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Research Article

Subchronic exposure to nitrocellulose thinner vapour induces persistent testicular dysfunction and reversible prostatic stress in male Wistar rats

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Abstract

Nitrocellulose thinner (NCT) is a widely used industrial solvent, yet the extent of its reproductive toxicity and the potential for recovery following cessation of exposure remain poorly understood. This study aimed to evaluate the subchronic effects of NCT vapor inhalation on the hypothalamic–pituitary–gonadal (HPG) axis, sperm quality, and prostatic integrity, as well as to determine the capacity for functional and structural recovery after a withdrawal period. Male Wistar rats were exposed to NCT vapor for 90 days, followed by a 90-day recovery (withdrawal) period. Serum levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH), testosterone, acid phosphatase (ACP), and prostate-specific antigen (PSA) were quantified. Sperm quality indices (count, motility, viability) and morphological defects were assessed, alongside histopathological examination of the prostate and testes. Subchronic NCTV exposure induced primary testicular dysfunction, characterized by significantly ($p < 0.05$) elevated FSH (0.74 ± 0.05 IU/L) and LH (0.55 ± 0.05 IU/L) alongside reduced testosterone (2.80 ± 0.47 ng/mL). Prostatic stress was evidenced by increased ACP and PSA levels and epithelial hyperplasia. Sperm quality was severely impaired, with a significant rise in total defects (15.50 ± 2.47%). Following withdrawal, prostatic markers and glandular architecture normalized, demonstrating significant regenerative potential. However, the HPG axis transitioned to secondary hypogonadotropic hypogonadism, with FSH, LH, and testosterone (0.71 ± 0.09 ng/mL) failing to recover. Furthermore, sperm parameters remained significantly lower than control levels, suggesting persistent damage to the germinal epithelium and spermatogonial stem cells. NCTV induces widespread reproductive toxicity. While prostatic damage is reversible upon withdrawal, testicular and endocrine impairments are persistent, likely due to oxidative injury and neuroendocrine toxicity. These findings highlight the significant long-term reproductive risks associated with industrial solvent exposure.

Keywords: Nitrocellulose Thinner, Hypothalamic-Pituitary-Gonadal Axis, Testosterone, Sperm Morphology, Prostatic Hyperplasia.

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Introduction

Nitrocellulose thinner (NCT) is a widely used industrial solvent employed in furniture finishing, spray painting, and coating processes. Commercial formulations typically comprise mixtures of volatile organic compounds (VOCs), including aromatic hydrocarbons such as toluene and ethylbenzene, as well as acetate esters such as butyl acetate. These compounds readily volatilize during use, contributing to indoor air contamination and increased occupational exposure in paint and manufacturing environments [1 - 2]. Of particular concern are volatile aromatic hydrocarbons within the benzene–toluene–ethylbenzene–xylene (BTEX) group, which are widely utilized industrially and have been associated with adverse effects on multiple physiological systems [3].

Exposure to organic solvent vapours occurs predominantly via inhalation, which represents the principal route of occupational exposure to these volatile compounds [4]. In Nigeria, studies among spray painters have reported significant health risks associated with such exposures, highlighting the need for further investigation into their long-term physiological consequences [5]. Upon inhalation, these lipophilic compounds rapidly diffuse across the alveolar membranes into systemic circulation and are distributed to highly perfused organs, including the liver, kidneys, brain, and reproductive tissues. Hepatic biotransformation of these solvents may generate reactive intermediates capable of inducing oxidative stress, cellular damage, and systemic toxicity [6].

Accumulating evidence suggests that exposure to organic solvents adversely affects reproductive health in both males and females [7]. In males, however, the disruption of androgenic hormones and prostate function remains insufficiently explored. Epidemiological and experimental studies have linked occupational exposure to solvent mixtures with reproductive dysfunction and endocrine disruption [8]. These endocrine-disrupting chemicals interfere with male fertility through complex physiological and molecular mechanisms [9]. Specifically, solvents such as toluene, benzene, and xylene have been reported to impair endocrine signaling and reproductive function [10].

In addition, inhalation of solvent mixtures has been associated with reductions in key reproductive hormones, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and testosterone, indicating disruption of the hypothalamic–pituitary–gonadal axis. Given that the prostate is an androgen-dependent organ, alterations in hormonal balance may consequently impair prostate function and secretory activity [11]. Such dysfunction is often reflected by changes in prostate biomarkers, including prostate-specific antigen (PSA) and prostatic acid phosphatase (ACP), which serve as important indicators of prostate physiological status [12].

Despite increasing concern regarding the toxicological effects of solvent exposure, there is limited information on the reversibility of reproductive alterations following cessation of exposure, particularly with respect to nitrocellulose thinner vapour (NCTV). Understanding the extent to which endocrine and reproductive disturbances recover after withdrawal is essential for accurate occupational risk assessment and the development of effective preventive strategies. Therefore, this study aimed to investigate the effects of withdrawal following chronic exposure to nitrocellulose thinner vapour on selected reproductive hormones and prostate biomarkers (PSA and ACP) in male Wistar rats.

Methods

Chemicals and reagents

All chemicals and reagents used in this study were of analytical grade. Enzyme-linked immunosorbent assay (ELISA) kits for testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), prostate-specific antigen (PSA), progesterone, and oestradiol were obtained from Monobind Inc. (Lake Forest, CA, USA). Acid phosphatase (ACP) assay reagents were of analytical grade. Other reagents included chloroform, deionized water, and appropriate buffer solutions.

Nitrocellulose thinner

Nitrocellulose thinner (NCT) of the Abro® brand was purchased from a chemical retail outlet located at Timber Market, Marian Road, Calabar, Cross River State, Nigeria.

Experimental animals and study design

Twenty-four (24) male Wistar rats weighing 100–110 g was obtained from the Animal House, Department of Biochemistry, University of Calabar, Nigeria. The animals were housed in standard laboratory cages under controlled environmental conditions (temperature: 25 ± 2°C; relative humidity: 50–62%; 12 h light/12 h dark cycle) and allowed free access to standard feed and water *ad libitum*.

The study was conducted over a period of approximately six (6) months, comprising a two-week acclimatization period, a 90-day exposure phase, and a 90-day withdrawal (recovery) phase.

The animals were randomly assigned into three groups (n = 8 per group):

- **Group I (Control):** No exposure to NCTV
- **Group II (Exposure):** Exposed to NCTV for 90 days
- **Group III (Recovery):** Exposed to NCTV for 90 days followed by a 90-day withdrawal period

Biochemical and histological analyses were conducted at the end of the exposure and recovery periods, respectively.

Ethical approval

All experimental procedures were carried out 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). Efforts were made to minimize animal suffering and to reduce the number of animals used.

Mode of exposure

Exposure to nitrocellulose thinner vapour (NCTV) was carried out using a modified whole-body inhalation method as previously described [13]. The exposure chambers consisted of locally fabricated glass-enclosed compartments (150 cm x 90 cm x 210 cm). Two highly perforated 1000 mL containers, each containing 500 mL of nitrocellulose thinner, were placed within the chamber to allow passive volatilization. Animals were exposed to the vapour for 4 hours daily (9:00 am to 1:00 pm). The chambers were adequately ventilated to ensure uniform vapour distribution and to prevent excessive accumulation. After daily exposure, animals were transferred to a fume-free section of the animal facility.

Determination of serum hormone levels

Testosterone, FSH, and LH

Serum concentrations of testosterone, FSH,

and LH were determined using microwell ELISA kits (Monobind Inc., USA) according to the manufacturer's instructions. Testosterone was measured using a competitive ELISA method, while FSH and LH were determined using the sandwich ELISA principle.

Briefly, serum samples were incubated in antibody-coated microwells, followed by washing steps to remove unbound components. Tetramethylbenzidine (TMB) substrate was added for colour development, and the reaction was stopped using a stop solution. Absorbance was read at 450 nm using a microplate reader.

The assays were validated for rat serum using spike-recovery and parallelism tests, confirming the absence of significant matrix interference. All samples were analyzed in duplicate to ensure intra-assay precision.

Prostate-specific antigen (PSA)

Serum PSA levels were determined using ELISA kits (Monobind Inc., USA) following the manufacturer's protocol.

Determination of serum acid phosphatase (ACP)

Serum ACP activity was determined using a colorimetric method based on the hydrolysis of disodium phenyl phosphate under acidic conditions. The liberated phenol reacts with 4-aminoantipyrine to form a red-coloured complex, which was measured spectrophotometrically at 520 nm.

Sperm quality assessment

Sperm parameters, including concentration, motility, viability, and morphology, were evaluated according to standardized protocols [14]. The cauda epididymis was excised and minced in 5 mL of phosphate-buffered saline (PBS) at 37°C to release spermatozoa. The suspension was incubated for 10 minutes to allow proper dispersion.

Sperm count was determined using an improved Neubauer hemocytometer under a light microscope at ×400 magnification and expressed as $\times 10^6$ cells/mL. Motility was assessed by evaluating 200 spermatozoa per sample and expressed as the percentage of motile cells.

Viability was determined using the 1% eosin exclusion method, where unstained cells were considered viable. Morphological assessment was performed on stained smears under oil immersion (×1000 magnification), evaluating abnormalities of the head, mid-piece, and tail, and expressed as the percentage of abnormal spermatozoa.

Histological examination

Reproductive tissues were excised immediately after sacrifice and fixed in 10% neutral buffered formalin. The tissues were processed using standard histological techniques, embedded in paraffin wax, sectioned at 5 µm thickness, and stained with hematoxylin and eosin (H&E). The stained sections were examined under a light microscope for histopathological alterations.

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. Differences were considered statistically significant at $p < 0.05$.

Results and Discussion

The present study investigated the effects of

subchronic exposure to nitrocellulose thinner vapour (NCTV) and its withdrawal on the hypothalamic–pituitary–gonadal (HPG) axis and reproductive organ integrity. To address the primary goal of determining the extent of NCTV-induced reproductive toxicity, we first evaluated the endocrine profile of the subjects.

Impact of NCTV exposure and withdrawal on serum gonadotropic hormones and testosterone

Subchronic exposure to NCTV for 90 days resulted in a significant ($p < 0.05$) increase in serum follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels (0.74 ± 0.05 IU/L and 0.55 ± 0.05 IU/L, respectively) compared with the control group. Conversely, serum testosterone levels were significantly ($p < 0.05$) decreased after 90 days of exposure (2.80 ± 0.47 ng/mL) (Table 1).

Table 1: Serum levels of gonadotropic hormones and testosterone in male wistar rats following exposure to and withdrawal from NCTV.

Parameter	Control	NCTV 90-day Exposure	NCTV 90 day Withdrawal
FSH (IU/L	0.63 ± 0.08	0.74 ± 0.05*	0.43 ± 0.07 *,a
LH (IU/L	0.35 ± 0.02	0.55 ± 0.05*	0.24 ± 0.03*,a
TEST.(ng/mL	3.70 ± 0.81	2.80 ± 0.47*	0.71 ± 0.09*,a

Values are expressed as mean ± SD (n = 8). $p < 0.05$ vs control, a = $p < 0.05$ vs 90-day exposure group FSH- Follicle Stimulating Hormone: LH – Luteinizing Hormone; TEST. –Testosterone.

The elevation in gonadotropins alongside reduced testosterone indicates a disruption of the HPG axis consistent with primary testicular dysfunction. However, during the withdrawal phase, FSH and LH declined to sub-baseline levels (0.43 ± 0.07 IU/L and 0.24 ± 0.03 IU/L, respectively), while testosterone suffered a further significant reduction (0.71 ± 0.09 ng/mL. This shift implies a transition to secondary hypogonadotropic hypogonadism, suggesting that prolonged NCTV exposure may impair hypothalamic–pituitary signaling, potentially through neuroendocrine toxicity [15 - 16]. The lack of testosterone recovery further points to sustained damage to Leydig cells or oxidative injury affecting key enzymes such as 17-beta-hydroxysteroid dehydrogenase. This aligns with findings by Yang et al. [17] who linked sustained androgen suppression to oxidative injury in steroidogenic pathways.

Effect of NCTV exposure and withdrawal on prostatic function and histology

To evaluate the impact on accessory reproductive organs, prostatic markers and architecture were assessed. Exposure resulted in a significant ($p < 0.05$) increase in serum acid phosphatase (ACP) activity (25.22 ± 1.20 IU/L) and prostate-specific antigen (PSA) levels (6.61 ± 1.83 ng/dL) (Table 2).

Table 2: Serum prostate-specific antigen (PSA) and acid phosphatase (ACP) levels in male wistar rats following exposure to and withdrawal from NCTV.

Parameter	Control	NCTV 90-day Exposure	NCTV 90-day withdrawal
ACP (IU/L)	18.28 ± 1.55	25.22 ±1.20*	16.84 ± 0.95[a]
PSA(ng/dL)	3.73 ± 0.75	6.61 ± 1.83*	3.89 ± 0.15[a]

Values are expressed as mean ± SD (n = 8). $p < 0.05$ vs control, a = $p < 0.05$ vs 90-day exposure group PSA-Prostate Specific Antigen (PSA). ACP - Acid Phosphatase.

Histological analysis of the control group showed normal glandular acini (Figure 1). In contrast, NCTV exposure caused marked structural alterations, including epithelial hyperplasia and reduced luminal secretions,

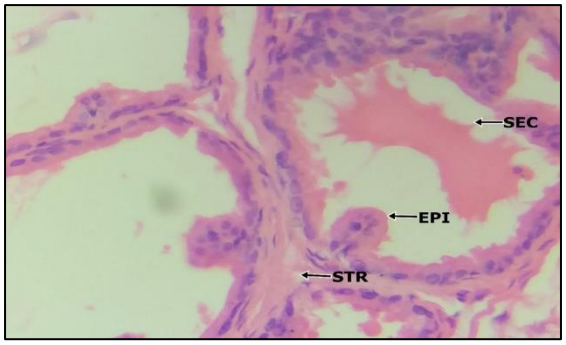

Figure 1: Photomicrograph of prostatic tissue from control rats showing normal glandular architecture with well-defined acini, intact epithelial lining, and abundant luminal secretions (H&E stain, ×400). (EPI – Epithelial Lining, STR – Stroma, SEC – Secretory material).

Figure 2 indicating significant prostatic stress [18 - 19]. Notably, following withdrawal, ACP and PSA levels normalized.

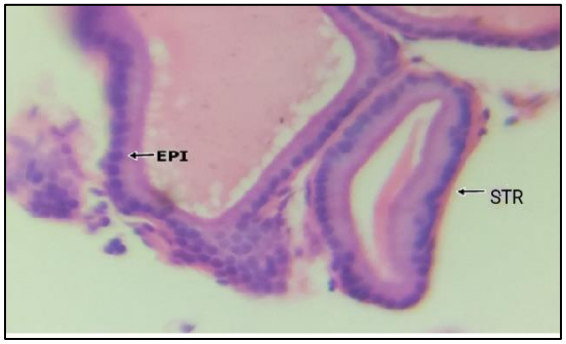

Figure 2: Photomicrograph of prostatic tissue from rats exposed to NCTV for 90 days showing reduced acinar size and disorganized epithelial lining. Note the depletion of secretions (H&E stain, ×400). (EPI – Epithelial Lining, STR – Stroma).

This was mirrored by the restoration of glandular architecture in Figure 3, suggesting that the prostatic tissue retains substantial regenerative potential once the toxicant is removed [20].

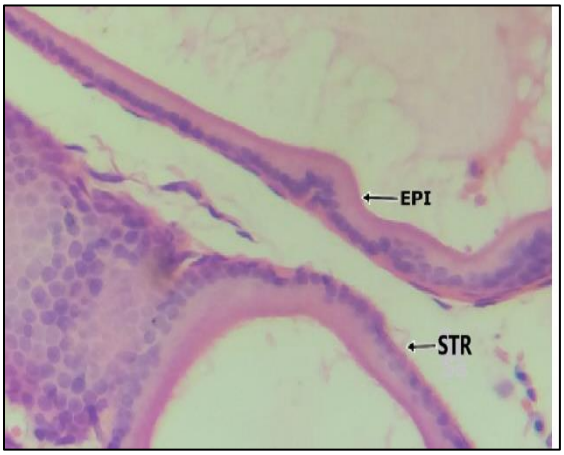

Figure 3: Photomicrograph of prostatic tissue following 90-day withdrawal showing significant restoration of glandular architecture and secretory material (H&E stain, ×400). (EPI – Epithelial Lining, STR – Stroma).

Impact on sperm quality and morphological characteristics

Sperm quality indices showed a significant ($p < 0.05$) reduction in motility (55.50 ± 3.10%), viability (33.75 ± 2.45%), and count (47.00 ± 3.22 × 10^6 cells/mL) after 90 days of exposure (Table 3).

Table 3: Effects of NCTV exposure and withdrawal on sperm quality indices in male Wistar rats.

Parameter	Control	NCTV 90-day Exposure	NCTV 90-day withdrawal
Sperm Motility (%)	80.00 ± 4.22	55.50 ± 3.10*	62.75 ± 3.85 [a]
Non-motile (%)	20.00 ± 1.85	45.50 ± 3.42*	37.25 ± 2.90[a]
Sperm Viability (%)	50.50 ± 3.14	33.75 ± 2.45*	42.00 ± 3.05[a]
Non-viable (%)	49.50 ± 2.88	66.25 ± 4.10*	58.00 ± 3.12[a]
Sperm pH	6.8	6.7	6.8
Sperm Count (x106 cells/mL)	62.00 ± 4.15	47.00 ± 3.22*	53.00 ± 3.75[a]

Values are expressed as mean ± SD (n = 8). $p < 0.05$ vs control, a = $p < 0.05$ vs 90-day exposure group.

These findings align with evidence that organic toxicants compromise sperm quality through oxidative stress [21]. Furthermore, total sperm defects increased from 6.00 ± 0.98% to 15.50 ± 2.47%, with tail defects being the most prominent (Table 4).

Such structural abnormalities, particularly in the tail, suggest that NCTV-induced oxidative injury interferes with the integrity of the sperm cytoskeleton and mitochondrial function during the late stages of spermiogenesis [22].

Table 4: Effect of NCTV exposure and withdrawal on sperm morphological characteristics in male wistar rats.

Parameter	Control	NCTV 90-day Exposure	NCTV 90-day Withdrawal
Head defect (%)	2.00 ± 0.35	5.25 ± 0.82*	4.50 ± 0.64[a]
Middle piece defect (%)	2.50 ± 0.41	4.00 ± 0.55*	3.25 ± 0.48[a]
Tail defect (%)	1.50 ± 0.22	6.25 ± 1.10*	4.50 ± 0.75[a]
Total defect (%)	6.00 ± 0.98	15.50 ± 2.47*	12.25 ± 1.87[a]

Values are expressed as mean ± SD (n = 8), $p < 0.05$ vs control. a = $p < 0.05$ vs 90-day exposure group.

While withdrawal resulted in significant improvements, values remained lower than the control. This incomplete restoration, despite a recovery period spanning multiple spermatogenic cycles, suggests persistent alterations in the germinal epithelium or damage to spermatogonial stem cells [23 - 24].

Testicular architecture and mechanistic discussion

The histopathological assessment of the testes provided structural evidence for the functional declines observed. While the control showed normal architecture (Figure 4), the 90-day exposure group exhibited thinning of the germinal epithelium and depleted lumina (Figure 5). Crucially, the withdrawal group (Figure 6) showed persistent alterations, including collapsed tubules and thickened basement membranes.

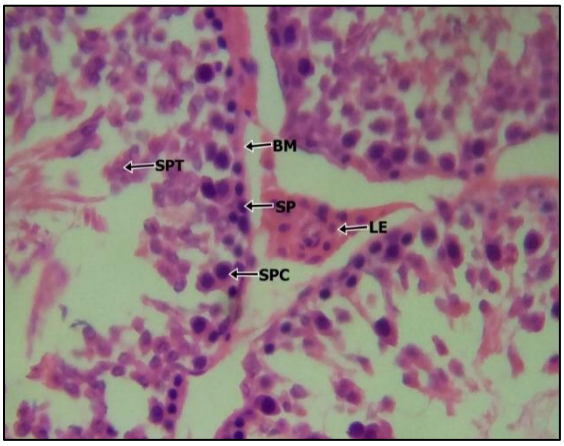

Figure 4: Photomicrograph of control testicular tissue showing normal seminiferous tubule architecture (H&E stain, ×400). (SPT – Seminiferous tubules, BM – Basement membrane, WristerSP – Spermatocytes, LE – Lumen, SPC – Spermatogonia cells).

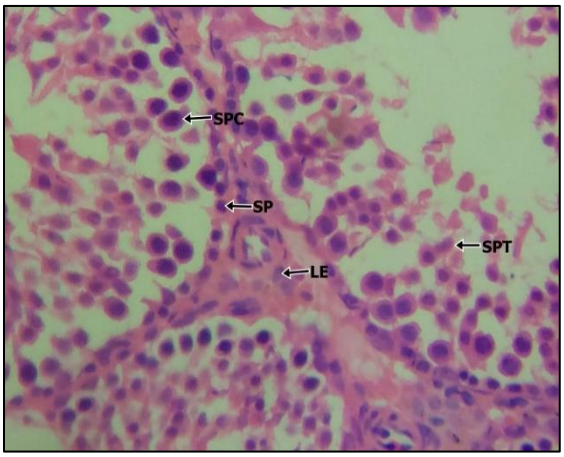

Figure 5: Photomicrograph of testicular tissue exposed to NCTV for 90 days showing disrupted architecture and depleted luminal contents (H&E stain, ×400). (SPT – Seminiferous tubules, SP – Spermatocytes, LE – Lumen, SPC – Spermatogonia cells).

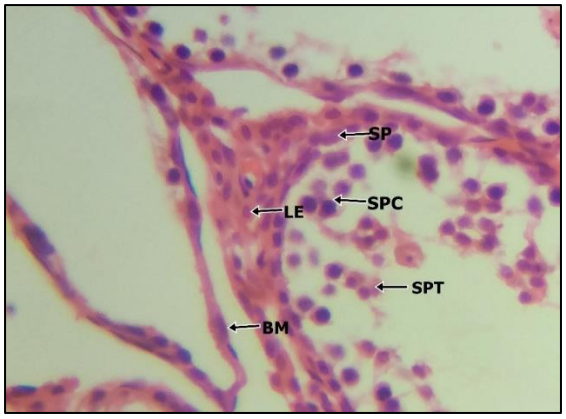

Figure 6: Photomicrograph of testicular tissue following withdrawal showing collapsed tubules and persistent depletion of luminal contents (H&E stain, ×400). **(**SPT – Seminiferous tubules, BM – Basement membrane, SP – Spermatocytes, LE – Lumen, SPC – Spermatogonia cells).

The differential recovery between the testis and prostate observed in this study likely reflects fundamental differences in cellular turnover and the unique vulnerability of the blood–testis barrier [25]. While the prostatic epithelium possesses a robust regenerative capacity, the complex architecture of the seminiferous tubules is more susceptible to permanent disruption.

Mechanistically, NCTV-induced oxidative stress may trigger ferroptosis—an iron-dependent form of regulated cell death—leading to the irreversible loss of germinal cells and the sustained endocrine suppression seen in the withdrawal group [26]. In conclusion, this study fulfills its initial objective by demonstrating that while NCTV induces widespread reproductive toxicity, the prostatic damage is reversible, whereas testicular and endocrine impairments are persistent.

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Conflict of interest

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

References

1. Li X, Zhang Y, Wang L. Occupational exposure to industrial solvents and male reproductive health: A systematic review. J Occup Health. 2024; 66(1):102-115.
2. Růžičková K, Novakova M, Prochazka J. Volatile organic compounds and the hypothalamic-pituitary-gonadal axis. Environ Toxicol Pharmacol. 2025;108:104432.
3. Ghahri S, Mohammadi S, Azari M. Impact of nitrocellulose thinner on endocrine function in industrial workers. Int J Hyg Environ Health. 2024;255:114290.
4. Khoshakhlagh AH, Ghasemi M, Chiamulera C. Neuroendocrine toxicity of solvent mixtures: Mechanisms and clinical implications. Toxicol Lett. 2025;392:45-58.
5. Ojo AO, Adienbo OM, Emudainohwo JO. Evaluation of the effect of nitrocellulose thinner on the reproductive hormones of male Wistar rats. J Pharm Biol Sci. 2020;15(4):34-40.
6. Dong J, Zhuang X. Chemical composition and toxicity profiles of commercial paint thinners. Chemosphere. 2024;341:140021.
7. Yang L, Zhou J, Chen H. General impacts of environmental toxicants on male reproductive outcomes. Reprod Toxicol. 2024;123:88-95.
8. Dutta S, Sengupta P, Slama P. Oxidative stress and the HPG axis: A detailed mechanistic approach. Cells. 2023;12(11):1502.
9. Lahimer M, Ben Saïd M, Ghorbel F. Chronic exposure to solvent vapors and testicular oxidative injury. Environ Sci Pollut Res. 2023;30:11245-11258.
10. Suaidi MH, Rahman AA, Bakar NA.

Toluene and xylene induced hormonal imbalance in animal models. Toxicology. 2022;476:153241.
11. Banerjee A, Singh A, Srivastava S. Evaluating prostate-specific antigen as a biomarker of toxicant-induced prostatic stress. Biomarkers. 2018; 23(5):412-420.
12. Elechi-Amadi K, Azuonwu O, Amadi PN. Sub-chronic vapor exposure and accessory sex organ function. J Reprod Biol Health. 2024;12:15-22.
13. Uboh FE, Ebong PE, Akpan HD, et al. Effects of vitamins C and E on gasoline vapor induced reproductive toxicity in male rats. Nig J Physiol Sci. 2005;20(1-2):43-47.
14. Seed J, Chapin RE, Clegg ED, et al. Methods for assessing health risks in children relevant to specific exposure scenarios: Reproductive toxicity. Environ Health Perspect. 1996; 104(S3):455-466.
15. Abdulai A, Mensah K, Boateng F. Neuroendocrine signaling disruption following prolonged solvent inhalation. NeuroToxicology. 2025;105:22-31.
16. van Os M, Peters R, de Vries S. Secondary hypogonadism in the recovery phase of chemical exposure. Endocrine. 2025;87(2):315-324.
17. Yang Q, Zhao D, Sun R. 17-β-Hydroxysteroid dehydrogenase inhibition and Leydig cell senescence under oxidative stress. J Biol Chem. 2025;301:105672.
18. Balakrishna S, Reddy P, Gupta S. Prostatic epithelial hyperplasia and the role of industrial pollutants. Toxicol Ind Health. 2021;37(8):489-501.
19. Singh R, Kumar M, Sharma A. Acid phosphatase as a marker for prostatic secretory dysfunction in chemical exposure. Life Sci. 2024;336:122310.
20. Korpal S, Jaddou H. Prostatic regeneration and recovery kinetics following chemical insult. J Appl Toxicol. 2025;45(3):210-222.
21. Marić D, Fučić A, Aghayanian G. Oxidative stress and sperm quality: From molecular mechanisms to clinical evidence. Antioxidants. 2021;10(12): 1954.
22. Nowicka-Bauer K, Nixon B. Molecular changes in the sperm cytoskeleton and mitochondria during spermiogenesis. Int J Mol Sci. 2020;21(11):4009.
23. Jagadesh N, Sridharan B. Spermatogonial stem cell niche damage following subchronic toxicant exposure. Stem Cell Res Ther. 2026; 17:44-56.
24. Mathur A, Gupta P, Jain S. Germinal epithelium restoration and spermatogenic cycles: A recovery study. Fertil Steril. 2023;120(4):812-825.
25. Li J, Wang H, Liu Y. Dynamics of the blood-testis barrier and organ-specific cellular turnover. Nat Rev Urol. 2024;21:156-170.
26. Chen Z, Xu M, Li L. Ferroptosis: A novel mechanism of germinal cell loss in environmental toxicology. Cell Death Dis. 2025;16:112-125.