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HEPATOPROTECTIVE ACTIVITY OF ETHYL ACETATE EXTRACT OF *MORINGA OLEIFERA* LEAVES IN D- GALACTOSAMINE INDUCED HEPATITIS IN ALBINO RATS

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ABSTRACT: The study was conducted to evaluate the hepatoprotective effect of ethyl acetate extract of *Moringa oleifera* leaves in D -galactosamine (D-GalN) induced hepatotoxicity in albino rats. The hepatoprotective effect of ethyl acetate extract of *M. oleifera* leaves at doses of 150mg/kg, 200mg/kg and 250 mg/kg body weight respectively per orally was evaluated by inducing hepatotoxicity with D-GalN at a dose of 300mg/kg body weight intraperitoneally and using Silymarin 100mg/kg per orally as the standard reference drug. The hepatoprotective activity was monitored biochemically by various biochemical assays (serum SGOT, SGPT, ALP, LDH, γ GT, total and direct bilirubin) using a Sys 200 pro autoanalyzer and histopathological examinations of the collected liver were carried out. The extract exhibited significant ($p < 0.05$) hepatoprotective effect in a dose-dependent manner in D-GalN intoxicated albino rats. Standard drug, silymarin (100mg/kg) also significantly reversed the hepatotoxicity. The histopathological findings were supportive of the biochemical findings which highlighted the protective role of *Moringa oleifera* against hepatic injury induced by D- GalN.

INTRODUCTION: Liver disorders represent a global health burden accounting for 2 million deaths annually (4% of all deaths world width) ¹. Hepatotoxicity, or drug-induced liver injury (DILI), is a significant clinical concern, often leading to acute liver failure which is a leading reason for drug withdrawals and regulatory warnings worldwide ². Varieties of toxic agents have been implicated in the pathogenesis of hepatic injury. D Galactosamine (D-GalN), which is generated from D-galactose, is a renowned hepatotoxic drug that causes a diffuse kind of liver injury with necrosis, inflammation, and regeneration which is comparable to human viral hepatitis.

D-GalN inhibits hepatocyte RNA synthesis and lowers the quantity of cellular uridine-50-triphosphate (UTP) to impede tran scription in the liver ³. In recent years, there has been a growing interest in the therapeutic application of medicinal plants to mitigate hepatotoxic damage. *Moringa oleifera* a member of the family Moringaceae and commonly known as drumstick, sajana, horse radish tree or miracle tree has diverse pharmacological properties with hepatoprotective effects.

Various parts of the plant including leaves, fruits, flowers and immature pods of this tree are used as a highly nutritive vegetable in various countries such as India, Pakistan, Philippines, Hawaii and many parts of South East Asia. Phytochemical investigations have revealed that the plant possess a wide range of biological properties including antioxidant, anti-inflammatory, antidiabetic, antihypertensive, anti- tumour, hepatoprotective,

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antiulcer, antipyretic, anticonvulsant and antibacterial properties⁴. In addition, the extract of leaves of *Moringa oleifera* exhibited significant activity against anti-tubercular drugs⁵, carbon tetrachloride⁶, paracetamol⁷, cadmium⁸, nickel⁹, thioacetamide¹⁰, alcohol¹¹ induced hepatotoxicity in animal models. Silymarin, a flavonolignan complex extracted from *Silybum marianum* is a well-tolerated and effective drug used in cases of hepatotoxicity produced by a number of hepatotoxic agents¹². However, there were less evidences regarding efficacy of *M. oleifera* against D-Galactosamine induced hepatotoxicity in rats. Accordingly, the present study was undertaken to evaluate the hepatoprotective role of *Moringa oleifera* against D-GalN induced hepatotoxicity in rats.

MATERIALS AND METHODS: The protocol containing the materials and methods employed in the present investigation was approved by Institutional Animal Ethics Committee of Regional Institute of Medical Sciences, Imphal (Registration no.1596/GO/a/12/CPCSEA). The present study was conducted in the Department of Pharmacology, JNIMS, Imphal, Manipur, in collaboration with the Department of Pathology and Biochemistry.

Collection and Identification of *Moringa oleifera* Leaves: The fresh leaves of *Moringa oleifera* were collected in the month of June, 2022 from Churachandpur. The plant was identified and authenticated by Life Science Department, Manipur University (Mrs. Yumkham Sanatombi Devi, guest faculty). A plant sample was deposited at the laboratory herbarium and allocated with voucher no. MUMP001325.

Preparation of Plant Extract: The ethyl acetate extract of leaves of *Moringa oleifera* was prepared by the method as described by Milon Mondal *et al*¹³. Freshly collected leaves of *M. oleifera* were thoroughly washed with cold water and dried in shade on a table at the room temperature. The dried leaves were finally powdered with the help of electric mixture grinder. 50 grams of the powder of *Moringa oleifera* leaves was extracted with 500 ml of ethyl acetate by using Soxhlet apparatus for 16 hours. The crude extract obtained was filtered using Whatman paper and filtrate was evaporated. The extract was collected in sterile glass petridish and

finally stored in air tight glass containers. In this way, the procedure was repeated several times to yield 20 gm of the extract and preserved in sterile container at 40C, which was used as test material throughout the study.

The Experimental Animals Used for the Study: 30 (Thirty) healthy albino rats of either sex weighing 150-200 grams were recruited from the animal house of JNIMS, Porompat, Imphal and housed in the departmental polypropylene cages for 10 days for acclimatization in the laboratory atmosphere. The rats were fed with standard laboratory diet and water *ad libitum*. 12 hours dark light cycle was maintained. The animals were fasted for 18 hrs prior to the experiment and care taken to avoid any coprophagy.

Acute Oral Toxicity Studies¹⁴: Acute toxicity study for the test drug i.e. ethyl acetate extract of *Moringa oleifera* leaves (EAELMO) was carried out using OECD/OCED Guidelines 425. The extract was found to be safe and no mortality was observed up to a dose of 2000 mg/kg body weight per oral after 14 days.

Experimental Design: The experiment was carried out for the period of 7 days. For this purpose, thirty healthy albino rats of either sex and weighing approximately 150-200 gm were used. Animals were weighed, recorded numbered and randomly divided into 6 groups of 5 animal each as follows:

Groups	Treatment
Group I (Normal control)	distilled water (per orally)
Group II (Positive control)	Distilled water (per orally)+D-Gal N (i. p.)
Group III (Standard drug)	Silymarin-100mg/kg body weight per orally)+D- Gal N (i. p.)
Groups IV (Treatment group)	EAELMO -150 mg/kg bodyweight (per orally) + D-Gal N (i. p.)
Group V (Treatment group)	EAELMO- 200mg/kg body weight (per orally) + D-Gal N (i. p.)
Group VI (Treatment group)	EAELMO- 250mg/kg body weight (per orally) + D-Gal N (i. p.)

EAELMO – Ethyl acetate extract of leaves of *Moringa oleifera*.

- All these protocols were continued for 7 days.
- On the final day of the treatment, animals of groups II–VI received a single dose of D-GalN intraperitoneally at 300 mg/kg body weight.

On the 8th day, all the animals were taken group wise and the rats from each group were anaesthetized using ether. Blood collected from each of them for assessing various biochemical parameters - (AST, ALT, ALP, LDH, γ GT, total and direct bilirubin) using SYS 200 Pro autoanalyzer. After collection of blood, the animals were sacrificed by cervical dislocation to collect the liver tissue for histopathological examination.

RESULTS: The values were expressed as the Mean $\pm$ SEM. The statistical significance was analyzed by using one way ANOVA, followed by Tukey- Kramer multiple comparisons test to compare between the different groups. The significance in the test was expressed by F- ratio and p- values of < 0.05 was considered significant.

TABLE 1: SERUM LEVEL OF ALT, AST, ALP, LDH IN ALL THE GROUPS (MEAN $\pm$ SEM)

Groups		ALT	AST	ALP	LDH
I		64.4 $\pm$ 8.56	157.6 $\pm$ 14.0	62.4 $\pm$ 3.61	170.9 $\pm$ 15.9
II		778.8 $\pm$ 41.2 [#]	936.6 $\pm$ 45.0 [#]	304 $\pm$ 22.8 [#]	908 $\pm$ 60 [#]
III		93.9 $\pm$ 6.5 [*]	188.4 $\pm$ 7.8 [*]	80 $\pm$ 3.1 [*]	471.2 $\pm$ 17 [*]
IV		330.8 $\pm$ 12.5 ^{*@}	445.4 $\pm$ 12.4 ^{*@}	275.6 $\pm$ 22.4 [@]	618 $\pm$ 7.61 [*]
V		181.4 $\pm$ 6.8 ^{*@}	283.6 $\pm$ 16.0 [*]	145.2 $\pm$ 4.61 ^{*@}	478 $\pm$ 26.5 [*]
VI		97.4 $\pm$ 6.7 [*]	248.6 $\pm$ 11.0 [*]	81 $\pm$ 6.64 [*]	340 $\pm$ 18.5 [*]
One way ANOVA	F	216.29	180.26	60.90	71.57
	df	5,24	5,24	5,24	5,24
	P	<0.05	<0.05	<0.05	<0.05

[#]P<0.05 when compared to the normal control group (Group I), ^{*}P < 0.05 when compared to the positive control group (Group II), [@]P < 0.05 when compared to the standard group (Group III).

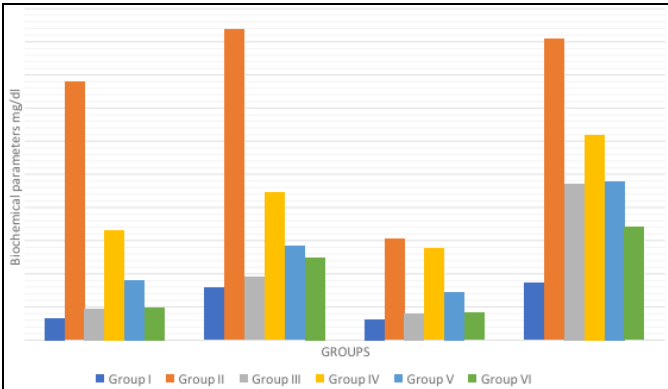


FIG. 1: BAR DIAGRAM SHOWING SERUM LEVELS OF ALT, AST, ALP, LDH IN ALL THE GROUPS. EACH VALUE IS EXPRESSED AS MEAN $\pm$ SEM

Administration of D-GalN in Group II (positive control) resulted in significant elevation ($p < 0.05$) of serum hepatic enzymes as compared to the normal control group. The levels are recorded as alanine aminotransferase ALT (778.8 $\pm$ 41.2), aspartate aminotransferase AST (936.6 $\pm$ 45.0), alkaline phosphatase ALP (304 $\pm$ 22.8) and lactate dehydrogenase LDH (908 $\pm$ 60). In Group III, which was pretreated with silymarin (100mg/kg) followed by D-GalN, when compared with group II showed a significant decrease ($p < 0.05$) in the ALT (93.9

$\pm$ 6.5), AST (188.4 $\pm$ 7.8), ALP (80 $\pm$ 3.1), LDH (471.2 $\pm$ 17) serum levels. In Group IV, animals pretreated with EAELMO at a dose of 150 mg/kg causes a significant reduction ($p < 0.05$) in the levels of these enzymes ALT (330.8 $\pm$ 12.5), AST (445.4 $\pm$ 12), LDH (618 $\pm$ 7.61) as compared to Group II but when compare to group III, significant difference was observed only in serum ALT, AST and ALP levels. In Group V, EAELMO given at a dose of 200 mg/kg showed a significant reduction ($p < 0.05$) in the levels of ALT (181.4 $\pm$ 6.8), AST (283.6 $\pm$ 16.0), ALP (145.2 $\pm$ 4.61), LDH (478 $\pm$ 26.5) as compared to group II but when compare to group III, significant difference was found only in in ALT and ALP levels. Group VI, which was pretreated with EAELMO at a dose of 250 mg/kg showed a significant reduction ($p < 0.05$) in elevated levels of ALT (97.4 $\pm$ 6.7), AST (248.6 $\pm$ 11.0), ALP (81 $\pm$ 6.64), LDH (340 $\pm$ 18.5) as compared to group II. Overall, the results indicate a clear dose – dependent hepatoprotective effect of EAELMO, evidenced by progressive reduction in the serum levels of ALT, AST, ALP and LDH with increasing extract dosage.

TABLE 2: SERUM LEVEL OF TOTAL AND DIRECT BILIRUBIN, GGT IN ALL GROUPS (MEAN $\pm$ SEM)

Groups		Total Bilirubin	Direct Bilirubin	GGT
I		0.44 $\pm$ 0.07	0.20 $\pm$ 0.02	2.9 $\pm$ 0.17
II		1.32 $\pm$ 0.19 [#]	0.84 $\pm$ 0.05 [#]	9.8 $\pm$ 0.3 [#]

III		0.45±0.19*	0.24±0.03*	3.7±0.2*
IV		0.67±0.02*	0.53±0.02*@	5.9±0.5*@
V		0.50±0.03*	0.43±0.02*@	5.2±0.4*@
VI		0.41±0.03*	0.22±0.01*	3.1±0.2*
One Way	F	17.39	69.41	59.83
ANOVA	dF	5,24	5,24	5,24
	P	<0.05	<0.05	<0.05

#P < 0.05, when compared to the normal control group (Group I), *P < 0.05, when compared to the positive control group (Group II), @P < 0.05 when compared to the standard group (Group III).

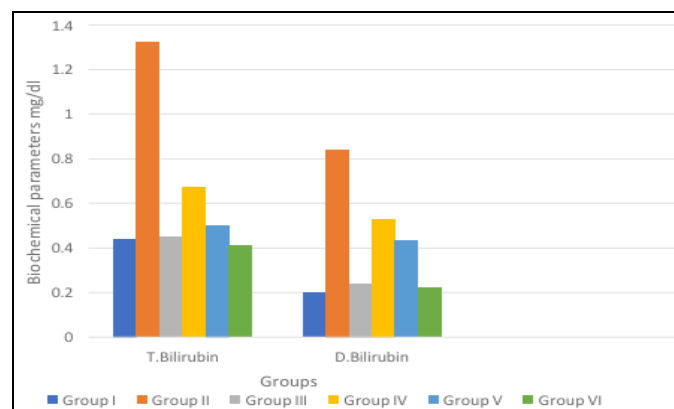


FIG. 2: BAR DIAGRAM SHOWING SERUM LEVELS OF T. BILIRUBIN, D. BILIRUBIN IN ALL GROUPS. EACH VALUE IS EXPRESSED AS MEAN ± SEM

Similarly, the total bilirubin levels and direct bilirubin levels were markedly ($p < 0.05$) increased in D-Gal N induced rats (Group II) with values of 1.32 ± 0.19 and 0.84 ± 0.05 respectively as compared with the control group. Group III, treatment with silymarin significantly reduced both the total bilirubin (0.45 ± 0.19) and direct bilirubin (0.24 ± 0.03) levels than D-Gal N treated rats. In Group IV, which was pretreated with EAELMO at a dose of 150 mg/kg causes a significant reduction ($p < 0.05$) in the level of serum total and direct bilirubin as compared to Group II and had significant difference in direct bilirubin level as compared to group III. In Group V, EAELMO pretreatment at a dose of 200 mg/kg causes a significant reduction T. Bilirubin D. Bilirubin Groups Group I Group II Group III Group IV Group V Group VI Biochemical parameters mg/dl ($p < 0.05$) in the level of serum total and direct bilirubin as compared to Group II and had significant difference in direct bilirubin level as compared to group III. Likewise, in group VI, EAELMO pretreatment at a dose of 250 mg/kg, the total and direct bilirubin levels were significantly reduced to 0.41 ± 0.03 and 0.22 ± 0.01 respectively when compared to group II. The elevated enzyme levels were significantly reduced ($p < 0.05$) in the EAELMO treated group in a dose - dependent

fashion as compared to Group II (Positive control). Administration of D-GalN significantly ($p < 0.05$) increased the level of GGT (9.8 ± 0.3) in group II, whereas administration of D-GalN in rats pretreated with EAELMO (150, 200 and 250 mg/kg) significantly reduced GGT levels in a dose-dependent manner with values of 5.9 ± 0.5 , 5.2 ± 0.4 , 3.1 ± 0.2 respectively as compared to group II. When compared with group III, significant difference was shown in group IV and V.

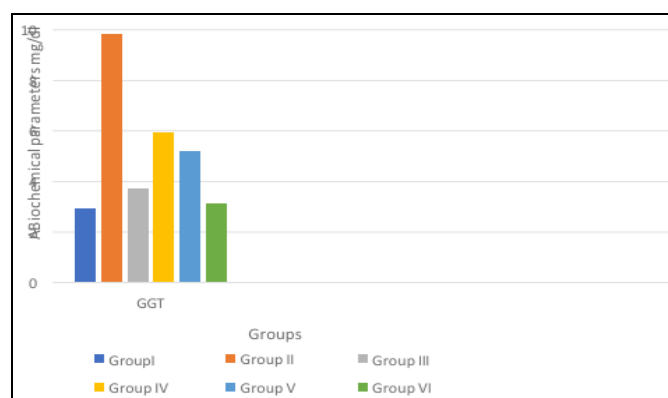


FIG. 3: BAR DIAGRAM SHOWING SERUM LEVELS OF GGT IN ALL GROUPS OF D-GALN INDUCED HEPATOTOXICITY IN ALBINO RATS. EACH VALUE IS EXPRESSED AS MEAN ± SEM

Histopathological Findings:

Gross Features: In normal control group, the liver was unremarkable as shown in **Fig. 4** whereas in D-galactosamine induced rats, there was mild hepatomegaly with congested external surface **Fig. 5**.

Microscopic Findings: In normal control group (Group I), histology of the liver exhibited normal lobular architecture and cellular structure **Fig. 6A**. Whereas in D-GalN group (Group II) showed portal tract inflammation and cell necrosis in wide area and severe inflammatory cell infiltration **Fig 6B**. The silymarin (100mg/kg dose) treated group (Group III) showed marked reduction of inflammation, absence of necrosis and preservation of all the histopathological features **Fig. 6C**.

Moderate tissue necrosis and inflammation were observed in group IV, where animals were pretreated with 150 mg/kg EAELMO followed by D-GalN **Fig. 6D**. Group V and VI, where animals were pretreated with 200mg/kg and 250 mg/kg respectively followed by D- GalN showed an

improvement in the pathological features with mild inflammation **Fig. 6E, 6F**. All these findings indicate the EAELMO have hepatoprotective role if pretreatment of the extract was administered to rats before D-GalN injection.



FIG. 4: GROSS PICTURE: NORMAL LIVER OF AN ALBINO RAT



FIG. 5: GROSS PICTURE: LIVER OF A D-GALACTOSAMINE INDUCED ALBINO RAT WHICH SHOWS MILD HEPATIC ENLARGEMENT AND CONGESTION OF EXTERNAL SURFACE

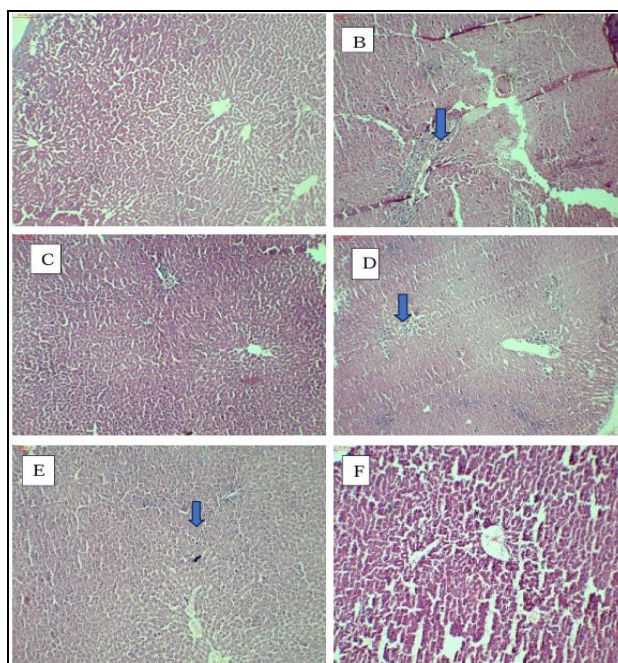


FIG. 6: HISTOPATHOLOGY OF RAT LIVER, H & E; (A) NORMAL CONTROL, (B) POSITIVE CONTROL: DENSE PORTAL TRACT INFLAMMATION WITH SURROUNDING PARENCHYMAL NECROSIS (C) STANDARD DRUG: D- GALN (300MG/KG B. W. I. P) + SILYMARIN (100MG/KG B. W.) SHOWED MARKED REDUCTION OF INFLAMMATION WITH ABSENCE OF NECROSIS AND PRESERVATION OF LOBULAR ARCHITECTURE (D), (E), (F) PRETREATED WITH EAELMO 150, 200, 250 MG/KG B.W., SUBSEQUENTLY + D- GALN (300MG/KG B. W. I. P) SHOWED IMPROVEMENT WITH PRESENCE OF MILD INFLAMMATION

DISCUSSION: The liver performs various vital physiological functions in the body and hepatic impairment resulting from disease or injury can lead to serious illness. Since ancient times medicinal plants have been employed in the treatment of hepatic disease. In previous studies, *Moringa oleifera* was found to have potent antioxidant, anticancer, antihypertensive, hepatoprotective, and nutritional effects⁴.

However, limited data are available regarding the hepatoprotective effect of EAELMO against D-GalN induced hepatotoxicity in experimental animal models. D-galactosamine is a well-established hepatotoxin which induces a diffuse type of liver injury closely resembling human viral hepatitis. A single injection with D-galactosamine can decrease the uracil nucleotides in the liver and heart. Galactosamine markedly depletes hepatic UDP glucuronic acid (UDP-GA) whereas extrahepatic UDP-GA is minimally affected. This suggests that galactosamine predominantly inhibits hepatic glucuronidation. It disrupts the synthesis of essential uridylate nucleotides resulting in organelle injury. Depletion of these nucleotides ultimately impairs the synthesis of protein and glycoprotein, which leads to progressive damage of cellular membranes resulting in a change in permeability of the cellular membrane which leads to enzyme leakage from the cells¹⁵.

Liver damage induced by D-GalN generally reflects disturbances of liver cell metabolism which leads to characteristic changes in the serum enzyme activities. The increased levels of AST, ALT, ALP, LDH and γ GT in this study may be resulted due to liver cell destruction or changes in the membrane permeability indicating the severity of hepatocellular damage induced by D-GalN. The rise in ALT and AST levels is indicative of hepatocellular damage. An increase in ALP reflects the pathological alteration in biliary flow. Increase LDH levels reflects a non-specific alteration in the plasma membrane integrity and/or permeability as a response to D-GalN. γ GT is an enzyme embedded in the hepatocyte plasma membrane, mainly in the canalicular domain and increase in its level indicates damage to the cell and thus injury to the liver. Samantha Beck *et al*¹⁶ evaluated that pretreatment with *Tridax procumbens* extract attenuated the increased activities of these enzymes

in serum caused by D-GalN. Recovery towards normalisation suggests that *Tridax procumbens* extract causes parenchymal cell regeneration in liver, thus protecting membrane fragility, thereby, decreasing enzyme leakage. Evidence of parenchymal cell regeneration in the groups treated with the extract as shown in the histopathological section could be one of the mechanisms for hepatoprotective effect of *Moringa oleifera* leaves extract in this study any abnormal elevation of serum bilirubin levels indicates hepatobiliary disease and liver injuries caused by toxicants. Previous reports have shown that D-GalN induced hepatitis is characterised by increased levels of bilirubin in serum. The extract mediated suppression of the increased bilirubin level suggests the possibility of the extract being able to stabilise biliary dysfunction¹³.

Pre-treatment with EAELMO at graded doses (150, 200 and 250 mg/kg) prior to the D- GalN induced hepatic damage prevents increase in enzyme levels and bilirubin levels. The 250mg/kg dose produced a maximum reduction of serum enzymes levels than the other doses, shows the dose response action of the extract against D-GalN induced hepatotoxicity. The protective action of ethyl acetate extract of leaves of *Moringa oleifera* is reliable as evidenced by the reversal of the altered values following administration probably by promoting regeneration of hepatocytes that restore integrity.

Silymarin, being a standard drug significantly reduced the serum enzyme and bilirubin levels as compared to the D-GalN treated group indicating the protective effect of the drug against the liver cell injury. The histopathological findings of liver tissue basically supported the result obtained from biochemical parameters of the study. Focal necrosis and inflammations induced by D- GalN was prevented by treatment with EAELMO at 250, 200 and 150 mg/kg doses. Phenolics, flavonoids, and tannins, largely found in medicinal plants, act as potent antioxidants and exhibit hepatoprotective activity by inhibiting the generation of these free radicals by donating hydrogen atoms or electrons¹⁷. Flavonoids such as quercetin, genistein, apigenin, kaempferol, and epigallocatechin 3-gallate modulate the expression and activation of a cytokine such as interleukin-1beta (IL-1 β), tumor

necrosis factor-alpha (TNF- α), IL-6 and IL-8, regulate the gene expression of many pro-inflammatory molecules such as nuclear factor kappa-light chain enhancer of activated B cells (NF- κ B)¹⁸. Ashutosh Pareek *et al* claimed that ethyl acetate extract of leaves of *Moringa oleifera* contain numerous bioactive constituents like flavonoids (quercetin, kaempferol, iso- quercetin, rhamnetin *etc.*), phenolic acid (coumaric acid), phenol (ferulic acid, gallic acid), polyphenol (ellagic acid)⁴. The possible mechanism of hepatoprotective activity may be due to the protective action of the natural antioxidants present in the ethyl acetate extract of leaves of *Moringa oleifera* and parenchymal cell regeneration as depicted by the histopathological findings. Thus, the findings of current study strongly suggest that hepatoprotective activity of EAELMO is mediated through its anti-oxidant properties and its ability to promote hepatocyte regeneration, as evidenced by both biochemically and histologically.

CONCLUSION: The present study demonstrated the significant hepatoprotective activity of ethyl acetate extract of *Moringa oleifera* leaves against D-GalN induced hepatic injury in rats. The protective effect is likely attributed to the presence of bio-active constituents like carotenoids, polyphenols, alkaloids, flavonoids, glycosides, terpenoids, tannins, saponins, isothiocyanates. Future research involving purified bioactive constituents may further elucidate its potential in hepatoprotection clinically.

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Declarations:

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Ethical Approval: The study was approved by Institutional Animal Ethics Committee, RIMS.

CONFLICTS OF INTEREST: None declared.

REFERENCES:

1. Devvarbhavi H, Asrani SK, Arab JP, Nartey YA, Pose E and Kamat PS: Global burden of liver disease. *J Hepatol* 2023; 79: 516–537.
2. Falke MM, Bonde KP, Thakur A, Daud ST, Kumar A and Saraswati: Hepatotoxicity: A detailed review of its types, mechanisms, drug interactions, and hepatoprotective interventions. *IJIRT* 2025; 11(9): 2349-6002.
3. Althuriwia HN, Altharawib A, Alharthya KM, Albaqamia FF, Alzareac SI and Al-Abbasid FA: Butin prevent liver damage triggered by D-galactosamine *via* regulating lipid peroxidation and proinflammatory cytokines in rodents. *J King Saud Univ Sci* 2023; 35: 102934.
4. Pareek A, Pant M, Gupta MM, Kashania P, Ratan Y and Pareek A: *Moringa oleifera*: An updated comprehensive review of its pharmacological activities, ethnomedicinal, phytopharmaceutical formulation, clinical, phytochemical, and toxicological aspects. *Int J Mol Sci* 2023; 24: 2098.
5. Jimenez-Arellanes MA, Gutierrez-Rebolledo GA, Meckes-Fischer M and Rosalba L: Medical plant extracts and natural compounds with a hepatoprotective effect against damage caused by antitubercular drugs: A review. *Asian Pac J Trop Med* 2016; 9(12): 1141–1149.
6. Fathy SM, Mohammed S, Mahmoud: *Moringa oleifera* Lam. leaf extract mitigates carbon tetrachloride-mediated hepatic inflammation and apoptosis *via* targeting oxidative stress and toll-like receptor 4/nuclear factor kappa B pathway in mice. *Food Sci and Human Wellness* 2021; 10(3): 383-391.
7. Altaee R, Kadhim FO and Al-Saedi SR: Assessment of the hepatoprotective effect of *Moringa oleifera* leaves extract (MOLE) on paracetamol-induced hepatotoxicity in Albino Wistar rats. *J Kerbala for Agric Sci* 2023; 4(10).
8. Hussein S, Ben Bacha A, Alonazi M, Alwaili MA, Mobasher MA and Alburae NA: *Urtica pilulifera* leaves extract mitigates cadmium induced hepatotoxicity *via* modulation of antioxidants, inflammatory markers and Nrf-2 signaling in mice. *Front Mol Biosci* 2024; 11:
9. Stephen AO, Sokolayemji AC and Adewumi AM: *Moringa oleifera* based diet protects against nickel induced hepatotoxicity in rats. *J Biomed Res* 2017; 31(4): 350-357.
10. Khalil MA, Eliwa A, El-Shiekh A, Al-Mokaddem K, M Hassan M and Tawfek M: Ashwagandha (*Withania somnifera*) root extract attenuates hepatic and cognitive deficits in thioacetamide-induced rat model of hepatic encephalopathy *via* induction of Nrf2/HO-1 and mitigation of NF- κ B/MAPK signalling pathways. *J Ethnopharmacol* 2021; 277: 114141.
11. Kim CG, Chang SN, Park SM, Hwang BS, Kang SA and Kim KS: *Moringa oleifera* mitigates ethanol-induced oxidative stress, fatty degeneration and hepatic steatosis by promoting Nrf2 in mice. *Phytomedicine* 2022; 100: 154037.
12. Oinam J, Raleng I, Meitankeishangbam P, Raj Kumari B and Laishram S: Study on hepatoprotective activity of ethanol extract of *Zanthoxylum armatum* dc (mukthruhi) leaves in experimental animal. *IJPSR* 2017; 8(7): 3025-3029.
13. Milon M, Hossain M, Hasan R, Towhidul IT, Islam F and Choudhuri MSK: Hepatoprotective and antioxidant capacity of *Mallotus repandus* ethyl acetate stem extract against D-galactosamine-induced hepatotoxicity in Rats. *ACS Omega* 2020; 5(12): 6523- 6531.
14. OECD guidelines for testing of chemicals (acute oral toxicity-up and down procedure). [cited 2022 Mar 20]. Available from <http://www.oecd.org>.
15. Banu S, Bhaskar B and Balasekar P: Hepatoprotective and antioxidant activity of *Leucas aspera* against d-galactosamine induced liver damage in rats. *Pharm Biol* 2012; 50(12): 1592–1595.

16. Beck S, Mathison H, Todorov T, Juárez EAC and Kopp OR: A review of medicinal uses and pharmacological activities of *Tridax procumbens* (L.). J Plant Studies 2018; 7(1): 19.
17. Wahed TB, Mondal M, Rahman MA, Hossen MS, Bhoumik NC and Saha S: Protective role of *Syzygium cymosum* leaf extract against carbofuran-induced

- hematological and hepatic toxicities. Chem Res Toxicol 2019; 32: 1619–1629.
18. Al-Khayri JM, Sahana GR, Nagella P, Joseph BV, Alessa FM and Al-Mssallem MQ: Flavonoids as potential anti-inflammatory molecules: A review. Molecules 2022; 27(9): 2901.

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