Hepatoprotective potential of *Telfairia occidentalis* fruit extract against Nickel chloride-induced toxicity in Wistar rats

Edobor Obayuwana[1], Priscilla Ekpabosi Oshianor[2], Njideka Jennifer Dim[3], Norris Osenele Obata-Godwin[4], Joseph Raymond Enoghase[5], Endurance Osakioduwa Imafidon[6], Martha Odion Obayuwana[7]

[1-6]Department of Anatomy, University of Benin, Benin-City, Edo State, Nigeria

[7]Department of Physiology, University of Benin, Benin-City, Edo State, Nigeria

Corresponding Author: Joseph Raymond Enoghase
University of Benin, Department of Anatomy
Benin-City, Edo State, Nigeria
Phone: +234 807 528 2878
Email: joseph.enoghase@bmedsci.uniben.edu

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ABSTRACT

Nickel chloride ($NiCl_2$) is an environmentally abundant heavy metal known to induce oxidative stress, metabolic disturbances, and hepatocellular injury. Identifying natural hepatoprotective agents remains crucial for mitigating heavy-metal toxicity. *Telfairia occidentalis* fruit contains rich phytochemicals with reported antioxidant and therapeutic properties. To evaluate the protective effects of aqueous *Telfairia occidentalis* fruit extract against $NiCl_2$-induced hepatic toxicity in Wistar rats. Twenty-four Wistar rats were divided into four groups (n = 6): Control, $NiCl_2$ (5 mg/kg, i.p.), *T. occidentalis* fruit extract (200 mg/kg p.o.) + $NiCl_2$ (pre-treated 1 hour before $NiCl_2$), and extract-only group (200 mg/kg p.o.). Treatments lasted 28 days. Body weight, relative liver weight, serum liver enzymes, oxidative stress biomarkers (MDA, GSH, SOD, CAT), and histopathology (H&E) were assessed. Data were analysed using ANOVA with Tukey's post-hoc test at $p < 0.05$. $NiCl_2$ exposure caused significant reductions in body weight, increased liver enlargement, elevated serum AST, ALT, ALP, increased MDA, and depleted antioxidant defences. Histology revealed periportal fibrosis, inflammation, hepatocyte vacuolation, and sinusoidal congestion. Pre-treatment with *T. occidentalis* fruit extract markedly improved body weight, reduced liver enlargement, stabilised liver enzymes, restored antioxidant biomarkers, and preserved hepatic microarchitecture. Extract-only rats showed normal hepatic and biochemical integrity. Aqueous *Telfairia occidentalis* fruit extract demonstrates significant hepatoprotective, antioxidant, and membrane-stabilising effects against $NiCl_2$-induced liver toxicity. The fruit possesses therapeutic potential for mitigating heavy-metal-related hepatic damage, supporting its relevance as a natural protective agent.

Keywords: antioxidants; epatoprotection; nickel toxicity; oxidative stress; *Telfairia occidentalis*; Wistar rats.

INTRODUCTION

Nickel is a widely distributed heavy metal with significant industrial applications, including electroplating, alloy production, and battery manufacturing (BARTZA et al., 2021). Environmental and occupational exposure to nickel compounds has increased over recent decades, with epidemiological studies indicating elevated exposure levels particularly among workers in mining, smelting, welding, and electroplating industries, as well as populations residing near industrial zones (NARAYANAN, 2026). Biomonitoring data from agencies such as the World Health Organization and the Agency for Toxic Substances and Disease Registry have demonstrated detectable nickel levels in blood and urine across both occupationally exposed and general populations, with higher burdens consistently reported in industrialized and urban regions (GENCHI et al., 2020). Additionally, longitudinal and cross-sectional studies have linked chronic nickel exposure to increased risks of respiratory cancers, dermatitis, and systemic toxicity, underscoring its growing public health relevance (BUXTON et al., 2019). Among various nickel salts, nickel chloride ($NiCl_2$) is particularly harmful because of its high solubility, rapid systemic distribution, and strong propensity to induce oxidative stress (SHARMA et al., 2023). The liver, being the primary organ responsible for xenobiotic metabolism, is especially vulnerable to nickel-induced cytotoxicity and metabolic derangement

Nickel chloride toxicity is mediated largely through the generation of reactive oxygen species (ROS), lipid peroxidation, mitochondrial dysfunction and disruption of endogenous antioxidant defences (SHARMA et al., 2023). Excessive ROS accumulation leads to oxidative damage of cellular membranes, proteins and nucleic acids, culminating in hepatocellular necrosis, inflammation and fibrosis. These pathological changes manifest biochemically as elevated liver enzyme activity - including alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) - and impaired metabolic function (HOMADY et al., 2025). Histologically, nickel exposure has been shown to cause periportal fibrosis, sinusoidal congestion, hepatocyte vacuolation and inflammatory cell infiltration, consistent with the morphological disruptions highlighted in the presented results.

Given the growing toxicity concerns associated with heavy metals, attention has shifted toward identifying plant-based therapeutic agents capable of ameliorating oxidative damage. Medicinal plants are rich sources of bioactive phytochemicals—such as flavonoids, alkaloids, tannins and phenolic compounds—that possess strong antioxidant, anti-inflammatory and

hepatoprotective activities (SAXENA et al., 2013; NWOZO et al., 2023). *Telfairia occidentalis*, commonly known as fluted pumpkin, is one such medicinal plant widely cultivated in West Africa. Traditionally used for nutritional and medicinal purposes, it contains antioxidant constituents including phenols, vitamins, carotenoids and bioflavonoids, which have been documented to protect against chemically induced organ toxicity (CHIJINDU et al., 2024).

Recent studies demonstrate that extracts of *Telfairia occidentalis* enhance endogenous antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT) and glutathione (GSH), thereby reducing lipid peroxidation and preserving organ structure and function during toxic insults (AIRAODION et al., 2019). This aligns with the present findings, where *T. occidentalis* ameliorated $NiCl_2$-induced alterations in body weight, liver enzymes and oxidative stress biomarkers, while also preserving hepatic architecture on histological examination. However, despite growing evidence, the mechanisms underlying its protective role against heavy-metal-induced liver injury require further elucidation.

This study therefore investigates the hepatoprotective effects of *Telfairia occidentalis* fruit extract against nickel chloride–induced toxicity, focusing on biochemical, oxidative stress and histopathological alterations. By providing updated experimental evidence, this research contributes to ongoing efforts to identify effective, affordable and plant-based interventions for mitigating heavy-metal-related hepatic injury.

MATERIALS AND METHODS

Experimental Animals

Twenty-four (24) adult male Wistar rats weighing 180-210 g were obtained from the Animal Housing Unit, Department of Anatomy, University of Benin, and housed in clean plastic cages under standard laboratory conditions (temperature 22 ± 2 °C, relative humidity 50–60%, 12-h light/dark cycle). Rats were allowed access to standard growers' feed and water ad libitum. All procedures conformed to institutional and international guidelines for the care and use of laboratory animals.

Plant Material and Authentication

Fresh mature fruits of *Telfairia occidentalis* were purchased from a local farm market in Benin City, Nigeria. Botanical authentication was carried out at the Department of Plant

Biology and Biotechnology, Faculty of Life Sciences, University of Benin, and a voucher specimen was deposited with authentication number UBH-T187.

Preparation of Telfairia occidentalis Fruit Extract

The fruit pulp was washed, sliced, and air-dried at room temperature until constant weight. The dried material was pulverised into fine powder using a mechanical grinder.

A total of 500 g of the powdered fruit material was macerated in 2.5 L of distilled water for 72 hours with intermittent shaking. The mixture was filtered through muslin cloth and Whatman No. 1 filter paper. The filtrate was concentrated using a rotary evaporator at 40 °C and further dried to obtain a semi-solid aqueous extract. The extract was stored at 4 °C in an airtight container. Working doses were freshly reconstituted in distilled water before administration.

Experimental Design

The rats were randomly assigned into four groups (n = 6 per group):

Group A – Control

Received oral distilled water and intraperitoneal (i.p.) injection of normal saline daily.

Group B – Nickel Chloride ($NiCl_2$).

Received 5 mg/kg $NiCl_2$ intraperitoneally (i.p.) once daily.

Group C – *Telfairia occidentalis* Fruit Extract (200 mg/kg) + $NiCl_2$.

Received 200 mg/kg aqueous fruit extract orally, and after 1 hour, were administered 5 mg/kg $NiCl_2$ intraperitoneally, daily.

Group D – *Telfairia occidentalis* Fruit Extract Only.

Received 200 mg/kg aqueous fruit extract orally once daily.

All treatments were administered for 28 consecutive days. Body weights were recorded at baseline and at the end of the experiment.

Ethical Approval

The College Ethics Committee of the College of Medical Sciences, University of Benin approved the research and an ethical approval number CMS/REC/2025/869 was assigned.

Sample Collection

At the end of the 28-day experimental period, rats were anaesthetised with light ether. Blood was collected by cardiac puncture into plain tubes, allowed to clot, and centrifuged at 3000 rpm for 10 minutes. Serum was separated for biochemical analysis.
Liver tissues were excised, rinsed in ice-cold saline, blotted dry, and divided into:

- a homogenate portion for oxidative stress assays
- a fixed portion placed in 10% neutral-buffered formalin

Relative Liver Weight Determination

Livers were weighed using a digital balance. Relative liver weight was computed as:

$$Relative\ Liver\ weight = \frac{Liver\ weight\ (g)}{Final\ Body\ weight\ (g)} x100$$

Serum Biochemical Assays

Serum concentrations of ALT, AST, ALP, total protein, and albumin were determined using standard colorimetric methods with commercial kits, following manufacturer protocols.

Oxidative Stress and Antioxidant Assays

Liver homogenates (10% w/v) were prepared in 0.1 M phosphate buffer (pH 7.4) and centrifuged at 5000 rpm for 10 minutes. The supernatant was used for:

- Malondialdehyde (MDA) - lipid peroxidation index
- Glutathione Peroxidase (GPx) activity
- Superoxide dismutase (SOD) activity
- Catalase activity

Spectrophotometric readings were taken at appropriate wavelengths.

Histopathological Examination

Formalin-fixed liver tissues were processed using standard paraffin-embedding techniques. Sections of 5 µm thickness were stained with haematoxylin and eosin (H&E). Microscopic evaluation was carried out at ×40 and ×100 magnifications, assessing:

- hepatic lobular architecture
- hepatocyte integrity
- periportal fibrosis
- inflammatory infiltration
- sinusoidal congestion or dilation
- vacuolar degeneration

Representative photomicrographs were captured.

Statistical Analysis

Data were expressed as mean ± SEM. One-way analysis of variance (ANOVA) was used to compare group means, followed by Tukey's post-hoc test for multiple comparisons. Within-group initial vs. final body weight was analysed using paired t-test. A significance level of $p < 0.05$ was adopted. Statistical analyses were performed using Graphpad Prism version 9.121.

RESULTS AND DISCUSSION

Nickel chloride ($NiCl_2$) exposure resulted in reduced body weight compared to the control group (Table 1), consistent with earlier findings that heavy metals impair metabolic function, appetite, and nutrient absorption (GENCHI et al., 2020). Weight loss during toxic exposure is attributed to oxidative stress–mediated catabolism and tissue injury.

Pre-treatment with *Telfairia occidentalis* fruit extract attenuated the $NiCl_2$-induced reduction in body weight, showing a stabilising effect on metabolic balance. This agrees with reports that *T. occidentalis* improves nutritional status and enhances physiological resilience through its rich antioxidant and micronutrient composition (ALI et al., 2024; CHIJINDU et al., 2024).

The extract-only group exhibited weight gain similar to control, confirming its nutritional safety and non-toxic profile.

Table 1. Body weight parameters - within group comparisons (Initial vs Final).

Group	**Initial Weight (g)**	**Final Weight (g)**	**p-value (Paired t-test)**
Control	185.33 ± 2.91	207.33 ± 1.76	0.005*
5mg/kg Nickel Chloride	198.33 ± 3.79	189.67 ± 3.18	0.110
5mg/kg Nickel chloride + 200mg/kg *Telfairia occidentalis* (After 30 mins)	236.50 ± 2.63	257.50 ± 0.87	0.018*
200mg/kg *Telfairia occidentalis*	210.00 ± 1.53	215.67 ± 2.33	0.023*

*Note: Values are expressed as mean ± SEM. *P<0.05 indicates statistical significance between initial and final weight within each group using paired t-test.*

A significant increase in relative liver weight in the $NiCl_2$ group (Table 2) indicates hepatic enlargement, which is a well-known outcome of heavy-metal-induced inflammation and oxidative injury (GUO et al., 2015). Hepatomegaly often reflects vascular congestion, inflammatory swelling, or early-stage fibrosis.

Pre-treatment with *T. occidentalis* fruit extract reduced the $NiCl_2$-mediated liver enlargement, highlighting its hepatomodulatory capacity. Studies have shown that *T. occidentalis* extracts possess anti-inflammatory and membrane-stabilizing phytochemicals capable of mitigating toxin-induced liver swelling (FEMI-AKINLOSOTU; IGADO; JUBRIL, 2022).

The extract-only group showed no deviation from control, supporting reports of its hepatic safety (ALI et al., 2024).

Table 2. Weight change and liver parameters across experimental groups.

Parameter/Group	**Control**	**5mg/kg Nickel Chloride**	**5mg/kg Nickel chloride + 200mg/kg *Telfairia occidentalis* (After 30 mins)**	**200mg/kg *Telfairia occidentalis***	**p-value**
Weight Change (g)	22.00 ± 2.16	-8.67 ± 0.62 *	21.00 ± 1.22 #	5.67 ± 0.62 *#	<0.001
Liver Weight (g)	6.65 ± 0.13	7.53 ± 0.21 *	6.25 ± 0.06 #	6.53 ± 0.06 #	<0.001
Hepato-Somatic Index (%)	3.21 ± 0.08	3.97 ± 0.07 *	2.43 ± 0.02 #	3.03 ± 0.05 #	<0.001

*Note: Values are expressed as mean ± SEM. *P<0.05 compared with the control group; #P<0.05 compared with the 5mg/kg Nickel Chloride only group based on Tukey's HSD post-hoc test following one-way ANOVA.*

$NiCl_2$ significantly elevated serum levels of AST, ALT, and ALP (Table 3), confirming hepatocellular damage and enzyme leakage into circulation. These biochemical changes reflect membrane disruption, mitochondrial dysfunction, and hepatocyte necrosis—common effects of nickel toxicity (HOMADY et al., 2025).

The reduction in total protein and albumin levels in the $NiCl_2$ group also indicates impaired liver synthetic function, aligning with the established metabolic toxicity of nickel compounds (HOMADY et al., 2025).

However, rats pre-treated with *T. occidentalis* fruit extract demonstrated marked improvement in these indices. Restoration of enzyme levels and protein concentrations suggests hepatoprotection mediated by the extract's antioxidant, anti-inflammatory, and membrane-stabilising components (OLADELE et al., 2021).

The extract-only group remained comparable to the control, confirming that *T. occidentalis* does not adversely alter liver enzyme function.

Table 3. Liver function parameters across experimental groups.

Parameter/Group	**Control**	**5mg/kg Nickel Chloride**	**5mg/kg Nickel chloride + 200mg/kg *Telfairia occidentalis* (After 30 mins)**	**200mg/kg *Telfairia occidentalis***	**p-value**
Total Protein (g/dL)	2.85 ± 0.00	1.49 ± 0.03 *	1.86 ± 0.02 *	1.98 ± 0.01 *#	<0.001
Albumin (g/dL)	1.86 ± 0.03	1.02 ± 0.08 *	1.63 ± 0.07 *#	1.82 ± 0.02 #	<0.001
Alanine Transaminase (U/L)	120.1 ± 0.8	187.8 ± 1.2 *	127.4 ± 0.3 *	101.5 ± 1.1 *#	<0.001
Aspartate Transferase (U/L)	235.6 ± 7.0	355.5 ± 5.9 *	251.9 ± 6.0 *	214.6 ± 2.0 #	<0.001
Alkaline Phosphatase (U/L)	56.6 ± 1.5	73.3 ± 0.8 *	28.7 ± 0.4 *#	64.0 ± 0.9 *#	<0.001
Total Bilirubin (mg/dL)	0.62 ± 0.01	1.57 ± 0.02 *	1.13 ± 0.08 *#	0.51 ± 0.04 #	<0.001
Conjugated Bilirubin (mg/dL)	0.30 ± 0.01	1.31 ± 0.04 *	0.38 ± 0.00 *#	0.28 ± 0.03 #	<0.001
*Note: Values are expressed as mean ± SEM. *P<0.05 compared with the control group; #P<0.05 compared with the 5mg/kg Nickel Chloride only group based on Tukey's HSD post-hoc test following one-way ANOVA.*					

$NiCl_2$ exposure significantly increased malondialdehyde (MDA) levels, indicating enhanced lipid peroxidation due to excessive generation of reactive oxygen species (ROS). Concurrently, GSH, SOD, and catalase levels were markedly reduced, demonstrating depletion of endogenous antioxidant defences (Table 4). These findings are consistent with literature that identifies oxidative stress as a central mechanism of nickel-induced hepatotoxicity (GENCHI et al., 2020).

Pre-treatment with *T. occidentalis* fruit extract reversed these alterations by reducing MDA levels and restoring antioxidant enzymes and GSH. This protective effect reflects the potent free-radical-scavenging capacity of the fruit's phenolic and flavonoid constituents (RUTH et al., 2023).

The extract-only group maintained normal oxidative profiles, confirming its inherent antioxidant potential and non-toxic nature.

Table 4. Oxidative stress parameters in liver tissue across experimental groups.

Parameter/Group	**Control**	**5mg/kg Nickel Chloride**	**5mg/kg Nickel chloride + 200mg/kg *Telfairia occidentalis* (After 30 mins)**	**200mg/kg *Telfairia occidentalis***	**p-value**
Superoxide Dismutase (SOD)	0.746 ± 0.007	0.262 ± 0.011 *	0.279 ± 0.008 *	0.777 ± 0.017 #	<0.001
Catalase	0.748 ± 0.014	0.198 ± 0.025 *	0.166 ± 0.004 *	0.729 ± 0.020 #	<0.001
Glutathione Peroxidase (GPx)	0.476 ± 0.012	0.118 ± 0.010 *	0.335 ± 0.065 *#	0.497 ± 0.011 #	<0.001
Malondialdehyde (MDA)	0.292 ± 0.011	0.947 ± 0.016 *	0.860 ± 0.060 *	0.177 ± 0.012 *#	<0.001
*Note: Values are expressed as mean ± SEM. *P<0.05 compared with the control group; #P<0.05 compared with the 5mg/kg Nickel Chloride only group based on Tukey's HSD post-hoc test following one-way ANOVA.*					

The control liver showed normal hepatic cords, intact sinusoids, and well-defined portal structures (Figure 1).

The $NiCl_2$-exposed group exhibited periportal fibrosis, inflammatory infiltration, hepatocyte disorganization, vacuolation, and sinusoidal congestion. Such lesions are consistent with heavy-metal-induced oxidative injury and inflammation reported in previous studies (AKINWUMI et al., 2020).

Pre-treatment with *T. occidentalis* fruit extract reduced these histological abnormalities, with improved hepatic architecture and minimal inflammatory changes. The extract's ameliorative effect reflects antioxidant and hepatoprotective mechanisms documented in literature (AGADA et al., 2024).

The extract-only group displayed normal hepatic architecture, confirming the extract's safety and compatibility with liver tissue.

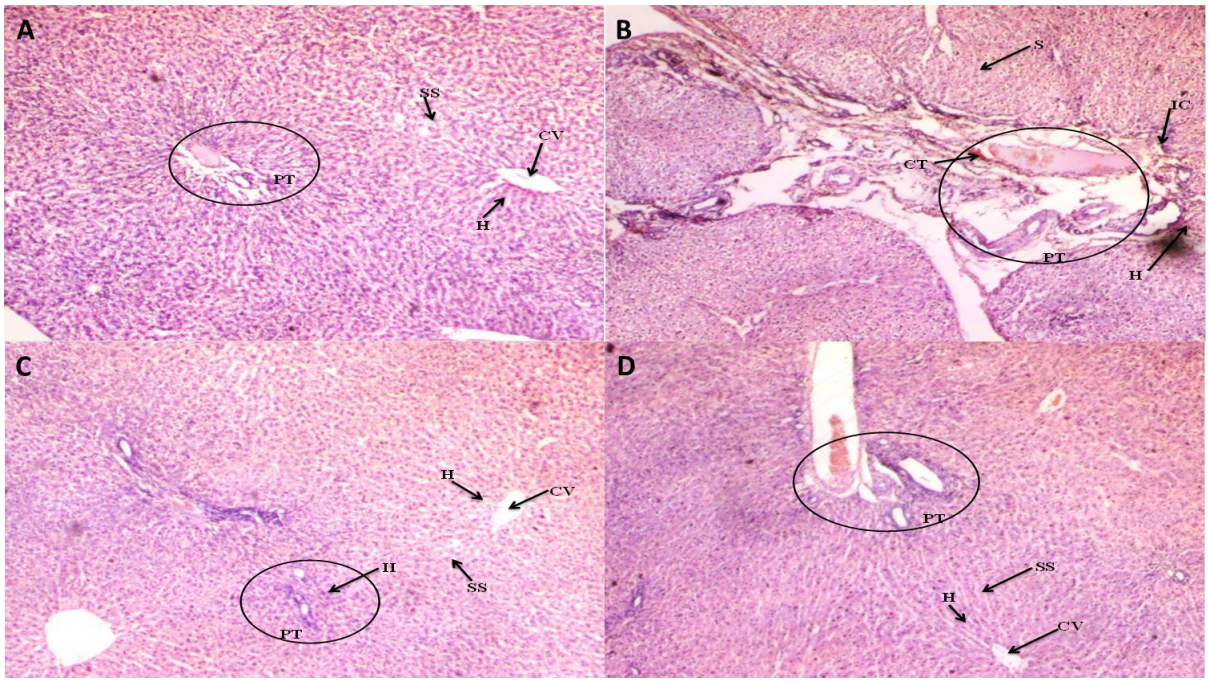

Figure 1. Plates A–D: Photomicrographs of liver sections (H&E, ×40) showing (A) normal hepatic architecture in the control with distinct central vein (CV), hepatocyte cords (H), and portal tract (PT); (B) Nickel chloride–treated liver with periportal fibrosis (CT), mild inflammatory infiltration (IC), and sinusoidal congestion (S); (C) pre-treatment with Telfairia occidentalis (200 mg/kg) and nickel chloride (5 mg/kg) showing preserved lobular organization with mild periportal inflammation and sinusoidal dilation; and (D) Telfairia occidentalis (200 mg/kg)–treated liver displaying intact hepatic cords, central vein, portal tract, and mildly dilated sinusoids.

CONCLUSION

Nickel chloride exposure produced significant biochemical, oxidative, and histological alterations characteristic of hepatotoxic injury. *Telfairia occidentalis* fruit extract demonstrated strong protective effects, evidenced by improved body weight, reduced liver enlargement, stabilization of liver enzymes, restoration of antioxidant systems, and preservation of hepatic microarchitecture. The protective actions of *Telfairia occidentalis* fruit extract are attributed to its rich repertoire of bioactive phytochemicals, including flavonoids, phenolic acids, saponins, alkaloids, and terpenoids which exhibit potent antioxidant and anti-inflammatory properties, as demonstrated through phytochemical screening, chromatographic (HPLC/GC–MS) profiling of phenolic constituents such as gallic acid and quercetin, FTIR-based functional group characterization, and spectroscopic identification of flavonoid structures. A limitation of this study is that no direct phytochemical or physicochemical characterization of the extract was

performed within the experimental design; thus, the observed effects could not be directly correlated with quantified or isolated active constituents. These findings support the potential therapeutic value of *T. occidentalis* fruit extract mitigating heavy-metal-induced liver toxicity and warrant further investigation into its active constituents and mechanisms of action.

REFERENCES

1. AGADA SA, ODAMA RI, KENECHUKWU CO, AONDOASEER K, EZEH CO, UTI DE, ALUM EU. Antioxidant and hepatoprotective effects of methanolic seed extract of *Telfairia occidentalis* on carbon tetrachloride induced hepatic damage in wistar rats. *Discov. Med* 1(75): 1-13, 2024.
2. AIRAODION AI, OGBUAGU EO, EKENJOKU JA, OGBUAGU U, AIRAODION EO. Therapeutic effect of methanolic extract of *Telfairia occidentalis* leaves against acute ethanol-induced oxidative stress in Wistar rats. *IJBST* 11(7): 179-189, 2019.
3. AKINWUMI KA, JUBRIL AJ, OLANIYAN OO, UMAR YY. Ethanol extract of Nigella sativa has antioxidant and ameliorative effect against nickel chloride-induced hepato-renal injury in rats. *Clin. Phytosc* 6(64): 1-12, 2020.
4. ALI AI, DANDAGO MA, ALI FI. Food applications of *Telfairia occidentalis* as a functional ingredient and nanoencapsulation as a promising approach toward enhancing food fortification. In: RAO V, SINGH P, SHARMA K. Phytochemicals in Agriculture and Food. IntechOpen, 2024, Chapter 6, p.1-18.
5. BARTZAS G, TSAKIRIDIS PE, KOMNITSAS K. Nickel industry: Heavy metal (loid) s contamination-sources, environmental impacts and recent advances on waste valorization. *Curr Opin Environ Sci Health* 21:100253, 2021, doi 10.1016/j.coesh.2021.100253.
6. BUXTON S, GARMAN E, HEIM KE, LYONS-DARDEN T, SCHLEKAT CE, TAYLOR MD, OLLER AR. Concise review of nickel human health toxicology and ecotoxicology. *Inorganics* 7(7): 89, 2019, doi 10.3390/inorganics7070089.
7. CHIJINDU PC, ONYEUKWU OB, OGHENEORUESE U. Evaluation of Nutritional, Phytochemicals and Antioxidant Capacity of *Telfairia Occidentalis* F. and *Vernonia Amygdalin*a Delile Leaves. *NJBMB* 39(3): 170-177, 2024.
8. FEMI-AKINLOSOTU OM, IGADO OO, JUBRIL AJ. Ethnomedicine and

Neuroscience in the developing world: plants most commonly used for neurological conditions. *Arch Basic Appl Med* 10(1): 5-16, 2022.

9. GENCHI G, CAROCCI A, LAURIA G, SINICROPI MS, CATALANO A. Nickel: Human health and environmental toxicology. *IJERPH 17*(3): 679, 2020, doi 10.3390/ijerph17030679.
10. GUO H, DENG H, CUI H, PENG X, FANG J, ZUO Z, DENG J, WANG X, WU B, CHEN K. Nickel chloride (NiCl2)-caused inflammatory responses via activation of NF-κB pathway and reduction of anti-inflammatory mediator expression in the kidney. *Oncotarget* 6(30): 28607-28620, 2015.
11. HOMADY MH, BILAL HS, OMER MM, ALNUAIMY SL, KHALID HS. The Impact of Different Doses of Nickel Chloride on Some Biochemical and Histopathological Changes in the Liver of Rats. *CUESJ* 9(2): 92-99, 2025.
12. NARAYANAN M. Heavy metal exposure among industrial workers mechanisms of toxicity biomonitoring strategies and public health risk assessment. *Discov Public Health* 23(1): 146, 2026, doi 10.1186/s12982-026-01431-1.
13. NWOZO OS, EFFIONG EM, AJA PM, AWUCHI CG. Antioxidant, phytochemical, and therapeutic properties of medicinal plants: A review. *Int. J. Food Prop* 26(1): 359-388, 2023.
14. OLADELE JO, ANYIM JC, OYELEKE OM, OLOWOOKERE BD, BAMIGBOYE MO, OLADELE OT, OLADIJI AT. *Telfairia occidentalis* mitigates dextran sodium sulfate-induced ulcerative colitis in rats via suppression of oxidative stress, lipid peroxidation, and inflammation. *J. Food Biochem* 45(9): e13873, 2021.
15. RUTH OO, ADEOYE RI, AFOLABI OB, AND ARISE RO. Free radical scavenging-related antioxidant properties and inhibitory activities of *Telfairia occidentalis* seed protein hydrolysates and peptide fractions against two key enzymes implicated in diabetes mellitus. *Int J Pept Res Ther* 29(5): 82, 2023, doi 10.1007/s10989-023-10555-w.
16. SAXENA M, SAXENA J, NEMA R, SINGH D, GUPTA A. Phytochemistry of medicinal plants. *J. pharmacogno phytochem* 1(6): 168-182, 2013.
17. SHARMA, M., Khan, FH. & Mahmood, R. Nickel chloride generates cytotoxic ROS that cause oxidative damage in human erythrocytes. *J Trace Elem Med Biol* 80: 127272, 2023, doi 10.1016/j.jtemb.2023.127272.