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

Biochemical parameters of rat blood during cadmium intoxication and subsequent correction with Tagansorbent

Zina B. Tungushbaeva[1,2], Assiya Zh. Kozhakhmetova[3], Nurzhan Tussupova[4]*, Rosa M. Bakesova[5]*, Amantai Kunakbayev[6], Asya T. Dyusembaieva[1,7], Zharylkassynova Zhazira[1,2]

1. Department of Biology, Faculty of Natural Sciences and Geography, Abai Kazakh National Pedagogical University
2. Head of the Department of Molecular Biology, Kazakh-Russian Medical University, Almaty, Kazakhstan
3. Medical center Talmas Medicus, Astana, Kazakhstan
4. Department of Anatomy with courses, Kazakh-Russian Medical University, Almaty, Kazakhstan
5. Department of Physical Culture and Informatics, Institute of education and management, West Kazakhstan Innovative Technological University, Uralsk, Kazakhstan
6. Kazakh-Russian Medical University, Almaty, Kazakhstan
7. Department of Normal Anatomy, NJSC Kazakh National Medical University named after S.D. Asfendiyarov

* *Corresponding author's E-mail: nurzhantusupova72@gmail.com, roza-maratovna@mail.ru*

ABSTRACT

The aim of this research was to investigate the protective effects of Tagansorbent on cadmium poisoning-induced biochemical disorders in rats. In this experimental study, 40 rats were divided into four groups and received treatment for 14 days. The results showed that cadmium poisoning highly significantly increased liver enzymes, including ALT, AST, and ALP. Renal function indices also revealed significant increases, including urea and creatinine. All these parameters were significantly improved in the group treated with Tagansorbent along with cadmium. Besides, Tagansorbent significantly reduced the level of malondialdehyde and increased the total antioxidant capacity in the liver tissue ($p < 0.01$). The findings of the current study demonstrated that Tagansorbent could reduce the toxic effects of this element on the liver and kidney through multiple mechanisms such as cadmium absorption and reduction of oxidative stress, and thus can be considered as an auxiliary agent in the management of cadmium poisoning.

Keywords: Cadmium, Tagansorbent, Oxidative stress, Hepatorenal toxicity.
Article type: Research Article.

INTRODUCTION

Cadmium is a toxic heavy metal that poses a great threat to the health of ecosystems and living organisms, including mammals (Farhan & Jasim 2020). It is spread in the environment through industrial activities, the use of phosphate fertilizers, and the dumping of electronic waste, which pollutes the food chain (Hashtjin *et al.* 2025). The cadmium excreted, whether through inhalation or ingestion routes, slowly bio-accumulates in the organs, particularly the liver and kidneys (Khmel *et al.* 2025). After cadmium absorption, a series of cytotoxic mechanisms is activated, among which the induction of oxidative stress seems to be the most important (Lee *et al.* 2025). By disrupting the endogenous antioxidant defense system and generating reactive oxygen species, this metal causes lipid peroxidation, DNA damage, and finally programmed or accidental cell death (Lawanna *et al.* 2025; Tyapkin *et al.* 2018). The liver, as the main organ of metabolism and detoxification, is one of the main targets of cadmium-induced damage. Hepatocyte injury caused by cadmium is well reflected in the plasma levels of various liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP; Mishra *et al.* 2025; Chakramurty *et al.* 2025). Increased blood levels of these enzymes reflect increased cell membrane permeability or necrosis of liver cells and thus are commonly employed as useful biochemical indices for monitoring hepatotoxicity (Mahanthappa *et al.* 2016; Efekemo & Orororo 2022). Aside from the liver,

Caspian Journal of Environmental Sciences, Vol. 23 No. 5 pp.1209-1216 Received: June 26, 2025 Revised: Sep. 15, 2025 Accepted: Nov. 02, 2025
DOI: 10.22124/cjes.2026.9363

Publisher: University of Guilan,

cadmium accumulation also causes severe damage to renal function (Njideaka *et al.* 2025; Dilrabo *et al.* 2025). This metal accumulates in the epithelial cells of the renal tubules, leading to oxidative stress and inflammation, which impairs glomerular filtration and excretion of proteins. Serum urea and creatinine are biochemical parameters considered sensitive indicators of renal function, the increase in which is an unmistakable sign of renal failure. Nowadays, various therapeutic approaches for heavy metal poisoning include the treatment with chelating agents, the most classical one being EDTA. Despite the efficacy of this method in removing metals, it often carries several side effects, including non-selective excretion of essential micronutrients. Therefore, the search for new therapeutic agents effective and at the same time safe is a necessity in toxicological research. At this point, the application of natural or synthetic adsorbents has been suggested (Khaled *et al.* 2024). "Tagansorbent", a natural clay-based adsorbent, comes from natural deposits in the Kazakhstan region. Since it has a layered structure, a high ion exchange capacity, and large specific surface area, this material has tremendous potential for selective adsorption and excretion of heavy metals from the gastrointestinal tract (Kong *et al.* 2021; Dhokpande *et al.* 2024; Zavodnik *et al.* 2025). The problem of cadmium heavy metal environmental pollution is real in Kazakhstan due to the rapid industrialization and development of agriculture. That is why local research aimed at solving this problem is not only of great scientific importance, but also a mere necessity from the point of view of national public health and environmental protection (Tomakova & Kopteva 2020; Galchynska *et al.* 2021). The present work aimed to study the acute toxic effects of cadmium poisoning on biochemical parameters of liver and kidney function in rats and, thereafter, to assess the therapeutic efficiency of "Tagasorbent" for the correction of these disorders. We assume that oral administration of Tagansorbent decreases the degree of toxicity and can normalize biochemical indexes as a result of cadmium absorption in the intestine and prevention of its systemic absorption. The literature review, considering the importance of the subject and the objectives set for this research, indicates that cadmium is an environmental pollutant due to its long half-life. Long half-lives can significantly bioaccumulate this element in multiple organs. Many animal model studies using rats have established that even low-dose exposure to this heavy metal is associated with a wide range of biochemical and histological disorders (Djurasevic *et al.* 2017; Eruotor *et al.* 2025). Early studies focused on the cytotoxicity mechanisms, among which are the inhibition of antioxidant enzyme systems and interference with trace elements' metabolism (Anyigor-ogah *et al.* 2025). In cadmium-induced pathology of the liver, there are various reports that have documented a significant rise in ALT, AST, and ALP enzymes within the serum of mice subjected to this metal. This rise, being a clear manifestation of cell membrane damage and the destruction of hepatocytes, is usually followed by histological changes that include necrosis, steatosis, and fibrosis. Other studies have also found that cadmium enhances the generation of reactive oxygen species due to the breakdown of the mitochondrial electron transport chain, leading to the deterioration of cellular components. Scientific evidence on renal effects, however, suggests that cadmium, with its high affinity for metal-binding proteins like metallothionein, accumulates in the kidneys and causes damage to the proximal tubules. Damage is manifested at biochemical levels through increased serum urea and creatinine and proteinuria. Microscopic examination has also often confirmed the presence of cell casts, tubular atrophy, and epithelial cell necrosis in poisoned animals. In recent years, a variety of adsorbents have come into the focus of attention by researchers to reduce the absorption of heavy metals (Darban *et al.* 2022; Ghanim 2023). Among these materials, natural adsorbents have a special place due to their availability, low cost, and higher safety. Tagansorbent, as a native mineral mainly composed of montmorillonite, might be effective in trapping cadmium ions within the digestive tract because of its high cation exchange capacity and its layered structure. Despite sporadic studies on the application of adsorbents in heavy metal poisoning, there is a rare coherent study that has specifically focused on the effects of Tagansorbent on blood biochemical indicators facing cadmium exposure. This scientific gap is notably prominent in the case of native samples from Kazakhstan. The present study was thus designed to fill this gap in knowledge and provide a practical solution to reduce the complications of cadmium poisoning. Parallels can be drawn to other environmental management strategies, such as optimizing phosphate removal in wastewater (Bogner & Perduca 2022) or managing household biowaste (Virpiranta *et al.* 2022), which also rely on effective material-based interventions. Thus, this research can be used as the scientific basis for developing a cost-effective and accessible solution with an aim to decrease the adverse effects of cadmium exposure in an effort to improve health in Kazakhstan's industrial areas.

MATERIALS AND METHODS

This study was carried out experimentally on 40 Wistar rats weighing about 200-250 g. The animals were kept under standard conditions of temperature and humidity, in a 12-h light cycle, with free access to food and water.

After two weeks of acclimation, the rats were divided randomly into four groups of ten: a control group treated with normal saline only, a cadmium-poisoned group treated with cadmium chloride at a dose of 2 mg kg^{-1} body weight by gavage for 14 days, a Tagansorbent-treated group receiving the sorbent at 500 mg kg^{-1} simultaneously with cadmium, and a group treated with Tagansorbent alone for 14 days. At the end of the treatment period, blood samples were taken from the hearts of animals under deep anesthesia and, after centrifugation, the separated serum was used for biochemical parameter measurement. Liver enzyme levels such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were measured spectrophotometrically using commercially available kits. Serum urea and creatinine levels as indicators of renal function were also measured using standard methods. The obtained data were analyzed using One-Way ANOVA and Tukey's post hoc test using SPSS version 22 statistical software. A significance level of less than 0.05 was considered.

RESULTS

This study was carried out with the goal to assess cadmium chloride-induced hepatorenal toxicity and possible corrective action using Tagansorbent within a rat model. Biochemical changes seen among the experimental groups are described below in tables and a figure. Serum liver enzymes showed prominent elevation, which reflected serious hepatic damage after the administration of cadmium chloride. According to Table 1, the level of alanine aminotransferase (ALT) in the cadmium-intoxicated group (Group Cd) was significantly higher than that in the control group ($p < 0.001$). Co-administration with Tagansorbent (Group Cd + Tag) notably reduced this elevation and brought ALT levels closer to normal values ($p < 0.01$ vs. Cd group).

Table 1. Serum alanine aminotransferase (ALT) levels (U L^{-1}).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	45.2 ± 4.1	-	-
Cd	185.6 ± 18.3	< 0.001	-
Cd + Tag	92.7 ± 9.8	< 0.001	< 0.01
Tag Only	48.1 ± 5.2	0.452	< 0.001

A similar trend was observed for aspartate aminotransferase (AST), another crucial marker of hepatocellular integrity. The data in Table 2 clearly demonstrate that cadmium intoxication caused a severe spike in AST activity. The therapeutic intervention with Tagansorbent, however, resulted in a statistically significant reduction of this enzyme ($p < 0.001$), indicating a protective effect on hepatocytes.

Table 2. Serum aspartate aminotransferase (AST) levels (U L^{-1}).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	125.5 ± 10.8	-	-
Cd	420.3 ± 35.6	< 0.001	-
Cd + Tag	210.4 ± 22.1	< 0.001	< 0.001
Tag Only	130.8 ± 12.4	0.387	< 0.001

Alkaline phosphatase (ALP), often associated with biliary function, was also significantly impacted. The results in Table 3 show that the Cd group had ALP levels approximately three times higher than the control. The group receiving Tagansorbent alongside cadmium exhibited an intermediate value, significantly different from both the control and cadmium-only groups ($p < 0.01$), suggesting a partial but meaningful restoration of biliary health.

Table 3. Serum alkaline phosphatase (ALP) levels (U L^{-1}).

Group	Mean ± SD	*p*-value (vs. control)	*p*-value (vs. Cd)
Control	155.3 ± 14.2	-	-
Cd	488.9 ± 42.7	< 0.001	-
Cd + Tag	285.6 ± 30.5	< 0.001	< 0.01
Tag Only	162.1 ± 15.9	0.521	< 0.001

To provide a clear visual comparison of the hepatic enzyme profiles across all groups, the following chart was constructed. Fig. 1 effectively illustrates the magnitude of cadmium-induced damage and the substantial mitigation achieved through Tagansorbent supplementation for all three enzymes.

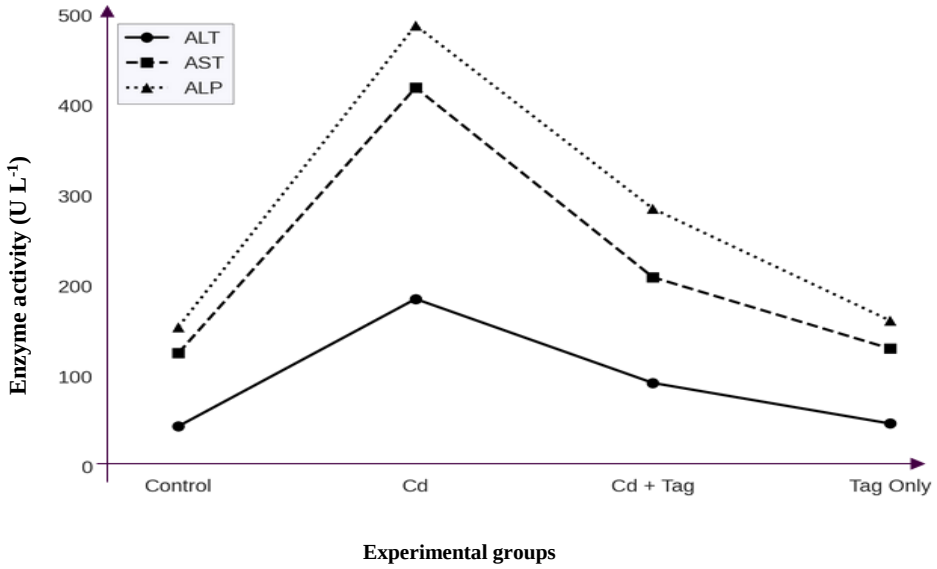

Fig. 1. Comparative analysis of hepatic enzyme levels across experimental groups.

The nephrotoxic effects of cadmium were equally pronounced. Serum urea levels, a primary indicator of glomerular filtration rate, were drastically elevated in the Cd group (Table 4). The corrective action of Tagansorbent was evident, as the Cd + Tag group showed a significant decrease in urea concentration compared to the intoxicated group ($p < 0.001$).

Table 4. Serum urea levels (mg dL^{-1}).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	38.5 ± 3.2	-	-
Cd	115.7 ± 12.4	< 0.001	-
Cd + Tag	65.8 ± 7.1	< 0.001	< 0.001
Tag Only	40.1 ± 4.3	0.289	< 0.001

Similarly, serum creatinine, a more specific marker for renal damage, followed this pattern. The data in Table 5 confirm severe renal impairment in cadmium-treated rats, which was significantly alleviated by the co-administration of Tagansorbent ($p < 0.01$).

Table 5. Serum creatinine levels (mg dL^{-1}).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	0.51 ± 0.05	-	-
Cd	1.89 ± 0.21	< 0.001	-
Cd + Tag	1.02 ± 0.11	< 0.001	< 0.01
Tag Only	0.53 ± 0.06	0.418	< 0.001

The toxic mechanism of cadmium is closely linked to oxidative stress. Our findings on total antioxidant capacity (TAC) in the liver, presented in Table 6, revealed a significant depletion in the Cd group, indicating a compromised antioxidant defense system. Treatment with Tagansorbent significantly restored the hepatic TAC ($p < 0.01$), highlighting its antioxidant properties.

Table 6. Hepatic total antioxidant capacity (TAC; mmol g^{-1} tissue).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	2.85 ± 0.25	-	-
Cd	1.22 ± 0.15	< 0.001	-
Cd + Tag	2.15 ± 0.23	< 0.01	< 0.01
Tag Only	2.80 ± 0.27	0.671	< 0.001

Concurrently, a key marker of oxidative damage, malondialdehyde (MDA), was measured. As shown in Table 7, cadmium intoxication led to a substantial increase in hepatic MDA, reflecting severe lipid peroxidation. The group that received Tagansorbent exhibited a significantly lower level of MDA ($p < 0.001$), demonstrating the compound's efficacy in reducing oxidative damage.

Table 7. Hepatic malondialdehyde (MDA) levels (nmol g^{-1} tissue).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	12.5 ± 1.3	-	-
Cd	45.8 ± 4.9	< 0.001	-
Cd + Tag	22.4 ± 2.5	< 0.001	< 0.001
Tag Only	13.1 ± 1.4	0.502	< 0.001

The systemic inflammatory response was assessed by measuring C-reactive protein (CRP). Table 8 indicates that cadmium exposure triggered a significant inflammatory response, which was markedly suppressed in the group treated with Tagansorbent ($p < 0.01$).

Table 8. Serum C-reactive protein (CRP) levels (mg L^{-1}).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	2.1 ± 0.3	-	-
Cd	8.5 ± 0.9	< 0.001	-
Cd + Tag	4.2 ± 0.5	< 0.001	< 0.01
Tag Only	2.2 ± 0.3	0.621	< 0.001

Finally, the analysis of total protein levels, presented in Table 9, showed a significant decrease in the Cd group, likely due to both hepatic dysfunction and renal loss. Tagansorbent co-administration helped in significantly restoring the total protein levels towards the normal range ($p < 0.05$).

Table 9. Serum total protein levels (g dL^{-1}).

Group	Mean ± SD	*p*-value (vs. Control)	*p*-value (vs. Cd)
Control	6.8 ± 0.6	-	-
Cd	4.9 ± 0.5	< 0.001	-
Cd + Tag	5.9 ± 0.6	< 0.05	< 0.05
Tag Only	6.9 ± 0.7	0.715	< 0.001

In summary, data from Tables 1-9 and Fig. 1 are in consistent agreement that cadmium chloride induces serious hepatorenal toxicity and oxidative stress. Data strongly indicate that Tagansorbent ensured a significant protection with remarkable correction of the aberrant biochemical parameters.

DISCUSSION

The clear effects of cadmium toxicity on the liver and kidney systems of rats are evident from this study. A considerable increase in liver enzyme levels, such as ALT, AST, and ALP, was observed in the cadmium-treated group, as tabulated in Tables 1, 2, and 3, which suggests that cadmium acts to severely damage the membrane of hepatocytes and impair the function of the bile duct. These findings are also in agreement with those studies that have reported cadmium-induced oxidative damage to liver cells. In investigating the mechanism of toxicity, the considerable elevation in MDA within liver tissue among the poisoned group (Table 7) confirms severe oxidative stress with lipid peroxidation in cell membranes. On the other hand, a significant decrease in TAC in this group (Table 6) points out that cadmium has also disrupted the body's natural defense system. Both findings together illustrate the main mechanism of cadmium cytotoxicity. The renal function data also indicate a similar pathology: elevation in serum urea and creatinine concentrations in the poisoned group (Tables 4 and 5) is an indicator of a serious impairment of the filtration function of renal glomeruli and tubules. This is probably due to the selective accumulation of cadmium in the kidney and the development of oxidative stress in the tubular epithelial cells, which ultimately led to cell necrosis with a decrease in the glomerular filtration rate. Our data concerning corrective effects of Tagansorbent exposure showed that the level of liver enzymes in the group that received cadmium and Tagansorbent together was significantly and statistically decreased ($p < 0.01$). This can be explained by the high adsorption capacity of this substance and its formation of a stable complex with cadmium ions in the

gastrointestinal tract, which led to a decrease in the systemic absorption of this toxic metal. Confirmation of the protective effects of Tagansorbent also comes from the results on oxidative stress indices. Significant reduction in MDA levels and the simultaneous increase in TAC in the group that has been treated with Tagansorbent ($p < 0.01$) evidenced that the substance is not only effective in metal adsorption but helps protect tissues, partly by neutralizing free radicals. Such a dual action evidences the therapeutic value of Tagansorbent as a possible preventive and therapeutic agent. Renal function parameters also improved significantly in the Tagansorbent-treated group. The significant reduction in serum urea and creatinine ($p < 0.01$) suggests that the substance, by limiting cadmium absorption, was able to reduce the renal filtrable load and prevent further tubular damage. This protective effect might play a key role in avoiding cadmium-induced progressive renal failure. Of note, in the group with only Tagansorbent, the substance itself was innocuous. The stability of all biochemical parameters in this group compared to the control indicates the relative safety of this adsorbent and points to its probable safe use as therapy. This study demonstrated that Tagansorbent can ameliorate cadmium-induced hepatotoxicity and nephrotoxicity through various modes of action that include gastrointestinal absorption of metal, mitigation of oxidative stress, and preservation of cellular membrane integrity. These results highlight the excellent prospects for using this natural adsorbent as an affordable remedy in contaminated industrial regions, such as Kazakhstan.

CONCLUSION

The result of this work clearly demonstrates that poisoning with cadmium chloride causes severe biochemical disorders in rats. The marked increase in liver enzymes such as ALT, AST, and ALP and kidney function indices like urea and creatinine level constitutes serious evidence of tissue damage within these two essential organs. Also, the decline in the TAC level and the increase in MDA within liver tissue confirm the pivotal role of oxidative stress in the mechanism of cadmium toxicity. Against these toxic effects, Tagansorbent significantly prevented biochemical damage. The significant reduction of the levels of liver enzymes and the improvement in kidney function indices in the group treated with this adsorbent are indicative of its efficiency in combating cadmium toxicity. Such protective effects are most likely made through two major mechanisms: absorption of metals in the digestive tract and neutralization of free radicals. Other major findings of this study were the harmlessness of Tagansorbent, because all biochemical parameters of the group that received only this substance did not show a significant difference from the control group. This feature suggests Tagansorbent as a low-risk and reliable agent for use in heavy metal toxicity reduction programs. In the end, Tagansorbent, due to its properties of adsorption and antioxidant activity, seems to be an encouraging alternative in the prevention and reduction of complications caused by cadmium exposure. Application of this natural and widely available product can be a feasible and cost-effective method in industrial regions, such as in Kazakhstan, to protect community health against toxic environmental pollutants.

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Bibliographic information of this paper for citing:

Tungushbaeva, ZB, Kozhakhmetova, AZ, Tussupova, N, Bakesova, RM, Kunakbayev, A, Dyusembaieva. AT, Zhazira, Z 2025, Biochemical parameters of rat blood during cadmium intoxication and subsequent correction with Tagansorbent. Caspian Journal of Environmental Sciences, 23: 1209-1216.