HEAVY METALS AND METALLOIDS IN LIVESTOCK SYSTEMS: EXPOSURE PATHWAYS, TOXIC MECHANISMS, REPRODUCTIVE RISK AND FOOD-CHAIN SAFETY

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Abstract

Heavy metals and metalloids are important contaminants in livestock production because they connect environmental pollution with animal health, productivity, reproduction and food safety. Some trace elements, including Cu, Zn, Fe, Mn, Co, Mo and Se, are essential for physiological, catalytic and regulatory functions, but excessive intake may convert them into toxicants. In contrast, Pb, Cd, Hg and As have no established nutritional role and are considered undesirable contaminants in feed and food chains. Livestock exposure occurs mainly through contaminated feed, forage, soil, water, industrial emissions, mining residues, fertilizers, pesticides, sewage sludge, wastewater irrigation and improperly formulated mineral supplements. Toxicity depends on dose, chemical form, route and duration of exposure, animal species, age and nutritional status. The major toxic mechanisms include oxidative stress, enzyme inhibition, displacement of essential minerals, mitochondrial dysfunction, endocrine disruption and tissue bioaccumulation. Kidney, liver, nervous system and reproductive organs are major target sites, whereas residues in milk, meat, eggs and edible offal create public-health concerns. This article synthesizes evidence from uploaded review articles and toxicological data to highlight exposure pathways, organ toxicity, reproductive impairment, feed–manure–soil cycling and prevention strategies in livestock systems.

Keywords: heavy metals, metalloids, livestock, nephrotoxicity, reproduction, food-chain safety, mineral nutrition

Introduction

Heavy metals and metalloids are now recognized as a livestock-system problem rather than only an environmental problem. They move through soil, water, feed, forage, animals, manure and animal-origin foods. Hejna et al. (2018) described this as a nutritional-ecology issue, where the same production system may contain both essential trace elements and undesirable contaminants. Essential elements such as Fe, Cu, Zn, Mn, Co, Mo and Se support metabolism, enzyme function, immunity, growth and reproduction; however, excessive supply can produce cellular or systemic toxicity. In contrast, As, Cd, Pb and Hg are considered contaminants or undesirable substances in animal feed because they have no established biological function and may damage organs after exposure. Toxicity is influenced by dose, route, chemical species, duration of intake, species, genetics and nutritional status.

The practical concern in livestock is often chronic exposure rather than obvious acute poisoning. Afzal and Mahreen (2024) emphasized that after ingestion, heavy metals may persist in the body and may produce acute, chronic, clinical, subclinical or subtle toxicity. Chronic exposure may reduce body condition, depress production potential and increase residues in consumable animal products.

Sources and exposure pathways

Livestock are mainly exposed through ingestion of contaminated feed, forage, soil and water. Important sources include industrial effluents, mining, automobile emissions, fertilizers, pesticides, sewage sludge, manure, wastewater irrigation, battery waste, paints, contaminated groundwater and mineral supplements. Afzal and Mahreen (2024) reported that industrial wastewater can introduce hazardous metals into soil and water bodies, while phosphate fertilizers, sewage sludge and animal manure may add metals to agricultural soils. Animals grazing in contaminated areas may then ingest polluted herbage, soil or water, leading to tissue accumulation.

Production system modifies the risk. In extensive systems, grazing animals may be exposed to contaminated soil, dust, forage and water, especially near industrial or mining areas. In intensive systems, exposure is more closely linked with compound feed, imported ingredients and mineral additives. Tahir and Alkheraije (2023) specifically identified industries, fertilizers, traffic, automobiles, paint, groundwater and animal feed as sources of heavy-metal contamination in cattle. Domestic animals are exposed mostly through plants, feed, soil and water, while inhalation becomes relevant near industrial and traffic-polluted environments.

Toxic mechanisms and target organs

The central mechanism of heavy-metal toxicity is disturbance of cellular homeostasis. Toxic metals generate reactive oxygen species, reduce antioxidant defenses, impair enzyme systems and disrupt mineral-dependent cellular processes. Afzal and Mahreen (2024) described oxidative stress as a key toxic pathway involving reactive oxygen and nitrogen species, depletion of intracellular antioxidants and reduced enzymatic detoxification capacity. These changes contribute to immunosuppression, poor body condition and impaired production.

Pb is particularly important in cattle because it is widespread in contaminated feed, soil, paints, machinery grease and industrial environments. It may interfere with calcium-dependent signaling, heme synthesis and sulfhydryl-containing enzymes. Tahir and Alkheraije (2023) noted that continuous low-level Pb exposure can still accumulate in tissues because of slow removal, and Pb exposure increases reactive oxygen species such as superoxide radicals, hydroxyl radicals, lipid peroxides and hydrogen peroxide.

Cd is a chronic accumulator, especially in kidney and liver. Reis et al. (2010) reported that the maximum tolerated Cd concentration in animal diets is about 0.5 mg/kg. In cattle, diets containing 5–30 mg Cd/kg may reduce performance, while diets containing ≥30 mg Cd/kg may disturb health. In sheep, diets containing >40 mg Cd/kg DM were associated with parakeratosis, reduced appetite, poor body-weight gain and testicular effects.

Cd toxicity is strongly linked with metallothionein. After intestinal absorption, Cd is transported to the liver, where it induces metallothionein synthesis. The Cd–metallothionein complex is released into blood, filtered by kidney glomeruli and degraded in tubular cells. Because metallothionein protection is limited, excessive Cd intake may result in accumulation and organ damage, particularly in liver, kidney, bone, nervous system, testis, intestine, skin and blood.

Cu requires careful interpretation because it is an essential trace element but becomes toxic when oversupplied. Reis et al. (2010) reported that sheep are especially susceptible to Cu poisoning because of limited capacity to increase biliary Cu excretion. Excessive Cu accumulates in the liver, causes oxidative hepatocyte injury, enters the bloodstream and induces intravascular hemolysis, anemia, jaundice and hemoglobinuria. In calves, milk containing up to 50 ppm Cu did not cause poisoning signs, whereas 200–500 ppm reduced weight gain and 1,000 ppm caused death within 3–5 days.

Nephrotoxicity and systemic effects

Kidney is one of the most important target organs, particularly for Pb, Cd and As. Tahir and Alkheraije (2023) stated that the nephrotoxic effects of Pb, As and Cd are well established in cattle. These metals are linked with oxidative stress, free-radical production and acute or chronic kidney injury. Pb may damage renal proximal tubules, alter antioxidant enzymes, induce apoptosis and contribute to interstitial nephritis. Cd mainly accumulates in proximal tubular regions and may impair reabsorption of proteins, bicarbonate, phosphate and amino acids. As exposure has been linked with tubular necrosis, glomerular sclerosis and oxidative renal damage.

Systemic signs depend on the metal and exposure pattern. Pb exposure in cattle may produce depression, blindness, ataxia, convulsions, coma, gastrointestinal disturbance and death. As may cause gastrointestinal and nervous signs, weight loss, mucosal lesions and reduced milk yield. Cu toxicity may produce hepatic damage followed by hemolytic crisis. Cd exposure is often chronic and may appear as poor growth, anemia, renal damage, bone effects or reproductive impairment.

Reproductive toxicity

Reproductive toxicity is important but must be interpreted cautiously because much mechanistic evidence comes from experimental animals or in vitro studies. Verma et al. (2018) reported that heavy-metal exposure can cause chronic reproductive toxicity, producing structural and functional cellular impairment. The affected processes include steroidogenesis, hormonal regulation, gametogenesis, Leydig-cell activity, spermatogenesis, granulosa-cell function, theca-cell activity, placental growth, pregnancy rate and fetal development.

Pb may accumulate in testes, epididymis, vas deferens and seminal vesicles. It can impair spermatogenesis and steroidogenesis, detach germinal cells from the basal membrane and cause Leydig-cell atrophy. It may also reduce seminal plasma constituents such as fructose and succinic dehydrogenase, contributing to azoospermia, asthenozoospermia, teratozoospermia and sperm tail abnormalities.

Cd interacts with Zn, Fe, Cu and Se due to chemical similarities and may also influence Ca and P metabolism in bone. Experimental reproductive evidence indicates that Cd may impair steroidogenesis, inhibit DNA repair, reduce antioxidant capacity and disturb placental progesterone synthesis. Verma et al. (2018) also noted that Hg can act as a spermatotoxic, steroidotoxic and fetotoxic agent, while As may damage androgen-binding protein and alter steroidogenic enzyme activity.

Food-chain safety and environmental recycling

Food-chain transfer depends on absorption, dose, chemical form, exposure duration and tissue distribution. Hejna et al. (2018) reported that animal-origin food contamination depends on carry-over into milk, eggs and meat, which is influenced by absorption, bioaccumulation, metabolism and excretion. Under standard diets below permissible limits, transfer of Cd, Pb, As and Hg into milk, eggs and muscle is generally low, but higher exposure increases residues in liver, kidney and bone. Afzal and Mahreen (2024), while summarizing previous residue studies, reported Pb concentrations of 0.05–0.58 mg/kg dry matter and Cd concentrations of 0.02–0.04 mg/kg in muscles of fattening pigs. Sheep under pasture and extensive systems may accumulate Cd in kidneys and udder, while Pb, Zn and Cu may predominate in ribs, liver and long bones. Backyard poultry may accumulate metals from landfills and contaminated soil, and ducks or geese near industrial areas may show higher As, Cd and Hg in eggs than birds from non-industrial sites. Manure also matters. Metals excreted through feces and urine may return to agricultural soil, especially when manure from supplemented animals is used as fertilizer. This makes heavy-metal control a circular issue: contaminated feed produces contaminated manure, which can contaminate soil, forage and future feed.

Prevention and control

Prevention is more effective than treatment. Farm-level control should include routine testing of feed, forage, water, mineral mixtures and suspicious environmental sources. Animals should be prevented from accessing old batteries, paints, machinery grease, mining waste, industrial discharge and contaminated ponds. Mineral supplementation should be requirement-based, not based on maximum legal limits. Environmental control should include safe manure management, avoidance of wastewater irrigation in fodder fields unless tested, and monitoring of farms near industrial zones. Afzal and Mahreen (2024) noted that composting, phytoremediation, intercropping and biochar may help reduce metal bioavailability or environmental burden. Biochar from pyrolysis may reduce metal mobility, improve soil quality and limit contamination risk.

Conclusion

Heavy metals and metalloids in livestock systems represent a connected problem of exposure, bioaccumulation, organ toxicity, reproductive impairment and food-chain risk. Essential trace elements are required only within physiological limits, whereas Pb, Cd, Hg and As remain major undesirable contaminants because of their persistence, tissue accumulation and toxic effects on kidney, liver, nervous and reproductive systems. Chronic low-level exposure is especially important, as it may silently reduce growth, milk yield, fertility and product safety before obvious clinical signs appear. Therefore, control should begin at the source through regular monitoring of feed, forage, water, soil, mineral supplements and edible tissues, particularly in industrial, mining, peri-urban and wastewater-irrigated areas. Precision mineral nutrition, safe manure recycling, avoidance of contaminated grazing and removal of farm-level hazards are essential to protect livestock productivity, reproductive efficiency and public health.

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