Physiological investigations on the effect of olive and rosemary leaves extracts in male rats exposed to thioacetamide

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KEYWORDS
Thioacetamide; Olive leaves; Rosemary leaves; Blood; Rats

Abstract Physiologically, it is known that thioacetamide (TAA) toxicity is generally associated with hepatic fibrosis induction, complicated metabolic disorders and health problems. The capability of extracts of olive and rosemary leaves to attenuate the severe physiological disturbances induced by thioacetamide (TAA) intoxication in male rats has been evaluated. Healthy male Wistar rats were used in the present study and were divided randomly into eight groups. Rats of the first group were served as normal control. Rats of the second group were administrated with TAA. Rats of the third, fourth and fifth groups were exposed to TAA plus olive leaves extract, TAA plus rosemary leaves extract and TAA plus olive and rosemary leaves extracts respectively. The sixth, seventh and eighth groups were supplemented with olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts respectively. After 12 weeks of experimental treatments, the levels of serum glucose, total protein, albumin and high density lipoprotein cholesterol were significantly decreased, while the levels of triglycerides, cholesterol, low density lipoprotein cholesterol, very low density lipoprotein cholesterol, creatine kinase and lactate dehydrogenase were statistically increased in rats exposed to TAA. Administration of the studied extracts inhibited the hematobiochemical parameters and improved the physiological disturbances induced by TAA intoxication. Additionally, most improvements were noted in rats administrated with rosemary leaves extract followed by olive and rosemary leaves extracts and olive leaves extract. These results suggested that the effect of these extracts might be due to their antioxidant activities against TAA toxicity.

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1. Introduction
The World Health Organization (WHO) estimates that more than 25% of the global burden of disease is linked to environmental factors, including exposures to toxic chemicals. Tens of thousands of chemicals are currently in use, and hundreds more are introduced every year. Because current chemical...
testing is expensive and time consuming, only a small fraction of chemicals have been fully evaluated for potential human health effects. Thioacetamide (TAA) was originally used to control the decay of oranges and then as a fungicide (Childs and Siegler, 1945). TAA is a potent hepatotoxicant which requires metabolic activation by the mixed-function oxidases. For its toxicity, thioacetamide requires oxidation to its S-oxide and then further to reactive S,S-dioxide form which ultimately attacks lipids and proteins (Hajovsky et al., 2012). Furthermore, the effects of TAA are not limited to the liver as profound structural and functional changes have been described in the thymus (Barker and Smuckler, 1974), the kidney (Barker and Smuckler, 1974; Caballero et al., 2001), the intestine (Ortega et al., 1997; Caballero et al., 2001), the spleen (Al-Bader et al., 2000) and the lung (Latha et al., 2003).

The pharmacological treatment of disease began long ago with the use of herbs (Schulz et al., 2001). Herbal medicine is the use of plants, plant parts, their water or solvent extracts, essential oils, gums, resins, exudates or other forms of advanced products made from plant parts used therapeutically to provide proactive support of various physiological systems; or, in a more conventional medical sense, to treat, cure, or prevent a disease in animals or humans (Weiss and Fintelmann, 2000). About 70–80% of the world populations, particularly in the developing countries, rely on non-conventional medicine in their primary healthcare as reported by the World Health Organization (Akerele, 1993). The use of herbal medicines and phytonutrients or nutraceuticals continues to expand rapidly across the world with many people now resorting to these products for treatment of various health challenges in different national healthcare settings (WHO, 2004). This interest in drugs of plant origin is due to several reasons, namely, conventional medicine can be inefficient (e.g. side effects and ineffective therapy), abusive and/or incorrect use of synthetic drugs results in side effects and other problems, a large percentage of the world’s population does not have access to conventional pharmacological treatment, and folk medicine and ecological awareness suggests that natural products are harmless.

Olive tree (*Olea europaea*, Oleaceae) is a longevous plant, anciently known in the Mediterranean basin (Melillo, 1994). The olive tree has been widely accepted as one of the species with the highest antioxidant activity via its oil, fruits, and leaves. It is well known that the activity of the olive tree byproduct extracts in medicine and food industry is due to the presence of some important antioxidant and phenolic components to prevent oxidative degradations. Olive leaves are considered a cheap raw material and a useful source of high-added value products (Briante et al., 2002; Jamai et al., 2008). The main phenolic compound in olive leaves is the glycosylated form of oleuropein (Amro et al., 2002; Visioli et al., 2002). It is a natural phenolic antioxidant, which is present in high concentration in olives, olive oil and olive tree leaves (Andreadou et al., 2007). Rosemary plant with the scientific name of Rosmarinus officinalis belongs to the Lamiaceae family. Four main categories of compounds found in rosemary include flavonoids, phenols, volatile oil, and terpenoids (Barnes et al., 2007). Leaves of rosemary possess a variety of bioactivities, including antioxidant, antiinflammatory, treat headaches and anti-HIV (Aruoma et al., 1996; Altinier et al., 2007). The present study is aimed to investigate the role of olive and rosemary leaves extracts against TAA toxicity in Wistar male rats.

2. Materials and methods

2.1. Extraction of olive and rosemary leaves

The methods of Sakr and Lamfon (2012), and Al-Attar and Abu Zeid (2013) were used for the preparation of olive and rosemary leaves extracts with some modifications. Fine qualities of olive and rosemary leaves were obtained from a commercial market, Jeddah, Saudi Arabia. The leaves were scientifically defined by the herbarium of Biological Sciences Department, Faculty of Sciences, King Abdulaziz University, Jeddah, Saudi Arabia. The leaves were thoroughly washed and dried at room temperature. The dried olive leaves (50 g) were powdered and added to 2 L of hot water in a flask. After 6 h, the mixture was slowly boiled for 1 h. After boiling period, the mixture was cooled at room temperature and it was gently subjected to an electric mixer for 10 min. Also, the dried rosemary leaves (50 g) were powdered and added to 2 L of hot water in a flask. After 6 h, the mixture was slowly boiled for 1 h. After boiling period, the mixture was cooled at room temperature and it was gently subjected to an electric mixer for 10 min. Thereafter the solutions of olive and rosemary leaves were filtered. Finally, the filtrates were evaporated in an oven at 40 °C to produce dried residues (active principles). With references to the powdered samples, the yield means of the olive and rosemary extracts were 18.7% and 20.6% respectively. Furthermore, these extracts were prepared every 2 weeks and stored in a refrigerator for experimentation.

2.2. Animal models

Forty-eight Wistar male rats weighing 72.6–103.4 g were obtained from the Experimental Animal Unit of King Fahd Medical Research Center, King Abdulaziz University, Jeddah, Saudi Arabia. Rats were acclimatized to the experimental laboratory having temperature 20 ± 1 °C, controlled humidity conditions (65%) and 12:12 h light:dark cycle. Rats were housed in standard plastic cages, fed with standard diet, and water ad libitum. All experimental procedures were conducted in accordance with ethical guidelines of the Animal Care and Use Committee of King Abdulaziz University.

2.3. Experimental design

The animals were divided into eight groups of six animals each and then subjected to one of the following treatments:

- **Group 1**: Rats were served as controls and intraperitoneally injected with saline solution (0.9% NaCl), twice weekly for 12 weeks.
- **Group 2**: Rats were given 300 mg/kg body weight of TAA (Sigma–Aldrich Corp., St. Louis, MO, USA) by intraperitoneal injection, twice weekly for 12 weeks.
- **Group 3**: Rats were intraperitoneally injected with TAA at the same dose given to group 2 and were orally supplemented with olive leaves extract at a dose of 200 mg/kg body weight/day for 12 weeks.
- **Group 4**: Rats were intraperitoneally injected with TAA at the same dose given to group 2 and were orally supplemented
with rosemary leaves extract at a dose of 200 mg/kg body weight/day for 12 weeks.

**Group 5:** Rats were intraperitoneally injected with TAA at the same dose given to group 2 and were supplemented with olive leaves extract (100 mg/kg body weight/day) and rosemary leaves extract (100 mg/kg body weight/day) for 12 weeks.

**Group 6:** Rats intraperitoneally received saline solution at the same dose given to group 1 and were orally supplemented with olive leaves extract at the same dose given to group 3 for 12 weeks.

**Group 7:** Rats were intraperitoneally received saline solution at the same dose given to group 1 and were orally supplemented with rosemary leaves extract at the same dose given group 4 for 12 weeks.

**Group 8:** Rats were intraperitoneally received saline solution at the same dose given to group 1 and were supplemented with olive and rosemary leaves extracts at the same dose given to group 5 for 12 weeks.

### 2.4. Blood sampling

After 12 weeks, the experimental animals were fasted for 12 h, water was not restricted, and then anesthetized with diethyl ether. Blood samples were collected from orbital venous plexus in non-heparinized tubes, centrifuged at 2500 rpm for 15 min. Blood sera were carefully separated and stored frozen. The level of serum glucose was determined using the method of Trinder (1969). The method of Peters (1968) was carried out to measure the level of serum total protein. The level of serum albumin was measured according to the method of Doumas et al. (1971). To estimate the triglycerides value, Fossati and Prinicip method (1982) was used. The method of Richmond (1973) was used to determine the level of serum cholesterol. The method of Warnick (1983) was used to determine the value of serum high density lipoprotein cholesterol (HDL-C). The level of serum low density lipoprotein cholesterol (LDL-C) was estimated according to the equation of Friedewald et al. (1972).

\[
LDL - C = \frac{Total\ cholesterol - HDL - triglycerides}{5}
\]

Serum very low density lipoprotein cholesterol (VLDL-C) was evaluated using the following equation:

\[
VLDL - C = \frac{Triglycerides}{2.175}
\]

Serum creatine kinase (CK) level was determined according to the method of Horder et al. (1991). The method of Weishaar (1975) was used to measure the value of serum lactate dehydrogenase (LDH).

### 2.5. Statistical analysis

The statistical differences of all data were determined by two-way analysis of variance (ANOVA). All values were expressed as mean ± standard deviation (SD) for six observations. Statistical probability of less than 0.05 was used as a criterion for significance. All data were evaluated for statistical significance using the Statistical Package for Social Sciences (SPSS) for Windows, version 12.0.

### 3. Results

The levels of serum glucose were statistically decreased in rats exposed to TAA (34.8%), TAA plus olive leaves extract (28.5%), TAA plus rosemary leaves extract (33.2%), TAA plus olive and rosemary leaves extracts (27.3%), olive leaves extract (18.5%), rosemary leaves extract (11.1%), and olive and rosemary leaves extracts (6.7%) when compared to control rats (Table 1).

In comparison with control data, the levels of serum total protein were significantly diminished in rats administrated with TAA (12.9%), TAA plus olive leaves extract (9.2%). No statistically significant difference was observed in this parameter in rats treated with TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extracts, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts. The level of serum albumin was significantly decreased from corresponding control values in rats treated with TAA (11.9%). In comparison with the control group, there were no significant changes in the levels of serum albumin in TAA plus olive leaves extract, TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extracts, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts treated rats. The levels of serum triglycerides were statistically increased in rats exposed to TAA (77.9%), TAA plus olive leaves extract (48.5%), TAA plus rosemary leaves extract (47.1%), and TAA plus olive and rosemary leaves extracts (48.5%) as compared with control rats. Remarkable elevations in the level of serum cholesterol were observed in rats treated with TAA (105.3%), TAA plus olive leaves extract (63.2%), TAA plus rosemary leaves extract (51.2%), and TAA plus olive and rosemary leaves extracts (67.9%) when compared to control rats. Insignificant changes of serum triglycerides and cholesterol levels were noted in olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts treated rats as compared with control rats (Table 1).

As shown in Table 2, a significant decline in the level of serum HDL-C was noted in rats exposed to TAA (17.8%) compared with control rats. In comparison with the control group, there were no significant alterations in the levels of serum HDL-C in TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extract, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts treated rats. The levels of serum LDL-C were statistically enhanced in rats treated with TAA (211.3%), TAA plus olive leaves extract (123.6%), TAA plus rosemary leaves extract (97.2%), and TAA plus olive and rosemary leaves extracts (130.2%) as compared with control rats. The levels of serum VLDL-C were statistically increased in rats subjected to TAA (80.7%), TAA plus olive leaves extract (54.8%), TAA plus rosemary leaves extract (48.4%), and TAA plus olive and rosemary leaves extracts (51.6%) as compared with control rats. Insignificant alterations of serum LDL-C and VLDL-C levels were observed in rats supplemented with olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts as compared with control rats. The level of serum CK was significantly increased from corresponding control values in rats treated with TAA (103.4%) and TAA plus olive leaves extract (21.8%). In comparison with the control group, there were no significant changes in the levels of serum
Table 1  The levels of serum HDL-C, LDL-C, VLDL-C, CK and LDH of control, TAA, TAA plus olive leaves extract, TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extracts, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts treated rats ($n = 6$). Percentage changes are included in parentheses.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Parameters</th>
<th>Glucose (mmol/L)</th>
<th>Total protein (g/L)</th>
<th>Albumin (g/L)</th>
<th>Triglycerides (mmol/L)</th>
<th>Cholesterol (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td>6.59 ± 0.35</td>
<td>69.33 ± 5.61</td>
<td>11.83 ± 0.75</td>
<td>0.68 ± 0.05</td>
<td>4.29 ± 0.53 (+105.3)</td>
</tr>
<tr>
<td>TAA</td>
<td></td>
<td>4.30 ± 0.14 (-34.8)</td>
<td>60.42 ± 2.25 (-12.9)</td>
<td>10.42 ± 1.28 (-11.9)</td>
<td>1.21 ± 0.20 (+77.94)</td>
<td>4.29 ± 0.53 (+105.3)</td>
</tr>
<tr>
<td>TAA + olive leaves</td>
<td></td>
<td>4.71 ± 0.27 (-28.5)</td>
<td>63.00 ± 2.10 (-9.2)</td>
<td>10.84 ± 1.33 (-8.4)</td>
<td>1.01 ± 0.13 (+48.5)</td>
<td>3.41 ± 0.50 (+63.2)</td>
</tr>
<tr>
<td>TAA + rosemary leaves</td>
<td></td>
<td>4.40 ± 0.25 (-33.23)</td>
<td>69.92 ± 6.73 (+0.9)</td>
<td>13.08 ± 1.11 (+10.4)</td>
<td>1.00 ± 0.11 (+47.1)</td>
<td>3.16 ± 0.32 (+51.2)</td>
</tr>
<tr>
<td>TAA + olive and rosemary leaves</td>
<td></td>
<td>4.79 ± 0.31 (-27.3)</td>
<td>68.75 ± 2.32 (-0.8)</td>
<td>12.81 ± 1.60 (+8.3)</td>
<td>1.01 ± 0.17 (+48.5)</td>
<td>3.51 ± 0.48 (+67.9)</td>
</tr>
<tr>
<td>Olive leaves</td>
<td></td>
<td>5.37 ± 0.39 (-18.5)</td>
<td>65.67 ± 2.26 (-5.3)</td>
<td>11.58 ± 0.49 (-2.1)</td>
<td>0.65 ± 0.06 (-4.4)</td>
<td>2.01 ± 0.05 (-3.8)</td>
</tr>
<tr>
<td>Rosemary leaves</td>
<td></td>
<td>5.86 ± 0.44 (-11.1)</td>
<td>66.42 ± 2.50 (-4.2)</td>
<td>12.00 ± 0.63 (+1.4)</td>
<td>0.65 ± 0.05 (-4.4)</td>
<td>1.98 ± 0.11 (-5.3)</td>
</tr>
<tr>
<td>Olive and rosemary leaves</td>
<td></td>
<td>6.15 ± 0.41 (-6.7)</td>
<td>64.17 ± 4.12 (-7.4)</td>
<td>11.25 ± 0.88 (-4.9)</td>
<td>0.66 ± 0.04 (-2.9)</td>
<td>2.05 ± 0.10 (-1.9)</td>
</tr>
</tbody>
</table>

Table 2  The levels of serum HDL-C, LDL-C, VLDL-C, CK and LDH of control, TAA, TAA plus olive leaves extract, TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extracts, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts treated rats ($n = 6$). Percentage changes are included in parentheses.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Parameters</th>
<th>HDL-C (mmol/L)</th>
<th>LDL-C (mmol/L)</th>
<th>VLDL-C (mmol/L)</th>
<th>CK (U/L)</th>
<th>LDH (U/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td>0.90 ± 0.03</td>
<td>1.06 ± 0.07</td>
<td>0.31 ± 0.03</td>
<td>284.67 ± 18.24</td>
<td>523.67 ± 32.14</td>
</tr>
<tr>
<td>TAA</td>
<td></td>
<td>0.74 ± 0.04 (-17.8)</td>
<td>3.30 ± 0.53 (+211.32)</td>
<td>0.56 ± 0.09 (+80.7)</td>
<td>579.12 ± 64.22 (+103.4)</td>
<td>1816.33 ± 282.29 (+246.9)</td>
</tr>
<tr>
<td>TAA + olive leaves</td>
<td></td>
<td>0.82 ± 0.08 (-8.9)</td>
<td>2.37 ± 0.52 (+123.6)</td>
<td>0.48 ± 0.06 (+54.8)</td>
<td>346.69 ± 40.58 (+21.8)</td>
<td>594.33 ± 104.09 (+13.5)</td>
</tr>
<tr>
<td>TAA + rosemary leaves</td>
<td></td>
<td>0.88 ± 0.03 (-2.22)</td>
<td>2.09 ± 0.35 (+97.2)</td>
<td>0.46 ± 0.05 (+48.4)</td>
<td>338.25 ± 61.56 (+18.8)</td>
<td>547.50 ± 70.83 (+4.6)</td>
</tr>
<tr>
<td>TAA + olive and rosemary leaves</td>
<td></td>
<td>0.86 ± 0.06 (-4.4)</td>
<td>2.44 ± 0.47 (+130.2)</td>
<td>0.47 ± 0.08 (+51.6)</td>
<td>328.33 ± 62.75 (+15.3)</td>
<td>589.67 ± 105.02 (+12.6)</td>
</tr>
<tr>
<td>Olive leaves</td>
<td></td>
<td>0.89 ± 0.04 (-1.1)</td>
<td>0.99 ± 0.04 (-6.6)</td>
<td>0.30 ± 0.03 (-3.3)</td>
<td>289.50 ± 18.58 (+1.7)</td>
<td>527.17 ± 27.88 (+0.7)</td>
</tr>
<tr>
<td>Rosemary leaves</td>
<td></td>
<td>0.90 ± 0.04 (0.0)</td>
<td>0.95 ± 0.11 (-10.4)</td>
<td>0.30 ± 0.02 (-3.3)</td>
<td>282.40 ± 21.53 (-0.8)</td>
<td>513.00 ± 16.35 (-2.0)</td>
</tr>
<tr>
<td>Olive and rosemary leaves</td>
<td></td>
<td>0.89 ± 0.03 (-1.1)</td>
<td>1.02 ± 0.09 (-3.8)</td>
<td>0.31 ± 0.02 (0.0)</td>
<td>284.26 ± 31.38 (-0.1)</td>
<td>494.67 ± 94.66 (-5.5)</td>
</tr>
</tbody>
</table>
CK in TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extracts, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts treated rats. In comparison with control, the administration of TAA alone significantly increased (246.9%) the activity of serum LDH. The activity of this enzyme in rats treated with TAA plus olive leaves extract, TAA plus rosemary leaves extract, TAA plus olive and rosemary leaves extracts, olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts was not significantly different from those of controls (Table 2).

4. Discussion

The present decline in levels of serum glucose, total protein and albumin with the increases of serum triglycerides, cholesterol, LDL-C and VLDL-C indicate disturbances in protein, carbohydrate and lipid metabolism induced by TAA intoxication. Kruszynska and McIntyre (1991) reported that the blood sugar level after overnight fasting in cirrhotic patients is believed to decrease only in severe hepatic failure. Al-Attar (2012) showed that the level of blood glucose was statistically decreased in the mice group treated with TAA compared with control group. Furthermore, several studies showed that carbon tetrachloride (CCL₄) administration depleted liver glycogen in cirrhotic rats (Favari and Perez-Alvarez, 1997; Muriel and Escobar, 2003; Muriel et al., 2003; Moreno et al., 2011).

Blood glucose concentration is known to depend on the ability of the liver to absorb or produce glucose. The liver performs its glucostatic function owing to its ability to synthesize or degrade glycogen according to the needs of the organism, as well as via gluconeogenesis (Ahmed et al., 2006).

The present study showed that the supplementation of olive leaves extract, rosemary leaves extract, and olive and rosemary leaves extracts significantly decreased the levels of serum glucose in normal rats. Jemai et al. (2009) reported that the oleuropein and tannins in olive leaves act as α-glucosidase inhibitors, reducing the absorption of carbohydrates in the gut. Moreover, the extract of olive leaves was shown to have an inhibitory effect on the postprandial blood increase in glucose in diabetic rats (Komaki et al., 2003). In humans treated with olive leaves extract, blood glucose was significantly declined after cooked rice loading compared with untreated controls (Komaki et al., 2003). There have been two possible mechanisms suggested to explain the hypoglycemic effect of the olive leaves extract: (1) oleuropein improved glucose-induced insulin release, and (2) increased peripheral uptake of glucose (El and Karakaya, 2009). The oleuropein in olive leaves has been shown to accelerate the cellular uptake of glucose, leading to reduced blood glucose (Gonzalez et al., 1992). Since oleuropein is a glycoside, it could potentially access a glycogen in the cytoplasm of the cell (Moss and Henderson, 1986; Lott and Nemensanszky; 1987). Generally, high concentrations of LDH are found in the liver, heart, erythrocytes, skeletal muscles and kidneys. Consequently, diseases affecting those organs, such as renal infarction, myocardial infarction and hemolysis, have been reported to be associated with significant elevations in total serum LDH activity. Such elevations have been widely applied as diagnostic indices for kidney, liver, heart and red blood cell dysfunction (Wills, 1971; Timmis and Nathan, 1993; Castaldo et al., 1994). Additionally, high serum LDH activity has also been reported in a variety of cancers (Kanowski and Clague, 1994). Al-Attar (2011) demonstrated that the chronic administration of TAA (300 mg/kg body weight/twice weekly for 10 weeks) induced cardiotoxicity manifested by a significant increase in serum of CK and LDH activities in rats. This is due to leakage from the heart as a result of TAA induced necrosis (Al-Attar, 2011).
The present obtained results showed that these extracts improved the physiological disturbances induced by TAA intoxication. Moreover, most improvements were observed in rats supplemented with rosemary leaves extract followed by olive and rosemary leaves extracts and olive leaf extract. TAA causes an elevation of oxidative stress, enhancing free radical mediated damage to proteins, lipids and DNA (Bruck et al., 2004; Tunez et al., 2005; Uskokovic-Markovic et al., 2007). Several studies have demonstrated the beneficial effect of antioxidants against TAA toxicity (Uskokovic-Markovic et al., 2007; Baskaran et al., 2010; Mustafa et al., 2013; Ali et al., 2014). Olive leaves are a source of several antioxidants (Briante et al., 2002; Bouaziz and Sayadi, 2005; Meirinhos et al., 2005; Ranalli et al., 2006). Popular medicine and phytotherapy use olive leaves to treat and prevent several diseases. Many of these properties have been described as resulting from the antioxidant character of oleanuropein (Visioli et al., 1998). Most pharmacological effects of rosemary are the consequence of high antioxidant activity of its main chemical constituents, which include carnosol, carnosic acid, ursolic acid, rosmarinic acid, and caffeic acid. The potent antioxidant properties of rosemary have been mainly attributed to its major diterpenes, carnosol and carnosic acid, as well as to the essential oil components (Ngo et al., 2011). In the light of this study, these extracts may play a role in the prevention of physiological disturbances caused by TAA. These results suggested that the effect of these extracts might be due to their antioxidant activities restraining the oxidative stress which is widely associated with TAA toxicity. Finally, the present study may help to understand the extent of interaction between the studied extracts’ administration in toxicants causing physiological alterations, its prevention in animal model and allowed for preventive applying in human status.

References


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