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Lead poisoning, pica, and their relationship with cognitive development in children: A comprehensive review

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Abstract
Lead poisoning remains one of the most significant environmental health concerns affecting children worldwide, particularly in low- and middle-income countries. Young children are especially vulnerable because of their developing nervous systems and increased gastrointestinal absorption of lead. One of the important behavioral conditions associated with childhood lead exposure is pica, characterized by the persistent ingestion of non-food substances such as soil, clay, paint chips, chalk, and dust. Pica not only increases the risk of lead exposure but is also associated with nutritional deficiencies, particularly iron deficiency, which further enhances lead absorption. Both lead toxicity and pica have profound consequences on cognitive development, including reduced intelligence quotient (IQ), impaired attention, poor academic performance, executive dysfunction, behavioral disorders, and delayed neurodevelopment. Emerging evidence indicates a bidirectional relationship in which nutritional deficiencies predispose children to pica, pica increases lead ingestion, and lead toxicity further impairs cognitive function and behavioral regulation. This review summarizes the epidemiology, sources, mechanisms, clinical manifestations, diagnosis, prevention, and management of lead poisoning and pica while emphasizing their combined impact on cognition. The review also highlights current research findings, public health interventions, and future directions aimed at reducing childhood lead exposure and improving neurodevelopmental outcomes.

Keywords: Lead poisoning, Pica, cognition, neurodevelopment, Children, Iron deficiency, environmental health, IQ

Introduction

Childhood is a critical period for brain growth and neurological development. Environmental exposures during this stage can produce lifelong consequences on cognitive performance, behavior, and overall health. Among environmental toxins, lead continues to be one of the most extensively studied because of its irreversible effects on the developing nervous system. Despite global efforts to eliminate lead from gasoline, paints, and industrial products, millions of children remain exposed through contaminated soil, water, household dust, traditional medicines, toys, and occupational exposure brought home by family members.

Children younger than six years of age are particularly susceptible because they absorb approximately 40–50% of ingested lead compared with only 10–15% in adults. Their frequent hand-to-mouth behavior, crawling, and exploratory activities further increase exposure.

Pica, defined as the persistent consumption of non-nutritive substances for at least one month beyond the age when such behavior is developmentally appropriate, represents another important pediatric concern. Common substances consumed include soil (geophagia), clay, chalk, paper, starch, paint chips, ash, hair, and ice (pagophagia). Although pica has multiple etiologies, iron deficiency remains one of its strongest associations.

The coexistence of pica and lead poisoning represents a major public health issue because ingestion of contaminated soil or paint chips substantially increases lead exposure. Additionally, iron deficiency associated with pica enhances intestinal lead absorption through shared transport mechanisms. Together, these factors create a vicious cycle leading to impaired neurodevelopment and poor cognitive outcomes.

This review examines current evidence regarding lead poisoning, pica, and their interaction in influencing cognitive development in children.

Epidemiology of Childhood Lead Poisoning

Childhood lead poisoning remains a major global public health challenge despite substantial reductions in environmental lead exposure in many high-income countries. The World Health Organization (WHO) identifies lead as one of the ten chemicals of major public health concern and emphasizes that there is no safe level of lead exposure, particularly for children, whose developing nervous systems are highly susceptible to its neurotoxic effects. Globally, an estimated one in three children (approximately 800 million) have blood lead levels (BLLs) at or above 5 µg/dL, with nearly 90–95% of affected children living in low- and middle-income countries (LMICs) where environmental regulations, waste management, and occupational safety measures are often inadequate (WHO, 2024; Larsen *et al*., 2023) [10]. According to WHO estimates, lead exposure contributed to more than 1.5 million deaths and over 33 million disability-adjusted life years (DALYs) worldwide in 2021, reflecting its substantial health burden beyond childhood neurotoxicity. The burden is particularly severe in LMICs due to informal recycling of used lead-acid batteries, mining, smelting industries, lead-contaminated spices, cosmetics, traditional medicines, paints, and electronic waste recycling. India represents one of the countries with the highest burden of childhood lead exposure, with reports estimating that over 275 million children have elevated blood lead levels, largely attributable to rapid industrialization, urban pollution, informal battery recycling, contaminated household dust,

and inadequate environmental monitoring. Studies have shown that children residing in urban industrial areas generally exhibit higher BLLs than those in rural regions because of greater exposure to vehicular emissions (historically), industrial emissions, construction activities, and contaminated housing; however, rural populations are increasingly affected through contaminated groundwater, agricultural soils, traditional remedies, and cottage industries. High-risk populations include children younger than six years, those with pica, iron deficiency, malnutrition, developmental disabilities, and children living near battery recycling units, mining sites, smelters, or hazardous waste disposal areas. Socioeconomic determinants such as poverty, overcrowded housing, poor sanitation, low parental education, inadequate nutrition, and limited access to healthcare substantially increase the risk of lead exposure and its adverse health consequences. Age is another important determinant, with children aged 1–5 years exhibiting the highest prevalence because of frequent hand-to-mouth behavior, increased gastrointestinal absorption of lead, and rapid brain development. Although gender differences are generally modest during early childhood, several epidemiological studies have reported slightly higher BLLs among boys, likely reflecting greater outdoor activity, exploratory behavior, and environmental exposure compared with girls. Collectively, these epidemiological findings underscore the urgent need for targeted surveillance, environmental remediation, nutritional interventions, and public health policies to reduce childhood lead exposure, particularly among socioeconomically disadvantaged populations.

Sources of Lead Exposure in Children

Include detailed discussion on:

Sources of Lead Exposure in Children

1. Environmental Sources of Lead Exposure

Environmental contamination remains the primary source of lead exposure among children worldwide. Contaminated soil serves as a major reservoir of lead due to historical emissions from leaded gasoline, industrial activities, mining operations, and deteriorating lead-based paints. Young children frequently ingest contaminated soil through hand-to-mouth behavior and pica, significantly increasing their risk of lead poisoning (Lanphear *et al*., 2005) [9]. Lead-based paint, particularly in homes built before regulations restricting lead-containing paints, continues to be a significant source of exposure. As painted surfaces deteriorate, peeling paint chips and contaminated dust become readily accessible to children, who may ingest them intentionally or inadvertently (Bellinger, 2016) [1]. Household dust is another important exposure pathway because lead particles originating from paint, contaminated soil, or occupational activities accumulate indoors and are easily inhaled or ingested by crawling infants and toddlers. Drinking water may become contaminated when water passes through lead service lines, plumbing fixtures, brass faucets, or solder containing lead, particularly in areas with corrosive water supplies. Episodes such as the Flint water crisis have highlighted the serious health consequences of lead-contaminated drinking water (Hanna-Attisha *et al*., 2016) [7]. Air pollution also contributes to environmental lead exposure through emissions from mining, smelting, battery manufacturing, waste incineration, electronic waste recycling, and industrial combustion. Although the global elimination of leaded gasoline has substantially reduced airborne lead concentrations, industrial emissions continue to expose nearby communities, particularly in low- and middle-income countries (WHO, 2024).

2. Industrial Sources of Lead Exposure

Industrial activities remain among the most significant contributors to environmental lead contamination. Informal battery recycling, particularly the recycling of used lead-acid batteries, is recognized as one of the largest global sources of lead exposure. Primitive recycling techniques often release lead dust and fumes into surrounding communities, exposing both workers and nearby children (UNICEF & Pure Earth, 2020) [14]. Mining operations release lead-containing ores into surrounding soil, air, and water, increasing exposure among nearby populations. Similarly, lead smelting emits substantial quantities of airborne lead particles that contaminate residential areas and agricultural land. Workers employed in mining and smelting industries may also transport lead dust home on their clothing, shoes, and equipment, creating secondary household exposure. Ceramic manufacturing, particularly where lead-containing glazes are used, can contaminate pottery and release lead into food during cooking or storage. Additionally, the rapidly expanding electronics industry, especially informal electronic waste (e-waste) recycling, has emerged as an important source of lead exposure. The dismantling and burning of electronic components release lead into the environment, posing significant health risks to children living near recycling sites (Grant *et al*., 2013) [6].

3. Household Sources of Lead Exposure

Numerous household products continue to contribute to childhood lead exposure. Certain toys, particularly inexpensive imported toys, painted wooden toys, jewelry, crayons, and plastic products, have occasionally been found to contain lead-containing pigments or plastics. Cosmetics, including traditional eye cosmetics such as kohl (surma), sindoor, and certain lipsticks, may contain elevated lead concentrations, especially when manufactured without adequate quality control. Traditional medicines, herbal remedies, Ayurvedic preparations, and folk medicines have also been identified as unexpected sources of lead because some intentionally incorporate heavy metals or become contaminated during production. Furthermore, imported products, including spices, candies, ceramics, cookware, and decorative items, have occasionally been reported to contain unsafe levels of lead, emphasizing the importance of regulatory surveillance and consumer awareness (CDC, 2024; WHO, 2024).

4. Dietary Sources of Lead Exposure

Diet represents another important route of childhood lead exposure. Contaminated food may accumulate lead from polluted agricultural soils, irrigation water, atmospheric deposition, food processing equipment, or packaging materials. Cereals, vegetables, leafy greens, spices, seafood, and root crops grown in contaminated environments may contain elevated lead concentrations. Lead-glazed pottery remains a significant concern because acidic foods facilitate the leaching of lead from ceramic glazes into food and beverages during cooking or storage. Similarly, food storage containers, including lead-soldered cans, improperly glazed

ceramic vessels, brass utensils, and certain plastic containers containing lead stabilizers, may contribute to chronic dietary lead exposure. Although regulatory agencies have significantly reduced food-related lead contamination in many countries, dietary exposure continues to be a concern in regions with inadequate food safety monitoring (EFSA, 2010 [4]; WHO, 2024).

Lead Toxicokinetics

Lead toxicokinetics describes the processes by which lead is absorbed, distributed, stored, and eliminated within the human body. Children absorb substantially more lead than adults because of physiological differences in gastrointestinal function, rapid skeletal growth, and immature detoxification systems. Once absorbed, lead circulates primarily in the blood, distributes to soft tissues, and ultimately accumulates in bones, where it may remain for decades. Blood lead concentration serves as the most commonly used biomarker for recent exposure; however, bone lead represents the largest long-term body burden. Lead readily crosses the placenta during pregnancy, exposing the developing fetus and increasing the risk of neurodevelopmental impairment even before birth. Although the kidneys excrete a portion of absorbed lead, elimination is generally slow, resulting in prolonged biological persistence. Multiple nutritional and physiological factors—including iron, calcium, and zinc deficiencies, fasting, age, pregnancy, and pica—can significantly enhance lead absorption and toxicity (Needleman, 2004 [11]; WHO, 2024).

1. Absorption

Lead enters the human body primarily through ingestion and inhalation, with ingestion representing the predominant route among children. Children absorb approximately 40–50% of ingested lead, whereas adults typically absorb only 10–15%, making children substantially more susceptible to lead toxicity. Gastrointestinal absorption is further enhanced by nutritional deficiencies, particularly deficiencies of iron, calcium, and zinc, because lead competes with these essential minerals for intestinal transport proteins such as divalent metal transporter-1 (DMT1). Pica, characterized by the ingestion of non-food substances such as soil or paint chips, further increases lead intake among young children (Wright *et al*., 2003) [16].

2. Distribution

Following absorption, approximately 95–99% of circulating lead binds to erythrocytes, while only a small fraction remains in plasma. The plasma fraction is biologically active and readily enters soft tissues, including the brain, liver, kidneys, spleen, and bone marrow. Lead readily crosses the blood-brain barrier in young children because the barrier is not fully developed, allowing greater accumulation within the central nervous system and increasing susceptibility to neurological damage (Needleman, 2004) [11].

3. Storage in Bone

Approximately 90–95% of the total body lead burden in adults and about 70–75% in children is eventually deposited in bones and teeth, where lead substitutes for calcium within hydroxyapatite crystals. Bone serves as a long-term reservoir of lead, with a biological half-life ranging from decades. During periods of increased bone turnover, such as pregnancy, lactation, osteoporosis, fractures, or aging, stored lead may be released back into the bloodstream, prolonging exposure even after external sources have been removed (Hu *et al*., 2007) [8].

4. Blood Lead Concentration

Blood lead level (BLL) is the standard biomarker used to assess recent lead exposure. The Centers for Disease Control and Prevention (CDC) currently uses a blood lead reference value of 3.5 µg/dL to identify children with higher-than-expected lead exposure. Numerous epidemiological studies have demonstrated that even BLLs below 5 µg/dL are associated with measurable reductions in intelligence quotient (IQ), impaired executive function, attention deficits, and behavioral disorders, indicating that no safe blood lead threshold has been identified (CDC, 2024; Lanphear *et al*., 2005) [9].

5. Placental Transfer

Lead readily crosses the placenta through passive diffusion, exposing the developing fetus throughout pregnancy. Maternal bone stores accumulated over previous years may be mobilized during pregnancy due to increased calcium demand, resulting in elevated maternal blood lead concentrations and fetal exposure. Prenatal lead exposure has been associated with reduced birth weight, impaired fetal growth, premature delivery, and long-term deficits in cognitive development, language acquisition, and behavioral regulation (Bellinger, 2016) [1].

6. Excretion

Lead is eliminated primarily through the kidneys into urine, with smaller amounts excreted in bile, sweat, hair, nails, and breast milk. However, because lead stored in bone continuously re-enters circulation, complete elimination may require several decades. The biological half-life of lead is approximately 30 days in blood, several months in soft tissues, and 20–30 years in bone, explaining the persistence of toxicity following chronic exposure (Needleman, 2004) [11].

7. Factors Affecting Lead Absorption

Several biological, nutritional, and environmental factors influence the extent of lead absorption. Young age is associated with increased gastrointestinal absorption and greater neurological vulnerability. Iron deficiency markedly enhances lead uptake through increased expression of DMT1 transporters, while deficiencies of calcium, zinc, and vitamin D similarly increase absorption by altering mineral metabolism. Fasting enhances lead absorption compared with food consumption, whereas diets rich in calcium, iron, and vitamin C reduce gastrointestinal uptake. Children with pica, malnutrition, developmental disorders, or those residing in deteriorating housing or near industrial sites experience substantially greater lead exposure. Genetic polymorphisms affecting metal transport and detoxification pathways may also contribute to individual susceptibility to lead toxicity (Wright *et al*., 2003 [16]; WHO, 2024).

Mechanisms of Lead Neurotoxicity

Lead exerts its neurotoxic effects through multiple interconnected molecular and cellular mechanisms that disrupt normal brain development, particularly during early childhood when the central nervous system is highly

vulnerable. One of the primary mechanisms is oxidative stress, in which lead exposure promotes excessive generation of reactive oxygen species (ROS) while simultaneously inhibiting endogenous antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase. The resulting oxidative damage affects neuronal lipids, proteins, and DNA, ultimately impairing neuronal survival and cognitive function (Flora *et al*., 2012) [5]. Lead also acts as a calcium mimic, competing with calcium ions for binding sites on voltage-gated calcium channels, calmodulin, and protein kinase C, thereby disrupting calcium-dependent signaling pathways essential for neurotransmitter release, neuronal migration, synaptic plasticity, and long-term potentiation. This calcium substitution interferes with hippocampal development and significantly impairs learning and memory processes (Sanders *et al*., 2009) [13]. Furthermore, lead induces synaptic dysfunction by reducing dendritic spine density, inhibiting synapse formation, and impairing N-methyl-D-aspartate (NMDA) receptor activity, resulting in defective neuronal communication and reduced cognitive performance. Alterations in neurotransmitter systems are also well documented, with lead disrupting the synthesis, release, and receptor function of dopamine, glutamate, γ-aminobutyric acid (GABA), acetylcholine, and serotonin, thereby contributing to attention deficits, hyperactivity, behavioral disturbances, and learning disabilities. At the cellular level, lead causes mitochondrial dysfunction by impairing oxidative phosphorylation, decreasing ATP production, disrupting mitochondrial membrane potential, and enhancing ROS production, ultimately leading to neuronal energy failure. Persistent oxidative damage further activates intrinsic apoptotic pathways through mitochondrial cytochrome c release, caspase activation, and DNA fragmentation, promoting neuronal apoptosis and reducing neuronal populations in critical brain regions such as the hippocampus and cerebral cortex. Emerging evidence also indicates that lead induces long-lasting epigenetic modifications, including altered DNA methylation, histone modifications, and microRNA expression, which dysregulate genes involved in neurodevelopment, synaptic plasticity, and cognitive function. These epigenetic alterations may persist throughout life and even contribute to transgenerational effects. Additionally, lead stimulates neuroinflammation by activating microglia and astrocytes, resulting in increased production of pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α), interleukin (IL)-1β, and IL-6. Chronic neuroinflammation further exacerbates oxidative stress, disrupts the blood-brain barrier, impairs neuronal repair mechanisms, and accelerates neurodegeneration. Collectively, these overlapping mechanisms explain why even relatively low levels of lead exposure during childhood can produce irreversible deficits in intelligence quotient (IQ), executive function, attention, memory, language development, and behavior, emphasizing the absence of a safe threshold for pediatric lead exposure (Lanphear *et al*., 2005; Bellinger, 2016 [1, 9]; World Health Organization [WHO], 2024) [15].

Relationship Between Pica and Lead Poisoning

Pica and lead poisoning share a complex, bidirectional relationship that creates a vicious cycle, significantly increasing the risk of neurodevelopmental impairment in children. Among the various forms of pica, geophagia (the intentional ingestion of soil or clay) is the most common behavior associated with elevated blood lead levels because contaminated soil and household dust often contain substantial concentrations of lead derived from deteriorating lead-based paint, industrial emissions, mining activities, battery recycling, and historical deposition from leaded gasoline. Young children with pica frequently ingest these contaminated materials through repetitive hand-to-mouth behavior, resulting in increased gastrointestinal absorption of lead and elevated blood lead concentrations (BLLs) (Lanphear *et al*., 2005) [9]. The relationship is further intensified by iron deficiency, one of the most common underlying causes of pica. Iron deficiency stimulates the expression of divalent metal transporter-1 (DMT1) in the intestinal mucosa to enhance iron uptake; however, because lead shares similar physicochemical properties with iron, DMT1 also transports lead more efficiently, resulting in significantly greater lead absorption from the gastrointestinal tract (Wright *et al*., 2003) [16]. Consequently, children with iron deficiency not only exhibit a stronger craving for non-food substances but also absorb larger amounts of lead from contaminated soil, dust, or paint chips. Once absorbed, lead crosses the blood-brain barrier and accumulates in the developing brain, where it induces oxidative stress, disrupts calcium-dependent neuronal signaling, impairs synaptic plasticity, alters neurotransmitter function, and promotes neuroinflammation and neuronal apoptosis. These neurotoxic effects contribute to reduced intelligence quotient (IQ), impaired attention, executive dysfunction, learning disabilities, hyperactivity, impulsivity, and behavioral abnormalities, including poor judgment and repetitive behaviors (Bellinger, 2016; Sanders *et al*., 2009) [1, 13]. Emerging evidence suggests that these behavioral and cognitive disturbances may further reinforce pica behavior by impairing self-regulation and increasing compulsive ingestion of non-food substances, thereby perpetuating additional lead exposure. Moreover, chronic lead toxicity frequently causes microcytic hypochromic anemia by interfering with heme synthesis, which can worsen pre-existing iron deficiency and further upregulate intestinal DMT1 expression, creating a self-perpetuating cycle of pica → ingestion of lead-contaminated materials → elevated blood lead levels → enhanced lead absorption due to iron deficiency → neurotoxicity → behavioral abnormalities → persistent pica. This vicious cycle highlights the importance of early identification of pica, assessment of iron status and blood lead levels, nutritional rehabilitation, environmental lead remediation, and behavioral interventions to prevent irreversible cognitive impairment and improve long-term neurodevelopmental outcomes in children (WHO, 2024; UNICEF & Pure Earth, 2020) [14].

Effects of Lead on Cognitive Development

Lead exposure during early childhood has profound and often irreversible effects on cognitive development because the immature brain is highly susceptible to toxic insults during periods of rapid neuronal growth, synaptogenesis, and myelination. Numerous epidemiological and experimental studies have demonstrated that there is no safe blood lead concentration, with measurable neurodevelopmental deficits occurring even at blood lead levels below 5 μg/dL and, in some cases, below 3.5 μg/dL (Lanphear *et al*., 2005 [9]; WHO, 2024). One of the most consistently reported outcomes is a reduction in intelligence

quotient (IQ), with pooled analyses indicating that each incremental increase in blood lead concentration is associated with a significant decline in cognitive performance, particularly verbal IQ, performance IQ, and overall intellectual functioning (Canfield *et al*., 2003) [2]. Lead-induced damage to the prefrontal cortex and hippocampus also impairs attention by disrupting dopaminergic neurotransmission and executive control networks, resulting in poor concentration, reduced sustained attention, distractibility, and an increased risk of attention-deficit/hyperactivity disorder (ADHD). Executive functions—including planning, decision-making, cognitive flexibility, inhibitory control, and problem-solving—are similarly affected, limiting children's ability to organize tasks and regulate behavior (Bellinger, 2016) [1]. Lead exposure additionally compromises several domains of memory, including working memory, visual memory, and long-term memory, through impairment of hippocampal synaptic plasticity and long-term potentiation, leading to difficulties in information retention, recall, and learning. Language development is also adversely affected, with exposed children frequently demonstrating speech delays, limited vocabulary acquisition, impaired verbal comprehension, and poor communication skills that hinder both academic achievement and social interaction. These cognitive deficits contribute directly to poorer academic performance, including difficulties in reading comprehension, writing ability, mathematical reasoning, classroom participation, standardized test performance, and increased rates of grade repetition and school dropout. Beyond cognitive impairment, lead toxicity significantly influences behavioral outcomes, increasing the prevalence of aggression, irritability, impulsivity, anxiety, depression, antisocial behavior, and conduct disorders through disruption of neurotransmitter systems and prefrontal cortical function. These behavioral abnormalities not only affect interpersonal relationships but also interfere with educational attainment and psychosocial development, often persisting into adolescence and adulthood. Longitudinal studies have further linked childhood lead exposure to reduced educational achievement, lower occupational success, increased delinquency, and diminished socioeconomic productivity later in life, underscoring the lifelong consequences of early-life lead exposure. Collectively, these findings emphasize that even low-level lead exposure can adversely affect multiple cognitive domains, reinforcing the need for primary prevention, routine screening in high-risk populations, nutritional interventions, and early developmental support to minimize irreversible neurological damage (Canfield *et al*., 2003; Lanphear *et al*., 2005; Sanders *et al*., 2009 [2, 9, 13]; WHO, 2024).

Biological Mechanisms Linking Iron Deficiency, Pica, Lead Poisoning, and Cognitive Impairment

The relationship between iron deficiency, pica, lead poisoning, and cognitive impairment represents a complex biological cascade that contributes significantly to adverse neurodevelopmental outcomes in children. Iron deficiency is often the initiating event, as inadequate iron stores impair dopamine metabolism, myelination, neurotransmitter synthesis, and brain energy metabolism while simultaneously increasing the expression of the intestinal divalent metal transporter-1 (DMT1), a protein responsible for absorbing iron from the gastrointestinal tract. Because lead shares similar physicochemical properties with iron, increased DMT1 expression inadvertently enhances intestinal lead absorption. Iron deficiency also predisposes children to pica, a behavioral disorder characterized by the persistent ingestion of non-food substances such as soil, clay, chalk, or paint chips, many of which may be contaminated with lead. Repeated lead ingestion through these materials elevates blood lead concentrations, allowing lead to accumulate in the developing brain where it induces oxidative stress by generating reactive oxygen species (ROS) and suppressing antioxidant defense systems. Excessive oxidative stress damages neuronal membranes, proteins, mitochondria, and DNA, resulting in widespread neuronal dysfunction. Lead further disrupts hippocampal calcium signaling and N-methyl-D-aspartate (NMDA) receptor activity, leading to reduced hippocampal plasticity, which compromises long-term potentiation (LTP), the cellular mechanism underlying learning and memory. Simultaneously, lead inhibits dendritic growth, decreases dendritic spine density, and suppresses synaptic protein synthesis, resulting in reduced synaptogenesis and impaired neuronal connectivity throughout the developing brain. These structural and functional abnormalities ultimately manifest as poor learning, characterized by deficits in attention, memory, language acquisition, problem-solving ability, and academic performance. Over time, persistent neurotoxicity culminates in cognitive impairment, including reduced intelligence quotient (IQ), executive dysfunction, behavioral abnormalities, learning disabilities, and diminished educational achievement. Thus, iron deficiency initiates a vicious cycle in which pica increases environmental lead exposure, enhanced lead absorption intensifies oxidative neuronal injury, and progressive impairment of hippocampal function and synaptic development results in long-lasting cognitive deficits, emphasizing the importance of early nutritional intervention, environmental lead control, and developmental monitoring in at-risk children (Canfield *et al*., 2003; Lanphear *et al*., 2005; Wright *et al*., 2003 [2, 9, 16]; World Health Organization [WHO], 2024) [15].

Conclusion

Lead poisoning and pica represent interrelated pediatric health problems with significant implications for cognitive development. Pica, particularly geophagia, increases the ingestion of lead-contaminated materials, while iron deficiency—a common underlying factor in pica—enhances gastrointestinal lead absorption, creating a cycle that exacerbates neurotoxicity. Even low levels of lead exposure have been associated with measurable reductions in intelligence quotient, impaired executive function, attention deficits, learning disabilities, and behavioral disorders. Because the neurological effects of lead are largely irreversible, early prevention, timely diagnosis, and comprehensive management are essential. Public health measures, including elimination of environmental lead sources, nutritional interventions to correct iron and micronutrient deficiencies, routine blood lead screening in high-risk populations, and behavioral management of pica, are critical for protecting children's cognitive health. Future multidisciplinary research integrating environmental science, nutrition, neuroscience, and pediatric medicine will be essential for developing more effective prevention strategies and improving long-term neurodevelopmental outcomes.

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