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Peanuts or an Isocaloric Lower Fat, Higher Carbohydrate Nighttime Snack Have Similar Effects on Fasting Glucose in Adults with Elevated Fasting Glucose Concentrations: a 6-Week Randomized Crossover Trial

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ABSTRACT

Background: The glycemic effects of peanuts are not well studied and no trials have been conducted in adults with elevated fasting plasma glucose (FPG). Furthermore, intake of peanuts as a nighttime snack, an eating occasion affecting FPG, has not been examined.

Objectives: The aim was to determine the effect of consuming 28 g/d of peanuts as a nighttime snack for 6 wk on glycemic control and cardiovascular disease risk factors, compared with an isocaloric lower fat, higher carbohydrate (LFHC) snack (whole grain crackers and low-fat cheese), in adults with elevated FPG.

Methods: In a randomized crossover trial, 50 adults (FPG 100 ± 8 mg/dL) consumed dry roasted, unsalted peanuts [164 kcal; 11% energy (E) carbohydrate, 17% E protein, and 73% E fat] or a LFHC snack (164 kcal; 54% E carbohydrate, 17% E protein, and 33% E fat) in the evening (after dinner and before bedtime) for 6 wk with a 4-wk washout period. Primary (FPG) and secondary end points [Healthy Eating Index-2015 (HEI-2015), weight, insulin, fructosamine, lipids/lipoproteins, central and peripheral blood pressure, and pulse wave velocity] were evaluated at the beginning and end of each condition. Linear mixed models were used for data analysis.

Results: FPG was not different between the peanut and LFHC conditions (end point mean difference: −0.6 mg/dL; 95% CI: −2.7, 1.6; $P = 0.67$). There were no between-condition effects for secondary cardiometabolic endpoints. The HEI-2015 score was not different between the conditions (3.6 points; $P = 0.19$), although the seafood/plant protein (2.0 points; $P < 0.01$) and added sugar (0.8 points; $P = 0.04$) components were improved following peanut intake. The whole grain component was lower with peanuts compared with LFHC (−2.6 points; $P < 0.01$).

Conclusions: In adults with elevated FPG, peanuts as a nighttime snack (28 g/d) did not affect FPG compared with an isocaloric LFHC snack after 6 wk. This trial was registered at clinicaltrials.gov as NCT03654651. *J Nutr* 2022;152:153–162.

Keywords: prediabetes, elevated fasting glucose, nutritional intervention, peanuts, randomized controlled trial

Introduction

In 2018, ~88 million (34.5%) US adults had prediabetes (1). For adults with prediabetes at age 45 y, the lifetime risk of progression to type 2 diabetes mellitus (T2DM) is 74% (95% CI: 67.6, 80.5) (2). Individuals with prediabetes have a higher risk (HR: 1.18; 95% CI: 1.08, 1.26) of developing cardiovascular disease (CVD) (3) and present with more CVD risk factors (hypertension, inflammation, dyslipidemia, and obesity) compared with individuals with normoglycemia (4). Intensive lifestyle therapies (i.e., weight loss, medical nutrition therapy, and physical activity) are recommended for all adults with prediabetes and T2DM, but compliance is often suboptimal. Simple strategies to improve fasting blood glucose and delay or prevent T2DM onset and CVD in individuals with prediabetes are needed.

Observational evidence suggests that habitual peanut and tree nut consumption (4 servings of 28.4 g/wk) is associated with a significantly lower incidence of T2DM (RR: 0.87; 95% CI: 0.81, 0.94), fatal ischemic heart disease (RR: 0.76; 95% CI: 0.69, 0.84), and nonfatal ischemic heart disease (RR: 0.78; 95% CI: 0.67, 0.92) (5). These findings are supported by randomized controlled trials showing improvements in glycemic outcomes with peanut and tree nut intake in healthy individuals and those with prediabetes or T2DM (6–8). Few studies have examined

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the effect of peanuts on glycemic outcomes (9–13), and no study has been conducted in individuals with prediabetes. In addition, limited research has examined whether nighttime nut intake affects glucose control.

The dawn phenomenon affects ∼50% of individuals with type 1 and type 2 diabetes and is the primary cause of elevated fasting plasma glucose (FPG), although the etiology is not well understood (14). The dawn phenomenon is characterized by an increase in blood glucose concentrations between 02:00 and 08:00, and is caused by dysregulated compensatory insulin secretion in response to hepatic glucose production at "dawn." Dietary strategies (i.e., reducing carbohydrate-to-protein ratio of nighttime snacks) are suggested to attenuate the dawn phenomenon, but limited empirical evidence is available. In 2 clinical trials, consumption of evening snacks containing primarily complex carbohydrates or a higher protein/fat to carbohydrate ratio for 3 nights improved fasting glucose in adults with T2DM (15, 16). Investigation of how healthy evening snacks affect fasting glucose in individuals with prediabetes is warranted.

We conducted a randomized controlled trial in individuals with elevated FPG to evaluate the effect of consuming 28 g/d of peanuts, as a nighttime snack, compared with an isocaloric lower fat, higher carbohydrate snack (LFHC), on FPG and risk factors for CVD. We hypothesized that consuming peanuts as an evening snack, after dinner and before bedtime, would lower FPG compared with an isocaloric LFHC in adults with elevated FPG.

Methods

Trial design

A single-blind, 2-period, randomized crossover controlled clinical trial was conducted at the Pennsylvania State University between October 2018 and January 2020. Eligible individuals were randomly assigned immediately prior to baseline testing in a 1-to-1 ratio to 2 randomization sequences generated using a computer-generated scheme (randomization.com) by an investigator who was not involved in data collection. The randomization code was held by the metabolic kitchen manager; the study coordinator who conducted screening, enrollment, and data collection was unaware of participants' randomization until data collection was completed. Participants were not blinded to the randomization because of the nature of the study. Participants consumed the following evening snacks for 6 wk: *1*) 28 g/d of dry-roasted unsalted peanuts; *2*) an isocaloric LFHC snack. Participants had a minimum 4-wk break between the diet periods (median break: 28 d; range: 28–41 d). Outcomes were measured at baseline and the end of each diet period. The Institutional Review Board of the Pennsylvania State University (University Park, PA) approved the protocol. Written informed consent was obtained from all participants before screening for this study. This trial is registered at clinicaltrials.gov (identifier NCT03654651).

Participants

Participants were recruited between November 2018 and September 2019 from the State College, Pennsylvania area. Recruitment was conducted using clinicaltrials.gov and StudyFinder (studyfinder.com) and our research group's webpage. Advertisements were posted on university and local businesses' bulletin boards, listservs, and in local newspapers and circulars. Men and women aged 18–75 y, who had an elevated FPG (≥100 mg/dL and ≤125 mg/dL) measured at screening, were nonsmokers, and had a BMI ≥20 and ≤40 kg/m^2 were eligible. Exclusion criteria were diagnosed diabetes or FPG >126 mg/dL; systolic blood pressure (BP) >160 mmHg, diastolic BP >100 mmHg; taking glucose, lipid, or BP-lowering medications; or taking antibiotics ≤6 wk prior to enrollment. Individuals with a history of CVD, stroke, liver, kidney, autoimmune disease, or inflammatory conditions were excluded. Participants taking supplements (psyllium, fish oil, soy lecithin, and phytoestrogens) and botanicals were excluded unless willing to abstain during the course of the study. Finally, pregnant or lactating women, individuals consuming >14 alcoholic beverages/wk, or those who had lost ≥10% of their body weight in the previous 6 mo were excluded.

A telephone screening was conducted to assess medical history and lifestyle to determine eligibility for a clinical screening visit. Eligible participants were scheduled for an in-person screening at the Pennsylvania State University Clinical Research Center. Each participant fasted for 12 h (no food or drink except for water) and avoided alcohol and over-the-counter medications for 48 h. During this visit, weight and height (without shoes) were measured. BP was measured (validated automated sphygmomanometer following a 5-min rest) 3 times (17). A mean of the last 2 of the 3 BP measurements was used to determine eligibility. Premenopausal women provided a urine sample for a pregnancy test. Finally, nurses collected a fasting blood sample for a complete blood count, blood chemistry, and plasma glucose assayed by a commercial laboratory (Quest Diagnostics).

Intervention

The experimental snack was 28 g/d (1 oz) of dry-roasted, unsalted, skinless peanuts [164 kcal (fat: 14 g and 73% E; carbohydrates: 5 g and 11% E; protein: 7 g and 17% E; saturated fat: 2.2 g; monounsaturated fat: 7.4 g; polyunsaturated fat: 2.8 g; fiber: 2.4 g; sodium: 5 mg)] and the comparison LFHC snack was 6 low-sodium whole grain crackers (28 g) and 1 slice (19 g) of low-fat prepackaged American cheese [165 kcal (fat: 6 g and 33% E; carbohydrates: 22 g and 54% E; protein: 7 g and 17% E; saturated fat: 2.0 g; monounsaturated fat: 1 g; polyunsaturated fat: 2 g; fiber: 3.0 g; sodium: 270 mg)]. This comparator was selected because it represents a snack choice that can be recommended for blood glucose control (low glycemic index) and therefore would not be expected to worsen blood glucose control in this cohort at risk of T2D. In addition, the LFHC snack had a similar saturated fat, fiber, and protein content to the peanuts. Participants were asked to consume the study food after dinner, but before bedtime, and avoid any other food or drink containing calories in the evening. A specific time for consuming the evening snack was not given because of the interindividual as well as daily variability in dinner time and bedtime. Nonprescriptive timing for the evening snack mimics real-world application and increases generalizability. Additionally, participants were asked to avoid any other peanuts or tree nuts, including nut butters, throughout the entirety of the study. Study food was provided every 14 d by the metabolic kitchen manager.

Adherence was assessed biweekly by the metabolic kitchen manger based on daily adherence checklists completed by the participants that included questions about consumption of the study foods, if the study foods were consumed after dinner, if other calorie-containing foods/beverages were consumed after the study food, if other peanuts/tree nuts were consumed, if any changes in health status or usual exercise occurred, and if any nonhabitual medications were taken. Adherence was calculated by dividing the total number of days the participants consumed the study food by the total number of days in the

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Abbreviations used: ASA24, automated self-administered 24-hour dietary assessment tool; BP, blood pressure; CVD, cardiovascular disease; E, energy; FPG, fasting plasma glucose; HEI, Healthy Eating Index; LFHC, isocaloric lower fat, higher carbohydrate snack; NCI, National Cancer Institute; PWV, pulse wave velocity; T2DM, type 2 diabetes mellitus.

diet period. Additionally, the percentage of study days the participant consumed the study food as directed (i.e., as an evening snack, without consuming any other food or drinks in the evening) was calculated.

Outcomes

Testing was conducted on 2 separate consecutive days at baseline and the end of each diet period for a total of 8 visits throughout the study. For 12 h prior to the testing visits participants fasted and avoided strenuous physical activity, and for 48 h prior participants were asked to refrain from drinking alcohol or taking over-the-counter medications. Each participant confirmed they had followed these protocols prior to beginning data collection. On both test days weight was measured using a calibrated electronic scale while participants wore light clothing and no shoes, and a fasting blood draw was taken for analysis of glucose, lipids and lipoproteins, insulin, and fructosamine. On 1 of the test days, vascular testing was performed.

Blood collection and assay methods.

Blood samples were drawn into serum separator and sodium fluoride/potassium oxalate tubes. The sodium fluoride/potassium oxalate tube was centrifuged immediately at 1590 × *g* (± 90) at room temperature for 15 min. Blood drawn into the serum separator tube was allowed to clot for 30 min and then centrifuged at 1590 × *g* (± 90) at room temperature for 15 min. Plasma samples were used to measure glucose. Serum samples were used to measure total cholesterol, LDL cholesterol (direct measurement), HDL cholesterol, triglycerides, and lipoprotein particle size. All samples were frozen at −80°C upon collection and analyzed in 1 batch at the end of the study. Plasma was analyzed for glucose at the Pennsylvania State University Biomarker Core Lab (University Park, PA) using a Cobas c311 chemistry analyzer (Roche Diagnostics). Serum samples were assayed for lipids/lipoproteins, insulin, and fructosamine at the Pennsylvania State University Biomarker Core Lab (University Park, PA) using a Cobas c311 chemistry analyzer (Roche Diagnostics) according to the manufacturer's instructions. Lipoprotein subfractions were analyzed using ion mobility, which enables direct particle quantification as a function of particle diameter (18) following a procedure to remove other plasma proteins (19). Particle concentrations (nanomoles per liter) were determined for subfractions defined by the following size intervals (nanometers): VLDL: large (42.40–54.70), medium (33.50–42.39), small (29.60–33.49); IDL: large (25.00–29.59), small (23.33–24.99); LDL: large (22.0–23.32), medium (21.41–21.99), small (20.82–21.40), very small (18.0–20.81); HDL: large (10.50–14.50) and small (7.65–10.49). Interassay variation was reduced by inclusion of 2 in-house controls in each preparatory process and triplicate analysis. The interassay CV was <15% for each subfraction measurement. apoB was analyzed by immunoturbidimetric assay (AMS Liasys 330 analyzer; Kamiya Biomedical Company) and the interassay CV was <15%.

Vascular testing methods.

Following a 5-min seated rest, a SphygmoCor ECEL (AtCor Medical) was used to assess peripheral and central BP in the seated position. A cuff was placed on the left arm for measurement of peripheral blood pressure and radial artery waveforms. A validated generalized transfer function was used to calculate central BP from peripheral BP and the radial artery pressure waveform. Augmentation index was adjusted to a heart rate of 75 beats per minute. Three measurements were taken and the last 2 results were averaged and used for the analyses.

Immediately following the BP assessment, carotid-femoral pulse wave velocity (PWV) was measured using the SphygmoCor ECEL while participants were in the supine position. A tonometer was placed on the carotid artery and a BP cuff was placed on the femoral artery. A 10-s recording of the carotid-femoral waveform was taken and the PWV was calculated by dividing the linear distance between the carotid and femoral sites by the transit time using the SphygmoCor system. On each test day, 3 PWV measurements were obtained and the average of the last 2 was used for analysis.

Dietary assessment methods.

Dietary intake was assessed by nonrandom, participant-completed 24-h recalls prior to baseline and in the last week of each diet period; each participant completed a total of four 24-h recalls throughout the study. The Automated Self-Administered 24-Hour Dietary Assessment Tool (ASA24) [National Cancer Institute (NCI)] was used and administered as recommended by the NCI Dietary Assessment Primer (20). Briefly, participants were e-mailed a unique username and password prior to baseline and end point data collection visits and asked to complete the 24-h recall prior to their study visit. Recall data were reviewed and cleaned to ensure the study snacks were reported correctly and consistently. Diet quality was assessed using the Healthy Eating Index (HEI)-2015, which consists of 13 components including 9 "adequacy" components (whole grains, total fruits, dairy, etc.) and 4 "moderation" components (saturated fats, sodium, refined grains, and sugars). A higher adequacy score reflects higher intake whereas a higher moderation score reflects a lower intake. The HEI-2015 was calculated using the Statistical Analysis System (SAS) code created by the NCI (21). Dietary recalls where energy intake was <600 or >4400 kcal/d for women and <650 or >5700 kcal/d for men were excluded from the analyses based on the NCI guidelines for reviewing and cleaning ASA24 data (22).

Statistical analyses

Sample size calculations indicated that completion of 45 participants would provide 80% power ($P < 0.05$) to detect a minimum 10 mg/dL difference in FPG between the conditions (SD: 23.4 mg/dL) based on previous studies (13, 15, 23). FPG is the primary outcome. All other outcomes are secondary.

All statistical analyses were performed using SAS (version 9.4; SAS Institute Inc). All data collected from randomly assigned participants were included in data analyses consistent with intent-to-treat principles. Univariate analysis (PROC UNIVARIATE) was used to assess normality of the residuals for each variable based on the distribution and normal probability plots (Q-Q plots). In the instance of skewed residuals, the variable was logarithmically transformed. Weight, glucose, lipids and lipoproteins, and insulin values taken on the 2 test days at each time point were averaged for analysis. Change from baseline for each diet period was calculated by subtracting the end point value from the baseline value. Data are presented as least-squares means ± SEMs unless otherwise stated.

The effect of each condition on outcome variables was examined using the mixed-models procedure (PROC MIXED) at a predetermined α level of 0.05. Subject nested within condition was modeled to account for the repeated-measures crossover design, and baseline value was included as a covariate. Sex and randomization sequence were included as fixed effects to assess sex and carryover effects. There were neither sex nor carryover effects based on nonsignificant condition × sex and condition × sequence interactions for each outcome variable; therefore, sex and sequence were removed from the final model. The primary analyses assessed between-diet differences in end point means for each outcome variable. Additionally, change from baseline was evaluated using the mixed-models procedure for each condition. The covariance structure for the models was based on optimizing fit statistics (lowest bayesian information criterion) and varied depending upon which analysis was being conducted. Exploratory analyses were conducted to assess end point–mean differences and change from baseline for each condition by eating occasion (breakfast, brunch, lunch, dinner/supper, snack, drinks, and supplements).

Results

Fifty-one adults were randomly assigned out of the 267 individuals who completed a telephone screening; further details are given in the CONSORT flow diagram (**Figure 1**). Of the 51 individuals randomly assigned, 50 (female $n = 25$) aged 42 ± 15 y with a BMI of 28.3 ± 5.6 kg/m^2 and FPG of 100 ± 8 mg/dL completed the study. Participant characteristics

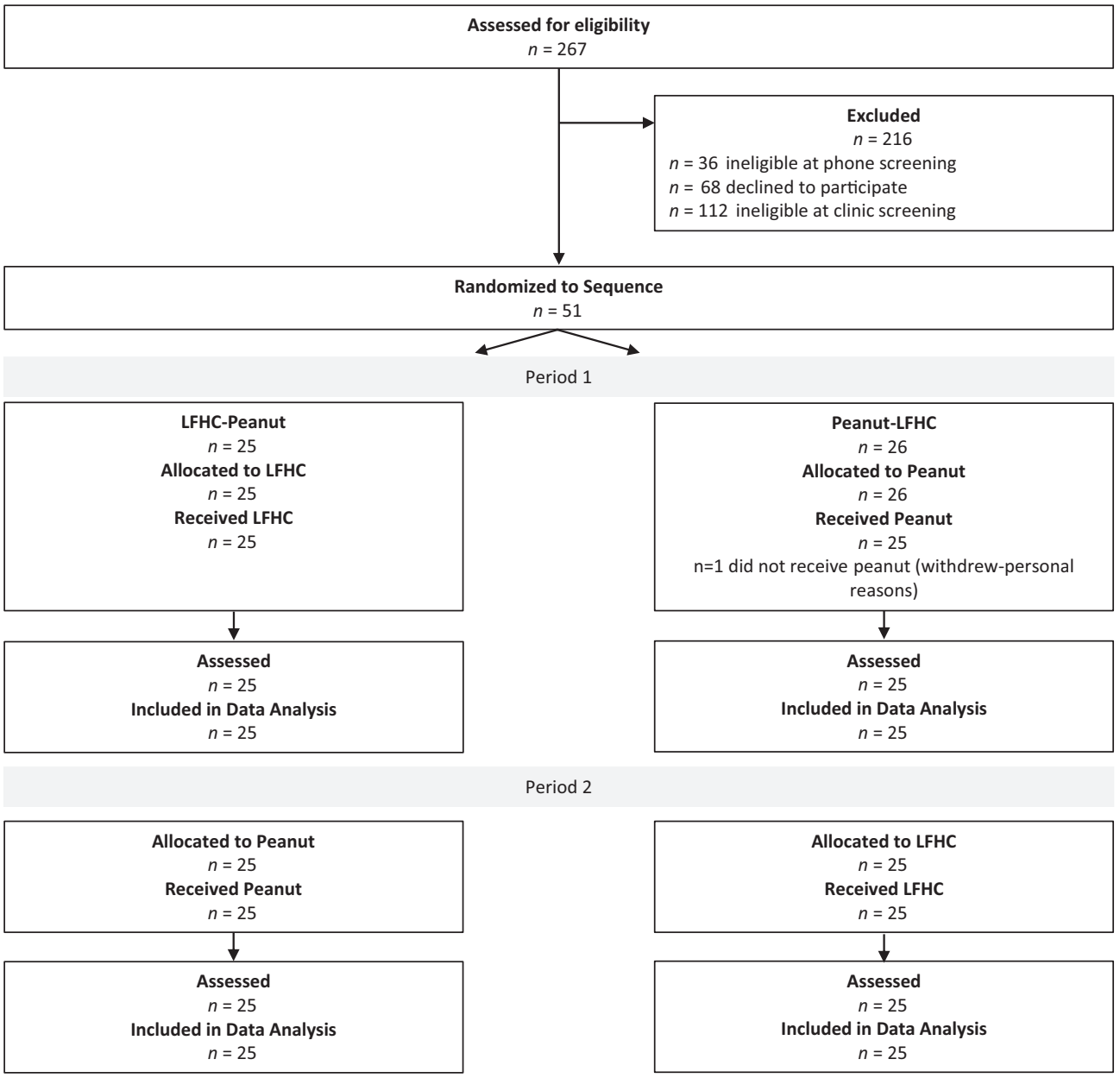

FIGURE 1 CONSORT flow diagram

were comparable between the randomization sequences at baseline (Table 1). On average, participants reported eating their study food on 87% of study days. Additionally, on 88% of study days, participants reported consuming the study food as directed (i.e., after dinner and before bedtime), although slightly greater adherence was observed during the LFHC condition compared with the peanut condition (89% compared with 87%). One subject withdrew without consuming any study food due to personal reasons. The 50 individuals who provided end point data were included in all analyses, unless otherwise stated.

TABLE 1 Baseline characteristics of study participants overall and by randomization sequence ($n = 50$)[1]

Characteristic	Peanut-LFHC	LFHC-Peanut	Total
n (% female)	24 (50%)	26 (46%)	50
Age, y	40 ± 15	43 ± 15	42 ± 15
Weight, kg	85.0 ± 21.1	82.7 ± 16.5	83.8 ± 18.7
Height, m	1.72 ± 0.1	1.71 ± 0.1	1.72 ± 0.1
BMI, kg/m^2	28.5 ± 6.0	28.2 ± 5.2	28.3 ± 5.6
Glucose, mg/dL	99 ± 8	101 ± 8	100 ± 8

[1]Values are mean ± SD unless otherwise stated. LFHC, lower fat, higher carbohydrate snack.

No significant between-condition effects were observed for FPG (peanut compared with LFHC mean difference: −0.6 mg/dL; 95% CI: −2.7, 1.6; $P = 0.67$), insulin (0.45 μIU/mL; 95% CI: −1.2, 2.1; $P = 0.60$), or fructosamine (0.7 μmol/L; 95% CI: −4.8, 3.4; $P = 0.74$) (Table 2). No differences were observed between the conditions for total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, weight, peripheral or central BP, or measures of arterial stiffness. Lipoprotein particle concentration analysis showed no differences between the conditions for VLDL, IDL, and HDL subparticles. LDL peak diameter was not different between the conditions, and no differences in the concentration of all LDL subparticles

TABLE 2 Between-condition differences and change from baseline for glycemic, lipid, lipoprotein, vascular, and anthropometric outcomes in adults with elevated FPG ($n = 50$)[1]

	Peanut			LFHC				
Outcome	Baseline[2]	End point	Within-condition difference[3]	Baseline[2]	End point	Within-condition difference[3]	Between-condition effect[3]	Between-condition *P* value
Glucose, mg/dL (primary outcome)	100 ± 1.0	99.2 ± 0.9	−0.9 (−2.7, 0.9)	100 ± 1.2	100 ± 0.9	−0.4 (−2.2, 1.4)	−0.6 (−2.7, 1.6)	0.67
Insulin, μIU/mL	11.35 ± 1.14	11.34 ± 0.59	−0.2 (−1.5, 1.0)	11.93 ± 1.34	10.89 ± 0.59	−0.8 (−2.1, 0.5)	0.45 (−1.2, 2.1)	0.60
Fructosamine, μIU/mL	228 ± 2.2	224 ± 1.7	−4.0 (−5.1, 5.4)	229 ± 2.6	225 ± 1.7	−3.8 (−5.1, 5.4)	0.7 (−4.8, 3.4)	0.74
TChol, mg/dL	191 ± 5	192 ± 2	0.1 (−4.9, 5.1)	194 ± 5	190 ± 2	−2.7 (−7.8, 2.3)	2.2 (−3.8, 8.2)	0.46
LDL-C, mg/dL	122 ± 4.1	126 ± 2.7	2.4 (−2.1, 6.9)	125 ± 4.2	122 ± 2.3	−2.3 (−6.8, 2.2)	4.3 (−1.4, 9.8)	0.13
HDL-C, mg/dL	53.2 ± 2.1	54.1 ± 0.6	0.6 (−0.6, 1.8)	53.8 ± 2.0	54.1 ± 0.8	0.6 (−1.6, 2.3)	0.0 (−2.3, 2.3)	0.99
TG, mg/dL	122 ± 11	103 ± 3	−17.0 (−29.1, −4.8)*	115 ± 9	111 ± 4	−5.7 (−17.1, 5.7)	−7.7 (−18.3, 2.8)	0.15
Weight, kg	83.9 ± 2.6	84.1 ± 0.2	0.2 (−0.1, 1.5)	83.8 ± 2.6	83.9 ± 0.2	0.0 (−0.5, 0.5)	0.2 (−0.3, 0.7)	0.51
bSBP, mmHg	125 ± 1.9	125 ± 1.3	0.4 (−2.5, 3.3)	125 ± 1.8	125 ± 1.3	−0.3 (−3.1, 2.6)	0.7 (−3.1, 4.6)	0.70
bDBP, mmHg	79 ± 1.3	80 ± 0.9	0.5 (−1.5, 2.5)	80 ± 1.3	80 ± 0.9	−0.0 (−2.0, 2.0)	0.4 (−2.1, 2.9)	0.73
cSBP, mmHg	114 ± 1.6	114 ± 1.1	0.6 (−1.9, 3.1)	114 ± 1.8	113 ± 1.1	−0.4 (−2.9, 2.1)	1.0 (−2.4, 4.4)	0.54
cDBP, mmHg	80 ± 1.3	81 ± 0.9	0.5 (−1.5, 2.6)	81 ± 1.4	80 ± 0.9	−0.4 (−2.4, 1.6)	0.8 (−1.7, 3.3)	0.54
AP, mmHg	6 ± 3.0	7 ± 0.5	0.3 (−0.8, 1.4)	7 ± 0.8	7 ± 0.5	−0.0 (−1.1, 1.1)	0.3 (−1.3, 1.9)	0.72
AIX,[4] %	16 ± 3.0	16 ± 1.6	−0.4 (−3.7, 2.9)	18 ± 2.6	17 ± 1.6	0.2 (−4.0, 4.3)	−1.4 (−5.9, 3.3)	0.55
PTT, ms	64 ± 1.3	62 ± 0.8	−1.2 (−2.7, 0.3)	63 ± 1.3	63 ± 0.8	−0.2 (−1.7, 1.3)	−0.9 (−3.0, 1.1)	0.36
PWV, m/s	6.8 ± 0.2	7.2 ± 0.1	0.3 (0.1, 0.6)*	7.0 ± 0.2	7.0 ± 0.1	0.1 (−0.1, 0.3)	0.2 (−0.1, 0.5)	0.19

[1]Data are presented as least squares means and SE unless otherwise stated. *Indicates within-condition significant difference ($P < 0.05$). Statistical analyses were performed with SAS version 9.4 (SAS Institute). The MIXED procedure was used to determine the effect of the conditions on each outcome measure, the between-condition *P* values represent the main effect of condition. The MIXED procedure was also used to assess change from baseline. AIX, augmentation index; AP, augmentation pressure; bDBP, brachial diastolic blood pressure; bSBP, brachial systolic blood pressure; cDBP, central diastolic blood pressure; cSBP, central systolic blood pressure; HDL-C, high-density-lipoprotein cholesterol; LDL-C, low-density-lipoprotein cholesterol; LFHC, lower fat, higher carbohydrate snack; PTT, pulse transit time; PWV, pulse wave velocity; TChol, total cholesterol; TG, triglycerides.
[2]Values are mean and SE.
[3]Values are least squares mean effect estimate and 95% CI.
[4]Adjusted to a heart rate of 75 bpm.

except LDL IIa were observed. LDL IIa particle size was higher following the peanut condition (peanut compared with LFHC mean difference: 16.1 nmol/L; 95% CI: 0.5, 31.7; $P = 0.04$). apoB was higher following the peanut condition compared with the LFHC condition (mean difference: 5.6 mg/dL; 95% CI: 1.4, 9.7; $P = 0.01$) (**Supplemental Table 1**).

Total energy intake (kilocalories) was similar for both conditions (peanut compared with LFHC mean difference: −43 kcal; 95% CI: −251, 236; $P = 0.71$). The percentage of energy from MUFAs and PUFAs was higher following peanut intake compared with the LFHC snack (1.7%; 95% CI: 0.0, 3.4; $P = 0.04$ and 1.9%; 95% CI: 0.0, 3.4; $P = 0.01$); no other significant differences were observed for nutrient intakes (**Table 3**). Intake of total protein foods (includes nuts) and oil-containing foods (includes nuts) was significantly higher with peanut intake compared with the LFHC snack (mean difference: 1.9 oz-eq; 95% CI: 0.1, 3.7; $P = 0.04$ and 8.3 g; 95% CI: 1.9, 14.6; $P = 0.01$). Whole grain consumption was significantly lower for the peanut condition compared with LFHC (−1.2 oz-eq; 95% CI: −1.6, −0.8; $P < 0.01$). No differences in intake of vegetables, total grains or refined grains, fruit, or dairy were observed between the conditions.

The HEI-2015 score was not significantly different between the conditions (mean difference: 3.6 points; 95% CI: −1.9, 9.0; $P = 0.19$). Compared with the LFHC snack, the seafood/plant protein (2.0 points; 95% CI: 1.0, 2.9; $P < 0.01$) and added sugar (0.8 points; 95% CI: 0.0, 1.5; $P = 0.04$) components were higher following the peanut condition. The whole grain component score was higher (mean difference: 2.6 points; 95% CI: 1.4, 3.8; $P < 0.01$) following the LFHC snack compared with peanuts. No other significant between-condition differences were observed for HEI components (**Table 4**).

The total calories from snacks were not significantly different between conditions (mean difference: −37 kcal; 95% CI: −165, 92; $P = 0.41$). Additionally, caloric intake at all other eating occasions (i.e., breakfast, lunch, dinner, supper, and drinks) was not significantly different between conditions. Following both conditions, no change in energy, nutrient, or food group intake for snacking occasions was observed (**Supplemental Tables 2–6**).

Discussion

The present study demonstrated that consuming 28 g/d of dry roasted, unsalted peanuts as an evening snack for 6 wk did not change FPG compared with an isocaloric lower fat, higher carbohydrate snack (whole grain crackers and low-fat cheese) in participants with elevated FPG. In addition, neither peanuts nor the LFHC snack significantly improved insulin, fructosamine, lipids, lipoproteins, vascular measures, or weight. However, LDL IIa and apoB concentration were higher following the peanut condition compared with the LFHC condition. Consumption of peanuts did not change overall diet quality compared with the LFHC snack, but did increase intake of protein from the seafood and plant sources category. Furthermore, the percentages of energy intake from PUFAs and MUFAs, oil-containing foods, and total protein foods were greater with peanut intake. Collectively, in individuals with elevated FPG, consumption of 28 g/d of peanuts as a nighttime snack, compared with an isocaloric LFHC nighttime snack, improved several components of diet quality without changing FPG, fructosamine, or CVD risk factors after 6 wk.

To our knowledge, this is the first clinical trial to examine evening snacking in adults with elevated FPG without T2DM. Dyer-Parziale (15) demonstrated significantly lower morning fasting glucose (114.2 mg/dL compared with 158.5 mg/dL; $P < 0.0001$]) after nighttime consumption of an Extend bar [30 g carbohydrate (5 g uncooked cornstarch), 3 g protein, and 3 g fat] compared with an isocaloric, macronutrient-matched snack without cornstarch in patients with T2DM after 3 d. Similarly, Abbie et al. (16) compared the effects of nighttime snacks high in protein and fat (egg) and carbohydrate (yogurt), as well as no snack on morning fasting glucose and glucose measured by continuous glucose monitor (CGM) in patients with T2DM. Morning fasting glucose and average CGM assessed glucose were significantly lower in the high-protein group (129.6 and 136.8 mg/dL) compared with the yogurt group (136.8 and 147.6 mg/dL). Our study compared similar nighttime snacks, but we did not observe comparable effects on FPG. The discordance could be because our study population did not have T2DM. In addition, our study had a longer study duration, which could have resulted in short-term acute effects similar to those observed in the aforementioned studies being missed.

Nuts (peanuts and tree nuts) have established short- and longer-term glucoregulatory properties. Several clinical trials show that peanuts improve postprandial glucose response. Liu et al. (9) conducted a clinical trial in men with overweight and obesity and demonstrated 4-h postprandial glucose was lower following a high-saturated fat beverage with 85 g peanuts compared with an isocaloric, macronutrient-matched, high–saturated-fat beverage without peanuts. Furthermore, in a study where 42.5 g peanuts or peanut butter was added to breakfast, improvements in postprandial (240–490 min) glucose were observed in women with obesity compared with a high-carbohydrate control breakfast (10). Peanuts contain 49.3 g/100 g fat (24.6 g is MUFAs) and likely improve postprandial glucose response by delaying gastric emptying, which delays carbohydrate absorption following a meal. Therefore, it is plausible that intake of meals containing peanuts improves postprandial glucose homeostasis without affecting fasting glucose. However, the current study was not designed to assess short-term glycemic response and is not directly comparable to these postprandial trials.

A recent systematic review and meta-analysis of 40 randomized controlled trials with a median duration of 12 wk, showed that nut intake (tree and peanuts; median dose 52 g/d) improved fasting insulin (weighted mean difference: −0.40 μIU/mL; 95% CI: −0.73, −0.07) and HOMA-IR (weighted mean difference:−0.23; 95% CI: −0.40, −0.06); fasting glucose was unaffected (weighted mean difference: −0.52 mg/dL; 95% CI: −1.43, 0.38) (7). However, heterogeneity existed in the interventions tested, particularly how the study foods were consumed, and only 4 studies examined peanut intake, which limits the direct comparability to our study. A recent 12-wk parallel arm, randomized controlled trial conducted in China evaluated the effect of peanuts [56 g/d (28 g 1 h before lunch and 28 g 1 h before dinner)] compared with an isocaloric white rice bar in participants with metabolic syndrome or at risk of metabolic syndrome (11). The peanut condition nonsignificantly reduced fasting glucose from 100 mg/dL (95% CI: 97.7, 102.1) to 94 mg/dL (95% CI: 91.8, 96.3) with similar reductions observed for the white rice bar. It is possible that the dose tested (i.e., 28 g/d) in our trial and the study duration were not sufficient to detect changes in glycemic control.

Consistent evidence shows that tree nuts and peanuts improve lipids and lipoproteins. A systematic review of 23

TABLE 3 Between-condition mean differences and change from baseline for daily nutrient and food group intake in adults with elevated FPG ($n = 46$)[1]

Outcome	Peanut		Within-condition difference[3]	LFHC		Within-condition difference[3]	Between-condition effect[3]	Between-condition *P* value
	Baseline[2]	End point		Baseline[2]	End point			
Energy, kcal	2090 ± 113	2036 ± 89	−60 (−304, 184)	2075 ± 114	2078 ± 88	−8 (−197, 244)	−43 (−251, 236)	0.71
Protein, g	94 ± 6	91 ± 5	−0.7 (−14.0, 12.6)	91 ± 6	90 ± 5	−0.1 (−13.4, 13.2)	1.1 (−14.6, 16.7)	0.89
Protein, E %	18.3 ± 1	18.4 ± 1	0.7 (−1.5, 3.0)	17.5 ± 1	17.7 ± 1	0.4 (−1.2, 1.9)	0.6 (−1.7, 2.9)	0.59
Carbohydrate, g	232 ± 15	211 ± 11	−21.2 (−50.8, 8.4)	232 ± 15	228 ± 11	−4.8 (−34.4, 24.8)	−17.1 (−44.7, 10.4)	0.22
Carbohydrate, E %	44.4 ± 2	41.4 ± 1	−3.2 (−6.7, 0.2)	44.7 ± 1	44.4 ± 1	−0.2 (−3.4, 3.0)	−3.0 (−7.4, 1.4)	0.17
Total fat, g	90 ± 7	93 ± 6	1.4 (−15.0, 16.8)	88 ± 5	90 ± 6	1.0 (−14.4, 16.4)	3.1 (−13.6, 19.8)	0.71
Fat, E %	38.6 ± 1	41.0 ± 1	2.0 (−1.2, 5.1)	38.2 ± 1	38.3 ± 1	−0.0 (−3.2, 2.8)	2.6 (−1.3, 6.6)	0.19
SFA, g	32 ± 3	29 ± 3	−2.9 (−9.8, 3,9)	29 ± 2	33 ± 3	3.1 (−3.8, 9.9)	−3.4 (−11.9, 5.2)	0.43
SFA, E %	13.6 ± 1	12.8 ± 1	−1.1 (−2.9, 0.7)	12.8 ± 1	13.6 ± 1	0.8 (−1.0, 2.7)	−0.8 (−2.8, 1.2)	0.41
MUFA, g	30 ± 2	33 ± 2	2.9 (−2.5, 8.2)	30 ± 2	31 ± 2	0.8 (−4.5, 6.2)	2.7 (−3.0, 8.3)	0.35
MUFA, E %	13.1 ± 1	14.8 ± 1	1.8 (0.0, 3.2)*	13.0 ± 1	13.1 ± 1	0.1 (−1.2, 1.5)	1.7 (0.0, 3.4)	0.04
PUFA, g	21 ± 2	23 ± 2	1.9 (−2.9, 6.7)	22 ± 2	19 ± 2	−3.3 (−8.1, 1.5)	4.1 (−0.0, 8.2)	0.05
PUFA, E %	8.8 ± 1	10.2 ± 1	1.4 (−0.0, 2.7)	9.4 ± 1	8.3 ± 1	−1.2 (−2.5, 0.0)	1.9 (0.0, 3.4)	0.01
Fiber, g	22 ± 2	19 ± 1	−1.9 (−5.3, 1.5)	21 ± 2	21 ± 1	−0.5 (−3.9, 2.9)	−1.2 (−4.0, 1.7)	0.42
Potassium, mg	2831 ± 144	2772 ± 136	26 (−291, 343)	2774 ± 144	2787 ± 136	59 (−258, 337)	−15 (−335, 305)	0.93
Sodium, mg	3752 ± 224	3678 ± 196	−63 (−564, 439)	3892 ± 263	3827 ± 196	50 (−451, 552)	−148 (−677, 380)	0.57
Total vegetables,[4] c-eq	2.1 ± 0.1	1.9 ± 0.2	−0.2 (−0.7, 0.4)	1.9 ± 0.2	2.1 ± 0.2	0.1 (−0.4, 0.7)	−0.1 (−0.7, 0.4)	0.62
Dark green vegetables,[4] c-eq	0.4 ± 0.1	0.4 ± 0.1	−0.0 (−0.3, 0.2)	0.5 ± 0.1	0.4 ± 0.1	−0.1 (−0.3, 0.2)	0.0 (−0.2, 0.3)	0.69
Red/orange vegetables,[4] c-eq	0.5 ± 0.1	0.6 ± 0.1	0.1 (−0.1, 0.3)	0.5 ± 0.1	0.5 ± 0.1	0.0 (−0.1, 0.2)	0.1 (−0.1, 0.3)	0.27
Starchy vegetables,[4] c-eq	0.5 ± 0.1	0.3 ± 0.1	−0.2 (−0.6, 0.1)	0.3 ± 0.1	0.4 ± 0.1	0.0 (−0.3, 0.4)	−0.0 (−0.4, 0.4)	0.93
Total grains,[4] oz-eq	5.9 ± 0.5	6.1 ± 0.5	−0.1 (−1.4, 1.2)	6.9 ± 0.5	6.7 ± 0.5	−0.2 (−1.5, 1.1)	−0.6 (−1.9, 0.7)	0.37
Whole grains,[4] oz-eq	1.1 ± 0.1	0.6 ± 0.2	−0.3 (−0.8, 0.2)	1.4 ± 0.2	1.8 ± 0.2	0.5 (0.0, 1.0)*	−1.2 (−1.6, −0.8)	<0.01
Refined grains,[4] oz-eq	5.0 ± 0.5	5.4 ± 0.5	0.2 (−1.0, 1.5)	5.5 ± 0.5	4.8 ± 0.5	−0.7 (−2.0, 0.6)	0.6 (−0.7, 1.9)	0.34
Total fruit,[4] c-eq	0.9 ± 0.1	1.1 ± 0.2	0.2 (−0.2, 0.6)	1.1 ± 0.2	1.1 ± 0.2	−0.0 (−0.4, 0.4)	0.0 (−0.4, 0.5)	0.84
Total dairy products,[4] c-eq	1.9 ± 0.3	1.6 ± 0.2	−0.3 (−0.9, 0.3)	2.0 ± 0.2	2.0 ± 0.4	−0.0 (−0.8, 0.7)	0.0 (−0.1, 0.2)	0.32
Total protein foods,[4] oz-eq	6.6 ± 0.7	7.9 ± 0.6	1.6 (−0.1, 3.2)	6.5 ± 0.7	6.0 ± 0.6	−0.5 (−2.2, 1.2)	1.9 (0.1, 3.7)	0.04
Oils, g	26.8 ± 3.3	30.7 ± 2.3	4.6 (−3.9, 13.1)	29.2 ± 3.4	22.4 ± 2.3	−7.6 (−16.9, 0.9)	8.3 (1.9, 14.6)	0.01

[1]Data are presented as least squares means and SE unless otherwise stated. *Indicates within-condition significant difference ($P < 0.05$). Statistical analyses were performed with SAS version 9.4 (SAS Institute). The MIXED procedure was used to determine the effect of the conditions on each outcome measure, the between-condition *P* values represent the main effect of condition. The MIXED procedure was also used to assess change from baseline. c-eq, cup-equivalent; E %, energy percentage; LFHC, lower fat, higher carbohydrate snack; oz-eq, ounce-equivalent.

[2]Values are mean and SE.

[3]Values are least squares mean effect estimate and 95% CI.

[4]Units are based on the Dietary Guidelines for American's definitions (27).

TABLE 4 Between-condition mean differences and change from baseline for the Healthy Eating Index (HEI) score and individual components in adults with elevated FPG ($n = 46$)[1]

Outcome	Peanut		Within-condition difference[3]	LFHC		Within-condition difference[3]	Between-condition effect[3]	Between-condition *P* value
	Baseline[2]	End point		Baseline[2]	End point			
HEI 2015 total score[4]	53.2 ± 1.7	56.5 ± 1.9	3.6 (−1.1, 8.2)	56.9 ± 2.1	52.9 ± 1.9	−2.9 (−7.5, 1.8)	3.6 (−1.9, 9.0)	0.19
Adequacy components								
Total vegetables[5]	3.7 ± 0.2	3.5 ± 0.3	−0.2 (−0.9, 0.4)	3.7 ± 0.2	3.5 ± 0.3	−0.1 (−0.8, 0.5)	−0.0 (−0.6, 0.6)	0.94
Greens and beans[5]	2.3 ± 0.3	2.8 ± 0.4	0.4 (−0.5, 1.3)	3.2 ± 0.3	2.2 ± 0.4	−0.7 (−1.6, 0.2)	0.5 (−0.5, 1.6)	0.32
Total fruits[5]	2.3 ± 0.3	2.3 ± 0.3	−0.0 (−0.7, 0.7)	2.6 ± 0.3	2.3 ± 0.3	−0.2 (−0.9, 0.5)	−0.0 (−0.9, 0.9)	0.96
Whole fruits[5]	2.7 ± 0.3	2.4 ± 0.3	−0.2 (−1.0, 0.5)	2.9 ± 0.3	2.7 ± 0.3	−0.1 (−0.8, 0.7)	−0.3 (−1.3, 0.7)	0.52
Whole grains[6]	3.1 ± 0.4	2.4 ± 0.5	−0.5 (−1.8, 0.7)	4.2 ± 0.6	5.0 ± 0.5	1.0 (−0.2, 2.2)	−2.6 (−3.8, −1.4)	<0.01
Total dairy[6]	5.8 ± 0.4	6.0 ± 0.4	0.0 (−1.1, 1.1)	6.8 ± 0.5	5.9 ± 0.4	−0.8 (−1.9, 0.3)	0.1 (−1.0, 1.2)	0.83
Total protein foods[5]	4.0 ± 0.2	4.7 ± 0.2	0.8 (0.3, 1.2)*	4.2 ± 0.2	4.2 ± 0.2	0.1 (−0.4, 0.6)	0.5 (−0.0, 1.0)	0.06
Seafood and plant proteins[5]	2.7 ± 0.3	3.8 ± 0.3	1.2 (0.3, 2.1)*	2.4 ± 0.3	1.8 ± 0.3	−0.6 (−1.5, 0.3)	2.0 (1.0, 2.9)	<0.01
Fatty acid ratio[6]	4.2 ± 0.5	5.1 ± 0.5	0.8 (−0.5, 2.2)	4.7 ± 0.5	3.6 ± 0.5	−1.0 (−2.3, 0.4)	1.5 (−0.1, 3.1)	0.07
Moderation components								
Sodium[6]	3.2 ± 0.5	4.0 ± 0.5	0.8 (−0.5, 2.1)	2.8 ± 0.5	3.3 ± 0.5	0.5 (−0.8, 1.9)	0.7 (−0.7, 2.0)	0.31
Refined grains[6]	6.9 ± 0.5	6.4 ± 0.5	−0.5 (−1.8, 0.8)	6.2 ± 0.6	7.3 ± 0.5	1.1 (−0.3, 2.5)	−0.9 (−2.1, 0.3)	0.12
Saturated fats[6]	4.3 ± 0.5	5.4 ± 0.5	1.0 (−0.1, 2.2)	4.6 ± 0.5	4.3 ± 0.5	−0.2 (−1.4, 1.0)	1.1 (−0.4, 2.5)	0.14
Added sugars[6]	8.0 ± 0.4	8.9 ± 0.3	0.8 (0.1, 1.5)*	8.6 ± 0.3	8.1 ± 0.3	−0.4 (−1.1, 0.3)	0.8 (0.0, 1.5)	0.04

[1]Data are presented as least squares means and SE unless otherwise stated. *Indicates within-condition significant difference ($P < 0.05$). Statistical analyses were performed with SAS version 9.4 (SAS Institute). The MIXED procedure was used to determine the effect of the conditions on each outcome measure, the between-condition *P* values represent the main effect of condition. The MIXED procedure was also used to assess change from baseline. HEI, Healthy Eating Index; LFHC, lower fat, higher carbohydrate snack.
[2]Values are mean and SE.
[3]Values are least squares mean effect estimate and 95% CI.
[4]Maximum total score = 100.
[5]Maximum score = 5.
[6]Maximum score = 10.

trials concluded that consuming 50–100 g peanuts and tree nuts 5 times/wk, as a part of a heart-healthy diet, lowers total cholesterol concentrations by 2–16% and LDL cholesterol by 2–19% (24). Del Gobbo et al. (25) conducted a systematic review and meta-analysis of 61 trials and reported a dose–response reduction per serving per day of peanuts and tree nuts for total cholesterol (−4.7 mg/dL), LDL cholesterol (−4.8 mg/dL), and triglycerides (−2.2 mg/dL). The response for LDL cholesterol was nonlinear, with greater reductions observed in studies that provided >60 g/d nuts. Many of the trials included in this systematic review evaluated tree nut intake in subjects with hyperlipidemia, and median nut intake was greater than our study. Recently, epidemiological studies have demonstrated that LDL subparticle concentrations [LDL subparticle classes I–IV (I is the largest)] are differentially associated with coronary heart disease risk. Small, dense LDL particles are positively associated with coronary heart disease risk (26). We observed a nonsignificant increase in LDL cholesterol between the peanut and LFHC condition (4.3 mg/dL; 95% CI: −1.4, 9.8; $P = 0.15$) and this was likely driven by the significant increase in LDL IIa, a larger, more buoyant particle. The increase in apoB concentration suggests more LDL cholesterol particles were present following the peanut condition, which is likely attributable to the increase in LDL IIa. We likely did not observe changes in lipids and lipoproteins because our participants were normolipidemic at baseline, the dose of peanuts tested might have been insufficient to affect lipids and lipoproteins, and peanuts, because of their fatty acid profile, have different lipid-lowering effects than some tree nuts.

The 2020–2025 (27) and 2015–2020 (28) Dietary Guidelines recommend consuming 5 oz-eq/wk of nuts, for those consuming 2000 kcal/d, as part of a healthy dietary pattern. In our study, participants were instructed to consume 14 oz-eq/wk and we did not observe an increase in diet quality measured by the HEI-2015 (3.6; 95% CI: −1.9, 9.0; $P = 0.19$) with the peanut condition compared with the LFHC snack. Baseline diet quality in our sample reflected average US diet quality (58 for adults aged 18–64 y, and 64 for adults ≥65 y) (29). The HEI-2015 changes we observed were driven by significant improvements in the seafood/plant protein component, and nonsignificant improvements in the total protein and fatty acid components, which is expected based on the composition of peanuts. Therefore, the observed dietary changes following both conditions reflect the intervention, suggesting that the participants' background diets did not change. Our findings suggest that intake of peanuts as a nighttime snack is a relatively simple dietary strategy to improve the fatty acid profile of the diet and protein intake from plants/seafood in individuals with elevated FPG. This dietary intervention together with other small changes could significantly improve overall dietary quality and benefit health (30).

Based on NHANES 2013–2016, the average US adult (≥18 y) engages in 1.56 snacking occasions per day, with an average of 236 kcal/snack; therefore snacking contributes ∼370 kcal/d to the US diet (31). Our participants consumed a similar number of calories from snacks at baseline. Calories, nutrient, and food group intake from snacks were unchanged following both conditions and no significant between-condition differences were observed. Additionally, we did not observe any compensatory eating behaviors (i.e., consuming more, or less, calories at other eating occasions) for either condition. These findings demonstrate that the addition of peanuts as a nighttime snack does not have adverse effects on total or snacking caloric intake and does not promote compensatory eating behaviors.

The strengths of this study are the design, glycemic control measures, and dietary assessment. The crossover design with baseline measures for each condition allows for the change from baseline to be calculated for each condition and determination of mean differences between the treatments. Assessing both FPG and fructosamine provides information about short- and longer-term glycemic control. The dietary assessment using ASA24 generated information about small, yet important, dietary changes that might not have been detected using other dietary assessment methods. Limitations of this study include the single-blinded protocol, participant screening procedures, lack of a no-snack condition, and nonrandom dietary assessment. The participants were not blinded to the intervention allocation, which could have introduced bias; however, double-blinding was not feasible given the study design. All outcome measures had a low risk for bias from the single-blind protocol and were collected/analyzed by blinded study personnel. We enrolled subjects based on a single FPG measure ≥100 mg/dL at screening. However, due to day-to-day variability, some individuals had a baseline FPG <100 mg/dL. We did not include a no-snack condition and therefore it remains unclear whether no nighttime snack is superior to nighttime snacking for FPG control. Nonrandom 24-h recalls were administered and participants were aware of the diet assessment days, which might have impacted reporting. Finally, the risk of type 1 statistical errors is inflated by of the number of analyses conducted for the secondary end points.

In summary, we demonstrated that 28 g/d of dry roasted, unsalted peanuts as a nighttime snack did not affect FPG, lipids/lipoproteins, vascular health, or weight in subjects with elevated FPG compared with an LFHC snack. However, this relatively low-calorie dietary intervention resulted in a greater percentage of energy from PUFAs and MUFAs being consumed, as well as higher intake of oil-containing foods, and total protein from plant/seafood sources. These findings suggest that peanuts can be consumed as a nighttime snack for those with elevated FPG and, importantly, do not have adverse effects on FPG. Further research is necessary to elucidate the effect of larger doses of peanuts as an evening snack on glycemic control in individuals with prediabetes.

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