Quinoa Seed Lowers Serum Triglycerides in Overweight and Obese Subjects: A Dose-Response Randomized Controlled Clinical Trial

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Abstract

Background: Quinoa (*Chenopodium quinoa*) is a pseudo-cereal originally cultivated in the Andean region. The popularity of its seeds has increased in recent years due to the claims of health benefits and superfood qualities. Studies to date on the health benefits of quinoa have been restricted to animal models, and the results provide weak to moderate evidence to support improved plasma lipid profiles. Clinical trials in humans to examine the claims of health benefits of quinoa are limited to a few prospective studies and one randomized trial carried out in postmenopausal women. To our knowledge, no studies have been conducted in the general population.

Objective: The objective of this randomized clinical trial was to investigate the effect of different quinoa doses (25 and 50 g/d) on body composition, serum lipids and hormones, and nutrient intakes in overweight and obese humans.

Methods: This was a dose-response randomized, controlled, single-blind trial with a parallel design (1 control and 2 treatment groups) that compared the effect of 25 and 50 g quinoa/d in 50 overweight and obese participants over a 12-wk intervention period.

Results: Body composition, nutrient intake, and total, LDL, and HDL cholesterol were not significantly altered by quinoa consumption ($P > 0.05$). Mean serum triglyceride (TG) concentration was reduced significantly in the 50-g quinoa group from 1.14 to 0.72 mmol/L at 12 wk ($P < 0.05$). The prevalence of metabolic syndrome (MetS) was also reduced in this group by 70%. No significant changes in TGs were observed in the control and 25-g quinoa groups. The prevalence of MetS was reduced by 40% (from $n = 7$ at baseline to $n = 4$ at 12 wk) in the 25-g group.

Conclusions: The consumption of 50 g quinoa/d lowers serum TGs in overweight and obese participants and reduces the prevalence of MetS. This trial was registered at clinicaltrials.gov as UTN U1111-1175-470. *Curr Dev Nutr* 2017;1:e001321.

Introduction

Quinoa (*Chenopodium quinoa*) is a pseudo-cereal originally cultivated in the Andean region between Bolivia and Peru. In recent years, the popularity of quinoa seeds has increased due to claims of health benefits and superfood properties. Research has shown that quinoa seeds have an improved macronutrient profile (1), including gluten-free characteristics, a particularly beneficial essential amino acid ratio (2), and a superior phytochemical composition (3) compared with other cereals and grains.

Despite quinoa's composition and properties, scientific evidence supporting health claims such as weight loss, antidiabetic effects, and appetite suppression in in vivo models is limited and the evidence is restricted to a few animal studies. Among this body of

evidence, it can be observed that a possible health benefit of quinoa consumption may be linked to a potential lipid-lowering effect (4–6). Human clinical studies have been limited to prospective studies on the effect of cereal bars containing quinoa on cardiovascular disease (CVD) markers and immunologic responses in a cohort of celiac patients after the consumption of quinoa flakes (7, 8). Both studies showed changes in total cholesterol and in TGs. In addition, LDL-cholesterol changes were reported in the study that used cereal bars (8). Furthermore, a randomized controlled trial was carried out in postmenopausal women analyzing the effect of quinoa consumption on lipid profiles and oxidative stress markers (9). The results of this trial were in line with previous data that reported favorable changes in TGs and total and LDL cholesterol.

Obesity has reached epidemic proportions according to the WHO in 2016, whereby ~1.9 billion adults were overweight and 600 million of these were considered obese (10). A cluster of metabolic factors, including abdominal fat measured as waist circumference, reduced HDL cholesterol, increased TGs, augmented fasting plasma glucose, and elevated blood pressure, have been termed the metabolic syndrome (MetS) (11, 12); and MetS has been linked to an increased risk of chronic disease states such as obesity, coronary artery disease, and type 2 diabetes. MetS has a substantive impact on the economy reflected in the uses of health care resources and the decrease in productivity. In the United States, the estimated annual cost for 2011 was $320.1 billion for CVD and stroke, with $195.6 billion associated with direct costs and $124.5 billion linked to loss of productivity due to premature deaths (13). Addressing the components of the MetS will lead to an increase in health and a potential reduction in costs to the economy.

To our knowledge, no previous studies have been conducted that analyzed the effect of quinoa consumption in people who are overweight and obese with regard to MetS risk factors and health benefits. The aim of this research was to investigate a dose-response effect of quinoa seed consumption in a randomized controlled trial design on body composition, circulating lipids, hormones, and nutrient intakes in overweight and obese humans.

Methods

Design
A dose-response randomized, controlled, single-blind trial that used a parallel design including 1 control and 2 treatment groups was undertaken. Researchers who assessed study outcomes were blinded from participant interventions and codes.

Participants
Overweight and obese adults were recruited via advertisements at La Trobe University by using flyers, social media, and e-mails after ethical approval was obtained. Participants were included if they were aged between 18 and 65 y and had a BMI (in kg/m^2) >25. Exclusion criteria were pregnancy, diagnosis of diabetes or heart disease, and current use of lipid-lowering medication. Participants gave written consent before the commencement of the study after being briefed about study procedures and expectations.

Food samples
White organic quinoa seeds were purchased from an Australia-based company (Quilla Foods Pty.) dedicated to the importation of Bolivian quinoa. Quinoa bags were carefully weighed and packed into transparent sachets of either 25 or 50 g and packaged in a box of 42 sachets, which constituted a 6-wk supply to each participant in the treatment groups.

Randomization
Participants were randomly allocated into 1 of 3 treatment groups (control and 25 and 50 g quinoa seeds/d). This randomization was carried out following a block randomization protocol stratified by sex with the use of a computer-generated numbering program retrieved from the website random.org. Allocation sequences and assignment of participant interventions were carried out by an external researcher before the appointments.

Study protocol
Participants attended appointments at La Trobe University's Bundoora campus at baseline and at 6 and 12 wk of the intervention period. Participants allocated to the 25- and 50-g quinoa groups were given a quinoa supply for 6 wk at baseline and again at their 6-wk appointment and were advised to consume 1 quinoa sachet/d. Participants were instructed on cooking methods for quinoa consumption, and recipes to incorporate quinoa were given to participants who requested them. Participants were not instructed on how and when to consume the quinoa and were advised to retain their normal lifestyle and diet throughout the study. Participants were given a calendar to self-report quinoa consumption for compliance analysis. Those in the control arm were advised to continue their normal routine and to avoid the consumption of quinoa meals during the study period.

Study outcomes
Primary outcomes included lipid profile, and secondary outcomes such as body composition and dietary intake were measured at baseline and at 6 and 12 wk. Hormone concentrations were also measured at baseline and at 12 wk.

Anthropometric measures
Anthropometric measures such as body weight and height were recorded by using a stadiometer (Wedderburn) and waist-to-hip ratio were assessed by using a measuring tape according to WHO criteria (14). Body composition was analyzed by using a DXA scan (Discovery QDR series; Hologic, Inc.) calibrated daily before measurement.

Serum lipids, hormones, and glucose
Fasting blood samples were collected in an 8.5-mL serum separation tube. After 30 min of incubation time at room temperature, blood was centrifuged at 1300 × g for 10 min at room temperature, and the separated serum was stored at −80°C until analysis. Lipid profile (total, HDL, and LDL cholesterol and TGs) and glucose concentrations were measured by using enzymatic assays in a chemistry analyzer (Indiko; Thermo Scientific) as per the manufacturer's protocol. Adiponectin, leptin, insulin, and C-peptide were determined by multiplex ELISA on a Bio-Plex 200 system (Bio-Rad Laboratories Pty. Ltd.)

following the manufacturer's instructions. Acquired fluorescence data were analyzed by the Bio-Plex Manager software version 6.1 (Bio-Rad Laboratories Pty. Ltd.).

Dietary intake

Participants recorded their food intake with the use of a 3-d food diary. They were provided with templates and were advised to record intake for 3 d after the first appointment and for 3 d before the second (6 wk) and final (12 wk) appointments. Data were analyzed by using FoodWorks version 8 (Xyris).

Compliance

Participants were advised to complete a compliance checklist calendar every time they consumed a quinoa sachet. In addition, leftover sachets were returned at the following appointment.

Prevalence of MetS

Analysis of abnormalities, including those related to MetS, were reported by using the criteria stated in the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults [Adult Treatment Panel III (ATP III)] (12), including waist circumference (men: >102 cm; women: >88 cm), circulating TGs (≥1.7 mmol/L), circulating HDL cholesterol (men: <1.04 mmol/L; women: <1.3 mmol/L), and fasting glucose (≥5.6 mmol/L).

Statistical analysis

Data were analyzed for normality by using Kolmogorov-Smirnov test. Baseline characteristics were assessed by 1-factor ANOVA for parametric data and Kruskal-Wallis test for nonparametric data. Results are presented as means ± SEMs, with the exception of lipid profile and hormones, which are reported as medians (IQRs). Treatment differences were adjusted for baseline values and analyzed by using a linear mixed model with fixed factors (baseline of the variable, treatment and time, and interaction between treatment and time) and a subject-specific intercept as a random effect. Nonnormally distributed variables, such as adiponectin, glucose, HDL cholesterol, and TGs were log-transformed in order to fit the model. When significant interactions were observed, pairwise comparisons with Bonferroni were carried out. Subgroup analysis was performed by a linear mixed model with the above considerations. The prevalence of MetS was analyzed by using 1-factor ANOVA for baseline values and repeated-measures ANOVA for between- and within-group comparisons. All data were analyzed by using SPSS, Inc., software (version 24.0). The level of significance was chosen as 5%.

The study was approved by the La Trobe University Human Ethics Committee (HEC 14- 065) and registered with the Australian New Zealand Clinical Trials Registry (ANZCTR http://www.anzctr.org.au/) as UTN U1111-1175-470.

Results

Participants

One hundred fifty-one participants expressed interest in the study. After screening, 131 met the inclusion criteria and were deemed eligible to participate. Sixty-seven eligible participants declined involvement, and 64 participants were randomly assigned. Nine participants discontinued the study after the baseline appointment (2 in the control group, 3 in the 25-g group, and 4 in the 50-g group) and 5 after the 6-wk appointment (1 in the control group, 3 in the 25-g group, and 1 in the 50-g group). Fifty participants successfully completed the 12-wk intervention period. **Figure 1** shows an overview of the recruitment randomization process and further analysis of the data. Seventy-one percent of participants were women (*n* = 53), and the study group was aged between 20 and 64 y (mean ± SEM: 37.96 ± 1.43 y). According to the WHO criteria for obesity classification (10), 42% (*n* = 31) were considered overweight (BMI >25), 28% (*n* = 21) were obese type I (BMI between 30 and 34.9), 15% (*n* = 11) were obese type II (BMI between 35 and 39.9), and 15% (*n* = 11) were obese type III (BMI ≥40). There were no significant differences between groups at baseline (control and interventions) (**Table 1**). Participants were randomly assigned from February 2015 to May 2016, and the follow-up period ended August 2016.

Effect of quinoa on anthropometric measures and body composition

As shown in **Table 2**, no significant differences were observed for body-composition measurements after the 6- and 12-wk intervention time periods between and within treatment groups. In the control group, body weight varied from a 3.6% decrease (−2.92 kg) at 6 wk to a 4.1% increase (3.19 kg) at 12 wk. In both intervention groups, changes in body weight were reported as a 0.5% increase (0.43 kg) at 6 wk, whereas a 1.5% (−1.32 kg) and a 1.7% (−1.43 kg) decrease at 12 wk in the 25- and 50-g groups, respectively, was observed. There were no differences in the percentage of body fat and total lean tissue. There was a decrease in waist circumference of 2.8 cm in the control group at 6 wk, but an increase of 3.3 cm at 12 wk. In the 25-g group, waist circumference increased by 0.6 cm at 6 wk and decreased by 1 cm at 12 wk, whereas in the 50-g group there was a reduction of 0.2 and 1.3 cm at 6 and 12 wk, respectively.

Serum lipids, hormones, and glucose

The interaction effects between lipids, lipoproteins, and fasting glucose with quinoa intake is reported in **Table 3**. No significant effect was observed for total, HDL, and LDL cholesterol and fasting glucose between and within groups at 6 and 12 wk of quinoa consumption. However, a significant difference was noted for TGs in the between-group analysis. A pairwise comparison adjusted with a Bonferroni correction showed that TGs were significantly lower in the 50-g group compared with the control group (0.72 and 1.25 mmol/L, respectively; *P* = 0.022). Furthermore, within-group comparison showed that the 50-g group had significantly lower TG concentrations at the 12-wk time point compared with baseline (1.14 and 0.72 mmol/L at baseline and 12 wk, respectively; *P* = 0.001). No significant difference was observed at 6 wk compared with baseline. These results indicate that there is a dose-response effect with daily consumption of 50 g quinoa, which reduced serum TGs by ∼28.1% after 6 wk of a dietary intervention and by 36.8% after 12 wk. TG changes in the 25-g group were not significant at either
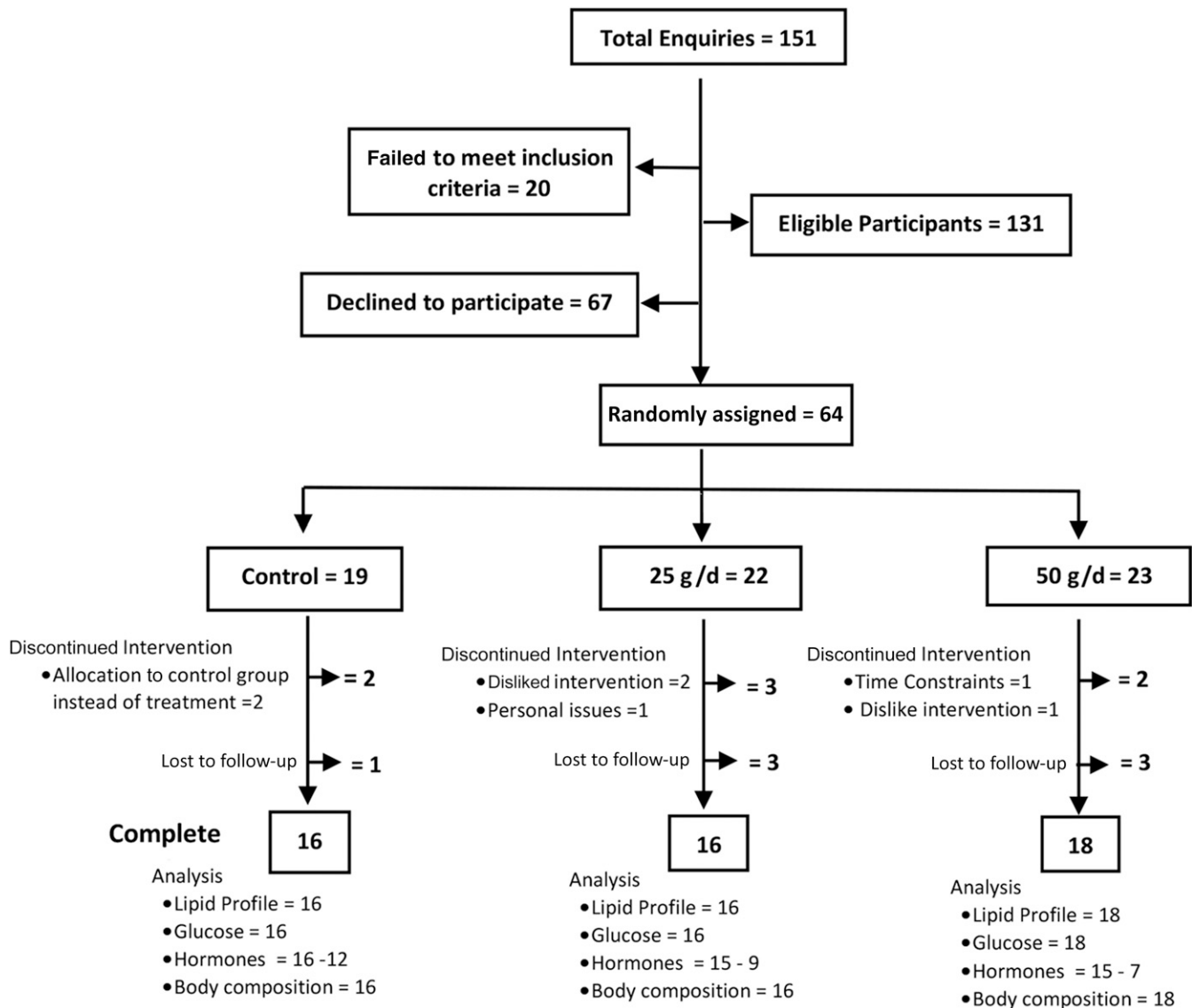
FIGURE 1 Participant flowchart: CONSORT guidelines. CONSORT, Consolidated Standards of Reporting Trials.

6 or 12 wk, although concentrations showed a slight reduction when compared with baseline values (1.36, 1.33, and 1.27 mmol/L at baseline, 6 wk, and 12 wk, respectively).

Further analysis showed that 15.8% of all of the participants in the study had TGs exceeding optimum concentrations of 1.7 mmol/L, the cutoff according to the ATP III MetS criteria (12). Analysis of the effect of quinoa on normo- and hypertriglyceridemic groups showed a significant change (P = 0.048) in participants with normal TGs who consumed 50 g quinoa/d compared with controls at 12 wk (**Figure 2**). Reductions were also observed within the 50-g group from 0.86 mmol/L at baseline to 0.76 mmol/L at 6 wk and 0.70 mmol/L at 12 wk, although these were nonsignificant (P = 0.336). This represents a reduction of 12.7% and 18.4% at 6 and 12 wk, respectively. No significant differences were observed in TGs in participants who consumed 25 g quinoa/d. In the hypertriglyceridemic group, a significant decrease was observed at 12 wk (P = 0.001) in participants

who consumed 50 g quinoa/d. TGs were reduced by 30.4% at 6 wk (from 2.83 mmol/L at baseline to 1.97 mmol/L) and by 58% at 12 wk (from 2.83 mmol/L at baseline to 1.18 mmol/L). Participants who consumed 25 g quinoa showed a reduction of 18.5% at 6 wk (2.7 mmol/L at baseline to 2.20 mmol/L) and 23.7% at 12 wk (from 2.7 mmol/L at baseline to 2.06 mmol/L). No significant difference was observed in the between-group comparison. Of note, TG concentrations were normalized to within the healthy range (<1.7 mmol/L) in the hypertriglyceridemic group who consumed 50 g quinoa/d.

Circulating hormones were not significantly affected by quinoa consumption. Adiponectin increased in the control group by 18% and by 21% and 6% in the 25-g and 50-g quinoa groups, respectively, during the intervention period. Leptin and C-peptide decreased by 21% in the control group and increased by 11% in the 25-g group. In addition, leptin decreased by 3%, whereas C-peptide increased in the 50-g group. Insulin

TABLE 1 Baseline characteristics of overweight and obese participants in the control and treatment groups

| | | Treatment group | | |
Characteristics	Control group	25 g quinoa/d	50 g quinoa/d	P[1]
Participants, n	16	16	18	
Sex (M/F), n (%)	5 (31.52)/11 (68.75)	7 (43.75)/9 (56.25)	4 (21.05)/14 (78.94)	
Age,[2] y	38 (22–59)	35 (21–61)	40 (21–64)	0.447
BMI,[2] kg/m²	29.91 (25.3–41.2)	32.84 (25.9–46.1)	31.49 (25–39.5)	0.287
Body composition				
Body weight, kg	81.28 ± 4.03[3]	89.91 ± 4.31	85.85 ± 2.80	0.286
Body fat, %	40.21 ± 2.0	41.41 ± 1.73	41.25 ± 1.72	0.894
Total lean tissue, %	61.22 ± 2.36	59.79 ± 2.14	58.75 ± 1.72	0.862
Bone mineral density, g/cm²	2.42 ± 0.12	2.46 ± 0.10	2.48 ± 0.09	0.927
Waist circumference, cm	102.97 ± 3.19	109.32 ± 3.64	106.63 ± 2.07	0.268
Lipid profile and glucose,[4] mmol/L				
Total cholesterol	4.69 (3.49–5.38)	4.45 (3.32–5.49)	4.36 (3.51–5.58)	0.729
LDL cholesterol	3.93 (2.90–4.45)	3.90 (3.10–4.69)	3.25 (2.87–3.53)	0.221
HDL cholesterol	1.20 (0.80–1.43)	1.04 (0.89–1.23)	1.28 (0.79–1.56)	0.749
TGs	1.07 (0.84–1.21)	1.36 (0.90–2.38)	1.14 (0.611–1.73)	0.345
Fasting glucose	4.71 (4.58–5.07)	4.85 (4.58–6.63)	4.65 (4.19–5.09)	0.481
Hormones[4]				
Adiponectin, μg/mL	15.33 (9.74–27.10)	10.73 (4.83–15.27)	13.91 (10.28–21.50)	0.176
Leptin, ng/mL	11.08 (3.85–17.18)	10.86 (5.52–15.82)	6.64 (3.45–20.4)	0.947
C-peptide, ng/mL	1.00 (0.71–1.12)	1.04 (0.88–1.72)	0.99 (0.84–1.43)	0.398
Insulin, mIU/L	11.31 (4.97–16.69)	11.60 (3.21–15.89)	11.46 (7.65–19.34)	0.551
Nutrient intake				
Energy, kJ	8066.71 ± 691.32	7908.55 ± 774.10	7618.25 ± 595.96	0.885
Protein, g	87.68 ± 11.14	85.37 ± 13.38	84.53 ± 6.13	0.972
Total fat, g	75.86 ± 7.54	73.65 ± 8.07	79.64 ± 7.38	0.851
Carbohydrates, g	201.96 ± 16.92	193.52 ± 14.59	167.23 ± 17.25	0.296
Dietary fiber, g	23.60 ± 2.42	24.20 ± 3.85	22.52 ± 2.26	0.908
Protein, % of energy	18.30 ± 1.32	17.57 ± 1.27	19.51 ± 1.27	0.559
Total fat, % of energy	34.80 ± 1.72	34.40 ± 1.51	38.54 ± 1.81	0.171
Carbohydrates, % of energy	41.64 ± 1.21	42.07 ± 2.15	39.64 ± 1.53	0.610
Fiber, % of energy	2.46 ± 0.27	2.43 ± 0.20	2.53 ± 0.22	0.948
Other nutrients, % of energy	0.94 ± 0.16	1.23 ± 0.20	1.04 ± 0.17	0.531

[1] Derived by using 1-factor ANOVA for body composition and nutrient intake and Kruskal-Wallis test for lipid profile, glucose, and hormones.
[2] Values are means (ranges).
[3] Values are means ± SEMs.
[4] Values are medians (IQRs).

concentrations remained stable in the control and 25-g groups and showed a decrease in the 50-g group (14%). Results are reported in Table 3.

Prevalence of MetS

The prevalence of MetS as defined by the ATP III criteria in the treatment groups is reported in **Figure 3**. According to the criteria for MetS, 24% (n = 12) of total participants had ≥3 risk factors, including high waist circumference (men >102 cm and women >88 cm), reduced HDL cholesterol (men <1.04 mmol/L and women <1.3 mmol/L), high TGs (≥1.7 mmol/L), and higher fasting blood glucose (≥5.6 mmol/L) at baseline. No changes in prevalence were observed at 6 wk for the control and 50-g groups, whereas the 25-g group showed a reduction of 14% in prevalence at this time point. At the completion of the study, there was a decrease in the occurrence of MetS in the 25-g group of 41% (n = 3 compared with n = 7 at baseline) and in the 50-g group of 70% (n = 2 compared with n = 3 at baseline). On the contrary, the

TABLE 2 Effect of quinoa on body composition in overweight and obese participants at 6 and 12 wk of intervention[1]

| Body-composition | Control | | 25 g/d | | 50 g/d | | P |
characteristics	6 wk	12 wk	6 wk	12 wk	6 wk	12 wk	(treatment)[2]
BMI, kg/m²	28.45 ± 0.72	29.85 ± 1.19	33.45 ± 1.38	32.81 ± 1.43	30.86 ± 1.11	30.20 ± 1.16	0.968
Body weight, kg	78.43 ± 2.80	81.62 ± 4.31	90.34 ± 4.19	89.02 ± 4.24	86.05 ± 2.80	84.59 ± 2.83	0.775
Body fat, %	39.47 ± 2.0	40.29 ± 1.99	41.28 ± 1.73	41.41 ± 1.93	40.96 ± 1.71	40.25 ± 1.87	0.380
Total lean tissue, %	61.86 ± 2.29	61.09 ± 2.31	59.85 ± 1.98	59.74 ± 2.15	59.04 ± 1.71	59.75 ± 1.86	0.341
Bone mineral density, g/cm²	1.87 ± 0.11	2.42 ± 0.13	1.95 ± 0.09	2.44 ± 0.11	1.84 ± 0.08	2.49 ± 0.10	0.477
Waist circumference, cm	100.15 ± 3.62	103.26 ± 3.62	109.88 ± 3.61	108.86 ± 3.54	106.34 ± 2.20	105.03 ± 2.07	0.265

[1] Values are means ± SEMs.
[2] Indicates the main effect of treatment observed between groups (baseline adjusted on the basis of a linear mixed model).

TABLE 3 Effect of quinoa intake on fasting biochemical measures in overweight and obese participants in the control and treatment groups at 6 and 12 wk of intervention[1]

Characteristics	Control group		25-g/d group		50-g/d group		P (treatment)[2]
	6 wk	12 wk	6 wk	12 wk	6 wk	12 wk	
Lipid profile and glucose, mmol/L							
Total cholesterol	4.65 (3.70–5.56)	4.71 (4.03–5.35)	4.69 (3.48–5.43)	4.27 (3.30–4.88)	4.38 (3.58–5.38)	4.63 (3.41–5.59)	0.787
LDL cholesterol	3.73 (3.06–4.86)	3.58 (2.81–4.70)	3.78 (2.93–4.46)	4.15 (3.62–4.61)	3.48 (2.96–4.56)	3.42 (2.64–4.02)	0.838
HDL cholesterol	1.41 (1.10–1.48)	1.20 (1.08–1.62)	1.05 (0.89–1.55)	1.02 (0.93–1.37)	1.26 (0.81–1.70)	1.48 (0.86–1.71)	0.350
TGs	1.22 (0.64–1.34)	1.25 (1.11–1.33)	1.33 (0.85–2.07)	1.27 (0.92–1.80)	0.82 (0.59–1.26)	0.72 (0.56–1.01)	0.025
Fasting glucose	4.72 (4.54–5.07)	4.96 (4.39–5.20)	4.85 (4.61–5.32)	5.04 (4.98–5.56)	4.65 (4.20–5.09)	4.90 (4.26–5.40)	0.111
Hormones							
Adiponectin, µg/mL	NM	18.15 (11.80–27.01)	NM	13.08 (5.49–22.89)	NM	14.76 (10.92–19.44)	0.446
Leptin, ng/mL	NM	8.84 (3.77–15.57)	NM	11.06 (5.65–17.68)	NM	6.41 (2.93–19.80)	0.973
C-peptide, ng/mL	NM	0.79 (0.69–1.35)	NM	1.16 (0.74–1.58)	NM	0.95 (0.69–1.33)	0.629
Insulin, mLU/L	NM	12.72 (5.15–22.68)	NM	11.75 (6.20–15.57)	NM	9.86 (5.82–16.01)	0.688

[1] Values are medians (IQRs). NM, value not measured.
[2] Indicates the main effect of treatment, observed between groups (baseline adjusted on the basis of a linear mixed model).

prevalence of MetS in the control group increased by 6.8%. The reduction in the prevalence of MetS was a result of the increase in HDL cholesterol and the decrease in TG concentration. No changes were observed in waist circumference and fasting glucose among the groups. Mean differences between and within groups were not significant.

Dietary intake

As summarized in **Table 4**, quinoa consumption did not significantly alter nutrient intake during the intervention time. There was an increase in reported energy intake in the control group of 359 kJ at 6 wk and a reduction of 252 kJ at 12 wk. In the 25-g group there was a reduction of 364 kJ at 6 wk and a further increase of 385 kJ at 12 wk. Conversely, the 50-g group showed no difference at 6 wk, followed by a decrease of 585 kJ at 12 wk. There were no reported trends in changes in macronutrients among all treatment groups. In the control group, protein and fiber intakes increased, whereas total fat intake decreased at both 6 and 12 wk; and carbohydrate intake increased at 6 wk but decreased at 12 wk. For the 25-g group there was an increase in protein and carbohydrate intakes and a reduction in total fat intake at 6 and 12 wk, and dietary fiber intake increased at 6 wk but decreased at 12 wk. Finally, in the 50-g group there was a decrease in total fat and dietary fiber intakes and an increase in carbohydrate intake at 6 and 12 wk, whereas protein intake showed an increase at 6 wk but a reduction at 12 wk.

Compliance

According to self-report diaries, participants in the 25-g group showed compliance to the prescribed quinoa consumption of >90.0% at 6 wk and 89.6% at 12 wk, whereas participants in the 50-g group showed compliance of >90.7% at 6 wk and 82.3% at 12 wk.

Discussion

The aim of this dose-response randomized controlled study was to assess the relation between quinoa consumption and anthropometric, biochemical, and dietary data in participants who are overweight or obese. There was no effect of quinoa on anthropometric measures or body composition, circulating hormones, glucose, or total, HDL, or LDL cholesterol. There was also no effect observed on dietary intake data. However, our results showed that the consumption of 50 g quinoa seeds/d for 12 wk reduced serum TGs in overweight and obese adults.

TGs have been extensively linked to CVDs as an independent risk marker. Early evidence showed that in individuals who survive cardiovascular events, serum TGs are augmented compared with healthy controls, even after controlling for other lipoproteins such as LDL (15, 16). Some authors also argue that the relative risk of a cardiovascular event may increase by ∼32% in men and 14% in women for each millimole per liter increase in circulating TGs (17). Furthermore, recent evidence suggests that TGs are not only a risk marker but also have an active pathogenic role in the development of cardiovascular events. Mutations in the intermediates of lipid metabolism lead to changes in cardiovascular risk. Modifications in proteins such as APO-CIII (18, 19) result in a
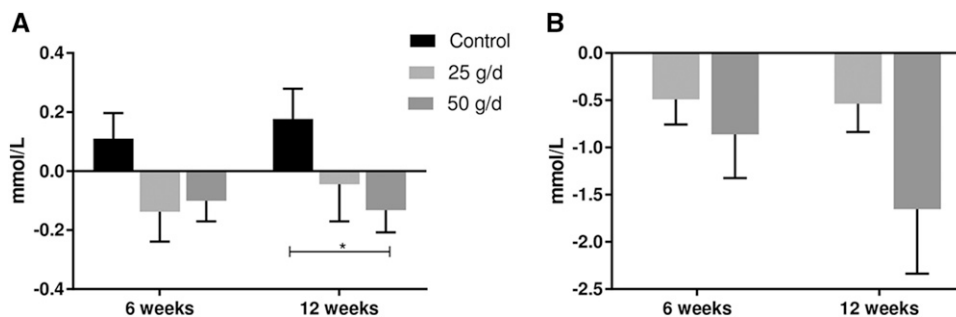
FIGURE 2 Changes in subgroup TG concentrations in participants with normal TGs (<1.7 mmol/L) (A) and those with high TGs (≥1.7 mmol/L) (B). Values were calculated as the difference between 6 and 12 wk and baseline and were compared by using a linear mixed-effects model with time as a within-subject factor and intervention group as a between-subject factor. Values are means ± SEMs. *Significant changes during the 12-wk intervention, $P < 0.05$.

reduction in the risk of coronary artery calcification. Conversely, alterations in enzymes such as lipoprotein lipase (LPL) (20) cause an increase in coronary artery disease. This confirms that the reduction in TGs achieved in this study may potentially reduce CVD risk.

Circulating TGs are the result of a complex network involving synthesis in the liver, absorption of chylomicrons by the intestine, peripheral lipolysis mediated by the action of LPL, and hepatic clearance of the remnant molecules. Early evidence showed that in obese participants (21), hypertriglyceridemia is the consequence of the increase in hepatic VLDL production due to an increased flux of FFAs from adipose tissue (22); this overproduction causes a further impairment in LPL activity in adipose tissue and muscle, which thus increases the circulating TG concentrations.

The reduction of 36% after the consumption of 50 g quinoa for 12 wk observed in our study is greater than the 16% reduction observed in previous reports in healthy participants who consumed 19.5 g quinoa for 4 wk (8) and the 4% reduction in overweight postmenopausal women who consumed 25 g quinoa for 4 wk (9). The mechanism by which quinoa consumption results in a reduction in TGs is not fully understood. A reduction in intestinal dietary fat absorption observed through the increase in lipid content in the feces was reported in rodents fed a diet containing quinoa protein extract and a quinoa extract enriched with 20-hydroxyecdysone (4, 23). A possible underlying mechanism for this quinoa effect may be based on bile acid activity. In an in vitro essay it was shown that quinoa proteins have a higher bile acid–binding capacity affecting the absorption of lipids (4). Bile acid emulsification of fats constitutes an essential part of intestinal lipid absorption.

Other approaches, such as increased dietary fiber intake, have been associated with improvement in lipid profile in a number of randomized clinical trials (24–26). Although human studies are inconclusive, some authors argued that dietary fiber may have an important role in hepatic cholesterol synthesis linked to bile acid regulation (27). Although quinoa has an enhanced content of soluble and insoluble fiber compared with other cereals and grains (28, 29), our results do not support these findings.

The reduction in TGs in our study is comparable to the reduction evidenced in pharmacologic therapy that used 40% nicotinic acid (30), 35% fibrates (31), and 20% statins (32). In addition, the consumption of 3.4 g omega-3 FAs/d reduced TGs by 23% in healthy participants with mild hypertriglyceridemia (33). Each of these treatments has a very different mechanism of action and cannot be compared with the possible TG-lowering mechanism of quinoa seed intake.

Quinoa consumption has increased steadily over recent years. In 2011, the per capita consumption of quinoa in Bolivia was reported to be ~1.11 kg/y; and in 2012, it escalated to 2.37 kg/y. In contrast, in nonproducer countries such as Canada and Australia, quinoa was not consumed in reported per capita quantities in 2011 and it reached just 227 g/y in 2014 in Canada and 81 g/y in Australia. Therefore the reduction in TGs observed in our study would require a significantly higher amount of quinoa consumption than shown in the current data. However, quinoa is a suitable replacement for grain consumption, whose intake was in the range of 112–175 g/d for the Australian population in 2011–2012 (34). In addition to providing a higher amount of nutrients such as protein and micronutrients, quinoa may have an important role in the reduction in the risk of CVDs.
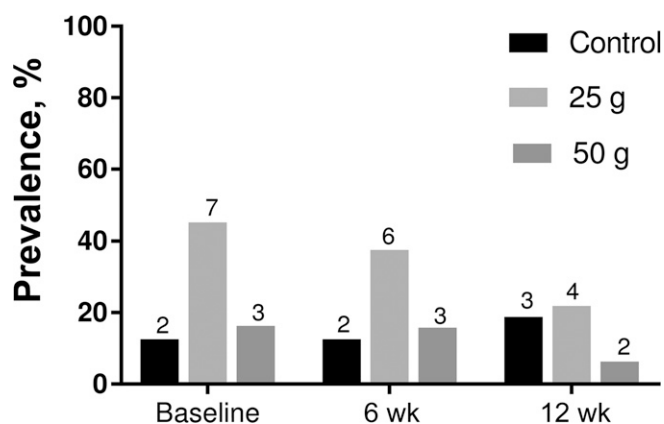

FIGURE 3 The prevalence of MetS in participants from the control and treatment groups across intervention periods. The number of participants is specified above each bar. MetS, metabolic syndrome.

TABLE 4 Effect of quinoa on nutrient intake outcomes of overweight and obese participants in the control and treatment groups at 6 and 12 wk of intervention[1]

Nutrient intake	Control group		25-g/d group		50-g/d group		P (treatment)[2]
	6 wk	12 wk	6 wk	12 wk	6 wk	12 wk	
Energy, kJ	8425.26 ± 670.43	8173.67 ± 577.70	7543.71 ± 657.56	7929.18 ± 541.45	7638.19 ± 414.77	7053.39 ± 623.47	0.305
Protein, g	90.68 ± 12.08	95.39 ± 11.52	95.21 ± 13.11	92.30 ± 11.31	90.35 ± 8.06	82.24 ± 7.98	0.460
Total fat, g	72.72 ± 8.44	74.24 ± 8.22	64.48 ± 6.27	69.31 ± 10.92	70.45 ± 6.39	65.51 ± 9.13	0.654
Carbohydrates, g	225.04 ± 17.43	199.70 ± 9.08	194.41 ± 19.04	198.87 ± 13.67	187.78 ± 13.13	175.15 ± 14.60	0.299
Dietary fiber, g	24.93 ± 2.23	24.92 ± 2.23	20.27 ± 2.34	24.86 ± 2.78	23.76 ± 2.19	25.78 ± 3.18	0.330
Protein, % of energy	17.71 ± 1.17	19.47 ± 1.55	21.17 ± 1.78	20.04 ± 2.31	19.98 ± 1.55	20.11 ± 1.59	0.537
Total fat, % of energy	31.05 ± 2.29	33.02 ± 2.12	32.12 ± 1.83	31.29 ± 3.13	33.87 ± 2.16	33.57 ± 1.76	0.794
Carbohydrates, % of energy	45.26 ± 2.35	41.34 ± 2.06	42.31 ± 2.24	41.98 ± 2.57	40.65 ± 1.69	41.57 ± 1.90	0.459
Fiber, % of energy	2.59 ± 0.37	2.53 ± 0.33	2.18 ± 0.24	2.52 ± 0.24	2.52 ± 0.24	2.95 ± 0.29	0.489
Other nutrients, % of energy	1.24 ± 0.37	1.20 ± 0.31	1.30 ± 0.26	1.24 ± 0.31	0.87 ± 0.13	1.08 ± 0.15	0.466

[1] Values are means ± SEMs.
[2] Indicates the main effect of treatment observed between groups (baseline adjusted on the basis of a linear mixed model).

Incorporating quinoa into the diet did not alter the overall nutrient intake of participants. There was a positive change with respect to a reduction in the prevalence of MetS due to the positive changes in HDL cholesterol and circulating TGs. Although the numbers were small, making small substitutions to diet quality without concomitant weight loss can improve the metabolic profile of overweight and obese participants.

Study limitations

This study showed a diminution of circulating TGs when quinoa seeds were consumed as part of the daily diet. However, due to the small sample size, further investigation must be conducted to determine the role of quinoa seed intake in participants with high circulating TGs. In addition, a compliance biomarker should be established in order to have an independent measure of quinoa consumption.

Conclusions

In our study, we showed that the consumption of 50 g quinoa seeds/d for 12 wk reduced serum TGs and therefore the prevalence of MetS in overweight and obese individuals. Further studies are needed to elucidate a clear mechanism of action by which quinoa seeds lower circulating TG concentrations.

Acknowledgments

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