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REVIEW ARTICLE

LEAD POISONING AMONG CHILDREN IN NIGERIA: A REVIEW OF EVIDENCE, GAPS, AND INTERVENTIONS

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ABSTRACT

Lead poisoning is a public health problem with a long-term impact on children globally. Nigerian children, in particular, are exposed to elevated blood lead levels (BLLs) from artisanal mining, unofficial battery recycling, occupational take-home contamination, leaded paint, legacy leaded gasoline pollution, and many other sources of pollution, despite decades of progress worldwide. This review provides information on the epidemiology, causes, health consequences, and prevention of pediatric lead poisoning in Nigeria, including children under five years of age, who are most impacted by lead neurotoxic effects.

We then synthesized the relevant studies narratively. Exposure context was a significant determinant of reported BLLs. Urban communities without point sources had geometric means of around 4-16 µg/dL, with a few above 10 µg/dL; artisanal-mining groups had means of 35-119 µg/dL, with a typical level of 45 µg/dL. The risks of uncontrolled exposure were highlighted by the 2010-2013 Zamfara mining disaster, which resulted in about 400 child deaths and a median BLL of 119 µg/dL in the most affected area.

Children of workers are also at risk of contamination from Nigeria's vast informal industry, which includes battery recycling, painting and metalworking. Neurodevelopmental damage, anemia, growth retardation, renal failure and, at high exposures, mortality are among the health effects. Systematic prevention is hampered by inadequate infrastructure, poverty, and lax regulation despite chelation therapy, environmental remediation, workplace safety measures, and community education.

Keywords: *lead poisoning, children, Nigeria, blood lead level, artisanal mining, environmental health*

INTRODUCTION

Childhood lead poisoning is a preventable environmental health challenge of the twenty-first century [1]. Lead, a cumulative toxicant, has no known safe exposure limits. Levels as low as 3.5 µg/dL have been linked to impaired learning, cognitive, and behavioral problems in children [1, 2]. Children absorb 40 to 50% of ingested lead compared to 10% in adults, making them vulnerable to acute and lifelong poisoning [3]. Global-burden-of-disease models show that low- and middle-income countries (LMICs), especially Nigeria, bear most of the burden [2].

In Nigeria, millions earn a living in fast-growing informal lead-related jobs (painting, battery recycling, artisanal mining, smelting, auto repair, etc.), frequently without safety regulations or protective equipment [4]. The 2010-2013 Zamfara crisis, the deadliest documented artisanal pediatric lead poisoning, brought Nigeria's lead problem to global attention and exposed critical failures in environmental health surveillance and emergency response [5, 6].

Growing evidence indicates sources other than mining. In one cluster survey in Jos, Nigeria, 70% of young children exceeded the 10 µg/dL action threshold [7, 8]. Children of lead-exposed workers have high blood lead levels (BLLs), suggesting exposure at home [9]. Higher-than-safe BLLs have been found in household dust, soil, water, and food through environmental sampling. Paint assessments show

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that most decorative paints exceed the WHO/UNEP 90 ppm guidelines, with solvent-based home-use paints exceeding by 74% [10].

Childhood lead exposure has multi-organ and multi-system clinical implications. Neurodevelopmental effects at 5 μg/dL include lower IQ, executive function, attention, and behavior [3, 11]. Hundreds of children developed acute encephalopathy, coma, and seizures at Zamfara's extreme exposures [5, 12]. Other effects include anemia due to lower haem production, nutritional impairment, and renal and cardiovascular complications.

Despite these complications, there is a lack of coordination on prevention: there is no monitoring and very few children at risk are being tested, regulatory frameworks are underfunded and poorly implemented, and intervention research is very limited. Low awareness of lead risks is widespread among the general public and health professionals. Because most vulnerable children are aged 5 years and younger, we present evidence to inform policy, practice, and research to address this problem. Our goals are to: (1) identify exposure prevalence and sources; (2) summarize health effects; (3) evaluate the effectiveness of interventions; (4) identify gaps; and (5) provide a framework for this preventable tragedy.

METHODS

Search strategy

We searched PubMed, Google Scholar, Web of Science, African Journals Online, and the WHO Global Health Library for studies on lead poisoning in Nigerian children from January 2004 to February 2025. Exposure and outcome concepts were combined (lead poisoning, lead exposure, blood lead, lead toxicity) with particular contexts (artisanal mining, battery recycling, occupational exposure, environmental contamination, leaded paint, leaded petrol) and geographical and age focus (Nigeria; children, pediatric, under-five, infants). Reference lists were manually searched, and WHO regional office reports, international NGO publications, and Nigerian government health ministry websites were used to search for gray literature.

Eligibility criteria

Studies that: (1) included Nigerian children under 18; (2) measured BLLs or assessed exposure through environmental sampling; (3) looked at health outcomes related to lead; (4) described interventions or policies; or (5) provided relevant contextual information on sources and pathways were included. Studies limited to adults, conducted outside Nigeria, lacking original data, or not available in English were excluded.

Study selection and data extraction

Two reviewers independently screened titles and abstracts, and then assessed the full texts. They resolved disputes by discussion and summarized the data flow in Figure 1. We extracted study characteristics, the sample age range, the BLL measurement method and its values, exposure sources, health outcomes, and interventions using a standardized form completed independently by two reviewers.

Synthesis and quality assessment

We grouped studies by exposure source, region, health outcome, and intervention type using narrative synthesis and not meta-analysis because of the diverse study designs, populations, and settings. Using criteria modified from the Newcastle-Ottawa Scale and the Cochrane Risk-of-Bias tool, we considered representativeness, exposure and outcome assessment, adjustment for confounding, and analysis to judge the quality of evidence. Higher-quality evidence was given more weight, and limitations were emphasized.

EPIDEMIOLOGY OF CHILDHOOD LEAD EXPOSURE IN NIGERIA

Figure 2 is a schematic presentation of the distribution of lead poisoning in Nigeria. Symbols distinguish artisanal-mining areas with documented outbreaks or high exposure (e.g., Zamfara and Niger states), occupational-exposure studies (e.g., Lagos, Ibadan, and Kano), urban environmental studies without specific point sources, and intervention sites. The map illustrates the geographic concentration of research in south-western Nigeria and Zamfara State, with large areas of the country unstudied.

Overview of blood lead levels

Major studies are presented in Table 1. Study results vary widely across sources, contexts, and populations. Typically, geometric means are 4-16 μg/dL in urban populations without high-intensity sources, and most are much higher [8, 13] than in high-income countries (i.e., less than 2 μg/dL), indicating that background exposure is still high even without point sources. The levels are even higher where there are sources: children in mining villages have an average of 35-119 μg/dL, some have much higher values than that, and many are in need of urgent chelation [5, 6]; children of occupationally exposed parents have an average of several-fold higher means [9], and there is a difference between the 18-35 μg/dL recorded from informal battery recycling or smelting [14]. Only a few years of longitudinal data are available, and there is no reliable way to know time-series trends. Nigeria's leaded-gasoline phase-out in 2008 probably reduced exposure from that source, but mining, informal

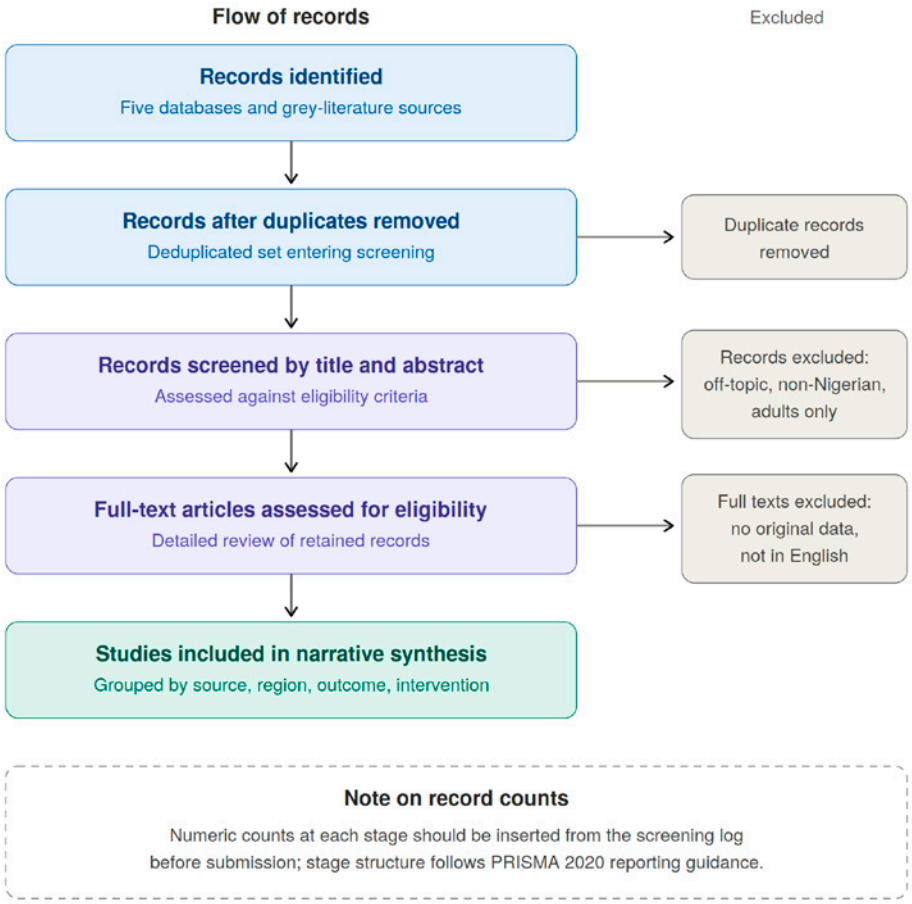

Figure 1. Flow of records through the review (PRISMA-style diagram)

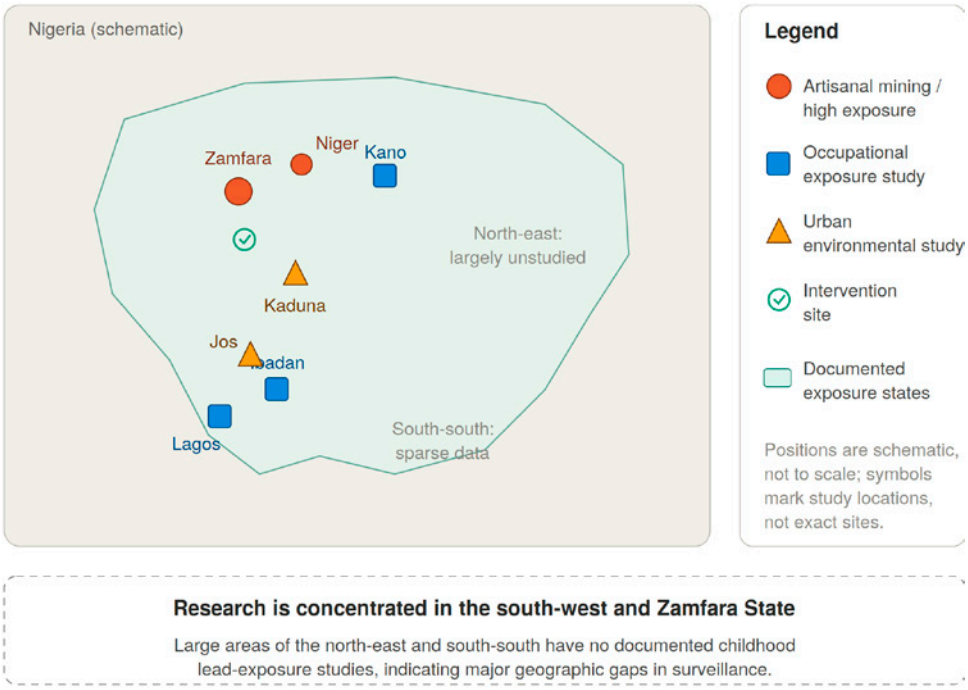

Figure 2. Geographic distribution of documented childhood lead-exposure studies in Nigeria, 2004-2025

Sources: Most Nigerian studies included in this review (1997-2025), especially [5-13, 15-18, 20, 21] (see references). Symbols distinguish artisanal-mining sites with documented outbreaks (triangles), occupational exposure studies (circles), urban environmental studies (squares), and intervention site (marked). Geographic locations are approximate; the map is a schematic representation only and does not reflect official administrative boundaries.

Table 1. Summary of blood lead studies in Nigerian children

Study	Location	Year	N	Age range (yr)	Design/exposure	GM or median BLL (µg/dL)	% ≥ 10 µg/dL	Key findings
Dooyema et al. [5]	Zamfara State	2010	204	0-5	Cross-sectional; artisanal gold mining	Median 119	~100%	Outbreak investigation; > 400 deaths
Tirima et al. [6]	Zamfara (Bagega)	2011-2013	535	0-10	Longitudinal; artisanal gold mining	71 (pre-remediation)	91.4%	~74% BLL reduction after remediation
Greig et al. [12]	Zamfara State	2010-2011	972	≤ 5	Retrospective: artisanal gold mining	All ≥ 45 (inclusion)	100%	Neurological features linked to higher BLL
Wright et al. [7]	Jos	2003-2004	275	0.5-3	Cross-sectional, urban environmental	Mean 8.7 ± 5.7 µg/dL	~70%	Car ownership/ tarred-road residence key risks
Adebamowo et al. [8]	Lagos/Ibadan	2005	40	1-5	Cross-sectional, urban environmental	NR (a qualitative study)	NR	Knowledge, attitudes, and exposure risk factors
Nriagu et al. [9]	Kaduna	1995	87	1-6	Cross-sectional; urban/traffic	Mean 10.6	NR, but 2% > 30 µg/dL	Highest in 5-year-olds

NR – not reported; BLL – blood lead level; GM – geometric mean. Dashes indicate values not reported. The Zamfara median of 119 µg/dL refers to Bagega at outbreak; the Tirima et al. [6] figure is a pre-remediation cohort value.

recycling, occupational exposure, and leaded paint are still present and could appear to be increasing or getting worse; therefore, if we do not get enough information, we cannot predict trends clearly.

Zamfara mining disaster

The most severe recorded childhood lead poisoning event occurred in Zamfara State from 2010 to 2013. Artisanal gold ore, rich in lead sulfide, was crushed in or next to homes, contaminating soil, dust, water, and food [6]. Investigations by the Nigerian government and MSF revealed lead poisoning; the first deaths were reported in March 2010, and over 400 child deaths were eventually recorded [5].

The median BLL in Bagega was 119 μg/dL (range 10-320 μg/dL), and most children were at least 45 μg/dL at the chelation level [5]. Soil lead levels exceeded 150,000 ppm in compounds and were far above the US EPA residential soil guideline of 400 ppm, which is used here as an internationally recognized benchmark, with no enforced Nigerian standard [5, 6].

Seizures, altered awareness, acute encephalopathy, and severe anemia were some of the symptoms of the disease in 972 children with venous BLLs ≥ 45 μg/dL, and neurological disorders were also in high correlation with higher concentrations [12]. Case-fatality among under-fives reached around 25% in the poorest areas before emergency care, and most were severely neurodevelopmentally damaged [5].

Mass screening, chelation of BLLs ≥ 45 μg/dL, environmental remediation, relocation of ore processing, and health education were part of the response, carried out by the Federal Ministry of Health, MSF, US CDC, WHO, and partners. While levels remained above safe criteria and re-contamination occurred when mining resumed [15], the Bagega median had dropped by about three-quarters from 119 to about 30 μg/dL [6]. The long-term effects of BLLs in survivors are still being investigated. Zamfara revealed systemic flaws in Nigeria's informal sector, including poor rural health facilities, lack of surveillance, lengthy response times, inadequate mining regulation, and low public awareness.

Occupational exposure and take-home contamination

Millions of people in Nigeria's informal sector work in lead-exposed jobs outside mining, putting their children at risk of exposure at home (Table 2). With several operations in Nigeria, informal lead-acid battery recycling is one of the biggest global sources of exposure; workers disassemble batteries in residential areas without protection, and children in the neighbourhood have high BLLs and elevated household dust lead levels [4, 14].

Take-home contamination from spray painters in African towns has been linked to increased child BLLs; artisan painters who use lead-pigmented spray paints and industrial paints carry lead home on their skin, hair, and clothing and store equipment close to dwelling areas [9]. BLLs are raised in children by workers who handle lead alloys and solders through informal metalworking, welding, and smelting.

Other understudied methods include garbage collection, lead-glazed ceramics, traditional medicine preparation, and recycling electronic waste. The pathway is facilitated by children's hand-to-mouth conduct and intimate parental touch, as well as dust on unmodified clothing, contaminated skin, hair, cars, and shared laundry. Longer, more intense exposure and the lack of washing facilities or protective clothing increase risk; workplace-home separation, changing and washing before returning home, and a clean home provide protection.

Environmental lead sources

According to Olawoyin et al. [16], the use of lead petrol up until 2008 resulted in decades of soil contamination along highways and in cities, which persisted through direct contact and resuspended dust. Urban soil lead levels are higher in areas with heavy traffic. Decorative paints often go beyond safety regulations: 74% of solvent-based home-use samples (40 of 54) exceeded 90 ppm, with some exceeding tens of thousands of ppm [10]. Children are exposed to paint dust, chips, and hand-to-mouth contact.

The Standards Organization of Nigeria set a 90 ppm limit, although it is not strictly enforced (section 'Regulatory and policy interventions') [17]. Lead enters drinking water from contaminated sources, lead-soldered connections, and corroding pipes; this is especially dangerous for neonates fed formula. There are few studies, but traditional medicines, holding containers and cans, and crop absorption can all contaminate food. Consumer products such as imported toys, kohl cosmetics, lead-glazed pottery, some vinyls, costume jewelry, and traditional treatments have modifiable, regulation-friendly channels.

There are regional differences in lead levels. In northwest mining regions (Zamfara, Niger, Katsina), there are the highest elevations; metropolitan areas (Lagos, Ibadan, Kano, Port Harcourt) have moderate elevations from a variety of sources; rural regions, apart from industry and mining, tend to have lower elevations; but very few rural surveys exist [8]. The relationship seems to be U-shaped because of socioeconomic patterns [8]. Wealthier families are also at risk from imported goods, renovations, high-lead paints, poor living conditions with paint deterioration, informal industry, and hazardous work (mothers are

Table 2. Sources of childhood lead exposure in Nigeria

Exposure source	Extent	Typical BLL range (μg/dL)	Primary pathway	Population at risk	Intervention priority
Artisanal mining	Localized (Zamfara, Niger, Katsina)	35-119	Environmental contamination: food and water	Children in mining villages	High – remediation, mining regulation [5, 6]
Occupational take-home	Widespread, urban/peri-urban	12-35	Contaminated clothing, skin, and household dust	Children of lead-exposed workers	High – occupational safety, hygiene [4, 9]
Lead-based paints	Nationwide	5-15 (contribution)	Hand-to-mouth contact; paint dust/chips	Children in painted environments	High – paint regulation and enforcement [17]
Leaded-gasoline legacy	Urban areas nationwide	3-12 (contribution)	Contaminated soil; suspended dust	Urban children, especially near roads	Medium – soil remediation in hotspots [16]
Battery recycling	Urban/peri-urban clusters	18-32	Environmental contamination; dust	Children near recycling sites	High – relocation, regulation, remediation [4, 14]
Contaminated water	Variable by location	2-8 (contribution)	Drinking-water consumption	Children with contaminated supplies	Medium – water system improvement [16]
Contaminated food	Nationwide, variable	2-6 (contribution)	Food consumption	All children, higher with local produce	Low-medium – food-safety monitoring [19]
Consumer products	Nationwide, sporadic	Variable	Product use: hand-to-mouth	Children using contaminated products	Medium – product regulation and testing [18]

BLL – blood lead level. "Contribution" indicates the source adds to the total body burden rather than acting as a dominant point source. BLL ranges represent approximate values reported in Nigerian studies and may vary by exposure intensity and population.

more likely to work), as well as nutrition (iron and calcium deficiency) [18]. BLLs are typically lower in infants unless they are exposed through formula, water, or breast milk; levels drop after 3 to 4 years, even with continued cumulative exposure [19]. Sex differences are negligible, and slightly higher levels are reported in boys [20]. There is little research on seasonal variation; dust exposure may increase during dry seasons, and water pollution may increase during wet seasons [21].

HEALTH IMPACTS OF CHILDHOOD LEAD EXPOSURE

Health effects of lead exposure in Nigerian children are described in Tabe 3.

Neurodevelopmental effects

Neurotoxicity is one of the most serious impacts of lead in childhood, and its long-term effects are profound. Lead alters neurotransmitter systems (dopamine, glutamate, GABA), synaptic plasticity, neuronal migration and neuronal differentiation, calcium signaling, oxidative stress and gene expression, and is associated with increased stress and impaired gene expression.

Limited neuropsychological evaluations on Nigerian children show some effects consistent with international studies. Evidence from Zamfara and elsewhere indicates high blood lead levels are linked to intellectual, behavioral, and motor deficits. Zamfara survivors experienced intellectual disability, motor issues, seizures, behavioral disorders, and acute encephalopathy at a median of 119 μg/dL, with long-term epilepsy, motor impairment, and severe intellectual disability.

At 10-40 μg/dL of blood lead, subtle but significant effects occur: each 10 μg/dL increase links to a 3-5 point IQ drop below 5 μg/dL [3, 11]. These include impacts on verbal ability, attention, executive function, memory, visuospatial skills, behavior (aggression,

Table 3. Health effects of lead exposure in Nigerian children: summary of evidence

Health effect	Evidence strength	Key findings from Nigerian studies	Typical BLL range for effect	Clinical significance
Neurodevelopmental impairment	Strong	Zamfara survivors: intellectual disability, motor dysfunction, seizures; developmental delay at moderate exposure [3, 11, 12]	Effects < 5 µg/dL; severe > 45 µg/dL	High – irreversible, lifelong
Anaemia	Strong	High prevalence in high-exposure groups; dose-response with hemoglobin [5]	Effects > 10 µg/dL; severe > 45 µg/dL	High – compound malnutrition burden
Growth impairment	Moderate	Mixed findings; associations with stunting in some studies [22]	Associations > 10 µg/dL	Moderate – hard to isolate from malnutrition
Renal dysfunction	Moderate	Documented at extreme BLL in Zamfara; subclinical effects possible at moderate BLL [12]	Clinical effects > 45 µg/dL	Moderate – long-term concerns uncertain
Acute encephalopathy	Strong	Seizures, coma, cerebral edema in severe Zamfara cases [12]	Occurs > 70-100 µg/dL	Very high – life-threatening emergency
Mortality	Strong	> 400 deaths in Zamfara; ~25% case-fatality in worst-affected villages [5]	Deaths > 70 µg/dL	Very high – preventable deaths

BLL – blood lead level.

hyperactivity, impulsivity, anxiety), movement, hearing, and social and academic problems.

Chelation reduces blood lead and might slow progression but doesn't restore lost function; effects are permanent. Lead damage lowers human capital and productivity. In low- and middle-income countries, economic models show substantial impact, but Nigeria's specific impact is not well assessed and needs further study [2].

Hematological effects

Lead buildup increases protoporphyrin, lowers hemoglobin, and causes anemia by inhibiting enzymes like ferrochelatase and δ-aminolaevulinic acid dehydratase. Studies in Nigeria show anemia worsens at high Zamfara blood lead levels and shows a dose-response with moderate exposure [5]. Lead-induced anemia raises iron shortages, malaria, parasites, and hemoglobinopathies, increasing mortality alongside malaria, pneumonia, and diarrhea. It also causes exhaustion, weakened immunity, delayed growth, and impaired cognition. Iron-deficient children absorb more lead due to upregulated DMT-1, which transports iron and lead, possibly creating a loop where iron supplements offer dual benefits.

Growth and nutritional effects

Lead may hinder growth by disrupting hormone signaling, bone health, and appetite, and indirectly by causing chronic illness and anemia [22]. Data in Nigeria is mixed: some studies show little correlation after adjusting for confounders, while others link elevated blood lead to stunting. Although lead's small effect is hard to detect amid high stunting rates, Nigeria's large pediatric population means many children are affected even by minor effects [22].

Renal effects

Lead causes impaired kidney function, hypertension, and tubular dysfunction, with effects known in adults but less studied in children [12]. In Nigeria, low-level exposure rarely causes renal

disease, but subclinical effects can develop. High Zamfara blood lead levels are linked to renal issues like increased creatinine, proteinuria, and electrolyte abnormalities, with uncertain long-term impacts [12].

Cardiovascular effects

Lead's cardiovascular effects are well known in adults but less so in children [1]. Long-term blood pressure rise links to blood lead levels, with adults more affected, associated with stroke, cardiovascular disease, and hypertension [1].

Other health effects

Other effects include gastrointestinal issues (abdominal pain, constipation, vomiting) at higher blood lead levels; immunological effects increasing infection risk; endocrine disruption affecting thyroid and puberty; dental effects like delayed eruption and more caries [1].

Mortality

At extreme Zamfara blood lead levels, lead poisoning caused over 400 child deaths from encephalopathy, cerebral edema, convulsions, and brainstem dysfunction [5]. Lead-induced anemia and immune suppression may raise mortality, warranting studies on lead's interaction with malaria, pneumonia, and diarrhea. Lower lead levels may also indirectly worsen anemia, immune issues, and health.

INTERVENTIONS AND THEIR EFFECTIVENESS

Chelation therapy

In Nigeria, agents such as $CaNa_2EDTA$, dimercaptosuccinic acid (DMSA), and dimercaprol (BAL) are used to bind lead for urinary excretion. Chelation is the main pharmacological treatment for high BLLs. The most important pediatric chelation experience in Africa was the Zamfara response: the median Bagega dropped from 119 to about 30 µg/dL, with symptoms resolved after MSF; the Federal Ministry of Health and partners screened and treated children with BLLs greater than 45 µg/dL, using $CaNa_2EDTA$ at each follow-up (Figure 3) [6].

Limitations include rebound when children return to contaminated homes, requiring simultaneous remediation. Adverse effects are minor and manageable with monitoring. Remote delivery is challenging, and chelation does not reverse established damage. Results are sparse outside Zamfara, with limited agents and services and high care costs. Recommended chelation for ≥ 45 µg/dL, consideration at 20-44 µg/dL if symptomatic or environmental control fails, and environmental priority below 20 µg/dL [6].

Environmental remediation

Contaminated materials, dust, and soil must be removed or contained to prevent rebound and new cases.

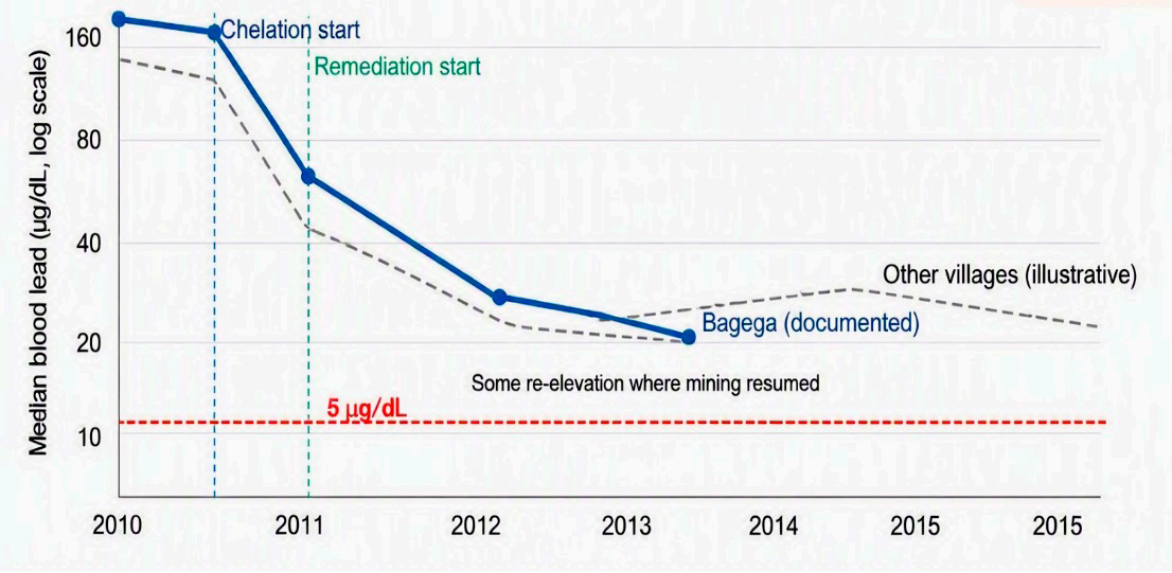

Figure 3. Temporal trends in median blood lead levels among children in Zamfara State mining villages, 2010-2015

Reading the figure: The Bagega trajectory (solid blue) reflects reported values, with a median of 119 µg/dL at the outbreak, falling below 30 µg/dL after remediation. The other-village line (dashed grey) illustrates the general pattern, not the reported point estimates. The y-axis is on a log scale; levels remained above the 5 µg/dL reference line throughout the follow-up. Separate lines for individual villages, with vertical markers indicating the timing of chelation therapy initiation and environmental remediation start. Median levels declined by roughly 70-75% from peak values following intervention but remained above the 5 µg/dL reference line, with some re-elevation where mining resumed. Data from Tirima et al., 2018 [6], and Nigerian Federal Ministry of Health surveillance reports.

Zamfara's efforts included removing contaminants, replacing soil, decontaminating buildings, changing ore processes, and educating locals, which lowered soil lead levels and reduced rebound in remediated areas [6, 15]. Challenges included vast contamination, safe soil management, community acceptance, temporary relocation, and recontamination risks. Elsewhere, remediation is limited, and pollution from legacy petrol remains unaddressed due to a lack of legal, technical, and financial resources.

Occupational safety interventions

Reducing occupational exposure and contamination is logical but limited in the informal sector. Components include protective gear, workplace hygiene, training, engineering controls, work-home separation, and worker BLL monitoring. Pilot programs with equipment and washing facilities for battery recyclers seem feasible and could reduce exposure, but adherence suffers from heat, cost, and behavioral issues; rigorous evaluations are needed. The main challenge is lack of enforcement and economic constraints, requiring innovations like subsidies, community monitoring, artisan engagement, consumer pressure, and formalization.

Regulatory and policy interventions

While Nigeria's source-directed regulation has authority, enforcement is weak. The Standards Organization set a 90 ppm paint limit [17], but most samples exceed it, hindered by limited testing, weak inspections, manufacturers' interests, low consumer awareness, and non-compliant imports [10]. International success shows compliance improves with clear standards, mandated testing, surveillance, sanctions, public awareness, and reformulation support. Lead industry regulations are covered by Nigeria's Factories Act, mainly for the official sector, but rarely enforced. Its informal extension may need voluntary certification, community monitoring, and incentives. Environmental laws exist but lack monitoring, requiring investments in labs, staff, and infrastructure.

Screening and surveillance

Although systematic screening and surveillance are essential, they are mainly used in research and emergencies in Nigeria due to the lack of a national program. Barriers include costly testing, insufficient trained personnel, poor follow-up, and low awareness. Strategies for expansion involve integrating testing into routine child health services in high-risk areas, using point-of-care devices, targeting high-risk populations (like mining and recycling communities), establishing low-cost laboratories, and implementing national surveillance to track levels and detect clusters. Donor and NGO efforts support context-specific screening, but sustainable domestic capacity remains limited. Less invasive methods should be promoted, and extended screening should include schools, clinics for under-5s, outpatient centers, immunization sites, and displaced camps.

Community education and mobilization

Without addressing structural barriers, education that raises knowledge of risks and prevention is crucial but insufficient. Effective programs use community meetings, skilled community health workers, information in the local language, safer practice demonstrations, leader engagement, and connections to tangible resources. Zamfara's extensive education was important to the acceptance of remediation and behavior change. Education must be combined with institutional interventions that support protective behaviors, since families frequently know protective measures but cannot afford them; for example, a painter may know to shower and change clothes but lack the facilities.

Nutritional interventions

Given lead-nutrition interactions, nutritional measures offer supplementary benefits: iron supplementation reduces absorption and treats anemia; calcium may reduce absorption and bone mobilization; and adequate intake of iron, calcium, zinc, and vitamin C is associated with lower BLLs. In Nigeria, where malnutrition and micronutrient deficiencies are common, these align with broader child health goals but should complement, not replace, source elimination.

Minimizing lead exposure in pregnancy

Lead exposure during pregnancy poses severe risks to child health, potentially causing adverse birth outcomes, because lead crosses the placenta [23-25]. Prenatal exposure can compromise fetal development, increasing the risk of structural abnormalities like neural tube defects and undermining a child's future neurological and physiological potential [26]. Addressing these risks requires clinical management and public health interventions to identify and eliminate sources of exposure [27]. Additionally, targeted nutritional interventions during pregnancy, such as calcium, iron, zinc, magnesium, Vitamin C, and Vitamin E, may serve as a supportive strategy, as certain nutrients have been evaluated for their potential to help lower maternal blood lead levels [28].

KNOWLEDGE GAPS AND RESEARCH PRIORITIES

Epidemiological gaps

Gaps include lack of nationally representative BLL data; lack of quantification of paint, water, food, and products contributions; poor temporal trends; lack of prenatal and infant data; and non-assessed geographic variation [29].

Priority: nationally representative biomonitoring, longitudinal cohorts, source apportionment (isotopic analysis and modeling), prenatal and early-life studies using cord and infant blood, and increased surveillance in neglected areas. Addressing these epidemiological gaps is a core objective of Pillar 2 ('Surveillance and screening systems').

Health-effects research gaps

Gaps include limited standardized neurodevelopmental assessment, inadequate longitudinal follow-up, little investigation below 10 µg/dL, inadequate assessment of lead interactions with malaria, malnutrition and infection, and little economic analysis [18].

Priority: longitudinal neurodevelopmental studies using validated instruments (e.g., Bayley, Wechsler), low-BLL effect studies, interaction studies, economic-burden studies and long-term follow-up of Zamfara survivors. The research priorities identified here inform Pillar 3 ('Clinical services and treatment') and provide the evidence base for Pillar 1 ('Regulatory strengthening and enforcement').

Intervention research gaps

Gaps include limited evaluation of effectiveness in Nigerian contexts, lack of comparative effectiveness research, poorly understood implementation barriers, limited economic assessment and insufficient acceptability and sustainability.

Priorities: randomized or quasi-experimental studies of household cleaning, occupational safety and education, comparative effectiveness and implementation science research; cost-effectiveness analyses and community-based design of contextually appropriate interventions. These gaps map directly to Pillar 4 ('Environmental remediation and source control') and Pillar 5 ('Occupational safety and informal sector').

Policy and systems gaps

Gaps include lack of knowledge of regulatory barriers, under-investigation of economic and political capacity, insufficient assessment of health system capacity, and lack of stakeholder representation [30]. Policy analysis of regulatory frameworks; political economy analysis of paint and occupational safety enforcement; health systems research on screening, treatment, and prevention capacity; and stakeholder engagement research to inform policy. Closing these systemic gaps is the principal aim of Pillar 1 ('Regulatory strengthening and enforcement') and the cross-cutting governance mechanisms described in section 'Cross-cutting elements'.

A STRUCTURED FRAMEWORK FOR ACTION

Reducing childhood lead exposure in Nigeria requires a multi-faceted response that integrates prevention, early detection, treatment, environmental restoration, and policy development (Figure 4). Based on evidence on the ground, we developed six complementary strategic pillars supported by cross-cutting governance mechanisms (Figure 5).

Pillar 1: Regulatory strengthening and enforcement

Aim: Control lead exposure at source. The focus of primary prevention still needs to be strengthening and enforcing regulations regarding lead-containing products. The priority is to establish a national paint lead limit of ≤ 90 ppm, to make workers' lives safer and the environment safer, to regulate lead in the products we use (toys, cosmetics, ceramics) and to make laboratory technology more efficient for product testing, inspection, and regulatory enforcement [31]. Appropriate penalties for non-compliance and incentives to produce and use safer products should be present in implementation.

Pillar 2: Surveillance and screening systems

The objective is to detect exposure early and take action in public health. A national surveillance system is necessary to identify the real burden of childhood lead exposure and identify high-risk groups [29]. This will include representative biomonitoring, targeted screening of children in high-risk populations, reliable laboratory services, electronic reporting systems, and routine environmental monitoring. Standardized follow-up protocols would support early intervention and timely response to emerging exposure clusters.

Pillar 3: Clinical services and treatment

Objective: Improve access to timely diagnosis and evidence-based care. Health systems must be better equipped to identify, diagnose, and respond to childhood lead poisoning with a common set of clinical guidelines, trained healthcare workers to manage it, and better access to diagnostic services, chelation therapy and treatment, and specialist referral pathways [32]. Long-term follow-up should be done to address not only lead toxicity but also associated

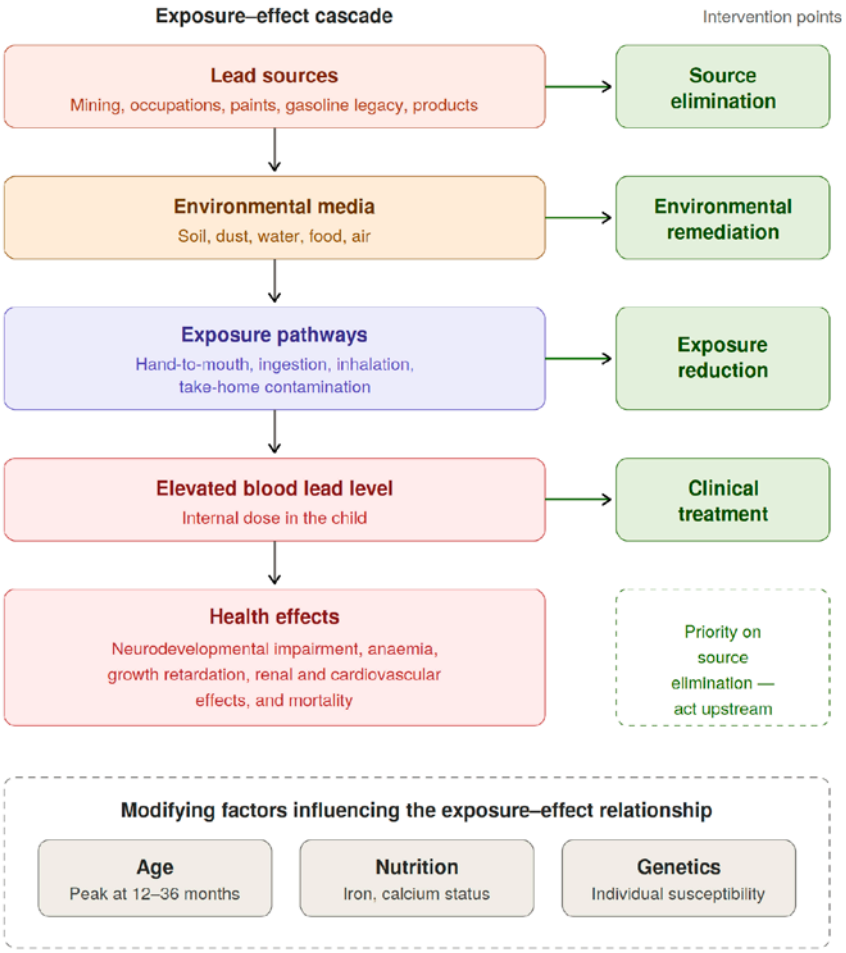

Figure 4. Conceptual framework of exposure pathways and intervention points

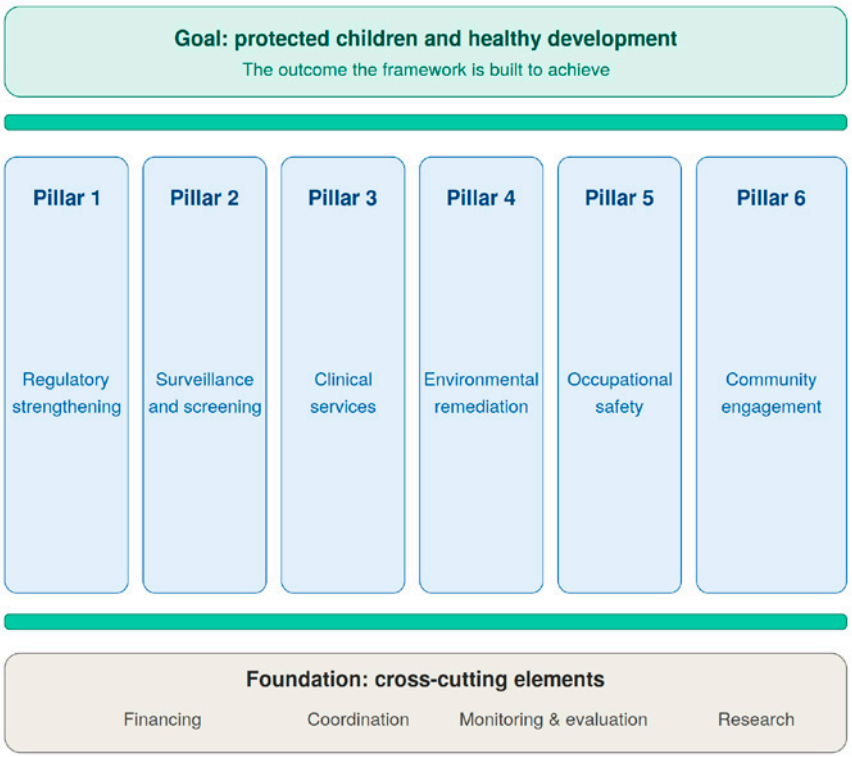

Figure 5. The six-pillar framework for addressing childhood lead poisoning in Nigeria

diseases (anemia, nutritional deficiencies, and neurodevelopmental dysfunction).

Pillar 4: Environmental remediation and source control

Goal: Eliminate environmental contamination and prevent repeat exposure. To prevent pollution for sustainable prevention, we need to remedy the environment (i.e., in mining communities, informal battery recycling sites, and urban areas). Environmental interventions should be complemented by engineering controls, safer industrial practices, and the removal of lead-containing materials in the future in order to reduce the risk of environmental exposure [33].

Pillar 5: Occupational safety and informal sector

Objective: Reduce occupational exposure and prevent take-home contamination. Occupational exposure is a major source of childhood lead poisoning via contamination into the home [6]. Preventive measures must include better workplace hygiene, access to appropriate personal protective equipment, education for workers in the informal sector and artisan associations to work safely, and regular monitoring of workplace exposure, if possible.

Pillar 6: Community engagement and education

Goal: Increase public awareness and prevention. Community involvement is critical for prevention to be sustainable [34]. Public education should focus on common sources of lead exposure, associated health risks, and practical preventive measures. Community health workers, schools, traditional and religious leaders, and local organizations can also play a key role in risk communication, behavior change, and early health-seeking behavior in the affected families.

Cross-cutting elements

Implementing these pillars needs ongoing political support, sufficient domestic funding, multisectoral cooperation, and robust monitoring. Partnerships among government, academia, professional groups, civil society, and development partners are vital for program success. Research should fill knowledge gaps, assess interventions in Nigeria, and generate evidence for policy and resource decisions.

Feasibility/prioritization of pillars under resource constraints

Given the resource constraints endemic to Nigeria's health system, simultaneous implementation of all six pillars is neither realistic nor necessary as a first step. A tiered approach is recommended: Pillars 1 (regulatory strengthening), 2 (surveillance and screening), and 6 (community engagement) offer comparatively high impact at lower initial cost and should be treated as priority entry points. Pillars 3 (clinical services) and 4 (environmental remediation) require sustained financing and external partnership, and should be developed progressively; Pillar 5 (occupational safety) can advance in parallel through existing labour-inspection frameworks at minimal additional cost.

CONCLUSIONS

Childhood lead poisoning in Nigeria remains a severe yet preventable public health crisis. Children face dangerously high blood lead levels from artisanal mining, leaded products, and legacy contamination, leading to cognitive impairment, anemia, stunted growth, and death. While acute disasters like Zamfara highlight the risks, chronic low-to-moderate exposure is widespread and equally damaging. This burden reflects broader environmental health threats driven by rapid urbanization, weak regulation, poverty, and unsafe housing.

Evidence shows interventions work: chelation and remediation saved lives in Zamfara, occupational safety and education pilots proved effective, and global paint regulation demonstrates prevention is possible. A coordinated six-pillar approach (strengthening regulation, expanding surveillance, improving occupational and environmental controls, and integrating prevention into child health services) can shift Nigeria from isolated projects to systematic protection.

The stakes are high: each year's exposure erodes lifelong cognition and costs more than prevention. Although research gaps exist, evidence is sufficient. Political will, resources, and proven strategies are needed to protect Nigeria's children.

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Conflicts of interest

The authors declare no conflicts of interest.

AI tools

AI tools were carefully used to refine outlines, schematic diagrams and figures, and English language grammar. The authors are responsible for the manuscript.

REFERENCES

1. World Health Organization. Lead poisoning and health (fact-sheets) [Internet]. Geneva: WHO; 2023 [cited 2026 Jul 19]. Available from: https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health.
2. Larsen B, Sánchez-Triana E. Global health burden and cost of lead exposure in children and adults: a health impact and economic modelling analysis. Lancet Planet Health. 2023;7(10):e831-e840. doi: 10.1016/S2542-5196(23)00166-3.
3. Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ Health Perspect. 2005;113(7):894-9. doi: 10.1289/ehp.7688. Erratum in: Environ Health Perspect. 2019;127(9):99001. doi: 10.1289/EHP5685.
4. Ericson B, Landrigan P, Taylor MP, Frostad J, Caravanos J, Keith J, et al. The Global Burden of Lead Toxicity Attributable to Informal Used Lead-Acid Battery Sites. Ann Glob Health. 2016;82(5):686-699. doi: 10.1016/j.aogh.2016.10.015.
5. Dooyema CA, Neri A, Lo YC, Durant J, Dargan PI, Swarthout T, et al. Outbreak of fatal childhood lead poisoning related to artisanal gold mining in northwestern Nigeria, 2010. Environ Health Perspect. 2012;120(4):601-7. doi: 10.1289/ehp.1103965.
6. Tirima S, Bartrem C, von Lindern I, von Braun M, Lind D, Anka SM, et al. Food contamination as a pathway for lead exposure in children during the 2010-2013 lead poisoning epidemic in Zamfara, Nigeria. J Environ Sci (China). 2018;67:260-272. doi: 10.1016/j.jes.2017.09.007.
7. Wright NJ, Thacher TD, Pfitzner MA, Fischer PR, Pettifor JM. Causes of lead toxicity in a Nigerian city. Arch Dis Child. 2005;90(3):262-6. doi: 10.1136/adc.2003.043562.
8. Adebamowo EO, Agbede OA, Sridhar MK, Adebamowo CA. An examination of knowledge, attitudes and practices related to lead exposure in South Western Nigeria. BMC Public Health. 2006;6(1):82. doi: 10.1186/1471-2458-6-82.
9. Nriagu J, Oleru NT, Cudjoe C, Chine A. Lead poisoning of children in Africa, III. Kaduna, Nigeria. Sci Total Environ. 1997;197(1-3):13-9. doi: 10.1016/s0048-9697(96)05408-3.
10. Sustainable Research and Action for Environmental Development/International Pollutants Elimination Network. Lead in solvent-based paints for home use in Nigeria [Internet]. Nigeria: SRADev/IPEN; 2017 [cited 2026 Jul 19]. Available from: https://ipen.org.
11. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. N Engl J Med. 2003;348(16):1517-26. doi: 10.1056/NEJMoa022848.
12. Greig J, Thurtle N, Cooney L, Ariti C, Ahmed AO, Ashagre T, et al. Association of blood lead level with neurological features in 972 children affected by an acute severe lead poisoning outbreak in Zamfara State, northern Nigeria. PLoS One. 2014;9(4):e93716. doi: 10.1371/journal.pone.0093716.
13. Adeyeye EI, Faleye FJ, Omolaja TM. Metal concentration of liquid effluents and surroundings of a pharmaceutical industry. Bull Chem Soc Ethiop 2007;21(1):23-31. doi: 10.5555/20073213054.
14. Gottesfeld P, Pokhrel AK. Review: Lead exposure in battery manufacturing and recycling in developing countries and among children in nearby communities. J Occup Environ Hyg. 2011;8(9):520-32. doi: 10.1080/15459624.2011.601710.
15. Bashir M, Umar-Tsafe N, Getso K, Kaita IM, Nasidi A, Sani-Gwarzo N, et al. Assessment of blood lead levels among children aged ≤ 5 years – Zamfara State, Nigeria, June-July 2012. MMWR Morb Mortal Wkly Rep. 2014;63(15):325-7. doi: 10.5555/20143163494.
16. Olawoyin R, Oyewole SA, Grayson RL. Potential risk effect from elevated levels of soil heavy metals on human health in the Niger delta. Ecotoxicol Environ Saf. 2012;85:120-30. doi: 10.1016/j.ecoenv.2012.08.004.
17. Standards Organisation of Nigeria. Nigerian industrial standard for paints and varnishes – limitation of lead content (NIS 269:2017) [Internet]. Abuja: SON; 2017. Available from: https://son.gov.ng/wp-content/uploads/2022/08/SNTC.pdf.
18. Madaki PD, Frazzoli C, Ibibama JO, Tengbeh AS, Orisakwe OE. Environmental Burden of Lead and Public Health Issues in Nigeria: A Systematic Review of Emerging Evidence. Biol Trace Elem Res. 2026;204(9):7103-7120. doi: 10.1007/s12011-026-05175-6.
19. Igweze ZN, Ekhator OC, Orisakwe OE. Lead and cadmium in infant milk and cereal based formulae marketed in Nigeria: a probabilistic non-carcinogenic human health risk assessment. Rocz Panstw Zakl Hig. 2020;71(3):303-11. doi: 10.32394/rpzh.2020.0129.
20. Bello O, Naidu R, Rahman MM, Liu Y, Dong Z. Lead concentration in the blood of the general population living near a lead-zinc mine site, Nigeria: Exposure pathways. Sci Total Environ. 2016;542(Pt A):908-14. doi: 10.1016/j.scitotenv.2015.10.143.
21. Ani NC, Ucheana IA, Ani CC, Abugu HO, Ihedioha JN, Okoye COB. Seasonal variability of soil metal contamination and risks in a Pb-Zn mining site, Southeastern Nigeria. Environ Geochem Health. 2025;47(12):557. doi: 10.1007/s10653-025-02855-5.
22. Kaji M, Nishi Y. Lead and growth. Clin Pediatr Endocrinol. 2006;15(4):123-8. doi: 10.1297/cpe.15.123.
23. Vigeh M, Yokoyama K, Sahebi L. Prenatal lead exposure and risk of Pregnancy-Induced hypertension: A systematic review and Meta-Analysis. BMC Pregnancy Childbirth. 2025;25(1):989. doi: 10.1186/s12884-025-08131-9.
24. Issah I, Duah MS, Arko-Mensah J, Bawua SA, Agyekum TP, Fobil JN. Exposure to metal mixtures and adverse

pregnancy and birth outcomes: A systematic review. Sci Total Environ. 2024;908:168380. doi: 10.1016/j.scitotenv.2023.168380.
25. Ou Y, Han J, Wei Y, Gao X. Meta-analysis of maternal exposure to heavy metals (lead, cadmium, mercury, chromium) and adverse pregnancy outcomes. BMC Pregnancy Childbirth. 2025;26(1):7. doi: 10.1186/s12884-025-08507-x.
26. Eaves LA, Choi G, Hall E, Sillé FCM, Fry RC, Buckley JP, et al. Prenatal Exposure to Toxic Metals and Neural Tube Defects: A Systematic Review of the Epidemiologic Evidence. Environ Health Perspect. 2023;131(8):86002. doi: 10.1289/EHP11872.
27. World Health Organization. Lead poisoning and health [Internet]. Geneva: WHO; 2024 [cited 2026 Jul 19]. Available from: https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health.
28. McElroy KG, Iobst SE, DeVance-Wilson C, Ludeman E, Barr E. Systematic Review and Meta-Analysis of the Effect of Nutrients on Blood Lead Levels in Pregnancy. J Obstet Gynecol Neonatal Nurs. 2020;49(3):243-253. doi: 10.1016/j.jogn.2020.02.004.
29. Centers for Disease Control and Prevention. Childhood Blood Lead Surveillance: National Data. Atlanta: CDC; 2025. [cited 2026 Jul 26]. Available from: https://www.cdc.gov/lead-prevention/php/data/national-surveillance-data.html.
30. LeBrón AMW, Torres IR, Valencia E, Dominguez ML, Garcia-Sanchez DG, Logue MD, et al. The State of Public Health Lead Policies: Implications for Urban Health Inequities and Recommendations for Health Equity. Int J Environ Res Public Health. 2019;16(6):1064. doi: 10.3390/ijerph16061064.
31. Environmental Protection Agency. Lead-Based Paint Laws & Regulations [internet]. Washington (DC): EPA; 2026. [cited 2026 Jul 26]. Available from: https://www.epa.gov/lead/lead-based-paint-laws-regulations.
32. World Health Organization. Guideline for clinical management of exposure to lead [Internet]. Geneva: WHO; 2021. [cited 2026 Jul 26]. Available from: https://www.who.int/publications/i/item/9789240037045.
33. Ohlander J, Kromhout H, van Tongeren M. Interventions to Reduce Exposures in the Workplace: A Systematic Review of Intervention Studies Over Six Decades, 1960–2019. Front Public Health. 2020;8:67. doi: 10.3389/fpubh.2020.00067.
34. Miller AL, Varisco R, Charles S, Haan P, Stein SF, Hernandez J, et al. Parenting and Lead Mitigation at Home: A Multifaceted Community Partnership Model Promoting Parent Engagement in Lead Exposure Prevention. Health Promot Pract. 2023;24(5):911-920. doi: 10.1177/15248399221092998.

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