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Review Article

Cadmium toxicity in poultry: Mechanisms, food safety concerns, and One Health implications

I. Kar[1*], G. P. Mandal[2], S. Das[3], A. Mukherjee[4], H. K. Maity[1], S. Mandal[5] and A. K. Das[6]

[1]Department of Avian Science, Faculty of Veterinary & Animal Sciences, West Bengal University of Animal and Fishery Sciences, Mohanpur, Nadia - 741 252, West Bengal, India; [2]Department of Animal Nutrition, Faculty of Veterinary & Animal Sciences, West Bengal University of Animal and Fishery Sciences, Kolkata - 700 037, West Bengal, India; [3]Department of Fish Nutrition, Faculty of Fishery Sciences, West Bengal University of Animal and Fishery Sciences, Kolkata - 700 094, West Bengal, India; [4]Department of Animal Biotechnology, Faculty of Veterinary & Animal Sciences, West Bengal University of Animal and Fishery Sciences, Mohanpur, Nadia - 741 252, West Bengal India; [5]Assistant Director, ARD (Fodder), Directorate of Animal Resources and Animal Health, Hooghly - 712 101, West Bengal, India; [6]Assistant Director, ARD (Micro), Toxicological Lab, Directorate of Animal Resources and Animal Health, Jhargram - 721 507, West Bengal, India

Abstract

Cadmium (Cd) is a highly toxic heavy metal of increasing global concern due to its persistence, bioaccumulation, and detrimental impacts on ecosystems, animal health, and food safety. In poultry, Cd exposure occurs mainly through contaminated feed and water, leading to impaired growth, reduced productivity, immunosuppression, and reproductive dysfunction. Once ingested, Cd demonstrates low absorption but extensive tissue sequestration, particularly in the liver, kidneys, and reproductive organs, with a very slow rate of excretion. Mechanistically, Cd induces oxidative stress, mitochondrial dysfunction, disruption of mineral metabolism, genotoxicity, and endocrine interference. These pathways culminate in hematological and biochemical alterations, hepatotoxicity, nephrotoxicity, skeletal fragility, and immune organ atrophy. In India, Cd contamination has been documented in poultry feed ingredients such as rice bran, fishmeal, and groundwater near industrial zones in Punjab, Uttar Pradesh, West Bengal, and Tamil Nadu, highlighting region-specific risks for poultry production. From a food safety perspective, residues in poultry meat and eggs pose significant risks to consumers, given Cd's long biological half-life and established role as a Group 1 human carcinogen. Chronic dietary exposure through poultry products is particularly concerning in populations relying heavily on these protein sources. Beyond direct effects on flock health and consumer safety, Cd-rich poultry manure perpetuates environmental contamination, underscoring the interconnectedness of animal, human, and ecosystem health. This review consolidates current knowledge on Cd toxicokinetics, mechanisms of toxicity, pathological effects, and food safety implications within a One Health framework. Special emphasis is placed on Indian contamination scenarios, regulatory gaps, and practical mitigation strategies relevant to the Indian poultry scenario.

Keywords: Cadmium toxicity, Food safety, Heavy metal contamination, One Health, Poultry health

Highlights

- Cd is a pollutant with serious impacts on poultry health and food safety.
- Cd exposure in poultry leads to poor growth, suppressed immunity, and organ damage.
- Cd residues in poultry meat and eggs pose health risks to humans due to long half-life.
- Poultry serve as sentinel species for Cd contamination, fitting the One Health framework.
- Mitigation requires integrated strategies: monitoring feed/water, nutritional supplementation, genetic approaches, and strong regulatory policies.

INTRODUCTION

Heavy metals are a critical class of environmental pollutants due to their persistence, bioaccumulative properties, and detrimental effects on ecosystems, food webs, and public health. Unlike organic contaminants, which may degrade over time, metals such as Cd, lead, mercury, and arsenic are non-biodegradable and remain in the environment for decades. These elements enter biological systems through air, soil, water, and food chains, and once incorporated, they persist in tissues and accumulate progressively (Tchounwou et al., 2012). Anthropogenic activities-including mining, smelting, fertilizer application, industrial effluents, and waste disposal-have intensified Cd contamination,

*Corresponding Author, E-mail: indrajitkar78@gmail.com

thereby increasing its potential entry into agricultural production systems (Shahid et al., 2017). Within food-producing animals, particularly poultry, this contamination poses dual consequences: economic losses due to impaired productivity and heightened food safety risks for consumers (Korish et al., 2020).

Cd is recognized as one of the most hazardous heavy metals because of its extreme toxicity, long biological half-life, and environmental persistence. It ranks seventh on the "Priority List of Hazardous Substances" by the Agency for Toxic Substances and Disease Registry (Agency for Toxic Substances and Disease Registry [ATSDR], 2011) and has been classified as a Group 1 human carcinogen by the International Agency for Research on Cancer [IARC] (2012). Unlike essential trace minerals, Cd has no physiological role and exerts toxic effects even at very low exposure levels. Mechanistically, Cd induces oxidative stress, disrupts mitochondrial function, interferes with calcium and zinc homeostasis, and triggers apoptosis (Cuypers et al., 2010; Thévenod & Lee, 2013).

In India, Cd contamination is increasingly reported in soils, groundwater, and agricultural by-products near industrial clusters in Punjab, Uttar Pradesh, Odisha, Jharkhand, and West Bengal. Several Indian studies have detected Cd in poultry feeds-including rice bran, maize, fishmeal, and drinking water-especially in regions where poultry farms operate close to battery recycling units, tanneries, or electroplating industries (Islam et al., 2024; Mamun et al., 2024) Backyard poultry in peri-urban areas of Kolkata, Kanpur, and Ludhiana have shown measurable Cd accumulation in tissues, underscoring the relevance of this issue in the Indian context.

Its strong affinity for tissues, especially the liver, kidney, and bone, combined with a biological half-life of 10–30 years in mammals, underscores its significance as a contaminant of concern for animal and human health (Satarug et al., 2011; Nordberg et al., 2022).

The risks posed by Cd are particularly important in poultry production, given the sector's growing contribution to global food security. Poultry meat and eggs are the most rapidly expanding sources of animal protein worldwide, valued for affordability, cultural acceptability, and a relatively low environmental footprint compared with ruminant systems (Mottet & Tempio, 2017). The Food and Agriculture Organization (FAO) estimates that poultry now accounts for over 40% of global meat consumption, with demand increasing in both developed and developing regions (Food and Agriculture Organization of the United Nations [FAO] & World Health Organization [WHO], 2011). Beyond their nutritional and economic importance, poultry act as effective sentinels of environmental contamination owing to their short production cycles, rapid metabolism, and continuous exposure to feed, water, and soil that may contain heavy metals (Suhani et al., 2021).

From a production perspective, Cd exposure in poultry is associated with reduced growth rates, impaired feed efficiency, suppressed immunity, poor reproductive performance, and elevated mortality (Hashem et al., 2019). From a food safety standpoint, Cd residues in edible tissues and eggs may exceed permissible levels, posing chronic health risks to consumers (European Food Safety Authority [EFSA], 2012).

While international bodies such as Codex Alimentarius and EFSA provide guidelines for Cd limits, Indian regulations through the Food Safety and Standards Authority of India (FSSAI) and Bureau of Indian Standards (BIS) specify maximum permissible levels for heavy metals in foods and animal feeds. However, periodic monitoring is limited, and state-wise contamination data remain fragmented, indicating the need for a stronger national surveillance system in India

Additionally, Cd-rich poultry manure reintroduces the metal into soils, perpetuating contamination cycles and emphasizing the interconnectedness of animal, human, and environmental health within the One Health framework (Alloway, 2012). Although Cd toxicity has been extensively studied in mammals and aquatic organisms, systematic documentation in poultry remains limited. Considering poultry's role as both a sustainable protein source and a bioindicator species, this knowledge gap warrants urgent attention. Accordingly, this review consolidates current evidence on Cd toxicity in poultry, focusing on toxicokinetics, mechanistic pathways, toxicopathological outcomes, and food safety implications within a One Health perspective. The review also integrates India-specific contamination trends, regulatory frameworks, and practical mitigation strategies relevant to Indian poultry systems.

Sources of cadmium exposure in poultry

Cd enters poultry production systems primarily through environmental contamination and agricultural practices that facilitate its transfer into feed and water. As a non-biodegradable heavy metal, Cd persists in

ecosystems and accumulates in soil, water, and air as a result of mining, smelting, electroplating, fossil fuel combustion, and improper disposal of effluents (Tchounwou et al., 2012). Atmospheric deposition further contaminates agricultural soils and water bodies, creating long-term reservoirs of exposure (Alloway & Steinnes, 1999; Adriano, 2001; Shahid et al., 2017). Agricultural inputs such as phosphate fertilizers, sewage sludge, and pesticides also increase Cd levels in croplands, with cereals, oilseeds, and forages serving as major dietary vectors. Once ingested, Cd accumulates mainly in the liver and kidney, but residues are also found in muscle and eggs, posing food safety risks (Nordberg et al., 2018; Suhani et al., 2021). Chronic exposure impairs growth, immunity, and productivity, underscoring the need for stringent monitoring.

Absorption, Distribution, Metabolism, and Excretion (ADME) of cadmium in poultry

The toxicokinetics of Cd in poultry are characterized by low absorption, extensive tissue sequestration, and very slow elimination, leading to progressive bioaccumulation and long-term health risks (Hossain et al., 2023). Understanding Cd absorption, distribution, metabolism, and excretion (ADME) in poultry is critical for evaluating both toxicological outcomes and food safety implications.

The gastrointestinal tract is the primary entry route for Cd. Absorption efficiency is generally low (2–10% of ingested Cd) but increases under calcium, iron, or zinc deficiency due to competition for shared divalent metal transporters (Satarug et al., 2011). Uptake occurs mainly in the small intestine, mediated by divalent metal transporter 1 (DMT1) and calcium channels. Following absorption, Cd enters the portal circulation bound to albumin and plasma proteins (Nordberg et al., 2022).

Cd exhibits high affinity for soft tissues, with the kidneys and liver being the principal depots (Table 1). Hepatic accumulation induces metallothionein (MT) synthesis, which facilitates Cd sequestration (Ruttkay-Nedecky et al., 2013). Over time, redistribution favors renal accumulation, reflecting the kidney's filtration role (Kar et al., 2018). Cd also deposits in muscle, bone, and reproductive organs. Skeletal muscle deposition raises food safety

Table 1. Cadmium concentrations (mg/kg) in different tissues of poultry (Wet weight basis)

Sl. No.	Poultry Species	Muscle	Kidney	Liver	Other Organ	Reference
1.	Poultry	0.020		0.061	Heart: 0.099	Skalicka & Korenekovd (2002)
2.	Chickens	<0.003	0.065-0.73	<0.003-0.12		Jevsnik & Doganoc (2003)
	Turkeys	<0.003	0.19 -0.51	<0.003		
3.	Poultry	0.31	0.45	0.49		Mariam et al. (2004)
4.	Chickens			0.029		Villar et al. (2005)
5.	Broiler chickens	0.18	0.39	0.17		Kumar et al. (2007)
6.	Mallards (*Anas platyrhynchos*)		0.06 to 0.87 (2001);0.01 to 0.65 (2002)			Bojar & Bojar (2008)
7.	Broiler chickens	0.05 to 4.00		0.5 to 18		Kurnaz & Filazi (2011)
8.	Layer chickens	0.00	<0.001	<0.001		Mohammed et al. (2013)
9.	Free range chickens	0.001-0.012	0.80 -7.60'!	0.25-0.36	Heart: 0.002-0.030	Yabe et al. (2013)
	Broiler chickens			<0.001-0.004		
10.	Chickens			0.003-0.077	Heart: <0.001-0.007	Ismail & Abolghait (2013)
11.	Chickens	0.007 -0.038	1.56 -3.98	1.12 -3.21	Lung:0.60 -1.78	Kar et al. (2018)

concerns, while ovarian and oviductal accumulation contributes to residues in eggs (Suhani et al., 2021). In bone, Cd disrupts calcium metabolism, leading to demineralization and fragility.

Metallothioneins are central to Cd detoxification. Hepatic Cd–MT complexes are relatively stable but may be transported to the kidneys. In renal lysosomes, partial degradation releases free Cd, contributing to nephrotoxicity (Nordberg et al., 2018). The strong Cd-MT affinity accounts for prolonged tissue retention.

Excretion occurs mainly via urine and bile, with minor elimination through feces, eggs, and feathers (Hashem et al., 2019). However, clearance is inefficient (<0.01% daily), and Cd exhibits a biological half-life of years, comparable to mammals (Nordberg et al., 2018). This persistence underscores its potential for chronic toxicity in poultry.

Mechanisms of cadmium toxicity in poultry

Cd exerts toxic effects in poultry through diverse cellular and molecular mechanisms that disrupt homeostasis, Cadmium has a significant impact on various physiological systems of poultry (Fig. 1). Unlike essential trace minerals, Cd lacks physiological functions but readily interacts with biomolecules, displaces vital metal ions, induces redox imbalance, and damages macromolecules. Major mechanistic pathways include oxidative stress, mitochondrial dysfunction, disruption of trace element metabolism, genotoxicity, and immunoendocrine dysregulation.

Oxidative stress is a central mechanism of Cd toxicity. Although non-redox active, Cd indirectly enhances reactive oxygen species (ROS) formation by impairing antioxidant systems (Cuypers et al., 2010). It inhibits superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) by displacing zinc, copper, and selenium cofactors, leading to superoxide, hydroxyl radical, and hydrogen peroxide accumulation. In poultry, Cd exposure elevates lipid peroxidation, protein oxidation, and glutathione (GSH) depletion (Hashem et al., 2019), promoting apoptosis and DNA damage.

Cd disrupts mitochondrial electron transport, reducing ATP synthesis and increasing ROS leakage (Thévenod & Lee, 2013). Loss of membrane potential, cytochrome c release, and caspase activation in hepatocytes and renal cells contribute to hepatotoxicity, nephrotoxicity, and impaired growth and reproduction.

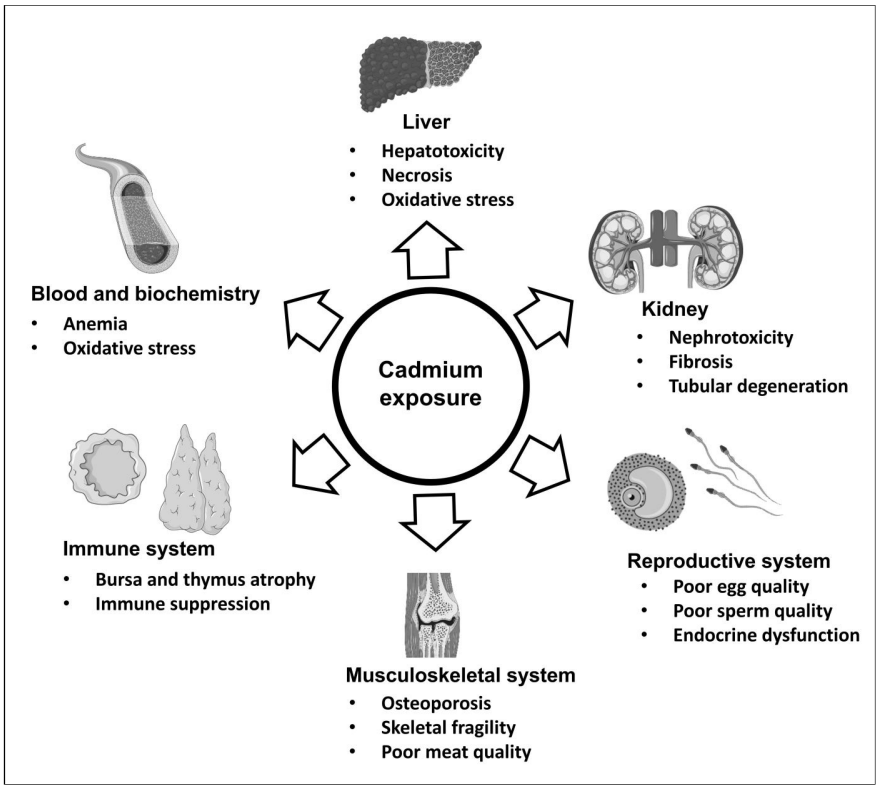

Fig. 1. The impact of cadmium exposure on different physiological systems of poultry

Cd competes with essential metals, impairing enzymatic and structural functions (Ruttkay-Nedecky et al., 2013). Substitution for zinc disrupts transcription factor activity; competition with iron interferes with heme synthesis and contributes to anemia. Selenium antagonism reduces GPx activity, worsening oxidative stress, while displacement of copper impairs cuproenzymes such as cytochrome c oxidase. These imbalances compromise immunity and systemic metabolism.

Cd induces DNA lesions, including 8-hydroxy-22 - deoxyguanosine (8-OHdG), and inhibits DNA repair enzymes such as PARP and ligase (Joseph, 2009). In poultry, chronic Cd exposure is associated with chromosomal aberrations, micronuclei, and

reduced lymphocyte proliferation. Persistent genotoxicity raises concerns about carcinogenic potential (Waalkes, 2023).

Cd suppresses immunity by reducing lymphocyte proliferation, antibody titers, and cytokine expression (Zhu et al., 2007; Xu et al., 2015) and also acts as an endocrine disruptor by binding estrogen and androgen receptors (Johnson et al., 2003).

In laying hens, Cd reduces egg production, impairs shell quality, and damages ovarian function (Kabir et al., 2004).

Toxicopathological effects of cadmium in poultry

Cd exposure induces a wide range of toxicopathological changes in poultry (Table 2), with implications for performance, organ function, immune competence, and food safety. Chronic Cd exposure in poultry is consistently associated with growth retardation, poor feed efficiency, and elevated mortality. Broilers and laying hens exposed to Cd exhibit reduced body weight gain, decreased feed intake, and poor feed conversion ratios (Kar & Patra, 2021). These effects are attributed to Cd's interference with nutrient absorption, mitochondrial energy metabolism, and protein synthesis. In broilers, such changes translate into diminished carcass yield and economic losses, while in layers, egg production declines significantly. At higher dietary doses, Cd exposure induces anorexia, weakness, neurological disturbances, and eventually mortality (Aljohani, 2023).

Table 2. Histomorphological alterations of different organs in cadmium toxicity in poultry

Sl. No.	Species	Liver	Kidney	Other organs	References
1.	Quails	* Necrosis of hepatocyte, pycnotic nucleus, and hepatic cord disarrangement, Congestion and hemorrhage	*Necrosis and deformation of normal structure of kidney tubules.	*Muscle and lung:* *Hemorrhage *Gizzard:* Mucosal erosion and discoloration.	Akter et al. (2019)
2.	Japanese quails			*Intestine:* *Sloughing of the cells into the intestinal lumen *Necrotized intestinal cells *Disruption of cell-cell junctions of intestinal epithelium	Cigánková et al. (2010)
3.	Chickens	*Cell damage and necrosis of hepatocytes *Cell size increased	*Degeneration of kidney cell	*Brain:* *Enlarges the brain cell size	Gabol et al (2014)
4.	Control, 50, and 300 mg/kg in diet for 90 days in Pekin ducks		*Inflammation of renal interstitium *Degeneration of tubular epithelium	*Testis:* *No spermatogenesis *Necrotic cells in testis	Hughes et al. (2000)
5.	Herring gulls	*Hepatic cordsdisruption	*Alterations in loop of Henle *Reduction in tubular cells around glomeruli	*Gizzard:* *Disruption of lining epithelium of gizzard	Raza et al. (2016)
6.	Japanese quails	*Cellular infiltration *Fibroblasts proliferation		*Testes:* *Arrested spermatogenesis * Elongated	Saleemi et al. (2019)

Cont. Table 2.

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Sl. No.	Species	Liver	Kidney	Other organs	References
		*Nuclear condensation,		spermatids	
7.	Broiler chickens	* Degenerative hepatocytes *Increased lymphocytes, plasma cells and macrophages in sinusoidal spaces of glomeruli	*Degeneration anddesquamation of lining epithelium of tubules *Eosinophilic hyaline mass *Hypercellularity		Singh et al. (2016)
8.	White hens			*Ovary* *Necrosis ovarian follicle *Apoptosis of growing follicles	Yang et al. (2012)

Cd disrupts hematological homeostasis, frequently causing anemia characterized by reduced hemoglobin, packed cell volume, and erythrocyte counts, largely attributable to impaired heme synthesis and increased erythrocyte fragility (Bhattacharya, 2022). Altered leukocyte profiles, including lymphopenia and heterophilia, reflect immunosuppression and systemic stress. Biochemical markers further demonstrate toxicity: serum hepatic enzymes-ALT, AST, ALP, and LDH-are elevated due to hepatocellular injury (Abdo & Abdulla, 2013), while increased creatinine and urea indicate renal dysfunction. In Japanese quails, high dietary Cd (150–300 mg/kg) increased creatinine, ALT, and AST and decreased total protein and albumin (Tahir et al., 2017). Oxidative stress, evidenced by elevated MDA and reduced SOD, CAT, and GPx, underscores Cd-induced redox imbalance (Cuypers et al., 2010).

The liver, a primary site of Cd sequestration, undergoes extensive pathological changes. Histopathological findings include hepatocellular vacuolation, fatty degeneration, sinusoidal congestion, and focal necrosis (Rani et al., 2014). Chronic exposure induces fibrosis and Kupffer cell hyperplasia, largely mediated by oxidative stress and metallothionein induction. Ultrastructural changes, such as mitochondrial swelling and rough endoplasmic reticulum disruption, impair protein synthesis and detoxification (El-Boshy et al., 2015). These alterations compromise hepatic lipid metabolism, negatively affecting growth and productivity.

The kidney, another major Cd depot, exhibits pronounced nephrotoxicity due to metallothionein-mediated accumulation. Lesions include renal tubular epithelial degeneration, glomerular shrinkage, and interstitial fibrosis, leading to impaired filtration, azotemia, electrolyte imbalance, and metabolic acidosis. Chronic renal lesions in poultry resemble Cd nephropathy in mammals, underscoring its comparative toxicological relevance (Zhang et al., 2018; Zhu et al., 2019; Abdelrahman et al., 2022).

Cd exerts severe reproductive toxicity. In males, it causes testicular degeneration, germ cell loss, and impaired spermatogenesis, often mediated by apoptosis in seminiferous tubules (Saleemi et al., 2019). In hens, Cd disrupts folliculogenesis, induces follicular atresia, and alters steroid hormone synthesis. These changes reduce egg production and shell quality. Acting as a xenoestrogen, Cd binds to steroid receptors (Johnson et al., 2003), exacerbating hormonal imbalance. Egg contamination with Cd poses direct food safety risks, with implications for public health.

Skeletal lesions include osteoporosis, decreased mineralization, and cortical thinning, largely due to Cd competition with calcium and zinc in bone remodelling (Hashem et al., 2019). Birds may present with lameness and fracture susceptibility, raising welfare and production concerns. In muscle, Cd reduces protein deposition and increases lipid peroxidation, thereby compromising meat quality, tenderness, and shelf life (Kar & Patra, 2021).

Cd exposure severely compromises immunity by targeting central and peripheral lymphoid organs. The bursa of Fabricius, critical for B-cell maturation, shows follicular atrophy, lymphocyte depletion, and necrosis (Kar et al., 2018), leading to impaired humoral immunity and reduced antibody titers. Broilers exposed to Cd exhibit diminished vaccine responses against Newcastle disease and infectious bursal disease (Ebrahimzadeh et al., 2018).

The thymus, essential for T-cell development, undergoes cortical thinning, lymphocyte apoptosis, and reduced thymocyte density (Ognik et al., 2018), impairing cell-mediated immunity. Similarly, the spleen shows lymphoid depletion, germinal center atrophy, and reduced macrophage activity (Guan et al., 2022). These lesions lower immunoglobulin levels (IgM, IgG) and impair complement activity, weakening host defence.

At the molecular level, Cd alters cytokine regulation, increasing pro-inflammatory cytokines (IL-1β, TNF-α, IFN-γ) while suppressing anti-inflammatory mediators (Genchi et al., 2020). This imbalance promotes chronic inflammation and immunosuppression. Furthermore, Cd-induced oxidative stress in lymphoid tissues triggers apoptosis and dysregulation of signalling pathways such as NF-κB, essential for immune activation (Cuypers et al., 2010; Thévenod & Lee, 2013).

Chronic Cd exposure results in progressive accumulation in the liver, kidney, brain, pancreas, intestine, and reproductive organs, promoting oxidative stress and molecular damage. These lesions collectively impair growth, reproduction, immunity, and survivability, while residues in meat and eggs pose significant public health concerns (Kar & Patra, 2021).

Food safety concerns and public health implications

The presence of Cd residues in poultry products represents a critical concern at the intersection of animal health, food safety, and public health. Poultry meat and eggs are major dietary sources of affordable, high-quality protein worldwide. However, their contamination with Cd provides a direct route of human dietary exposure. Studies have documented Cd accumulation in edible poultry tissues, particularly the liver and kidneys, which serve as primary depots, but also in muscle-the tissue most frequently consumed by humans. Deposition in eggs, although typically lower than in visceral organs, can become significant with prolonged exposure (Kar et al., 2018). Such findings underscore the importance of Cd contamination as a tangible global food safety issue.

Cd residues in poultry-derived foods are of particular concern due to Cd's persistence and prolonged half-life in humans. Once absorbed, Cd accumulates mainly in the liver and kidneys, with a biological half-life of 10–30 years, making even low-level chronic exposure hazardous (Satarug et al., 2011). Eggs represent an additional exposure pathway, with long-term feeding of Cd-contaminated diets leading to measurable residues in yolk and albumen (Genchi et al., 2020). This is particularly concerning in vulnerable populations, including children, pregnant women, and the elderly, who have higher absorption efficiency and lower detoxification capacity.

To safeguard public health, regulatory agencies have established maximum permissible limits for Cd in foods of animal origin. The Codex Alimentarius Commission and European Food Safety Authority (EFSA) provide guidance to minimize dietary exposure (EFSA, 2009; Food and Agriculture Organization of the United Nations [FAO] & World Health Organization [WHO], 2011). EFSA has defined a tolerable weekly intake (TWI) of 2.5 μg/kg body weight, while the Joint FAO/WHO Expert Committee on Food Additives (JECFA) recommends a provisional tolerable monthly intake of 25 μg/kg body weight. Exceeding these thresholds increases lifetime risks of renal dysfunction, osteoporosis, cardiovascular disorders, and carcinogenic outcomes (Jarup & Akesson, 2009; Thévenod & Lee, 2013). Given Cd's persistence, accumulation over time may surpass toxic thresholds even when short-term exposure appears minimal.

Populations living in regions with high environmental Cd burdens-particularly near mining, smelting, or industrial discharge sites—are especially vulnerable (Suhani et al., 2021). Products derived from the poultry industry may pose substantial health hazards owing to contamination with toxic heavy metals via the food chain and environmental exposure (Pappuswamy et al., 2021). Risks are further amplified in backyard and smallholder poultry systems, where birds scavenge on contaminated soils, consume untreated water, or feed on agricultural by-products. In rural communities where poultry products constitute the primary protein source, Cd contamination threatens both food security and human health (Okoye et al., 2011; Tchounwou et al., 2012; Unsal et al., 2020; Kar & Patra, 2021). Despite the classification of Cd as a Group 1 human carcinogen by the International Agency for Research on Cancer (IARC, 2012), consumer awareness of contamination in poultry products remains low. The cumulative, insidious nature of Cd toxicity-marked by bioaccumulation without immediate symptoms-complicates early detection and public health responses. Consequently, chronic exposure often remains unnoticed until irreversible renal or skeletal damage emerges.

Mitigation and management strategies

Surveillance and monitoring of cadmium residues: Mitigation strategies require strengthened surveillance

of Cd residues in poultry meat and eggs. Systematic monitoring of feed ingredients, water sources, and finished products should be prioritized, coupled with stricter enforcement of regulatory standards. Improved feed quality control is essential, as contaminated feedstuffs remain a primary entry route for Cd into poultry systems. Public health strategies should also emphasize raising awareness among consumers, producers, and policymakers regarding Cd's long-term risks (EFSA, 2009; Unsal et al., 2020). To control environmental cadmium (Cd) toxicity effectively, strategies must combine source reduction with active soil and water management. Primary prevention entails the stringent regulation of agricultural inputs to mitigate the entry of cadmium, including the restriction of cadmium-rich phosphate fertilizers and the prohibition of untreated wastewater or sewage sludge application on agricultural land. To address existing contamination, the application of soil amendments such as lime, farmyard manure, and biochar elevates soil pH and sequesters metals, therefore markedly diminishing their mobility and absorption by crops. These initiatives are bolstered by remediation methods, such as phytoremediation employing hyperaccumulator plants (e.g., *Brassica napus*) to extract cadmium from soils and aquatic plants (e.g., water hyacinth) to cleanse water bodies, in conjunction with precision agriculture technologies that reduce chemical runoff and leaching.

Public health approaches to fight cadmium (Cd) toxicity include stringent regulation, enhanced monitoring, and community engagement. Authorities enforce maximum permissible quantities (3-5 µg L^{-1}) in water in accordance with EU Drinking Water Directive and WHO norms, ensuring traceable, safe food supply chains. This is supported by "One Health" surveillance systems that map contamination hotspots and use IoT and AI for real-time pollution detection. Informing consumers and farmers about health concerns through public education generates demand for certified safe products and helps enforce safety measures (Vasile Scãeteanu et al., 2025).

Cadmium and the One Health perspective: The One Health paradigm highlights the intrinsic interconnectedness of human, animal, and environmental health, emphasizing that sustainable solutions to modern health challenges require a multidisciplinary approach. Cd, a persistent and highly toxic environmental pollutant, exemplifies this interconnectedness. With no physiological function in living systems, Cd exerts adverse effects across ecosystems, animal production, and human health (Fig. 2). Its capacity for environmental persistence, bioaccumulation, and biomagnification along food chains positions it as a contaminant of critical concern within the One Health framework.

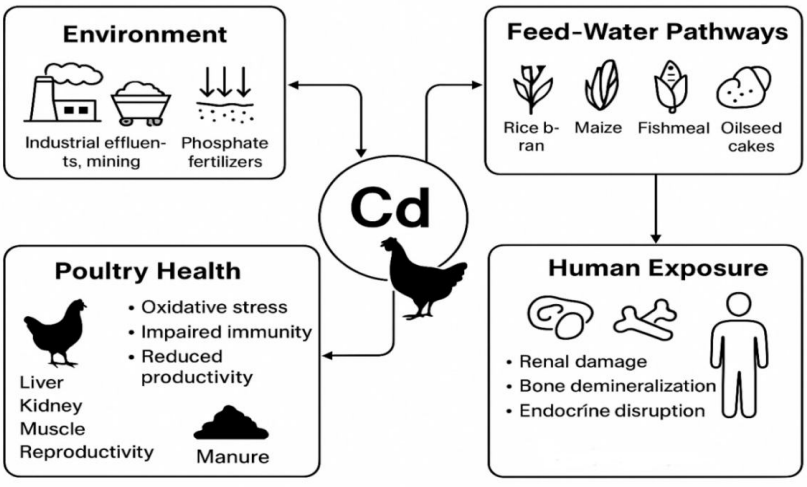

Fig. 2. A conceptual diagram illustrating the cadmium contamination cycle within the One Health framework - Environmental pollution translates into poultry and human health risks. The visual summary depicts four interconnected domains: Environment, Feed–Water Pathways, Poultry Health, and Human Exposure, linked by directional arrows to demonstrate Cd flow

Linkages between environmental contamination, poultry health, and human health: The environmental burden of Cd arises primarily from anthropogenic activities, including mining, smelting, fossil fuel combustion, waste incineration, and phosphate fertilizer application (Jarup & Akesson, 2009). These practices contaminate soil, water, and air, creating long-term reservoirs of Cd that subsequently enter agricultural systems. Poultry, as one of the most intensively reared livestock species globally, are highly dependent on feed and water quality, which makes them particularly vulnerable to Cd exposure. Feed ingredients such as cereals, legumes, and oilseed by-products often accumulate Cd from contaminated soils and fertilizers, thereby serving as primary exposure pathways for poultry (Kar & Patra, 2021).

Once ingested, Cd induces a spectrum of toxicopathological effects in poultry, including hepatotoxicity, nephrotoxicity, immunosuppression, and reproductive impairments, which cumulatively compromise flock health, productivity, and economic sustainability (Wang et al., 2021). Of greater concern, however, is the deposition of Cd residues in poultry meat and eggs—staple protein sources consumed worldwide. Transfer of these residues into the human food chain heightens dietary exposure risks, particularly in populations with high poultry consumption (Suhani et al., 2021). In humans, chronic Cd exposure has been unequivocally linked to renal dysfunction, osteoporosis, endocrine disruption, and carcinogenesis (IARC, 2012; Genchi et al., 2020). Thus, poultry act not only as production animals but also as sentinel species, reflecting the broader environmental burden of Cd and its potential translation into public health hazards.

Ecosystem-level impacts and the bioindicator role of poultry: Beyond direct effects on poultry and consumers, Cd contamination exerts far-reaching consequences at the ecosystem level. In terrestrial systems, Cd impairs soil fertility by reducing microbial diversity and disrupting nutrient cycling, thereby negatively influencing crop productivity and feed availability (Satarug et al., 2011). In aquatic ecosystems, Cd accumulates in sediments and propagates through food webs, inducing toxicity in fish and invertebrates. This contamination has indirect consequences for poultry production, as fishmeal and other aquatic-derived ingredients are commonly incorporated into poultry diets, introducing an additional exposure pathway (Das et al., 2023).

Given their widespread distribution, standardized production systems, and continuous interaction with feed and water, poultry can function as effective bioindicators of environmental Cd contamination. Monitoring Cd residues in poultry tissues and eggs provides a cost-efficient tool for assessing both environmental contamination and potential food safety hazards (Korish & Attia, 2020). This bioindicator role directly supports One Health objectives by linking environmental surveillance with early detection of risks to animal and human health.

Relevance for the Global One Health framework: The One Health approach emphasizes the need for cross-sectoral collaboration in managing health threats that transcend ecological and disciplinary boundaries. Cd contamination necessitates such coordinated efforts, as its impacts extend across environmental, veterinary, agricultural, and public health domains.

At the environmental level, reducing Cd release requires stringent regulation of industrial emissions, improved waste management practices, and judicious use of phosphate fertilizers. In agricultural systems, feed quality monitoring, soil testing, and the adoption of remediation strategies such as phytoremediation or soil amendments are critical to reducing Cd transfer into the poultry production cycle (FAO/WHO, 2011). From the veterinary perspective, preventing Cd accumulation in poultry requires both proactive monitoring of feed ingredients and the adoption of dietary interventions, such as the use of mineral supplements or adsorbents, to mitigate Cd absorption.

Public health authorities also play a central role by establishing and enforcing maximum residue levels (MRLs) in poultry meat and eggs to safeguard consumers. The European Food Safety Authority [EFSA] (2009) has established a tolerable weekly intake (TWI) of 2.5 µg/kg body weight, while the Joint FAO/WHO Expert Committee on Food Additives (JECFA) recommends a provisional tolerable monthly intake of 25 µg/kg body weight. These international guidelines provide benchmarks for regulatory frameworks but must be adapted for low- and middle-income countries, where monitoring capacity is often limited and poultry serves as a primary source of affordable protein.

Ultimately, Cd contamination illustrates the core principles of One Health- protecting environmental quality directly reduces Cd entry into food chains, thereby preserving poultry health and productivity and ensuring safe, nutritious food for human populations. Addressing Cd toxicity thus requires integrated strategies that bridge environmental regulation, veterinary medicine, agricultural practices, and public health interventions. By embedding poultry production into the broader One Health agenda, it becomes possible to break the cycle of Cd contamination, ensuring sustainable production systems and healthier outcomes across species.

Surveillance and intervention strategies: Along with scientific methods, public education is essential for controlling pollution. Once pollutants, especially complex ones like heavy metals (HMs), enter the environment, they cannot be fully removed and are very hard to manage. Therefore, raising awareness about the sources, impacts, and prevention of heavy metal pollution is crucial. Promoting sustainable farming practices, such as reduced chemical use, organic farming, and precision agriculture, offers cleaner alternatives (Vasile Scãeteanu et al., 2025). Though

these methods may slightly lower yields, they help prevent long-term environmental harm. In summary, heavy metal pollution from agriculture is a growing concern, but with proper management and greater public awareness, its risks can be significantly reduced.

Reducing cadmium contamination in feed and water: Feed and water represent the primary pathways of Cd exposure in poultry, making reduction at these levels a key mitigation priority. Feed ingredients, particularly cereals, oilseeds, and animal by-products, may accumulate Cd when produced in contaminated soils or irrigated with polluted water, often linked to industrial effluents, phosphate fertilizers, or sewage sludge (McLaughlin et al., 1999; Alloway, 2012). Rigorous screening of feed ingredients and sourcing from low-Cd regions are critical preventive measures. Adsorbents such as zeolite, bentonite, activated carbon, and biochar have demonstrated the ability to bind Cd in the gastrointestinal tract, reducing its bioavailability (Duruibe et al., 2007). Similarly, water purification techniques—including reverse osmosis, ion exchange, and nanofiltration-effectively lower Cd levels in drinking water. At the farm level, secure feed storage and minimizing dust exposure near industrial zones further reduce contamination risks.

Nutritional interventions: antioxidants, trace minerals, and phytochemicals: Nutritional interventions provide an important defense against Cd-induced toxicity, primarily by mitigating oxidative stress. Antioxidants such as vitamin E and selenium synergistically enhance glutathione peroxidase activity, prevent lipid peroxidation, and stabilize cell membranes (Hashem et al., 2019). Vitamin C supports redox balance by scavenging reactive oxygen species and regenerating other antioxidants (Patra et al., 2011).

Trace minerals play complementary roles in reducing Cd absorption and toxicity (Table 3). Zinc decreases intestinal Cd uptake by competing for transporters and induces metallothionein synthesis, thereby promoting the formation of less toxic Cd–MT complexes (Wen et al., 2018). Iron reduces Cd absorption by saturating divalent metal transporters (Blalock et al., 1988), while selenium provides both antioxidant protection and detoxification (Kar et al., 2018). However, boron supplementation has shown no protective effect in hens (Olgun & Bahtiyarca, 2015). In contrast, co-exposure studies in ducks revealed synergistic nephrotoxicity with Cd and molybdenum, characterized by oxidative stress and mitochondrial damage (Xia et al., 2015).

Phytochemicals also contribute to mitigation through their antioxidant, chelating, and detoxifying properties (Table 3). Compounds such as curcumin, allicin, gingerols, and green tea polyphenols have demonstrated protective effects. Herbal extracts from *Withania somnifera* and *Ocimum sanctum* enhanced antioxidant defense by decreasing lipid peroxidation and restoring SOD, CAT, and GSH activity (Bharavi et al., 2011). Polysaccharides from *Agaricus blazei* lowered Cd deposition, malondialdehyde (MDA), inflammatory cytokines, and HSPs while improving

Table 3. Amelioration of cadmium (Cd) toxicity in poultry by phytomedicinal plants and trace minerals

SL No.	Species	Ameliorative agent	Effects	Reference
1.	Chickens	*Ocimum sanctum* leaf, *Spirulina platensis*, *Withania somnifera* root powder	● ↑ Body weight gain ● ↑ GSH and ↓ TBARS level in liver and kidney ● ↓ Serum ALT, creatinine and urea concentration level	Bharavi et al. (2011)
2.	Chickens	Ferrous sulfate	● Fe decreased Cd content in kidney and liver	Blalock & Hill (1988)
3.	Hailan cocks	Sodium selenite	● Se alleviated oxidative stress in livers caused by Cd.	Cong et al. (2019)
4.	Broiler chickens	Sodium selenite and vitamin E	● ↓ MDA and ↑ GPx activity Se and vitamin E ↑ IL-2 and 10	Hashem et al. (2019)
5.	Laying chickens	*Agaricus blazei* Murill	● ↑ Antioxidant enzyme activities, ↓ MDA content, expressions of inflammatory cytokines in livers.	Hu et al. (2017)

Cont. Table 3.

Table 3., Cont. ...

SL No.	Species	Ameliorative agent	Effects	Reference
6	Laying Japanese quails	Zinc sulfate in water at 4 mg/day per quail for 58 day	● Zn ↑ eggshell thickness and strength, which are reduced by Cd	Korénekova et al. (2007)
7.	Laying Hens	Boron	● ↓ Growth and eggshell thickness (at higher doses).	Olgun & Bahtiyarca (2015)
8.	Broiler chickens	Emblica Officinalis fruit	● ↓ MDA concentration and ↑ the activity of liver catalase and SOD.	Swapna et al. (2010)
9.	Chickens	Sodium Selenite	● ↓ Cd content in ovarian tissue ↓ apoptosis via ER stress pathway	Wan et al. (2018)
10.	Ducks	Molybdenum	● ↓ Total antioxidative capacity and the activity of xanthine oxidase and ↑ MDA and NO-synthase level in the kidney mitochondria synergistically	Xia et al. (2015)

antioxidant status (Hu et al., 2017; Song et al., 2018; Xie et al., 2018). Moreover, supplementation with *Lactobacillus* spp. facilitated Cd excretion by binding Cd ions in the gut, thereby reducing tissue burden and oxidative damage (Zhai et al., 2016; Djurasevic et al., 2017). These nutritional strategies not only counteract toxicity but also enhance growth, immunity, and product quality.

Breeding and genetic approaches: Long-term mitigation may be achieved through genetic strategies that enhance resistance to Cd toxicity. Variation in Cd absorption, distribution, and detoxification has been observed in poultry, often linked to differences in metallothionein and antioxidant enzyme expression (Ruttkay-Nedecky et al., 2013). Lines with higher basal levels of metallothioneins or heat shock proteins may exhibit improved tolerance (Cuypers et al., 2010). Advances in transcriptomics, proteomics, and genome-wide association studies provide biomarkers for resistance and inform selective breeding (Joseph, 2009). Emerging tools such as CRISPR/Cas9 also hold promise for enhancing detoxification pathways, though ethical and regulatory challenges remain (Thévenod & Lee, 2013).

Policy, monitoring, and regulatory frameworks: Effective management also requires robust regulatory oversight. International bodies such as EFSA and Codex Alimentarius have established maximum limits for Cd in poultry feed and products, including 0.5 mg/kg in feed and 0.05 mg/kg in meat (EFSA, 2012). Systematic monitoring of Cd in feed, water, and poultry-derived foods is essential to ensure compliance. Preventive policies must address upstream contamination sources, including stricter controls on industrial emissions, reduced use of Cd-rich fertilizers, and limitations on sewage sludge application (Kabata-Pendias, 2011). Farmer education enhances awareness and adoption of best practices in feed sourcing, water management, and supplementation.

Given the globalization of feed supply chains and poultry markets, international collaboration and harmonization of standards are crucial. Coordinated action under the One Health framework underscores Cd as a shared environmental, veterinary, and human health hazard, necessitating integrative solutions (FAO/WHO, 2014).

Permissible standards and residue-monitoring practices for cadmium in India: Codex Alimentarius Commission, European Food Safety Authority, Joint Expert Committee on Food etc are several bodies to regulate the Cd levels in the environment. They provide guidelines for health based on maximum residue limits. In India, for monitoring of heavy metals, including Cd other standard-setting bodies are there like Food Safety and Standards Authority of India, BIS, CGWB and NCDC. They work on heavy metals to assess human exposure, guide public health policy and also the maximum intake limitation of Cd (e.g., EFSA TWI = 2.5 µg/kg bw/week; JECFA PTMI = 25 µg/kg bw/month). Tissue-specific maximum

residue limits, a surveillance programme and a proper monitoring system are recommended to improve the health status. Indian guidelines are not as systematic surveillance infrastructure as international ones- this is the basic difference between the guidelines of India and International ones.

Conclusion

Cd is a persistent environmental pollutant with profound implications for poultry health, food safety, and the broader One Health continuum. Evidence shows that Cd exposure impairs growth, alters hematological and biochemical parameters, induces organ-specific lesions, and weakens immunity, thereby reducing flock productivity and raising risks of Cd residues in meat and eggs. These residues pose serious public health threats, particularly in regions with high environmental Cd burden, despite established regulatory limits. Addressing this issue requires comprehensive monitoring, stricter enforcement of residue standards, and farm-level interventions. From a One Health perspective, poultry act as both sentinels and vectors of Cd entry into the food chain, highlighting the interconnectedness of environmental, animal, and human health. Mitigation strategies should include reducing Cd contamination in feed and water, adopting nutritional and genetic interventions, and strengthening environmental and food safety regulations to ensure sustainable poultry production and consumer protection.

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