Hepatoprotective effect of jojoba oil against lead-induced toxicity in rats

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Background
Previous investigations showed that jojoba oil exhibited a protective effect against hepatotoxicity caused by different toxicants, however, to the best of our knowledge, no prior research has been done to determine the effectiveness of jojoba oil in protecting against lead toxicity.
Objective
This study assessed the hepatoprotective properties of jojoba oil against lead toxicity in rats.
Materials and methods
The study included four groups, each consisting of six Sprague Dawley male rats, and orally administered jojoba oil (JO group), lead acetate (LA group), and lead acetate plus jojoba oil (protective group)
Results and conclusion
The results showed that lead acetate-induced hepatotoxic effects were revealed by increased serum alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase activities with hepatic histomorphological changes. There was a significant increase in serum total cholesterol, triglycerides, and low-density lipoprotein-cholesterol, while levels of high-density lipoprotein-cholesterol significantly declined compared to normal rats. Additionally, lead acetate triggered oxidative damage of hepatocytes, evidenced by a significant increase of malondialdehyde levels and a decrease of reduced glutathione levels and activities of superoxide dismutase and glutathione S-transferase. Administration of lead was associated with a change in the distribution of cells over different cell cycle phases, characterized by a marked increase in the sub G1 cell population and a significant decrease in the G0/G1 cell population. Supplementation of jojoba oil with lead acetate relieved the toxic impacts of lead acetate with an enhancement of the liver enzyme activities, antioxidant status, lipid profile parameters, and histopathological alterations. In conclusion, jojoba oil might be an effective natural product that offers a promising preventive action towards lead-induced liver damage in rats.

Keywords:
hepatoprotective, jojoba oil, lead toxicity, oxidative stress

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Introduction

Throughout their lifespan, humans are subjected to a variety of environmental pollutants in an increasing manner. The majority of these exposures are detrimental [1]. Many cellular organelles and many enzymes required for detoxification, metabolism, and damage recovery are disrupted by heavy metals [2]. Lead is an environmental pollutant and one of the most poisonous heavy metals. Great attention has been directed toward health hazards related to lead toxicity. The majority of body organs are affected by lead poisoning, these involve the digestive, neurological, and circulatory systems, the blood, besides the liver, kidney, and bone [3].

The primary routes of lead exposure are inhalation of Pb-contaminated aerosols or dust and utilization of nutrients and paints contaminated with lead [2]. Lead that has been ingested is assembled in the liver, and a little portion is excreted in the urine. The remainder is collected within other body portions, with the liver being the biggest store of Pb in soft tissues, followed by the kidney [3,4].

One of the key processes of lead toxicity is oxidative damage, as lead exposure results in an imbalance of the redox potential [5]. Lead exposure results in the generation of reactive oxygen species (ROS), which cause nephrotoxicity, hepatotoxicity, and behavioral and reproductive problems. These issues may persist even after Pb levels have decreased [3,6].

Due to the connection between lead toxicity and oxidative destruction in numerous human organs, active dietary phytochemicals were consequently used to improve the antioxidant system of cells and protect them from damage caused by lead [5,7]. Additionally, the development of treatment methods that strengthen the antioxidant protection of the body could stop liver toxicity induced by lead [8].

Today, natural plant oils are increasingly utilized in medicine all around the world. They often have fewer side effects, are efficient, and are readily available at low prices. Numerous oils contain components that have anti-inflammatory and antioxidant characteristics, making them useful for therapeutic purposes [2,9].

A promising oil seed plant in the Simmondsiaceae family is jojoba (Simmondsia chinensis) [10]. It is indigenous to the deserts of North and Central America, but its cultivation is also entrenched in other countries, involving Chile, Egypt, and Argentina [11,12]. Jojoba oil is easily extracted in large amounts from plant sources. It nearly constitutes 50% of the entire seed weight [13]. This oil has a unique composition because it is primarily made up of lengthy monounsaturated esters, whereas other oils are primarily triglycerides. As a result, jojoba oil has distinctive features and characteristics that are crucial for industrial chemistry and pharmaceutical purposes [9,14].

Several studies showed the protective outcomes of jojoba oil towards hepatotoxicity caused by different toxicants [9], however, up to the best of our knowledge, no prior research was carried out to examine the effectiveness of jojoba oil in protecting against lead toxicity. Thus, this work aimed to study the possible jojoba oil alleviating properties towards lead hepatotoxicity in rats.

Materials and methods

Chemicals

Jojoba oil was brought from Egyptian Natural Oil Co., Cairo, Egypt. Test kits supplied by Biodiagnostic and Spectrum Co., Cairo, Egypt, were employed for biochemical analysis. Lead acetate ($Pb(CH_3CO_2)_2 .3H_2O$) was of analytical grade (Merck, Germany). Chemicals of analytical grade were used in biochemical analysis.

Animals

Adult Sprague Dawley male rats, weighting 170–180 g, were provided from the Animal Breeding House, National Research Center, Dokki, Cairo, Egypt. The research was approved by Mansoura University Animal Care and Use Committee, MU-ACUC (SC. R.23.01.5). The ethical standards of experiments are in accordance with the guidelines provided by the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA) and World Medical Association Declaration of Helsinki. Animals were retained in clean stainless steel cages and supplied with libitum feeding and water. They were kept for two weeks to acclimatize before performing the practical work under controlled conditions (50% humidity, 23±2 °C temperature, and 12 h light/ dark period).

Experimental design

The study included four groups of male adult Sprague Dawley rats, each with six animals. Rats received these treatments for consecutive 7 days as follows: The 1st group was provided with standard food and water and represented the normal control group. The 2nd group was the lead acetate (LA) group, which was given a single dose of lead acetate ($Pb(CH_3CO_2)_2 \cdot 3H_2O$) by oral gavage (150 mg Pb/kg bw) [15]. The 3rd group was the jojoba oil group (JO group), which received jojoba oil by oral gavage (2 ml/Kg) for 7 days [16]. The 4th group was the protective group (JO+LA group), which received jojoba oil seven days by oral gavage (2 ml/Kg), followed by a single dose of lead acetate by oral gavage (150 mg Pb/kg bw).

After finishing the experiment, the sacrification of rats was done under light anesthesia. Serum was separated from blood samples by centrifugation at 2500 rpm for 5 min. Biochemical parameters, including liver enzymes and lipid profile parameters were evaluated in the serum samples.

Liver samples from different groups were dissected and rinsed in cold saline. The liver was split into 2 parts; one part was stored at −80°C for the estimation of oxidant/antioxidant biomarkers and flow cytometry analysis, while the other part was preserved for histopathological analysis in formalin solution (10% neutral buffered).

Liver function tests

Test kits from Biodiagnostic, Egypt, were utilized for the estimation of liver function. Assays included measuring serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) according to the kit's instructions.

Lipid profile parameters

Assay kits from Spectrum, Egypt, were used for the assessment of lipid profile parameters. Estimations included measuring total cholesterol (TC), HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), and triglycerides (TG), according to the kit's instructions.

Preparation of liver homogenates

Liver tissue homogenates were prepared from each group. Tissue samples were washed in ice-cold saline buffer composed of 20 mM Tris–HCl and 0.14 M NaCl (pH 7.4). Tissue homogenization was carried out using ice-cold phosphate-buffered saline (pH 7.4). The supernatants were collected after centrifugation of the cellular suspension and kept at −20°C.

Oxidant/antioxidant parameters

Kits purchased from Biodiagnostic, Egypt, were used for the determination of liver homogenate malondialdehyde (MDA), reduced glutathione (GSH), glutathione S-transferase (GST), and superoxide dismutase (SOD), as described by the manufacturers.

Flow cytometry analysis

Absolute ice-cold alcohol was used for the fixation of liver tissues and the tissues were stored at +4°C for further analysis. After a minimum of 12 h of fixation, the samples were centrifuged, and excess alcohol was eliminated. Propidium iodide staining was performed in accordance with Vindelov's 1977 instructions [17]. Within one hour after adding propidium iodide, the samples were processed through a FACS caliber flow cytometer (Becton Dickinson, Sunnyvale, California, USA). Analysis of DNA data was done by a MODFIT program (verify software house, Topsham, ME 04086 USA).

Histopathology

The liver tissues preserved in formalin solution were used for histopathological analysis. Tissues were trimmed, washed, dried out in ascending ethanol levels, and cleared in methyl benzoate. The standard paraffin embedding method was used for tissue processing. The paraffin blocks were used for the preparation of 3–5-μm thick sections, and then the sections were stained with hematoxylin and eosin routinely. Light microscopy was utilized for the examination of stained slides. A digital Leica photomicroscope was used for the development the histopathological photos.

Statistical analyses

The Statistical Package for Social Sciences, version 23 (SPSS Software, SPSS Inc., Chicago, USA) was used for data analysis. Results were presented as mean ±standard deviation (SD). For data with a Gaussian distribution and homogeneity of differences, statistical analysis was conducted using analysis of variance (One Way ANOVA), followed by a Tukey's multitest. Data with a Gaussian distribution and heterogeneity of differences were analyzed using analysis of variance (One Way ANOVA), followed by Tamhane's T2 test. The Kruskal-Wallis test and Mann-Whitney U-test were used to examine multiple comparisons of parameters with a non-Gaussian distribution. The significance threshold was established at P 0.05.

Results

Activities of serum ALT, AST, and ALP were statistically different among the studied groups ($P<0.05$). The LA group had the highest activities of liver enzymes. Regarding the jojoba group, no significant differences were observed in the levels of ALT, AST, and ALP compared to controls ($P>0.05$). The protective group recorded a significant decline compared to the LA group, which almost returned normally as the control group (Table 1, Fig. 1).

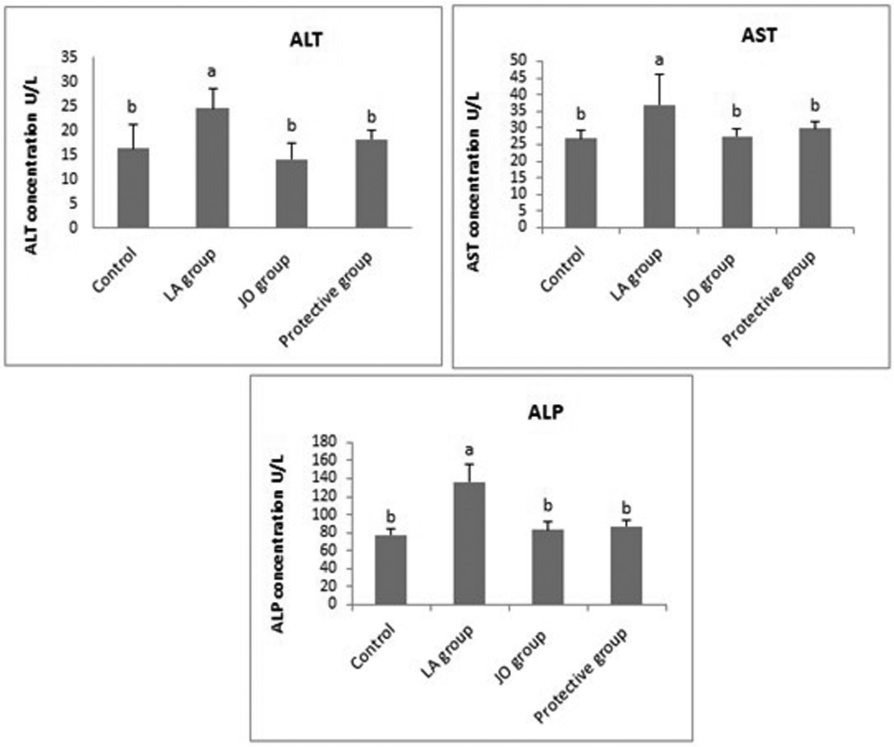
Lead administration resulted in a significant increase ($P<0.05$) in TG, TC, and LDL-C levels in

Table 1 Biochemical parameters of liver function and lipid profile in the studied groups

Groups Parameter	Control group (negative control)	Lead acetate group (LA) (positive control)	Jojoba oil group (JO)	Protective group (LA+JO)
ALT(U/ml)	16.30±4.81	24.49±4.16[a]	14.19±3.34[b]	18.24±1.82[b]
AST(U/ml)	26.70±2.48	36.95±8.94[a]	27.44±2.15[b]	29.89±1.99[b]
ALP(IU/l)	77.18±7.61	136.21±19.69[a]	83.35±9.31[b]	87.00±6.31[b]
Triglycerides (mg/dl)	120.97±21.14	197.05±66.48[a]	117.58±24.04[b]	114.57±34.63[b]
Total cholesterol (mg/dl)	79.83±10.71	109.72±18.02[a]	87.79±11.18[b]	88.04±6.41[b]
HDL-C (mg/dl)	55.04±6.81	33.49±4.70[a]	62.06±6.04a[b]	48.97±6.44[b]
LDL-C (mg/dl)	26.39±6.26	54.58±10.98[a]	24.54±7.14[b]	29.58±4.85[b]

*Values are presented as mean±SD (n=6). [a]Significance at $P<0.05$ compared to controls. [b]Significance at $P<0.05$ compared to the LA group. †ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, ALP: Alkaline phosphatase, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol.

Figure 1



Liver function tests in the studied groups.

comparison to normal rats. Levels of HDL-C were significantly decreased in the LA group compared to controls. In the protective group, levels of TG, TC, and LDL were significantly decreased compared to the LA group. However, HDL-C levels significantly increased in the protective group after jojoba protection. No significant differences were observed in the levels of TG, TC, and LDL-C in the JO group and controls. JO group had significantly higher levels of HDL-C compared to controls (Table 1, Fig. 2).

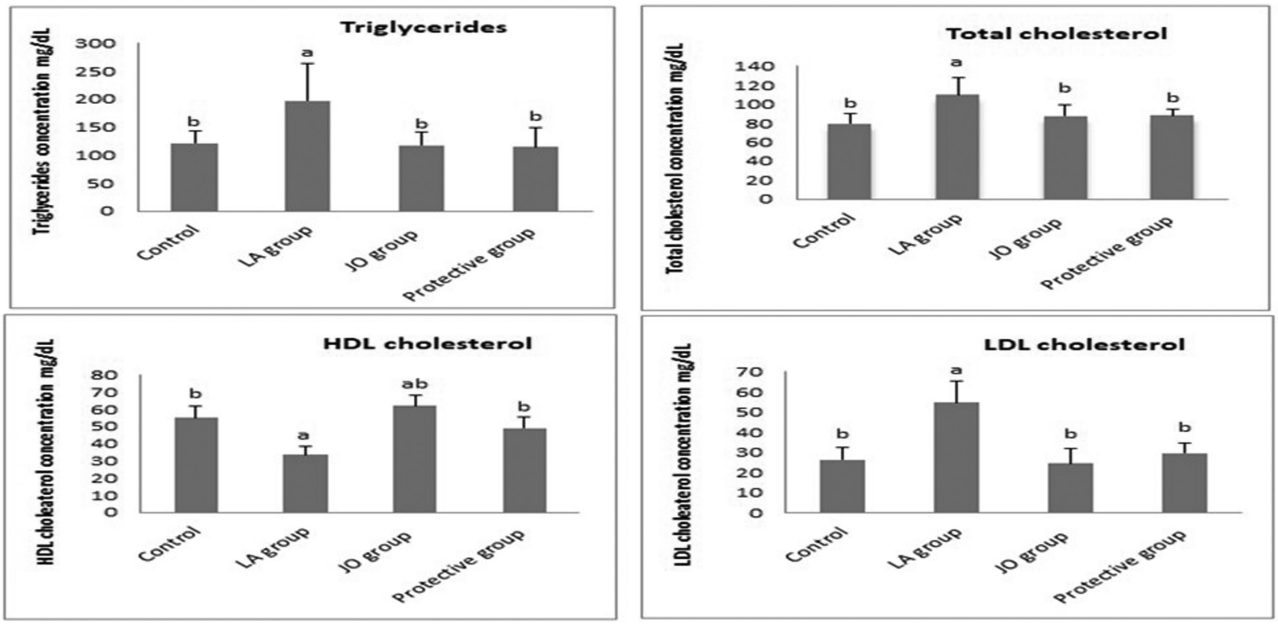
Lead administration caused a significant ($P \leq 0.05$) elevation of liver MDA levels compared to controls (Table 2, Fig. 3). In addition, lead administration resulted in a significant decrease in the concentration of GSH and the activities of SOD and GST. Therefore, these findings showed that lead has a negative effect on the liver by increasing the generation of free radicals and the extent of peroxidation and in the meantime decreasing the levels of antioxidant protective enzymes.

There were no significant differences in the levels of MDA and antioxidant enzymes in the jojoba group and normal controls. Although the levels of all oxidant/antioxidant parameters were significantly different from controls, the protective group showed significant recovery of all oxidant/antioxidant parameters ($P \leq 0.05$) (Table 2, Fig. 3). This indicates that the administration of jojoba oil has no negative effect on the liver and that its administration with lead in rats can beneficially conserve the protective enzymatic balance.

Table 3 shows the proliferation of hepatocytes and changes in the cell cycle phases in the studied groups, there were no changes in the number of hepatocytes in different cell cycle phases of control, jojoba, or protective groups, with the highest number of cells in G0/G1 phase. On the other hand, the lead acetate group showed a significantly lower number of hepatocytes in G0/G1 phase compared to other studied groups (Fig. 4).

The histopathological results of the normal controls and the jojoba group showed that the hepatic cords were normally and radially arranged around the central veins (CV), it also showed normal portal tracts and sinusoids. In the specimens examined after lead acetate administration, the liver showed marked congestion in portal blood vessels, mixed micro-and macro-vesicular steatosis in hepatocytes, and biliary epithelium hyperplasia with periductal sections. Examination of liver sections of the protective group revealed some

Figure 2



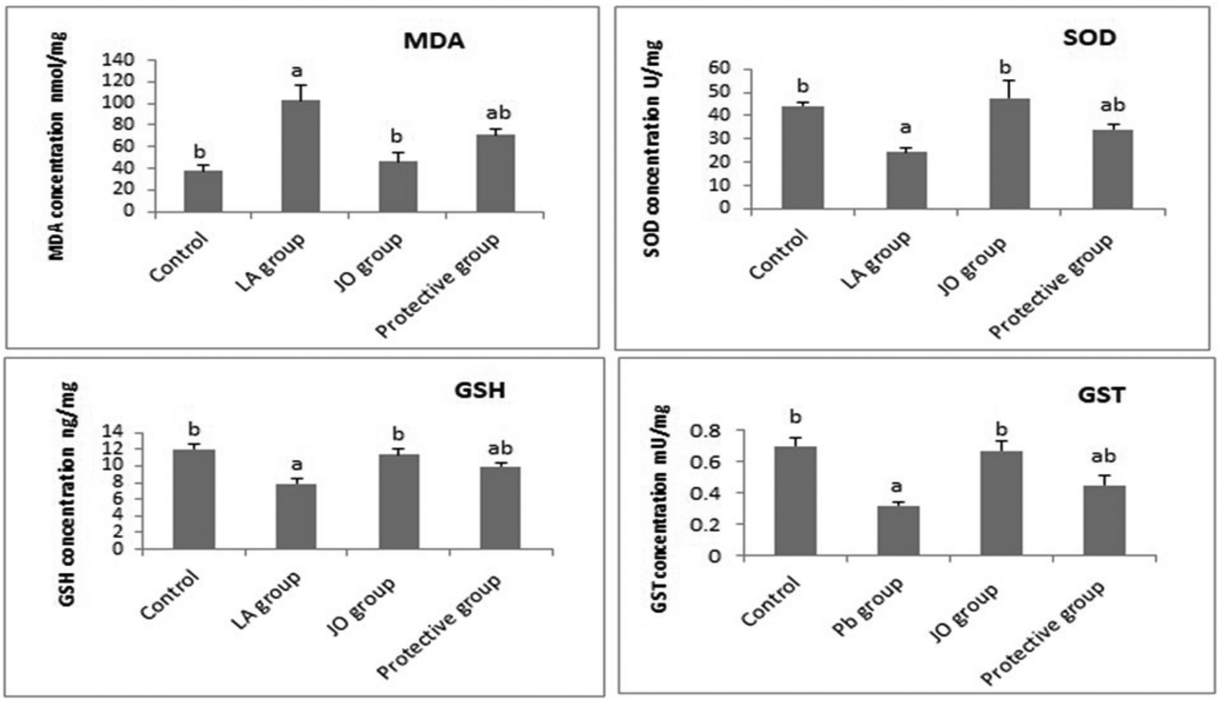
Lipid profile parameters in the studied groups.

Table 2 Oxidant/antioxidant biomarkers in the studied groups

Groups Parameter	Control group (negative control)	Lead acetate group (LA) (positive control)	Jojoba oil group (JO)	Protective group (LA+JO)
MDA (nmol/mg)	37.48±6.00	102.82±14.44[a]	46.05±8.73[b]	70.56±6.12[ab]
SOD (U/mg)	44.24±1.32	24.39±2.11[a]	47.40±7.82[b]	34.11±2.01[ab]
GSH (ng/mg)	11.97±.64	7.87±.65[a]	11.30±.75[b]	9.98±.47[ab]
GST (Mu/mg)	0.70±0.05	0.32±0.02[a]	0.67±0.06[b]	0.45±0.06[ab]

*Values are presented as mean±SD (n=6). [a]Significance at $P<0.05$ compared to controls. [b]Significance at $P<0.05$ compared to the LA group. †MDA: Malondialdehyde, SOD: superoxide dismutase, GSH: Reduced glutathione, GST: Glutathione-S-transferase.

Figure 3



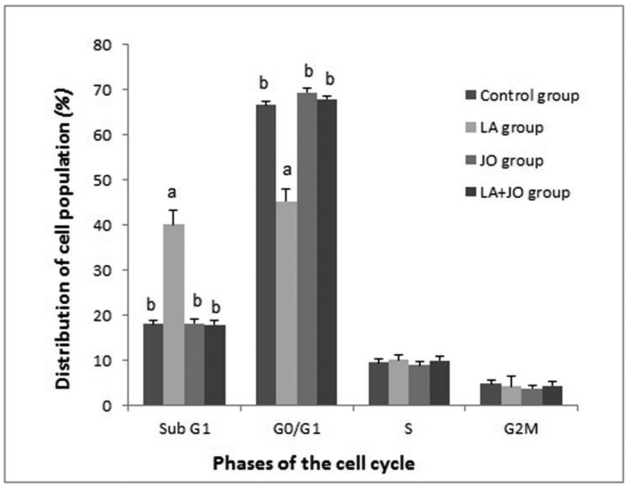
Oxidant/antioxidant biomarkers in different groups.

Table 3 Liver cell cycle profile in the studied groups

Groups Parameter	Control group (negative control)	Lead acetate group (LA) (positive control)	Jojoba oil group (JO)	Protective group (LA+JO)
Sub G1 (%)	18.03±0.75	40.15± 0.81[a]	18.20±0.89[b]	17.90±0.91[b]
G0/G1 (%)	66.56±3.04	45.20±2.84[a]	69.30±1.09[b]	67.85±2.13[b]
S (%)	9.50±0.89	10.04±0.97	8.97±0.85	9.88±0.76
G2M (%)	4.80±0.92	4.25±0.82	3.8±0.89	4.39±0.94

*Values are presented as mean±SD (n=6). [a]Significance at $P<0.05$ compared to controls. [b]Significance at $P<0.05$ compared to the LA group.

Figure 4



Distribution of cell population in different phases of the cell cycle in different groups.

healthy appearance, as the liver tissues showed mild congestion with normal hepatic cords, bile ducts, and sinusoids (Fig. 5).

Discussion

This study assessed the toxic outcomes of lead and the potential protective properties of jojoba oil administration on rat hepatic tissues. Levels of serum AST, ALT, and ALP were evaluated to assess the therapeutic impact of jojoba on liver damage brought on by lead acetate.

Our results demonstrated that rats exposed to lead showed significant alterations in liver function tests as showed by the significant increase in the levels of ALT, AST, and ALP in the LA group. These biochemical markers are important indicators for hepatocellular injury where changes in cell permeability cause the release of these enzymes from hepatocytes into the circulation [8]. According to several experimental researches, these parameters were significantly increased after Pb exposure and this increase was accompanied by histological changes in the structure of the liver [18–20].

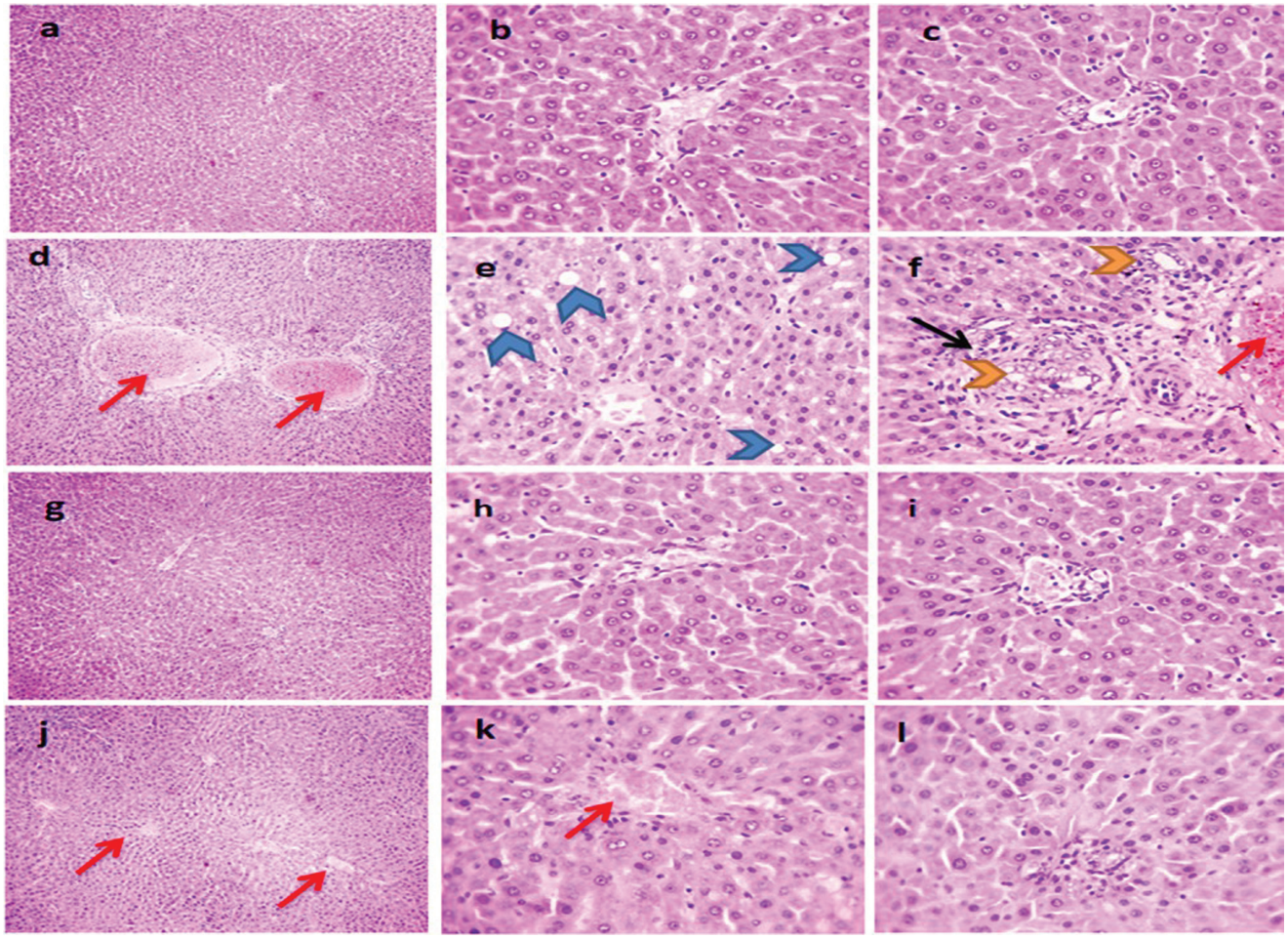
Administration of jojoba oil alone in experimental rats caused no significant changes in the levels of these biochemical parameters compared to controls. This shows that the jojoba oil administration has no damaging impact on the liver. Intake of jojoba oil with lead acetate significantly lowered serum levels of these parameters compared to the LA group. This shows that jojoba could reduce liver damage caused by lead and preserve the integrity of hepatocytes.

In consistence with the alterations in the levels of liver enzymes, the histopathological examination of the hepatic tissue of lead-treated rats indicated marked congestion in portal blood vessels, mixed micro-and macro-vesicular steatosis in hepatocytes, biliary epithelium hyperplasia with periductal sections. Similar results were previously reported by other studies [21,22].

Histological results of the jojoba-treated group showed hepatic cords normally and radially arranged around central veins (CV), normal portal tracts, and normal sinusoids similar to that of the control group, this confirms the non-harmful effect of jojoba oil treatment on liver cells. Examination of liver sections after administration of jojoba oil with lead in the protective group showed some healthy appearance, as the liver tissues displayed mild congestion with normal hepatic cords, bile ducts, and sinusoids. This confirms the ability of jojoba oil to sustain the integrity of hepatocytes and protect against lead-induced hepatotoxicity.

The obtained data revealed that lead administration caused significant elevations in the levels of triglycerides, TC, and LDL-C, while the levels of HDL-C were significantly decreased in the LA group. These findings suggest that the metabolism of lipids and carbohydrates was severely disrupted by Pb exposure. Similar observations were reported by other studies [23–25]. As a result of oxidative stress, oxidized LDL (O-LDL) and oxidized cholesterol are produced, which promotes the accumulation of cholesterol in vascular tissues [26].

Figure 5



Microscopic images of H&E stained liver slices showing hepatic cords normally and radially arranged around central veins (CV), normal portal tracts and normal sinusoids in controls (a,b,c) and in JO group (g,h,i). In contrast, marked congestion in portal blood vessels (red arrows), mixed micro-and macro-vesicular steatosis in hepatocytes (blue arrowheads), biliary epithelium hyperplasia (orange arrowhead) with periductal fibrosis (black arrow) (d,e,f) in LA group. Mild congestion (red arrows) is seen in protective group with normal hepatic cords, bile ducts and sinusoids (j,k,l).

When jojoba oil was administered alone, the levels of HDL-C significantly increased in comparison to controls. Also, its administration caused no changes in the levels of TC, TG, and LDL-C. In the protective group, the administration of jojoba with lead acetate caused a significant decrease in the levels of serum TC, TG, and LDL-C. However, HDL-C levels were significantly increased in comparison with normal rats.

Regarding the impact of jojoba oil, our results are in line with the results of Shahwan (2014) who demonstrated that in rabbits on a high-cholesterol diet, jojoba oil dramatically raised HDL-C levels and lowered serum levels of TG and TC [27].

The natural antioxidants in jojoba oil, which is hypothesized to be an allylic derivative of hydroxytoluene, omega-3 fatty acids, and monounsaturated fatty acids may be responsible for the oil's hypolipidemic action. Moreover, numerous studies have demonstrated that monounsaturated fatty acid-rich oils can lower TG, TC, and LDL-C levels [28]. Also, monounsaturated fatty acids may decrease LDL-C and increase HDL-C levels [26,29].

To elucidate the molecular processes behind lead-induced liver damage and the mitigating effects of jojoba oil on this toxicity, the oxidant/antioxidant profile was examined in liver tissues of rats, and levels of MDA, GSH, SOD, and GPx were employed to identify oxidative damage in hepatocytes. It has been proposed that the main mechanism of Pb-mediated tissue injury is Pb-induced oxidative stress or alteration of the oxidant/antioxidant equilibrium in the body tissues and blood [30–32].

The present study indicated that lead encouraged oxidative stress as shown by the increase of MDA levels and the significant e of SOD, GSH-Px, and GSH levels. These findings are in line with that of Omobowale *et al.*, 2014 [33,34]. Under physiological circumstances, a balance exists between oxidants and antioxidant levels. However, even minor alterations in the quantities of oxidants or/and antioxidants can disrupt the balance causing oxidative stress [35].

According to liver pathological analysis and serum biochemical indicators, lead accumulates in the liver and causes liver damage. This is likely due to the fact that lead increases the attack of target organs by ROS and results in a significant decline of the antioxidant enzymes involving SOD, CAT, GSH-Px, and GSH [8].

Our results showed that no significant differences were observed in the levels of MDA and antioxidant enzymes in the jojoba group and the control group. Administration of jojoba with lead acetate resulted in significant recovery of all oxidant/antioxidant biomarkers. This shows that the jojoba oil administration has no damaging impact on the liver tissues and that administering jojoba oil together with lead can successfully maintain the protective enzymatic balance [9].

Some elements have been observed to reduce lead-induced toxicities, these include some chelating factors and some antioxidants, such as vitamins C, and E, green tea, lipoic acid, pectin, N-acetylcysteine, homocysteine, and methionine. This could support the idea that lead can induce reactive oxygen species. Also, animal experiments showed that both plant extracts and components of animal origin might protect against the toxicity caused by lead. The antioxidant properties of the components included in these extracts were thought to be responsible for their ability to alleviate these toxicities [36,37].

Cell development and differentiation are governed by the cell cycle, which is a strictly regulated process [38]. A quick, precise, and quantitative examination of the DNA ploidy, proliferative activity, and distribution of cells in the various cell cycle phases can be done using the semi-quantitative technique of DNA flow cytometry [16].

Using flow cytometry, we examined the cell cycle profile of hepatocytes in the studied groups to determine the impact of lead administration and the hepatoprotective benefits of jojoba oil on cell cycle behavior. Administration of lead acetate was associated with a change in the distribution of cells over different cell cycle phases. Lead acetate significantly increased sub G1 cell population from 18.03% in the control group to 40.15% in the lead acetate group, G0/G1 cell population decreased from 66.56% in the control group to 45.20% in the lead acetate group.

Administration of jojoba oil alone resulted in no significant alterations of cell cycle distribution compared to controls. Also, the protective group showed a similar cell cycle profile as the control group with no significant alterations in cell cycle distribution.

In this work, jojoba oil mitigated the negative outcomes of lead in rats as shown by enhanced liver enzymes and decreased lipid peroxidation as shown by liver MDA content. Additionally, jojoba oil improved cellular GSH levels, GSH-PX, and SOD activities, which improved the antioxidant status. Additionally, it reduced the liver's histomorphological changes brought on by lead exposure.

Previous studies support jojoba oil's ability to defend against the toxic effects brought on by different toxicants. Based on jojoba oil's antioxidant and antiapoptotic effects, Abouzeid *et al.*, 2021 demonstrated that jojoba oil offered considerable protection against the liver and nervous system damage brought on by fipronil [9].

The harmful effects of diethyl nitrosamine in rats were reduced by jojoba oil as shown by a decrease in the activity of serum transaminases, the amount of MDA in the liver, and histological changes [16]. Also, the hepatoprotective effect of jojoba oil has been supported against different toxicants, including aflatoxins, cadmium, and CCl4 [39–41]. Moreover, a similar hepatoprotective effect was shown by ethanolic extract of jojoba in rats intoxicated with fumonisin [42–44].

More importantly, jojoba oil was nontoxic and did not alter the tested biochemical and hematological markers or the normal histology of the hepatocytes. In this regard, recent studies demonstrated that no toxic effects on body metabolism or blood markers were caused by jojoba extract [45,46].

Nitric oxide and 2,2-Diphenyl-1-picrylhydrazyl (DPPH) scavenging assays have been used to

demonstrate the antioxidant properties of jojoba oil [47,48]. The high antioxidant constituent of jojoba oil, which allows it to protect against ROS and mitigate the resulting oxidative injury, may be responsible for its protective effect. According to Kara (2018), the high total phenolic content of jojoba is what gives the extract its antioxidant properties [49]. Owing to its high phenolic components and flavonoid content, jojoba oil has been proven to have good antioxidant capabilities compared to ascorbic acid [50,51].

Additionally, compounds such as gallic acid, catechin, quercetin, quercetin 3-glucoside, and caffeic acid may contribute to the antioxidant effects of jojoba seed extract. It was shown that jojoba oil contains a hydroxytoluene allylic derivative which is a natural antioxidant [52]. Depending on the oil's source, jojoba oil contains varying amounts of tocopherol isomers, with the γ- isomer being the most common. Jojoba oil also functions as an antioxidant and aids in storing and using vitamins A and E in the liver [26].

Jojoba plant free radical protection has also been attributed to its content of lipoxygenase inhibitors, which reduce the production of leukotriene and, as a result, inflammation [9,53,54]. A further mechanism underpinning jojoba pharmacological actions is provided by jojoba seed extract jojobenoic acid which has antioxidant properties as well as the capacity to bind metal ions [26,55].

Conclusion

In conclusion, the current work shows that jojoba oil can be taken into consideration as a possible protective factor against lead-induced hepatotoxicity. Based on the promising results of the hepatoprotective properties of jojoba oil against lead toxicity in rats, this study indicates that jojoba oil might be a helpful protective agent for countering lead toxicity.

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The manuscript has been read and approved by all the authors, each author believes that the manuscript represents honest work and the information is not provided in another form.

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Nil.

Conflicts of interest

The authors declare there are no conflicts of interest.

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