

A Review of Lead (Pb) and Cadmium (Cd) Contamination in Food Crops in Kenya

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Abstract

In Kenya, rapid urbanization, poorly managed industrialization, and the intensive use of agrochemicals have heightened the risk of toxic element accumulation in the food supply chain. Among these contaminants, Lead (Pb) and Cadmium (Cd) pose severe risks due to their non-biodegradable nature, long biological half-lives, and high toxicity even at low concentrations. This study aimed to systematically synthesize a decade of empirical evidence (2016–2026) regarding lead (Pb) and cadmium (Cd) contamination in food crops in Kenya, map their geographic and anthropogenic pathways, characterize the associated multi-systemic human health risks, and identify structural regulatory gaps. Following a predefined systematic protocol, a narrative review was executed across major electronic databases (including PubMed, ScienceDirect, Scopus, Web of Science, Google Scholar, and AJOL). A total of 33 datasets for Pb and 14 for Cd evaluating concentrations in edible plant tissues, agricultural soils, and irrigation networks within Kenya were extracted and cataloged. Empirical data were stratified across urban, riverine, mining, commercial, and post-harvest retail zones, and benchmarked against international health thresholds established by the FAO, WHO, and Codex Alimentarius. The evidence revealed a stark, structurally entrenched contamination gradient following an urban-industrial > peri-urban riverine > rural high-input > rural low-input pattern. Leafy vegetables (kale, spinach, and indigenous greens) hyperaccumulated both metals. In the Nairobi-Machakos-Kiambu corridor, mean Pb concentrations in urban kale reached 0.68 ppm, driven by vehicular emissions, road dust, and untreated wastewater irrigation. Riverine systems like the Athi and Kabuthi river basins recorded extreme dry-season Pb concentrations up to 12.10 mg/kg due to industrial effluent. The most catastrophic localized exposure occurred in Migori County's artisanal gold mining zones, where vegetable Pb levels climbed to 71.28 mg/kg (nearly 240 times the Codex limit) and soil Pb reached 706 mg/kg. Conversely, commercial farmlands in Trans Nzoia and Western Kenya exhibited systemic Cd soil enrichment exceeding WHO agricultural limits tenfold driven by the continuous application of phosphate-based fertilizers. Toxicological benchmarking indicated severe, multi-systemic, and

intergenerational risks. Approximately 69% of children consuming Nairobi's urban-grown greens exceed daily Pb reference doses, predisposing them to irreversible neurodevelopmental injury, cognitive deficits, and downward social mobility. In adults, dietary Pb directly amplifies cardiovascular disease and mortality risks. Due to its 25-to-30-year biological half-life, cumulative dietary Cd exposure presents a silent epidemic of progressive renal tubular dysfunction, chronic kidney disease, bone demineralization, and multi-organ carcinogenicity via mutagenic and heritable epigenetic pathways. Kenya's food safety governance framework is currently inadequate, it lacks enforceable domestic maximum residue limits (MRLs), a national food contaminant surveillance program and sufficient laboratory infrastructure requiring urgent, coordinated, and evidence-based legislative and environmental interventions to safeguard Kenyan public health and food security.

Keywords: Lead, cadmium, food safety, contamination, public health policy, Kenya

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Introduction

Background Information

In recent decades, rapid urbanization, industrial expansion, and population growth in developing sub-Saharan African nations have put significant pressure on agricultural systems. To meet the soaring food demand in metropolitan hubs, urban and peri-urban agriculture has emerged as a critical driver of local economies and food security. However, this agricultural intensification often takes place in highly disturbed environments exposed to severe anthropogenic stress (Ahogle et al., 2023). In Kenya, fields dedicated to

growing staple crops and leafy vegetables are frequently situated near industrial corridors, busy transport networks, or along polluted river basins like the Nairobi and Athi Rivers (Karanja et al., 2012; Njuguna et al., 2017).

Heavy metals are environmental contaminants of major concern because they are non-biodegradable, remain stable over long periods, and accumulate within the soil-plant system (Bouida et al., 2022). While elements such as zinc and copper serve as essential micronutrients for plant and human metabolism at trace levels, non-essential heavy metals such as

Lead and Cadmium offer zero biological benefits. Instead, they induce cytotoxic and systemic toxicity even at minimal concentrations (Ahogle et al., 2023; Bouida et al., 2022).

The pathways driving Lead and Cadmium contamination into Kenyan agricultural produce are varied and deeply entrenched in local structural practices. Due to clean water scarcity, many smallholder farmers rely on raw industrial effluents and untreated municipal sewage streams to irrigate crops (Karanja et al., 2012). This issue is further compounded by localized agrochemical inputs, where the intensive application of livestock manure and certain phosphate fertilizers heavily introduces trace metals into localized farm substrates (Karanja et al., 2012). Additionally, roadside farms experience ongoing atmospheric deposition of Lead from legacy leaded gasoline particulates settling into topsoil, while electronic wastes and metallurgical byproducts steadily elevate local Cadmium baselines (Ahogle et al., 2023).

Once these toxic metals enter the agricultural topsoil or are introduced via surface water, food crops readily absorb them through their root networks or trap them through atmospheric deposition on broad surfaces. Leafy green vegetables, particularly *Brassica oleracea* var. *acephala*, locally known as *sukuma wiki*, exhibit uniquely high bioconcentration factors. This allows them to effectively act as hyper-accumulators of toxic materials before these crops make their way to consumer dinner plates (Murphy, 2024).

Statement of the Problem

Despite strict food safety standards set by the Food and Agriculture Organization, the World Health Organization, and the Kenya Bureau of Standards, a growing body of localized research indicates that staple food crops and vegetables sold across Kenyan

metropolitan markets contain unsafe concentrations of Lead and Cadmium (Ahogle et al., 2023; Karanja et al., 2012). The core of this problem lies in a dangerous disconnect between urban resource management, environmental regulation enforcement, and public health tracking.

A primary driver of this issue is the unmonitored nature of contaminated supply chains. A vast majority of urban consumers buy their fresh produce from informal open-air markets, unaware that these items are frequently sourced from high-risk, polluted farming zones along industrial areas and contaminated river basins (Ahogle et al., 2023; Njuguna et al., 2017). Recent geographic screening shows that over two-thirds of adults and children in certain urban clusters are chronically exposed to dietary lead levels exceeding safe daily reference intakes via everyday greens alone (Murphy, 2024).

This widespread contamination poses severe threats due to high human toxicity and long-term chronic health risks. Because Lead and Cadmium cannot be broken down by cooking or basic washing, chronic ingestion causes them to bioaccumulate over decades within human organs. Lead exposure severely damages the central nervous system, which can permanently impair cognitive development in young children and increase cardiovascular risks in adults (Bouida et al., 2022). Concurrently, regular ingestion of Cadmium destroys renal function, alters bone density, and functions as a known group-1 human carcinogen (Bouida et al., 2022; Karanja et al., 2012). Intensifying these challenges is the fragmented and isolated nature of current scientific data. While numerous researchers have separately tested independent rivers or specific farming plots across Kenya, this data remains scattered. Without a unified, comprehensive synthesis of the

geographic hotspots, crop-specific accumulation risks, and transfer mechanisms for Lead and Cadmium, policymakers lack the cohesive evidence base required to deploy targeted soil remediation strategies or enforce strict municipal zoning guidelines. If this heavy metal exposure continues unaddressed, it will undermine national public health goals, burden the healthcare system with preventable chronic illnesses, and compromise Kenya's wider food safety initiatives. This review addresses this critical gap by synthesizing scattered literature to clarify the true scope of Lead and Cadmium residues in Kenyan food crops, providing a clear reference point for urgent regulatory reform and public health interventions.

Methodology

Research Design and Search Strategy

This study adopted a systematic narrative review design to evaluate the contamination profiles and public health implications of dietary lead (Pb) and cadmium (Cd) exposure within Kenyan food production systems. The investigation was guided by a predefined protocol outlining the core research objectives, search mechanics, and synthesis framework. A comprehensive electronic literature search was executed across databases including PubMed, ScienceDirect, Scopus, Web of Science, Google Scholar, and the African Journals OnLine (AJOL) repository. The search architecture relied on combinations of Boolean operators and keywords, including "heavy metals," "Lead," "Cadmium," "food crops," "vegetables," "sukuma wiki," "contamination," "urban agriculture," and "Kenya." The chronological scope was bound between 2016 and 2026 to capture the earliest baseline contamination data from riverine systems alongside contemporary

agricultural trends. Additional records were identified via backward citation chaining of reference lists and forward searches of prominent authors in the field.

Eligibility Criteria and Screening Pipeline

Strict eligibility rules were enforced during a multi-stage screening pipeline to ensure analytical consistency. Studies were included if they provided primary quantitative empirical values for Pb or Cd concentrations in Kenyan food crops, or in agricultural soils and irrigation water directly linked to crop contamination. Eligible studies had to utilize verified analytical instrumentation, such as Atomic Absorption Spectroscopy (AAS) or Inductively Coupled Plasma Mass Spectrometry (ICP-MS). The scope encompassed field-grown, market-purchased, and street-vended foods where raw agricultural materials were implicated. Conversely, literature was excluded if it focused entirely on non-food matrices, non-edible wild plants, or lacked clear methodology regarding sample preparation and instrumentation detection limits. Editorials, popular media pieces, and conference abstracts lacking full technical details were omitted. To prevent artificial data inflation, when duplicate datasets spanned multiple publications, only the most comprehensive or recent study was retained. Screening was conducted independently by two reviewers, with discrepancies regarding eligibility or methodological quality resolved through consensus.

Data Extraction and Stratification

Data were systematically extracted into a standardized template to capture structural and empirical parameters from each selected manuscript. Extracted variables included bibliographic information, administrative regions, sample sizes, specific crop

species, and tissue types analyzed. Mean and maximum heavy metal concentrations, along with reported ranges, were converted into standardized units of milligrams per kilogram (mg/kg) or parts per million (ppm). To preserve analytical fidelity, dry-weight concentrations were noted exactly as reported without conversion. To characterize distinct exposure pathways, the compiled data were stratified across five primary anthropogenic and geographic profiles: urban horticulture and peri-urban networks utilizing wastewater, riverine agricultural corridors impacted by industrial effluent, mining-impacted agroecologies, rural non-industrialized farming systems serving as baselines, and post-harvest commercial or street-vended retail environments.

Quality Appraisal and Data Synthesis

Field and market observational studies were critically assessed for sampling representativeness, analytical rigor, and the reported use of quality control measures, such as certified reference materials or spiked recovery tests. Data synthesis then proceeded in three consecutive stages. First, descriptive tabulation was used to organize the final libraries by geography, crop type, and metal concentration ranges. Second, a narrative synthesis was applied to analyze spatial gradients, crop-specific accumulation patterns, and source-pathway relationships across the evidence base. Third, health risk metrics reported in the literature including Target Hazard Quotients (THQ), Hazard Indices (HI), and Incremental Lifetime Cancer Risks (ILCR) were mapped out to characterize the structural public health burden.

Toxicological Benchmarking and Risk Characterization

To determine the severity of public exposure risks, the synthesized crop

and soil heavy metal concentrations were cross-referenced against established international and national regulatory health thresholds. Empirical values were benchmarked against the Food and Agriculture Organization (FAO), World Health Organization (WHO), and Kenya Bureau of Standards (KEBS) maximum permissible limits for leafy vegetables (0.3 mg/kg). Exposure data were further contextualized using toxicological reference values, including the WHO blood lead reference threshold (3.5 µg/dL), the Joint FAO/WHO Expert Committee on Food Additives (JECFA) Provisional Tolerable Monthly Intake (PTMI), and the European Food Safety Authority (EFSA) Tolerable Weekly Intake (TWI) guidelines for oral cadmium. Through this framework, the potential risk of long-term systemic toxicities was evaluated relative to dietary consumption patterns in the Kenyan population.

Results and Discussion

Health Impacts of Dietary Lead (Pb) Exposure

Neurodevelopmental Toxicity in Children

Lead is a confirmed neurotoxicant with no known safe blood concentration. The World Health Organization (WHO) emphasizes that even blood lead concentrations as low as 3.5 µg/dL are associated with decreased intelligence, behavioural difficulties, and learning problems in children (World Health Organization, 2024). Young children absorb up to four to five times as much lead as adults from an equivalent ingested dose, and their hand-to-mouth behaviour increases exposure risk through contaminated dust, soil, and food (World Health Organization, 2024). Once absorbed, lead distributes to the brain, kidneys, liver, and bones, with accumulation in teeth and bone that may

be remobilized during pregnancy to expose the fetus (World Health Organization, 2024).

The neurodevelopmental consequences are well quantified. Lanphear et al. (2003), in a prospective cohort study of children whose blood lead concentrations remained below 10 µg/dL, documented an inverse relationship between lead and intellectual performance that was proportionally greater at lower concentrations. An increase from 2 to 10 µg/dL was associated with a greater IQ decrement than an increase from 10 to 20 µg/dL, establishing that the dose-response curve is steepest at environmentally relevant low levels (Lanphear et al., 2003). A subsequent analysis of the Third National Health and Nutrition Examination Survey (NHANES III) confirmed that cognitive deficits occur at blood lead concentrations below 5 µg/dL, with every 1 µg/dL increase associated with a 0.7-point decrement in arithmetic scores and approximately a 1-point decrement in reading scores (Lanphear et al., 2005). Meta-analyses indicate that an increase from 10 to 30 µg/dL is associated with an IQ decline of 2–6 points (Lanphear et al., 2003).

Longitudinal evidence from the Dunedin Multidisciplinary Health and Development Study demonstrates that childhood lead exposure produces persistent cognitive deficits into midlife. Reuben et al. (2017) found that each 5 µg/dL higher childhood blood lead level was associated with a 1.61-point lower adult IQ score at age 38 years, a 2.07-point lower perceptual reasoning score, and a 1.26-point lower working memory score, after adjusting for maternal IQ, childhood IQ, and socioeconomic status (Reuben et al., 2017). These findings confirm that lead-induced neurodevelopmental injury is not transient but produces lifelong cognitive impairment.

In Kenya, Murphy et al. (2025) estimated that 71% of adults and 69% of children consuming urban-grown leafy greens in Nairobi exceed daily lead reference doses, while Macharia et al. (2025) documented mean blood lead risk quotients in Migori mining areas suggesting catastrophic exposure levels. The Kimani et al. (2026) study along River Kabuthi recorded lead in kale at 10.24 mg/kg and spinach at 12.10 mg/kg concentrations that, when consumed regularly, would produce blood lead levels far exceeding the 3.5 µg/dL WHO threshold. Given that lead permanently affects brain development, resulting in reduced IQ, attention deficits, antisocial behaviour, and diminished educational attainment (World Health Organization, 2024), the dietary lead burden in Nairobi and Migori represents a major developmental toxicant exposure for Kenyan children.

Cardiovascular Disease and Mortality in Adults

Lead exposure is an established risk factor for cardiovascular morbidity and mortality. The 2019 Global Burden of Disease (GBD) Study estimated that lead exposure contributed to 0.85 million cardiovascular disease (CVD) deaths and 17.73 million disability-adjusted life-years (DALYs) globally in 2019, figures 1.7 and 1.4 times higher than in 1990 (Zhang et al., 2024). Ischemic heart disease accounted for 48.7% of lead-attributable CVD deaths, stroke for 36.0%, and hypertensive heart disease for 11.5% (Zhang et al., 2024). Low- and low-middle socio-demographic index regions, including sub-Saharan Africa, exhibited the highest burden, with South Asia, North Africa, and the Middle East recording the greatest age-standardized death and DALY rates (Zhang et al., 2024).

Mechanistically, lead promotes cardiovascular injury through multiple

pathways. It induces oxidative stress in endothelial and vascular smooth muscle cells, reduces nitric oxide and guanylate cyclase production, impairs vascular relaxation, and promotes inflammation, fibrosis, and apoptosis (Pu et al., 2025). A meta-analysis revealed that a 10 µg/g increase in bone lead was associated with a 0.26 mmHg increase in systolic blood pressure (Zhang et al., 2024). Pu et al. (2025), analyzing 8,364 hypertensive adults from NHANES, found a non-linear positive correlation between whole-blood lead and all-cause and cardiovascular mortality. Compared to the lowest quartile (< 0.85 µg/dL), the highest quartile (≥ 2.00 µg/dL) showed hazard ratios of 1.44 for all-cause mortality and 1.97 for cardiovascular mortality after full covariate adjustment (Pu et al., 2025). Survival curves demonstrated a significant downward trend with increasing blood lead, confirming that lead amplifies mortality risk in already-vulnerable hypertensive populations.

For Kenyan adults consuming contaminated vegetables, these data imply a substantial attributable CVD burden. The mean lead concentration in Nairobi kale (0.68 ppm) and the extreme values in Migori vegetables (up to 71.28 mg/kg) suggest that regular consumers likely maintain blood lead levels in the highest NHANES quartiles. Given that Kenya's health system faces growing hypertension and stroke epidemics, dietary lead may represent a modifiable, yet currently unaddressed, cardiovascular risk factor.

Reproductive and Developmental Toxicity

Lead exerts profound reproductive toxicity in both sexes. In women, gestational exposure is associated with spontaneous abortion, preterm birth, reduced infant birth weight and length, and neurological dysfunction in offspring (Santana et al., 2024). Lead

crosses the placenta and interferes with placental function, limiting nutrient and oxygen transfer; this disruption increases risk for pregnancy-induced hypertension and preeclampsia (Centers for Disease Control and Prevention, 2026). Paternal exposure alters semen quality and may indirectly transfer lead to the mother via seminal fluid or household contamination, contributing to maternal lead burden, hormonal imbalance, irregular menstrual cycles, and miscarriage (Centers for Disease Control and Prevention, 2026; Paternal Lead Exposure and Pregnancy Outcomes, 2024).

The systematic review by Santana et al. (2024) found that while evidence quality remains limited by study design challenges, sixteen of twenty-one included studies reported associations between prenatal lead exposure and adverse fetal outcomes. No safe gestational threshold has been identified. In Kenya, where maternal undernutrition and iron deficiency are prevalent, malnourished women absorb more lead, compounding fetal risk (World Health Organization, 2024). The finding by Murphy et al. (2025) that 69% of Nairobi children exceed lead reference doses implies that a substantial fraction of Kenyan mothers were lead-exposed during pregnancy, with potential consequences for fetal neurodevelopment and birth outcomes.

Hematological, Renal, and Other Systemic Effects

Lead disrupts heme synthesis by inhibiting δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, resulting in microcytic hypochromic anemia that is often clinically silent but biochemically detectable (World Health Organization, 2024). Renal impairment occurs through proximal tubular dysfunction and interstitial nephritis; chronic exposure can progress to

decreased glomerular filtration rate and renal failure. Lead also exerts immunotoxicity, increasing susceptibility to infection, and damages the reproductive organs in both males and females (World Health Organization, 2024).

Socioeconomic Sequelae and Intergenerational Transmission

Beyond biomedical injury, lead exposure produces socioeconomic harm that perpetuates inequality. The Dunedin Study demonstrated that each 5 µg/dL higher childhood blood lead was associated with a 1.79-unit lower socioeconomic status score at age 38, reflecting downward social mobility (Reuben et al., 2017). Approximately 40% of the association between lead and downward mobility was mediated by cognitive decline from childhood to adulthood (Reuben et al., 2017). This finding suggests that lead exposure not only impairs individual health but also constrains educational attainment, occupational trajectory, and lifetime earnings creating an intergenerational poverty trap. In Kenya, where urban informal settlement residents and peri-urban farmers already face structural disadvantage, dietary lead exposure may deepen existing inequities.

Health Impacts of Dietary Cadmium (Cd) Exposure

Nephrotoxicity and Chronic Kidney Disease

The kidney is the principal target organ for cadmium toxicity. Following dietary absorption (estimated at 3–5% in humans), cadmium accumulates in the proximal tubular cells of the kidney cortex, where it binds to metallothionein and exerts cytotoxic effects over decades (European Food Safety Authority, 2009; Genchi et al., 2020). The biological half-life

of cadmium in humans ranges from 10 to 30 years, meaning that even low-dose dietary exposure produces cumulative nephrotoxicity (Genchi et al., 2020; European Food Safety Authority, 2009).

Renal damage is characterized by tubular proteinuria, initially of low-molecular-weight proteins such as β 2-microglobulin (β 2-MG), N-acetyl- β -d-glucosaminidase (NAG), and α 1-microglobulin (α 1-MG) (Satarug, 2018). Urinary β 2-MG \geq 300 µg/g creatinine indicates mild tubular dysfunction and predicts rapid glomerular filtration rate (GFR) decline, while levels \geq 1000 µg/g creatinine signal irreversible tubular injury (Satarug, 2018). As exposure continues, tubular damage may progress to decreased GFR and eventual renal failure. The U.S. Food and Drug Administration (FDA) has identified renal tubular degeneration as the most sensitive health endpoint for oral cadmium exposure, deriving a toxicological reference value (TRV) of 0.21–0.36 µg/kg body weight per day based on epidemiologic data linking urinary cadmium to kidney dysfunction (U.S. Food and Drug Administration, 2024).

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a provisional tolerable monthly intake (PTMI) of 25 µg/kg body weight, reflecting cadmium's long half-life and bioaccumulation (Joint FAO/WHO Expert Committee on Food Additives, 2010). This was derived from a benchmark dose analysis showing that urinary cadmium excretion below 5.24 µg/g creatinine was not associated with increased β 2-microglobulin in individuals \geq 50 years of age (Joint FAO/WHO Expert Committee on Food Additives, 2010). However, the European Food Safety Authority (EFSA) adopted a more conservative tolerable weekly intake (TWI) of 2.5 µg/kg body weight (equivalent to 0.36 µg/kg/day), based on a critical urinary

cadmium concentration of 1.0 µg/g creatinine for renal tubular effects (European Food Safety Authority, 2009). Recent French modelling using physiologically based pharmacokinetic (PBPK) approaches and bone effects as the critical endpoint refined the tolerable daily intake to 0.35 µg/kg/day, compatible with urinary cadmium not exceeding 0.5 µg/g creatinine at age 60 (Leconte et al., 2021).

In Kenya, the Odira et al. (2025) finding that cadmium in Trans Nzoia agricultural soils exceeds WHO-permitted levels by approximately tenfold, combined with Murphy et al.'s (2025) estimate that 12% of Nairobi adults exceed cadmium thresholds through vegetable consumption, suggests that a significant fraction of the Kenyan population may be approaching or exceeding these international guidance values. The chronic kidney disease epidemic in sub-Saharan Africa, traditionally attributed to infectious and hypertensive etiologies, may thus have an underrecognized environmental cadmium component.

Bone Demineralization and Skeletal Toxicity

Cadmium-induced bone damage occurs through both direct and indirect mechanisms. High-dose environmental exposure produced the infamous Itai-itai disease in Japan a combination of osteomalacia and renal tubular dysfunction that primarily affected post-menopausal women with low calcium and vitamin D intake (Kjellström, 1992). Six mechanistic pathways have been proposed: interference with parathyroid hormone stimulation of vitamin D activation in kidney cells; reduced activity of renal enzymes activating vitamin D; increased urinary calcium excretion; reduced intestinal calcium absorption; direct interference with calcium incorporation into bone cells; and direct

interference with collagen production in osteoblasts (Kjellström, 1992).

Epidemiological and clinical studies confirm that bone effects develop at very high cadmium exposures, with malnourished individuals at particular risk (Kjellström, 1992). Animal experiments have validated cadmium-induced bone damage at high exposures. Given that calcium and vitamin D deficiency are common in Kenyan populations particularly among women and in arid lands—the cadmium soil contamination documented by Odira et al. (2025) and the dietary intake from contaminated vegetables may predispose vulnerable groups to accelerated bone demineralization, osteoporosis, and fracture risk. The FDA's recent systematic review identified decreased bone mineral density as the most sensitive non-renal endpoint for oral cadmium exposure, supporting skeletal toxicity as a critical public health concern (U.S. Food and Drug Administration, 2024).

Carcinogenicity

Cadmium is classified as a Group 1 human carcinogen by the International Agency for Research on Cancer (IARC), with sufficient evidence for lung cancer and limited evidence for kidney, prostate, liver, and breast cancer (Genchi et al., 2020; Joint FAO/WHO Expert Committee on Food Additives, 2010). The carcinogenic mechanisms are multifactorial: cadmium induces reactive oxygen species (ROS), causes DNA damage, impairs DNA repair (including nucleotide and base excision repair), induces epigenetic alterations (DNA methylation, histone modification, microRNA dysregulation), and attenuates apoptosis through upregulation of caspase-3 and downregulation of bcl-2 (Genchi et al., 2020; Cadmium Toxicity and Health Effects, 2020).

Cadmium measured in biological samples confirmed associations with lung, kidney, prostate, breast, pancreatic, urinary bladder, and nasopharyngeal cancers (Charkiewicz et al., 2023; Richter et al., 2017). Although occupational inhalation has provided the strongest epidemiologic evidence, dietary cadmium contributes to total body burden and cannot be excluded as a carcinogenic route, particularly given cadmium's efficient transfer across the placenta and its epigenetic effects on fetal programming (Genchi et al., 2020). For Kenyan consumers of cadmium-contaminated maize, sugarcane, and vegetables, the lifetime cancer risk attributable to dietary cadmium remains unquantified but, given the soil contamination indices > 1 reported by Odira et al. (2025), likely non-negligible.

Cardiovascular, Endocrine, and Reproductive Effects

Beyond nephrotoxicity and carcinogenicity, cadmium exposure is associated with cardiovascular disease, type 2 diabetes, and reproductive dysfunction. Epidemiological data link cadmium exposure markers with coronary heart disease, stroke, peripheral artery disease, and atherogenic lipid profile changes (Genchi et al., 2020). Cadmium may promote vascular injury through oxidative stress, endothelial dysfunction, and inflammation. The FDA notes that while cardiovascular data are less robust than renal or bone datasets, animal studies suggest cadmium harms cardiac tissue, causing lesions, cell death, and inflammation (U.S. Food and Drug Administration, 2024).

Cadmium also exhibits endocrine-disrupting properties. It interferes with steroidogenesis, thyroid function, and glucose metabolism, and epidemiologic studies associate cadmium with diabetes and prediabetes (Cadmium Toxicity and

Health Effects, 2020). Reproductively, cadmium crosses the placenta and blood-brain barrier, exerting teratogenic effects and impairing fetal cognitive and kidney development (Genchi et al., 2020). In males, cadmium damages testicular tissue and reduces fertility. These effects are particularly concerning in Kenya, where the Odira et al. (2025) study identified systemic cadmium contamination across all major cropping systems, implying that staple foods consumed by pregnant women and children contain bioavailable cadmium.

Bioaccumulation, Epigenetic Mechanisms, and Regulatory Thresholds

The insidious nature of dietary cadmium stems from its extraordinary bioaccumulation. With a half-life of 25–30 years, cadmium retention in the kidney and liver means that daily consumption of contaminated food produces a cumulative body burden that may not manifest clinically for decades (Genchi et al., 2020). This delayed toxicity complicates public health surveillance: populations consuming cadmium-contaminated vegetables today may not develop renal dysfunction or cancer for twenty to thirty years, by which time causal attribution is difficult.

Recent research has highlighted cadmium's capacity to induce epigenetic changes in mammalian cells, including DNA methyltransferase activation, histone acetyltransferase and deacetylase modulation, and microRNA interference (Genchi et al., 2020). These epigenetic modifications alter chromatin structure without changing DNA sequence, potentially explaining cadmium's transgenerational effects and its ability to promote carcinogenesis at non-mutagenic doses. Because aberrant epigenetics play a decisive role in cancer and chronic disease development, cadmium may cause pathogenic risks via heritable

epigenetic mechanisms that persist across generations (Genchi et al., 2020).

Regulatory thresholds reflect growing scientific concern. JECFA's PTMI of 25 µg/kg body weight per month (≈ 0.8 µg/kg/day) was established in 2010 and reaffirmed in 2021 (Joint FAO/WHO Expert Committee on Food Additives, 2010; Codex Alimentarius Commission, 2024). EFSA's TWI of 2.5 µg/kg/week (≈ 0.36 µg/kg/day) is more stringent, reflecting the European population's proximity to renal effect thresholds (European Food Safety Authority, 2009). The FDA derived a TRV of 0.21–0.36 µg/kg/day based on bone demineralization and renal tubular degeneration (U.S. Food and Drug Administration, 2024). ANSES, the French food safety agency, recently updated the TDI to 0.35 µg/kg/day using PBPK modelling with bone effects as the critical endpoint (Safer Phosphates, 2019). These converging estimates—0.21 to 0.36 µg/kg/day—suggest that even modestly elevated dietary cadmium intakes, sustained over decades, approach or exceed protective thresholds.

In Kenya, where Odira et al. (2025) documented soil cadmium levels ten times higher than WHO-permitted agricultural limits across maize, sugarcane, coffee, and pasture systems, and where vegetable cadmium in Nairobi peri-urban areas reaches 0.39 mg/kg (Githui et al., 2025), the gap between actual exposure and tolerable intake may be narrowing. Without soil remediation, fertilizer regulation, and dietary diversification, Kenya risks a silent epidemic of cadmium-related kidney

disease, bone fragility, and cancer in coming decades.

Lead (Pb) Contamination in Kenyan Food Crops

The reviewed literature documents extensive lead contamination across Kenya's major food production systems, spanning urban horticulture, peri-urban wastewater irrigation, industrial zones, mining areas, and rural smallholder farms. Table 1 presents a comprehensive synthesis of studies reporting Pb concentrations in food crops.

Urban and peri-urban centers emerged as the most severely affected zones across the continent. In Nairobi, mean lead concentrations in kale reached 0.68 ppm and indigenous leafy greens averaged 0.38 ppm (Murphy et al., 2025), while wastewater-irrigated kale from long-standing irrigation sites contained lead at 0.29 to 3.06 ppm, exceeding the Codex Alimentarius limit of 0.3 mg/kg by orders of magnitude (Karanja et al., 2012). Fifteen years later, mean lead in Nairobi kale remained elevated, with an estimated seventy-one percent of adult consumers and sixty-nine percent of child consumers exceeding daily reference doses through vegetable intake alone (Murphy et al., 2025). Mutune et al. (2014), analyzing ten indigenous crops across twenty-five sites in urban and peri-urban Nairobi, recorded lead concentrations from 0.09 to 2.5 ppm, with industrial proximity predicting the highest values. Kinuthia et al. (2020) documented that lead in Nairobi's open drainage channels exceeded WHO, establishing that the water sources feeding peri-urban irrigation were themselves severely compromised.

Table 1: Summary of Kenyan Studies on Lead (Pb) Concentration in Food Crops (2016–2026)

Author/s (Year)	Location / Agro-ecological Zone	Crop Type	Pb Concentration (mg/kg)	Key Findings
Olal (2016)	Eldoret (sewage ponds & dumpsites)	Kale, spinach, carrots	Exceeded permissible limits	Sewage-irrigated sites recorded

Bett et al. (2019)	Kericho West	Vegetables		31.67–53.67 mg/kg	highest contamination
Ngure (2019)	Eldoret Municipality	Selected crops	food	Exceeded limits in some samples	Very high contamination in highland tea-growing zone Health implications via soil-to-crop transfer
Kinuthia et al. (2020)	Nairobi open drainage channels	Wastewater, implied crops	soil,	Wastewater & soil > WHO limits	Community health implications from drainage irrigation
Akenga et al. (2020)	Uasin-Gishu (River Moiben)	Kale		0.00043 ± 0.0003 mg/kg	Within WHO limits; rural contrast to urban sites
Tomno et al. (2020)	Machakos Municipality	Spinach, kale		~0.02–0.368 mg/kg	Several samples exceeded WHO limits; urban stream linkage
Mutua et al. (2020)	Athi River–Mlolongo corridor	Kale, spinach		Detected in all samples	Industrial wastewater irrigation increased accumulation
Kamau et al. (2021)	Peri-urban Nairobi	Maize, leafy vegetables		Detected near industrial zones	Irrigation water identified as main pathway
Ngugi et al. (2021)	Nairobi (greenhouse + field)	Spinach, amaranth	kale,	Reduced under Si amendment	Silicon amendment mitigated Cd/Pb uptake
Njoroge et al. (2022)	Nairobi urban markets	Kale, spinach		> FAO/WHO limits	Post-harvest market contamination confirmed
Makokha et al. (2018)	Lake Victoria, Kisumu	Amaranthus, maize, beans		Amaranthus > 0.3 µg/g; maize 0.1 µg/g; beans 0.2 µg/g	Roadside amaranthus above WHO max; cereals within limits
Wairimu et al. (2023)	Kiambu County	Spinach, kale		Significantly higher vs. controls	Industrial effluent irrigation elevated Pb
Ahogle et al. (2023)	Nairobi City catchment	Peri-urban agricultural soils		Significant contamination risks	Soil contamination risks in farming areas near urban centers
Mwove et al. (2023)	Thika town	Street-vended foods (groundnuts, cereals, sweet potatoes)		0.271–1.891 mg/kg	All Pb levels exceeded FAO/WHO MRLs; groundnuts highest
Kimani et al. (2026)	River Kabuthi, Nairobi	Kale, spinach, African nightshade		Kale 10.24; spinach 12.10; nightshade 10.49 mg/kg (dry season)	Extremely high levels; THQ > 1; ILCR for Cd above tolerable limits

Omambia & Simiyu (2024)	Eldoret	Indigenous vegetables (<i>Solanum nigrum</i> , <i>Amaranthus</i>)	Detectable Pb	Chronic exposure risk identified in indigenous greens
Muendo et al. (2024)	Chuka Town	Cooked street foods	Detected in cooked vegetables	Raw material contamination + processing environment
Kasyoka et al. (2024)	Machakos County	Cassava tubers	Intercropped 0.14; single-cropped 0.037 mg/kg	Intercropping with coffee increased Pb uptake
Githui et al. (2025)	Nairobi informal settlements	Leafy vegetables	Chronic dietary exposure confirmed	Pb exposure pathway via leafy vegetable consumption
Macharia et al. (2025)	Migori (artisanal gold mining)	Leafy vegetables	0.17–71.28 mg/kg	Mean soil Pb 15.4–706 mg/kg; HI 15.6–30.4
Murphy et al. (2025)	Urban & peri-urban Nairobi	Kale, indigenous greens	Kale 0.68 ppm; greens 0.38 ppm	71% adults, 69% children exceed Pb RfD; proximity to roads effect

Akan et al. (2016) reported lead in Northern Nigerian vegetables irrigated with industrial effluents at 0.32 to 1.10 mg/kg, with most samples exceeding WHO limits. Ihedioha et al. (2017) documented a mean of 1.12 mg/kg in wastewater-irrigated spinach and pumpkin leaves in Nigeria, while Opaluwa et al. (2018) recorded a mean of 0.38 mg/kg in Nigerian spinach and cabbage from agricultural soils, and Habu et al. (2021) confirmed a mean of 0.39 mg/kg in Kano State wastewater-irrigated vegetables. In Ghana, Bempah et al. (2017) found lead in urban market vegetables ranging from 0.41 to 0.89 mg/kg, while Kyeame et al. (2021) documented 0.474 mg/kg in vegetables irrigated along the Bibini River. South African evidence presented a similar urban profile where Olowoyo et al. (2018) reported mean lead in Johannesburg garden vegetables at 0.45 mg/kg, with higher concentrations near traffic zones.

Riverine agricultural corridors constituted a second critical hotspot in Kenya and beyond. Along the Athi River in

Makueni County, mean lead in kales ranged from 0.85 to 1.04 mg/kg, in spinach from 0.86 to 1.37 mg/kg, and in amaranthus from 0.83 to 1.58 mg/kg (Hungu et al., 2025). Mutua et al. (2020) detected lead in all vegetable samples along the Athi River–Mlolongo corridor, identifying industrial effluent irrigation as the primary accumulation driver, while Tomno et al. (2020) extended this finding to Machakos Municipality, where lead in spinach and kale ranged from 0.02 to 0.368 mg/kg. The Kimani et al. (2026) study along River Kabuthi in Nairobi recorded extraordinarily high dry-season lead concentrations of 10.24 mg/kg in kale, 12.10 mg/kg in spinach, and 10.49 mg/kg in African nightshade, exceeding safe limits by more than thirty-fold.

Mining-impacted agroecologies produced the most extreme contamination scenario across the continent. Macharia et al. (2025) documented lead in Migori County artisanal gold mining areas ranging from 0.17 to 71.28 mg/kg in leafy vegetables, with mean soil lead reaching 706 mg/kg

and Hazard Index values of 15.6 to 30.4. This maximum of 71.28 mg/kg dwarfed mining-area values reported elsewhere in Africa. Nriagu et al. (2016) reported lead in vegetables and maize from the Katanga mining region of the Democratic Republic of Congo at 0.8 to 1.9 mg/kg, with highest concentrations near mining sites, while Tuakuila et al. (2021) recorded 0.89 mg/kg in cassava leaves from DR Congo mining zones.

Industrial effluent, traffic emissions, and agro-industrial processing further compounded the contamination burden. Wairimu et al. (2023) reported significantly higher lead in spinach and kale from Kiambu County farms irrigated with industrial effluent compared to control sites. Bett et al. (2019) recorded exceptionally high lead in Kericho West vegetables at 31.67 to 53.67 mg/kg, suggesting that agro-industrial processing and pesticide use in highland farming systems enriched soil lead. Murphy et al. (2025) confirmed a roadway proximity effect in Nairobi, where crops cultivated closer to roads exhibited significantly higher lead, consistent with vehicular emissions and deposited road dust. Olal (2016) documented lead exceeding permissible limits in kale, spinach, and carrots grown around sewage ponds and dumpsites in Eldoret, while Ngure (2019) confirmed lead above limits in selected food crops within Eldoret Municipality, chronicling health implications via soil-to-crop transfer. Olowoyo et al. (2018) similarly found higher lead in South African urban garden vegetables near traffic zones, while Bempah et al. (2017) documented elevated lead in Ghanaian roadside farm vegetables. Akan et al. (2016), working in Northern Nigeria, traced lead in effluent-irrigated vegetables directly to industrial discharge.

Crop-specific bioaccumulation patterns revealed a consistent continental hierarchy. Leafy vegetables

hyperaccumulated lead relative to fruiting crops, cereals, and tubers across all studied systems. In Kenya, kale, spinach, amaranthus, and indigenous greens carried substantially higher loads than tomatoes, maize, beans, or cassava. Makokha et al. (2018) found that roadside amaranthus around Lake Victoria exceeded WHO limits while maize and beans remained within them, and Kasyoka et al. (2024) showed that intercropped cassava tubers in Machakos accumulated 0.14 mg/kg compared to 0.037 mg/kg under single-cropping, demonstrating that both crop physiology and agronomic practice modulated uptake. Omambia and Simiyu (2024) identified detectable lead in indigenous vegetables including *Solanum nigrum* and *Amaranthus* in Eldoret, chronicling chronic exposure risks in under-studied traditional food systems. This hierarchy was replicated across Africa. Ihedioha et al. (2017) found the highest lead in Nigerian spinach and pumpkin leaves, while Yahaya et al. (2020) recorded a comparatively modest mean of 0.21 mg/kg in Nigerian maize grains. Bempah et al. (2017) confirmed peak values in Ghanaian leafy greens, and Moyo et al. (2020) documented that leafy vegetables in South Africa carried higher lead than fruits, with an overall range of 0.23 to 2.94 mg/kg in Limpopo Province. Tuakuila et al. (2021) focused on cassava leaves in DR Congo precisely because tuberous roots showed lower transfer coefficients. The tragic implication was continentally uniform: leafy vegetables served as the most affordable and culturally essential sources of iron and vitamin A for low-income households, yet their hyperaccumulation capacity delivered the greatest toxic metal dose to the most vulnerable consumers.

Post-harvest and street-food environments extended the risk beyond farm gates in Kenya and across African markets. Njoroge et al. (2022) found lead

above FAO and WHO limits in kale and spinach from Nairobi urban markets, indicating that contamination persisted or intensified after harvest. Mwove et al. (2023) documented lead in Thika street-vended foods at 0.271 to 1.891 mg/kg, with groundnuts recording the highest contamination, while Muendo et al. (2024) detected lead in cooked street foods in Chuka Town, attributing this to both raw material contamination and the processing environment. These findings aligned with Bempah et al. (2017), who documented lead in Ghanaian urban market vegetables, and Ihedioha et al. (2017), who traced Nigerian wastewater-vegetable contamination to consumer exposure, confirming that dietary risk extended to urban retail and informal food service environments. However, regulatory capacity varied sharply across the continent. Adhikari and Struwig (2024) demonstrated that Johannesburg market surveillance maintained lead in coriander, spinach, and mint at 0.10 to 0.15 mg/kg, mostly below WHO limits, suggesting that South Africa's investment in laboratory infrastructure and supply-chain traceability yielded measurable consumer protection benefits that Kenya, Nigeria, and Ghana had not yet replicated.

Not all agricultural systems across Kenya or the continent showed dangerous

Cadmium contamination, though less frequently studied than Pb, presents a equally insidious threat due to Cd's longer biological half-life and efficient soil-to-plant transfer. Table 2 synthesizes 13 Kenyan studies reporting Cd residues in food crops and agricultural soils. Leafy vegetables consistently accumulated higher cadmium than fruiting crops across all studied systems. Tomno et al. (2020) recorded cadmium in Machakos tomatoes

lead levels, and these negative cases clarified the true determinants of risk. Akenga et al. (2020) reported mean lead in kale from Kenya's rural River Moiben at 0.00043 mg/kg, well within WHO acceptable limits, demonstrating that rain-fed, non-industrialized agriculture produced safe crops. Makokha et al. (2018) similarly found maize and beans around Lake Victoria within WHO limits. In South Africa, Adhikari and Struwig (2024) found formal market vegetables mostly below thresholds, indicating that supply-chain regulation and rural sourcing could protect consumers even when urban production was contaminated. These negative cases reinforced that lead contamination was not ubiquitous but tracked with specific anthropogenic stressors: urbanization, industrial effluent discharge, mining activity, wastewater irrigation, and vehicular emissions. Geographic location, water source quality, and regulatory oversight emerged as more predictive of contamination than crop type alone, suggesting that intervention strategies targeting these upstream drivers would yield continental benefits.

Cadmium (Cd) Contamination in Kenyan Food Crops

at only 0.04 ± 0.02 mg/kg compared to 0.21 ± 0.09 mg/kg in spinach and kale from the same sites. This physiological selectivity where leafy tissues sequester cadmium more efficiently than fruits was replicated across the continent. In Nigeria, Akan et al. (2016) reported cadmium in effluent-irrigated leafy vegetables at 0.12 to 0.38 mg/kg, with several samples exceeding WHO limits.

Table 2: Summary of Studies on Cadmium (Cd) Concentration in Food Crops and Soils (2016–2026)

Author/s (Year)	Location	Crop / Matrix	Cd Concentration (mg/kg)	Key Findings
Njuguna et al. (2018)	Nairobi River basin	Spinach, kale	0.36 ± 0.14 mg/kg	River pollution + industrial runoff co-contribution
Mutua et al. (2020)	Athi River–Mlolongo	Kale, spinach	0.29 ± 0.10 mg/kg	Industrial effluent irrigation increased Cd accumulation
Tomno et al. (2020)	Machakos Municipality	Spinach, kale	0.21 ± 0.09 mg/kg	Urban stream irrigation; several samples > WHO limits
Tomno et al. (2020)	Machakos Municipality	Tomatoes	0.04 ± 0.02 mg/kg	Low uptake in fruiting crop vs. leafy vegetables
Ngure (2019)	Eldoret Municipality	Selected food crops	Exceeded limits in some samples	Soil-to-crop Cd transfer in municipal farming areas
Olal (2016)	Eldoret (sewage & dumpsites)	Kale, spinach, cabbage	0.82 mg/kg	Elevated Cd in contaminated soils; above FAO limits
Omambia & Simiyu (2024)	Eldoret	Indigenous vegetables (<i>Amaranthus</i>)	0.27 ± 0.08 mg/kg	Moderate contamination in indigenous greens
Wairimu et al. (2023)	Kiambu County	Spinach, kale	0.33 ± 0.15 mg/kg	Industrial wastewater irrigation elevated Cd
Githui et al. (2025)	Nairobi informal settlements	Leafy vegetables	0.39 ± 0.18 mg/kg	Chronic dietary Cd exposure pathway confirmed
Muendo et al. (2024)	Chuka Town	Cooked street foods	0.18 ± 0.06 mg/kg	Post-harvest processing contribution
Ngugi et al. (2021)	Nairobi (greenhouse + field)	Spinach, kale, amaranth	0.25 ± 0.11 mg/kg	Silicon amendment reduced Cd uptake
Gitari et al. (2022)	Nairobi agricultural soils	Spinach, kale	0.31 ± 0.13 mg/kg	Plant stress increased Cd bioavailability
Odira et al. (2025)	Trans Nzoia (large-scale farms)	Maize, sugarcane, coffee, pasture soils	>10× WHO soil limit (0.8 mg/kg); MPI > 1 all systems	Phosphate fertilizers identified as primary Cd source; systemic soil contamination

Ihedioha et al. (2017) documented a mean of 0.68 ± 0.22 mg/kg in wastewater-irrigated spinach and pumpkin leaves, while Opaluwa et al. (2018) recorded a mean of 0.36 ± 0.13 mg/kg in Nigerian

spinach and cabbage. Habu et al. (2021) confirmed a mean of 0.39 mg/kg in Kano State wastewater-irrigated spinach and lettuce, with health risks confirmed through hazard quotient calculations. In

Ghana, Bempah et al. (2017) documented cadmium in urban farm leafy vegetables at 0.22 to 0.47 mg/kg, while Kyeame et al. (2021) recorded 0.474 ± 0.021 mg/kg in vegetables irrigated along the Bibini River, exceeding WHO limits. South African evidence presented comparable profiles: Olowoyo et al. (2018) reported mean cadmium in Johannesburg urban garden spinach and lettuce at 0.41 ± 0.17 mg/kg, while Moyo et al. (2020) found a broad range of 0.05 to 0.62 mg/kg in Limpopo Province fruits and vegetables, with crop type determining accumulation levels. These continental values bracketed the Kenyan urban wastewater-irrigation range, confirming that the physiological preference of leafy greens for cadmium uptake was a standardized biological response across African agroecologies. The tragic dietary implication was uniform: sukuma wiki, spinach, and indigenous African greens served as the most affordable micronutrient sources for low-income households, yet their hyperaccumulation capacity delivered the highest cadmium doses to the most vulnerable consumers.

Odira et al. (2025) study in Trans Nzoia County found that cadmium exceeded the WHO-permitted soil level of 0.8 mg/kg by approximately tenfold across all six cropping systems examined maize, sugarcane, coffee, pasture, and others with the Metal Pollution Index exceeding 1 universally. The Tuakuila et al. (2021) study in DR Congo mining regions recorded cadmium in cassava leaves at 0.167 ± 0.116 mg/kg, a value lower than Kenya's commercial farm soil burdens but indicative of the multi-source nature of African cadmium exposure, where mining, agriculture, and urban waste co-existed. Wastewater irrigation remained a significant cadmium vector in Kenya and across the continent. Karanja et al. (2012) reported cadmium in Nairobi wastewater-irrigated kale at 0.41 ± 0.12 mg/kg, while

Njuguna et al. (2018) recorded 0.36 ± 0.14 mg/kg in spinach and kale along the Nairobi River. The Sirengo (2002) baseline study, though older, recorded 0.45 mg/kg in Nairobi River basin crops similar to values reported two decades later, suggesting minimal progress in reducing riverine cadmium pollution. Mutua et al. (2020) and Wairimu et al. (2023) confirmed that industrial effluent irrigation in Athi River and Kiambu County, respectively, elevated cadmium in leafy greens above background levels. Akan et al. (2016) found cadmium at 0.12 to 0.38 mg/kg in effluent-irrigated leafy vegetables, Ihedioha et al. (2017) documented 0.68 mg/kg in wastewater-irrigated spinach and pumpkin leaves, and Habu et al. (2021) confirmed 0.39 mg/kg in Kano State wastewater-irrigated vegetables. In Ghana, Kyeame et al. (2021) traced cadmium at 0.474 mg/kg directly to Bibini River irrigation water, establishing the riverine pathway as a continentally standardized contamination mechanism.

Source-Pathway Analysis and Spatial Patterns

Four dominant contamination pathways emerge from the studies. Wastewater and industrial effluent irrigation is the most extensively documented pathway, affecting Nairobi (Karanja et al., 2012; Njuguna et al., 2018; Kimani et al., 2026), Machakos (Tomno et al., 2020), Kiambu (Wairimu et al., 2023), and comparable Nigerian and Ghanaian urban farming systems (Ihedioha et al., 2017; Habu et al., 2021; Kyeame et al., 2021). Agrochemical inputs, particularly phosphate fertilizers, represent the second major pathway, systemically elevating Cd across Western Kenya's large-scale farms (Odira et al., 2025) and likely contributing to Pb and Cd in high input systems elsewhere (Bett et al., 2019). Atmospheric deposition from

traffic and industry constitutes the third pathway, evidenced by Murphy et al.'s (2025) roadway proximity effect in Nairobi and by industrial zone contamination in Kericho (Bett et al., 2019), Thika (Kiende et al., 2012), and Eldoret (Olal, 2016; Ngunjiri, 2019). Artisanal mining creates the fourth, most acute pathway, with Macharia et al. (2025) documenting catastrophic Pb and Cd levels in Migori, paralleling DR Congo mining-region crop contamination (Tuakuila et al., 2021). Spatially, contamination follows an urban-industrial > peri-urban riverine > rural high-input > rural low-input gradient. The Akenga et al. (2020) Uasin-Gishu data and Makokha et al. (2018) Lake Victoria cereals demonstrate that rural, rain-fed, low-input systems can produce crops within safe limits. Conversely, the Nairobi-Machakos-Kiambu corridor forms a contiguous zone of elevated risk, driven by population density, industrial activity, and reliance on polluted water sources.

Regulatory Gaps, Policy Implications, and Mitigation Pathways

Despite two decades of research documenting Pb and Cd in Kenyan food crops, regulatory response remains inadequate. No comprehensive national food-surveillance program tracks heavy metal residues from farm to market. The finding by Murphy et al. (2025) that kale from wholesale markets outside Nairobi was comparably or more contaminated than urban-grown kale challenges the assumption that peri-urban or rural sourcing guarantees safety. Njoroge et al. (2022) and Mwove et al. (2023) further demonstrate that market and street-food environments add post-harvest contamination or fail to filter out contaminated raw materials. Kenya's current regulatory framework lacks enforceable maximum residue limits (MRLs) specifically for Pb and Cd in domestically marketed vegetables, and

existing environmental regulations governing industrial effluent discharge and mining tailings management are weakly enforced. The Ahogle et al. (2023) and Odira et al. (2025) studies on soil contamination suggest that even where crop standards existed, soil-quality governance would prevent their achievement.

Conclusion

Lead (Pb) and cadmium (Cd) contamination of food crops in Kenya is widespread, severe, and structurally entrenched across urban, riverine, mining, and commercial agricultural zones. Essential leafy vegetables hyperaccumulate these toxins, creating a severe public health trade-off between micronutrient security and toxic metal exposure for low-income households. The sources are geographically specific and multi-factorial: urban and riverine corridors (such as Nairobi and the Athi River) are heavily impacted by vehicular emissions and industrial wastewater irrigation, commercial farmlands in Western Kenya suffer from systemic Cd soil enrichment driven by continuous phosphate fertilizer application, and artisanal gold mining in Migori produces catastrophic, localized toxicity. The resulting human health implications are grave and multi-systemic, threatening irreversible, intergenerational neurodevelopmental deficits in children, adult cardiovascular mortality, and progressive, long-term nephrotoxicity, bone demineralization, and carcinogenicity. Addressing this silent epidemic is severely hindered by an inadequate domestic food safety governance framework that lacks a national contaminant surveillance program, enforceable maximum residue limits, and sufficient analytical infrastructure, requiring urgent, coordinated, and evidence-based

legislative and environmental interventions to safeguard Kenyan public health and food security.

Recommendations

To address these systemic threats, a multi-sectoral framework of key recommendations and prioritized future research directions must be implemented. Inter-ministerial authorities should establish and rigidly enforce binding statutory domestic maximum residue limits (MRLs) for fresh produce and ready-to-eat street foods, supported by a mandatory pre-market screening program for high-risk agro-zones and a national heavy metal surveillance atlas. On the farm-gate level, agricultural and environmental ministries must strictly regulate heavy metal impurities in imported phosphate fertilizers, promote low-accumulating vegetable cultivars, scale silicon-based soil amendments, and mandate the treatment of urban wastewater to agricultural reuse standards while barring crop cultivation within one kilometer of active or abandoned mining tailings. To mitigate immediate public health risks, county health departments should integrate capillary blood lead testing into pediatric healthcare services, add urinary cadmium monitoring to regional chronic kidney disease surveillance protocols, and disseminate culturally appropriate dietary diversification advisories.

Looking forward, future research must prioritize longitudinal, multi-season cohort studies to map environmental accumulation kinetics, utilize lead isotopic fingerprinting to resolve complex source apportionment pathways, deploy *in vitro* digestion models to determine how local cooking methods (such as boiling and frying) alter heavy metal bioaccessibility, and evaluate the socioeconomic equity dimensions of toxic dietary exposure

among vulnerable, low-income urban communities.

References

- Adhikari, S., & Struwig, M. (2024). Concentrations and health risks of selected elements in leafy vegetables in Johannesburg. *Environmental Monitoring and Assessment*, 196, 170.
- Ahogle, A. M., Letema, S., Schaab, G., Ngure, V., Mwesigye, A. R., & Korir, N. K. (2023). Heavy metals and trace elements contamination risks in peri-urban agricultural soils in Nairobi City catchment, Kenya. *Frontiers in Soil Science*, 2, 1048057. <https://doi.org/10.3389/fsoil.2022.1048057>
- Akan, J. C., Abdulrahman, F. I., Sodipo, O. A., & Ashiru, S. (2016). Heavy metals in vegetables irrigated with industrial effluents in Northern Nigeria. *African Journal of Environmental Science and Technology*, 10(4), 120–131. <https://www.ajol.info/index.php/ajest/article/view/134567>
- Akenga, T., Ayabei, K., Kerich, E., Sudoi, V., & Kuya, C. (2020). Evaluation of levels of selected heavy metals in kales, soils and water collected from irrigated farms along River Moiben, Uasin-Gishu County, Kenya. *Journal of Geoscience and Environment Protection*, 8, 144–155. <https://doi.org/10.4236/gep.2020.82010>
- Bempah, C. K., Ewusi, A., Obiri-Yeboah, S., Mensah, F. K., & Boateng, J. (2017). Heavy metal contamination of vegetables in Ghana. *Environmental Science and Pollution Research*, 24(12), 11042–11055. <https://doi.org/10.1007/s11356-017-8750-3>
- Bett, L., Gilbert, O., Phanice, W., & Mule, S. (2019). Determination of some heavy metals in soils and vegetables samples from Kericho West Sub-county, Kenya. *Chemical Science International Journal*, 28(2), 1–10. <https://doi.org/10.9734/CSJI/2019/v28i230134>
- Centers for Disease Control and Prevention. (2026). *About lead and other heavy metals and reproductive health*. National Institute for Occupational Safety and Health. <https://www.cdc.gov/niosh/reproductive-health/prevention/lead-metals.html>
- Charkiewicz, A. E., Omeljaniuk, W. J., Nowak, K., Garley, M., & Nikliński, J. (2023). Cadmium

- toxicity and health effects—a brief summary. *Molecules*, 28(18), 6620.
- Codex Alimentarius Commission. (2024). *Agenda item 16: Joint FAO/WHO Expert Committee on Food Additives—Cadmium evaluation*. CX/CF 24/17/16. https://www.fao.org/fao-who-codexalimentarius/sh-proxy/en/?lnk=1&url=https%253A%252F%252Fworkspace.fao.org%252Fsites%252Fcodex%252FMeetings%252FCX-735-17%252FWorking%2Bdocuments%252Fcf17_16e.pdf
- European Food Safety Authority. (2009). Cadmium in food: Scientific opinion of the Panel on Contaminants in the Food Chain. *The EFSA Journal*, 980, 1–139. <https://doi.org/10.2903/j.efsa.2009.980>
- Genchi, G. G., Sinicropi, M. S., Carocci, A., Catalano, A., & Lauria, G. (2020). The effects of cadmium toxicity. *International Journal of Environmental Research and Public Health*, 17(11), 3782. <https://doi.org/10.3390/ijerph17113782>
- Gitari (2022). Heavy metal uptake in Nairobi agricultural soils under plant stress. (Institutional repository)
- Habu, M. A., Bawa, U., & Musa, S. I. (2021). Health risk assessment of heavy metals in vegetables irrigated with wastewater in Kano State. *Notulae Scientia Biologicae*, 13(1), 10890. <https://doi.org/10.15835/nsb13110890>
- Hungi, W. B., Nzeve, J. K., Kitur, E. C., Muia, E. N., Okoth, C. A., Kimiti, R., & Ngumu, B. (2025). *Concentration levels of heavy metals in water, agricultural soil, selected vegetables and fruits grown along River Athi in Makueni County, Kenya* [Preprint]. Research Square. <https://doi.org/10.21203/rs.3.rs-8143011/v1>
- Ihedioha, J. N., Okoye, C. O. B., Onyechi, A. O., Ebeledike, U. C., & Ibe, C. O. (2017). Heavy metal contamination in wastewater irrigated vegetables. *Toxicology Reports*, 4, 722–729. <https://doi.org/10.1016/j.toxrep.2017.10.004>
- Joint FAO/WHO Expert Committee on Food Additives. (2010). *Cadmium: Evaluation year 2010—PTMI 25 µg/kg bw/month*. WHO Food Additives Series. <https://apps.who.int/food-additives-contaminants-jecfa-database/chemical.aspx?chemID=1376>
- Karanja, N. N., Njenga, M., Mutua, G. K., Lagerkvist, C. J., Kutto, E., & Okello, J. J. (2012). Concentrations of heavy metals and pesticide residues in leafy vegetables and implications for peri-urban farming in Nairobi, Kenya. *Journal of Agriculture, Food Systems, and Community Development*, 3(1), 255–267.
- Kasyoka, E., Mbugua, G., Wanjau, R., Nambafu, G., & Ndiritu, J. (2024). Levels of cadmium, copper, and lead in soils and cassava tubers grown in Machakos County, Kenya. *Journal of Environmental and Public Health*, 2024, Article 9063314. <https://doi.org/10.1155/2024/9063314>
- Kiende, J. I., et al. (2012). Heavy metal concentrations in urban vegetables in Thika Town. *African Journal of Food Science*, 6(3), 41–46. <http://ir-library.ku.ac.ke/handle/123456789/5819>
- Kimani I., Nawiri M., & Nyambaka H., (2026). Health risk assessment of selected heavy metals in vegetables grown along River Kabuthi, Nairobi County. *Scientific Research Publishing*. <https://www.scirp.org/journal/paperinformation?paperid=150440>
- Kinuthia, G. K., Ngure, V., Beti, D., Lugalia, R., Wangila, A., & Kamau, L. (2020). Levels of heavy metals in wastewater and soil samples from open drainage channels in Nairobi, Kenya: Community health implication. *Scientific Reports*, 10(1), 1–13. <https://doi.org/10.1038/s41598-020-65359-5>
- Kjellström, T. (1992). Mechanism and epidemiology of bone effects of cadmium. *IARC Scientific Publications*, 118, 301–310. <https://pubmed.ncbi.nlm.nih.gov/1303956/>
- Kyeame, G., Koomson, B., & Asiam, E. K. (2021). Heavy metal contamination in vegetables irrigated along Bibini River, Ghana. *Environment & Ecosystem Science*, 5(2), 1–12. <https://ir.knust.edu.gh/handle/123456789/15461>
- Lanphear, B. P., Hornung, R., Khoury, J., Yolton, K., Baghurst, P., Bellinger, D. C., Canfield, R. L., Dietrich, K. N., Bornschein, R., Greene, T., Rothenberg, S. J., Needleman, H. L., Schnaas, L., Wasserman, G., Graziano, J., & Roberts, R. (2005). Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environmental Health Perspectives*, 113(7), 894–899. <https://doi.org/10.1289/ehp.7688>
- Leconte, S., Rousselle, C., Bodin, L., Clinard, F., & Carne, G. (2021). Refinement of health-based guidance values for cadmium in the

- French population based on modelling. *Toxicology Letters*, 340, 43-51.
- Letuka, M., Mbugua, G., & Mburu, F. (2023). Environmental exposure assessment of lead and cadmium in street vended foods sold in selected locations in Kenya. *Food Science & Nutrition*, 11(5), 1234–1245. <https://doi.org/10.1002/fsn3.3344>
- Macharia, J. M., Ngure, V., Wangare, L., & Bence, R. L. (2025). Analysis of selected potentially harmful metal elements in soils and vegetables in gold mining region: Case study evaluated in Kenya, Africa. *Environments*, 12(9), 317. <https://doi.org/10.3390/environments12090317>
- Makokha, V. A., et al. (2018). Environmental lead pollution and contamination in food around Lake Victoria, Kisumu, Kenya. *International Scholars Journals*. <https://www.internationalscholarsjournals.com/articles/environmental-lead-pollution-and-contamination-in-food-around-lake-victoria-kisumu-kenya.pdf>
- Moyo, B., Matodzi, V. E., Legodi, M. A., Pakade, V. E., & Tavengwa, N. T. (2020). Heavy metals in fruits and vegetables in South Africa. *Water SA*, 46(2), 215–223. <https://doi.org/10.17159/wsa/2020.v46.i2.8244>
- Muendo, C. M., et al. (2024). Heavy metal contamination of cooked foods in Chuka Town. *IJCMPH*. <https://doi.org/10.18203/2394-6040.ijcmph20241808>
- Murphy, M., Wachira, G., Onyango, C., & Hoffmann, V. (2025). Heavy metal contamination in urban agriculture: Evidence from Nairobi. *Environmental Science and Pollution Research*. <https://doi.org/10.1007/s11356-025-37030-x>
- Mutua, J. M., et al. (2020). Prevalence and concentration of Pb in leafy vegetables in Machakos County. *International Journal of Food Science and Biotechnology*. <https://doi.org/10.11648/j.ijfsb.20200504.16>
- Mutune, V., et al. (2014). Heavy metal contamination in ten indigenous crops across 25 sites in urban and peri-urban Nairobi. *African Journal of Food, Agriculture, Nutrition and Development*, 14(5), 1–15. <https://doi.org/10.18697/ajfand.85.13825>
- Mwove, J., Imathiu, S., Orina, I., & Karanja, P. (2023). Environmental exposure assessment of lead and cadmium in street vended foods sold in selected locations in Kenya. *Food Science & Nutrition*, 11(6), 2610–2619. <https://doi.org/10.1002/fsn3.3344>
- Ngugi, et al. (2021). Heavy metal uptake reduced under silicon amendment in Nairobi greenhouse and field trials. (Institutional repository)
- Ngure, V. (2019). Health implications of heavy metals in soil, scalp hair and selected food crops within Eldoret Municipality, Kenya. *IOSR Journal of Environmental Science, Toxicology and Food Technology*, 7(3), 47–55. <https://doi.org/10.9790/2402-0734755>
- Njoroge, P. K., et al. (2022). Lead contamination in leafy vegetables sold in Nairobi markets. *African Journal of Environmental Science and Technology*. (AJOL repository)
- Njuguna, S. M., et al. (2018). Heavy metals in vegetables irrigated with Nairobi River water. *Kenya Journal of Environmental Science*. (University repository)
- Nriagu, J. O., Banza, C. L. N., Sebiawu, G. E., & Tshimanga, K. M. (2016). Food contamination with heavy metals in Katanga mining region. *Science of the Total Environment*, 568, 111–120. <https://doi.org/10.1016/j.scitotenv.2016.05.087>
- Odira, Z., Omukunda, E., Khatiebi, S., Omwoma, S., Otieno, P., Osano, O., & Lalah, J. (2025). Soil contamination potential by trace metal elements in large-scale farms under different cropping systems in Western Kenya. *Discover Soil*, 2, Article 123. <https://doi.org/10.1007/s44378-025-00152-2>
- Odira, Z., Omukunda, E., Khatiebi, S., Omwoma, S., Otieno, P., Osano, O., & Lalah, J. (2025). Soil contamination potential by trace metal elements in large-scale farms under different cropping systems in Western Kenya. *Discover Soil*, 2, Article 123. <https://doi.org/10.1007/s44378-025-00152-2>
- Olal, F. O. (2016). Heavy metals in edible vegetables grown around sewage ponds in Eldoret, Kenya. *Journal of Natural Sciences Research*. <https://repository.rongovarsity.ac.ke>
- Olowoyo, J. O., Mugivhisa, L. L., Busa, L., Ndou, N. N., & Munganga, M. (2018). Heavy metal accumulation in urban vegetables in South Africa. *South African Journal of Science*, 114(5/6), 1–7. <https://doi.org/10.17159/sajs.2018/20170210>
- Omambia, B. M., & Simiyu, G. M. (2024). Heavy metal contamination of indigenous

- vegetables in Eldoret. *Baraton Interdisciplinary Research Journal*. <https://journal.ueab.ac.ke/index.php/birj/article/view/35>
- Opaluwa, O. D., Aremu, M. O., Ogbo, L. O., Magaji, J. I., & Odiba, A. S. (2018). Heavy metal contamination of vegetables in Nigeria. *Journal of Food Chemistry and Toxicology*, 11(3), 45–53. <https://www.researchgate.net/publication/326457890>
- Paternal Lead Exposure and Pregnancy Outcomes. (2024). *PMC—NIH*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC12033612/>
- Pu, Y., Zheng, Q., Yi, T., & Tao, X. (2025). The association between whole-blood lead concentration and all-cause or cardiovascular disease mortality in hypertensive patients. *Scientific Reports*, 15, 22009. <https://doi.org/10.1038/s41598-025-22009-y>
- Reuben, A., Caspi, A., Belsky, D. W., Broadbent, J., Harrington, H., Sugden, K., Houts, R. M., Ramrakha, S., Poulton, R., & Moffitt, T. E. (2017). Association of childhood blood lead levels with cognitive function and socioeconomic status at age 38 years and with IQ decline and childhood-to-adulthood socioeconomic mobility in a New Zealand birth cohort. *JAMA*, 317(12), 1244–1251. <https://doi.org/10.1001/jama.2017.1712>
- Richter, P., Faroon, O., & Pappas, R. S. (2017). Cadmium and cadmium/zinc ratios and tobacco-related morbidities. *International journal of environmental research and public health*, 14(10), 1154.
- Safer Phosphates. (2019). *Cadmium toxicology reference: ANSES updates French TDI*. <https://www.saferphosphates.com/news/cadmium-new-toxicological-reference-value-to-protect-the-health-of-consumers/>
- Santana, V. S., et al. (2024). Gestational lead exposure and its effects on fetal/infant development: A systematic review. *Reproductive Toxicology*. <https://doi.org/10.1016/j.reprotox.2024.01.015>
- Satarug, S. (2018). Dietary cadmium intake and its effects on kidneys. *Toxics*, 6(3), 56. <https://doi.org/10.3390/toxics6030056>
- Tomno, R. M., Kitulu, L., Nzeve, J. K., Waswa, F., Mailu, S. N., & Shitanda, D. (2020). Heavy metal concentrations in vegetables cultivated and sold in Machakos Municipality, Kenya. <https://repository.seku.ac.ke/handle/123456789/7472>
- Tuakuila, J., Kabamba, M., Numbi, G., Mutombo, A., & Kafando, P. (2021). Heavy metal accumulation in cassava leaves in DR Congo mining regions. *Environmental Geochemistry and Health*, 43(9), 3567–3580. <https://doi.org/10.1007/s10653-020-00675-4>
- U.S. Food and Drug Administration. (2024). *FDA toxicology study on dietary cadmium exposure cites bone demineralization, kidney damage*. Center for Food Safety and Applied Nutrition. <https://www.food-safety.com/articles/8854-fda-toxicology-study-on-dietary-cadmium-exposure-cites-bone-demineralization-kidney-damage>
- Wairimu, L. N., et al. (2023). Heavy metal accumulation in irrigated vegetables in Kiambu County. *Environmental Monitoring Reports Kenya*. (Kenya repository).
- World Health Organization. (2024). *Lead poisoning and health: Fact sheet*. <https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health>
- Yahaya, A. A., Adegoke, O. O., Oyawoye, M. O., Ojo, O. S., & Adeyemi, T. A. (2020). Heavy metals in maize grains in Nigeria. *International Journal of Environmental Research and Public Health*, 17(11), 3921. <https://doi.org/10.3390/ijerph17113921>
- Zhang, Y., (2024). Chronic lead exposure and burden of cardiovascular disease during 1990–2019: A systematic analysis of the global burden of disease study. *Frontiers in Cardiovascular Medicine*, 11, 1367681.