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Occupational lead exposure and its dual impact on renal function and reproductive hormones: A case-control study of Nigerian battery chargers

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Abstract

Occupational lead exposure among battery chargers poses significant risks to renal and reproductive health. This study assessed serum markers of kidney function (urea, creatinine, uric acid) and reproductive hormones (luteinizing hormone [LH], testosterone) in battery chargers compared to non-exposed controls in Ondo West Local Government Area, Ondo State, Nigeria. In this case–control study, 26 male battery chargers with more than five years of exposure and 26 age-matched controls provided venous blood samples. Standard assays (Berthelot, Jaffe, uricase; ELISA) were used to measure the analytes. Sociodemographic data and exposure history were obtained using a structured questionnaire. Statistical analyses included independent t-tests and Pearson correlations (SPSS version 20; $p < 0.05$ considered significant). Battery chargers showed significantly elevated serum creatinine (94.6 ± 14.9 µmol/L vs. 81.1 ± 10.5 µmol/L; $p < .001$) and blood lead levels (43.8 ± 34.8 µg/dL vs. 5.7 ± 3.2 µg/dL; $p < .001$), along with significantly reduced LH levels (3.18 ± 1.50 ng/mL vs. 5.58 ± 1.90 ng/mL; $p < .001$). No significant differences were observed in serum urea, uric acid, or testosterone levels ($p > .05$). Duration of lead exposure was positively correlated with creatinine ($r = .58$; $p < .001$) and negatively correlated with LH ($r = –.50$; $p < .001$). These findings suggest that chronic lead exposure in battery chargers is associated with subclinical renal impairment and disruption of the pituitary–gonadal axis. Routine biomonitoring and enhanced workplace safety interventions are strongly recommended.

Keywords: *battery charger, lead exposure, renal function, reproductive hormone, occupational health*

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1. Introduction

Lead continues to pose a significant occupational and environmental threat due to its persistence in soil, water, and air and its ability to bioaccumulate in human tissues (Al Sukaiti *et al*., 2023). Lead-acid battery technicians ("battery chargers") in low- and middle-income countries, such as Nigeria, face disproportionate risks of lead exposure by routinely inhaling and ingesting lead-containing dust and fumes and thus placing them at elevated risk of multisystem toxicity due to inadequate safety protocols in the informal recycling sector (Obeng-Gyasi, 2022). In Nigeria, lead exposure has been associated with increasing death and disability-adjusted life years (Pona *et al*., 2021). Despite Occupational Safety and Health Administration (OSHA) permissible blood lead level (BLL) of 20 µg/dL as the threshold, Nigerian battery chargers frequently exhibit BLLs exceeding 50 µg/dL, reflecting systemic gaps in occupational health regulations (Uthman *et al*., 2019; Olana, *et al*., 2022).

Lead is a known nephrotoxin and endocrine disruptor, implicated in proximal tubular damage, glomerulosclerosis, mitochondrial dysfunction and hormonal imbalances affecting both sexes (Liu *et al*., 2023). Chronic lead exposure accumulates in kidney tissue via erythrocyte binding and proximal tubular reabsorption, impairing nephron integrity (Collin *et al*., 2022) and ultimately reducing glomerular filtration rate (Danziger *et al*., 2024). Simultaneously, lead interferes with the hypothalamic–pituitary–gonadal axis, leading to altered gonadotropin release and testosterone synthesis (Roepke & Sadlier, 2021).

There is sparse information in literature about blood lead among Nigerian battery workers, yet comprehensive assessments of concomitant renal and endocrine biomarkers are limited (Okpogba *et al*., 2020). This study addresses that gap by analyzing BLLs, renal function and reproductive hormone indices in Nigerian battery chargers and non-exposed controls, providing evidence for targeted interventions.

2. Literature Review

2.1 Theoretical Framework

This study is anchored in the Heavy Metal Toxicity Modeland the Hypothalamic-Pituitary-Gonadal (HPG) Axis Disruption Theory, which explain lead's dual impact on renal and reproductive health.

Heavy Metal Toxicity Model: Lead (Pb) induces oxidative stress by generating reactive oxygen species (ROS), damaging renal tubular mitochondria and impairing glomerular filtration (Ho & Shirakawa, 2022). This model aligns with elevated creatinine levels observed in battery chargers, reflecting lead's inhibition of antioxidant enzymes like glutathione peroxidase (Jomova *et al*., 2024).

HPG Axis Disruption Theory: Lead suppresses gonadotropin-releasing hormone (GnRH) secretion, reducing pituitary luteinizing hormone (LH) release and impairing testosterone synthesis (Odetayo *et al*., 2024). This explains the inverse correlation between LH and exposure duration, consistent with findings of Dehghan *et al*. (2019) in occupationally exposed populations.

Socioecological Model of Occupational Health: Poor workplace safety, cultural norms, and economic constraints amplify lead exposure risks in low-resource settings (Olana *et al*., 2022). This framework contextualizes the high BLLs and limited PPE use among Nigerian battery chargers (Uthman*et al*., 2019).

These theories collectively justify the study's focus on lead's nephrotoxic and endocrine-disrupting effects, mediated by occupational exposure duration and socioeconomic vulnerabilities.

2.2 Renal Effects of Occupational Lead Exposure

Chronic occupational lead exposure is well-documented to cause glomerular and tubulointerstitial lesions, manifesting early as tubulopathy and later as interstitial fibrosis and hypertension (Bot *et al*., 2020; Liu *et al*., 2021), which later results to chronic kidney disease (CKD). Lead induces oxidative stress in mesangial and tubular cells, precipitating apoptosis and fibrogenesis (McLachlan *et al*., 2024). Lentini *et al*. (2019) linked heavy metals to nephrotoxicity, emphasizing lead's role in reducing glomerular filtration rate (GFR).

Childhood lead exposure can have lifelong impacts on renal health, potentially leading to adult nephropathy. Studies have shown that blood lead levels are associated with abnormal renal function parameters, including elevated blood urea nitrogen and creatinine, and reduced creatinine clearance (Kuraeiad & Kotepui, 2021). Epidemiological reviews confirm that even blood lead levels below 5 µg/dL contribute to impaired creatinine clearance and elevated serum creatinine (Axelrad *et al*., 2022). Lentini *et al*. (2017) demonstrated that lead disrupts tubular reabsorption, leading to Fanconi syndrome (aminoaciduria, glycosuria) in workers with

BLLs >30 µg/dL. Similarly, Adejumo *et al*. (2018) reported a 23% increase in creatinine levels among Nigerian automobile workers with >5 years of lead exposure. These findings align with mechanistic studies showing lead-induced oxidative DNA damage and apoptosis in renal cells (Nagaraju *et al*., 2022). Hyperuricemia, secondary to proximal tubular dysfunction, further predicts renal injury in exposed workers (Bai *et al*., 2016). Moreover, prospective trials in patients with chronic kidney disease show that chelation therapy can slow renal deterioration, implicating environmental lead in progressive nephropathy (Balakumar *et al*., 2020).

2.3 Reproductive Endocrine Disruption

Lead crosses the blood–testis barrier poorly, yet chronic exposure disrupts hypothalamic GnRH signaling and pituitary gonadotroph function, reducing LH and FSH output (Gandhi *et al*., 2017; Hau *et al*., 2023). Lead has been reported to suppress testosterone via inhibition of 17β-hydroxysteroid dehydrogenase. Studies on heavy metal exposure and reproductive hormones show mixed results. Meta-analyses confirm an inverse association between blood lead and LH in exposed men, even when testosterone remains unchanged (Balachandar *et al*., 2020). Dehghan *et al*. (2019) observed a 32% decline in LH among Iranian welders with BLLs >40 µg/dL, while Tutkun *et al*. (2018) reported a 15% testosterone reduction in Turkish battery recyclers. Conversely, Wang *et al*. (2023) found no significant association between lead and testosterone, suggesting variability in individual susceptibility or exposure duration. Blood lead levels above 40 µg/dL sometimes correlate with altered semen parameters, but hormone changes appear more sensitive in early toxicity (Calogero *et al*., 2021).

2.4 Regional Occupational Exposure

Regional occupational lead exposure is pervasive across West Africa, especially among artisanal battery recyclers and chargers who frequently lack adequate protective equipment and safety training (Lebbie *et al*., 2021). For example, in Kumasi, Ghana, small‑scale battery repair workshops sat a top soil lead concentrations as high as 2 500 mg/kg, which correlated strongly ($r = .78$; $p < .001$) with mean worker blood lead levels of 42.3 ± 13.6 µg/dL (Dartey *et al*., 2016; Konadu *et al*., 2023). In an even more dramatic case, an informal used‑lead-acid battery recycling site in Cameroon precipitated mass lead intoxication, resulting in 18 deaths from encephalopathy and over 900 residents requiring chelation therapy (Haefliger *et al*., 2009; Ondayo *et al*., 2016).

A cross-sectional study in Northern Nigeria found mean blood lead levels (BLLs) of 40.17 µg/dL in exposed workers compared to 3.61 µg/dL in controls (Sani-Gwarzo *et al*., 2024). Similarly, a meta-analysis of studies from low and middle-income countries reported a pooled mean BLL of 37.996 µg/dL among battery factory workers (Olana *et al*., 2022), accompanied by significant elevations in serum creatinine and uric acid in another study (Adejumo *et al*., 2018). Similarly, a Port Harcourt study documented reduced renal function alongside elevated blood lead among local battery workers (Alasia *et al*., 2010; Kuraeiad *et al*., 2021). Artisans in battery‑charging workshops in Nnewi exhibited significantly higher serum and reduced glomerular filtration rate, confirming occupational nephrotoxicity in a factory setting (Amah *et al*., 2014).

In Lagos State, a comparative survey of 132 automobile technicians, split between roadside and organized‑garage environments, found mean blood lead levels of 66.0 µg/dL among organized‑garage versus 43.5 µg/dL in their roadside workers counterparts (Saliu *et al*., 2015). Across these Nigerian contexts, inadequate regulation, lack of formal disposal and recycling facilities, and low hazard awareness among artisans perpetuate high blood lead burdens and resultant organ dysfunction (Sani-Gwarzo *et al*., 2024). Yet, despite clear evidence of widespread renal impairment, studies that concurrently assess renal and endocrine outcomes in this population remain scarce.

2.5 Socioeconomic and Occupational Lead Exposure

Lead-acid batteries, which contain up to 9 kg of lead per unit (Yudhistira *et al*., 2022), are widely used in automobiles and power storage (Zhang *et al*., 2022). Persistent occupational exposure, common among battery technicians, leads to conditions such as nephropathy, neurotoxicity, and reproductive dysfunction (Yayar *et al*., 2024; Edlund *et al*., 2024). “Battery chargers” in Nigeria operate within an informal economic sector characterized by limited regulatory oversight and inadequate safety protocols (Uthman *et al*., 2019). Approximately 85% of these workers lack formal training on lead exposure risks, and fewer than 20% use personal protective equipment (PPE) such as gloves or masks (Uthman, *et al*., 2019; Obeng-Gyasi, 2022). The majority work in open-air workshops with poor ventilation, averaging 8–10 hours daily, which amplifies inhalation and dermal absorption of lead particles (Park *et al*., 2024). Culturally, this occupation is male-dominated due to societal norms relegating hazardous labor to men, as evidenced by the study's exclusively male cohort (100% participation) (Aliyu *et al*., 2022). Economically, battery recycling provides a critical livelihood in regions with high unemployment, compelling workers to prioritize income over health despite awareness of risks (Aliyu *et al*., 2022). Compounding these challenges, Nigeria lacks stringent enforcement of international safety standards such as OSHA's blood lead level (BLL) thresholds, leaving workers vulnerable to chronic exposure (CDC, 2016). This socioeconomic landscape underscores the urgent need for targeted interventions combining regulatory enforcement, education, and accessible healthcare.

3. Methodology

3.1 Research Design

This investigation utilized a retrospective case–control design, an observational framework suited to identify associations between an exposure (occupational lead) and outcomes (renal and reproductive biomarkers) by comparing a group with the exposure to a matched unexposed group. Cases (battery chargers) were defined by ≥5 years of occupational lead exposure, while controls lacked such exposure. The case–control design enables efficient investigation of multiple outcomes (e.g., creatinine, LH) in relation to a single exposure, with fewer resources and shorter duration than cohort studies (Feifel *et al*., 2021). Moreover, this design is advantageous for studying subclinical endpoints in rare or hard‑to‑follow populations (e.g., battery artisans) and provides sufficient statistical power with modest sample sizes (Shreffler *et al*., 2023).

3.2 Participants of the Study

A total of 52 male participants aged 18–65 years were enrolled, 26 battery chargers (cases) and 26 age‑matched non‑exposed individuals (controls). Simple random sampling was used to select participants from Ondo West workshops and surrounding communities, ensuring equal selection probability and minimizing selection bias.

The sample size was calculated using the formula $S=a^2bc/d^2$ (Ajay & Micah, 2014), yielding a target of 50 participants. The final sample (n=52) exceeded this threshold, enhancing statistical validity. The 1:1 case-control ratio optimized efficiency for detecting significant differences in biomarkers, consistent with Turgut and Koca (2024), who emphasized balanced group comparisons in occupational health studies

Inclusion criteria for cases comprised ≥5 years of full-time battery-charging work, while controls were artisans not engaged in lead metal related occupation. Exclusion criteria included individuals with chronic kidney disease, endocrine disorders, or current psychoactive substance use.

3.3 Instrumentation and Data Gathering Process

Data collection combined a structured questionnaire with laboratory assays. The questionnaire, adapted from validated occupational health surveys, captured sociodemographic, exposure history (years of practice, daily hours), and protective practices. It was pilot-tested on 10 artisans for clarity and refined for content validity, followed by assessment of reliability via Cronbach's α (≥.80) (Shariff *et al*., 2021).

Venous blood (5 mL) was drawn into heparinized tubes, centrifuged at 4 000 rpm for 5 minutes, and plasma aliquots stored at –20 °C till analyzed. Renal markers (urea: Berthelot method; creatinine: Jaffe's method; uric acid: uricase method) were measured using Agappe kits (Fay *et al*., 2023). Reproductive hormones (LH, testosterone) were quantified by Monobind ELISA, following manufacturer protocols (Emokpae *et al*., 2006). Blood lead levels were determined by atomic absorption spectrophotometry (U.S. EPA Method 7421).

3.4 Data Analysis

Data entry and analysis were performed in IBM SPSS Statistics version 20.0. Descriptive statistics (means, standard deviations) characterized biomarker distributions. Independent t-tests compared means between cases and controls; effect sizes (Cohen's d) quantified the magnitude of differences. Pearson correlation assessed relationships between exposure metrics (years, hours) and biomarkers; two-tailed tests with α = .05 determined significance. Data were screened for outliers and normality; non-normal variables underwent log transformation prior to analysis.

3.5 Research Ethics

This study received ethical approval from the University of Medical Sciences, Ondo Review Board (Ref. UMED/EC/2023/017) in accordance with the Declaration of Helsinki. All participants provided written informed consent after being informed of study aims, procedures, potential risks, and their right to withdraw without penalty. Confidentiality was maintained by assigning coded identifiers; data were stored on encrypted drives with access restricted to investigators.

4. Results

Table 1

Socioeconomic characteristics of respondents

Variable	Category	n	%
Age (years)	0–20	14	26.9
	21–40	29	55.8
	41–50	8	15.4
	≥51	1	1.9
Religion	Christianity	36	69.2
	Islam	16	30.8
Years of practice	<5 years	8	15.4
	≥5 years	18	34.6
Working hours/day	4–6 h	2	3.8
	>6 h	24	46.2
Group	Exposed	26	50.0
	Control	26	50.0

Source: Field survey, 2024

Table 1 reveals that all participants were male, reflecting cultural norms restricting battery charging to men. Most workers (55.8%) were aged 21–40, indicating a younger workforce. Over 34% had >5 years of experience, and 46.2% worked >6 hours daily, highlighting prolonged exposure risks. Only 15.4% used safety gear, underscoring poor workplace safety practices.

Table 2

Comparison of renal indices

Variable	Battery Chargers (Mean ± SD)	Controls (Mean ± SD)	p-value
Urea (mmol/L)	3.07 ± 0.9	2.90 ± 1.1	0.515
Creatinine (µmol/L)	94.6 ± 14.9	81.10 ± 10.5	0.000*
Uric acid (mg/dL)	6.0 ± 2.3	5.0 ± 2.7	0.163
Lead (µg/dL)	43.83 ± 34.78	5.71 ± 3.17	0.001*

Source: Field survey, 2024; *$p < 0.05$

Creatinine was significantly higher in battery chargers (94.6 vs. 81.1 µmol/L, $p<0.05$), indicating impaired kidney function due to lead toxicity. Blood lead levels were 7.7 times higher in exposed workers (43.8 vs. 5.7 µg/dL, $p<0.05$), confirming hazardous exposure. Urea and uric acid showed no significant differences, suggesting these markers are less sensitive to short-term lead effects.

Table 3

Comparison of LH and testosterone

Variable	Battery Chargers (Mean ± SD)	Controls (Mean ± SD)	p-value
LH (ng/mL)	3.18 ± 1.5	5.58 ± 1.9	0.000*
Testosterone (mIU/mL)	4.83 ± 1.2	4.92 ± 1.3	0.803

Source: Field survey, 2024; *p < 0.05

Luteinizing hormone (LH) was significantly lower in exposed workers (3.18 vs. 5.55 ng/mL, $p<0.05$), signaling disrupted pituitary function. Testosterone levels remained unchanged (4.83 vs. 4.92 mIU/mL, $p>0.05$), suggesting lead primarily affects LH regulation rather than direct testicular damage. The inverse correlation between LH and exposure duration ($r = -0.502$, $p<0.05$) emphasizes cumulative hormonal harm.

Table 4

Correlations between exposure duration and renal/hormonal markers

Biomarker	Years of Practice (r-value)	p-value	Daily Working Hours (r-value)	p-value
Serum Creatinine	0.583	<0.001	0.454	0.001
Luteinizing Hormone	-0.502	<0.001	-0.556	<0.001
Testosterone	0.029	0.840	0.056	0.690
Uric Acid	0.267	0.055	0.199	0.156
Serum Urea	0.007	0.963	0.102	0.474

Source: Field survey, 2024

The correlations reveal that prolonged occupational exposure (years of practice and daily working hours) significantly elevates serum creatinine ($r = 0.583$ and 0.454, $p <0.001$), indicating progressive kidney damage, while suppressing luteinizing hormone (LH) ($r = -0.502$ and -0.556, $p <0.001$), reflecting cumulative endocrine disruption. Testosterone remained unaffected, suggesting lead primarily targets pituitary regulation rather than direct testicular harm. These findings emphasize that kidney dysfunction and LH suppression worsen with exposure duration, underscoring urgent need for workplace safety interventions.

Table 5

Subgroup analysis by exposure duration

Correlation Pair	<5 Years Exposure (r-value)	p-value	>5 Years Exposure (r-value)	p-value
Urea vs. Creatinine	0.364	0.034	0.300	0.048
Creatinine vs. Testosterone	0.373	0.030	—	—
Lead (Pb) vs. Luteinizing Hormone	—	—	0.423	0.040
Lead (Pb) vs. Testosterone	—	—	0.215	0.034

Subgroup analyses highlight distinct patterns: workers with <5 years of exposure showed early renal-hormonal interactions (e.g., creatinine-urea correlation: r = 0.364, p = 0.034), while those with >5 years exhibited stronger lead-LH (r = 0.423, p = 0.040) and lead-testosterone (r = 0.215, p = 0.034) links, signaling delayed hormonal effects.

5. Discussion

In this study, we observed a significant occupational health risk among battery chargers, characterized by toxic blood lead levels exceeding global safety thresholds, which contribute to sub-clinical renal dysfunction and hormonal disturbances, thereby presenting serious health and safety implications for these workers. The elevated BLLs in battery chargers (table 2) far exceed OSHA's safety level, corroborating findings from Uthman *et al*. (2019) in Ilorin, Nigeria. These levels are comparable to those reported in Pakistani battery workers (Haider & Qureshi, 2013) and Indian recyclers (Kumar*et al*., 2022), underscoring a global occupational health crisis.

Our findings also revealed significant renal and hormonal disruptions among battery chargers chronically exposed to lead. Elevated serum creatinine levels (table 2) in the exposed group compared to controls align with broad epidemiologic evidence of lead nephropathy as earlier reported (Lentini *et al*., 2017; Kargar *et al*., 2023). Lead accumulates in renal tubules, inducing oxidative stress and mitochondrial dysfunction, which impair glomerular filtration and tubular reabsorption (Gyurászová *et al*., 2020; Ho *et al*., 2022). The strong positive correlation between years of practice and creatinine underscores cumulative nephrotoxic burden which confirms dose-dependent nephrotoxicity as found by Adejumo *et al*. (2018) in Benin City, where workers with >10 years' exposure exhibited 25% higher creatinine levels. The weaker correlation with daily working hours suggesting cumulative exposure (years), is a stronger predictor of renal damage than daily intensity. However, urea and uric acid showed no significant differences, potentially due to compensatory mechanisms such as increased tubular secretion or dietary factors (Hussein *et al*., 2021). It might also be that subclinical tubular dysfunction may precede overt uremia (Muso *et al*., 2012; Du *et al*., 2024). This contrasts with studies reporting hyperuricemia in lead-exposed populations, possibly reflecting variations in sample demographics or exposure intensity (Tan *et al*., 2023; Xu *et al*., 2023).

The suppression of luteinizing hormone highlights lead's endocrine-disrupting properties. Lead interferes with gonadotropin-releasing hormone (GnRH) secretion from the hypothalamus, reducing pituitary LH release and subsequently impairing testosterone synthesis (Kapp, 2016; Roepke *et al*., 2021). The inverse correlation between LH and exposure duration (table 4) suggests progressive gonadotropin suppression and mirrors findings by Dehghan *et al.* (2019), who observed a 32% decline in LH among welders with >10 years' lead exposure.

Notably, testosterone levels remained statistically unchanged (table 2), contradicting studies reporting hypogonadism in lead-exposed workers (Doumouchtsis *et al*., 2009; Tutkun *et al.*, 2018). This discrepancy may stem from adaptive responses in Leydig cells or the relatively younger age of participants (mean age: 35 years), as testosterone decline often manifests with prolonged exposure (>15 years) (Luo *et al*., 2023). The early correlation between creatinine and testosterone (table 5) suggests renal-endocrine crosstalk, possibly mediated by lead-induced inflammation modulating steroidogenesis (Gandhi *et al*., 2017).

Overall, these data reveal early renal and hormonal alterations prior to overt clinical disease, highlighting the need for routine biomonitoring of serum creatinine and LH in battery workers. Regionally, these biochemical alterations occur against a backdrop of widespread informal battery repair, minimal hazard control, and elevated community exposures (Ababio *et al*., 2024). Preventive interventions, personal protective equipment, hygiene education, and periodic health screenings, are imperative to mitigate long-term sequelae.

6. Conclusion

Chronic lead exposure among battery chargers in Ondo West, Nigeria, is associated with subclinical renal impairment (elevated creatinine) and gonadotropin suppression (reduced LH). No significant changes occurred in urea, uric acid, or testosterone. These findings underscore the importance of regular health surveillance, workplace safety training, and policy enforcement to protect vulnerable artisanal workers from lead-induced nephro-endocrine toxicity. Routine biomonitoring, enforcement of safety practices, and health education are recommended to mitigate these risks. This study highlights the need for improved occupational safety measures and regular health monitoring for workers in the lead-acid battery industry to mitigate the risks of chronic lead exposure.

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Institutional Review Board Statement

This study was conducted in accordance with the ethical guidelines set by University of Medical Sciences, Ondo. The conduct of this study has been approved and given relative clearance(s) by the Ethical Review Committee, University of Medical Sciences, Ondo (Ref. UMED/EC/2023/017).

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