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

Asmaa. H. Ahmed¹ and Basma R. M. Khateib²
Dept. of Home Economics, Faculty of Specific Education, Menoufia University, Egypt¹, Dept. of Nutrition & Food Science, Faculty of Home Economics, Menoufia University, Egypt².

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 ± 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).

Biological Experiment

Twenty mature male albino rats weighing 184 ± 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°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).

\[
\text{LDL Concentration mg/dl} = \text{LDL} = \text{TC} - (\text{VLDL} + \text{HDL})
\]

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±1 g, 0.029±0.001 g and 15.5±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**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Control -ve (G1)</th>
<th>Control +ve (G2)</th>
<th>Quinoa 150 g/kg diet (G3)</th>
<th>Quinoa 300 g/kg diet (G4)</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>FI (g) Mean ± SD</td>
<td>12²±1.04</td>
<td>18&lt;sup&gt;a&lt;/sup&gt;±1</td>
<td>16&lt;sup&gt;b&lt;/sup&gt;±1</td>
<td>13&lt;sup&gt;c&lt;/sup&gt;±1</td>
<td></td>
<td>1.8</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-33.3%</td>
<td>-</td>
<td>-11.11%</td>
<td>-27.77%</td>
<td>-</td>
<td>--</td>
</tr>
<tr>
<td>FER (g) Mean ± SD</td>
<td>0.028&lt;sup&gt;a&lt;/sup&gt;±0.00</td>
<td>0.029&lt;sup&gt;b&lt;/sup&gt;±0.001</td>
<td>-0.027&lt;sup&gt;c&lt;/sup&gt;±0.002</td>
<td>-0.045&lt;sup&gt;d&lt;/sup&gt;±0.005</td>
<td></td>
<td>0.008</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-3.44%</td>
<td>-</td>
<td>-193.0%</td>
<td>-255.17%</td>
<td>-</td>
<td>--</td>
</tr>
<tr>
<td>BWG (%) Mean ± SD</td>
<td>9.78&lt;sup&gt;b&lt;/sup&gt;±3.08</td>
<td>15.5&lt;sup&gt;a&lt;/sup&gt;±0.78</td>
<td>-12.6&lt;sup&gt;c&lt;/sup&gt;±0.77</td>
<td>-16.8&lt;sup&gt;d&lt;/sup&gt;±0.378</td>
<td></td>
<td>3.1</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-36.9%</td>
<td>-</td>
<td>-181.29%</td>
<td>-208.38%</td>
<td>-</td>
<td>--</td>
</tr>
</tbody>
</table>

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 ≤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**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control -ve (G1)</th>
<th>Control +ve (G2)</th>
<th>Quinoa 150 g/kg diet (G3)</th>
<th>Quinoa 300g/kg diet (G4)</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart (g) Mean±SD</td>
<td>0.16±0.023</td>
<td>0.35±0.05</td>
<td>0.263±0.023</td>
<td>0.193±0.023</td>
<td>0.062</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-54.29%</td>
<td>-</td>
<td>-25.71%</td>
<td>-0.45%</td>
<td>-</td>
</tr>
<tr>
<td>Kidney (g) Mean±SD</td>
<td>0.393±0.035</td>
<td>1.063±0.125</td>
<td>0.533±0.041</td>
<td>0.393±0.057</td>
<td>0.107</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-63.20%</td>
<td>-</td>
<td>-50.00%</td>
<td>-63.20%</td>
<td>-</td>
</tr>
<tr>
<td>Liver (g) Mean±SD</td>
<td>1.85±0.052</td>
<td>3.656±0.060</td>
<td>2.94±0.202</td>
<td>2.24±0.061</td>
<td>0.235</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-49.45%</td>
<td>-</td>
<td>-19.97%</td>
<td>-38.79%</td>
<td>-</td>
</tr>
</tbody>
</table>

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 (T.C) of hypercholesterolemic rat. Control (+ve) groups showed significant increase in total cholesterol as compared to healthy rats which were 174.0±3.60mg /dl and 110 ±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±3.60) and (77.33±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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control -ve (G1)</th>
<th>Control +ve (G2)</th>
<th>Quinoa 150 g/kg diet (G3)</th>
<th>Quinoa 300g/kg diet (G4)</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mg/dl)</td>
<td>110 ±2</td>
<td>174 a±3.60</td>
<td>144 b±3.60</td>
<td>123 c±3.60</td>
<td>3.31</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of change</td>
<td>-36.78%</td>
<td>--</td>
<td>-17.24%</td>
<td>-29.21%</td>
<td></td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>77.33±2.51</td>
<td>149 a±3.60</td>
<td>124.33b±4.50</td>
<td>93 c±3.60</td>
<td>8.039</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of change</td>
<td>-48.10%</td>
<td>--</td>
<td>-16.55%</td>
<td>-37.58%</td>
<td></td>
</tr>
<tr>
<td>Atherogenic Index (AI) (mg/dl)</td>
<td>1.13c±0.09</td>
<td>6.06d±0.805</td>
<td>3.53b±0.263</td>
<td>1.7a±0.235</td>
<td>0.925</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of change</td>
<td>-81.35</td>
<td>--</td>
<td>-41.7%</td>
<td>-71.94%</td>
<td></td>
</tr>
</tbody>
</table>

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

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

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 ± 3.50 and 63.00± 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±3.50 nmol/g as compared to hypercholestermic 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 hypercholesteromic rats in SOD which were 9.16 ± 1.04 and 2.96 ± 0.50 U/mg tissue, receptively. 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 ±2.00 and 59.00±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 ±1.00 and 19.50±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 quinoa300g/kg. diet which showed (CAT) enzyme value were 16.30 ± 1.52 U/g tissue.

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

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Control -ve (G1)</th>
<th>Control +ve (G2)</th>
<th>Quinoa 150 g/kg diet (G3)</th>
<th>Quinoa 300g/kg diet (G4)</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MDA (nmol/gtissue) Mean + SD</td>
<td>63.1±3.60</td>
<td>95.5±3.5</td>
<td>84.5±1.80</td>
<td>71.66±3.05</td>
<td>6.569</td>
</tr>
<tr>
<td>Percent of change</td>
<td>-34.30%</td>
<td>--</td>
<td>-11.51%</td>
<td>-24.96%</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SOD (U/mg tissue) Mean + SD</td>
<td>9.16±1.04</td>
<td>2.96±0.50</td>
<td>5.5±0.5</td>
<td>7±0.5</td>
<td>1.497</td>
</tr>
<tr>
<td>Percent of change</td>
<td>2.945%</td>
<td>--</td>
<td>85.81%</td>
<td>136.48%</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td></td>
<td>GSH (mg/g tissue) Mean + SD</td>
<td>94±2</td>
<td>59±1</td>
<td>72±3</td>
<td>86±2</td>
<td>4.75</td>
</tr>
<tr>
<td>Percent of change</td>
<td>59.32%</td>
<td>--</td>
<td>2203%</td>
<td>45.76%</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CAT (U/g tissue) Mean + SD</td>
<td>19.5±1.80</td>
<td>5±1</td>
<td>12±2</td>
<td>16.3±1.52</td>
<td>3.61</td>
</tr>
<tr>
<td>Percent of change</td>
<td>290%</td>
<td>--</td>
<td>140%</td>
<td>226%</td>
<td>--</td>
<td></td>
</tr>
</tbody>
</table>

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 γ-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


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.


Lamothe, L.M.; Sri Chuwong, 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.


المكملات غذائية من مسحوق بذور الكينوا وتأثيرها على فرط كولسترول الدم والأسكدرة في الجرذان البيضاء

أسماء حسن عبد العظيم أحمد، بسمة رمضان محمد خطيب

المقدمة

الهدف الرئيسي من هذا البحث هو استخدام مكملات غذائية من مسحوق بذور الكينوا تحت ترقيم مختلفة (100 جم / كجم من الغذاء) لإعطاء مزيد من الحماية ضد مرض فرط كولسترول الدم. تم استخدام جشر من ذكور القران البيضاء يتراوح وزنها ما بين 184 ± 2 جرامًا في هذه الدراسة. تم تقسيم القران إلى 4 مجموعات (5 قرَن في كل مجموعة) استخدمت واحدة منها كمجموعة ضابطة سلبية بينما تغذت الثلاث مجموعات الأخرى على وجبة محتوية على 1.5% من الكولسترول بالاضافة إلى 10% من دهن الأغذية لمدة 15 يومًا وذلك للإصابة بمرض ارتفاع الكولسترول، تركت واحدة من هذه المجموعات كمجموعة ضابطة موجبة وتحذت المجموعتين الاحترتين على جرعتين من الكينوا (100 جم و300 جم / كجم من الوجبة) لمدة 28 يومًا. تم تقدير المأخوذ من الطعام وزن الجسم المكشوف معبدة الاستفادة من الغذاء وكذلك وزن الأعضاء الداخلية، كما تم تقدير الكولسترول الكلي والتراب جليسريد والليبيدويترات مرتقب الكثافة والليبيدويترات مخفض الكثافة جدادًا (كما تقدم الزيادات المؤكدة (الكالونيديه) ) وكذلك الزيادات المؤكلة للكسدرة (السوير أوكسيد ديسومتريز والجلوتانين والكتاب) وأظهر أن النتائج أن معاملة بذور الكينوا قد حسنت المعاملات السابقة وذلك بالمقارنة بالمجموعة الضابطة الموجبة وأفضل علاج لوحظ في المجموعة التي غذت على 300 جم/كجم من الغذاء. ولذلك، توصي الدراسة بأن مكملات الكينوا لها تأثيرات إيجابية كبيرة على التمثيل الغذائي وفرط كولسترول الدم وأمراض القلب.

الكلمات المفتاحية: الكينوا - ارتفاع الكولسترول - الأكسدرة - الأمثلة المستحقة للأكسدرة - مستوى الدهون