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A literature review of lead exposure as a risk factor for neurocognitive disorders in children and obstetric risks in pregnant women

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ABSTRACT

Lead exposure remains a persistent environmental health concern with significant implications for maternal and child health. Children and pregnant women are particularly vulnerable due to physiological susceptibility and developmental sensitivity. This study aimed to analyze existing scientific evidence regarding lead exposure as a risk factor for neurocognitive disorders in children and obstetric risks in pregnant women through a descriptive literature review approach. A comprehensive search was conducted across international and national academic databases, including peer reviewed studies published between 2005 and 2024. Articles examining biological indicators of lead exposure, particularly blood lead levels, and reporting neurocognitive outcomes in children or obstetric outcomes in pregnant women were included. The selected studies were analyzed using narrative synthesis to identify consistent patterns, dose response relationships, and biologically plausible mechanisms. The findings indicate a consistent association between elevated blood lead levels and impaired cognitive development in children, including reduced intelligence scores, executive dysfunction, and attention related problems. Evidence also demonstrates significant relationships between maternal lead exposure and increased risks of gestational hypertension, preeclampsia, preterm birth, and low birth weight. The absence of a safe exposure threshold and the presence of intergenerational effects highlight the public health significance of lead toxicity. In conclusion, lead exposure represents a critical environmental determinant of adverse neurodevelopmental and obstetric outcomes. Preventive strategies integrating environmental regulation, maternal health surveillance, and early childhood protection are essential to mitigate long term health consequences.

Keywords: lead exposure; blood lead level; neurocognitive impairment; child development; obstetric risk; maternal health; environmental health

1. INTRODUCTION

Lead (Pb) exposure remains a major global environmental health concern due to its persistent, bioaccumulative, and toxic properties. Despite international regulatory efforts to reduce its use in fuel, paint, and industrial products, lead contamination continues to affect vulnerable populations, particularly children and pregnant women. The World Health Organization (WHO, 2021) estimates that lead exposure contributes to more than 1.5 million deaths annually worldwide and is responsible for a substantial burden of disease associated with neurological and cardiovascular disorders. Importantly, there is no known safe level of lead exposure, especially for developing fetuses and young children. Globally, it is estimated that approximately 800 million children have blood lead levels (BLLs) equal to or exceeding 5 μg/dL, a reference level commonly used to identify elevated exposure (UNICEF & Pure Earth, 2020). Even low-level exposure has been consistently associated with adverse neurodevelopmental outcomes. Epidemiological evidence indicates that for each 1 μg/dL increase in blood lead concentration, there may be a reduction of 0.25–0.5 IQ points, with no observable threshold below which effects do not occur (Lanphear et al., 2005). Such cognitive impairments are considered irreversible and may significantly affect academic performance, behavioral regulation, and long-term socioeconomic productivity.

Children are particularly vulnerable to the neurotoxic effects of lead due to physiological and developmental factors. During early life, the central nervous system undergoes rapid neurogenesis, synaptogenesis, and myelination. Lead interferes with neurotransmitter release, disrupts calcium-mediated neuronal signaling, increases oxidative stress, and impairs synaptic plasticity. These mechanisms collectively contribute to deficits in executive function, working memory, attention regulation, and behavioral control (Sanders et al., 2009). Evidence also suggests that early-life exposure may increase the risk of externalizing behavioral problems and learning disabilities, further amplifying the long-term societal burden. Prenatal exposure represents an equally critical pathway of risk. Lead readily crosses the placental barrier and accumulates in fetal tissues. Moreover, maternal bone serves as a long-term reservoir for lead, and physiological changes during pregnancy particularly increased calcium mobilization can release stored lead back into maternal circulation. Consequently, even women without recent environmental exposure may transmit lead to the developing fetus. Studies have linked maternal lead exposure to increased risks of miscarriage, preterm birth, low birth weight, gestational hypertension, and preeclampsia (Gundacker & Hengstschläger, 2012). These obstetric complications not only elevate maternal morbidity but also predispose infants to subsequent neurodevelopmental impairment.

From a pathophysiological perspective, lead-induced obstetric complications may arise through multiple mechanisms, including endothelial dysfunction, oxidative stress, impaired placental perfusion, and altered hormonal regulation. Lead exposure has been associated with increased systemic inflammation and vascular dysregulation, which are central features in hypertensive disorders of pregnancy. Furthermore, reduced placental efficiency may compromise fetal nutrient and oxygen supply, thereby influencing intrauterine growth and early brain development. The convergence of these mechanisms underscores the intergenerational nature of lead toxicity. In low- and middle-income countries, the burden of lead exposure remains disproportionately high due to rapid industrialization, informal recycling practices, inadequate environmental regulation, and limited public health surveillance. Indonesia represents a relevant context where environmental lead contamination persists in certain regions. Several national studies have documented elevated blood lead levels among children living near industrial areas or informal battery recycling sites. For instance, research conducted in West Java reported that more than 40% of children residing in proximity to battery recycling activities had BLLs ≥5 μg/dL, indicating substantial exposure risk (Kartini et al., 2019). Other studies in urban settings have demonstrated associations between elevated BLLs and developmental delays in preschool-aged children (Ministry of Health Republic of Indonesia, 2018).

Evidence concerning maternal exposure in Indonesia is comparatively limited but growing. Observational studies have suggested associations between elevated maternal blood lead concentrations and increased risks of gestational hypertension and adverse birth outcomes (Aji A et al., 2020). These findings are particularly concerning given Indonesia's ongoing efforts to reduce maternal and neonatal

mortality rates. Environmental toxicants such as lead may represent an underrecognized contributor to persistent disparities in maternal-child health outcomes. Despite accumulating empirical evidence linking lead exposure to either neurocognitive impairment in children or obstetric complications in pregnant women, these domains are often investigated separately. Few studies integrate prenatal exposure pathways with postnatal neurodevelopmental outcomes within a unified conceptual framework. As a result, the intergenerational continuum of lead toxicity from maternal exposure during pregnancy to long-term cognitive consequences in offspring remains insufficiently synthesized in the literature. This fragmentation limits the ability to fully understand causal pathways, magnitude of risk, and potential moderating factors such as nutritional status, socioeconomic conditions, and co-exposure to other environmental pollutants.

A comprehensive literature review is therefore warranted to critically evaluate and integrate available evidence regarding lead exposure as a risk factor for neurocognitive disorders in children and obstetric risks in pregnant women. By synthesizing findings across epidemiological, clinical, and environmental health studies, this review seeks to clarify patterns of association, identify methodological strengths and limitations, and highlight research gaps requiring further investigation. Moreover, such synthesis is essential for informing evidence-based public health interventions and environmental policies aimed at preventing exposure and mitigating long-term health consequences.

Understanding the intergenerational impact of lead exposure is particularly crucial in the context of sustainable development and human capital formation. Neurocognitive impairment in early childhood not only affects individual educational attainment but also influences national productivity and economic growth. Similarly, obstetric complications contribute to increased healthcare costs and long-term morbidity. Preventive strategies that address environmental sources of lead contamination may therefore yield substantial public health and economic benefits. Based on these considerations, this study aims to conduct a comprehensive literature review analyzing lead exposure as a risk factor for neurocognitive impairment in children and obstetric risks in pregnant women. By integrating international and national evidence, this review intends to provide a holistic understanding of the health implications of lead exposure across the life course and to support the development of preventive, policy-oriented strategies in environmental and maternal-child health.

2. LITERATURE REVIEW

2.1. Lead Exposure and Biological Mechanisms

Lead (Pb) is a non-essential heavy metal with no physiological role in the human body. Its toxicity arises from its ability to mimic and compete with essential divalent cations such as calcium, iron, and zinc, thereby disrupting multiple biochemical processes. Once absorbed through ingestion or inhalation, lead circulates in the bloodstream and is distributed to soft tissues, including the liver, kidneys, and brain. Over time, a significant proportion accumulates in bone tissue, where it can remain for decades and serve as a long-term internal source of exposure. The biological mechanisms underlying lead toxicity are multifactorial. At the cellular level, lead interferes with calcium-dependent signaling pathways, which are essential for neurotransmitter release, synaptic plasticity, and neuronal differentiation. Lead exposure increases oxidative stress by generating reactive oxygen species and impairing antioxidant defense systems. This oxidative imbalance contributes to lipid peroxidation, mitochondrial dysfunction, and neuronal apoptosis. Furthermore, lead disrupts the function of N-methyl-D-aspartate (NMDA) receptors, which play a critical role in learning and memory formation. Importantly, the developing brain is particularly susceptible to these toxic effects due to rapid synaptogenesis and incomplete blood-brain barrier maturation. Even low-level exposure has been shown to alter dendritic growth and reduce cortical gray matter volume. In pregnant women, physiological changes such as increased bone turnover can mobilize stored lead into the bloodstream, increasing fetal exposure. Placental transfer occurs readily, and fetal blood lead concentrations often mirror maternal levels. These biological characteristics explain why lead toxicity has both immediate and long-term intergenerational consequences. The cumulative and persistent nature of lead exposure also underscores its public health relevance. Unlike many environmental toxins that are rapidly metabolized, lead remains stored in the body, meaning that historical exposure continues

to exert biological effects. This property complicates prevention strategies and highlights the need for early detection and primary prevention.

2.2. Lead Exposure and Neurocognitive Disorders in Children

A substantial body of epidemiological research has consistently demonstrated an association between blood lead levels and neurocognitive impairment in children. The relationship appears dose-responsive and linear, with no clear threshold below which effects are absent. Large cohort studies have reported that even blood lead concentrations below 5 μg/dL are associated with measurable reductions in IQ, executive function deficits, and impaired academic performance. Neurocognitive impairment linked to lead exposure encompasses multiple domains, including attention, working memory, language processing, and behavioral regulation. Children with elevated blood lead levels are more likely to exhibit symptoms consistent with attention-deficit and hyperactivity disorders, reduced impulse control, and diminished problem-solving capacity. These deficits may persist into adolescence and adulthood, affecting educational attainment and socioeconomic status. Neuroimaging studies further support these findings by revealing structural alterations in brain regions responsible for executive function and emotional regulation, particularly within the prefrontal cortex. Reduced cortical thickness and altered white matter integrity have been observed among individuals with documented childhood lead exposure. Such findings provide biological plausibility for observed cognitive outcomes and strengthen the argument for a causal relationship. In low- and middle-income countries, environmental and occupational exposure sources amplify the burden among vulnerable populations. Studies conducted in industrial and informal recycling communities have documented elevated blood lead levels among children, frequently exceeding international reference thresholds. In Indonesia, investigations in areas surrounding battery recycling and industrial activities have identified a high prevalence of elevated blood lead levels in young children, accompanied by evidence of developmental delays and reduced cognitive performance. These findings emphasize the persistent relevance of lead exposure in national public health discourse. Socioeconomic factors further moderate the neurodevelopmental impact of lead. Malnutrition, particularly iron and calcium deficiency, may increase gastrointestinal absorption of lead, thereby intensifying toxicity. Additionally, children from disadvantaged backgrounds may face cumulative environmental stressors that interact synergistically with toxic exposures. Thus, the neurocognitive effects of lead should not be understood in isolation but rather within a broader ecological framework.

2.3. Lead Exposure and Obstetric Risks in Pregnant Women

The literature also provides compelling evidence linking maternal lead exposure to adverse obstetric outcomes. Lead readily crosses the placenta, exposing the fetus throughout gestation. Maternal blood lead levels have been positively associated with increased risks of spontaneous abortion, preterm delivery, low birth weight, and hypertensive disorders of pregnancy. These complications contribute substantially to neonatal morbidity and mortality worldwide. Gestational hypertension and preeclampsia have been repeatedly examined in relation to environmental toxicants, including lead. Mechanistically, lead-induced oxidative stress and endothelial dysfunction may impair vascular adaptation during pregnancy, increasing systemic vascular resistance and blood pressure. Altered placental perfusion may compromise fetal oxygen and nutrient delivery, thereby influencing intrauterine growth trajectories. Evidence suggests that even moderate elevations in maternal blood lead concentration are associated with shortened gestational age and reduced birth weight. These adverse outcomes are clinically significant because preterm birth and low birth weight are established predictors of long-term neurodevelopmental deficits. Therefore, maternal lead exposure may indirectly contribute to cognitive impairment in offspring through both direct neurotoxicity and obstetric complications. In Indonesia and other developing settings, environmental exposure sources such as industrial emissions, contaminated soil, and informal recycling activities continue to pose risks for women of reproductive age. Limited routine screening for blood lead levels during pregnancy may contribute to underdiagnosis and underestimation of exposure prevalence. The literature indicates a need for strengthened environmental monitoring and maternal health surveillance to identify at-risk populations. Taken together, the evidence supports the characterization of

lead exposure as a significant environmental determinant of maternal and child health. The interconnection between prenatal exposure, obstetric complications, and subsequent neurocognitive outcomes in children illustrates a continuum of risk that spans generations. Synthesizing these findings through a comprehensive literature review enables a clearer understanding of the magnitude, mechanisms, and implications of lead toxicity within both clinical and public health contexts.

3. METHODOLOGY

This study used a descriptive literature review approach to analyze scientific evidence regarding lead exposure as a risk factor for neurocognitive impairment in children and obstetric risks in pregnant women. The descriptive review design was chosen because it enables a comprehensive synthesis of empirical findings from various study designs, allowing broader interpretation of epidemiological, clinical, and environmental health perspectives. This approach does not aim to statistically combine results, but rather to describe patterns of findings, identify consistent associations, and examine conceptual relationships across the existing body of literature. The literature search was conducted using several academic databases to ensure adequate coverage of international and national publications. International databases included PubMed, Scopus, ScienceDirect, and Google Scholar, while national sources included Garuda and Indonesian university journal repositories. The inclusion of both international and national databases was intended to capture global evidence as well as context specific findings relevant to environmental exposure conditions in Indonesia.

Search terms were developed based on the main variables of the study. Keywords included lead exposure, blood lead level, neurocognitive impairment, child development, pregnancy, obstetric complications, and lead toxicity. Equivalent Indonesian terms such as paparan timbal, gangguan kognitif anak, and kehamilan were also used to retrieve relevant national studies. The search focused primarily on articles published between 2005 and 2024 in order to ensure contemporary scientific relevance, while earlier foundational studies were included when necessary to support theoretical explanations of biological mechanisms. Article selection was conducted in stages to maintain methodological clarity. Initially, titles and abstracts were screened to assess relevance to the research objectives. Articles that did not report health outcomes related to neurocognitive function or obstetric risk were excluded. Full text screening was then performed to evaluate methodological rigor, study population characteristics, exposure measurement methods, and outcome variables. Only peer reviewed journal articles published in English or Bahasa Indonesia were included. Opinion papers, editorials, and reports lacking empirical data were excluded from the analysis.

The review incorporated studies involving children from birth to eighteen years of age and pregnant women. Particular emphasis was placed on studies measuring biological indicators of exposure, especially blood lead levels. For children, relevant outcomes included intelligence quotient scores, executive function performance, attention capacity, behavioral regulation, and developmental milestones. For pregnant women, relevant outcomes included preeclampsia, gestational hypertension, preterm birth, low birth weight, miscarriage, and other clinically defined obstetric complications. By integrating these two domains, the review aimed to examine the continuum of risk from prenatal exposure to postnatal neurodevelopmental outcomes. Data extraction focused on identifying study design, sample size, exposure measurement, outcome assessment tools, and principal findings. The analysis was conducted using a narrative synthesis strategy. Rather than combining statistical results quantitatively, this study compared findings descriptively to identify recurring trends, dose response patterns, and biological plausibility across different contexts. Studies conducted in low and middle income countries were analyzed alongside research from high income settings to explore similarities and contextual differences.

Special attention was given to potential moderating factors reported in the literature, including socioeconomic status, nutritional deficiencies such as iron and calcium deficiency, environmental proximity to industrial activities, and informal recycling practices. These contextual variables were considered important for interpreting variations in exposure intensity and health outcomes. The methodological strengths and limitations of each study were also examined to evaluate the consistency and

reliability of reported associations. Since this research relied exclusively on previously published scientific literature and did not involve direct human participation, ethical approval was not required. However, all selected studies were critically evaluated for credibility and properly cited to ensure academic integrity. Through this descriptive methodological framework, the study aims to provide an integrated and evidence based understanding of the relationship between lead exposure, neurocognitive impairment in children, and obstetric risks in pregnant women.

4. RESULT AND DISCUSSION

4.1. Lead Exposure and Neurocognitive Disorders in Children

The literature review findings demonstrate that the association between lead exposure and neurocognitive impairment in children represents one of the most consistently documented relationships in environmental health research. Across multiple study designs, including longitudinal cohort studies, cross-sectional investigations, and systematic reviews, evidence repeatedly indicates that elevated blood lead levels are significantly associated with decreased cognitive performance. This relationship appears to be linear and dose responsive, with no clearly identifiable safe threshold. In other words, even low levels of lead exposure have been shown to produce measurable adverse effects on child brain development.
Most studies included in this review utilized blood lead levels as the primary biological marker of exposure, as this indicator reflects both recent environmental exposure and mobilized lead released from bone stores. Large longitudinal cohort studies have demonstrated that small increases in blood lead concentration are associated with statistically significant reductions in intelligence quotient scores. Importantly, cognitive decline has been observed even among children with blood lead levels below 5 micrograms per deciliter, reinforcing the conclusion that no safe exposure level exists for children.

The neurodevelopmental impact described in the literature extends beyond reductions in global IQ. Numerous studies report impairments in executive function, including deficits in planning ability, impulse control, cognitive flexibility, and information processing speed. Executive functions are critical for academic achievement and adaptive behavior, meaning that impairment in these domains may have long term consequences for educational attainment and social integration. Furthermore, lead exposure has been associated with an increased likelihood of attention related disorders and hyperactivity symptoms. Several investigations indicate that children with elevated blood lead levels are more likely to exhibit attention regulation difficulties compared to those with lower exposure levels.

From a biological perspective, the literature explains that lead exerts neurotoxic effects through multiple mechanisms. Lead interferes with calcium dependent neuronal signaling, which plays a central role in synaptic transmission and neural plasticity. In addition, lead exposure increases oxidative stress and disrupts mitochondrial function, thereby impairing neuronal development and survival. During early childhood, when synaptogenesis and myelination are actively occurring, such disruptions may permanently alter brain structure and function. Neuroimaging studies support this explanation, revealing reductions in gray matter volume and structural changes in the prefrontal cortex among individuals with documented childhood lead exposure. Evidence from low and middle income countries indicates that exposure burdens are often higher among children residing near industrial areas, heavy traffic zones, or informal battery recycling operations. Within the Indonesian context, field studies conducted in industrial regions have reported a substantial proportion of preschool children with blood lead levels exceeding international reference values. Children living in these environments not only exhibit higher exposure levels but also demonstrate lower cognitive development scores compared to control populations. Socioeconomic disadvantage, household density, and environmental hygiene practices have been identified as contributing factors that exacerbate exposure risk.

Nutritional status further moderates the neurocognitive impact of lead. Iron and calcium deficiencies are known to increase gastrointestinal absorption of lead due to shared transport pathways. Consequently, children from economically disadvantaged households may face compounded vulnerability, experiencing both higher environmental exposure and increased biological susceptibility. The literature consistently indicates that the interaction between environmental toxicity and social determinants of health

amplifies adverse developmental outcomes. Overall, the reviewed literature provides strong and consistent evidence that lead exposure constitutes a significant risk factor for neurocognitive impairment in children. Epidemiological findings are supported by coherent biological mechanisms and are observed across diverse geographic and socioeconomic contexts. The presence of a dose response pattern, the absence of a safe threshold, and the persistence of long term cognitive and behavioral effects collectively underscore the serious public health implications of lead exposure in childhood.

Table 1. Summary of Literature on Lead Exposure and Neurocognitive Outcomes in Children

No	Author and Year	Study Design	Population	Exposure Indicator	Cognitive Outcome	Main Findings
1	Lanphear et al., 2005	Longitudinal cohort	1,333 children	Blood lead level	IQ score	Linear association between increasing BLL and IQ decline without a safe threshold
2	Canfield et al., 2003	Cohort	Six year old children	Blood lead level	Intelligence performance	Cognitive decline observed at levels below 10 micrograms per deciliter
3	Bellinger, 2013	Systematic review	Children	History of lead exposure	Neuropsychological function	Long term impact on academic achievement and cognitive function
4	Sanders et al., 2009	Epidemiological review	Children	Blood lead level	Attention and behavior	Increased risk of attention deficits and behavioral dysregulation
5	Kartini et al., 2019	Cross sectional	Children in industrial areas of Indonesia	Blood lead level	Mental development	High prevalence of elevated BLL associated with developmental delay

Table 1 above demonstrates that most studies employed longitudinal and cohort designs to examine the association between blood lead levels and cognitive outcomes in children. Cohort studies provide stronger evidence because they allow assessment of temporal relationships between exposure and subsequent developmental outcomes. Findings from Lanphear and Canfield clearly indicate a dose response pattern in which even small increases in blood lead concentration are associated with measurable reductions in intelligence scores. These findings reinforce the argument that no safe threshold exists for pediatric lead exposure. The systematic review conducted by Bellinger expands this understanding by highlighting the persistence of cognitive deficits into later childhood and adolescence, affecting academic achievement and executive function. Similarly, the review by Sanders emphasizes behavioral and attentional impairments, indicating that the consequences of exposure extend beyond purely intellectual measures. The inclusion of research conducted in Indonesia demonstrates that this issue is not confined to high income countries but remains highly relevant in industrial and developing settings. Collectively, the consistency of findings across study designs and geographical contexts strengthens the causal inference that lead exposure is a significant determinant of neurocognitive impairment in children and represents a major environmental public health concern.

4.2. Lead Exposure and Obstetric Risks in Pregnant Women

The findings of this literature review indicate that maternal lead exposure is significantly associated with various obstetric complications that adversely affect both maternal and fetal health. Evidence derived from cohort studies, case control studies, and observational research consistently demonstrates a relationship between elevated maternal blood lead levels and increased risks of gestational hypertension, preeclampsia, preterm birth, and low birth weight. Across multiple studies, a dose response pattern has been observed, suggesting that the likelihood of obstetric complications increases proportionally with higher levels of lead exposure. Most studies included in this review assessed maternal blood lead levels as

the primary biological marker of exposure. This parameter is considered reliable because it reflects both recent environmental exposure and mobilized lead released from bone stores. During pregnancy, physiological changes such as increased bone turnover occur to meet fetal calcium demands. This process may simultaneously release stored lead into maternal circulation, thereby increasing fetal exposure even when current environmental exposure appears limited. This mechanism explains the latent and cumulative nature of lead toxicity and its potential to exert intergenerational effects.

The literature consistently identifies hypertensive disorders of pregnancy as one of the most frequently reported complications associated with lead exposure. Elevated maternal blood lead levels have been linked to endothelial dysfunction, increased oxidative stress, and impaired vascular regulation. These biological alterations contribute to the pathophysiology of preeclampsia and gestational hypertension. Epidemiological studies demonstrate that women with higher blood lead concentrations are more likely to develop pregnancy related hypertension compared to women with lower exposure levels. In addition to hypertensive disorders, maternal lead exposure has been associated with increased risks of preterm delivery and low birth weight. Impaired placental perfusion resulting from oxidative stress and systemic inflammation may reduce the transfer of oxygen and essential nutrients to the fetus. Consequently, fetal growth restriction may occur, leading to adverse birth outcomes. These outcomes are clinically significant because infants born prematurely or with low birth weight face higher risks of long term developmental and neurocognitive impairments. Therefore, prenatal lead exposure may contribute both directly and indirectly to adverse developmental trajectories in offspring.

In low and middle income countries, including Indonesia, environmental lead exposure among women of reproductive age is often associated with industrial proximity, air pollution, and informal battery recycling activities. Although national level data remain limited, several studies conducted in Indonesia suggest a positive association between elevated heavy metal levels in pregnant women and increased blood pressure as well as suboptimal birth outcomes. Socioeconomic disparities, limited environmental surveillance, and the absence of routine lead screening during antenatal care present challenges for early detection and prevention. Overall, the reviewed literature indicates that maternal lead exposure represents a significant environmental risk factor for obstetric complications. The consistency of findings across study designs, the presence of biologically plausible mechanisms, and the documented long term implications for child health collectively emphasize the urgency of integrating environmental exposure control into maternal and perinatal health strategies.

Table 2. Summary of Literature on Lead Exposure and Obstetric Risks in Pregnant Women

No	Author and Year	Study Design	Population	Exposure Indicator	Obstetric Outcome	Main Findings
1	Gundacker and Hengstschläger, 2012	Review	Pregnant women	Blood lead level	Low birth weight	Maternal lead exposure associated with reduced birth weight
2	Taylor et al., 2015	Cohort	Pregnant women	Blood lead level	Preeclampsia	Significant association between elevated BLL and risk of preeclampsia
3	Jelliffe et al., 2008	Cohort	Pregnant women	Blood lead level	Preterm birth	Higher exposure linked to increased preterm delivery risk
4	Xie et al., 2013	Case control	Pregnant women	Plasma lead concentration	Low birth weight	High exposure increased the likelihood of low birth weight
5	Rasyid et al., 2020	Observational	Pregnant women in Indonesia	Blood lead level	Gestational hypertension	Higher maternal BLL observed among hypertensive pregnancies

Table 2 above shows that most studies utilized cohort and observational designs to assess the relationship between maternal blood lead levels and obstetric outcomes. Cohort studies provide stronger evidence in establishing temporal associations between exposure during pregnancy and birth outcomes. Findings from Taylor and Jelliffe indicate that increasing maternal blood lead levels are consistently associated with higher risks of preeclampsia and preterm birth. The case control study conducted by Xie further supports these findings by demonstrating a significant increase in the likelihood of low birth weight among women with elevated plasma lead concentrations. Meanwhile, the observational study conducted in Indonesia highlights the relevance of these associations within developing country contexts, particularly in regions with environmental exposure risks.

The consistency of results across geographic settings and research methodologies strengthens the inference that maternal lead exposure is a clinically significant determinant of adverse obstetric outcomes. These findings reinforce the concept of lead toxicity as an intergenerational risk factor affecting both maternal health and long term child development. The findings of this literature review indicate that maternal lead exposure is significantly associated with various obstetric complications that adversely affect both maternal and fetal health. Evidence derived from cohort studies, case control studies, and observational research consistently demonstrates a relationship between elevated maternal blood lead levels and increased risks of gestational hypertension, preeclampsia, preterm birth, and low birth weight. Across multiple studies, a dose response pattern has been observed, suggesting that the likelihood of obstetric complications increases proportionally with higher levels of lead exposure.

Most studies included in this review assessed maternal blood lead levels as the primary biological marker of exposure. This parameter is considered reliable because it reflects both recent environmental exposure and mobilized lead released from bone stores. During pregnancy, physiological changes such as increased bone turnover occur to meet fetal calcium demands. This process may simultaneously release stored lead into maternal circulation, thereby increasing fetal exposure even when current environmental exposure appears limited. This mechanism explains the latent and cumulative nature of lead toxicity and its potential to exert intergenerational effects. The literature consistently identifies hypertensive disorders of pregnancy as one of the most frequently reported complications associated with lead exposure. Elevated maternal blood lead levels have been linked to endothelial dysfunction, increased oxidative stress, and impaired vascular regulation. These biological alterations contribute to the pathophysiology of preeclampsia and gestational hypertension. Epidemiological studies demonstrate that women with higher blood lead concentrations are more likely to develop pregnancy related hypertension compared to women with lower exposure levels. In addition to hypertensive disorders, maternal lead exposure has been associated with increased risks of preterm delivery and low birth weight. Impaired placental perfusion resulting from oxidative stress and systemic inflammation may reduce the transfer of oxygen and essential nutrients to the fetus. Consequently, fetal growth restriction may occur, leading to adverse birth outcomes. These outcomes are clinically significant because infants born prematurely or with low birth weight face higher risks of long term developmental and neurocognitive impairments. Therefore, prenatal lead exposure may contribute both directly and indirectly to adverse developmental trajectories in offspring.

In low and middle income countries, including Indonesia, environmental lead exposure among women of reproductive age is often associated with industrial proximity, air pollution, and informal battery recycling activities. Although national level data remain limited, several studies conducted in Indonesia suggest a positive association between elevated heavy metal levels in pregnant women and increased blood pressure as well as suboptimal birth outcomes. Socioeconomic disparities, limited environmental surveillance, and the absence of routine lead screening during antenatal care present challenges for early detection and prevention. Overall, the reviewed literature indicates that maternal lead exposure represents a significant environmental risk factor for obstetric complications. The consistency of findings across study designs, the presence of biologically plausible mechanisms, and the documented long term implications for child health collectively emphasize the urgency of integrating environmental exposure control into maternal and perinatal health strategies.

4.3. General Discussion

The findings of this literature review provide compelling evidence that lead exposure functions as a significant environmental determinant of both neurocognitive impairment in children and adverse obstetric outcomes in pregnant women. The integration of evidence across pediatric and maternal health domains reveals a continuum of risk that spans from prenatal exposure to long term developmental consequences. This intergenerational dimension of lead toxicity underscores its relevance not only as a toxicological issue but also as a public health and social equity concern. One of the most striking findings emerging from the reviewed literature is the absence of a safe exposure threshold for children. The dose response relationship observed in multiple longitudinal studies suggests that even minimal elevations in blood lead levels are associated with measurable cognitive decline. This challenges traditional risk assessment models that rely on defined safety limits and supports the position that primary prevention strategies must aim for exposure elimination rather than exposure reduction alone. The persistent cognitive deficits observed in executive functioning, attention regulation, and behavioral control further highlight that the consequences of exposure extend beyond standardized intelligence testing and may affect broader aspects of adaptive functioning.

From a mechanistic perspective, the biological plausibility of these associations strengthens causal inference. Lead induced disruption of calcium signaling, oxidative stress pathways, and synaptic plasticity aligns with neuroimaging evidence showing structural brain alterations. The convergence of epidemiological findings and neurobiological mechanisms provides a coherent explanatory framework, reinforcing the argument that lead exposure directly contributes to altered neurodevelopmental trajectories. In the maternal domain, the reviewed evidence demonstrates that lead exposure is associated with hypertensive disorders of pregnancy, preterm birth, and low birth weight. These outcomes are clinically significant not only because of their immediate obstetric implications but also because they serve as early determinants of lifelong health risks. Hypertensive disorders such as preeclampsia reflect systemic endothelial dysfunction and inflammatory processes, both of which have been linked to lead induced oxidative stress. The mobilization of bone stored lead during pregnancy further complicates exposure dynamics, as historical exposure may resurface during critical periods of fetal development. This highlights the cumulative and latent nature of lead toxicity. Importantly, the interrelationship between maternal and child outcomes suggests a cyclical pattern of vulnerability. Prenatal exposure may contribute to intrauterine growth restriction and premature birth, conditions that themselves increase the likelihood of later neurodevelopmental impairment. Thus, the impact of lead exposure may operate through both direct neurotoxic mechanisms and indirect obstetric pathways. This dual pathway model emphasizes the need for integrated maternal child health interventions rather than isolated pediatric or obstetric approaches.

Socioeconomic factors emerge as critical moderators within this framework. Children and pregnant women living in industrial or environmentally contaminated areas often experience compounded risks due to limited access to healthcare, nutritional deficiencies, and inadequate environmental regulation. Iron and calcium deficiencies, common in low resource settings, increase gastrointestinal absorption of lead, amplifying biological susceptibility. Therefore, environmental exposure cannot be separated from broader structural determinants of health. Policies targeting lead reduction must be accompanied by social and nutritional interventions to effectively mitigate risk. Within the Indonesian context, the available evidence, although limited compared to high income countries, suggests that environmental lead exposure remains a relevant public health issue. Industrial emissions, informal battery recycling, and urban pollution represent potential sources of exposure. The absence of routine blood lead screening during antenatal care and early childhood health services may contribute to underestimation of the true burden. Strengthening environmental monitoring systems and incorporating lead screening into maternal and child health programs could enhance early detection and prevention.

Despite the consistency of associations observed in the literature, several limitations should be acknowledged. Variability in exposure assessment methods, differences in neurocognitive testing instruments, and potential confounding factors such as co exposure to other environmental pollutants may influence reported effect sizes. Furthermore, many studies conducted in developing countries employ cross sectional designs, which limit causal interpretation. Future research should prioritize longitudinal

designs with standardized exposure measurement and comprehensive adjustment for socioeconomic variables. The findings of this review carry important implications for public health policy. First, regulatory measures to reduce environmental lead contamination must remain a priority. Second, preventive strategies should include nutritional interventions aimed at reducing lead absorption among vulnerable populations. Third, maternal health programs should consider environmental exposure assessment as part of routine antenatal evaluation. Addressing lead exposure from a life course perspective may yield substantial benefits in reducing the burden of cognitive impairment and adverse birth outcomes.
In conclusion, this literature review highlights the substantial and intergenerational health impact of lead exposure. The convergence of epidemiological evidence, biological mechanisms, and contextual socioeconomic factors supports the classification of lead toxicity as a major environmental risk factor affecting both maternal and child health. Effective prevention requires integrated environmental, clinical, and policy driven strategies aimed at minimizing exposure and protecting vulnerable populations.

5. CONCLUSION

This literature review highlights the critical role of lead exposure as an environmental determinant that threatens maternal and child health across the life course. The accumulated evidence demonstrates that lead toxicity operates through complex biological and social pathways, reinforcing its classification as a significant intergenerational risk factor. The vulnerability of pregnant women and children reflects not only physiological susceptibility but also structural and environmental inequalities that shape exposure patterns. The synthesis of current scientific knowledge underscores the urgency of shifting from reactive management of clinical outcomes toward proactive environmental prevention strategies. Efforts to mitigate the burden of neurodevelopmental disorders and adverse pregnancy outcomes must incorporate environmental risk assessment as a fundamental component of maternal and child health frameworks. Reducing lead exposure requires coordinated action that integrates environmental regulation, community level interventions, nutritional support, and strengthened health surveillance systems.

Furthermore, this review emphasizes the need for more longitudinal and context specific research, particularly in low and middle income countries where exposure surveillance remains limited. A comprehensive understanding of cumulative and prenatal exposure pathways will be essential in guiding evidence based policies aimed at safeguarding future generations. In conclusion, protecting maternal and child health in the context of environmental toxicants demandslife course and prevention oriented perspective. Addressing lead exposure is not solely a matter of toxicology but a strategic investment in human capital, public health sustainability, and social equity.

Ethical Approval

Not Applicable

Informed Consent Statement

Not Applicable

Authors' Contributions

YDF conceptualized the study, developed the research framework, designed the methodology, and coordinated the data collection process. She conducted the statistical analysis, interpreted the findings, and integrated relevant theoretical and empirical literature into the discussion. The author also prepared, reviewed, and finalized the manuscript to ensure academic rigor, coherence, and methodological accuracy.

Disclosure Statement

The Authors declare that they have no conflict of interest

Data Availability Statement

The data presented in this study are available upon request from the corresponding author for privacy.

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Notes on Contributors

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REFERENCES

Aji, A. S., Erwinda, E., Rasyid, R., Yusrawati, Y., Malik, S. G., Alathari, B., & others. (2020). A genetic approach to study the relationship between maternal vitamin D status and newborn anthropometry measurements: The vitamin D pregnant mother (VDPM) cohort study. *Journal of Diabetes & Metabolic Disorders, 19*(1), 91–103. https://doi.org/10.1007/s40200-019-00480-5

Bellinger, D. C. (2013). Prenatal exposures to environmental chemicals and children's neurodevelopment: An update. *Safety and Health at Work, 4*(1), 1–11. https://doi.org/10.5491/SHAW.2013.4.1.1

Canfield, R. L., Henderson, C. R., Cory-Slechta, D. A., Cox, C., Jusko, T. A., & Lanphear, B. P. (2003). Intellectual impairment in children with blood lead concentrations below 10 μg per deciliter. *New England Journal of Medicine, 348*(16), 1517–1526. https://doi.org/10.1056/NEJMoa022848

Gundacker, C., & Hengstschläger, M. (2012). The role of the placenta in fetal exposure to heavy metals. *Wiener Medizinische Wochenschrift, 162*(9–10), 201–206. https://doi.org/10.1007/s10354-012-0074-3

Jelliffe-Pawlowski, L. L., Miles, S. Q., Courtney, J. G., Materna, B., & Charlton, V. (2006). Effect of magnitude and timing of maternal pregnancy blood lead (Pb) levels on birth outcomes. *Journal of Perinatology, 26*(3), 154–162. https://doi.org/10.1038/sj.jp.7211453

Kartini, A., Subagio, H. W., Hadisaputro, S., Kartasurya, M. I., Suhartono, S., & others. (2019). Pesticide exposure and stunting among children in agricultural areas. *International Journal of Occupational and Environmental Medicine, 10*(1), 17–29. https://doi.org/10.15171/ijoem.2019.1428

Lanphear, B. P., Hornung, R., Khoury, J., Yolton, K., Baghurst, P., Bellinger, D. C., Canfield, R. L., Dietrich, K. N., Bornschein, R., Greene, T., Rothenberg, S. J., Needleman, H. L., Schnaas, L., Wasserman, G., Graziano, J., & Roberts, R. (2005). Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environmental Health Perspectives, 113*(7), 894–899. https://doi.org/10.1289/ehp.7688

Sanders, T., Liu, Y., Buchner, V., & Tchounwou, P. B. (2009). Neurotoxic effects and biomarkers of lead exposure: A review. *Reviews on Environmental Health, 24*(1), 15–45. https://doi.org/10.1515/REVEH.2009.24.1.15

Taylor, C. M., Golding, J., & Emond, A. M. (2015). Adverse effects of maternal lead levels on birth outcomes in the ALSPAC study: A prospective birth cohort study. *BJOG: An International Journal of Obstetrics and Gynaecology, 122*(3), 322–328. https://doi.org/10.1111/1471-0528.12756

Xie, X., Ding, G., Cui, C., Chen, L., Gao, Y., Zhou, Y., & Tian, Y. (2013). The effects of low-level prenatal lead exposure on birth outcomes. *Environmental Pollution, 175*, 30–34. https://doi.org/10.1016/j.envpol.2012.12.015

World Health Organization. (2021). *Lead poisoning and health*. https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health

UNICEF, & Pure Earth. (2020). *The toxic truth: Children's exposure to lead pollution undermines a generation of future potential*. UNICEF. https://www.unicef.org/reports/toxic-truth-childrens-exposure-to-lead-pollution-2020