Protective role of *Moringa oleifera* seed oil against hepatotoxicity induced by Diazinon in male rats

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Abstract

Diazinon (DZN) is a widely used organophosphate pesticide has environmental risks and health concerns via oxidative stress. This study investigated whether Moringa oleifera seed oil (MSO)can act as a protective shield against DZN-induced hepatotoxicity in male rats. **Methods:** Twenty-four adult male Wistar albino rats (150-200g) were randomly distributed into four groups represented by Control (corn oil, 1.0 ml/kg),second group was administrated by DZN (12.5 mg/kg body weight), third group was recieved MSO (200 mg/kg/day), and last group was received mixed(MSO+DZN) as protective for four weeks . Biochemical parameters including liver enzymes (, ALP, LDH, ALT& AST), lipid profile, antioxidant markers (SOD, CAT, GPx, GSH), and lipid peroxidation (MDA) were analyzed. Histopathological examination of liver tissues was performed using H&E staining. **Results:** Dzn Exposure Significantly Elevated Serum It is more scientifically accurate to say that it showed "a significant decrease in liver enzymes (ALT,AST and LDH) compared to the DZN group Also, always include a p-value next to the main results (e.g., $p < 0.05$ or $p < 0.01$) to enhance the statistical power of the conclusion.Compared to controls and MSO and protective group showed clear improvement among study parameters ($P<0.05$). Description of the histological results: It is preferable to link the histological results to the infected group first clearly and then mention the improvement in the protection group. showing clear signs of cell death and distress, such as hyperchromatic nuclei and vacuolar degeneration. **Conclusions:** Our findings highlight DZN's ability to compromise hepatic integrity through oxidative mechanisms. Interestingly, MSO demonstrated a significant capacity to shield the liver from this damage.**Keywords:** Water stress, Zea mays, Physiological, Chlorophyll content.

Keywords: Diazinon; Hepatotoxicity; Moringa oleifera; Oxidative stress; Antioxidants; Environmental toxicology; Pesticide poisoning

Introduction

GPoisoning from organophosphate (OP) insecticides continues to be a major global health concern. Although efforts have been made to limit their use in certain countries, these chemicals remain widely used across many parts of the world, leaving agricultural workers vulnerable to excessive exposure. (Gisila, 2018; Goetzman&Prochownik, 2018). The mechanistic basis of OPP of hepatotoxicity by mitochondrial dysfunction, increase ROS production, peroxidation of lipid , and depletion of cellular antioxidant defense systems. These oxidative stress-potentiate pathways finally result damage of membrane, reduce cellular function, and extracellular death(Su et al., 2021; Yang, Wei, Zhang,&Li, 2021).

Moringa oleifera commonly favorite and has garnered significant attractive due to its nutritional and medicinal properties (Chaudhary et al., 2023).On the other aspect it, has demonstrates multi-therapeutic activities, including antioxidant, antiviral, anti-diabetic , anti-inflammatory, antimicrobial, and protectiveness of liver (Zeinali, Meybodi, Rezaee, Rafatpanah,&Hosseinzadeh, 2018).

The seed oil of *Moringa oleifera* particularly rich in natural antioxidants involve tocopherols, phenolic compounds, flavonoids, and other bio-active phytochemicals(Karimani, Heidarpour,&Moghaddam Jafari, 2019)These agents have potent free radical scavenging capabilities and can potentiate cellular antioxidant enzyme systems. Despite growing interest in M. oleifera's health benefits, limited research has specifically examined the protective efficacy of its seed oil against organophosphate pesticide-induced hepatotoxicity (Hong et al., 2022).This study was aimed to evaluate the protectiveness role of *Moringa oleifera* seed oil versus DZN-induced oxidative liver damage in a rat model, providing insights into potential therapeutic strategies for pesticide poisoning management and environmental health protection.

Material and Methods

Technical-grade diazinon (98% purity, CAS No. 333-41-5) was obtained from El-Helb Pesticides and Chemicals Company (New Damietta, Egypt). DZN solutions were prepared in normal saline (0.9% NaCl) and confirmed for stability over 7 days at room temperature according to U.S. Environmental Protection Agency guidelines. Fresh dosing solutions were prepared twice weekly to ensure consistency and prevent degradation of the active compound.

Experimental animals: Twenty-four adult male Wistar rats, aged 3–4 months and weighing between 200 and 250g, were sourced from the Animal House at AL-Qassim Green University. The animals were housed in plastic cages under appropriate environmental conditions, with unrestricted access to pelleted feed and filtered tap water. Prior to the experimental procedures, a 14-day acclimatization period was observed to minimize stress-related variables.

Experimental design: After period of acclimatization, animals were distributed randomly allocated to four experimental groups (n=6 per group) using a computerized randomization procedure:

Group I (Control): Received corn oil vehicle (1.0 ml/kg body weight) via oral gavage1.0 ml/kg body weight) via gavage for 28 consecutive days.

Group II (DZN): Received orally diazinon (12.5 mg/kg body weight, equivalent to 1/100 of the reported oral LD50 value) dissolved in saline, administered orally for 4 weeks. This dose was selected based on previous toxicological studies demonstrating subacute toxicity without acute mortality.

Group III (MSO): Received Moringa oleifera seed oil (200 mg/kg/day) administered PO for 28 days. **Group IV (MSO+DZN):** Received MSO (200 mg/kg/day) followed by DZN administration (12.5 mg/kg) after a 30-minute interval, for same periods. The time interval was designed to allow MSO absorption before DZN exposure.

All treatments were administered between 09:00 and 11:00 hours to minimize circadian variations. Body weights were recorded weekly throughout the experimental period. Animals were monitored daily for signs of toxicity, behavioral changes, and general health status.

Sample collection and preparation

After end of experimental study at 28-day , animals were fasted overnight (12 hours) with free access to water. Animals were sacrificed under light choloroform anesthesia. Blood samples (5 ml) were collected via cardiac puncture into plain tubes and placed in refrigerator for 30 minutes. Samples were then centrifuged at 4000 rpm for 10 minutes at 4°C to get serum, which was stored at -20°C until biochemical analysis .

Liver tissues samples were immediately excised, washed with ice-cold normal saline to remove blood, blotted dry, and weighed. Portions of liver tissue were fixed in 10% phosphate-buffered formalin (pH 7.4) for histopathology inspection . The anthoer part of liver tissue was homogenized (10% w/v) in ice-cold phosphate buffer (pH 7.4) using a Potter-Elvehjem homogenizer. Homogenates were centrifuged at 10,000 rpm for 20 minutes at 4°C, and the supernatants were collected and stored at -20°C for biochemical assays of oxidative stress markers.

Biochemical analysis

Serum biochemical parameters were analyzed using a Shimadzu UV-VIS Recording 2401 PC spectrophotometer (Shimadzu Corporation, Japan) with commercial diagnostic kits. Liver enzyme activities including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were measured using Biodiagnostic kits (Biodiagnostic Company, Dokki, Giza, Egypt) according to the manufacturer's protocols. Serum lactate dehydrogenase (LDH) activity was analyzed using Spinreact kits (Spinreact S.A., Santa Coloma, Spain). Total protein and albumin concentrations were determined using colorimetric methods with Biodiagnostic kits. Serum triglycerides and total cholesterol were measured using enzymatic colorimetric methods.

Oxidative stress markers

Hepatic oxidative stress markers were evaluated in tissue homogenate supernatants as follows:

Malondialdehyde (MDA): Lipid peroxidation was assessed by measuring MDA levels using the thiobarbituric acid reactive substances (TBARS) method [22]. Briefly, tissue homogenate was mixed with TBA reagent and heated at 95°C for 60 minutes. After cooling, the absorbance was measured at 532 nm. MDA concentration was calculated using a molar extinction coefficient of 1.56×10^5 $M^{-1}cm^{-1}$ and expressed as nmol/g tissue.

Reduced glutathione (GSH): GSH content was measured using the method of Beutler et al. [23]. This method is based on the reaction of GSH with 5,5'-dithiobis(2-nitrobenzoic acid) (DTNB) to produce a colored compound measured at 412 nm. Results were expressed as μmol/g tissue.

Catalase (CAT): CAT activity was assessed by monitoring the decomposition of hydrogen peroxide (H_2O_2) at 240 nm according to the method of Aebi [24]. **Superoxide dismutase**

(SOD): SOD activity was measured through the inhibition of pyrogallol auto-oxidation at 440 nm according to Marklund and Marklund [25]. One unit of SOD activity was defined as the amount of enzyme that inhibits pyrogallol auto-oxidation by 50%. Results were expressed as U/g tissue.

Glutathione peroxidase (GPx): GPx activity was evaluated by measuring the rate of NADPH oxidation at 240 nm in the presence of glutathione reductase, according to the method of Paglia and Valentine [26]. One unit of GPx activity was defined as the amount of enzyme that oxidizes 1 μmol of NADPH per minute. Activity was expressed as U/g tissue.

All spectrophotometric measurements were performed in triplicate, and protein concentrations in tissue homogenates were determined using the Bradford method with bovine serum albumin as standard.

Histopathological examination

Hepatic tissue was collected after animal sacrificed and fixed in 10% formalin and then dehydrated by a graded ethanol series (70%–100%). Next step was remove alcohol by xylene, the specimen were embedded in paraffin. We using a rotary micro-tome to done 5\mu sections, which were then mounted, de-paraffinized, and stained with common hematoxylin and eosin (H&E). A comprehensive light microscopic inspection (magnifications of ×100, ×200, and ×400) was done to record histopathology shifts. Our data appear specific lesions of injury, involving hepatic architecture, morphology, and inflammatory cell infiltration.

Statistical analysis

Data are presented as mean ± standard deviation (SD). Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test to determine significant differences between groups. Statistical significance was set at $P<0.05$. All analyses were conducted using SPSS statistical software (version 20.0 or higher) with minimum n=6 per group to ensure adequate statistical power.

Results and Discussion

Liver function

As seen in Tables 1, the levels of AST, ALT, ALP, LDH , triglycerides and total cholesterol in ٥ serum waswere statistically ($P < 0.05$) elevated, while the level of albumen & total protein and were significantly ($P < 0.05$) reduced in animals exposed to DZN as compared with negative control and other treated groups. Insignificant alterations in the levels of all studied parameters were noted in animals supplemented with MSO while pretreatment of rats administered DZN reduced the alteration in tested biochemical parameters (Tables 1).

Table 1 Effects of diazinon on some biochemical parameters of hepatic and lipid profile

Parameters				Groups
MSO + DZN	MSO	DZN	Control	
5.89 ± 0.11^{abc}	6.72 ± 0.35	4.32 ± 0.3^{acd}	6.59 ± 0.24	Total protein (g/dl)
3.43 ± 0.06^{abc}	4.189 ± 0.11	3.029 ± 0.06 ^{acd}	4.172 ± 0.16	Albumin (g/dl)
169.57 ± 2.6^{abc}	144.98 ± 3.92	196.94 ± 3.69^{acd}	141.69 ± 3.29	AST (U/L)
53.98 ± 1.08^{abc}	47.13 ± 0.87	65.29 ± 1.06^{acd}	46.19 ± 1.39	ALT (U/L)
356.67 ± 12.4^{abc}	307.78 ± 14.98	463.23 ± 15.3^{acd}	308.3 ± 11.03	ALP (U/L)
1872.7 ± 64.6^{abc}	1644.87 ± 29.33	2536.5 ± 62.23^{acd}	1640.3 ± 34.23	LDH (U/L)
120.86 ± 5.3^{abc}	149.99 ± 2.71	188.72 ± 6.43^{acd}	148.92 ± 6.89	Triglycerides (mg/dl)
116.12 ± 7.1^{abc}	91.22 ± 5.94	151.63 ± 6.8^{acd}	94.98 ± 7.98	Total cholesterol (mg/dl)

Values are data mean plus SD of 6 rats in each group. Significance at P <0.05. DZN:

abc :Small letter to explain asignificant difference between groups

3.2 Lipid Peroxidation

Lipid peroxidation (LPO) occurs when free radicals attack and oxidize lipids, and is widely recognized as a key indicator of oxidative damage in cells and tissues. In the present study, hepatic LPO was notably elevated in DZN-treated rats, as reflected by a significant (P<0.05) rise in malondialdehyde (MDA) levels (expressed as nmol MDA/g tissue) compared to control animals (Fig. 1). Interestingly, co-administration of MSO alongside DZN resulted in a significant (P<0.05) reduction in MDA levels relative to the DZN-only group, suggesting a potential protective role of MSO against DZN-induced oxidative stress.

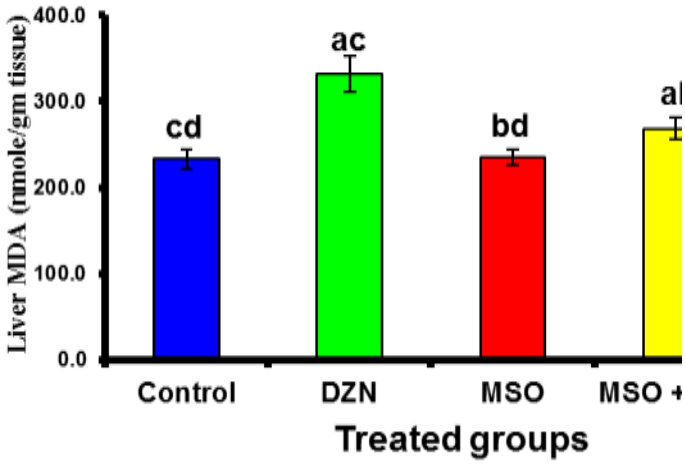

Figure 1. influence of long period administration of DZN on MDA nmole \ gm concentration in the liver tissues of rats.

Antioxidant Enzymes

The antioxidant parameters such as SOD , CAT and (GPx) are essential for evaluation defence of cellular antioxidant system. In the present study, the levels of these enzymes (U/g tissue) were significantly (P < 0.05) reduced in the livers of rats expose to DZN when compared to healthy animals. Therapy regimes with MSO, however, suitable to significant (P < 0.05) restoration of enzyme activities compared to the positive toxic group.

As shown in Figures 2, 3, and 4, routine and protective therapy has clear-improvement of hepatic SOD, CAT, and GPx activities, respectively. At the end of the 4th week, rats receiving both MSO and MSO-DZN appear significantly higher SOD, CAT, and GPx activities in liver tissue compared to those treated with DZN alone (Figure 2).

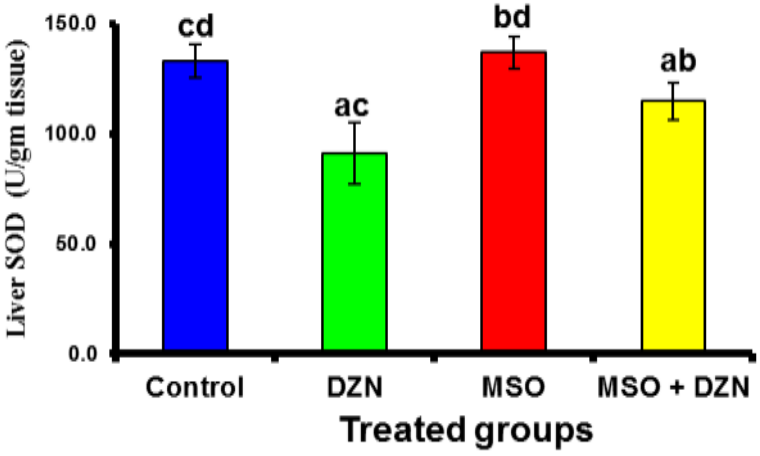

Figure 2. Chemical defense it is very helpful to make sure that the figures have bars showing the standard deviation and statistical symbols (such as a star * or the letters a, b, c) above the bars to show significant differences between groups

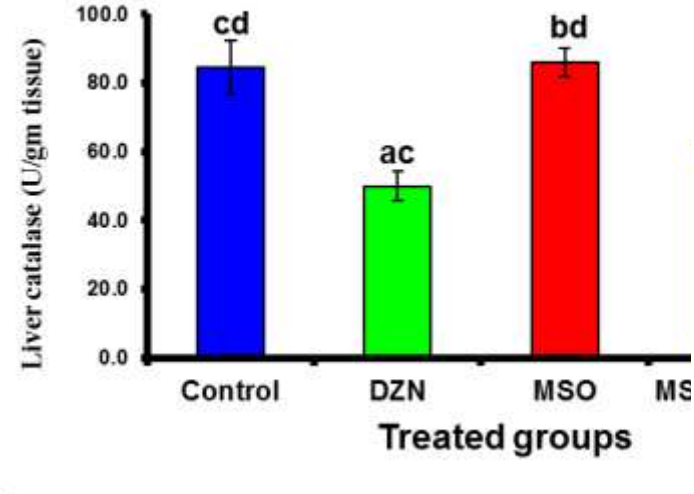

Figure 3. exposure influence of sub acute to of DZN on catalase activity in the liver tissues of rats.

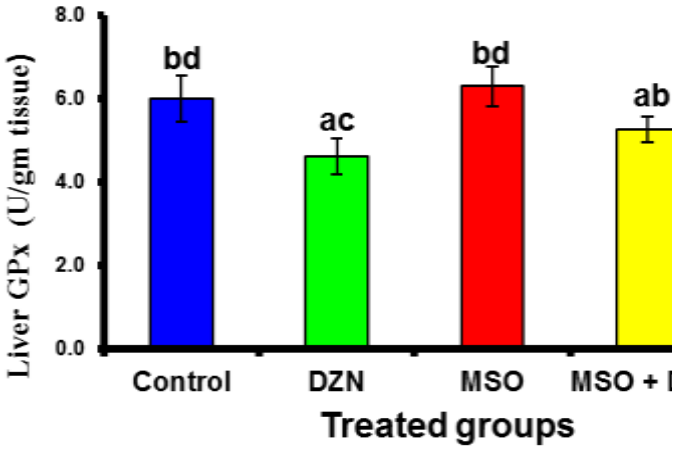

Figure 4. Effects of long periods administration diazinon on GPx activity in the liver tissues of rats.

4.4. Glutathione

Administration of DZN for 28 days reduced (P < 0.05) liver CSH concentration as compared to negative control. In-fact , combination therapy of rats with MSO and DZN elevated (P < 0.05) the GSH concentration in rat liver as compared with those treated with DZN alone (Figure 5).

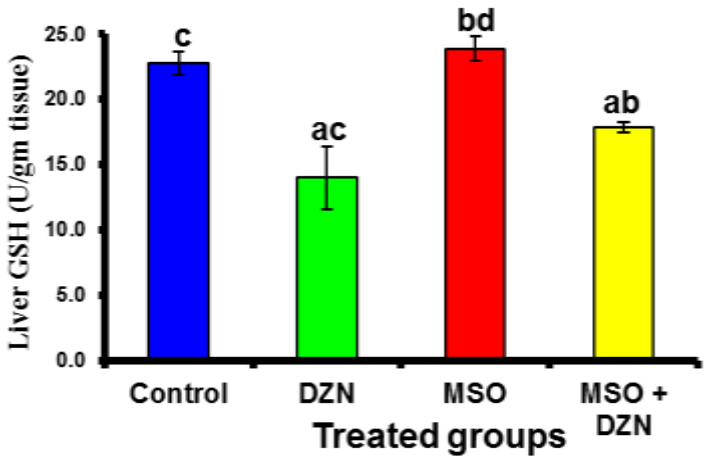

Figure 5. Effects of long therapy of diazinon on GSH content in the liver tissues of rats.

Histological changes:

Microscopic inspection of liver section of rats appear clear histopathological alteration across the treatment groups. In Group I (Control), as appear in Figure 6, the hepatic architecture showed entirely healthy. Hepatocytes were healthy in normal, binucleated cells were nearly encountered, a finding consistent with normal physiological regenerative activity. The hepatic sinusoids maintained patent lumina, and the portal triads were unremarkable, showing no evidence of inflammatory cell infiltration.

Protective rats with MSO (Group III) in figure (7) appeared hepatocytes arranged in single-cell plates and active cytoplasm appearing staining in

normal basophilic state, indicating good metabolic activity. The morphology of nuclear still in normal with no syptoms of degeneration. Moderate inflammation in central vein with sinusoidal dilation were recorded in some sections. Kupffer cells showed clear but not activated, and no significant pathological alterations were appear, confirming the safety of MSO at the administered dose.

DZN treatment (Group II) induced clear hepatic deleterious characterized by prominents lession in hepatic architecture. Proliferation hepatocyte cords extended toward dilated portal tracts, guiding compensatory regeneration in re-correct to healing of injury process in figure (8). Hepatocytes showed vacuolation of cytoplasmic ,due to lipid accumulation or hydropic degeneration. Nuclear alteration were obvious, appear hyperchromatic nuclei , karyolysis and nuclear shrinkage. Areas of focal necrosis were observed all the parenchyma, recognized via disappear of cellular detail and infiltration of inflammatory cell .

These histopathological changes refer to high extracellular injury, inability of lipid metabolism, and compromised liver function.

MSO protective therapy (Group IV) may be ameliorated hepatotoxicity. Liver sections appear hepatocyte cords radiating toward moderate dilated portal tracts with restore hepatic architecture figure (9) . The vacuolation of cytoplasmic was markedly diminished as compared to toxic zenobiotic groups with OPP, and hepatocytes appear clear mimic normally nuclear morphology with little degenerative alteration. Mild inflammatory cell infiltration was appear in some areas, may be due to ongoing tissue repair mechanism and inflammatory resolution. Fibrotic lesions were little or absent, and necrotic areas were clear diminshed as compared to positive hepatotoxic.

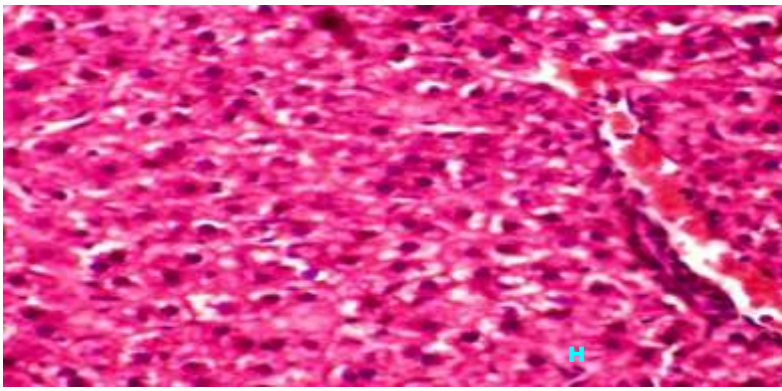

Figure 6: Photomicrographs of normal control rat liver section, show a cords of hepatocytes radiating to mild dilated and congested portal tracts (PT). Polygonal hepatocytes with centric rounded nuclei and some have binucleated (H&E stains, Bar=50µm)

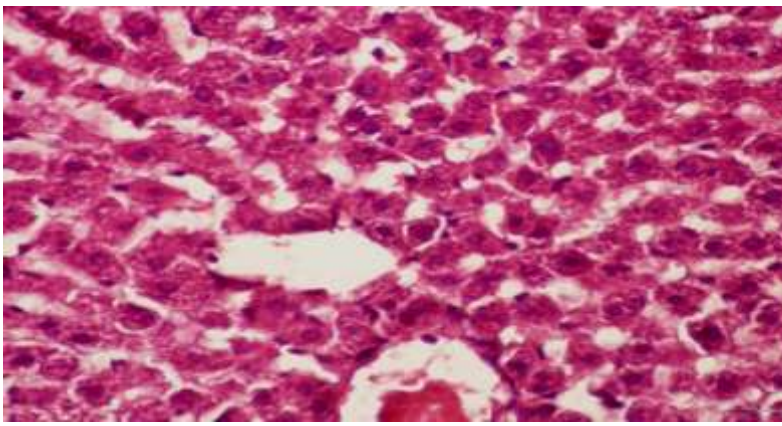

Figure 7: Photomicrographs of rat liver section administrated MSO hepatic architecture hepatocytes arranged in one cell plates with high activity

cytoplasm and normal rounded nuclei. Mild inflammation of central vein (CV), sinusoidal (S) dilation and Kupffer cells were noted. (H&E stains, Bar=50µm)

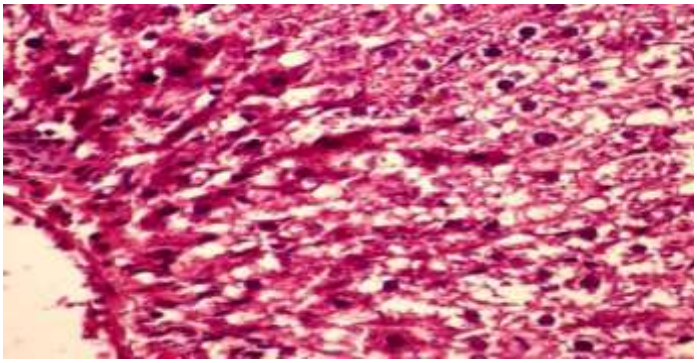

Figure 8: Photomicrographs of rat liver section administrated DZN, show a cords of proliferative hepatocytes radiating to dilated and portal tracts (pt) Vacuolated hepatocytes (VH) with centric hyperchromatic (CH) nuclei and some karyolitic hepatocytes (KH) as well as area of necrotic cells (H&E stains, Bar=50µm)

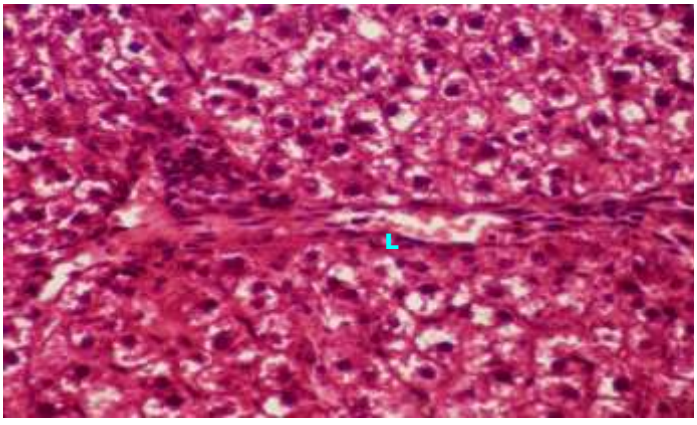

Figure 9: Photomicrographs of rat liver section administrated MSO+DZN, show a cords of hepatocytes radiating to mild dilated and congested portal tracts (pt). Area of infiltrating lymphocytes (L), regenerative hepatocytes with less vacuolated cytoplasm and normal rounded centric nuclei was noticed. Reduction of fibrotic sheath and area of necrotic cells (H&E stains, Bar=50µm)

There is growing interest in identifying plant-based antioxidants, largely because natural dietary sources are generally perceived as safer and free from the adverse effects associated with synthetic alternatives (Ogah et al., 2024; Sajad et al., 2024). While *Moringa oleifera* seed oil (MSO) possesses a wide range of biological activities, its therapeutic efficacy is primarily attributed to its antioxidant capacity. Consequently, the present study evaluates the ability of MSO to mitigate diazinon-induced (DZN) hepatotoxicity in rat models(Shahbaz et al., 2024).

The our data appear reduction in biochemical parameters like serum total protein within the DZN- group likely stems from hepatic defect and reduce protein biosynthesis(Abdulkarim, Jassim,&Hassan, 2025; AL-CHARAK et al., 2025; Ali, Sami, Haider, Ashfaq,&Javed, 2024). This clear reduction reported by previous articles involving various organophosphate , where a significant diminished serum blood protein levels were noted in experimental rats (Nassarawa, Nasiru, Magaji,&Abdullahi, 2025; Pal&Mahant, 2025). On the other aspect, DZN administration

make huge defect via significant elevations in standard parameters for liver as serum ALT, AST, and ALP enzymes typically localized in the restricted cytoplasm that leak into the bloodstream after cellular injury (Pal&Mahant, 2025; Rabee, Shaher, Obaid, Jasim,&Al-Gnami, 2025; Villegas-Vazquez, Gómez-Cansino, Marcelino-Pérez, Jiménez-López,&Quintas-Granados, 2025). These biochemical markers of hepatocyte damage were further corroborated by histopathological evidence of liver and kidney alterations, consistent with prior studies on DZN exposure (Zhou, Li,&Achal, 2025). The current study confirmed that OPP exposure leads to highly increase in serum TG and CHO, signaling a clear damage in lipid metabolism. These findings confirmed with others study recorder different lipid profile changes in rats exposed to OPP (Chaudhary et al., 2023; Mali et al., 2023). In facts, OPP support in accumulation of TG may lead to formation an imbalance between synthesis of hepatocyte and the secretion of TG into blood circulation (Al-Mansury, Aboktifa, Jassim, Balakit,&Alkazazz, 2022; Oz et al., 2023). In the present study, DZN treatment significantly reduced GPx activity, likely due to the surge in free radical production evidenced by elevated malondialdehyde (MDA) levels. This decline in GPx may also result from direct oxidative inhibition of the enzyme itself. Alongside GPx, enzymes such as superoxide dismutase (SOD) and catalase (CAT) constitute the primary enzymatic defense against macromolecular oxidative damage (Rajak et al., 2022; Zeinali et al., 2022).

The extract Moringa against CCl4" seems incomplete; it would be better phrased as: Previous studies have shown that Moringa extract protects against CCl4-induced hepatic injury CCl4 for hepatic injury and fibrosis by leveraging its antioxidant and anti-inflammatory properties, specifically through the minimize of hepatic stellate cell activation (Rajak et al., 2022; Zeinali et al., 2022). Islam et al. (2021)confirm a high concentration of monounsaturated fatty acids (MUFAs), with oleic acid accounting for 66–76% of the total composition (Kaushal, Khatri,&Arya, 2021; Naji, Jabber,&Jasim, 2021; Yang et al., 2021). This clinical program is able to other health-promoting plant extract like olive and high-oleic sunflower oil, which are represent as functional foods that reduce coronary heart disease risk. The therapeutic potential of *Moringa* seeds is largely attributed to their diverse phytochemical makeup. They are exceptionally rich in tocopherols and phenolic compounds, which work in tandem with essential vitamins to neutralize free radicals. The inclusion of unique compounds like isothiocyanates and glucosinolates distinguishes the seeds as a potent bio-resource for managing chemical-induced injury and systemic inflammation (Hamzah, Jasim,&Mohammed, 2020; Vuolo, Lima,&Junior, 2019).

Conclusion

The conclusion should be formulated as the main points: Improvement of Liver Enzymes: Combined treatment with MSO resulted in a significant decrease in the elevated serum levels of ALT, AST, ALP, and LDH enzymes, which are stimulated by DZN, indicating a reduction in fluid leakage from liver cells.

Restoration of Antioxidant Defense: MSO demonstrated potent hepatoprotective properties by enhancing the enzyme defense system (SOD, CAT, and GPx) and reducing lipid peroxidation (MDA).Structural Protection: MSO prevented the occurrence of serious pathological histological changes, such as vacuolar degeneration and focal necrosis, thus preserving the integrity of the liver's structure.

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