The effect of a high-egg diet on cardiovascular risk factors in people with type 2 diabetes: the Diabetes and Egg (DIABEGG) study—a 3-mo randomized controlled trial[1-4]

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ABSTRACT

Background: Previously published research that examined the effects of high egg consumption in people with type 2 diabetes (T2D) produced conflicting results leading to recommendations to limit egg intake. However, people with T2D may benefit from egg consumption because eggs are a nutritious and convenient way of improving protein and micronutrient contents of the diet, which have importance for satiety and weight management.
Objective: In this randomized controlled study, we aimed to determine whether a high-egg diet (2 eggs/d for 6 d/wk) compared with a low-egg diet (<2 eggs/wk) affected circulating lipid profiles, in particular high-density lipoprotein (HDL) cholesterol, in overweight or obese people with prediabetes or T2D.
Design: A total of 140 participants were randomly assigned to one of the 2 diets as part of a 3-mo weight maintenance study. Participants attended the clinic monthly and were instructed on the specific types of foods and quantities to be consumed.
Results: There was no significant difference in the change in HDL cholesterol from screening to 3 mo between groups; the mean difference (95% CI) between high- and low-egg groups was +0.02 mmol/L (−0.03, 0.08 mmol/L; $P = 0.38$). No between-group differences were shown for total cholesterol, low-density lipoprotein cholesterol, triglycerides, or glycemic control. Both groups were matched for protein intake, but the high-egg group reported less hunger and greater satiety postbreakfast. Polyunsaturated fatty acid (PUFA) and monounsaturated fatty acid (MUFA) intakes significantly increased from baseline in both groups.
Conclusions: High egg consumption did not have an adverse effect on the lipid profile of people with T2D in the context of increased MUFA and PUFA consumption. This study suggests that a high-egg diet can be included safely as part of the dietary management of T2D, and it may provide greater satiety. This trial was registered at the Australia New Zealand Clinical Trials Registry (http://www.anzctr.org.au/) as ACTRN12612001266853. *Am J Clin Nutr* 2015;101:705–13.

INTRODUCTION

Type 2 diabetes (T2D)[5] is one of the most-common chronic diseases worldwide (1). Therefore, interventions to manage T2D and its complications are a priority. Currently, guidelines differ between countries regarding egg consumption and total dietary cholesterol intake (2–4). The Australian National Heart Foundation recommends a maximum of 6 eggs/wk for healthy people and those with T2D (2), whereas US guidelines for people with T2D recommend that dietary cholesterol be limited to <300 mg/d (one egg contains ~200 mg cholesterol) and <4 eggs/wk (3). However, in the United Kingdom, there is no suggested limit on the number of eggs consumed, and instead, emphasis is placed on a dietary reduction of SFAs (4).

Eggs contain a number of nutrients (5, 6) that may reduce risk of T2D and cardiovascular disease (CVD), including folate (7) and arginine (8, 9). Eggs were also shown to increase circulating concentrations of HDL cholesterol (10, 11), which is associated with reduced CVD risk (12). Although eggs are rich in cholesterol, the total amount of fat is not high (5.2 g) and is predominantly unsaturated (51% MUFAs; 16% PUFAs) (5). In addition, their high protein content might improve satiety (13) thereby assisting with weight management.

Despite the positive nutritional value of eggs, there is a negative perception toward egg consumption for people with T2D. A number of epidemiologic studies indicated that high egg consumption, although not associated with adverse CVD outcomes in the general population, may be associated with worse CVD outcomes in people with T2D (14–16). However, the findings in such studies were affected by many confounding factors. For example, at the time that these epidemiologic studies were being conducted, a public health campaign advised people to limit

their cholesterol intake, including their consumption of eggs. Individuals who consumed >6 eggs/wk at that time may have been less likely to follow healthy dietary and lifestyle advice in general.

Furthermore, there is a lack of good-quality prospective data on the effects of high egg consumption in people with T2D. To our knowledge, there has only been one short-duration randomized controlled trial in people with T2D (10). In this study, people with T2D who consumed a high-cholesterol (2 eggs/d), reduced-energy diet lost the same amount of weight and had similar improvements in circulating lipid concentrations, blood pressure, and glycemic control as did those who consumed an isoenergetic low-cholesterol diet.

To address the limitations of previous research, this prospective randomized controlled study had a 3-mo active-intervention period of weight maintenance to determine the potential health effects of a high-egg diet in people with prediabetes and T2D. We hypothesized that there would be a significant increase in HDL cholesterol with high egg intake and that subjects in the high-egg group would report greater satiety.

METHODS

Study design and participants

This prospective, randomized controlled, parallel-arm study was conducted in accordance with International Conference on Harmonization–Good Clinical Practice guidelines. A total of 140 participants, aged ≥18 y, who were diagnosed with prediabetes or T2D and had BMI (in kg/m^2) ≥25 were recruited from the Boden Institute database, the Sydney Local Health District intranet, the University of Sydney website, and advertising in local newspapers. Prediabetes and T2D inclusion criteria were based on American Diabetes Association Guidelines (17). Participants diagnosed with T2D and taking antidiabetic medications were included.

Participants were excluded if they had T2D and glycated hemoglobin (Hb A_{1c}) >9.5%; unstable angina or recent onset of CVD (≤1 mo of screening); a history of significant liver, kidney, or gastrointestinal disease; untreated thyroid disease; a history or presence of malignancy; a history of alcohol abuse or illicit drug use; previous gastric surgery or gastric banding; used weight loss medications or other drugs that affect body weight ≤3 mo of screening; commenced a new medication or a change in dose regimen of prescription medication ≤3 mo of screening; were pregnant, breastfeeding, or planning a pregnancy during the study; followed vegetarian eating practices; had an egg allergy; or changed smoking habits or ceased smoking ≤6 mo before screening.

Participants who were taking hypoglycemic or hypolipidemic agents were not excluded from the study. When possible, participants were required to keep their diabetic and cholesterol medications and dosages constant throughout the study. This was done in conjunction with their primary care providers.

All experimental procedures were approved by the University of Sydney Human Ethics Review Committee, and all participants provided written informed consent. All clinic visits took place at the Boden Institute, the University of Sydney. This trial was registered at the Australia New Zealand Clinical Trials Registry (http://www.anzctr.org.au/) as ACTRN12612001266853.

Dietary interventions

Participants were randomly assigned in a 1:1 ratio to either a high- or low-egg diet. Participants met the study dietitian at the baseline visit (≤1 wk of the screening visit) and were advised about their diet allocation and diet prescription for weight maintenance. Participants who consumed the high-egg diet were instructed to eat 2 eggs/d at breakfast for 6 d/wk (12 eggs/wk). Subjects in the low-egg group were directed to consume <2 eggs/wk and match the protein intake of the high-egg group at breakfast with 10 g lean animal protein (meat, chicken, or fish) or other protein-rich alternatives such as legumes and reduced fat dairy products. Participants were given a booklet as a guide to the specific types of foods and quantities to be consumed with particular emphasis on replacing foods containing saturated fats with foods containing MUFAs and PUFAs to improve diet quality and maintain energy intake over the 3 mo (**Supplemental Material 1**). The diets were energy and macronutrient matched. Participants were instructed not to change their activity level. To aid with compliance, participants who consumed the high-egg diet were given the prescribed quota of eggs, and participants who consumed the low-egg diet received a grocery voucher of equivalent value (5 Australian dollars/wk; 20 Australian dollars/mo).

Primary and secondary outcomes

All major assessments were conducted at screening (anthropometric measures, vital signs, and pathology), baseline (nutritional analysis and questionnaires), and 3 mo (all outcomes). Body weight, waist circumference, the recording of side effects, any medication changes, and a dietetic review were conducted at monthly visits. Participants attended the clinic at week 2 of the study to ensure they were not losing weight after the implementation of the initial dietary changes. The primary outcome for the study was the change in HDL cholesterol at 3 mo. All other assessments were secondary outcomes.

Pathology

Blood samples were collected for fasting blood glucose, Hb A_{1c}, total cholesterol, HDL cholesterol, LDL cholesterol, high-sensitivity C-reactive protein, and apolipoprotein B. Blood was also collected for a full blood count, thyroid function, and liver and renal function to assess eligibility criteria at the screening visit.

Change in anthropometric measures and vital signs

Height was measured by using a wall-mounted stadiometer (accurate to 0.5 cm), body weight was measured by using a calibrated scale (correct to the nearest 0.1 kg), waist circumference was measured at the midpoint between the highest point of the iliac crest and lowest part of the costal margin in the midaxillary line (to the nearest 0.5 cm), mean systolic and diastolic blood pressures were measured twice in the same arm each time (if a difference >10 mm Hg was shown in either reading, a third reading was taken; each measure was taken by using the same digital sphygmomanometer), heart rate was measured by using the radial pulse, and total body fat and fat-free mass were measured by using a bioelectrical impedance analysis (Tanita BC Analyzer BC-418; Tanita).

Nutritional analysis

A weighed 5-d (self-reported) food diary (4 working days and one weekend day) was collected by the dietitian at the baseline and 3-mo visits. Food diaries were analyzed with Food Works 7 Professional (version 7) software (2013; Xyris Software) on the basis of Australian Food Composition tables and food manufacturers' data. Dietary compliance was defined and measured primarily by the quantity of eggs reported in the 5-d food diaries. Subjects who consumed ≥12 eggs/wk were compliant in the high-egg group, and subjects who consumed <2 eggs/d were compliant in the low-egg group. We also assessed intakes of saturated fat, MUFAs, and PUFAs as a measure of compliance because this was in line with the dietary prescription that was provided (Supplemental Material 1).

Questionnaires

The following questionnaires were completed by participants at baseline and 3 mo: 1) the Three-Factor Eating Questionnaire-R21 (18), 2) the International Physical Activity Questionnaire–short version (19), 3) the Impact of Weight on Quality of Life Questionnaire-Lite Version (20), 4) The Food Acceptability Questionnaire (21, 22), and 5) a visual analog scale [an appetite score for before and after breakfast (23)] whereby participants were required to answer questions 30 min before and after breakfast).

Statistical analysis

Sample size. A total of 140 participants were recruited into the study. With the assumption of a detectable difference in HDL cholesterol of 0.12 mmol/L between the 2 groups at 3 mo and an SD of 0.24 mmol/L, 126 participants were required to achieve 80% power of detecting a treatment effect (2-sided significance level of 5%). With allowance for a 10% dropout rate over the 3 mo, a sample size of 140 was needed to enable adequate power to assess our primary outcome of a 10% difference in HDL cholesterol between the 2 groups as shown in a previous study (11).

Random assignment. Treatment groups were stratified during random assignment according to age, sex, cholesterol medication treatment, and diabetes status (prediabetes or T2D). Once stratified, participants were randomly allocated by using computer-generated random numbers to either the high- or low-egg–diet group. This allocation was performed by one of the investigators (GD) who did not have any involvement with participant contact or clinic visits. Furthermore, outcome assessors were blinded to the treatment allocation during the data analysis.

Data analysis. The statistical analysis was performed with SPSS 19.0 software (SPSS). All participants who were randomly assigned and completed an initial assessment were included in the final results by using an intention-to-treat analysis. Multiple imputation with the use of linear regression was used to impute missing values from screening/baseline to 3 mo and was based on the assumption that data were missing at random. Five imputed data sets were created for each variable. An ANCOVA was used to compare treatment groups. Analyses were adjusted for the screening/baseline observation. An ANOVA with repeated measures was used for within-group comparisons. A completer's analysis was also performed for the primary and secondary outcomes, whereby only subjects who completed the outcome measures and were dietary compliant to the high- or low-egg

diet were included as assessed by the number of eggs reported in the food diary per week. A 2-sided $P < 0.05$ was considered statistically significant. Values are presented as mean (±SDs). Between-group differences are presented as means (95% CIs) and represent the δ change between screening/baseline and 3 mo for the 2 groups. Differences in screening/baseline characteristics were analyzed by using independent sample t tests.

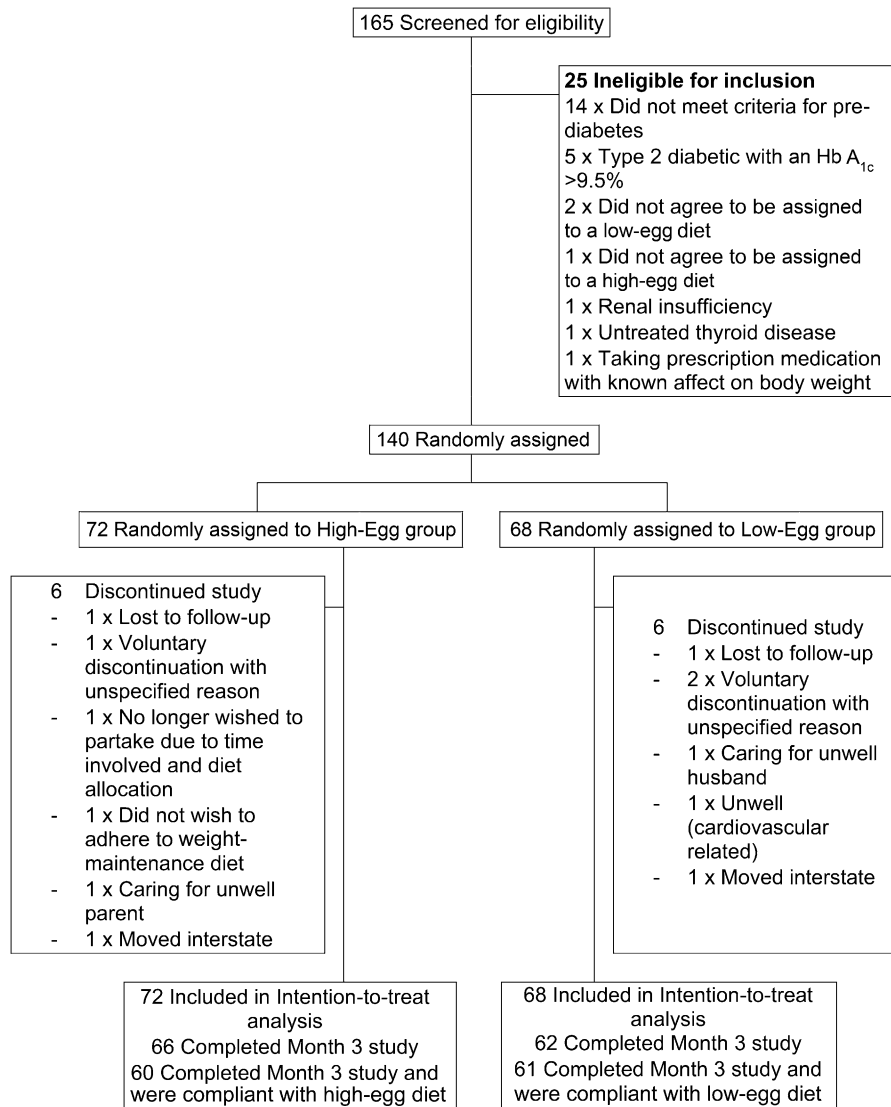## RESULTS

Trial disposition

A flowchart that detailed the participant disposition is provided in **Figure 1**. The study was conducted between January and September 2013. There was a dropout rate of 9% [8.6% = 12 participants (6 subjects from each group)] at the end of the 3-mo intervention. Of subjects who completed the 3 mo, 7 participants were considered to be noncompliant to their allocated diet (i.e., did not follow the high- or low-egg–diet prescription) (Figure 1).

Baseline characteristics

Baseline characteristics of participants are detailed in **Table 1**. Demographic and clinical characteristics were well matched across the 2 groups, although there were significant differences at baseline in total cholesterol (0.30; $P = 0.04$) and HDL cholesterol (0.10; $P = 0.03$) with higher values shown in the high-egg group. There was no significant difference in mean years diagnosed with T2D, which were 7.3 and 5.5 y for high and low-egg–diet groups, respectively. Baseline dietary intake was the same in both groups (**Table 2**).

Medication changes

Changes in hypolipidemic agents during the 3-mo study occurred in 3 participants in the low-egg group (one subject ceased rosuvastatin, and 2 subjects had an increase in statin dose). One participant in the high-egg group commenced a psoriasis medication (acitretin) that is an agent with a known adverse effect on lipid profile. A combined antihypertensive and hypolipidemic agent (amlodipine/atorvastatin) was commenced by one participant in the high-egg group. Two participants in the high-egg group had changes in hypoglycemic medication during the 3-mo study (one subject commenced exenatide, and one subject had a decreased dosage of 30% soluble insulin aspart:70% protamine-crystallized insulin aspart), and 5 participants in the low-egg group recorded changes [one subject commenced gliclazide, one subject increased the dosage of insulin, isophane (rys), one subject changed medication from sitagliptin/metformin to vildagliptin/metformin, one subject commenced metformin, and one subject commenced sitagliptin]. Five participants in the high-egg group had changes in antihypertensive medication during the 3-mo study (one subject increased the dosage of candesartan, one subject commenced labetalol, one subject increased the dosage of perindopril/amlodipine, one subject reduced the dosage of perindopril, and one subject reduced the dosage of perindopril/amlodipine), and 3 participants in the low-egg group recorded changes (one subject commenced candesartan, one subject increased the dosage of perindopril/amlodipine, and one subject reduced the dosage of ramipril and commenced amlodipine/telmisartan). These medication changes did not influence the

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┌─────────────────────────────┐
│ 165 Screened for eligibility │
└─────────────────────────────┘
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25 Ineligible for inclusion
14 x Did not meet criteria for pre-diabetes
5 x Type 2 diabetic with an Hb A_{1c} >9.5%
2 x Did not agree to be assigned to a low-egg diet
1 x Did not agree to be assigned to a high-egg diet
1 x Renal insufficiency
1 x Untreated thyroid disease
1 x Taking prescription medication with known affect on body weight

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┌──────────────────────┐
│ 140 Randomly assigned │
└──────────────────────┘
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72 Randomly assigned to High-Egg group | 68 Randomly assigned to Low-Egg group

6 Discontinued study
- 1 x Lost to follow-up
- 1 x Voluntary discontinuation with unspecified reason
- 1 x No longer wished to partake due to time involved and diet allocation
- 1 x Did not wish to adhere to weight-maintenance diet
- 1 x Caring for unwell parent
- 1 x Moved interstate

6 Discontinued study
- 1 x Lost to follow-up
- 2 x Voluntary discontinuation with unspecified reason
- 1 x Caring for unwell husband
- 1 x Unwell (cardiovascular related)
- 1 x Moved interstate

72 Included in Intention-to-treat analysis
66 Completed Month 3 study
60 Completed Month 3 study and were compliant with high-egg diet

68 Included in Intention-to-treat analysis
62 Completed Month 3 study
61 Completed Month 3 study and were compliant with low-egg diet

FIGURE 1 Flow diagram of participants through the study. Hb A_{1c}, glycated hemoglobin.

primary or secondary outcome results because the findings were the same regardless of whether or not these participants' data were included in the intention-to-treat statistical analyses (see primary and secondary descriptive results).

Primary outcome

There was no significant difference in HDL cholesterol from screening to 3 mo between the 2 groups (mean difference for high-egg group compared with low-egg group: +0.02 mmol/L; 95% CI: −0.03, 0.08 mmol/L; $P = 0.38$) (**Table 3**). This result was the same for participants who completed the 3-mo study and were dietary compliant ($n = 121$; high-egg group: $n = 60$; low-egg group: $n = 61$; mean difference for high-egg group compared with low-egg group:+0.04 mmol/L; 95% CI: −0.01, 0.09 mmol/L; $P = 0.15$). Again, when we analyzed only subjects with T2D, there was no statistical difference between groups ($n = 103$; mean difference for high-egg group compared with low-egg group: +0.02 mmol/L; 95% CI: −0.05, 0.08 mmol/L; $P = 0.65$), and this result held when participants who maintained

stable lipid-medication usage during the study ($n = 135$) were analyzed (mean difference for high-egg group compared with low-egg group: +0.03 mmol/L; 95% CI: −0.02, 0.08 mmol/L; $P = 0.26$).

Secondary outcomes

Change in total cholesterol, LDL cholesterol, and triglycerides

There were no significant differences in total cholesterol, LDL cholesterol, triglycerides, or apolipoprotein B from screening to 3 mo between the 2 groups (Table 3). These results were consistent when we analyzed only those participants who maintained stable lipid medication during the study (i.e., no change in medications from baseline), completers only, or only subjects who were compliant with their respective diet (results not shown).

Change in glycemic control

There were no significant differences in fasting glucose or Hb A_{1c} (%) from screening to 3 mo between the 2 groups (Table 3).

TABLE 1
Screening characteristics of study participants ($n = 140$)

Characteristics	High-egg group ($n = 72$)	Low-egg group ($n = 68$)
Sex (F), n (%)	39 (54)	38 (56)
Prediabetic, n (%)	21 (29)	16 (23.5)
Type 2 diabetic, n (%)	51 (71)	52 (76.5)
Years diagnosed with type 2 diabetes	7.3 ± 7.6^1	5.5 ± 4.8
Age, y	59.5 ± 9.7	60.1 ± 11.1
>45 y, n (%)	66 (92)	60 (88)
BMI, kg/m^2	35.4 ± 6.4	33.7 ± 5.9
Taking cholesterol medication, n (%)	40 (56)	35 (52)
HDL cholesterol, mmol/L	1.3 ± 0.3	1.2 ± 0.2^2
LDL cholesterol, mmol/L	2.9 ± 0.9	2.6 ± 1.0^3
Total cholesterol, mmol/L	5.0 ± 1.1	4.7 ± 1.1^4
Triglycerides, mmol/L	1.7 ± 0.7	1.8 ± 0.8
Fasting blood glucose, mmol/L	6.7 ± 1.7	6.9 ± 1.7
Hb A$_{1c}$,[5] %	6.6 ± 1.0	6.6 ± 0.9
Hb A$_{1c}$, mmol/mol	48.0 ± 11.4	48.9 ± 9.9
C-reactive protein, mg/L	5.1 ± 5.4	4.9 ± 8.5
Apolipoprotein B, g/L	1.1 ± 0.3	1.0 ± 0.3
Weight, kg	98.0 ± 20.4	93.3 ± 17.8
Waist circumference, cm	111.4 ± 13.7	109.6 ± 13.8
Total body fat, %	29.4 ± 11.0	29.0 ± 10.5
Fat-free mass, kg	69.7 ± 19.7	66.5 ± 17.7
Systolic blood pressure, mm Hg	136.0 ± 18.0	136.6 ± 14.6
Diastolic blood pressure, mm Hg	82.2 ± 10.2	82.4 ± 7.6
Radial pulse rate, beats/min	70.3 ± 9.2	71.7 ± 11.3

[1]Mean $\pm$ SD (all such values).
[2-4]P values of baseline differences between groups (t tests were used for the comparison of means between groups):
[2]$P = 0.03$, [3]$P = 0.05$, [4]$P = 0.04$.
[5]Hb A$_{1c}$, glycated hemoglobin.

This result was also consistent when we analyzed participants who maintained stable antidiabetic medication throughout the study, completers only, or only subjects who were compliant to their respective diet (results not shown).

Change in anthropometric measures and vital signs

Participants successfully maintained their body weight ≤1 kg of their initial weight, and there were no significant differences in weight change over the 3-mo period between groups. Similarly, no differences in waist circumference, total body fat, fat-free mass, blood pressure, or heart rate between groups were identified (Table 3).

Dietary changes

At 3 mo, when all participants ($n = 140$) were included, the high-egg group reported consuming a mean ($\pm$SD) of 11.8 $\pm$ 3.6 eggs/wk; whereas the low-egg group reported consuming 1.0 $\pm$ 1.8 eggs/wk.

There was no difference between groups for protein intake from baseline to 3 mo. A small but significant difference was evident between groups from baseline to 3 mo for carbohydrate, total fat, and fiber intake (**Table 4**). Saturated fat intake decreased significantly in both groups from 14.0% and 13.7% to 13.3% and 11.4% of total fat intake at 3 mo for high- and low-egg groups, respectively. PUFA and MUFA intakes significantly

TABLE 2
Baseline dietary intake of study participants ($n = 138$)[1]

Characteristics	High-egg group ($n = 70$)	Low-egg group ($n = 68$)
Energy, kJ	7795 ± 2259	7374 ± 1837
Protein, percentage of energy	20.4 ± 3.9	20.7 ± 4.9
Carbohydrates, percentage of energy	40.3 ± 7.3	41.6 ± 7.7
Total fat, percentage of energy	33.4 ± 5.9	32.2 ± 6.1
Alcohol, percentage of energy	2.6 ± 4.2	2.4 ± 3.8
Saturated fat, percentage of total fat	42.0 ± 5.3	42.7 ± 6.2
Polyunsaturated fat, percentage of total fat	17.3 ± 3.7	17.2 ± 4.6
Monounsaturated fat, percentage of total fat	40.6 ± 3.9	40.1 ± 4.1
Dietary fat, g	22.5 ± 8.0	20.3 ± 6.3
Cholesterol, mg	318.2 ± 140.6	298.8 ± 141.0

[1]All values are means $\pm$ SDs. Two participants (high-egg group) did not complete a baseline food diary. Dietary intakes are written as a percentage of total energy intake unless otherwise indicated. t tests were used for the comparison of means between groups. No significant differences were shown for any of the characteristics between groups.

TABLE 3
Change from screening for blood pathology results, anthropometric variables, and vital signs at 3 mo ($n = 140$)[1]

Variables	High-egg group ($n = 72$)[2]	Low-egg group ($n = 68$)[2]	Between-group comparison (high egg relative to low egg)[3]
HDL cholesterol (primary efficacy), mmol/L	0.034 ± 0.158 (0.07)	0.029 ± 0.170 (0.17)	0.024 (−0.030, 0.077) [0.38]
LDL cholesterol, mmol/L	−0.06 ± 0.64 (0.44)	0.05 ± 0.59 (0.54)	−0.02 (−0.22, 0.17) [0.81]
Total cholesterol, mmol/L	−0.04 ± 0.75 (0.62)	0.02 ± 0.69 (0.85)	0.03 (−0.20, 0.26) [0.81]
Triglycerides, mmol/L	−0.07 ± 0.51 (0.22)	−0.11 ± 0.58 (0.12)	0.00 (−0.16, 0.16) [0.99]
Fasting blood glucose, mmol/L	−0.35 ± 3.61 (0.42)	−0.06 ± 1.31 (0.69)	−0.39 (−1.29, 0.51) [0.39]
Hb A$_{1c}$, %	−0.07 ± 0.43 (0.20)	−0.11 ± 0.46 (0.05)	0.04 (−0.11, 0.19) [0.61]
Hb A$_{1c}$, mmol/mol	−0.58 ± 5.01 (0.33)	−1.25 ± 5.00 (0.04)	0.60 (−1.05, 2.25) [0.48]
hsCRP, mg/L	−0.59 ± 4.44 (0.26)	−0.76 ± 9.75 (0.52)	0.32 (−1.70, 2.34) [0.75]
Apolipoprotein B, g/L	−0.04 ± 0.37 (0.32)	−0.05 ± 0.17 (0.02)	0.02 (−0.07, 0.12) [0.64]
Weight, kg	−0.48 ± 2.13 (0.06)	−0.91 ± 1.96 (<0.0001)	0.43 (−0.25, 1.12) [0.21]
Waist circumference, cm	0.35 ± 2.60 (0.26)	−0.16 ± 3.96 (0.74)	0.51 (−0.60, 1.62) [0.37]
Total body fat, %	1.33 ± 3.95 (<0.01)	1.03 ± 4.14 (0.04)	0.30 (−1.05, 1.65) [0.66]
Fat-free mass, kg	−1.94 ± 5.12 (<0.01)	−1.86 ± 4.56 (0.001)	−0.08 (−1.70, 1.54) [0.92]
Systolic blood pressure, mm Hg	−1.54 ± 17.61 (0.46)	−5.42 ± 13.63 (<0.01)	3.88 (−1.40, 9.16) [0.15]
Diastolic blood pressure, mm Hg	−3.05 ± 6.60 (<0.0001)	−3.82 ± 6.83 (<0.0001)	0.77 (−1.47, 3.01) [0.50]
Radial pulse rate, beats/min	−1.06 ± 6.89 (0.90)	1.57 ± 6.82 (0.06)	−1.68 (−3.97, 0.61) [0.15]

[1]An ANCOVA was used to compare treatment groups. Analyses were adjusted for the screening observation. An ANOVA with repeated measures was used for within-group comparisons. Hb A$_{1c}$, glycated hemoglobin, hsCRP, high-sensitivity C-reactive protein.

[2]All values are mean differences ± SDs; P values in parentheses. P values represent within-group differences from screening to 3 mo.

[3]All values are mean differences; 95% CIs in parentheses; P values in brackets. P values represent between-group differences from screening to 3 mo after adjustment for the screening value.

increased from baseline in both groups (Table 4). These findings were in keeping with the dietary prescription for both groups to replace "bad" fats with "good" fats. However, there were no significant differences in the change in saturated fat, polyunsaturated fat, and monounsaturated fat intakes between groups with the dietary intervention. As expected, there was a significant difference between groups from baseline to 3 mo for dietary cholesterol (Table 4).

Food acceptability

Both groups reported an increase in overall satisfaction of the diet they were allocated compared with baseline dietary habits

with results favoring the high-egg group (**Table 5**). Analyses between groups indicated significant differences that favored the high-egg group for the enjoyment of the foods they were eating and being less bored with food choices. A trend toward being more satisfied with the high-egg diet compared with low-egg diet was also evident (Table 5).

Appetite

There were no differences in the before-breakfast appetite ratings between the 2 groups at 3 mo. However, the high-egg group reported significantly less hunger after breakfast (question 1: "How hungry are you?") and significantly greater satiety

TABLE 4
Change from baseline in mean dietary intake at 3 mo ($n = 135$)[1]

Variables	High-egg group ($n = 67$)[2]	Low-egg group ($n = 68$)[2]	Between-group comparison (high egg relative to low egg)[3]
Energy, kJ[4]	−346.4 ± 3006.2 (0.35)	−11.2 ± 2028.0 (0.96)	0.5 (−672.9, 673.8) [0.99]
Protein, percentage of energy	2.7 ± 8.9 (0.02)	0.9 ± 5.3 (0.17)	1.7 (−0.5, 3.9) [0.14]
Carbohydrates, percentage of energy	−3.9 ± 7.2 (<0.0001)	−1.1 ± 8.3 (0.29)	−3.5 (−5.8, −1.2) [<0.01]
Total fat, percentage of energy	1.4 ± 7.6 (0.15)	−0.5 ± 7.4 (0.57)	2.5 (0.2, 4.8) [0.04]
Alcohol, percentage of energy	−0.3 ± 2.7 (0.42)	0.0 ± 2.7 (0.97)	−0.3 (−1.2, 0.6) [0.57]
Saturated fat, percentage of total fat	−3.7 ± 11.7 (0.01)	−6.5 ± 8.2 (<0.0001)	2.3 (−0.8, 5.4) [0.14]
Polyunsaturated fat, percentage of total fat	1.8 ± 6.6 (0.03)	3.6 ± 5.5 (<0.0001)	−1.7 (−3.7, 0.2) [0.08]
Monounsaturated fat, percentage of total fat	2.5 ± 5.3 (<0.0001)	2.6 ± 5.7 (<0.0001)	0.3 (−1.2, 1.7) [0.71]
Dietary fiber, g	−0.2 ± 8.0 (0.85)	3.6 ± 7.2 (<0.0001)	−2.6 (−4.9, −0.4) [0.02]
Cholesterol, mg	281.5 ± 267.6 (<0.0001)	−36.2 ± 196.3 (0.13)	337.2 (271.7, 402.7) [<0.0001]

[1]Five participants did not return more than one food diary. Each dietary variable is written as a percentage of total energy intake unless otherwise indicated. An ANCOVA was used to compare treatment groups. Analyses were adjusted for baseline observation. An ANOVA with repeated measures was used for within-group comparisons.

[2]All values are mean differences ± SDs; P values in parentheses. P values represent within-group differences from baseline to 3 mo.

[3]All values are mean differences; 95% CIs in parentheses; P values in brackets. P values represent between-group differences from baseline to 3 mo after adjustment for the baseline value.

[4]1 kJ = 0.239 kcal.

TABLE 5
Average scores for each group at baseline and 3 mo for the FAQ[1]

| | High-egg diet (n = 70)[2] | | Low-egg diet (n = 67)[2] | | Between-group comparison (high egg relative to low egg)[3] |
	Baseline	Δ 3 mo	Baseline	Δ 3 mo	Δ 3 mo
Question 1	5.5 ± 1.0	0.0 ± 1.0	5.4 ± 0.9	−0.2 ± 1.1	0.3 (0.0, 0.6) [0.04]
Question 2	5.5 ± 0.9	−0.1 ± 0.9	5.4 ± 0.9	−0.3 ± 0.9	0.2 (−0.0, 0.5) [0.07]
Question 3	5.2 ± 1.1	−0.2 ± 1.1	5.0 ± 1.0	−0.1 ± 1.2	0.1 (−0.2, 0.4) [0.6]
Question 4	3.1 ± 1.4	−0.2 ± 1.4	3.3 ± 1.6	0.3 ± 1.6	−0.6 (−1.0, −0.2) [≤0.01]
Question 5	5.5 ± 1.4	−0.2 ± 1.5	5.3 ± 1.4	−0.2 ± 1.4	0.1 (−0.3, 0.5) [0.6]
Question 6	5.8 ± 1.2	−0.2 ± 1.3	6.0 ± 1.1	−0.4 ± 1.3	0.1 (−0.3, 0.4) [0.7]
Question 7	4.0 ± 1.6	−0.1 ± 1.6	4.5 ± 1.5	−0.5 ± 1.4	0.2 (−0.2, 0.7) [0.3]
Question 8	4.3 ± 1.6	0.0 ± 1.8	4.5 ± 1.5	−0.2 ± 1.5	0.2 (−0.3, 0.6) [0.4]
Question 9	5.0 ± 1.4	0.2 ± 1.3	4.9 ± 1.3	0.2 ± 1.5	0.0 (−0.3, 0.4) [0.9]
Question 10	4.6 ± 1.4	0.6 ± 1.4	4.8 ± 1.3	0.1 ± 1.3	0.3 (−0.0, 0.7) [0.06]

[1]The FAQ was composed of the following 10 questions scored on a 7-point linear scale with 1 being dislike and 7 being total acceptance (the exception was for question 4 for which the reverse applied): Question 1: How well do you like the food that you have been eating in the past 2 wk? Question 2: How well do you like the taste of these foods? Question 3: How appealing or unappealing do you find the appearance of these foods? Question 4: How boring are these foods? Question 5: How easy or difficult has it been for you to prepare these foods? Question 6: How easy or difficult has it been for you to purchase these foods? Question 7: How easy or difficult has it been for you to maintain your current diet at restaurants? Question 8: How much effort does it take for you to stay on this diet? Question 9: How satisfied or dissatisfied do you feel after eating a meal on this diet? Question 10: Overall, how satisfied or dissatisfied are you with this diet? Positive Δ values represent an increase from baseline; negative Δ values represent a decrease from baseline. An ANCOVA was used to compare treatment groups. Analyses were adjusted for the baseline observation. An ANOVA with repeated measures was used for within-group comparisons. P values represent between-group differences from baseline to 3 mo after adjustment for the baseline value. FAQ, Food Acceptability Questionnaire.

[2]All values are means ± SDs.

[3]All values are means; 95% CIs in parentheses; P values in brackets.

(question 4: "How much food do you think you can eat?") than the low-egg group (**Table 6**).

Eating behavior, physical activity, and quality of life

There were no significant differences between groups in the 3 domains of eating behavior (as determined by using the Three-Factor Eating Questionnaire-R21) over the 3 mo. Similarly, there was no difference between groups in the total time spent partaking in physical activity per week (median difference for high-egg group compared with low-egg group: 0; IQR: −999 to 1449; $P = 0.92$). With respect to quality of life, there were no differences between groups over the 3-mo period (mean difference for the high-egg group compared with the low-egg group: −2.2; 95% CI: −5.3, 0.9; $P = 0.17$).

DISCUSSION

With the rising prevalence of T2D, there is an urgent need to provide clear messages for both its treatment and prevention. On the basis of published research to date, it is unclear whether eggs are a safe and suitable dietary protein source for people with T2D who are at high risk of cardiovascular complications.

There is little or no association between egg intake and CVDs in a range of healthy populations (24), and eggs may actually improve risk factors associated with CVD in this group (25). Therefore, the current prospective randomized controlled trial was designed to address the confounding factors of the published epidemiologic evidence to date, which suggests that a high-egg diet may be associated with detrimental outcomes in people with T2D (14–16). At the end of this 3-mo study, there were no significant differences in circulating concentrations of HDL

cholesterol, LDL cholesterol, total cholesterol, or triglycerides between subjects in the 2 groups (the high- or low-egg diet). Although both groups significantly reduced their saturated fat intakes, there was a within-group trend toward an improvement in HDL cholesterol only in the high-egg group. This finding was in keeping with several studies in nondiabetic populations that showed increases in HDL cholesterol and apolipoprotein A-I concentrations with increased egg intake, with minimal effects on LDL cholesterol, particularly in obese insulin-resistant subjects in whom dietary cholesterol absorption is reduced (10, 11, 26). A possible explanation for this finding is that the formation of intestinal apolipoprotein A-I, which is the major protein component of HDL cholesterol, was shown to be increased with cholesterol and fat feeding in both insulin-sensitive and, albeit to a lesser extent, insulin-resistant subjects (26). This is a possible area of future research to confirm whether a high-egg diet in individuals with T2D is beneficial in raising HDL cholesterol. Interventions that were associated with increases in HDL cholesterol, such as the use of fenofibrate, were shown to reduce cardiovascular events in people with T2D (27). In the Fenofibrate Intervention and Event Lowering in Diabetes study, in which fenofibrate or placebo was given to participants with T2D (27), a 0.05-mmol/L increase in HDL cholesterol was evident over 4 mo for subjects taking fenofibrate. This increase was sustained to a lesser extent throughout the duration of the study, which perhaps contributed to the positive finding in relation to cardiovascular events.

Thus far, there has been only one prospective randomized controlled trial that examined the effect of eggs in people with T2D (10). Although there was an improvement in blood lipid profiles, the result was possibly confounded by the trial being

TABLE 6
Average scores for each group at baseline and 3 mo for the VAS[1]

	High-egg diet (n = 68)		Low-egg diet (n = 66)		Between-group comparison (high egg relative to low egg)	
	Before breakfast, baseline score	After breakfast, baseline score	Before breakfast, baseline score	After breakfast, baseline score	Before breakfast, Δ 3 mo	After breakfast, Δ 3 mo
Question 1	5.7 ± 2.4[2]	1.6 ± 2.0	5.2 ± 2.6	1.0 ± 1.4	0.6 (−0.3, 1.5) [0.2][3]	−0.7 (−1.2, −0.1) [0.02]
Question 2	3.5 ± 2.4	7.7 ± 2.2	3.6 ± 2.8	7.4 ± 2.4	−0.4 (−1.2, 0.4) [0.4]	0.2 (−0.6, 1.0) [0.6]
Question 3	6.2 ± 2.6	1.7 ± 2.0	5.6 ± 2.8	1.6 ± 2.3	0.1 (−0.8, 1.0) [0.8]	−0.5 (−1.1, 0.2) [0.1]
Question 4	6.1 ± 2.0	1.7 ± 2.0	5.5 ± 2.2	1.8 ± 2.1	0.0 (−0.7, 0.7) [0.9]	−0.6 (−1.2, −0.0) [0.04]
Question 5	3.5 ± 2.6	3.3 ± 3.2	3.4 ± 2.8	2.8 ± 3.2	−0.3 (−1.1, 0.5) [0.5]	−0.2 (−1.0, 0.6) [0.6]
Question 6	6.2 ± 2.4	1.7 ± 2.2	5.3 ± 2.9	1.6 ± 2.3	0.4 (−0.5, 1.4) [0.3]	−0.5 (−1.2, 0.2) [0.2]
Question 7	5.6 ± 2.7	7.3 ± 2.5	5.5 ± 2.5	7.4 ± 2.5	−0.1 (−1.0, 0.7) [0.8]	0.1 (−0.7, 0.9) [0.8]
Question 8	2.6 ± 2.3	1.6 ± 2.2	2.2 ± 2.3	1.1 ± 1.6	−0.2 (−0.9, 0.5) [0.6]	−0.5 (−1.1, 0.2) [0.2]
Question 9	1.8 ± 2.2	1.7 ± 2.2	1.5 ± 2.1	1.2 ± 1.7	0.2 (−0.5, 0.9) [0.6]	−0.3 (−1.0, 0.4) [0.4]
Question 10	7.5 ± 2.0	7.9 ± 2.0	7.4 ± 2.1	7.6 ± 2.3	−0.6 (−1.3, 0.1) [0.1]	−0.3 (−1.0, 0.3) [0.3]

[1]The VAS was composed of the following 10 questions scored on a 10-point linear scale with 0 being not at all and 10 being very: Question 1: How hungry are you? (extreme left of scale = not at all hungry; extreme right of scale = very hungry); Question 2: How full do you feel? (extreme left = not at all full; extreme right = very full); Question 3: How strong is your desire to eat? (extreme left = not at all strong; extreme right = very strong); Question 4: How much food do you think you could eat? (extreme left = none at all; extreme right = a large amount); Question 5: How strong is your desire for sweet food? (extreme left = not at all strong; extreme right = very strong); Question 6: How strong is your desire for savory food? (extreme left = not at all strong; extreme right = very strong); Question 7. How contented are you? (extreme left = not at all contented; extreme right = very contented); Question 8. How irritable are you? (extreme left = not at all irritable; extreme right = very irritable); Question 9. How depressed are you? (extreme left = not at all depressed; extreme right = very depressed); Question 10. How mentally alert are you? (extreme left = not at all alert; extreme right = very alert). Positive Δ values represent an increase from baseline; negative Δ values represent a decrease from baseline. An ANCOVA was used to compare treatment groups. Analyses were adjusted for baseline observation. An ANOVA with repeated measures was used for within-group comparisons. *P* values represent between-group differences from baseline to 3 mo after adjustment for the baseline value. VAS, visual analog scale.

[2]Mean ± SD (all such values).

[3]Mean; 95% CI in parentheses; *P* value in brackets (all such values).

a weight-loss intervention. Weight loss itself has effects on blood lipid profiles that might outweigh any effects of eggs on such variables. In the current study, participants were instructed to maintain their weight by replacing energy-dense, nutrient-poor foods that were high in saturated fats with energy-dense, nutrient-rich alternatives that were high in monounsaturated and polyunsaturated fats. Both groups successfully maintained their weight to ≤1 kg of their initial body weight. Despite a within-group significant weight loss or reduction in body fat percentage in both groups, the amount of weight loss was small and not clinically meaningful such that our observations were unlikely to be confounded by concurrent weight changes.

Despite the high protein content of eggs, the protein intake of both groups was matched for the period of the 3-mo study. A small but significant difference in carbohydrate and total fat intake was noted between groups at 3 mo, which may have been due to the higher fat and lower carbohydrate content of eggs compared with other protein-rich sources prescribed at breakfast for the low-egg group (e.g., skim/low fat dairy, baked beans, and lean meat). Intakes of saturated fat decreased significantly for both groups and intakes of polyunsaturated and monounsaturated fats increased significantly for both groups. However, the American Diabetes Association guidelines suggest saturated fat should be <10% of the total energy in the diet for the general population and people with T2D (3), and in our study, participants' intakes were still above this target. Participants in the high-egg group also consumed approximately double the current American Diabetes Association guidelines for cholesterol intake (3). Despite being above the American Diabetes Association guidelines for saturated fat and cholesterol intakes, these results show that a high-egg diet can be incorporated into the dietary

management of people with T2D in conjunction with an increase in monounsaturated and polyunsaturated fats without adversely affecting blood lipid profiles. However, it is not known how high egg consumption would influence the metabolic profile of people consuming a low-MUFA and -PUFA diet, compared with that of control subjects consuming a low-MUFA and -PUFA diet because this was not a part of our study design.

Although both diets were well adopted over the 3-mo study, the high-egg diet showed a significantly greater food-acceptability score compared with that with the low-egg diet. This finding suggests a high-egg diet does not result in boredom and may be more likely to improve compliance when incorporated into nutritional management for people with T2D. Appetite was shown to be reduced after breakfast in the group with high-egg intake by using the visual analog scale, which is a subjective analysis that has proven to be reproducible, sensitive to exposures of food components, and predictive of food intake (23, 28). Thus, the high-egg diet resulted in less hunger and greater satiety after breakfast than did the low-egg diet, implying that eggs may have benefits extending beyond their nutritional constituents.

Strengths of this study included the adequate sample size and low dropout rate of 9%. Despite some small differences in nutritional contents, both groups were well matched for protein intake. Both groups maintained their starting body weight ≤1 kg so that weight loss was not a confounder. The inability to blind intervention groups when obtaining visual analog scale ratings of appetite was a limitation as was the short duration of the study. Furthermore, a significant difference for the primary outcome may have been shown if the study had a crossover design or used a larger sample size because the study was powered for a 10% difference in HDL cholesterol, which was not shown.

In conclusion, individuals with prediabetes or T2D who consumed a high-egg diet for 3 mo while maintaining weight and reducing dietary saturated fat intake (in conjunction with concomitant increases in MUFA and PUFA consumption) had no adverse changes in circulating lipid profiles compared with in participants who consumed a low-egg diet. Therefore, a diet including more eggs than generally recommended may be used safely as part of the nutritional management of this group.

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