

Vitamin C supplementation attenuates cadmium-associated renal biochemical and oxidative alterations in Wistar Albino rats

Sarra Derbal^{1*}, Abdelmalek Arabi²

¹ Laboratory of Animal Ecophysiology, Badji Mokhtar - Annaba University, 12, P.O.Box, 23000 Annaba, Algeria

² Research Laboratory on Phenoculture "Phoenix," Kasdi Merbah University - Ouargla 30000, Algeria

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* Corresponding author:

Sarra Derbal

Email: mllederbalsara23@gmail.com,

Sarra.derbal@univ-annaba.dz

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Abstract

The present study aimed to evaluate the nephroprotective potential of vitamin C on biochemical parameters and oxidative biomarkers in rats exposed to subchronic dose of cadmium chloride (200 mg/L) for four weeks. The rats were placed in eight cages, with five rats per cage. The cages were divided into four groups: a control group, a vitamin C group with vitamin C supplementation in drinking water (1 g/L), a cadmium chloride group receiving cadmium chloride in drinking water (200 mg/L), and a combined-treatment group receiving both cadmium chloride (200 mg/L) and vitamin C (1 g/L) in drinking water (Cd-vit C). Results revealed that cadmium-exposed group had significantly lower body weight, with increased absolute and relative kidney weights, along with higher circulating levels of urea, creatinine, and uric acid. Cadmium exposure reduced glutathione content and the activities of glutathione peroxidase, catalase, and superoxide dismutase in kidneys, whereas the malondialdehyde levels increased. Cadmium chloride exposure adversely affected body weight and renal weight, along with adverse effects on renal biochemical and antioxidants parameters. However, co-supplementation with vitamin C partially attenuated these negative changes.

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1. Introduction

The increasing health risks posed by heavy metals to human and animal populations constitute a major global environmental challenge (Zhang et al. 2024). Cadmium is a widespread and highly toxic heavy metal that has emerged as a major industrial and environmental pollutant, contaminating soil, aquatic ecosystems, the atmosphere, and food chains (Nordberg and Nordberg 2025). The entry of cadmium into surface waters occurs mainly through industrial effluents, while its accumulation in soil ecosystems is largely attributed to the leaching of sewage sludge (Cinar et al. 2010). In addition, cadmium is a biological toxic substance with an exceptionally prolonged half-life ranging from 20 to 40 years. Cadmium accumulates mainly in liver and kidney tissues (Genchi et al. 2020). Chronic cadmium exposure is the main cause of renal tubulopathy and is also involved in the development of osteomalacia and diffuse osteoporosis, which underlines an acute vulnerability of the kidneys to the bioaccumulation of cadmium (Genchi et al. 2020). Cadmium-induced free radicals play a role not only in all stages of cancer development but also in the activation of certain proto-oncogenes (Badawi et al. 2024). Furthermore, exposure to reactive oxygen species can promote oxidative DNA damage, including the oxidation of guanine to 8-hydroxy-2'-deoxyguanosine (Chirocea-Paquim 2022). Toxicity of cadmium is largely linked to oxidative alterations of cellular components, primarily due to the overproduction of reactive oxygen species (Irato and Santovito 2021). Prolonged exposure leads to renal tubular dysfunction because the metal accumulates

continuously until it exceeds a critical tissue threshold within the kidneys. The physiological disturbances due to cadmium toxicity highlight the role of antioxidant defense mechanisms in protecting the body against structural and functional alterations (Al sammak et al. 2021).

Ascorbic acid acts as a prolific electron donor that has a potential to protect essential biomolecules, including proteins, lipids, carbohydrates, and nucleic acids, against intracellular oxidative degradation. Furthermore, it mitigates the inflammatory cascades and regulate vital biological processes, thereby modulating the immune functioning of body (Al sammak et al. 2021). In addition to its antioxidant role, vitamin C has been frequently investigated for its potential to combat liver and kidney toxicity induced by pharmaceutical substances (Alsudani 2022). Previous research has shown that ascorbic acid prophylaxis effectively protects against marked histopathological changes in the kidneys of rats exposed to chronic cadmium toxicity (Okwuonu et al. 2020). However, further studies are required to ascertain the extent to which vitamin C administration protects the renal tubular cells against chronic cadmium-induced oxidative damage in rat models. Therefore, in this study it was hypothesized that preventive co-supplementation with vitamin C protects against cadmium-induced renal tubular damage in Wistar Albino rats by suppressing oxidative stress and preserving renal tissue architecture. To test this hypothesis, the present study evaluated whether co-supplementation of vitamin C attenuates cadmium-associated renal dysfunction.

2. Materials and Methods

2.1 Animal Ethics Statement

All experimental procedures were conducted on rats over four weeks in accordance with applicable international and national animal ethical guidelines that were in line with National Institutes of Health guidelines. The research was approved by the Animal Ethics Committee (approval number: D01N01UN230120150001).

2.2 Reagents and Animals

All chemicals used in the experiments were obtained from Sigma Chemical Co. (St. Louis, Missouri, USA). A total of 40 adult female Wistar albino rats (8-10 weeks old), obtained from the Pasteur Institute of Algeria, were acclimatized for two weeks under standardized conditions of 12 h light/13 h dark photoperiod, 40% relative humidity, and an ambient temperature of $22 \pm 2^\circ\text{C}$. The animals were randomly assigned to four groups and housed in standard polypropylene cages, with five rats per cage and eight cages in total. Dust-free wood shavings were replaced daily to maintain hygiene; and ensure minimize cage-effect bias and prevent overcrowding. Throughout the 28-day experimental period, rats had *ad libitum* access to water and standard commercial feed (ONAB, El-Harrouch, Algeria), with each animal serving as the primary experimental unit for all body weight, physiological, and biochemical evaluations.

2.3 Experimental Design and Grouping

Forty rats were housed in plastic cages inside the animal facility, with five animals per cage, and were provided with water and food throughout the 28-day experimental period. One group served as the control group, while the others received, respectively, vitamin C in their drinking water (1 g/L), cadmium chloride (200 mg/L), and cadmium chloride (200 mg/L) plus vitamin C (1 g/L). The concentrations of the chemicals were based on previous data in the literature (Okwuonu et al. 2020; Shalan et al. 2018). All conditions strictly complied with the guidelines of the National Institutes of Health. The institutional ethics committee approved the experimental method. The treatments were co-administered continuously over a four-week period, during which individual body weight, daily food intake and water intake were regularly monitored and recorded. Cadmium chloride (200 mg/L) and vitamin C (1 g/L) were administered in distilled drinking water *ad libitum* for four weeks. Fresh solutions were prepared daily in light-protected bottles. Based on daily water intake (Table 1), the estimated daily doses were 22.23 - 29.89 mg CdCl_2 /kg body weight and 149.48 - 181.95 mg vitamin C/kg body weight.

2.4 Tissue Collection and Biochemical Assays

After four weeks of the experimental period, the rats were euthanized, using the method approved by Institutional Animal Ethics Committee, by a trained personnel to minimize the stress induced biochemical changes to the maximum possible extent. Blood samples were collected in appropriate test tubes and serum was harvested from clotted blood after centrifugation to evaluate the renal biochemical indicators. The kidneys were then removed, weighed, and stored in plastic bags at -20°C . The frozen kidney tissues were subsequently thawed for the estimation of antioxidant enzyme activities, malondialdehyde (MDA) concentration as a marker of lipid peroxidation, and reduced glutathione (GSH) levels as a non-enzymatic antioxidant.

2.5 Analytical methods

2.5.1 Determination of renal biochemical markers

Uric acid, creatinine, and urea concentrations were measured using commercially available diagnostic kits (Spinreact, Spain; ref. Creatinine-1001111, Urea-1001329, and Uric acid-1401001011).

2.5.2 Tissue preparation and determination of oxidative stress parameters

Approximately 1 g of kidney tissue was homogenized in 2 mL of Tris-buffered saline (TBS; pH 7.4). The homogenates were then centrifuged at $10,000 \times g$ for 15 minutes at 4°C , and the resulting supernatant was used to determine MDA, GSH, GSH-Px, CAT, and SOD activities and total renal protein content.

2.5.3 Assessment of lipid peroxidation (MDA)

The extent of lipid peroxidation in kidney homogenates was determined by assaying malondialdehyde (MDA), which reacts with thiobarbituric acid (TBA) to generate a red-colored Thiobarbituric Acid Reactive Substances (TBARS) adduct. Absorbance recorded at 532 nm, based on the protocol of Ohkawa et al. (1979), with minor modifications.

2.5.4 Determination of reduced glutathione (GSH)

Based on the procedure described by Jollow et al. (1974), with minor modifications, renal GSH content was measured colorimetrically through the reaction of sulfhydryl groups with 5, 5'-dithiobis-2-nitrobenzoic acid (DTNB). Protein precipitation was done by adding 0.3 mL of 25% sulfosalicylic acid to 0.8 mL of renal supernatant, followed by centrifugation at $3,000 \times g$ for 10 min at 4°C . Next, 0.2 mL of the recovered supernatant was mixed with 1.0 mL of 0.1 M phosphate buffer (pH 7.4) and 0.25 mL DTNB (0.01 M), followed by incubation at 25°C for 5 min. Optical density was measured at 412 nm. GSH content was calculated using the molar extinction coefficient ($\epsilon = 13.6 \text{ mM}^{-1}\text{cm}^{-1}$) as follows:

$$\text{GSH (nmol/mg tissue)} = (A_{412} \times V_{\text{total}}) / (\epsilon \times l \times V_{\text{sample}} \times W)$$

using the precipitation dilution factor $D = 1.375$, where, A_{412} is absorbance at 412 nm, l is optical path length (cm), V_{total} is total reaction volume, V_{sample} is volume of tissue extract, W is tissue weight (g).

2.5.5 Assay of glutathione peroxidase (GSH-Px)

Glutathione peroxidase activity was measured according to the method described by Weydert and Cullen (2010), with minor modifications. The supernatant obtained after centrifuging the kidney tissue homogenate (1 g of tissue in 2 mL of TBS buffer, 1:2 w/v) at $10,000 \times g$ for 15 min at 4°C was used for the assay. The 1 mL reaction mixture contained 0.3 mL of phosphate buffer (0.1 M, pH 7.4), 0.2 mL of GSH (2 mM), 0.1 mL of sodium azide (10 mM), 0.1 mL of H_2O_2 (2.5 mM), and 0.3 mL of kidney supernatant. After incubation at 37°C for 15 min, the reaction was stopped by adding 0.5 mL of 5% trichloroacetic acid (TCA). The tubes were centrifuged at $1,500 \times g$ for 5 min and the supernatant was collected. Then 0.2 mL of phosphate buffer and 0.7 mL of DTNB (5,5'-dithiobis-2-nitrobenzoic acid, 0.4 mg/mL) were added to 0.1 mL of the reaction supernatant. The reaction was incubated at 25°C for 5 min and absorbance was measured at 420 nm. GSH-Px activity was calculated from the rate of GSH oxidation as follows:

$$\text{GSH-Px Activity} = (\text{GSH}_{\text{control}} - \text{GSH}_{\text{sample}}) / (\Delta t \times W),$$

The values were expressed as $\mu\text{mol GSH consumed/min/g tissue}$, whereas Δt is incubation time (min), and W is tissue weight (g).

2.5.6 Catalase assay (CAT)

Catalase activity was determined using the method described by Weydert and Cullen (2010), with minor modifications. A reaction mixture with 0.78 mL of phosphate buffer (0.1 M, pH 7.4), 0.2 mL of renal supernatant, and 0.02 mL of H₂O₂ (0.5 M) was prepared. The reaction was initiated by the addition of H₂O₂, and its decomposition was monitored by measuring the drop in absorbance at 240 nm every 15 seconds for 1 minute at 25 °C. Enzyme activity was calculated with an extinction coefficient (ϵ) of 0.043 mM⁻¹cm⁻¹ as follows:

$$\text{CAT Activity (U/mg protein)} = (\Delta A_{240/\text{min}} \times V_{\text{total}}) / (\epsilon \times l \times V_{\text{sample}} \times C_{\text{protein}})$$

where, $\Delta A_{240/\text{min}}$ is change in absorbance per minute at 240 nm, V_{total} is total reaction volume, l is optical path length (cm), V_{sample} is volume of enzyme extract added to the reaction, C_{protein} is protein concentration in the enzyme extract (mg/mL).

2.5.7 Superoxide dismutase assay (SOD)

SOD activity was measured by the modified procedure described Ehimigbai et al. (2025). Briefly, the procedure involved dilution of 10 μ L tissue supernatant in 970 μ L of EDTA-sodium carbonate buffer (0.05 M, pH 10.2). The reaction was initiated with 20 μ L of 30 mM epinephrine, and the absorbance was measured at 480 nm for 4 min at 25°C. One unit (U) of SOD activity was defined as the amount of enzyme causing 50% inhibition of epinephrine autoxidation. The activity was calculated as follows:

$$\% \text{ Inhibition} = [(\Delta A_{\text{control}} - \Delta A_{\text{sample}}) / \Delta A_{\text{control}}] \times 100$$

$$\text{SOD Activity (U/mg protein)} = \% \text{ Inhibition} / (50 \times C_{\text{protein}} \times V_{\text{sample}})$$

where, $\Delta A_{\text{control}}$ is change absorbance of control, ΔA_{sample} is change absorbance of sample, C_{protein} is protein concentration of extract, and V_{sample} is volume of the tissue extract added.

2.5.8 Protein determination in tissue

Protein levels were estimated using the method described by Kieljopf et al. (2020), with bovine serum albumin serving as the calibration standard.

2.6 Statistical analysis

Data are presented as mean $\pm$ standard error of the mean (Mean $\pm$ SEM).

Before statistical analysis, normality of the data and homogeneity of the variances were assessed using the Anderson-Darling test and Bartlett tests, respectively. The differences between the four independent treatment groups were analyzed by one-way analysis of variance (ANOVA), followed by the Tukey's post hoc test. The significance level was set at $p < 0.05$. The mean values with different superscript letters were significantly different from each other at $p < 0.05$. All statistical analyses were performed using MINITAB software (version 18.0, Minitab Inc., State College, PA, USA).

3. Results

3.1 Effect on weight parameters

Animals exposed to cadmium revealed a significant decline in body weight, food intake, and water intake compared with the control animal group ($p < 0.05$). However, absolute and relative kidney weights increased significantly in rats exposed to cadmium ($p < 0.05$). In rats exposed to cadmium and treated with vitamin C (Cd-Vit C), vitamin C intake partially attenuated these changes (Table 1).

3.2 Effect on renal biochemical parameters

Cadmium exposure resulted in significant elevation of blood urea, creatinine, and uric acid levels compared with the control cohort ($p < 0.05$). In rats exposed to cadmium and treated with vitamin C (Cd-Vit C), vitamin C co-supplementation partially attenuated these biochemical alterations, bringing the values closer to those in the control group (Table 2).

3.3 Effects of the treatment on renal antioxidant parameters

As shown in Fig. 1A – E, cadmium exposed rats showed a significant increase in MDA levels ($p < 0.05$) paralleled by decrease in the levels of GSH and tissue protein, and activities of SOD, GSH-Px, and CAT ($p < 0.05$). In rats exposed to cadmium and treated with vitamin C (Cd-Vit C), vitamin C co-supplementation partially attenuated these biochemical alterations, bringing their values closer to those in the control cohort.

4. Discussion

To assess the protective effects of ascorbic acid against cadmium-induced renal dysfunction and oxidative alteration, rats were exposed to cadmium chloride for a four-week duration. Cadmium exposure

Table 1. Initial body weight, final body weight, food intake, water intake, and absolute and relative kidney weights of rats

Parameters	Experimental groups			
	Control	Vitamin C	Cadmium	Cadmium-Vitamin C
Initial Body Weight (g)	192 $\pm$ 9.35 ^a	193 $\pm$ 11.19 ^a	209 $\pm$ 14.34 ^a	208 $\pm$ 9.41 ^a
Final Body Weight (g)	248 $\pm$ 11.51 ^a	219 $\pm$ 15.11 ^b	170 $\pm$ 19.16 ^c	222 $\pm$ 9.53 ^b
Kidney Absolute Weight (g)	1.03 $\pm$ 0.07 ^c	1.04 $\pm$ 0.02 ^c	1.27 $\pm$ 0.2 ^b	1.16 $\pm$ 0.40 ^a
Kidney Relative Weight (%body weight)	0.41 $\pm$ 0.02 ^c	0.47 $\pm$ 0.01 ^c	0.74 $\pm$ 0.01 ^a	0.52 $\pm$ 0.18 ^b
Food Intake (g/day/rat)	22 $\pm$ 2.59 ^a	22 $\pm$ 2.63 ^a	11 $\pm$ 2.25 ^c	15 $\pm$ 1.95 ^b
water Intake (mL/day/rat)	37.2 $\pm$ 1.80 ^a	36.48 $\pm$ 2.70 ^a	22.29 $\pm$ 7.28 ^c	29.97 $\pm$ 3.91 ^b

Values are expressed as mean $\pm$ SEM ($n = 5$ per group). Means with different superscript letters (a, b, c) indicate statistically significant differences among groups ($p < 0.05$) based on tukey's post-hoc test

Table 2. Serum renal function biomarkers (creatinine, uric acid, and urea) of rats

Parameters	Experimental groups			
	Control	Vitamin C	Cadmium	Cadmium-Vitamin C
Serum creatinine (mg/dl)	1.14 ± 0.06 ^c	1.10 ± 0.05 ^c	1.74 ± 0.07 ^a	1.27 ± 0.15 ^b
Serum urea (mg/dl)	50.33 ± 17.82 ^c	51.5 ± 5.43 ^c	75.83 ± 0.98 ^a	56.00 ± 11.54 ^b
Serum uric acid (mg/dl)	8.10 ± 0.99 ^c	8.15 ± 0.49 ^c	11.25 ± 1.22 ^a	9.07 ± 1.75 ^b

Values are expressed as mean ± SEM (n = 5 per group). Means with different superscript letters (a, b, c) indicate statistically significant differences among groups (p < 0.05) based on tukey's post-hoc test

reduced body mass gain, food intake, and water intake relative to control group. According to the existing literature reports, such metabolic alterations may be associated with metal-induced perturbations in lipid metabolism, accelerated protein catabolism, or impaired intestinal nutrient absorption (Haidar et al. 2023; Erdogan et al. 2005). An increase in the absolute weight of the kidneys was observed in cadmium-exposed rats and previous studies have linked similar weight changes to tissue hypertrophy, cellular swelling, or renal edema (Poli et al. 2022). However, the ameliorative effects of vitamin C co-supplementation was evident in partially mitigating these systemic impairments, improving food and water intake, final body weight, and organ-to-body-weight ratios. These results are consistent with established empirical studies demonstrating that antioxidant agents, such as vitamin C, zinc, and N-acetylcysteine, are effective in mitigating the toxicity caused by cadmium, attenuating disruption in metabolic homeostasis, thereby partially restoring food and water consumption

and organ weight parameters (Poli et al. 2022). A significant response to cadmium toxicity was the concurrent systemic body weight loss and the increased kidney mass. The increase in relative kidney weight suggests potential organ swelling or structural enlargement, as described in studies on heavy metal toxicity (Poli et al. 2022).

Higher circulating urea and creatinine levels in cadmium-exposed rats suggest renal impairment. Given that these waste products are routinely used to assess glomerular filtration and clearance efficiency, their increased levels underscore the potent nephrotoxic action of cadmium (Almaimouni and Roshdy 2022; Ammari et al. 2025). Furthermore, elevated serum urea levels accompanied by decreased total serum protein levels suggest potential increased systemic protein catabolism. These observation are in line with previous studies reporting cadmium-induced nephrotoxicity (El-Demerdash et al. 2009; Bautista et al. 2024). Vitamin C co-administration partially preserves renal function through its ability to attenuate cadmium-induced renal

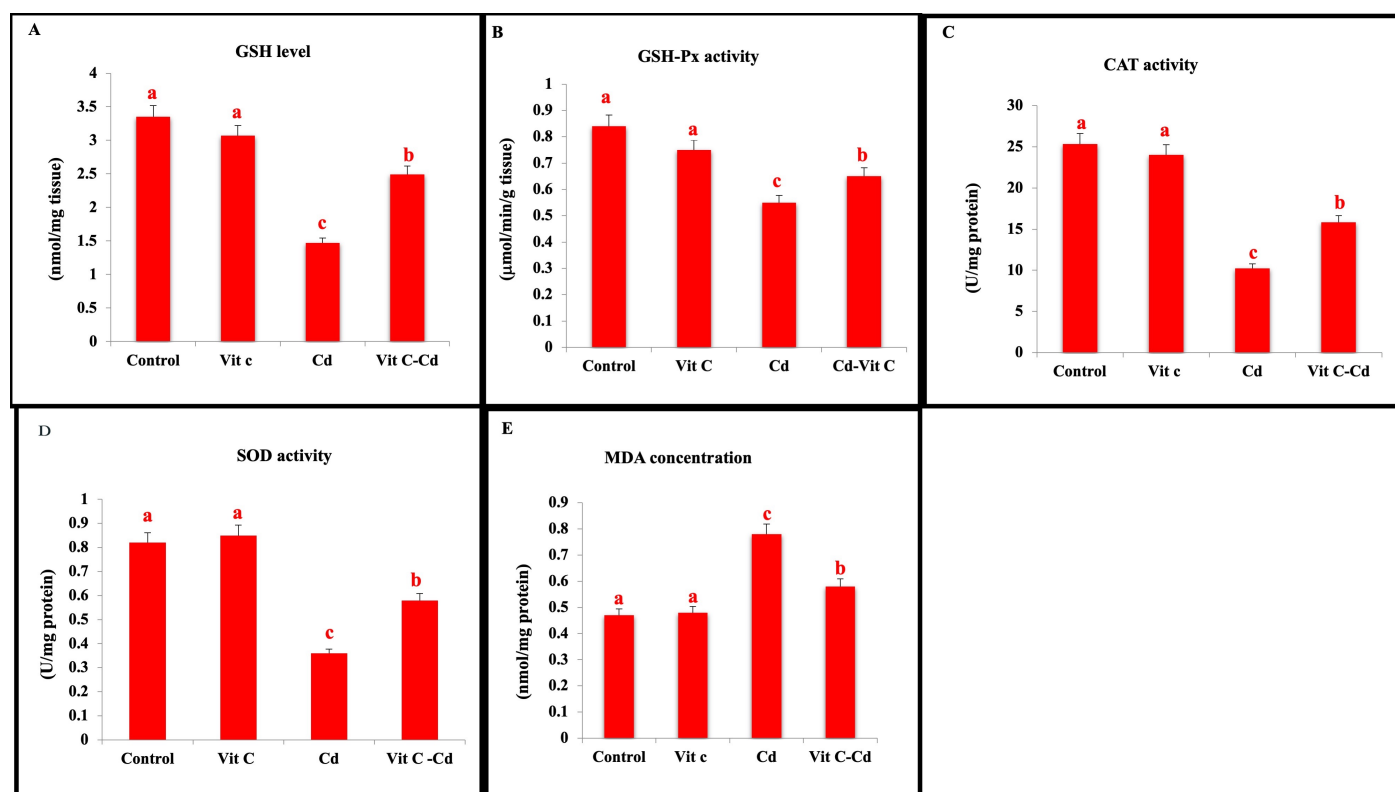


Fig. 1. Renal reduced glutathione (GSH) content (A), antioxidant enzyme activities - GSH-Px (B), CAT (C), SOD (D), and malondialdehyde (MDA) levels (E) of rats in control, cadmium (Cd), vitamin C (Vit C), and cadmium combined with vitamin C (Cd-Vit C) groups after four weeks of exposure. Values are expressed as mean ± SEM (n = 5 per group). Means with different superscript letters (a, b, c) indicate statistically significant differences among groups (p < 0.05) based on tukey's post-hoc test

tubular dysfunction (Ouyang et al. 2022). Under physiological conditions, the generation of free radicals is an integral component of cell metabolism, including mitochondrial electron transport. Cadmium exposure potentially increases the formation of reactive oxygen species (ROS) under oxidative stress and consequently, an imbalance develops between ROS production and antioxidant defenses (Qu and Zheng 2024; Badawi et al. 2024). At the cellular level, exposure of Wistar rats to cadmium chloride increases lipid peroxidation and decreases the activities of CAT, SOD, and GPx activities, resulting in significant renal oxidative damage (Karami et al. 2022). The observations made in this study are consistent with previous studies that oral administration of CdCl₂ increases tissue MDA levels while depleting endogenous GSH reserves and SOD activity in the liver and kidney parenchyma (Karabulut-Bulan et al. 2008). The present study suggests the protective role of vitamin C against the cadmium-induced renal oxidative stress, which helps maintain the normal kidney biochemical parameters (Prabu et al. 2010). Furthermore, co-administration of vitamin C with cadmium led to the restoration of antioxidant defenses in the renal tissue towards control levels. This nephroprotective effect of vitamin C can be partly linked to its ability to scavenge free radicals and thereby enhance the renal resistance to oxidative stress (El-Sokkary and Awadalla 2011).

5. Conclusions

In conclusion, cadmium exposure of Wistar albino rats adversely affected the kidney function and antioxidant indicators. However, the co-administration of vitamin C partially ameliorated these adverse effects, indicating the potential protective role of vitamin C against the cadmium-induced renal damage.

Declarations

Funding: None

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Data availability statement: Data associated with this study is available from the corresponding author upon reasonable request

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