



HISTOLOGICAL EFFECTS OF ETHANOL LEAF EXTRACT OF *Mangifera indica* ON THE LIVER OF ACETAMINOPHEN-INDUCED HEPATOTOXICITY IN ADULT MALE WISTAR RATS

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ABSTRACT

Introduction: Acetaminophen overdose is a major cause of drug-induced hepatotoxicity, the leaves of *Mangifera indica* have attracted considerable scientific interest because they are rich in bioactive phytochemicals like Mangiferin, making it a promising candidate for the prevention and treatment of hepatotoxicity. **Aim:** This study investigated the histological effects of ethanol leaf extract of *Mangifera indica* on acetaminophen-induced hepatotoxicity in adult male Wistar rats. **Methodology:** Forty-five rats were divided into five groups (I-V) of 9 rats each. Group I is the control, Group II acetaminophen-induced untreated, Group III acetaminophen-induced treated with low-dose extract, Group IV acetaminophen-induced treated with high-dose extract, and Group V acetaminophen-induced treated with silymarin. **Results:** Biochemical analysis revealed a significant increase in liver enzymes and a statistical reduction in the plasma protein levels. Histological analysis revealed dose-dependent hepatoprotection by *M. indica*, with reduced necrosis, preserved hepatic cords and attenuated inflammatory infiltration in the high dose and silymarin groups. **Conclusion:** It can be concluded from this study that ethanol leaf extract of *Mangifera indica* significantly ameliorated hepatic tissue damage in acetaminophen-induced hepatotoxicity in Wistar rats, with biochemical evidence of hepatoprotection.

KEYWORDS: Hepatotoxicity, Wistar rats, Acetaminophen, Ethanol leaf extract, *Mangifera indica*.

The liver is the largest internal organ and one of the most metabolically active organs in the body, playing a central role in maintaining physiological homeostasis. It performs numerous vital functions, including the metabolism of carbohydrates, lipids, and proteins; detoxification of endogenous and exogenous substances; synthesis of plasma proteins and clotting factors; bile production; storage of glycogen, vitamins, and minerals; and regulation of immune responses (Hall & Hall, 2021; Kumar et al., 2021). Owing to its strategic position within the portal circulation and its involvement in the biotransformation of xenobiotics, the liver is continuously exposed to toxic substances and is therefore highly susceptible to injury. Consequently, liver diseases remain a major global public health concern, contributing substantially to morbidity and mortality and imposing a significant socioeconomic burden worldwide (Sharma, 2014; Gan et al, 2025).

Hepatotoxicity refers to liver injury resulting from exposure to chemical agents, therapeutic drugs, environmental toxins, herbal products, alcohol, or infectious agents that impair the structural and functional integrity of hepatocytes. It remains a significant clinical challenge because the liver is the primary organ responsible for the metabolism and detoxification of xenobiotics, rendering it particularly vulnerable to toxic insults (Kumar et al., 2021). The pathogenesis of hepatotoxicity involves complex mechanisms, including excessive generation of reactive oxygen species, lipid peroxidation, mitochondrial dysfunction, inflammatory responses, and depletion of endogenous antioxidant defenses, all of which culminate in hepatocellular degeneration, apoptosis, or necrosis (Allameh et al., 2023). Progressive liver injury may ultimately compromise hepatic function, leading to fibrosis,

cirrhosis, liver failure, or even death if left untreated (Skat-Rørdam et al., 2025).

Acetaminophen (paracetamol) is one of the most widely used analgesic and antipyretic drugs because of its effectiveness and favorable safety profile at therapeutic doses. However, overdose or prolonged use can result in severe hepatotoxicity and remains one of the leading causes of acute liver failure worldwide (Yoon et al., 2016; Ramachandran & Jaeschke, 2018). Under normal physiological conditions, acetaminophen is predominantly metabolized in the liver through glucuronidation and sulfation pathways. A small fraction is oxidized by cytochrome P450 enzymes, particularly CYP2E1, to form the highly reactive metabolite N-acetyl-p-benzoquinone imine (NAPQI). Under therapeutic conditions, NAPQI is rapidly detoxified through conjugation with reduced glutathione. However, following acetaminophen overdose, excessive NAPQI formation depletes hepatic glutathione stores, resulting in oxidative stress, mitochondrial dysfunction, inflammatory responses, and centrilobular hepatocellular necrosis, which collectively impair liver structure and function (Jaeschke et al., 2023).

The structural consequences of acetaminophen-induced hepatotoxicity are characterized by marked histomorphological alterations that reflect the severity of hepatocellular injury. These changes commonly include centrilobular necrosis, hepatocellular degeneration, cytoplasmic vacuolation, sinusoidal congestion, inflammatory cell infiltration, ballooning degeneration, and nuclear changes such as pyknosis, karyorrhexis, and karyolysis (Kumar et al., 2021; Yoon et al., 2016). Persistent oxidative stress and inflammation further disrupt the normal hepatic architecture, impair cellular regeneration, and compromise liver function (Kopylchuk et al., 2026).

The management of acetaminophen-induced hepatotoxicity primarily involves the administration of N-acetylcysteine (NAC), which replenishes hepatic glutathione stores, enhances detoxification of the reactive metabolite N-acetyl-p-benzoquinone imine (NAPQI), and reduces oxidative liver injury. Although NAC is highly effective when administered within the early stages of acetaminophen overdose, its therapeutic efficacy declines significantly with delayed treatment, and it may not completely prevent hepatic injury in severe cases (Ramachandran & Jaeschke, 2018; Chiew et al., 2018). These limitations have stimulated growing interest in identifying natural products with antioxidant, anti-inflammatory, and hepatoprotective properties that may complement existing therapies and improve liver recovery following toxic injury.

Mangifera indica L. (Anacardiaceae), commonly known as the mango tree, is a perennial tropical plant widely cultivated throughout Asia, Africa, and other tropical and subtropical regions for its nutritional and medicinal

value. Beyond its edible fruits, various parts of the plant, including the leaves, bark, seeds, roots, and stem bark, have long been utilized in traditional medicine for the management of numerous ailments such as diabetes mellitus, hypertension, diarrhea, microbial infections, inflammation, and liver disorders (Ediriweera et al., 2017; Shah et al., 2010). In particular, the leaves of *M. indica* have attracted considerable scientific interest because they are rich in bioactive phytochemicals with diverse pharmacological properties, making them promising candidates for the prevention and treatment of oxidative stress-related diseases.

The hepatoprotective potential of *Mangifera indica* leaves is largely attributed to their rich phytochemical composition, which includes mangiferin, quercetin, gallic acid, catechins, flavonoids, phenolic compounds, tannins, and saponins. These bioactive constituents exhibit potent antioxidant, anti-inflammatory, anti-apoptotic, and free radical-scavenging activities that protect hepatocytes against oxidative and inflammatory damage (Shah et al., 2010; Nadella & Kumar, 2016). Experimental studies have demonstrated that these phytochemicals enhance endogenous antioxidant defense systems, inhibit lipid peroxidation, suppress pro-inflammatory cytokine production, stabilize cellular membranes, and promote tissue repair. Consequently, *M. indica* leaf extract has emerged as a promising natural therapeutic agent for mitigating oxidative stress-mediated liver injury and preserving hepatic structural integrity.

Several experimental studies have demonstrated the hepatoprotective effects of *Mangifera indica* leaf extract in models of chemically induced liver injury. These studies have reported improvements in liver function biomarkers, attenuation of oxidative stress and inflammatory responses, and preservation of hepatic architecture following treatment with the extract. The observed protective effects have largely been attributed to the plant's abundant antioxidant and anti-inflammatory phytochemicals, particularly mangiferin and other polyphenolic compounds (Shah et al., 2010; Rasool et al., 2011). However, despite these promising findings, there is limited information on the histomorphological influence of ethanol leaf extract of *Mangifera indica* in acetaminophen-induced hepatotoxicity. Further investigation is therefore warranted to elucidate its potential in preserving or restoring normal hepatic architecture following acetaminophen-induced liver injury.

Given that oxidative stress and inflammation are central mechanisms underlying acetaminophen-induced liver injury, therapeutic agents with antioxidant and cytoprotective properties, such as *Mangifera indica*, may help preserve hepatic architecture and function. Although *M. indica* has demonstrated promising hepatoprotective, antioxidant, and anti-inflammatory activities, its influence on the histomorphological alterations

associated with acetaminophen-induced hepatotoxicity remains inadequately characterized. Therefore, this study was designed to evaluate the histomorphological influence of ethanol leaf extract of *Mangifera indica* on the liver of acetaminophen-induced hepatotoxicity in adult male Wistar rats.

MATERIALS AND METHODS

Experimental Animals

Forty-five adult male Wistar rats weighing between 180–220 g were procured from Animal house of the college of health Sciences, Obafemi Awolowo University, Ile-Ife, Osun-State. The rats were housed in plastic cages under standard laboratory conditions, with natural light/dark cycle. Animals were acclimatized for two weeks prior to the commencement of the experiment and provided with standard rat chow and free access to water. All experimental procedures were conducted in accordance with institutional ethical guidelines for animal research and approved by Health Research Ethics Committee (HREC), Institute of Public Health (IPH) Obafemi Awolowo University (OAU) Ile-Ife. The animals were given humane care according to the guidelines of HREC, IPH, OAU.

Plant Material and Extract Preparation.

Fresh leaves of *Mangifera indica* were collected, authenticated by a botanist, and assigned a voucher specimen number deposited at the herbarium of the Department of Botany for reference purpose. The leaves were air-dried at room temperature, pulverized into fine powder, and subjected to Soxhlet extraction using 95% ethanol. The extract was concentrated under reduced pressure using a rotary evaporator and stored at 4 °C until use. The yield was calculated, and doses were prepared by dissolving the extract in distilled water.

Percentage yield (%) = (weight of dried extract/ Weight of dried leaves) X 100.
(Abubakar et al, 2017)

Experimental Design

The rats were randomly divided into five groups (n=9 per group).

Group I (Control): Received only distilled water orally.

Group II (Negative control): Received acetaminophen (750 mg/kg, orally) to induce hepatotoxicity and left

untreated. **Group III (Low-dose extract):** Received acetaminophen (750 mg/kg) followed by *M. indica* ethanol leaf extract (200 mg/kg, orally). **Group IV (High-dose extract):** Received acetaminophen (750 mg/kg) followed by *M. indica* ethanol leaf extract (400 mg/kg, orally). **Group V (Positive control):** Received acetaminophen (750 mg/kg) followed by silymarin (100 mg/kg, orally).

Induction of Hepatotoxicity

Hepatotoxicity was induced by a single oral administration of acetaminophen at 750 mg/kg body weight after overnight fasting. Rats in treatment groups received their respective interventions 2 hours post-acetaminophen administration and continued daily for 7 consecutive days.

Sacrifice and Tissue Collection

At the end of the experimental period, rats were anesthetized with ketamine (50 mg/kg, intraperitoneally) and sacrificed by cervical dislocation. The liver was excised, rinsed in ice-cold saline, blotted dry, and weighed. Representative samples were fixed in 10% neutral buffered formalin for histological analysis.

Histological Processing

Liver tissues were processed using standard paraffin embedding techniques. Sections of 5 µm thickness were cut using a rotary microtome and stained with hematoxylin and eosin (H&E). Slides were examined under light microscopy at magnifications of ×100 and ×400. Histomorphological parameters assessed included hepatocellular necrosis, vacuolation, sinusoidal congestion, inflammatory infiltration, and preservation of hepatic cords.

Statistical Analysis

Data were expressed as mean ± standard deviation (SD). Statistical comparisons between groups were performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test. A p-value < 0.05 was considered statistically significant.

RESULTS

BIOCHEMICAL ANALYSES OF LIVER FUNCTION PARAMETERS FOR ETHAOL LEAF EXTRACT OF *MANGIFERA INDICA*

GROUPS	AST (IU/L)	ALT (IU/L)	ALP (IU/L)	TP (g/L)	ALB(g/L)	GLB (g/L)
NORMAL CONTROL	59.43±5.62 ^b	15.20±5.72 ^b	98.31±8.39 ^b	69.88±5.13 _b	50.28±6.42 _b	19.6±4.09 ^b
ACETAMINOPHEN ONLY	96.68±6.67 ^a	51.80±6.72 ^a	267.50±6.73 _a	27.83±8.11 _a	12.58±3.42 _a	15.25±5.31
ACETAMINOPHEN +LOW DOSE ELEMI	75.96±7.18	40.13±1.63 ^a _b	202.68±4.81 ^a _b	39.07±4.22 _{a b}	24.77±8.92 _{a b}	14.30±5.44 _{a d}
ACETAMINOPHEN +HIGH DOSE ELEMI	69.56±3.62 ^b	24.73±2.12 ^a _b	108.12±2.09 _b	58.72±4.64 _b	39.54±2.67 _b	19.18±2.25
ACETAMINOPHEN + SILYMARIN	63.45±3.22 ^d _b	19.76 ±2.13 _b	103.72±1.01 _b	66.94±4.35 _b	45.89±9.11 _b	21.05±1.23 _b

ALT – Alanine aminotransferase, AST- Aspartate aminotransferase, ALP – Alkaline Phosphatase, TP- Total Protein, ALB- Albumin, GLB- Globulin.

^a- significantly different from the normal control

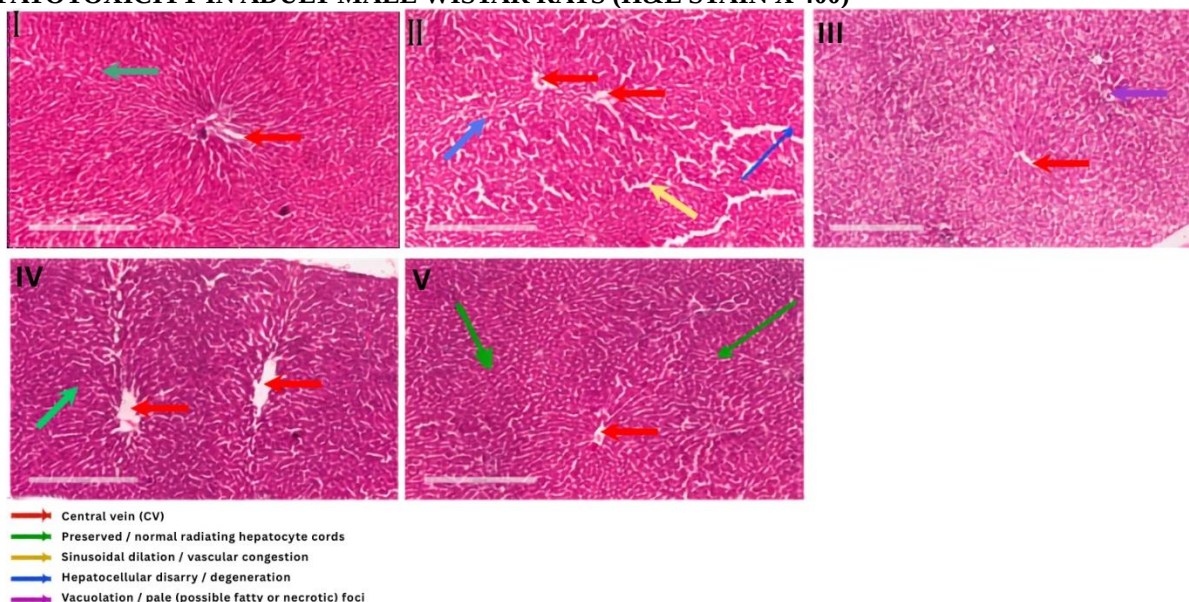
^b- significantly different from the toxic group

^d- significantly different from Silymarin group

In the table above (Table 1). The normal control for liver enzymes (AST, ALT, ALP) and plasma proteins (TP, ALB, GLB) were all within normal ranges, this represents healthy liver function in the normal control, whereas in the Acetaminophen only (Toxic Group) the liver enzymes (AST, ALT, ALP) were generally observed to be markedly elevated when compared with the normal control which indicates severe hepatocellular injury. In group 3 the liver enzymes slightly reduced but not significant compared to toxic group but still higher than normal which indicates partial protection of the liver cell by the ethanol leaf extract of *Mangifera indica* (ELEM1). In group 4 the liver enzymes were shown to be significantly reduced ($p < 0.05$) and close to normal values when compared with the normal control and other test groups, this suggests that high dose extract provided strong hepatoprotection when compared to other test groups II and III. Moreover group 4 significantly reduced the elevated liver enzyme ($p < 0.05$) and restored the values to near-normal serum protein levels when compared to groups II and III, this is in keeping with the work of Ilukho et al., 2022, who stated that extract of *Mangifera indica* possesses hepatoprotective effect, possibly mediated through induction of anti-oxidant enzymes to prevent the occurrence of oxidative stress damage and anti-inflammatory mediators which are being induced by the lethal dose of the hepatotoxicant.

• The plasma proteins were found to be within normal range in the normal control as seen in the table above, there was however significantly reduced plasma proteins especially the total protein and albumin in group 2 animals ($p < 0.05$) while reduction in globulin was noticed to be slightly reduced but not significant when compared with the normal control. Group 3 revealed slightly improved plasma protein which was not significant in the experimental group. The group 4 and 5 plasma proteins were observed to be significantly increased ($p < 0.05$) when compared to the toxic and the low dose group, which brings the plasma protein level to near normal almost similar to the normal control. This implies that acetaminophen caused acute hepatotoxicity with massive liver cell damage and impaired synthetic function, which corroborates the work of Mutiat et al., 2015 who stated in his research that a significant reduction ($p < 0.05$) in total protein and albumin was observed in paracetamol-induced hepatotoxicity compared with the normal control group, while there was significant increase ($p < 0.05$) in the treated groups especially high dose and Silymarin groups. In essence acetaminophen caused severe hepatotoxicity (elevated enzymes, reduced proteins). *Mangifera indica* ethanol leaf extract showed dose-dependent hepatoprotective effects, which mechanism likely involved antioxidant activity, membrane stabilization, and improved protein synthesis.

REPRESENTATIVE PHOTOMICROGRAPHS OF THE HISTOMORPHOLOGICAL EFFECTS OF ETHANOL LEAF EXTRACT OF *Mangifera indica* ON THE LIVER OF ACETAMINOPHEN-INDUCED HEPATOTOXICITY IN ADULT MALE WISTAR RATS (H&E STAIN X 400)



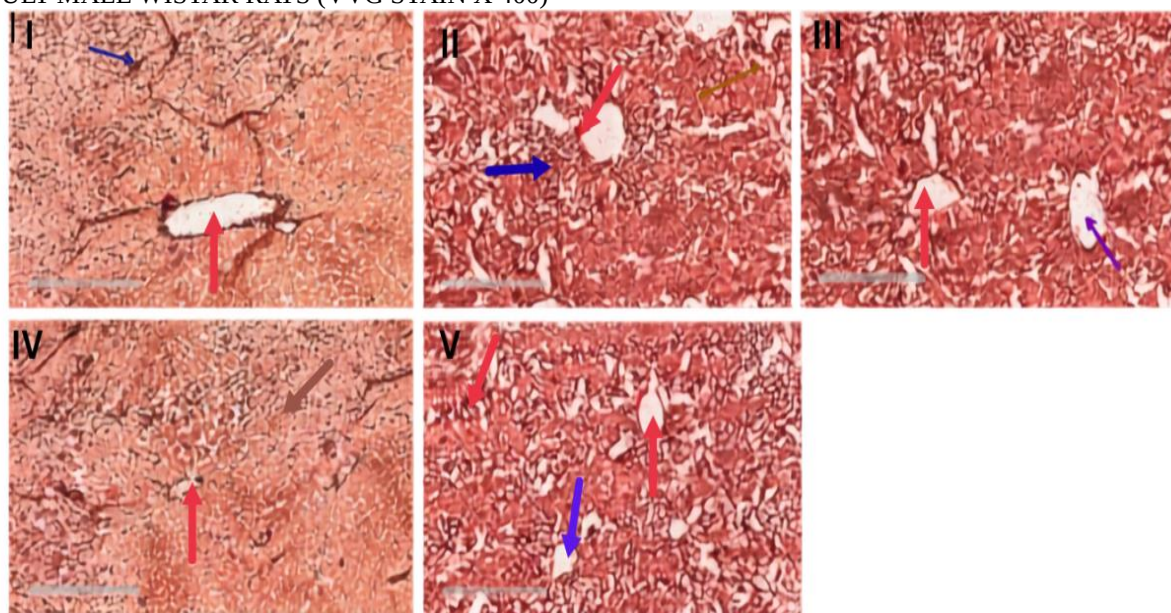
Central Vein (in red arrows) forms the main landmark in hepatic lobules, often dilated or congested in

hepatotoxicity. Hepatocytes (in green arrows) are polygonal liver cells radiating from the central vein.

Normal hepatocytes in treated groups while, there are degenerating hepatocytes (in blue arrow) with ballooning, vacuolation and necrosis, in untreated acetaminophen groups. Sinusoids (yellow arrow) Capillary-like spaces between hepatocyte cords which appear congested or collapsed in toxic group but more open and regular in extract-treated groups. The necrotic areas are the regions of hepatocyte death, showing loss of nuclei and cytoplasmic fragmentation, however there are regenerative changes (in treated groups) as evidenced by hepatocyte mitosis, restoration of sinusoidal architecture, and absence of necrosis in groups IV and V.

Group I (Control) showed normal hepatic architecture, intact hepatocytes, clear sinusoids. Group II (Acetaminophen only) showed severe necrosis, sinusoidal congestion, hepatocyte ballooning. Group III (Low-dose extract + acetaminophen) revealed partial protection, reduced necrosis and minor congestion. Group IV (High-dose extract + acetaminophen) characterized with marked hepatoprotection, near-normal hepatocytes and restored sinusoids. Group V (acetaminophen + Sylimarín): almost normal histology, showing no toxicity from the Sylimarín treated group.

REPRESENTATIVE PHOTOMICROGRAPHS OF THE HISTOMORPHOLOGICAL EFFECTS OF ETHANOL LEAF EXTRACT OF *Mangifera indica* ON THE LIVER OF ACETAMINOPHEN-INDUCED HEPATOTOXICITY IN ADULT MALE WISTAR RATS (VVG STAIN X 400)



Legend:

- Vascular lumen (central vein / vascular channel)
- Elastic fibers (VVG black staining), regular/thin pattern
- Collagen-rich matrix (VVG red-pink staining), denser pattern
- Vacuolated / lucent spaces within vessel lumen

Group I revealed loosely arranged, pale-staining stroma with a well-defined, patent vascular lumen and fine, thin elastic fibers around the vessel wall, collagen background was observed in group I which is consistent with normal control.

Group II showed a denser red-pink (collagen) background surrounding a dilated central lumen with fibrous strands appear thicker and more closely packed in the toxic group when compared with the normal control.

Group III revealed also similar dense collagen-rich matrix to group II, with two vascular spaces and a small vacuolated focus adjacent portal vessel in the low dose *Mangifera indica* group.

Group IV showed moderately dense stroma with a smaller, less distorted vessel and patchier collagen distribution which appears intermediate when compared with groups I and other test groups (II III).

Group V showed dense collagen background similar to groups II and III, with a vacuolated space close to portal vessel.

The ethanol leaf extract of *M. indica* demonstrated dose-dependent hepatoprotection against acetaminophen-induced liver injury, which are consistent with recent findings of Kabbashi *et al.*, 2024 whose research stated that ethanolic *M. indica* seed extract protected against

CCl₄-induced hepatotoxicity via antioxidant mechanisms. These studies support the hypothesis that *M. indica* phytochemicals act through antioxidant defense and stabilization of hepatocyte membranes. The

comparable efficacy of high-dose extract and silymarin highlights its therapeutic potential.

CONCLUSION

Ethanol leaf extract of *Mangifera indica* significantly ameliorates acetaminophen-induced hepatotoxicity in Wistar rats, with histomorphological evidence of hepatoprotection. This reinforces its potential as a natural hepatoprotective agent.

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CONTRIBUTIONS OF AUTHORS:

A.B.E. Initiating the research, writing of the literature, methodology and discussion.

A.O.S. Conceptualizing the work and provision of funds and guidance.

I.M.S. The laboratory bench work and data analyses.

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