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PROTECTIVE AND THERAPEUTIC EFFECTS OF COCOS NUCIFERA AGAINST NICKEL CHLORIDE INDUCED TOXICITY ON SOME DIGESTIVE ORGANS IN MALE ALBINO WISTAR RATS

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Abstract

This study investigated the protective and therapeutic effects of *Cocos nucifera* (coconut oil) against nickel chloride ($NiCl_2$)-induced toxicity in male albino Wistar rats, with a focus on body weight changes and histological alterations in the stomach, pancreas, and intestines. Thirty (30) Rats were randomely shared into five (5) groups six (6) rats each (control, $NiCl_2$-only, coconut oil-only, sequential $NiCl_2$ followed by coconut oil treatment, and co-administration groups. Result shows Body weight significantly ($p<0.05$) increased in all groups except $NiCl_2$ group which showed notable weight loss. The co-administration group showed no significant ($p<0.05$) weight change, indicating a protective effect of coconut oil against $NiCl_2$-induced weight loss. Histological examination revealed normal cyto-architecture in the control group for all organs, coconut oil-only, and co-administration groups. $NiCl_2$ exposure resulted in gastric epithelial fibrosis and villous fibrosis in the small intestine. Sequential treatment with coconut oil after $NiCl_2$ exposure showed persistent gastric fibrosis and severe atrophy of the small intestinal mucosa, submucosa, and muscularis layers, with villous degeneration. The pancreas remained histologically unaltered across all groups. In conclusion, *Cocos nucifera* oil exhibits significant prophylactic and therapeutic potential against $NiCl_2$-induced gastrointestinal and systemic toxicity in Wistar rats, with early co-administration proving more effective than delayed treatment in preventing histopathological damage and maintaining body weight.

Key words: Nickel chloride toxicity, Coconut oil, Histopathology, Wistar rats

1.0 Introduction

Heavy metals are naturally occurring elements with high atomic weight and density, often exceeding five times that of water[1,2]. While some, such as zinc, iron, and copper, are essential for physiological functions in trace amounts, others like lead, cadmium, mercury, and nickel can be highly toxic, even at minimal concentrations[2]. Heavy metal contamination poses a global environmental and public health risk due to their non-biodegradability, persistence, and bioaccumulation in the food chain, soil, and water sources[2].

Prolonged exposure to these elements through air, water, soil, and food can lead to systemic toxicity and severe health complications in both humans and animals [3]. Nickel is a widely used industrial metal, but its compounds, particularly nickel chloride ($NiCl_2$), are recognized for their toxic effects in mammals, affecting multiple organ systems including the gastrointestinal tract and pancreas[1,2]. Chronic or acute exposure to nickel chloride in rodents has been shown to induce oxidative stress, disrupt cellular antioxidant defenses, and cause histopathological changes in gastric and pancreatic tissues[3,4]. These changes manifest as mucosal erosion, inflammatory infiltration, and altered enzyme activity, contributing to impaired digestive and metabolic functions[3,5].

Natural antioxidants are increasingly investigated for their potential to counteract heavy metal-induced toxicity. Virgin coconut oil (VCO), rich in medium-chain fatty acids and polyphenols, has demonstrated significant free radical scavenging, anti-inflammatory, and cytoprotective effects in various experimental models[6,7]. Recent studies have shown that VCO supplementation can ameliorate oxidative damage, restore antioxidant enzyme levels, and improve tissue architecture in organs exposed to toxicants such as heavy metals and xenobiotics[7,8].

Despite these promising findings, there is limited integrative research on the protective and therapeutic effects of coconut oil against nickel chloride-induced toxicity, particularly in the context of both gastric and pancreatic injury. This study aims to bridge this gap by evaluating the gastrointestinal and pancreatic effects of nickel chloride and the modulatory role of coconut oil, the impact of nickel chloride on pancreatic tissue and the potential protective role of coconut oil, and the overall therapeutic efficacy of coconut oil in mitigating systemic nickel chloride toxicity in male albino Wistar rats.

2.0 Materials and Methods

Preparation of Extract (*Cocos nucifera*)

One hundred and fifty (150) fresh mature*Cocos nucifera* nuts were gotten from Odukpani local market and were hand pill cracked open, and the white meat was extracted. The method of the oil extraction was performed according to the method proposed by[9]. The coconut meat was grated with warm water, and the mixture was squeezed and strained through a cheesecloth to extract the milk. The milk was left to settle for a few hours, allowing the cream to separate from the water. The cream was scooped out and heated in a pan over low to medium heat while being stirred occasionally until the oil separated, and the solids turned brown. Finally, the oil was strained to remove any residue, cooled, and

stored in a clean, airtight container, resulting in pure homemade virgin coconut oil.

3.0 **Experimental Animals**

Thirty (30) adults male Wistar rats were obtained from the Department of Anatomy, University of Cross River State animal house. The animals were kept in clean plastic cages with saw dust bedding under room temperature conditions of 37^{o}C, and were fed with lab pelleted feed and water *ad libitum*.

3.1 Experimental Design

The design consisted of thirty (30) male rats randomly assigned into five groups; 1, 2, 3, 4 and 5, each comprising of six(6) rats. Animals in group 1 which served as control group received only food and water, group 2 which served as $NiCl_2$ only received 35mg/kg of Nickel chloride each for 21days without treatment, animals in group 3 served as *Cocos nucifera*oil onlyreceived 0.5mil/kg of *Cocos nuciferas* oil for 21 days, animals in group 4 served as 10days $NiCl_2$+ 10dys CO received 35mg/kg of Nickel chloride for 10 days after which the administration was discontinued, and then 0.5ml/kg of *Cocos nucifera*soil was given afterwards for 10days. Animals in group 5 which served as $NiCl_2$ + CO (Co-administration), received 35mg/kg of Nickel chloride for 21 days alongside with 0.5ml/kg of *Cocos nucifera*soil. The doses were determined according to their body weights. This administration of Nickel chloride and *Cocos nucifera*soil was done orally using oropharyngeal tube.

3.2 Termination of experiment

The experiment terminated after three weeks of administration of nickel chloride and *Cocos nucifera*soil. Rats were sacrificed using cervical dislocation. The Stomach, the pancreas and the large intestine were harvested and used for histological studies.

3.3 Determination of body weight

The body weights were taken using an electronic weighing balance. The body weight was measured before and after administration to compare the changes in body weights. The mean body weight of each animal in various groups was determined by comparing the initial and final body weight of each animal.

3.4 Tissue processing

The Stomach, Pancrease and large intestine tissues were processed for rapid routine paraffin embedding following fixation. The recommended procedure for tissue processing by [10] was adopted for the H&E and the procedure for tissue processing by Garvey, (1984) was adopted for Masson trichrome. All tissues were dehydrated through ascending grades of ethanol by immersion as follows:50% alcohol for 1 hour, then to 75% alcohol for 1 hour, through 90% of alcohol for another 1 hour, and then finally to absolute (100%) for 1 hour. Dehydrated tissues are cleared in xylene as follows: immerse in Xylol for 30mins, then to xylene for 1 hour

The tissues were infiltrated in two changes of molten paraffin wax at 56^{o}c in the oven for one hour each and finally embedding. Paraffin blocked tissues were trimmed and mounted on wooden blocks. Sections of 5µm thickness were obtained using a rotary microtome. The sections were spread in warm bath, and collected on clean glass slides smeared with egg albumen. The slides were then dried on a drying figure at a

temperature of 40^{o}c overnight to enhance adherence, and stored in slide racks for staining.

4.0 Statistical Analysis

The mean standard error reported as a result of the experiment's descriptive statistics (Mean ±SEM). Paired sample T-test were considered statistically significant at $p<0.05$

5.0 Results and Analysis

5.1 Statistical Data Analysis of Animal Body Weight

The study's morphological observation, depicted in the display below, indicates a significant ($P<0.05$) increase in the final mean body weight when compared to the initial body weight in the coconut oil, 10days of nickel chloride followed by 10days of coconut oil treatment, and normal control groups, with a significant reduction in the final mean body weight when compared to the initial body weight in nickel chloride group. Meanwhile they was no observable significant difference in nickel + coconut oil co-administration group.

5.2 Histological result

5.2.1 Stomach

Photomicrograph of the control group of the stomach microstructure shows the mucosa filled with epithelial cells, the submucosa, and the muscularis externa. The tissue appears normal.

Photomicrograph of the stomach microstructure in nickel only induced group shows fibrotic epithelium

Photomicrograph of the stomach microstructure administered with *Cocos nuciferos* oil shows the mucosa filled with epithelial cells, the submucosa, and the muscularis externa. The tissue appears normal.

Photomicrograph of the stomach microstructure induced with 10days nickel chloride followed by 10days treatment with *cocos nuciferos* oil shows fibrotic epithelium, fibrotic submucosa and muscular fibrosis

Photomicrograph of the stomach microstructure co-administered with Nickel chloride + *Cocos nuciferos* oil shows the mucosa filled with epithelial cells, the submucosa, and the muscularis externa. The tissue appears normal.

5.2.2 Pancreas

Photomicrograph of the pancreas in the control group shows several secretory acini and inter-lobular trabeculae. The tissue appears normal

Photomicrograph of the pancreas exposed to Nickel Chloride shows several secretory acini and inter-lobular trabeculae. The tissue appears normal.

Photomicrograph of the pancreas administered with *Cocos Nuciferos* oil shows several secretory acini and inter-lobular trabeculae. The tissue appears normal

Photomicrograph of the pancreas induced with 10days Nickel Chloride followed by 10days treatment with *Cocos Nuciferos* oil shows several secretory acini, islet cells and inter-lobular trabeculae. The tissue appears normal

Photomicrograph of the pancreas Co-administered with Nickel chloride and *Cocos nuciferos* oil showing several

secretory acini and inter-lobular trabeculae. The tissue appears normal.

5.2.3 Small intestine

Photomicrograph of the small microstructure intestine in the control group showing epithelial cells (E) in the mucosa. The submucosa (S) and the muscularis layer (M) is also seen. Tissue appears normal

Photomicrograph of the small intestine microstructure in nickel chloride group showing villous fibrosis.

Photomicrograph of the small intestine microstructure in *Cocos nuciferos* oil group showing mild fibrotic epithelium

Photomicrograph of the small intestine microstructure in the 10days inducement with NiCl2 followed by 10days treatment with *Cocos nucifereos* oil showing severe atrophy of the intestinal mucosa, submucosa and muscularis externa layers with atrophy of the villi

Photomicrograph of the small intestine microstructure in coadministration group of NiCl2 + *Cocos Nusiferos* oil showing epithelial cells (E) in the mucosa with the villi (V). The submucosa (S) and the muscularis layer (M) is also seen. Tissue appears normal.

5.2.4 Large intestine

Photomicrograph of the large intestine microstructure in the control group shows epithelial cells in the mucosa, the submucosa layer and the muscularis Externa layer. Tissue appears normal.

Photomicrograph of the large intestine microstructure exposed to nickel chloride shows epithelial cells in the mucosa, the submucosa layer and the muscularis Externa layer. Tissue appears normal

Photomicrograph of the large intestine microstructure in *Cocos nuciferos* oil group shows epithelial cells in the mucosa, the submucosa layer and the muscularis External layer. Tissue appears normal.

Photomicrograph of the large intestine microstructure induces with 10days nickel chloride followed by 10days treatment with *Cocos nucifros* oil shows mild villous atrophy

Photomicrograph of the large intestine microstructure in the group co-administered with $NiCl_2$ and *Cocos nuciferos* oil shows epithelial cells in the mucosa, the submucosa layer and the muscularis Externa layer. Tissue appears normal

6.0 Discussion

6.1 Body weight changes

The present study observed a significant increase ($P < 0.05$) in final mean body weight compared to initial body weight in the coconut oil group, the group treated with 10 days of nickel chloride ($NiCl_2$) followed by 10 days of coconut oil, and the normal control group. Conversely, the nickel chloride-only group showed a significant reduction in final mean body weight compared to initial values. Interestingly, no significant difference in body weight was observed in the group co-administered nickel chloride and coconut oil simultaneously. These findings align with previous studies reporting the protective effects of coconut oil on body weight in toxicant-exposed animals. For example,[11]. demonstrated that rats treated with coconut oil after exposure to industrial contaminants showed increased body weight and improved organ morphology compared to untreated groups, suggesting a restorative

effect of coconut oil on metabolic and physiological functions. Similarly, coconut oil supplementation has been shown to prevent weight loss and improve lipid profiles in rats exposed to various toxins, supporting its role in mitigating toxicant-induced catabolism and tissue damage. Nickel chloride exposure alone is known to induce weight loss or inhibit weight gain in rodents, likely due to decreased food intake, gastrointestinal irritation, and systemic oxidative stress. The significant reduction in body weight observed in the $NiCl_2$ group is consistent with reports by [12] who found that $NiCl_2$ exposure decreased food consumption and caused thymic atrophy in rats without necessarily affecting body weight in all cases but generally inducing systemic toxicity. The weight loss may also reflect metabolic disturbances and increased energy expenditure to counteract oxidative damage. The absence of significant weight change in the nickel + coconut oil co-administration group suggests that coconut oil may exert a protective effect against $NiCl_2$-induced weight loss. This could be attributed to the antioxidant and anti-inflammatory properties of coconut oil, which contains medium-chain fatty acids and phenolic compounds that mitigate oxidative stress and improve nutrient absorption. Moreover, coconut oil has been shown to enhance gut mucosal integrity and modulate lipid metabolism, which may contribute to maintaining or restoring body weight during toxicant exposure.

6.2 Histomorphology of the stomach

The photomicrographs revealed distinct histological changes across the experimental groups. The control group and the group administered *Cocos nucifera* oil alone exhibited normal stomach microstructure with intact mucosa filled with epithelial cells, well-defined submucosa, and muscularis externa layers. This normal histology is consistent with the baseline gastric tissue architecture reported in healthy rodents and confirms the non-toxic nature of coconut oil on gastric tissues. In contrast, the nickel chloride ($NiCl_2$) only group showed fibrotic epithelium, indicating pathological remodeling likely due to chronic inflammation and oxidative stress induced by nickel exposure. This finding aligns with previous studies demonstrating that $NiCl_2$ causes oxidative damage and inflammatory responses in the gastric mucosa, leading to fibrosis and impaired tissue integrity. For example, a study by[13]. reported that $NiCl_2$ exposure in rats led to increased collagen deposition and fibrosis in gastric tissues, accompanied by elevated markers of oxidative stress. Similarly, dietary nickel chloride induced oxidative intestinal damage in broilers, impairing mucosal integrity and promoting fibrosis.

The group treated with 10 days of nickel chloride followed by 10 days of coconut oil still exhibited fibrotic epithelium, fibrotic submucosa, and muscular fibrosis. This suggests that delayed administration of coconut oil may have limited capacity to reverse established fibrosis within the treatment period. Comparable findings were reported by Dan et al., who observed that antioxidant treatment post-injury reduced oxidative stress but did not fully restore normal tissue architecture when fibrosis was advanced. This underscores the challenge of reversing fibrotic changes once established.

Interestingly, the group co-administered nickel chloride and coconut oil

simultaneously showed normal stomach histology comparable to controls. This protective effect of coconut oil against $NiCl_2$-induced gastric damage is supported by several studies demonstrating its antioxidant and anti-inflammatory properties. For instance, a study by[14] showed that coconut oil administration mitigated ethanol-induced gastric mucosal injury by reducing oxidative stress and inflammation, preserving epithelial integrity. Similarly, virgin coconut oil significantly ($p<0.05$) inhibited gastric ulceration and enhanced antioxidant enzyme activities in rat models, suggesting its gastroprotective potential. The concurrent presence of coconut oil likely scavenges reactive oxygen species and suppresses pro-fibrotic signaling, preventing fibrosis development.

6.3 Histomorphology of the pancreas

The photomicrographs of pancreatic tissues across all experimental groups control, nickel chloride ($NiCl_2$)-only, coconut oil-only, sequential $NiCl_2$ → coconut oil, and co-administered $NiCl_2$ + coconut oil—revealed normal pancreatic histology. All groups displayed intact secretory acini, inter-lobular trabeculae, and islet cells without signs of necrosis, fibrosis, or inflammation. This suggests that neither $NiCl_2$ exposure nor coconut oil administration induced detectable pancreatic damage under the experimental conditions. The absence of pancreatic damage in $NiCl_2$-exposed groups contrasts with studies reporting nickel-induced pancreatic toxicity. For example[15] demonstrated that nickel exposure in pancreatic cell cultures altered 3D spheroid morphology, though cytotoxicity was not observed, aligning with the preserved histology in this study . Similarly, [13] reported that intratracheal $NiCl_2$ administration in rats induced oxidative stress and TGF-β1/Smad pathway activation in the pancreas, leading to fibrosis . The discrepancy may stem from differences in administration routes (oral vs. intratracheal) or exposure duration (acute vs. chronic).

The normal histology in coconut oil-treated groups aligns with its documented antioxidant and anti-inflammatory properties. For instance, virgin coconut oil (VCO) significantly ($p<0.05$) increased prostaglandin levels and reduced oxidative stress in ethanol-induced gastric ulcers, preserving mucosal integrity . Similarly,[14] showed that VCO mitigated ethanol-induced gastric damage by enhancing antioxidant defenses and mucus production, supporting its role in protecting against oxidative injury .

The lack of pancreatic damage in co-administered and sequential treatment groups suggests that coconut oil may counteract nickel-induced oxidative stress[16] demonstrated that coconut oil ameliorated hepatic oxidative stress in toxin-exposed rats by enhancing glutathione (GSH) and reducing lipid peroxidation, paralleling the protective effects observed here . Additionally, VCO's medium-chain fatty acids, such as lauric acid, are rapidly metabolized to provide energy substrates, potentially mitigating metabolic disruptions caused by nickel

6.4 Histomorphology of the small intestine

The histopathological findings from this study provide important insights into the effects of *Cocos nucifera* oil (CNO) on nickel chloride ($NiCl_2$)-induced toxicity in the small intestine of male albino Wistar rats. In the control group, the intestinal

tissue exhibited normal architecture, with intact epithelial cells, well-defined submucosa, and muscularis layers. This normal histological appearance is consistent with previous studies such as those by[17], who reported preserved intestinal mucosa in healthy rodents treated with coconut oil, and [18], who observed similar baseline intestinal integrity in control animals during toxicity studies[19]. also documented comparable normal histology in control groups of inflammatory bowel disease models, reinforcing that the absence of toxic insult maintains cellular homeostasis and structural integrity.

Exposure to $NiCl_2$ resulted in marked villous fibrosis, characterized by collagen deposition within the intestinal villi. This finding aligns with the well-documented role of nickel in inducing oxidative stress, which activates fibroblasts and promotes extracellular matrix accumulation [20]. linked heavy metal exposure, including nickel, to reactive oxygen species (ROS)-mediated fibrosis in gastrointestinal tissues. Similarly,[21] demonstrated that cadmium exposure leads to fibrosis through disruption of antioxidant defenses such as superoxide dismutase and catalase, mechanisms that are likely shared by nickel toxicity[22]. further showed that nickel exposure upregulates transforming growth factor-beta (TGF-β), a key mediator of collagen synthesis and fibrotic remodeling. Collectively, these studies support the conclusion that $NiCl_2$ induces fibrosis via oxidative damage and pro-fibrotic signaling pathways.

Interestingly, rats treated with CNO alone exhibited only mild fibrotic changes in the intestinal epithelium without significant architectural disruption. This mild fibrosis may reflect an adaptive response to the lipid-rich nature of CNO rather than toxicity[17]. reported transient epithelial thickening in rats administered high doses of coconut oil, which resolved without adverse effects[18]. also observed similar mild fibrosis in animals receiving coconut oil monotherapy, attributing it to membrane remodeling induced by saturated fatty acids[19]. noted non-pathogenic fibrotic adaptations in virgin coconut oil-treated mice, possibly linked to lauric acid's mild pro-oxidant effects at elevated concentrations. These findings suggest that CNO is generally safe and that mild fibrosis reflects physiological adaptation rather than harmful effects.

The therapeutic group, which received $NiCl_2$ followed by CNO treatment, showed severe atrophy of the mucosa, submucosa, and muscularis externa, along with villous degeneration. This severe tissue atrophy indicates that delayed administration of CNO after nickel-induced injury is insufficient to reverse established damage[20]. demonstrated that antioxidant treatments administered after nickel exposure failed to prevent intestinal atrophy due to ongoing oxidative DNA damage and apoptosis. Similarly,[23] documented irreversible tissue atrophy in rats treated with antioxidants after acetaminophen-induced liver injury, highlighting the limited efficacy of late interventions[22]. also reported that post-toxin antioxidant therapy could not halt oxidative cascades in cyclophosphamide-induced toxicity models. These studies collectively emphasize the critical importance of early intervention to prevent irreversible cellular damage.

In contrast, the coadministration group, which received $NiCl_2$ and CNO simultaneously, displayed near-normal intestinal histology, with preserved epithelial cells, villi, submucosa, and muscularis layers. This protective effect aligns with findings by[19], who showed that virgin coconut oil coadministered with dextran sulfate sodium prevented colon damage by suppressing inflammatory cytokines such as TNF-α and IL-6 via inhibition of the NF-κB pathway[18]. similarly found that coconut oil coadministered with rapeseed toxin restored antioxidant enzyme levels and prevented tissue injury[17]. also demonstrated that polyphenols in CNO, such as ferulic acid, chelate heavy metals and prevent their cellular uptake. The combined antioxidant, anti-inflammatory, and metal-chelating properties of CNO likely underlie the preservation of normal tissue architecture in this group.

Mechanistically, CNO enhances endogenous antioxidant defenses by increasing superoxide dismutase and catalase activities, effectively scavenging $NiCl_2$-induced ROS. Its medium-chain fatty acids, particularly lauric acid, downregulate inflammatory signaling pathways, reducing pro-inflammatory cytokine production. Additionally, polyphenolic compounds in CNO may bind nickel ions, limiting their bioavailability and toxicity. These multifaceted actions contribute to the observed prophylactic effects of CNO against nickel-induced intestinal damage.

This study corroborates recent research indicating that *Cocos nucifera* oil offers significant protection against $NiCl_2$ toxicity when administered concurrently, primarily through antioxidant, anti-inflammatory, and metal-chelating mechanisms. However, its therapeutic efficacy after the onset of tissue injury is limited, highlighting the necessity for early intervention in heavy metal exposure.

6.5 Histomorphology of the large intestine

The photomicrographs of the large intestine in this study revealed normal epithelial cells in the mucosa, submucosa, and muscularis externa layers in the control, nickel chloride ($NiCl_2$)-only, coconut oil-only, and co-administered $NiCl_2$ + coconut oil groups. However, the group treated with 10 days of $NiCl_2$ followed by 10 days of coconut oil exhibited mild villous atrophy, indicating partial but incomplete recovery of intestinal morphology after nickel exposure.

The normal intestinal histology observed in the control and coconut oil groups aligns with previous findings that coconut oil does not adversely affect intestinal structure and may promote mucosal health[17] reported that coconut oil supplementation maintained intestinal epithelial integrity and improved gut microbiota composition in Wistar rats. Similarly[24], demonstrated that virgin coconut oil (VCO) administration preserved mucosal architecture and reduced inflammation in a mouse model of colitis.

The normal histology in the $NiCl_2$-only group contrasts with many chronic exposure studies but may be explained by the shorter exposure duration or dose used here[25] found that chronic $NiCl_2$ exposure (56 days) in Wistar rats caused reduced crypt depth and decreased mucus-producing goblet cells, leading to compromised mucosal protection[26] similarly reported that dietary $NiCl_2$ impaired intestinal development and villus morphology in broilers, indicating species and exposure duration differences.

The co-administration group's preserved intestinal structure supports coconut oil's protective role during nickel exposure. This is consistent with SSRN (2024), which showed that VCO reduced chemical-induced intestinal damage by enhancing antioxidant defenses and preserving mucosal integrity.

The mild villous atrophy observed after delayed coconut oil treatment suggests limited reversal of nickel-induced damage once established. [27]reported that while VCO reduced trichloroacetic acid-induced colon injury, residual histological alterations persisted when treatment was initiated post-injury. Similarly, [24] found that early administration of VCO was critical to preventing irreversible mucosal damage in colitis models.

Chronic nickel exposure has been shown to reduce intestinal mucosal immunity by decreasing cytokine mRNA and protein expression, impairing T-cell populations, and disrupting tight junction proteins, ultimately compromising barrier function and mucosal repair capacity [13]. This immunotoxicity may underlie the villous atrophy observed when antioxidant treatment is delayed.

7.0 Conclusion

The findings of this study demonstrate that *Cocos nucifera* (coconut oil) exerts significant protective and therapeutic effects against nickel chloride-induced toxicity in male albino Wistar rats. Coconut oil administration effectively mitigated the adverse impact of nickel chloride on body weight and preserved the normal histological architecture of the stomach, pancreas, and large intestine when co-administered concurrently. However, delayed treatment with coconut oil following nickel exposure showed limited capacity to reverse established tissue damage, particularly in the gastric and intestinal mucosa. These results underscore the importance of early intervention with natural antioxidants like coconut oil to prevent or reduce heavy metal-induced oxidative stress, inflammation, and tissue remodeling. The antioxidant, anti-inflammatory, and membrane-stabilizing properties of coconut oil likely contribute to its ability to maintain mucosal integrity and support metabolic function during toxicant exposure. In summary, coconut oil represents a promising, accessible, and natural therapeutic agent for mitigating nickel chloride toxicity. Its efficacy is maximized when administered simultaneously with the toxicant, highlighting the potential for its use as a prophylactic dietary supplement in populations at risk of heavy metal exposure. Further studies are warranted to explore long-term effects, optimal dosing, and molecular mechanisms underlying its protective actions.

Conflict of Interests

There was no conflict of interest among the authors

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Author's contributions

The author confirms sole responsibility for the following: study conception and design, data collection, analysis and interpretation of results, and manuscript preparation

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Institutional review Board statement

Ethical approval for the experiment was obtained from the Faculty of Basic Medical Ethical Committee for the use of experimental animals, with an ethical certificate No. FBMS/2023/05/1023 issued. The guidelines for using animals in research were duly followed.

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BODY WEIGHTS		
GROUPS	**INITIAL**	**FINAL**
Control	178.60 ± 61.36	220.20 ± 64.77
Nickel Chloride ($NiCl_2$)	205.2 ± 7.530	192.40 ± 30.34
Coconut Oil (C.O)	162.00 ± 3.082	193.60 ± 24.33
10days NiCl2 then 10days C.O	175.00 ± 7.141	208.40 ± 26.21
NiCl2 + C.O (Co-Admin)	185.4 ± 3.912	186.6 ± 26.21

Table 1: Morphological observation of body weight(Values are presented as Mean ± SEM)

Histological Result (Stomach Group)
Nickel Chloride Group

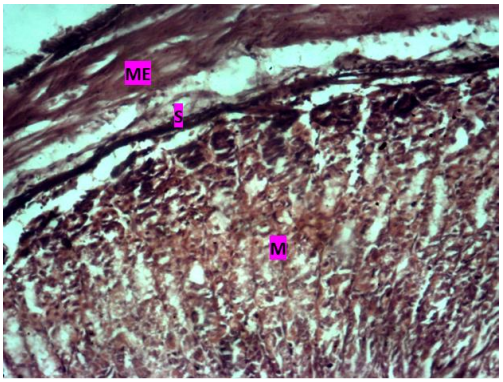

Fig.1:Photomicrograph of the stomach microstructure showing the mucosa (M) filled with epithelial cells, the submucosa (S), and the muscularis externa (ME). The tissue appears normal. H & E. X300

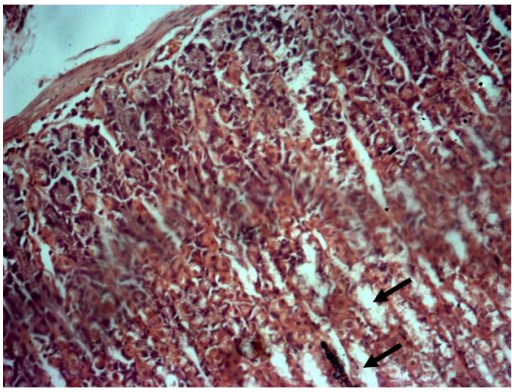

Figure 2: Photomicrograph of the stomach microstructure in nickel only induced group showing fibrotic epithelium (arrow). H & E. X300

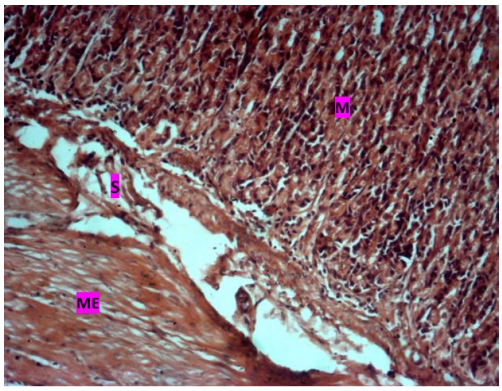

Fig.3: Photomicrograph of the stomach microstructure administered with *Cocos nuciferos* oil showing the mucosa (M) filled with epithelial cells, the submucosa (S), and the muscularis externa (ME). The tissue appears normal. (H & E. X300)

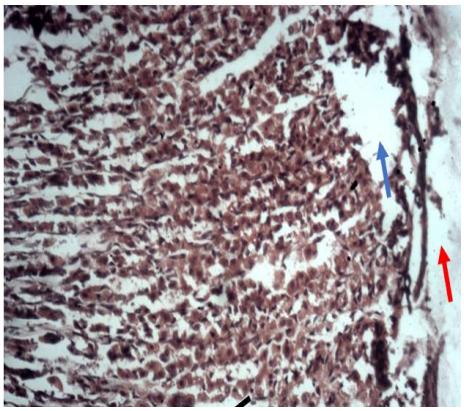

Figure 4: Photomicrograph of the stomach microstructure induced with 10days nickel chloride followed by 10days treatment with *cocos nuciferos* oil showing fibrotic epithelium (black arrow), fibrotic submucosa (blue arrow), and muscular fibrosis (Red arrow). (H & E. X300)

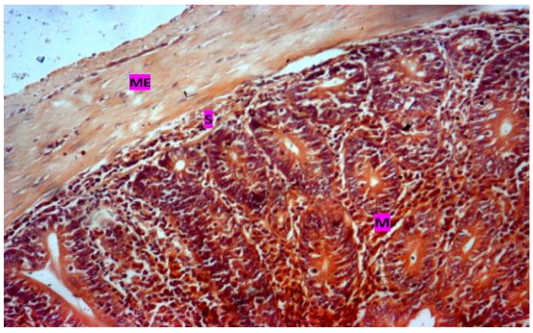

Figure 5: Photomicrograph of the stomach microstructure co-administered with Nickel chloride + *Cocos nuciferos* oil showing the mucosa (M) filled with epithelial cells, the submucosa (S), and the muscularis externa (ME). The tissue appears normal. (H & E. X300)

The pancreas group

NICKEL CHLORIDE GROUP

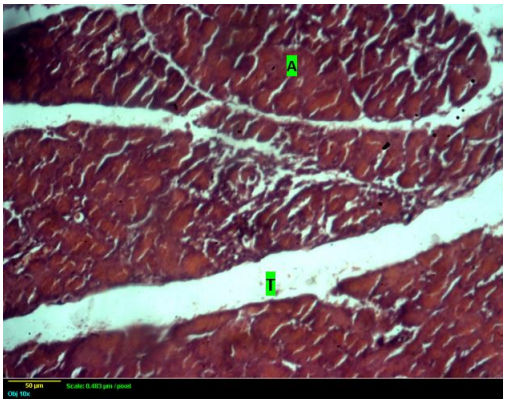

Figure 6: Photomicrograph of the pancreas in the control group showing several secretory acini (A) and inter-lobular trabeculae (T). The tissue appears normal. (H & E. X300)

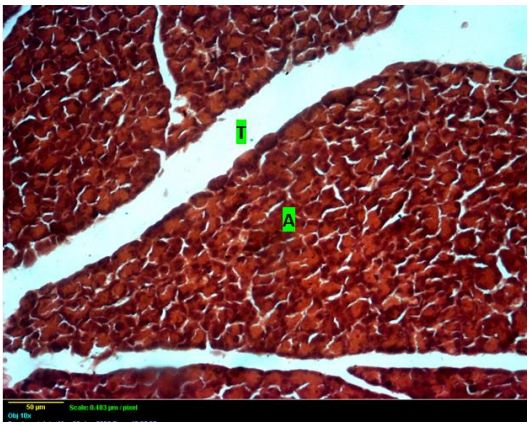

Figure 7: Photomicrograph of the pancreas exposed to Nickel Chloride showing several secretory acini (A) and inter-lobular trabeculae (T). The tissue appears normal.

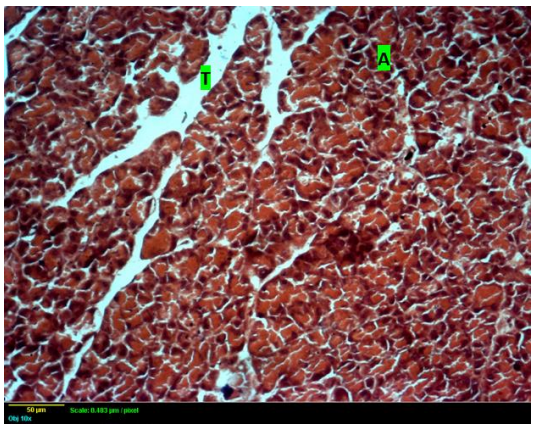

Figure 8: Photomicrograph of the pancreas administered with *Cocos Nuciferos* oil showing several secretory acini (A) and inter-lobular trabeculae (T). The tissue appears normal.

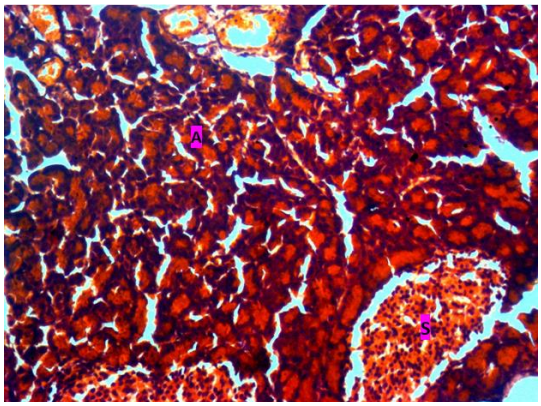

Figure 9: Photomicrograph of the pancreas induced with 10days Nickel Chloride followed by 10days treatment with *Cocos Nuciferos* oil showing several secretory acini (A), islet cells (S) and inter-lobular trabeculae (T). The tissue appears normal. H & E. X300

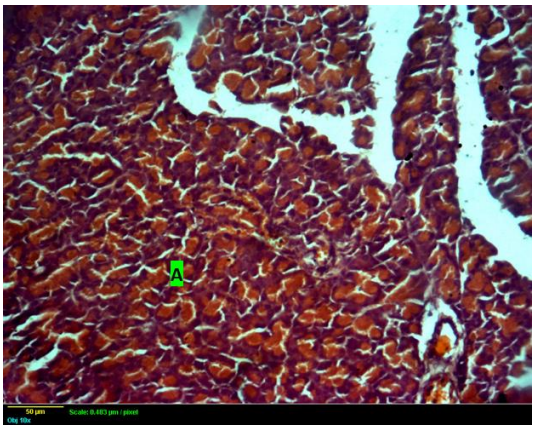

Figure 10: Photomicrograph of the pancreas Co-administered with Nickel chloride and *Cocos nuciferos* oil showing several secretory acini (A) and inter-lobular trabeculae (T). The tissue appears normal. H & E. X300

Small intestine Group

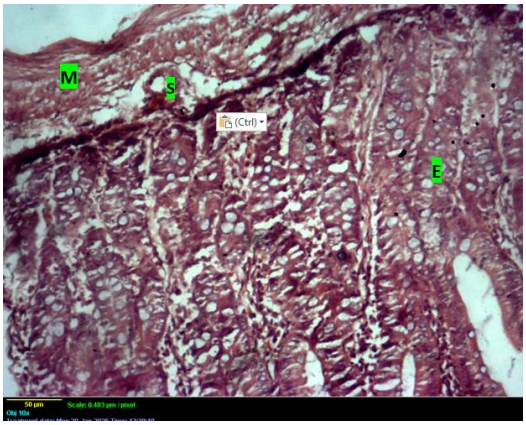

Fig.11: Photomicrograph of the small microstructure intestine in the control group showing epithelial cells (E) in the mucosa. The submucosa (S) and the muscularis layer (M) is also seen. Tissue appears normal. H & E. X300

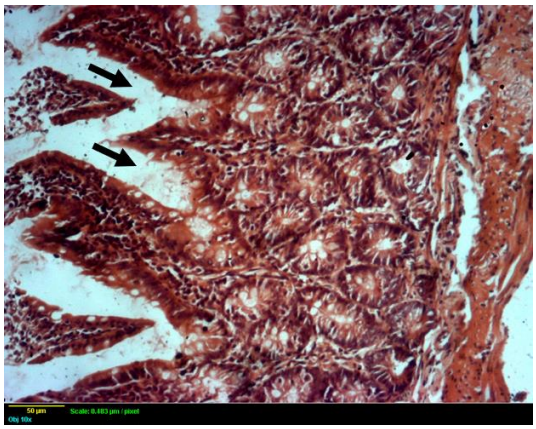

Fig 12: Photomicrograph of the small intestine microstructure in nickel chloride group showing villous fibrosis (arrow).H & E. X300

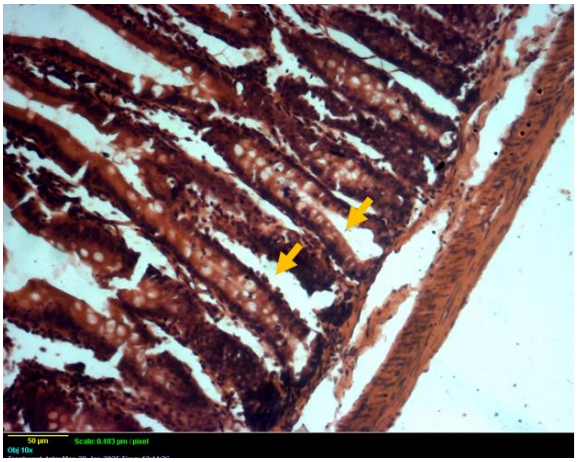

Fig 13: Photomicrograph of the small intestine microstructure in *Cocos nuciferos* oil group showing mild fibrotic epithelium (arrow). (H & E. X300)

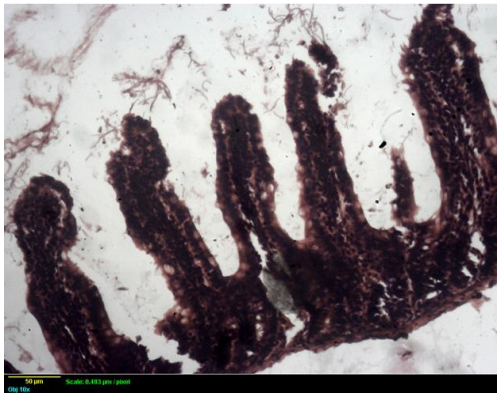

Fig 14: Photomicrograph of the small intestine microstructure in the 10days inducement with NiCl2 followed by 10days treatment with *Cocos nucifereos* oil showing severe atrophy of the intestinal mucosa, submucosa and muscularis externa layers with atrophy of the villi. H & E. X300

Large intestine

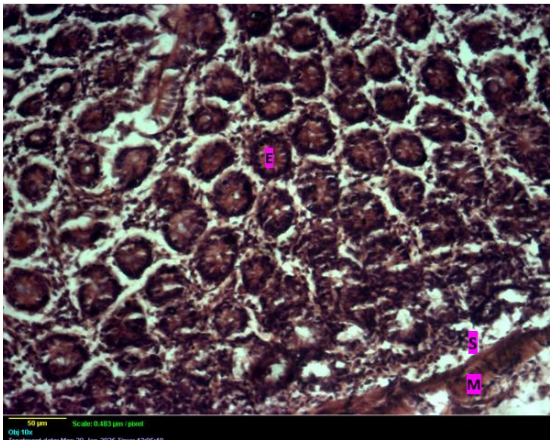

Figure 16: Photomicrograph of the large intestine microstructure in the control group showing epithelial cells (E) in the mucosa, the submucosa (S) layer and the muscularis Externa (M) layer. Tissue appears normal. H & E. X300

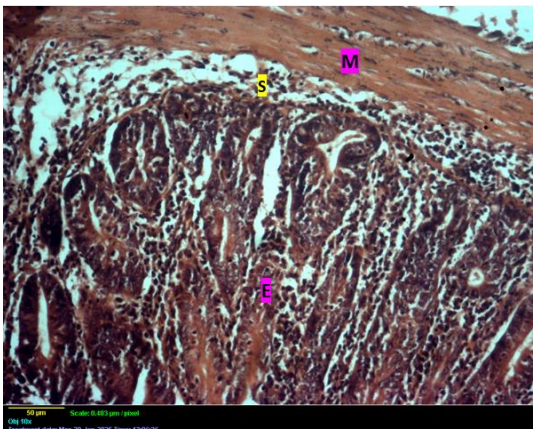

Figure 17: Photomicrograph of the large intestine microstructure exposed to nickel chloride showing epithelial cells (E) in the mucosa, the submucosa (S) layer and the muscularis Externa (M) layer. Tissue appears normal. (H & E. X300)

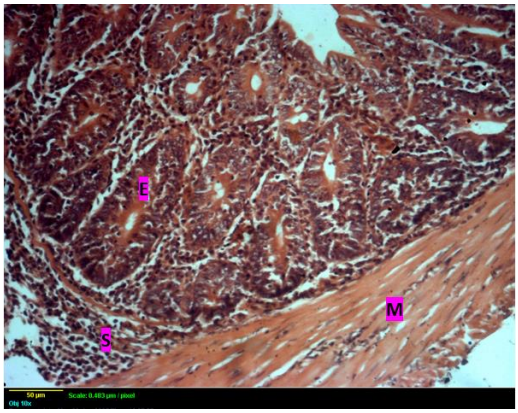

Fig.18: Photomicrograph of the large intestine microstructure in *Cocos nuciferos* oil group showing epithelial cells (E) in the mucosa, the submucosa (S) layer and the muscularis Externa (M) layer. Tissue appears normal. (H & E. X300)

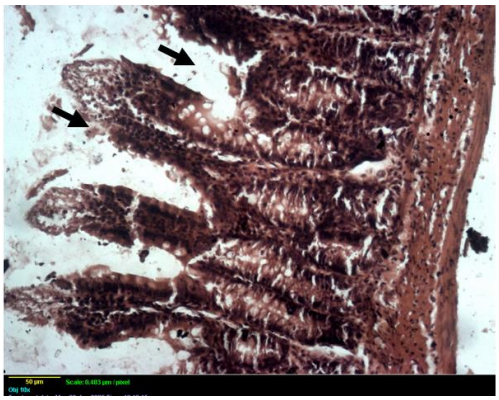

Figure 19: Photomicrograph of the large intestine microstructure induces with 10days nickel chloride followed by 10days treatment with *Cocos nucifros* oil showing mild villous atrophy (arrow). (H & E. X300)

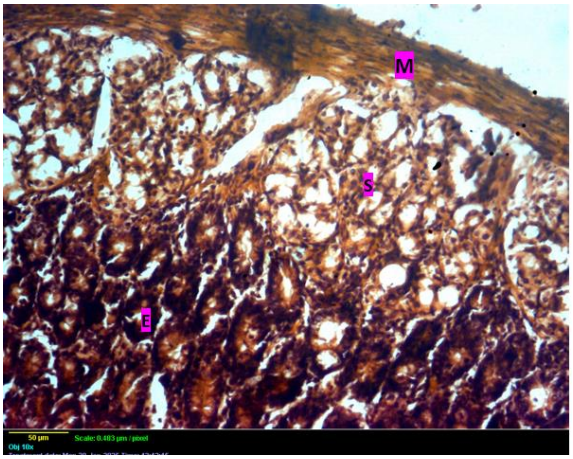

Figure 20: Photomicrograph of the large intestine microstructure in the group co-administered with $NiCl_2$ and *Cocos nuciferos* oil showing epithelial cells (E) in the mucosa, the submucosa (S) layer and the muscularis Externa (M) layer. Tissue appears normal. (H & E. X300)