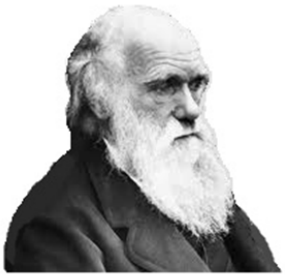
Available online at https://www.worldnewsnaturalsciences.com/



World News of Natural Sciences

An International Scientific Journal

WNOFNS 68 (2026) 129-153 EISSN 2543-5426

Comparative Toxicological Profiles of Copper, Zinc, and Lead in Urban Environments. Exposure Pathways and Long-Term Public Health Implications. A Systematic Review

Clement Akutam Azure[1], Omuru Uyouyouoghene Hannah[2], Oluwafemi Shittu Bakare[3], Akanimo Gordon Essiet[4], Bassey Okon Effanga[5], Jessica Augustine Udeh[6,*]

[1] Department of Physical Science, Faculty of Liberal Arts and Science, Eastern New Mexico University, Portales, New Mexico, USA

[2] Department of Environmental Management and Toxicology, Federal University of Petroleum Resources, Effurun, Nigeria

[3] Department of Biochemistry, Faculty of Science, Adekunle Ajasin University, Akungba Akoko, Ondo State, Nigeria

[4] School of Basic Sciences, Department of Public Health (Epidemiology), Federal College of Medical Laboratory Science and Technology, Jos, Nigeria

[5] Department of Human Nutrition and Dietetics, University of Calabar, Nigeria

[6] Department of Chemistry, University of Uyo, Uyo, Nigeria

*E-mail address: Jessicaaugustine98@gmail.com

ABSTRACT

Transport activity, industrial emissions, waste, atmospheric deposition, contaminated water systems, and dust resuspension have resulted in persistently redistributing copper (Cu), zinc (Zn), and lead (Pb) in urban environments as they are continuously redistributed in the air, dust, soil, water, food, and interior surfaces. This paper systematically reviewed the toxicological comparison of Cu, Zn, and Pb in urban environments, with reference to exposure pathways and long-term effects on the population. The study adopted the PRISMA 2020 framework and followed a comparative narrative synthesis design. A search of the literature was conducted in Scopus, Web of Science Core Collection,

(Received 19 June 2026; Accepted 22 July 2026; Date of Publication 23 July 2026)

PubMed/MEDLINE, ScienceDirect, and Google Scholar using Boolean combinations of metal-specific, urban-exposure, and toxicological keywords. The search generated 1,248 records (36 of which were identified by hand), of which 268 were duplicates, leaving 980 distinct records. Among them, 671 failed on the title stage, 167 on the abstract stage, and 62 on the full-text eligibility assessment; thus, 80 peer-reviewed studies were retained in the final synthesis. Data were systematically obtained on the type of study location, environmental matrix, types of metal contamination, sources of contamination, exposure pathways, toxicological endpoints, biomarkers, and the effects of contamination on public health. Findings indicated that the most frequently encountered media of exposure were road dust, street dust, and urban soils; drinking water; indoor dust; and food systems, but the most prevalent routes of exposure were inhalation, dust ingestion, water ingestion, dietary intake, dermal contact, and occupational exposure. The best result was that Pb was the most consistently risky metal, and became not only essential but also toxic when exposed in a chronic excess manner, while Cu and Zn displayed the dual essential yet toxic quality. Generally, exposure to urban metals is cumulative, multipathway and a key long-term community health issue.

***Keywords*:** Copper, Zinc, Lead, Urban Exposure Pathways, Toxicological Profiles, Public Health Risk

1. INTRODUCTION

Cities and ethnic areas have become key sites of convergence of metal contamination due to the concentration of transport, industrial processes, waste, building materials, commercial density, and excessive human exposure to metal-contaminated air, dust, soil, water, and food chains. It is in this extended contamination setting that copper, zinc, and lead are of particular concern, as they are continuously identified in the urban environment by the media and are closely associated with cumulative and chronic toxicological loads and accruing societal health hazards. It is demonstrated in urban road dust, street dust, soils, airborne particles, indoor dust, and water systems that these metals persist in circulating through mixed environments, including high-traffic routes, mixed territories, industrial neighbourhoods, informally organised workshop groups, and poorly regulated waste zones [1-3].

The environmental value of this trend lies not only in the availability of the metals but also in their resilience, mobility, bio-accessibility, and ability to move through various compartments of the city, thereby rendering them long-term depositories and routes of toxic contact. However, this is not the case for copper, zinc, and lead, which is exactly why a comparative review is required. Copper and zinc are toxic; both are trace elements and are necessary for enzyme regulation, redox homeostasis, immune function, and several other physiological activities. However, they become dangerous when their concentration network either rises above homeostatic control or falls below homeostatic control in the environment or in the body. Lead is different: there is no established positive biological activity, and a person is toxic despite relatively low exposure levels. The extant literature has demonstrated that copper and zinc can shift from physiological requirements to toxicological liabilities under conditions of contaminated exposure, and that lead has exhibited pronounced neurotoxic, haematological, hepatic, nephrotoxic, neuropathogenic, and endocrinogenic-disruptive effects across the age spectrum [4, 5].

This difference poses a significant scientific issue. Even when copper and zinc are discussed together with lead as heavy-metal contaminants, the processes, limits, and other long-term biological consequences of their contact are not comparable. The comparative synthesis

is thus rigorous and must be used to explain the point of convergence and divergence of these metals, with respect to which the toxicological profiles should be explained in the context of actual urban exposure systems.

The simplicity seems to be the key factor that keeps urban contamination with such metals a grave concern for people's general health, as there are numerous ways to be exposed. The urban populations are not exposed in a single manner, but rather they are cumulative and tend to be confirmed at the same time through inhaling the contaminated particulate materials and dust, ingesting the contaminated water and food, getting the contaminated soil and dust particles incidentally, and dermal exposure to the contaminated surfaces and urban green-space soils. Urban dust and street dust studies have indicated that bioaccessible fractions of copper and zinc and, in particular, lead may contribute to increasing actual human risk by more than bulk concentration alone may indicate, as well as by multipathways interactions starting to demonstrate that actual human exposure and urban ecological contamination are closely connected [6-8] Similar data pertaining to urban road dust, urban airborne dust, and urban soils also suggest that land-use type, particle size and sources of the emission locally and physicochemical properties influence the level of exposure and the degree of risk, especially on children and other susceptible urban dwellers [9-11].

These will demonstrate the fact that toxicological significance of copper, zinc, and lead in cities cannot be interpreted in a proper way without stating explicitly that pathway-based exposure science was combined with toxicological interpretation in the review. These metals have toxic effects that are not only mechanistically complex but, owing to their nature, often chronic and not immediately apparent. There has been a wide association of lead with unhealthy hematopoiesis, oxidative stress, enzyme activity, lipid peroxidation, brain damage, liver dysfunction, kidney dysfunction, and developmental derailment [12-14]. Although it is crucial, copper may cause oxidative tissue damage, hepatic perturbation, hematological change, as well as pathological damage in case of excessive exposure to copper-induced toxicity, whereas altered zinc balance could either contribute to toxicity aggravation or show a protective interaction in some cases of metal-exposure conditions [15, 16] Additional experimental and observational data can also indicate the interference with blood indices, liver enzymes, kidney biomarkers, and other similar metabolic parameters, in mixed and occupationally relevant metal exposure scenarios [17-19]. When combined, these studies indicate that biochemical toxicity in urban residents can result at some low-to-moderate levels of exposure over time, with cumulative physiological outcomes that are always underestimated when pollution investigations focus solely on environmental levels and not on related biological impacts in the body.

These long-term population health effects of copper-, zinc-, and lead-related urban exposures are especially severe because they are socially disproportionate and frequently concentrated among populations already disadvantaged by infrastructure and the environment. The high-exposure situation can be experienced by children living close to smelters and industrial areas, people living in traffic-restricted areas, workers in battery smelting and automobile repair areas, residents of contaminated urban districts, and, in some cases, along several routes simultaneously [20-22]. The issue is exacerbated in most low- and middle-income urban areas, particularly in sub-Saharan Africa, where rapid urbanisation, lax pollution regulations, informal waste management, and insufficient environmental surveillance may further increase exposure and under-reporting [23-25]. Within these environments, the impacts of chronic metal exposure may extend beyond classical toxicology to encompass broader

burdens related to child development, work-related susceptibility, food-chain pollution, endocrine disruption, chronic illness progression, and intergenerational health disparities. Although the literature on heavy metals in urban settings is sizeable and continues to grow, one significant conceptual gap remains. Several studies consider copper, zinc, and lead as a generalised set of potentially toxic metals, whereas others consider only lead or discuss environmental occurrence in an integrated manner, not considering sufficient exposure pathways, the biochemical processes involved, and the resulting long-term health effects. Others still study contamination in a single matrix (e.g., road dust, water, or soil) without determining how urban dwellers are exposed to and internalise these metals in everyday life.

This has led to a lot of information being provided in the literature, which is, however, disparate across the fields of environmental monitoring, exposure science, biomedical toxicology, and the assessment of population risk. If the toxicological profiles of copper, zinc, and lead are not only compared systematically but also synthesised more coherently, the key environmental exposure route supporting urban health risk, the long-term consequences of chronic exposure on the health of the population in modern cities are being unveiled due to these processes [26, 27].

It is against this backdrop that the current research paper is a systematic review of the comparative toxicological nature of copper, zinc, and lead in urban environments, with the emphasis laid on how the metals are distributed within the environmental media, exposure avenues in urban environments, and how long-term exposure can lead to long-term health effects on the population. The objective of the review is not only to describe reports on contamination events, but also to bridge environmental prevalence, exposure, toxicology, and health importance into a single analytical framework. In this way, it will seek to provide an enhanced basis for monitoring the environment, interpreting toxicological impacts, city risk management, and health-protective policy in cities where heavy-metal exposure has been endemic, multiplex, and inadequately controlled.

2. METHODOLOGY

2. 1. Study Design

The study employed a systematic review design to integrate and compare assessments of existing evidence on the toxicological characteristics of copper, zinc, and lead in urban settings, particularly regarding environmental exposure pathways and long-term health effects on the populace. The systematic review method was chosen because the topic spans various disciplines, including environmental chemistry, urban pollution science, toxicology, biomonitoring, epidemiology, and the assessment of population health risks. Given this extent, a systematic evidence synthesis methodology was deemed the most appropriate to locate, filter, evaluate, and combine the results of published research in a clear and reproducible way. The review was conducted using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) framework, which guided the identification, screening, eligibility, and final inclusion of studies. The design of the review was in the form of a comparative narrative synthesis, instead of a meta-analysis, due to the fact that those studies that were included in the review were assumed to be very different in terms of study design, environmental matrices, the procedures applied in the study, measurements of exposure, toxicological endpoints, and report forms. As a result, the systematic comparison, thematic

comparison, and integration of evidence through narrative discussion and structured tables were emphasised.

2. 2. Focus and Scope

The review has addressed three significant urban and toxicologically important metals. copper (Cu), zinc (Zn), and lead (Pb). These metals were chosen because they are frequently reported in urban environmental media, i.e., street dust, road dust, urban soil, urban water, atmospheric particulates, sediments, and food items, and because they exhibit wide variability in their toxicological effects. The essential trace metals were copper and zinc, which are toxic at high concentrations, whereas the non-essential metal was lead, which is already toxic and for which there is no safe level of exposure. The framework of the review aimed to capture evidence spanning the two ends of the urban sources of contamination and ecological events, including biochemical disturbances, toxicological effects, and final long-term effects on humans. The relevance criteria of studies included were accordingly. The presence of Cu, Zn or Pb in urban systems; source of contamination in urban settings; human exposure to contamination through inhalation, ingestion, dermal intake, professional exposure, and diet transfer; toxicological or biochemical outcome; response of biomarkers; risk of health in terms of characterisation; and an overall impact on health in urban populations.

2. 3. Eligibility Criteria

The screening process had pre-specified eligibility criteria to ensure consistency and methodological transparency. The studies had to be peer-reviewed journal articles published in the English language and had to target at least one of the target metals, that is, copper, zinc or lead, in an urban, peri-urban, metropolitan, municipal, industrial-urban or city-associated area. To be considered, a study also needed to present evidence on environmental occurrence, sources of contamination, exposure routes, toxicological effects, biochemical indicators, biomonitoring results, estimates of health risks, or implications for population health. Both primary empirical research and pertinent review papers that directly contributed to the comparative understanding of toxicological processes or exposure-health relationships were eligible. The studies were excluded in case they only dealt with a rural, forest, mining-only, or remote non-urban setting, analyzed heavy metals other than copper, zinc, or lead, or were limited to dealing with metallurgy, materials engineering, industrial processing, or laboratory chemistry only, were conference abstracts, editorials, commentaries, book chapters, theses, dissertations, or other types of grey literature, or could not be extracted due Experiments that involved experimental exposure only of an animal under artificial laboratory conditions were also not looked at unless the results were expressly related to the environmental toxicological interpretation that applied to the urban human exposures.

2. 4. Sources of Information

The literature search was conducted across five key academic databases to cover the subject matter in a broad, multidisciplinary way. Scopus, Web of Science Core Collection, PubMed/MEDLINE, Science Direct and Google Scholar were searched. This was done due to the large range of the environmental science, toxicology, and public health journals that Scopus and Web of Science index. PubMed/MEDLINE was also added to include toxicological, biomedical, and epidemiological studies, and, with the help of ScienceDirect, access to full-text

articles in environmental chemistry and exposure science was obtained. Google Scholar searches were conducted as auxiliary sources to identify additional peer-reviewed studies not covered by the main databases. In addition to searching electronic databases, the reference lists of the relevant studies were manually screened to identify additional articles on the review topic.

2. 5. Search Strategy

The search strategy incorporated terms related to the target metals, the city environment, and toxicological or health outcomes in the population. Maximum retrieval efficiency was maximised using the use of Boolean operators, truncation where necessary and searching of phrases. The keywords were narrowed down to the following. Copper or CU, zinc or Zn, lead or Pb, urban environment, urban pollution, city, metropolitan, road dust, street dust, urban soil, urban water, air pollution, PM, exposure pathway, human exposure, toxicality, toxicalogical profile, health risk, biomarker, oxidative stress, human health and long-term health effects. The representative database string was formatted by the following way. (Copper or CU or Zinc or Zn or Lead or Pb and urban or city or metropolitan or street dust or road dust or urban soil or urban water or long-term health effect or biomarker or health risk or public health or toxicity or toxicological profile or exposure pathway). The search strategy was modified to fit the search syntax of every database. Further selective searches were carried out on them that were related to the exposure of children, oxidative stress, neurotoxicity, hepatic toxicity, renal dysfunction, and chronic exposure to enhance them in terms of biochemical and public health aspects. Backwards citation searching, in addition to manual citation searching, was also conducted for select full-text papers.

2. 6. Search Yield and Study Identification

A total of 1,248 records were obtained through the literature search conducted across all electronic databases and additional searches. In particular, 312 records were found in Scopus, 241 in Web of Science, 226 in PubMed/MEDLINE, 189 in ScienceDirect, 244 in Google Scholar, and 36 by manual search of the reference list. After all records were exported into a reference management file and merged, it was found that 268 garbage records contained duplicate information. This yielded 980 unique records for stage I of the screening.

2. 7. Screening Process

The screening process was conducted in accordance with the PRISMA order. title screening, abstract screening, and full-text eligibility screening. The 980 unique records were reviewed at the title screening stage to identify those directly relevant to the topic under review. One thousand six hundred seven hundred records were weeded out at this stage as they were obviously not related to urban heavy metal contamination, had no bent towards copper, zinc or lead, and had no toxicological orientation, or were located in a non-urban or industrially isolated environment without environmental contact involvement. Title screening was completed, resulting in 309 records qualifying for abstract screening. The remaining 309 articles were screened at the abstract level against the inclusion criteria. This eliminated 167 records because they were insufficient to cover the toxicological profiles of Cu, Zn, or Pb; were irrelevant to the exposure pathways of contaminants in the environment or their impact on the human population; were about different contaminants; or were unclear whether they were from an

urban environment. This led to the retrieval of 142 full-text articles, which were evaluated for eligibility.

2. 8. Full-Text Eligibility Assessment

The entire manuscript of 142 articles was retrieved and attentively reviewed in accordance with the preset inclusion and exclusion criteria. After the review, 62 full-text articles were excluded for various reasons. Amongst them 18 studies were excluded by virtue of being located in non-urban or rural areas; 11 studies were excluded by virtue of not having the right amount of information on copper, zinc, or lead per se; 9 studies were eliminated because they were review commentaries, conference papers, or non-peer-reviewed materials; and 9 studies were excluded because they were largely about industrial or laboratory process issues, not giving them a Concluding the eligibility tests, 80 articles were left in the end after considering all the criteria.

2. 9. Final Inclusion

The final review included 80 studies. These researchers formed the evidence base for a comparative synthesis of the environmental occurrence, exposure pathways, toxicological consequences, biochemical effects, and long-term effects of copper, zinc, and lead on the urban population. The articles included at the end represented a wide spectrum of urban environmental matrices, geographical sites, exposure conditions, and health outcomes, providing a sufficient variety of study areas for comparative toxicological interpretation.

2. 10. Data Extraction Procedure

An extraction template was prepared to ensure that the main features and results of the included trials are systematically extracted. The information that was extracted on each of the 80 studies that were included was as follows; author name(s), year of publication, study location or city, study design, environmental matrix of interest, target metal(s) of interest, contamination or concentration data, significant contamination sources, reported exposure pathway, method of analytical approach to data, biomarkers or toxicological outcome, health outcome, and significant conclusions to the objectives of the review. Further information was extracted where available, including the groups in need and those in extreme need, the type of sample, biomonitoring indicators, and the cancer- or no-cancer-risk measures. Hazard quotient, hazard index, contamination factor, and enrichment factor. This method of extraction made the literature consistent and allowed conducting comparative thematic analysis between the studies.

2. 11. Quality Appraisal and Risk of Bias taking

The methodological quality of the studies in the included literature was appraised based on the heterogeneity of the literature. A flexible set of appraisal tools was used, as the final pool of 80 studies included environmental monitoring, exposure assessment, human biomonitoring, epidemiological investigations, and mechanistic toxicology reports. All the studies were evaluated based on the clarity of objectives, adequacy of study design, description of sampling, relevance of the environmental/biological context, adequacy of the analytical method, clarity of the exposure description, and applicability of the toxicological/public health interpretation. The possible sources of bias that were given particular focus included a lack of description of sampling methods, a lack of clarity in the quality control of the laboratories, a lack of connection

between the contamination of the environment and health effects, poor characterisation of the urban areas, and exaggerated conclusions in the end beyond the statistics. Rather than excluding only the weaker studies, quality appraisal results were utilised to weight interpretive confidence during synthesis. Higher-quality empirical research and explicitly defined exposure-outcome logic were accorded more analysis in the results and discussion.

2. 12. Synthesis and Analysis Method

The review utilised a comparative synthesis of narratives, employing thematic grouping and integrating evidence in tabular form. A formal meta-analysis could not be considered suitable due to high heterogeneity among the 80 included studies, which varied across environmental media, population groups, metal combinations, methodologies, exposure measures, outcome measures, and reporting styles. The evidence was thus conceptualised across various domains that are related to one another. First, the studies were compared with respect to the environmental occurrence of copper, zinc, and lead in dust, soil, water, air, sediment, and food. Second, the literature synthesis was based on the most prevalent sources of urban contamination, such as transportation activity, industrial effluents, waste burning, e-waste management, residual paint/construction debris, urban runoff, and informal recycling. Third, the included studies were compared by exposure route, particularly inhalation, ingestion, skin contact, diet, and occupational/household second-hand exposure. Fourth, the toxicology of Cu, Zn, and Pb was comparatively discussed in relation to bioavailability, essentiality, threshold behaviour, induction of oxidative stress, organ toxicity, haematological disruption, neurotoxicity, inflammatory pathways and developmental outcome. Fifth, the review compiled the long-term potential impacts on population health identified in the literature, including the susceptibility of children, the chronic disease burden on health, reproductive and developmental consequences, and health issues in urban communities. This overlay synthesis strategy helped move the review beyond descriptive enumeration to a comparative analysis of common trends and mechanistic oppositions among the incorporated evidence.

2. 13. Outcome Domains

To provide analytical coherence, the review combined the 80 included studies, and the outcomes were divided into pre-defined outcome domains. They included the level of environmental occurrence and contamination; the source of contamination and urban emission patterns; the exposure pathways; the biochemical and toxicological effects; the biomarkers of exposure or effect; the reported health outcomes; and the wider implications for public health. This helped establish the structure of the synthesis in a way that indirectly addressed the review's overall objective and, as a result, contributed to a comparative interpretation of the specific and general toxicological activity of copper, zinc, and lead.

2. 14. Ethical Considerations

The research was based solely on existing literature, without conducting any primary research, direct contact with human subjects, research involving animals, or confidential disclosure of personal records. In this regard, no formal ethics approval was necessary. However, the review was conducted in accordance with acceptable ethical standards for synthesising scholarly evidence, such as clear study selection, accurate reflection of the results

of the 80 included studies, avoidance of misinterpretation, and credible references to all sources through continuous citation.

3. RESULTS

Table 1. General characteristics of the included studies.

S/N	Study cluster	Representative studies	Main study emphasis	Broad contribution to the review
1	Urban dust and road dust contamination studies	[1-3, 11, 27-30]	Quantification of Cu, Zn, Pb and other toxic metals in road dust, street dust, and surface dust	Established urban dust as one of the most recurrent and dominant environmental reservoirs of toxic metal exposure
2	Urban soil contamination studies	[26, 31-33]	Metal distribution in urban soils and human health implications	Showed that urban soils serve as long-term sinks and secondary sources of exposure through resuspension and contact
3	Airborne dust and particulate exposure studies	[34-36]	Pulmonary toxicity and inhalation-related metal exposure	Demonstrated that fine particles and airborne dust facilitate respiratory uptake and deepen chronic exposure risk
4	Water contamination and drinking water studies	[24, 37, 38]	Detection of metals in drinking water and associated health hazards	Confirmed water ingestion as a critical pathway where environmental control is weak
5	Food-chain and vegetable contamination studies	[39, 40]	Metal occurrence in foodstuffs and urban-grown vegetables	Showed dietary transfer as an important long-term exposure route
6	Bioaccessibility and multipathway risk studies	[6-8]	Bioaccessible fraction, hazard quotients, and pathway-integrated health risk	Shifted emphasis from total concentration to human-relevant exposure potential
7	Occupational and industrial exposure studies	[20-22, 41-43]	Smelter, battery, workshop, and traffic-related exposure	Identified high-risk occupational and peri-occupational populations
8	Toxicological and mechanistic studies	[12-15, 44-46]	Oxidative stress, organ toxicity, hematological and pathological effects	Clarified the mechanistic basis of biochemical injury and long-term toxicity

9	Trace-metal physiology and interaction studies	[4, 14, 47-49]	Essentiality-to-toxicity transition and metal interaction effects	Helped distinguish Cu and Zn as essential-but-toxic-at-high-dose, unlike Pb
10	Broad environmental health review studies	[23, 25, 50]	Environmental pollution patterns and health implications	Provided wider contextual understanding of urban metal pollution and environmental governance issues

Table 2. Major environmental matrices in which copper, zinc, and lead were reported.

Environmental matrix	Studies indicating occurrence	Metals emphasized	Result pattern emerging from the review
Road dust	[1, 2, 28, 35]	Cu, Zn, Pb	Road dust was among the most consistently reported urban exposure media, especially near traffic corridors and dense urban activity zones
Street dust	[7, 10, 27, 29]	Cu, Zn, Pb	Street dust repeatedly acted as a mixed-source reservoir reflecting vehicular, industrial, atmospheric, and surface deposition inputs
Surface dust / urban dust	[3, 6, 11, 30]	Cu, Zn, Pb	Urban dust carried both bulk metal load and bioaccessible fractions, making it central to non-dietary ingestion risk
Urban soils	[26, 31-33]	Cu, Zn, Pb	Soils were long-term accumulation sites and were implicated in dermal, inhalation, and incidental ingestion pathways
Park and green-space soils/dust	[9, 32]	Cu, Zn, Pb and related metals	Recreational environments were not risk-free; children and frequent users may have meaningful contact exposure
Airborne dust / PM	[34-36]	Cu emphasized in airborne dust; Pb relevant in particulate mixtures	Airborne fractions heightened inhalation risk and increased concern for pulmonary and systemic absorption
Indoor dust	[52]	Mixed metal contamination, including Pb and other metals	Indoor environments reflected carry-over contamination and sustained chronic household exposure
Drinking water / rainwater	[24, 37, 38]	Cu, Zn, Pb	Water acted as a direct ingestion pathway, particularly where storage systems, plumbing, or source contamination existed

Foodstuffs and vegetables	[39, 40]	Cu, Zn, Pb and other trace metals	Urban food-chain transfer reinforced the importance of chronic dietary intake in metal exposure
Biological tissues / blood markers	[14, 18, 41, 42]	Pb, Cu, Zn	

Table 3. Dominant urban sources and emission patterns of copper, zinc, and lead.

Source category	Metals commonly linked	Supporting studies	Synthesized result
Vehicular activity, brake/tire wear, traffic emissions	Cu, Zn, Pb	[1, 2, 28, 35]	Traffic-related surfaces were repeatedly associated with elevated contamination, making transport systems a major urban driver
Industrial discharge and smelting	Pb, Zn, Cu	[17, 18, 20]	Smelter-linked exposure emerged as one of the most serious pathways for chronic and community-level contamination
E-waste and electronics manufacturing environments	Pb, Cu, Zn	[21, 31]	Electronics-related environments contributed to multi-metal exposure and biomarker disruption
Automobile workshops and mechanical service zones	Pb, Cu, Zn	[22, 43]	Informal and poorly regulated workshop environments were important local sources in sub-Saharan urban settings
Waste accumulation and urban mixed refuse	Pb, Cu, Zn	[23, 25]	Waste systems amplified diffuse contamination and broadened community exposure beyond industrial districts
Atmospheric deposition and resuspension	Pb, Cu, Zn	[34, 35, 52]	Deposited particles re-entered the breathing zone and indoor environments, sustaining repeated exposure
Urban runoff and contaminated water pathways	Cu, Zn, Pb	[24, 37, 38]	Waterborne transport linked environmental contamination with domestic ingestion exposure
Construction materials, paint residues, built infrastructure	Pb and Cu particularly	[12, 54]	Legacy and contemporary building materials contributed to persistent exposure burdens in older and mixed-use urban areas

Table 4. Comparative exposure pathways identified across the included studies.

Exposure pathway	Environmental medium	Key supporting studies	Comparative result for Cu, Zn, and Pb
Inhalation	Airborne dust, PM, resuspended road/street dust	[34-36]	Inhalation was especially important in traffic-dense and dust-prone environments; Pb and Cu were particularly relevant in particulate-associated exposure
Incidental ingestion of dust and soil	Road dust, surface dust, street dust, urban soil	[6-8, 10]	This was one of the strongest pathways for children and highly exposed urban residents, especially where Pb bioaccessibility was high
Direct water ingestion	Drinking water, stored rainwater, urban water sources	[24, 37, 38]	Water ingestion provided a direct and repeated pathway with important long-term health relevance
Dietary intake	Vegetables, foodstuffs, contaminated urban-grown produce	[39, 40]	Chronic low-dose dietary exposure likely contributes to long-term internal burden
Dermal exposure	Urban soils, green-space soils, surface contact media	[32]	Dermal exposure was less emphasized historically, but recent work indicates it remains toxicologically relevant under repeated contact conditions
Indoor household exposure	Indoor settled dust	[52]	Indoor dust extended exposure beyond outdoor spaces, especially through hand-to-mouth transfer and inhalation of fine particles
Occupational exposure	Smelters, battery sites, workshops, polluted industrial settings	[20, 22, 41, 42]	Occupational populations showed stronger internal exposure signals and biochemical disturbance
Multipathway cumulative exposure	Combined dust, soil, water, inhalation, and ingestion routes	[7, 8]	

Table 5. Comparative toxicological profiles of copper, zinc, and lead synthesized from the included studies.

Metal	Biological status	Principal toxicological concern	Key target systems/organs	Representative studies	Result pattern
Copper (Cu)	Essential trace metal	Toxic at elevated concentrations; oxidative stress; hepatic and hematological disturbance	Liver, blood, kidney, respiratory system	[4, 5, 16, 45]	Copper showed a dual profile. physiologically necessary, but harmful when exposure exceeds homeostatic buffering
Zinc (Zn)	Essential trace metal	Dysregulation under excess exposure; interaction with other metals; hematological and endocrine implications	Blood, endocrine system, liver, kidney	[4, 14, 16, 49]	Zinc was frequently discussed as both a nutrient and a modifying factor in toxic metal interactions
Lead (Pb)	Non-essential toxic metal	Neurotoxicity, hematotoxicity, nephrotoxicity, hepatotoxicity, oxidative stress, developmental injury	Nervous system, blood, kidney, liver, developmental systems	[12, 13, 42, 44, 53]	Lead emerged as the most consistently harmful and least biologically defensible of the three metals
Cu + Pb co-exposure	Mixed essential-toxic combination	Additive oxidative and tissue injury potential	Blood, liver, kidney	[44, 45]	Mixed exposure intensified pathological interpretation because essentiality did not prevent toxicity
Pb with Zn/Cu interaction	Competitive and modifying relationship	Altered trace metal balance and possible disruption of protective micronutrient status	Hematological and metabolic systems	[14, 41, 47]	Lead exposure often coincided with disturbed Zn and Cu homeostasis, suggesting interaction effects in exposed populations

Table 6. Major biochemical and pathological effects reported across the literature.

Effect domain	Reported pattern	Metals implicated	Supporting studies
Oxidative stress	Increased oxidative injury, lipid peroxidation, redox imbalance	Pb, Cu; mixed metal exposure	[13, 44, 45]
Hematological alteration	Changes in blood indices, hematopoiesis-related disturbance	Pb, Cu, Zn interactions	[14, 15, 41, 42]
Hepatic dysfunction	Elevated liver enzymes, tissue injury, metabolic disruption	Pb, Cu, co-exposure states	[5, 17, 18, 45]
Renal impairment	Kidney biomarker changes and renal function disruption	Pb, Cu, mixed exposure	[17, 19, 54]
Pulmonary toxicity	Respiratory irritation and lung-related injury from metal-bearing particulates	Cu and soluble metals in dust mixtures; Pb mixtures	[34]
Histopathological damage	Tissue and organ structure alteration	Cu, Pb, Zn in experimental systems	[46, 55]
Endocrine and thyroid relevance	Metal-related endocrine disruption and thyroid implications	Zn, Pb and broader trace metal mixtures	[48, 49]

Table 7. Population groups and settings identified as most vulnerable.

Vulnerable group / setting	Why vulnerable	Supporting studies
Children in polluted urban areas	Greater hand-to-mouth behavior, dust ingestion, developmental sensitivity	[6, 9, 20]
Occupationally exposed workers	Direct chronic contact with metal-rich dust, fumes, and residues	[22, 41-43]
Residents near smelters and industrial zones	Continuous environmental deposition and multi-route exposure	[17, 20, 21]
Residents in traffic-dense corridors	Constant exposure to road dust, resuspended particles, and roadside contamination	[1, 2, 35]

Households with contaminated indoor dust	Persistent low-dose exposure through domestic contact	[52]
Consumers relying on contaminated water and food sources	Repeated ingestion over long durations	Teschke and Xuan (2025); Khanna (2011); El-Kady and Abdel-Wahhab (2018)

Table 8. Long-term public health implications synthesized from the included studies.

Public health domain	Main implication from the reviewed evidence	Metals most strongly linked	Representative studies
Neurological and developmental health	Persistent concern for cognitive, developmental, and nervous-system injury, especially under Pb exposure	Pb	[12, 20, 44]
Hematological health	Disruption of blood parameters, metal-binding proteins, and hematopoietic balance	Pb, Cu, Zn interaction states	[14, 41, 42]
Liver health	Chronic exposure may contribute to hepatic stress and clinical liver injury	Cu, Pb	[5, 18, 45]
Kidney health	Long-term exposure may impair renal biomarkers and kidney function	Pb and mixed exposures	[14, 19, 54]
Endocrine and thyroid health	Trace-metal imbalance may influence endocrine pathways and disease vulnerability	Zn, Pb, mixed trace metals	[48, 49]
Respiratory health	Inhaled dust-associated metals may drive pulmonary toxicity and prolonged respiratory burden	Cu and mixed urban particulate metals	[34, 35]
Community environmental health	Exposure becomes a broader urban governance and environmental justice issue	Cu, Zn, Pb collectively	[23, 25, 33]
Public health prevention and policy	Monitoring, nutritional protection, occupational control, and environmental regulation remain essential	Pb primarily, but also Cu and Zn under excess conditions	[12, 37, 56]

Table 9. Geographic spread and contextual distribution of the included evidence.

Region / context	Examples from included studies	Result trend
China and East Asia	[1, 9, 10, 18, 21]	Strong evidence base on road dust, industrial contamination, and health-risk assessment
Middle East / West Asia	[3, 30]	Strong urban dust and land-use comparative evidence
Europe / Türkiye	[29]	Strong source-dynamics framing for street dust exposure
South Asia	[11, 42]	Important evidence on urban dust and occupational internal burden
Africa / sub-Saharan Africa	[22-24, 33, 35, 43]	Evidence confirms growing urban metal burden, but region remains comparatively underrepresented in detailed biomarker-based studies
Latin America	[27]	Urban dust contamination and risk framing clearly documented
Broad/global reviews	[4, 25, 37]	Reinforce the global relevance of comparative metal toxicology in urban environments

4. DISCUSSION OF THE RESEARCH FINDINGS

The findings of this systematic review indicate that such toxicological meaning of copper, zinc, and lead in cities cannot be perceived as a straightforward case of contamination of isolated impact. Instead, the evidence synthesised in Tables 1 to 9 demonstrates that the system of exposures is structurally connected, in which environmental opportunity, urban land-use stress, the multiplicity of pathways, metal-specific toxicodynamics, and social vulnerability enhance one another. Studies are largely focused on urban dust, road dust, street dust, soil, and water, as well as particulate matter, occupational environments, and biomarker-based toxicology; this literature has increasingly shifted toward less integrated studies of human health interpretation. The data that the road dust, surface dust, urban soils, and water systems were repeatedly represented in the studies incorporated is a strong indication that modern cities are more of an exposure landscape in which metals are mobilised, redistributed, inhaled, ingested, and internalised during a period of time rather than just a contaminated site. Such a trend can be observed in the evidence base, which includes urban dust, street dust, indoor dust, water, soil, airborne particulates, biomonitoring, and mechanistic toxicology. One of the key insights from Table 1 is that the literature is evenly distributed across all toxicological dimensions. The majority of research activities focus on environmental monitoring and risk characterisation, particularly dust and soil investigations, but very few address the direct relationship between environmental burden and long-term biochemical harm in exposed human

populations. This aspect is noteworthy. It indicates that research on urban toxic metals has now been highly effective at identifying areas of contamination, yet the matter remains somewhat unfinished regarding how such exposures lead to long-term physiological dysfunction. This suggestion is both methodological and substantive. In terms of methodology, this implies that numerous evidence-based pieces remain based on inference. Contamination of dust, soil, and water is typically regarded as an indicator of a health risk. The fact that there are occupational biomonitoring studies, haematological studies, and mechanistic toxicology papers in the included literature, however, indicates that the problem of the environmental burden is not a hypothetical notion; it has biological implications. Therefore, the review not only indicates that copper, zinc and lead do exist in cities but also that the literature is leaning more towards establishing a link between occurrence and effect, albeit that lead has a stronger link to effect than copper and zinc.

As shown in Table 2, road dust, street dust, surface dust, and urban soil have the most studies documenting their repeated occurrence in the analysed papers, and this observation is crucial. Not only is the dust in the urban environment abundant and readily sampled, but it is also toxicologically strategic because it incorporates traffic and industrial emissions, atmospheric fallout, waste systems, building materials, and resuspended past deposits. Practically, dust can be described as a condensed war journal of urban pollution and an expositional medium in the present. The fact that road dust appears on top of the list several times in the review, as well as street dust, is indicative of the fact that transport infrastructure may be considered one of the most significant points of contamination affecting public health.

This is in line with the principles of the science of urban exposure. The closer pollutants are to the daily human movement, the higher the ability to enter into the respiratory tract, onto the surface of skin, into the house and, ultimately, into the systemic body. These findings thus show that road-related matrices are not merely environmental inferences but epidemiological agents of chronic exposure. In contrast, the fact that the evidence in question includes indoor dust, park soils, green-space soils, foodstuffs, and rainwater demonstrates that the reducing factor is not confined to the roadside. It also infiltrates domestic spaces, including during recreation and ingestion routes, and thus poses contamination as an exposure problem throughout the entire life cycle, not just in public spaces.

This interpretation is further supported by the trend shown in Table 3. Automobile activity, industry, smelting, e-waste treatment, informal automobile workshops, accumulation of waste, and atmospheric deposition are the leading urban sources. The subtle implication of this is that there is no single urban process that is causing the contamination of Cu, Zn, and Pb. It is being fabricated through an overlaid city metabolism where formal and informal practices have an interplay between them. There are mechanical wear and suspended particulates brought about by traffic, direct emissions, and localised deposition by the industry; diffuse contamination by waste and e-waste; and redistribution of the atmosphere, ensuring that even those not in direct occupational contact could still be exposed. This variety of sources shows that urban pollution is usually hard to eradicate, even when it is treated locally. Other channels continue to feed into the environmental load even if one of the sources has been diminished. The review consequently endorses a systems explanation.

The exposure of urban metals is cumulative since the urban sources are cumulative. The theoretical implications of this are enormous, as it removes the point-source toxicology of the debate, replacing it with distributed-source toxicology, in which the cumulative effects of

numerous low- to moderate-intensity emissions, rather than a spectacular poisoning incident, cause changes in the health of the population.

It is possible that Table 4 is among the most significant in the study, as it demonstrates that exposure pathways are multiple, overlapping, and cumulative. The included studies all represent inhalation, incidental ingestion of dust and soil, water ingestion, dietary intake, dermal exposure, indoor contact, occupational contact, and multipathway contact. This is a conclusive finding, as it implies that issues concerning the toxicological profiles of copper, zinc, and lead cannot be justifiably debated on a one-way basis. The ancient tradition of preference to the inhalation or simply drinking of water cannot suit the city. Children can ingest dust via hand-to-mouth contamination; workers can inhale contaminated particulates and simultaneously transport them to their homes; individuals living in cities may drink polluted water and eat unhygienic foods cultivated in polluted soil; and long-term dermal contact may impose additional burdens in certain microenvironments. The more general point is that risk is not additive but interactive. Although both pathways may appear humble individually, a combination of prolonged exposure may result in an internal dose that is significantly large. It is particularly significant to lead, where there is no physiological usefulness; it is also true of copper and zinc, where repeated excess exposures can defeat the body's homeostatic mechanisms. The review thus substantiates that one of the fundamental causes of why urban metal pollution continues to be a perennial issue for the masses is not a temporary environmental irritant.

Table 5 is the most insightful comparative profile of the specific toxicological identity of the three metals. Copper and Zinc are biologically essential, and lead is bioessential but intrinsically toxic. This difference is inherent, since it explains why the toxicological explanation of Cu and Zn should be more subtle than that of Pb. Copper and zinc are located in a toxicological space, which is threshold (dependent). Systems of enzymes, immune controls, redox regulation, and cell structure require these elements at physiological levels, but at excessively high or uncontrolled levels, damagingly influence the system. Lead, on the other hand, is toxic in that it is dangerous in nature and builds up. The review consequently maintains a discerned toxicology as opposed to the homogeneous model of the toxicity of heavy metals being bad. This is important in science as it is conceptually flattening. Copper and zinc cannot be treated in the same manner and are regarded as weaker forms of lead. Their toxicity depends on context and is conditional, partly determined by disruption of homeostasis, co-exposure, nutrition, and metabolic rivalry. On the other hand, lead poisoning is less indirect, more reliably unwell, and clearly associated with haematological, neurological, renal, hepatic, and developmental damage. This point of comparative difference is one of the most powerful ideas in the conceptual framework of the current review.

In the same table, it is also shown that mixed exposure warrants special attention. In cases of co-occurring lead with copper-zinc, the toxicological explanation becomes more complicated, since lead can affect the standard levels of required trace metals, whereas interference with the balance between zinc and copper can both enhance and, to some extent, soften the intensity of toxicity. This is an interaction-based interpretation, which is vital. It means that the toxicology of metals in the urban environment is not only related to the individual dose of metals, but to the ecology of combined exposure. Nutritional physiology, competitive transport, metalloprotein binding, buffering oxidative stress and displacement of trace-elements become enforced. The review, therefore, upholds the notion that urban exposure to Cu, Zn, and Pb can be explained and understood within a mixed-metal framework rather than

by single-metal toxicology. Such a conclusion has both theoretical and practical health consequences, since in an actual urban setting, it is almost never the case that a population can be exposed to a single metal. This argument is supported by Table 6, which indicates that oxidative stress, haematological and hepatic dysfunction, renal impairment, pulmonary toxicity, histopathological injury, and endocrine relevance are repeated across the studies used. Oxidative stress is one of the most widespread pathways, and it seems to be unifying according to the mechanistic approach.

This is particularly significant since oxidative stress serves as an intermediary in disease exposure. It assists in describing how prolonged environmental exposure can lead to cellular damage, membrane damage, inflammation, enzyme disruption, and ultimate tissue dysfunction. Oxidative stress in the context of toxicology does not represent only a single endpoint among a range of many other endpoints but rather a pathway of mechanistic centrality, through which essential and non-essential metals can cause overall physiological harm. Haematological effects are also significant, indicating that the internal disturbance does not seem to be limited to functional organs and may extend to systemic transport, oxygenation, and metabolic processes.

The recurrence rate of blood-related abnormalities in the included literature supports the case that chronic exposure to metals has system-wide effects on the organism and is not just a case of localised toxicity. The fact that liver and kidney disorders repeatedly recur also points to the fact that the burden of exposure to metals is cumulative and depends on detoxification. In cases of repeated exposure, the organs responsible for metabolism, filtration, and excretion become the locus of sustained stress.

Table 7 indicates that the vulnerability is highly patterned rather than randomly distributed. The children, those who work in industries where they are exposed to industrial hazards, people living near mines and smelting plants, along traffic-congested routes, families with contaminated indoor dust, and communities relying on damaged food or water systems all turn out to be prime targets. This outcome is highly significant, as it sets aside the debate over contamination and focuses on environmental inequality. The exposure cannot be described as socially neutral. Residents in the immediate vicinity of improperly regulated industrial activity, informal repairs and waste systems, and high-traffic density areas are at higher risk of experiencing heavy metal burdens on a permanent basis. Children cannot be overlooked in this regard, since, biologically, they are in the process of development; behaviorally, they are inclined towards ingesting dust; and physiologically, they are more prone to neurodevelopmental and haematological damage.

The sheer exposure of workers to direct forms of contamination is recurring, but the review also suggests a para-occupational threat, whereby contaminants can enter the home through work-related clothing, dust, and even contaminated surfaces. Therefore, the negative scope of copper, zinc and lead toxicology in the urban environment is also to be taken as a sign of injustice in healthcare. Uneven distribution of exposure raises city toxicity, which is in part a product of the city's organisation, control and occupation. The results in Table 8 show that it has long-term, general impacts on health across the population. The evidence base is again demonstrated by neurological and developmental damage, disruption of the blood system, dysfunction of the liver and kidneys, respiratory and endocrine disturbances, and the impact of environmental health on the community. The greatest harbour of systemic neurodevelopmental harm and chronic neurotoxicity is a product of lead, particularly in comparison to the other two metals. Although less homogeneous in their effects, copper and zinc nonetheless become toxicologically significant during prolonged or excessive exposure, particularly regarding liver

function, blood parameters, and metabolic imbalance. Of particular significance to the health of the public is the fact that most of the consequences established are chronic, subliminal in the early phases of development and accumulative. It means that urban populations can have toxic loads that are meaningful before critical, apparently disease-free conditions strike. It is apparent in the review that urban metal pollution could be interpreted more on a preventive than a reactionary basis. It is scientifically and ethically disadvantageous to wait up until an overt poisoning is discovered. Exposure to the sharp results might have become long-term and deeply ingrained within the city setting by the time the harsh consequences are revealed.

The issue of geographical dimension is introduced to the discussion in Table 9. The studies included display high levels of evidence in China, wider Asia, sections of the Middle East, and select urban cities in other parts, but sub-Saharan Africa is included, although as a proportion of evidence compared with other areas. The implication of this is huge. First, it implies that the international evidence base is actual but ineffective. Second, it hints at otherness and the under-representation of risk in African cities. Third, it points out the necessity of not assuming that the more studied regions are the high-risk ones. Actually, the availability of studies in Kenya and sub-Saharan urban workshop settings, reviews of African water contamination, and reviews of African-based systematic syntheses provide evidence that the region is experiencing a heavy burden under less stringent monitoring and regulation. It means that the lack of evidence can not be misconstrued as safety. Quite the contrary, in fast-urbanising areas, there is limited data, which can obscure the significant toxicological burden that has not been properly quantified. The findings indicated that there were certain, most pressing gaps in research. The literature continues to be too heavily based on concentration-based contamination studies; it is also too geographically sloppy, too few longitudinal human biomonitoring designs, and in many cases, too generically based on copper and zinc as being toxic metals without a sufficient consideration of their key physiological functions. These loopholes are important since they define the conceptualisation of risk. When the focus of the research is on the total concentration level, it might overstate the role of bioaccessibility, internal dose, and cumulative effects. In case of non-combat over the dermal exposure, an aspect of the urban contact system is under-excavated. Discussion of copper and zinc without a physiological implication makes the toxicological interpretation too simplistic.

The current review, thus, not only synthesises the existing knowledge but also clarifies the conceptually immature one. More specifically, the review supports the necessity of future research that incorporates environmental monitoring with biomarkers, toxicokinetic and metabolic interpretation, nutritional status, combined-metal interactions, and pathway-resolved exposure studies. The results of Tables 1 to 9, as a whole, support a single conclusion: copper, zinc, and lead in urban areas should be viewed from an integrated exposure-toxicity perspective. Disparate parts of urban architecture, rural dust on the road, and indoor dust are not separate entities but rather structural parts in chronic exposure architecture. A large part of the emissions overlaps, and thus neither traffic, industry, waste, informal workshops, nor smelting is a solitary source. Other available pathways include inhalation, ingestion, dermal contact, and occupational carryover, which would be coexisting pathways contributing to cumulative internal burden. The health outcomes, including oxidative stress, blood disturbances, organ damage, developmental damage, and community susceptibility, are not incidental endpoints of long-term exposure to contaminated urban systems. This is the greatest implication of the review. Urban metal toxicology cannot be a one-substance issue, nor a one-pathway issue, nor just an environmental-measures issue. It is a public health problem at the systems level that

requires interdisciplinary surveillance, source control, biomonitoring, and prevention-based government.

5. CONCLUSIONS

The evidence synthesised in this systematic review shows that copper, zinc, and lead remain major toxicologically relevant metals in the urban environment, though their significance to population health cannot be applied uniformly. It is evident in the reviewed literature that urban dust, road dust, street dust, soils, water systems, indoor dust, and food-related pathways are consistent sources and transmission routes for how these metals enter human life and biological systems. In comparison with the other two metals, lead was the safest since it is not essential, it is highly toxic and has a close relationship with neurological, haematological, renal, hepatic, and developmental damage. Copper and zinc, which are important to physiology, were shown to become toxicologically significant when environmental concentrations exceed homeostatic regulation, particularly in cases of chronic exposure, oxidative stress, mixed-metal interactions, and repeated contact through various pathways. The review also confirms that exposure to urban metals is cumulative, multipathway, and socially patterned, with children, workers, roadside people, and people living in industrial or informal urban areas with low regulation being more vulnerable. In general, the research finds that the toxicological burden of copper, zinc, and lead in cities can be considered a systems-level environmental health issue in which the sources of contamination, exposure routes, biochemical processes, and the ultimate effects on human health are closely interrelated.

References

[1] Shi, G., Chen, Z., Bi, C., Wang, L., Teng, J., Li, Y., & Xu, S. (2011). A comparative study of health risk of potentially toxic metals in urban and suburban road dust in the most populated city of China. *Atmospheric Environment*, 45(3), 764-771

[2] Lu, H., Shen, Y., Maurya, P., Chen, J., Li, T., & Paz-Ferreiro, J. (2025). Spatial Heterogeneity of Heavy Metals Contamination in Urban Road Dust and Associated Human Health Risks. *Land*, 14(4), 754

[3] Mihankhah, T., Saeedi, M., & Karbassi, A. (2020). A comparative study of elemental pollution and health risk assessment in urban dust of different land-uses in Tehran's urban area. *Chemosphere*, 241, 124984

[4] Leal, M. F. C., Catarino, R. I., Pimenta, A. M., & Souto, M. R. S. (2023). The influence of the biometals Cu, Fe, and Zn and the toxic metals Cd and Pb on human health and disease. *Trace Elements and Electrolytes*, 40(1), 1-22

[5] Teschke, R. (2024). Copper, iron, cadmium, and arsenic, all generated in the universe. elucidating their environmental impact risk on human health including clinical liver injury. *International Journal of Molecular Sciences*, 25(12), 6662

[6] Bi, X., Li, Z., Sun, G., Liu, J., & Han, Z. (2015). In vitro bioaccessibility of lead in surface dust and implications for human exposure. A comparative study between industrial area and urban district. *Journal of Hazardous Materials*, 297, 191-197

[7] Dehghani, S., Moore, F., Vasiluk, L., & Hale, B. A. (2018). The influence of physicochemical parameters on bioaccessibility-adjusted hazard quotients for copper, lead and zinc in different grain size fractions of urban street dusts and soils. *Environmental Geochemistry and Health*, 40(3), 1155-1174

[8] Qader, M. Q., Ayanlade, O. S., Ayoola, P. O., Fatah, K. K., Oluwatimilehin, I. A., & Ayanlade, A. (2026). Multipathway Assessment of Heavy Metal Exposure in Urban Dust. Linking Ecological and Human Health Risks. *Journal of Applied Toxicology* 1–13. https://doi.org/10.1002/jat.70126

[9] Han, X., Lu, X., Qinggeletu, & Wu, Y. (2017). Health risks and contamination levels of heavy metals in dusts from parks and squares of an industrial city in semi-arid area of China. *International Journal of Environmental Research and Public Health*, 14(8), 886

[10] Jiang, Y., Shi, L., Guang, A. L., Mu, Z., Zhan, H., & Wu, Y. (2018). Contamination levels and human health risk assessment of toxic heavy metals in street dust in an industrial city in Northwest China. *Environmental Geochemistry and Health*, 40(5), 2007-2020

[11] Nasim, I., Akram, T., Nawaz, R., Irshad, M. A., Nasim, M., & Rahman, M. A. U. (2025). Assessment of human health risk of some heavy metals in surface dust of selected urban Areas in Pakistan. *Pollution*, 11(2), 369-383

[12] Patrick, L. (2006). Lead Toxicity, a review of the literature. Part I. Exposure, Evaluation, and treatment. *Alternative Medicine Review*, 11(1).

[13] Campana, O., Sarasquete, C., & Blasco, J. (2003). Effect of lead on ALA-D activity, metallothionein levels, and lipid peroxidation in blood, kidney, and liver of the toadfish Halobatrachus didactylus. *Ecotoxicology and Environmental Safety*, 55(1), 116-125

[14] Choi, J. W., & Kim, S. K. (2005). Relationships of lead, copper, zinc, and cadmium levels versus hematopoiesis and iron parameters in healthy adolescents. *Annals of Clinical & Laboratory Science*, 35(4), 428-434

[15] Singh, D., Nath, K., Trivedi, S. P., & Sharma, Y. K. (2008). Impact of copper on haematological profile of freshwater fish, Channa punctatus. *Journal of Environmental Biology*, 29(2), 253

[16] Effah-Yeboah, E., et al., (2021). Effects of copper and zinc supplementation on haematological, renal and liver function in healthy Wistar rats. *Asian Journal of Immunology*, 5(4), 27-36

[17] Mohajeri, G., Norouzian, M. A., Mohseni, M., & Afzalzadeh, A. (2014). Changes in blood metals, hematology and hepatic enzyme activities in lactating cows reared in the vicinity of a Lead–zinc smelter. *Bulletin of Environmental Contamination and Toxicology*, 92(6), 693-697

[18] Chen, Y., Xu, X., Zeng, Z., Lin, X., Qin, Q., & Huo, X. (2019). Blood lead and cadmium levels associated with hematological and hepatic functions in patients from an e-waste-polluted area. *Chemosphere*, 220, 531-538

[19] Andjelkovic, M., et al., (2019). Toxic effect of acute cadmium and lead exposure in rat blood, liver, and kidney. *International Journal of Environmental Research and Public Health*, 16(2), 274

[20] Baker Jr, et al., (1977). A nationwide survey of heavy metal absorption in children living near primary copper, lead, and zinc smelters. *American Journal of Epidemiology*, 106(4), 261-273

[21] Zhang, X., Yang, L., Li, Y., Li, H., Wang, W., & Ye, B. (2012). Impacts of lead/zinc mining and smelting on the environment and human health in China. *Environmental Monitoring and Assessment*, 184(4), 2261-2273

[22] Lawal, O., Arokoyu, S. B., & Udeh, I. I. (2015). Assessment of automobile workshops and heavy metal pollution in a typical urban environment in sub-Saharan Africa. *Environmental Research, Engineering and Management*, 71(1), 27-35

[23] Fayiga, A. O., Ipinmoroti, M. O., & Chirenje, T. (2018). Environmental pollution in Africa. *Environment, Development and Sustainability*, 20(1), 41-73

[24] Karim, A. Y. A., Azokpota, E., Dovonon, L. F., Moussa, A. K. A., Avocefohoun, A. S., Mama, D., & Sohounhloue, D. C. K. (2023). Assessment of heavy metals (As, Cd, Hg, Pb) in sources of water for human consumption in Sub-Saharan Africa. A literature review. *Science Journal of Chemistry*, 11(1), 1-9

[25] Nyika, J., & Dinka, M. O. (Eds.). (2023). Global industrial impacts of heavy metal pollution in sub-saharan Africa. IGI Global Scientific Publishing. https://doi.org/10.4018/978-1-6684-7116-6

[26] Adimalla, N. (2020). Heavy metals pollution assessment and its associated human health risk evaluation of urban soils from Indian cities. A review. *Environmental Geochemistry and Health*, 42(1), 173-190

[27] Aguilera, A., et al. (2022). Heavy metal contamination (Cu, Pb, Zn, Fe, and Mn) in urban dust and its possible ecological and human health risk in Mexican cities. *Frontiers in Environmental Science*, 10, 854460

[28] Rehman, A., Zhong, S., Du, D., Zheng, X., Ijaz, S., Haider, M. I. S., & Hussain, M. (2025). Unveiling sources, contamination, and eco-human health implications of potentially toxic metals from urban road dust. *Scientific Reports*, 15(1), 10673

[29] Öncü, T., Yazman, M. M., Ustaoğlu, F., Hristova, E., & Yüksel, B. (2025). Source dynamics and environmental risk of street dust as a vector of human exposure to potentially toxic elements in Istanbul, Türkiye. *Scientific Reports*, 15(1), 30550

[30] Jahandari, A. (2020). Pollution status and human health risk assessments of selected heavy metals in urban dust of 16 cities in Iran. *Environmental Science and Pollution Research*, 27(18), 23094-23107

[31] Wu, W., Wu, P., Yang, F., Sun, D. L., Zhang, D. X., & Zhou, Y. K. (2018). Assessment of heavy metal pollution and human health risks in urban soils around an electronics manufacturing facility. *Science of the Total Environment*, 630, 53-61

[32] Cheng, Y., Cui, D., Guo, Z., Hong, W., Li, Y., Lai, C. W., & Xiang, P. (2026). Dermal Exposure to Heavy Metals in Urban Green Space Soils. A Review of Bioavailability, Toxic Mechanisms, and Precision Risk Assessment. *Toxics*, 14(3), 236

[33] Mkhonza, N. P., Zondo, S., & Vilakazi, S. (2026). Toxic heavy metals distribution in urban soils of Africa. a systematic review. *Environmental Monitoring and Assessment*, 198(2), 205

[34] Prieditis, H., & Adamson, I. Y. R. (2002). Comparative pulmonary toxicity of various soluble metals found in urban particulate dusts. *Experimental Lung Research*, 28(7), 563-576

[35] Maina, E. G., Gachanja, A. N., Gatari, M. J., & Price, H. (2018). Demonstrating PM2. 5 and road-side dust pollution by heavy metals along Thika superhighway in Kenya, sub-Saharan Africa. *Environmental Monitoring and Assessment*, 190(4), 251

[36] Samani, M., Ahlawat, Y. K., Golchin, A., Bybordi, A., Sharma, N., & Alikhani, H. A. (2025). Evaluating seasonal health risks of copper, nickel, and chromium in airborne dust. *Air Quality, Atmosphere & Health*, 18(4), 1147-1167

[37] Teschke, R., & Xuan, T. D. (2025). Heavy Metals Like Aluminum, Arsenic, Cadmium, Chromium, Copper, Iron, Lead, Manganese, Mercury, Nickel, and Zinc Polluting the Drinking Water. Their Individual Health Hazards. *International Journal of Molecular Sciences*, 26(23), 11656

[38] Chubaka, C. E., Whiley, H., Edwards, J. W., & Ross, K. E. (2018). Lead, zinc, copper, and cadmium content of water from South Australian rainwater tanks. *International Journal of Environmental Research and Public Health*, 15(7), 1551

[39] El-Kady, A. A., & Abdel-Wahhab, M. A. (2018). Occurrence of trace metals in foodstuffs and their health impact. *Trends in Food Science & Technology*, 75, 36-45

[40] Khanna, P. (2011). Assessment of heavy metal contamination in different vegetables grown in and around urban areas. *Research Journal of Environmental Toxicology*, 5(3), 162

[41] Kasperczyk, A., Prokopowicz, A., Dobrakowski, M., Pawlas, N., & Kasperczyk, S. (2012). The effect of occupational lead exposure on blood levels of zinc, iron, copper, selenium and related proteins. *Biological Trace Element Research*, 150(1), 49-55

[42] Memon, M. S., Ujjan, I. U., Shaikh, M., Arain, S. Q., Naz, A., & Abbasi, H. (2024). Analysis of serum lead, copper, iron, and zinc and hematological parameters in battery smelting workers. assessing lead toxicity. *Biometals*, 37(6), 1529-1535

[43] Olanrewaju Lawal, Samuel Bankole Arokoyu, and Innocent I. Udeh (2015). Assessment of automobile workshops and heavy metal pollution in a typical Sub-Saharan African. *Environmental Research, Engineering and Management,* 71(1), 27-35

[44] Generalova, A., Davidova, S., & Satchanska, G. (2025). The mechanisms of lead toxicity in living organisms. *Journal of Xenobiotics*, 15(5), 146

[45] Al-Attar, A. M. (2011). Vitamin E attenuates liver injury induced by exposure to lead, mercury, cadmium and copper in albino mice. *Saudi Journal of Biological Sciences* 18(4), 395-401

[46] de Souza Teixeira, M. B., Barcarolli, I. F., Saito, M. E., Traverso, S. D., Branco, M. A., Perrone, R. F., & de Moraes, A. N. (2025). Toxicological, Hematological, and Pathological Effects of Acute Copper and Lead Intoxication in Grass Carp (Ctenopharyngodon idella). *Ecotoxicology*, 34(6), 1093-1104

[47] Bhattacharya, S. (2022). Essential trace metals as countermeasure for lead toxicity. *Journal of Environmental Pathology, Toxicology and Oncology*, 41(2)

[48] Henkin, R. I. (1976). Trace metals in endocrinology. *Medical Clinics of North America*, 60(4), 779-797

[49] Stojsavljević, A., & Rovčanin, B. (2021). Impact of essential and toxic trace metals on thyroid health and cancer. a review. *Exposure and Health*, 13(4), 613-627

[50] Mudgal, V., Madaan, N., Mudgal, A., Singh, R. B., & Mishra, S. (2010). Effect of toxic metals on human health. *Open Nutraceuticals J*, 3(1), 94-99

[51] Tipping, E., Jarvis, A. P., Kelly, M. G., Lofts, S., Merrix, F. L., & Ormerod, S. J. (2009). Ecological indicators for abandoned mines, Phase 1. Review of the literature. Environment Agency, Rio House, Waterside Drive, Aztec West, Almondsbury, Bristol, BS32 4UD.

[52] Halpern, E. R., et. al., (2026). Comparative analysis of metal Contaminations and environmentally persistent free radicals in indoor dust from urban and rural households. Environmental perspectives. Environmental Science. *Advances,* 5(3), 866-877

[53] Winecker, R. E., Ropero-Miller, J. D., Broussard, L. A., & Hammett-Stabler, C. A. (2002). The toxicology of heavy metals. Getting the lead out. *Laboratory Medicine*, 33(12), 934-947

[54] Satarug, S., Nishijo, M., Ujjin, P., Vanavanitkun, Y., Baker, J. R., & Moore, M. R. (2004). Evidence for concurrent effects of exposure to environmental cadmium and lead on hepatic CYP2A6 phenotype and renal function biomarkers in nonsmokers. *Environmental Health Perspectives*, 112(15), 1512

[55] KZ, H., & AM, C. (1998). The pollutant effects of copper, zinc and lead on the histological patterns of fish kidney. *Egyptian Journal of Aquatic Biology and Fisheries*, 2(3), 15-41

[56] Mahaffey, K. R. (1995). Nutrition and lead. strategies for pulic health. *Environmental Health Perspectives*, 103(Suppl 6), 191