

Functional recovery and compensatory glomerular hypertrophy following withdrawal from subchronic nitrocellulose thinner vapour exposure in Wistar rats

Original Article

Abstract:

Nitrocellulose thinner is a volatile industrial solvent whose prolonged inhalation poses renal risks, though the reversibility of such effects remains under-researched. This study evaluated renal biochemical and histological changes after withdrawal from subchronic nitrocellulose thinner vapour (NCTV) exposure in male Wistar rats. Twenty-four rats were assigned into three groups: control, 90-day NCTV exposure, and 90-day exposure followed by 90-day withdrawal. Serum electrolytes, urea, and creatinine were determined using colorimetric and enzymatic techniques. Kidney tissues were processed for histological examination and quantitative morphometric analysis (Glomerular Occupancy %) using ImageJ. Results showed significant ($p < 0.05$) increases in serum urea and creatinine following 90-day exposure, alongside reduced glomerular occupancy indicating atrophy. Following withdrawal, biochemical markers returned to control levels, demonstrating functional reversibility. However, morphometric analysis revealed persistent adaptive remodeling; glomerular occupancy in the withdrawal group (72.1%) significantly exceeded control levels (64.5%), indicating compensatory glomerular hypertrophy. We conclude that while NCTV-induced biochemical dysfunction is reversible, and that the recovery process involves significant structural adaptation to restore renal homeostasis.

Key words:

nitrocellulose thinner; nephrotoxicity; renal recovery; glomerular alterations; wistar rats; subchronic exposure; solvent inhalation

Apstrakt:

Funkcionalni oporavak i kompenzatorna glomerularna hipertrofija nakon prestanka subhronične izloženosti parama nitroceluloznog razređivača kod Wistar pacova

Nitrocelulozni razređivač je isparljivi industrijski rastvarač čije produženo udisanje predstavlja rizik za bubrege, iako je reverzibilnost njegovih efekata i dalje nedovoljno istražena. Ova studija ispitala je biokemijske i histološke promene u bubrezima nakon prestanka subhronične izloženosti parama nitroceluloznog razređivača (NCTV) kod mužjaka Wistar pacova. Dvadeset četiri pacova podeljena su u tri grupe: kontrolnu grupu, grupu izloženu NCTV tokom 90 dana i grupu izloženu tokom 90 dana nakon čega je usledio period oporavka od 90 dana bez izloženosti. Elektroliti u serumu, urea i kreatinin određeni su kolorimetrijskim i enzimskim metodama. Tkiva bubrega su pripremljena za histološki pregled i kvantitativnu morfometrijsku analizu (procenat površine koju zauzima glomerul) pomoću programa ImageJ. Rezultati su pokazali značajan ($p < 0,05$) porast uree i kreatinina u serumu nakon 90 dana izloženosti, uz smanjenje površine koju zauzima glomerul, što ukazuje na atrofiju. Nakon prestanka izloženosti, biokemijski markeri su se vratili na nivoe kontrolne grupe, što dokazuje funkcionalnu reverzibilnost. Međutim, morfometrijska analiza je otkrila postojano adaptivno remodelovanje; procenat površine koju zauzima glomerul u grupi koja je prošla period oporavka (72,1%) bio je značajno veći u poređenju sa kontrolnom grupom (64,5%), što ukazuje na kompenzatornu glomerularnu hipertrofiju. Zaključujemo da, iako je biokemijska disfunkcija izazvana parama NCTV reverzibilna, proces oporavka uključuje značajnu strukturnu adaptaciju u cilju ponovnog uspostavljanja homeostaze bubrega.

Ključne reči:

nitrocelulozni razređivač; nefrotoksičnost; oporavak bubrega; glomerularne promene; Wistar pacovi; subhronična izloženost; udisanje rastvarača

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Received: April 04, 2026

Revised: May 18, 2026

Accepted: May 18, 2026



Introduction

The kidney plays a critical role in maintaining systemic homeostasis. This is achieved through the regulation of fluid and electrolyte balance, excretion of metabolic waste products, acid–base equilibrium, and blood pressure control. Renal function is commonly assessed using biochemical markers such as serum electrolytes, urea, and creatinine, which reflect glomerular filtration efficiency and tubular integrity (Lousa et al., 2020; Chafekar, 2025). Alterations in these indices often indicate impaired renal function and may precede overt clinical manifestations of kidney disease.

Renal dysfunction may arise from pathological conditions or exposure to nephrotoxic agents, including environmental pollutants and industrial solvents. Volatile organic compounds (VOCs), hydrocarbon mixtures, pesticides, and heavy metals have been reported to induce varying degrees of nephrotoxicity in both humans and experimental models (Liu et al., 2023; Zhang et al., 2024). Chronic exposure to such agents has been shown to impair renal filtration and structure. This often results in elevations in serum creatinine and urea, electrolyte disturbances, and histopathological alterations, including glomerular and tubular injury. Inhalation of a mixed organic solvent in rats has been shown to cause marked tubular epithelial damage and glomerular alterations. This supports relationships between chemical exposures and kidney injury. Systematic reviews also indicate an increased risk of chronic kidney disease in humans following prolonged solvent exposure (Lim & Oh, 2023).

Nitrocellulose thinner (NCT) is a widely used industrial solvent mixture containing volatile organic compounds that can cause systemic toxicity following inhalational exposure. The systemic toxicity of inhaled NCT fumes has been demonstrated in studies involving common commercial brands such as FIAB®, ABRO®, and SPRINT®. Exposure to these vapors for 4 h daily has been shown to significantly elevate serum urea levels and induce severe histopathological alterations in visceral organs, confirming that chronic inhalation poses a significant risk for multi-organ toxicity (Patrick-Iwuanyanwu et al., 2013). Furthermore, previous studies have demonstrated that exposure to nitrocellulose thinner vapor triggers significant elevations in serum urea and creatinine, alongside a decrease in antioxidant defenses, which leads to structural distortions in the glomerular basement membrane (Uboh et al., 2014).

Despite these documented hazards, there remains a critical gap in understanding the „toxicological window” and the potential for recovery following

the cessation of exposure. Understanding whether solvent-induced glomerular alterations are permanent or reversible is essential for occupational health risk assessment and clinical management. Therefore, the present study investigated the effects of subchronic inhalation of nitrocellulose thinner vapour (NCTV) on serum renal function indices and kidney histoarchitecture in male Wistar rats, with a particular emphasis on evaluating the extent of structural recovery through quantitative histomorphometric analysis following a 90-day withdrawal period

Materials and Methods

Study area and duration

This study was conducted at the Animal House Facility, Department of Biochemistry, University of Calabar, Calabar, Cross River State, Nigeria. The facility maintained controlled environmental conditions with temperature at 25 ± 2 °C, relative humidity of 50–62%, and a 12 h light/12 h dark cycle. The experimental duration was approximately six (6) months, comprising a two-week acclimatization period, a 90-day exposure phase to nitrocellulose thinner vapour (NCTV), and a subsequent 90-day withdrawal (recovery) period for the designated group.

Ethical approval

All experimental procedures were conducted in accordance with internationally accepted guidelines for the care and use of laboratory animals. 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; Approval Date: 5 March 2023). All efforts were made to minimize animal suffering and to reduce the number of animals used.

Materials and reagents

All chemicals and reagents used in this study were of analytical grade. Agappe diagnostics kits (Agappe Diagnostics Ltd., India) for the estimation of serum electrolytes, urea and creatinine were employed for the analysis. The Abro® brand of nitrocellulose thinner (NCT) was obtained from a commercial chemical outlet in Calabar, Nigeria.

Experimental animals

Twenty-four (24) male Wistar rats (*Rattus norvegicus*; Family: Muridae) weighing 100–110 g were obtained from the Animal House of the Department of Biochemistry, University of Calabar.

The animals were housed in standard laboratory cages under controlled environmental conditions (25 ± 2 °C; 50–62% humidity; 12 h light/dark cycle). They were acclimatized for two (2) weeks prior to the commencement of the experiment and were provided with standard pelletized rat chow and water *ad libitum*.

Animal handling and grouping

The rats were randomly divided into three (3) groups with each group containing eight (8) rats ($n=8$):

Group 1 (Control): Animals received no NCTV exposure.

Group 2 (Exposure): Animals were subjected to 90 days of NCTV exposure.

Group 3 (Recovery): Animals were subjected to 90 days of NCTV exposure followed by a 90-day withdrawal period.

Exposure procedure

Exposure to nitrocellulose thinner vapour was carried out using a modified whole-body inhalation method adapted from Uboh et al. (2005). The exposure system consisted of a locally fabricated chamber ($150 \text{ cm} \times 90 \text{ cm} \times 210 \text{ cm}$) placed within a fume cupboard. A measured volume (500 mL) of nitrocellulose thinner was placed in two perforated 1000 mL containers within the chamber to allow gradual volatilization. Animals in the exposure groups were placed inside the chamber and exposed to the vapour for 4 h daily (09:00 to 13:00 h) throughout the 90-day exposure period. Although the exact vapour concentration within the chamber was not analytically determined, a constant volume of nitrocellulose thinner and uniform chamber conditions were maintained throughout the exposure period to ensure consistency and reproducibility of exposure.

Collection of serum samples

Twenty-four (24) h after the final exposure and starvation, the rats were anaesthetized with chloroform. Blood was collected by cardiac puncture into plain screw-cap sample bottles. The samples were allowed to clot and the supernatant (sera) separated with Pasteur pipette (after spinning with MSE model [England] table-centrifuge at 3000 rpm for 10 minutes), and stored in a refrigerator. All biochemical analyses were carried out within 24 h.

Biochemical analysis

Biochemical indices were determined using Agappe diagnostic kits (Agappe Diagnostis Ltd., Kochi India). The standard and established protocols of the manufacturer were strictly followed.

Determination of sodium concentration in serum

The principle involved precipitation of sodium as the triple salt, sodium magnesium, and uranyl acetate. The excess uranium reacted with ferrocyanide to produce a chromophore whose absorbance (colour intensity) was inversely proportional to the concentration of sodium in the test specimen (serum) (Tietz, 1994).

Determination of concentration of potassium in serum

Sodium tetraphenyl boron was used as the indicator for the determination of potassium concentration. The intensity of which was proportional to potassium concentration in the range of 2–7 mmol/L (Tietz, 1994).

Determination of concentration of chloride in serum

Chloride reacted with mercuric thiocyanate to liberate thiocyanate ions, which on reaction with ferric ions formed a coloured complex whose intensity was proportional to the chloride concentration in the sample (Tietz, 1994).

Estimation of serum urea level

Serum urea concentration was determined using an enzymatic kinetic method (Urease-GLDH). Urea was hydrolyzed by urease, and the resulting ammonia was reacted with α -ketoglutarate and NADH via glutamate dehydrogenase. The rate of NADH oxidation was monitored spectrophotometrically at 340 nm. Results were expressed in mmol/L.

Estimation of serum creatinine level

Serum creatinine was estimated using the kinetic Jaffe method. Creatinine reacted with picric acid in an alkaline medium to form a red-orange alkaline picrate complex. The rate of complex formation was measured at 505 nm using a two-point kinetic approach to minimize interference from non-creatinine chromogens, ensuring high analytical specificity. Results were expressed in mmol/L.

Histological investigation

Histological processing and staining were carried out according to the standard protocols described by Suvarna et al. (2019). Five-micrometer ($5 \mu\text{m}$) thick sections were obtained and stained with Hematoxylin and Eosin (H&E) for light microscopic examination.

Quantitative morphometric analysis

Quantitative evaluation of glomerular structural changes was performed using ImageJ software (Fiji

version 1.54) following the standardized protocols for histological quantification established by Jensen (2013). For each experimental group representative micrographs (x400 magnification) were analyzed. The Glomerular Occupancy (%) was calculated by measuring the surface area of the glomerular tuft and the total area of the Bowman’s capsule. The occupancy ratio was derived using the formula:

Glomerular Occupancy % = $\frac{A_g}{A_b} \times 100$

All measurements were performed by a blinded observer to prevent bias. Statistical differences between groups were analyzed using a One-Way ANOVA followed by Tukey’s post hoc test, with significance set at $p<0.05$.

Statistical analysis

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

Results

Impact of NCTV exposure and withdrawal on renal function indices

The results of the impact of 90-day nitrocellulose thinner vapor (NCTV) exposure and subsequent withdrawal on renal function indices in male Wistar rats are presented in **Tab. 1**. Serum electrolytes (sodium, Na⁺; chloride, Cl⁻; and potassium, K⁺) were elevated in the 90-day NCTV exposure group compared with the control; however, these alterations were not statistically significant ($p>0.05$). Following the withdrawal period, electrolyte levels in the recovery group were comparable to those of the control group.

In contrast, serum urea and creatinine levels in the 90-day NCTV exposure group increased significantly ($p<0.05$) compared with the control group. Following cessation of exposure (NCTV withdrawal group), serum urea and creatinine levels

were significantly lower ($p<0.05$) than in the 90-day exposure group. Furthermore, these values returned to a range comparable to the control levels, indicating a reversal of the biochemical impairment. While this recovery was evident biochemically, morphometric analysis revealed an adaptive structural response in the kidney. The glomerular occupancy ratio in the withdrawal group (**Fig. 1D**) did not simply return to baseline but significantly exceeded that of the control group. This indicates a state of compensatory glomerular hypertrophy, where the expansion of the glomerular tuft served as the structural mechanism for the observed restoration of renal filtration capacity.

Impact of NCTV exposure and withdrawal on renal histology

A unified qualitative and quantitative analysis of the renal parenchyma was performed (**Fig. 1**)

The control, **Fig. 1A** reveals normal renal architecture with organized capillary tufts. The Glomerular occupancy was calculated as 64.5% (**Fig. 1D**). The Bowman’s space is distinct, and renal tubules (RT) appear intact.

The 90-day exposure to NCTV results in significant glomerular alterations (**Fig. 1B**). The tissue shows glomerular atrophy and mesangial congestion. Quantitative morphometry confirmed a reduction in glomerular occupancy to 61.8% (**Fig. 1D**). This led to relative expansion in the Bowman’s space. These structural distortions provide a morphological basis for the elevated urea and creatinine levels observed in **Tab. 1**.

The 90-day recovery showed substantial architectural restoration. Remarkably, the glomeruli exhibit compensatory hypertrophy, with the glomerular occupancy increasing to 72.1% (**Fig. 1D**). This expansion of the glomerular tuft resulted in a functional narrowing of the Bowman’s space. The surrounding renal tubules showed a return to normal epithelial integrity, which, in conjunction with the glomerular remodeling, facilitates the observed biochemical recovery.

Table 1. Renal function indices of male Wistar rats following subchronic nitrocellulose thinner vapour (NCTV) exposure and a 90-day withdrawal period. Values are expressed as mean ± SD (n=8). Data were analysed using one way ANOVA followed by Tukey’s post hoc test

Groups	Na ⁺ (mmol/L)	Cl ⁻ (mmol/L)	K ⁺ (mmol/L)	Urea (mmol/L)	Creatinine (mmol/L)
Control	126.43±1.99	74.54±4.64	4.91±0.63	4.43±0.94	0.06±0.02
NCTV(90-day) Exposure	127.39±3.53	75.68±8.25	5.43±0.37	6.64±0.49*	0.10±0.03*
NCTV (90-day)Withdrawal	126.28±3.44	72.82±1.49	4.72±0.23	4.62±0.84 [#]	0.07±0.02 [#]

* $p<0.05$ vs. Control.; [#] $p<0.05$ vs. NCTV exposure.

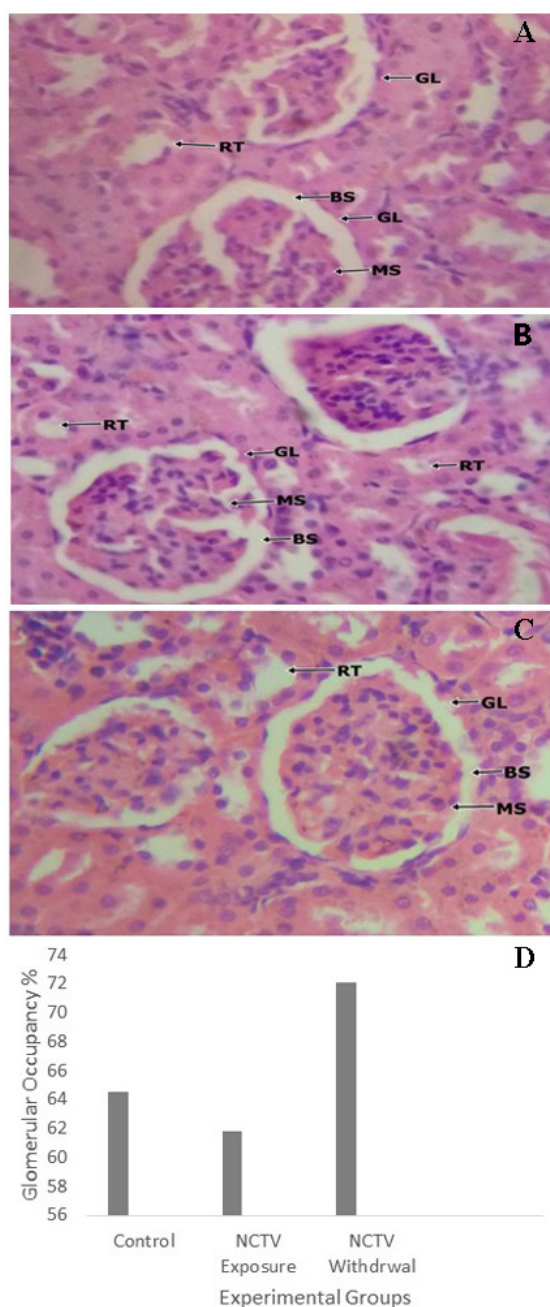


Fig. 1. Integrated histopathological and morphometric analysis of rat kidney tissue. (A) Control group showing normal glomerular architecture (B) 90-day NCTV exposure group showing glomerular atrophy and congestion (C) 90-day withdrawal group showing compensatory hypertrophy restorative remodeling of the renal parenchyma. (D) Quantitative morphometric analysis of glomerular occupancy (%), data illustrate a reduction in occupancy during NCTV exposure followed by an adaptive expansion during the withdrawal period. (All micrographs: H&E, $\times 400$) Abbreviations: RT, Renal Tubule; GL, Glomerulus; BS, Bowman's Space; MS, Mesangium.

Discussion

The present study investigated the effects of withdrawal from prolonged nitrocellulose thinner vapor (NCTV) exposure on renal biochemical markers and kidney histoarchitecture in male Wistar rats. This dual approach provides insight into both functional and structural implications of NCTV-induced nephrotoxicity and the potential for renal recovery following cessation of exposure.

Biochemically, serum electrolyte levels (Na^+ , Cl^- , and K^+) were slightly elevated in rats exposed to NCTV for 90 days compared with controls, but these differences were not statistically significant ($p > 0.05$). The lack of significant electrolyte disturbances suggests that NCTV exposure, while affecting glomerular filtration, did not markedly impair tubular ion transport processes. This observation aligns with established toxicological profiles, in which tubular reabsorptive mechanisms often remain resilient during transient glomerular stress (Radi, 2019; Skorecki et al., 2020). Electrolyte homeostasis is highly regulated by renal tubular function, and the mild, non-significant deviations observed in this study may indicate subclinical tubular stress rather than overt failure. This suggests preservation of renal functional reserve, with tubular ion transport mechanisms adapting to maintain homeostasis despite NCTV-induced glomerular injury (Ronco et al., 2019; Jufar et al., 2020).

In contrast, serum urea and creatinine levels were significantly elevated in the NCTV-exposed group compared with the control group. This indicates a reduction in glomerular filtration rate (GFR) and compromised renal excretory function (Adetunji et al., 2025). These biochemical findings are quantitatively corroborated by morphometric analysis (Fig. 1D), which revealed a significant contraction of the glomerular tuft and a decrease in the glomerular occupancy ratio to 61.8%. Elevations in these markers are consistent with impaired renal clearance resulting from toxicant-induced injury to glomerular and tubular structures, a phenomenon widely employed as an experimental indicator of renal dysfunction in Wistar rat models (Adetunji et al., 2025; Al-Mohamadi et al., 2025). These findings are corroborated by experimental studies of solvent and nephrotoxic exposures, which have similarly documented increases in serum waste products linked to oxidative injury and glomerular dysfunction (Hsu et al., 2025). Collectively, this supports the notion that chemical vapours can disrupt renal filtration processes by inducing oxidative stress-mediated damage to the glomerular filtration barrier (Abubakar et al., 2025).

Mechanistically, oxidative stress has emerged as

a central contributor to the renal toxicity of a wide range of environmental contaminants, including organic solvents and toxic vapors (Hsu et al., 2025). Chronic or subchronic exposure to toxicants increases the production of reactive oxygen species (ROS), leading to lipid peroxidation, mitochondrial dysfunction, and depletion of endogenous antioxidant defenses (Jomova et al., 2023; Abubakar et al., 2025). While the current study focused on the structural and functional reversibility of renal injury, the biochemical mechanisms driving these alterations are closely linked to the organ's specific antioxidant status. Previous investigations into nitrocellulose thinner (NCT) toxicity have consistently demonstrated that renal impairment is mediated by a significant depletion of superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) alongside increased lipid peroxidation (Uboh et al., 2014). The glomerular contraction and basement membrane distortions observed in our NCTV-exposed group likely stem from this oxidative pressure, which disrupts cellular integrity and triggers the documented histopathological changes. This redox imbalance disrupts cellular homeostasis and contributes to both tubular epithelial and glomerular injury (Radi, 2019; Arendshorst et al., 2024). Such pathways likely underpin the significant rise in urea and creatinine observed in the NCTV-exposed rats, as impaired cell membrane function and compromised nephron integrity directly reduce the GFR. Furthermore, elevated oxidative stress may initiate pro-apoptotic signaling and inflammatory cascades, exacerbating structural kidney damage (Radi, 2019; Abubakar et al., 2025).

Remarkably, withdrawal from NCTV exposure resulted in significant normalization of serum urea and creatinine, which were no longer significantly different from controls. While the NCTV-exposed group showed moderate glomerular atrophy and altered mesangial architecture (**Fig. 1B**), the withdrawal group demonstrated compensatory hypertrophy, characterized by an increase in glomerular occupancy to 72.1% (**Fig. 1D**). This structural expansion suggests a robust adaptive response where the surviving glomeruli undergo hypertrophy to restore filtration capacity. Such a recovery pattern suggests that the NCTV-induced damage was functional and manageable. However, instead of the renal structure returning to the native state, it exercised an inherent regenerative and compensatory capacity, thereby, remodeling the nephron architecture to maintain homeostasis before the onset of permanent fibrosis or scarring (Maremonti et al., 2022; Naved et al., 2022). This adaptation explains the divergence between normalized biochemical parameters and the persistent structural

changes observed in the withdrawal group.

Conclusion

This study demonstrates that subchronic NCTV exposure induces significant renal dysfunction. This was evidenced by elevated serum urea and creatinine levels as well as measured glomerular atrophy. Our findings establish that these insults are functionally reversible at the subchronic stage, as a 90-day withdrawal period allows for biochemical normalization facilitated by compensatory glomerular hypertrophy. This inherent regenerative and compensatory capability of the kidney can prevent progression from functional stress to irreversible, permanent damage if exposure is terminated in a timely manner.

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