was refined using the Mifflin-St. Jeor equation and an activity factor.[16] Individualized diet plans providing a 500 kcal/d energy deficit and based on Canada's Guidelines for Healthy Eating (~30% fat, 20% protein and 50% carbohydrates) were devised. The majority of carbohydrates were whole grains, vegetables, and fruit. Dietary intake during the study was measured via 3d food records at 3, 6, 9, and 12 wk. Diet Analysis Plus 8.0 software was used for analysis (Thomson Wadsworth, Toronto, ON).

Physical activity

Participants were instructed not to change their exercise habits during the study. Exercise levels were quantified at baseline and wk 12 using Godin's Leisure Score Index Questionnaire.[17]

Physical characteristics

At baseline and wk 12, body composition was assessed with DXA (Hologic QDR 4500, Hologic, Inc., Bedford, MA). Weight was measured using a balance beam scale at baseline, wk 3, 6, 9 and 12. At baseline and wk 12, height, waist circumference and blood pressure were measured.

Meal tolerance test and blood sampling

At baseline and wk 12, a blood sample was collected in the morning after 12 hr of fasting. Participants then consumed a standardized meal consisting of 50 g white bread, 50 g rye bread, 30 g cheddar cheese, 10 g butter, 20 g fruit jam, and 200 mL unsweetened orange juice [605 kcal; 56% carbohydrate, 11% protein, and 32% fat].[18] Postprandial blood samples were collected via antecubital vein cannula at 30, 60, 90, 120, and 240 min following the first bite of the meal according to our previous protocol.[19]

Plasma analysis

Glucose was determined via Trinder assay (Stanbio Laboratory, Boerne, TX). Ghrelin (active), GLP-1 (active), GIP (total), leptin, insulin, and PYY (total) concentrations were quantified using a Human Gut Hormone Milliplex kit (Millipore, St. Charles, MO). Concentrations of interleukin 1 beta (IL-1β), IL-6, monocyte chemoattractant protein-1 (MCP-1), and tumor necrotic factor alpha (TNFα) were quantified using Human Adipokine Milliplex kits (Millipore). Calgary Laboratory Services (Calgary, AB, Canada) measured HbA1c.

Subjective appetite scores

Subjective sensations of appetite were determined with 100 mm visual analog scales (VAS).[20] Weekly VAS were distributed at baseline for completion by the participants each week at home. Participants were asked to complete VAS following a meal, at the same time on the same day each week. In addition, each subject was asked to complete VAS throughout the MTT. Questions took the form of “How full do you feel?” or “How much do you think you can eat?” and were anchored by “not at all full” or “nothing at all” and “totally full” or “a lot”.

Statistical analysis

Data is presented as mean±SE and only includes those who completed the trial. Physiological measures, food record and VAS data were analyzed via 2-factor repeated measures ANOVA with Bonferroni adjustment [time (wk 0 and wk12) and diet (CONTROL or DAIRY/CA)]. Change from baseline was determined by subtracting initial value from final value and analyzed by ANOVA. Hormone and glucose concentrations during the MTT were analyzed via 2-factor repeated measures ANOVA with a Bonferroni adjustment [time (0–240 min) and diet] as variables or 2-factor analysis with week (0, 3, 6, 9, 12 wk) and diet]. Data were analyzed with SPSS v. 17.0 software (SPSS Inc, Chicago, IL).

Results

Baseline characteristics

Forty-nine individuals were enrolled with 38 participants completing the study (Table 1). Reasons for dropping out included pregnancy (n=1), change in employment (n=2), illness (n=2), or personal (n=6). There were no differences in the baseline characteristics between groups (all: P>0.05) except for bone mineral density (P=0.04) (Table 1). Physical activity did not change during the study (P>0.05).

Body composition

Weight loss was −2.2±0.5 kg and −3.3±0.6 kg (P=0.16) in the CONTROL and DAIRY/CA groups respectively (Table 1). The change in lean body mass (LBM), from wk 0 to wk 12, was significantly different between groups (P=0.03) with a slightly greater decrease in DAIRY/CA versus CONTROL (Table 1). Bone mineral content (BMC) was significantly higher in DAIRY/CA compared to CONTROL at wk 12 and the change in BMC showed a decrease in CONTROL (−2.8±9.6 g) and increase (32.2±12.9 g) in DAIRY/CA (P=0.04) (Table 1).

Satiety and hunger hormones

The change in PYY tAUC from baseline to wk 12 was significantly greater for DAIRY/CA compared to CONTROL (P=0.01) (Fig. 1A). During the MTT, the change from baseline to wk 12 was significantly greater for DAIRY/CA versus CONTROL at 0 min (P=0.04), 30 min (P=0.01) and at 240 min (P=0.01) (Fig. 1B). PYY, GLP-1, GIP and ghrelin curves are shown in Supplemental Fig. 2. At the end of the study, the change between baseline and wk 12 GLP-1 concentrations at 240 min was significantly greater with DAIRY/CA (P=0.02) (Supplemental Fig. 2B). There were no differences in leptin (Supplemental Figure 3A & D), although there was a positive correlation between body fat and fasting leptin (r=0.70, P<0.01) and leptin tAUC (r=0.68, P<0.01).

Subjective appetite scores

DAIRY/CA reported feeling ‘more satisfied’ in the weekly assessment of subjective appetite sensations (P=0.01) (Fig. 2A). There was a significant interaction between time and diet (P=0.03) for the question ‘how comfortable do you feel?’ wherein CONTROL felt less comfortable from baseline (63 ± 7 mm) to wk 12 (45 ± 6mm) and DAIRY/CA felt more comfortable at wk 12 (59 ± 4 mm) compared to wk 0 (50 ± 5 mm). At wk 0 and wk 12, participants also completed VAS during the MTT (Supplementary Figure 4), wherein there was a significant effect of time for ratings related to hunger, satisfaction, fullness, prospective consumption, and desire to eat something sweet, salty or meat and fish (P<0.05). There were no diet differences at wk 12, although there was a trend (P=0.1) for greater fullness in DAIRY/CA versus CONTROL when ratings were normalized for fasting scores.

Food intake and ‘feed efficiency’

Energy Intake—Total energy intake was significantly higher at baseline compared to wk 3, 6, 9, and 12 (P<0.05) (Table 2) but did not differ between groups. As expected, there was

a reduction in daily energy intake throughout the study in both groups (Table 2) (P<0.01). There was an effect of week (P<0.01) and diet (P<0.03) on fat intake. Both groups reduced their fat intake during the study, however, DAIRY/CA consumed less energy as fat compared to CONTROL (P=0.02). Expressed as a function of body weight, DAIRY/CA consumed less fat (0.58±0.04 g/kg) versus CONTROL (0.78±0.07 g/kg) (P=0.015). Using the self-reported reduction in energy intake, we calculated the percent expected weight loss achieved by subjects (observed weight loss/expected weight loss*100). DAIRY/CA achieved a greater percent of expected weight loss (82.1±19.4%) compared to CONTROL (32.2±7.7%) (P=0.03). The correlation between percent expected weight loss achieved and delta energy intake approached significance (r=0.351;P=0.086). Feed efficiency, classically measured as weight gain per unit energy consumed in animal studies[21], was similarly calculated in this study to capture weight loss per unit restricted energy intake. There was no significant difference (P=0.35) between CONTROL (0.053 ± 0.012 g weight loss/kcal restricted) and DAIRY/CA (0.100 ± 0.038 g weight loss/kcal restricted).

Calcium and Vitamin D Intake—Daily Ca intake during the study was significantly higher in DAIRY/CA versus CONTROL (Table 2)(P<0.001). Baseline dairy intake was similar between groups but as expected higher in DAIRY/CA versus CONTROL during the study. Vitamin D intake at baseline and during the study was significantly higher in DAIRY/CA (P<0.05, Table 2). The relationship between delta energy intake from baseline to wk 12 (kcal) and Ca (mg) intake was significant (R=0.40, r^2 =0.16, df=28, P=0.027)(Fig. 2B). The r^2 value implies that 16.3% of variation in total energy intake can be explained by Ca intake. There was no significant relationship between anthropometric measures (LBM or fat mass) and Ca intake (P>0.05).

Glucose homeostasis and inflammation

The change in glucose tAUC from wk 0 to wk12 was −111±48.4 mmol·L^{-1}240min^{-1} in DAIRY/CA and −23.3±71.7 mmol·L^{-1}240min^{-1} in CONTROL, which did not differ (P>0.05). No differences in fasting or tAUC were detected for insulin (Supplemental Fig. 3C, F).

Fasting IL6, TNFα, MCP-1, and IL1β remained constant over the 12 wk (Supplemental Table 1). There was a significant reduction (P=0.04) in baseline adjusted MCP-1 concentration at 30 min for DAIRY/CA (−18.7±7.5 pg mL^{-1}) versus CONTROL (4.6±7.5 pg mL^{-1}).

Discussion

Across a range of human and rodent studies there have been inconsistent results regarding the role of dairy and/or Ca in regulating body weight, appetite, and glucose homeostasis.[1] Our findings demonstrate that in the context of energy restriction, a dietary pattern rich in dairy foods and Ca results in modest increases in plasma PYY concentrations, enhanced subjective ratings of feeling satisfied and reduced dietary fat intake, but does not accelerate weight loss compared to a low dairy/Ca diet.

Participants consuming dairy/Ca had weight loss that was similar in magnitude to control. This is consistent with findings from Van Loan et al.[22] showing no difference in body weight or fat loss between low dairy or high dairy energy-restricted diets. Similarly, a recent meta-analysis showed no differences in body weight changes between dairy intervention and control groups.[3] With subgroup analysis, however, dairy was shown to reduce body weight (−0.79 kg) in studies imposing energy restriction.[3] This is similar to the meta-analysis of Abargouei et al. [4] that found no overall difference for the effect of dairy on body weight but did find a significant reduction in body weight and fat mass within the energy restricted subgroup. Although weight loss did not differ in our study, the modest indications of improved appetite regulation with dairy may be clinically relevant. In the context of weight management for consumers, foods that oppose the physiological consequences of energy restriction and the feelings of deprivation that accompany restriction are meaningful targets.[23]

In contrast to the decrease observed in CONTROL, DAIRY/CA resulted in a modest increase in PYY concentration during energy restriction. Attenuated PYY concentrations and blunted meal responses have been reported in obesity[24]; whereas administration of 100–200 μg/kg systemic PYY3-36 reduced motivation to seek high-fat food in a rodent relapse model.[25] Furthermore, while PYY levels increase following Roux-en-Y gastric bypass surgery[26], weight loss induced by energy-restricted low-fat or low-carbohydrate diets has been shown to reduce serum PYY levels.[27] Although weight loss did not differ in the current study, it is possible that the modest increase in PYY levels and spontaneous reduction in dietary fat intake seen with DAIRY/CA could help prevent relapse to a maladaptive high-fat diet.[25]

Analysis by linear regression suggests that Ca intake (reflecting both dairy and Ca supplement) was related to the change in energy intake over the 12 weeks. In contrast, there was no correlation between Ca intake and BMI or body weight. This relationship suggests that participants who consumed more Ca also consumed more energy, but did not gain weight relative to the increased energy. This finding is consistent with Barr *et al*[28], who found that in free-living elderly, those who drank 3 cups of milk per day did not gain the amount of weight predicted based on additional energy intake. We probed this phenomenon further by calculating the percent of expected weight loss achieved by our subjects. We acknowledge the limitations that self-reported reductions in energy intake introduce to our calculation but the percent of expected weight loss achieved by DAIRY/CA was 2.5 times that of the CONTROL (82.1% versus 32.2%). We also calculated a modified version of feed efficiency. Although numerically double, we did not see a significant difference between DAIRY/CA (0.100 ± 0.038 g weight loss/kcal restricted) and CONTROL (0.053 ± 0.012 g weight loss/kcal restricted). Calculating feed efficiency in the traditional sense, Thomas et al.[21] did show significantly lower feed efficiency (mg weight gain/kJ consumed) in diet-induced obese mice fed non-fat dry milk (2.3±0.1) compared to control (3.5±0.1) and high-Ca alone (3.8±0.1). The explanation for the altered ‘efficiency’ of dairy/Ca rich diets may include increased fecal fat excretion[29], increased fat oxidation[30], blunted parathyroid hormone release[31], suppression of circulating calcitriol[32] or potentially increased PYY concentrations.

In *ob/ob* mice, Pittner *et al*[33] demonstrated that a 4 wk pharmacologic infusion of PYY reduced weight gain without a concomitant decrease in total energy intake. Likewise in humans, subcutaneous injections of PYY3-36 result in a lipolytic effect.[34] It is important, however, to identify whether physiological concentrations of PYY influence energy homeostasis. To this end it was shown that peak PYY concentrations after a meal were negatively associated with 24-hour respiratory quotient (RQ) and fasting PYY negatively correlated with resting metabolic rate.[35] While it was impossible to establish causation in this study, the findings do suggest a potential for increased fat oxidation with higher endogenous PYY levels.[35] In mice treated with subcutaneous infusion of 1 mg/kg PYY3-36, it was similarly shown that RQ was decreased during the dark cycle, although the effect was transient.

When appetite was assessed subjectively, ratings over the 12 wk intervention were improved with DAIRY/CA. Specifically, as determined via weekly VAS, DAIRY/CA reported feeling more satisfied. Recently, Gilbert et al. [7] showed that a milk supplement was associated with a smaller increase in desire to eat and hunger during weight loss. A Ca-specific effect on appetite appears less robust given that only in a small subset of subjects (n=7 out of 63) with very low baseline Ca intake demonstrated a spontaneous decreased in fat intake with a Ca and vitamin D supplement. [9]

Our results for LBM and inflammatory markers are in contrast with some but not all studies. With regards to LBM, several studies show protective effects with a high dairy diet[36,37], while others have shown no difference.[22] The high proportion of branched chain amino acids in dairy proteins, particularly leucine, has been suggested to play a role in regulating muscle protein synthesis[38] and help reduce LBM loss during energy restriction. Although no benefits for LBM retention were observed in our study, the increase in BMC in the DAIRY/CA group is worth noting when contrasted against the slight decrease seen in CONTROL. Weight loss induced via energy restriction is a risk factor for rapid bone loss. [39]

With regards to inflammation, previous work in cells, rodents and humans by Zemel et al. [40,41] shows a decrease in inflammatory cytokines with increased dairy. Specifically, reductions in TNFα, IL6, and MCP-1 were seen in participants with metabolic syndrome that consumed a high versus low dairy diet.[40] We did not observe any change in IL6, TNFα or IL1β levels and MCP-1 was only reduced in DAIRY/CA at 30 min during the final MTT. The difference in findings may relate to the composition of the control diet in the two studies (Stancliffe *et al*[40] control diet included prepackaged processed foods, some of which contained *trans* fatty acids) or the presence of significant weight loss. For example, in mice fed control, high Ca or high nonfat dry milk, diet-associated differences in most inflammatory marker mRNA levels disappeared when body weight was included as a covariate.[21] The notion that reduced body weight drives the anti-inflammatory effects of dairy/Ca is interesting in light of a study by Van Loan et al.[22], which similar to us did not find a difference in body weight and no difference in circulating cytokines.

Our study adds to others designed to examine a high versus low dairy/Ca dietary pattern in free-living adults. We recognize that dietary Ca is not the same as dairy foods, and we tested a “portfolio-style” diet similar in principle to that devised by Jenkins *et al*. for cardiovascular

health.[42] We combined two bioactive food components aimed at maximizing efficacy and to address suggestions that at a population level, elemental Ca, either as a supplement or fortified foods may be needed to reach target Ca intake levels.[43,44] Our study is limited in that we did not measure energy expenditure and fecal fat loss, both of which would provide valuable information given the discrepancy we saw between energy intake and estimated weight loss.

In conclusion, a dietary pattern rich in dairy foods and Ca did not influence weight loss during a 12 wk energy-restricted period. However, DAIRY/CA resulted in a modest increase in circulating PYY in response to a standardized meal and was associated with reduced dietary fat intake and enhanced feelings of satisfaction. Given the demonstration that energy-restricted diets can reduce PYY levels[27], the ability of DAIRY/CA to prevent this decline and result in a modest increase in PYY levels may be important for appetite regulation during weight loss. While the ability of DAIRY/CA to augment weight loss during energy restriction is not substantiated by this study, the higher percent of expected weight loss achieved is intriguing and warrants further investigation into the effect of dairy/Ca on energetics.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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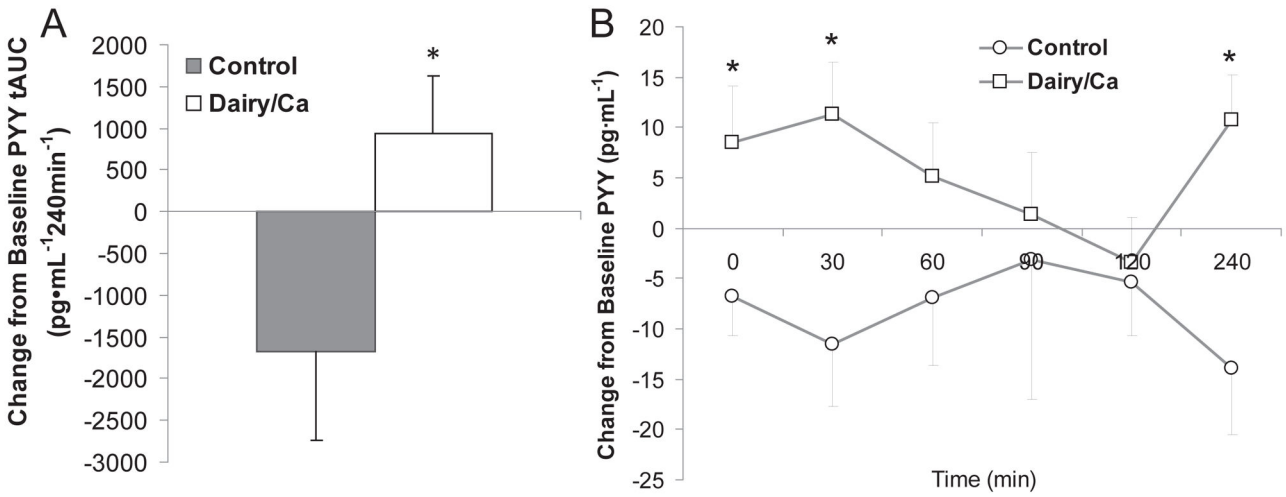

Figure 1.

Delta total area under the curve (**A**) and change from baseline values for plasma PYY (**B**) during a 4 h meal tolerance test in participants consuming control or dairy/Ca at wk 0 and wk 12. Values are means ± SE, n=18 (control) and n=20 (dairy/Ca). ○ Control, □ Dairy/Ca * Different from control at indicated time point, P<0.05.

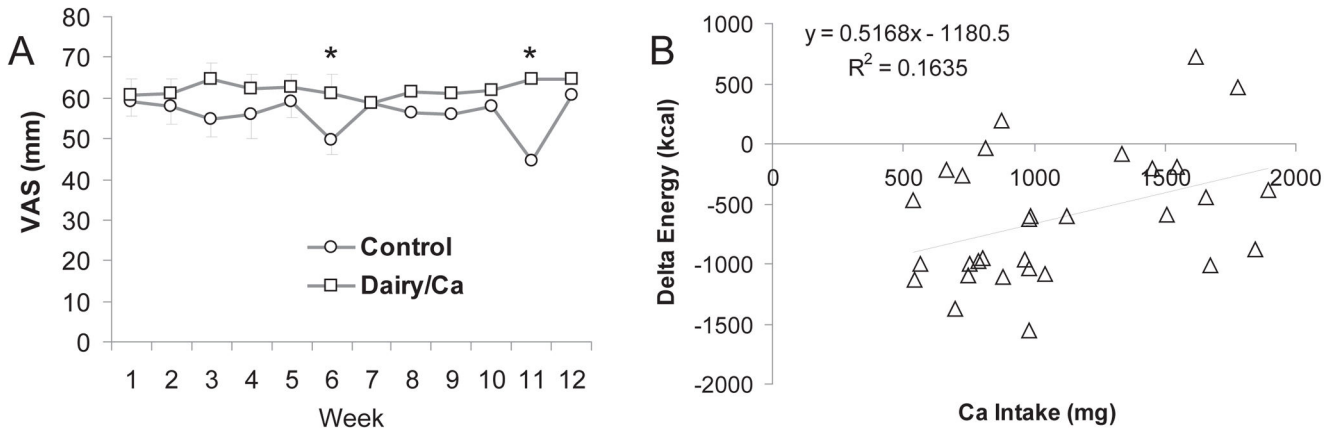

Figure 2.

Weekly subjective rating of satisfaction (**A**) and regression analysis (**B**) showing the relationship between delta energy intake from wk 0 to wk 12 (kcal) and Ca (mg) intake. In Panel A, values are means ± SE, *n*=18 (control) and *n*=20 (dairy/Ca). ○ Control, □ Dairy/Ca.

* Different from control at indicated time point, $P < 0.05$. In Panel B, the regression is represented by R=0.40, r^2 =0.16, df=28, $P = 0.027$.

Table 1

Clinical and biochemical characteristics of participants at baseline and after 12 wk of dietary treatment in the control or dairy/Ca groups[1].

	Baseline		Week 12	
Characteristic	**Control**	**Dairy/Ca**	**Control**	**Dairy/Ca**
Females/males, *n*	11/7 (18)	13/7 (20)	11/7 (18)	13/7 (20)
Age, *y*	50.1 ± 2.7	52.1 ± 1.5	50.1 ± 2.7	52.1 ± 1.5
Body weight, *kg*	84.0 ± 4.3	93.8 ± 3.6	81.8 ± 4.5	90.5 ± 3.5
Lean body mass, *kg*	55.6 ± 3.7	61.7 ± 3.0	55.2 ± 3.8	60.0 ± 2.9
Body fat, *kg*	28.4±2.0	32.1 ± 1.7	26.6 ± 1.9	30.5 ± 1.6
Body fat, *%*	34.2 ± 1.9	34.5 ± 1.4	32.9 ± 1.8	33.9 ± 1.4
BMI, $kg\ (m^2)^{-1}$	31.7 ± 0.9	32.9 ± 0.8	30.6 ± 0.9	31.7 ± 0.8
Waist circumference, *cm*	104 ± 2.8	110 ± 1.9	98±2.9	106±2.1
Bone mineral density, $g\ (cm^3)^{-1}$	1.09 ± 0.02	1.16 ± 0.02*	1.10±0.02	1.16±0.02*
Bone mineral content, *g*	2240 ± 97	2530 ± 109	2240 ± 100	2560 ± 109*
Total cholesterol, $mmol\ L^{-1}$	5.4 ± 0.4	4.8 ± 0.2	4.9 ± 0.4	4.6 ± 0.2
HDL-C, $mmol\ L^{-1}$	1.2 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	1.1 ± 0.1
LDL-C, $mmol\ L^{-1}$	3.4 ± 0.4	2.9 ± 0.2	3.0 ± 0.3	2.8 ± 0.2
Total-C:HDL-C Ratio	4.7 ± 0.3	4.3 ± 0.3	4.4 ± 0.3	4.5 ± 0.3
Triglycerides, $mmol\ L^{-1}$	1.7 ± 0.3	1.8 ± 0.2	1.7 ± 0.3	2.0 ± 0.2
HbA1c, *%*	5.8 ± 0.1	5.9 ± 0.1	5.7 ± 0.1	5.7 ± 0.1
Systolic blood pressure, *mmHg*	126 ± 3.9	128 ± 3.7	123 ± 3.1	121 ± 2.4
Diastolic blood pressure, *mmHg*	76 ± 2.3	78 ± 2.7	73 ± 2.6	71 ± 2.5
Fasting glucose, $mmol\ L^{-1}$	5.7 ± 0.4	6.2 ± 0.4	5.6 ± 0.4	6.3 ± 0.6
Fasting insulin, $pg\ mL^{-1}$	471 ± 85	700 ± 114	406 ± 120	559 ± 89
HOMA-IR	2.6 ± 0.5	4.3 ± 0.6	2.4 ± 0.9	3.6 ± 0.6

[1] Data are means ± SE, n=18 (control) and n=20 (dairy/Ca).

* Different from control within wk 0 or wk 12, $P < 0.05$.

Table 2

Daily dietary intake of participants at baseline and during the 12 wk of dietary treatment in the control or dairy/Ca groups[1]

	Baseline		Study[1]	
	Control	**Dairy/Ca**	**Control**	**Dairy/Ca**
Energy, *kcal*	2274.0 ± 172.0	2049.5 ± 97.4	1720.5 ± 136.6 *	1661.8 ± 83.8 *
Fat, *%*	38.9 ± 1.9	33.9 ± 1.4	32.2 ± 1.6	26.6 ± 1.5 †
Protein, *%*	18.5 ± 0.8	17.6 ± 0.9	21.5 ± 1.1	22.1 ± 0.7
Carbohydrate, *%*	45.7 ± 2.1	48.8 ± 2.2	45.5 ± 2.9	50.9 ± 1.5
Low-fat milk[2], *mL*	129 ± 34.0	195 ± 46.2	89.2 ± 20.8	463 ± 61.3 †
Low-fat yogurt[2], *g*	20 ± 7.2	26 ± 9.8	31 ± 8.2	126 ± 35.7 †*
Calcium, $mg\ d^{-1}$	943 ± 127	884 ± 82	736 ± 37	1400 ± 79 †
Vitamin D, $\mu g\ d^{-1}$	2.4 ± 0.6	5.6 ± 0.6 †	2.7 ± 0.6	6.1 ± 0.6 †

[1] Mean intake during the study as reported via 3-d food records at wk3, wk6, wk9, and wk12. Data is presented mean ± SE, n=18 (control) and n=20 (dairy/Ca).

[2] Serving size is 250 mL for milk and 175 g for yogurt

† Different from control at respective time point (baseline or during study), $P < 0.05$.

* Different from baseline within treatments, $P<0.05$.