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EFFECTS OF QUINOA SEED ON SOME BIOCHEMICAL PARAMETERS IN OVERWEIGHT AND OBESE IRAQI WOMEN

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Abstract
Obesity is a leading preventable cause of death worldwide, with increasing prevalence in adults and children, Obesity is one of the most important health problems in human beings and it increases the likelihood of various diseases, such as type 2 diabetes, and particularly heart disease, systemic hypertension, hyperlipidemia, cardiovascular diseases, and certain types of cancer and osteoarthritis and authorities view it as one of the most serious public health challenges of the 21st century. The consumption of bioactive compounds from the diet or dietary supplementation is one possible way to control obesity and to prevent or reduce the risks of getting various obesity-related diseases. Recently, there has been a remarkable interest in finding natural lipid inhibitors from natural products to replace synthetic compounds. Natural substances are presumed to be safe since they occur in plant foods, and are therefore seen as more desirable than their synthetic counterparts. Quinoa (*Chenopodium quinoa*) is plant that recently has been successfully grown in Iraq, providing seeds rich in nutrients and bioactive compounds. The popularity of its seeds has increased in recent years due to the claims of health benefits and super food qualities. 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 50 and 100 g quinoa/d in 50 overweight and obese women over a 12-wk intervention period. Body composition, 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 100-g quinoa group from 1.38 to 0.85 mmol/L and in the 50-g quinoa group from 1.25 to 1.02 mmol/L at12 wk ($P< 0.05$). No significant changes in TGs were observed in the control groups.

Keyword: heart disease, systemic hypertension, hyperlipidemia, cardiovascular disease

Introduction

Obesity is an increasing public health concern due to increased risk of related disorders. However, appropriate prevention and early management of obesity are changes in lifestyle patterns including physical activity and diet. Additional use of dietary agents for the prevention of obesity would be of tremendous benefits [1, 2].

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The burden of overweight and obesity is one of the biggest worldwide public health problems and is the 5th leading risk factor for global deaths [3, 4]. According to the report of the World Health Organization (WHO), 35 % and 11% of adults (≥ 20 years) were overweight and obese in 2008 [1]. Moreover, about 65 % of worldwide population lives in regions where more people are dying from overweight and obesity than from underweight [5, 6]. It is also estimated that by the year 2030, about 57.8% (3.3 billion) of adult population worldwide might be overweight or obese, with higher rates in developing countries than in developed countries[7, 8].Overweight and obesity are associated with increasing burden of chronic diseases such as type II diabetes, cardiovascular diseases, cancers, hypertension, gallbladder diseases, musculoskeletal disorders and reduced health related quality of life [9].Globally, overweight and obesity accounts for 44% of the overall diabetes burden, 23% of the ischemic heart disease and from 7% -41% of some types of cancers [10, 11].Each year about 2.8 million people die globally due to health problems associated with being overweight and obese. Furthermore, being overweight/obese can have significant effects on adolescents self-image and psychological wellbeing [12, 13]. Adolescents who are overweight are often maltreated by their peers and experience higher rates of isolation, grief, anxiety, low body satisfaction and low self-worth than adolescents with normal weight [14, 15]. 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, including gluten-free characteristics [16]. A particularly beneficial essential amino acid ratio and a superior phytochemical composition 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 [17]. 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 [18]. 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 [19] . Both studies showed changes in total cholesterol and in TGs. In addition, LDL-cholesterol changes were reported in the study that used cereal bars. Furthermore, a randomized controlled trial was carried out in postmenopausal women analyzing the effect of quinoa consumption on lipid profiles and oxidative stress markers [20]. The results of this trial were in line with previous data that reported favorable changes in TGs and total and LDL cholesterol. 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. The objective of this randomized clinical trial was to investigate the effect of different quinoa doses (50 and 100 g/d) on body composition and lipids profile in overweight and obese Iraqi women's.

Material and Methods

The current study is 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. An overweight and obese woman's who visit the Obesity clinics in Baghdad city. Participants were included if they were aged between 20 and 60 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. White organic quinoa seeds were purchased from local super market in Iraq. Quinoa bags were carefully weighed and packed into transparent Sachets of either 50g or 100 g and packaged in a box of 42 sachets, which constituted a 6-wk supply to each participant in the treatment groups [21]. An overweight and obese woman's attended appointments at Obesity clinics at baseline and at 6 and 12 wk of the intervention period. Participants were randomly allocated into 1 of 3 treatment groups. 50- and 100-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.

Measurment of Body Mass Index

Body weight was measured without shoes and light clothing and recorded in Kg. Body height was measured without a shoe and recorded in the meter. Depending on these two anthropometric parameters a mathematical formula was designed by Quetelet in the 1830s.

$BMI = \frac{Weight(kg)}{heigh\ (m^2)}$ The classification of adult BMI according to WHO was listed in Table (2-1)

Table (1) the classification of weight according to the biomass index (20).

Weight classification	BMI (Kg/m²)	Obesity	Risk of Disease
Under weight	< 18.5		Low
Normal	18.5-24.9		Normal
Overweight	25.0-29.9		Increased
Obesity	30.0-34.9	Obese	High
	35.0-39.9	Sever obese	Very high
	≥40.0	Morbidly obese	Extremely High

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.

Lipids profile 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 [22]. Statistical calculations were made using the Statistical Package for the Social Sciences (SPSS) (version 20.0) program (IBM SPSS Statistics, SPSS Inc., Chicago, Illinois, USA). The Anderson-Darling test was performed to test the adherence of continuous, parametric, variables to the normal distribution. Normally distributed continuous parametric variables, with no significant outlier, presented using their mean and standard deviation (mean±SD) and parametric tests were used; independent 2 samples student t-test was used to analyze the differences between the mean of two groups, while one-way ANOVA was used to analyze the differences between the mean of more than two groups. The statistical tests were approved by assuming a null hypothesis of no difference between the mean of variables, a P-value ≤ 0.05 was considered statistically significant.

Results and Discussion

Seventy five participants expressed interest in the study. After screening, Fifty met the inclusion criteria and were deemed eligible to participate. 50 participants were randomly assigned in the control group, 2 in the 50-g group, and 3 in the 100-g group). Fifty participants successfully completed the 12-wk intervention period; The recruitment randomization process and further analysis of the data. The study group was aged between 20 and 60 y (mean ± SEM: 36.85 ± 1.98 y). According to the WHO criteria for obesity classification [21]), The Biomass index categories showed (46%) overweight, (32%) obese, (22%) severe obese; There were no significant differences between groups at baseline (control and interventions) (**Table1)**

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

parameter	Treatment group			
Characteristics	**Control group**	**50 g quinoa/d**	**100 g quinoa/d**	**(p value)**
Participants	10	20	20	
Age(year)	35 (22–60)	38 (20–58)	43 (21–60)	0.32
BMI (kg/m^2)	28.30 (25.5–29.2)	30.38 (28.2–40.2)	32.20 (27–38.5)	0.18
Body composition				
Body weight, kg	78.28 ± 3.02	85.91 ± 3.28	89.85 ± 3.50	0.16
Waist circumference, cm	100.12 ± 2.19	103.11 ± 2.88	105.12 ± 3.20	0.30
Lipid profile and glucose, mmol/L				
Total cholesterol	4.22 (3.12–5.20)	4.88 (3.45–5.60)	4.95 (3.88–5.96)	0.12
LDL cholesterol	2.99 (2.90–3.45)	3.20 (3.10–4.40)	3.60 (2.75–4.53)	0.23
HDL cholesterol	1.00 (0.70–1.20)	1.10 (0.90–1.11)	1.30 (0.90–1.60)	0.35
TGs	1.15 (0.70–1.18)	1.25 (0.92–1.70)	1.38 (0.88–1.93)	0.22
Fasting glucose	4.22 (4.00–4.92)	5.15 (4.58–6.20)	4.80 (4.56–5.50)	0.19

Effect of quinoa on anthropometric measures and body composition

In this study, As shown in (Table2) no significant differences were observed for body-composition measurements after the 6- and 12-wk intervention time periods between and within treatment groups.

TABLE (2): Effect of quinoa on body composition in overweight and obese participants at 6 and 12 wk of intervention

Body-composition characteristics	Control		50 g/d		100 g/d		(p value)
	6 wk	**12 wk**	**6 wk**	**12 wk**	**6 wk**	**12 wk**	
BMI, kg/m^2	28.15 ± 0.50	28.88 ± 1.20	30.32 ± 1.22	30.12 ± 1.05	32.08 ± 1.16	31.30 ± 1.02	0.110
Body weight, kg	80.11 ±	82.42 ±	86.18 ±	85.20 ±	88.90 ±	89.59 ±	0.120

	2.12	2.30	3.05	3.10	3.83	3.80	
Waist circumference, cm	100.18 ± 3.15	100.88 ± 3.15	103.88 ± 3.89	103.86 ± 3.54	105.15 ± 3.00	105.20 ± 3.00	0.21

Lipid Profile and glucose

The interaction effects between lipids, lipoproteins, and fasting glucose with quinoa intake is reported in (Table3).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 100-g group compared with the control group (0.85 and 1.138 mmol/L, respectively; $P = 0.015$). and in the 50-g quinoa group from 1.25 to 1.02 mmol/L at12 wk ($P < 0.05$) 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 100g and 50 g quinoa, which reduced serum TGs by ~37.5% and 22.3% after 12 wk of a dietary intervention.

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

Characteristics	Control group		50-g/d group		100-g/d group		(p value)
	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	1.40 (1.10–1.42)	1.20 (1.08–1.42)	1.11 (0.90–1.50)	1.16 (0.92–1.35)	1.32 (0.85–1.60)	1.35 (0.86–1.70)	0.33
TGs	1.22 (0.64–1.34)	1.25 (1.11–1.33)	1.20 (0.85–2.07)	1.02(0.92–1.70)	0.90 (0.59–1.26)	0.85 (0.56–1.01)	**0.015**
Fasting glucose	4.72 (4.54–	4.90 (4.39–	4.95 (4.61–	5.04 (4.98–	4.75 (4.20–	4.40 (4.26–5.40)	0.111

	5.07)	5.20)	5.32)	5.56)	5.09)

The aim of this dose-response randomized controlled study was to assess the relation between quinoa consumption and anthropometric, and biochemical data in participants who are overweight or obese. There was no effect of quinoa on anthropometric measures or body composition, glucose, or total, HDL, or LDL cholesterol. However, our results showed that the consumption of 100 and 59 g quinoa seeds/d for 12 wk reduced serum TGs in overweight and obese adults. 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. 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); 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 [22].

The aim of this dose-response randomized controlled study was to assess the relation between quinoa consumption and anthropometric and biochemical parameter in participants who are overweight or obese. There was no effect observed of quinoa on anthropometric measures or body composition, glucose, or total, HDL, or LDL cholesterol.. However, our results showed that the consumption of 50 g and 100 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 roteins such as APO-CIII [18, 19] result in a reduction in the risk of coronary artery calcification. Conversely, alterations in enzymes such as lipoprotein lipase (LPL) [23] 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 (18); this overproduction causes a further impairment in LPL activity in adipose tissue and muscle, which thus increases the circulating TG concentrations. The reduction of 37.5% after the consumption of 100 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, 3). 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 (21; 22). 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 (23). Although quinoa has an enhanced content of soluble and insoluble fiber compared with other cereals and grains (24, 25), 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 (26), 35% fibrates (27), and 20% statins (28). In addition, the consumption of 3.4 g omega-3 FAs/d reduced TGs by 23% in healthy participants with mild hypertriglyceridemia (29). 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 non producer 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 (30). 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. Further studies are needed to elucidate a clear mechanism of action by which quinoa seeds lower circulating TG concentrations. Overall the results suggest some potential

benefit of consuming quinoa on glucose response but this requires further investigation, possibly with larger doses of quinoa. The mechanisms by why quinoa may have this effect remain undetermined

Conclusions

The consumption of 50 g and 100 g quinoa/d lowers serum TGs in overweight and obese women's. Body composition, and total, LDL, and HDL cholesterol were not significantly altered by quinoa consumption ($P > 0.05$). 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. It

is important to note that studying the role of environmental and genetic factors in the susceptibility of obese. Increasing the sample size will provide more visible results; Further studies are needed to elucidate a clear mechanism of action by which quinoa seeds lower circulating TG concentrations.

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