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ORIGINAL ARTICLE

Clinical Trials and Investigations

Almonds vs. carbohydrate snacks in an energy-restricted diet: Weight and cardiometabolic outcomes from a randomized trial

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Abstract

Objective: This study evaluated weight and cardiometabolic outcomes after a 3-month energy-restricted diet (−30%) containing almonds (almond-enriched diet [AED]) or containing carbohydrate-rich snacks (nut-free control diet [NFD]) (Phase 1), followed by 6 months of weight maintenance (Phase 2).

Methods: Participants (25–65 years old) with overweight or obesity (BMI 27.5–34.9 kg/m^2) were randomly allocated to AED ($n = 68$) or NFD ($n = 72$).

Results: Both groups lost weight during Phase 1 ($p < 0.001$) (mean [SE], −7.0 [0.5] kg AED vs. −7.0 [0.5] kg NFD, $p = 0.858$) and Phase 2 ($p = 0.009$) (−1.1 [0.5] kg AED vs. −1.3 [0.6] NFD, $p = 0.756$), with improvements in percentage lean mass after Phase 2 (4.8% [0.3%], $p < 0.001$). Reductions occurred in fasting glucose (−0.2 [0.07] mmol/L, $p = 0.003$), insulin (−8.1 [4.0] pmol/L, $p = 0.036$), blood pressure (−4.9 [0.8] mm/Hg systolic, −5.0 [0.5] mm/Hg diastolic, $p < 0.001$), total cholesterol (−0.3 [0.1] mmol/L), low-density lipoprotein (LDL) (−0.2 [0.1] mmol/L), very low-density lipoprotein (−0.1 [0.03] mmol/L), and triglycerides (−0.3 [0.06] mmol/L) (all $p < 0.001$), and high-density lipoprotein increased (0.1 [0.02] mmol/L, $p = 0.011$) by the end of Phase 2 in both groups. There were group by time interactions for lipoprotein particle concentrations: very small triglyceride-rich (−31.0 [7.7] nmol/L AED vs. −4.8 [7.9] nmol/L NFD, $p = 0.007$), small LDL (−109.3 [40.5] nmol/L AED vs. −20.7 [41.6] nmol/L NFD, $p = 0.017$), and medium LDL (−24.4 [43.4] nmol/L AED vs. −130.5 [44.4] nmol/L NFD, $p = 0.045$).

Conclusions: An energy-restricted AED resulted in weight loss and weight loss maintenance comparable to an energy-restricted NFD, and both diets supported cardiometabolic health. The AED resulted in greater improvements in some lipoprotein subfractions, which may enhance reductions in cardiovascular risk.

INTRODUCTION

Obesity is a leading cause of preventable death and a major risk factor for the development of hypertension, type 2 diabetes, and cardiovascular disease (CVD) [1]. Studies prescribing energy-restricted diets and lifestyle modification have induced weight loss among participants; however, weight loss maintenance is more difficult to achieve. Dietary strategies that help participants reduce energy intake and sustain changes in the long term are needed.

Nuts may play a role in weight management. Epidemiological studies and clinical trials indicate an inverse association between nut consumption and body mass index (BMI), suggesting that nut consumption could be protective against accumulation of adiposity [2–7]. The mechanisms underlying this relationship are unclear but they may be related to appetite regulation, increased resting energy expenditure, and inefficient energy absorption from nuts [4, 8–12].

Dietary patterns that include nuts (e.g., Mediterranean, Portfolio, Dietary Approaches to Stop Hypertension [DASH]) are recommended for CVD prevention and management [13]. A Mediterranean diet supplemented with nuts (30 g/d) reduced the risk of cardiovascular events by approximately 30% [14], and nut consumption of >3 servings per week (84 g/wk) was associated with a decreased risk of metabolic syndrome and diabetes [15]. Nuts are high in protein, fiber, and unsaturated fatty acids, and their inclusion in the diet contributes to improved diet quality. These nutritional qualities likely contribute to their beneficial cardiometabolic effects [16–18], and studies have suggested that regular nut intake reduces the risk of CVD [19–21].

Despite these findings, there is a widespread perception that nut consumption will lead to increased body weight and long-term health risks due to their high fat content and, consequently, that nuts should be avoided because they might increase body weight or impair weight loss when a person is trying to lose weight. This perception undermines the public health call for increased nut consumption [22] as a strategy to manage obesity and cardiometabolic risk factors. Therefore, this study aimed to contribute to the growing research surrounding nuts, specifically to evaluate whether the inclusion of 15% of dietary energy from almonds [almond-enriched diet (AED)] compared with carbohydrate-rich snacks in an otherwise nut-free diet [nut-free control diet (NFD)] would improve weight loss during 3 months of dietary energy restriction and limit weight regain during 6 months of weight maintenance (eucaloric). We hypothesized that the AED would lead to greater weight loss during the energy restriction phase of 3 months and limit weight regain during the weight maintenance period of 6 months compared with the NFD. The study also aimed to assess the effects of the AED compared with the NFD on cardiometabolic risk factors.

Study Importance

What is already known?

- Studies have indicated an inverse association between nut consumption and BMI, suggesting that nut consumption may have a protective effect against accumulation of adiposity.
- Nuts are high in protein, fiber, and unsaturated fatty acids, properties that likely promote their beneficial effects on cardiometabolic abnormalities.

What does this study add?

- This is the largest study to date to assess benefits of incorporating almonds into an energy-restricted diet for weight loss and weight loss maintenance, and it contributes to the growing evidence that nuts can support a healthy diet for weight management.
- It also contributes to the limited knowledge of the effects of nuts on lipoprotein subfractions.

How might these results change the direction of research?

- Future studies should consider the dose of almonds and effects in populations with elevated cardiometabolic risk factors.
- Lipoprotein subfractions should be included when profiling lipid and lipoprotein responses to dietary changes.

METHODS

Study design

The full protocol including the study design, eligibility criteria, and a detailed description of the outcome measures has been reported previously [23]. Briefly, 140 male and female volunteers, aged 25 to 65 years with BMI of 27.5 to 34.9 kg/m^2, enrolled in a 9-month, randomized controlled, parallel-arm dietary intervention conducted between January 15, 2019, and March 10, 2021. The study was approved by the University of South Australia Human Research Ethics Committee (no. 201436) and registered with the Australian New Zealand Clinical Trials Registry (no. 12618001861246). Prior to commencement, written informed consent was obtained.

Participants were assigned to the AED or the NFD group using minimization [24] based on age, sex, and BMI. A staff member independent of the study outcomes and analysis performed the randomization, and staff conducting clinical assessments were blinded to treatment groups. Participants completed a 12-week hypocaloric weight loss phase (Phase 1: weeks 0 to 12) followed by a 24-week eucaloric weight maintenance phase (Phase 2: weeks 13 to 36). Participants attended the clinic at baseline and the end of each study phase to assess outcomes (weeks 0, 12, and 36). The primary outcome was weight loss and weight loss maintenance in the respective phases. Secondary outcomes were body composition (measured by dual-energy x-ray absorptiometry), waist circumference, and total energy expenditure (objective data collected via

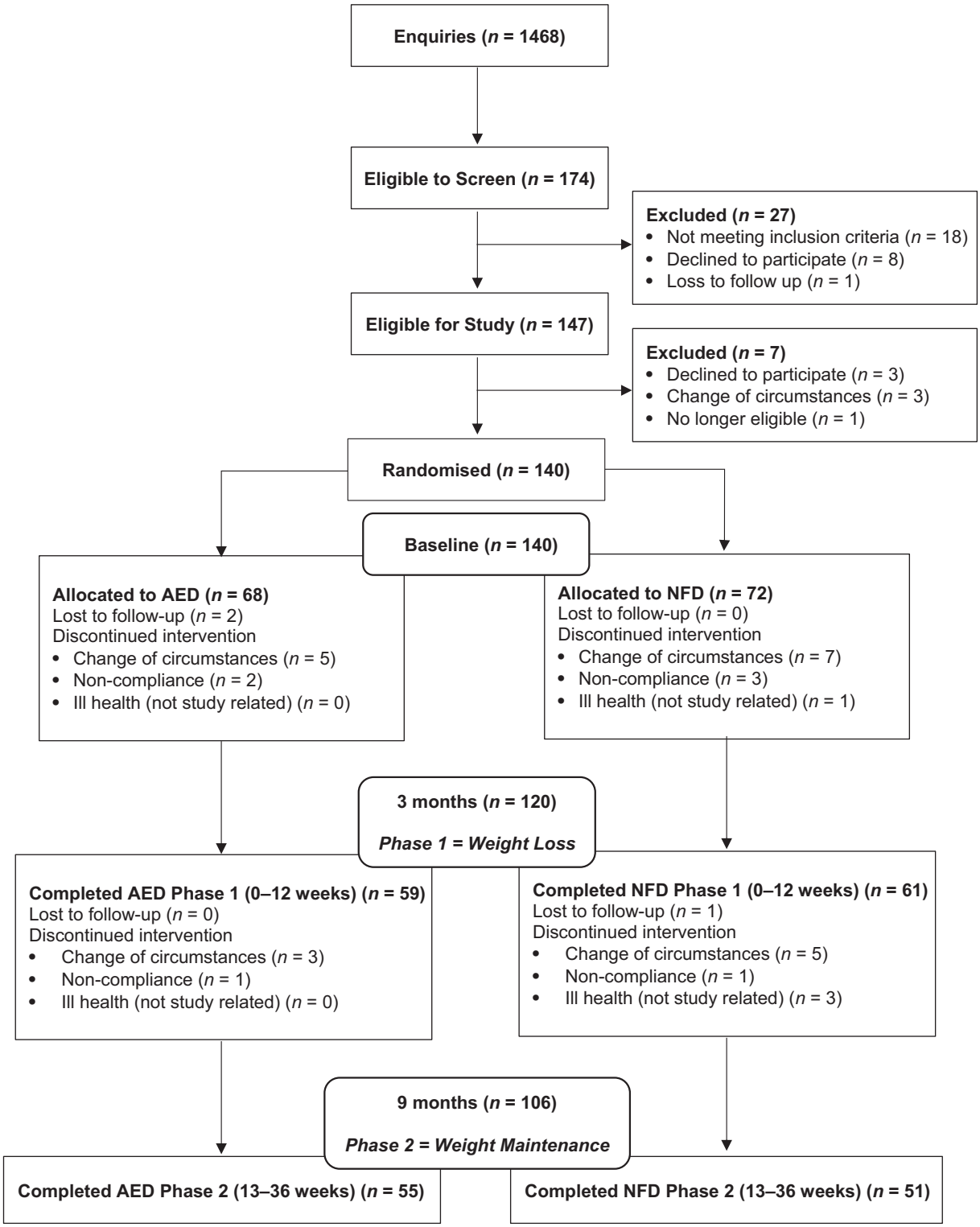

FIGURE 1 Consort flow diagram. AED, almond-enriched diet; NFD, nut-free diet

GENEActiv triaxial accelerometers and subjective data via the International Physical Activity Questionnaire [IPAQ]). Tertiary outcomes were systolic blood pressure (SBP), diastolic blood pressure (DBP), blood lipid profile (including subfraction particle size and concentration), glucose, insulin, insulin resistance (homeostasis model assessment index 2 of insulin resistance [HOMA2-IR]), insulin sensitivity (homeostasis model assessment index 2 of insulin sensitivity [HOMA2-%S]), and pancreatic β cell function (homeostasis model assessment index 2 of pancreatic β cell function [HOMA2-%B]). Metabolic syndrome score was also calculated [25].

Outcome assessment procedures are detailed in the protocol paper [23].

COVID-19

COVID-19 restrictions interrupted clinic visits between April and June 2020. During this time, some anthropometric data (waist circumference and body composition) and blood samples were not collected. Participants were provided with Bluetooth-enabled scales (Withings/Nokia WBS06, Nokia) to capture body weight at home. Weight captured by the Bluetooth-enabled scales was used in analyses after determining that there was no difference in the magnitude of weight loss achieved by participants assessed using Bluetooth-enabled scales compared with participants assessed using clinic scales.

Diet intervention

During the 9-month study period, participants were prescribed an energy target to facilitate weight loss (Phase 1, 3 months) or maintenance of weight loss (Phase 2, 6 months). In addition, during both phases, participants in the AED group incorporated 15% of their energy as unsalted, whole, natural Californian almonds with skins (e.g., 30–50 g of almonds), whereas participants in the NFD group included 15% of their energy from carbohydrate-rich snack foods (oven-baked fruit cereal bar and rice crackers) (Supporting Information Table S1) (see protocol paper for more details [23]). The control foods were chosen because they are commonly consumed snacks that are lower in beneficial micro- and macronutrients found in almonds but they provide similar energy. Participants were provided with the test foods to consume 6 days per week at any time of the day with 1 day per week free from consuming the test foods. This approach has been found to enhance compliance with the test food regimen. Compliance was monitored by reports of remaining test foods in grams or count (collected every 2–4 weeks). An ≥80% compliance was set as a reasonable expectation of compliance during a 9-month study. All participants were asked to avoid all other nuts/nut products for the duration of the trial.

Individual estimated energy requirements were calculated using the Schofield equation based on sex, age, and initial body weight and multiplied by physical activity level [26]. A moderate energy restriction (30% less than estimated energy requirement) was prescribed to facilitate weight loss for Phase 1 (weeks 0 to 12). To achieve weight maintenance for Phase 2 (weeks 12 to 36), participants were counseled to increase their overall energy intake as required. Participants received comprehensive dietary advice from a qualified dietitian at baseline and every 2 weeks during Phase 1. During Phase 2, participants received individual advice from a dietitian every 2 weeks for the first month and then monthly in small groups for the remainder of the study. Weighed 4-day food records were obtained 2 weeks before baseline and at the end of Phases 1 and 2 to assess compliance with the dietary intervention using Foodworks Nutritional Analysis Software V.10 (Xyris Software).

Statistical analysis

Statistical analyses were performed using SPSS Statistics version 28.0 (IBM Corp.). Sample size calculations were based on the primary outcome (change in weight), and they are detailed in the protocol paper [23]. Non-normally distributed data (HOMA-IR) were log transformed before analysis. The effects of the different interventions over time were assessed using an intention-to-treat analysis (including all participants who commenced the study) using mixed effects modeling. The fixed effects included treatment (AED or NFD) and time (baseline, 12 weeks, and 36 weeks) with participants as the random effect. Age, sex, and BMI were included in the models as covariates. Where main effects were identified, post hoc comparisons were performed with Bonferroni adjustments for multiple comparisons to determine differences between group means. Data are presented as mean ± standard error (SE), and statistical significance was set at $p < 0.05$. Results are reported as Phase 1 (0–12 weeks), Phase 2 (13–36 weeks), and end of trial (0–36 weeks).

RESULTS

Participants

Of the 140 participants randomized (AED $n = 68$, NFD $n = 72$), 120 completed Phase 1 (AED $n = 59$, NFD $n = 61$), and 106 completed Phase 2 (AED $n = 55$, NFD $n = 51$) (Figure 1, Table 1). There were no differences between those who did versus did not complete the study at baseline for any variables.

Dietary compliance

Compliance to intervention (i.e., almond consumption or carbohydrate-based snack consumption) was achieved by all participants, and

TABLE 1 Baseline characteristics

Participant demographics	AED ($n = 68$)	NFD ($n = 72$)
Age (y), mean ± SE	48.2 ± 1.3	46.8 ± 1.3
Sex, male: female (%)	20:48 (29:71)	22:50 (31:69)
Ethnicity, *n* (%)		
Caucasian	53 (79)	62 (86)
Asian	7 (10)	7 (10)
Hispanic/Latino	4 (6)	3 (4)
African	3 (4)	0 (0)
SEIFA decile, mean ± SE	7.0 ± 2.2	6.7 ± 2.5
Antihypertensive medication, *n* (%)	6 (9)	8 (11)
Lipid-lowering medication, *n* (%)	5 (7)	2 (3)

Note: Mixed models were used. Values are mean ± SE or *n* (%). All *p* values are >0.05. Ethnicity data available for AED $n = 67$, NFD $n = 72$. SEIFA was collated using the Australian Bureau of Statistics' Socio-Economic Indexes for Areas (SEIFA), Australia, 2016, State Suburb Indexes (1 to 10 = Disadvantaged to Advantaged).
Abbreviations: AED, almond-enriched diet; NFD, nut-free diet.

TABLE 2 Diet and physical activity data

	Baseline	3 Months	Change in Phase 1	9 Months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
Energy (kJ)									
AED	9208 ± 217	5964 ± 217	−3244 ± 227	6648 ± 225	684 ± 234	−2559 ± 234	0.268	<0.001	0.623
NFD	8810 ± 228	5608 ± 231	−3201 ± 241	6554 ± 230	946 ± 243	−2255 ± 239			
Carbohydrate (g)									
AED	227.2 ± 6.4	135.9 ± 6.4	−91.8 ± 6.6	154.7 ± 6.6	19.3 ± 6.9	−72.5 ± 6.8	0.162	<0.001	0.002
NFD	217.6 ± 6.7	155.9 ± 6.8	−61.7 ± 7.0	176.0 ± 6.8	20.1 ± 7.1	−41.6 ± 7.0			
Carbohydrate (% energy)									
AED	40.5 ± 0.9	37.4 ± 0.9	−3.0 ± 0.9	38.3 ± 0.9	0.9 ± 1.0	−2.2 ± 1.0	<0.001	0.235	<0.001
NFD	40.3 ± 0.9	45.5 ± 0.9	5.3 ± 1.0	44.3 ± 0.9	−1.2 ± 1.0	4.1 ± 1.0			
Dietary fiber (g)									
AED	24.3 ± 1.0	26.7 ± 1.0	2.4 ± 1.0	25.9 ± 1.0	−0.8 ± 1.1	1.6 ± 1.1	0.304	<0.001	0.105
NFD	24.3 ± 1.0	27.4 ± 1.1	3.2 ± 1.1	28.9 ± 1.0	1.5 ± 1.1	4.7 ± 1.1			
Protein (g)									
AED	92.5 ± 2.8	77.9 ± 2.8	−14.6 ± 3.2	83.8 ± 3.0	5.8 ± 3.3	−8.7 ± 3.3	0.898	<0.001	0.197
NFD	97.5 ± 3.0	74.6 ± 3.0	−22.8 ± 3.4	83.3 ± 3.0	8.7 ± 3.4	−14.2 ± 3.3			
Protein (% energy)									
AED	17.2 ± 0.5	22.4 ± 0.5	5.2 ± 0.6	21.8 ± 0.5	−0.6 ± 0.7	4.6 ± 0.7	0.128	<0.001	0.097
NFD	19.2 ± 0.5	23.1 ± 0.6	3.9 ± 0.7	21.8 ± 0.5	−1.3 ± 0.7	2.6 ± 0.7			
Total fat (g)									
AED	87.5 ± 2.6	55.0 ± 2.6	−32.5 ± 2.9	61.5 ± 2.7	6.5 ± 3.0	−26.1 ± 3.0	<0.001	<0.001	0.009
NFD	83.1 ± 2.8	37.5 ± 2.8	−45.6 ± 3.1	47.8 ± 2.8	10.4 ± 3.2	−35.2 ± 3.1			
Total fat (% energy)									
AED	35.1 ± 0.8	34.0 ± 0.8	−1.2 ± 0.8	34.0 ± 0.8	0.05 ± 0.9	−1.2 ± 0.9	<0.001	<0.001	<0.001
NFD	34.5 ± 0.8	25.0 ± 0.8	−9.9 ± 0.9	26.6 ± 0.8	2.0 ± 0.9	−7.9 ± 0.9			
Saturated fat (g)									
AED	34.8 ± 1.1	15.6 ± 1.1	−19.3 ± 1.3	18.4 ± 1.2	2.9 ± 1.4	−16.4 ± 1.4	0.170	<0.001	0.666
NFD	33.3 ± 1.2	12.9 ± 1.2	−20.4 ± 1.4	17.5 ± 1.2	4.7 ± 1.4	−15.8 ± 1.4			
Saturated fat (% energy)									
AED	14.0 ± 0.4	9.4 ± 0.4	−4.6 ± 0.5	10.1 ± 0.4	0.7 ± 0.5	−4.0 ± 0.5	0.196	<0.001	0.513
NFD	13.8 ± 0.4	8.4 ± 0.4	−5.4 ± 0.5	9.7 ± 0.4	1.2 ± 0.5	−4.1 ± 0.5			

(Continues)

TABLE 2 (Continued)

	Baseline	3 Months	Change in Phase 1	9 Months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
MUFA (g)									
AED	32.2 ± 1.1	24.2 ± 1.1	−8.0 ± 1.3	26.3 ± 1.2	2.1 ± 1.4	−5.9 ± 1.4	<0.001	<0.001	<0.001
NFD	30.7 ± 1.2	14.1 ± 1.2	−16.6 ± 1.4	17.6 ± 1.2	3.3 ± 1.4	−13.1 ± 1.4			
MUFA (% energy)									
AED	12.9 ± 0.4	15.1 ± 0.4	2.2 ± 0.4	14.6 ± 0.4	−0.5 ± 0.5	1.7 ± 0.5	<0.001	0.083	<0.001
NFD	12.7 ± 0.4	9.2 ± 0.4	−3.5 ± 0.5	10.0 ± 0.4	0.7 ± 0.5	−2.8 ± 0.5			
PUFA (g)									
AED	13.0 ± 0.5	10.2 ± 0.5	−2.8 ± 0.6	10.8 ± 0.5	0.6 ± 0.6	−2.1 ± 0.6	<0.001	<0.001	0.007
NFD	11.4 ± 0.5	6.1 ± 0.5	−5.3 ± 0.6	7.6 ± 0.5	1.5 ± 0.6	−3.8 ± 0.6			
PUFA (% energy)									
AED	5.2 ± 0.2	6.4 ± 0.2	1.2 ± 0.2	6.0 ± 0.2	−0.4 ± 0.2	0.8 ± 0.2	<0.001	0.491	<0.001
NFD	4.8 ± 0.2	3.9 ± 0.2	−0.8 ± 0.2	4.3 ± 0.2	0.3 ± 0.3	−0.5 ± 0.3			
Alcohol (g)									
AED	13.2 ± 1.5	3.7 ± 1.5	−9.5 ± 1.6	5.4 ± 1.6	1.8 ± 1.6	−7.8 ± 1.6	0.272	<0.001	0.230
NFD	9.0 ± 1.6	2.8 ± 1.6	−6.2 ± 1.7	4.7 ± 1.6	1.8 ± 1.7	−4.3 ± 1.6			
Alcohol (% energy)									
AED	4.1 ± 0.5	1.5 ± 0.5	−2.6 ± 0.5	2.1 ± 0.6	0.6 ± 0.6	−2.0 ± 0.6	0.628	<0.001	0.393
NFD	3.2 ± 0.6	1.3 ± 0.6	−1.8 ± 0.6	2.2 ± 0.6	0.9 ± 0.6	−0.9 ± 0.6			
Alpha-tocopherol (mg)									
AED	11.2 ± 0.5	16.4 ± 0.5	5.2 ± 0.6	16.0 ± 0.5	−0.4 ± 0.6	4.8 ± 0.6	<0.001	0.016	<0.001
NFD	10.6 ± 0.5	7.4 ± 0.5	−3.2 ± 0.6	8.0 ± 0.5	0.6 ± 0.6	−2.6 ± 0.6			
Sodium (mg)									
AED	2692.7 ± 114.9	1894.0 ± 114.9	−798.7 ± 123.1	1976.2 ± 119.1	82.3 ± 127.0	−716.5 ± 127.0	0.628	<0.001	0.349
NFD	2907.2 ± 120.5	1895.4 ± 122.4	−1011.9 ± 130.9	1952.1 ± 121.4	56.7 ± 131.7	−955.2 ± 130.0			
Potassium (mg)									
AED	3261.7 ± 108.2	3204.5 ± 108.2	−57.2 ± 104.5	3163.2 ± 111.6	−41.3 ± 108.0	−98.5 ± 108.0	0.798	0.030	0.125
NFD	3338.9 ± 113.5	2990.9 ± 115.0	−348.0 ± 111.2	3198.7 ± 114.2	207.8 ± 111.9	−140.2 ± 110.4			
Magnesium (mg)									
AED	347.6 ± 12.0	359.5 ± 12.0	11.9 ± 10.9	363.2 ± 12.3	3.7 ± 11.2	15.6 ± 11.2	0.026	0.028	<0.001
NFD	350.8 ± 12.6	296.9 ± 12.7	−53.9 ± 11.6	321.9 ± 12.7	25.0 ± 11.6	−28.9 ± 11.5			

TABLE 2 (Continued)

	Baseline	3 Months	Change in Phase 1	9 Months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
Calcium (mg)									
AED	953.6 ± 40.9	848.4 ± 40.9	−105.2 ± 39.5	887.5 ± 42.1	39.1 ± 40.8	−66.1 ± 40.8	0.648	<0.001	0.185
NFD	990.9 ± 42.9	784.4 ± 43.4	−206.5 ± 42.0	846.2 ± 43.1	61.8 ± 42.3	−144.7 ± 41.7			
Iron (mg)									
AED	11.0 ± 0.4	9.8 ± 0.4	−1.2 ± 0.4	10.0 ± 0.4	0.2 ± 0.4	−0.9 ± 0.4	0.205	<0.001	0.005
NFD	11.3 ± 0.4	8.2 ± 0.4	−3.1 ± 0.4	9.4 ± 0.4	1.2 ± 0.4	−1.9 ± 0.4			
Total MET-min/wk									
AED	2846 ± 478	2730 ± 490	−115.8 ± 368.0	3231 ± 513	500.8 ± 390.1	384.9 ± 397.4	0.531	0.067	0.648
NFD	2947 ± 472	3245 ± 477	−298.5 ± 359.1	3776 ± 497	829.5 ± 386.0	829.5 ± 386.0			
GENEA sleep (min/d)									
AED	484 ± 6.0	485 ± 6.8	0.6 ± 6.7	471 ± 7.2	−14.3 ± 7.7	−13.7 ± 7.1	0.815	0.013	0.649
NFD	490 ± 5.8	482 ± 7.0	−8.2 ± 6.9	474 ± 6.9	−7.3 ± 7.7	−15.5 ± 6.8			
GENEA Sed (min/d)									
AED	634 ± 11.3	637 ± 12.1	3.7 ± 8.7	642 ± 12.5	4.8 ± 9.9	8.5 ± 9.3	0.071	0.402	0.042
NFD	624 ± 11.0	605 ± 12.2	−18.9 ± 8.9	602 ± 12.1	−2.6 ± 9.9	−21.5 ± 8.8			
GENEA light (min/d)									
AED	216 ± 7.5	208 ± 8.0	−8.3 ± 5.3	219 ± 8.2	11.3 ± 6.1	3.0 ± 5.7	0.210	0.037	0.066
NFD	219 ± 7.4	227 ± 8.9	8.4 ± 5.5	235 ± 8.0	7.4 ± 6.1	15.8 ± 5.4			
GENEA MV (min/d)									
AED	90 ± 5.6	90 ± 6.0	−0.5 ± 4.2	84 ± 6.2	−5.9 ± 4.8	−6.4 ± 4.5	0.089	0.843	0.089
NFD	97 ± 5.5	101 ± 6.0	4.0 ± 4.3	104 ± 6.0	3.3 ± 4.8	7.3 ± 4.3			

Note: Values are mean ± SE. Mixed models were used to compare outcomes at the three time points, controlling for age, sex, and BMI. Significance set at $p < 0.05$. Diet data available for AED $n = 68$, NFD $n = 71$. Diet diaries analyzed in Foodworks Nutritional Analysis Software V.10 (Xyris Software). Total MET-min/wk available for AED $n = 62$, NFD $n = 63$. GENEA data available for AED $n = 66$, NFD $n = 70$. Diet data were excluded due to extremes of total energy intakes $n = 1$.

Abbreviations: AED, almond-enriched diet; GENEA, GENEActiv triaxial accelerometers; MET, metabolic equivalent of task; MUFA, monounsaturated fatty acid; MV, moderate-vigorous activity; NFD, nut-free diet; PUFA, polyunsaturated fatty acid; Sed, sedentary.

TABLE 3 Changes in weight and body composition

	Baseline	3 Months	Change in Phase 1	9 Months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
Weight (kg)									
AED	87.8 ± 0.8	80.9 ± 0.9	−7.0 ± 0.5	79.8 ± 0.9	−1.1 ± 0.5	−8.0 ± 0.5	0.818	<0.001	0.963
NFD	87.7 ± 0.8	80.7 ± 0.8	−7.0 ± 0.5	79.4 ± 0.9	−1.3 ± 0.6	−8.3 ± 0.6			
BMI (kg/m^2)									
AED	30.7 ± 0.1	28.3 ± 0.1	−2.4 ± 0.2	27.9 ± 0.2	−0.4 ± 0.2	−2.8 ± 0.2	0.865	<0.001	0.967
NFD	30.7 ± 0.1	28.2 ± 0.1	−2.5 ± 0.2	27.8 ± 0.2	−0.4 ± 0.2	−2.9 ± 0.2			
Waist circumference (cm)									
AED	101.8 ± 0.8	94.3 ± 0.8	−7.5 ± 0.8	93.2 ± 0.9	−1.1 ± 0.8	−8.6 ± 0.8	0.614	<0.001	0.587
NFD	102.4 ± 0.7	95.4 ± 0.8	−7.0 ± 0.8	93.0 ± 0.8	−2.4 ± 0.9	−9.4 ± 0.8			
Total body fat mass (kg)									
AED	35.9 ± 0.6	30.1 ± 0.6	−5.8 ± 0.5	28.4 ± 0.6	−1.7 ± 0.6	−7.5 ± 0.5	0.848	<0.001	0.810
NFD	35.8 ± 0.5	30.2 ± 0.6	−5.5 ± 0.5	28.7 ± 0.6	−1.5 ± 0.6	−7.0 ± 0.5			
Total body fat mass (%)									
AED	42.7 ± 0.5	38.8 ± 0.5	−3.9 ± 0.4	37.2 ± 0.6	−1.6 ± 0.5	−5.4 ± 0.4	0.953	<0.001	0.646
NFD	42.5 ± 0.5	38.8 ± 0.5	−3.7 ± 0.4	37.6 ± 0.5	−1.2 ± 0.4	−4.9 ± 0.4			
Android fat mass (kg)									
AED	3.4 ± 0.1	2.6 ± 0.1	−0.8 ± 0.1	2.5 ± 0.1	−0.2 ± 0.1	−0.9 ± 0.1	0.506	<0.001	0.746
NFD	3.4 ± 0.1	2.7 ± 0.1	−0.7 ± 0.1	2.6 ± 0.1	−0.1 ± 0.1	−0.9 ± 0.1			
Android fat mass (%)									
AED	49.8 ± 0.8	43.9 ± 0.8	−5.9 ± 0.7	41.4 ± 0.9	−2.5 ± 0.7	−8.4 ± 0.7	0.932	<0.001	0.438
NFD	49.5 ± 0.7	43.6 ± 0.8	−5.8 ± 0.6	42.2 ± 0.8	−1.4 ± 0.7	−7.2 ± 0.7			
Gynoid fat mass (kg)									
AED	6.0 ± 0.1	5.0 ± 0.1	−1.0 ± 0.1	4.8 ± 0.1	−0.2 ± 0.1	−1.2 ± 0.1	0.649	<0.001	0.473
NFD	6.0 ± 0.1	5.1 ± 0.1	−0.9 ± 0.1	4.9 ± 0.1	−0.2 ± 0.1	−1.1 ± 0.1			
Gynoid fat mass (%)									
AED	44.0 ± 0.5	40.1 ± 0.6	−3.9 ± 0.4	39.0 ± 0.6	−1.1 ± 0.4	−5.0 ± 0.4	0.552	<0.001	0.435
NFD	44.0 ± 0.5	40.7 ± 0.5	−3.3 ± 0.4	39.6 ± 0.6	−1.1 ± 0.4	−4.4 ± 0.4			
Total body lean mass (kg)									
AED	48.6 ± 0.6	47.6 ± 0.6	−1.0 ± 0.2	47.7 ± 0.6	0.1 ± 0.2	−0.9 ± 0.2	0.966	<0.001	0.980
NFD	48.6 ± 0.6	47.6 ± 0.6	−1.0 ± 0.2	47.7 ± 0.6	0.1 ± 0.2	−0.9 ± 0.2			
Total body lean mass (%)									
AED	55.5 ± 0.5	59.1 ± 0.5	3.6 ± 0.4	60.6 ± 0.5	1.5 ± 0.4	5.1 ± 0.4	0.991	<0.001	0.617
NFD	55.7 ± 0.5	59.1 ± 0.5	3.4 ± 0.4	60.3 ± 0.5	1.2 ± 0.4	4.6 ± 0.4			
Android lean mass (kg)									
AED	3.4 ± 0.1	3.3 ± 0.1	−0.1 ± 0.03	3.3 ± 0.1	0.03 ± 0.03	−0.1 ± 0.03	0.659	<0.001	0.686
NFD	3.4 ± 0.1	3.4 ± 0.1	−0.1 ± 0.03	3.4 ± 0.1	−0.01 ± 0.03	−0.1 ± 0.03			
Android lean mass (%)									
AED	49.9 ± 0.8	55.5 ± 0.8	5.7 ± 0.7	58.1 ± 0.9	2.6 ± 0.7	8.3 ± 0.7	0.936	<0.001	0.371
NFD	50.2 ± 0.7	55.9 ± 0.8	5.7 ± 0.6	57.2 ± 0.8	1.4 ± 0.7	7.1 ± 0.6			
Gynoid lean mass (kg)									
AED	7.6 ± 0.1	7.4 ± 0.1	−0.2 ± 0.04	7.4 ± 0.1	0.002 ± 0.04	−0.2 ± 0.04	0.725	<0.001	0.892
NFD	7.6 ± 0.1	7.4 ± 0.1	−0.2 ± 0.04	7.4 ± 0.1	0.01 ± 0.04	−0.2 ± 0.04			
Gynoid lean mass (%)									
AED	54.9 ± 0.5	58.6 ± 0.5	3.7 ± 0.4	59.6 ± 0.5	1.0 ± 0.4	4.7 ± 0.4	0.550	<0.001	0.371
NFD	54.9 ± 0.5	58.0 ± 0.5	3.1 ± 0.4	59.0 ± 0.5	1.0 ± 0.4	4.1 ± 0.4			

TABLE 3 (Continued)

	Baseline	3 Months	Change in Phase 1	9 Months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
VAT volume (cm^3)									
AED	1340.9 ± 63.4	975.7 ± 66.8	−365.2 ± 39.4	903.3 ± 68.4	−72.4 ± 45.1	−437.6 ± 42.1	0.325	<0.001	0.926
NFD	1416.5 ± 61.7	1073.1 ± 65.7	−343.5 ± 39.4	984.9 ± 66.1	−88.1 ± 45.0	−431.6 ± 40.2			
VAT mass (g)									
AED	1.3 ± 0.1	920.5 ± 63.0	−344.5 ± 37.2	852.1 ± 64.5	−68.4 ± 42.5	−412.9 ± 39.7	0.325	<0.001	0.926
NFD	1.3 ± 0.1	1012.3 ± 62.0	−324.0 ± 37.2	929.1 ± 62.4	−83.2 ± 42.5	−407.2 ± 37.9			

Note: Values are mean ± SE. Mixed models were used to compare outcomes at the three time points, controlling for age, sex, and BMI. Significance set at $p < 0.05$. Change in Phase 1 (weeks 0–12); change in Phase 2 (weeks 12–26); overall change (weeks 0–36).
Abbreviations: AED, almond-enriched diet; NFD, nut-free diet; VAT, visceral adipose tissue.

tolerability to the weight loss and weight maintenance approach was discussed in regular dietetic counseling appointments. Dietary intake data are provided in Table 2. Weighed 4-day food records with extremes of total energy intakes, <500 or >4000 kcal/d (<2090 or >16,720 kJ), were excluded ($n = 1$) [27]. Energy intake decreased in both groups by the end of Phase 1 ($p < 0.001$), with similar reductions between groups for both intervention phases ($p = 0.623$). Compared with the NFD group, the AED group consumed significantly more total fat (grams, $p = 0.009$; percentage energy, $p < 0.001$), monounsaturated fatty acids (grams, $p < 0.001$; percentage energy, $p < 0.001$), and polyunsaturated fatty acids (grams, $p = 0.007$; percentage energy, $p < 0.001$) over the duration of the trial and significantly less carbohydrate (grams, $p = 0.002$; percentage energy, $p < 0.001$). Compared with the NFD group, the AED group consumed significantly more α-tocopherol and magnesium ($p < 0.001$) over the duration of the trial and significantly more iron at Phase 1 ($p = 0.009$).

Physical activity

There was no change in physical activity (time spent walking and in moderate-vigorous physical activity) captured by the IPAQ over time ($p = 0.067$) or between groups ($p = 0.648$ for group by time interaction, Table 2). Accelerometer-recorded sleep time decreased by the end of Phase 2 ($p = 0.013$), with no differences between groups ($p = 0.649$ for group by time interaction, Table 2). Accelerometer-recorded sedentary time decreased in the NFD compared with the AED group ($p = 0.042$, Table 2), but no differences in light ($p = 0.07$, Table 2) or moderate-vigorous activity ($p = 0.089$, Table 2) were observed between groups.

Weight

During Phase 1, both the AED and the NFD energy-restricted diets resulted in significant reductions in weight ($p < 0.001$) (Table 3). The proportion of participants who lost ≥5% but <10% and ≥10% of initial body weight during Phase 1 was similar between groups (≥5% but <10%: AED 30 of 59 [51%] and NFD 36 of 60 [60%]; ≥10%: AED 17 of 59 [29%] and NFD 14 of 60 [23%]; $p = 0.604$). There was a small amount of additional weight loss in both groups during Phase 2 (−1.2 kg, $p = 0.009$). Overall, there was an average 9.3% ± 0.4% reduction in body weight over the trial ($p < 0.001$), with no significant differences between groups ($p = 0.963$ for group by time interaction, Table 3), and the proportion of participants who lost ≥5% but <10% and ≥10% of initial body weight by end of trial was similar between the groups (≥5% but <10%: AED 20 of 55 [36%] and NFD 26 of 51 [51%]; ≥10%: AED 23 of 55 [42%] and NFD 18 of 51 [35%]; $p = 0.278$). There was no difference in weight loss between participants who underwent the intervention during COVID-19 lockdown periods compared with those whose participation was not interrupted by COVID-19 restrictions ($p = 0.587$).

Body composition (waist circumference and dual-energy x-ray absorptiometry)

Waist circumference decreased significantly during Phase 1 ($p < 0.001$), Phase 2 ($p = 0.012$), and over the duration of the trial ($p < 0.001$), with no differences between the groups ($p = 0.587$ for group by time interaction, Table 3). There was a significant reduction in total fat mass at all time points ($p < 0.001$) and total lean body mass during Phase 1 ($p < 0.001$) and by end of trial ($p < 0.001$), with both groups showing a similar response ($p = 0.810$ and $p = 0.980$, respectively, for group by time interaction, Table 3). Percentage fat mass decreased, and percentage lean mass increased at all time points ($p < 0.001$), with no group by time interactions ($p = 0.646$ and $p = 0.617$, respectively, Table 3). Android and gynoid fat mass decreased at all time points (all $p = 0.05$) with no group by time interactions ($p = 0.746$ and $p = 0.473$, respectively, Table 3). Android and gynoid lean mass decreased during Phase 1 (both $p < 0.001$) and over the duration of the trial (both $p < 0.01$), with no group by time interactions ($p = 0.686$ and $p = 0.892$, respectively, Table 3). Visceral adipose tissue also decreased at all time points (Phase 1 and end of trial $p < 0.001$, Phase 2 $p = 0.038$), with both groups showing a similar response ($p = 0.926$ for group by time interaction, Table 3).

TABLE 4 Changes in cardiometabolic outcomes (blood pressure and lipid profile)

	Baseline	3 months	Change in Phase 1	9 months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
SBP (mm Hg)									
AED	118.3 ± 1.3	114.8 ± 1.4	−3.5 ± 1.1	113.4 ± 1.5	−1.4 ± 1.3	−4.9 ± 1.2	0.770	<0.001	0.650
NFD	119.2 ± 1.3	114.3 ± 1.4	−4.9 ± 1.2	114.4 ± 1.5	0.1 ± 1.3	−4.8 ± 1.2			
DPB (mmHg)									
AED	82.5 ± 1.0	78.7 ± 1.0	−3.8 ± 0.7	78.1 ± 1.1	−0.6 ± 0.8	−4.4 ± 0.8	0.566	<0.001	0.241
NFD	84.2 ± 1.0	78.6 ± 1.0	−5.5 ± 0.7	78.6 ± 1.0	0.0 ± 0.8	−5.6 ± 0.8			
Total cholesterol (mmol/L)									
AED	5.3 ± 0.1	4.8 ± 0.1	−0.5 ± 0.1	4.9 ± 0.1	0.1 ± 0.1	−0.4 ± 0.1	0.673	<0.001	0.262
NFD	5.3 ± 0.1	4.8 ± 0.1	−0.5 ± 0.1	5.1 ± 0.1	0.3 ± 0.1	−0.1 ± 0.1			
HDL (mmol/L)									
AED	1.5 ± 0.04	1.4 ± 0.04	−0.1 ± 0.03	1.6 ± 0.04	0.1 ± 0.03	0.1 ± 0.03	0.395	<0.001	0.222
NFD	1.5 ± 0.04	1.3 ± 0.04	−0.1 ± 0.03	1.5 ± 0.04	0.2 ± 0.03	0.1 ± 0.03			
LDL (mmol/L)									
AED	3.2 ± 0.1	2.9 ± 0.1	−0.3 ± 0.1	2.9 ± 0.1	0.02 ± 0.1	−0.3 ± 0.1	0.272	<0.001	0.257
NFD	3.3 ± 0.1	3.0 ± 0.1	−0.2 ± 0.1	3.1 ± 0.1	0.1 ± 0.1	−0.1 ± 0.1			
VLDL (mmol/L)									
AED	0.6 ± 0.04	0.5 ± 0.04	−0.1 ± 0.03	0.4 ± 0.04	−0.1 ± 0.04	−0.1 ± 0.04	0.530	<0.001	0.507
NFD	0.5 ± 0.04	0.5 ± 0.04	−0.1 ± 0.03	0.5 ± 0.04	−0.01 ± 0.04	−0.1 ± 0.04			
Non-HDL (mmol/L)									
AED	3.8 ± 0.1	3.4 ± 0.1	−0.4 ± 0.1	3.3 ± 0.1	−0.04 ± 0.1	−0.5 ± 0.1	0.429	<0.001	0.133
NFD	3.8 ± 0.1	3.5 ± 0.1	−0.3 ± 0.1	3.6 ± 0.1	0.1 ± 0.1	−0.2 ± 0.1			
Total cholesterol:HDL ratio									
AED	3.8 ± 0.1	3.5 ± 0.1	−0.3 ± 0.1	3.3 ± 0.1	−0.22 ± 0.1	−0.5 ± 0.1	0.284	<0.001	0.116
NFD	3.8 ± 0.1	3.7 ± 0.1	−0.1 ± 0.1	3.5 ± 0.1	−0.17 ± 0.1	−0.3 ± 0.1			
Triglycerides (mmol/L)									
AED	1.4 ± 0.1	1.2 ± 0.1	−0.2 ± 0.1	1.1 ± 0.1	−0.1 ± 0.1	−0.4 ± 0.1	0.468	<0.001	0.461
NFD	1.3 ± 0.1	1.1 ± 0.1	−0.2 ± 0.1	1.1 ± 0.1	0.001 ± 0.1	−0.2 ± 0.1			
ApoA1 (g/L)									
AED	1.4 ± 0.02	1.3 ± 0.03	−0.1 ± 0.02	1.4 ± 0.03	0.1 ± 0.03	0.01 ± 0.02	0.292	<0.001	0.317
NFD	1.4 ± 0.02	1.2 ± 0.03	−0.1 ± 0.02	1.4 ± 0.03	0.1 ± 0.03	0.002 ± 0.02			
ApoB (g/L)									
AED	1.0 ± 0.02	1.0 ± 0.03	−0.1 ± 0.02	0.9 ± 0.03	−0.02 ± 0.02	−0.1 ± 0.02	0.387	<0.001	0.324
NFD	1.0 ± 0.02	1.0 ± 0.03	−0.1 ± 0.02	1.0 ± 0.03	0.02 ± 0.02	−0.04 ± 0.02			
Total TRLP (nmol/L)									
AED	165.4 ± 7.8	142.4 ± 8.4	−23.0 ± 6.6	134.6 ± 8.8	−7.8 ± 7.6	−30.8 ± 7.1	0.847	<0.001	0.050
NFD	154.1 ± 7.8	152.1 ± 8.5	−2.0 ± 6.8	142.1 ± 8.9	−10.0 ± 8.0	−11.9 ± 7.3			
Very large TRLP (nmol/L)									
AED	0.3 ± 0.1	0.2 ± 0.1	−0.04 ± 0.1	0.2 ± 0.1	−0.03 ± 0.2	−0.1 ± 0.2	0.260	0.492	0.873
NFD	0.2 ± 0.1	0.1 ± 0.1	−0.1 ± 0.1	0.04 ± 0.1	−0.03 ± 0.2	−0.1 ± 0.2			
Large TRLP (nmol/L)									
AED	4.7 ± 0.5	2.5 ± 0.6	−2.1 ± 0.6	2.8 ± 0.6	0.3 ± 0.7	−1.9 ± 0.6	0.575	<0.001	0.874
NFD	4.8 ± 0.5	3.1 ± 0.6	−1.7 ± 0.6	3.0 ± 0.6	−0.1 ± 0.7	−1.8 ± 0.6			
Medium TRLP (nmol/L)									
AED	19.8 ± 1.6	15.4 ± 1.8	−4.4 ± 1.6	17.6 ± 1.9	2.2 ± 1.8	−2.2 ± 1.7	0.216	0.002	0.968
NFD	17.0 ± 1.6	13.2 ± 1.8	−3.8 ± 1.6	14.9 ± 1.9	1.7 ± 1.9	−2.2 ± 1.7			

TABLE 4 (Continued)

	Baseline	3 months	Change in Phase 1	9 months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
Small TRLP (nmol/L)									
AED	46.5 ± 4.5	52.0 ± 5.1	5.6 ± 5.4	52.6 ± 5.5	0.6 ± 6.2	6.2 ± 5.8	0.112	0.973	0.273
NFD	45.1 ± 4.5	39.6 ± 5.3	−5.4 ± 5.6	40.8 ± 5.6	1.1 ± 6.5	−4.3 ± 5.9			
Very small TRLP (nmol/L)									
AED	94.2 ± 6.6	72.8 ± 7.4	−21.4 ± 7.1	63.3 ± 8.0	−9.5 ± 8.9	−30.9 ± 7.7	0.178	0.006	0.007
NFD	86.9 ± 6.7	95.1 ± 7.6	8.2 ± 7.4	82.1 ± 8.0	−13.0 ± 8.6	−4.8 ± 7.9			
Total HDLP (μmol/L)									
AED	20.4 ± 0.3	18.9 ± 0.3	−1.5 ± 0.3	20.2 ± 0.4	1.3 ± 0.4	−0.2 ± 0.3	0.236	<0.001	0.209
NFD	20.3 ± 0.3	18.1 ± 0.4	−2.3 ± 0.3	19.7 ± 0.4	1.7 ± 0.4	−0.6 ± 0.3			
Large HDLP (μmol/L)									
AED	2.1 ± 0.2	2.3 ± 0.2	0.2 ± 0.1	2.7 ± 0.2	0.4 ± 0.1	0.6 ± 0.1	0.314	<0.001	0.176
NFD	2.0 ± 0.2	1.9 ± 0.2	−0.1 ± 0.1	2.4 ± 0.2	0.5 ± 0.1	0.4 ± 0.1			
Medium HDLP (μmol/L)									
AED	6.5 ± 0.3	5.7 ± 0.3	−0.8 ± 0.3	5.9 ± 0.3	0.2 ± 0.3	−0.6 ± 0.3	0.998	0.003	0.798
NFD	6.3 ± 0.3	5.8 ± 0.3	−0.5 ± 0.3	5.8 ± 0.3	−0.003 ± 0.3	−0.5 ± 0.3			
Small HDLP (μmol/L)									
AED	11.9 ± 0.3	11.0 ± 0.4	−0.9 ± 0.3	11.7 ± 0.4	0.8 ± 0.4	−0.2 ± 0.4	0.485	<0.001	0.331
NFD	12.0 ± 0.3	10.3 ± 0.4	−1.7 ± 0.3	11.4 ± 0.4	1.1 ± 0.4	−0.5 ± 0.4			
Total LDLP (nmol/L)									
AED	1493.8 ± 41.8	1374.8 ± 44.7	−119.0 ± 32.1	1328.3 ± 46.6	−46.5 ± 36.9	−165.5 ± 34.8	0.300	<0.001	0.235
NFD	1525.4 ± 41.8	1407.1 ± 45.3	−118.3 ± 33.5	1439.1 ± 46.8	32.0 ± 39.0	−86.3 ± 35.8			
Large LDLP (nmol/L)									
AED	432.2 ± 27.2	402.9 ± 29.3	−29.4 ± 22.3	409.3 ± 30.7	6.4 ± 25.7	−23.0 ± 24.2	0.853	0.268	0.091
NFD	404.0 ± 27.2	403.1 ± 29.8	−0.9 ± 23.2	457.4 ± 30.8	54.2 ± 27.1	53.3 ± 24.8			
Medium LDLP (nmol/L)									
AED	612.0 ± 36.7	516.5 ± 41.2	−95.6 ± 40.2	587.6 ± 44.3	71.2 ± 46.2	−24.4 ± 43.4	0.429	0.018	0.045
NFD	663.3 ± 36.8	628.7 ± 42.4	−34.6 ± 41.7	532.9 ± 44.7	−95.9 ± 48.4	−130.5 ± 44.4			
Small LDLP (nmol/L)									
AED	449.5 ± 37.9	456.8 ± 41.9	7.4 ± 37.5	340.2 ± 44.7	−116.6 ± 43.1	−109.3 ± 40.5	0.872	0.071	0.017
NFD	458.1 ± 38.0	374.4 ± 42.9	−83.7 ± 39.0	437.4 ± 45.0	63.0 ± 45.4	−20.7 ± 41.6			
TRL size (nm)									
AED	48.4 ± 0.9	44.6 ± 1.0	−3.8 ± 1.0	44.0 ± 1.1	−0.6 ± 1.2	−4.4 ± 1.1	0.089	<0.001	0.648
NFD	49.6 ± 0.9	47.1 ± 1.1	−2.4 ± 1.1	46.0 ± 1.1	−1.1 ± 1.3	−3.6 ± 1.2			
HDL size (nm)									
AED	9.1 ± 0.04	9.1 ± 0.1	0.1 ± 0.03	9.2 ± 0.1	0.1 ± 0.04	0.1 ± 0.03	0.881	<0.001	0.358
NFD	9.1 ± 0.04	9.1 ± 0.1	0.04 ± 0.03	9.2 ± 0.1	0.1 ± 0.04	0.2 ± 0.03			
LDL size (nm)									
AED	21.2 ± 0.1	21.2 ± 0.1	−0.01 ± 0.04	21.3 ± 0.1	0.1 ± 0.1	0.1 ± 0.1	0.706	0.007	0.252
NFD	21.2 ± 0.1	21.3 ± 0.1	0.1 ± 0.1	21.3 ± 0.1	0.1 ± 0.1	0.2 ± 0.1			

Note: Values are mean ± SE. Mixed models were used to compare outcomes at the three time points, controlling for age, sex, and BMI. Significance set at $p < 0.05$. Blood was unavailable for $n = 5$ in NFD (AED $n = 68$, NFD $n = 67$). Change in Phase 1 (weeks 0–12); change in Phase 2 (weeks 12–26); overall change (weeks 0–36).

Abbreviations: AED, almond-enriched diet; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; DBP, diastolic blood pressure; HDL, high-density lipoprotein; HDLP, high-density lipoprotein particle; LDL, low-density lipoprotein; LDLP, low-density lipoprotein particle; NFD, nut-free diet; SBP, systolic blood pressure; TRL; triglyceride-rich lipoprotein; TRLP, triglyceride-rich lipoprotein particle.

TABLE 5 Changes in cardiometabolic outcomes (glycemic control)

	Baseline	3 Months	Change in Phase 1	9 Months	Change in Phase 2	Overall change	*p* value		
							Group	Time	Group × time
Fasting glucose (mmol/L)									
AED	5.0 ± 0.1	4.9 ± 0.1	−0.1 ± 0.1	4.9 ± 0.1	−0.1 ± 0.1	−0.2 ± 0.1	0.694	<0.001	0.395
NFD	5.2 ± 0.1	4.9 ± 0.1	−0.3 ± 0.1	4.9 ± 0.1	0.02 ± 0.1	−0.3 ± 0.1			
Interstitial glucose (mmol/L)									
AED	5.0 ± 0.1	5.2 ± 0.1	0.2 ± 0.1	5.0 ± 0.1	−0.2 ± 0.1	0.1 ± 0.1	0.409	0.046	0.515
NFD	5.1 ± 0.1	5.2 ± 0.1	0.1 ± 0.1	5.1 ± 0.1	−0.04 ± 0.1	0.1 ± 0.1			
HbA_{1c} (%)									
AED	4.7 ± 0.1	4.9 ± 0.1	0.1 ± 0.1	4.8 ± 0.1	−0.1 ± 0.1	0.02 ± 0.1	0.403	0.024	0.526
NFD	4.8 ± 0.1	4.9 ± 0.1	0.1 ± 0.1	4.9 ± 0.1	−0.02 ± 0.1	0.1 ± 0.1			
Fasting insulin (pmol/L)									
AED	50.3 ± 4.9	46.3 ± 5.4	−4.6 ± 5.2	49.3 ± 5.8	3.0 ± 5.9	−1.6 ± 5.6	0.716	0.036	0.244
NFD	55.8 ± 4.7	42.7 ± 5.8	−13.1 ± 5.6	41.3 ± 6.0	−1.4 ± 6.5	−14.5 ± 5.8			
HOMA2-%B									
AED	90.0 ± 4.7	85.1 ± 5.4	−4.9 ± 5.6	90.2 ± 5.8	5.1 ± 6.8	0.2 ± 5.9	0.754	0.753	0.718
NFD	88.2 ± 4.6	87.1 ± 5.9	−1.2 ± 6.0	84.6 ± 6.0	−2.5 ± 6.9	−3.7 ± 6.1			
HOMA2-%S									
AED	125.6 ± 6.4	150.4 ± 7.3	24.8 ± 7.2	144.3 ± 7.9	−6.1 ± 8.3	18.6 ± 7.9	0.462	<0.001	0.996
NFD	131.0 ± 6.1	156.2 ± 7.8	25.2 ± 7.7	150.7 ± 8.0	−5.5 ± 8.9	19.7 ± 8.0			
HOMA2-IR									
AED	1.0 ± 0.1	0.9 ± 0.1	−0.1 ± 0.1	1.0 ± 0.1	0.1 ± 0.1	−0.01 ± 0.1	0.501	<0.001	0.716
NFD	1.1 ± 0.1	0.8 ± 0.1	−0.3 ± 0.1	0.8 ± 0.1	−0.01 ± 0.1	−0.3 ± 0.1			

Note: Values are mean ± SE. Mixed models were used to compare outcomes at the three time points, controlling for age, sex, and BMI. Significance set at $p < 0.05$. Fasting glucose AED $n = 62$, NFD $n = 68$; interstitial glucose AED $n = 67$, NFD $n = 70$; HbA_{1c} AED $n = 68$, NFD $n = 71$; fasting insulin AED $n = 61$, NFD $n = 66$; HOMA AED $n = 60$, NFD $n = 65$. Change in Phase 1 (weeks 0–12); change in Phase 2 (weeks 12–26); overall change (weeks 0–36). Abbreviations: AED, almond-enriched diet; HbA_{1c}, hemoglobin A_{1c}; HOMA2-%B, homeostasis model assessment index 2 of pancreatic β cell function; HOMA2-%S, homeostasis model assessment index 2 of insulin sensitivity; HOMA2-IR, homeostasis model assessment index 2 of insulin resistance; NFD, nut-free diet.

Cardiometabolic outcomes

Blood pressure

SBP and DBP fell during Phase 1 (both $p < 0.001$) and over the duration of the trial (both $p < 0.001$), with no difference between groups for either variable ($p = 0.650$, $p = 0.241$, respectively, Table 4).

Blood lipids

There was a reduction in total cholesterol in Phase 1 ($p < 0.001$), and although there was a small increase during Phase 2 ($p = 0.027$), there was a reduction by end of trial ($p = 0.001$), with no differences between groups ($p = 0.262$ for group by time interaction, Table 4). Triglycerides, low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and non-high-density lipoprotein (HDL) decreased during Phase 1 ($p < 0.001$; VLDL, $p = 0.002$) and over the course of the trial ($p < 0.001$; LDL, $p = 0.002$). There were no group by time interactions ($p = 0.461$, $p = 0.257$, $p = 0.507$, $p = 0.133$, respectively, Table 4). For both groups, HDL decreased during Phase 1 ($p < 0.001$), and it increased during Phase 2 ($p < 0.001$) and from baseline to end of trial ($p = 0.011$). Group by time interactions were not significant ($p = 0.222$, Table 4). Apolipoprotein A1 decreased during Phase 1 ($p < 0.001$) and increased during Phase 2 ($p < 0.001$), but over the course of the trial, there was no change from baseline values ($p > 0.999$) and no difference between groups ($p = 0.317$ for group by time interaction, Table 4). There was a reduction in apolipoprotein B in Phase 1 ($p < 0.001$) and at end of trial ($p < 0.001$), with both groups showing a similar response ($p = 0.324$ for group by time interaction, Table 4).

Particle concentrations were collected for triglyceride-rich lipoproteins (TRL), LDL, and HDL in diameter size subclasses of very large (TRL only), large, medium, small, and very small (TRL only). The AED group had an overall greater reduction in very small TRL particles (TRL-P) ($p = 0.007$) and small LDL particles (LDL-P) ($p = 0.017$) when compared with the NFD group (Table 4). However, the NFD group had a greater reduction in medium LDL-P ($p = 0.045$) than the AED group (Table 4).

Glycemic control and metabolic syndrome score

Reductions in fasting glucose occurred in Phase 1 ($p = 0.007$) and over the duration of the trial ($p = 0.003$), with no difference between groups ($p = 0.395$ for group by time interaction, Table 5). Hemoglobin A_{1c} and

flash interstitial glucose increased during Phase 1 ($p = 0.020$ and $p = 0.040$, respectively), but changes were not significant over the course of the trial (both $p > 0.999$), and there were no group by time interactions ($p = 0.526$, $p = 0.515$, respectively, Table 5). Insulin decreased over the duration of the trial ($p = 0.036$), and there were no group by time interactions ($p = 0.244$, Table 5). HOMA2-%S increased and HOMR2-IR decreased in Phase 1 (both $p < 0.001$) and over the duration of the trial compared with baseline ($p = 0.001$ and $p = 0.002$, respectively), with no differences between groups ($p = 0.927$, $p = 0.918$, respectively, for group by time interaction, Table 5). No significant changes were noted for HOMA2-%B by time ($p = 0.753$) or by group by time ($p = 0.718$, Table 5). Metabolic syndrome score was calculated [25], and it decreased in Phase 1 ($p = 0.021$) and over the duration of the trial ($p < 0.001$). There were no differences between groups at any time point (Phase 1, $p = 0.279$; Phase 2, $p = 0.918$; end of trial, $p = 0.778$).

DISCUSSION

In this study, both the AED and NFD groups demonstrated comparable efficacy in achieving weight loss and improving cardiometabolic risk factors, rejecting the predicted hypothesis. The comparable improvements observed in both groups can be attributed to two main factors: the isocaloric nature of the diets and the equal level of dietetic support provided. Most improvements occurred during the weight loss phase and they were maintained during the weight maintenance phase. At the end of weight loss, most participants (82%) had lost ≥5% of their body weight, with a mean overall weight loss of 9.3% at the end of the trial (89% fat mass and 11% lean mass) in both groups. This clinically significant weight loss is likely responsible for the improvements in cardiometabolic risk factors seen in both groups [28]. Moderate weight loss (5%–10%) is associated with reductions in blood pressure and triglycerides, improved glycemic control, and increased HDL [29]. A 5 mm Hg reduction in SBP reduces risk of major cardiovascular events by 10% [30], and in this study, there was a 4.9 mm Hg reduction in both groups.

Of note are the differential effects of the AED on lipoprotein subfraction concentrations, specifically the reductions seen in very small TRL-P and small LDL-P compared with the NFD. Lipoprotein subfractions provide a more sensitive and specific measurement of lipid metabolism and cardiovascular risk than measuring total lipoprotein levels alone. Very small TRL-P are independently associated with the presence, severity, and progression of atherosclerosis, and small LDL-P are highly atherogenic and strongly related to CVD risk [31, 32]. Although medium LDL-P decreased in the AED group, there were significantly greater reductions in the NFD group. There are mixed findings relating to medium LDL-P and cardiovascular outcomes, with some studies reporting links with CVD risk and other studies finding statistically nonsignificant associations [33]. However, it is established that small LDL-P confer the greatest atherogenic risk due to ease of penetration into the subendothelial space and greater susceptibility to oxidation [34–36].

The specific effects of nuts within weight loss interventions have been examined in only a few previously published randomized controlled trials, with mixed results. Studies have varied considerably in length (3 months [37, 38], 6 months [39], or 18 months [40]), have incorporated different amounts of nuts (50 to 84 g), and have implemented different energy restriction targets (either reducing energy intake by 500 kcal [37] or 1000 kcal [38] or prescribing 1000 kcal/d [achieved via liquid diet] [39] or 1200–1800 kcal/d [40]). Studies of longer duration and implementing a greater degree of restriction have achieved greater weight loss overall, with significantly better outcomes for the nut-enriched diets observed in two studies [38, 39]. Interestingly, the greater degree of weight loss observed in the studies conducted by Wien et al. and Abazarfard et al. did not consistently result in better cardiometabolic outcomes. Abazarfard et al. reported significant reductions in total cholesterol, triglycerides, total cholesterol: high density lipoporotein cholesterol ratio, fasting glucose, and DBP in the AED group (50 g) compared with the NFD group [38], whereas Wien et al. reported no between-group difference for cardiometabolic outcomes except for a greater reduction in SBP in the almond low-calorie diet compared to the carboydrate low-calorie diet [39]. In contrast, Foster et al. reported that the inclusion of almonds (56 g) was less effective at achieving weight loss over 6 months compared with an NFD, but after 18 months, this difference no longer remained [40]. Despite less weight loss in the AED group at 6 months, the AED group experienced greater improvements in total cholesterol and triglycerides compared with the NFD group at 6 months [40]. These cardiometabolic improvements were similar between groups at 18 months [40]. Similarly, Dhillon et al. saw no change in cardiometabolic risk measures other than SBP in both groups; this was likely due to the small weight loss achieved by both groups in response to the relatively modest energy restriction [37]. The energy restriction used in Abazarfard et al. and Foster et al. was similar to that used in the present study; however, the present study saw greater weight loss [38, 40]. This may have been due to the different level of dietary support provided. Furthermore, the dose of almonds in Wien et al. (84 g), Foster et al. (56 g), and Abazarfard et al. (50 g) was larger than the dose of almonds used in the present study (30–50 g) (Abazarfard et al. used a fixed amount of almonds, whereas in the present study the amount varied), which may explain the significant between-group differences in cardiometabolic risk measures seen in these studies, which were not observed in the present study [38–40].

In the present study, clinically significant and very similar weight loss in both groups may have masked any beneficial effect associated with almond consumption. Several mechanisms could potentially explain why some studies have seen greater weight loss with almond consumption compared with control [8]. Almonds are high in fiber, protein, and unsaturated fats. These properties promote satiety and increase resting energy expenditure [8, 10]. In addition, not all energy from almonds is available for digestion due to the poor bioavailability of fats [41]. However, we provided control foods that ensured that the overall nutrient profiles of both treatment groups were similar and, as such, that changes in cardiometabolic parameters were not the result of a deterioration in nutrient profile.

Evidence suggests that there are cardiovascular benefits of nuts without weight loss, as seen in the Prevention with Mediterranean Diet study

in which subjects were assigned to a Mediterranean diet supplemented with nuts and had low incidence of major cardiovascular events within the 4.8-year follow-up period compared with those on the low-fat diet [42]. Almonds are also rich in α-tocopherol, an antioxidant that is associated with a lower risk of CVD [43]. We saw macronutrient differences between groups, specifically higher monounsaturated fatty acid and polyunsaturated fatty acid intake in the AED group compared with the NFD group, as well as higher α-tocopherol. Almonds also contain high amounts of protein that is rich in arginine, a known precursor of nitric oxide, which inhibits platelet adhesion and aggregation [44, 45]. These properties may help to explain the statistically significant changes in the highly atherogenic very small TRL-P and small LDL-P following the AED, which may lead to improved cardiometabolic health in the longer term [42].

A strength of this study is that participants were able to maintain their weight loss for 6 months with reduced and more realistic (real-world applicable) dietary support. There are some limitations that should be considered when interpreting the results. Firstly, our sample size may not have provided the statistical power to detect smaller between-group differences. Additionally, the population group was free from chronic disease, so cardiometabolic parameters generally fell within recommended ranges. Future studies may like to examine effects in participants with metabolic syndrome, type 2 diabetes, or CVD to allow for greater impact of diet on cardiometabolic parameters.

These findings provide further evidence that an energy-restricted AED can promote weight loss and maintenance comparable to an energy-restricted NFD and that both diets support cardiometabolic health. Replacing typical snacks with almonds can have a meaningful impact on lipoprotein subfractions, shifting to a less atherogenic pattern, and as such, health professionals can recommend almonds as part of a balanced weight loss diet. Future studies should consider the dose of almonds and testing in populations with elevated cardiometabolic risk factors, such as populations with metabolic syndrome. The satiating effects of almonds need further investigation as almonds may assist with hunger management, and this may explain why some intervention studies have seen differences in weight loss between groups.O

AUTHOR CONTRIBUTIONS

Alison M. Coates was the principal investigator for the study. Alison M. Coates, Jonathan D. Buckley, Alison M. Hill, Sze-Yen Tan, and Geraint B. Rogers were co-investigators on the grant application and, as such, were involved with the original design. Sharayah Carter, Alison M. Hill, Catherine Yandell, Jonathan D. Buckley, and Alison M. Coates were involved with the study coordination and were responsible for the day-to-day running of the trial, participant recruitment, and sample collection. Lauren C. Mead and Hoi Y. Wong contributed to the data collection. All authors contributed to the conceptualization, data curation, analysis, statistical interpretation, and writing and preparation of this manuscript for publication. All authors have read and approved the final manuscript.

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CONFLICT OF INTEREST STATEMENT

AMC has consulted for Nuts for Life (an initiative of the Australian Tree Nut Industry). S-YT has previously been involved in studies funded by the Californian Walnut Commission. AMC, JDB, AMH, and S-YT have previously been involved in studies funded by the International Nut and Dried Fruit Council. The other authors declared no conflict of interest.

CLINICAL TRIAL REGISTRATION

Australian New Zealand Clinical Trials Registry (ANZCTR) reference number ACTRN12618001861246.

DATA AVAILABILITY STATEMENT

Data will be made available on request.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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