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Research Article

Cadmium-Induced Hepato-Renal Toxicity in Rats: Protection by Cinnamic Acid Via Antioxidative System

Ige Olaoye[1,*], Olubukola Agboola[1], Babatunde Oso[1], Adepeju Aberuagba[1], Adebukola Adeyanju[2], Olayinka Adebayo[1], Oluwapelumi Olaniyi[1], Opeyemi Edu[1], Omowumi Sunday[1],

[1]*Department of Biochemistry, McPherson University, Seriki Sotayo, Nigeria*
[2]*Department of Biological Sciences, Kola Daisi University, Ibadan, Nigeria*

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CORRESPONDENCE
Agboola, O.
bkbakanni@gmail.com
igelaoye@gmail.com

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ABSTRACT

Cadmium chloride ($CdCl_2$), a highly toxic compound poses significant risks to human health such as oxidative injury. The use of antioxidants such as cinnamic acid has been used in the treatment of oxidative stress. Hence, this study investigated the ameliorative effects of cinnamic acid on cadmium chloride–induced hepato-renal injury. Thirty male Wistar rats, divided into six groups (n=5), were treated with corn oil (control), $CdCl_2$, $CdCl_2$ + CA (50 mg/kg), $CdCl_2$ + CA (100 mg/kg CA (50 mg/kg) and CA (100 mg/kg). Rats were pretreated with CA (*p.o*) for 7 days and challenged with intraperitoneal injection of two doses of $CdCl_2$ (1.5 mg/kg) at the last 72 hours. Administration of $CdCl_2$ caused a significant ($p<0.05$) increase in the levels of serum urea, creatinine and renal lipid peroxidation (LPO) by 79%, 25% and 29%, respectively when compared with the control. However, the activity of renal superoxide dismutase and reduced glutathione were significantly ($p<0.05$) decreased by 50% and 26%, respectively, relative to the control. $CdCl_2$ also significantly increased the activities of serum AST and levels of liver LPO by 76% and 71%, respectively while the activity of SOD in the liver was insignificantly ($p>0.05$) decreased. Interestingly, pre-treatment with cinnamic acid significantly reduced the serum indices and restored the antioxidant parameters close to normal. Cinnamic acid ameliorates cadmium chloride-induced kidney and liver dysfunction via antioxidative pathway.

Keywords: *Cadmium chloride; Cinnamic acid; Oxidative injury; Antioxidant*

INTRODUCTION

The prevalence of heavy metal poisoning, which results from both occupational and environmental exposure, poses a serious threat to public health. This widespread risk to human and animal health results from acute or on-going exposure to heavy metals, which can cause serious organ damage and, in the worst circumstances, mortality (Ali et al., 2019). Epidemiological studies have repeatedly demonstrated a connection between the amount of heavy metals in the body and their effects on health (Szczygłowska et al., 2014). Notably, it has long been known that exposure to cadmium (Cd^{2+}) compounds, particularly cadmium chloride ($CdCl_2$), which has a rapid tissue distribution, may be harmful to both humans and animals (Deveci and Deveci, 2011).

The extensive use of cadmium (Cd^{2+}) compounds in numerous industrial processes such as plating operations, production of polyvinyl chloride (PVC), dyeing of plastics and glass, manufacture of nickel-cadmium batteries, production of commercial phosphate fertilizers, tobacco and chemical industries, has made these compounds widely prevalent in the environment (Oladele et al., 2017). Since it is present in

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soil and plants, cadmium chloride can be ingested through food and drinks (Shahid et al., 2016). Previous research on Cadmium poisoning has indicated its accumulation in the liver and kidneys following occupational and environmental exposure (Mershiba et al., 2013; Gabr et al., 2019). The precise mechanism by which $CdCl_2$ compounds induces hepatotoxicity is not completely understood but the main possible toxic mechanism is the induction of oxidative stress (Arroyo et al., 2012; Andjelkovic et al., 2019). Cadmium has also been reported to be toxic to the kidney. The main toxicological mechanism hypothesized to Cd-induced nephrotoxicity is oxidative stress (Andjelkovic et al., 2019) which occurs when the cellular antioxidant defense is overwhelmed by the excessive production of reactive oxygen species (ROS) and free radicals. According to Sanjeev *et al.* (10 2019), this causes lipid peroxidation, damage to membrane proteins, disruption of the antioxidant system, DNA damage, inhibition of DNA repair enzymes, regulation of gene expression, and eventually, apoptosis. Cadmium chloride can also cause inflammation, interfere with cell cycle control, and disrupt apoptotic signalling pathways (Jiménez-Ortega et al., 2012; Valko et al., 2015; Everson et al., 2018). Therefore, there is a need to identify agents with antioxidant properties that can alleviate cadmium-induced toxicity.

Cinnamic acid, a naturally occurring chemical that belongs to the phenylpropanoid family, is a key phenolic component of cinnamon (*Cinnamomum cassia*). It is widely present in honey, cinnamon oils, coca leaves, fruits, and vegetables (Ruwizhi and Aderibigbe, 2020). It has been used in several medicinal formulations and as a flavoring agent in the food and beverage sectors (Gupta and Wakhloo, 2007). Cinnamic acid has drawn much interest as a potential therapeutic agent for the prevention and treatment of numerous diseases because of its various pharmacological effects, including antioxidant, anti-inflammatory, anti-diabetic, anti-cancer, and hepatoprotective properties (Hemmati et al., 2018). In addition, research has shown that cinnamic acid has protective effects against liver and kidney damage caused by various substances for instance acetaminophen. However, to the best of our knowledge, the therapeutic efficacy of cinnamic acid on cadmium induced toxicity has not been reported. Therefore, this study was designed to evaluate the possible protective effects of cinnamic acid on Cd-induced hepato-renal toxicity in male Wistar rats.

MATERIALS AND METHODS

Chemicals and Reagents

5,5'-dithios-bis-2-nitrobenzoic acid (DTNB), Epinephrine, reduced Glutathione and Cinnamic acid were purchased from Sigma Chemical Co., Saint Louis, MO USA. Thiobarbituric acid, cadmium chloride, trichloroacetic acid, sodium hydroxide and potassium chloride were procured from British Drug House (BDH) Chemical Ltd., Poole, UK. Other chemicals were of analytical grade.

Animals

Adult male Wistar rats (110 and 170 g) from the animal house of the Department of Veterinary Physiology, Biochemistry and Pharmacology, University of Ibadan, Nigeria were used for this study. The rats were kept in well aired cages at room temperature (28-30^{o}C) and fed with rat feed, from Ladokun Feeds, Ibadan, Nigeria, and water *ad libitum*. All experimental procedures were carried out in accordance with the NIH Guidelines for the Care and Use of Laboratory Animals.

Study design

Thirty male rats were randomly divided into six groups (n = 5). Group A (Control) received corn oil, group B received cadmium chloride (1.5 mg/kg), group C received cadmium chloride (1.5 mg/kg) and cinnamic acid (50 mg/kg), group D received cadmium chloride (1.5 mg/kg) and cinnamic acid (100 mg/kg), group E received cinnamic acid (50 mg/kg) only, and group F received cinnamic acid (100 mg/kg) only. The rats were pretreated with cinnamic acid via oral gavage for seven consecutive days, while the rats were challenged with two doses of cadmium chloride intraperitoneally at the last 72 h of the study. The doses used for cadmium chloride was based on our previous toxicity studies (Adaramoye and Akanni, 2015) while that of cinnamic acid was according to Babaeenezhad *et al*. (2021).

Preparation of samples

After the last dose of treatment, rats were fasted overnight and sacrificed under mild ether anaesthesia. Blood was collected from the animals into plain centrifuge tubes and allowed to stand for 1 hour before centrifugation. The supernatants (serum) were decanted into already labelled tubes and stored for biochemical analysis. Upon dissection, the kidneys were also excised and washed in ice-cold 1.15% KCl solution, dried and weighed. A kidney was fixed in 10% buffered formalin for histological examination. The remaining was homogenized in 4 volumes of 5 mM phosphate buffer (pH 7.4) and centrifuged at 10,000 g for 15 minutes to obtain post-mitochondrial supernatant fraction for biochemical indices.

Alanine and aspartate aminotransferases determination

Activities of aspartate aminotransferases (AST) and alanine aminotransferase (ALT) in the serum of rats were determined using the methods of Reitman and Frankel, (1957) and Mohun and Cook, (1957) respectively.

Urea and creatinine determination

The levels of urea and creatinine in the serum of rats were estimated by the methods of Talke and Schubert, (1965) and Jaffe, (1886) respectively.

Antioxidant assays

The activity of superoxide dismutase (SOD) was measured by the method of McCord and Fridovich, (1969). The level of reduced glutathione (a non-enzymatic antioxidant) in the liver and kidney was measured according to the technique of

Beutler *et al*. (1963), which is based on the development of a relatively stable (yellow) colour when 5',5'-dithiobis-(2-nitrobenzoic acid) (Ellman's reagent) is added to sulfhydryl compounds. The chromophoric product resulting from the reaction of Ellman's reagent with the reduced glutathione (2-nitro-5-thiobenzoic acid) possesses a molar absorption at 412 nm that is proportional to the level of reduced glutathione in the test sample. The level of lipid peroxidation in the liver and kidney were assayed by the reaction between 2-thiobarbituric acid (TBA) and malondialdehyde (MDA), an end product of lipid peroxidases described by Buege and Aust, (1978).

Histology

The tissues fixed in 10% formalin were dehydrated in 95% ethanol and then cleared in xylene before embedded in paraffin. Micro sections (4 µm) were prepared and stained with haematoxylin and eosin (H&E) dye, and were examined under a light microscope by a Histopathologist who was ignorant of the treatment groups. µmol/mg protein

Statistical analysis

Data were expressed as the Mean ± S. D. The values were analyzed using one-way ANOVA followed by the post-hoc Duncan's multiple range tests for analysis of biochemical data using SPSS (20.0). Values were considered statistically significant at $p< 0.05$.

RESULTS

Effects of cinnamic acid on body weight and relative weight of organs of cadmium chloride-exposed male wistar rats

There is significant decrease ($p<0.05$) in the body weight-gain in the cadmium-exposed rats when compared to the control (Table 1). Also, exposure to cadmium significantly ($p<0.05$) decreased the weight of liver relative to the control while no significant decrease was observed in the weight of kidney when compared to the control. The group treated with $CdCl_2$+CA1 (50 mg/kg) showed significant decrease in the weight gained when compared to the $CdCl_2$ group. However, treatment of Cd-exposed group with CA at 100 mg/kg significantly ($p<0.05$) increased both the weight and relative weight of the liver (Table 1).

Table 1: Effects of Cinnamic Acid on Body Weight and Relative Weight of Liver and Kidney in Cadmium Chloride-Exposed Male Wistar Rats

Treatments	Body Weight			Organ Weight			Relative Body Weight
	Initial (g)	Final (g)	Weight Gain (g)	Liver (g)	Kidney (g)	Liver (g)	Kidney (as % of body weight)
Control	119.97±5.41	147.38±13.86	27.41±8.45	6.62±0.48	0.93±0.05	4.26±0.02	0.63±0.03
$CdCl_2$	155.75±2.63	167.73±2.38	11.98±0.74*	6.16±0.08	1.09±0.02	3.73±0.10*	0.65±0.03
$CdCl_2$ + CA1	147.75±2.21	144.78±5.28	-2.97±3.07**	6.02±0.10	0.92±0.05	4.23±0.15**	0.64±0.05
$CdCl_2$ + CA2	138.25±1.25	150.65±1.74	12.24±0.24	7.19±0.08**	0.89±0.02	4.77±0.10**	0.59±0.02
CA1	131.00±15.68	170.98±24.98	39.98±9.30*	5.74±0.27*	1.08±0.05	3.40±0.40*	0.61±0.02
CA2	170.25±9.22	181.31±13.43	11.06±4.22*	6.83±0.40	1.06±0.04	3.36±0.19*	0.57±0.03*

Values are expressed as Mean ± S.D. (n=5)

* Significantly different from control at $p<0.05$, ** Significantly different from Cadmium Chloride group at $p<0.05$

$CdCl_2$ = Cadmium Chloride, CA1 = Cinnamic Acid at 50 mg/kg, CA2 = Cinnamic Acid at 100 mg/kg

Table 2: The Protective Effects of Cinnamic Acid on the Serum Indices of Cadmium Chloride – Exposed Male Wistar Rats

Treatments	CREATININE (mg/dL)	UREA (mg/dL)	AST (U/L)	ALT (U/L)
Control	1.00±0.00	2.25±0.19	106.08±5.33	57.86±0.13
$CdCl_2$	1.25±0.29*	4.20±1.04*	125.43±1.28*	55.63±0.86
$CdCl_2$ + CA1	0.5±0.00**	1.47±0.15**	107.07±6.58	51.43±0.62
$CdCl_2$ + CA2	0.75±0.35**	1.52±0.14**	114.43±4.38	58.04±2.65
CA1	0.50±0.00*	1.44±0.14*	113.73±2.25	57.47±0.36
CA2	0.50±0.00*	2.53±0.73	108.79±1.61	56.34±1.22

Values are expressed as Mean ± S.D. (n=5)

* Significantly different from control ($p<0.05$), ** significantly different from Cadmium Chloride group ($p<0.05$)

$CdCl_2$ = Cadmium Chloride, CA1 = Cinnamic Acid at 50 mg/kg, CA2 = Cinnamic Acid at 100 mg/kg

Effects of cinnamic acid on serum biochemical indices of kidney and liver function of cadmium chloride-exposed male wistar rats

The Table 2 depicts the level of serum creatinine, urea, AST and ALT where the creatinine and urea level significantly ($p<0.05$) increased in $CdCl_2$ exposed group by 20% and 86%, respectively, when compared to the control. Interestingly, pre-treatment with CA at 50 and 100 mg/kg before exposure to $CdCl_2$ significantly ($p<0.05$) decreased creatinine level by 60% and 40%. Similar patterns were observed for urea levels in groups pre-treated with CA at 50 and 100 mg/kg, the urea level in these groups significantly ($p<0.05$) decrease by 65% and 64%, respectively.

In addition, the activity of serum AST was significantly ($p<0.05$) increased by 18% in the $CdCl_2$ group when compared to the control. The observed increase in AST activity was restored to normal in the pre-treated groups with CA (50 and 100 mg/kg). On the other hand, no significant difference was

seen in the activity of ALT between the control and $CdCl_2$ exposed group.

Effects of cinnamic acid on antioxidant parameters, and histology of tissues of cadmium chloride-exposed male wistar rats

In Figure 1, the level of reduced glutathione (GSH) in the kidney tissue was significantly ($p<0.05$) decreased by 26% in the rats exposed to cadmium for 72 hours when compared to the control rats. However, no significant increase was observed in the level of reduced glutathione in the liver of $CdCl_2$ rats when compared to the control. Pre-treatment with cinnamic acid significantly ($p<0.05$) increased the level of hepatic GSH by 24% and 36% at 50 mg/kg and 100 mg/kg, respectively when compared to the $CdCl_2$ group. Conversely, insignificant ($p>0.05$) increase was observed in the level of renal GSH in the groups pre-treated with Cinnamic acid when compared to the $CdCl_2$ group. While in Figure 2, $CdCl_2$ exposure caused a significant ($p<0.05$) increase in the level of both renal and hepatic lipid peroxidation (LPO) by 41% and 71%, respectively when compared to the control rats.

hepatic LPO were observed in the $CdCl_2$ rats pre-treated with CA (100 mg/kg) when compared with the $CdCl_2$ group. In addition (Figure 3), $CdCl_2$ exposure significantly decreased the activity of renal superoxide dismutase (SOD) when compared to the control by 50% while pre-treatment with Cinnamic acid at 50 mg/kg and 100 mg/kg significantly increased the activity by 27% and 84%, respectively. In the liver, however, there was no significant difference in the activity of SOD of the cadmium-exposed rats when compared to the control rats but pre-treatment of the $CdCl_2$ rats with cinnamic acid (50 mg/kg) resulted in a significant ($p<0.05$) increase of 100% in the activity of SOD (Figure 3). Also, Figure 4 is the photomicrograph of the liver tissues. The results revealed normal architecture of the liver tissues in the control rats while there is fatty degeneration in the focal area of hepatocytes of the cadmium-exposed rats.

The rats pretreated with CA at 50 mg/kg also have normal hepatocytes while those pretreated with CA at 100 mg/kg and those rats that received only CA (50 and 100 mg/kg) had mild infiltration of the inflammatory cells (Figure 4D-F). Figure 5 shows the photomicrograph of the kidney tissues. Normal kidney tissue architecture was observed for rats in the control, cadmium-exposed rats and the group pretreated with CA at 50 mg/kg (Figure 5A – C_ On the other hand, the groups pretreated with CA at 100 mg/kg and those treated with CA only (50 and 100 mg/kg) revealed glomeruli with peripheral infiltration of inflammatory cells (Figure 5D-F)

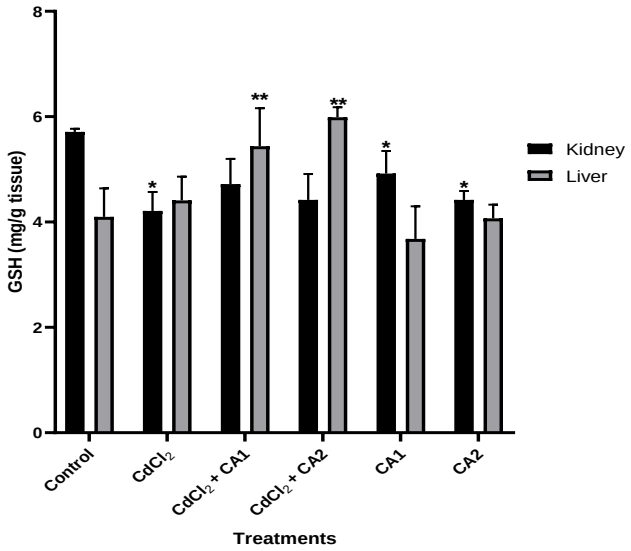


Figure 1: The effects of cinnamic acid on the level of reduced glutathione (GSH) in the kidney and liver of Cadmium-chloride exposed male Wistar rats.

Values are expressed as Mean ± S.D. (n=5)
*Significantly different from control ($p<0.05$), **Significantly different from Cadmium Chloride group ($p<0.05$)
$CdCl_2$ = Cadmium Chloride, CA1 = Cinnamic Acid at 50 mg/kg, CA2 = Cinnamic Acid at 100 mg/kg

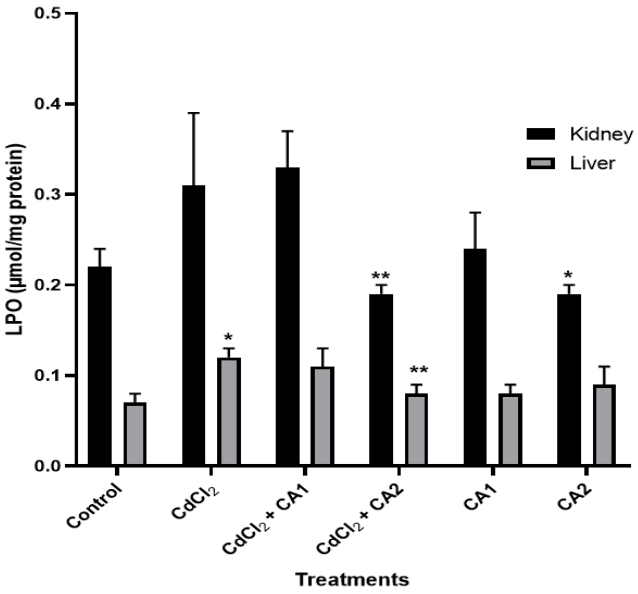


Figure 2: The effects of cinnamic acid on the level of lipid peroxidation (LPO) in the kidney and liver of Cadmium-chloride exposed male Wistar rats.

Values are expressed as Mean ± S.D. (n=5)
*Significantly different from control ($p<0.05$), **Significantly different from Cadmium Chloride group ($p<0.05$)
$CdCl_2$ = Cadmium Chloride, CA1 = Cinnamic Acid at 50 mg/kg, CA2 = Cinnamic Acid at 100 mg/kg

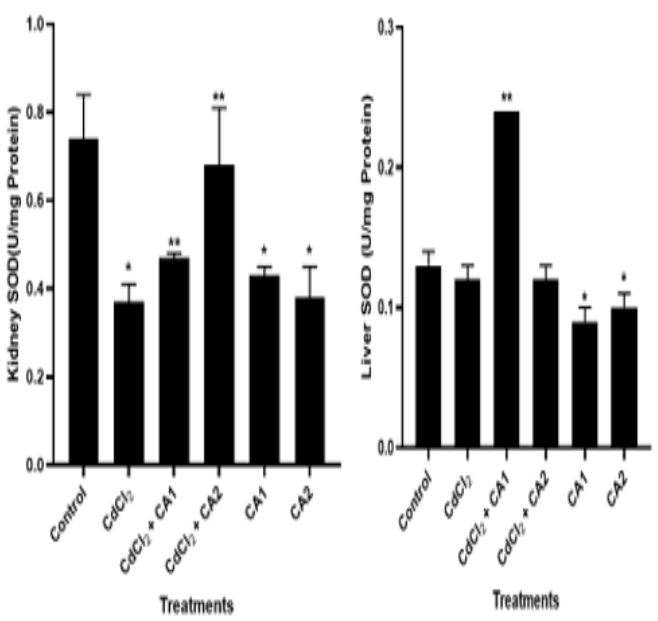


Figure 3: The effects of Cinnamic acid on the activity of Superoxide dismutase (SOD) in the kidney and liver of Cadmium-chloride exposed male Wistar rats.

Values are expressed as Mean ± S.D. (n=5)
*Significantly different from control ($p<0.05$), **Significantly different from Cadmium Chloride group ($p<0.05$)
$CdCl_2$ = Cadmium Chloride, CA1 = Cinnamic Acid at 50 mg/kg, CA2 = Cinnamic Acid at 100 mg/kg

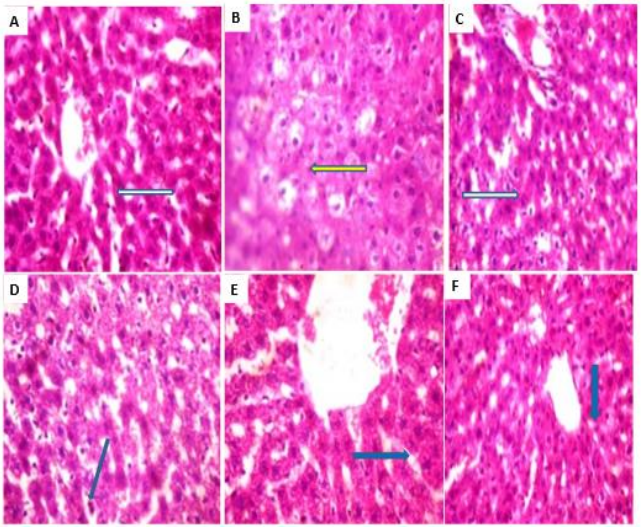


Figure 4: The photomicrograph of the liver of Cadmium-exposed rats pre-treated with Cinnamic acid for seven days (M X 400).

A = Control, B = $CdCl_2$ group (rats that received cadmium chloride only), C = $CdCl_2$ + CA1 (rats that received cadmium chloride and cinnamic acid at 50 mg/kg), D = $CdCl_2$ + CA2 (rats that received cadmium chloride and cinnamic acid at 100 mg/kg), E = CA1 group (rats that received cinnamic acid at 50 mg/kg only), F = CA2 (rats that received Cinnamic acid at 100 mg/kg only).

White arrows show normal hepatocytes, yellow arrow shows focal area of hepatocytes with fatty degeneration while the blue arrows show the sinusoids with mild infiltration of inflammatory cells.

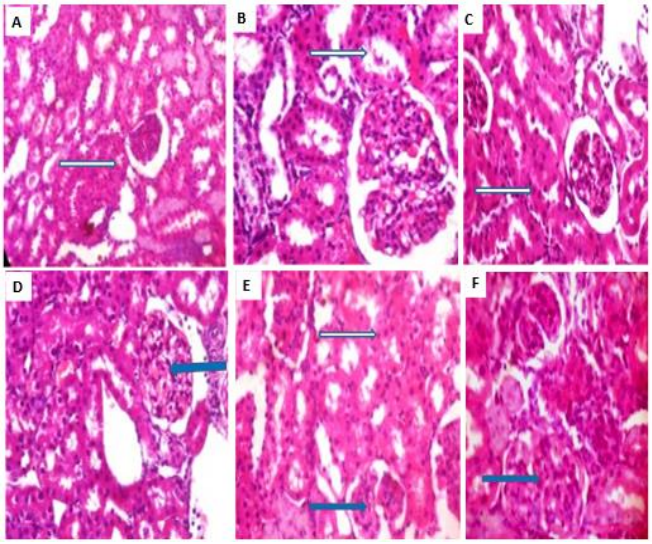


Figure 5: The photomicrograph of the kidney of Cadmium-exposed rats pre-treated with Cinnamic acid for seven days (M X 400)

A = Control, B = $CdCl_2$ group (rats that received cadmium chloride only), C = $CdCl_2$ + CA1 (rats that received cadmium chloride and cinnamic acid at 50 mg/kg), D = $CdCl_2$ + CA2 (rats that received cadmium chloride and cinnamic acid at 100 mg/kg), E = CA1 group (rats that received cinnamic acid at 50 mg/kg only), F = CA2 (rats that received cinnamic acid at 100 mg/kg only).

White arrows show normal glomeruli with normal mesengial cells and capsular spaces while the blue arrows show glomeruli with peripheral infiltration of inflammatory cells

DISCUSSION

Cadmium exposure via water, air, contaminated food, manufactured goods, commercial phosphate fertilizers or occupational hazards can result in its intoxication (Hayat et al., 2019). The most susceptible organs to cadmium toxicity are the liver and kidney according to Ojo *et al.* (2014). Previous studies have highlighted oxidative stress, apoptosis, inflammation and autophagy to be significant in the etiology and pathophysiology of hepatotoxicity and nephrotoxicity from cadmium exposure (Patra et al., 2011, Yuan et al., 2014; El-Boshy et al., 2015). Even though cadmium intoxication has been linked to testicular damage, its accumulation in human tissues like the kidney and liver is a matter of concern in many medical conditions (Ojo et al., 2014; Adaramoye and Akanni, 2015).

In order to control the progression of oxidative stress-related diseases, several plant-based compounds with antioxidant properties have been explored as chemopreventive agents in experimental studies. Cinnamic acids have been recognized as interesting compounds with anti-inflammatory, antioxidant and cytotoxic properties (Abozaid et al., 2019). In this study, the protective effects of cinnamic acid on cadmium chloride-induced hepato-renal damage were evaluated. The result of the study shows that pre-treatment with cinnamic acid could prevent liver and kidney damage in rats exposed to cadmium intoxication.

The results presented in this study show a significant increase in the levels of urea and creatinine in cadmium chloride exposed rats when compared to the control. Kidneys have been documented to be the one of the target tissues affected with the toxic effects of cadmium (Wang et al., 2014; Satarug et al., 2017). Cadmium toxicity can lead to tubular dysfunction, reduced glomerular filtration and ultimately kidney failure (Fahim et al., 2012) characterized by paler cytoplasm, disrupted nuclei, and scanty chromatin. Also, the rate at which urea is produced in the serum is usually greater than the rate of its clearance in renal diseases and this will result in uremia (Adaramoye and Akanni, 2016) which was established in this study. Interestingly, pre-treatment with cinnamic acid was able to reduce the urea and creatinine level significantly and this is an indication of its nephro-protective properties.

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are known as definite marker enzymes that are raised in cases of hepatic toxicity (EL-Geddawy et al., 2019). Indeed, it has been reported consistently that the activity of serum AST and ALT are significantly increased in rats exposed to cadmium chloride (Ibraheem et al., 2016; Alkushi et al., 2018). Findings in this study however only showed significant increase in the activity of serum AST with no significant change in that of ALT.

Antioxidant enzymes are major defensive mechanisms against reactive oxygen species. Superoxide dismutase is an endogenous antioxidant enzyme that catalyses the dismutation of superoxide radical to oxygen molecule and hydrogen peroxide, by reduction leading to the accumulation of hydrogen peroxide (Wang et al., 2014) which is the less reactive species while reduced glutathione (GSH) is a tripeptide, non-enzymatic antioxidant, that protects against reactive oxygen species and detoxification of exogenous and endogenous toxins of an electrophilic nature. A decline in the level of these antioxidants can lead to the generation of oxidative stress. The results in this study showed a significant decrease in the renal activity and level of both enzymatic (SOD) and non-enzymatic antioxidant enzyme (GSH), respectively. This could be an indication that cadmium chloride generates free radicals that can interact with the polyunsaturated fatty acids thereby causing lipid peroxidation. The result is tandem with the results of Navaneethan and Rasool. (Navaneethan and Rasool, 2014) and Prabu *et al*. (2013) which reported that cadmium exposure increases lipid peroxidation and causes inhibition of antioxidant enzymes, resulting in oxidative damage to heart, kidney and testes. On the other hand, the hepatic activities of SOD and GSH in cadmium chloride-exposed group did not seem to show any significant difference with what was observed in the control. This may mean that the concentration of cadmium chloride used in this study was not high enough to inhibit SOD and GSH activities in the liver. However, pre-treatment with cinnamic acid before exposure to cadmium chloride raised hepatic GSH levels, which is in support of the report of Abozaid et al. (2019) which states that cinnamic acid improved GSH level due to its ability to decrease ROS levels and improving the GSH/GSSG ratio thereby counteracts increased ROS levels.

Cadmium chloride has been reported to stimulate lipid peroxidation by increasing the rate of production of superoxide radical that can act as a primary oxidant (Ojo et al., 2023). This progression can result in direct cell damage by destroying membranes and indirect cell damage by generating reactive carbonyl products. One of the end products of lipid peroxidation is malondialdehyde which is an indicator of oxidative stress that may even damage the membrane proteins and lipids. In this study, we observed a significant ($p<0.05$) increase in the level of hepatic and renal lipid peroxidation (LPO) in the rats exposed to cadmium chloride.

The photomicrograph of the hepatocytes shows area with fatty degeneration in rats exposed to cadmium chloride while rats pre-treated with cinnamic acid at 50 mg/kg had normal hepatocytes. Although mild infiltration of inflammatory cells was observed in groups pretreated with 100 mg/kg and those treated with cinnamic acid only. Also, peripheral infiltration of inflammatory cells was observed in the glomeruli of $CdCl_2$-exposed rats and those pretreated with 100 mg/kg of cinnamic acid as well as those treated with CA only (50 mg/kg and 100mg/kg) suggesting the pro-oxidant effect of cinnamic acid. This finding supports the pro-oxidant likelihood of cinnamic acid reported in the work of Lee and his team (Lee et al., 2022). However, rats pretreated with 50 mg/kg of cinnamic acid shows normal glomeruli as well as normal mesengial cells.

In conclusion, our study reveals that cinnamic acid protects against cadmium-induced hepato-renal toxicity by reducing the elevated level of malondialdehyde and restoring the serum and antioxidant indices in the liver and kidney tissue of male Wistar rats. Further studies are necessary to reveal detailed molecular mechanism of action of cinnamic acid against cadmium-induced toxicity.

CONCLUSION

The overall result of this study provides epidemiological data on the prevalence of *A. lumbricoides, E. histolytica/dispar*, Hookworm and *T. trichiura* infections which will enhance the baseline data in providing suitable control strategies. The result shows that enteric parasites are more prevalent among non-breastfeeding mothers and their infants. But whether lactation and/or breastfeeding reduce risk of maternal parasitic infections is uncertain and needs further investigation. Proper sanitary practice and exclusive breastfeeding as already being advocated should therefore be encouraged.

AUTHORS' CONTRIBUTIONS

Oluwapelumi Olaniyi: Writing – original draft, Methodology,
Opeyemi Edu: Writing – original draft, Project administration, Methodology, Investigation
Omowumi Sunday: Writing – original draft, Methodology.
Olubukola Agboola: Writing – original draft, Methodology, Conceptualization.

Adebukola Adeyanju: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization.
Babatunde Oso: Investigation, Formal analysis, Conceptualization.
Olayinka Adebayo: Writing – review & editing, Writing – original draft, Methodology
Adepeju Aberuagba: Investigation, Formal analysis, Conceptualization

CONFLICT OF INTEREST

We had no conflicts of interest associated with this publication and no significant financial support for this work that could influence its outcome.

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