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 ($KBrO_3$)-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 $KBrO_3$ 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 $KBrO_3$ induced oxidative stress in rats by exhibiting stronger antioxidant activity. The mixture of SO with JO provide the preferable effects.

Keywords: Potassium Bromate ($KBrO_3$) – 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 ($KBrO_3$) 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 $KBrO_3$-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 $KBrO_3$-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 ($KBrO_3$) in the form of a white powder was purchased from El-Gomhoryia 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 feed 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 = Final Body Weight - 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 centrifuged for 10 minutes at 3000 rpm for serum separation. Serum samples were carefully aspired 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 $KBrO_3$-intoxicated rats (positive control group) were decreased significantly, compared with those of the normal rats. On the other hand, treated $KBrO_3$-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 $KBrO_3$

Groups	Parameters as Mean ± SD			
	FI (g/d)	**IBW (g)**	**FBW (g)**	**BWG (g)**
G 1: Normal control rats	18.29 ± 1.25^{a}	199.29 ± 4.50^{a}	244.00 ± 3.37^{a}	44.71 ± 6.18^{a}
G2: Positive control rats ($KBrO_3$ treated)	14.43 ± 1.13^{c}	200.70 ± 3.55^{a}	229.86 ± 6.84^{c}	29.14 ± 5.18^{c}
G 3: 5% SO + $KBrO_3$	17.00 ± 0.82^{b}	200.29 ± 4.07^{a}	239.00 ± 3.16^{ab}	38.00 ± 6.14^{ab}
G 4: 5% JO + $KBrO_3$	16.57 ± 1.13^{b}	200.68 ± 3.27^{a}	236.71 ± 4.72^{b}	35.86 ± 5.70^{b}
G 5: 5% SO+JO+ $KBrO_3$	17.71 ± 0.78^{ab}	199.00 ± 3.87^{a}	240.43 ± 5.13^{ab}	41.43 ± 6.95^{ab}

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

SD: Standard Deviation

Table 2: Serum levels of TG, TC, LDL-c and HDL-c in normal rats and intoxicated rats with $KBrO_3$

Groups	Parameters as Mean ± SD			
	TG (mg/dL)	**TC (mg/dL)**	**LDL-C (mg/dL)**	**HDL-C (mg/dL)**
G 1: Normal control rats	117.14 ± 3.39^{b}	54.43 ± 4.24^{c}	16.00 ± 2.58^{c}	36.71 ± 2.14^{a}
G2: Positive control rats ($KBrO_3$ treated)	142.43 ± 4.58^{a}	88.29 ± 3.73^{a}	41.00 ± 3.37^{a}	25.57 ± 3.78^{b}
G 3: 5% SO + $KBrO_3$	115.29 ± 4.54^{b}	58.29 ± 3.73^{bc}	23.57 ± 3.95^{b}	34.29 ± 1.70^{a}
G 4: 5% JO + $KBrO_3$	117.71 ± 2.93^{b}	60.29 ± 3.73^{b}	24.00 ± 1.83^{b}	34.14 ± 1.68^{a}
G 5: 5% SO+JO+ $KBrO_3$	116.00 ± 3.92^{b}	58.00 ± 3.46^{bc}	21.29 ± 1.25^{b}	36.28 ± 2.22^{a}

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 $KBrO_3$

Groups	**Serum Levels as Mean ± SD**		
	UN (mg/dL)	**UA(mg/dL)**	**Cr (mg/dL)**
G 1: Normal control rats	17.14 ± 2.34^{c}	3.71 ± 0.99^{b}	2.03 ± 0.51^{b}
G2: Positive control rats ($KBrO_3$ treated)	28.71 ± 2.22^{a}	6.36 ± 0.90^{a}	4.86 ± 0.85^{a}
G 3: 5% SO + $KBrO_3$	23.43 ± 3.10^{b}	3.79 ± 0.91^{b}	2.64 ± 0.80^{b}
G 4: 5% JO + $KBrO_3$	24.29 ± 2.43^{b}	4.50 ± 1.55^{b}	2.81 ± 0.72^{b}
G 5: 5% SO+JO+ $KBrO_3$	21.86 ± 3.02^{b}	3.29 ± 0.99^{b}	2.46 ± 0.42^{b}

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 $KBrO_3$- 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, $KBrO_3$-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 $KBrO_3$

Groups	Parameters as Mean ± SD			
	AST (U/L)	ALT (U/L)	ALP (U/L)	TBr (mg/dl)
G 1: Normal control rats	19.71±3.20[b]	16.29±1.98[b]	40.43±4.58[c]	0.37±0.08[b]
G2: Positive control rats ($KBrO_3$ treated)	31.43±3.51[a]	23.29±1.98[a]	66.87±4.67[a]	2.34±0.37[a]
G 3: 5% SO + $KBrO_3$	20.71±3.45[b]	18.43±3.26[b]	56.43±4.76[b]	0.54±0.16[b]
G 4: 5% JO + $KBrO_3$	21.29±2.56[b]	19.29±3.04[b]	57.86±4.88[b]	0.54±0.13[b]
G 5: 5% SO+JO+ $KBrO_3$	20.57±1.51[b]	17.43±2.51[b]	55.14±3.02[b]	0.43±0.08[b]

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 $KBrO_3$

Groups	Parameters as Mean ± SD	
	MDA (nmol/dL)	GSH (nmol/dL)
G 1: Normal control rats	30.29±4.27[c]	172.86±8.10[a]
G2: Positive control rats ($KBrO_3$ treated)	80.43±4.24[a]	71.43±7.48[b]
G 3: 5% SO + $KBrO_3$	32.14±4.87[b c]	166.43±5.56[a]
G 4: 5% JO + $KBrO_3$	36.71±4.15[b]	167.14±5.67[a]
G 5: 5% SO+JO+ $KBrO_3$	29.71±3.72[c]	168.57±7.48[a]

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 $KBrO_3$ - 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 $KBrO_3$- intoxicated rats caused significant increase in serum

activity GPx, SOD and CAT enzymes and total antioxidant level toward the normal levels.

Comparing $KBrO_3$- 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 $KBrO_3$-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 $KBrO_3$-intoxicated rats.

Table 8, shows a marked significant decrease in the activity of antioxidant GPx, SOD and CAT enzymes in liver tissues of $KBrO_3$-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 + $KBrO_3$, JO + $KBrO_3$ and SO+JO+ $KBrO_3$ treated groups compared with those of $KBrO_3$-intoxicated group alone.

Table 6: Serum activity of GPx, SOD and CAT enzymes and total antioxidant in normal and intoxicated rats with $KBrO_3$

Groups	Parameters as Mean ± SD			
	GPx (mmol/dL)	**SOD (U/ml)**	**CAT (mmol/dl)**	**Total antioxidant (mmol trolox/liter)**
G 1: Normal control rats	36.43±5.56^{a}	10.00±2.52^{a}	73.57±5.56^{a}	3.90±0.93^{a}
G2: Positive control rats ($KBrO_3$ treated)	16.00±1.53^{b}	3.50±1.08^{d}	32.14±4.22^{c}	2.01±0.52^{c}
G 3: 5% SO + $KBrO_3$	32.86±3.93^{a}	8.43±1.99^{ab}	71.43±4.76^{ab}	3.29±0.57^{ab}
G 4: 5% JO + $KBrO_3$	32.86±3.93^{a}	8.57±2.23^{ab}	76.43±2.44^{a}	3.79±0.70^{ab}
G 5: 5% SO+JO+ $KBrO_3$	36.43±2.44^{a}	7.57±1.40^{c}	67.86±5.67^{b}	3.00±0.65^{b}

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 $KBrO_3$

Groups	Concentrations as Mean ± SE	
	MDA (nmol/gm tissues)	**GSH (mmol/gm tissues)**
G 1: Normal control rats	50.00±4.08[b]	15.86±1.21[a]
G2: Positive control rats ($KBrO_3$-intoxicated)	80.57±4.47[a]	5.50±1.38[c]
G 3: 5% SO + $KBrO_3$	51.57±3.78[b]	14.14±2.41[ab]
G 4: 5% JO + $KBrO_3$	53.57±3.78[b]	12.71±2.14[b]
G 5: 5% SO+JO+ $KBrO_3$	50.29±3.82[b]	14.14±2.41[ab]

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 $KBrO_3$

Groups	Enzyme activities as Mean ± SE		
	GPx nmol/min/mg issues)	**SOD (U/mg tissues)**	**CAT (nmol/min/mg tissues)**
G 1: Normal control rats	70.14±4.78[a]	2.24±0.37[a]	0.33±0.21[a]
G2: Positive control rats ($KBrO_3$ treated)	56.29±7.06[b]	0.71±0.21[b]	0.12±0.02[b]
G 3: 5% SO + $KBrO_3$	68.86±3.98[a]	1.89±0.39[a]	0.31±0.12[a]
G 4: 5% JO + $KBrO_3$	67.14±2.67[a]	1.84±0.35[a]	0.27±0.10[a]
G 5: 5% SO+JO+ $KBrO_3$	71.71±3.72[a]	1.99±0.40[a]	0.37±0.11[a]

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 $KBrO_3$ 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 $KBrO_3$-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 KBrO3 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.

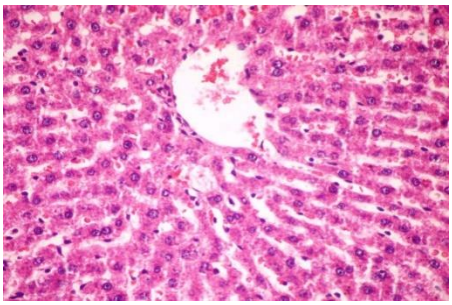

Figure (3): Liver sections of normal rats from negative control group showing normal histological structure of hepatic lobule (H and E x 400).

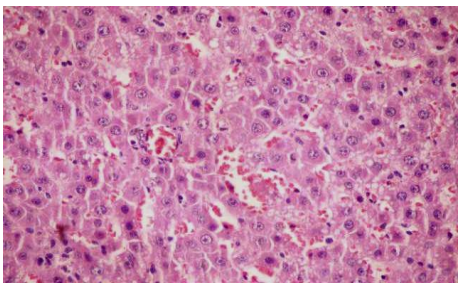

Figure (4): Liver sections of $KBrO_3$-intoxicated rats from positive intoxicated control group showing congestion of hepatic sinusoid, vacuolization and necrosis of sporadic hepatocytes (H and E X 200).

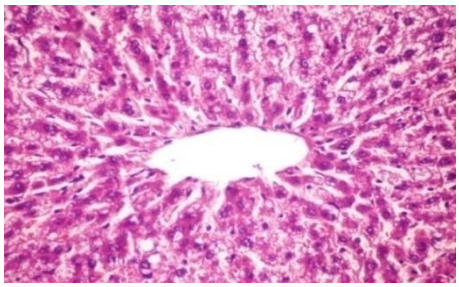

Figure (5): Liver sections of treated $KBrO_3$-intoxicated rats with SO showing small vacuoles in the cytoplasm hepatocytes (H and E x 400).

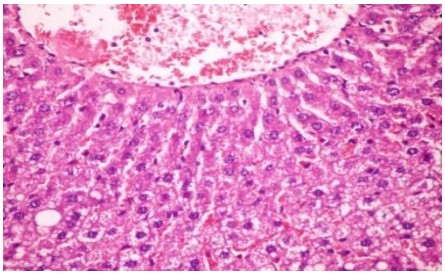

Figure (6): Liver sections of treated $KBrO_3$-intoxicated rats with JO showing small vacuoles in the cytoplasm hepatocytes (H and E x 400).

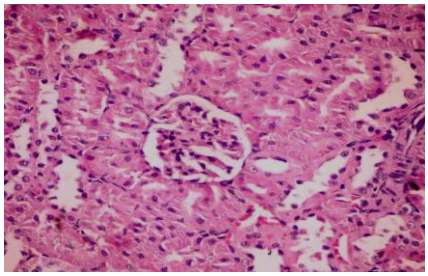

Figure (7): Kidney sections of normal rats from negative control group showing normal histopathological structure of hepatic lobules (H and E X 200).

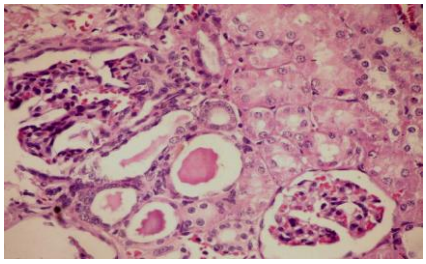

Figure (8): Kidney sections of $KBrO_3$-intoxicated rats from positive intoxicated control group showing thickening of glomerular basement membrane and dilatation of renal tubules with eosinophilic protein lost (H and E X 200).

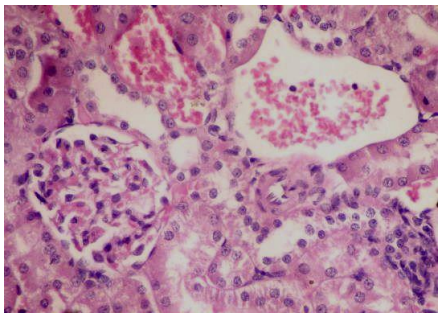

Figure (9): Kidney sections of treated $KBrO_3$-intoxicated rats with SO showing small focal leucocytic cells aggregation (H and E x 200).

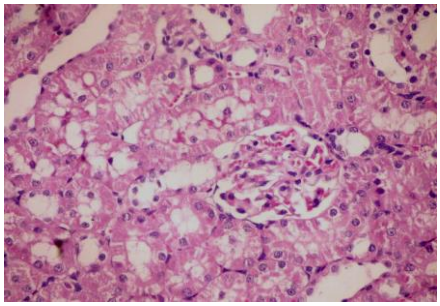

Figure (10): Kidney sections of treated $KBrO_3$-intoxicated rats with JO showing slight congestion of renal blood vessels (H and E X 200).

4. DISCUSSION

The present study was designed to used sesame oil and jojoba oil to attenuate the $KBrO_3$-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 ($KBrO_3$)-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 $KBrO_3$ 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 $KBrO_3$. 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 $KBrO_3$.

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 $KBrO_3$-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 $KBrO_3$ exposure **(Chipman *et***

***al.*, 1998)**. 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 $KBrO_3$-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 $KBrO_3$ 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 $KBrO_3$-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 $KBrO_3$ **(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 $KBrO_3$-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 hypocholestermic 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 $KBrO_3$-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 KBrO3 are implicated in its toxic and carcinogenic effects, especially because KBrO3 produced 8-hydroxydeoxy-guanosine 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 createnine, 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 perioxidation. 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 $KBrO_3$ 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. $KBrO_3$ 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 $KBrO_3$-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 $KBrO_3$-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 Fe^{2}-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 Fe^{2}-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 antioxidative properties as they improve total antioxidant capacity (TAC), suppress destructive oxygen-free radicals, and prevent oxidative stress damage (**Wichitsranoi *et al.*, 2011**). **Karatzi *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 attenuated 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 Fe^{2} -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 Fe^{2} -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 $KBrO_3$-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

Abou-Gharbia, H.A., Shehata, A.A. and Shahidi, F. (2000). Effect of processing on oxidative stability and lipid classes of sesame oil. Food Res. Int. 33(5): 331–340.

Adikwu, E. and Nelson, B. (2013). Hepatoprotective effect of vitamin E. Am. J. Pharmacol. Toxicol., 7(4): 154- 163.

Aebi, H. (1984). Catalase *in vitro*. Methods Enzymol., 105:121-126.

Ahmad ,M.K., Amani, S. and Mahmood, R. (2014). Potassium bromate causes cell lysis and induces oxidative stress in human erythrocytes. Environ Toxicol., 29(2);139-148.

Ahmad, M.K. Khan, A.A. Ali, S.N. and Mahmood, R. (2015). Chemoprotective effect of Taurine on potassium bromate-induced DNA damage, DNA-protein cross-linking and oxidative stress in rat Intestine.PLOS ONE. pp,1-16.

Ahmad, M.K., Naqshbandi, A., Fareed. M. and Mahmood, R. (2012). Oral administration of a nephrotoxic dose of potassium bromate, a food additive, alters renal redox and metabolic status and inhibits brush border membrane enzymes in rats. Food Chem., 134: 980-985.

Ahmad, M.K., Zubair, H. and Mahmood, R. (2013). DNA damage and DNA-protein cross-linking induced in rat intestine by the water disinfection by-product potassium bromate. Chemosphere. 91(8): 1221–1224.

Ahmed, M.K., and Mahmood, R. (2012). Oral administration of potassium bromate, a major water disinfection byproduct, induces oxidative stress and impairs the antioxidant power of rat blood. Chemosphere., 87(7):750-756.

Akimoto, K., Kitagawa, Y., Akamatsu, T., Hirose, N., Sugano, M., Shimizu, S. and Yamada, H. (1993). Protective effects of sesamin caused by alcohol or carbon tetra chloride in rodents. Ann Nutr Met., 37(4): 218–224.

Albro, P.W., Corbett, J.T. and Schroeder, J..L. (1986). Application of the thiobarbiturate assay to the measurement of lipid peroxidation products in microsomes. J. Biochem. Biophys. Methods.13:185-194.

Ali, W.S. and Al-Firdous, F.A. (2012). Effect of Consumption of Kiwi Fruit on Potassium Bromate Induced Oxidative Stress In Rats. Australian Journal of Basic and Applied Sciences, 6(3): 519-524

Alvarez, A.M. and Rodríguez, M.L. (2000). Lipids in pharmaceutical and cosmetic preparations. Grasas y Aceites., 51(1-2):74-96.

Ballet, F., (1997).Hepatotoxicity in drug development: Detection, significance and solutions. J. Hepatol., 26(suppl2): 26-36.

Bergmeyer, H.U., Schreiber, P. and Wahlefeld, A.W. (1978). Optimization of methods for aspartate and alanine aminotransferase. Clin. Chem., 24(1): 58-61.

Beutler, E., Duron, O. and Kelly, B.M. (1963). Improved method for the determination of blood glutathione. J. Lab. Clin. Med., 61:882-888.

Bhat, S.V., Nagasampagi. B.A. and Sivakumar. M. (2005). Chemistry of natural products, (1st ed.), India, Narosa.pp840

Boozer, C.N. and Herron, A.J. (2006). Simmondsin for weight loss in rats. Int. J. of Obe., 30(7):1143-1148.

Bouali, A., Bellirou, A., Boukhatem, N., Hamal, A. and Bouammali, B. (2008). Enzymatic detoxifi cation of jojoba meal and effect of the resulting meal on food intake in rats. Nat Prod Res., 22(7):638-47.

Bull, R.J. (1990). Water In: Marquard H, Schafer SG, McClennon RO, Welsh F, editors. Toxicology. Academic Press, San Diego; 1999. pp. 1041–1050.

Busson-Breysse, J., Farines, M. and Soulier, J. (1994). Jojoba wax: Its esters and some of its minor components. J. of the Am. Oil Chem. Society. 71 (9): 999–1002.

Caetano, A.C., da-Veiga, L.F., Capaldi, F.R., de- Alencar, S.M. and Bezerra, R.M. (2013). The antioxidant response of the liver of male swiss mice raised on a AIN 93 or commercial diet. BMC Physiol., 13: 3-9.

Chavali, S.R., Utsunomiya, T. and Forse, R.A. (2001). Increased survival after cecal ligation and puncture in mice consuming diets enriched with sesame seed oil. Crit. Care. Med., 29(1):140–143.

Chiagoziem, A.O. and Ebenezer, O.F. (2012). Comparative studies on the antioxidant and scavenging activities of *Garcinia kola* extract and

vitamin E: Modulatory effects on KBrO - induced oxidative stress in rats. J. Chem. Pharm. Res., 4(7): 3676-3683.

Chipman, J.K., Davies, J.E., Parsons, J.L., Nair, J., O'Neill, G. and Fawell, J.K. (1998). DNA oxidation by potassium bromate; a direct mechanism or linked to lipid peroxidation? Toxicology. 126(2):93–102.

Circu, M.L. and Aw, T.K. (2011). Redox biology of the intestine. Free Radic Res., 45: 1245–1266.

Cooney, R. V., Custer, L. J., Okinaka, L. and Frunk, A. A. (2001). Effects of dietary seeds on plasma tocopherol levels. Nutr. Cancer, 39(1): 66-71.

Deangelo, A., George, M., Kilburn, S., Moore, T. and Wolf, D. (1998). Carcinogenicity of potassium bromate administered in the drinking water to male B6C3F1 mice and F344/N rats. Toxicol Pathol., 26(5): 724-729.

Dimkpa, U., Ukoha, U.U., Anyabolu, E.A., Uchefuna, R.C., Anikeh, L.C., Oji, O.J., Besong, E.E. and Emenjo, O.A. (2013). Hepatotoxic effects of potassium bromate on adult wistar rats. J. of Biolo. Agricu. and Healthcare. 3(7)111-116.

Draper, H. and Hadley, M. (1990). Malondialdehyde determination as index of lipid peroxidation. Methods Enzymol., 186: 421-431.

Drury, R.A. and Wallington, E.A. (1980). Carlton's Histological Technique. 5th ed. New York: Oxford University Press; 1980. p. 235-241.

El-Laithy, H.M. and El-Shaboury, M.F. (2002).The Development of cotina lipogels and gel microemulsion for topical admin-istration of fluconazole. APS. Pharm. Sci.Tech., 3(4): 1-9.

Elleuch, M., Besbes, S., Roiseux, O., Blecker, C. and Attia, H. (2007). Quality characteristics of sesame seeds and by-products. Food Chem., 103(2): 641-650.

El-Mallah, M. H. and El-Shami, S. M. (2009). Investigation of liquid wax components of egyptian jojoba seeds. J. of Oleo Sci., 58 (10): 543-548.

El-Shamy, A.M., Shehata, A.H., Sanad, O.A. and El Halawany, A.M. (2001). Chemical and biological evaluation of jojoba oil. Bull.Fac. Pharm. Cairo Univ. 39(2): 65-82.

El-Sokkary, G.H. (2006). Melatonin protect against oxidative stress induced by the kidney carcinogen potassium bromate. Neuroendocrinol. 21(6): 461-468.

Erukainure, O.L., Oke, O.V., Owolabi, F.O., Kayode, F.O., Umanhonlen, E.E. and Aliyu, M. (2012). Chemical properties of Monodora myristica and its protective potentials against free radicals *in vitro*. Oxid. Antioxid. Med. Sci., 1(2): 127-132.

FAO/WHO. (2010). Fats and fatty acids in human nutrition. Report of an Expert Consultation; FAO/WHO: Geneva, Switzerland.

Farombi, E.O., Alabi, M.C. and Akuru, T.O. (2002). Kolaviron modulates cellular redox status and impairment of membrane protein activities induced by potassium bromate (KBrO3) in rats. Pharm. Res., 45:63-68.

Fossati, P., Prencipe, L. and Berti, G. (1980). Use of 3, 5 dichloro-2-hydroxyl benzene sulfonic acid/4- amylphenazone chromogenic system in direct enzymatic assay of uric acid in serum and urine. Clin. Chem., 26(2): 227-231.

Gao, Z.L. and Rao, E.C. (1993). Synthesis of formyl (acetyl) amino acid and dipeptide derivatives of amino ketones. Yao Xue Xue Bao., 28(10): 744–757.

Habashy, R.R., Abdel-Naim, A.B., Khalifa, A.E. and Al-Azizi, M.M. (2005). Anti-inflammatory effects of jojoba liquid wax in experimental models. Pharmacol Res., 51(2):95-105.

Habila, O. H., Taha, N. M. and Mandour, A. E. (2013). Biochemical effect of chromium element on lipid profile of broilers. AJVS., 39 (1): 74-81.

Haidari, F., Mohammadshahi, M., Zarei, M. and Gorji, Z. (2016). Effects of sesame butter (Ardeh) versus sesame oil on metabolic and oxidative stress markers in streptozotocin-induced diabetic rats. Iran. J. Med. Sci., 41(2): 102–109.

Halliwell, B. and Gutteridge, J.(1999). Free radicals in biology and medicine. 3rd Edn., Oxford University Press, Oxford.

Hampl, R., Drábková, P., Kanďár, R. and Stěpán J. (2012). Impact of oxidative stress on male infertility. Ceska Gynekol., 77(3):241–245.

Hanan, M. S.; Riad, E. M. and Zaki, E. R. (1998). Studies on jojoba oil and its effect as antibacterial and growth promoters. Egypt.J. Comp. Path. and Clinic. Path.,11(2):221-234.

Hemalatha, S., Raghunath, M. and Ghafoorunissa A. (2004). Dietary sesame (*Sesamum indicum* cultivar Linn) oil inhibits ironinduced oxidative stress in rats. Bri. J. Nutr., 92(4): 581–587.

Hissin, P. L. and Hiff, R. (1976). A fluorometric method for determination of oxidized and reduced glutathione in tissues. Anal. Biochem., 74 (1): 214-226.

Holser, R.A. and Abbott, T.P.(1999). Extraction of simmondsins from defatted jojoba meal using aqueous ethanol. Ind Crops Prod., 10:41-46.

Hou, R.C., Huang, H.M., Tzen, J.T. and Jeng, K.C. (2003). Protective effects of sesamin and sesamolin on hypoxic neuronal PC12 cells. J. Neurosci. Res., 74(1): 123–133.

Hsu, D.Z. and Liu, M,Y. (2004). Sesame oil protects against lipopolysaccharide-stimulated oxidative stress in rats. Crit. Care. Med., 32(1):227–231.

Hsu, D.Z., Chien, S.P., Li, Y.H., Chuang, Y.C., Chang, Y.C. and Liu, M,Y. (2008). Sesame oil attenuates hepatic lipid peroxidation by inhibiting nitric oxide and superoxide anion generation in septic rats. JPEN J Parenter Enteral Nutr., 32:154-159.

Husdan, H. and Rapoport, A. (1968). Estimation of creatinine by the jaffe reaction a comparison of three methods. Clin. Chem., 14(3): 222-238.

Jedlinska, M., Bomba, G., Jakubowski, K., Rotkiewicz, T. and Jana, B. and Penkowski, A. (2006). Impact of oxidative stress and supplementation with vitamins E and C on testes morphology in rats. J. Reprod., 52(2): 203-209

Jensen, J.E., Stainberg, S.E., Freese, P. and Marino, E. (2004). Liver function tests. J. Digest. Disord., 6: 1-3.

Johnson, W. (1992). Final report on the safety assessment of jojoba oil and jojoba wax. J. of the Am. Coll. of Toxico., 11(1):57-74.

Juan X., Chen S. and Qiuhui, H. (2005). Antioxidant activity of brown pigment and extracts from black sesame seed (*Sesamum indicum* L.). Food Chem., 91(1): 79-83.

Kakkor, P., Das, B. and Viswanathan. P. N. (1984). A modified spectrophotometric assay of superoxide dismutase. Ind. J. Biochem. Biophyso., 21: 130-132.

Kapil, A., Suri, O.P. and Koul, I.B. (1995). Antihepatotoxic effects of chorogenic acid from Anthrocephalus cadamba. Phytother. Res., 9(3): 189-193.

Karatzi, K., Stamatelopoulos, K., Lykka, M., Mantzouratou, P., Skalidi, S., Manios, E., Georgiopoulos, G., Zakopoulos, N., Papamichael, C. and Sidossis, L.S. (2012). Acute and long-term hemodynamic effects of sesame oil consumption in hypertensive men. J. Clin. Hypertens. (Greenwich). 14(9):630-6.

Khan, N. and Sultana, S. (2004). Abrogation of potassium bromate induced renal oxidative stress and tumor promotion response by soy isoflavones in wistar rats. Toxicology 201(1-3):173-84.

Khan, N., Sharma, S. and Sultana, S. (2003). *Nigella sativa* (black cumin) ameliorates potassium bromate-induced early events of carcinogenesis: Diminution of oxidative stress. Hum. Exp. Toxicol., 22(4): 193-203.

Khan, R.A., Khan, M.R. and Sahreen, S. (2013). Protective effects of Sonchusasper (L.) against KBrO -induced oxidative stress in rat testis. Pak J Pharm Sci., 26: 567–570.

Khan, R.A., Khan, M.R., Sahreen, S., Shah, N.A., Bokhari, J., Shabbir, M., Rashid, R. and Jan, S. (2011). Protective effects of Launaea procumbens against KBrO3-induced hepatic serum marker enzymes. African J. of Pharm. and Pharmac., 5(23): 2639-2641.

Kothavade, R.J., Joylekar, S.N. and Barodavalla, S.A. (1996). Protective effect of indigenous drug livomyn on Ketoconazole induced hepatotoxicity. Indian J. Pharm. Sci., 58(4): 142-146.

Krajcovicova-Kudlackova, M., Valachovicova, M. and Mislanova C. and Pribiojova, J. (2012). Antioxidative vitamins and oxidative lipid and DNA damage in relation to nutrition. Oxid. Antioxid. Med. Sci., 1(2): 147-151.

Kumar, C.K., Madem, T., Satheesh, k.D., Ramya M., Revathi, K., Kumar, G. and Reddy, A. (2012), A Review on Antioxidants. 2(2): 98-114.

Kurokawa, Y., Mackawa, A., Takahashi, N. and Hayeshi, Y. (1990). Toxicity and carcinogenicity of potassium bromate a new renal carcinogen. Environ. Health Perspect, 87: 309-355.

Labib, E.M., Zaki, M.A. and Mabrouk, H.A.(2012). Nutritional studies on partial and total replacement of fishmeal by jojoba meal

(*Simmondsia chinensis*) in Nile Tilapia (*Oreochromis niloticus*) Fingerlings Diets. APCBEE Procedia ;4:196-203.

Lee, S.Y., Son, D.J., Lee, Y.K., Lee, J.W., Lee, H.J, Yun, Y.W., Ha, T.Y., Hong, J.T. (2006). Inhibitory effect of sesaminol glucosides on lipopolysaccharide-induced Nf-Kb activation and target gene expression in cultured rat astrocytes. Neurosci. Res. 56(2): 204-212.

Li, A.N., Li, S., Zhang, Y.J., Xu, X.R., Chen, Y.M. and Li, H.B. (2014). Resources and biological activities of natural polyphenols. Nutrients. 6(12):6020–6047.

Lim, J.S., Adachi, Y., Takahashi, Y. and Ide, T. (2007). Comparative analysis of sesame lignans (sesamin and sesamolin) in affecting hepatic fatty acid metabolism in rats. Br J Nutr., 97(1):85-95.

Liu, C., Chien,S., Hsu,D., Periasamy, S. and Liu, M.(2015). Curative effect of sesame oil in a rat model of chronic kidney disease. Nephrology (Carlton). 20(12):922-30

Maharjan, B.R., Jha, J.C., Adhikari, D., Akila, S. R., Alurkar, V.M. and Singh, P.P. (2008). Oxidant stress antioxidant status and lipid profile in ischemic heart disease patients from western Nepal. Nepal Med J., 10:20–24.

Makni, M, Fetoui, H., Garoui, M., Gargouri, N.K., Jaber, H., Makni, J., Boudawara, T. and Zeghal, N. (2010). Hypolipidemic and hepatoprotective seeds mixture diet rich in omega-3 and omega-6 fatty acids. Food Chem Toxicol., 48(8-9):2239-2246.

Martin, P. and Friedman, L.S. (1992). Assessment of liver function and diagnostic studies. In: Hand Book of Liver Disease. Friedmann, L.s. and E.B. Keeffe (Eds.), Churchill Livingstone, Philadelphia, pp 1-14.

Medina, J. and Moreno-Otero, R. (2005). Pathophysiological basis for antioxidant therapy in chronic liver disease. Drugs. 65(17):2445–2461.

Mir-Kaisar, A., Samreen, A. and Riaz, M. (2011). Potassium bromate causes cell lysis and induces oxidative stress in human erythrocytes. Environ. Toxicol., 29(2):138-145.

Mohamed, M.A. Khalil, R. H. Saad, T.T. and Amira A.Z. (2015). Role of some herbs in controlling aflatoxicosis in cultured *Oreochromisniloticus*. World J.of Fish and Marine Sc., 7 (4): 325-333.

Moller, P. and Wallin, H. (1998). Adduct formation, mutagenesis and nucleotide excision repair of DNA damage produced by reactive oxygen species and lipid peroxidation product. Mutat. Res., 410(3):271–290.

Montgomery, H.A. and Dymock, J. F. (1961). Colorimetric determination of nitrite. Analyst. pp 86- 414.

Morris, J.B. (2002). Food, industrial, nutraceutical, and pharmaceutical uses of sesame genetic resources In: Trends in new crops and new uses. Janick, J. and Whipkey, A. (eds.), ASHS Press, Alexandria, VA., pp: 153–156.

Naqvi, H.H. and Ting, I.P. (1990). Jojopa a unique liquid wax producer from the american desert. in Advances in new crops. (Janick; Simon ed.). Timper 11. Press, Portland,OR. pp. 247-251

Nishikimi, M., Appaji, N. and Yagi, K. (1972). The occurrence of superoxide anion in the reaction of reduced phenazine methosulfate and molecular oxygen. Biochem. Biophys. Res. Commun.,46(2):849-854.

Ogawa, K., Watanabe, T., Ikeda, Y. and Kondo, S. (1997). A new glycoside, 1D-2-O-a-D-galactopyranosyl-chiro-inositol from jojoba beans. Carbohydr Res., 302:219-21.

Paglia, D.F. and Valentaine, W.N. (1979). Studies on glutathione and glutathione characterization of erythrocytes glutathione peroxidase. J. Lab.Clin.Med., 70(1):158-169.

Patton, C.J. and Crouch, S.R. (1977). Enzymatic colorimetric method for determination of urea in serum. Anal. Chem., 49: 464-465.

Periasamy, S., Liu, C.T., Hsu, D.Z. and Liu, M.Y. (2010). Sesame oil accelerates kidney healing following gentamicin-induced kidney injury in rats. Am. J. Nephrol., 32(5):383-92.

Ranzatoa, E., Martinotti, S., and Burlando, B. (2011). Wound healing properties of jojoba liquid wax: an *in vitro* study. J Ethnopharmacol.134(2):443-449.

Reeves, P. G., Nielson, F. H. and Fahmy, G.C. (1993). Reports of the American Institute of Nutrition, adhoc-wiling committee on reformulation of the AIN 93, Rodent diet. J Nutr., 123(11): 1939-1951.

Rice-Evans, C.A., Miller, N.J. and Paganga, G. (1996). Structure-antioxidant activity relationships of flavonoids and phenolic acids. Free Radic. Biol. Med., 20(7): 933- 956.

Roghani, M., Jalali-Nadoushan, M.R., Baluchnejadmojarad, T., Vaez Mahdavi, M.R., Naderi, G., Roghani, D.F. and Joghataei, M.T. (2013). Endothelium-dependent Effect of Sesame Seed Feeding on Vascular Reactivity of Streptozotocin-diabetic Rats: Underlying Mechanisms. Iran. J. Pharm. Res., 12(3):377-85.

Rogi, T., Tomimori, N., Ono, Y. and Kiso, Y. (2011). The mechanism underlying the synergetic hypocholesterolemic effect of sesamin and alpha-tocopherol in rats fed a high-cholesterol diet. J. Pharmacol. Sci., 115(3):408-416.

Roy, S.E. (1970). Colorimetric determination of serum alkaline phosphatase. Clin. Chem., 16: 431-432.

Sahreen, S., Khan, M.R. and Khan, R.A. (2011). Hepatoprotective effects of methanol extract of Carissa opaca leaves on CCl_4-induced damage in rat. BMC Compl. Alter. Med., 11: 48, 1-9.

Schiffman, S.S., Graham, B.G., Sattely-Miller, E.A. and Warwick, Z.S. (1998). Orosensory perception of dietary fat. Curr. Dir. Psychol. Sci., 7:137-143.

Sedigheb, A., Mahmoud, RafieianK., Somayeh, N., Esfandiar ,H. and Amirhossein, S. (2013). Antihyperlipidemic effects of sesamum indicum L. in rabbits fed a high-fat diet. The Scie.c Wor. J. Article ID 365892, pp. 1-5.

Seyedeh, M. N., Fatemeh, K., and Soheila, L. (2013).Olive and sesame oil effect on lipid profile in hypercholesterolemic patients, which better? Int. J. Prev. Med., 4(9): 1059–1062.

Shahwan, M.J. (2014). Antihyperlipidimic effect of simmondsia Chinensis sedds extracts in Rabbits. Int.J of App.Biolo. and Pharm.Tech., 5(1): 184-188.

Shalaby, M.A., El Zorba, H.Y. and Gehan, M.K. (2004). Effect of alpha-tocopherol and simvastatin on male fertility in hypercholesterolemic rats. Pharmacol. Res., 50(2): 137-142.

Shani, A. (1995). The struggles of jojoba. Chemthch. 25(5), 49-54.

Shelbaya, L.A. El Mehairy, H. F. and El-ZainyAntioxidant, A.R. (2014). Activities of Marjoram (*Origanum majoranum* L.) added to frozen

beef kofta and its therapeutic effect against kidney damage in rats. Wor. App.Sci. J., 31 (8): 1406-1414.

Siedel, J. (1993). AACC meeting abstract 34.Clin Chem., 39: 1127. Young, D. S. (1995). Effect of drugs on clinical tests, 4th ed. AACC Press, Washington, D.C.

Sinha, K. A. (1972). Colorimetric assay of catalase enzyme. Anal, Biochem., 47(2): 389-394.

Sirato-Yasumoto, S. M., Katsuta, Y., Okuyama, Y., Takahashi Y. and Ide T. (2001). Effect of sesame seeds rich in sesamin and sesamolin on fatty acid oxidation in rat liver. J. Agri. Food Chem., 49(5): 2647-2651.

Snedecor, G.W. and Cochran, W.G. (1981). Statistical methods. 7th. Ed. Iowa, USA: Iowa Uni. Press. Ames. p. 175-91.

Sobhy, H.M. Abo Elmagd, M.K., El-kholy, M.M., Abo Elmagd, E.K. and Laz, E.S. (2016). Hepatoprotective effect of jojoba oil and Nigella Sativa seeds in rats fed diet containing Aflatoxin. Egypt. J. Chem. Environ. Health, 2 (2):21-37.

Sobhy, H.M., Mansour, M.K., Zaki, A.A. and Elkholy, M.M. (2015). Hepatoprotective effect of jojoba oil on DNA damage and antioxidant enzymes induced by cadmium in rats. Egypt. J. Chem. Environ. Health, 1 (1):94-112.

Stasiak, M., Zasada, K., Lewinski, A. and Karbownik-Lewinska, M. (2010). Melatonin restores the basal level of lipid peroxidation in rat tissues exposed to potassium bromate in vitro. Neuro. Endocrinol. Lett., 31(3): 363–369.

Tietz, N. W. (1991). Clinical Chemistry, 2nd Edition, Saunders, Pp: 477-540.

Umemura, T., Takagi, A., Sai, K., Hasegawa, R. and Kurokawa, Y. (1998). Oxidative DNA damage and cell proliferation in kidneys of male and female rats during 13-weeks exposure to potassium bromat e (KBrO3). Arch. Toxicol., 72(5):264-269.

Unnikrishnan, M. K., Kumar, M. S., Satyamoorthy, K. and Joshi R. (2005). Free radical reactions and antioxidant activity of sesamol: Pulse radiolytic and biochemical studies. J, Agric. Food Chem., 53(7): 2696-2703.

Van Boven, M., Busson, R., Cokelaere, M., Flo, G. and Decuypere, E. (2000). 4-Demethyl simmondsin from Simmondsia chinensis. Ind Crops Prod., 12:203-208.

Venukumar, M.R. and Latha, M.S. (2004). Indian effect of Coscinium fenestatum on hepatotoxicity in rats. J. Exp. Biol. 42(8): 792-797.

Wahba, H.M. and Ibrahim, T.A. (2013). Protective effect of flaxseed oil and vitamin E on potassium Bromate-induced oxidative stress in male rats. Int. J. Curr. Microbiol. App.Sci., 2(9): 299-309.

Wichitsranoi, J., Weerapreeyakul, N., Boonsiri, P., Settasatian, C., Settasatian, N., Komanasin, N., Sirijaichingkul, S., Teerajetgul, Y., Rangkadilok, N. and Leelayuwat, N. (2011). Antihypertensive and antioxidant effects of dietary black sesame meal in pre-hypertensive humans. Nutr J., 10:82.

Wisniak, J. (1994). Potential uses of jojoba oil and meal : a review. Ind Crops Prod ;3:43-68.

Woodford, F.P. and Whitehead , T.P. (1998). Is measuring serum antioxidant capacity clinically useful? Ann. Clin. Biochem., 35:48–56.

Yamashita, K., Lizuka, Y., Imai, T. and Namiki, M, (1995). Sesame seed and its lignans produce marked enhancement of vitamin E activity in rats fed a low tocopherol diet. Lipids. 30(11): 1019–1028.

Young, D. S., 1995. Effects of Drugs on Clinical Laboratory Tests, 4th ed. Washington, DC: AACC Press, 500–506.

الآثار المحتملة للوقاية والتحسين من تأثير زيت السمسم وزيت الجوجوبا ضد الاجهاد التأكسدي المُحدث ببرومات البوتاسيوم في الجرذان

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الملخص العربي:

للأكسدة دوراً مهماً في إحداث الكثير من الأمراض المزمنة مثل تصلب الشرايين وارتفاع ضغط الدم وداء السكري والسرطان. ويؤدي تناول الأغذية الغنية بمضادات للأكسدة يمكن ان 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 بأستخدام الجرذان كحيوانات تجارب. وقد تم تقسيم خمسة وثلاثين جرذ عشوائيا علي خمس مجموعات وتحتوي كل مجموعة علي سبعة من الجرذان. واستخدمت المجموعة الاولي كمجموعة ضابطة سالبة (مجموعة طبيعية) وكانت تتناول الغذاء العادي. بينما المجموعات الاربعة الاخري تم حقنها داخل الغشاء البريتوني بجرعة واحدة (١٢٥ ملجم/كجم من وزن الجسم) من برومات البوتاسيوم لاحداث الاجهاد التأكسدي. واستخدمت المجموعة الثانية كمجموعة ضابطة موجبة تتغذي علي الغذاء العادي. بينما المجموعات ٣ و ٤ و ٥ تم تغذيتها على الغذائي العادي المضاف إليه ٥٪ زيت السمسم، وزيت الجوجوبا وخليطهما على التوالي. وأظهرت النتائج أن الجرذان المحقونة ببرومات البوتاسيوم بجانب تناولها الغذاء المضاف اليه زيت السمسم أو زيت الجوجوبا أو الخليط منهما لديها نقص واضح في مستويات االكولستيرول، الجلسريدات الثلاثية، الليبوبروتينات منخفضة الكثافة، البيليروبين الكلي، يوريا نيتروجين، حمض البوليك، والكرياتينين، مالوندالي الدهيد، نشاط إنزيمات اسبارتييز امينوترانسفيريز، الألانين امينوترانسفيريز والفوسفاتيز. بينما كان لديها زيادة كبيرة في كمية الغذاء المستهلك، وزيادة وزن الجسم، ومستويات الليبوبروتينات مرتفعة الكثافة، مضادات الأكسدة الكلية، الجلوتاثيون، ونشاط الانزيمات المضادة للأكسدة (الجلوتاثيون بيروكسيديز، السوبر اوكسيد ديسميوتيز والكتاليز). كما أظهرت النتائج وجود نقص معنوي في تركيز المالوندالي الدهيد وزيادة معنوية في تعزيز

الجلوتاثون ونشاط الانزيمات المضادة للأكسدة (الجلوتاثيون بيروكسيديز، السوبر اوكسيد ديسميوتيز والكتاليز) في خلايا الكبد وذلك كمقارن بما كانت موجودة في الجرذان المعالجة ببرومات البوتاسيوم فقط (المجموعة الضابطة الموجبة). كما كان هناك تحسن واضح في انسجة الكبد والكلى. وكانت أفضل النتائج لجميع المؤشرات البيوكيميائية والهستوباثولوجية لانسجة الكبد والكلى والتي كانت تميل نحو النتائج الطبيعية في الجرذان المعالجة بخليط زيت السمسم مع الجوجوبا. وفي النهاية، اقترحت نتائج الدراسة الحالية أن التناول المنتظم لزيت السمسم اوالجوجوبا فائدة في تحسين وظائف الكبد والكلى والحماية ضد الإجهاد التأكسدي المٌحدث ببرومات البوتاسيوم في الجرذان وذلك من خلال إظهار النشاط المضاد للأكسدة كما وجد أن الخليط من زيت السمسم والجوجوبا يعطي أفضل النتائج.