



The 6<sup>th</sup> international- 20<sup>th</sup> Arabic  
conference for Home Economics  
Home Economics and Educational  
quality assurance December 23rd -  
24th, 2018

---

---

**Journal of Home  
Economics**

---

---

<http://homeEcon.menofia.edu.eg> ISSN 1110-2578

## **Supplementations Dietary of Quinoa Seeds Powder and their Effect on Hypercholesteremia & Oxidation in Albino Rats**

**Asmaa. H. Ahmed<sup>1</sup> and Basma R. M. Khateib<sup>2</sup>**

Dept. of Home Economics, Faculty of Specific Education, Menoufia  
University, Egypt<sup>1</sup> , Dept. of Nutrition & Food Science, Faculty of Home  
Economics, Menoufia University, Egypt<sup>2</sup>.

---

### **Abstract:**

The main target of this research was to study the effect of dietary supplementation of quinoa seeds powder (QSP) under two different concentrations (150 and 300 g/kg diet) to give more protection against hypercholesteremia disease. Twenty (20) mature male albino rats weighing  $184 \pm 2$  g were used in this study. The rats were divided into 4 groups (5 rats in each group) one of them used as control negative group while other three groups fed on diet containing 1.5% cholesterol plus 10 % sheep tail for 15 days to induce hypercholesteremia disease , one of these left as control positive group and other two groups fed on two doses of quinoa (150 and 300 g/kg diet) for 28 days. Body weight gain, FER and FI were estimated as well as internal organs weight . Also, lipid profile (total cholesterol, triglyceride, atherogenic index, HDL, LDL and VLDL) in addition, the oxidants (MDA) and antioxidants enzymes activities including SOD, GSH and CAT of hypercholesteremic rats were analyzed. Results indicated that treatment with quinoa seeds powder improved the previous parameters when compared to control positive group. The best treatment observed in the group administrated with quinoa (300 g/kg diet). Therefore, the study recommended that quinoa supplementation exerts significant positive effects on metabolic, hypercholesterolemia and cardiovascular.

**Keywords:** Quinoa- Hypercholesteremia - Oxidation- antioxidant activities- lipid profile

## **Introduction**

Familial hypercholesterolemia (FH) is a prevalent autosomal dominant disorder of lipoprotein metabolism, caused by mutations in the genes encoding for the low-density lipoprotein (LDL) receptor, apolipoprotein B (ApoB) or proprotein convertase subtilisin/kexin-type 9 (PCSK9). Patients with FH are characterized by elevated serum LDL-cholesterol (LDL-C) levels and an increased risk for premature cardiovascular disease (CVD). LDL-C levels vary widely amongst FH patients. Because of the continuous and direct interaction between the digestive tract and foods, dietary compounds represent an interesting source of chemopreventive agents for gastrointestinal health ( **Hovingh et al., 2013 and Ruel et al., 2018**).

Hypercholesterolemia is defined as extravagantly high level of plasma cholesterol , and is a high risk factor for many harmful cardiovascular events. Total cholesterol levels above 200 mg/dl leads to development of peripheral vascular and coronary artery disease. High attention has been directed toward evaluating mechanisms by which hypercholesterolemia may impact vascular outcomes; these include both results of direct cholesterol lowering therapies and alternative interventions for improving vascular function ( **Stapleton et al., 2010** ) .

Quinoa (*Chenopodium quinoa* Willd.) belongs to the family *Amaranthaceae*, native to the Andean regions. It can adapt to various types of climatic and soil. Quinoa has attracted the attention of nutrition scientists because of its high nutritional value. It is rich in proteins, dietary fiber, unsaturated fats , minerals and vitamins. It is also distinguished by its amino acid balance. Quinoa has the advantage of being a gluten-free grain making it suitable for celiac patients ( **Alvarez-Jubete et al., 2010 ; Filho et al., 2017 and Gordillo et al., 2016** )

Quinoa is easy to preparation and cook ( **Filho et al., 2017** ). Similar to rice, its seeds are used in soups, by puffing them

to do breakfast cereal, or by grinding them to make baked products such as cookies, bread, biscuits, pasta, crisps, tortilla, and pancake (**Bhargava et al., 2006**).

**Reguera et al., (2018)** reported that the unique nutritional value of quinoa seeds depends on their high antioxidants activity. **Zhu et al., (2001)** extracted six glycosylated flavonols from quinoa seeds and showed that quinoa may play an important role in inhibition of free radicals .Quinoa considered as a natural antioxidant at the cell membrane level, protecting fatty acids from damage of free radicals (**Ng et al., 2007 and Ryan et al., 2007**) . Quinoa seeds also play a good role in lowering blood sugar and reducing weight (**Mihaela et al., 2014**).

Despite all these health benefits, quinoa is not widely used due to several reasons, such as high import costs of the grain and lack of informations about it among consumers. As we believe that further research is needed to provide more information about quinoa, so we will show the effect of dietary supplementation with quinoa seeds powder (QSP) to find out more protection against hypercholesterolemia disease .

## **Materials and methods**

### **Materials**

#### **Preparation of basal diet**

The basal diet was prepared according to **Reeves et al., (1993)**.

### **Methods**

#### **Preparation of sample**

Quinoa seeds were purchased from Harraz for Food Industry and Natural Products, Bab Alkhalq, Cairo, Egypt. Quinoa seeds were well grinded for powder and then added in the above amounts to the basal diet.

### **Induction of experimental hypercholesterolemia**

High cholesterol was induced in normal healthy male albino rats by feeding on diet containing 1.5% cholesterol plus 10% sheep tail fat for 15 days sheep according to **AIN (1993)**.

### **iological Experiment**

Twenty mature male albino rats weighing  $184 \pm 2$  g were used in this study. The rats were divided into 4 groups (5 rats in each group) one of them used as control (-ve) while other three groups had given 1.5% cholesterol plus 10% sheep tail for 15 days, one of these left as control positive group and other two groups fed on two doses of quinoa (150 and 300 g/kg diet) for 28 days

### **Blood sampling collections**

At the end of experiment period, blood samples were collected after 14 hours fasting from the portal vein; the rats were sacrificed under ether anesthesia. Blood samples were received into clean dry centrifuge tubes and left to clot at room temperature, then centrifuged for 10 minutes at 3000 r.p.m to separate the serum. Serum was carefully aspirated and transferred into clean covette tubes and stored frozen at  $-20^{\circ}\text{C}$  for analysis (**Malhotra, 2003**).

### **Biological evaluation**

During the experimental period, the diet consumed was recorded every day, and body weight recorded every week. The body weight gain (BWG) and feed efficiency ratio (FER) were determined according to **Chapman et al., (1959)** using the following equations.

$$\text{B.W.G. \%} = \frac{(\text{Final weight} - \text{Initial weight}) \times 100}{\text{Initial weight}}$$

$$\text{FER} = \frac{\text{Body weight gain (g/day)}}{\text{Feed intake (g/day)}}$$

Relative organ weight calculated by the following formula:

$$\text{Relative organ weight (ROW)} = \frac{\text{Organ weight}}{\text{Total body weight}} \times 100$$

### **Biochemical parameters**

Serum triglycerides (T.G) , total cholesterol (T.C) and high density lipoprotein cholesterol (HDL-c) were measured according to **Fossati and Principe (1982); Richmond (1973) and Allain (1974)**. Low density lipoprotein cholesterol (LDL-c) was measured according to **Castelli *et al.*, (1977)**.

**LDL Concentration mg/dl = LDL= TC- (VLDL+ HDL)** Serum very low-density lipoprotein cholesterol (VLDL-c) was calculated as mg/dl according to **Lee and Nieman (1996)** equation:

$$\text{VLDL-C concentration mg/dl} = \frac{\text{T. G}}{5}$$

**atherogenic index (AI) =  $\frac{\text{LDL} + \text{VLDL}}{\text{HDL}}$**

Malondialdehyde (MDA), superoxide dismutase (SOD), glutathione (GSH) and Catalase(CAT) were estimated in liver tissue according to **Ohkawa *et al.*, (1979); Kakkar *et al.*, (1984); Rotruck *et al.*, (1973) and Goth, (1991)**, respectively .

### **Statistical analysis of data:**

Data were statistically analyzed using a computerized program at the Scientific Computer Center, Faculty of Home Economics, Menoufia University, using ANOVA test according to **Armitage and Berry, (1987)**.

### **Results and Discussion**

#### **Effect of quinoa on feed intake (FI) , feed efficiency ratio (FER) and body weight gain (BWG %) of hypercholesteremic rats**

Data presented in Table (1) show the effect of quinoa on hypercholesterolemic rats. It is evident that the value of feed intake (FI), feed efficiency ratio (FER) and body weight gain of rats in control (+ve) was high compared to negative control group . The mean values were  $18.00 \pm 1g$  ,  $0.029 \pm 0.001 g$  and  $15.5 \pm 0.78\%$ , respectively .

Regarding group 3 and 4, it is observed that quinoa can be used for management hypercholesterolemia and to reduce BWG especially in G4 which fed on quinoa at 300g / kg diet.

**Table (1): Effect of quinoa on feed intake (FI) g , feed efficiency ratio (FER) g and body weight gain BWG(%) of hypercholesterolemic rats**

Groups Parameters	Control -ve (G1)	Control +ve (G2)	Quinoa 150 g/kg diet (G3)	Quinoa300 g/kg diet (G4)	LSD
FI (g) Mean ± SD	12 <sup>c</sup> ±1.04	18 <sup>a</sup> ±1	16 <sup>b</sup> ±1	13 <sup>c</sup> ±1	1.8
Percent of change	- 33.3%	-	-11.11%	-27.77%	--
FER (g) Mean ± SD	0.028 <sup>a</sup> ±0.00	0.029 <sup>a</sup> ± 0.001	-0.027 <sup>b</sup> ±0.002	-0.045 <sup>c</sup> ± 0.005	0.008
Percent of change	-3.44 %	-	-193.0%	-255.17%	--
BWG (%) Mean ± SD	9.78 <sup>b</sup> ±3.08	15.5 <sup>a</sup> ±0.78	-12.6 <sup>c</sup> ±0.77	-16.8 <sup>d</sup> ±0.378	3.1
Percent of change	-36.9%	-	-181.29%	- 208.38%	--

Values with different letters indicate significant differences between the groups (P≥0.05), and vice versa.

LSD: least significant Differences (P≤0.05).

From this point of view, the above results coincided with the results of the researchers , **Thomas *et al.*, (2015)** they reported that quinoa consumption can decrease weight gain, improve lipid profile and improve the adverse effect of oxidative stress. These physiological effects may be due to the presence of saponins and protein in the quinoa seed.

**Mihaela *et al.*, (2014)** showed that diet fortified with 30% and 40% quinoa seeds can decrease the body weight, feed consumption, feed efficiency ratio, blood cholesterol and other lipids as well as improve liver and kidney functions compared to positive control group. Diet with 40% QSP reduced the adverse effect of hypercholesterolemia.

Also, **Mithila and Khanum (2015)** reported that rats fed on quinoa had little food consumption and had a decrease in weight gain as compared to control. Quinoa is one of the most nutritious

plants. Its seeds are rich in proteins, minerals, B vitamins and fibre which makes quinoa a complete and modern food to decrease weight.

**Effect of quinoa on relative weight of heart, kidneys and liver of hypercholesterolemic rats**

Data illustrated in Table (2) indicated the effect of treatment with quinoa on relative weight of heart, kidneys and liver of hypercholesterolemic rats. It is clear that rats fed on hypercholesterolemic diet (c+ve group) showed significant increase ( $p \leq 0.05$ ) in relative heart, kidneys and liver weight when compared to healthy (-ve group). Result denote that there were significant decreases in treated groups as compared to (c+ve group). It could be noticed that rats fed on quinoa powder (300 g/kg diet) showed the lowest decrease in precedent organs as compared to (c+ve group). The percent of change for quinoa powder (300 g/kg diet) were -0.45%, -63.20% and -38.79%, respectively.

**Table (2) : Effect of quinoa on relative weight of heart, kidney and liver of hypercholesterolemic rats**

Groups Parameters	Control -ve (G1)	Control +ve (G2)	Quinoa 150 g/kg diet (G3)	Quinoa 300g/kg diet (G4)	LSD
Heart (g) Mean $\pm$ SD	0.16 <sup>c</sup> $\pm$ 0.023	0.35 <sup>a</sup> $\pm$ 0.05	0.263 <sup>b</sup> $\pm$ 0.023	0.193 <sup>c</sup> $\pm$ 0.023	0.062
Percent of change	-54.29%	-	-25.71%	-0.45%	-
Kidney (g) Mean $\pm$ SD	0.393 <sup>c</sup> $\pm$ 0.035	1.063 <sup>a</sup> $\pm$ 0.125	0.533 <sup>b</sup> $\pm$ 0.041	0.393 <sup>c</sup> $\pm$ 0.057	0.107
Percent of change	-63.20%	-	-50.00%	-63.20%	-
liver (g) Mean $\pm$ SD	1.85 <sup>d</sup> $\pm$ 0.052	3.656 <sup>a</sup> $\pm$ 0.060	2.94 <sup>b</sup> $\pm$ 0.202	2.24 <sup>c</sup> $\pm$ 0.061	0.235
Percent of change	-49.45%	-	-19.97%	-38.79%	-

Values with different letters indicate significant differences between the groups ( $P < 0.05$ ), and vice versa.

LSD: least significant Differences ( $P < 0.05$ )

These results similar to **Pawel et al., (2010)** findings who demonstrated that quinoa seeds can act as a moderate protective agent against potential of fructose-induced changes in rats by reducing lipid peroxidation and by enhancing the antioxidant capacity of blood (plasma) and heart, kidney, testis, lung and pancreas.

**González et al., (2014)** showed that quinoa seeds can decrease liver, heart and kidney weight. They reported also that consumption quinoa seeds caused increase in vitamin E in the heart, liver, lungs, spleen, and kidneys and increase the antioxidant protection power of these organs in addition to their tissues.

**Effect of quinoa on total cholesterol (T.C), triglyceride (T.G) and atherogenic index (A.I) of hypercholesterolemic rats**

Data recorded in Table (3) show the effect of quinoa powder on total cholesterol (TC) of hypercholesterolemic rat. Control (+ve) groups showed significant increase in total cholesterol as compared to healthy rats which were  $174.0 \pm 3.60$  mg /dl and  $110 \pm 2$  mg/dl, respectively. Feeding on quinoa led to a decrease in total cholesterol (Tc). Maximum decrease of total cholesterol recorded for group 4 which fed on quinoa at 300g/kg diet.

Regarding data illustrated in Table (4) mentioned the effect of treatments on hypercholesterolemic rats with quinoa powder, there was a significant increase of TG in hypercholesterolemic rats (control + ve group) as compared to healthy group control (- ve) which were  $(149.00 \pm 3.60)$  and  $(77.33 \pm 2.51)$  mg /dl, respectively. All rats fed on quinoa at two doses showed significant decreases in TG as compared to control (+ve) group. It is evident that the best treatment was of groups 4 that given quinoa powder (300 g/kg diet).



**Table(3): Effect of Quinoa on total cholesterol (T.C.), triglyceride (T.G.) and atherogenic index (A.I) of hypercholesterolemic rats**

Groups Parameters	Control -ve (G1)	Control +ve (G2)	Quinoa 150 g/kg diet (G3)	Quinoa 300g/kg diet (G4)	LSD
TC (mg/dl) Mean ± SD	110 <sup>d</sup> ±2	174 <sup>a</sup> ±3.60	144 <sup>b</sup> ±3.60	123 <sup>c</sup> ±3.60	3.31
Percent of change	-36.78%	--	-17.24%	-29.21%	--
TG (mg/dl) Mean ± SD	77.33 <sup>d</sup> ±2.51	149 <sup>a</sup> ±3.60	124.33 <sup>b</sup> ±4.50	93 <sup>c</sup> ±3.60	8.039
Percent of change	-48.10%	--	-16.55%	-37.58%	--
Atherogenic Index (AI) (mg/dl) Mean ± SD	1.13 <sup>c</sup> ±0.090	6.06 <sup>a</sup> ±0.805	3.53 <sup>b</sup> ±0.263	1.7 <sup>c</sup> ±0.235	0.925
Percent of change	-81.35	--	-41.7%	-71.94%	--

Values with different letters indicate significant differences between the groups (P<0.05), and vice versa.

LSD: least significant Differences (P<0.05)

The present study is in accordance with **Farinazzi-Machado et al., (2012)** they reported that daily intake of quinoa pills for 30 days significantly decreased triglycerides. Several clinical studies have indicated that quinoa consumption can lower cholesterol due to the presence of main ingredients (proteins, fibers, vitamins), tocopherol and carotenoids, minerals (iron, zinc, and magnesium), saponins, plant sterols, and polyphenols (**Abderrahim et al., 2015 and Pellegrini et al., 2018**) . **Yao and Rong (2017)** focused on the phytochemical composition of quinoa and amaranth seeds, the antioxidant and anti- inflammatory activities of hydrophilic (e.g. phenolics, betacyanins) and lipophilic (e.g. fatty acids, tocopherols, and carotenoids) nutrients, and how these contribute to the potential health benefits, especially in lowering the risk of the oxidative stress related diseases cancer, cardiovascular disease, diabetes, and obesity.

**Effect of Quinoa on the serum levels of lipoproteins fractions (HDLc- LDLc and VLDLC) of hypercholesterolemic rats**

To summarize the results presented in this in Table (4) It is evident that rats fed on hypercholesteremic diet control(+ve group) indicated significant decrease in HDLc but significant increase in

LDL-c and VLDL-c as compared to normal healthy rats. Rats fed on quinoa with 300 g/kg diet showed a significant increase in HDL-c but significant decrease in LDL-c and VLDL-c levels. Treatment of quinoa with 300 g/kg diet showed the highest increase in HDLc but the highest decrease in LDLc and VLDLc as compared to control positive group.

**Table (4) : Effect of quinoa on high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) of hypercholesterolemic rats**

Groups Parameters	Control -ve (G1)	Control +ve (G2)	Quinoa 150 g/kg diet (G3)	Quinoa 300g/kg diet (G4)	LSD
HDL (mg/dl) Mean $\pm$ SD	51.5 <sup>a</sup> $\pm$ 3.12	24.83 <sup>c</sup> $\pm$ 2.84	31.83 <sup>b</sup> $\pm$ 2.56	45.66 <sup>a</sup> $\pm$ 3.51	6.723
Percent of change	107.4%	--	28.19%	83.89	--
LDL (mg/dl) Mean $\pm$ SD	43.03 <sup>d</sup> $\pm$ 1.76	119.36 <sup>a</sup> $\pm$ 4.21	87.3 <sup>b</sup> $\pm$ 1.93	58.73 <sup>c</sup> $\pm$ 5.44	5.49
Percent of change	-63.94	--	-26.85%	-50.79%	--
VLDL(mg/dl) Mean $\pm$ SD	15.46 <sup>d</sup> $\pm$ 0.503	29.8 <sup>a</sup> $\pm$ 0.721	24.86 <sup>b</sup> $\pm$ 0.90	18.6 <sup>c</sup> $\pm$ 0.721	1.607
Percent of change	-48.12%	--	-16.57%	-37.58%	--

Values with different letters indicate significant differences between the groups (P<0.05), and vice versa.

LSD: least significant Differences (P<0.05)

These results similar to **Paško *et al.*, (2010)** who came to same result that quinoa seeds can reduce serum TC, LDL and TG when compared to the control positive group. Quinoa seeds also can reduce serum glucose level and plasma total protein level. Fructose significantly decreased HDL level in control group but when the diet was fortified with quinoa seeds, the level of HDL increased.

**Flávia *et al.*, (2012)** indicated that quinoa can reduce the levels of total cholesterol, triglycerides, and LDL-c . It can be concluded that the consumption of quinoa in diet is beneficial for protecting against risk factors related to cardiovascular diseases that are among the leading causes of death in the world.

Result from **Wu *et al.*, (2015)** and **Lamothe *et al.*, (2015)** agreed with previous result they showed that greater consumption of fiber-rich whole grains is useful to reduce risk of type 2 and cardiovascular disease. Quinoa is an important source of dietary fiber, it contains about 2.6%-10% of the total weight of the grain. About 78% of its fiber content is insoluble and 22% soluble.

Also, **Rubén and Blanca (2017)** reported that quinoa has been found to contain numerous phytochemicals including saponins, phytosterols, phytoecdysteroids, phenolics and bioactive peptides. These compounds may exert beneficial effects on hypercholesteremia, metabolic, cardiovascular, and gastrointestinal health.

**Effect of quinoa on malondialdehyde (MDA), superoxide dismutase (SOD), Glutathione (GSH) and catalase (CAT) in liver tissue of hypercholesteremic rats.**

Data illustrated in Table (5) showed the treatment of quinoa on hypercholesteremic rats reflected apparent increase in lipid peroxidation malondialdehyde (MDA) of control +ve group as compared to control -ve group. MDA values which were  $95.50 \pm 3.50$  and  $63.00 \pm 3.60$  nmol/g tissue for control (+ve) and control (-ve) groups, respectively. All treated showed significant decreases in malondialdehyde as compared to control +ve, the best treatment for group 4 which fed on quinoa (300 kg/ diet) because this group caused the highest decrease in malondialdehyde which was  $71.66 \pm 3.50$  nmol/g as compared to hypercholesteremic rats. Concerning the percent decrease of control -ve group it was -34.3% as compared to control +ve group.

Regarding superoxide dismutase (SOD) data recorded in Table (6) showed that there was a significant increase in normal rats as compared to hypercholesteremic rats in SOD which were  $9.16 \pm 1.04$  and  $2.96 \pm 0.50$  U/mg tissue, respectively. All treated group showed a significant increase in SOD as compared to control +ve group.

Concerning glutathione (GSH), it is observed that there was a significant increase of glutathione in healthy rats (control -ve) as

compared to positive rats, values were  $94.00 \pm 2.00$  and  $59.00 \pm 1.00$  mg/g tissue respectively. Results denoted that there was a significant increase with all tested quinoa as compared to control positive group (control +ve). The best treatment was that of group 4 which fed quinoa 300g/kg diet .

The effect of treated groups with quinoa on CAT enzyme recorded in Table (6). The mean value of control positive group was lower than negative group (healthy rats), which were  $5.00 \pm 1.00$  and  $19.50 \pm 1.80$  U/g tissue, respectively and the results showed a significant difference with percent of increase 4% folds than control positive group. All treated group showed a significant increase in (CAT) enzyme as compared to control + ve groups. It is evident that the best treatment was quinoa 300g/kg. diet which showed (CAT) enzyme value were  $16.30 \pm 1.52$  U/g tissue

**Table (5) : Effect of Quinoa on oxidant and antioxidant parameters in liver Tissue of hypercholesterolemic rats**

Groups Parameters	Control -ve (G1)	Control +ve (G2)	Quinoa 150 g/kg diet (G3)	Quinoa 300g/kg diet (G4)	LSD
MDA (nmol/g tissue) Mean $\pm$ SD	$63^d \pm 3.60$	$95.5^a \pm 3.5$	$84.5^b \pm 1.80$	$71.66^c \pm 3.05$	6.569
Percent of change	-34.30%	--	-11.51%	-24.96%	--
SOD (U/mg tissue) Mean $\pm$ SD	$9.16^a \pm 1.04$	$2.96^d \pm 0.50$	$5.5^c \pm 0.5$	$7^b \pm 0.5$	1.497
Percent of change	2.945%	--	85.81%	136.48%	--
GSH (mg/g tissue) Mean $\pm$ SD	$94^a \pm 2$	$59^d \pm 1$	$72^c \pm 3$	$86^b \pm 2$	4.75
Percent of change	59.32%	--	2203%	45.76%	--
CAT (U/g tissue) Mean $\pm$ SD	$19.5^a \pm 1.80$	$5^c \pm 1$	$12^b \pm 2$	$16.3^a \pm 1.52$	3.61
Percent of change	290%	--	140%	226%	--

Values with different letters indicate significant differences between the groups (P<0.05), and vice versa.

LSD: least significant Differences (P<0.05)

These results agree with Pawel *et al.*, (2010) who mentioned that quinoa seeds effected on the oxidative stress by decreasing

MDA in plasma, and increasing the activities of antioxidant enzymes. Also, **De Carvalho et al., (2014)** reported a potential benefit of quinoa seeds consumption against oxidative stress. **Park et al., (2017)** reported that quinoa seeds have antioxidant activity due to the presence of important active ingredients such as polysaccharides

Quinoa is an excellent source of vitamin E, the amount of  $\gamma$ -tocopherol was higher than the amount present in corn oil so quinoa oil has a long shelf life due to the antioxidant power of this substance. vitamin E, is very important because it acts as a natural antioxidant to protect fatty acids from damage by free radicals (**Ng et al., 2007 and Ryan et al., 2007**)

**Zhu et al., (2001)** extracted six glycosylated flavonols from quinoa seeds and showed that quinoa may play an important role in inhibition of free radicals.

### **Conclusion:**

To summarize the results presented in this section, quinoa emerges as a food of particular interest to celiac patients, as the potential cornerstone of a healthy, gluten-free diet. Furthermore, we hypothesize that including quinoa in the diet could decrease oxidative stress, improve serum lipid profile, help to control body weight and serum glucose, and decrease cardiovascular disease and type 2 diabetes risk factors; quinoa may even prove beneficial in reversing the effects of these diseases.

**Reference**

- Abderrahim, F.; Huanatico, E.; Segura, R.; Arribas, S.; Gonzalez, M.C. and Condezo-Hoyos, L. (2015):** Physical features, phenolic compounds, betalains and total antioxidant capacity of coloured quinoa seeds (*Chenopodium quinoa Willd.*) from Peruvian Altiplano. *Food chemistry*, 183: 83-90.
- AIN,A.(1993):** American institute of nutrition purified diet for laboratory rodent : final report to J. Nutrition, 123: 1939-1951.
- Allain, C.C. (1974):** Cholestrol enzymatic colorimetric method. *J. of Clin. Chem.*, 20: 470.
- Alvarez-Jubete, L.; Wijngaard, H.; Arendt, E. K. and Gallagher, E. (2010):** Polyphenol composition and in vitro antioxidant activity of amaranth, quinoa buckwheat and wheat as affected by sprouting and baking. *Food chemistry*, 119(2): 770-778.
- Armitage, P. and Berry, G. (1987):** *Statistical Methods in Medical Research*. Black Well. Oxford. UK; 93-213.
- Bhargava, A., Shukla, S. and Ohri, D. (2006):** *Chenopodium quinoa an Indian perspective*. *Industrial crops and products*, 23(1): 73-87.
- Castelli, W. P.; Doyle, J. T.; Gordon, T.; Hames, C. G.; Hjortland, M. C.; Halley, S. B.; Kagan, A. and Zuckel, W. J. (1977):** HDL cholesterol and other lipids in coronary heartdisease: The cooperative lipoprotein phenotyping study. *Circulation* , 55: 767-772.
- Champman, D. G.; Gastilla, R. and Cambell, J. A. (1959):** Evaluation of protein in food.LA. method for the determination of protein efficiency ratio. *Can. J.Biochem. Phosiol.*,37:679-686.
- De Carvalho, F. G.; Ovídio, P. P.; Padovan, G. J.; Jordao Junior, A. A.; Marchini, J. S. and Navarro, A. M. (2014):** Metabolic parameters of postmenopausal women after quinoa or corn flakes intake—a prospective and double-blind study. *International Journal of Food sciences and nutrition*, 65(3), 380-385.

- Farinazzi-Machado, F.M.V.; Barbalho, S.M.; Oshiiwa, M.; Goulart, R. and Pessan Junior, O.( 2012):** Use of cereal bars with quinoa (*Chenopodium quinoa W.*) to reduce risk factors related to cardiovascular diseases. Food Science and Technology, 32(2), 239-244
- Filho, A. M. M.; Pirozi, M. R.; Borges, J. T. D. S.; Pinheiro Sant'Ana, H. M.; Chaves, J. B. P. and Coimbra, J. S. D. R. (2017)** : Quinoa: nutritional, functional, and antinutritional aspects. Critical reviews in food science and nutrition, 57(8), 1618-1630.
- Flávia, M. V.; Sandra, M. B.; Marie, O.; Ricardo, G. and Osvaldo, P. J. (2012):** Use of cereal bars with quinoa (*Chenopodium quinoa W.*) to reduce risk factors related to cardiovascular diseases. Food Science and Technology, 32(2), 239-244.
- Fossati, P. and Principe, L. (1982):** Determination of triglycerides. Clinical Chemistry, 28: 2077-2078.
- González Martín, M. I.; Wells Moncada, G.; Fischer, S. and Escuredo, O. (2014):** Chemical characteristics and mineral composition of quinoa by near-infrared spectroscopy. Journal of the Science of Food and Agriculture, 94(5), 876-881.
- Gordillo, B.; E1-Díaz-Rizzolo, D. A.;Roura, E.;Massanés, T.and Gomis, R.(2016):** Quinoa (*Chenopodium quinoa Willd*), from nutritional value to potential health benefits: an integrative review. J. Nutr. Food Sci., 6(497), 10-4172.
- Goth, L. (1991):** A simple method for determination of serum catalase activity, and revision of reference range. Clinica Chimica Acta,196:143– 52.
- Hovingh,G.K.; Davidson, M.H.; Kastelein, J.J.P. and O'Connor, A.M.(2013):** Diagnosis and treatment of familial hypercholesterolemia. European heart journal, 34(13), 962-971.
- Kakkar, P.; Das, B. and Viswanathan, P. N. (1984):**A modified spectrophotometric assay of superoxide dismutase. Indian J. of Biochemistry and Biophysics, (21):130-132.
- Lamothe, L.M.; Srichuwong, S.; Reuhs, B.L. and Hamaker, B.R. (2015):** Quinoa (*Chenopodium quinoa W.*) and amaranth (*Amaranthus caudatus L.*) provide dietary fibres

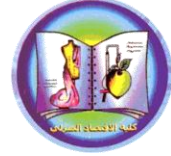
high in pectic substances and xyloglucans. *Food chemistry*, 167:490-496.

- Lee, R. and Nieman, D. (1996):** Nutrition Assessment. 2nd Ed. Mosby, Missouri, USA. 591 – 594.
- Malhotra, V.K. (2003):** Practical Biochemistry for Students. Fourth Edition Jaypee Brothers Medical Publishers (P) LTD. New Delhi .
- Mihaela, A.; Ovidiu, T.; Adriana, D. and Alexandra (2014):** Research Concerning Physico-Chemical and Microbiological Characteristics Of Quinoa, Dried Milk and Oat Bran Yogurt *Journal of Faculty of Food Engineering*, Volume XII, (1) : 28 – 33.
- Mithila M.V.and Khanum, F( 2015):** Effectual comparison of quinoa and amaranth supplemented diets in controlling appetite; a biochemical study in rats. *Journal of food science and technology*, 52(10), 6735-6741.
- Ng, S. C.; Anderson, A.; Coker, J. and Ondrus, M. (2007):** Characterization of lipid oxidation products in quinoa (*Chenopodium quinoa*). *Food Chemistry*, 101(1): 185-192.
- Ohkawa, H.; Ohishi, W. and Yagi, K. (1979):** Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Anal. Bio. Chem.*, 95: 351–358.
- Park, J. H., Lee, Y. J., Kim, Y. H., & Yoon, K. S. (2017):** Antioxidant and antimicrobial activities of Quinoa (*Chenopodium quinoa* Willd.) seeds cultivated in Korea. *Preventive nutrition and food science*, 22(3), 195.
- Paško, P.; Zagrodzki, P.; Bartoń, H.; Chłopicka, J. and Orinstein, S. (2010):** Effect of quinoa seeds (*Chenopodium quinoa*) in diet on some biochemical parameters and essential elements in blood of high fructose-fed rats. *Plant Foods Hum Nutr.*, 65(4):33-38.
- Pawel, P.; Henryk, B.; Pawel, Z.; Aleksandra, I.; Miroslaw, K.; Malgorzata, G.; Maciej, G. and Shela, G. (2010):** Effect of diet supplemented with quinoa seeds on oxidative status in plasma and selected tissues of high fructose-fed rats. *Plant foods for human nutrition*, 65(2), 146-151.



- Pellegrini, M.; Lucas-Gonzales, R.; Ricci, A.; Fontecha, J.; Fernández-López, J.; Pérez-Álvarez, J.A.; Viuda-Martos, M.(2018):** Chemical, fatty acid, polyphenolic profile, techno-functional and antioxidant properties of flours obtained from quinoa (*Chenopodium quinoa* Willd) seeds. *Industrial Crops and Products*, 111: 38-46.
- Reeves, D.W.; Mask, P.L.; Wood, C.W. and Delaney, D.P. (1993):** Determination of wheat nitrogen status with a handheld chlorophyll meter: Influence of management practices. *J. Plant Nutri.*,109 (16): 781-796.
- Reguera, M.; Conesa, C.M.; Gil-Gómez, A.; Haros, C.M.; Pérez-Casas, M.Á.; Briones-Labarca, V.; Bolaños, L.; Bonilla, I.; Álvarez, R.; Pinto, K.; Mujica, Á. and Bascuñán-Godoy, L. (2018):**The impact of different agroecological conditions on the nutritional composition of quinoa seeds. *PeerJ*, 6, e4442
- Richmond, W. (1973):** Preparation and properties of a cholesterol oxidase from *Nocardia sp.* and its application to the enzymatic assay of total cholesterol in serum. *Clin.Chem.*, 19 (12): 1350.
- Rotruck .J. ; T.; Pope, L.; Ganther, H. E. and Swanson, A. B. (1973):** Selenium: biochemical role as a component of glutathione peroxidase. *Science*, 179(4073): 588-590.
- Rubén, V. and Blanca, H.L. (2017):** Nutritional and biological value of quinoa (*Chenopodium quinoa* Willd.). *Current Opinion in Food Science*, 14: 1-6.
- Ruel. I.;Brisson, D .; Aljenedil, S .; Awan, Z.; Baass, A .; Bélanger, A.; Bergeron, J., Bewick. D.; Brophy, J.M. ; Brunham, L.R.; Couture, P et al., (2018):** Simplified Canadian definition for familial hypercholesterolemia. *Canadian Journal of Cardiology*, 34(9), 1210-1214.
- Ryan, E.; Galvin, K.; O'Connor, T. P.; Maguire, A. R. and O'Brien, N. M. (2007):** Phytosterol, squalene, tocopherol content and fatty acid profile of selected seeds, grains, and legumes. *Plant Foods for Human Nutrition*, 62(3), 85-91.

- Stapleton, P. A., Goodwill, A. G., James, M. E., Brock, R. W. and Frisbee, J. C. (2010):** Hypercholesterolemia and microvascular dysfunction: interventional strategies. *Journal of inflammation*, 7(1): 1-10.
- Thomas, G.; Simnadis, L. and Linda, C.T. (2015):** Physiological effects associated with Quinoa consumption and implications for research involving humans: a review. *Plant foods for human nutrition*, 70(3), 238-249.
- Wu, Y.; Qian, Y.; Pan, Y.; Li, P.; Yang, J.; Ye, X. and Xu, G.(2015):** Association between dietary fiber intake and risk of coronary heart disease: A meta-analysis. *Clinical nutrition*, 34(4), 603-611.
- Yao, T. and Rong, T. (2017):** Phytochemicals in quinoa and amaranth grains and their antioxidant, anti-inflammatory, and potential health beneficial effects: a review. *Molecular Nutrition & Food Research*, 61(7), 1600767.
- Zhu, N.; Sheng, S.; Li, D.; LAVOIE, E. J.; KARWE, M. V.; Rosen, R. T. and HO, C. T. (2001) :** Antioxidative flavonoid glycosides from quinoa seeds (*Chenopodium quinoa* Willd). *Journal of Food Lipids*, 8(1): 37-44.



## مكملات غذائية من مسحوق بذور الكينوا وتأثيرها على فرط كوليسترول الدم والأكسدة في الجرذان البيضاء

أسماء حسن عبد العظيم أحمد ، بسمة رمضان محمد خطيب

مدرس بقسم الاقتصاد المنزلي (تخصص التغذية وعلوم الأطعمة) - كلية التربية النوعية، جامعة المنوفية،  
مصر<sup>١</sup>، مدرس بقسم التغذية وعلوم الأطعمة - كلية الاقتصاد المنزلي - جامعة المنوفية، مصر<sup>٢</sup>

الهدف الرئيسي من هذا البحث هو استخدام مكملات غذائية من مسحوق بذور الكينوا تحت تركيزات مختلفة (١٥٠ و ٣٠٠ جم / كجم من الغذاء) لإعطاء مزيد من الحماية ضد مرض فرط كوليسترول الدم. تم استخدام عشرون من ذكور الفئران البيضاء يتراوح وزنها ما بين  $184 \pm 2$  جراماً في هذه الدراسة. تم تقسيم الفئران إلى ٤ مجموعات (٥ فئران في كل مجموعة) استخدمت واحدة منها كمجموعة ضابطة سالبة بينما تغذت الثلاث مجموعات الأخرى على وجبة محتوية على ١.٥٪ من الكولسترول بالإضافة إلى ١٠٪ من دهن الأغنام لمدة ١٥ يوماً وذلك للإصابة بمرض ارتفاع الكوليسترول ، تركت واحدة من هذه المجموعات كمجموعة ضابطة موجبة وتغذت المجموعتين الأخرتين على جرعتين من الكينوا (١٥٠ جم و ٣٠٠ جم / كجم من الوجبة) لمدة ٢٨ يوماً. تم تقدير المأخوذ من الطعام ووزن الجسم المكتسب ومعدل الاستفادة من الغذاء وكذلك وزن الأعضاء الداخلية، كما تم تقدير الكوليسترول الكلي والتراى جليسيريد والليوبروتين مرتفع الكثافة والليوبروتين منخفض الكثافة والليوبروتين منخفض الكثافة جدا وكذلك تم تقدير الانزيمات المؤكسدة (المالونالدهيد) وكذلك الانزيمات المضادة للاكسدة (السوبر اوكسيد ديسميوتيز والجلوتاثيون والكتايز). وقد أظهرت النتائج أن المعاملة ببذور الكينوا قد حسنت المعاملات السابقة وذلك بالمقارنة بالمجموعة الضابطة الموجبة وأفضل علاج لوحظ في المجموعة التي غذيت على ٣٠٠ جم/كجم من الغذاء. ولذلك، توصي الدراسة بأن مكملات الكينوا لها تأثيرات إيجابية كبيرة على التمثيل الغذائي وفرط كوليسترول الدم وامراض القلب.

**الكلمات المفتاحية:** الكينوا - ارتفاع الكولسترول - الأكسدة - الأنشطة المضادة للأكسدة - مستوى الدهون

