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# Whole-fat dairy products do not adversely affect adiposity or cardiometabolic risk factors in children in the Milky Way Study: a double-blind randomized controlled pilot study

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## ABSTRACT

**Background:** Limited evidence supports the common public health guideline that children >2 y of age should consume dairy with reduced fat content.

**Objectives:** We aimed to investigate the effects of whole-fat compared with reduced-fat dairy intake on measures of adiposity and biomarkers of cardiometabolic risk in healthy 4- to 6-y-old children.

**Methods:** The Milky Way Study enrolled 49 children (mean  $\pm$  SD age:  $5.2 \pm 0.9$  y; 47% girls) who were habitual consumers of whole-fat dairy, then randomly assigned them in a double-blind fashion to remain on whole-fat dairy or switch their dairy consumption to reduced-fat products for 3 mo. Primary endpoints included measures of adiposity, body composition, blood pressure, fasting serum lipids, blood glucose, glycated hemoglobin (HbA1c), and C-reactive protein (CRP) and were assessed at baseline and study end. Pre- and postintervention results were compared using linear mixed models, adjusted for growth, age, and sex.

**Results:** Dairy fat intake was reduced by an adjusted (mean  $\pm$  SEM)  $12.9 \pm 4.1$  g/d in the reduced-fat compared with the whole-fat dairy group (95% CI:  $-21.2, -4.6$  g/d;  $P = 0.003$ ), whereas dietary energy intakes remained similar ( $P = 0.936$ ). We found no significant differential changes between dairy groups in any measure of adiposity, body composition, blood pressure, or fasting serum lipids, glucose, HbA1c, and CRP.

**Conclusions:** Our results suggest that although changing from whole-fat to reduced-fat dairy products does reduce dairy fat intake, it does not result in changes to markers of adiposity or cardiometabolic disease risk in healthy children. This trial was registered at [www.anzctr.org.au](http://www.anzctr.org.au) as ACTRN12616001642471. *Am J Clin Nutr* 2021;00:1–18.

**Keywords:** Milky Way Study, dairy fat, dietary fat, pediatric, randomized controlled trial, cardiometabolic disease, air displacement plethysmography, BodPod, cholesterol, child-centered care

## Introduction

Systematic reviews and meta-analyses of prospective adult cohorts have shown that core dairy whole foods—milk, cheese, and yogurt—are associated with lower risks of cardiometabolic dysfunction (1), including obesity (2); cardiovascular disease (CVD) (3–6); coronary heart disease (3–8); heart failure (7); hypertension (5, 9); stroke (5–8, 10); metabolic syndrome (MetS) (5, 11, 12) and its components (12); and all-cause mortality (3). In children, increased total dairy consumption is associated with

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No funding body played any role in the Milky Way Study design, implementation, analysis or interpretation of the data, or publication. The Milky Way Study received no funding from any dairy or food industry organization or affiliation toward study research, dairy product purchase or provision, child assessments, project personnel, or publication.

Supplemental Study Protocol and Supplemental Tables 1–3 are available from the “Supplementary data” link in the online posting of the article and from the same link in the online table of contents at <https://academic.oup.com/ajcn/>.

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Abbreviations used: CRP, C-reactive protein; CVD, cardiovascular disease; ECU, Edith Cowan University; EER, estimated energy requirement; FMI, fat mass index; HbA1c, glycated hemoglobin; LMI, lean mass index; MetS, metabolic syndrome; RCT, randomized controlled trial.

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lower adiposity (13). Associations between dairy intake and type 2 diabetes risk are more nuanced (14), with mostly beneficial associations (8, 15–20).

Although dairy forms part of a healthy diet in many countries (21–23), whole-fat dairy is thought to increase cardiometabolic risk (24–27). In Australia, whole milk is considered essential for infant growth, but everyone >2 y old is advised to consume mostly reduced-fat dairy (22). Traditionally, 2 main reasons are given: the “adiposity hypothesis” suggests that higher energy density in whole-fat dairy may increase ad libitum energy intake and body weight. The “saturated fat hypothesis” considers that the 60%–70% saturated fat fraction in dairy fat raises serum LDL cholesterol, considered a major risk factor for CVD (27–30). However, cardiometabolic risk projection is generally based on single-nutrient research or component outcomes in adults (31–33), and it is increasingly evident that matrix effects in complex whole foods modify the health impacts of individual nutrients (34). Hence, consuming dairy fat in cheese lowers serum LDL cholesterol compared with similar amounts of dairy fat in butter (35, 36).

Observational studies of adults show that dairy fat is not commonly associated with weight gain or cardiometabolic dysfunction when consumed as part of typical dietary patterns (28, 31, 32, 37, 38). The National Heart Foundation of Australia recently changed position, from recommending reduced-fat dairy choices for all adults toward considering whole-fat milk, yogurt, and cheese to be acceptable for healthy adults (27, 39). This represents a substantial shift toward accepting whole-food dairy as part of a healthy dietary pattern (40). In children, the largely observational evidence shows consistently that public health policy encouraging reduced-fat dairy after 2 y of age is unlikely to prevent or reduce childhood obesity or excess adiposity (41–43).

Observational studies may suffer from reverse causality or confounding (44, 45), and good-quality evidence in the form of clinical trials is needed to inform pediatric dairy guidelines. To date, as far as we know there have been no double-blind randomized controlled trials (RCTs) directly comparing the effects of whole-fat and reduced-fat dairy diets on comprehensive measures of child body composition (outside of BMI and waist circumference) or cardiometabolic risk factors. We aimed to investigate the effects of 12 wk of whole-fat compared with reduced-fat dairy intake on cardiometabolic risk factors in healthy 4- to 6-y-old children in an RCT. We hypothesized there would be no significant between-group differences in adiposity or in cardiometabolic risk factors.

## Methods

### Study design and trial registration

The Milky Way Study is a double-blinded comprehensive pilot RCT investigating the effects of dairy fat on health-related outcomes in young Western Australian children. The study involved assessments and sample collection at study clinics before and after a 3-mo dairy intervention. The Milky Way Study was registered prospectively with the Australian New Zealand Clinical Trials Registry as ACTRN12616001642471 (<https://www.anzctr.org.au/Trial/Registration/TrialReview.aspx?id=371803>) and approved by the Edith Cowan University (ECU) Human Research Ethics Committee (Project no. 14990).

Participants were recruited and completed the dairy intervention and relevant assessments for this study between January and December 2017, with all clinical assessments carried out at the ECU Joondalup campus in Perth. Primary study outcomes were 1) changes in a variety of common adiposity measures including anthropometrics (body weight, height, waist circumference, and neck circumference, and derived indexes such as BMI) and body composition (body fat mass and body fat percentage measured using a COSMED BodPod with pediatric option), and 2) changes in cardiometabolic risk factors {serum lipids (fasting total cholesterol, fasting HDL cholesterol, fasting triglycerides, and derived fasting LDL cholesterol); serum glycemic measures [fasting blood glucose and fasting glycated hemoglobin (HbA1c)]; the serum inflammatory biomarker fasting C-reactive protein (CRP); and systolic and diastolic blood pressure, assessed using a Dinamap recorder with pediatric cuff}. Secondary outcome measures included intakes of different food groups to provide data on dairy intake and overall diet composition, and erythrocyte fatty acid composition to verify compliance with the intervention.

### Development of the study protocol

The Milky Way study protocol (**Supplemental Study Protocol**) details prestudy preparation, such as a blind taste-test by children ( $n = 8$ ) to check the acceptability of the proposed study dairy products. We have previously published details of our pretrial community consultation (46) and child-centered research model: procedures developed to optimize parental study involvement and child compliance with clinical assessments have been detailed in our feasibility study (47). Study details conducted as per the supplied protocol have been shortened here in favor of clinically relevant detail.

### Study participants

We recruited participants from the coordinating university, community childcare centers, and parent social communities and organizations, and via social media snowball recruitment, articles in local newspapers, and a current affairs segment on television. Although Australian guidelines to change to reduced-fat milk apply to children from the age of 2 y (22), we chose slightly older children owing to the nature of the assessments to be used and feedback from our community consultation. Healthy children aged 4–6 y were eligible for inclusion in the study if they were daily consumers of  $\geq 1$  serving of whole-fat dairy, with >70% of their dairy consumed or prepared at home; lived within 20 km of the study university campus; and were able to complete the study protocol. Key exclusion criteria included diagnosis of or medications for cardiometabolic or gastrointestinal dysfunction; dairy allergy; antibiotic use over the past 3 mo; or having a body weight < 9.5 kg, because guidelines for the total blood volume that can safely be taken from children stipulate age-related body weights (48). This low body weight was chosen to exclude extreme outliers in advance, as detailed in the study protocol, ensuring safe blood draws of  $\leq 7$  mL from all participants.

Parents were recruited by telephone and sent parent and child information leaflets by email. At the start of the first clinic visit, child informed assent was obtained (47) and  $\geq 1$  parent signed

informed consent. After their first baseline clinic visit, each child was block randomly assigned, stratified by age and sex, into the whole-fat or the reduced-fat dairy group by an independent researcher picking an opaque sealed envelope for each participant from the appropriate one of 6 buckets (6 strata, comprising 2 sexes and 3 ages for each: 4, 5, or 6 y). The envelopes provided equal proportions of each dairy group; 6 envelopes/bucket were replenished when empty.

### Dairy intervention

In this double-blind RCT, 4- to 6-y-old children who were habitual consumers of whole-fat dairy were randomly assigned to either continue consuming whole-fat dairy foods, or to switch to reduced-fat versions of these dairy foods. The 3-mo dairy intervention began when the first dairy products were provided to take home, immediately after completion of all baseline assessments. The goal of a real-world study changed our original intention to recommend daily dairy intake according to dietary guidelines (22); instead, we requested ongoing intake of  $\geq 1$  serving of dairy per day, with no order limits. Children continued their habitual diet but replaced all dairy with the study dairy products, provided at no cost.

### Dairy product selection, blinding, and delivery.

Study dairy products were all purchased at local supermarkets, relabeled by independent researchers, packaged for optimum cold storage, and, after the first on-site collection, delivered regularly to most families at home. Apart from fat content, each product pair was closely matched for brand and nutrient content to minimize product variations, including differences in bovine diet (37, 49) and sugar content. Fat content for whole-fat products was  $\sim 3.5$  g/100 mL for milk,  $\sim 35$  g/100 g for cheese,  $\sim 9$  g/100 g for yogurt, and  $\sim 3.5$  g/100 g for dairy dessert and custard; we note the greater accuracy in reporting 3.5% dairy fat (50) than the 4% estimates in the prospective study documentation. For reduced-fat dairy, we aimed for 2 g/100 mL for milk, 15 g/100 g for cheese, 2 g/100 g for yogurt, and 2 g/100 g for dairy dessert and custard, based on product availability, suitability, likely child acceptability, and compliance with Food Standards Australia New Zealand criteria for reduced-fat foods (51). All cream and butter items provided were whole fat, or 50% fat-reduced, to help boost group dairy fat intake differences in an ad libitum diet. We offered a wide range of dairy foods to help maintain child interest and intake. This was balanced against possible variability in outcomes, given that different dairy products have potentially different effects on health (34–36, 40).

Final decisions on products were informed by child prestudy blind taste assessments, supply availability, and the ability to blind product pairs to prevent identification of the dairy group from a product's unique shape, color, or markings. A dietitian recorded each participant's habitual diet at their first clinic visit, using a "typical day" dietary recall (referencing the past 7 d, weekdays compared with weekends compared with holidays) combined with a study-specific dairy FFQ, with prompting around situational dairy intake contexts. Given the paucity of validated dairy FFQs for this age group (52), we designed these tools to enable provision of appropriate dairy products and to

provide support for the detailed food record data collected at baseline and end of intervention.

Dairy products offered to replace habitual intake included the core dairy products, milk, cheese, and yogurt, along with custard and chocolate dairy desserts, (sour) cream, and butter. Further choice requests were accommodated if a suitable product pair could be sourced. Yogurt was supplied in individual tubs (all  $\leq 200$  g) and options available included vanilla (French-style, unstrained pot-set compared with conventional, sweeter versions) and strawberry or berry (real fruit pieces); plain yogurt was supplied if specifically requested. Cheese choices comprised mild hard cheese (supplied as a whole food block or sliced; we did not offer highly processed cheese slices) and cream cheese (supplied as foil-wrapped wedges). Cream was provided as fresh pouring/whipping cream or sour cream. Dairy desserts comprised individual chocolate desserts (all  $\leq 175$  g) and plain custard ( $\sim 150$  g/serving, in 500-g containers). We did not supply ice cream, owing to food safety concerns around maintaining temperatures during deliveries.

To ensure compliance with the study dairy protocol, we included advice to create a dedicated fridge space for each child at a suitable height, and relabeled all participant dairy with each child's name prominent for their own "dairy section"; recommended that parents regularly involve children in their food preparation, and that children remind parents to use their study dairy; noted the suitability of individual items like cheese slices, cream cheese, and yogurts specifically for school lunchboxes, and of individual dairy desserts for temporary carers, to ensure study dairy consumption out of home; suggested making and freezing typical family communal dishes, such as lasagna, specifically for the child; recommended preparing and storing the child's own grated cheese, sour cream toppings, ice cream, etc., and adding these to child portions served first from communal family dishes prepared without these ingredients; and asked families to share successful recipes (e.g., ice cream) that used our study dairy. Family instructions were particularly detailed around separation and use of dairy when siblings were participating simultaneously.

Participant data relating to dairy requests, supply, and consumption (weight of dairy product supplied less weight returned) were stored in an electronic database (Microsoft Access 2016 database management system), coded to maintain clinical researcher blinding. All weekly or fortnightly home deliveries were in accordance with the study protocol, and ensured we maintained the dairy temperature at  $\leq 2^\circ\text{C}$  throughout; parents who worked nearby could choose to collect the dairy at prearranged times.

### Assessment of dairy intervention compliance and adverse event reporting.

Eligibility and ongoing compliance criteria included participants consuming  $\geq 1$  serving of dairy (preferably milk) per day, where a serving comprised a 250-mL glass of milk, 40 g cheese, or a 200-g tub of yogurt (22). Fortnightly compliance checks, initially by telephone and later by email, enabled assessment of each child's mean daily intake of the supplied dairy products; potential compliance issues and solutions detailed in the study protocol proved relevant. Parental feedback requested included details of suspected adverse events, including any untoward

medical occurrence affecting their child and not necessarily due to the dairy intervention.

### Child clinic assessments

Anthropometric and body composition assessments, blood pressure measurement, and blood draws were generally split over 2 preintervention baseline clinics, and repeated at a single final clinic visit in the last week of the dairy intervention. We have noted here where equipment varies from the study protocol: such changes were dictated by manufacturer upgrades or by practical experience gained in prestudy practice clinics with 4- to 6-y-old children, and all changes provided equal or improved accuracy of results. Parents completed a sociodemographic questionnaire at baseline. Children were excluded from further participation if they dissented to both body fat percentage and blood tests at baseline. Although we have previously defined assessment “success” (a result was obtained) as distinct from “compliance” (the child was prepared to have a go on the day) for our study child-centered research (47), “compliance” as used here has the more traditional meanings: that a procedure was followed satisfactorily and/or a quantifiable result was obtained.

### Anthropometrics and body composition.

A trained researcher measured child waist and neck circumference in the standing position using a Lufkin Executive Thinline 2-m clinical steel tape measure (W606PM), according to standard protocols (53, 54), and used a SECA 763 digital stadiometer (SECA Ltd.) to determine height. Each measurement included  $\geq 3$  readings taken to the nearest 0.1 cm;  $\geq 2$  that agreed to within 0.1 cm were averaged. Body composition was measured in the BodPod (COSMED) chamber using radiation-free air displacement plethysmography and a 2-compartment model (55) to determine fat mass and lean (fat-free) mass to within 0.001 kg and 0.01%. Body weight was reported to within 0.001 kg (1 g), using the integral calibrated electronic scale supplied with the BodPod.

The BodPod was fitted with a pediatric option (BodPod Pediatric Option™ GS model, COSMED USA Inc.), validated for use in children aged 2–6 y (55). Participants refrained from food, drink, and physical activity for 1.5 h before clinics, removed jewelry, and dressed in close-fitting bathing suits or active wear with bathing caps. All related parental communication included warnings to ensure that children had no recent ear problems, in anticipation of slight pressure changes in the BodPod chamber.

Anthropometric indexes calculated included BMI {weight (kg)/[height (m)]<sup>2</sup>}; waist-to-height ratio [waist (cm)/height (cm)]; neck-to-waist [neck (cm)/waist (cm)], neck-to-height [neck (cm)/height (cm)], and neck-to-waist-to-height ratios [neck (cm)/waist (cm)/height (m)]; fat mass index {FMI; body fat mass (kg)/[height (m)]<sup>2</sup>}; and lean (fat-free) mass index {LMI; lean mass (kg)/[height (m)]<sup>2</sup>} (56). US CDC child growth charts were used to determine BMI-for-age percentiles (57). The commonly used BMI is regarded as a limited surrogate measure of obesity, including in children, because it does not account for the distribution of body lean and fat mass (56, 58). Given that no 1 measure has been clearly identified as superior to others in children, we therefore aimed to measure “adiposity”

in a variety of different ways. Waist-to height ratio includes a measure of abdominal obesity, and may prove a better predictor of CVD risk than BMI (58), whereas according to the equation  $BMI = FMI + LMI$ , relative health risk could be assumed to increase with fat mass but to decrease with lean mass (56) [in the 2-compartment model used, body fat (or %) + lean mass (or %) = body mass (or 100%)].

### Blood pressure.

Blood pressure measurements were taken seated at rest, using a calibrated Dinamap ProCare 300 Monitor (GE Medical Systems) with the appropriate pediatric cuff determined by arm circumference. A minimum of 2 measurements was averaged for statistical analysis (59).

### Blood tests.

All blood test clinics followed overnight fasting. A 23 G BD Vacutainer blood collection set (Becton Dickinson and Company) was used to collect  $\leq 4$  vials (6.0 mL total) of blood according to pediatric research guidelines (48, 57, 60). The lithium heparin collection tube was centrifuged for 10 min at 4°C and 2500  $\times$  g; together with an unprocessed K2EDTA tube, it was transported to a local pathology laboratory for analysis within 2 h of the blood draw. A second K2EDTA tube was centrifuged as before; plasma was removed, and erythrocytes were saline washed (0.1% NaCl) and centrifuged again, as previously. All plasma (500  $\mu$ L) and erythrocyte (300  $\mu$ L) samples were stored on-site at  $-80^{\circ}\text{C}$  for later analysis.

### Blood analysis

#### Cardiometabolic biomarkers.

Blood samples were analyzed by PathWest, a National Association of Testing Authorities–accredited laboratory. Unless specified otherwise, all analyses were performed on an Abbott Architect c16000 (Abbott Laboratories) clinical chemistry analyzer, using the centrifuged samples from the lithium heparin tubes. Lipid profiling was performed using standard enzymatic assay techniques. LDL cholesterol was calculated using the Friedewald equation (61) and we calculated a total-to-HDL cholesterol ratio (total cholesterol/HDL cholesterol) and non-HDL cholesterol (total cholesterol minus HDL cholesterol). Immunoassay was used to determine fasting serum plasma glucose and ferritin. We included testing for the inflammatory marker CRP, using latex immunoassay; however, CRP values  $> 5.0$  mg/L were deleted from analyses based on suspected acute illness or injury. HbA1c was determined on a Cobas c501 system (Roche Holding AG), using the unprocessed K2EDTA tube samples.

#### Erythrocyte fatty acids.

Erythrocyte samples stored and transported at  $-80^{\circ}\text{C}$  were analyzed at the University of Western Australia Medical Research Foundation for red cell membrane fatty acids by GLC (Agilent Technologies Model 7980A Gas Chromatograph), using an SPTH 2560 column (100 m  $\times$  0.25 mm, 0.2- $\mu$ m film thickness; Supelco), with temperature programmed from 140°C to 240°C

at 4°C/min, and hydrogen as carrier gas (1 mL/min) at a split ratio of 30:1. Individual fatty acids were calculated as a relative percentage of the peak total area, with the total set at 100%.

### Dietary assessment

We chose weighed 3-d food records for Milky Way Study dietary assessment as a reasonable burden to ask of parents. At the first baseline clinic parents were asked to record everything their child consumed over 3 consecutive days. We provided electronic scales (Proport 5 kg Slimline stainless-steel digital scales), and a dietitian demonstrated accurate measurement techniques using food models. Parents were asked to note daily whether the record accurately represented their child's usual eating habits, and if not to describe why. Parents returned completed records at the final baseline clinic. These 3-d food records were repeated over the final weekend of the intervention. A dietitian reviewed each record on receipt at clinic visits and sought parental clarification for any potentially ambiguous or implausible items or quantities.

Each food record, together with supplied recipe and food packaging nutrient information, was entered into FoodWorks 10 Professional software for dietary energy, nutrient, and dairy product analysis (Xyris Software; updated after our study protocol, this version includes major food composition tables: AusFoods 2019, AusBrands 2019, Australian Food Composition Database) and independently verified.

### Assessment of dietary compliance and validity of food record reporting.

Individual food records were adjusted to exclude single days where a child's diet was deemed unusual; the remaining days were then averaged. Unusual daily intake was determined by a dietitian after parental clarification (e.g., due to reported illness). We allowed for natural large daily variations in children's intake, given innate adjustment for energy needs (62); in addition, we compared daily energy intake (kJ) against national standardized estimated energy requirements (EERs) for children (63). Each participant's final mean energy intake was then assessed against upper and lower cutoffs, calculated as 3 SDs from the mean population daily energy intake at that time point, to eliminate participants with prolonged extreme intakes.

Measuring dairy fat intake can be difficult within studies because dairy fat is included in many foods and fat content can vary across similar foods (64, 65). Erythrocyte odd-numbered SFAs, pentadecanoic acid (15:0) and heptadecanoic acid (17:0), as well as *trans*-palmitoleic acid (t16:1n-7), were investigated as dairy-specific biomarkers based on previous research studies (66–71) to provide objective verification of food record reporting.

### Statistical analyses

#### Power calculation.

For the primary outcomes, the pilot study was estimated to have the following power to detect realistic between-group changes in 40 healthy children over a 12-wk dairy intervention: 1) body fat percentage: 80% power to detect  $\geq 3.3\%$ , or 50% power to detect  $\geq 2\%$ , based on reported child study mean  $\pm$  SD

of  $19.2\% \pm 7.8\%$  (determined in 3- to 8-y-olds, using DXA) (72) and  $25.6\% \pm 4.1\%$  (in 2- to 6-y-olds, using a BodPod) (55); 2) LDL cholesterol: 80% power to detect  $\geq 0.73$  mmol/L, or 50% power to detect  $\geq 0.51$  mmol/L; and 3) HDL cholesterol: 80% power to detect  $\geq 0.27$  mmol/L, or 50% power to detect  $\geq 0.19$  mmol/L (73, 74). We therefore aimed to recruit  $\leq 55$  children, to allow for sample loss.

### Data analysis.

Clinic data were entered into spreadsheets via independent double-entry, and exported to IBM SPSS Statistics version 26.0 for Windows (2019) for all data analysis. Sociodemographic, body composition, blood test, dairy, and dietary variables were compared for between-group differences at baseline, using chi-square tests for categorical variables (reported as counts/frequencies and percentages) and independent-sample *t* tests for continuous variables (reported as mean  $\pm$  SD or mean  $\pm$  SEM). Where data were below the threshold of detection, a median midpoint value was used. Changes ( $\Delta$ ) in dependent variables (cardiometabolic endpoints, nutrients, and erythrocyte fatty acids) over the intervention were compared using paired-sample *t* tests and reported as mean  $\pm$  SEM, as per the equation:

$$3\text{-mo (time) change} = \Delta_{\text{Combined group}} (\text{final} - \text{baseline}) \quad (1)$$

Nonparametric Mann–Whitney *U* tests and Wilcoxon signed rank tests replaced independent-sample and paired-sample *t* tests, respectively, where variable distribution was not normal.

Relevant postintervention correlations are reported between promising erythrocyte fatty acid dairy biomarkers and dairy fat intakes (in grams per day and adjusted for dietary energy), for comparison with reported correlations in both intervention (75, 76) and observational dairy fat studies (70, 77, 64). Where variables were not normally distributed, Kendall's tau-beta ( $\tau$ ) was used instead of Pearson's correlation coefficient (*r*) for all bivariate correlations. In variation from the original protocol to use a repeated-measures ANOVA we used linear mixed models instead, because these are more effective in handling missing data. Analysis undertaken using repeated-measures ANOVA yielded substantively similar results. Linear mixed models in SPSS compared intervention changes ( $\Delta$ ) from baseline in cardiometabolic, nutrient, and erythrocyte fatty acid variables, producing parameter estimates (contrast hypotheses) as per the programmed equation:

$$\begin{aligned} 3\text{-mo (dairy group} \times \text{time) change} \\ = \Delta_{\text{Reduced-fat group}} (\text{final} - \text{baseline}) \\ - \Delta_{\text{Whole-fat group}} (\text{final} - \text{baseline}) \quad (2) \end{aligned}$$

The 3-mo change, or dairy group  $\times$  time interaction, reported for mixed models represents the net difference for the combined group, with each dairy group adjusted for baseline. Where group means moved in opposite directions during the intervention, such as an increase in one group and a decrease in the other, a larger net difference was produced. The final mean "direction" of change of all participants from baseline is recorded as positive or negative as per equation 2.

SPSS paired-sample analyses included only those who achieved a numerical assessment result at both time points, by

definition, but linear mixed models have the potential to allow for individual participant variables missing at 1 time point. Linear mixed models can adjust for covariates at each time point, not just at baseline, hence providing an allowance for growth effects. In addition to traditional growth variables of weight and height, we also adjusted for growth via changes in neck and waist circumference. This provided additional data on growth and on weight distribution. Where body composition-dependent variables or indexes included  $\geq 1$  growth variable (e.g., BMI includes height and weight), these were not used in growth adjustment. Because there is a possibility that type of dairy consumption may have influenced both growth rate and health markers, as a sensitivity analysis we repeated the models adjusting only for changes in height and weight. Refitting without adjustment for growth in neck or waist circumference did not affect the significance of any of the reported findings, and hence has not been reported here.

No sociodemographic variables improved the fit or significance of these models and none was included in the final models. We modeled an additional random effect to account for sibling pairs in the study; given that this did not substantively affect the parameter estimates, and the variance component associated with sibling pairs was small and nonsignificant for all key analyses, our results presented in the article do not include clustering for sibling pairs. We used a random intercept and first-order autoregressive variance structure to account for repeated measures and adjusted for fixed effects—the covariates age, sex, and the 4 growth variables—as appropriate. Values are reported as means  $\pm$  SEMs, and 95% CIs from adjusted mixed-model analyses have been included to indicate effect size for changes in primary and secondary outcomes.

The main purpose of this study was to determine if dietary guidelines should continue to recommend mainly reduced-fat dairy for children, via a real-world intervention—such as a healthy population maintaining their habitual diet. Although our investigation was an RCT, this primary purpose took precedence over determining if providing children and families with blinded dairy products would achieve certain outcomes, such as reducing children's body fat. Unforeseen considerations, such as extensive sample losses for some endpoints, in addition factored into our decision not to pursue intention-to-treat considerations, as originally intended, but to conduct a per-protocol analysis only. Although we appreciate that not all families base their dietary choices on dietary guidelines, and revisions to guidelines will not necessarily change dietary habits for all families, the study aims to contribute useful information to help inform decisions about what should be included in dietary guidelines. All tests were 2-tailed and applied a significance threshold of 0.05.

## Results

We assessed 176 applications for children to participate in the Milky Way Study, of which 119 were excluded and 8 children were withdrawn after enrolment. Randomization followed completion of the first baseline clinic. In total, 25 children were allocated to the whole-fat dairy (control) group, of which 23 completed baseline testing and 22 completed the intervention, and 24 were allocated to the reduced-fat (intervention) group,

with no losses. For the Consolidated Standards of Reporting Trials (CONSORT) flow diagram showing the Milky Way Study participant numbers for the 3-mo dairy fat intervention, see [Figure 1](#).

## Participant characteristics

The Milky Way Study included 49 children (47% girls) with a mean age of  $5.2 \pm 0.9$  y at the first baseline clinic. The mean intervention duration was  $12.3 \pm 0.9$  wk. Three children (2 girls) withdrew during the study (dropout rate of 6%), owing to assessment refusal ( $n = 1$ ) and parent burden with sample collection ( $n = 1$ ) at baseline, and refusal of further study participation ( $n = 1$ ) during the intervention. Baseline sociodemographic, clinical, and lifestyle characteristics, including key endpoint variables, are compared between dairy groups in [Table 1](#).

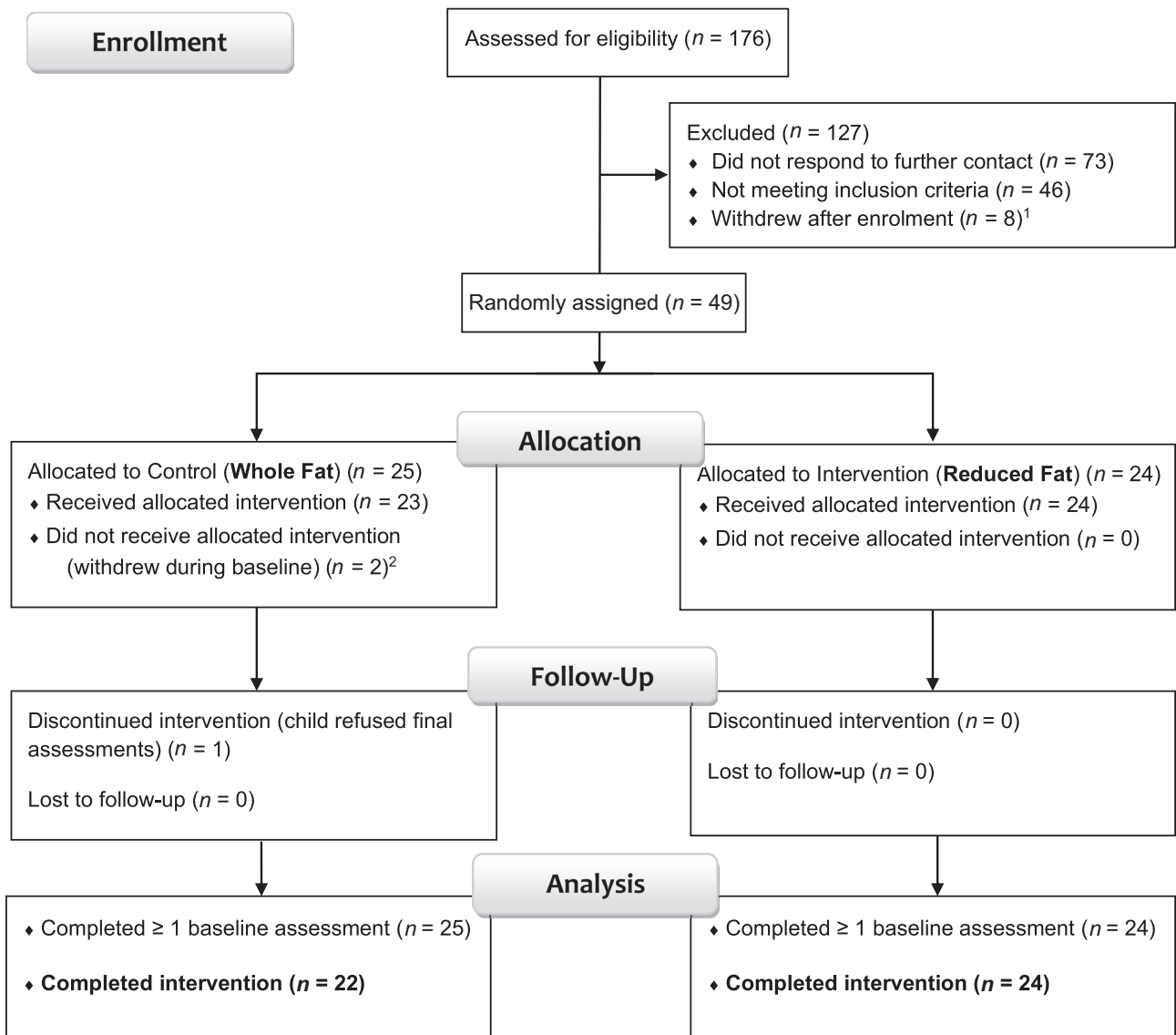
Including nonidentical twins and 2 siblings who participated at separate times owing to age constraints, 6 eligible sibling pairs participated in the study. Each child was individually randomly assigned, producing 3 pairs of siblings split between the dairy groups, as well as 2 pairs in the whole-fat dairy group and 1 pair in the reduced-fat group. Fitting models to account for any effects of first-degree relatives showed a negligible impact of variance and covariance parameters on key analysis results at the sibling pair level. In addition, close comparison of siblings' food records showed reasonable parental compliance with instructions around separate provision and recording of foods, portion sizes, and dairy foods.

## Participant compliance with the study protocol

We have previously published detailed participant compliance rates for body composition, blood pressure, and phlebotomy (47). We recruited an additional 9 children, 22.5% above the 40 required for the pilot study, to achieve adequate statistical power for our primary study outcomes. This was achieved for body fat percentage changes over the intervention ( $n = 40$ ). Despite anticipating some sample losses due to child dissent, an integral part of our child-centered approach, participant sickness and/or refusal were the major factors affecting the final outcome numbers at each time point; BodPod malfunction at the study midpoint affected mainly final assessments ( $n = 5$ ). Analysis for each endpoint variable was conducted using the full data available for that variable; however, participant numbers for intervention-related changes were particularly affected where statistical analysis excluded missing items pairwise.

All participants maintained their dairy intakes at or above the minimum requirement of 1 serving/d, based on mean dairy calcium intake of 250–300 mg/d, calculated from total dairy product supply and consumption over 3 mo (dairy nutrient data are provided in [Table 2](#); additional data on dairy product consumption are included in [Supplemental Table 1](#)). No adverse effects from the supplied dairy were reported. Childhood illnesses affected intake results variably at both time points, particularly over winter assessments. One participant was excluded from the final food record dietary analysis when illness restricted her mean energy intake over each of the 3 d to below one-quarter of her EER, with the final mean well below our cutoff





**FIGURE 1** Milky Way Study Consolidated Standards of Reporting Trials (CONSORT) flow diagram detailing study participant recruitment, random assignment, and retention pre- and postintervention. <sup>1</sup>Unanticipated family and/or child health-related issues ( $n = 6$ ); prescribed antibiotics after enrolment ( $n = 2$ ). <sup>2</sup>Parent time burden ( $n = 1$ ); child declined both primary outcome assessments (blood test and body composition analysis) ( $n = 1$ ).

for extreme intakes (population mean  $\pm 3$  SD). Water intake proved difficult to track and had to be estimated; however, milk and other drink items appeared to be appropriately reported. No participants appeared to over- or underreport intake to a degree that would have led us to exclude their reported intakes from analyses.

### Intervention cardiometabolic changes

#### *Anthropometrics and body composition.*

There were no significant between-group changes in body composition over the intervention after adjustment for growth, sex, age, and group baseline differences in linear mixed models (Table 3), as per Equation 2, provided above. Individual children's height increased by  $\leq 3.5$  cm over the 3 mo, with weight changes of  $\leq 1.7$  kg. We observed a trend toward a

differential change in the BMI percentile, with a borderline significant reduction in the whole-fat compared with the reduced-fat group [ $P$  (dairy group  $\times$  time) = 0.054].

There were no meaningful differences in changes in neck or waist circumference between the intervention and control groups in the study. Child postintervention neck circumference results correlated strongly with their BMI (Pearson's  $r = 0.70$ ;  $P < 0.001$ ), weight ( $r = 0.86$ ;  $P < 0.001$ ), waist circumference ( $r = 0.75$ ;  $P < 0.001$ ), and age (Kendall's tau-beta,  $\tau = 0.27$ ;  $P = 0.008$ ). There was a consistent ratio of  $\sim 1:2:4$  for participant neck:waist:height measurements (cm) across clinic visits.

#### *Blood pressure.*

There was no significant effect of the dairy intervention on either systolic or diastolic blood pressure (Table 4).

**TABLE 1** Comparison of Milky Way Study baseline sociodemographic, clinical, dietary, and lifestyle characteristics for children randomly assigned to the WF (control) or to the RF (intervention) dairy group<sup>1</sup>

Characteristics <sup>2</sup>	WF dairy <sup>2</sup>		RF dairy <sup>2</sup>		Comparison <sup>3</sup> <i>P</i>
	<i>n</i>	Percentage/ mean ± SD/SEM	<i>n</i>	Percentage/ mean ± SD/SEM	
Participant	25	51	24	49	
Female	13	57	10	43	
Male	12	46	14	54	
Age, y	25	5.2 ± 0.9	24	5.2 ± 0.9	0.555
Female	13	5.2 ± 0.8	10	5.1 ± 0.7	
Male	12	5.3 ± 1.0	14	5.3 ± 1.0	
Mother <sup>4</sup>	23		23		
BMI at baseline, kg/m <sup>2</sup>	21	24.8 ± 4.5	22	25.1 ± 5.0	0.821
Education	23		23		0.681
Year 12 or less	3	13.0	4	17.4	
Tertiary	20	87.0	19	82.0	
Marital status	23		23		0.406
Living with partner	1	4.3	4	17.4	
Divorced/separated	2	8.7	2	8.7	
Married	19	82.6	17	73.9	
Single	1	4.3	0	0	
Father	23		24		
BMI at baseline, kg/m <sup>2</sup>	19	26.2 ± 3.0	22	27.0 ± 3.2	0.446
Education	23		23		0.501
Year 12 or less	5	21.7	6	26.1	
Trade	1	4.3	3	13.0	
Tertiary	17	73.9	14	60.9	
Household income, AUD	21		24		0.935
<\$100,000	5	23.8	7	29.2	
\$100,000–\$150,000	7	33.3	8	33.3	
>\$150,000	5	23.8	4	16.7	
Rather not say	4	19.0	5	20.8	
Child clinical and lifestyle					
Child weight, kg	25	18.3 ± 0.6	24	19.1 ± 0.5	0.331
BMI, kg/m <sup>2</sup>	25	15.1 ± 0.2	24	15.1 ± 0.2	0.972
Body fat, %	23	20.1 ± 1.3	24	20.4 ± 1.4	0.876
Systolic BP, mm Hg	19	94 ± 3	20	100 ± 2	0.048
Diastolic BP, mm Hg	19	56 ± 1	20	58 ± 1	0.076
Total cholesterol, mmol/L	15	4.3 ± 0.1	21	4.1 ± 0.1	0.193
LDL cholesterol, mmol/L	15	2.6 ± 0.1	21	2.3 ± 0.1	0.113
HDL cholesterol, mmol/L	15	1.4 ± 0.1	21	1.5 ± 0.1	0.340
Triglycerides, mmol/L	15	0.60 [0.40, 0.70]	21	0.50 [0.40, 0.75]	0.391
Non-HDL cholesterol, mmol/L	15	2.9 ± 0.1	21	2.5 ± 0.1	0.104
Total/HDL cholesterol	15	3.1 ± 0.2	21	2.7 ± 0.1	0.078
Dietary energy, kJ/d	23	5767 ± 179	24	5628 ± 209	0.619
Dietary total fat, kJ/d	23	32.2 ± 1.4	24	34.1 ± 0.9	0.257
Dairy fat, g/d	23	15.0 [11.8, 21.3]	24	15.5 [11.1, 23.5]	0.915
Dairy fat, kJ/d	23	10.7 [8.6, 14.2]	24	10.3 [7.5, 16.4]	0.831
Outdoor activity, h/d	23	3.0 ± 1.2	24	2.6 ± 1.9	0.167
Indoor physical activity, h/d	21	2.9 ± 1.6	24	2.6 ± 1.4	0.574

<sup>1</sup>Family sociodemographic characteristics from Milky Way Study Family Questionnaires [total returned *n* = 47 (94%); 2 surveys not returned owing to child withdrawals]; some individual data items were not completed, e.g., child indoor activity was not reported for 2 children in these returned surveys. BP, blood pressure; kJ/d, percentage of total dietary kilojoules per day, or adjusted for energy intake; RF, reduced-fat; WF, whole-fat.

<sup>2</sup>Values for sociodemographic and child activity variables are percentages of the total number/counts for categorical variables, or means ± SDs for continuous variables (all included here are normally distributed). Values for child clinical and dietary variables are means ± SEMs (or medians [IQRs] where data proved not normally distributed).

<sup>3</sup>Dairy group comparisons used chi-square tests for categorical variables and independent-samples testing for continuous variables: *t* tests where data showed a normal distribution and Mann–Whitney *U* tests where data proved not normally distributed.

<sup>4</sup>Parent-reported details about participant's mother: no details provided for 1 birth mother (child adopted); 46 responses fully or partially completed.

**TABLE 2** Comparison of Milky Way Study mean daily intervention dairy product nutrient consumption (products supplied less weighed returns) by the WF (control) and the RF (intervention) dairy groups over 3 mo<sup>1</sup>

Dairy (per day) <sup>3</sup>	WF group	RF group	Group comparison <sup>2</sup>	
	Mean ± SEM	Mean ± SEM	Difference ± SEM	P
Total intake, g	432 ± 34	471 ± 46	−39 ± 58	0.613
Energy, kJ	2441 ± 197	1578 ± 131	864 ± 233	0.001
Energy, % diet kJ	41.6 ± 2.9	25.7 ± 2.1	15.8 ± 3.6	<0.001
Total fat, g	41.8 ± 3.7	16.6 ± 1.5	25.3 ± 4.0	<0.001
Total fat, % dairy kJ	62.5 ± 1.4	38.9 ± 1.4	23.6 ± 2.0	<0.001
Total fat, % diet kJ	26.1 ± 2.0	10.1 ± 0.9	16.0 ± 2.2	<0.001
Saturated fat, g	26.9 ± 2.3	10.2 ± 0.9	16.7 ± 2.5	<0.001
Saturated fat, % dairy kJ	40.3 ± 0.7	23.9 ± 0.9	16.4 ± 1.1	<0.001
Saturated fat, % diet kJ	16.8 ± 1.2	6.2 ± 0.6	10.6 ± 1.4	<0.001
Protein, g	19.2 ± 1.5	21.5 ± 1.8	−2.3 ± 2.4	0.429
Protein, % dairy kJ	13.7 ± 0.5	23.3 ± 0.6	−9.6 ± 0.8	<0.001
Protein, % diet kJ	5.7 ± 0.4	5.9 ± 0.5	−0.3 ± 0.7	0.696
Carbohydrate, g	29.8 ± 2.6	32.4 ± 2.9	−2.6 ± 3.9	0.629
Carbohydrate, % dairy kJ	20.0 ± 1.0	32.7 ± 1.2	−12.7 ± 1.5	<0.001
Carbohydrate, % diet kJ	8.2 ± 0.7	8.4 ± 0.7	−0.2 ± 1.0	0.848
Sugar, g	27.1 ± 2.5	29.4 ± 2.7	−2.3 ± 3.6	0.582
Sugar, % dairy kJ	18.2 ± 0.9	29.6 ± 1.1	−11.4 ± 1.4	<0.001
Sugar, % diet kJ	7.5 ± 0.6	7.6 ± 0.7	−0.2 ± 0.9	0.865
Calcium, mg	636 ± 49	746 ± 66	−110 ± 84	0.613
Calcium, mg/1000 kJ dairy	268 ± 10	472 ± 11	−205 ± 15	<0.001
Calcium, mg/1000 kJ diet	110 ± 8	120 ± 10	−10 ± 13	0.443
Sodium, mg	402 ± 30	388 ± 31	14.4 ± 43.3	0.253
Sodium, mg/1000 kJ dairy	268 ± 10	472 ± 11	−205 ± 15	<0.001
Sodium, mg/1000 kJ diet	69.2 ± 5.0	65.1 ± 5.6	4.1 ± 7.5	0.591

<sup>1</sup>A total of 46 participants (94%) on WF ( $n = 22$ ) or RF ( $n = 24$ ) dairy completed the 3-mo intervention. mg/1000 kJ dairy, milligrams per 1000 kJ of daily dairy intake; mg/1000 kJ diet, milligrams per 1000 kJ of daily dietary energy intake; RF, reduced-fat; WF, whole-fat; % dairy kJ, percentage of total dairy kilojoules per day (adjusted for dairy energy intake); % dietary kJ, percentage of total dietary kilojoules per day (adjusted for dietary energy intake).

<sup>2</sup>Intervention dairy product nutrient intakes over 3 mo were compared using independent-sample  $t$  tests; values are means ± SEMs. Mann-Whitney  $U$  tests were used where products were not normally distributed. Results are all considered significant where  $P$  (2-tailed) < 0.05.

<sup>3</sup>Dairy products were supplied to match usual intake, with orders adjusted fortnightly to maintain interest. Product pairs were closely matched for brand and nutrient content: fat content for WF dairy products was ~3.5 g/100 mL for milk, ~35 g/100 g for cheese, ~9 g/100 g for yogurt, and ~3.5 g/100 g for dairy dessert and custard; for RF dairy, we aimed for 2 g/100 mL for milk, 15 g/100 g for cheese, 2 g/100 g for yogurt, and 2 g/100 g for dairy dessert and custard; all creams and butters were whole fat, or 50% fat-reduced. Adjustment for dietary energy intake was derived from final 3-d food records:  $n = 21$  for the RF group ( $n = 24$  completed the intervention: 2 participants did not return food records and 1 participant was excluded when sickness over the 3 d reduced energy intake to <25% of estimated energy requirements and outside the population cutoff of mean ± 3SD);  $n = 22$  for the WF group.

### Lipid profile, glycaemic, and inflammatory markers.

There were no significant between-group differences in lipid concentrations in adjusted models over the intervention (Table 5). Both dairy groups showed a similar small, nonsignificant increase in LDL-cholesterol (+0.1 mmol/L) and decline in HDL-cholesterol concentration (−0.1 mmol/L). Seventy-six percent of children (75% at baseline; 77% postintervention) had normal-sensitivity CRP values <1 mg/L, preventing trend analysis within the healthy range (<5 mg/L). Supplemental Table 2 provides a fuller range of cardiometabolic endpoint variables.

### Intervention dietary and dairy nutrient changes

A subset of relevant nutrient intervention-related changes from weighed 3-d food records is shown in Table 6, with a fuller range of nutrients, including those contributed by dairy intake, provided in Supplemental Table 3. Headings and footnotes account for final participant numbers and missing data in each dairy group. We found a lower mean daily energy intake (15.7% at baseline;

13.5% at end of intervention) than predicted by child standardized EERs. Erythrocyte fatty acid dairy biomarkers used to assess intervention compliance have been reported below; however, analysis of individual dietary fatty acids and fat-soluble vitamins has not been included here.

Dairy fat intake was reduced by an adjusted  $12.9 \pm 4.1$  g/d (95% CI: −21.2, −4.6 g/d;  $P = 0.003$ ) in the reduced-fat compared with the whole-fat dairy group, although participant dietary energy intakes remained similar ( $P = 0.936$ ). After adjustment for dietary energy, this differential reduction amounted to  $7.7 \pm 2.2$  kJ/d (95% CI: −12.2, −3.2 kJ/d;  $P = 0.001$ ). The dairy saturated fat fraction showed similar results (adjusted 3-mo net reduction =  $8.8 \pm 2.5$  g/d; 95% CI: −14.0, −3.7 g/d;  $P = 0.001$ , or  $5.1 \pm 1.4$  kJ/d; 95% CI: −7.9, −2.3 kJ/d;  $P = 0.001$ ).

As a biomarker of dairy fat, erythrocyte pentadecanoic acid was positively associated with dairy fat intake changes over the intervention, both in grams per day (Kendall's  $\tau = 0.47$ ;  $P < 0.01$ ) and adjusted for dietary energy (Pearson's  $r = 0.53$ ;  $P < 0.01$ ). This significant response to the dairy fat intervention was confirmed in adjusted mixed models (3-mo net comparative

TABLE 3 Comparison of anthropometric and adiposity changes in the WF and RF dairy groups over the 3-mo dairy intervention<sup>1</sup>

Variable <sup>3,4,5</sup>	<i>n</i> <sup>2</sup> (paired samples)		WF group		RF group		Unadjusted test differences <sup>3,4</sup> (paired-sample tests)		Adjusted model differences <sup>5</sup> (dairy group × time)	
	WF group	RF group	WF group, mean ± SEM	RF group, mean ± SEM	Mean ± SEM	<i>P</i>	Mean ± SEM	95% CI	<i>P</i>	
Growth variables										
Weight, kg	21	24								
Baseline			18.2 ± 0.6	19.1 ± 0.5						
3-mo change			0.5 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	<0.001	—			
Height, cm	22	24								
Baseline			110.1 ± 1.3	112.1 ± 1.1						
3-mo change			2.0 ± 0.2	1.8 ± 0.2	1.9 ± 0.1	<0.001	—			
Waist circumference, <sup>4</sup> cm	22	24								
Baseline			51.3 ± 0.6	52.6 ± 0.5						
3-mo change			0.2 ± 0.4	0.1 ± 0.4	0.2 ± 0.3	0.887	—			
Neck circumference, <sup>4</sup> cm	22	24								
Baseline			25.1 ± 0.2	25.4 ± 0.3						
3-mo change			0.0 ± 0.1	0.2 ± 0.1	0.1 ± 0.1	0.116	—			
Body composition and adiposity										
Body fat % <sup>4</sup>	19	21								
Baseline			20.6 ± 1.5	20.0 ± 1.6						
3-mo change			-1.3 ± 0.8	-1.4 ± 0.7	-1.4 ± 0.5	0.032	0.0 ± 1.1	-2.2, 2.2	0.973	
BMI, kg/m <sup>2</sup>	21	24								
Baseline			15.0 ± 0.2	15.1 ± 0.2						
3-mo change			-0.1 ± 0.1	0.0 ± 0.1	-0.1 ± 0.1	0.310	0.1 ± 0.1	0.0, 0.3	0.092	
BMI percentile <sup>4</sup>	21	24								
Baseline			45.7 ± 5.7	43.2 ± 4.8						
3-mo change			-5.5 ± 2.1	0.4 ± 2.1	-1.7 ± 1.5	0.155	4.5 ± 2.2	-0.1, 9.0	0.054	
BMI z score	21	24								
Baseline			-0.12 ± 0.22	-0.01 ± 0.18						
3-mo change			-0.02 ± 0.09	0.13 ± 0.08	0.06 ± 0.06	0.302	0.15 ± 0.09	-0.03, 0.32	0.099	
FMI, <sup>4</sup> kg/m <sup>2</sup>	19	21								
Baseline			3.1 ± 0.2	3.0 ± 0.2						
3-mo change			-0.2 ± 0.1	-0.2 ± 0.1	-0.2 ± 0.1	0.030	0.0 ± 0.2	-0.3, 0.4	0.767	
LMI, kg/m <sup>2</sup>	19	21								
Baseline			11.9 ± 0.2	12.1 ± 0.3						
3-mo change			0.1 ± 0.1	0.2 ± 0.1	0.1 ± 0.1	0.075	0.1 ± 0.2	-0.2, 0.4	0.573	

<sup>1</sup>FMI, fat mass index; LMI, lean mass index; RF, reduced-fat; WF, whole-fat.

<sup>2</sup>Sample losses: of 49 children that attended the baseline clinic visit and 46 that attended the final clinic (94% of the total) provided most anthropometric readings; BodPod malfunction at study midpoint (4 wk awaiting repairs) prevented measurement of 2 children at baseline (including 1 that dropped out) and 5 children at end of intervention, reducing the number of paired samples to 40 (82% of the total).

<sup>3</sup>Unadjusted paired differences: paired-samples *t* tests were used to compare dairy group intervention changes where the data set was normally distributed. Differences are considered significant where  $P < 0.05$ .  
<sup>4</sup>Unadjusted paired differences: Wilcoxon signed rank tests were used to compare dairy group intervention changes where the data set was not normally distributed. Differences are considered significant where  $P < 0.05$  (asymptotic; 2-sided test). The *P* value for the appropriate test has been reported in each case. Variables are reported in the same format throughout, to ensure consistency across different models.

<sup>5</sup>Adjusted model differences: linear mixed models were used to adjust for fixed confounders in dairy group comparisons (no random factors achieved significance or improved the fit of any of the models), using contrast hypotheses (parameter estimates) to estimate group variable changes over the intervention. Each variable was adjusted for sex, age changes (incorporating individual length of intervention, between 11.5 and 15 wk), and child growth over the intervention. Child growth variables that achieved significance in models included changes in height, weight, neck circumference, and waist circumference: the latter 2 were considered to better allow for adipose tissue deposition (or loss) during growth spurts. Covariates in the model: mean age over the intervention = 5.4 y; mean weight over the intervention = 19.0 kg; mean height over the intervention = 111.9 cm; mean waist circumference over the intervention = 52.0 cm; and mean neck circumference over the intervention = 25.3 cm. Linear mixed models do not necessarily remove participants missing either initial or final test results. Significant differences are indicated where  $P$  (dairy group × time) < 0.05.

**TABLE 4** Comparison of BP changes in the WF and RF dairy groups over the 3-mo dairy intervention<sup>1</sup>

Variable <sup>3</sup>	<i>n</i> <sup>2</sup> (paired samples)		WF group, mean ± SEM	RF group, mean ± SEM	Unadjusted test differences <sup>3</sup> (paired-sample tests)		Adjusted model differences <sup>4</sup> (dairy group × time)		
	WF	RF			Mean ± SEM	<i>P</i>	Mean ± SEM	95% CI	<i>P</i>
Systolic BP, mm Hg	17	18							
Baseline			93 ± 3	100 ± 2					
3-mo change			3 ± 2	−1 ± 2	1 ± 1	0.677	−3 ± 2	−8, 2	0.239
Diastolic BP, mm Hg	17	18							
Baseline			55 ± 1	58 ± 1					
3-mo change			2 ± 1	−1 ± 1	0 ± 1	0.646	−2 ± 2	−6, 2	0.251

<sup>1</sup>BP, blood pressure; RF, reduced-fat; WF, whole-fat.

<sup>2</sup>Sample losses: of 49 children that attended the baseline clinic visit and 46 that attended the final clinic (94%; 3 dropped out before the final clinic), *n* = 43 children (88% of the total) provided ≥2 BP readings; 8 of these (4 each at baseline and postintervention) provided ≥2 readings at only a single time point, reducing the number of paired samples to 35 (71% of the total); child dissent was responsible for all sample losses.

<sup>3</sup>Unadjusted paired differences: paired-samples *t* tests were used to compare dairy group intervention changes where the data set was normally distributed. Differences are considered significant where *P* < 0.05.

<sup>4</sup>Adjusted model differences: linear mixed models were used to adjust for fixed confounders in dairy group comparisons (no random factors achieved significance or improved the fit of any of the models), using contrast hypotheses (parameter estimates) to estimate group variable changes over the intervention. Each variable was adjusted for sex, age changes (incorporating individual length of intervention, between 11.5 and 15 wk), and child growth over the intervention. Child growth variables that achieved significance in models included changes in height, weight, neck circumference, and waist circumference: the latter 2 were considered to better allow for adipose tissue deposition (or loss) during growth spurts. Covariates in the model: mean age over the intervention = 5.4 y; mean weight over the intervention = 19.0 kg; mean height over the intervention = 111.9 cm; mean waist circumference over the intervention = 52.0 cm; and mean neck circumference over the intervention = 25.3 cm. Linear mixed models do not necessarily remove participants missing either initial or final test results. Significant differences are indicated where *P* (dairy group × time) < 0.05.

reduction in reduced-fat group =  $-0.036\% \pm 0.008\%$ ; 95% CI:  $-0.053\%, -0.018\%$ ; *P* < 0.001). A smaller, nonsignificant association of heptadecanoic acid with dairy fat intake (*r* = 0.18) achieved borderline significance for the adjusted intervention dairy group × time interaction (3-mo net comparative reduction in reduced-fat group =  $-0.024\% \pm 0.012\%$ ; 95% CI:  $-0.049\%, 0.001\%$ ; *P* = 0.055). We were unable to distinguish *trans*-palmitoleic acid from the *cis*-isoform. (Data for these dairy biomarkers are not included in either the article or the supplemental tables.)

The reduced-fat dairy group increased their intake of dietary sodium over the dairy intervention by  $301 \pm 109$  mg/d. After adjustment for dietary energy (mg Na/1000 kJ), this amounted to a substantial increase in sodium intake compared with the whole-fat dairy group (for dairy group × time, adjusted 3-mo net difference =  $54 \pm 19$  mg/1000 kJ; *P* = 0.008). When adjusted further for intake changes in dairy sodium, the reduced-fat dairy group effectively increased their dietary sodium by 241 mg/d (15.2%) from baseline, amounting to an additional 44 mg/1000 kJ (15.6%) consumed per day compared with the whole-fat group (baseline mean sodium intake:  $1583 \pm 53$  mg/d or  $282 \pm 9$  mg/1000 kJ; *n* = 47; these baseline population mean values are not included in either the article or the supplemental tables).

## Discussion

Our RCT provided no evidence for differential effects of whole-fat compared with reduced-fat dairy on measures of adiposity or cardiometabolic risk factors in healthy 4- to 6-y-old children over 3 mo, despite substantially increased intake of core dairy products. For adiposity measures, 2 previous interventions to reduce dairy fat in habitual diet in schoolchildren similarly found no significant differences between intervention and control

groups for body weight, BMI, or waist circumference in 145 Australian 4- to 13-y-olds (65), or in indigenous Mexican 6- to 16-y-old school boarders drinking reduced-fat (*n* = 180) or skim milk (*n* = 148), compared with whole-milk controls (*n* = 134) (78). Our RCT shows in addition no significant differences in body fat percentage or FMI, all in direct opposition to the so-called “adiposity hypothesis.” Our data are further supported by the observational evidence. A systematic review and meta-analysis of observational studies reported lower odds of child overweight or obesity (OR: 0.61; 95% CI: 0.52, 0.72; *P* < 0.0001) with regular intake of whole-fat milk (41); a systematic review of observational and intervention studies found no associations between whole-fat dairy products and increased child body weight or adiposity (42), while a comprehensive analysis of the evidence-base in children found little evidence for the influence of dairy fat content on body fatness (43).

Furthermore, cross-sectional (79, 80) and prospective cohort (81) studies have shown that 2- to 6-y-old children with (severe) obesity were less likely to consume whole milk than those in the healthy weight range (adjusted OR: 0.77; 95% CI: 0.60, 0.98; *P* = 0.031) (81); this effect was repeated in cohort studies of overweight or obese participants aged 2–20 y (81–83). Researchers have questioned whether this relation could be bidirectional, causal, or due to public health recommendations affecting parental choices around supply (81). Our RCT recruited healthy young children consuming mostly whole-fat dairy. However, we noted a trend toward a lower BMI percentile in the whole-fat relative to the reduced-fat group (*P* = 0.054), which matches emerging observational evidence (41, 81) questioning the recommendation to provide reduced-fat dairy to healthy children. In adults, whole-fat compared with reduced-fat dairy has been associated with neutral or beneficial effects on body composition in intervention studies (84–86), and in a systematic review (37) and a meta-analysis (2) of observational studies,

**TABLE 5** Comparison of fasting serum lipid profile and glycemic changes in the WF and RF dairy groups over the 3-mo dairy intervention<sup>1</sup>

Variable <sup>3,4,5</sup>	<i>n</i> <sup>2</sup> (paired samples)		WF group, mean ± SEM		RF group, mean ± SEM		Unadjusted test differences <sup>3,4</sup> (paired-sample tests)		Adjusted model differences <sup>5</sup> (dairy group × time)			
	WF	RF	WF	RF	WF	RF	Mean ± SEM	<i>P</i>	Mean ± SEM	95% CI	<i>P</i>	
Total cholesterol, mmol/L	13	16										
Baseline			4.3 ± 0.1		4.0 ± 0.2							
3-mo change			0.0 ± 0.1		0.1 ± 0.1		0.745	0.0 ± 0.1	0.0 ± 0.2	-0.3, 0.4	0.793	
LDL cholesterol, mmol/L	13	16										
Baseline			2.6 ± 0.1		2.3 ± 0.2							
3-mo change			0.1 ± 0.1		0.1 ± 0.1		0.192	0.1 ± 0.1	0.1 ± 0.1	-0.2, 0.4	0.656	
HDL cholesterol, mmol/L	13	16										
Baseline			1.5 ± 0.1		1.5 ± 0.1							
3-mo change			-0.1 ± 0.1		-0.1 ± 0.0		0.057	-0.1 ± 0.0	0.0 ± 0.1	-0.2, 0.1	0.809	
Non-HDL cholesterol, mmol/L	13	16										
Baseline			2.9 ± 0.2		2.5 ± 0.2							
3-mo change			0.1 ± 0.1		0.1 ± 0.1		0.230	0.1 ± 0.1	0.1 ± 0.2	-0.3, 0.4	0.726	
Total:HDL cholesterol ratio	13	16										
Baseline			3.1 ± 0.2		2.7 ± 0.2							
3-mo change			0.2 ± 0.1		0.2 ± 0.1		0.044	0.2 ± 0.1	0.0 ± 0.2	-0.3, 0.4	0.844	
Triglycerides, <sup>4</sup> mmol/L	13	16										
Baseline			0.65 ± 0.07		0.51 ± 0.05							
3-mo change			-0.02 ± 0.07		0.02 ± 0.06		0.963	0.00 ± 0.05	-0.04 ± 0.10	-0.24, 0.16	0.693	
Fasting glucose, <sup>4</sup> mmol/L	13	15										
Baseline			4.6 ± 0.1		4.6 ± 0.1							
3-mo change			0.0 ± 0.1		-0.2 ± 0.2		0.583	-0.1 ± 0.1	-0.1 ± 0.2	-0.5, 0.2	0.411	
HbA1c, %	13	14										
Baseline			5.2 ± 0.0		5.0 ± 0.1							
3-mo change			0.0 ± 0.0		0.0 ± 0.0		0.908	0.0 ± 0.0	0.0 ± 0.1	-0.1, 0.2	0.494	

<sup>1</sup>HbA1c, glycated hemoglobin; RF, reduced-fat; WF, whole-fat.

<sup>2</sup>Sample losses: of 47 children that attended the baseline phlebotomy clinic visit (94%; 2 dropped out before this clinic and 1 during the intervention), *n* = 38 children (78% of the total) provided fasting blood samples for successful analysis; 9 of these (7 at baseline; 2 postintervention) provided a usable sample at only a single time point, reducing the number of paired samples to 29 (59%); inadequate samples or sample spoilage/clotting further reduced the number of sample pairs for fasting blood glucose and HbA1c tests.

<sup>3</sup>Unadjusted paired differences: paired-samples *t* tests were used to compare dairy group intervention changes where the data set was normally distributed. Differences are considered significant where *P* < 0.05.

<sup>4</sup>Unadjusted paired differences: Wilcoxon signed rank tests were used to compare dairy group intervention changes where the data set was not normally distributed. Differences are considered significant where *P* < 0.05 (asymptotic; 2-sided test). The *P* value for the appropriate test has been reported in each case. Variables are reported in the same format throughout, to ensure consistency across different models.

<sup>5</sup>Adjusted model differences: linear mixed models were used to adjust for fixed confounders in dairy group comparisons (no random factors achieved significance or improved the fit of any of the models), using contrast hypotheses (parameter estimates) to estimate group variable changes over the intervention. Each variable was adjusted for sex, age changes (incorporating individual length of intervention, between 11.5 and 15 wk), and child growth variables that achieved significance in models included changes in height, weight, neck circumference, and waist circumference; the latter 2 were considered to better allow for adipose tissue deposition (or loss) during growth spurts. Covariates in the model: mean age over the intervention = 5.4 y; mean weight over the intervention = 19.0 kg; mean height over the intervention = 111.9 cm; mean waist circumference over the intervention = 52.0 cm; and mean neck circumference over the intervention = 25.3 cm. Linear mixed models do not necessarily remove participants missing either initial or final test results. Significant differences are indicated where *P* (dairy group × time) < 0.05.

TABLE 6 Comparison of group mean daily dietary and dairy product nutrient intakes over the dairy intervention, using data from weighed 3-d food records<sup>1</sup>

Dietary and dairy nutrients <sup>2,3,4</sup>	WF dairy group, <sup>5</sup>		RF dairy group, <sup>5</sup>		Unadjusted test differences <sup>2,3</sup> (paired-sample tests)		Adjusted model differences (dairy group × time) <sup>4</sup>	
	mean ± SEM	mean ± SEM	Mean ± SEM	P	Mean ± SEM	95% CI	P	
Estimated energy requirements, <sup>3</sup> kJ/d								
Baseline	6710 ± 114	6904 ± 103						
3-mo change	75 ± 14	64 ± 25	69 ± 14	<0.001	—			
Dietary energy, kJ/d								
Baseline	5761 ± 187	5730 ± 229						
3-mo change	218 ± 284	138 ± 192	179 ± 171	0.302	-28 ± 347	-729, 672	0.936	
Core dairy, <sup>3,6</sup> servings/d								
Baseline	1.6 ± 0.2	1.6 ± 0.2						
3-mo change	0.3 ± 0.2	0.6 ± 0.1	0.4 ± 0.1	0.001	0.2 ± 0.2	-0.2, 0.7	0.240	
Total fat, <sup>3</sup> g/d								
Baseline	50.0 ± 3.1	53.0 ± 2.7						
3-mo change	8.4 ± 3.3	-6.5 ± 2.5	1.1 ± 2.3	0.913	-14.5 ± 4.1	-22.8, -6.2	0.001	
Dairy total fat, <sup>3</sup> g/d								
Baseline	19.3 ± 2.7	19.0 ± 2.8						
3-mo change	6.9 ± 3.3	-6.4 ± 2.2	0.4 ± 2.2	0.668	-12.9 ± 4.1	-21.2, -4.6	0.003	
Total fat, % dietary kJ/d								
Baseline	32.2 ± 1.5	34.2 ± 1.1						
3-mo change	3.6 ± 1.1	-5.1 ± 0.9	-0.6 ± 1.0	0.518	-8.5 ± 1.4	-11.3, -5.7	<0.001	
Dairy total fat, <sup>3</sup> % dietary kJ/d								
Baseline	12.3 ± 1.5	11.9 ± 1.4						
3-mo change	3.5 ± 1.8	-3.9 ± 1.2	-0.1 ± 1.2	0.562	-7.7 ± 2.2	-12.2, -3.2	0.001	
Saturated fat, <sup>3</sup> g/d								
Baseline	21.9 ± 1.6	23.5 ± 1.5						
3-mo change	5.8 ± 1.7	-2.7 ± 1.4	1.6 ± 1.3	0.391	-8.3 ± 2.2	-12.8, -3.8	0.001	
Dairy saturated fat, <sup>3</sup> g/d								
Baseline	12.3 ± 1.7	12.2 ± 1.8						
3-mo change	4.4 ± 2.0	-4.6 ± 1.4	0.0 ± 9.2	0.507	-8.8 ± 2.5	-14.0, -3.7	0.001	
Dairy saturated fat, <sup>3</sup> % dietary kJ/d								
Baseline	7.8 ± 0.9	7.7 ± 0.9						
3-mo change	2.3 ± 1.1	-2.8 ± 0.8	-0.1 ± 1.2	0.440	-5.1 ± 1.4	-7.9, -2.3	0.001	
Calcium, <sup>3</sup> mg/d								
Baseline	727 ± 62	704 ± 60						
3-mo change	62 ± 54	195 ± 51	127 ± 38	0.003	125 ± 71	-18, 267	0.085	
Calcium, <sup>3</sup> mg · 1000 kJ <sup>-1</sup> · d <sup>-1</sup>								
Baseline	126 ± 10	123 ± 9						
3-mo change	5 ± 9	31 ± 10	18 ± 7	0.005	25 ± 13	-1, 52	0.059	

(Continued)

**TABLE 6** (Continued)

Dietary and dairy nutrients <sup>2,3,4</sup>	WF dairy group, <sup>5</sup> mean ± SEM		RF dairy group, <sup>5</sup> mean ± SEM		Unadjusted test differences <sup>2,3</sup> (paired-sample tests)		Adjusted model differences (dairy group × time) <sup>4</sup>		
	Mean ± SEM	P	Mean ± SEM	P	Mean ± SEM	95% CI	P		
Sodium, mg/d									
Baseline	1537 ± 64		1639 ± 96						
3-mo change	57 ± 118		301 ± 109	0.037	176 ± 82	284 ± 152	-23, 591	0.069	
Sodium, mg · 1000 kJ <sup>-1</sup> · d <sup>-1</sup>									
Baseline	270 ± 12		289 ± 15						
3-mo change	-6 ± 14		45 ± 15	0.097	19 ± 11	54 ± 19	14, 93	0.008	

<sup>1</sup>g (or mg or μg) · 1000 kJ<sup>-1</sup> · d<sup>-1</sup>, grams (or milli- or micrograms) of each nutrient per 1000 kJ dietary energy consumed that day; RF, reduced-fat; WF, whole-fat; % dietary kJ/d, percentage of total dietary energy intake (kJ) consumed per day.

<sup>2</sup>Unadjusted test differences: paired-samples *t* tests were used to compare dairy group intervention changes where the data set was normally distributed. Differences are considered significant where *P* < 0.05.

<sup>3</sup>Unadjusted test differences: Wilcoxon signed rank tests were used to compare dairy group intervention changes where the data set was not normally distributed. Differences are considered significant where *P* < 0.05 (asymptotic; 2-sided test). The *P* value for the appropriate test has been reported in each case. Variables are reported in the same format throughout, to ensure consistency across different models.

<sup>4</sup>Adjusted model differences: linear mixed models were used to adjust for fixed effects (confounders) in dairy group comparisons (no random effects achieved significance or improved the fit of any of the models), using contrast hypotheses (parameter estimates) to estimate group variable changes over the intervention. Each variable was adjusted for sex, age changes (incorporating individual length of intervention, between 11.5 and 15 wk), and child growth over the intervention. Child growth variables that achieved significance in models included changes in height, weight, neck circumference, and waist circumference; the latter 2 were considered to better allow for adipose tissue deposition (or loss) during growth spurts. Covariates in the model are evaluated as the mean of the baseline and final variable values; mean age over the intervention = 5.4 y; mean weight over the intervention = 19.0 kg; mean height over the intervention = 111.9 cm; mean waist circumference over the intervention = 52.0 cm; and mean neck circumference over the intervention = 25.3 cm. Linear mixed models do not necessarily remove participants missing either initial or final test results. Significant differences are indicated where *P* (dairy group × time) < 0.05.

<sup>5</sup>Sample size and losses for 3-d food records: of 49 children in the study, 47 (96%) completed baseline 3-d food records (WF dairy group: *n* = 23, with 2 dropouts after the first clinic visit; RF dairy group: *n* = 24, with no losses); 43 children (88% of the total) provided a paired 3-d record postintervention [WF dairy group: *n* = 22, with 1 dropout; RF dairy group: *n* = 21, with 2 unreturned records and 1 participant excluded for mean daily energy intake < 25% of estimated energy requirements over all 3 d and 3-d mean energy outside population cutoffs (mean ± 3 SD) owing to sickness].

<sup>6</sup>Core dairy servings include daily servings of milk, cheese, and yogurt only. Butter and cream are generally not considered dairy foods in Australian public health guidelines.



although some interventions have found adverse effects on body weight where energy intake is ad libitum (87, 88).

We found no significant effect of the dairy intervention on blood pressure. Our mean results were similar to the relevant 50th percentile ranges established for nonoverweight children ( $n = 13,547$ ; age 2–9 y) participating in the European multicenter IDEFICS (Identification and prevention of Dietary- and lifestyle-induced health Effects In Children and infantS) study (59). In male adolescents, diastolic blood pressure was inversely associated with both types of dairy (89). In adult observational and intervention studies, neither systolic nor diastolic blood pressure has been associated with increased intake of dairy products (5, 12, 90) or dairy fat (84, 86, 91).

Fasting serum glucose and lipids in both Milky Way Study groups were not differentially affected by the dairy intervention: not even a trend was apparent. Likewise, in an Australian child cluster education intervention (65), reducing dairy fat did not significantly affect adjusted serum lipids after 12 wk, while in Mexican boarding schools (78), changing to reduced-fat or skim milk for 4 mo did not significantly affect serum triglycerides or total:HDL cholesterol ratios, because both total- and HDL-cholesterol concentrations reduced (42). In contrast, a significant relative change in LDL ( $-0.28$  mmol/L) in the skim milk group (78) may have been influenced by intervention-related dietary changes, as explored below. The observational evidence-base (92) also largely shows that saturated fat, dairy fat, or high-fat core dairy foods are not detrimentally associated with CVD risk, risk indexes, or proxy markers (particularly LDL) in adults (37, 90, 93), in adolescents (89), and in children (1–16 y old) (94).

Our study demonstrated significant changes in dairy fat consumption over 3 mo, supported by significant correlations of erythrocyte pentadecanoic acid with dairy fat intake. Despite this, daily energy intakes remained similar. This finding matches that from the child cluster intervention to reduce dairy fat or screen time (65). However, adults with MetS ( $n = 72$ ) supplied with 3.3 servings of whole- or reduced-fat dairy per day increased their dietary energy compared with limited dairy consumption ( $P < 0.001$ ), suggesting relative failure to balance ad libitum energy intake (87). Whereas young children generally do eat according to their energy needs, environmental and social factors can affect intake in older children (95). In the home food choices are limited to what parents provide, and depend largely on cost, convenience, and perceived health benefits (96). Changing 1 aspect of a diet can have ramifications for overall diet quality (97): for example, in Mexican boarding schools children randomly assigned to reduced-fat milk increased intake of daily tortillas by 26%, while those on skim milk ate 57% more tortillas than the control group (78). Although evaluation of food group changes was not an aim of our study, the reduced-fat group increased their dietary sodium intake by 241 mg/d, or an additional 44 mg Na for each 1000 kJ consumed, after adjustment for small changes in the whole-fat dairy group and in total dairy intake. This amounted to a substantial comparative increase in this group of 15.2% from the mean population baseline, or 15.6% higher when adjusted for dietary energy intake.

As a double-blind RCT, our comprehensive pilot study has many notable strengths. Our provision of blinded dairy products at no cost to participants removed cost obstacles toward compliance, and the design of the study helped limit researcher and family bias. Another important strength of our study was the

use of repeated 3-d weighed food records, with verification by a dietitian, as well as erythrocyte fatty acid analysis to validate group differences in dairy fat intake. We recruited healthy, routine consumers of whole-fat dairy to extend the transferability of our research to the larger population of healthy children. To allow for rapid and individual child growth spurts, we adjusted statistical models for changes in waist and neck circumference, as well as height and weight, as significant indicators of childhood growth and distribution of body weight between adipose and lean tissue. Sensitivity analysis to refit the models without adjustment for growth in neck or waist circumference did not affect the significance of our reported findings.

We note some important limitations. Three months was potentially inadequate to detect all effects of differential dairy fat intake. The pilot study was not powered to detect small changes in main outcomes. Although we took care to follow a child-centered approach (47), blood draws were potentially distressing for children. We respected children's decisions to dissent to assessments. This contributed to a lack of complete data, particularly for serum-based risk biomarkers. Hence, our null results may have been partly due to a lack of power. Lack of an intent-to-treat analysis in our RCT may be considered a limitation. However, given our primary goal was to inform dietary guidelines, our focus was on the biological effects of actual consumption of reduced- compared with whole-fat dairy foods on healthy children. Loss of sample size for serum biomarker results added extra information to inform our decision to conduct statistical analyses on a per-protocol basis only, because we were unable to analyze the full randomized groups for some endpoints. However, because we could not find even the slightest trend for a differential change for serum lipids and glucose, it is unlikely that a lack of power or of intent-to-treat analyses caused us to miss major diet effects on these endpoints.

Future research following a larger number of children over a longer time would further strengthen our findings; in addition, this could investigate whether different dairy products have different effects on study endpoints. Given cost considerations, however, a community-based setting where advice is given on the types of dairy to consume (similar to the dietary guidelines), rather than providing dairy, might prove more feasible, less prohibitively expensive, and better reflect the real-world setting. We recommend future research continue to use child-friendly principles to provide a positive participant experience, and make appropriate allowances for equipment malfunction.

In conclusion, our results suggest that healthy children can safely consume whole-fat dairy products without increased adiposity or adverse cardiometabolic effects. With consideration of our results and previous research, future revisions of dietary guidelines should consider recommending that children can consume either whole-fat or reduced-fat dairy: this would help simplify parental dairy choices and child health concerns. To our knowledge, our study is the first such dairy intervention in preadolescent children, and our findings support the accumulated evidence that public health policy encouraging reduced-fat dairy after 2 y of age is unlikely to prevent or reduce childhood obesity or excess adiposity, or improve biomarkers of cardiometabolic disease risk.

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## Data Availability

An anonymized data set including all data described in the article, code book, and analytic code will be made available upon request to the Chief Investigator (TAOS).

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