The Journal of Nutrition xxx (xxxx) xxx

THE JOURNAL OF NUTRITION

journal homepage: www.journals.elsevier.com/the-journal-of-nutrition

Nutrition and Disease

Dairy Product Consumption and Incident Prediabetes in the Australian Diabetes, Obesity, and Lifestyle Study With 12 Years of Follow-Up

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A B S T R A C T

Background: Investigating modifiable risk factors for the early stages of the development of type 2 diabetes is essential for effective prevention. Some studies show protective associations between dairy and prediabetes; however, associations are heterogeneous by the type and fat content of dairy foods.

Objective: To examine the relationship between the consumption of dairy, including different types of dairy products and risk of prediabetes.

Methods: The study included 4891 participants with normal glucose tolerance (aged 49.0 ± 12.3 y, 57% female) of the Australian Diabetes, Obesity, and Lifestyle (AusDiab) study, a longitudinal population-based study. Dairy intake was measured at baseline using a food frequency questionnaire. Prediabetes at the 5-y and 12-y follow-ups was defined according to the WHO criteria as fasting plasma glucose levels of 110–125 mg/dL or 2-h plasma glucose levels of 140–199 mg/dL. Associations were analyzed using Poisson regression, adjusted for social demographics, lifestyle behaviors, a family history of diabetes, and food group intake.

Results: In total, 765 (15.6%) incident cases of prediabetes were observed. The mean intake of dairy foods was 2.4 ± 1.2 servings/d, mostly consisting of low-fat milk (0.70 ± 0.78 servings/d) and high-fat milk (0.47 ± 0.72 servings/d). A higher intake of high-fat dairy ($RR_{servings/d}$: 0.92; 95% CI: 0.85, 1.00), high-fat milk (0.89; 95% CI: 0.80, 0.99), and total cheese (0.74; 95% CI: 0.56, 0.96) was associated with a lower risk of prediabetes. Low-fat milk intake was associated nonlinearly with prediabetes risk. Low-fat dairy foods, total milk, yogurt, low-fat cheese, and ice cream were not associated with prediabetes risk.

Conclusion: In this large Australian cohort, protective associations were found for high-fat dairy types, whereas neutral associations were seen for low-fat dairy types. Studies with more detail on sugar content of types of dairy foods and products eaten with dairy foods (e.g., cereals or jam), and studies into potential causal mechanisms of the health effects of dairy intake are required.

Keywords: dairy, milk, impaired fasting glucose, impaired glucose tolerance, nutritional epidemiology

Introduction

Prediabetes is defined as the intermediate stage between normal glucose tolerance (NGT) and type 2 diabetes, including impaired fasting glucose and/or impaired glucose tolerance [1]. People in this early risk stage of type 2 diabetes already display metabolic disturbances and are prone to develop microvascular and macrovascular complications [2–5]. The prevalence of prediabetes is increasing worldwide [6,7]; in particular, a high prevalence is observed in people with obesity and of older age [8]. Prevention is needed because a significant proportion of people with prediabetes will develop type 2 diabetes over time (cumulative incidence of 9%–84%, depending on the follow-up duration and prediabetes definition) [9], and the incidence of cardiovascular disease among people with prediabetes is substantial [relative risk (RR): 1.15; 95% CI: 1.11, 1.18 compared with NGT] [5]. Furthermore, people with prediabetes may revert to NGT [9,10]. Lifestyle modification is the recommended

Abbreviations: CVD, cardiovascular disease; FPG, fasting plasma glucose; HDL, high-density lipoprotein; 2hPG, 2-hour glucose levels; LDL, low-density lipoprotein; NGT, normal glucose tolerance; RCT, randomized controlled trial; WC, waist circumference.

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https://doi.org/10.1016/j.tjnut.2023.03.032

Received 20 January 2023; Received in revised form 7 March 2023; Accepted 28 March 2023; Available online xxxx

approach to prevent and treat prediabetes, and its effectiveness has been shown in randomized controlled trials (RCTs) [11,12].

Dairy food is a key component in many diets and has, therefore, received ample attention in the literature. Nevertheless, the health effects of dairy foods are heterogeneous, partly underlined by differences in study populations and variations in consumed dairy types [13,14]. Few prospective cohort studies have presented associations between dairy intake and incident prediabetes. In the Framingham Offspring Cohort (FHS-OC) (n = 1867, 12-y of follow-up), total, low-fat, and high-fat dairy were associated with a 39%, 32%, and 25% lower risk of incident prediabetes, respectively, for the top compared with the bottom intakes [15]. In addition to this study in the United States, our research group conducted analyses in 2 large Dutch cohort studies because dairy consumption is much higher and more widespread in the Netherlands compared with that in other countries. In the Hoorn studies (pooling 2 cohorts, n = 2262, 6.4-y of follow-up), high-fat fermented dairy, total cheese, and high-fat cheese were associated with a lower risk of prediabetes, but total dairy and other types of dairy were not associated with prediabetes [16]. On the contrary, in the Rotterdam studies (pooling 3 cohorts, n = 6053, 11.4-y of follow-up), high-fat yogurt and high-fat milk intake were strongly associated with a lower prediabetes risk, but low-fat dairy and low-fat milk were associated with a higher prediabetes risk [17].

In a previous analysis of the Australian Diabetes, Obesity, and Lifestyle (AusDiab) study (n = 5582, 5-y follow-up), a nonsignificant association of total dairy and type 2 diabetes incidence was found (OR: 0.71; 95% CI: 0.48, 1.05 for the third compared with the first tertile) [18]. Low-fat milk was significantly associated with lower odds of diabetes incidence (OR: 0.65; 95% CI: 0.44, 0.94), whereas there was no association with full-fat milk, yogurt, and cheese. The relationship between dairy intake and prediabetes has not yet been investigated in the AusDiab population. Investigating this early stage is essential because potential associations between dairy and prediabetes have important implications for effective early-stage prevention of diabetes and cardiovascular disease.

Therefore, the aim of this study was to examine the relationship between the consumption of dairy, including different types of dairy products, and prediabetes risk in the nationwide, on a population-based longitudinal AusDiab study with 12 y of follow-up.

Methods

The AusDiab study is a national, population-based survey of 11,247 adults aged older than 25 y in 1999–2000, with follow-up measurements in 2004–2005 and 2011–2012; details are described previously [19]. The AusDiab study aimed to provide national benchmark data on the prevalence, incidence and risk factors of diabetes, obesity, hypertension, and kidney disease in Australia. In short, a stratified cluster sample was drawn from 42 randomly selected census collector districts across Australia, including mostly participants with an Australian, New Zealand or British background (85%).

Study participants

Participants were interviewed at home after which they received a biomedical examination at the center. All eligible participants were invited to attend the follow-up measurement, excluding those who were deceased, had moved overseas or into a nursing facility classified for high care, or had a terminal illness. Of those completing the baseline household interview, 55% completed the biomedical examination. Differences between responders and nonresponders have been described previously [19]. The baseline measurements were repeated at 5 y (response rate 0.6% of eligible participants) and 12 y of follow-up (response rate, 59.8% of eligible participants) [20,21,66]. The study was approved by the human research ethics committee of the International Diabetes Institute, and the Alfred Hospital (Melbourne, Australia). All participants provided written informed consent.

For this analysis, we excluded pregnant participants (n = 60), participants with missing baseline dietary data (n = 203), or those with implausible energy intakes (defined as men <800 or >4200 kcal, women <500 or >3500 kcal) (n = 276) [22,23] (Supplemental Figure 1). Furthermore, we excluded participants with prediabetes or diabetes at baseline (n = 2823) or with missing information related to glucose testing at baseline (n = 94), resulting in 7791 participants without prediabetes or diabetes at baseline. Participants with complete follow-up information for the 5-y and/or 12-y examination and without diabetes or missing prediabetes information at the follow-up were included in the analysis, resulting in an analytical sample of 4891 participants.

Assessment of dairy intake

Dietary intake in the last 12 mo was measured without the interviewer's assistance using a self-administered 74-item FFQ designed by the Cancer Council Victoria [24]. This FFQ was compared with 7-d weighted food records in 63 women of childbearing age, with correlation coefficients of 0.39 for protein, 0.64 for saturated fat, and 0.30 for sodium. Diet quality was measured with the food-based Australian Guideline Index, comprising 15 items, with scores ranging from 0 to 150, with higher scores reflecting better adherence to dietary guidelines [25]. Questions on dairy intake included milk (full-fat milk, reduced fat milk, and skimmed milk), flavored milk, yogurt, cheese (hard cheese, firm cheese, soft cheese, low-fat cheese, ricotta/cottage cheese, and cream cheese), and ice cream. For milk intake, participants were asked to report quantity of intake per day (from "none" to "3 cups or more"). For other dairy food types, participants were asked to select from 10 frequency responses ("never" to "3 or more times per day") for each item on the FFQ. Daily intakes in grams were calculated using sex-specific standard portion sizes derived from weighted food records. Nutrient intakes were calculated using the NUTTAB95 food composition table [26]. Intake in grams were converted to standard serving size, that is, milk: 250 g, yogurt: 200 g, cheese: 40 g, and ice cream: 50 g [27]. In the combined total dairy and fermented dairy category, a serving of liquid dairy products was defined as 200 mL and cheese as 20 g. Dairy types analyzed included total dairy, high-fat dairy (liquid products >2%, cheese >20%; including full-fat milk, hard cheese, firm cheese, soft cheese, cream cheese, and ice cream), low-fat dairy (liquid products ≤2%, cheese ≤20%; including reduced-fat milk, skimmed milk, low-fat cheese, and ricotta/cottage cheese), and fermented dairy (yogurt and hard cheese, firm cheese, soft cheese, and low-fat cheese) (Supplemental Table 1). Because the FFQ did not distinguish between high-fat and low-fat yogurt types, yogurt

intake was divided equally across the high-fat and low-fat dairy category according to the Australian Health Survey showing that ~ 45% of yogurt consumption was low-fat and ~48% was regular or high-fat yogurt [6].

Outcome assessment

At baseline and both follow-up measurements, blood samples were collected after an overnight fast (≥8 h) [28]. All participants except those on diabetes medication or who were pregnant underwent a standard 75-g oral glucose tolerance test [1]. Fasting plasma glucose (FPG) levels and 2-h glucose levels (2hPG) based on the oral glucose tolerance test were determined with a glucose oxidase method at baseline and with a spectrophotometric-hexokinase method at both follow-up measurements [21]. Prevalent prediabetes at baseline and incident prediabetes at the follow-up was defined as FPG levels in the range 110 and 125 mg/dL (6.1 and 6.9 mmol/L) or 2hPG between 140 and 199 mg/dL (7.8 and 11.0 mmol/L) based on the WHO criteria [1,20]. Additional sensitivity analyses were performed with FPG levels in the range 100 and 125 mg/dL (5.6 and 6.9 mmol/L) based on the American Diabetes Association (ADA) criteria [29]. Type 2 diabetes was defined as FPG levels of ≥126 mg/dL (≥7.0 mmol/L) or 2hPG levels of ≥200 mg/dL (≥11.1 mmol/L) or current treatment with insulin or oral hypoglycemic agents.

Incident prediabetes was defined as occurrence of the outcome at 5-y or 12-y follow-up. Thus, participants with prediabetes at 5-y follow-up were coded as prediabetes regardless of their 12-y follow-up status ($n = 255$) or if they had missing 12-y follow-up status ($n = 153$). Participants with NGT at 12 y and NGT or missing information at 5 y were coded as having NGT ($n = 2,869$).

Assessment of covariates

Interviewer-administered questionnaires were used to collect data on demographic and health-related information. Educational level was categorized into primary school/never attended, some secondary school, completed secondary school, or university/further higher education. A smoking history was assessed using a validated questionnaire, and participants were categorized as current, past, and never smoker [30]. Total leisure-time physical activity was measured using the Active Australia questionnaire [31,32]. Total physical activity was calculated as the sum of time spent on walking (if continuous and for ≥10 min) or moderate-intensity activities, plus double the time spent in vigorous-intensity activities in the past week [33]. Physical activity was categorized as none, insufficient, 1–149 min/wk or sufficient, ≥150 min/wk. A family history of diabetes was defined as having a parent diagnosed with diabetes. The presence of cardiovascular disease (CVD) was obtained during the interviewer-administered questionnaire. BMI was calculated as weight (in kilograms) divided by height (in meters) squared. Waist circumference (WC) was measured in duplicate halfway between the lower border of the ribs and the iliac crest on a horizontal plane [34]. Serum triacylglycerol, total cholesterol, LDL-cholesterol and HDL-cholesterol concentrations were measured using standard enzymatic methods (Olympus AU600 analyzer; Olympus Optical) in serum fasting samples. Systolic and diastolic blood pressure levels were measured in a seated position using a Dinamap oscillometric blood pressure recorder (GE Healthcare), except in Victoria, where a standard mercury sphygmomanometer was used with appropriate adjustments to calculate blood pressure levels [35]. Hypertension was defined as systolic blood pressure of ≥140 mm Hg, diastolic blood pressure of ≥90 mm Hg, and/or reporting the use of antihypertensive medication.

Statistical analysis

Descriptive data were presented as means and SDs for continuous variables, medians and IQRs for nonnormally distributed continuous variables, and frequencies and percentages for categorical variables. Poisson regression models with robust variance were used because of the high incidence of prediabetes, in which case an odds ratio would overestimate the strength of the association [36]. Relative risks and 95% CIs for associations between dairy types and prediabetes incidence were calculated for dairy types in tertiles (reference lowest) and continuously (servings/d). For dairy types with many zero-consumers, a nonconsumer category (reference) was made, and consumers were dichotomized at the median value. The *P*-trend was calculated by incorporating the median values of dairy tertiles as continuous variables in the model. For each model, we examined whether nonlinear terms of continuous dairy types (second-order polynomials or natural splines with 3 to 5 knots depending on the intake range, excluding outliers) significantly improved model fit compared with the linear model assessed by likelihood ratio tests. To adjust for potential bias associated with missing data, a multiple imputation procedure ($n = 10$) was used for missing data on covariates (Supplemental Table 2). No corrections for multiple testing were made as most exposures were correlated, and corrections may result in a type II error [37]. Statistical procedures were performed with the software STATA (version 15.1).

Confounders were selected based on the literature [38–40]. The basic model (model 1) adjusted for age, sex, and energy intake. Model 2 was additionally adjusted for educational level, alcohol intake, smoking status, physical activity, and genetic background with a family history of type 2 diabetes. Model 3 additionally adjusted for food group intake associated with type 2 diabetes including fruits, vegetables, whole grains, legumes, nuts, red and processed meat, and fruit juice intake. To further examine possible confounding of associations by a healthy lifestyle, we presented descriptive data stratified by the dairy food types significantly associated with the outcomes. Potential effect modification by age, sex, and WC were explored in model 3, and stratified associations were presented in case of significant interactions ($P < 0.05$).

Multiple sensitivity analyses were performed in model 3 to examine the robustness of the findings. First, we additionally adjusted for baseline WC, change in WC from baseline to follow-up, LDL-cholesterol and hypertension as these variables are potential mediators. Second, we additionally adjusted for intake of all other dairy types to assess whether associations of certain dairy types were independent of each other. Third, participants with prevalent CVD were excluded to address reverse causation by change of diet and lifestyle ($n = 211$). Fourth, associations

were calculated with energy-adjusted intake of dairy types using the residual method [23]. Fifth, analyses were repeated using the ADA cutoff levels for prediabetes [29].

Results

Participant characteristics

In 4891 participants with NGT as measured by blood glucose at baseline, the mean age was 49.0 ± 12.3 y, 57% were female, and 12% were current smokers (Table 1). The mean WC was 88.0 ± 12.8 cm, the mean BMI was 26.1 ± 4.3 kg/m^2, and 15.7% were obese (BMI ≥ 30 kg/m^2). The mean total dairy intake was 2.4 ± 1.2 servings/d, mostly consisting of low-fat milk (0.70 ± 0.78, consumed by 55% of participants) and high-fat milk (0.47 ± 0.72, consumed by 37% of participants) intake (Figure 1, Supplemental Table 1). Participants in the highest (3.7 ± 0.9 servings/d) compared with the lowest (1.2 ± 0.5 servings/d) tertile of total dairy intake recorded a higher educational level (47.9 vs. 39.1% with university/further education level), were more physically active (58.8 vs. 52.2% with sufficient (≥150 min/wk) level), and were less likely to be hypertensive (19.9 vs. 24.7%) (Table 1). Furthermore, the average diet quality, energy intake, and intake of fruits, vegetables, grains, and fruit juice were higher in participants with the highest dairy intake that those with the lowest intake. Characteristics by the intake of specific dairy food types are presented in Supplemental Table 3.

Dairy intake and prediabetes risk

A total of 765 incident prediabetes cases were identified (15.6%): 408 at the 5-y follow-up (of 4383, 9.3%) and 357 at the 12-y follow-up (of 3474, 10.3%). A higher intake of total dairy was significantly associated with a lower prediabetes risk in

TABLE 1
Baseline characteristics of the study population according to tertiles of total dairy intake*

	Total (n = 4891)	T1 (n = 1646)	T2 (n = 1624)	T3 (n = 1621)
Total dairy consumption (serving/d)	2.4 ± 1.2	1.2 ± 0.5	2.3 ± 0.3	3.7 ± 0.9
Range	0.0–9.1	0.0–1.8	1.8–2.7	2.7–9.1
Follow-up time (5 y/12 y)	34.0/66.0	35.7/64.3	34.0/66.0	32.5/67.6
Age at baseline (y)	49.0 ± 12.3	49.4 ± 11.7	49.0 ± 12.7	48.7 ± 12.4
Sex, female	56.7	56.7	56.5	56.9
Educational level				
Primary school/never attended school	3.4	4.0	3.3	2.8
Completed some high school	33.1	37.4	32.3	29.5
Completed high school	19.6	19.6	19.3	19.8
University/further education	44.0	39.1	45.1	47.9
Smoking				
Current	11.5	12.3	11.2	11.2
Former	28.1	28.3	29.4	26.7
Never	60.4	59.4	59.5	62.2
Physical activity level				
Inactive (0 min/wk)	14.6	17.9	13.0	13.0
Insufficient (1–149 min/wk)	29.8	29.9	31.1	28.2
Sufficient (≥150 min/wk)	55.6	52.2	56.0	58.8
Family history of diabetes	17.7	18.4	17.8	16.8
BMI (kg/m^2)	26.1 ± 4.3	26.1 ± 4.4	26.1 ± 4.3	26.0 ± 4.3
Waist circumference (cm)	88.0 ± 12.8	88.3 ± 12.9	88.1 ± 12.8	87.8 ± 12.7
Total cholesterol (mmol/L)	5.6 ± 1.0	5.6 ± 1.1	5.6 ± 1.0	5.6 ± 1.0
LDL-cholesterol (mmol/L)	3.5 ± 0.9	3.5 ± 0.9	3.5 ± 0.9	3.5 ± 0.9
HDL-cholesterol (mmol/L)	1.5 ± 0.4	1.5 ± 0.4	1.5 ± 0.4	1.5 ± 0.4
TAG (mmol/L)	1.1 (0.8–1.7)	1.2 (0.8–1.7)	1.1 (0.8–1.7)	1.1 (0.8–1.6)
Hypertension	22.6	24.7	23.4	19.9
Dietary intake				
Energy intake (kcal/d)	1910 ± 657	1689 ± 590	1876 ± 610	2170 ± 677
Diet quality	84.6 ± 13.9	80.3 ± 14.5	85.0 ± 13.0	88.6 ± 12.9
Fruit (g/d)	198.0 ± 136.5	193.8 ± 144.8	191.9 ± 129.8	208.5 ± 133.9
Vegetables (g/d)	104.1 ± 49.1	100.6 ± 50.9	104.2 ± 47.7	107.6 ± 48.4
Grains (g/d)	164.3 ± 122.3	152.1 ± 120.6	158.3 ± 118.3	182.7 ± 125.7
Legumes (g/d)	27.7 ± 19.3	27.4 ± 20.0	27.5 ± 19.2	28.2 ± 18.6
Nuts (g/d)	3.4 ± 6.4	3.6 ± 7.0	2.7 ± 4.6	3.9 ± 7.2
Meat (red and processed) (g/d)	97.8 ± 73.8	93.5 ± 75.6	96.8 ± 72.8	103.3 ± 72.5
Fruit juice (g/d)	86.3 ± 120.8	80.3 ± 128.4	83.3 ± 109.2	95.5 ± 123.3
Total fat (EN%)	36.0 ± 5.7	36.4 ± 5.6	35.8 ± 5.6	35.8 ± 5.8
Saturated fat (EN%)	14.4 ± 3.5	13.7 ± 3.3	14.4 ± 3.4	14.9 ± 3.6
Carbohydrates (EN%)	45.2 ± 6.0	45.1 ± 6.4	45.4 ± 5.9	45.0 ± 5.5
Protein (EN%)	19.3 ± 3.0	19.0 ± 3.1	19.2 ± 3.0	19.6 ± 2.8
Calcium (mg/d)	908 ± 326	619 ± 172	875 ± 148	1233 ± 277
Sodium (mg/d)	2612 ± 991	2318 ± 909	2575 ± 932	2950 ± 1026
Alcohol (g/d)	13.0 ± 16.7	13.8 ± 17.9	12.9 ± 16.6	12.3 ± 15.5

Abbreviations: EN%, percentage of total energy intake; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TAG, triacylglycerol.
* Values are mean ± SD for continuous variables with a normal distribution (pooled) or median (IQR) for continuous variables with a skewed distribution and percentages for categorical variables, on the basis of unimputed data.

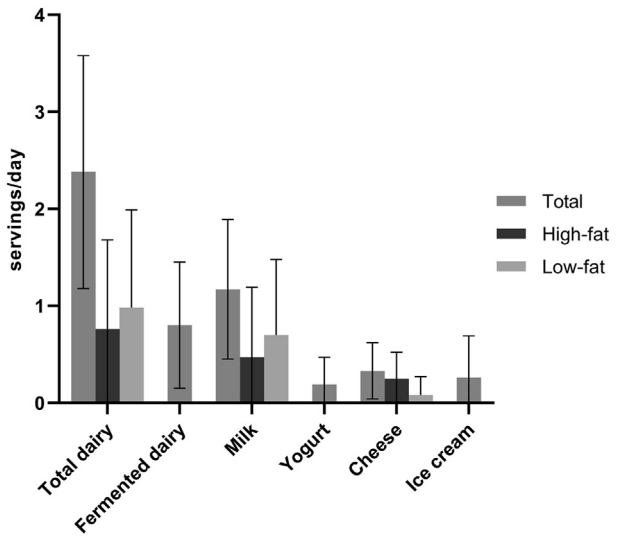

FIGURE 1. Dairy type intake in the AusDiab study in serving per day (mean ± SD): milk, 250 mL; yogurt, 200 mL; cheese, 40 g; and ice cream, 50 g. Combined total dairy category: liquid dairy products, 200 mL; cheese, 20 g.

model 2 ($RR_{servings/d}$: 0.94; 95% CI: 0.89, 1.00; $P = 0.04$), but this association was no longer significant after additional adjustment for dietary intake in model 3 (Table 2). Furthermore, a higher intake of fermented dairy was associated with a lower prediabetes risk in model 2 ($RR_{servings/d}$: 0.88; 95% CI: 0.78, 0.99; $P = 0.04$) but nonsignificantly in model 3 ($RR_{servings/d}$: 0.91; 95% CI: 0.80, 1.02; $P = 0.10$). A higher intake of high-fat dairy (44.4% of total dairy intake) was significantly associated with a lower prediabetes risk in fully adjusted models when analyzed on a continuous scale ($RR_{servings/d}$: 0.92; 95% CI: 0.85, 1.00; $P = 0.04$) but not when analyzed in tertiles. In line with the results of high-fat dairy intake, a higher intake of high-fat milk (39.8% of total milk) was associated with a lower prediabetes risk on a continuous scale in model 3 ($RR_{servings/d}$: 0.89; 95% CI: 0.80, 0.99; $P = 0.03$) and borderline significant in the third compared with those of the first intake category (RR_{T3}: 0.79; 95% CI: 0.65, 0.97; $P_{trend} = 0.05$). Furthermore, a higher intake of total cheese was associated with a lower prediabetes risk ($RR_{servings/d}$: 0.74; 95% CI: 0.56, 0.96; $P = 0.03$). A higher intake of high-fat cheese (77.1% of total cheese) was associated with a lower prediabetes risk in model 2 ($RR_{servings/d}$: 0.71; 95% CI: 0.54, 0.95; $P = 0.02$), but this association was similar but no longer significant in model 3 (0.75; 95% CI: 0.57, 1.00; $P = 0.05$). The third compared with the first intake category of low-fat milk (59.8% of total milk) intake was associated with a higher prediabetes risk in model 3 (RR_{T3}: 1.15; 95% CI: 0.97, 1.35; $P_{trend} = 0.04$), although continuous associations were not significant. A better fit of a nonlinear association was found for low-fat milk ($P = 0.03$) consumption, showing an increased risk of up to 1.5 servings/d and a lower risk with higher intakes (Figure 2). Low-fat dairy, total milk, total yogurt, low-fat cheese, and ice cream were not associated with risk of prediabetes in multivariate-adjusted models.

Sensitivity analyses

None of the interactions of types of dairy food with sex, age, and baseline WC were statistically significant. Only total cheese remained associated with a lower prediabetes risk with additional adjustment for the potential mediators baseline WC, change in WC from baseline to follow-up, LDL-cholesterol, and hypertension ($RR_{servings/d}$: 0.74; 95% CI: 0.57, 0.96; $P = 0.03$) (Supplemental Table 4). The associations for the intake of high-fat dairy and high-fat milk were similar but no longer statistically significant. All associations were similar after adjustment for intake of all other dairy types, after excluding participants with prevalent CVD and with dairy types adjusted for energy intake. Using the ADA cutoffs resulted in attenuation of the associations for high-fat dairy and high-fat milk but not for total cheese.

Discussion

In this large, prospective Australian cohort, high-fat dairy, high-fat milk, and total cheese were associated with a lower incidence of prediabetes. These associations were independent of age, sex, energy intake, educational level, smoking status, physical activity, alcohol consumption, a family history of diabetes, and background dietary intake. By contrast, a nonlinear association for low-fat milk intake was found; risk was highest at 1.5 servings/d, with a decreasing risk at lower and higher intakes. Total dairy foods, fermented dairy, and high-fat cheese were associated with a lower incidence of prediabetes, but not when considering dietary intake of other food groups. Low-fat dairy foods, total milk, total yogurt, low-fat cheese, and ice cream were not associated with risk of prediabetes in multivariable models.

Dairy should not be regarded as one single product but as a heterogeneous group of foods because associations with (markers of) disease risk vary by the product type [41]. Furthermore, our results showed that distinguishing the fat content is important for the assessment of the health effects of dairy. For instance, we found a protective association for high-fat milk and prediabetes and a nonlinear positive association for low-fat milk. These associations were in accordance with those of the Dutch Rotterdam studies, where we found significant associations for high-fat milk ($HR_{serving/d}$: 0.88, 95% CI 0.79, 0.99) and in the other direction for low-fat milk ($HR_{serving/d}$: 1.07, 95% CI 1.01, 1.13) [17]. However, low-fat milk and high-fat milk were not associated with prediabetes in the Dutch Hoorn studies [16]. In the FHS-OC, no contrast in associations by the fat content of milk was found; they reported significant dose–response associations for both skimmed milk (HR: ≥14 vs. 0–1 servings/wk, 0.82; 95% CI: 0.61, 1.10) and whole milk (HR: ≥1 vs. 0 servings/wk, 0.84; 95% CI: 0.69, 1.01) [15].

A higher intake of total and high-fat cheese was associated with a lower prediabetes risk, which is consistent with the Hoorn Study (total cheese, RR top vs. bottom quartile: 0.86; 95% CI: 0.73, 1.02), high-fat cheese, $RR_{serving/d}$: 0.94; 95% CI: 0.88, 1.00; and RR top vs. bottom quartile: 0.79; 95% CI: 0.66, 0.94) [16] and the FHS-OC (total cheese, HR: ≥ 4 vs. 0–1 servings/wk, 0.86; 95% CI: 0.69, 1.07) [15] but not with the Rotterdam study [17]. In addition, this association was reported in several cross-sectional studies with prediabetes outcomes [42,43] and meta-analyses of prospective cohort studies on cheese and type 2 diabetes [44].

Many guidelines worldwide recommend low-fat dairy types to limit the intake of saturated fat [45]. However, there is

TABLE 2
The associations of dairy product types and prediabetes risk in the AusDiab study (n = 4891)

	Intake range categories (servings/d)			P_{trend}	Continuous (servings/d), RR (95% CI)
	T1	T2	T3		
	RR	RR (95%CI)	RR (95%CI)		
Total dairy					
n/N	280/1646	250/1624	235/1621		765/4891
Median intake	1.3	2.3	3.4		
Model 1	1 (ref)	0.92 (0.78–1.07)	0.88 (0.75–1.04)	0.14	0.93 (0.88–0.99)*
Model 2	1 (ref)	0.93 (0.80–1.09)	0.91 (0.77–1.07)	0.26	0.94 (0.89–1.00)*
Model 3	1 (ref)	0.97 (0.82–1.13)	0.97 (0.82–1.15)	0.72	0.96 (0.91–1.02)
High-fat dairy					
n/N	258/1637	280/1645	227/1609		
Median intake	0.05	0.3	1.9		
Model 1	1 (ref)	1.06 (0.90–1.24)	0.90 (0.76–1.06)	0.08	0.91 (0.84–0.98)*
Model 2	1 (ref)	1.07 (0.92–1.25)	0.89 (0.75–1.06)	0.06	0.90 (0.83–0.98)*
Model 3	1 (ref)	1.10 (0.94–1.28)	0.92 (0.77–1.09)	0.12	0.92 (0.85–1.00)*
Low-fat dairy					
n/N	237/1608	270/1664	258/1619		
Median intake	0.01	1.0	1.9		
Model 1	1 (ref)	1.13 (0.96–1.32)	1.09 (0.93–1.28)	0.29	1.03 (0.97–1.10)
Model 2	1 (ref)	1.15 (0.98–1.35)	1.13 (0.96–1.34)	0.14	1.05 (0.98–1.11)
Model 3	1 (ref)	1.16 (0.98–1.36)	1.16 (0.98–1.37)	0.08	1.06 (0.99–1.13)
Fermented dairy					
n/N	294/1656	231/1530	240/1703		
Median intake	0.2	0.7	1.4		
Model 1	1 (ref)	0.89 (0.76–1.04)	0.83 (0.71–0.98)	0.03*	0.87 (0.78–0.98)*
Model 2	1 (ref)	0.89 (0.76–1.04)	0.85 (0.72–0.99)	0.05	0.88 (0.78–0.99)*
Model 3	1 (ref)	0.91 (0.78–1.07)	0.88 (0.75–1.04)	0.16	0.91 (0.80–1.02)
Total milk					
n/N	269/1568	165/1017	331/2306		
Median intake	0.8	0.8	1.5		
Model 1	1 (ref)	1.05 (0.87–1.25)	0.89 (0.76–1.03)	0.04*	0.94 (0.86–1.03)
Model 2	1 (ref)	1.05 (0.88–1.25)	0.90 (0.78–1.05)	0.08	0.95 (0.87–1.04)
Model 3	1 (ref)	1.06 (0.89–1.27)	0.94 (0.81–1.10)	0.22	0.98 (0.89–1.08)
High-fat milk					
n/N	502/3080	153/923	110/888		
Median intake	0.0	0.8	1.5		
Model 1	1 (ref)	1.02 (0.87–1.21)	0.77 (0.64–0.94)	0.03*	0.88 (0.78–0.99)*
Model 2	1 (ref)	1.00 (0.85–1.18)	0.77 (0.63–0.94)	0.02*	0.88 (0.79–0.97)*
Model 3	1 (ref)	0.99 (0.84–1.17)	0.79 (0.65–0.97)	0.05*	0.89 (0.80–0.99)*
Low-fat milk					
n/N	312/2192	242/1342	211/1357		
Median intake	0.0	0.8	1.5		
Model 1	1 (ref)	1.27 (1.09–1.48)	1.10 (0.94–1.29)	0.11	1.04 (0.97–1.13)
Model 2	1 (ref)	1.28 (1.10–1.49)	1.13 (0.96–1.33)	0.06	1.06 (0.98–1.14)
Model 3	1 (ref)	1.27 (1.09–1.49)	1.15 (0.97–1.35)	0.04*	1.07 (0.99–1.16)
Total yogurt					
n/N	219/1195	220/1537	326/2159		
Median intake	0.0	0.01	0.36		
Model 1	1 (ref)	0.91 (0.76–1.08)	0.94 (0.80–1.10)	0.83	1.05 (0.84–1.32)
Model 2	1 (ref)	0.92 (0.77–1.10)	0.97 (0.82–1.15)	0.89	1.10 (0.87–1.38)
Model 3	1 (ref)	0.93 (0.78–1.11)	0.99 (0.84–1.17)	0.69	1.14 (0.90–1.43)
Total cheese					
n/N	278/1583	297/1894	190/1414		
Median intake	0.05	0.3	0.7		
Model 1	1 (ref)	0.91 (0.79–1.06)	0.81 (0.68–0.97)	0.02*	0.69 (0.53–0.91)**
Model 2	1 (ref)	0.92 (0.79–1.06)	0.82 (0.69–0.97)	0.02*	0.69 (0.53–0.91)**
Model 3	1 (ref)	0.94 (0.81–1.09)	0.85 (0.72–1.01)	0.07	0.74 (0.56–0.96)*
High-fat cheese					
n/N	293/1656	332/2218	140/1017		
Median intake	0.0	0.2	0.7		
Model 1	1 (ref)	0.86 (0.74–0.99)	0.81 (0.67–0.98)	0.04*	0.72 (0.54–0.95)*
Model 2	1 (ref)	0.85 (0.74–0.99)	0.81 (0.67–0.98)	0.04*	0.71 (0.54–0.95)*
Model 3	1 (ref)	0.87 (0.75–1.01)	0.84 (0.70–1.02)	0.11	0.75 (0.57–1.00)
Low-fat cheese					
n/N	591/3726	71/457	103/708		
Median intake	0.0	0.1	0.4		

(*continued on next page*)

TABLE 2 (*continued*)

	Intake range categories (servings/d)			P_{trend}	Continuous (servings/d), RR (95% CI)
	T1	T2	T3		
	RR	RR (95%CI)	RR (95%CI)		
Model 1	1 (ref)	1.02 (0.81–1.29)	0.97 (0.80–1.18)	0.77	0.85 (0.59–1.22)
Model 2	1 (ref)	1.02 (0.82–1.29)	0.98 (0.81–1.19)	0.87	0.86 (0.60–1.22)
Model 3	1 (ref)	1.02 (0.81–1.28)	0.99 (0.82–1.20)	0.94	0.87 (0.61–1.25)
Ice cream					
n/N	273/1676	242/1581	250/1634		
Median intake	0.02	0.11	0.5		
Model 1	1 (ref)	0.95 (0.81–1.11)	0.87 (0.74–1.04)	0.14	1.02 (0.86–1.20)
Model 2	1 (ref)	0.95 (0.81–1.12)	0.87 (0.74–1.04)	0.13	1.02 (0.86–1.20)
Model 3	1 (ref)	0.95 (0.81–1.11)	0.88 (0.75–1.05)	0.18	1.03 (0.87–1.21)

Continuous analysis in servings/d: milk, 250 mL; yogurt, 200 mL; cheese, 40 g; and ice cream, 50 g. Combined total dairy category: liquid dairy products, 200 mL; cheese, 20 g. Model 1 included age (continuous) and sex and energy intake (continuous). Model 2 was additionally adjusted for education (3 categories), smoking (3 categories), physical activity (3 categories), alcohol consumption (continuous), and a family history of diabetes (yes/no). Model 3 was additionally adjusted for food groups associated with type 2 diabetes, including intakes of fruit, vegetables, grains, legumes, nuts, red and processed meat, and fruit juice (continuous). *P* value significance level: *0.05, **0.01, ***0.001.

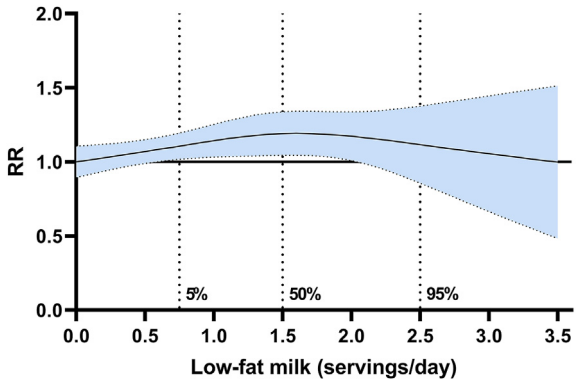

FIGURE 2. A nonlinear association between low-fat milk intake and prediabetes risk in the AusDiab study. The solid line indicates risk estimate fitted with a restricted cubic spline regression with 3 knots specified at the 5th, 50th, and 95th percentile of low-fat milk intake as indicated by the dotted vertical lines. The colored area indicates the 95% confidence interval. The model was adjusted for age, sex, energy intake, education, smoking, physical activity, alcohol consumption, a family history of diabetes, fruit, vegetables, grains, legumes, nuts, red and processed meat, and fruit juice intake.

currently little evidence that high-fat dairy regardless of its high saturated fat content is harmful for health [46–48]. Differences in nutrient content are marginal: for example, high-fat milk contains 3.5% fat, semiskimmed milk contains 1%–1.5% fat, and skimmed milk contains no more than 0.15% fat, but high-fat milk contains higher concentrations of fat-soluble vitamin A (36, 15, and 1 μg, respectively) [26] and vitamin K (1.4, 0.7, and 0 μg, respectively) [49]. It is unlikely that these nutritional differences completely accounted for the sign reversal of our observed associations for milk. Possibly, low-fat foods have a lower satiety value, which could result in overconsumption of carbohydrates, particularly harmful to health if fats are substituted for refined starches and sugar [50,51]. Furthermore, consumers may prefer sweetened low-fat milk to compensate for the reduced flavor by removing fat globules and cream, subsequently increasing sugar intake. Indeed, in our study, the diet of participants in the top compared with those in the bottom tertiles of low-fat milk and low-fat cheese contained higher proportions of carbohydrates. Compared with high glycemic carbohydrates, saturated fat increases HDL-cholesterol levels, resulting in similar total cholesterol/HDL-cholesterol and lower triglyceride levels [52]. In addition, the effects of saturated fatty acids depend on the type. Dairy contains palmitic acid associated with increased type 2 diabetes risk but also various fatty acids with potential opposite effects. A meta-analysis of 16 studies showed that higher levels of odd-chained saturated fatty acids (C15:0 and C17:0), and natural ruminant *trans*-fats [t16:1(n-7)] were associated with a lower type 2 diabetes risk [53].

We did not find that yogurt intake was associated with prediabetes in our study, in line with previous prospective studies [15,16]. Only in the Rotterdam study, we previously found that high-fat yogurt was associated with a lower prediabetes risk, but low-fat yogurt showed neutral associations [17]. A nonlinear inverse association was found between yogurt intake and type 2 diabetes (at 80 g/d, RR: 0.86; 95% CI: 0.83, 0.90), with no additional benefit at higher intake levels [54]. Furthermore, in 3 large cohorts among US individuals and in the Iranian Tehran Lipid and Glucose study, increased intake of yogurt during the follow-up was associated with a lower risk of type 2 diabetes [55, 56]. Compared with US and Western-European cohorts, intake levels of yogurt were considerably lower in our current study (0.19 ± 0.28 servings/d), which might explain the neutral association we observed. Low intake levels in this sample were in line with the Australian Health Survey (0.12 servings/d) [57]. Furthermore, in Australia, 76% of yogurt was flavored or had added fruit, and 46% was of low fat. The FFQs used in many population-based studies do not consider the variety in sugar, protein, and fat content of commercially available yogurts [58, 59]. Moreover, many consumers add sweeteners such as sugar, jam, or honey to plain yogurt [60]. Unmeasured differences in nutrient content of consumed yogurts in each cohort might contribute to inconsistent findings. Future studies should collect more detailed information on yogurt composition and consumer behavior to elucidate potential heterogeneity.

Our results are not in line with population-based studies showing inverse associations for low-fat dairy and yogurt intake with type 2 diabetes [44]. Furthermore, our findings are not in line with the previous analysis of the AusDiab study, which found an inverse association between low-fat milk intake and

type 2 diabetes incidence at the 5 y follow-up (OR: 0.65; 95% CI: 0.44, 0.94) and a nonsignificant association for full-fat milk (OR: 1.18; 95% CI: 0.78, 1.79) [18]. Nevertheless, associations between yogurt and cheese were similar. We excluded approximately 2000 participants with prediabetes at baseline, and thus, these different findings may result from a baseline sample with less variation in the glycemic status compared with studies on type 2 diabetes. It could be that dairy has differential effects according to individual's metabolic state or degree of insulin resistance [15], and more research is needed in that regard.

Current evidence from long-term RCTs with dairy consumption as the main intervention and diabetes related outcomes is inconclusive owing to differences in design, duration, and geographic location [61]. Results are further affected by the diet consumed parallel or in replacement of high dairy diets, physical activity levels, and weight variation during the study. Two recent RCTs studying the effects of diets high in dairy did not find differences in glucose measurements after the intervention [62,63]. However, one of these RCTs showed that insulin sensitivity was decreased in the high dairy diets compared with that in the low dairy control, possibly because of the insulinotropic effect of dairy and alterations in the gut microbiota [62]. Short-term controlled feeding trials showed that milk proteins attenuated acute hyperglycemia [64] and regulated lipid changes induced by glucose ingestion [65]. Future studies are required to detangle the role of different dairy types and dairy fat content in type 2 diabetes development, particularly RCTs.

Strengths and limitations

This study was performed within a large population-based study with up to 12 y of follow-up. This study adds to the body of evidence by differentiating associations by the fat content of dairy types. Prediabetes was defined both based on FPG and 2hPG, representing impaired fasting glucose and impaired glucose tolerance, respectively, 2 distinct states of type 2 diabetes development [1]. The results should be interpreted carefully by considering the following limitations. First, we used only baseline data on dairy intake, and people might have changed their diet over time, resulting in misclassification of the exposure, biasing estimates toward the null. However, in our previous analyses of the Rotterdam study, we showed that the inclusion of repeated measurements of dairy consumption did not change associations [17]. Second, the FFQ is useful in estimating the intake of frequently used foods such as dairy and ranking participants according to their food intake in observational studies; however, the FFQ relies on participants' memory and ability to estimate portion sizes and is, therefore, prone to recall bias. Thus, misclassification of exposure is possible, resulting in bias toward the null. Third, reverse causality might be an issue. Prediabetes is commonly an asymptomatic condition, making reverse causality due to a prediabetes diagnosis unlikely. Moreover, the exclusion of people with CVD at the baseline or follow-up in sensitivity analyses did not change the estimates. However, presence of obesity or other risk factors might have induced behavioral changes, such as a shift from high-fat to low-fat dairy consumption to reduce fat and caloric intake. Fourth, residual confounding cannot be ruled out considering the observational nature of our study; nevertheless, we carefully adjusted for a wide range of confounders, such as background diet and CVD risk factors. Finally, the AusDiab is not entirely representable of the general population, as socioeconomic status of responders was somewhat higher than of nonresponders, and some healthy volunteer selection bias is likely [19]. Furthermore, there was a considerable loss to the follow-up in the AusDiab study.

Conclusions

In conclusion, in the long-term, population-based Australian cohort, associations of dairy and prediabetes differ by both type and fat content. High-fat dairy foods, high-fat milk, and total cheese were associated with a lower prediabetes incidence. Further prospective studies should collect more specific information on fat and sugar content of various milk and yogurt types and examine the influence of reverse causation.

Funding

The AusDiab study, initiated and coordinated by the International Diabetes Institute, and subsequently coordinated by the Baker Heart and Diabetes Institute, was supported by the National Health and Medical Research Council (NHMRC grants 233200 and 1007544), Australian Government Department of Health and Ageing, Abbott Australasia Pty Ltd, Alphapharm Pty Ltd, Amgen Australia, AstraZeneca, Bristol-Myers Squibb, City Health Centre-Diabetes Service-Canberra, Department of Health and Community Services – Northern Territory, Department of Health and Human Services – Tasmania, Department of Health – New South Wales, Department of Health – Western Australia, Department of Health – South Australia, Department of Human Services – Victoria, Diabetes Australia, Diabetes Australia Northern Territory, Eli Lilly Australia, Estate of the Late Edward Wilson, GlaxoSmithKline, Jack Brockhoff Foundation, Janssen-Cilag, Kidney Health Australia, Marian & FH Flack Trust, Menzies Research Institute, Merck Sharp & Dohme, Novartis Pharmaceuticals, Novo Nordisk Pharmaceuticals, Pfizer Pty Ltd, Pratt Foundation, Queensland Health, Roche Diagnostics Australia, Royal Prince Alfred Hospital, Sydney, Sanofi Aventis, Sanofi-Synthelabo, and the Victorian Government's OIS Program. This project was funded with an unrestricted grant by the Dutch Dairy Association (NZO) to SSS-M.

The supporting source was not involved in the study design, collection, analysis, interpretation of data, and writing the report or restrictions regarding the submission of the report for publication.

Author disclosures

SSS-M has received unrestricted grants from the Global Dairy Platform, Dairy Research Institute, and Dairy Australia for a meta-analysis on cheese and blood lipids (2012) and a meta-analysis of dairy and mortality (2015). She received the Wiebe Visser International Dairy Nutrition Prize and has received recent research funding (2019) for epidemiological studies on dairy products and cardiometabolic diseases from the Dutch Dairy Association and the Danish Dairy Research Foundation. All other authors declare that they have no conflict of interest.

Acknowledgments

The AusDiab study, initiated and coordinated by the International Diabetes Institute, and subsequently coordinated by the

Baker Heart and Diabetes Institute, gratefully acknowledges the support and assistance given by:K Anstey, B Atkins, B Balkau, E Barr, A Cameron, S Chadban, M de Courten, D Dunstan, A Kavanagh, S Murray, N Owen, K Polkinghorne, J Shaw, T Welborn, P Zimmet and all the study participants.

Author contributions

IALS, SSS-M: designed the research; IALS, LC: wrote the statistical analysis plan; LC: conducted the data analysis; IALS: drafted the manuscript, supervised by SSS-M; IALS: had responsibility for the final content; and all authors: contributed to the study design and read and approved the final manuscript.

Data availability

The AusDiab team at the Baker Heart and Diabetes Institute (AusDiab) welcomes approaches from bona fide research workers for access to the accumulated data and biological materials and for participation in ongoing and new data collection activities. Researchers and potential collaborators wishing to access the AusDiab data sets should complete the AusDiab data access form that can be found on their website: https://www.baker.edu.au/ausdiab/

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.tjnut.2023.03.032.

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