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Potential Effects of Ungerminated, and Germinated Quinoa Seeds (Chenopodium quinoa, W.) on Hypercholesterolemic Rats

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Abstract:
Several diseases in humans may have hypercholesterolemia as a precursor. Quinoa is a nutritional food with several properties that lower the risk of developing chronic diseases. This study aimed to determine how different doses of 5% and 10% powdered ungerminated and germinated quinoa affected hypercholesterolemic male albino rats. Thirty-six male albino rats weighing 140g±10g were divided into six groups, one of which was the negative control group (-ve). At the same time, the other five were given Triton-X-100 (100 mg/kg of the rat's weight b.w.) to cause hypercholesterolemia. The following analysis has measured blood glucose, renal functions (urea, uric acid, and creatinine), liver enzymes (alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), and lipid profile (total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL-c), low-density lipoprotein (LDL-c), very low-density lipoprotein (LDL-c) and atherogenic index (AI). According to the results, both ungerminated and germinated quinoa significantly improved in all biochemical parameters, including lipid profile. The most effective result was for 10% germinated quinoa. In conclusion, quinoa that has been germinated may be a potent nutraceutical therapeutic option for the treatment of hypercholesterolemic rats.

Key Words: *Hypercholesterolemia, Germination Seeds, Rats, Biochemical Analysis.*

Introduction

Hyperlipidemia is defined as an increase in one or more plasma lipids, including triglycerides, cholesterol, cholesterol esters, and phospholipids, as well as plasma lipoproteins, including very low-density lipoprotein and low-density lipoprotein, and a decrease in high-density lipoprotein levels (1). The oxidative modification of LDL, protein glycation, gluco-seauto oxidation with excess formation of free radicals, and lipid peroxidation products, which represent key risk factors for ischemic heart disorders, may be caused by

hypertriglyceridemia and hypercholesterolemia. Hypercholesterolemia also causes several human diseases or tissue damage (2). Familial hypercholesterolemia (FH), a hereditary disorder that accelerates arteriosclerotic cardiovascular disease, is characterized by high LDL-c plasma concentrations (ASCVD). On average, 1/250 persons carry the heterozygous FH mutation (3).

The use of medicinal herbs to treat hypercholesterolemia has expanded in recent years, as has consumer desire for meals that are nutritious, secure, natural, and quick to prepare (4). Quinoa (*Chenopodium quinoa,* W.), an annual dicotyledon that is normally herbaceous and grows to a height of 0.2 to 3.0 m, is a member of the *Amaranthaceae* family. It has been produced for many years in the Andean region as a staple food, and it is currently gaining popularity in the western region. Quinoa seeds' colors range from yellow to black and are produced by this plant. Although this plant is usually referred to as a pseudocereal, it is classified as a crop (5). One excellent example of a "functional food," which aims to reduce the risk of numerous diseases, is quinoa seeds. Its functional elements come from vitamins, minerals, antioxidants, and fatty acids that principally contribute to human nutrition, especially to protect cell membranes, with favorable results in neuronal processes of the brain. Its minerals also serve as cofactors in antioxidant enzymes, enhancing the usefulness of its abundant proteins. These seeds have an additional benefit over other plant meals for human nutrition in that contain phytohormones (6). Quinoa is free of gluten and other harmful ingredients and is a useful source of good-quality protein, fiber, carbohydrates, vitamins, minerals, phytochemicals, and bioactive peptides. The antinutritional components of quinoa, including saponins, tannins, and phytic acids, may decrease the bioavailability by forming insoluble complexes with minerals like zinc and iron. Reduction of these compounds can be enhanced by germination (7). It's a frequent practice to employ germination to enhance a seed's nutritional content (8). The biochemical component of the grains undergoes significant changes during the germination process: The starch reserves are depleted by the action of the enzyme amylase, which acts on the granule's surface and creates pores; the nitrogen-containing fractions are shifted toward oligopeptides free amino acids; and the amino acid composition also changes. Triglycerides start to hydrolyze, and the proportion of saturated to unsaturated fatty acids changes. At the same time, the level of tannins, phytates, and other antinutritional substances falls dramatically and bioactive substances like phenols, phytosterols, and folates rise. Because practically all nutrients are fully available in sprouted grains and a variety of antioxidants are present in increased amounts, sprouts can be considered "functional meals." (9). Quinoa seeds should be suggested for commercial production in Egyptian industries, food, and pharmaceuticals since they can improve blood lipid levels, provide greater protection against hypercholesterolemia disease, and lessen risks to the liver and kidneys (10). (11) observed that the rat's group which fed on a high cholesterol diet supplemented with quinoa seeds powder at 40% showed the highest outcomes in lipid fractions for all treated groups since this therapy reduced serum cholesterol and triglyceride levels. Furthermore, the findings confirmed (12), who stated that increasing the HDL-c ratio is one of the most important needs for an anti-hypercholesterolemia treatment. The results were consistent with those of (13), who

discovered that rats given a high-cholesterol meal along with two different amounts of quinoa displayed lower mean lipid profile values than the positive control group. This may be because there was less cholesterol absorbed and synthesized, and more cholesterol and bile acid were excreted in the feces.

The current study aimed to investigate the biochemical changes that might occur in rats fed a high-cholesterol diet, resulting in hypercholesterolemia, as well as the potential effects of quinoa seed powder supplements on adult male albino rats.

Material & Methods

Materials

Source of quinoa seeds

The quinoa (*Chenopodium quinoa,* Willd.), seeds, was purchased at the herbalist in Cairo City, Cairo Governorate, Egypt.

Triton- X-100

Sigma Chemical Company provided the compound Triton X-100 (a polyethylene glycol-based non-ionic surfactant)-induced hypercholesterolemia.

Experimental animals

A total of forty-eight adult normal male albino rats "Sprague Dawley" strain weighing 140 g was provided by the Vaccine and Immunity Organization, Ministry of Health, Helwan Farm, Cairo, Egypt.

The chemicals and kits

Egypt's Sigma Chemical Company provided pure white crystalline cholesterol powder. While casein, cellulose, choline chloride powder, and DL methionine powder were provided by Morgan Co. in Cairo, Egypt. The chemical kits (TC, TG, HDL-c, ALT, AST, ALP, urea, uric acid, and creatinine) used in this study were given by El-Gomhoria Company for Trading Drugs, Chemical, and Medical Instruments in Cairo, Egypt.

Methods

Preparations of germination quinoa seeds

Quinoa seeds were germinated according to **(14)** with minor adjustments, with the germination temperature being 20 °C and the germination durations being zero hr. (used as a germination control sample) and 48 hr. To inhibit enzyme activity, the seeds were drying time at 40 °C. Finally, the seeds were ground to a fine powder in an air mill, then blended with a high-speed mixer (Molunix, Al-Araby Company, Benha, Egypt) and stored in polyethylene bags at freezing temperatures until use.

The induction of experimental hypercholesterolemia:

According to the **(15)** method, after an overnight fast of 18 hours, normal healthy male albino rats were given a single intraperitoneal injection of freshly produced Triton X-100 (100 mg/kg b.w.) in physiological solution. The cholesterol level in the blood should have been measured after the injection of Triton X-100.

Experimental design

The research was carried out and approved at Menoufia University's Animal House, Department of Nutrition and Food Science, Faculty of Home Economics.

Thirty-six adult male white albino "Sprague Dawley" rats, 10 weeks old, weighing (140), was used in this research. All rats were given a casein diet prepared in accordance with **(16)** for 7 days as a means of adaptation. Following this period of adaptation, rats were separated into 6 groups of six each as follows: Group (1): Rats were fed on basal diet only as negative control. Group (2): Hypercholesterolemic rats were fed on basal diet only as a positive control group. Group (3): Hypercholesterolemic rats were fed on basal diet and un-germinated quinoa powder by 5% of kg/diet/day. Group (4): Hypercholesterolemic rats were fed on basal diet and un-germinated quinoa as powder by 10% of kg/diet/day. Group (5): Hypercholesterolemic rats were fed on basal diet and germinated quinoa as powder by 5% of kg/diet/day. Group (6): Hypercholesterolemic rats were fed on basal diet and germinated quinoa as powder by 10% of kg/diet/day. The experiment lasted for 28 days; at the end of the trial, each rat was weighed independently, slaughtered, and blood samples were taken.

Blood sampling

Rats were fasted for 12 hours at the end of the experiment (which lasted 28 days), after which they were scarified. For serum separation, blood samples were taken from the portal vein and placed in dry, clean centrifuge tubes. The blood samples were centrifuged for 10 minutes at 4000 rpm to separate the serum **(17)**. At -18 °C, serum samples were kept frozen for chemical analysis.

Biochemical analysis

To measure serum total cholesterol, the colorimetric technique described by **(18)**. Enzymatic measurement of serum triglycerides was performed using kits in accordance with **(19)** and (20). The procedure described by **(21&22)** and used to determine HDL-c. According to **(23)** the VLDL-c was computed in mg/dl using the following formula: Triglycerides (mg/dl) = VLDL-c / 5. According to **(23)** LDL-c was determined in mg/dl as follows: LDL-c (mg/dl) = Total cholesterol - (HDL + VLDL-c).

Serum glucose was measured using the modified kinetic method **(24)** by using kit supplied by spin react. Spain.

The procedures given by **(25, 26 and 27)**, respectively, were used to measure the serum alanine aminotransferase (ALT), serum aspartate aminotransferase (AST), and serum alkaline phosphatase (ALP).

According to the method, serum urea and serum creatinine were determined using an enzymatic technique **(28) and (29)**. While serum uric acid was measured using a calorimeter using the method of **(30)**.

Statistical analysis

The data were analyzed using a completely randomized factorial design **(31)** when a significant main effect was detected; the means were separated with the Student-Newman-Keuls Test. Using the Costat Program, differences between treatments ($P \leq 0.05$) were considered significant. One Way ANOVA was used to assess the biological results.

Results and Discussion

The data shown in Table (1) illustrates how different concentrations of ungerminated and germinated quinoa powder affected the serum total cholesterol and triglycerides of

hypercholesterolemic rats. Total cholesterol levels in the negative control group were 104.50 mg/dl, whereas those in the positive control group were 236.15 mg/dl, with significant differences. (P≤0.05).

However, among the treated groups, the hypercholesterolemic group rats fed 10% germinated quinoa powder had the lowest levels of total cholesterol, while the hypercholesterolemic group rats fed 5% ungerminated quinoa powder had the highest levels significantly (P≤0.05), with mean value of 201.40 mg/dl, respectively. When compared to the control positive group (236.15 mg/dl), group 6 (10% germinated quinoa powder, 116.50 mg/dl) had the lowest serum total cholesterol levels.

In terms of triglycerides, the data revealed a significant difference (P≤0.05) between the positive control group and the negative control group. The relative mean values were 181.00 and 96.50 mg/dl. For the treated groups, rats in the hypercholesterolemic group that received 10% germinated quinoa powder had the lowest triglyceride levels. However, hypercholesterolemic group rats fed 5% ungerminated quinoa powder showed the highest value with a significant difference (P≤0.05). The relative mean values were 103.75 and 149.75 mg/dl. Compared to the control positive group (181.00 mg/dl), group 6 had the lowest serum triglyceride levels (10% germinated quinoa powder, 103.75 mg/dl). Compared to the control positive group (181.00 mg/dl), group 6 had the lowest serum triglyceride levels (10% germinated quinoa powder, 103.75 mg/dl). These results are consistent with a study by (32) which discovered that quinoa seed consumption of 50 g /day for 12 weeks reduced serum TGs and, as a result, the prevalence of metabolic syndrome in overweight and obese individuals.

The capacity of sprouted quinoa to favorably relieve hyperlipidemia may be due to the rise and synergistic effects of various endogenous bioactive chemicals, which have allegedly been boosted by the germination process (33).

In addition, quinoa proteins had a better potential to bind bile acids, which has an impact on how well lipids are absorbed. An integral component of intestinal lipid absorption is the emulsification of fats by bile acids (34).

Table (1) Effect of ungerminated and germinated quinoa powder on serum total cholesterol, and triglycerides of hypercholesteremic rats

Parameters Groups	Total cholesterol mg/dl	Triglycerides mg/dl
G_1 Negative control	104.50^{f}±0.31	96.50^{f}±0.25
G_2 Positive control	236.15^{a}±0.82	181.00^{a}±0.71
G3 (5%Ungerminated quinoa)	201.40^{b}±0.64	149.75^{b}±0.50
G4 (10% Ungerminated quinoa)	175.35^{c}±0.60	123.59^{c}±0.31
G5 (5% Germinated quinoa)	137.25^{d}±0.52	119.65^{d}±0.41
G6 (10% Germinated quinoa)	116.50^{e}±0.40	103.75^{e}±0.37
LSD (P≤ 0.05)	3.640	3.461

Each value is represented as mean ± SD; means in the same column with different letter are significantly different (P≤0.05).

The effects of different levels of ungerminated and germinated quinoa on the high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), and very low-density lipoprotein cholesterol (VLDL-c) of hypercholesteremic rats are shown in Table (2). The collected data showed that there were significant differences (P≤0.05) in the levels of HDL-c between the positive control group and the negative control group, with the positive control group having the lowest levels. There were 51.13 and 27.09 mg/dl on average, respectively. The highest levels of HDL-c were found in hypercholesteremic groups in 10% germinated quinoa, whereas the lowest levels were found in 5% ungerminated quinoa. These differences were significantly at (P≤0.05), and the values were 47.50 and 38.14 mg/dl, respectively.

As for LDL-c, the positive control group had the highest levels of LDL-c, whereas the negative control group had the lowest levels, with a significant difference (P≤0.05), which were 172.86 mg/dl and 34.07 mg/dl, respectively. On the other hand, in hypercholesteremic groups fed 5% ungerminated quinoa had the highest levels of LDL-c, whereas 10% germinated quinoa had the lowest, with a significant difference (P≤0.05). The average levels were 48.25 mg/dl and 133.31 mg/dl, respectively.

With statistically significant differences (P≤0.05), it can be stated that the positive control group had the highest levels of VLDL-c, whereas the negative control group had the lowest levels. There were 36.20 and 19.30 mg/dl on average, respectively. The 5% ungerminated quinoa group had the highest hypercholesteremic VLDL-c values, whereas the 10% germinated quinoa powder group had the lowest levels, with significant differences (P≤0.05), which were 29.95 and 20.75 mg/dl, respectively.

According to the findings, the atherogenic index (AI) values between the positive control group had the greatest levels of the compared to the negative control group, which showed a significant difference (P≤0.05), which were 7.72 and 1.04%, respectively.

The highest AI values of the hypercholesteremic groups were found in 5% ungerminated quinoa, while the lowest were found in 10% germinated quinoa powder. These differences were significant (P≤0.05), and they were 4.28 and 1.45%, respectively. These results are consistent with those of (11), who discovered that the mean values of the lipid profiles of the rats fed a high-cholesterol diet supplemented with quinoa seeds powder at 30% and 40% were lower than those of the positive control group. A decrease in cholesterol absorption and production as well as an increase in bile acid and cholesterol excretion from the faeces may be related to this variation.

According to the current study, daily quinoa eating may support older adults who live independently experience favorable (i.e., reduced), significant improvements in body weight, BMI, and serum levels of total cholesterol and LDL-c. Longer-term studies should now be conducted to confirm this finding (35).

Table (3) shows the effect of ungerminated, and germinated quinoa as powdered on hypercholesterolemic rats on fasting serum blood glucose levels. There are significant differences (P≤0.05) between the negative control and positive control groups, with mean values of 117.15 and 201.52 mg/dl, respectively. For all the treated groups, the hypercholesterolemic group of rats had the lowest blood glucose levels when they were fed

on 10% germinated quinoa powder. While the highest value was seen in rats with hypercholesterolemic that received 5% ungerminated quinoa powder, which were 131.10 and 173.00 mg/dl, respectively with significant difference (P≤0.05). These findings are consistent with those of (36), who found that diabetic rats given (10-40%) quinoa powder in their diets had significantly lower blood glucose levels than diabetic rats given a basal diet, demonstrating quinoa's significant advantages over other crops for human nutrition and health maintenance.

Table (2): Effect of ungerminated and germinated quinoa powder on lipid fractions of hypercholesteremic rats

Parameters Groups	HDL-c mg/dl	LDL-c mg/dl	VLDL-c mg/dl	AI %
G_1 Negative control	51.13^{a}±0.60	34.07^{f}±0.12	19.30^{e}±0.20	1.04^{e}±0.10
G_2 Positive control	27.09^{e}±0.11	172.86^{a}±4.63	36.20^{a}±0.51	7.72^{a}±0.65
G3 (5%Un-germinated quinoa)	38.14^{d}±0.43	133.31^{b}±2.32	29.95^{b}±0.40	4.28^{b}±0.41
G4 (10% Un-germinated quinoa)	42.65^{c}±0.52	107.98^{c}±1.27	24.72^{c}±0.30	3.11^{c}±0.40
G5 (5% Germinated quinoa)	44.19^{c}±0.35	69.13^{d}±0.50	23.93^{c}±0.40	2.11^{d}±0.35
G6 (10% Germinated quinoa)	47.50^{b}±0.21	48.25^{e}±041	20.75^{d}±0.33	1.45^{d}±0.23
LSD (P≤ 0.05)	2.401	2.713	1.125	0.725

HDL-c= High-density lipoprotein. LDL-c= Low-density lipoprotein. VLDL-c= Very low-density lipoprotein. Each value is represented as mean ± SD; means in the same column with different letter are significantly different (P≤0.05).

Table (3) Effect of ungerminated and germinated quinoa powder on serum glucose level of hypercholesteremic rats

Parameters Groups	Glucose level mg/dl
G_1 Negative control	117.15^{f}±0.21
G_2 Positive control	201.52^{a}±0.63
G3 (5%Ungerminated quinoa)	173.00^{b}±0.40
G4 (10% Ungerminated quinoa)	164.45^{c}±0.32
G5 (5% Germinated quinoa)	148.75^{d}±0.25
G6 (10% Germinated quinoa)	131.10^{e}±0.13
LSD (P≤ 0.05)	4.240

Each value is represented as mean ± SD; means in the same column with different letter are significantly different (P≤0.05).

The effect of ungerminated and germinated quinoa powders on the liver enzymes ALT, AST, and ALP in hypercholesteremic rats is shown in Table (4). The ALT liver enzyme levels between the negative and positive control groups differed significantly. The average values were respectively 62.34 and 129.75 U/L. The ALT liver enzyme was lowest in the hypercholesteremic group of rats fed on 10% germinated quinoa powder. 5% ungerminated quinoa powder was given to the hypercholesteremic group of rats, who had the highest value and the greatest difference (P≤0.05). The relative mean values were 72.34 and 115.90 U/L.

The results showed a significant difference (P≤0.05) between the negative and positive control groups for the liver enzyme AST. The corresponding mean values were 132.20 and 274.75 U/L. The AST enzyme was lowest in hypercholesteremic rats fed a 10% germinated quinoa powder than in any other treated group. But the highest value was observed in hypercholesteremic group rats fed 5% ungerminated quinoa powder. With a significant difference (P≤0.05), the mean values were 172.25 and 219.57 U/L, respectively.

It is evident that liver enzyme ALP differed significantly (P≤0.05) between the positive and negative control groups. The relative mean values were 134.55 and 290.00 IU/L. The rats in the hypercholesteremic group were fed 10% germinated quinoa powder, and their ALP enzyme levels were the lowest among the treatment groups. While the highest value observed was for the hypercholesteremic group of rats fed on 5% ungerminated quinoa powder with a significant difference (P≤0.05), values which were 163.75 and 263.65 U/L, respectively. These findings are consistent with (13) demonstrated that consuming high-cholesterol meals fortified with quinoa seeds powder at 30% and 40% led to a significant decrease in blood AST and ALT levels when compared to the positive control group, with a P-value of 0.05. The best liver function results were observed in hypercholesterolemic rats fed a diet enhanced with quinoa seeds powder at 40%.

Table (4) Effect of ungerminated and germinated quinoa powder on liver functions of hypercholesteremic rats

Parameters Groups	ALT U/L	AST U/L	ALP U/L
G_1 Negative control	62.34^{f}±0.11	132.20^{f}±0.25	134.55^{f}±0.40
G_2 Positive control	129.75^{a}±0.70	274.75^{a}±0.81	290.00^{a}±0.86
G3 (5%Un-germinated quinoa)	115.90^{b}±0.60	219.57^{b}±0.63	263.50^{b}±0.71
G4 (10% Un-germinated quinoa)	100.87^{c}±0.50	189.40^{c}±0.51	223.65^{c}±0.60
G5 (5% Germinated quinoa)	85.00^{d}±0.30	181.00^{d}±0.44	197.80^{d}±0.41
G6 (10% Germinated quinoa)	72.34^{e}±0.14	172.25^{e}±0.54	163.75^{e}±0.53
LSD (P≤ 0.05)	2.4304	2.815	2.713

ALT= alanine aminotransferase. AST = aspartate aminotransferase. ALP= alkaline phosphatase.
Each value is represented as mean ± SD; means in the same column with different letter are significantly different (P≤0.05).

The effects of different levels of ungerminated, germinated quinoa powder on hypercholesterolemic rats' kidney functions (serum urea, uric acid, and creatinine) are shown in Table (5) shows. It should be noted that the positive control group's serum urea was much higher than the negative control group's serum urea, which were 24.80 and 52.20 mg/dl, respectively. The hypercholesterolemic group rats given 5 % ungerminated quinoa powder had the highest serum urea level of all the treated groups. When fed 10% germinated quinoa powder, hypercholesterolemic rats' lowest recorded value showed a significant difference (P≤0.05), which were 48.64 and 36.68 mg/dl, respectively.

When it came to serum uric acid, the findings showed that the positive control group's value was significantly (P≤0.05) higher than that of the negative control group, which were 5.74 and 2.87 mg/dl, respectively. On the other hand, among all treated groups,

hypercholesterolemic rats exhibited the highest serum uric acid levels when given 5% ungerminated quinoa powder, with the lowest value being achieved for hypercholesterolemic group rats fed on 10% germinated quinoa powder, which were 5.04 and 3.54 mg/dl, respectively, with significant differences (P≤0.05).

The serum creatinine levels of the negative control groups were significantly (P≤0.05) lower than those of the positive control groups. The relative mean values were 1.31 and 0.70 mg/dl. The hypercholesterolemic group rats given 5 % ungerminated quinoa powder had the highest serum creatinine level of the treatment group. While the lowest result was reported for hypercholesterolemic rats fed a 10% germinated quinoa powder, the mean values were 1.00 and 0.79 mg/dl, respectively, with significant differences (P≤0.05). These findings concurred with those of (37), who found that elevated cholesterol levels may also contribute to renal failure, which is indicated by high creatinine levels.

According to (38) the renal system may benefit from quinoa seeds, which also reduce oxidative stress. For those with hyperuricemia, adding quinoa seed to the diet may be beneficial.

Table (5): Effect of un-germinated and germinated quinoa powder on kidney functions of hypercholesteremic rats

Parameters Groups	Urea mg/dl	Uric acid mg/dl	Creatinine mg/dl
G_1 Negative control	24.80^{f}±0.15	2.87^{b}±0.26	0.70^{b}±0.13
G_2 Positive control	52.20^{a}±0.61	5.74^{a}±0.50	1.31^{a}±0.40
G3 (5%Un-germinated quinoa)	48.64^{b}±0.50	5.04^{a}±0.41	1.00^{a}±0.41
G4 (10% Un-germinated quinoa)	41.29^{d}±0.33	4.61^{a}±0.32	0.99^{a}±0.35
G5 (5% Germinated quinoa)	43.19^{c}±0.42	4.24^{b}±0.31	0.90^{b}±0.38
G6 (10% Germinated quinoa)	36.68^{e}±0.29	3.54^{b}±2.14	0.79^{b}±0.22
LSD (P≤ 0.05)	1.203	1.415	0.370

Each value is represented as mean ± SD; means in the same column with different letter are significantly different (P≤0.05).

Conclusions

Quinoa that has been germinated has improved serum lipid profiles and reduced hyperglycemia, liver enzymes, and renal functions. As a result, this study suggests the possible use of quinoa seeds that have been germinated as dietary supplements that may enhance foods like baked products and lower risk factors for chronic diseases, particularly hypercholesterolemia and obesity.

References

1. Mishra, P.R.; Panda, P.K.; Apanna, K.C. and Panigrahi, S. Evaluation of acute hypolipidemic activity of different plant extracts in Triton WR-1339 induced hyperlipidemia in albino rats. *Pharmacology online.,* (2011); 3: 925-934.
2. Olorunnisola, O.; Bradley, G. and Afolayan, A. Protective effect of *T. violacea* rhizome extract against hypercholesterolemia-induced oxidative stress in Wistar rats. *Molecules,* (2012); 17: 6033-6045.

3. Roman, I. and Ramos, R. Representing the XULA and ISV-Girona groups. Familial hypercholesterolemia in a European Mediterranean Population-Prevalence and clinical data from 2.5 million primary care patients. *Journal of Clinical. Lipidology,* (2017); 11: 1013-1022.

4. Yang, H. and U. Ludewig, Lysine catabolism, amino acid transport, and systemic acquired resistance: What is the link. *Plant Signal Behav.,* (2014); 9: e28933.

5. Abugoch-James, L.E. Quinoa (*Chenopodium quinoa,* Willd.): Composition, chemistry, nutritional, and functional properties. *Adv. Food Nutr. Res.*, (2009); 58: 1-31.

6. Vega-Gálvez, A.; Miranda, M.; Vergara, J.; Uribe, E. and Puente, L. et al. Nutrition facts and functional potential of quinoa (*Chenopodium quinoa,* Willd.), an ancient Andean grain: A review. *J. Sci. Food Agric.,* (2010); 90:2541-2547.

7. Jarvis, D. E., Ho, Y. S., Lightfoot, D. J., Schmöckel, S. M. et al. The genome of *Chenopodium quinoa*. *Nature,* (2017); 542 (7641): 307-312.

8. Benincasa, P.; Falcinelli, B.; Lutts, S. and Stagnari, F. et al. Sprouted grains: A comprehensive review. *Nutrients,* (2019); 11, 421.

9. Guardianelli, L.M.; Salinas, M.V.; Brites, C. and Puppo, M.C. Germination of white and red quinoa seeds: Improvement of nutritional and functional quality of flours. *Foods,* (2022); 11: 1-18.

10. Halaby, M.S.; Abdel-Rahman, M.K. and Hassan, R.A. Protective influence of quinoa on hypercholesterolemia in male rats. *Current Science International,* (2017); 6 | (1): 259-270.

11. Zevallos, V.; Herencia, L.; Chang, F.; Donnelly, S. et al. Gastrointestinal effects of eating quinoa (*Chenopodium quinoa* Willd.) in celiac patients. *Am. J. Gastroenterol.,* (2014); 109: 270-278.

12. Wang, L.; Sun, J.; Yi, Q.; Wang, X. and Ju, X. Protective effect of polyphenols extracts of adlay (*Coix lachryma*-jobi L. var. ma-yuen Stapf) on hypercholesterolemia-induced oxidative stress in rats. *Molecules,* (2012); 17(8): 8886-8897.

13. Del Hierro, J.N.; Hidalgo, G.C. and Reglero, G. The hydrolysis of saponin-rich extracts from fenugreek and quinoa improves their pancreatic lipase inhibitory activity and hypocholesterolemic effect. Food Chemistry, (2021); 338 (5):128113.

14. Guardianelli, L.M.; Salinas, M.V. and Puppo, M.C. Chemical and thermal properties of flours from germinated amaranth seeds. J. Food Meas. Charact., (2019); 13: 1078-1088.

15. Venkatesham, A.; Keshetty, V.; Gudipati, R. and Kandhukari, J.M., Antihyprlipidemic activity of methanolic extract of garlic (*Allium sativum,* L.) in Triton X-100 induced hyperlipidemic rats. *Journal of Pharmacy Research,* (2009); 2: 777-780.

16. Reeves, P.G.; Nielsen, F.H. and Fahmy, G.C. Reported of the American Institute of Nutrition adhocwriling committee on the reformulation of the AIN -76 a Rodent diet. *Journal Nutrition,* (1993);123:1939-19351.

17. Schermer, A. The Blood Morphology of Laboratory Animal. Longmans, Printed in Great Britain, *Green and Co. Ltd.,* (1967); pp.350.

18. Thomas, L. Labor and diagnose, 4th Ed. Marburg: *Die Medizinischi Verlagsgesellschaft*, (1992), (Chemical Kits).

19. Young, D. Effects of drugs on clinical laboratory tests. *Pestaner, L. Clin. Chem.,* (1975), 21 (5): 14- 32. (Chemical Kits).
20. Fossati, P. and Pricipe, I. Determination of serum triglycerides. *Clin. Chem.,* (1982), 28: 2077.
21. Friedwaid, W.T. Determination of HDL. *Clin. Chem.,* (1972), 18: 499.
22. Grodon, T. and Amer, M. Determination of HDL. *Clin. Chem.,* (1977), 18: 707.
23. Lee, R. and Nieman, D. Nutrition Assessment. 2nd Ed. *Mosby, Missouri,* (1996), U.S.A.
24. Kaplan, L.A. Clinical Chemistry. The C.V. *Mosby Co. St Louis. Toronto. Princent.,* (1984), 1032-1036.
25. Hafkenscheid, J.C. Determination of GOT. *Clin. Chem.,* (1979), 25:155.
26. Henary, R.J. Clinical Chemist: Principals and Techniques. 2nd, Edition, *Hagerstoun (MD), Harcer, ROW,* (1974), P. 882.
27. Moss, D.W. Alkaline phosphatase isoenzymes. *Clin. Chem.,* (1982), 28: 2007-2016.
28. Patton, C.J. and Crouch, S.R. Enzymatic determination of urea. *J. of Anal. Chem.,* (1977), 49: 464-469.
29. Henry, R.J. Clinical Chemist: Principles and Techniques, 2nd Edition, *Hagerstoun (MD), Harcer,* (1974), *ROW*, 882.
30. Barham, D. and Trinder, P. Determination of uric acid. *Analyst,* (1972), 97: 142.
31. SAS. SAS Users Guide: Statistics version 5th Ed. SAS. *Institute Inc.,* (1988), Cary N.C.
32. Navarro-Perez, D.; Radcliffe, J.; Tierney, A. and Jois, M. Quinoa seed lowers serum triglycerides in overweight and obese subjects: A dose-response randomized controlled clinical trial. Curr. Dev. Nutr., (2017); 1 (9): 1-9.
33. Jung, E.H.; Kim, S.R.; Hwang, I.K. and Ha, T.Y. Hypoglycemic effects of a phenolic acid fraction of rice bran and ferulic acid in C57BL/KsJ-db/db mice, *J. Agric. Food Chem.,* (2007); 55: 9800-9804.
34. Foucault, A.S.; Even, P.; Lafont, R.; Dioh, W. and Veillet, S. et al. Quinoa extract enriched in 20-hydroxyecdysone affects energy homeostasis and intestinal fat absorption in mice fed a high-fat diet. *Physiol. Behav.,* (2014); 128: 226-231.
35. Pourshahidi, L.K.; Caballero, E.; Osses, A. and Hyland, B.W. et al. Modest improvement in CVD risk markers in older adults following quinoa (*Chenopodium quinoa,* Willd.) consumption: a randomized-controlled crossover study with a novel food product. *Eur. J. Nutr.,* (2020); 59: 3313-3323.
36. AL-Sayed, H.M.; Zidan, N.S. and Abdelaleem, M. A. Nutritional applications of quinoa seeds (*Chenopodium quinoa,* W.) and their effect on diabetic rats. *Int. J. Pharm. Res. Allied Sci.,* (2019); 8 (4): 23-36.
37. Barakat, L., Hypolipidemic and antiatherogenic effects of dietary chitosan and wheat bran in high fat- high cholesterol fed rats. *Australian Journal of Basic and Applied Sciences,* (2011); 5 (10): 30-37.
38. El-Kewawy, H.E. and Morsy, G.M. Beneficial role of quinoa and Nigella sativa seeds as antihyperuricemia in rats. *Bulletin of the National Nutrition Institute of the Arab Republic of Egypt,* (2019); 53: 62-84.

التأثيرات المحتملة لبذور الكينوا الغير منبتة والمنبتة على الفئران المصابة بارتفاع الكوليسترول

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الملخص العربي:

العديد من الأمراض التي تصيب الانسان قد تكون بسبب ارتفاع كوليسترول الدم. الكينوا غذاء له العديد من الخصائص التي تقلل من خطر الإصـابة بالأمراض المزمنة. الهدف من هذه الدراسـة معرفة تأثير تركيزات مختلفة ٥٪، ١٠٪ من مسحوق الكينوا غير المنبتة والمنبتة على ذكور الفئران البيضاء المصابة بارتفاع الكولسترول. تم استخدام ما مجموعه ٣٦ فأرًا من الذكور، يتراوح وزن كل منها بين ١٤٠ جم، في الدراسـة. تم تقسـيم إلى سـت مجموعات متسـاوية كل مجموعة بها سـتة فئران، مع ترك واحدة كمجموعة ضـابطة سـالبة، والخمس مجاميع الأخرى التي تم معاملتها بمادة تريتون اكس ١٠٠ (١٠٠ مجم / كجم من وزن الفأر) للحث على ارتفاع كوليسـترول الدم. الجلوكوز، وظائف الكلى (اليوريا، وحمض البوليك، والكرياتينين)، ووظائف الكبد (ALT، و AST، و ALP)، وصـورة دهون الدم (الكوليسـترول الكلي، والدهون الثلاثية، والبروتين الدهني عالي الكثافة، والبروتين الدهني منخفض الكثافة ، والبروتين الدهني منخفض الكثافة جدا ، ومؤشر تصلب الشرايين. وفقًا للنتائج المتحصل عليها، أدى إضافة كل من الكينوا غير المنبتة والمنبتة إلى تحسـن بشـكل ملحوظ في جميع التحاليل الكيميائية الحيوية، بما في ذلك الكوليسـترول الكلى، والدهون الثلاثية والبروتين الدهني عالي الكثافة ومنخفض الكثافة ومنخفض الكثافة جدا ومؤشر تصـلب الشرـايين. كانت النتيجة الأكثر فاعلية مع الكينوا المنبتة بنسبة ١٠٪، ونستخلص من هذا ان الكينوا المنبتة قد تكون خيارًا علاجيًا قويًا للتغذية لعلاج الفئران المصابة بارتفاع كوليسترول الدم.

الكلمات المفتاحية: بذور الحبوب، ارتفاع دهون الدم، الفئران، التحاليل الحيوية.