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1-Nitropyrene exposure induces oxido-inflammatory stress in the renal tissues of Wistar rats

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ABSTRACT

Background: The mechanism underlying 1-nitropyrene (1-NP)- induced kidney damage remains largely unclear.
Methods: This study examined renal oxido-inflammatory stress in 32 adult male Wistar rats (8 per group) administered graded oral doses of 62.5, 125, or 250 mg/kg body weight 1-NP daily for seven consecutive days. A vehicle control group received corn oil (2 mL/kg body weight).
Results: 1-NP administration induced a marked, dose-dependent reduction in body weight gain, with a significant increase in kidney weight at the highest dose (250 mg/kg body weight). Renal function was impaired, as evidenced by significant dose-dependent elevations in serum urea and creatinine levels. Furthermore, 1-NP exposure significantly reduced the activities of catalase, superoxide dismutase, glutathione-S-transferase, and glutathione peroxidase, and depleted reduced glutathione levels, while concurrently increasing lipid peroxidation and reactive oxygen and nitrogen species. Significant increases in nitric oxide concentration and myeloperoxidase activity confirmed the induction of renal inflammation. These biochemical findings were corroborated by histopathological evidence of glomerular degeneration, focal inflammatory infiltration, and vascular congestion in the renal cortex.
Conclusion: These findings provide experimental evidence that 1-NP induces dose-dependent renal injury through an oxido-inflammatory mechanism in male Wistar rats, with implications for the nephrotoxic risks of environmental nitropyrene exposure.

Keywords: 1-nitropyrene, renal toxicity, xenobiotics, oxido-inflammatory stress, public health.

INTRODUCTION

Urbanization and industrialization have substantially increased the release of xenobiotics, toxins, and environmental pollutants, particularly through the combustion of fossil fuels (1, 2). Nitro-aromatic compounds, including 1-nitropyrene (1-NP), are generated during the incomplete combustion of fossil fuels and other organic materials (3). Environmental 1-NP is predominantly associated with diesel exhaust fumes from motor vehicles and industrial generators and has also been detected in foodstuffs, road dust particles, and open-fire grilled foods (3, 4). Human exposure to 1-NP typically occurs via inhalation, ingestion of contaminated food and water, and dermal contact (5). Several studies have reported that 1-NP is metabolically activated to reactive intermediates that bind covalently to DNA and proteins, forming adducts implicated in hepatotoxicity, pulmonary injury, renal toxicity, cardiovascular diseases, and the induction of oxidative, nitrosative, and inflammatory stress (3, 6, 7, 8).

The induction of lipid peroxidation, protein oxidation, and depletion of both enzymatic and non-enzymatic antioxidants are hallmarks of excessive reactive intermediate production (8, 9). Under physiological conditions, endogenous antioxidant enzymes including catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GPX) maintain redox homeostasis by scavenging reactive oxygen intermediates (10). The detoxification of 1-NP depends on glutathione-S-transferase (GST), which conjugates its reactive metabolites with reduced glutathione (GSH), thereby neutralizing their electrophilic toxicity (10). Ikponmwosa-Eweka and Maduako (2025) recently reported the involvement of GST in 1-NP-induced lipid peroxidation (LPO), reactive oxygen and nitrogen species (RONS) generation, nitric oxide (NO) production, and myeloperoxidase (MPO) activity in hepatopulmonary tissue (3).

Despite these advances, the renal consequences of 1-NP exposure remain poorly characterized. The kidney is a principal site of xenobiotic accumulation and detoxification, rendering it particularly susceptible to oxidative and inflammatory insult; yet, no study to date has specifically examined the dose-dependent nephrotoxic potential of 1-NP. This knowledge gap is clinically significant given the widespread human exposure to 1-NP from diesel-contaminated environments in developing nations. Accordingly, the present study aimed to investigate the acute dose-dependent effects of 1-NP on renal function, oxidative stress indices, inflammatory markers, and histological architecture in male Wistar rats.

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MATERIALS AND METHODS

Reagents

1-Nitropyrene (1-NP), 5,5′-dithiobis (2-nitrobenzoic acid), 1-chloro-2,4-dinitrobenzene, o-dianisidine, and thiobarbituric acid were purchased from Sigma-Aldrich (St. Louis, MO, USA). Urea and creatinine assay kits were obtained from Randox Laboratories (Crumlin, UK). All other reagents were of analytical grade and sourced from BDH Chemicals (Poole, UK).

Animal and experimental setup

Thirty-two adult male Wistar rats (8–10 weeks of age, body weight 185–190 g) were obtained from the animal facility of the Department of Veterinary Medicine, University of Ibadan, Nigeria. Animals were housed in groups of four per cage under controlled environmental conditions (temperature: 22 ± 2°C; relative humidity: 50 ± 5%; 12-hour light/dark cycle) and provided with standard rat chow and fresh water ad libitum throughout the study. Following a seven-day acclimatization period, animals were randomly assigned to four experimental groups (n = 8 per group) using a simple randomization procedure. Sample size was determined on the basis of similar published studies (3, 9). Animals were monitored daily for signs of pain, distress, or adverse reactions. All experimental procedures were conducted in accordance with the NIH Guide for the Care and Use of Laboratory Animals and received ethical approval from the Benson Idahosa University Animal Care and Use Research Ethics Committee (File No.: BIU-ACUREC/013–112/14). Every effort was made to minimize the number of animals used and to reduce pain and distress throughout the experimental period.

Animal groupings and sample collection

Animals were allocated into four groups and treated by oral gavage daily for seven consecutive days as shown in Table 1.

Table 1. Experimental groups, treatment, dose, route and duration

Groups	Treatments	Dose	Route	Duration	n
I (Control)	Corn oil (vehicle)	2 mL/kg BW	Oral gavage	7 days	8
II	1-NP (low dose)	62.5 mg/kg BW	Oral gavage	7 days	8
III	1-NP (mid dose)	125 mg/kg BW	Oral gavage	7 days	8
IV	1-NP (high dose)	250 mg/kg BW	Oral gavage	7 days	8

BW: body weight; 1-NP: 1-nitropyrene; n: number of animals per group.

Final body weights were recorded 24 hours after the last treatment. Animals were then anesthetized by intraperitoneal injection of ketamine (80 mg/kg body weight) and xylazine (10 mg/kg body weight) prior to blood collection by cardiac puncture (11). Animals were humanely euthanized immediately following blood collection. Blood samples were allowed to clot and centrifuged at 3,000 × g for 10 minutes to obtain serum. Kidney tissues were excised, rinsed in ice-cold phosphate-buffered saline, and homogenized in 10 volumes of cold 0.1 M phosphate buffer (pH 7.4). The homogenate was centrifuged at 10,000 × g at 4°C for 15 minutes to obtain the post-mitochondrial supernatant for biochemical analyses.

Assessment of renal function biomarkers

Serum creatinine and urea concentrations were determined using commercially available Randox diagnostic kits (Randox Laboratories, UK) according to the manufacturer's instructions.

Assessment of kidney oxidative stress biomarkers

Total protein concentration in the renal supernatant was determined using the Bradford method (12). Catalase (CAT) and superoxide dismutase (SOD) activities were measured according to the methods of Claiborne (13) and Misra and Fridovich (14), respectively. Glutathione-S-transferase (GST) and glutathione peroxidase (GPX) activities were determined according to Habig et al. (15) and Rotruck et al. (16), respectively. Reduced glutathione (GSH) levels were assessed using the method of Jollow et al. (17). Malondialdehyde (MDA), a product of lipid peroxidation, was quantified by the thiobarbituric acid reactive substances method (18).

Determination of reactive oxygen and nitrogen species (RONS)

Renal RONS production was quantified based on the oxidation of 2′,7′-dichlorodihydrofluorescein diacetate (DCFH-DA) to fluorescent DCF, as previously described (9).

Assessment of renal inflammatory indices

Nitric oxide (NO) concentration was determined colorimetrically using the Griess reagent method (19). Myeloperoxidase (MPO) activity was quantified spectrophotometrically according to the protocol of Granell et al. (20).

Pathology assessment of kidney architecture

The kidney tissue samples were fixed in 10% neutral-buffered formalin for 72 hours, dehydrated through a graded ethanol series, cleared in xylene, and embedded in paraffin wax. Sections of 5 µm thickness were cut using a rotary microtome, mounted on glass slides, and stained with hematoxylin and

eosin (H&E). Sections were examined under an Olympus light microscope (21). Representative photomicrographs from each group were selected and captured by a certified pathologist who was blinded to the group assignments.

Statistical analysis

Prior to statistical testing, data normality was verified using the Shapiro-Wilk test, and homogeneity of variance was confirmed using Levene's test. Data are expressed as mean ± standard deviation (SD) and were analyzed using one-way analysis of variance (ANOVA) followed by Bonferroni's post hoc test for multiple comparisons (GraphPad Prism, version 8.0; GraphPad Software, San Diego, CA, USA). A P value of less than 0.05 was considered statistically significant.

RESULTS

Effects of 1-NP on the body and the kidney weights

Table 2 shows that 1-NP administration at 62.5 and 125 mg/kg body weight did not produce statistically significant alterations in body weight gain or kidney weight relative to the control group ($P > 0.05$). However, exposure to 250 mg/kg body weight 1-NP resulted in a significant reduction in body weight gain ($P < 0.05$) and a concomitant significant increase in kidney weight compared with the control group ($P < 0.05$), suggesting early renal edema or inflammatory engorgement.

Effects of 1-NP on the renal function biomarkers

In Table 3, shows a significant ($P < 0.05$), dose-dependent increase in serum urea and creatinine concentrations across all 1-NP-treated groups compared with the control group.

Effects of 1-NP on renal redox status indices

Figures 1 and 2 show that 1-NP exposure at all doses produced a significant ($P < 0.05$), dose-dependent reduction in renal CAT, SOD, GST, and GPX activities compared with the control group. A concomitant significant dose-dependent increase in LPO and RONS levels and a significant decrease in GSH concentration were observed in 1-NP-treated rats compared with controls.

Effects of 1-NP on renal inflammatory markers

As shown in Figure 3, oral administration of graded doses of 1-NP produced a significant ($P < 0.05$), dose-dependent increase in renal MPO activity and NO concentration compared with the control group.

Effects of 1-NP on renal histology of exposed rats

As shown in Figure 4, histopathological examination of H&E-stained renal sections from 1-NP-treated rats revealed dose-dependent morphological alterations. The control group exhibited normal renal architecture with intact glomeruli and tubular epithelium. Rats administered 62.5 mg/kg body weight 1-NP displayed mild inflammatory cell infiltration. At 125 and 250 mg/kg body weight, severe cortical deterioration, glomerular degeneration, hyperplasia, epithelial tubular damage, and disseminated vascular congestion were observed.

Table 2. Effect of 1-NP on body weight gain and kidney weight in Wistar rats following seven days of oral exposure

Parameters	Control	62.5 mg/kg	125 mg/kg	250 mg/kg
Body weight gain	20.01 ± 1.11	19.89 ± 0.35	19.96 ± 0.22	11.16 ± 0.21*
Kidney weight	2.00 ± 0.08	2.01 ± 0.16	1.98 ± 0.10	2.86 ± 0.17*

Values are expressed as mean ± SD; n = 8 per group. *Significantly different from control group; $P < 0.05$. BW: body weight.

Table 3. Effect of 1-NP on renal function indices in Wistar rats following seven days of oral exposure

Parameters	Control	62.5 mg/kg	125 mg/kg	250 mg/kg
Urea (mmol/L)	2.11 ± 0.95	9.04 ± 1.22*	13.49 ± 2.81*	26.27 ± 1.88*
Creatinine (µmol/L)	21.20 ± 3.09	49.54 ± 2.60*	65.81 ± 4.01*	81.18 ± 3.15*

Values are expressed as mean ± SD; n = 8 per group. *Significantly different from control group; $P < 0.05$. BW: body weight.

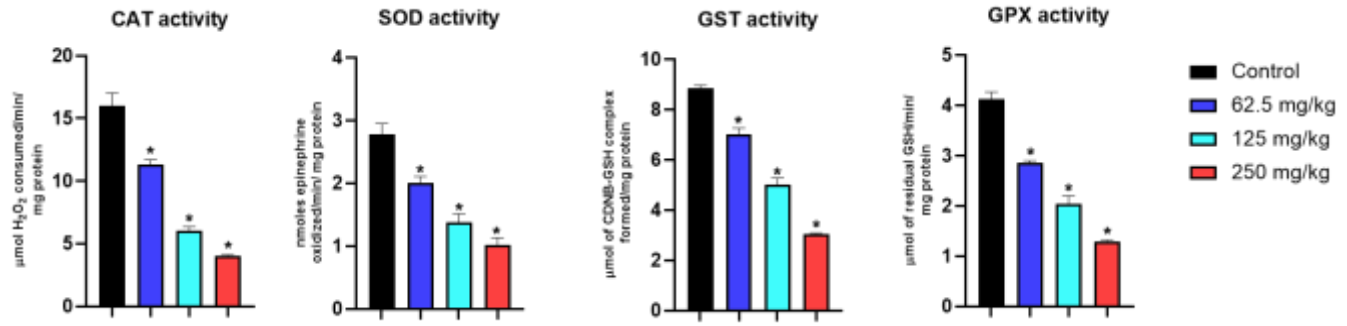


Figure 1. Effects of seven consecutive days of oral 1-NP exposure on renal antioxidant enzyme activities (CAT, SOD, GST, and GPX) in Wistar rats. Data are expressed as mean ± SD (n = 8 per group). *P < 0.05, significantly different from the control group.

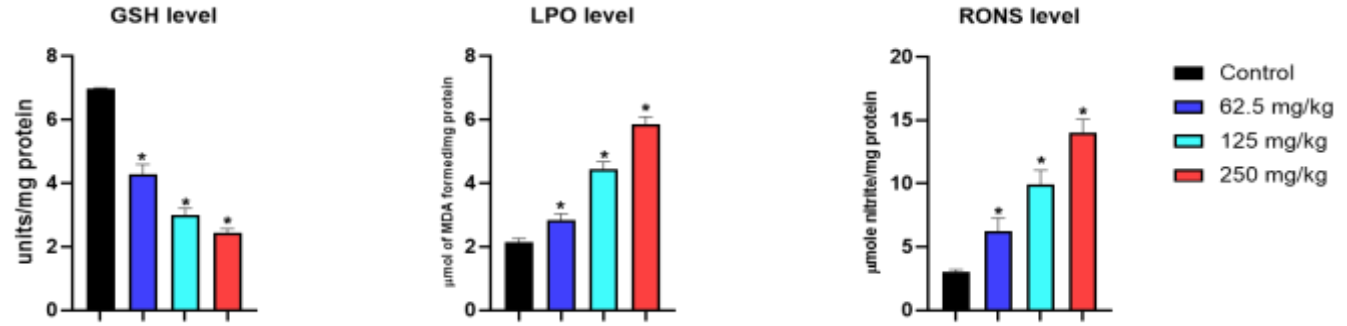


Figure 2. Effects of seven consecutive days of oral 1-NP exposure on renal GSH levels, lipid peroxidation (LPO), and reactive oxygen and nitrogen species (RONS) in Wistar rats. Data are expressed as mean ± SD (n = 8 per group). *P < 0.05, significantly different from the control group

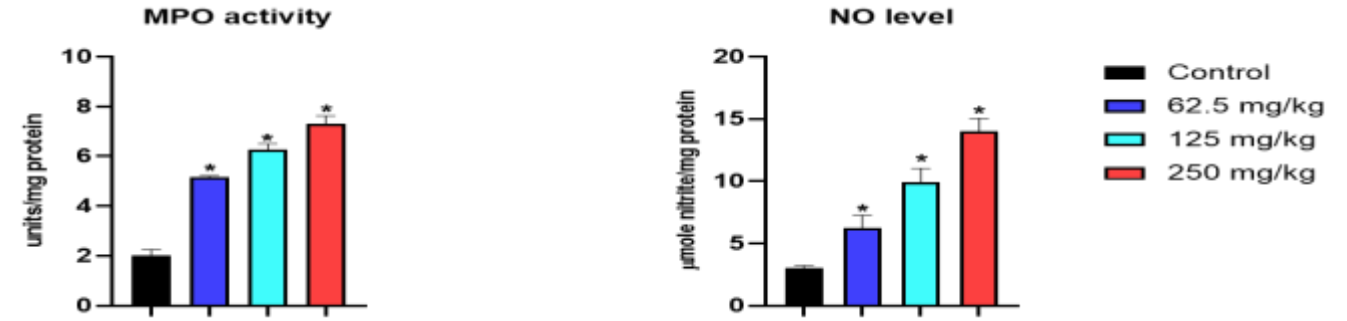


Figure 3. Effects of seven consecutive days of oral 1-NP exposure on renal myeloperoxidase (MPO) activity and nitric oxide (NO) concentration in Wistar rats. Data are expressed as mean ± SD (n = 8 per group). *P < 0.05, significantly different from the control group.

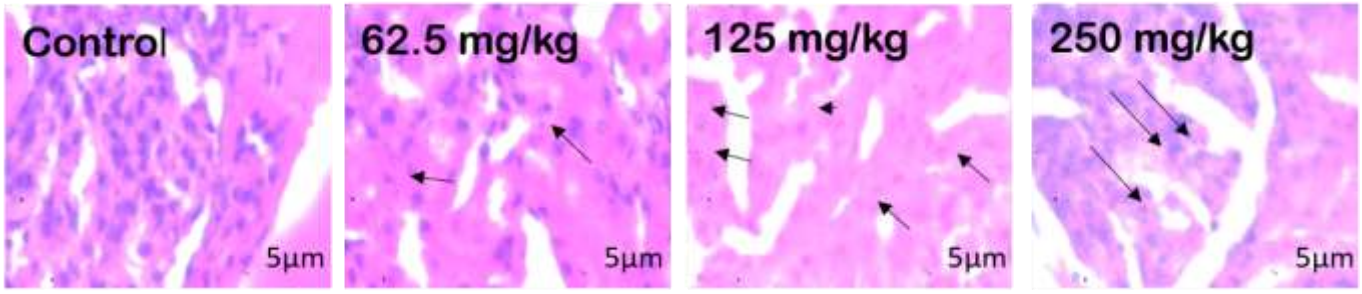


Figure 4. Representative photomicrographs of H&E-stained renal cortex sections from Wistar rats following seven consecutive days of oral 1-NP exposure. (A) Control: normal glomerular and tubular architecture. (B) 62.5 mg/kg BW 1-NP: mild inflammatory cell infiltration. (C) 125 mg/kg BW 1-NP: cortical deterioration and tubular epithelial degeneration. (D) 250 mg/kg BW 1-NP: severe glomerular lesions, hyperplasia, and vascular congestion. Stain: hematoxylin and eosin (H&E). Magnification: ×400. Scale bar: 50 µm.

DISCUSSION

Increasing urbanization and industrial activity in developing nations have elevated human exposure to environmental contaminants, particularly nitroarenes derived from fossil fuel combustion, with long-term consequences for organ health, especially the kidneys (1, 3, 9). The present study demonstrates, for the first time, that 1-NP induces dose-dependent nephrotoxicity in male Wistar rats through an oxido-inflammatory mechanism, as evidenced by significant alterations in renal function markers, antioxidant enzyme activities, oxidative stress indices, inflammatory mediators, and histopathological architecture.

A significant reduction in body weight gain was observed only at the highest dose (250 mg/kg body weight), consistent with previously reported systemic toxicity of 1-NP at elevated concentrations (3, 23). The concurrent significant increase in kidney weight at 250 mg/kg body weight is likely attributable to oedema and inflammatory cellular infiltration, phenomena characteristic of acute nephrotoxic injury. Serum urea and creatinine levels were significantly elevated in a dose-dependent manner across all treated groups. As filtration byproducts of protein catabolism and muscle metabolism, elevations in these markers reflect impaired glomerular filtration and tubular dysfunction (24). These findings are consistent with reports of xenobiotic-induced renal impairment in rodent models (3, 9).

The pro-oxidant activity of 1-NP in renal tissue was clearly evidenced by a significant dose-dependent increase in LPO and RONS concentrations, consistent with extensive laboratory evidence linking 1-NP-induced toxicity to elevated oxidative stress (1, 3, 23). Malondialdehyde, a stable end-product of polyunsaturated fatty acid peroxidation, serves as a reliable biomarker of lipid membrane damage under conditions of ROS overload (25). The concomitant depletion of GSH reflects excessive consumption of this critical non-enzymatic antioxidant in scavenging free radicals generated by 1-NP (26). Significant dose-dependent decreases in CAT, SOD, GST, and GPX activities were also observed, indicating that cytotoxic radical production suppresses endogenous renal antioxidant defenses. SOD catalyzes the dismutation of superoxide radicals to hydrogen peroxide, which is subsequently reduced to water by CAT, thereby maintaining renal redox homeostasis (26). GPX utilizes GSH to neutralize lipid hydroperoxides, while GST detoxifies electrophilic xenobiotic metabolites, both processes requiring adequate GSH availability (27). Disruption of these systems by 1-NP thus amplifies oxidative tissue injury.

The significant dose-dependent increases in renal MPO activity and NO concentration confirmed the induction of a local inflammatory response. MPO is a marker of neutrophil and monocyte recruitment to sites of tissue injury (28, 29), and its elevation supports the histopathological evidence of inflammatory infiltration observed in the renal cortex. Inducible nitric oxide synthase (iNOS)-mediated NO overproduction, which contributes to peroxynitrite formation and nitrosative tissue damage, is a recognized feature of oxido-inflammatory injury (30). These findings collectively suggest that 1-NP activates converging oxidative and inflammatory pathways in the kidney, ultimately leading to the morphological damage demonstrated on histological examination.

Histopathological analysis corroborated the biochemical findings, revealing dose-dependent glomerular lesions, hyperplasia, inflammatory infiltration, and vascular congestion consistent with the observed elevations in serum urea and creatinine, and in alignment with published data (3).

This study has several limitations that should be acknowledged. First, only acute (seven-day) exposure was examined; the renal consequences of subchronic or chronic 1-NP exposure remain to be characterized. Second, the study was conducted exclusively in adult male rats; therefore, the findings may not be directly generalizable to females or to juvenile or aged populations. Future studies should investigate the molecular signaling pathways mediating 1-NP nephrotoxicity, potential sex differences in susceptibility, and the efficacy of antioxidant interventions in attenuating renal injury.

Conclusion: The findings of the present study provide experimental evidence that acute oral exposure to 1-NP induces dose-dependent renal injury in male Wistar rats through an oxido-inflammatory mechanism characterized by depletion of antioxidant defenses, elevation of lipid peroxidation and reactive species, activation of inflammatory mediators, and histopathological disruption of renal architecture. These findings underscore the nephrotoxic risk of environmental nitroarene exposure and highlight the need for further mechanistic and epidemiological investigation.

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Conflicts of Interest: The authors declare no conflicts of interest.

Data availability: The data that support the findings of this study are available on request

Ethical Consideration: This study was approved by Benson Idahosa University Research Ethics Committee BIUREC/2026/122 with date of approval 11th January 2026. All animal procedures were conducted in strict accordance with the ARRIVE 2.0 guidelines and the National Institute of Health (NIH) Guide for the Care and Use of Laboratory Animals (1985).

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Author Contributions: MIC: Conceptualization; supervision; data curation; writing – original draft preparation; review, editing. ICN: Methodology, resources, validating writing – review and editing. OIE: Supervision, data curation, resources, writing – review and editing.

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