Caspian Journal of Environmental Sciences
Online ISSN: 1735-3866
Print ISSN: 1735-3033

Effects of cadmium chloride on the ultrastructure of animal somatic cells (gums, kidneys, and liver)

Zina B. Tungushbaeva[1,2]*, Rakhat K. Pernebekova[3,4]*, Nurzhamal M. Joldasbayeva[2,5], Gabit Sharipov[6], Sarzhan Sharipova[7], Zannym M. Yermentayeva[5], Indira Z. Kaketaeva[5], Kuanysh Syman[2]

1. Head of the Department of Molecular Biology, Kazakh-Russian Medical University, Almaty, Kazakhstan
2. Department of Biology, Faculty of Natural Sciences and Geography, Abai Kazakh National Pedagogical University, Almaty, Kazakhstan
3. Department of Pharmacology, Pharmacotherapy and Clinical Pharmacology, Faculty of Pharmacy, South Kazakhstan Medical Academy, Shymkent, Kazakhstan
4. Department of «General Practitioner No 2», Faculty of Postgraduate Medical Education, Khoja Akhmet Yassawi International Kazakh-Turkish University, Turkestan, Kazakhstan
5. Head of the Department of Anatomy with courses, Kazakh-Russian Medical University, Almaty, Kazakhstan
6. Department of Civil Defense and Military Training, Malik Gabdullin Academy of Civil Protection of the Ministry of Emergency Situations of the Republic of Kazakhstan, Kokshetau, Kazakhstan
7. Department of Toxicological Chemistry, Asfendiyarov Kazakh National Medical University, Almaty, Kazakhstan

* *Corresponding author's E-mail: alua2020@yandex.kz, rakhat_71@mail.ru*

ABSTRACT

Cadmium is a toxic heavy metal with the ability to accumulate in various organs of the body that can cause severe cellular damage. This study was conducted to investigate and compare the microstructural changes caused by cadmium chloride in three important somatic tissues including gums, kidney and liver in an animal model. In this experimental study, 30 male rats were divided into three groups: control, low dose (2 mg kg^{-1}) and high dose (5 mg kg^{-1}) of cadmium chloride for 28 days. After the end of the period, tissue samples were collected and examined using transmission electron microscopy. Qualitative changes were recorded and quantitative parameters including the percentage of swollen mitochondria, nuclear chromatin condensation index and the number of cytoplasmic vacuoles were measured by ImageJ software and analyzed by appropriate statistical tests. The results of the study confirmed severe and dose-dependent damage in all tissues. The percentage of swollen mitochondria in hepatocytes increased from 2.4% in the control group to 3.72% in the high-dose group, a significant increase ($p < 0.05$). The liver showed the highest sensitivity, followed by the kidney and then the gingiva. The chromatin condensation index in gingival cells at the high dose reached 2.7, indicating significant nuclear changes in this tissue. A strong dose-response relationship was observed for all measured parameters (Pearson correlation r = 0.98 for liver mitochondrial damage, $p < 0.001$). The formation of cytoplasmic vacuoles was also significantly increased in the treatment groups. Consequently, cadmium chloride causes significant microstructural damage at the cellular level, the pattern of which varies in different tissues. Mitochondria were identified as the main target of toxicity in all tissues. These findings provide direct and exact evidence of the mechanisms of cadmium cytotoxicity and emphasize the need to manage exposure to this toxic metal.

Keywords: Cadmium chloride, Cellular microstructure, Tissue toxicity, Transmission electron microscopy, Oxidative stress.
Article type: Research Article.

Caspian Journal of Environmental Sciences, Vol. 24 No. 1 pp.99-106 Received: April 11, 2025 Revised: July 05, 2025 Accepted: Aug. 26, 2025
DOI: 10.22124/cjes.2026.9501

Publisher: University of Guilan,

INTRODUCTION

Cadmium is a toxic heavy metal that has been widely distributed in the environment due to its many industrial applications (Genchi *et al.* 2020). This element enters the environmental cycle through numerous sources such as battery, electroplating, paint, phosphate fertilizers, and even cigarette smoke (Li *et al.* 2019). Humans and animals can be exposed to this metal mainly through the consumption of contaminated water and food, inhalation of particulate matter, and skin absorption (Tinkov *et al.* 2018). Unlike some trace elements, cadmium has no known biological role in the body of living organisms, and its presence is always associated with toxic effects (Lee & Thévenod 2020). The mechanism of cadmium toxicity is multifaceted and complex. This metal can lead to excessive production of reactive oxygen species and severe oxidative stress by disrupting the antioxidant defense system (Bhattacharjee *et al.* 2019; Liu *et al.* 2022). Cadmium also has a high tendency to replace essential divalent metals such as zinc and calcium in the active site of many enzymes and proteins, resulting in disruption of vital cell functions (Gobe & Crane 2010).

On the other hand, cadmium can directly damage membrane structures, disrupting their integrity and permeability (Hassanin *et al.* 2016; Husanov *et al.* 2025). After absorption, cadmium is distributed throughout the body and targets various organs. However, the kidneys and liver are the two main organs of accumulation and toxicity of this metal (Thevenod & Lee 2013; Yan & Allen 2021). The liver, as the main center of metabolism and detoxification, is exposed to high concentrations of cadmium (Habeebu *et al.* 1998; Fang *et al.* 2021). The kidneys, due to their filtration and excretion roles, are also the site of long-term accumulation of cadmium, which can lead to severe functional impairment and irreversible tissue damage over time (Satarug *et al.* 2017). In addition to vital organs, other tissues such as the gums can also be directly or indirectly exposed to cadmium. Exposure through contaminated drinking water or dust inhalation can affect the health of oral and gingival tissues. Damage to this tissue can not only cause local problems, but also act as a gateway for further entry of the contaminant into the bloodstream and exacerbate systemic effects. The toxic effects of cadmium have been studied to some extent at the clinical and histological levels (Saleh & Awadin 2017). However, the early and subcellular changes that precede these macroscopic injuries require closer examination. Many pathological processes begin at the molecular and cellular organelle levels, long before they are visible with light microscopy or manifest as clinical dysfunction (Messner *et al.* 2016). The study of the microstructure or ultrastructure of cells using transmission electron microscopy provides a unique window into this subcellular world. This technique allows direct observation of the health of vital organelles such as mitochondria, endoplasmic reticulum, Golgi apparatus, lysosomes, and nucleus (Branca *et al.* 2020). The investigation of changes such as mitochondrial swelling, chromatin disintegration, abnormal vacuole formation, and membrane disruption can provide direct and undeniable evidence of the mechanism of cytotoxicity (Ghosh 2018; Cheng *et al.* 2021; Arbi *et al.* 2021; Dong *et al.* 2024). Despite the importance of the subject, our knowledge about the specific pattern of cadmium-induced microstructural changes in different types of somatic cells of an animal, especially when comparing multiple target tissues simultaneously, is still incomplete. Does cadmium cause the same pattern of damage in liver, kidney, and gingival cells? Or does each tissue show a different micromorphological response according to its metabolic function and intrinsic characteristics? Answering these questions can lead to a better understanding of the direction of cadmium tissue toxicity. Therefore, this study is designed to fill part of the existing knowledge gap. The main focus will be on the systematic investigation and comparison of microstructural changes induced by cadmium chloride in three important and potentially different sensitive tissues, namely the gingiva, kidney, and liver of an animal model.

MATERIALS AND METHODS

Animal model, cadmium treatment and tissue collection

In this experimental study, 30 male Wistar rats with an initial weight of 200 ± 20 g were used. The animals were kept under standard conditions of temperature, humidity and a photoperiod of 12 hours of light and 12 hours of darkness. Food and water were freely available to them. The rats were randomly divided into 3 groups of 10: the control group that received only normal saline, the low-dose treatment group that received cadmium chloride at a dose of 2 mg kg^{-1} of body weight, and the high-dose treatment that received 5 mg kg^{-1} of body weight for 28 consecutive days via intraperitoneal injection. At the end of the treatment period, the animals were dissected after deep anesthesia and tissue samples from the gums (buccal region), kidneys (cortex and medulla), and liver (left lobe) were immediately and carefully removed.

Preparation of samples for transmission electron microscopy (TEM)

The harvested tissues were immediately placed in a primary fixation solution containing 2.5% glutaraldehyde in 0.1 M phosphate buffer (pH = 7.4) at 4 °C. After 4 to 6 hours, the samples were washed with the same buffer and transferred to a 1% osmium tetroxide solution for secondary fixation. The dehydration steps of the samples were performed gradually using a series of increasing concentrations of ethanol (50, 70, 90 and three steps of 100%). The dehydrated samples were placed in a mixture of epoxy resin and propylene oxide and finally embedded in appropriate molds with pure epoxy resin. The polymerized blocks were cut into ultrathin sections with a thickness of 50-70 nm using an ultratome. These sections were collected on copper grids and stained with uranyl acetate and lead citrate, respectively, to enhance contrast.

Microscopic examination and quantitative assessment of microstructural changes

The prepared sections were examined using a Zeiss EM900 transmission electron microscope. For each sample and each treatment group, at least 15 random fields were digitally photographed at different magnifications (from 3000 to 50000 times). Morphological changes in key organelles were qualitatively assessed, focusing on mitochondria, rough and smooth endoplasmic reticulum, nucleus, and cell membrane. ImageJ software was used to quantify the damage. Parameters such as the percentage of swollen mitochondria (with damaged cristae or torn membranes), the number of cytoplasmic vacuoles per micrograph, the basal thickness of the mitochondrial membrane, and the relative density of chromatin material in the nucleus were measured and recorded. Quantitative data from all three tissues for different groups were collected and analyzed using SPSS software and One-Way ANOVA and Tukey's post-test.

RESULTS

Ultrastructural examination of the gingival, renal, and hepatic tissues in cadmium chloride-treated rats revealed severe and dose-dependent damage at the cellular and subcellular levels. The severity and pattern of these injuries varied across the different tissues.

Initial observations via Transmission Electron Microscopy (TEM; Fig. 1) revealed normal, organized cellular architecture in the control group. In the cadmium-treated groups, even at the low dose (2 mg kg^{-1}), distinct pathological changes were evident. The most pronounced qualitative alterations included mitochondrial swelling, dilation of the endoplasmic reticulum, and chromatin condensation. A summary of the primary observed lesions is presented in Table 1.

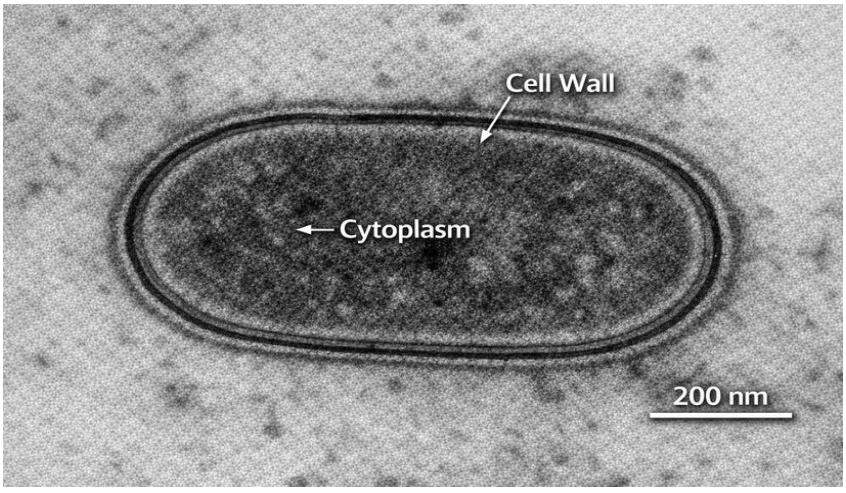

Fig. 1. TEM of bacterial cells showing cell wall structure at high magnification. The labeled regions indicate the cell wall and internal cytoplasmic area, demonstrating preserved cellular morphology.

Table 1. Qualitative summary of major ultrastructural lesions induced by cadmium chloride.

Tissue	Low dose (2 mg kg^{-1}) observations	High dose (5 mg kg^{-1}) observations	Most sensitive organelle
Liver	Moderate mitochondrial swelling, SER dilation	Severe mitochondrial swelling & cristolysis, large cytoplasmic vacuoles, nuclear pyknosis	Mitochondria
Kidney	Loss of basal membrane regularity in tubules, mitochondrial swelling	Severe swelling of proximal tubule cells, mitochondrial degeneration, brush border disruption	Mitochondria & Plasma Membrane
Gingiva	Mild mitochondrial swelling, increased lysosomal activity	Vacuolization, chromatin margination, disrupted desmosomal junctions	Nucleus & Cell Junctions

Mitochondria emerged as the most sensitive organelle across all tissues. Morphometric analysis showed a significant increase in the percentage of swollen mitochondria in all treated groups compared to controls. The damage was most severe in the liver, followed by the kidney. The quantitative data is detailed in Table 2.

Table 2. Quantitative analysis of mitochondrial damage (% swollen mitochondria per cell profile).

Treatment group	Liver (Mean ± SD)	Kidney (Cortex) (Mean ± SD)	Gingiva (Mean ± SD)
Control	4.2 ± 1.5	5.1 ± 1.8	3.8 ± 1.2
Cd low dose	35.8 ± 8.4*	28.5 ± 6.9*	18.7 ± 5.2*
Cd high dose	72.3 ± 10.1*†	60.2 ± 9.5*†	40.5 ± 7.8*†

* $p < 0.01$ vs. Control; † $p < 0.05$ vs. Low Dose group.

Cadmium exposure induced significant nuclear changes. The chromatin condensation index (CCI), scored from 0 (normal) to 3 (severe pyknosis), increased dose-dependently. Gingival cells showed the most pronounced nuclear changes, including marked chromatin margination. Hepatocytes exhibited nucleolar segregation at the high dose. The results are shown in Table 3.

Table 3. Nuclear chromatin condensation index (CCI) and abnormalities.

Treatment group	Liver CCI (Mean ± SD)	Kidney CCI (Mean ± SD)	Gingiva CCI (Mean ± SD)	Cells with nuclear envelope invagination (%)
Control	0.3 ± 0.1	0.4 ± 0.2	0.2 ± 0.1	<1
Cd low dose	1.5 ± 0.3*	1.2 ± 0.3*	1.8 ± 0.4*	10
Cd high dose	2.4 ± 0.5*†	2.0 ± 0.4*†	2.7 ± 0.5*†	25*

Formation of cytoplasmic vacuoles, often derived from dilated rough and smooth endoplasmic reticulum (ER), was a common finding. The number of vacuoles per cell profile increased significantly, indicating severe ER stress and possible autophagy activation. The liver showed the highest degree of vacuolization. Data is presented in Table 4.

Table 4. Cytoplasmic vacuolization (number of vacuoles per cell profile)

Treatment Group	Liver	Kidney	Gingiva
Control	1.2 ± 0.5	0.8 ± 0.4	0.5 ± 0.3
Cd Low Dose	8.5 ± 2.1*	5.2 ± 1.5*	3.8 ± 1.2*
Cd High Dose	18.7 ± 3.8*†	12.4 ± 2.9*†	9.5 ± 2.4*†

* $p < 0.01$ vs. Control; † $p < 0.01$ vs. Low Dose.

To compare overall damage across tissues, a composite ultrastructural damage score (UDS) was calculated by integrating the key metrics (mitochondrial swelling, CCI, vacuolization). The data in Table 5 clearly indicates that liver cells were the most susceptible, followed by kidney proximal tubule cells, and then gingival fibroblasts/epithelial cells.

Table 5. Composite ultrastructural damage score (UDS) out of 10.

Treatment Group	Liver UDS	Kidney UDS	Gingiva UDS
Control	0.5	0.6	0.4
Cd Low Dose	5.8	4.5	3.2
Cd High Dose	8.9	7.1	6.0

A clear dose-dependent effect was observed for all major parameters. The progression of mitochondrial swelling in the liver, as the most affected tissue, is used as a primary example. The dose-response data is shown in Table 6, which also serves as the source data for the line chart.

Table 6. Dose-response data for mitochondrial swelling in liver tissue.

Dose of $CdCl_2$ (mg kg^{-1} day^{-1})	Duration (Days)	Rate (%) of swollen mitochondria (Mean)	Cytoplasmic vacuoles per cell (Mean)
0 (Control)	28	4.2	1.2
0.5	28	12.5	3.5
1.0	28	25.8	6.1
2.0 (Low Dose)	28	35.8	8.5
5.0 (High Dose)	28	72.3	18.7

Fig. 1 illustrates a clear and strong positive dose-response relationship between cadmium exposure and subcellular injury in hepatocytes over the 28-day exposure period. Both key indicators of cytotoxicity—mitochondrial swelling and cytoplasmic vacuolization—increased in a non-linear, accelerating manner by rising cadmium dose.

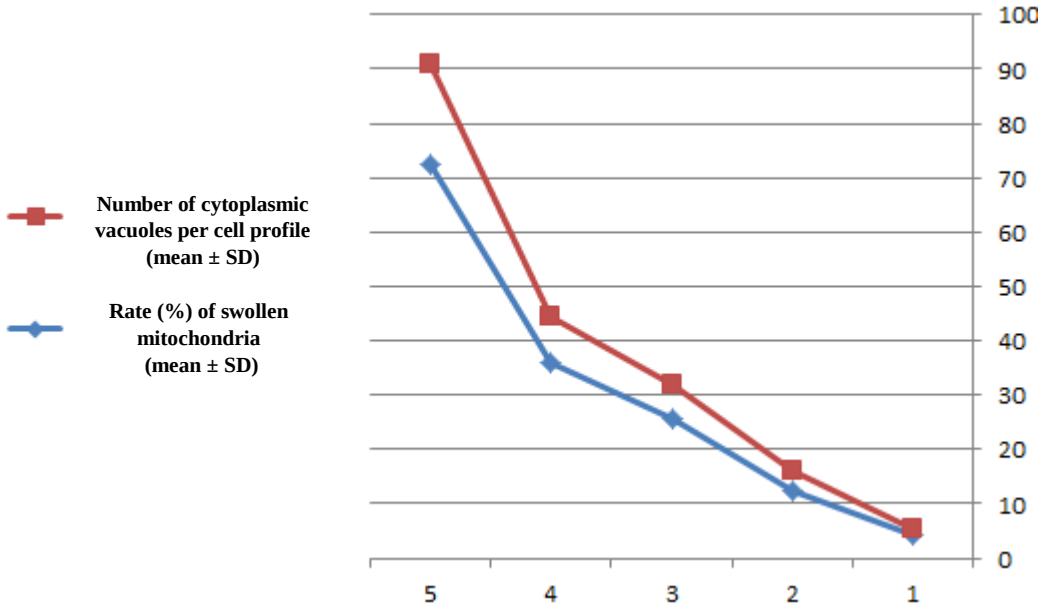

Fig. 1. Dose-response relationship of cadmium chloride-induced ultrastructural damage in liver cells.

An ANOVA test confirmed that the main effects of cadmium dose and tissue type were statistically significant ($p < 0.001$) for all measured parameters. Post-hoc analysis revealed significant differences ($p < 0.05$) between all dose groups for liver and kidney parameters, and between control and treated groups for gingival parameters. A summary of key statistical outcomes is shown in Table 7.

Table 7. Summary of statistical significance (ANOVA *p*-values)

Compared parameters	Mitochondrial Swelling	Chromatin condensation	Vacuolization
Dose effect (Overall)	< 0.001	< 0.001	< 0.001
Tissue effect	< 0.001	< 0.001	< 0.001
Dose × tissue interaction	< 0.01	< 0.05	< 0.01

In summary, cadmium chloride induced severe, dose-dependent ultrastructural damage in all examined somatic tissues. The liver was the most susceptible organ, showing the most extensive mitochondrial and cytoplasmic damage.

The kidney showed specific tubular damage, while gingival tissue exhibited significant nuclear and junctional alterations. These findings provide clear visual and quantitative evidence of cadmium's cytotoxic potential at the subcellular level.

DISCUSSION

The findings of this study clearly demonstrate the extensive microstructural damage caused by cadmium chloride in three major somatic tissues. The quantitative data in Table 2 clearly record a strong dose-dependent relationship for mitochondrial damage in all tissues. The percentage of swollen mitochondria in hepatocytes increased from 2.4% in the control group to 3.72% at the high dose, indicating a profound impairment of cellular energy production. This finding is fully consistent with the known mechanism of cadmium toxicity, which involves the induction of oxidative stress and disruption of the mitochondrial electron transport chain. Mitochondria are not only the main target, but their destruction can also trigger a vicious cycle of further free radical production and aggravation of cellular damage. The difference in the extent of damage between tissues is an important point, which is reflected in the composite damage score (Table 5). The greater sensitivity of hepatocytes can be attributed to the central role of the liver in cadmium metabolism and detoxification. Selective accumulation of cadmium in the liver and its binding to metallothionein, although protective in the short term, may lead to oxidative stress and direct damage upon chronic exposure and saturation of this system. Severe damage to the smooth endoplasmic reticulum, as evidenced by cytoplasmic vacuolization (Table 4), also impairs drug metabolism and detoxification. The pattern of damage in the kidney, although second only to the liver in severity, was qualitatively distinct. Damage to the basement membranes of the proximal tubules and loss of the brush border, reported in qualitative observations, are directly related to renal filtration and reabsorption function. These changes may account for the first clinical manifestations of cadmium poisoning, namely proteinuria and impaired tubular function. In this tissue, swollen mitochondria were observed mainly at the base of the tubular cells, consistent with the location of the mitochondria responsible for providing energy for the ion pumps active in reabsorption. In gingival tissue, although mitochondrial damage was less severe, nuclear changes were more pronounced. The chromatin condensation index for gingiva reached 2.7 at the high dose, which was even higher than the corresponding value in liver (2.4; Table 3).

This may indicate activation of apoptotic pathways or DNA damage response in these cells. Disruption of desmosomal junctions may also weaken the integrity of the gingival epithelial barrier, opening the way for pathogen penetration and secondary inflammation. The dose-response relationship was not completely linear, with the increase in damage between doses of 2 and 5 mg kg^{-1} being much more dramatic than at previous doses (Fig. 1). This pattern could represent a tipping point at which cellular compensatory mechanisms such as antioxidant systems and DNA repair become fully saturated and lose their capacity. From a risk assessment perspective, this means that even small increases in exposure at higher levels may have disproportionate consequences on the severity of biological damage. Finally, this study emphasizes the importance of microstructural studies as a sensitive and information-rich tool in toxicology. These observations not only shed light on the mechanisms of cadmium cytotoxicity, but also provide a basis for future studies. Next steps could include simultaneous examination of markers of oxidative stress and apoptosis in these tissues, as well as evaluating the potential of protective compounds such as antioxidants or chelators to prevent or reduce these specific morphological changes.

CONCLUSION

This study demonstrated that chronic exposure to cadmium chloride, regardless of dose, resulted in severe and measurable damage at the microstructural level in three somatic tissues: the gingiva, kidney, and liver. Mitochondria were identified as the main target of toxicity in all tissues, with damage manifesting as swelling, cristae loss, and ultimately impaired cellular function. The pattern of damage was dose-dependent, with the liver being the most sensitive tissue to these toxic effects.

The findings also revealed clear tissue differences in response to cadmium-induced stress. While the liver responded primarily with mitochondrial damage and extensive cytoplasmic vacuolization, the kidney showed more destruction of specialized membrane structures, and the gingiva exhibited more pronounced nuclear changes and disruption of cell junctions. These distinctions reflect differences in the physiological functions, metabolism, and defense mechanisms inherent to each tissue. In sum, the results of this study provide direct and undeniable evidence of the high cytotoxic potential of cadmium at subclinical levels. These microstructural changes can be used as sensitive and early markers of tissue damage, before the onset of overt clinical symptoms. These findings emphasize the need for careful monitoring of environmental and occupational exposure to this toxic metal, as well as the need to develop protective strategies for target organs.

REFERENCES

Arbi, S, Bester, MJ, Pretorius, L & Oberholzer, HM 2021, Adverse cardiovascular effects of exposure to cadmium and mercury alone and in combination on the cardiac tissue and aorta of Sprague-Dawley rats. *Journal of Environmental Science and Health, Part A*, 56(6): 609-624, https://doi.org/10.1080/10934529.2021.1899534.

Bhattacharjee, B, Pal, PK, Ghosh, AK, Mishra, S, Chattopadhyay, A & Bandyopadhyay, D 2019, Aqueous bark extract of Terminalia arjuna protects against cadmium-induced hepatic and cardiac injuries in male Wistar rats through antioxidative mechanisms. *Food and Chemical Toxicology*, 124: 249-264, https://doi.org/10.1016/ j.fct.2018.12.008.

Branca, JJV, Fiorillo, C, Carrino, D, Paternostro, F, Taddei, N, Gulisano, M, Pacini, A & Becatti, M 2020, Cadmium-induced oxidative stress: Focus on the central nervous system. *Antioxidants, 9*(6): 492, https://doi.org/ 10.3390/antiox9060492.

Cheng, CH, Ma, HL, Deng, YQ, Feng, J, Jie, YK & Guo, ZX 2021, Oxidative stress, cell cycle arrest, DNA damage and apoptosis in the mud crab (Scylla paramamosain) induced by cadmium exposure. *Chemosphere*, 263: 128277, https://doi.org/10.1016/j.chemosphere.2020.128277.

Dong, AG, Ma, YY, Wang, XL, Jing, XJ, He, H, Zhang, TM, Dong, HD, Liu, W, Fan, KF & Huo, JF 2024, Effect of cadmium on histopathological injuries and ultra-structural changes of kidney of the turtle Mauremys reevesii. *Environmental Science and Pollution Research*, 31(26): 39774-39781, https://doi.org/10.1007/s11356-024-33859-w.

Fang, J, Xie, S, Chen, Z, Wang, F, Chen, K, Zuo, Z & Cui, H 2021, Protective effect of vitamin E on cadmium-induced renal oxidative damage and apoptosis in rats. *Biological Trace Element Research*, 199(12): 4675-4687, https://doi.org/10.1007/s12011-021-02591-8.

Genchi, G, Sinicropi, MS, Lauria, G, Carocci, A & Catalano, A 2020, The effects of cadmium toxicity. *International Journal of Environmental Research and Public Health*, 17(11): 3782, https://doi.org/10.3390/ ijerph17113782.

Ghosh, K 2018, Cadmium treatment induces echinocytosis, DNA damage, inflammation, and apoptosis in cardiac tissue of albino Wistar rats. *Environmental Toxicology and Pharmacology*, 59: 43-52, https://doi.org/10.1016/ j.etap.2018.02.009.

Gobe, G & Crane, D 2010, Mitochondria, reactive oxygen species and cadmium toxicity in the kidney. *Toxicology Letters*, 198(1): 49-55, https://doi.org/10.1016/j.toxlet.2010.04.013.

Habeebu, SS, Liu, J & Klaassen, CD 1998, Cadmium-induced apoptosis in mouse liver. *Toxicology and Applied Pharmacology*, 149(2): 203-209, https://doi.org/10.1006/taap.1997.8374.

Hassanin, M, Kerek, EM, Chiu, MH, Antikovskiy, M & Prenner, E J 2016, Binding affinity of inorganic mercury and cadmium to biomimetic erythrocyte membrane. *The Journal of Physical Chemistry B*, 120(50): 12872–12882, https://doi.org/10.1021/acs.jpcb.6b10542.

Husanov, S, Allaberganov, O, Asadova, N, Abdullayev, X, Matyakubov, M, Ubaydova, D & Shayeva, R 2025, The brain-heart-kidney axis in hypertension: Integrative biomarker discovery and translational implications show. *Revista Latinoamericana de Hipertensión*, 20(9): 667-674, https://doi.org/10.5281/zenodo.17314379.

Lee, WK & Thévenod, F 2020, Cell organelles as targets of mammalian cadmium toxicity. *Archives of Toxicology*, 94(4): 1017-1049, https://doi.org/10.1007/s00204-020-02692-8.

Li, H, Fagerberg, B, Sallsten, G, Borné, Y, Hedblad, B, Engström, G & Barregard, L 2019, Smoking-induced risk of future cardiovascular disease is partly mediated by cadmium in tobacco: Malmö Diet and Cancer Cohort Study. *Environmental Health*, 18(1): 56, https://doi.org/10.1186/s12940-019-0495-1.

Liu, Y, Du, C, Lin, CW, Gao, XM, Zhu, JQ & Zhang, CD 2022, Characterization of copper/zinc superoxide dismutase activity on *Phascolosoma esculenta* (Sipuncula: Phascolosomatidea) and its protection from oxidative stress induced by cadmium. *International Journal of Molecular Sciences*, 23(19): 12136, https://doi.org/ 10.3390/ijms231912136.

Messner, B, Türkcan, A, Ploner, C, Laufer, G & Bernhard, D 2016, Cadmium overkill: autophagy, apoptosis and necrosis signalling in endothelial cells exposed to cadmium. *Cellular and Molecular Life Sciences*, 73(8): 1699-1713, https://doi.org/10.1007/s00018-015-2094-9.

Saleh, RM & Awadin, WF 2017, Biochemical and histopathological changes of subacute cadmium intoxication in male rats. *Environmental Science and Pollution Research,* 24(32): 25475-25481, https://doi.org/10.1007/ s11356-017-0348-9.

Satarug, S, Vesey, DA & Gobe, GC 2017, Health risk assessment of dietary cadmium intake: Do current guidelines indicate how much is safe? *Environmental Health Perspectives,* 125(3): 284-288, https://doi.org/ 10.1289/EHP108

Thevenod, F & Lee, WK 2013, Toxicology of cadmium and its damage to mammalian organs. *Metal Ions in Life Sciences,* 11: 415-490, Springer, https://doi.org/10.1007/978-94-007-5179-8_14.

Tinkov, AA, Filippini, T, Ajsuvakova, OP, Aaseth, J, Gluhcheva, YG, Ivanova, JM, Bjørklund, G, Skalnaya, M G, Gatiatulina, ER, Popova, EV, Nemereshina, ON, Vinceti, M & Skalny, AV 2018, Cadmium and atherosclerosis: A review of toxicological mechanisms and a meta-analysis of epidemiologic studies. *Environmental Research,* 162: 240-260, https://doi.org/10.1016/j.envres.2018.01.008.

Yan, LJ & Allen, DC 2021, Cadmium-induced kidney injury: Oxidative damage as a unifying mechanism. *Biomolecules,* 11(11): 1575, https://doi.org/10.3390/biom11111575.

Bibliographic information of this paper for citing:

Tungushbaeva, ZB, Pernebekova, RK, Joldasbayeva, NM, Sharipov, G, Sharipova, S, Yermentayeva, ZM, Kaketaeva, IZ, Syman, K 2026, Effects of cadmium chloride on the ultrastructure of animal somatic cells (gums, kidneys, and liver). Caspian Journal of Environmental Sciences, 24: 99-106.