

The association between dairy food intake and the incidence of diabetes in Australia: the Australian Diabetes Obesity and Lifestyle Study (AusDiab)

Narelle M Grantham¹, Dianna J Magliano^{1,*†}, Allison Hodge², Jeremy Jowett¹, Peter Meikle¹ and Jonathan E Shaw¹

¹Baker IDI Heart and Diabetes Institute, Melbourne, Victoria, Australia; ²Melbourne University, St Vincent's Hospital, Fitzroy, Victoria, Australia

Submitted 23 February 2011: Final revision received 15 December 2011: Accepted 24 February 2012: First published online 7 June 2012

Abstract

Objective: Several studies have suggested that dairy food may reduce the risk of obesity and metabolic abnormalities but few have been able to conclusively demonstrate that it reduces the risk of diabetes. The aim of the present analysis was to investigate if dairy food intake independently reduces the risk of diabetes.

Design: The Australian Diabetes Obesity and Lifestyle Study (AusDiab) is a national, population-based, prospective survey conducted over 5 years. Baseline measurements included a 121-item FFQ, anthropometrics and an oral glucose tolerance test.

Setting: Forty-two randomly selected clusters across Australia.

Subjects: Adults aged ≥ 25 years who participated in the baseline survey and returned to follow-up 5 years later.

Results: A total of 5582 participants with complete data were eligible for analysis, 209 of whom had incident diabetes. Compared with men in the first tertile of dairy food intake, men in the third tertile had a significantly reduced risk of developing diabetes after adjustment for age, sex, total energy intake, family history of diabetes, education, physical activity, smoking status, fasting serum TAG and HDL cholesterol, systolic blood pressure, waist circumference and hip circumference (OR = 0.53, 95% CI 0.29, 0.96; $P = 0.033$). A similar non-significant association was observed in women.

Conclusions: Dietary patterns that incorporate high intakes of dairy food may reduce the risk of diabetes among men. Further investigation into the relationship between dairy food intake and diabetes needs to be undertaken to fully understand the potential mechanism of this observation.

Keywords
Diabetes mellitus
Epidemiology
Nutrition assessments
Dairy products
Prospective studies
Incidence

The prevalence of type 2 diabetes is increasing at an alarming rate worldwide. The ageing of populations and the effects of modernization of lifestyle are contributing to this dramatic increase. It is estimated that 285 million people worldwide had diabetes in 2010⁽¹⁾. This number is expected to increase to 439 million by the year 2030, with the majority of these cases being type 2 diabetes. This represents a projected increase in the prevalence of diabetes from 6.4% in 2010 to 7.7% by 2030⁽¹⁾.

A number of epidemiological studies have suggested that dairy consumption may have beneficial effects on diabetes risk factors such as body weight, blood pressure and insulin resistance^(2–6), leading to an increased interest in research exploring directly the relationship between

dairy food intake and diabetes risk. Some investigators have suggested that lactose and protein in dairy products enhance satiety, having a favourable impact on weight loss^(2,6), while others have suggested that Ca intake plays a central role in any effects of dairy food⁽⁷⁾.

Several large prospective cohorts have shown an association between dairy intake (particularly low-fat products) and incident self-report diabetes^(2–4). However, in the only study to use objective measurements of baseline parameters and of diabetes status (the Hoorn Study), no association was seen between baseline dairy food intake and change in blood glucose over 6 years⁽⁸⁾. Thus, uncertainty remains over the existence and nature of a relationship between dairy food intake and diabetes risk.

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) included dietary assessment and objective measures of diabetes status as well as measures of a wide

† Address for correspondence: PO Box 6492, St Kilda Road Central, VIC 8008, Australia.

range of potential confounders in a national, population-based longitudinal study. It therefore provided an ideal opportunity to examine the relationship between dairy food intake and diabetes incidence.

Methods

The AusDiab baseline study methods and response rates are described in detail elsewhere⁽⁹⁾. In brief, the baseline study was a national, population-based survey of 11 247 adults aged ≥ 25 years in 1999–2000. Over 85% of the sample was from an Australian, New Zealand or British background. A stratified cluster sample was drawn from forty-two randomly selected census collector districts across Australia. Information was collected using a brief household interview, followed by a biomedical examination. Of the eligible adults, 70% completed the household interview, and 55% of these completed the baseline biomedical examination⁽⁹⁾. In 2004–2005, all living eligible participants were invited to attend follow-up. Those who were considered ineligible included those who were deceased, had moved overseas or into a nursing facility classified for high care, or had a terminal illness. Among the 11 005 eligible participants, 6537 returned for the 5-year follow-up at which the baseline assessments were repeated; the response rate was 60%.

Differences between follow-up attendees and people who did not attend have been described previously⁽¹⁰⁾. Only those people with complete data for the variables of interest and who did not have diabetes at baseline were included in the present analyses (n 5582).

At baseline and follow-up, the protocol included questionnaires, anthropometric measurements and collection of blood samples. All participants except for those currently receiving treatment for diabetes or who were pregnant underwent a standard 75 g oral glucose tolerance test⁽¹¹⁾. Blood was collected after an overnight fast (≥ 9 h). Serum TAG, total cholesterol, LDL cholesterol and HDL cholesterol (HDL-C) were measured by enzymatic methods (Olympus AU600 analyser; Olympus Optical Co. Ltd, Tokyo, Japan). Blood pressure was measured using a Dinamap[®] oscillometric blood pressure recorder (GE Healthcare, Australia) or a standard mercury sphygmomanometer with appropriate adjustments made as previously described⁽¹²⁾. The study was approved by the International Diabetes Institute Ethics Committee.

Risk factors

Data on education, smoking, physical activity and family history of diabetes were collected by an interviewer-administered questionnaire. Education was classified into four categories: (i) university/further education, (ii) completed secondary school, (iii) some secondary school or (iv) primary school/never attended school. Smoking history (current smoker, past smoker or never

smoked) was collected using a questionnaire which has been validated in Australian adults⁽¹³⁾. Total leisure-time physical activity reported for the previous week (none; insufficient, 1–149 min/week; sufficient, ≥ 150 min/week) was measured using the Active Australia questionnaire, which is a standard instrument for population surveillance⁽¹⁴⁾. Family history of diabetes was defined as mother or father being diagnosed as having diabetes.

Measurement of dairy food intake

Dietary information was collected using an FFQ designed by the Cancer Council Victoria and self-administered by participants at the local testing site. This questionnaire enquires about dietary intake over the last 12 months⁽¹⁵⁾. Information on the amount or frequency of consumption was collected on milk (full-fat, low-fat and skimmed), cheese (hard, firm, soft, low-fat cheese, ricotta/cottage cheese and cream cheese), yoghurt, ice cream and flavoured milk. Dairy products classified as low-fat included reduced-fat milk, skimmed milk, low-fat cheese, ricotta cheese and yoghurt. Dairy products classified as full-fat included full-fat milk, hard cheese, firm cheese, soft cheese and cream cheese. Participants were asked to select from ten frequency responses ('never' to '3 or more times per day') for each item on the FFQ except for milk, where they were asked to report quantity of milk intake per day (from 'none' to '3 cups or more'). Daily intakes of each food were calculated using sex-specific standard portion sizes derived from weighed food records and the reported frequencies converted to daily equivalents. From these data, intakes of nutrients, including energy and Ca, were calculated using NUTTAB95 food composition data⁽¹⁶⁾. Daily number of servings of dairy products was calculated from the estimated food intakes in grams, using the following definitions: 1 serving of milk = 250 g, 1 serving of yoghurt = 200 g and 1 serving of cheese = 40 g. Dairy food intake (in servings/d) was also categorized into tertiles (see Table 1 for distribution). Dairy product intakes were analysed as total dairy food (i.e. milk, cheese and yoghurt together), as individual foods and in high-fat and low-fat dairy categories. The focus of the analysis of individual products was on milk, yoghurt and cheese, as these products made up the bulk of dairy products consumed by our study population. Ca intake was assessed from all foods as indicated in the NUTTAB95 database. This variable does not include Ca intake from supplements.

Statistical analysis

Incident cases of diabetes were defined as individuals without diabetes at baseline but who had developed diabetes at follow-up. At both baseline and follow-up, diabetes was classified on the basis of fasting plasma glucose ≥ 7.0 mmol/l or 2 h post-load plasma glucose ≥ 11.1 mmol/l or current treatment with insulin or oral hypoglycaemic agents⁽¹⁰⁾.

Potential confounding factors included age, sex, total energy intake, family history of diabetes, education,

Table 1 Baseline characteristics according to diabetes status at follow-up: adults aged ≥ 25 years, the Australian Diabetes Obesity and Lifestyle Study (AusDiab)

	Diabetes status at follow-up				P value
	With diabetes (n 209)		Without diabetes (n 5373)		
	% or Mean or Median	SD or 25th, 75th	%, Mean or Median	SD or 25th, 75th	
Males (%)	50.7		44.9		0.09
Age (years)	55.7	12.1	50.6	12.5	<0.01
Age range (years)	26–83		25–88		
Waist circumference (cm)					
Males	103.8	11.2	96.4	10.5	<0.01
Females	92.5	14.7	83.7	12.4	<0.01
Hip circumference (cm)					
Men	107.2	0.8	103.9	14.6	<0.01
Women	109.7	1.3	104.5	0.2	<0.01
Smoking status (%)					
Current smoker	15.2		10.8		0.03
Ex-smoker	33.3		29.1		
Never smoked	51.5		60.0		
Education level (%)					
Primary school/never attended school	11.0		3.9		<0.01
Completed some high school	38.3		34.9		
Completed high school	18.7		18.9		
University/further education	32.1		42.3		
Level of physical activity (%)					
Inactive (0 min/week)	20.8		14.9		<0.01
Insufficient (1–149 min/week)	37.7		30.7		
Sufficient (≥150 min/week)	41.5		54.4		
Family history of diabetes (%)	30.1		17.7		<0.01
HDL cholesterol (mmol/l)	1.3	0.4	1.4	0.4	<0.01
TAG (mmol/l)	1.7	1.2, 2.5	1.2	0.8, 1.8	
Hypertension (%)*	52.4		27.3		<0.01
Dairy food intake (servings/d)					
Tertile 1 (0–1.2) (%)	42.6		32.6		
Tertile 2 (>1.2–1.9) (%)	33.0		33.3		
Tertile 3 (>1.9–5.8) (%)	24.4		34.0		

Data are presented as percentage, or mean and standard deviation, or median and 25th, 75th percentile.

*Hypertension was defined as systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg or reporting use of antihypertensive medication.

physical activity, smoking status, fasting serum TAG and HDL-C, systolic blood pressure, waist circumference and hip circumference⁽¹⁰⁾.

Skewed variables (TAG, Ca) were log-transformed before inclusion in regression models. In order to control for FFQ that may not have been completed correctly and therefore do not represent usual dietary intake, the top and bottom 1% (sex-specific) of energy intake were excluded (*n* 114). In order to control for outliers or potential unrealistic intake of dairy food, individuals with the top 1% (sex-specific) of dairy food intake were excluded from the analysis (*n* 61) as has been done in similar analyses⁽¹⁷⁾. Medians were calculated on consumers only.

Logistic regression analyses were performed to investigate the association between dairy food intake and the incidence of diabetes. These associations were expressed as odds ratios and 95% confidence interval. Groups were compared using the *t* test and a *P* value of <0.05 was considered statistically significant.

Analysis was conducted using the STATA statistical software package version 10.0 (Stata Corp, College Station, TX, USA).

Results

The response rate for the follow-up was 60.0% (6537 of 11 005). Compared with those who did not attend (*n* 4710), attendees (*n* 6537) were significantly less likely to be hypertensive, to have a lower level of education attainment and to be smokers, and had lower 2 h post-load plasma glucose and smaller waist circumference at baseline⁽¹⁰⁾.

Of the 5842 attendees who were free of diabetes at baseline, eighty-five did not complete a dietary questionnaire at baseline, leaving 5757 participants among whom 220 cases of incident diabetes were observed. After excluding participants in the top and bottom 1% of energy intake and those in the top 1% of dairy intake, 5582 participants remained eligible for the analysis, among whom 209 cases of incident diabetes were observed.

Table 1 describes the baseline characteristics of those who developed diabetes over follow-up *v.* those who did not. Table 2 shows that the intake of total dairy food was similar between men and women. However, there were significant differences between men and women with regard to individual dairy food groups, such that women were more likely than men to consume low-fat milk and yoghurt.

Table 2 Median and 25th, 75th percentile of total dairy and individual dairy food intakes and proportion of the population with zero intake: adults aged ≥ 25 years, the Australian Diabetes Obesity and Lifestyle Study (AusDiab)

Dairy food (g)	Men			Women			<i>P</i> *
	Median	25th, 75th	% with zero intake	Median	25th, 75th	% with zero intake	
Total dairy	346	346, 408	0.6	351	217, 430	0.7	0.67
Total milk	254	200, 376	10.4	313	200, 376	11.6	0.83
Low-fat milk	313	200, 375	52.1	375	200, 375	39.3	0.01
Full-fat milk	200	200, 375	55.8	200	200, 375	68.8	0.06
Yoghurt	20	3, 73	35.3	41	10, 73	18.4	<0.001
Cheese	11	6, 20	7.5	14	6, 22	5.9	0.18

**P* value is for the difference in median intake between men and women for all participants. Presented medians were calculated for consumers only.

Table 3 The association between total dairy food intake (servings/d) and the incidence of diabetes over 5 years: adults aged ≥ 25 years, the Australian Diabetes Obesity and Lifestyle Study (AusDiab)

	Men		Women		Total	
	OR*	95% CI	OR*	95% CI	OR*	95% CI
Tertile 1	1.00	Ref.	1.00	Ref.	1.00	Ref.
Tertile 2	0.98	0.62, 1.56	0.79	0.48, 1.33	0.90	0.64, 1.27
Tertile 3	0.53	0.29, 0.96	0.86	0.50, 1.45	0.71	0.48, 1.05

*Odds ratios from logistic regression models of diabetes and dairy intake adjusted for age, sex (for total population only), energy intake, family history of diabetes, education level, level of physical activity, smoking status, TAG, HDL cholesterol, systolic blood pressure, waist circumference and hip circumference. Only those with complete data were included in these analyses (*n* 5582).

We observed that an increased total baseline dairy food intake was associated with a significantly reduced risk of developing diabetes in men (Table 3; adjusted OR = 0.53, 95% CI 0.29, 0.96 for third *v.* first tertile; *P* = 0.033). For women, dairy intake was also associated with reduced risk of diabetes (albeit at a lower magnitude than men), but this did not reach statistical significance (Table 3; adjusted OR = 0.86, 95% CI 0.45, 1.45 for third *v.* first tertile; *P* = 0.516). When we additionally adjusted for Ca intake this relationship was not attenuated (data not shown). Further adjustment for fibre intake had little impact on most results, but the odds ratio for incident diabetes for the third dairy tertile in the whole population was strengthened and became marginally significant (OR = 0.67, 95% CI 0.45, 0.995; *P* = 0.047). Examination of the types of dairy food consumed indicated that the benefit associated with total dairy intake in men was predominantly driven by the effects of low-fat milk and cheese (Table 4). There was a decreased risk of diabetes in the third tertile compared with the first tertile of low-fat milk intake in both men and women, although this was significant only in the total population (Table 4). Further adjustment for fibre intake in models for individual dairy items such as low-fat milk, high-fat milk, cheese and yoghurt had little impact on the results.

Discussion

In the large, prospective AusDiab study, a relationship between increased total dairy food intake and reduced

risk of diabetes among men, independent of age, family history of diabetes, energy intake, smoking status, level of education, level of physical activity, TAG, HDL-C, systolic blood pressure, waist circumference, hip circumference and Ca intake, was evident. However, a similar significant relationship was not present among women.

This finding has previously only been reported in studies in which all data (including diabetes status) were self-reported. In a 12-year prospective study of 41 254 males, Choi *et al.* found that men with a higher intake of dairy also had a reduced risk of diabetes. After adjusting for potential confounders, Choi *et al.* found that men in the fifth quintile of dairy food intake had a relative risk of developing diabetes of 0.77 (95% CI 0.62, 0.95) compared with those in the first quintile⁽²⁾. Lui *et al.* explored the relationship between dairy food intake and incidence of diabetes among women and found an inverse association that was largely attributable to low-fat dairy food intake and not full-fat dairy food intake⁽⁴⁾. In our analyses, low-fat milk intake was inversely associated with risk of diabetes among men and women, but there was no evidence of such an association for full-fat milk. The small difference in saturated fat intake is unlikely to account for this difference. However, other unmeasured characteristics of people who choose low-fat dairy products may be relevant⁽¹⁸⁾. This might indicate that dairy food itself does not contain the active component providing protection against diabetes or that, despite the intake of a protective factor in dairy food, people consuming large amounts of high-fat dairy have other behavioural characteristics that offset this benefit. For total dairy intake

Table 4 The association between individual dairy products (servings/d), calcium (g/d) and the incidence of diabetes over 5 years, after multiple adjustments: adults aged ≥ 25 years, the Australian Diabetes Obesity and Lifestyle Study (AusDiab)

	Men		Women		Total	
	OR*	95 % CI	OR*	95 % CI	OR*	95 % CI
Low-fat milk						
Tertile 1	1.00	Ref.	1.00	Ref.	1.00	Ref.
Tertile 2	1.07	0.66, 1.70	0.69	0.41, 1.14	0.85	0.60, 1.2
Tertile 3	0.57	0.32, 1.02	0.67	0.40, 1.12	0.65	0.44, 0.94
Full-fat milk						
Tertile 1	1.00	Ref.	1.00	Ref.	1.00	Ref.
Tertile 2	1.37	0.84, 2.22	1.39	0.83, 2.32	1.38	0.97, 1.97
Tertile 3	1.43	0.83, 2.45	0.89	0.45, 1.75	1.18	0.78, 1.79
Yoghurt						
Tertile 1	1.00	Ref.	1.00	Ref.	1.00	Ref.
Tertile 2	0.91	0.55, 1.51	0.87	0.52, 1.46	0.88	0.62, 1.26
Tertile 3	1.02	0.56, 1.88	1.23	0.74, 2.04	1.14	0.78, 1.67
Cheese						
Tertile 1	1.00	Ref.	1.00	Ref.	1.00	Ref.
Tertile 2	0.78	0.49, 1.24	0.80	0.50, 1.30	0.81	0.58, 1.12
Tertile 3	0.69	0.39, 1.21	0.83	0.48, 1.45	0.78	0.53, 1.15
Ca						
Tertile 1	1.00	Ref.	1.00	Ref.	1.00	Ref.
Tertile 2	1.05	0.63, 1.73	0.61	0.36, 1.04	0.83	0.57, 1.19
Tertile 3	0.66	0.34, 1.27	0.79	0.43, 1.45	0.74	0.48, 1.15

Ref., referent category.

*Odd ratios from logistic regression models of diabetes and dairy intake adjusted for age, sex (for total population only), energy intake, family history of diabetes, education level, level of physical activity, smoking status, TAG, HDL cholesterol, systolic blood pressure, waist circumference and hip circumference. Only those with complete data were included in these analyses (n 5582).

in the total population, the association was slightly strengthened by adjusting for fibre, a finding that has been previously demonstrated in the literature⁽¹⁹⁾.

Our findings appear inconsistent with the findings of the Hoorn Study, which found no association between baseline dairy consumption and the change in fasting blood glucose over 6.4 years⁽⁸⁾. It should be noted that participants in the Hoorn Study were all aged over 50 years baseline, compared with 25 years or more in AusDiab, although in our data the relationship between dairy intake and diabetes was retained when the analysis was restricted to those aged over 50 years (n 2699, data not shown) but not in the younger participants (n 2883).

The mechanism behind the possible relationship between dairy food intake and reduction in diabetes risk remains unclear. It has been suggested that it could be due to the satiety effect of dairy components such as protein and Ca⁽⁴⁾, although it is noteworthy that our findings were independent of total energy intake; and obesity defined using both waist and hip circumference. Nevertheless, some studies have reported that higher dairy food intake may protect against obesity^(5,20–22). It has been suggested that Ca and Mg may be the key dairy components that lower the risk of type 2 diabetes^(2,23–26). However, in our analysis, the relationship between Ca intake and incident diabetes was weaker than that between total dairy food intake and diabetes. Furthermore, when we adjusted for Ca intake, the relationship between total dairy food intake and diabetes was not attenuated. Thus, our data do not support the hypothesis

that it is the Ca component of dairy food that provides protection against the development of diabetes.

The inverse association between dairy food intake and diabetes risk was significant only in men and not women. Gender disparity has also occurred in two other prospective studies^(7,27). Kirii *et al.* reported the opposite effect of gender, in which a marginally significant risk reduction associated with the highest intake of Ca and dairy was seen in women but not men, and suggested that this may be due to women having an overall higher Ca and dairy food intake⁽⁷⁾. The difference between the total dairy intakes among men and women in our study was small and not significant (median total dairy food intake: 346 g/d for men and 354 g/d for women, $P=0.2$). Furthermore, we calculated the grams of dairy food per kilojoule of energy intake for men and women to be 3.8% and 5.1%, respectively. Since women have a higher relative intake from dairy food than men, the absence of an association in women is not likely to be explained by a low dairy intake.

Strengths and limitations

There were a number of limitations to the present study. The FFQ was developed for the Melbourne Collaborative Cohort Study (MCCS) in the late 1980s, based on weighed food records from men and women between the ages of 40 and 69 years who were born in Australia, Italy or Greece, and thus may not include all relevant foods for the AusDiab participants. A limitation of FFQ in general is that participants can only provide responses for food

items listed in the questionnaire, leaving room for potential omission of important components of an individual's dietary intake. For example, soft drinks were excluded from this FFQ as it was found that very few people from the MCCS population consumed this item. This exclusion may have had an impact on our overall results as it contributes to total energy consumption, and the intake of sugar-sweetened beverages has been associated with type 2 diabetes⁽²⁸⁾. However, this needs to be taken into consideration with the overall difficulties of measuring diet in large cohorts, and although FFQ may have their limitations they are logistically and financially much easier to implement in large studies than other dietary measurement methods.

The response rate at baseline was 55%⁽⁹⁾ and only 60% of participants were followed up. Therefore, the results may not reflect the general Australian population. However, the findings that the incidence of self-reported diabetes was very similar in those who attended the biomedical follow-up testing and the 2200 participants who completed only the self-report questionnaires for follow-up⁽¹⁰⁾, and that the predictors of self-reported diabetes did not change if these non-attendees were grouped with attendees (data not shown), suggest that the impact of the response rate on our findings is likely to be small.

Conclusions

In men with total dairy food intake in the third tertile compared with the first tertile, the risk of developing diabetes over 5 years was reduced by nearly 50%. Further, among men in the third tertile of low-fat milk intake, compared with men in the first tertile, the odds of developing diabetes was approximately 40% lower, but this was of borderline significance ($P=0.059$). There were no similar significant relationships in women. Further investigations into the relationship between dairy food intake and diabetes risk need to be undertaken to fully understand the potential mechanism and implications of this observation.

Acknowledgements

Funding from The Dairy Health and Nutrition Consortium (DHNC) is gratefully acknowledged. The DHNC is a consortium of Tatura Milk Industries & Bega Cheese, National Foods, Fonterra Australia, Parmalat Australia, Dairy Australia, Geoffrey Gardiner Foundation, Murray Goulburn Co-operative, Warrnambool Cheese & Butter Factory, and Dairy Innovation Australia. The AusDiab study, co-coordinated by the Baker IDI Heart and Diabetes Institute, gratefully acknowledges the generous support given by: the National Health and Medical Research Council (NHMRC grant 233200); the Australian

Government Department of Health and Ageing; Abbott Australasia Pty Ltd; Alphapharm Pty Ltd; AstraZeneca; Bristol-Myers Squibb; the City Health Centre, Diabetes Service, Canberra; the Department of Health and Community Services – Northern Territory; the Department of Health and Human Services – Tasmania; the Department of Health – New South Wales; the Department of Health – Western Australia; the Department of Health – South Australia; the Department of Human Services – Victoria; Diabetes Australia; Diabetes Australia Northern Territory; Eli Lilly Australia; the Estate of the Late Edward Wilson; GlaxoSmithKline; the Jack Brockhoff Foundation; Janssen-Cilag; Kidney Health Australia; the Marian & FH Flack Trust; Menzies Research Institute; Merck Sharp & Dohme; Novartis Pharmaceuticals; Novo Nordisk Pharmaceuticals; Pfizer Pty Ltd; the Pratt Foundation; Queensland Health; Roche Diagnostics Australia; Royal Prince Alfred Hospital, Sydney; Sanofi Aventis; and Sanofi Synthelabo. D.J.M. is supported by a Victorian Cancer Agency Public Health Fellowship. J.E.S. is supported by an NHMRC Senior Research Fellowship (540103). All authors declare no conflict of interest with regard to this manuscript. All authors contributed to the conception and design of the study. N.M.G. conducted the analysis and drafted the manuscript. All authors were involved in the preparation of the final manuscript. All authors were involved in the preparation of the final manuscript. For their invaluable contribution to the set-up and field activities of AusDiab, the authors are enormously grateful to A. Allman, B. Atkins, S. Bennett, A. Bonney, S. Chadban, M. de Courten, M. Dalton, D. Dunstan, T. Dwyer, H. Jahangir, D. Jolley, D. McCarty, A. Meehan, N. Meinig, S. Murray, K. O'Dea, K. Polkinghorne, P. Phillips, C. Reid, A. Stewart, R. Tapp, H. Taylor, T. Whalen, F. Wilson and P. Zimmet. Finally, the authors thank the AusDiab participants for volunteering their time to participate in the study.

References

1. Shaw JE, Sicree RA & Zimmet PZ (2010) Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* **87**, 4–14.
2. Choi HK, Willett WC, Stampfer MJ *et al.* (2005) Dairy consumption and risk of type 2 diabetes mellitus in men: a prospective study. *Arch Intern Med* **165**, 997–1003.
3. Elwood PC, Pickering JE & Fehily AM (2007) Milk and dairy consumption, diabetes and the metabolic syndrome: the Caerphilly prospective study. *J Epidemiol Community Health* **61**, 695–698.
4. Liu S, Choi HK, Ford E *et al.* (2006) A prospective study of dairy intake and the risk of type 2 diabetes in women. *Diabetes Care* **29**, 1579–1584.
5. Moore LL, Bradlee ML, Gao D *et al.* (2006) Low dairy intake in early childhood predicts excess body fat gain. *Obesity (Silver Spring)* **14**, 1010–1018.
6. Snijder MB, van der Heijden AA, van Dam RM *et al.* (2007) Is higher dairy consumption associated with lower body weight and fewer metabolic disturbances? The Hoorn Study. *Am J Clin Nutr* **85**, 989–995.

7. Kirii K, Mizoue T, Iso H *et al.* (2009) Calcium, vitamin D and dairy intake in relation to type 2 diabetes risk in a Japanese cohort. *Diabetologia* **52**, 2542–2550.
8. Snijder MB, van Dam RM, Stehouwer CD *et al.* (2008) A prospective study of dairy consumption in relation to changes in metabolic risk factors: the Hoorn Study. *Obesity (Silver Spring)* **16**, 706–709.
9. Dunstan DW, Zimmet PZ, Welborn TA *et al.* (2002) The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) – methods and response rates. *Diabetes Res Clin Pract* **57**, 119–129.
10. Magliano DJ, Barr EL, Zimmet PZ *et al.* (2008) Glucose indices, health behaviors, and incidence of diabetes in Australia: the Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care* **31**, 267–272.
11. World Health Organization & International Diabetes Federation (2006) *Definition and Diagnosis of Diabetes Mellitus and Intermediate Hyperglycaemia. Report of WHO/IDF Consultation*. Geneva: WHO.
12. Briganti EM, Shaw JE, Chadban SJ *et al.* (2003) Untreated hypertension among Australian adults: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Med J Aust* **179**, 135–139.
13. Australian Institute of Health and Welfare (1998) *Standard Questions on the Use of Tobacco Among Adults*. Canberra: AIHW.
14. Australian Institute of Health and Welfare (2003) *The Active Australia Survey: A Guide and Manual for Implementation, Analysis and Reporting*. Canberra: AIHW.
15. Hodge A, Patterson AJ, Brown WJ *et al.* (2000) The Anti Cancer Council of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in young to middle-aged women in a study of iron supplementation. *Aust N Z J Public Health* **24**, 576–583.
16. Lewis J, Milligan G, Hunt A (1995) *NUTTAB95 Nutrient Data Table for Use in Australia*. Canberra: Australian Government Publishing Service.
17. Hodge AM, English DR, O'Dea K *et al.* (2007) Dietary patterns and diabetes incidence in the Melbourne Collaborative Cohort Study. *Am J Epidemiol* **165**, 603–610.
18. Mensink RP (2006) Dairy products and the risk to develop type 2 diabetes or cardiovascular disease. *Int Dairy J* **16**, 1001–1004.
19. de Munter JS, Hu FB, Spiegelman D *et al.* (2007) Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. *PLoS Med* **4**, e261.
20. Barba G, Troiano E, Russo P *et al.* (2005) Inverse association between body mass and frequency of milk consumption in children. *Br J Nutr* **93**, 15–19.
21. Eagan MS, Lyle RM, Gunther CW *et al.* (2006) Effect of 1-year dairy product intervention on fat mass in young women: 6-month follow-up. *Obesity (Silver Spring)* **14**, 2242–2248.
22. Zemel MB (2005) The role of dairy foods in weight management. *J Am Coll Nutr* **24**, 6 Suppl., 537S–546S.
23. Colditz GA, Manson JE, Stampfer MJ *et al.* (1992) Diet and risk of clinical diabetes in women. *Am J Clin Nutr* **55**, 1018–1023.
24. Pittas AG, Dawson-Hughes B, Li T *et al.* (2006) Vitamin D and calcium intake in relation to type 2 diabetes in women. *Diabetes Care* **29**, 650–656.
25. Simmons D, Joshi S & Shaw J (2010) Hypomagnesaemia is associated with diabetes: not pre-diabetes, obesity or the metabolic syndrome. *Diabetes Res Clin Pract* **87**, 261–266.
26. van Dam RM, Hu FB, Rosenberg L *et al.* (2006) Dietary calcium and magnesium, major food sources, and risk of type 2 diabetes in US black women. *Diabetes Care* **29**, 2238–2243.
27. Mennen LL, Feskens EJM, Novak M *et al.* (2000) Possible protective effect of bread and dairy products on the risk of the metabolic syndrome. *Nutr Res* **20**, 335–347.
28. Malik VS, Popkin BM, Bray GA *et al.* (2010) Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes Care* **33**, 2477–2483.