Potential Protective and Ameliorate Effects of Sesame Oil and Jojoba Oil Against Potassium Bromate (KBrO$_3$)-induced oxidative stress in rats

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ABSTRACT
Oxidative stress plays an important role in the etiology and pathogenesis of many chronic diseases such as atherosclerosis, hypertension, diabetes mellitus and cancers. Dietary intake of antioxidants can inhibit or delay the oxidation of susceptible cellular substrates so prevent oxidative stress. The present study was designed to investigate potential protective and ameliorate effects of sesame oil and jojoba oil against potassium bromate (KBrO₃)-induced oxidative stress using experimental rats. Thirty five of rats were randomly divided into five groups, seven rats each. Group 1 was fed on the basal diet and kept as a negative control group (normal rats). The other 4 groups were injected by a single intraperitoneal dose of KBrO₃ at dose of 125 mg/ kg body weight for induction of oxidative stress. Group 2 was left as a positive control group and groups 3, 4 and 5 were fed on supplemented diet with 5% sesame oil, jojoba oil and mixture of them, respectively. The obtained results revealed that the injected intoxicated groups with sesame oil (SO) or jojoba oil (JO) or mixture of them had significant reduced in serum levels of total cholesterol, triglycerides, LDL-c, total bilirubin, blood urea nitrogen, uric acid, creatinine, malondialdehyde (MDA), activity of aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) enzymes and significant increased in feed intake, body weight gain, serum levels of HDL-c, total antioxidant capacity, reduced glutathione (GSH), activity of glutathione peroxidase (GPx) superoxide dismutase (SOD) and catalase (CAT) enzymes. Moreover, there is a significant decrease in MDA and increase in GSH content and activity of antioxidant enzymes (GPx, SOD and CAT) in liver tissues as well as partially improvements in liver structures of liver and kidneys compared to those of positive intoxicated control group. The best improvements of all the biochemical parameters and histological
structures of liver and kidneys which were tended toward normal results were observed in treated KBrO3-intoxicated rats with mixture of SO with JO. In conclusion, the present findings suggested that regular intake of SO or JO may be useful in improving liver and kidney functions and may protect against KBrO3 induced oxidative stress in rats by exhibiting stronger antioxidant activity. The mixture of SO with JO provide the preferable effects.

**Keywords:** Potassium Bromate (KBrO3) – Liver enzymes – Kidney functions – Antioxidant enzymes – Oxidative stress

1. INTRODUCTION

Oxidative stress is a condition that reflects an imbalance between the systemic manifestation of reactive oxygen species (ROS) and a biological system's ability to readily detoxify (antioxidant defenses) the reactive intermediates or to repair the resulting damage (Hampl et al., 2012). Oxidative stress plays an important role in the etiology and pathogenesis of many chronic diseases such as atherosclerosis, hypertension, diabetes mellitus and cancers (Krajcovicova-Kudlackova et al., 2012).

Potassium bromate (KBrO3) is a food additive that is extensively used as a maturing agent for flour and as a dough conditioner. It is also used in cosmetics and a component of permanent hair weaving solutions. Disinfection of drinking water by ozonation, which has emerged as a promising alternative to chlorination since it does not result in the production of hazardous agents like trihalomethanes, also generates bromate as a by-product (Bull, 1990). Bromate is now considered as a probable human carcinogen and a complete carcinogen in animals. Increased production of reactive oxygen species (ROS) and free radicals has been implicated in mediating KBrO3-induced toxicity (Ahmad et al., 2012). Supporting the involvement of ROS in its action, several antioxidants (AO) have been shown to ameliorate the bromate-induced multiple organ toxicity (Stasiak et al., 2010 and Khan et al., 2013).

Dietary intake of antioxidants is important to protect cellular system against oxidative stress that represents as a risk factor for many chronic diseases (Erukainure et al., 2012). Sesame (Sesamum indicum L.) is one of the most important oil seed crops, and its oil is highly valued as a
traditional healthy food ingredient because of its antioxidant contents (Abou-Gharbia et al., 2000). Sesame oil contains sesamin, sesamolin, sesaminol and lignan fractions, which are known to play an important role in its oxidative stability and antioxidative activity (Elleuch et al., 2007). Sesame oil may be useful in managing oxidative stress-associated diseases such as atherosclerosis, diabetes mellitus, obesity, chronic renal failure, rheumatoid arthritis, and neurodegenerative diseases (Lee et al., 2006), cancer and aging (Habila et al., 2013).

Jojoba seeds (Simmondsia chinensis L.) as shown in Fig. 1, are a perennial woody shrub native to semi-arid regions all over the world (Ranzatoa et al., 2011). The jojoba plant produces seeds that contain up to 50% liquid by weigh wax esters used as a lubricant additive in cosmetics (Wisniak, 1994). Jojoba seeds possess anti-inflammatory activity (Habashy et al., 2005). Jojoba liquid, naturally golden liquid wax ester (Fig. 2), is a stable highly lipophilic nontoxic oily material obtained from the desert plant jojoba. More than 80% of these are esters of C18-, C20-, C22-, and C24- chain monounsaturated alcohols and fatty acids (Shani, 1995). Numerous studies have demonstrate the feasibility of incorporating jojoba as an oil phase in formulas containing active compounds to enhance the efficiency of topical drugs (El-Laithy and El-Shaboury, 2002). Jojoba oil is used as a potential low-calorie edible oil and coating material for fruits and pills (Naqvi and Ting, 1990).

Based on these facts the present study was designed to investigate effects of sesame oil and jojoba oil on attenuate the KBrO₃-induced oxidative stress in rats as the animal model.

Fig. 1: Jojoba seeds (Simmondsia chinensis L)
Fig. 2: Jojoba seed oil (Simmondsia chinensis L)
2. MATERIALS AND METHODS

Materials

Oils: Pure sesame oil (SO) and jojoba oil (JO) were obtained from the Egyptian Company of Natural Oils, Cairo, Egypt.

Animals and diets: Thirty five male adult of Sprague-Dawley rats 7–8 weeks old and weighing 200 ± 5 g were purchased from the Laboratory Animal Colony, Helwan, Egypt. Diet consists of casein, soybean oil, Choline chloride, vitamin mixture, mineral mixture, fibers, L-Cystine, sucrose and corn starch were obtained from El- Gomhorya Company for Chemical and Pharmaceutical, Cairo, Egypt.

Chemicals and kits: Potassium bromate (KBrO₃) in the form of a white powder was purchased from El-Gomhorya Company, Cairo Egypt. Kits for all biochemical analysis were obtained from the Gamma Trade Company for Pharmaceutical and Chemicals, Dokki, Giza, Egypt.

Methods

Preparation of basal diet: The dietary supply of protein, fat, carbohydrates, vitamins and minerals to meet the recommended dietary allowances for rats was prepared according to Reeves et al., (1993).

Experimental design and grouping of rats: All animals were housed at a controlled room temperature of 23±3°C, 55% humidity and under a 12 hr light/12-hr dark schedule. The animals were fed on the basal diet and water was provided ad libitum for one week before starting the experiment for acclimatization. Then, rats were randomly distributed into 5 equal groups, of 7 rats each. Group 1 was feed on the basal diet and kept as a negative control group (normal rats). The other 4 groups were injected by a single intraperitoneal dose of potassium bromate at dose of 125 mg/kg body weight for induction of oxidative stress according to the described methods by Khan and Sultana (2004). Group 2 was left as a positive control group and groups 3, 4 and 5 were fed on supplemented diet with 5% sesame oil, jojoba oil and mixture of them, respectively.

Assay of food intake and body weight gain: Food intake (FI) was calculated every day. Body weight gain (BWG) was calculated using the following formula:

\[ BWG = \text{Final Body Weight} - \text{Initial Body Weight} \]
Blood collection: At the end of the experimental period (6 weeks), animals were fasted for 12-hr., except for water. Animal blood samples were collected from the posterior vena cava into serum-separation tubes under light ethyl ether anesthesia. Then, blood samples were allowed to clot for 30 min at room temperature, and were then centrifuged at 10 minutes at 3000 rpm for serum separation. Serum samples were carefully aspirated using a needle and transferred into dry clean test tubes and kept frozen at -20°C for biochemical analysis.

Preparation of liver tissue homogenate: Part of liver of all animals were cut into small pieces and immediately homogenized in 5-10 ml ice-cold medium containing buffer (50 mm potassium phosphate, PH 7.5, containing 2mM EDTA) per gram tissue using tissue homogenizer (Sonicator, model 4710, Cole-Parmer Instrument Company, USA). The homogenates tissues were centrifuged at 4000 rpm for 15 min at 4°C (Montgomery and Dymock, 1961). Then, supernatant was carefully separate for the determinations of for biochemical analysis.

Biochemical Assay
Assay of serum lipid profile: Serum levels of triglyceride (TG) were estimated as described by Siedel (1993) method. Levels of serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-c) and high-density lipoprotein (HDL-c) were determined as described by Young (1995) method.

Assay of renal functions: Serum urea nitrogen levels (UN) was determined according to the method of Patton and Crouch (1977). Serum levels of uric acid were determined using the enzymatic colorimetric method as described by Fossati et al., (1980). Serum creatinine concentrations were colorimetrically determined as described by Husdan and Rapoport, (1968) method.

Assay of liver functions: Serum activities AST, ALT enzymes were assayed colorimetric using described method by Bergmeyer et al., (1978). Serum activity of ALP was determined as described by Roy (1970) method. Serum TBr level was determined colorimetric as described by Tietz (1991) method.

Assay Serum levels of lipid peroxidation and GSH and activity of antioxidant enzyme: Serum level of malondialdehyde (MDA) was assayed quantitatively in serum as described by Draper and Hadley (1990). Serum reduced glutathione (GSH) level was determined as
described by Beutler et al., (1963). Serum activity of glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) enzymes were assayed according to the method of Hissin and Hiff (1976), Kakkor et al., (1984) and Sinha (1972), respectively.

**Assay levels of serum total antioxidant capacity:** Total Antioxidant capacity (TAC) was assayed quantitatively in serum using spectrophotometric method as described by Woodford and Whitehead (1998).

**Assay levels of lipid peroxidation, GSH and activity of antioxidant enzyme in liver tissues:** Oxidant and antioxidant status in liver tissues includes lipid peroxides as MDA concentration was measured according to the method of Albro et al., (1986). Non-enzyme antioxidant as GSH concentration was determined in liver tissues according to the method of Beutler et al., (1963). Activities of antioxidant enzyme of GPx, SOD and CAT in liver tissues were measured according to the described methods by Paglia and Valentaine (1979), Nishikimi et al., (1972) and Aebi (1984), respectively.

**Preparation of histological sections:** Specimens of liver and kidneys were taken and fixed in 10% neutral buffered formalin for 24 hr and processed for light microscope. Tissue specimens were embedded in paraffin wax using a conventional method and stained by Harris hematoxylin and eosin stain for histopathological studies (Drury and Wallington, 1980).

**Statistical analysis:** The results were expressed as means ± standard deviations in each group. Differences between groups were assessed using computerized statistical package of social sciences (SPSS) program (SPSS. 20 software version) with one-way analysis of variance (ANOVA) followed by Duncan's multiple range tests. P<0.05 values were considered to be statistically significant (Snedecor and Cochran, 1981).

3. RESULTS

Results of feed intake (FI), initial body weight (IBW), final body weight (FBW) and body weight gain (BWG) of experimental rats are presented in Table 1. It showed that FI, FBW and BWG of KBrO₃-intoxicated rats (positive control group) were decreased significantly, compared with those of the normal rats. On the other hand, treated KBrO₃-intoxicated rats which feeding on supplemented diet with SO or
JO or mixture of them had significantly increased in FI, FBW and BWG compared with those of intoxicated positive rats. Moreover, treated KBrO$_3$-intoxicated rats with JO had significantly decreased in FI, FBW and BWG compared with those of the normal rats.

The obtained results in Table 2 showed a marked significant increase in serum TG, TC and LDL-c levels and significant decrease in serum HDL-c levels in positive control group compared with those of the normal rats (negative control group). Comparing untreated KBrO$_3$-intoxicated rats with those treated with SO, JO and mixture of them revealed a marked significant decrease in serum levels of TG, TC and LDL-c and significant increase in serum HDL-c, which nearly returned toward the normal levels.

Table 1: FI, FBW and BWG of normal rats and intoxicated rats with KBrO$_3$

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters as Mean ± SD</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>FI (g/d)</td>
<td>IBW (g)</td>
<td>FBW (g)</td>
<td>BWG (g)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>18.29±1.25$^a$</td>
<td>199.29±4.50$^a$</td>
<td>244.00±3.37$^a$</td>
<td>44.71±6.18$^a$</td>
</tr>
<tr>
<td>G2: Positive control rats (KBrO$_3$ treated)</td>
<td>14.43±1.13$^c$</td>
<td>200.70±3.55$^a$</td>
<td>229.86±6.84$^c$</td>
<td>29.14±5.18$^c$</td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO$_3$</td>
<td>17.00±0.82$^a$</td>
<td>200.29±4.07$^a$</td>
<td>239.00±3.16$^a$</td>
<td>38.00±6.14$^a$</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO$_3$</td>
<td>16.57±1.11$^b$</td>
<td>200.68±3.27$^b$</td>
<td>236.71±4.72$^b$</td>
<td>35.86±5.70$^b$</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO$_3$</td>
<td>17.71±0.78$^{ab}$</td>
<td>199.00±3.87$^a$</td>
<td>240.43±5.13$^{ab}$</td>
<td>41.43±6.95$^{ab}$</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p< 0.05.

Table 2: Serum levels of TG, TC, LDL-c and HDL-c in normal rats and intoxicated rats with KBrO$_3$

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters as Mean ± SD</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TG (mg/dL)</td>
<td>TC (mg/dL)</td>
<td>LDL-C (mg/dL)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>117.14±3.39$^b$</td>
<td>54.43±4.24$^c$</td>
<td>16.00±2.58$^c$</td>
</tr>
<tr>
<td>G2: Positive control rats (KBrO$_3$ treated)</td>
<td>142.43±4.58$^a$</td>
<td>88.29±3.73$^a$</td>
<td>41.00±3.37$^a$</td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO$_3$</td>
<td>115.29±4.54$^b$</td>
<td>58.29±3.73$^{bc}$</td>
<td>23.57±3.95$^b$</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO$_3$</td>
<td>117.71±2.93$^b$</td>
<td>60.29±3.73$^b$</td>
<td>24.00±1.83$^b$</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO$_3$</td>
<td>116.00±3.97$^b$</td>
<td>58.00±3.46$^{bc}$</td>
<td>21.29±1.25$^b$</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p< 0.05.

SD: Standard Deviation
Tabulated data in Table 3 showed that intoxicated positive rats have significant increase in serum levels of urea nitrogen (UN), uric acid (UA) and creatinine (Cr) compared with those of normal control rats. In contrast, rats feeding on supplemented diet with SO or JO or mixture of them had significantly decreased serum levels of UN, UA and Cr when compared with those of positive control group.

**Table 3**: Serum levels of UN, UA, Cr in normal rats and intoxicated rats with KBrO₃

<table>
<thead>
<tr>
<th>Groups</th>
<th>Serum Levels as Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>UN (mg/dL)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>17.14±2.34</td>
</tr>
<tr>
<td>G2: Positive control rats</td>
<td>28.71±2.22</td>
</tr>
<tr>
<td>(KBrO₃ treated)</td>
<td></td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO₃</td>
<td>23.43±3.10</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO₃</td>
<td>24.29±2.43</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO₃</td>
<td>21.86±3.02</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p<0.05.

SD: Standard Deviation

Results in Table 4 showed significant elevations in serum activity of AST, ALT and ALP enzymes and levels of TBr compared with those of the normal rats. Treated KBrO₃-intoxicated rats with SO, JO and mixture of them have significant ameliorate in serum activity of AST, ALT and ALP enzymes and levels of TBr compared with those of the positive control rats. There was no significant changes in the activities of liver enzymes and serum levels of TBr between treated intoxicated groups with SO, JO, mixture of them and normal rats.

Table 5 shows serum malondialdehyde (MDA) and reduced glutathione (GSH) levels in experimental rats. In comparison to the normal control rats, KBrO₃-intoxicated caused a significant increase in serum MDA and decrease in serum GSH levels. Administration of SO, JO and mixture of them caused significant reversal in serum MDA and GSH levels toward the normal levels.
Table 4: Serum activity of AST, ALT, ALP enzymes and levels of TBr in normal rats and intoxicated rats with KBrO₃

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters as Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AST (U/L)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>19.71±3.20ᵇ</td>
</tr>
<tr>
<td>G 2: Positive control rats (KBrO₃ treated)</td>
<td>31.43±3.51ᵃ</td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO₃</td>
<td>20.71±3.45ᵇ</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO₃</td>
<td>21.29±2.56ᵇ</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO₃</td>
<td>20.57±1.51ᵇ</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p<0.05.

SD: Standard Deviation

Table 5: Serum levels of MDA and GSH in normal and intoxicated rats with KBrO₃

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters as Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MDA (nmol/dL)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>30.29±4.27ᶜ</td>
</tr>
<tr>
<td>G 2: Positive control rats (KBrO₃ treated)</td>
<td>80.43±4.24ᵃ</td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO₃</td>
<td>32.14±4.87ᵇ</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO₃</td>
<td>36.71±4.15ᵇ</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO₃</td>
<td>29.71±3.72ᶜ</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p<0.05.

SD: Standard Deviation

Table 6, shows a marked significant decrease in serum activity of glutathione peroxidase (GPx) superoxide dismutase (SOD) and catalase (CAT) enzymes as well as total antioxidant level in KBrO₃-intoxicated rats, compared with the normal rats. In comparison to the untreated KBrO₃-intoxicated rats, the administration of SO or JO or mixture of them to KBrO₃-intoxicated rats caused significant increase in serum
activity GPx, SOD and CAT enzymes and total antioxidant level toward
the normal levels.

Comparing KBrO₃- intoxicated rats with normal control group, the
present results revealed a marked significant increase in MDA and
decrease in GSH contents in liver tissues homogenates (Table 7). Treated
KBrO₃-intoxicated rats with SO or JO or mixture of them caused
significant decrease in MDA and increase in GSH content in liver tissues
homogenates compared to those of untreated KBrO₃-intoxicated rats.

Table 8, shows a marked significant decrease in the activity of
antioxidant GPx, SOD and CAT enzymes in liver tissues of KBrO₃-
intoxicated rats (positive control group ) compared to the normal rats
(negative control group). On the other hand, there was a significant
increased in the activity of antioxidant enzymes (GPx, SOD and CAT) in
SO + KBrO₃, JO + KBrO₃ and SO+JO+ KBrO₃ treated groups compared
with those of KBrO₃-intoxicated group alone.

Table 6: Serum activity of GPx, SOD and CAT enzymes and total
antioxidant in normal and intoxicated rats with KBrO₃

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters as Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GPx (mmol/dL)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>36.43±5.56ᵃ</td>
</tr>
<tr>
<td>G2: Positive control rats</td>
<td>16.00±1.53ᵇ</td>
</tr>
<tr>
<td>(KBrO₃ treated)</td>
<td></td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO₃</td>
<td>32.86±3.93ᵃ</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO₃</td>
<td>32.86±3.93ᵃ</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO₃</td>
<td>36.43±2.44ᵃ</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at
p< 0.05.
SD: Standard Deviation
Table 7: Concentrations of MDA and GSH in liver tissues of normal rats and intoxicated rats with KBrO₃

<table>
<thead>
<tr>
<th>Groups</th>
<th>Concentrations as Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MDA (nmol/gm tissues)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>50.00±4.08ᵇ</td>
</tr>
<tr>
<td>G 2: Positive control rats (KBrO₃-intoxicated)</td>
<td>80.57±4.47ᵃ</td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO₃</td>
<td>51.57±3.78ᵇ</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO₃</td>
<td>53.57±3.78ᵇ</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO₃</td>
<td>50.29±3.82ᵇ</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p< 0.05.
SD: Standard Deviation

Table 8: Activity of GPx, SOD and CAT enzymes in liver tissues of normal and intoxicated rats with KBrO₃

<table>
<thead>
<tr>
<th>Groups</th>
<th>Enzyme activities as Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GPx (nmol/min/mg tissues)</td>
</tr>
<tr>
<td>G 1: Normal control rats</td>
<td>70.14±4.78ᵃ</td>
</tr>
<tr>
<td>G 2: Positive control rats (KBrO₃ treated)</td>
<td>56.29±7.06ᵇ</td>
</tr>
<tr>
<td>G 3: 5% SO + KBrO₃</td>
<td>68.86±3.98ᵃ</td>
</tr>
<tr>
<td>G 4: 5% JO + KBrO₃</td>
<td>67.14±2.67ᵃ</td>
</tr>
<tr>
<td>G 5: 5% SO+JO+ KBrO₃</td>
<td>71.71±3.72ᵃ</td>
</tr>
</tbody>
</table>

Means with different letters in each column are significantly different at p< 0.05.
SD: Standard Deviation

Microscopically, liver sections of normal rats showing normal histological structure of hepatic lobule (Fig. 3). However, injected KBrO₃ induced morphopathological changes in the liver involved congestion of hepatic sinusoid, vacuolization of hepatocytes and necrosis of sporadic hepatocytes (Fig. 4). Liver sections of treated KBrO₃-intoxicated rats
with SO or JO revealed small vacuoles in the cytoplasm hepatocytes as shown in Figs. 5 and 6, respectively. There is no apparently histopathological changes were observed in liver sections of treated KBrO$_3$-intoxicated rats with the mixture of SO and JO.

Light micrograph of kidney sections from normal rats observed no histopathological changes of renal parenchyma as showed in Figure (7). In contrast, injected KBrO$_3$ induced sever damage in the kidney including thickening of glomerular basement membrane and dilatation of renal tubules with eosinophilic protein lost (Fig. 8). Administrations of SO and JO to KBrO$_3$-intoxicated rats induced a partial improvement in renal tissues which revealed small focal leucocytic cells aggregation (Fig. 9) and slight congestion of renal blood vessels (Fig. 10), respectively. However, mixture of SO with JO caused totally improvement in renal tissues.

Figure (3): Liver sections of normal rats from negative control group showing normal histological structure of hepatic lobule (H and E x 400).

Figure (4): Liver sections of KBrO$_3$-intoxicated rats from positive intoxicated control group showing congestion of hepatic sinusoid, vacuolization and necrosis of sporadic hepatocytes (H and E X 200).
Figure (5): Liver sections of treated KBrO₃-intoxicated rats with SO showing small vacuoles in the cytoplasm hepatocytes (H and E x 400).

Figure (6): Liver sections of treated KBrO₃-intoxicated rats with JO showing small vacuoles in the cytoplasm hepatocytes (H and E x 400).

Figure (7): Kidney sections of normal rats from negative control group showing normal histopathological structure of hepatic lobules (H and E X 200).

Figure (8): Kidney sections of KBrO₃-intoxicated rats from positive intoxicated control group showing thickening of glomerular basement membrane and dilatation of renal tubules with eosinophilic protein lost (H and E X 200).
4. DISCUSSION

The present study was designed to use sesame oil and jojoba oil to attenuate the KBrO₃-induced oxidative stress in rats as the animal model. In order to ensure the effectiveness of sesame oil and jojoba oil, the present study estimated their effects on food consumed, body weight, liver and kidney functions, oxidant process and antioxidant defense as well as histopathological effects on liver and kidneys in potassium bromate (KBrO₃)-induced oxidative stress in rats.

Small intestine is exposed on a continuous basis to high levels of ROS and requires a strong antioxidant defense system, which may preserve its endocrine, metabolic, digestive and absorptive functions (Circu and Aw, 2011). In this work, the amount of feed intake and values of body weight gain were significantly decreased after KBrO₃ treatment compared with those of the negative control group. This finding is consistent with the researches carried out by Ali and Al-Firdous (2012) and Wahba and...
Ibrahim (2013) who showed that oxidative stress induced by KBrO$_3$ in rats caused significant decreases in feed intake, body weight gain and feed efficiency ratio. The reduction in body weight is possibly attributed to lower food intake or from the direct toxic effect of KBrO$_3$ on the gastrointestinal tract which perhaps results in poor digestion of food or malabsorption of nutrients. It is well known that the major function of small intestine is to absorb important ions and molecules, which in turn depends on the structural integrity and available energy supplied by various metabolic pathways. Thus, it is possible that any alterations in these metabolic pathways caused by toxicants would affect the function of the small intestine (Ahmad et al., 2013). The brush border membrane lining the epithelial cells of small intestine is one of the most important cellular membranes, owing to its role in the digestion and absorption of nutrients. This process of digestion and absorption can be altered by drugs, chemicals, nutritional status and toxic pollutants (Circu and Aw, 2011). Injected KBrO$_3$ to rats induced oxidative stress (OS) and lowers the activities of several enzymes in the intestinal brush border membrane and induces several changes in the intestinal as revealed by extensive damage of villi and intestinal glands components with the lumen being filled with debris (Ahmad et al., 2013). The decrease in the activities of brush border membrane enzymes may be related the direct modification and consequent inactivation of these enzymes by KBrO$_3$ generated free radicals and reactive oxygen species (ROS) which lead to leakage or loss of these enzymes into the lumen of the intestine following ROS induced damage to the epithelial cells, especially the membrane. Also, increased lipid peroxidation affects intestinal membrane structure and function and result in a decrease in the activities of these enzymes Ahmad et al., (2015). In contrast, the administration of SO or JO or mixture of them to KBrO$_3$-intoxicated rats significantly increased feed intake and body weight gain compared to those of untreated KBrO$_3$-intoxicated rats. Since, KBrO$_3$ is well known to induce oxidative stress in the intestinal cells (Ahmad et al., 2014), antioxidant defense system may preserve its endocrine, metabolic, digestive and absorptive functions (Circu and Aw, 2011). The protective effect of SO or JO on KBrO$_3$-induced enzyme inactivation could have been due to the effectiveness of them in inhibiting the chain reactions of KBrO$_3$ generated free radicals before they reached their cellular targets. The consequent reduction in lipid peroxidation and
oxidative modification of brush border membrane enzymes might have contributed to the efficacy of SO or JO in attenuating the effects of KBrO₃. The present result was in accordance with Hsu et al., (2004) who showed that treated rats with sesame oil after Cecal Ligation and Puncture (CLP) -induce chronic septic shock caused increase in body weight and decrease oxidative stress. Sesame oil contains powerful natural antioxidants (sesamin, sesamol, sesamolin, and phytosterol) which give the oil very good oxidative stability (Alvarez and Rodríguez, 2000). Sesame oil potently decreases lipid peroxidation by inhibiting hydroxyl radical production and nitric oxide generation (Hsu et al., 2004). With regard to the effect of jojoba oil, previous studies revealed that jojoba oil helps to stabilizes oxidatively sensitive natural and synthetic active ingredients. The two most dominant factors inherent in natural lipid materials that affect their oxidative stability are molecular configuration and the presence of antioxidants (Johnson, 1992). Natural jojoba oil is one of the rich sources of tocopherols (Bhat et al., 2005) such as β, α and γ (El-Mallah and El-Shami, 2009) that are known to act as free radical scavengers. Therefore, administration of SO or JO have a positive effects protect gastrointestinal tract against oxidative stress induced of KBrO₃.

As mentioned in the present study, administration of jojoba oil caused significant decrease in feed intake and body weight compared to that of the normal rats. This observation provide the fact that an increase in body weight is independent on the amount of food consumed by the animals, but the content of the diet Schiffman et al., (1998). The decrease in feed intake and body weight gain in treated KBrO₃-intoxicated rats with JO as compared to those of normal rats may be due to its content of simmondsin which was reported to induce food restriction and growth retardation (Labib et al., 2012 and Mohamed et al, 2015). Rats feed on SO mixed with JO did not show an acute decrease in body weight gain or feed intake which may be due to the low levels of simmondsin (Ogawa et al., 1997 and Holser and Abbott, 1999). The mechanism of action is thought to be involved in the appetite suppressant effect as reported by Boozer and Herron (2006).

Hyperlipidemia and hypercholesterolemia are two of well-known complications of oxidative stress. Altered lipid metabolism and lipid peroxidation have been associated with KBrO₃ exposure (Chipman et
Cellular fatty acids are readily oxidized by reactive oxygen species to produce lipid peroxyl radicals and lipid hydroperoxides (Rice-Evans and Burdon, 1993). It is generally accepted that oxidative stress can lead to the oxidative degradation of lipids (Moller and Wallin, 1998), which can disrupt normal lipid metabolism. The present findings revealed that KBrO₃-induced toxicity rats have significant increase in serum levels of TG, TC, and LDL-c and decrease serum level of HDL-c. The present results are in accordance with those reported of Khan et al., (2011) and Shelbaya et al., (2014) who found that administration of KBrO₃ for rats resulted in a significant increase in serum total cholesterol, triglyceride and LDL-c levels and decrease HDL-c, compared to that of the normal rats. The abnormally high concentration of serum TG, TC, and LDL-c and decreased the serum level of HDL-c in KBrO₃-induced toxicity rats may be mainly due to raised oxidative stress as a result of increased production of reactive oxygen species and free radicals of KBrO₃ (Ahmed and Mahmood, 2012). ROS produced oxidative stress, attack many molecules such as protein, DNA and lipids (Halliwell and Gutteridge, 1999). Oxidative stress promotes several undesirable pathways including the formation of oxidized LDL (O-LDL) and oxidized cholesterol which encourages cholesterol accumulation in arterial tissues (Maharjan et al., 2008). Administration of SO or JO or mixture of them significantly reduced serum TC, TG and LDL-C concentrations and significantly increased serum HDL-c concentration, compared to that of the untreated KBrO₃-intoxicated rats. These results agreed with Haidari et al., (2016) that showed that diabetic treated rats with sesame oil had significantly lower levels of TG, TC and LDL-c, and higher levels of HDL-c than did the untreated diabetic control group. The hypolipidemic and hypocholesteremic effect of sesame oil may be attributed to its antioxidant properties. Sirato-Yasumoto et al., (2001) who reported that sesame oil have desirable physiological effects including antioxidant activity, serum lipid and cholesterol lowering potential and maintain good cholesterol (HDL-c) as proven in experimental animals and humans. In addition, sesame seed consumption increases plasma γ-tocopherol and enhances vitamin E activity, which is reported to prevent cancer and heart diseases (Cooney et al., 2001).. One of these molecules is sesamol that have antioxidant and free radical scavenging activities (Unnikrishnan et al., 2005 and Juan et al., 2005).
Seyedeh et al., (2013) found that after sesame oil consumption, TG, LDL-c, and VLDL-c, were significantly decreased and increased HDL due to presence of sesamin which have lipid lowering effect and inhibits the absorption of cholesterol from the intestine. In this regard, apart from increasing the fat oxidation, sesamin has also been proven to decrease lipogenesis by decreasing lipogenic enzymes of liver. Sesamin has been shown to decrease the lipogenic gene expression of sterol regulatory element binding protein-1 (SREBP-1), acetyl-CoA carboxylase and fatty acid synthase, which means less fat, is esterified in the liver and therefore less fat synthesis. It has also been posited that lignans present in sesame oil may play a role in the improvement of lipid profile. Sesame lignans such as sesamin and episesamin modulate cholesterol metabolism by inhibiting the synthesis and absorption of cholesterol in stroke-prone spontaneously hypertensive rats (Lim et al., 2007). In another study, confirmed that the ingestion of sesamin together with α-tocopherol synergistically reduced the concentration of blood cholesterol following a high-cholesterol diet (Rogi et al., 2011). Moreover, Sedigheh et al., (2013) mentioned that rabbits supplemented with 5% sesame oil were found to have lower circulating concentrations of TG, TC, LDL-c and increase HDL-c due to phytoestrogen content as estrogen increasing HDL cholesterol with lowering LDL-c cholesterol.

With regard to the effect of jojoba oil, the present results agreed with Shahwan (2014) who reported that jojoba oil (Simmondsia chinensis) significantly reduce serum levels of TG and TC and increase HDL-c in rabbits fed on high-cholesterol diet. The hypolipidemic effect of jojoba oil may be attributed to its content of natural antioxidant which postulated to be anallylic derivative of hydroxytoluene (Johnson, 1992), omega-3 fatty acid (Bouali et al., 2008) and monounsaturated fatty acids (Busson-Breysse et al., 1994). Numerous studies have demonstrated that oils containing high amounts of monounsaturated fatty acids (MUFAs) decrease TG, TC, and LDL-c levels (Makni et al., 2010). It has been documented that MUFAs may reduce LDL cholesterol, while it might possibly increase high-density lipoprotein (HDL) cholesterol (FAO/WHO, 2010). The effectiveness of mixing SO with JO in reducing TC, TG, LDL-c and increasing HDL-c may be related to the synergistic effect of both oils against KBrO3-induced oxidative stress in rats.
Increased production of reactive oxygen species (ROS) and free radicals has been implicated in KBrO$_3$-induced toxicity. These radicals can cause extensive tissue damage by reacting with macromolecules like proteins, nucleic acids and membrane lipids which causes an imbalance in homeostasis and leads to tissue injury (Ahmad and Mahmood, 2012). Active oxygen radicals generated from KBrO$_3$ are implicated in its toxic and carcinogenic effects, especially because KBrO$_3$ produced 8-hydroxydeoxyguanosine in the rat kidneys (Kurokawa et al., 1990). Therefore, KBrO$_3$ has a potent nephrotoxic agent that can mediate renal oxidative hydrogen peroxide formation with reduction in renal antioxidant enzymes and contributes to the cellular redox status that impairs membrane protein activities in rats (Chiagoziem and Ebenezer, 2012). The present study revealed that KBrO$_3$-intoxicated caused marked significant increase in serum levels of urea nitrogen (UN), uric acid (UA) and creatinine (Cr) compared to those of normal rats. These results were confirmed with the obtained results of histopathological investigation which showed that injected of KBrO$_3$ induced sever damage in the kidney including thickening of glomerular basement membrane and dilatation of renal tubules with eosinophilic protein lost. The elevation in serum urea nitrogen, uric acid and creatinine, indicate to the impairment in renal functions which may be result from intrinsic renal lesions and decreased perfusion of the kidney caused by KBrO$_3$-induced oxidative stress and the alterations in the antioxidant defensive system. This result was in accordance with the obtained results by Khan et al., (2011) and Shelbaya et al., (2014) who reported that administration of KBrO$_3$ rats resulted in a significant increase in serum urea, creatinine and uric acid levels. Also, Ali and Al-Firdous (2012), reported that KBrO$_3$-intoxicated rats caused significant increase in serum urea nitorge, uric acid and creatine, compared to that of the normal rats. The adverse effect of KBrO$_3$ may be related to the oxidative stress action of it as reported by Ahmed and Mahmood (2012) who that showed a decline in renal function following subchronic and chronic exposure KBrO$_3$ based on the oxidative stress action of bromate. The bromate toxicity in rat kidney includes changes in energy consumption and utilization in renal cells that involved up-regulation of glycolytic processes, possibly resulting from altered mitochondrial function. In addition to, Khan and Sultana (2004) reported that kidney damage is attributed to reduction in renal glutathione
content, activities of renal antioxidant enzymes and phase-II metabolising enzymes with enhancement in xanthine oxidase, lipid peroxidation, gamma-glutamyl transpeptidase and hydrogen peroxide ($H_2O_2$).

Liver is a major organ attacked by ROS, when the ROS is excessive, the homeostasis will be disturbed, resulting in oxidative stress, which plays a critical role in liver diseases, degenerative disorders and other chronic (Li et al., 2014). Reactive oxygen species (ROS) exposure causes variation at biochemical level. It affects the level of liver marker enzymes in serum, antioxidant enzymes and non enzymatic antioxidant compounds (Khan et al., 2011). Sahreen et al., (2011) reported that free radicals significantly elevate serum marker enzyme activities (AST, ALP, ALT) and serum levels of total protein and bilirubin indicated to severe necrosis of liver in rats. Assessment of liver function can be made by estimating the activities of serum AST, ALT and ALP which are enzymes originally present in higher concentration in cytoplasm which leak into blood stream in conformity with the extent of liver damage (Venukumar and Latha, 2004). Indication of hepatocellular integrity most commonly measured in clinical toxicology studies are the enzymes AST, ALT and bilirubin levels (Ballet, 1997). In the present investigation, the increase in serum activities of AST, ALT and ALP enzymes and serum bilirubin level have been observed in intoxicated rats with potassium promate (positive control group) compared to that of the normal control group, indicating to the hepatotoxic role of potassium promate. This finding agreed with a previous studies showed that the administration $KBrO_3$-induced serum level of hepatic marker enzymes (ALT, AST and ALP) (Khan et al., 2011, Dimkpa et al., 2013 and Shelbaya et al., 2014). Also, Ali and Al-Firdous (2012) reported that $KBrO_3$-intoxicated rats had significant increase in serum activity of AST, ALT, ALP and serum total bilirubin as compared to the normal rats. Therefore, an elevation in the levels of serum marker enzymes is generally regarded as one of the most sensitive index of the hepatic damage (Kapil et al., 2005). As indicated in literature, the enzymes ALT and AST, are present for assessing of hepatic injury in the hepatic and biliary cells (Jensen et al., 2004). ALP reaches the liver mainly from bone and its elevation in serum occurs through biliary excretion during hepatobiliary diseases (Kothavade et al., 1996). These elevated enzymes in hepatocytes usually released into circulation causing increase in their serum levels under hepatocellular injury or
inflammation of the biliary tract cells (Jensen et al., 2004). Determination of serum bilirubin represents an index for the assessment of hepatic function and any abnormal increase in the levels of bilirubin in the serum indicate hepatobiliary diseases and severe disturbance of hepatocellular function (Martin and Friedman, 1992). The present results were confirmed by the obtained results of histopathological investigation, which revealed that injected of KBrO$_3$ induced morphopathological changes in the liver involved congestion of hepatic sinusoid, vacuolization of hepatocytes and necrosis of sporadic hepatocytes.

Enzymatic and non-enzymatic antioxidant systems are essential for cellular response in order to deal with oxidative stress under physiological condition. Therefore, activities of antioxidant enzyme (CAT, SOD, and GPx) and non-enzymatic electron receptors such as GSH are affected and used as indexes to evaluate the level of oxidative stress (Medina and Moreno-Otero, 2005). The impairment of antioxidant defense system is considered a critical event in KBrO$_3$-induced oxidative stress. Exposure of KBrO$_3$ is characterized by the depletion of tissue and circulating enzymatic and non-enzymatic antioxidants. Toxicological studies have suggested that KBrO$_3$ is an oxidizing agent and increased lipid peroxidation (Deangelo et al., 1998).

In the current study, lipid peroxidation level was estimated by the measurement of MDA, its level was significantly elevated in serum and liver tissues of intoxicated rats with KBrO$_3$ compared to those of the normal rats, thus suggesting increased oxidative stress. In addition, serum and liver tissues of GSH levels and activity of antioxidant enzymes (GPx, SOD and CAT) were significantly increased in KBrO$_3$-intoxicated rats compared to that of the normal rats. The present results agreed with Khan et al., (2003) who showed reduction in antioxidant enzymes and enhancement of lipid peroxidation in injected rats with 125 mg/kg b.wt. KBrO$_3$ intraperitoneally. Also, El-Sokkary (2006) observed significant increase in malondialdehyde in liver tissues as an indicator of lipid peroxidation. Therefore, KBrO$_3$ induces oxidative stress in human erythrocytes through the generation of reactive oxygen species and alters the cellular antioxidant defense system (Mir-Kaisar et al., 2011). Also, Ali and Al-Firdous, (2012) showed significant decrease in the activity of serum SOD, GPx and catalase, and liver SOD, GPX and glutathione
transferase (GST) in treated rats with potassium bromated. It has been showed that KBrO₃ produces free oxygen radicals which cause oxidative stress and DNA damages (Umemura et al., 1998) and cause nephrotoxicity and hepatotoxicity; decreases the tissue soluble proteins, antioxidant enzymes. The decrease in antioxidant enzymes (SOD and CAT) may be due to the increase in reactive oxygen species (ROS) produced by metabolism of KBrO₃. KBrO₃ depleted glutathione (GSH) content in various tissues which causes decrease in phase II metabolizing enzymes like glutathione peroxidase (GPx) and glutathione reductase (GSH). It also increase thiobarbituric acid reactive substances (TBARS) contents, causes lipid peroxidation and disrupts liver profile including gammaglutamyltransferase (γ-GT), alkaline phosphatase (ALP) and protein concentration (Farombi et al., 2002).

Regarding to the biological and histopathological effects of sesame oil (SO), jojoba oil (JO) and mixture of them on KBrO₃-intoxicated rats, the most important result drawn from the current study is the powerful ability of them in induce significant amelioration in kidneys function as indicated by lower serum levels of urea nitrogen, uric acid and creatinine, and liver functions as indicated by lower activity of AST, ALT, ALP enzymes and serum bilirubin level, as well as antioxidant defense as indicated by increased total antioxidant capacity, lower serum and liver concentrations of MDA and increased GSH concentration, and activity of GPx, SOD and CAT enzymes. In addition to, the improvement in histopathological structure of kidneys and liver, compared with untreated KBrO₃-intoxicated rats. These amendments were more detectable in treated rats with mixture of SO with JO. These results were in accordance Hsu and Liu, (2004) who indicated that sesame oil might have therapeutic value and potently reduces oxidative stress and attenuates hepatic and renal dysfunctions. Periasamy et al., (2010) reported that sesame oil significantly decreased serum urea nitrogen and creatinine, renal osteopontin expression, superoxide anion, nitric oxide, peroxynitrite radical and lipid peroxidation levels as well as recovered and regenerated renal tubules in gentamicin-treated rats. Liu et al., (2015) revealed that sesame oil significantly decreased hydroxyl radical, peroxynitrite level and lipid peroxidation in deoxycorticosterone acetate induced chronic kidney disease in rats. Recently, Haidari et al., (2016) found a significant increase in serum TAC and decrease in MDA concentration following the
treatment of the diabetic rats with sesame oil. Morris (2002) reported that sesame oil is more effective in the treatment of high-fat diet toxicity through its ability to decrease the elevated activity of ALT and AST at the cellular level. SO contain some powerful antioxidants (IP-6, phytate, lignin, pinoresionoly, vitamin E, Lecithin, myristic acid and linolate) which may prevent free radical formation and scavenge free radicals that already formed. Additionally, Hemalatha et al., (2004) reported that rats fed on supplemented diet with sesame oil had significant increase in the activities of hepatic antioxidant enzymes and protect the liver against Fe²-induced oxidative damage due to the presence of lignans. Also, previous study demonstrated that sesame oil may be useful in managing oxidative stress-associated diseases such as atherosclerosis, diabetes mellitus, obesity, chronic renal failure, rheumatoid arthritis, and neurodegenerative diseases including Alzheimer's disease (Lee et al., 2006). Moreover, sesame oil has multiple physiological functions such as decreasing blood lipids and arachidonic acid levels, increasing antioxidative ability and γ-tocopherol bioavailability, and providing anti-inflammatory function and potential estrogenic activity. Many health promoting effects are attributed to its lignans (Sedigheb et al., 2013). The effectiveness effect of sesame oil may be related to its content of sesamin, sesamolin and sesaminol lignan fractions, which are known to play an important role in its oxidative stability and antioxidative activity (Elleuch et al., 2007). It is widely known as one of the natural health promoting foods that have the potential to prevent various disorders such as hypertension, hypercholesterolemia cancer and aging (Habila et al., 2013). Also, Chavali et al., (2001) reported that nonfat sesame oil lignans, such as sesamin or sesamol contribute to the antioxidation of sesame oil in rats with sepsis. Akimoto et al., (1993) showed that sesamin lowered the ethanol inhalation-induced increase in SGPT activity. It has been shown that lignans (schisantherin compounds) containing the methylenedioxy group protect the liver from injury against viral hepatitis in mice and human subjects. Therefore, the hepatoprotective effects of sesame oil observed in the present study could be due to the methylenedioxy group present in sesame lignans (sesamin and sesamolin) (Gao and Rao, 1993). In addition, Yamashita et al., (1995) reported that rats fed the sesame-oil had maximum protection against Fe²-induced lipid peroxidation; this appears due to the increase in hepatic α-tocopherol. Other, several studies
have demonstrated that sesame and its constituent lignans (sesamin, sesamol, episesamin, and sesamolin) possess antioxidantive properties as they improve total antioxidant capacity (TAC), suppress destructive oxygen-free radicals, and prevent oxidative stress damage (Wichitsranoi et al., 2011). Karatzis et al., (2012) reported that sesame oil consumption (35 g/d) significantly increase plasma TAC after 2 weeks in their male subjects with hypertension. These findings are in accordance with several studies on rats which reported that sesame oil may reduce oxidative stress (Hsu et al., 2008). Wichitsranoi et al., (2011) demonstrated that the administration of black sesame meal capsules (2.52 g/d) for 4 weeks significantly decrease serum MDA levels in human subjects with prehypertension. In addition, Roghani et al., (2013) showed that sesamin treatment at a dose of 20 mg/kg for 7 weeks attenuate the increase in MDA content and reduced the activity of superoxide dismutase in diabetic rats. It has been suggested that dietary lignans provided through the consumption of sesame seeds or oil may protect the liver against Fe-induced oxidative damage. Hemalatha et al., (2004) showed that superoxide dismutase activity was greater in Fe²⁻-induced oxidative stress rats following the administration of sesame oil (100 g/kg) plus sesamin (0.4 g/kg). The authors concluded that sesame lignans might enhance the ability to mop up superoxide radicals formed during Fe²⁻-induced oxidative stress. Likewise, Hou et al., (2003) studied the effects of sesame lignans (i.e., sesamin and sesamolin) on antioxidant enzyme activities in in vitro systems using cell lines and reported that the sesame antioxidants spared superoxide dismutase and catalase in hypoxia-stressed PC12 cells in a dose-dependent manner, an effect that may be related to their radical scavenging effect.

With regard to the effect of jojoba oil (JO) the present results agreed with El-Shamy et al., (2001) who reported that jojoba oil proved many valuable uses and has a hepatoprotective and anti inflammatory effects. Sobhy et al., (2015) reported that jojoba oil caused significant decrease in serum levels of ALP and urea nitrogen and concentration of MDA in liver tissues and increased liver concentration of GSH and activity of CAT enzyme in cadmium- intoxicated rats. Sobhy et al., (2016) revealed that treated rats fed on aflatoxin -contaminated diet with jojoba oil had significant decrease in serum levels of urea nitrogen, creatinine and activity of AST, ALT, ALP and levels of MDA in liver tissues and...
increased activity of antioxidant enzyme and level of GSH in liver tissues. The effectiveness of jojoba oil in protective kidney and liver as well as improve antioxidant defense in KBrO3-inoxicated rats may be related to its antioxidant properties. Natural Jojoba oil esters help in stabilizes oxidative sensitive natural and synthetic active ingredients (Johnson, 1992). Natural Jojoba oil contains tocopherols (Bhat et al., 2005) such as β, α and γ- tocopherols which are known to act as free radical scavengers (El-Mallah and El-Shami, 2009). The presence of α-tocopherol, which is the pharmacological active form (Bhat et al., 2005) may explain the biological activities of the antioxidant activity, because of the antioxidant activity of vitamin E (Kumar et al., 2012). Vitamin E regulates oxidation processes in the body and acts as a powerful antioxidant. Previous studies showed that vitamin E can ameliorate the damaging effects of reactive oxygen species (ROS) (Shalaby et al., 2004). Moreover, vitamin E protected DNA from oxidative stress of free radicals (Jedlinska et al., 2006) in rats and increased the activity of antioxidant enzymes involved in the removal of ROS generated by cell metabolism (Caetano et al., 2013). In addition to Adikwu and Nelson (2013) reported that vitamin E exhibited hepatoprotective effect and decreased oxidative stress in the liver manifested through decrease in microsomal lipid peroxidation. It normalized activity of AST, ALT, ALP, glutathione superoxide dimutase, lactose dehydrogenase, and level of bilirubin, MDA and improved histopathological changes in the liver induced by chemical agents. Moreover, Jojoba contains a natural antioxidant postulated to be an allylic derivative of hydroxytoluene and was isolated eight glucoside compounds from jojoba seeds (Van Boven et al., 2000). Additionally, Jojobenoic acid in jojoba seed extract also has antioxidant activity and the ability to bind metal ions, representing an additional mechanism underlying their pharmacological effects (Bouali et al., 2008). Also, Jojoba oil helps in the storage and utilization of vitamin A and E in liver and also act as antioxidant (Hanan et al., 1998).

5. CONCLUSION

The present study concluded that sesame oil and jojoba oil had hypolipidemic and hypocholestermic effect and exhibited improvement in liver and kidneys functions against injury induced by potassium bromate. Sesame oil and jojoba oil had the ability in reduced oxidative stress and
augmented antioxidants in KBrO$_3$-intoxicated rats. The mixture consisting of sesame oil and jojoba oil provide the best improvement in all biochemical measurements and histological structure. Hence, regular intake of them or using it for enriching food product may help functional foods to improve health status.

6. REFERENCES


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الأثر المحتمل للوقاية والتحسين من تأثير زيت السمسم وزيت الجوجوبا ضد الإجهاد التأكسدي المحدث ببرومات البوتاسيوم في الجرذان

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المملخص العربي:

للأساسية دوراً هاماً في إحداث الكثير من الأمراض المزمنة مثل تصلب الشرايين وارتفاع ضغط الدم وداء السكري والسرطان. ويؤدي تناول الأغذية الغنية بمضادات للأكسدة يمكن أن تمنع أو تؤخر من التأثيرات الضارة الناجمة من عمليات الأكسدة. وقد تم تصميم هذه التجربة لدراسة تأثير زيت السمسم وزيت الجوجوبا في الوقاية وتخفيف الإجهاد التأكسدي الناجم عن برومات البوتاسيوم وذلك باستخدام الجرذان كحيوانات تجارب. وقد تم تقسيم خمسة وثلاثين جرذ عشوائياً على خمس مجموعات وتحتوي كل مجموعة على سبعون من الجرذان. واستخدمت المجموعة الأولى كمجموعة ضابطة سالبة (مجموعة طبيعية) وكانت تتناول الغذاء العادي. بينما المجموعات الاربعة الأخرى تم حقنها داخل الغشاء البريتوني بجرعة واحدة (125 ملجم/كم من وزن الجسم) من برومات البوتاسيوم لحداث الإجهاد التأكسدي. واستخدمت المجموعة الثانية كمجموعة ضابطة موجبة على الغذاء العادي. بينما المجموعات 3 و 4 و 5 تم تغذيتها على الغذاء الدائري المضاف إليه 5% زيت السمسم، زيت الجوجوبا وخلطهما على التوالي. وأظهرت النتائج أن الجرذان المحونون ببرومات البوتاسيوم بجانب تناولها الغذاء المضاف إليه زيت السمسم أو زيت الجوجوبا أو الخلط منهما لديهم نقص واضح في مستويات الكولسترول، الجليريدات الثلاثية، الليبروتيينات منخفضة الكثافة، البيليروبين الكلي، بيروبين، هيم بروتين، حمض البوليك، الكريتيتين، مالوندالديهيد، نشاط إنزيمات اسبرتنيز أمينوتانسفيريز، الآلانين امينوتانسفيريز والفوسفاتيز. بينما كان لديهم زيادة كبيرة في كمية الغذاء المستهلك، زيادة وزن الجسم، ومستويات الليبروتيينات مرتفعة الكثافة، مضادات الأكسدة الكلية، الجلوتاثيون، ونشاط الإنزيمات المضادة للأكسدة (الجلوتاتيون برواكسيد، السوبر أوكسيد ديميونوارس، والكاليتاز). كما أظهرت النتائج وجود نقص معنوي في تركيز المالوندالديهيد وزيادة معنوية في تراكيز
الجلوتاثيون ونشاط الأنزيمات المضادة للأكسدة (الجلوتاثيون بروكسيداز، السوبر أوكسيد ديميوتاز والكتاليز) في خلايا الكبد وذلك كمقارن بما كانت موجودة في الجرذان المعالجة ببرومات البوتاسيوم فقط (المجموعة الضابطة الموجبة). كما كان هناك تحسن واضح في انسيجة الكبد والكلى. وكانت أفضل النتائج لجميع المؤشرات البيوكيميائية والهستوبيولوجية لانسجة الكبد والكلى والتي كانت تميل نحو النتائج الطبيعية في الجرذان المعالجة بخليط زيت السمسم مع الجوجوبا. وفي النهاية، اقترحت النتائج الحالية أن التناول المنتظم لزيت السمسم والجوجوبا فائدة في تحسين وظائف الكبد والكلى والحماية ضد الإجهاد التأكسدي المحدث ببرومات البوتاسيوم في الجرذان وذلك من خلال إظهار النشاط المضاد للأكسدة كما وجد أن الخليط من زيت السمسم والجوجوبا يعطي أفضل النتائج.