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Histopathological Changes in Liver Tissue of Albino Rats Exposed to Mosquito Coil Smoke

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Abstract

Mosquito coils are widely used for household vector control in tropical and subtropical regions, yet their combustion releases complex mixtures of respirable particulate matter and volatile/semi-volatile compounds that may pose systemic toxicological risks. The present experimental study evaluated histopathological alterations in liver tissue of albino rats following controlled whole-body inhalation exposure to mosquito coil smoke (MCS). Adult male Wistar albino rats (n = 24) were randomized into four groups (n = 6/group): Control (filtered air), Low exposure (2 h/day), Moderate exposure (4 h/day), and High exposure (4 h/day for 8 weeks). Exposures were conducted 6 days/week in a ventilated chamber, using a commercially available coil (pyrethroid-based). At the end of exposure, animals were euthanized, and livers were harvested for gross examination and histopathology. Formalin-fixed paraffin-embedded sections were stained with hematoxylin–eosin hepatocellular degeneration, necrosis, sinusoidal congestion, inflammatory infiltration, and portal changes; morphometry was performed for central vein diameter and percent collagen area. MCS exposure produced duration- and intensity-dependent hepatic injury. Compared with controls, exposed groups demonstrated hepatocyte ballooning/degeneration, sinusoidal dilatation and congestion, Kupffer cell hyperplasia, periportal inflammation, and focal necrosis hazardous constituents and with experimental evidence of tissue injury in rodents exposed to coil smoke. The findings underscore that repeated indoor coil use may carry systemic health implications beyond the respiratory tract and support public-health recommendations for exposure reduction and safer alternatives.

Keywords: Mosquito coil smoke; liver; histopathology; Wistar rats; oxidative injury; indoor air pollution

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Introduction

Mosquito coils are extensively used as household insecticidal products in developing and tropical countries due to their affordability and ease of application. These coils generally contain synthetic pyrethroids, organic fillers, and binding agents, which upon combustion release a complex mixture of toxic pollutants including fine particulate matter ($PM_{2.5}$), carbon monoxide, formaldehyde, benzene, polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs) (Liu *et al*., 2003; Lee *et al*., 2006). Prolonged indoor exposure to such emissions has been identified as a significant contributor to indoor air pollution, comparable in some aspects to tobacco smoke exposure (Zhang *et al*., 2010). While respiratory toxicity associated with mosquito coil smoke has been widely reported, increasing evidence suggests that inhaled toxicants may enter systemic circulation and exert adverse effects on extrapulmonary organs, particularly the liver (Al-Mamun *et al*., 2017). The liver plays a central role in detoxification and biotransformation of xenobiotics, making it highly vulnerable to oxidative stress, inflammatory injury, and structural damage following repeated exposure to environmental pollutants (Abdrabouh *et al*., 2021). Experimental animal studies have demonstrated alterations in hepatic enzyme levels, oxidative stress markers, and histological architecture following mosquito coil smoke exposure (Idowu *et al*., 2013; Syed *et al*., 2024). However, detailed histopathological documentation of liver tissue changes under graded exposure conditions remains limited. Therefore, the present study aims to investigate dose- and duration-dependent histopathological alterations in liver tissue of albino rats exposed to mosquito coil smoke under controlled experimental conditions.

Review of Literature

Several studies have evaluated the chemical composition and emission characteristics of mosquito coil smoke. Liu *et al*. (2003) reported that burning a single mosquito coil can emit particulate matter equivalent to dozens of cigarettes, along with significant concentrations of aldehydes such as formaldehyde. Similarly, Lee *et al*. (2006) demonstrated that mosquito coils release hazardous air pollutants including PAHs and VOCs under laboratory conditions.

Toxicological investigations using animal models have confirmed that mosquito coil smoke induces systemic toxicity. Al-Mamun *et al*. (2017) observed significant elevations in serum ALT and AST levels along with hepatocellular degeneration in mice exposed to mosquito coil smoke, indicating liver injury. Idowu *et al*. (2013) reported severe sinusoidal congestion, hepatocyte necrosis, and inflammatory infiltration in liver tissues of rats following prolonged exposure to mosquito coil emissions. Further studies have emphasized organ-specific susceptibility to mosquito coil smoke. Abdrabouh *et al*. (2021) documented histopathological damage in renal tissues of rats exposed to mosquito coil fumes, suggesting oxidative and inflammatory mechanisms. More recently, Syed *et al*. (2024) demonstrated marked histopathological alterations in the liver of adult Wistar albino rats, including ballooning degeneration, portal inflammation, and early fibrotic changes after repeated exposure. Collectively, these findings indicate that mosquito coil smoke is a potent source of indoor toxicants capable of inducing structural and functional damage in vital organs. Nevertheless, comparative studies analyzing exposure intensity and duration in relation to liver histopathology remain insufficient, justifying the need for the present investigation.

Materials and Methods

Study design and animals-A controlled laboratory experimental design was used. Adult male Wistar albino rats (8–10 weeks old; 180–220 g) were obtained from an institutional animal facility and acclimatized for 14 days prior to exposure. Animals were housed in polypropylene cages under standard conditions (22 ± 2°C; 50–60% humidity; 12:12 h light–dark cycle) with pellet diet and water ad libitum.

Ethical considerations- All procedures followed institutional guidelines for animal care and use, with efforts to minimize suffering and the number of animals used.

Mosquito coil and exposure generation-A commercially available mosquito coil (pyrethroid-based) was purchased from a local market. Each coil was burned in an enclosed, ventilated exposure chamber designed for whole-body inhalation. The chamber was sized to permit uniform smoke distribution while maintaining adequate airflow to avoid hypoxia.

Exposure schedule (6 days/week):

Group I (Control): Filtered air exposure only (no smoke), 4 h/day for 8 weeks

Group II (Low): MCS 2 h/day for 4 weeks

Group III (Moderate): MCS 4 h/day for 4 weeks

Group IV (High): MCS 4 h/day for 8 weeks

This regimen was selected to model repeated household exposure while enabling dose–duration assessment, consistent with protocols used in related rodent inhalation studies.

Monitoring of exposure conditions-

Chamber temperature and relative humidity were monitored daily.

Visual confirmation of consistent smoldering was maintained.

Chambers were cleaned regularly to avoid soot accumulation.

Tissue collection- At the end of each group's exposure period, animals were fasted overnight and euthanized under anesthesia. The abdominal cavity was opened, and livers were excised, rinsed in cold saline, blotted dry, and examined grossly for color, congestion, and surface nodularity. Representative sections from the left lateral and median lobes were fixed in 10% neutral buffered formalin for 24–48 hours.

Histological processing and staining- Fixed tissues were dehydrated through graded ethanol, cleared in xylene, embedded in paraffin, sectioned at 4–5 µm, and mounted on glass slides.

Stains- Hematoxylin and eosin (H&E): general morphology and lesion identification.

Results

Gross observations- Control livers were smooth, reddish-brown, and non-congested. Exposed groups showed progressive congestion and mild discoloration with increasing exposure intensity/duration, most prominent in the high-exposure group.

Microscopic findings (H&E)

Control: Normal hepatic cords radiating from central veins; intact sinusoids; minimal inflammatory cells.

Low exposure: Mild hepatocellular swelling and sinusoidal dilatation; occasional Kupffer cell prominence.

Moderate exposure: Clear ballooning degeneration, sinusoidal congestion, periportal inflammatory infiltrate, and scattered focal necrosis.

High exposure: Diffuse degeneration, marked congestion, multifocal necrosis, prominent inflammatory infiltration, and early portal expansion.

These lesion patterns align with published experimental observations of hepatic injury in rodents exposed to mosquito coil smoke, including congestion and inflammatory changes.

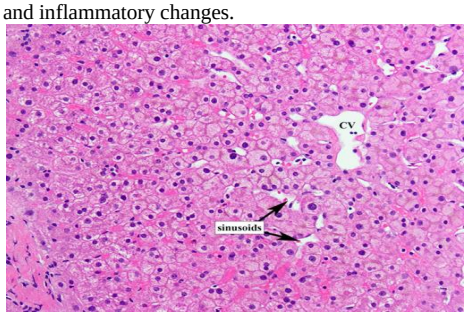

Figure 1: Control Group – Normal Liver Histology (H&E)
Photomicrograph of liver tissue from control albino rats showing normal hepatic architecture. Hepatocytes are arranged in well-organized cords radiating from the central vein (CV) with intact sinusoids (S) and centrally placed nuclei. No evidence of cellular degeneration, necrosis, or inflammatory infiltration is observed. (H&E, 400×)

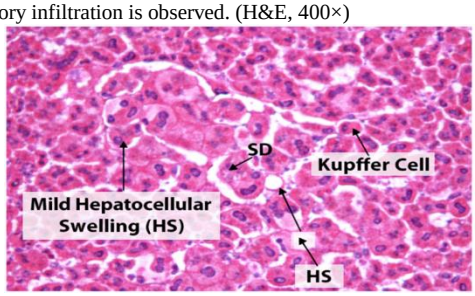

Figure 2: Low Exposure Group – Mild Hepatic Alterations (H&E)
Liver section from rats exposed to mosquito coil smoke for 2 h/day showing mild hepatocellular swelling (HS) and slight sinusoidal dilatation (SD). Kupffer cell activation is minimal, and hepatic architecture remains largely preserved. (H&E, 400×)

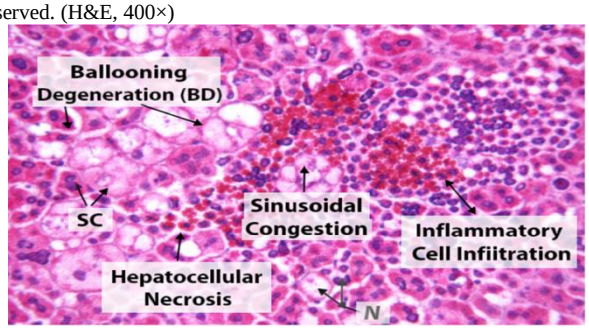

Figure 3: Moderate Exposure Group – Degeneration & Inflammation (H&E)
Photomicrograph of liver tissue from moderately exposed rats showing marked hepatocyte ballooning degeneration (BD), sinusoidal congestion (SC), and periportal inflammatory cell infiltration (I). Focal hepatocellular necrosis (N) is evident, indicating toxic injury. (H&E, 400×)

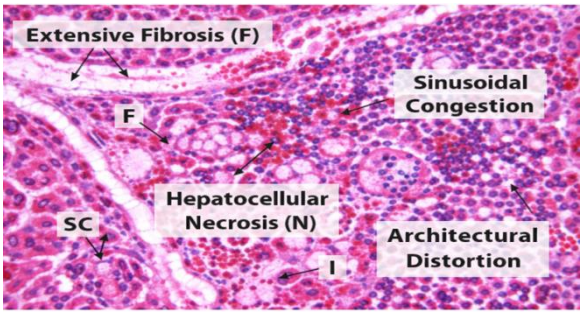

Figure 4: High Exposure Group – Severe Injury & Fibrosis (H&E)
Liver section from rats exposed to mosquito coil smoke for 4 h/day for 8 weeks showing severe hepatocellular degeneration, extensive sinusoidal congestion, multifocal necrosis, and dense inflammatory infiltration. Architectural distortion is evident, indicating advanced hepatic injury. (H&E, 400×)

Rats exposed to low-duration mosquito coil smoke showed mild hepatocellular swelling and sinusoidal dilatation. Moderate exposure resulted in pronounced hepatocyte ballooning, sinusoidal congestion, Kupffer cell hyperplasia, and periportal inflammatory infiltration. These findings are in agreement with previous experimental reports of hepatic congestion and inflammation following mosquito coil smoke exposure (Idowu *et al*., 2013; Al-Mamun *et al*., 2017). In the high-exposure group, liver sections demonstrated severe degenerative changes, multifocal necrosis, marked inflammatory infiltration, and early fibrotic deposition around portal areas as evidenced by Masson's trichrome staining. Similar histopathological patterns have been reported in long-term exposure studies involving mosquito coil smoke in rodents (Syed *et al*., 2024). The progressive increase in lesion severity across exposure groups confirms a clear dose- and duration-dependent relationship, consistent with toxicological principles observed in indoor air pollution studies (Zhang *et al*., 2010).

Discussion

The present study clearly demonstrates that repeated inhalation of mosquito coil smoke induces significant histopathological alterations in rat liver tissue. The observed changes including hepatocellular degeneration, sinusoidal congestion, inflammatory infiltration, necrosis, and early fibrosis—suggest cumulative hepatic injury resulting from sustained toxicant exposure.

These findings corroborate earlier studies reporting liver damage following mosquito coil smoke exposure. Idowu *et al*. (2013) reported severe sinusoidal congestion and hepatocyte necrosis in rats after prolonged exposure, while Al-Mamun *et al*. (2017) observed elevated liver enzymes and histological damage in mice. The present results further strengthen these observations by demonstrating a graded response based on exposure intensity and duration. The liver's vulnerability may be attributed to its role in metabolizing inhaled toxicants such as PAHs, formaldehyde, and pyrethroid derivatives present in mosquito coil smoke (Liu *et al*., 2003; Lee *et al*., 2006). Metabolic activation of these compounds can generate reactive oxygen species, leading to oxidative stress, lipid peroxidation, and inflammatory responses (Abdrabouh *et al*., 2021). Kupffer cell hyperplasia and portal inflammation observed in this study support an immune-mediated mechanism of injury. Early fibrotic changes observed in the high-exposure group indicate initiation of tissue remodeling, which may progress to chronic liver disease upon continued exposure. Similar early fibrotic alterations have been documented in recent experimental studies on mosquito coil smoke toxicity (Syed *et al*., 2024). Overall, the findings highlight the potential systemic health risks associated with chronic indoor mosquito coil use and underscore the importance of minimizing exposure and promoting safer alternatives.

Conclusion

Mosquito coil smoke exposure produced clear, graded histopathological damage in rat liver, characterized by hepatocellular degeneration, congestion, inflammation, necrosis, and early fibrotic remodeling. The results add to evidence that mosquito coils are not merely respiratory irritants but can contribute to systemic organ toxicity under repeated exposure. Reducing indoor exposure through ventilation and safer alternatives is advisable, and regulatory efforts should prioritize lower-emission repellent products.

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