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Hepatotoxic Effects of Sub-Chronic Nitrocellulose Thinner Vapor Exposure and Incomplete Recovery Following Withdrawal in Male Wistar Rats

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ABSTRACT

Background: Nitrocellulose thinner is a widely used industrial solvent mixture with recognized toxicity. Occupational exposure, particularly among painters and other industrial workers, is common; however, the extent of hepatic recovery following prolonged inhalational exposure remains poorly understood. This study evaluated the hepatotoxic effects of sub-chronic inhalation of nitrocellulose thinner vapor (NCTV) and the degree of hepatic recovery following withdrawal in male Wistar rats.

Materials and Methods: Twenty-four adult male Wistar rats were randomly allocated into three groups (n = 8 per group): a control group, an NCTV exposure group (4 hours/day for 90 consecutive days), and a withdrawal group exposed to NCTV for 90 days followed by a 90-day recovery period without further exposure. Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total and conjugated bilirubin, total protein, albumin, and globulin levels were measured using standard colorimetric methods. The albumin-to-globulin (A/G) ratio was calculated, and liver tissues were examined histologically using hematoxylin and eosin staining.

Results: Sub-chronic NCTV inhalation significantly increased serum ALT, AST, ALP, and total bilirubin levels ($p < 0.05$) and significantly reduced total protein and globulin concentrations, resulting in an increased A/G ratio. Histopathological examination revealed vascular congestion, sinusoidal dilatation, inflammatory cell infiltration, and hepatocellular swelling. Following the 90-day withdrawal period, serum ALT, ALP, and total bilirubin levels declined significantly and approached the control values. However, AST remained significantly elevated ($p < 0.05$), albumin concentrations decreased significantly compared with both the control and exposure groups, resulting in a marked reduction in the A/G ratio (0.43 ± 0.06), and conjugated bilirubin remained significantly higher than that in controls ($p < 0.05$). Histological examination revealed only partial recovery, with persistent vascular congestion and mild hepatic architectural abnormalities.

Conclusion: Sub-chronic inhalation of nitrocellulose thinner vapor induces significant hepatotoxicity in male Wistar rats. Although withdrawal leads to partial biochemical and histological recovery, persistent alterations in liver function biomarkers and hepatic architecture suggest incomplete hepatic recovery following prolonged exposure. These findings underscore the potential for long-term liver injury associated with chronic occupational exposure and highlight the importance of preventive measures and adequate recovery times.

Keywords: Nitrocellulose thinner, hepatotoxicity, inhalation exposure, liver recovery, occupational exposure, Wistar rats.

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INTRODUCTION

The liver is the primary organ responsible for the metabolism, detoxification, and elimination of endogenous and exogenous substances. Although it constitutes only about 2% of total body weight, it receives approximately 25% of the cardiac output, reflecting its central role in maintaining metabolic homeostasis (1). As the principal site of xenobiotic biotransformation, the liver is particularly vulnerable to toxic injury. Many xenobiotics undergo cytochrome P450-mediated metabolism, generating reactive metabolites that induce oxidative stress, lipid peroxidation, mitochondrial dysfunction, and hepatocellular damage (2).

Occupational exposure to organic solvents represents an important but often underrecognized cause of liver injury and other systemic health effects. Nitrocellulose thinner (NCT), a solvent mixture widely used in the automotive, painting, furniture, and woodworking industries, contains several volatile organic compounds (VOCs), including toluene, acetone, and butyl acetate. These compounds readily evaporate at room temperature, creating substantial inhalation exposure, particularly in poorly ventilated workplaces. Clinical reports have documented cases of solvent-induced toxic hepatitis among workers exposed to paint thinners, while epidemiological studies have linked chronic VOC exposure to neurological impairment, respiratory disorders, and other adverse health outcomes (3,4).

Following inhalation, volatile organic compounds are rapidly absorbed through the pulmonary alveoli into the systemic circulation, largely bypassing first-pass gastrointestinal metabolism (5). Owing to their lipophilic properties, these compounds readily cross biological membranes and accumulate in highly perfused organs, particularly the liver, where they undergo extensive biotransformation. Although hepatic metabolism facilitates detoxification, it may also generate reactive intermediates capable of inducing oxidative stress, inflammation, and cellular injury (2).

In many low- and middle-income countries, occupational exposure to solvent vapours remains common because of inadequate workplace ventilation, inconsistent use of personal protective equipment, and limited enforcement of occupational health and safety regulations. Consequently, painters, automobile repair workers, furniture makers, and other artisans are frequently exposed to low concentrations of mixed organic solvents over prolonged periods. While the hepatotoxic effects of individual solvent components have been documented (6), considerably less is known about the cumulative hepatic effects of prolonged exposure to mixed solvent vapours such as nitrocellulose thinner.

An equally important but poorly understood question is whether liver injury induced by chronic nitrocellulose thinner vapour (NCTV) exposure is reversible following cessation of exposure. Most experimental studies have focused on the toxic effects of exposure itself, with limited attention given to the extent and duration of hepatic recovery after withdrawal. Determining whether biochemical and histopathological alterations persist after exposure has ceased is essential for understanding the long-term consequences of occupational solvent exposure and for informing workplace interventions, including exposure reduction, work rotation, and medical surveillance.

Therefore, this study aimed to evaluate the hepatotoxic effects of sub-chronic nitrocellulose thinner vapour inhalation and to determine the extent of hepatic recovery following a withdrawal period using biochemical markers of liver function and histopathological assessment in male Wistar rats.

METHODS

Study Design and Setting

This experimental study was conducted at the Animal House Facility, Department of Biochemistry, University of Calabar, Cross River State, Nigeria. The animals were maintained under standard laboratory conditions (temperature: 25 ± 2°C; relative humidity: 50–62%; 12-hour light/12-hour dark cycle) with free access to standard pellet diet and water.

The study lasted approximately six months, comprising a two-week acclimatization period, a 90-day exposure period to nitrocellulose thinner vapour (NCTV), and a subsequent 90-day withdrawal (recovery) period for the designated group.

Ethical Approval

All efforts were made to minimize animal suffering and to reduce the number of animals used.

The study was conducted in accordance with internationally accepted guidelines for the care and use of laboratory ani-

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mals. Ethical approval was obtained from the Faculty Research Animal Ethics Committee (FAREC-FBMS), Faculty of Basic Medical Sciences, University of Calabar, Nigeria (Approval No. 418BCM2628; approved on 5 March 2023). All efforts were made to minimize animal suffering and to reduce the number of animals used.

Experimental Animals

Twenty-four adult male Wistar rats weighing 100–110 g was obtained from the Animal House, Department of Biochemistry, University of Calabar. Following a two-week acclimatization period, the animals were randomly allocated into three groups (n = 8 per group):

- Group I (Control): Unexposed to NCTV.
- Group II (Exposure): Exposed to NCTV for 90 consecutive days.
- Group III (Withdrawal): Exposed to NCTV for 90 days followed by a 90-day recovery period without further exposure.

All animals completed the study without mortality.

Nitrocellulose Thinner and Exposure Protocol

Commercial nitrocellulose thinner (Abro® brand) was purchased from a local distributor in Calabar, Nigeria. Whole-body inhalation exposure was conducted using a locally fabricated exposure chamber, adapted from the method described by Uboh et al. (7). Briefly, 500 mL of nitrocellulose thinner was placed in two perforated 1000-mL containers within the chamber to allow gradual volatilization. Rats in the exposure groups were exposed to NCTV for 4 hours daily (09:00–13:00) over a 90-day period.

Although the airborne concentration of NCTV was not directly measured, the exposure conditions, chamber dimensions, volume of thinner, and exposure duration were standardized throughout the experiment to ensure consistency across all exposure sessions.

Sample Collection

Twenty-four hours after the final exposure (or completion of the withdrawal period), animals were fasted overnight and anesthetized using inhaled chloroform immediately before euthanasia. Blood samples were collected by cardiac puncture into plain tubes, allowed to clot, and centrifuged at 3000 rpm for 10 minutes. Serum was separated and stored at 4°C until biochemical analysis, which was completed within 24 hours.

Liver tissues were excised immediately after sacrifice, rinsed with normal saline, and fixed in 10% neutral buffered formalin for histopathological examination.

Biochemical Analysis

Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, conjugated bilirubin, total protein, and albumin were measured using commercially available diagnostic kits (Agappe Diagnostics Ltd., India) according to the manufacturer's instructions.

Total protein was determined using the Biuret method, whereas albumin was measured using the bromocresol green dye-binding method. Serum globulin concentration was calculated by subtracting albumin from total protein, and the albumin-to-globulin (A/G) ratio was subsequently calculated.

Histopathological Examination

Fixed liver tissues were processed using standard histological procedures. Sections (5 µm thick) were stained with hematoxylin and eosin (H&E) and examined under a light microscope for evidence of hepatocellular injury and architectural changes.

Statistical Analysis

Data were analyzed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test for multiple comparisons. Results are presented as mean ± standard deviation (SD). Statistical significance was established at $p < 0.05$.

RESULTS

Effect of Withdrawal Following Sub-Chronic NCTV Exposure on Serum Liver Enzymes

The effects of withdrawal following a 90-day exposure to nitrocellulose thinner vapour (NCTV) on serum liver enzyme activities are presented in **Table 1**.

Compared with the control group (Group I), rats exposed to NCTV for 90 days (Group II) exhibited significantly higher serum alkaline phosphatase (ALP), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) activities (all $p < 0.05$). Following the 90-day withdrawal period (Group III), serum ALT and ALP activities decreased significantly ($p < 0.05$) compared with the exposure group and returned to levels comparable to those of the control group. In contrast, although AST activity declined after withdrawal, it remained significantly higher than that of the control group ($p < 0.05$) (Table 1).

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Table 1: Serum levels of liver enzymes in male Wistar rats following withdrawal from NCTV exposure

	ALP (IU/L)	AST(IU/L)	ALT(IU/L)
Group I(Control)	28.60 ± 3.40	15.20 ± 3.30	18.00 ± 2.70
Group II (NCTV Exposure)	37.59 ± 0.60*	26.40 ± 4.50*	33.00 ± 5.70*
Group III (Withdrawal)	29.38 ± 3.30[a]	22.40 ± 1.70*	21.00 ± 4.10[a]

ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Values are expressed as mean ± SD, n = 8;

[*] *Significantly different from control at p < 0.05;*

[a] *Significantly different from NCTV 90- day exposure group at p < 0.05.*

Effect of Withdrawal on Serum Protein Profile

The effects of withdrawal on serum protein concentrations are presented in **Table 2**.

Compared with the control group, NCTV exposure significantly reduced serum total protein and globulin concentrations ($p < 0.05$), whereas serum albumin concentrations did not differ significantly ($p > 0.05$). Consequently, the albumin-to-globulin (A/G) ratio was significantly higher in the exposure group than in the control group ($p < 0.05$).

Following withdrawal, serum total protein and globulin concentrations increased significantly compared with the exposure group ($p < 0.05$). However, serum albumin concentration decreased significantly relative to both the control and exposure groups ($p < 0.05$), resulting in a marked reduction in the A/G ratio compared with both groups (Table 2).

Table 2: Serum levels of proteins in male Wistar rats following withdrawal from NCTV exposure.

	Total Protein(mg/dL)	Albumin (mg/dL)	Globulin (mg/dL)	A/G Ratio
Group I(Control)	8.07 ± 0.53	3.26 ± 0.24	4.80 ± 0.44	0.68 ± 0.08
Group II (NCTV Exposure)	6.60 ± 0.57*	3.25 ± 0.14	3.35 ± 0.59*	0.97 ± 0.18*
Group III (Withdrawal)	7.52 ± 0.18 [a]	2.25 ± 0.29 *, [a]	5.27 ± 0.23 [a]	0.43± 0.06 *, [a]

Values are expressed as mean ± SD, n = 8.

** Significantly different from control at p < 0.05;*

[a] *Significantly different from NCTV 90-day exposure group at p < 0.05.*

Effect of Withdrawal on Serum Bilirubin Levels

The effects of withdrawal on serum bilirubin concentrations are presented in **Table 3**.

Rats exposed to NCTV for 90 days showed significantly higher total bilirubin concentrations than the control group ($p < 0.05$). Following withdrawal, total bilirubin concentrations decreased significantly compared with the exposure group ($p < 0.05$) and were no longer significantly different from those of the control group.

Conjugated bilirubin concentrations were significantly higher in both the exposure and withdrawal groups than in the control group ($p < 0.05$). However, no significant difference was observed between the exposure and withdrawal groups ($p > 0.05$) (Table 3).

Table 3: Serum levels of bilirubin in male Wistar rats following withdrawal from NCTV exposure.

	Total Bilirubin (mg/dL)	Conjugated Bilirubin (mg/dL)
Group I(Control)	0.53 ± 0.08	0.27± 0.01
Group II (NCTV Exposure)	0.73 ± 0.09 *	0.34 ± 0.07*
Group III (Withdrawal)	0.59 ± 0.11 [a]	0.38 ± 0.03 *

Values are expressed as mean ± SD, n = 8.

** Significantly different from control (Group I) at p < 0.05;*

[a] *Significantly different from NCTV 90-day exposure group (Group II) at p < 0.05.*

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Histopathological Findings

Representative photomicrographs of liver tissues are shown in Figures 1A–1C.

Liver sections from the control group (Figure 1A) exhibited normal hepatic architecture characterized by well-arranged hepatocyte cords radiating from the central vein, intact sinusoidal spaces, and normal portal tracts.

In contrast, liver sections from the NCTV-exposed group (Figure 1B) demonstrated congested central veins, sinusoidal dilatation, mild centrilobular inflammatory cell infiltration, and hepatocellular swelling, indicating hepatic injury.

Following the withdrawal period (Figure 1C), partial restoration of hepatic architecture was observed, with improved organization of hepatocyte plates and preservation of portal tracts. Nevertheless, residual vascular congestion, mild sinusoidal dilatation, and hepatocellular swelling persisted, indicating incomplete structural recovery after cessation of NCTV exposure.

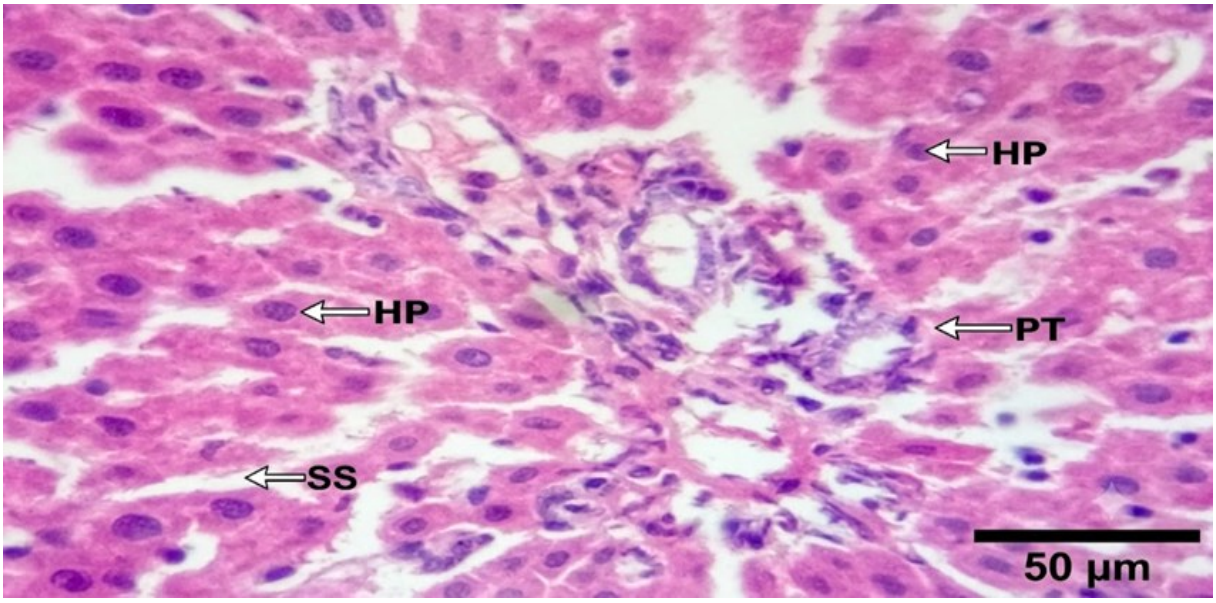

Figure 1a: *Photomicrograph of liver section from control group showing normal hepatic architecture with well-organized hepatocyte plates and intact portal tracts (H&E stain, ×400).*

Key: *HP = hepatocytes; SS = sinusoidal spaces; PT = portal tract. Scale bar = 50 µm.*

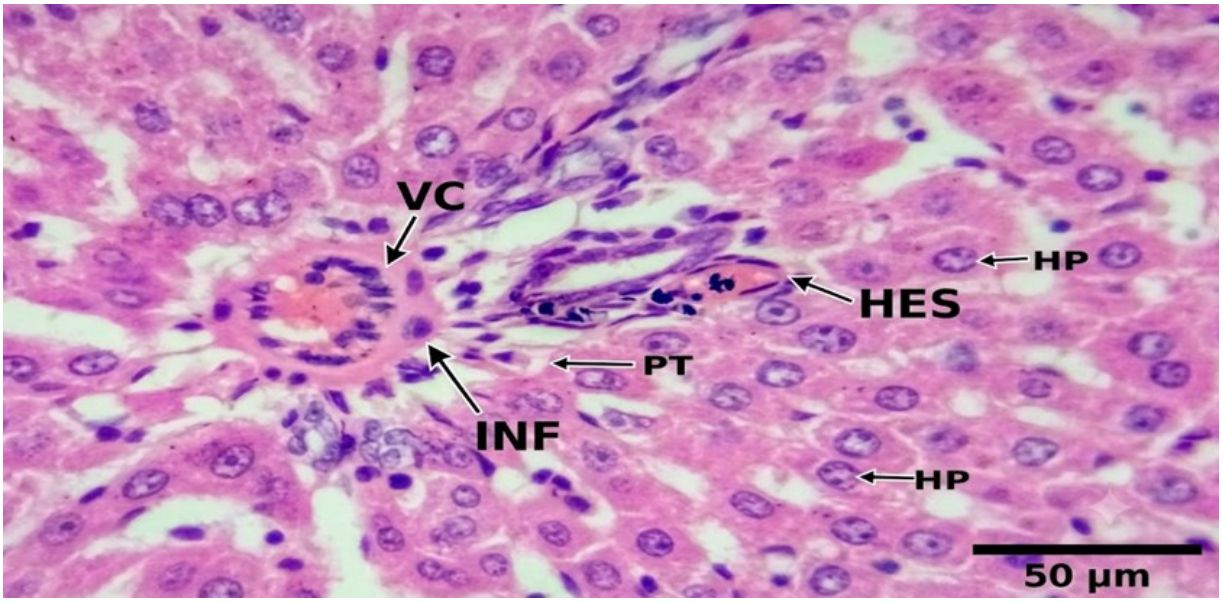

Figure 1b: *Photomicrograph of a liver section from the NCTV 90-day exposure group showing vascular congestion, portal tract inflammation, and hepatocellular swelling (H&E stain, ×400).*

Key: HP = hepatocytes; PT = portal tract; VC = vascular congestion; INF = inflammatory cell infiltration; HES = hepatocellular swelling. Scale bar = 50 µm.

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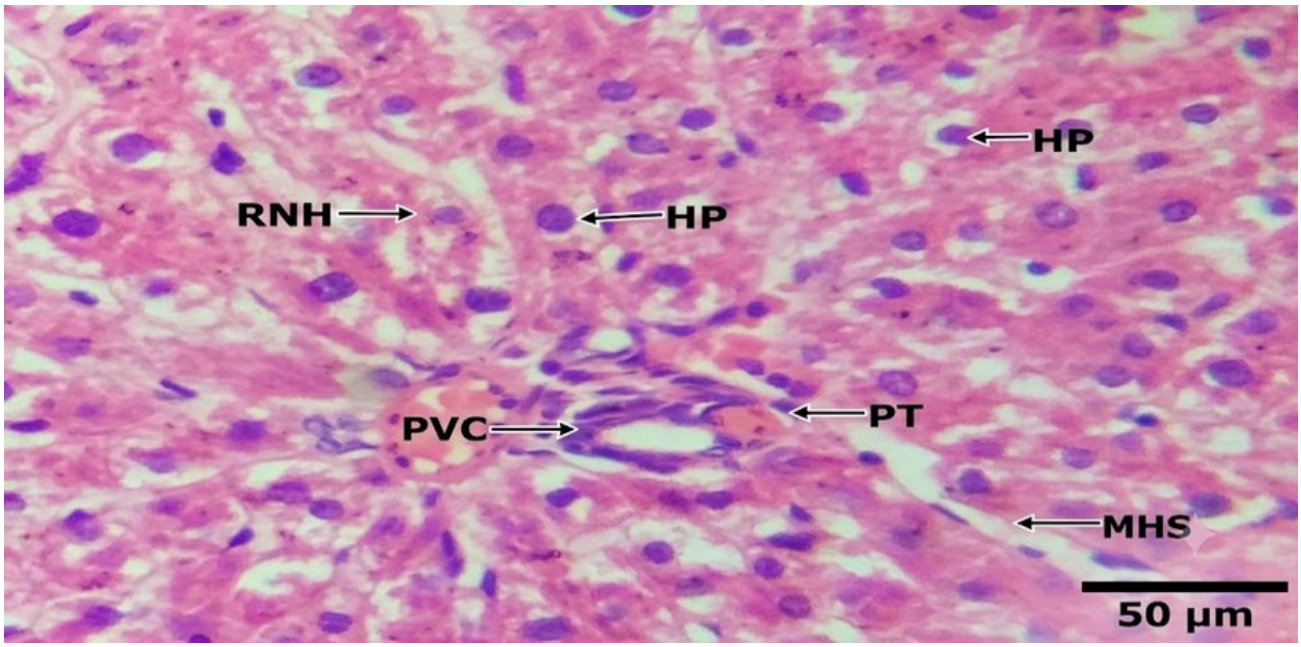

Figure 1c: *Photomicrograph of a liver section from the NCTV withdrawal group showing signs of partial tissue recovery alongside persistent microstructural alterations (H&E stain, ×400).*
Key: HP = hepatocytes; PT = portal tract; RNH = regenerated hepatocytes; PVC = persistent vascular congestion; MHS = mild hepatocellular swelling. Scale bar = 50 µm.

DISCUSSION

This study evaluated the effects of withdrawal following sub-chronic exposure to nitrocellulose thinner vapour (NCTV) on hepatic function in male Wistar rats. Prolonged NCTV exposure produced significant biochemical and histopathological evidence of liver injury. Although a 90-day withdrawal period resulted in partial recovery of several liver function parameters, persistent abnormalities in selected biochemical markers and liver architecture indicated incomplete hepatic recovery.

The significant increases in serum ALT, AST, and ALP activities following NCTV exposure are consistent with hepatocellular injury and disruption of membrane integrity. ALT and AST are established indicators of hepatocellular damage, whereas elevated ALP suggests impairment of hepatobiliary function (9). Following withdrawal, ALT and ALP activities returned to values comparable to the control group, while AST remained significantly elevated. Because AST is present in both the cytosol and mitochondria, persistent elevation may indicate continued mitochondrial injury or delayed recovery of hepatocellular function following prolonged solvent exposure. Similar observations have been reported in experimental models of chemically induced hepatotoxicity (10).

Nitrocellulose thinner contains volatile organic compounds, including toluene and xylene, which undergo hepatic metabolism through cytochrome P450 enzymes. Their metabolism generates reactive oxygen species that promote oxidative stress, lipid peroxidation, and cellular injury (10). These mechanisms likely contributed to the biochemical and histological alterations observed in the present study.

NCTV exposure also altered serum protein profiles, as evidenced by reductions in total protein and globulin concentrations. Although total protein and globulin concentrations improved following withdrawal, albumin levels declined significantly, resulting in a marked reduction in the albumin-to-globulin ratio. Albumin is synthesized exclusively by the liver and has a relatively long biological half-life; therefore, delayed normalization of albumin may reflect persistent impairment of hepatic synthetic function. Alternatively, the observed changes may represent a continuing inflammatory response following prolonged solvent exposure (11–13). Additional mechanistic studies are needed to clarify these findings.

The increase in total bilirubin observed after NCTV exposure suggests impaired hepatobiliary function, consistent with previous studies of volatile organic solvent toxicity (14). Although total bilirubin normalized following withdrawal, conjugated bilirubin remained elevated, indicating that recovery of biliary excretory function may require a longer period than recovery of other biochemical parameters.

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Histopathological examination supported the biochemical findings. Liver sections from exposed animals demonstrated vascular congestion, sinusoidal dilatation, inflammatory cell infiltration, and hepatocellular swelling, confirming hepatic injury. Although partial restoration of normal hepatic architecture was evident after withdrawal, persistent vascular congestion and mild hepatocellular alterations indicated incomplete structural recovery. These findings are consistent with the persistent elevation of AST and abnormalities in serum protein concentrations observed after withdrawal (15).

The present findings have important occupational health implications. Workers involved in automobile refinishing, furniture manufacturing, spray painting, and related industries are frequently exposed to nitrocellulose thinner vapours, often under conditions of inadequate ventilation and inconsistent use of personal protective equipment. The persistence of biochemical and histological abnormalities after withdrawal suggests that cessation of exposure alone may not be sufficient to achieve complete hepatic recovery following prolonged exposure. These findings reinforce the importance of effective exposure prevention, adequate workplace ventilation, appropriate personal protective equipment, and periodic health surveillance for workers routinely exposed to organic solvents.

Strengths and Limitations

A major strength of this study was the inclusion of a withdrawal group, which enabled assessment of hepatic recovery following prolonged exposure to nitrocellulose thinner vapour. The combined evaluation of biochemical markers and histopathological changes also provided complementary evidence of liver injury and recovery.

Several limitations should be acknowledged. First, the airborne concentration of nitrocellulose thinner vapour was not directly measured, limiting precise characterization of exposure intensity. Second, the study relied primarily on routine biochemical markers and histopathological assessment; molecular analyses of oxidative stress, inflammation, and apoptosis were not performed and therefore the underlying mechanisms could not be directly evaluated. Finally, chloroform was used as the terminal anesthetic because of institutional resource limitations. Although it was administered uniformly across all groups immediately before euthanasia, a potential confounding effect on liver function cannot be completely excluded.

Conclusion

Sub-chronic inhalation of nitrocellulose thinner vapour produced significant hepatocellular injury in male Wistar rats, as demonstrated by alterations in liver enzymes, serum proteins, bilirubin concentrations, and hepatic histology. Although withdrawal resulted in partial recovery of several biochemical and structural parameters, persistent abnormalities in AST activity, albumin concentration, conjugated bilirubin, and liver architecture indicated incomplete hepatic recovery after 90 days without exposure. These findings suggest that prolonged occupational exposure to nitrocellulose thinner may result in sustained liver injury and underscore the importance of effective exposure prevention and long-term monitoring of exposed workers. Further studies incorporating quantitative exposure assessment and molecular biomarkers are warranted to better understand the mechanisms and time course of hepatic recovery.

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Conflict of Interest

The authors hereby declare no conflict of interest regarding the publication of this manuscript.

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