RESEARCH ARTICLE

Hanaa M. Serag

EFFICACY OF QUINCE AND DILL ON EXPERIMENTAL HYPERCHOLESTEROLEMIA IN MALE RATS

ABSTRACT:
The main purpose of this study was to evaluate the effectiveness of quince fruit powder and dill leaves extract in the treatment of hypercholesterolemia induced experimentally by feeding male albino rats with 0.5% w/w cholesterol in diet for 4 weeks. The results revealed that the cholesterol-fed rats were characterized by high serum levels of total lipids, total cholesterol, triglycerides, LDL-cholesterol and VLDL-cholesterol, as well as a low level of HDL-cholesterol. Also an elevation in LDL/HDL-cholesterol and a reduction in HDL-cholesterol/total cholesterol ratio. The present results also recorded highly significant increase in the indicative enzymes of hepatic and cardiac activities, aspartate aminotransferase (AST), alanine aminotransferase (AST), and creatine kinase (CK) in hypercholesterolemia rats. Moreover, the hypercholesterolemia diet produced oxidative stress as achieved by significant increase in liver and heart thiobarbituric acid reactive substances (TBARS) indicating increase in lipid peroxidation (LPO), in addition, a significant decrease in liver and heart glutathione content (GSH) and superoxide dismutase (SOD). However, the dietary supplementation with quince (10 % w/w in diet) or orally administration of dill at a dose of 50 mg/kg BW/ day for 4 weeks to hypercholesterolemic rats, showed a remarkable amelioration in the lipid profiles, hepatic and cardiac enzymes activities as well as oxidative stress markers. Hence the present findings give an overview about the beneficially intake of quince and dill as hypocholesterolemic agents.

KEY WORDS:
Quince (Cydonia oblonga), Dill (Anethum graveolens), Hypercholesterolemia, Oxidative stress.

CORRESPONDENCE:
Hanaa M. Serag
Department of zoology, Faculty of science, Mansoura University, Mansoura, Egypt
E-mail: dr.hanaaserag@gmail.com

ARTICLE CODE: 15.01.15

INTRODUCTION:
Hypercholesterolemia is a major risk factor of cardiovascular disease that remains the most common cause of death in several developing countries (Goldstein et al., 2001; Parsaee et al., 2006). Cardiovascular disease is characterized by the accumulation of cholesterol, lipid peroxides, and oxyysterols in the arterial walls. Lipoprotein retention in the arterial wall, as well as lipoprotein oxidation are two major hypothesis attributed to the pathogenesis of atherosclerosis (Berliner and Heinecke, 1996).

The association between a diet rich in fruits and vegetables and a decreased risk of cardiovascular diseases is supported by considerable epidemiological evidence (Hertog et al., 1993). The protection that fruits and vegetables provide against some diseases has been attributed to several antioxidant compounds, such as ascorbic acid, α-tocopherol and β-carotene (Cao et al., 1996). However, these compounds are not the only ones contributing to the antioxidant activity of these fruits and vegetables but also many studies showed that the presence of polyphenol compounds, such as flavonoids also contribute to their beneficial effects (Vinson et al., 2005). Flavonoids are used as substitutes for synthetic antioxidant (Maron, 2004)

Quince (Cydonia oblonga) is a tree belonging to the Rosaceae family, different parts of quince have been used as traditional remedies for cardiovascular, respiratory, and gastrointestinal and urinary tract symptoms. Oliveira et al. (2007) have showed that
quince is an excellent natural source of phenolic acids and flavonoids, which are considered potent antioxidants. Jouyban et al. (2011) revealed that the fruit and leaves of quince have cell-protecting properties owing to the abundance of antioxidants they contain. Nowadays, quince fruit is recognized as a good, cheap and important dietary source of health promoting compounds, due to its biologically active phytochemicals which are able to operate as reducing agents, hydrogen donors, free radicals scavengers, and singlet oxygen quenchers, and therefore, as cell savours, these phytochemical are characterized by their antioxidant, antimicrobial, and anti-ulcerative properties (Fattouch et al., 2007).

Many plant extracts have been shown to have hypcholesterolemic activity in rats and the effects of several extracts have been described (Zhang et al., 2002). Anethum graveolens known as dill is used both medicinally and as an aromatic herb and spice and cookery. The presence of flavonoids, phenolic compounds and essential oil in dill has been reported (Ishikawa et al., 1974). Some pharmacological effects of the plant such as antimicrobial, antispasmodic, anti-hypercholesterolemic and anti-hyperlipidemic activities also reported (Yazdanparast and Bahramikia, 2008). Bahramikia and Yazdanparast (2009), concluded that dill extract, beside its hypolipidemic property, it could protect the liver against the high fat diet--induced oxidative damage in rats.

The present study aims to clarify the cholesterol lowering efficacy of quince and dill and their roles in the improvement of hepatic and cardiac enzymes activity as well as the oxidative stress induced by hypercholesterolemia in male rats.

**MATERIAL AND METHODS:**

**Chemicals:**

Cholesterol was purchased from Sigma Chemical Co. (St. Louis MO). Quince fruits was purchased from the market and then prepared in powder form; the powdered quince was given to rats in their diet (10% w/w) for four weeks. Dill leaves was purchased from the market and then water extract prepared and given orally at a dose of 50 mg/kg BW/day for four weeks. Dill leaves was purchased from the market and then water extract prepared and given orally at a dose of 50 mg/kg BW/day for four weeks.

**Animals and design of the experiment:**

Male albino rats (Rattus rattus) weighing 120-140 g were used in the present study. Rats were placed in separate cages and allowed food and water ad libitum. They were kept under suitable air flow and temperature during the whole period of experimentation.

Rats were divided into six groups (6 rats each) as follows:

<table>
<thead>
<tr>
<th>Group</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1</strong></td>
<td>Normal untreated control group. This group was fed on standard diet.</td>
</tr>
<tr>
<td><strong>2</strong></td>
<td>Quince-fed group, rats given quince fruit powder supplemented diet (10% w/w) for 4 weeks (Fattouch et al., 2007).</td>
</tr>
<tr>
<td><strong>3</strong></td>
<td>Dill-fed group, rats given orally water extract of dill leaves at a dose of 50 mg/kg BW/day for 4 weeks (Bahramikia et al., 2008).</td>
</tr>
<tr>
<td><strong>4</strong></td>
<td>Cholesterol-fed group, rats were fed on a normal diet supplemented with cholesterol (0.5% w/w) for 4 weeks to induce hypercholesterolemia (Moussavi et al., 1989), then continued to receive the cholesterol-supplemented diet for further 4 weeks.</td>
</tr>
<tr>
<td><strong>5</strong></td>
<td>Quince – fed hypercholesterolemic group. Rats were fed on cholesterol-supplemented diet (0.5% w/w) for 4 weeks to induce hypercholesterolemia and given quince fruit supplemented diet (10% w/w) besides the same diet of cholesterol for another 4 weeks.</td>
</tr>
<tr>
<td><strong>6</strong></td>
<td>Dill - fed hypercholesterolemic group. The rats were fed on cholesterol – supplemented diet (0.5% w/w) for 4 weeks to induce hypercholesterolemia and given orally dill leave extract at a dose of 50 mg/kg BW/day besides the same diet of cholesterol for another 4 weeks.</td>
</tr>
</tbody>
</table>

**Sampling:**

At the end of the experimental period, overnight fasted animals were sacrificed and blood samples were collected, sera were separated for biochemical measurements. Small pieces of liver and heart were carefully excised on ice, washed in ice-cold isotonic NaCl saline blotted dry and weighed. The tissue was immediately homogenized in ice-cold phosphate buffer (10% W/V) at 22,000 rpm for 20 S. The homogenates were centrifuged at 2000 x g in cooling centrifuge at 4°C for 15 min and the supernatant of each one was saved in two equal volume parts. The first one used for immediate measurements of thiobarbituric acid reactive substance (TBARS). The other one was again centrifuged at 6000 x g in cooling centrifuge at 4°C for 15 min and the yielded supernatant which contains the cytosolic and mitochondrial enzymes was saved for the measurements of enzyme activities and oxidative stress parameters.

**Biochemical assay:**

Total lipids (TL), total cholesterol (TC) triglycerides, (TG), concentration levels were determined according to the methods of Frings et al. (1972), Allian et al. (1974), and Fossati and Prencipe (1982), respectively.
using kit from Diamond Diagnostic Chemical Company (Egypt). HDL-cholesterol level was estimated according to the methods of Burstein et al. (1970). The activity of the (AST and ALT) and creatine kinase (CK), were assayed by the methods of Reitman and Frankel (1957) and Young (1997), respectively. Serum LDL-cholesterol and VLDL-levels were calculated according to Friedewald et al. (1972) and Norbert (1995) formula:

\[
\text{VLDL - C} = \frac{\text{Triglycerides concentration}}{5}
\]

\[
\text{LDL - C} = \text{Total cholesterol concentration} - (\text{VLDL-C} + \text{HDL concentration})
\]

The terminal product formed in the decomposition of polynsaturated fatty acids mediated by free radicals (MDA) was quantified as thiobarbituric acid reactive substances according to the method of Esteribauer and Cheeseman (1990). Liver and heart glutathione concentration (GSH) were assayed according to the method of Prins and Loose (1969), and superoxide dismutase (SOD) activity was assayed according to the method of Niskikimi et al. (1972) in both liver and heart tissues.

**Statistical analysis:**

The data obtained in the present study were evaluated by One Way ANOVA (analysis of variance) test and post-comparison was carried out with Tukey-test. The results were expressed as means ± standard error. The value of P ≤ 0.05 was considered statistically significant (Snedecor and Cochran, 1982).

**RESULTS:**

Data presented in table 1, showed that the feeding with high cholesterol diet for 4 weeks resulted in significant increases in serum total lipids, total cholesterol, triglycerides, LDL-C and VLDL-C levels, but a significant decrease in HDL-C was recorded when compared to control rats group fed on normal diet. In the same time an elevation in LDL/HDL-cholesterol and a reduction in HDL-C/total cholesterol ratios was recorded in rats fed cholesterol. In addition hepatic and cardiac enzymes activity, AST, ALT, and CK were increased in high cholesterol-fed rats. Meanwhile, dietary supplementation of hypercholesterolemic rats with quince powder or dill extract succeeded to improve the above mentioned parameters.

As shown in table 2, feeding on high cholesterol diet, induces an oxidative stress as indicated by the significant increase in hepatic and cardiac lipid peroxidation content if compared with control rats, however, hepatic and cardiac GSH content, as well as SOD activity were significantly declined. Treatment with quince powder or dill extract markedly improved the recorded oxidative stress markers and antioxidant parameters.

### Table 1. Serum biochemical parameters in control and different treated groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Animal Groups</th>
<th>Control</th>
<th>Quince</th>
<th>Dill</th>
<th>Cholesterol fed rats</th>
<th>Cholesterol + Quince</th>
<th>Cholesterol + Dill</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total lipids (mg/dl)</td>
<td></td>
<td>583 ± 5.6</td>
<td>588 ± 5.2</td>
<td>583 ± 8.9</td>
<td>699 ±5.9 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>614 ± 8.3 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>639 ± 3.7 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td></td>
<td>88.2 ± 2.5</td>
<td>86.2 ± 1.2</td>
<td>86.4 ± 0.7</td>
<td>154 ± 2.0 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>105 ± 2.3 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>99.2 ± 1.4 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td></td>
<td>77.4 ± 2.6</td>
<td>78.0 ± 1.4</td>
<td>72.4 ± 1.4</td>
<td>121 ± 2.9 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>105 ± 2.3 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>99.2 ± 1.4 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td></td>
<td>30.3 ± 0.7</td>
<td>28.6 ± 0.8</td>
<td>28.4 ± 0.9</td>
<td>99.8 ± 3.2 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>51.2 ± 1.7 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>64.4 ± 1.7 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>VLDL - C (mg/dl)</td>
<td></td>
<td>15.7 ± 1.3</td>
<td>15.6 ± 0.7</td>
<td>14.5 ± 0.7</td>
<td>23.8 ± 0.9 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>21.2 ± 0.6 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>19.2 ± 0.9 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td></td>
<td>42.4 ± 1.4</td>
<td>40.4 ± 1.6</td>
<td>44.2 ± 0.9</td>
<td>30.8 ± 1.5 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>32.6 ± 1.6 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>35.8 ± 1.2 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>LDL/HDL ratio</td>
<td></td>
<td>0.75 ± 0.1</td>
<td>0.74 ± 0.5</td>
<td>0.62 ± 0.02</td>
<td>3.5 ± 0.2 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.2 ± 0.1 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>1.7 ± 0.06 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>HDL/total cholesterol ratio</td>
<td></td>
<td>0.5 ± 0.03</td>
<td>0.46 ± 0.02</td>
<td>0.50 ± 0.04</td>
<td>0.2 ± 0.03 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.33 ± 0.01 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.35 ± 0.01 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>AST (u/L)</td>
<td></td>
<td>35 ± 0.7</td>
<td>33.2 ± 1.2</td>
<td>33.6 ± 0.6</td>
<td>58.4 ± 0.6 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>40.4 ± 1.0 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>42.6 ± 0.8 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>ALT (u/L)</td>
<td></td>
<td>20.0 ± 1.6</td>
<td>19.0 ± 0.6</td>
<td>21.2 ± 0.7</td>
<td>40.06 ± 0.9 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>25.8 ± 0.9 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>27.6 ± 0.9 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>C.K. (u/L)</td>
<td></td>
<td>150.8 ± 2.9</td>
<td>141.4 ± 1.5</td>
<td>150 ± 2.5</td>
<td>213 ± 1.8 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>165 ± 3.5 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>175 ± 3.0 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are the means S.E of six rats; S: significantly different from control at P ≤ 0.05; S<sup>a</sup>: Significantly different from cholesterol + fed rats group at P ≤ 0.05.

### Table 2 Hepatic and cardiac oxidative stress and antioxidant status in control and different treated groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Animal Groups</th>
<th>Control</th>
<th>Quince</th>
<th>Dill</th>
<th>Cholesterol fed rats</th>
<th>Cholesterol + Quince</th>
<th>Cholesterol + Dill</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBRAS nmol / g wet tissue</td>
<td>Liver</td>
<td>44.2 ± 1.0</td>
<td>44.4 ± 1.1</td>
<td>40.8 ± 0.9</td>
<td>89.8 ± 0.7 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>68.0 ± 2.0 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>61.2 ± 0.5 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Heart</td>
<td>50.0 ± 1.5</td>
<td>48.6 ± 1.2</td>
<td>51.2 ± 1.9</td>
<td>64.4 ± 1.4 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>58.0 ± 1.0 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>57.4 ± 1.0 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>GSH Mg/g wet tissue</td>
<td>Liver</td>
<td>4.8 ± 0.23</td>
<td>4.6 ± 0.11</td>
<td>5.0 ± 0.26</td>
<td>2.5 ± 0.10 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.0 ± 0.13 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.4 ± 0.07 S&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Heart</td>
<td>5.3 ± 0.20</td>
<td>5.0 ± 0.14</td>
<td>5.2 ± 0.14</td>
<td>3.6 ± 0.1 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.2 ± 0.15 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.3 ± 0.12 S&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>SOD u/g wet tissue</td>
<td>Liver</td>
<td>32.3 ± 1.1</td>
<td>29.5 ± 0.8</td>
<td>31.0 ± 1.1</td>
<td>12.4 ± 0.8 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>23.0 ± 1.2 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>25.0 ± 0.6 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Heart</td>
<td>29.4 ± 0.5</td>
<td>29.0 ± 0.7</td>
<td>28.2 ± 0.4</td>
<td>20.0 ± 0.7 S&lt;sup&gt;a&lt;/sup&gt;</td>
<td>23.0 ± 1.2 S&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>25.8 ± 0.7 S&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are the means S.E of six rats; S: significantly different from control at P ≤ 0.05; S<sup>a</sup>: Significantly different from cholesterol – fed rats group at P ≤ 0.05.
DISCUSSION:

The obtained data in the hypercholesterolemic rats group indicated a significant increase in the level of serum TL, TC, TG, LDL-C, VLDL-C, and LDL-C/HDL-C ratio, but HDL-C level and HDL-C/TC total cholesterol ratio were declined significantly. These findings are in concomitance with those recorded by Hoyos et al. (2000), where they reported changes in lipid and lipoprotein profiles of rats in response to the administration of cholesterol-supplemented diet. It has been reported that hyperlipidemia (increased level of TC, TG, and LDL-C) is one important risk factor for development and progression of chronic heart disease (Libby, 2000). The obtained results of hyperlipidemia may be due to an increase in fatty acids synthesis in the liver or may be due to liver cholestasis (Owings and Georgeson, 2000). However, the disturbance in lipoprotein profile may be due to over production of very low density lipoprotein VLDL-C by the liver or to the decrease in the removal of VLDL-C and LDL-C from the circulation (Tsutsumi et al., 1995).

The observed increase in the activity of serum AST and ALT enzymes of hypercholesterolemic rats may be due to the hyperlipidaemia, which leads to liver tissue injury. The damage in the cell membrane, so the enzymes located in the cytosol, leak to the blood circulation, (Shyamala et al., 2003). The activity of CK enzyme was increased in the serum of the rats feeding on high fat diet, which may be attributed to the increase in membrane permeability, this result is in agreement with Heibashy (2005) who recorded elevation in CK activity in heart diseases.

Moreover, hypercholesterolemic rats in this result recorded significant elevation in hepatic and cardiac lipid peroxidation, and a significant decrease in GSH concentration and the activity of the antioxidant enzyme SOD. These results may be due to the increase in the production of reactive oxygen species and the abnormal function of the antioxidant system that may contribute to the development of cardiovascular disease (Betteridge, 2000). Crowther (2005) had illustrated that a high-fat diet causes severe oxidative stress in the vascular tissue. Wang et al. (2011) believed that it can be induced from a simple dysfunction of endothelial lining. Moreover, the factors causing heart failure are life style, fatty regimen, hypertension, and a fatty diet. Particularly LDL cholesterol, are mainly responsible for hypercholesterolemia that is related to increased damage in vascular tissue by free radical oxidative stress (Hansson, 2005).

Supplementation of the rats with natural products and the diet rich with fruits or vegetables caused a highly significant improvement in the most tested parameters and ameliorated the alteration in lipid profile and hepatic and cardiac oxidative stress and antioxidant status (Magalhaes et al., 2009). Plants as a source of natural antioxidants, and due to the adverse effects associated with synthetic lipid-lowering drugs, the quest for natural products with lipid-lowering potential and without or minimal side effects is necessary (du Toit et al., 2001).

Phytochemicals, especially the phenolic compounds and flavonoids of fruits and vegetables, have been proposed as the major bioactive compounds which provide the health benefits in diet which are rich in plant foods (Weggemans and Trautwein, 2003). Administration with quince dry fruit powders have a protective action against hypercholesterolemia induced by the high fat diet resulted in the improvement of the lipid profile, as well as liver enzymes activity. This findings are in agreement with Umar et al. (2015). They reported that in comparison with hyperlipidemic rats group, quince total flavonoids significantly reduced serum TC, TG, LDL-C, and increased HDL-C, as well as a reduction in both liver enzymes ALT and AST was recorded.

At the same time dill was used for a long time in folk medicine as anti-hypercholesterolemic plant. Our data reported amelioration in most parameters tested in the hypercholesterolemic rats administered with dill. These results are in agreement with the findings of Yazdanparast and Bahramikia (2008). They indicated that the treatment of hyperlipidemic rats with dill extract reversed the serum lipid levels compared to rats which were fed only high fat diet. The presence of phenolic compounds mainly flavonoids in the dill may be responsible for lowering TC and LDL-C and elevating HDL-C (Vera and Chaneming, 1998). Several fundamental mechanisms have been proposed, may be due to the decrease in cholesterol absorption from the intestine, through binding to bile acids and an increase in bile acids excretion (Senanayake et al., 2004). In addition, the change in triglyceride might be due to a decrease in fatty acids synthesis (Bopanna et al., 1997), or enhanced LDL receptors (Khanna et al., 2002) and therefore, increase in the uptake of LDL (Slater et al., 1980). Injury altered the transport function of the hepatocytes and membrane permeability, leading to leakage of enzymes from their cells (Krishna et al., 2007). This leakage causes an increase in the levels of serum AST and ALT, the treatment with dill extract appears to protect animals against hepatic injury resulted from hypercholesterolemia and hyperlipidaemia (Yazdanparast and Bahramikia, 2008). The
amelioration in the cardiac enzymes function of hypercholesterolemic rats may be due to the hypolipidemic and hypocholesterolemic effect of quince and the dill (Silva et al., 2004; Khademi et al., 2013).

As well as, quince or dill supplementation resulted in amelioration in the lipid peroxidation and antioxidant parameters. The results were in accordance with Hamauzu et al. (2006), who reported that the antioxidant and the antiulcerative properties of phenolic from quince fruits. Mehta et al. (1999) also recorded an improvement in the GSH, the main protective intracellular sulphhydryl peptide of the hepatocytes after the supplementation with quince. As suggested by Silva et al. (2004), caffeic acid derivatives seem to be the main responsible for the antioxidant activity of quince fruits. Moreover, Magalhães et al. (2009) indicated that the antioxidant activity of quince pulp, peel and seed methanolic extracts is statistically correlated with total phenolic content. Oliveira et al. (2007) showed that quince is an excellent natural source of phenolic acids and flavonoids, which are considered potent antioxidants. At the same time, Bahramikia and Yazdanparast (2009) recorded that the treatment with different fractions of dill extract significantly increased hepatic antioxidant system activities such as SOD and GSH along with decreased lipid peroxidation in high fat diet treated rats. The presence of flavonoids, phenolic compounds and essential oil in dill has been reported (Justesen and Knuthsen, 2001). Flavonoids are a group of phenolic compounds having antioxidant potential and play an important role in protection against oxidative stress. Functional hydroxyl groups in flavonoids mediate their antioxidant effects by scavenging free radicals and/or by chelating metal ions (Zhishen et al., 1999).

**CONCLUSION:**

In conclusion, the obtained data strongly suggested the hypocholesterolemic and hypolipidemic effect of quince fruit and dill leaves, as well as the antioxidant status and the beneficial action in hepatic and cardiac enzymes activity. Further scientific efforts are certainly required to establish the exact mechanism of action using purified active components of quince or dill.

**REFERENCES:**


ISSN: 2090 - 0511 On Line ISSN: 2090 - 0503

http://my.ejmanager.com/ejebz


