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POTENTIAL PROTECTIVE AND THERAPEUTIC EFFECTS OF JOJOBA OIL AGAINST CHLORPYRIFOS-INDUCED HEPATOTOXICITY IN RATS

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ABSTRACT

Chlorpyrifos (CPF) is an extensively used and effective broad-spectrum organophosphorus insecticide. CPF exposure elicits liver injury mediated via oxidative stress; it disrupts the oxidant/antioxidant detoxification system, which consequently causes hepatotoxicity. Jojoba is widely used as a medicinal plant cultivated worldwide. Jojoba oil, extracted from Jojoba seeds, contains a high percentage of phenols and flavonoids, which make it a powerful antioxidant and enable it to get rid of free radicals. Therefore, the current investigation aimed to evaluate the potential protective and therapeutic effects of Jojoba oil against the oxidative effect of chlorpyrifos-induced hepatotoxicity in rats. Thirty adult male albino rats were divided into five groups of six animals in each group. Control group, Jojoba group, CPF-administration group, Jojoba prophylactic group, and Jojoba therapeutic group. Blood samples and hepatic tissues were obtained from each rat for biochemical and histopathological examinations. Results indicated that CPF-induced hepatotoxic effects were evidenced by elevated serum alkaline phosphatase and γ-glutamyltransferase, with histopathological alterations in hepatic tissue. Additionally, our results showed involvement of oxidative stress in liver injury after intoxication with CPF manifested by increased malondialdehyde level and decreased superoxide dismutase and catalase activities in hepatic tissues. Conversely, these results were reversed by Jojoba oil administration. Collectively, our data indicated that Jojoba oil can alleviate CPF-induced oxidative stress in hepatic tissues and reduce histopathological alterations. In conclusion, Jojoba oil could provide significant protective and therapeutic effects against CPF-induced hepatotoxicity, making it a potentially advantageous natural protective and therapeutic agent.

Keyword: *Chlorpyrifos, Jojoba oil, oxidative stress, hepatotoxicity.*

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INTRODUCTION

Pesticides are widely employed in agricultural and public health initiatives, which has led to serious environmental contamination and possible health risks, such as acute and chronic human poisoning (Abubakar *et al.,* 2020). One of the most effective kinds of pesticides used for both agricultural and landscape pest management is organophosphorus insecticides (OP), accounting for about 50% of the global insecticide use (Ambali *et al.,* 2018). It is estimated that the fatality rate among OPs poisoned people reaches 10% every year. Human exposure can occur through several pathways, including inhalation, direct contact, or consuming food contaminated with OPs (Sidhu *et al.,* 2019).

Chlorpyrifos (CPF), one of the most widely used OPs, can persist in food in significant quantities, with the greatest rate reaching 40% (Wołejko *et al.,* 2022). CPF exposure poses severe human health concerns, including hepatotoxicity, neurotoxicity, reproductive dysfunction, developmental abnormalities, cardiotoxicity, and nephron-toxicity (Albasher *et al.,* 2019, Biosca-Brull *et al.,* 2023, Fu *et al.,* 2024, Maggio *et al.,* 2021, Pallotta *et al.,* 2019, Zafiropoulos *et al.,* 2014). The principal mode of action of CPF, like other OP insecticides, is irreversible inhibition of acetylcholinesterase (AChE-ase) enzyme. This enzyme hydrolyzes acetylcholine at cholinergic synapses and neuromuscular junctions. The persistent inhibition of AChE-ase enzyme results in neurotoxic effects and disrupts cholinergic function in the nervous system (George *et al.,* 2014). CPF can induce toxicity by other mechanisms, including oxidative stress, mitochondrial dysfunction, and inflammatory pathways (Abduh *et al.,* 2023, Montanarí *et al.,* 2024).

Oxidative stress can occur when there is an imbalance between oxidant and antioxidant conditions, because of either excess reactive oxygen species (ROS) production and/or depletion of antioxidants (Pisoschi *et al.,* 2021). It has been reported that CPF enhances the generation of ROS by disrupting the respiratory chain components' electron reflux and altering the activities of antioxidant enzymes, such as catalase (CAT) and superoxide dismutase (SOD), which results in oxidative stress and lipid peroxidation (AlKahtane *et al.,* 2020). Therefore, antioxidants may prevent CPF-induced oxidative processes and adverse effects by reducing reactive oxygen species (ROS) (Albasher *et al.,* 2019).

Today, natural oils are progressively utilized in the field of medicine on a global scale. These oils frequently exhibit a reduced incidence of adverse effects, demonstrate efficacy, and are readily accessible at economical prices. A multitude of these oils contain constituents that possess anti-inflammatory and antioxidant properties, rendering them beneficial for medicinal applications (Saber *et al.,* 2022, Abou-Zeid *et al.,* 2021). A promising oil seed plant belonging to the Simmondsiaceae family is Jojoba (Simmondsia chinensis) (Verbiscar and Banigan, 1978).

Jojoba oil is readily obtainable in large quantities from plant sources. It constitutes approximately 50% of the total weight of the seed (Miwa, 1971). The composition of this oil is unique, as it is predominantly comprised lengthy monounsaturated esters, in contrast to other oils which are primarily composed of triglycerides. Consequently, Jojoba oil possesses distinctive features and properties essential for applications in industrial chemistry and pharmaceutical fields (Abou-Zeid *et al.,* 2021, Sánchez *et al.,* 2016).

Numerous research studies demonstrated the preventive effects of Jojoba oil against hepatotoxicity induced by different toxicants (Abou-Zeid *et al.,* 2021). However, to the best of our knowledge, no prior research was carried out to examine the effectiveness of Jojoba oil in protecting against CPF toxicity. Thus, this work aimed to study the possible Jojoba oil alleviating properties towards CPF

hepatotoxicity in rats, which may have implications in managing individuals accidentally exposed to such substances.

MATERIALS AND METHODS

Chemicals

Chlorpyrifos was obtained from CHEMA INDUSTRIES, Cairo, Egypt, as a commercial formulation (dofos 50%). Crude Jojoba oil (100% purity) was obtained from Organic Company for Natural Oils, Egypt.

Experimental animals

Thirty adult male albino rats, 8-12 weeks old, weighing about 170-200g were used in this experiment. They were obtained from the Laboratory Animal House of the Faculty of Veterinary Medicine, Assiut University, Egypt. The animals were housed in plastic cages on wood chips for bedding, and allowed to acclimatize two weeks before starting the experiment. Rats were fed standard food pellets and tap water ad libitum. The rats were housed at 24-25 °C, with a humidity of 65% and in a daily dark/light cycle. Throughout the experiment, the animals were maintained in accordance with the guidelines of the Animal House of the Faculty of Veterinary Medicine, Assiut University. The study protocol was approved by the Ethics Committee of the Faculty of Veterinary Medicine, Assiut University, Assiut, Egypt, according to the OLE standards for use of animals in research under No. 06/2024/0227.

Experimental design

The rats were randomly divided into five groups (six rats each).

Group I (Control group): Rats received corn oil every other day for 4 months by oral gavage.

Group II (jojoba group): Rats were fed on a basic diet mixed with 2.5% jojoba oil for 4 months (Nassar *et al.*, 2017).

Group III (CPF-administration group): Rats were administered CPF dissolved in corn oil every other day for 4 months at dose level of 20 mg/kg body weight, orally intubated by gavage (Abd El-Moneim Ibrahim *et al.*, 2020).

Group IV (prophylactic group): Rats were fed on a basic diet mixed with 2.5% jojoba oil with CPF dissolved in corn oil every other day for 4 months at dose level of 20 mg/kg body weight, orally intubated by gavage.

Group V (therapeutic group): Rats were administered CPF by regimen as group III for 3 months, and then rats were fed on a basic diet mixed with 2.5% jojoba oil for a month.

Sample collection

By the end of the experimental period, the rats were anesthetized, blood was obtained from the retro-orbital venous plexus via glass capillaries, and then sacrificed (Stone, 1954). The liver tissues from each animal were isolated and divided into 2 parts; one part was dried and fixed in 10% formalin for histopathological examination. The other part was flash frozen in liquid nitrogen and stored at -80 °C to be used for oxidant/antioxidant assays.

Preparation of serum and tissue homogenate

Blood samples were put in a serum-separating tube, left to clot at room temperature for 30 min and centrifuged at 3000 rpm for 10 min. Then, the serum was separated immediately and stored at –20°C until used. For preparation of tissue homogenate, 5–10 ml cold buffer (50 mM potassium phosphate, pH 7.5) per gram of tissue was added to the liver tissue sample. Then tissue was homogenized mechanically in an ice water bath by rotor-stator homogenizer (GlassCol; catalog no. 009ck4424, Terrue Haute, USA) followed by centrifugation (4^{o}C, 4000 rpm, 15 min) by cooling centrifuge (Sigma 3-18 k s, Germany). The supernatant was cautiously collected and preserved at -80^{o}C until used. The supernatant was centrifuged again after thawing before the assay.

Biochemical estimations

The serum alkaline phosphtatase (ALP) level and the serum γ-glutamyltransferase (γGT)

level were estimated by a kinetic method using the (ALP) Liquizyme (9 + 1) IFCC E.C.3.1.3.1. kit (Cat. No: 214 001) and (γGT)-Liquizyme (9+1) kit (Cat. No: 246 001) obtained from Egyptian Co. for Biotechnology-Spectrum Diagnostics (S.A.E), Egypt.

The oxidant/antioxidant biomarkers were investigated in the hepatic tissues of different animal groups. Malondialdehyde (MDA), superoxide dismutase (SOD), and catalase (CAT) were measured using assay kits purchased from Biodiagnostic, Egypt (Cat. No. MD2529, SD2521 and CA2517, respectively).

Histopathological examination

Specimens from hepatic tissue were excised, fixed in 10% neutral buffered formalin with phosphate for 24 h, then dehydrated through ascending grades of ethanol, followed by clearing with xylol and impregnation in pure soft paraffin for 2 h at 50˚C, which was then embedded in hard paraffin blocks. Tissue sections (5 µm-thick) were cut using a microtome, mounted on glass slides and stained with haematoxylin and eosin (H&E) for general histological examination by light microscopy to detect the histopathological changes.

Statistical analysis

The statistical analysis was performed with the statistical package GraphPad Prism Version 7 (GraphPad Software Inc., San Diego, USA). The data was presented as mean ± SD. A comparison of the data among the groups was performed using one way analysis of variance (ANOVA) followed by Tukey's multiple comparison method to compare means of different parameters in different groups. The difference was considered to be statistically significant at $P<0.05$.

RESULTS

1. Prophylactic and therapeutic effects of Jojoba oil on serum hepatotoxicity indices

The results revealed that CPF administration significantly increased the serum levels of ALP and GGT ($P<0.001$) compared to their levels in the control group and Jojoba groups. On the other hand, the Jojoba prophylactic group showed a significant decrease in the serum levels of ALP and GGT levels ($P<0.05$) when compared to the CPF-administrated group. Regarding the Jojoba therapeutic group, the estimation of serum levels of GGT and ALP revealed a significant decreased serum levels of GGT levels ($P<0.05$) with a decrease in ALP level but not reaching statistical significance ($P=0.08$), compared with the CPF-administrated group (Figure 1 and Table 1).

2. Prophylactic and therapeutic effects of Jojoba oil on hepatic oxidative stress markers

To investigate the oxidative stress in the liver tissue, malondialdehyde (MDA) concentration as well as the antioxidant superoxide dismutase (SOD) and catalase (CAT) activities were assessed in different animal groups. The results showed that CPF significantly increased the concentration of MDA ($P<0.001$) and significantly reduced SOD and CAT activities ($P<0.001$) in the hepatic tissues compared to the control and Jojoba groups.

On the other hand, both Jojoba prophylactic and therapeutic groups exhibited a significant decrease in MDA level ($P<0.01$) and a significant increase in SOD and CAT activities ($P<0.01$) and ($P<0.05$), respectively, compared to their levels in the hepatic tissues of the CPF-administrated group (Figure 2 and Table 2).

Table 1: Prophylactic and therapeutic effect of Jojoba oil on serum hepatotoxicity indices.

Variables	Control group	Jojoba group	CPF group	Jojoba prophylactic group	Jojoba therapeutic group
ALP (U/dl)	209.5 ± 14.68	208.0 ± 15.60	268.7 ± 20.68 **a***b*****	239.3 ± 14.31 **a*b*c***	242.5 ± 18.25 **a*b***
GGT (U/dl)	2.09 ± 0.66	2.26 ± 0.38	4.78 ± 0.79 **a***b*****	3.47 ± 0.78 **a*b*c***	3.51 ± 0.69 **a**b*c***

ALP: alkaline phosphatase; CPF: chlorpyrifos group; GGT: γ – Glutamyltransferase.
Data are expressed as mean ± SD (n = 6 in each group) and analyzed by One-way analysis of variance (ANOVA) followed by Tukey test.
a Significantly different from the value in the control group.
b Significantly different from the value in the Jojoba group.
c Significantly different from the value in the CPF-administrated group.
***p ≤ 0.001, **p ≤ 0.01, and *p ≤ 0.05.

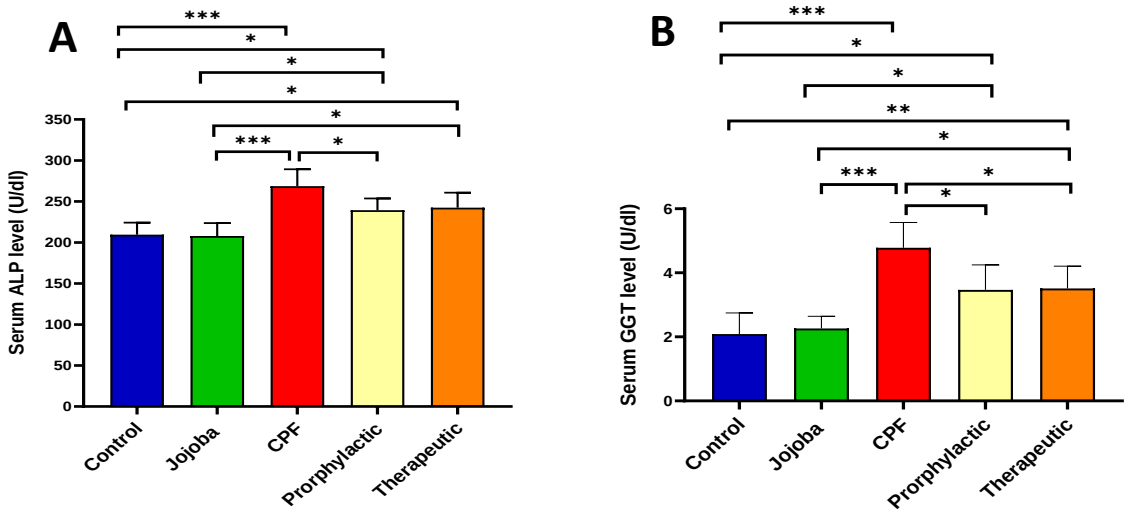

Figure 1: Prophylactic and therapeutic effect of Jojoba oil on serum hepatotoxicity indices: (A) ALP and (B) GGT (n = 6 in each group). Data are expressed as mean ± SD. ***p ≤ 0.001, **p ≤ 0.01, and *p ≤ 0.05. ALP: alkaline phosphatase; CPF: chlorpyrifos group; GGT: γ – Glutamyltransferase.

Table 2: Prophylactic and therapeutic effect of Jojoba oil on hepatic oxidative stress markers.

Variables	Control group	Jojoba group	CPF group	Jojoba prophylactic group	Jojoba therapeutic group
MDA (nmol/g)	25.6 ± 3.39	26.45 ± 3.8	50.02 ± 5.19 **a***b*****	36.32 ± 5.66 **a*b*c****	37.53 ± 7.26 **a**b**c****
SOD (U/g)	20.65 ± 1.46	20.9 ± 2.09	12.07 ± 1.89 **a***b*****	16.93 ± 2.19 **a*b*c****	15.97 ± 2.13 **a**b**c***
CAT (U/g)	3.12 ± 0.23	3.22 ± 0.25	1.72 ± 0.34 **a***b*****	2.48 ± 0.44 **a*b*c****	2.37 ± 0.43 **a**b**c***

CAT: catalase; CPF: chlorpyrifos group; SOD: superoxide dismutase; MDA: Malondialdehyde.
Data are expressed as mean ± SD (n = 6 in each group) and analyzed by One-way analysis of variance (ANOVA) followed by Tukey test.
a Significantly different from the value in the control group.
b Significantly different from the value in the Jojoba group.
c Significantly different from the value in the CPF-administrated group.
***p ≤ 0.001, **p ≤ 0.01, and *p ≤ 0.05.

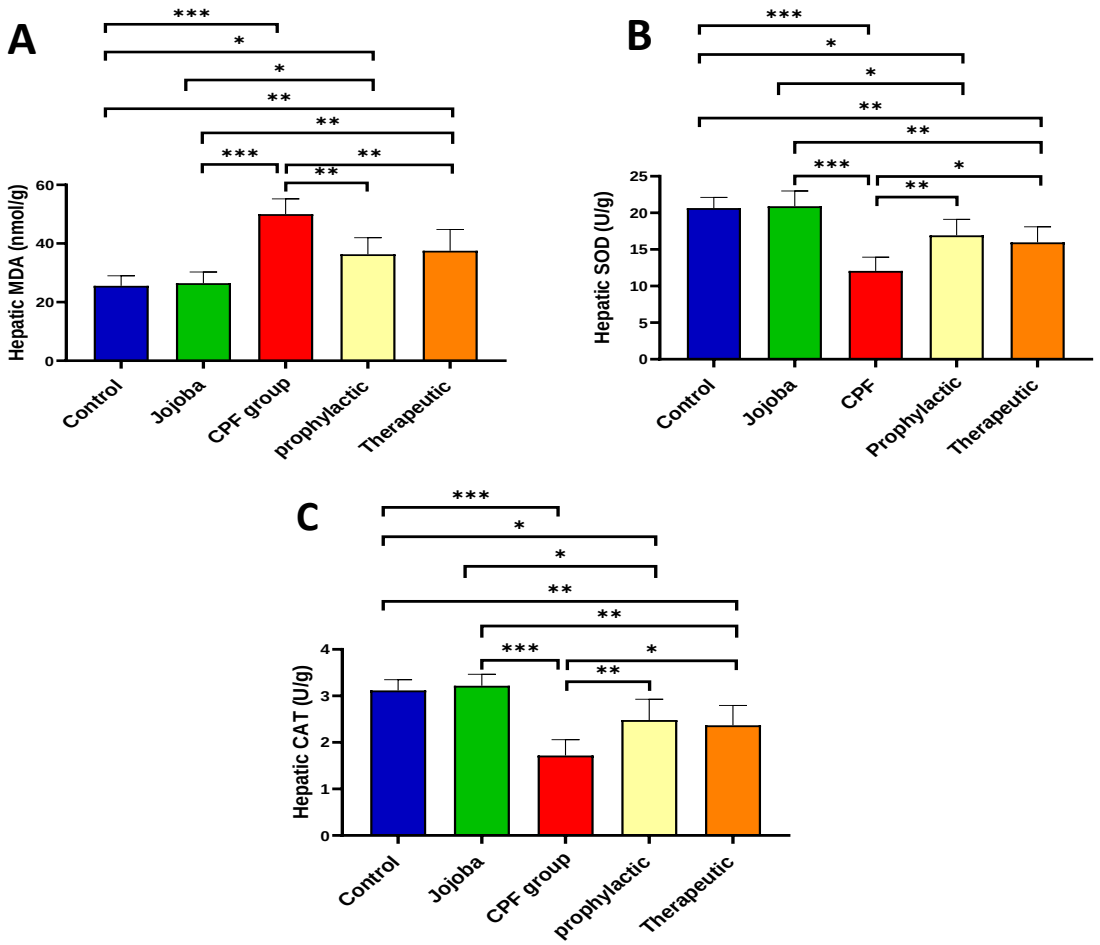

Figure 2: Prophylactic and therapeutic effect of Jojoba oil hepatic oxidative stress markers :(A) hepatic MDA, (B) hepatic SOD and (C) hepatic CAT (n = 6 in each group). Data are expressed as mean ± SD. ***p ≤ 0.001, **p ≤ 0.01, and *p ≤ 0.05. CAT: catalase; CPF: chlorpyrifos group; SOD: superoxide dismutase; MDA: malondialdehyde.

3. Prophylactic and therapeutic effects of Jojoba oil on histopathological examination of liver tissue

The liver tissue sections from control and Jojoba groups showed normal histological architecture of hepatic lobules with adjacent sinusoids radiating from the central veins (CV) toward the periphery of the liver lobules and normal portal triad. The hepatocytes were of normal shape with well-defined nuclei. The cytoplasm appeared uniform and regular (Figure 3 A1, A2, B1 and B2). In contrast, the liver of CPF-treated group showed congestion of central veins, severe hydropic degeneration (reticulated cytoplasm), degeneration of the wall of blood vessels, hemorrhage, hyperplasia of bile duct with infiltration of inflammatory cells, focal areas of necrosis and some apoptotic cells (Figure 3 C1, C2, C3 and C4).

On the other hand, liver tissue sections from the Jojoba prophylactic group and the Jojoba therapeutic group were more or less similar to those of the control group, revealing a normal central vein and hepatic cords and an absence of necrosis and hemorrhage. While some hepatic cells suffered from hydropic degeneration, mild hyperplasia of bile duct and mild inflammatory cells infiltration in the Jojoba prophylactic group and there was moderate hydropic degeneration, mild hyperplasia and mild inflammatory cell infiltration of bile duct in liver sections from the Jojoba therapeutic group (Figure 3 D1, D2, E1 and E2).

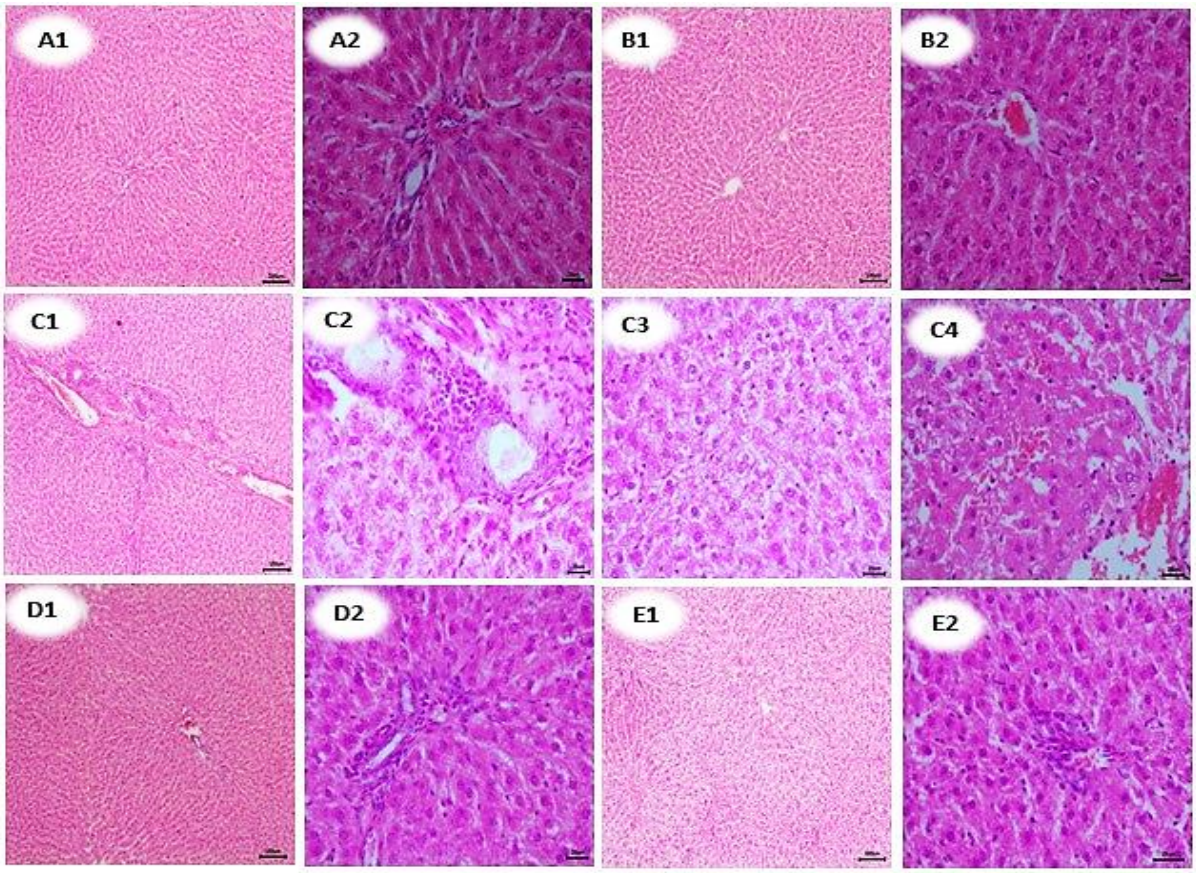

Figure 3: Liver section from control group (A1) and higher magnified (A2) showing normal histological architecture (scale bar =100 µm, 20 µm, respectively). Liver section from Jojoba group (B1) and higher magnified (B2) showing normal histological (scale bar =100 µm, 20 µm, respectively). Liver section from CPF group (C1) showing hyperplasia of bile duct, inflammatory cell infiltration, severe hydropic degeneration and degeneration of vascular wall (scale bar =100 µm) and higher magnified (C2) showing hyperplasia of bile duct, inflammatory cell infiltration, severe hydropic degeneration and degeneration of vascular wall, (C3) showing sever hydropic degeneration (reticulated cytoplasm), (C4) showing focal area of necrosis, hemorrhage, hydropic degeneration and degeneration of vascular wall (scale bar =20 µm). Liver section from Jojoba prophylactic group (D1) showing nearly normal histological architecture of hepatic lobules, normal central veins and mild hydropic degeneration (scale bar =100 µm) and higher magnified (D2) showing normal blood vessels, mild hyperplasia of bile duct and mild hydropic degeneration (scale bar =20 µm). Liver section from Jojoba therapeutic group (E1) and higher magnified liver (E2) showing normal blood vessels, moderate hyperplasia of bile duct and moderate hydropic degeneration (scale bar =100 µm, 20 µm, respectively).

DISCUSSION

Chlorpyrifos (CPF) has been classified among the most used organophosphorus poisoning pesticides for agricultural and household purposes. Its application has been linked with severe adverse reactions in farm animals and humans (Ore *et al.*, 2023). The liver is a vital organ in the human body and plays a crucial role in detoxification and is an important metabolic organ that converts toxic substances into less harmful or excretable forms, making it particularly susceptible to damage from various environmental toxins, including CPF (Liu *et al.*, 2021). Jojoba oil exhibits significant antioxidant properties. The unique composition of Jojoba oil, primarily consisting of long monounsaturated esters, enhances its bioactivity, making it valuable in various health applications (Chakrabarty *et al.*, 2024). The oil's antioxidant capacity

is largely attributed to its phenolic content, which has been shown to effectively scavenge free radicals, thereby reducing oxidative stress, a key factor in CPF toxicity (Kara, 2017). Therefore, the present study was carried out to investigate the antioxidant effects of Jojoba oil in CPF-induced hepatotoxicity in rats.

Experimental investigations have demonstrated that CPF is metabolized in the liver through hydrolytic ester cleavage and oxidative pathways by cytochrome P450 enzymes, resulting in enhanced production of reactive oxygen species (ROS) which may explain its toxic effects (Taha *et al.*, 2021). Lipid peroxidation is a major outcome of the free radical mediated-injury due to the high concentration of polyunsaturated fatty acids in cells (Gupta *et al.*, 2024). It can cause structural damage to cellular membranes and the generation of oxidized products like malondialdehyde (MDA) (Ozturk Kurt and Ozdemir, 2023).

The results of the present study revealed that CPF leads to the generation of ROS and lipid peroxidation, evidenced by increased MDA levels and histopathological changes in hepatic tissue of CPF intoxicated animals. These changes include severe hydropic degeneration, blood vessel wall degeneration, bile duct hyperplasia, and hemorrhage, possibly due to its toxic effect on the capillary endothelium (Altun *et al.*, 2017). These findings were in line with previous studies (Küçükler *et al.*, 2021, Owumi *et al.*, 2022, Abdel-Naim *et al.*, 2023, Montanarí *et al.*, 2024).

Antioxidant enzymes are proteins involved in the catalytic transformation of ROS and their byproducts into stable and non-toxic molecules. Therefore, it is the most important defense mechanism against cellular damage caused by oxidative stress (Thowfeik, 2016). Enzymatic defense, consisting of superoxide dismutase (SOD) and catalase (CAT), is a crucial subcellular defense mechanism in the antioxidant system, which counteracts free radicals produced during xenobiotic exposure by converting superoxide radicals into hydrogen peroxide (H_2O_2) and hydrogen peroxide into water (H_2O) (Mansour and Mossa, 2010).

Consistently and in line with earlier research (Albasher *et al.*, 2019, AlKahtane *et al.*, 2020, Xun *et al.*, 2020). Our results revealed that rats intoxicated with CPF showed lower SOD and CAT activities, possibly due to the utilization of these enzymes in conversion of the O_2^- to H_2O_2 and H_2O_2 to H_2O, respectively. On the other hand, the findings of the present study found that Jojoba oil, used in both prophylactic and therapeutic groups, significantly increased SOD and CAT activities, while decreasing MDA levels. Notably, Jojoba prophylactic group showed more improvement in SOD and CAT activities than the jojoba therapeutic group, indicating that Jojoba oil's use has more beneficial effects.

Jojoba oil's antioxidant properties are attributed to its high content of natural antioxidants like α, γ, and δ tocopherols, which are major forms of vitamin E (Awad *et al.*, 2022). Vitamin E acts as a powerful antioxidant, regulating oxidation processes and reducing the damaging effects of reactive oxygen species (ROS) (Ungurianu *et al.*, 2021). In addition, Jojoba oil's fatty acid composition mainly consists of unsaturated fatty acids (up to approximately 97%), such as eicosanoic, docosenoic, oleic, linoleic, and linolenic acids (Atteya *et al.*, 2021). The existence of double bonds in these unsaturated fatty acids enables them to contribute hydrogen atoms and neutralize free radicals, hence reducing oxidative damage (Ma *et al.*, 2023) and representing an additional mechanism of Jojoba oil's antioxidant effect.

A previous study reported that Jojoba oil has hepatoprotective and anti-inflammatory properties (Abou-Zeid *et al.*, 2021). In agreement, our histopathological investigation showed that Jojoba oil administration improved liver tissue changes, revealing normal central vein and

hepatic cords and no necrosis or hemorrhage. However, some hepatic cells still experienced hydropic degeneration, hyperplasia of the bile duct, and inflammatory cell infiltration with lesser hepatic tissue alterations observed in the prophylactic group compared to the therapeutic group.

Alkaline phosphatase (ALP) is present in the liver, mucosal epithelia of the small intestine, proximal convoluted tubule of the kidney, bone, and placenta. The serum ALP activity is primarily from the liver, with 50% from bone. Gamma glutamyl-transferase (GGT) is present in hepatocytes and biliary epithelial cells, renal tubules, pancreas and intestine (Rifai, 2017). Serum GGT activity correlates closely with the activities of ALP in various forms of liver disease, and is used in the evaluation of hepatic damage (Agnihotram *et al.,* 2010).

The results of the present study revealed that CPF administration increased the activities of GGT and ALP in male rats' sera, possibly due to liver dysfunction and disruptions in their biosynthesis. Additionally, the lipophilicity of CPF allows large amounts of CPF residue to penetrate cell membranes, causing changes in membrane permeability and subsequent leakage of these enzymes from the liver cytosol into the bloodstream (Fu *et al.,* 2024, Mansour and Mossa, 2010, Uzun and Kalender, 2013).

Elevations of GGT and ALP enzyme activities are observed in diseases that particularly affect the biliary tract (Gowda *et al.,* 2009). This was correlated with histopathological lesions observed in the current investigation. These lesions included hyperplasia of bile duct, which might be attributed to CPF excretion in the bile (Eaton *et al.,* 2008). Furthermore, the portal area was significantly affected by severe hydropic degeneration, necrotic areas, and infiltration of inflammatory cells since CPF can reach the liver via the portal vein (El-Bendary *et al.,* 2013).

Current findings Jojoba oil has been found to reverse altered serum levels of ALP and GGT, improving the condition of the bile duct due to its antioxidant activities. This is consistent with a 2015 study by Sobhy *et al.,* which found a significant decrease in ALP and MDA levels in liver tissues and increased liver activity of CAT enzyme in cadmium-intoxicated rats. The Jojoba prophylactic group showed more ameliorative effects compared to the Jojoba therapeutic group.

The findings of the current study showed that Jojoba oil reverted the altered serum levels of ALP and GGT, which might be attributed to Jojoba oil's antioxidant activities, thus protecting cells from oxidative stress and preventing cellular damage. These results are in line with a previous study, which reported that Jojoba oil significantly lowered ALP and MDA levels in cadmium-intoxicated rats while increasing CAT enzyme activity (Sobhy *et al.,* 2015). It is worth mentioning that the Jojoba prophylactic group showed more ameliorative effects compared to the Jojoba therapeutic group. Collectively, Jojoba oil's antioxidant properties may contribute to its effectiveness in protecting the liver and improving antioxidant defense in rats intoxicated with CPF.

CONCLUSION

The present study concluded that Jojoba oil had antioxidant and hepatoprotective effects and exhibited improvement in liver function against injury induced by CPF. Jojoba oil had the ability to reduce oxidative stress and augment antioxidants in CPF-intoxicated rats. The usage of Jojoba oil as prophylactic provides the best improvement in all biochemical measurements and histological structure.

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التأثيرات الوقائية و العلاجية المحتملة لزيت الجوجوبا ضدد التسمم الكبدي الناجم عن الكلوربيريفوس في الجرذان

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الكلوربيريفوس هو مبيد حشري فوسفاتي عضوي فعال يُستخدم علي نطاق واسع. التعرض لمبيد الكلوربيريفوس يسبب تلف الكبد من خلال الإجهاد التأكسدي؛ حيث يعطل نظام إزالة السموم (المؤكسد/مضاد الأكسدة) ، مما يؤدي إلى السمية الكبدية. تستخدم الجوجوبا على نطاق واسع كنبات طبي حيث يتم زراعته في جميع أنحاء العالم. يحتوي زيت الجوجوبا المستخرج من بذور الجوجوبا على نسبة عالية من الفينولات والفلافونويدات، مما يجعله مضادًا قويًا للأكسدة ويمكنه التخلص من الشوارد الحرة. لذلك، يهدف البحث الحالي إلى تقييم التأثيرات الوقائية والعلاجية المحتملة لزيت الجوجوبا ضد التأثير التأكسدي للتسمم الكبدي الناتج عن الكلوربيريفوس في الجرذان. تم تقسيم ثلاثين من ذكور الجرذان البالغين إلى خمس مجموعات، كل مجموعة تحتوي على ستة حيوانات. المجموعة الضابطة، مجموعة الجوجوبا، مجموعة الكلوربيريفوس، مجموعة الجوجوبا الوقائية، ومجموعة الجوجوبا العلاجية. تم الحصول على عينات دم وأنسجة كبدية من كل جرذ لإجراء الفحوصات الكيميائية الحيوية والأنسجة المرضية. أظهرت النتائج أن تأثيرات الكلوربيريفوس السامة على الكبد تمثلت بارتفاع مستوى الفوسفاتاز القلوي والغلوتاميل ترانسفيراز في المصل، مع تغييرات هيستوباثولوجية في نسيج الكبد. بالإضافة إلى ذلك، أظهرت نتائجنا تورط الإجهاد التأكسدي في تلف الكبد بعد التسمم بمبيد الكلوربيريفوس، والذي تجلى بزيادة مستوى المالونديالديهايد وانخفاض نشاط إنزيمات السوبر أوكسيد ديسموتاز والكاتالاز في الأنسجة الكبدية. على الجانب الاخر، تم عكس هذه النتائج من خلال إعطاء زيت الجوجوبا. أشارت بياناتنا إلى أن زيت الجوجوبا يمكن أن يخفف من الإجهاد التأكسدي الناجم عن الكلوربيريفوس في الأنسجة الكبدية ويقلل من التغيرات النسيجية المرضية. في الختام، يمكن أن يوفر زيت الجوجوبا تأثيرات وقائية وعلاجية كبيرة ضد السمية الكبدية الناتجة عن الكلوربيريفوس، مما يجعله عامل حماية وعلاج طبيعي محتمل الفائدة.