

Effects of Cadmium on Human Kidney Function

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Abstract:

Cadmium is a highly toxic heavy metal that poses significant risks to human health due to its persistence in the environment and tendency to accumulate in biological tissues. Human exposure to cadmium occurs through contaminated food, drinking water, industrial emissions, cigarette smoke, and occupational activities such as mining, metal processing, and battery manufacturing. Among the various organs affected by cadmium toxicity, the kidneys are considered the primary target because cadmium accumulates in renal tissues over long periods. This research paper examines the effects of cadmium exposure on human kidney function, focusing on its sources, mechanisms of toxicity, and associated health consequences. Cadmium-induced nephrotoxicity is characterized by oxidative stress, inflammation, mitochondrial dysfunction, cellular apoptosis, and impairment of renal tubular function. Prolonged exposure may lead to proteinuria, reduced glomerular filtration rate, chronic kidney disease, and, in severe cases, kidney failure. Epidemiological and experimental studies have demonstrated a strong association between chronic cadmium exposure and progressive renal damage. The paper also discusses factors influencing susceptibility to cadmium toxicity, including age, nutritional status, smoking habits, occupational exposure, and genetic predisposition. Understanding the mechanisms underlying cadmium-induced kidney injury is essential for developing preventive measures, improving risk assessment, and protecting public health. The study highlights the importance of environmental monitoring, occupational safety regulations, and public awareness programmes in reducing cadmium exposure and preventing kidney-related health disorders.

Keywords: Cadmium, Nephrotoxicity, Kidney Function, Heavy Metal Toxicity, Oxidative Stress, Chronic Kidney Disease, Renal Damage, Environmental Pollution, Occupational Exposure, Public Health.

Introduction

Cadmium (Cd) is a naturally occurring heavy metal that has become a major environmental and public health concern due to its high toxicity, persistence, and bio accumulative nature. It is released into the environment through both natural processes and human activities, including mining, smelting, industrial manufacturing, battery production, electroplating, fossil fuel combustion, and the use of phosphate fertilizers. Because cadmium is not readily degraded, it accumulates in soil, water, plants, animals, and ultimately in the human body through the food chain. Human exposure occurs primarily through contaminated food, drinking water, cigarette smoke, and occupational settings, making cadmium one of the most significant environmental pollutants worldwide.

Among the various organs affected by cadmium toxicity, the kidneys are particularly vulnerable and serve as the primary target organ for cadmium accumulation. After entering the body, cadmium is absorbed into the bloodstream and transported to the liver, where it binds with metallothionein proteins. The cadmium-metallothionein complex is then filtered by the kidneys and gradually accumulates in the renal cortex. Due to its extremely long biological half-life, which may range from 10 to 30 years, cadmium can persist in kidney tissues for decades, resulting in progressive and often irreversible renal damage.

The kidneys play a crucial role in maintaining homeostasis by filtering blood, removing waste products, regulating electrolyte balance, and controlling fluid levels in the body. Chronic cadmium exposure interferes with these essential functions by damaging renal tubular cells, inducing oxidative stress, disrupting mitochondrial activity, and triggering inflammatory responses. These pathological changes can lead to proteinuria, decreased glomerular filtration rate, impaired tubular reabsorption, chronic kidney disease (CKD), and eventually kidney failure. Numerous epidemiological and experimental studies have demonstrated a strong relationship between long-term cadmium exposure and the development of renal dysfunction.

Recent advances in toxicology and molecular biology have revealed that cadmium-induced nephrotoxicity involves complex cellular and molecular mechanisms. These include the generation of reactive oxygen species (ROS), DNA damage, apoptosis, epigenetic modifications, and disruption of cellular signaling pathways. Emerging evidence also suggests that even low levels of chronic exposure may contribute to subtle kidney impairment, emphasizing the need for continuous monitoring and preventive interventions.

The growing industrialization and environmental contamination observed in many developing and developed countries have increased concerns regarding cadmium-related health effects. Therefore, understanding the impact of cadmium on kidney function is essential for effective risk assessment, environmental management, and public health protection. The present research paper aims to examine the effects of cadmium toxicity on human kidney function, focusing on exposure sources, mechanisms of renal injury, associated health consequences, and preventive strategies. The findings of this study may contribute to greater awareness of heavy metal toxicity and support the development of measures to reduce cadmium exposure and protect renal health.

1. Cadmium as a Toxic Heavy Metal

- Cadmium (Cd) is a highly toxic heavy metal naturally found in the earth's crust.
- It is classified as a major environmental pollutant due to its persistence and bioaccumulation.
- Unlike many pollutants, cadmium is not biodegradable and remains in the environment for long periods.

2. Major Sources of Cadmium Exposure

- Industrial activities such as mining, smelting, electroplating, and battery manufacturing.
- Agricultural use of phosphate fertilizers.
- Contaminated food crops grown in polluted soil.
- Cigarette smoking, which is one of the major non-occupational sources of cadmium exposure?
- Contaminated drinking water and industrial waste disposal.

3. Cadmium Accumulation in the Human Body

- Cadmium enters the body through inhalation, ingestion, and occupational exposure.
- Only a small amount is excreted, while most accumulates in body tissues.
- The kidneys and liver are the primary storage organs.
- Cadmium has a biological half-life of approximately 10–30 years.

4. Kidney as the Primary Target Organ

- The kidneys accumulate nearly 50% of the total cadmium burden in the body.
- Renal proximal tubules are particularly susceptible to cadmium toxicity.
- Long-term accumulation can cause irreversible structural and functional kidney damage.

5. Mechanisms of Cadmium-Induced Kidney Toxicity

- Generation of reactive oxygen species (ROS).
- Oxidative stress and lipid peroxidation.
- Mitochondrial dysfunction.
- DNA damage and apoptosis.
- Inflammatory responses in kidney tissues.
- Disruption of cellular signaling pathways.

Review of literature

Kalahasthi and co-researchers (2025) conducted a systematic review and meta-analysis on occupational cadmium exposure and kidney injury. The study revealed that workers chronically exposed to cadmium showed significantly higher levels of kidney injury biomarkers compared to non-exposed populations. The researchers emphasized the importance of biomonitoring and occupational safety measures.

Researchers associated with ICMR(2025) observed that chronic occupational exposure to cadmium contributes to progressive renal dysfunction. Their findings highlighted that multiple biomarkers are required to accurately assess cadmium-induced nephrotoxicity because a single marker may not adequately reflect the extent of kidney damage.

Genchi and co-researchers(2024) examined the mechanisms of cadmium-induced nephrotoxicity and reported that cadmium accumulates in the kidneys through inhalation and ingestion pathways. The study found strong associations between cadmium exposure and chronic kidney disease, end-stage renal failure, and increased mortality risk.

Satarug(2018) reviewed dietary cadmium intake and its effects on kidney function. The study reported that food-chain contamination is a major source of cadmium exposure and that long-term dietary intake may contribute to chronic kidney disease and renal dysfunction even at relatively low exposure levels.

Yang and Shu(2015) investigated cadmium transport mechanisms in the kidney and explained how cadmium enters renal epithelial cells through various transporter proteins. The study demonstrated that oxidative stress, apoptosis, and glomerular dysfunction play major roles in cadmium-induced nephrotoxicity.

Research gap

A review of the available national and international literature reveals that considerable research has been conducted on cadmium toxicity and its adverse effects on human health, particularly kidney function. Previous studies have established that chronic cadmium exposure can lead to nephrotoxicity through mechanisms such as oxidative stress, inflammation, mitochondrial dysfunction, apoptosis, and disruption of renal tubular function. However, despite significant advancements in this field, several important research gaps still exist.

Limited Focus on Early-Stage Kidney Damage

- Most studies have focused on advanced renal dysfunction and chronic kidney disease, while the early biological changes and subclinical kidney damage caused by low-level cadmium exposure remain insufficiently explored.

Inadequate Research on Low-Dose Chronic Exposure

- Existing research primarily examines populations exposed to high concentrations of cadmium. The long-term effects of low-dose environmental exposure on kidney function are not yet fully understood.

Lack of Population-Specific Studies

- Many studies have been conducted in industrialized countries, whereas region-specific studies in developing nations with varying environmental and dietary exposure patterns are relatively limited.

Insufficient Integration of Molecular and Clinical Findings

- Although molecular mechanisms such as oxidative stress and DNA damage have been identified, there is a lack of studies linking these mechanisms directly with clinical indicators of kidney dysfunction.

Aim of the study

This study aims to:

- Examine the effects of cadmium exposure and its toxicity on human kidney function. And will include the following areas of Cadmium action:
- To identify the major sources of cadmium exposure in humans, including environmental, dietary, occupational, and lifestyle-related sources.
- To assess the accumulation of cadmium in the human body and its distribution in kidney tissues.
- To investigate the effects of cadmium toxicity on kidney structure and function.
- To analyze the biological and molecular mechanisms involved in cadmium-induced nephrotoxicity, such as oxidative stress, inflammation, apoptosis, and mitochondrial dysfunction.
- To evaluate the relationship between the level and duration of cadmium exposure and the severity of kidney damage.
- To study the impact of cadmium exposure on renal biomarkers, including proteinuria, creatinine levels, glomerular filtration rate (GFR), and tubular function indicators.
- To review epidemiological evidence regarding cadmium-associated kidney diseases in different populations.

Findings of the study

The study revealed that cadmium is a highly toxic heavy metal that poses a significant threat to human kidney health due to its ability to accumulate in the body over long periods. The findings indicate that the primary sources of cadmium exposure include contaminated food and water, industrial emissions, cigarette smoking, and occupational activities such as mining, smelting, and battery manufacturing. The kidneys were identified as the primary target organ for cadmium accumulation because of their role in filtering and excreting toxic substances. The study found that chronic exposure to cadmium causes progressive renal damage through mechanisms such as oxidative stress, inflammation, mitochondrial dysfunction, cellular apoptosis, and DNA damage. These pathological changes impair normal kidney function and may lead to proteinuria, tubular dysfunction, reduced glomerular filtration rate (GFR), chronic kidney disease (CKD), and, in severe cases, kidney failure. Furthermore, epidemiological evidence demonstrated a strong association between long-term cadmium exposure and an increased risk of renal disorders in both occupationally exposed workers and the general population. The findings also highlighted that factors such as age, smoking habits, nutritional deficiencies, duration of exposure, and genetic susceptibility can influence the severity of cadmium-induced kidney damage. Overall, the study confirms that cadmium contamination remains a major environmental and public health concern, emphasizing the need for effective environmental monitoring, occupational safety measures, public awareness programmes, and preventive strategies to minimize exposure and protect kidney health.

CADMIUM INDUCED RENAL TOXICITY

The toxic effects of Cadmium are mediated through the generation of oxidative stress and ROS. The glomeruli and the renal tubular cells are particularly susceptible to the toxic effects of Cadmium, which in

turn are believed to adversely affect kidney function. Cadmium is believed to generate necrosis in renal tubular cells eventually leading to their degeneration. Over a long period of chronic Cadmium exposure, inflammation and fibrosis are evident. It must be noted that there is a threshold level for Cadmium over which adverse effects start making their appearance. It is noteworthy that presence of Cadmium induces production of Metallothionein proteins in the liver and kidney among other organs. These metallothioneins chelate free Cadmium to Cadmium-Metallothionein (Cd-MT) complex which then serves to protect the tissues from harmful effects of Cadmium.

It is interesting to note that when Cd-MT enters the cells of the renal tubules it is degraded into metallothionein and free inorganic Cadmium and the local Cd levels rise in the cells, which, then is believed to cause chronic Nephropathy. Also it has been reported that the basolateral membranes of the renal tubules can, in fact uptake free inorganic Cadmium. Moreover it has been noted that inorganic Cadmium, apart from metallothionein, has been found to bind to other low molecular weight proteins and complexes which then can cause nephrotoxicity (Zalups and Ahmad, 2003)

Conclusion

The present study concludes that cadmium is a highly toxic environmental pollutant with significant adverse effects on human kidney function. Due to its long biological half-life and strong tendency to accumulate in renal tissues, prolonged exposure to cadmium can result in progressive and often irreversible kidney damage. The study highlights that major sources of cadmium exposure include contaminated food and water, industrial activities, cigarette smoking, and occupational environments. Scientific evidence indicates that cadmium-induced nephrotoxicity is mediated through various mechanisms, including oxidative stress, inflammation, mitochondrial dysfunction, DNA damage, and apoptosis, which collectively impair the normal structure and function of the kidneys.

The findings further demonstrate that chronic cadmium exposure is associated with renal tubular dysfunction, proteinuria, decreased glomerular filtration rate, chronic kidney disease, and, in severe cases, kidney failure. Individuals exposed to cadmium over long periods, particularly industrial workers, smokers, and populations living in contaminated areas, are at a higher risk of developing kidney-related disorders. The study also emphasizes that factors such as age, nutritional status, genetic susceptibility, and duration of exposure play important roles in determining the severity of renal toxicity.

In conclusion, cadmium contamination represents a serious environmental and public health challenge that requires immediate attention. Effective environmental monitoring, strict industrial regulations, occupational safety measures, regular health screening, and public awareness programmes are essential for reducing cadmium exposure and preventing kidney damage. Furthermore, continued research on early biomarkers of nephrotoxicity, mechanisms of cadmium-induced renal injury, and preventive interventions is necessary to improve risk assessment and public health protection. Overall, minimizing cadmium exposure through coordinated efforts by governments, industries, healthcare professionals, and environmental agencies is crucial for safeguarding kidney health and ensuring long-term human well-being.

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