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Comparative evaluation of heat stress and fuel Vapour exposure on hepatic histology and liver function Biomakers in Adult Wistar Rats

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Abstract

Environmental stressors such as extreme heat and fuel vapour exposure have become significant public health concerns due to their adverse effects on various organs of the body, particularly the liver, which plays a central role in metabolism, detoxification, and maintaining physiological homeostasis. Prolonged exposure to these stressors may induce oxidative stress, inflammation, and structural damage to hepatic tissues, thereby impairing liver function.

This study was designed to comparatively evaluate the effects of evaluate the effects of heat stress and fuel vapour exposure on hepatic histology and liver function biomakers in Adult wistar . A total of 12 healthy adult albino Wistar rats weighing between 150-200g were used for the study and randomly assigned into 3 groups, comprising a control group, an extreme heat exposure group, and an extreme fuel vapour exposure group. The experimental exposures were carried out for 2 weeks. At the end of the exposure period, blood samples were collected for biochemical analysis of liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). Liver tissues were also harvested and processed for histological examination using Haematoxylin and Eosin staining techniques.

The results showed significant increases in liver enzyme activities in both the heat-exposed and fuel vapour-exposed groups compared with the control group ($p < 0.05$). The heat-exposed group recorded AST, ALT, and ALP values of 117.50 ± 1.44 IU/L, 126.50 ± 0.86 IU/L, and 182.24 ± 2.19 IU/L, respectively, compared with control values of 94.00 ± 2.31 IU/L, 92.00 ± 0.58 IU/L, and 121.31 ± 1.77 IU/L. The fuel vapour-exposed group demonstrated the highest enzyme levels, with AST, ALT, and ALP values of 187.50 ± 4.33 IU/L, 237.50 ± 1.44 IU/L, and 316.22 ± 3.51 IU/L, respectively, indicating significantly greater hepatotoxicity than heat exposure ($p < 0.05$).

Histological examination revealed normal hepatic architecture in the control group, characterized by preserved hepatic lobules, well-arranged hepatocyte cords, and normal central veins. The fuel vapour-exposed group exhibited mild diffuse inflammatory cell infiltration and mild disorganization of the central vein. The heat-exposed group also demonstrated diffuse inflammatory cell infiltration but showed moderate stromal degeneration, indicating structural alterations in hepatic tissue.

In conclusion, both extreme heat exposure and fuel vapour exposure adversely affected liver structure and function in adult Wistar rats. However, fuel vapour exposure produced more pronounced biochemical alterations and greater hepatotoxic effects than extreme heat exposure. These findings highlight the potential occupational and environmental health risks associated with prolonged exposure to these stressors and emphasize the need for preventive measures to minimize liver damage in exposed populations.

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Keywords: Extreme Heat Exposure; Fuel Vapour Exposure; Liver Histology; Liver Enzymes; Hepatotoxicity; Adult Wistar Rats.

1. Introduction

Rising global temperatures and increasing occupational exposure to petroleum-based fuels represent two of the most pervasive environmental stressors affecting biological systems today. While these two forms of stress differ markedly in their route of induction, thermal versus chemical, both have been independently linked to disruptions in cellular homeostasis, oxidative damage, and organ-level toxicity, with the liver emerging as a particularly vulnerable target in both cases.

Heat stress occurs when an organism's core body temperature rises beyond its thermoneutral zone, overwhelming its physiological capacity for thermoregulation. In animals, prolonged exposure to elevated ambient temperature is known to disturb the steady-state balance between pro-oxidants and antioxidants, resulting in excessive generation of reactive oxygen species and consequent cellular and mitochondrial damage (Belhadj Slimen et al., 2016). Beyond its molecular effects, heat stress also reorganizes the way the body allocates its resources, altering fat, protein, and energy metabolism independent of any accompanying reduction in feed intake (Belhadj Slimen et al., 2016). These disruptions are not restricted to livestock or production animals; comparable oxidative and physiological consequences of heat exposure have been documented in laboratory and companion species as well. In female rabbits, for instance, prolonged thermal stress has been shown to alter organ weights, reduce red blood cell counts, and elevate kidney malondialdehyde levels while suppressing key antioxidant enzymes such as catalase, superoxide dismutase, and glutathione peroxidase, changes that collectively point to systemic oxidative injury and impaired physiological function (Mutwedu et al., 2021). Reviews of thermotolerance across species similarly emphasize that heat stress compromises production, reproduction, and welfare largely through mechanisms rooted in oxidative and cellular stress pathways, underscoring the need for continued investigation of species- and organ-specific responses (Sejian et al., 2018).

Parallel to thermal stress, exposure to petroleum-derived fuels such as petrol, diesel, and kerosene has become an increasingly recognized occupational and environmental hazard, particularly in regions where fuel handling, generator use, and informal refining are common. Inhalation of gasoline fumes has been shown to elevate serum markers of hepatic dysfunction, including alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase, alongside histopathological alterations in liver tissue of exposed rats, with the severity of damage increasing with duration of exposure (Owagboriaye et al., 2017). Renal function is similarly compromised following gasoline vapor exposure, with significant increases in serum creatinine, urea, and blood urea nitrogen reported in exposed rats relative to controls, and female rats appearing more susceptible than males (Uboh et al., 2008). Diesel fume exposure produces a comparable pattern of injury, with elevated packed cell volume and leucocyte counts accompanied by histopathological evidence of hepatic portal congestion, periportal connective tissue changes, and renal tubular degeneration in exposed Wistar rats (Okoh et al., 2023). Taken together, this body of work establishes fuel exposure, whether through inhalation or ingestion, as a potent hepatotoxic and nephrotoxic insult in rodent models.

Despite the substantial and separately growing literature on heat stress and on fuel exposure, few studies have directly compared the relative severity, mechanisms, or histopathological signatures of these two stressors within a single experimental framework. This is a notable gap, given that both stressors converge on many of the same downstream pathways, oxidative stress, disrupted membrane-bound enzyme activity, and impaired organ-level detoxification, yet arise from fundamentally different routes of insult (physical versus chemical). Understanding whether extreme heat and extreme fuel exposure produce comparable, additive, or distinct patterns of hepatic damage has both toxicological and occupational health relevance, particularly for populations simultaneously exposed to hot climates and fuel-handling occupations, such as generator operators, roadside fuel vendors, and mechanics in tropical regions.

The albino Wistar rat remains one of the most widely validated models for hepatotoxicological and stress-physiology research, owing to its physiological similarity to humans, well-characterized baseline hematological and biochemical parameters, and sensitivity to both thermal and chemical insult. Accordingly, the present study was designed to comparatively evaluate the effects of extreme heat exposure and extreme fuel exposure on liver histology and liver enzyme profiles (ALT, AST, and ALP) in albino Wistar rats. By subjecting experimental groups to each stressor independently under controlled conditions, this study aims to characterize and contrast the degree of hepatocellular injury induced by each, thereby contributing to a more integrated understanding of how distinct environmental stressors converge on, or diverge in, their hepatotoxic outcomes.

Aim

The aim of this study was to comparatively evaluate the effects of heat stress and fuel vapour exposure on hepatic histology and liver function biomakers in Adult wistar Rats.

Study area

This study was conducted at the Federal University of Technology Owerri (FUTO) in Owerri West Local Government Area, Imo State, Nigeria. FUTO is bounded by four surrounding communities: Eziobodo, Ihiagwa, Obinze and Umuchinma. The university is situated approximately 25 kilometers south of Owerri, the capital of Imo State, and sits on a sprawling campus of about 10,000 acres (4,048 hectares) along the banks of the Otamiri River. Established in 1980, FUTO is the first and premier federal university of technology in Nigeria and remains the only one of its kind in the South-Eastern part of the country. Owerri West, the local government area within which the university is located, has an estimated population of 99,265 people according to the Nigerian census of 2006. The local government area spans approximately 295 square kilometers in Imo State, which is in the Southeastern part of Nigeria, and is located at approximately latitude 5°23′N and longitude 6°59′E

2. Materials used

- Male adult wistar rats
- Petrol as fuel vapour from a nearby fueling station
- 40 watt bulb for heat source
- Exposure chambers
- perforated water bottle
- standard rat chow
- clean tap water (Provided ad libitum)
- sawdust bedding
- wooden cages with metal wire covers
- digital thermometer
- mild detergent (Viva)
- gloves
- feeding plates
- cotton wool
- broom
- parker
- tape
- markers
- normal saline
- weighing scale
- distilled water
- ethanol
- lab coats
- plain bottles
- petri dish
- thermostat
- dissecting set
- chloroform.

2.1. Sampling technique

Samples were obtained using cardiac puncture and placed in a plain tube before proceeding to the laboratory for analysis

2.2. Experimental animals

Adult wistar rat weighed around 150-200g. A total of 12 male adult wistar rats were used because of their similar physiological and morphological similarities with humans and also the low hormone interference when exposed to toxins compared to that female. The rats were grouped into 3 groups (control group, Heat group, Fuel group), having 4 rats per group.

2.3. Ethical clearance

Ethical clearance was sought and obtained from the Ethics and Research Committee School of Basic Medical Sciences, Federal University of Technology, Owerri, Imo state before the commencement of the research.

2.4. Experiment Design

Group 1 (Control): Rats were acclimatized for 21 days and received standard feed and water only They were maintained under standard laboratory conditions: Temperature: 25 ± 2°C. Humidity: 50–60%. Light/Dark Cycle: 12-hour each with no exposure to stressors/toxins.

Group 2 (Heat Group): The rats assigned to the heat exposure group were subjected to extreme heat conditions in a controlled environment throughout the experimental period. Prior to the commencement of the exposure, the animals were acclimatized to the laboratory environment for a period of 21 days under standard housing conditions with unrestricted access to food and water.

During the experiment, the animals in the heat exposure group were placed inside a heat chamber with side openings to avoid suffocation and maintained a temperature of 40 ± 2°C for 4 hours daily for 21 days. The temperature was continuously monitored using a Thermostat to ensure consistency throughout the exposure period.

At each daily exposure session, the rat's rectal temperature was checked before and after exposure, using a digital thermometer and were returned to their cages and allowed free access to feed and clean drinking water. Throughout the experimental period, the animals were observed for signs of heat stress, behavioral changes, and general wellbeing At the completion of the exposure period, body weight was checked for either an increase or decrease in body weight, blood samples were collected for biochemical analysis, and the liver tissues were harvested for histological examination.

Group 3 (Fuel vapor): The rats assigned to the fuel vapour exposure group were exposed to fuel vapour using an inhalation exposure method in a specially designed exposure chamber.

A measured quantity of fuel (60ml of fuel soaked in cotton wool) was placed in a perforated plastic bottle and inside the exposure chamber without direct contact between the animals and the liquid fuel. This allowed the animals to inhale the generated vapour while preventing ingestion or dermal exposure. The animals were exposed for 4 hours per day for 21 days. Throughout the exposure period, adequate ventilation and standard animal welfare procedures were maintained.

After each exposure session, the rats were returned to their cages and provided unrestricted access to food and water. The animals were monitored daily for behavioral and physiological changes.

At the end of the experimental period, their body weight was taken to check for either an increase or decrease in body weight, blood samples were collected for biochemical analysis, and liver tissues were excised and processed for histological examination using standard Haematoxylin and Eosin (H&E) staining procedures.

3. Results and Discussion

Table 1 Effects of heat and fuel exposure on liver enzymes (AST, ALT, and ALP)

	AST Level (IU/l)	**ALT Level (IU/l)**	**ALP Level (IU/l)**
	MEAN±SEM	MEAN±SEM	MEAN±SEM
Group A (Distilled Water Only)	94.00±2.31	92.00±0.58	121.31±1.77
Group B (Heat exposure)	117.50±1.44*	126.50±0.86*	182.24±2.19*
Group C (Fuel exposure)	187.50±4.33*#	237.50±1.44*#	316.22±3.51*#
f-ratio	271.23	547.60	146.67

Data obtained from the assessments were analyzed using one-way Analysis of Variance (ANOVA), and significant differences among toxins means were further evaluated using Tukey's Honest Significant Difference (HSD) test at $p < 0.05$.

Table 1 result revealed a significant increase in groups B and C compared to group A. Conversely, group C showed a significant increase in the AST result compared to group B.

3.1. Histological findings

Control 1:

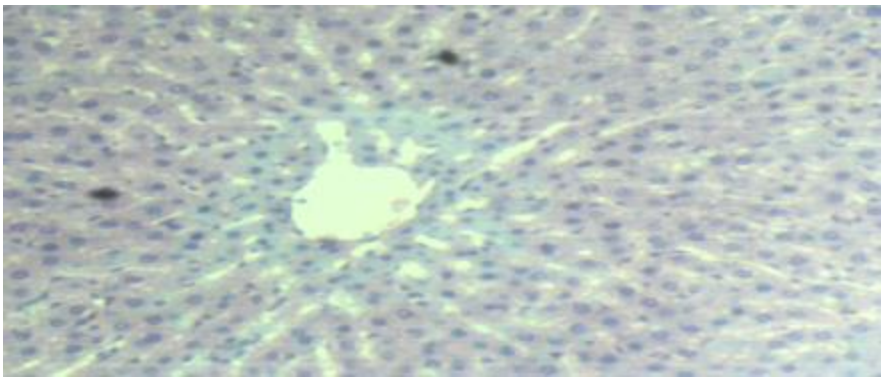

Figure 1 Control Liver histology plate A1

Control 2:

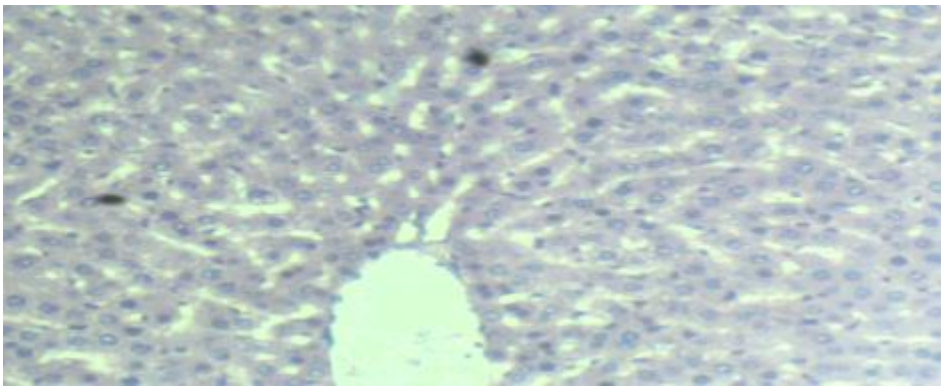

Figure 1 Control Liver histology plate A2

Plate A1 and A2: Photomicrographs of sections of liver tissue of an albino rat show normal hepatic architecture with preserved lobular pattern. Hepatocytes (arrow) are arranged in cords separated by sinusoids. The central vein shows normal histological features (arrowhead). There is no evidence of steatosis, inflammation, fibrosis or cholestasis observed (H&E X400).

Fuel 1:

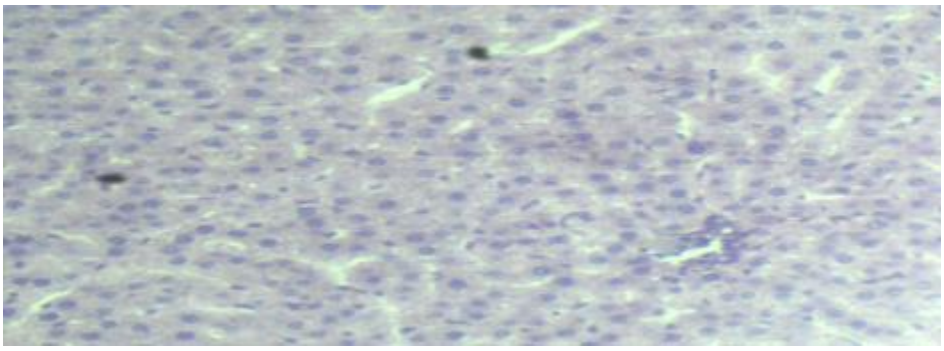

Figure 2 Fuel vapour-exposed liver histology plate B1

Fuel 2:

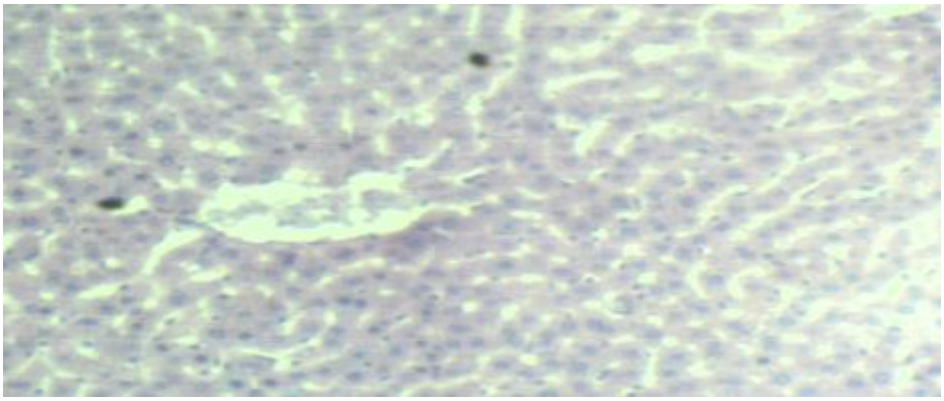

Figure 2 Fuel vapour-exposed liver histology plate B2

Plate B1 and B2: Photomicrographs of sections of liver tissue of an albino rat show normal parenchymal histological features with partially well-organised hepatic lobules showing radiating hepatocyte cords (arrow). The section, however, exhibits a mild but diffuse infiltration of inflammatory cells and mild signs of histological disorganisation of the central vein (arrowhead) (H&E X400).

Heat 1:

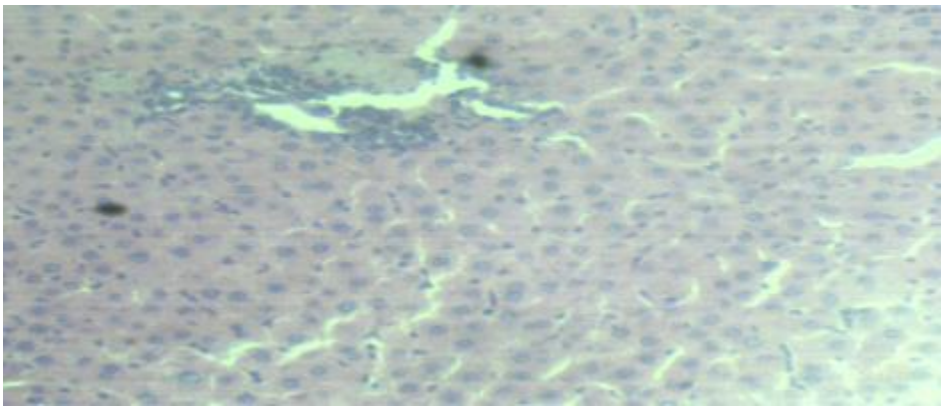

Figure 3 Heat-exposed Liver histology plate C1

Heat 2:

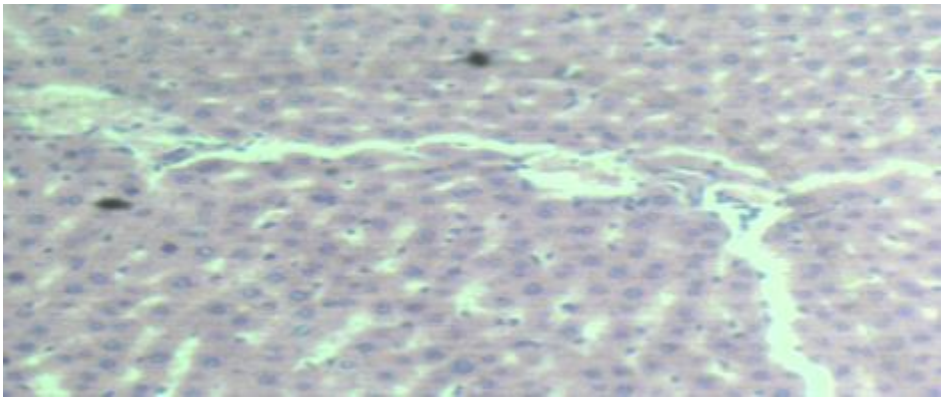

Figure 3 Heat-exposed liver histology plate C2

Plate C1 and C2: Photomicrographs of sections of liver tissue of an albino rat show normal parenchymal histological features with partially well-organised hepatic lobules showing radiating hepatocyte cords (arrow). The section, however, exhibits a mild but diffuse infiltration of inflammatory cells and moderate stromal degeneration (arrowhead) (H&E X400).

4. Discussion

The results of this study clearly demonstrate that both heat and fuel exposure cause statistically significant hepatic injury in albino rats, as evidenced by elevations in all three liver enzymes and by histopathological changes.

AST and ALT Elevations: AST is found in hepatocytes, cardiac muscle, skeletal muscle, and erythrocytes, making its elevation a general indicator of cell damage. ALT, however, is predominantly hepatic in location, and its disproportionate rise, especially in Group C, points strongly to hepatocellular injury as the dominant mechanism. The ALT: AST ratio in Group C (237.50/187.50 ≈ 1.27) is consistent with toxic or chemical hepatitis rather than alcoholic liver disease, further confirming direct hepatotoxicity from fuel vapour inhalation or absorption.

ALP Elevation: ALP is a marker of both hepatocellular damage and cholestasis. The substantial rise in ALP, particularly in the fuel group, may indicate that fuel exposure affects not only hepatocytes but also the biliary canalicular system. This is consistent with the histological finding of inflammatory cell infiltration, which, when occurring around portal tracts, can impede bile flow.

Heat Exposure vs. Fuel Exposure: Fuel exposure proved to be the more potent biochemical stressor, producing roughly double the enzyme elevations compared to heat exposure across all three markers. This is likely attributable to the direct cytotoxicity of petroleum-derived volatile organic compounds (VOCs), which can cause oxidative stress, lipid peroxidation of hepatocyte membranes, and mitochondrial dysfunction. Heat stress, while damaging, may operate through protein denaturation, heat shock protein activation, and micro vascular changes, producing a less severe biochemical phenotype in this model.

Histopathology Corroboration: The presence of inflammatory cell infiltration in both exposed groups confirms that hepatic inflammation is a shared mechanism. The additional finding of moderate stromal degeneration in the heat group implies damage to the connective tissue scaffold of the liver, which can compromise hepatocyte function over time. The apparent discrepancy, biochemically greater injury in fuel group but more structural damage histologically in heat group, may reflect the greater sensitivity of enzyme markers to early subcellular injury, whereas H&E histology detects structural changes at a later stage.

4.1. Fuel Exposure Studies

Owagboriaye et al. (2017) conducted an experimental study on forty albino rats exposed to gasoline fumes for varying durations (1, 3, 5, and 9 hours daily for 12 weeks) and reported a significant dose- and time-dependent increase in serum ALT, AST, and ALP levels compared to unexposed controls, along with progressive histopathological distortion of hepatic architecture. These findings are in strong agreement with the present study's Group C (fuel exposure) results, where all three liver enzymes were significantly elevated and histology revealed mild inflammatory infiltration of the liver. The authors attributed the hepatocyte membrane damage to the abnormal dynamic properties of hepatocyte membranes induced by hydrocarbon constituents of fuel fumes.

Neghab et al. (2015), in a cross-sectional occupational study of 200 petrol station workers in Shiraz, Iran, exposed to benzene, toluene, and xylenes (BTX) at concentrations below threshold limit values, reported that serum alanine aminotransferase, aspartate aminotransferase, blood urea, and plasma creatinine were significantly higher in exposed workers than in an unexposed comparison group of 200 employees. This study is particularly significant because it demonstrates that even sub-threshold levels of petroleum hydrocarbons are sufficient to elevate liver enzymes, lending weight to the biochemical elevations observed in the present study's fuel-exposed group. The present study's finding of a 2.58-fold increase in ALT in the fuel-exposed group is consistent with the pattern of hepatocellular injury described by Neghab et al. (2015).

Dedeke et al. (2021), in their mechanistic study on 72 male albino rats exposed to gasoline fumes for 1–9 hours daily over 10 weeks, demonstrated that gasoline fume exposure caused elevated serum hepatic markers, increased oxidative stress, disruption of mixed function oxygenase (MFO) activity, and histopathological lesions including dilated sinusoids, degenerated hepatocytes, and hepatic necrosis. The inflammatory cell infiltration observed in the fuel-exposed group (Plates B1 and B2) of the present study parallels the hepatic inflammatory response described by these authors.

4.2. Heat Exposure Studies

Gupta et al. (2018) investigated the effect of heat stress on liver microstructure and function in rats exposed to a heat simulation chamber at 45 ± 0.5°C. They categorised rats as moderately heat-stressed (core temperature 40°C) and severely heat-stressed (core temperature 42°C), and reported significant elevations in ALT, AST, and ALP levels in both heat-stressed groups relative to controls. They also documented significantly elevated reactive oxygen species (ROS) and nitric oxide (NO) in the liver, upregulation of heat shock proteins (HSPs) and caspases, and histological evidence of extensive hepatic parenchymal cell loss and lobular disorganisation. The elevated AST, ALT, and ALP observed in Group B (heat exposure) of the present study, along with the histological finding of mild inflammatory infiltration and moderate stromal degeneration, are consistent with and supported by the findings of Gupta et al. (2018).

Skibba et al. (1991), in a landmark study using isolated, haemoglobin-free perfused rat livers subjected to hyperthermic perfusion, demonstrated that oxidative stress precedes the appearance of cytosolic enzymes in the perfused state and subsequent cell death. Their observation that heat-induced hepatotoxicity is initiated by oxidative stress provides a physiological basis for the enzyme elevations observed in the heat-exposed group of the present study, and further explains the moderate stromal degeneration seen histologically, which may reflect oxidative injury to the hepatic connective tissue matrix.

The histological pattern of mild inflammatory infiltration without gross necrosis or fibrosis in both exposed groups of the present study suggests that the duration or intensity of exposure represents an early or subacute phase of hepatotoxicity. This is consistent with the literature: Owagboriaye et al. (2017) and Dedeke et al. (2021) noted that more prolonged gasoline fume exposure progressively worsened histopathological findings, and Gupta et al. (2018) similarly observed that more severe hyperthermia produced more extensive hepatic damage. Taken together, the comparison confirms that the present study's findings are well within the expected range of hepatotoxic responses to these stressors

5. Conclusion

This study demonstrated that both extreme heat exposure and fuel vapour exposure induce significant hepatic injury in adult Wistar rats, as evidenced by marked elevations in serum liver enzymes (AST, ALT, and ALP) and histopathological alterations in liver tissue. Although both environmental stressors adversely affected liver integrity, fuel vapour exposure produced substantially greater biochemical evidence of hepatocellular damage, indicating a higher degree of hepatotoxicity. Histological examination further confirmed that both exposures elicited inflammatory changes, with heat exposure causing moderate stromal degeneration, while fuel vapour exposure was characterized by inflammatory cell infiltration and disruption of normal hepatic architecture.

These findings suggest that prolonged exposure to either extreme heat or petroleum fuel vapours compromises liver function through mechanisms likely involving oxidative stress, inflammation, and cellular injury. The greater biochemical derangement observed following fuel vapour exposure highlights the heightened risk associated with occupational and environmental exposure to petroleum products, particularly among individuals such as fuel station attendants, mechanics, generator operators, and workers in hot tropical environments.

Overall, this study contributes to the growing body of evidence on environmentally induced hepatotoxicity and underscores the importance of implementing effective occupational safety measures, reducing unnecessary exposure to fuel vapours, and adopting appropriate heat-mitigation strategies. Future studies should investigate the underlying molecular mechanisms, including oxidative stress and inflammatory biomarkers, evaluate the long-term effects of combined heat and fuel vapour exposure, and explore potential therapeutic interventions capable of protecting the liver against these environmental insults.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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