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Review

Silent threats of lead-based paints in toys and households to children's health and development

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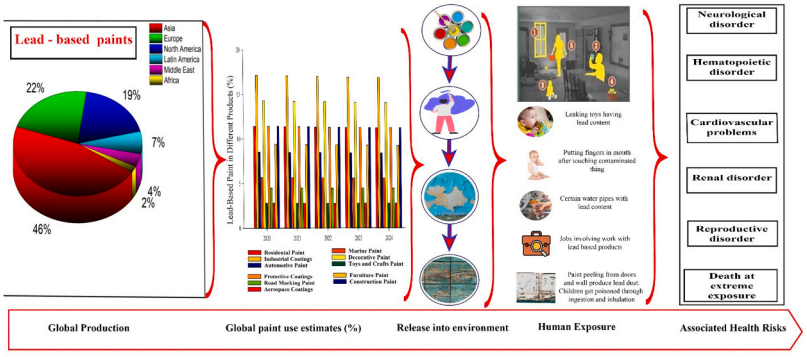
HIGHLIGHTS

- The global scenario of lead-based paint production and use was reviewed.
- Environmental release of lead, exposure pathways and safety standards were assessed.
- Insight into children's exposure, health impacts along the mechanism of toxicity.
- Mental retardation among children has been found at concentrations < 0.01 mg/dL.

GRAPHICAL ABSTRACT



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ABSTRACT

Lead (Pb), a highly toxic heavy metal, poses a significant global health risk, particularly to children. Widely used in paint manufacturing for its remarkable corrosion-resistance properties Pb exposure has been linked to severe health issues, including reduced neurotransmitter levels, organ damage, potentially leading to death in extreme cases. Children Are particularly vulnerable, with Pb toxicity primarily affecting the brain, reproductive, kidneys, and cardiovascular systems. Approximately 0.6 million children worldwide suffer from cognitive impairments caused by Pb exposure. Despite varying Pb content regulations across countries, research has found that Pb concentration in paints often exceed permissible levels. A 0.01 mg/dL blood Pb level (BLL) is considered the

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threshold level as per the World Health Organization. However, recent studies reveal that significant health effects, including cognitive impairments in children, occur even at BLLs < 0.01 mg/dL. This review provides critical insights into the global production and use of Pb-based paints, release mechanisms of Pb, exposure pathways, and safety standards. It also highlights the harmful effects of Pb on human health, particularly in children, and its detailed toxicity mechanisms. Finally, this review identifies critical knowledge gaps and offers perspectives for future research.

1. Introduction

Lead (Pb) exposure in humans is a notable global health concern because of its considerable environmental pollution [1]. The World Health Organization (WHO) estimated that in 2016, exposure to Pb resulted in more than half a million deaths [2], of which 82 % occurred in low- and middle-income developing nations [3].

Although all individuals are at health risk due to Pb exposure, children are particularly susceptible because of their higher Pb absorption rate compared with adults [4]. In children, organs of the neurological, reproductive, renal, cardiovascular, and nervous systems are the main ones affected by Pb poisoning. The brain is particularly susceptible to Pb exposure [5]. Exposure to Pb substantially affects synapse development in the cerebral cortex of a developing child's brain. This disrupts the ion channels and the synthesis of neurochemicals, such as neurotransmitters [6]. Approximately 0.6 million children worldwide suffer from Pb exposure-induced moderate mental impairment throughout their childhood [7].

Research has shown that Pb exposure detrimentally affects the enduring intelligence quotient (IQ). In particular, IQ scores at the age of five years and more decreased by two to six points with each increase of 0.01 mg/dL in blood Pb concentration [8,9]. Women with elevated Pb exposure suffered several adverse pregnancy concerns, including early membrane rupture, low birth weights, premature delivery, spontaneous abortion, and hypertension during pregnancy period [10]. Exposures (<0.01 mg/dL) induced adverse physiological effects even at deficient levels [11]. WHO and the US Centers for Disease Control and Prevention (CDC) predicted that blood Pb levels of 0.01 mg/dL or more should be seriously considered. However, there exists no specific minimum limit at which Pb exposure can be deemed safe, as the damaging effects and impaired development have been observed at lower doses as well [12, 13].

With an abundance of ~0.002 % in the earth's crust, Pb is a bluish-gray metallic element with an atomic number of 82 and a relative atomic mass of 207.2. The element has a density of 11.34 g/cm^3 and a melting point of 327.5 °C [14]. There exist many different Pb sources, which may be natural or anthropogenic. Pb naturally occurs in the soil. It can periodically originate because of weathering of Pb-containing rocks. Furthermore, industrial operations such as mining and smelting that discharge Pb fragments into the water and the atmosphere are examples of human activities [15,16]. Aging drinking water infrastructure is another important factor contributing to Pb contamination, mainly using Pb fittings, pipes, and solders in plumbing installations. Because of corrosion, these supplies can gradually release Pb particles into the drinking water supply [15].

Moreover, Pb exposure can result from incorrect handling and disposal of Pb acid batteries used in automobiles and other machinery [17]. With all these sources, old buildings with Pb paint are the primary sources of Pb contamination. Pb has been a component of mass-market consumer items, such as paint, for almost a hundred years. Historically, manufacturers have added Pb into paints because of its protecting qualities, which can increase their durability and adhesion to surfaces and enhance the intensity of color [18]. Lead carbonate ($PbCO_3$) is used as a white pigment, while lead chromate ($PbCrO_4$) is used as a yellow pigment. As per Crow (2007), paint coatings can maintain their toughness, flexibility, and resistance to cracking for an extended period by using $PbCO_3$ to neutralize the acidic breakdown products from certain paint oils [19]. Environmental laws related to Pb-based paints are often more strictly enforced in developed countries than in developing ones. The United States enacted several laws regulating Pb-based paints to safeguard residences and establishments occupied by children [20].

The threshold level of Pb was reduced to 600 mg/kg in 1977, which was further dropped to 90 mg/kg in 200In 9. Even after this regulation, old houses continued to cause notable Pb exposure for children living there [21]. While Pb paint application has ceased, this in no way indicates that contamination has stopped; because of its durability, Pb paint lasts for decades or even centuries on buildings. Pb paint continues to chip and peel from these houses because of weather, contaminating the surrounding soil and household dust. Hence, Pb-based paint continues to be a severe problem throughout most of the United States, Asian, and African countries, particularly for children who are more likely to play in these types of environments and are biologically more susceptible [22].

In light of potential hazards to human health and surroundings associated with continuous consumption of Pb-based paints, a thorough review of academic research on this subject is critical to accurately understanding the problem. Consequently, this literature review presents notable perspectives on the global scenario of Pb-based paint production with its use, Pb release into the environment, exposure pathways, safety standards of Pb exposure, and associated health impacts with its toxicity mechanisms. Finally, this study provides perspectives for future research as well.

2. Global scenario of lead-based paints: production and use

Global demand for paint and coatings was estimated to rise by 3.7 % per year, with 54.7 million metric tons in 2020 valued at US$193 billion [23]. As per the annual report of the Polymers Paint and Color Journal (2023), the worldwide paint and coatings business was valued at US $195 billion in 2023. Details of global Pb-based paint production are shown in Fig. 1. Asia is the largest geographical region worldwide, accounting for 46 % of the total paint production standing at 32.4 million tons. Europe and North America follow accounting for 22 % and 19 % of

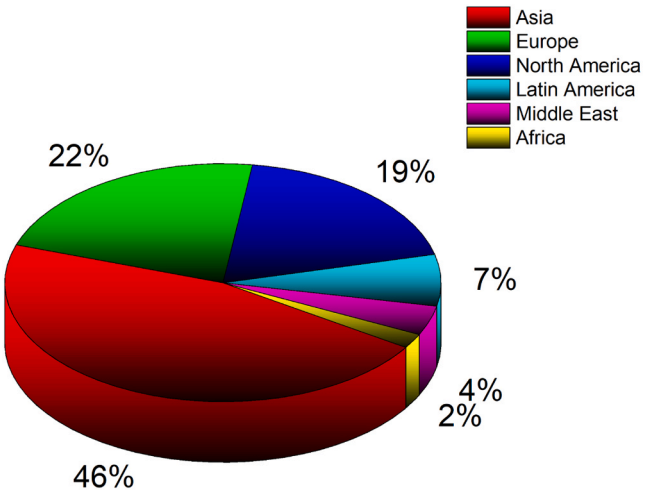

Fig. 1. Global lead-based paint production (%).
Data Source: Annual Review: PPCJs, 2023 Global Coatings Market Update.

the worldwide market, respectively, while Latin America and the Middle East account for 7 % and 4 %, respectively. Africa produces the least amount of paint (2 %), i.e., 1 million tons. Decorative paints represent the world's largest paint category [24]. All Asian nations combining manufacture 50 %–55 % of the world's paints, with China being the most notable and fastest-expanding paint producer and user [25]. China produced 19.0 million tons of paints in 2016. It has enforced a restriction on use of Pb in paints [26]. In 2001, the limit for soluble Pb was tightened from the erstwhile 250 mg/kg to 90 mg/kg [27]. However, despite these laws, Pb-based paints with Pb concentrations exceeding the acceptable range are frequently observed on the market. [28]. Lin et al. (2009) observed that of 58 fresh paint samples, Pb levels in 29 (50 %) were 600 mg/kg or higher and those in 14(24 %) were 5000 mg/kg or higher. Among the paints currently used, Pb concentrations were equal to or exceeded 600 mg/kg in 16 of 28 samples (57 %) acquired from 24 kindergartens and primary schools, including six samples (21 %) with Pb values of 5000 mg/kg or higher [27]. Details regarding paints with Pb levels exceeding 90 mg/kg are illustrated in Fig. 2.

Furthermore, the Indian paint manufacturing industry is divided into two sectors: (a) the organized sector, comprising 10–12 notable firms and holding 57 % of the total market share and (b) the unorganized sector, consisting of ~2000 small firms and accounting for 43 % of the total market share [29]. In 2011, the permissible Pb level in Indian paints was reduced to 300 mg/kg [30] from the previous content of 1000 mg/kg [31]. A study evaluated 148 samples from different organized (such as Asian Paints, ICI, and Kansai Nerolac Paints) and unorganized (such as Kingcoat, Glaxci, Globe, and Ujjala) paint production companies in India [30]. They observed that of the 91 paint samples from the organized sector, only 5 % contained Pb content exceeding 300 mg/kg. However, of the 57 paint samples from the unorganized sector, 93 % had $>$ 300 mg/kg Pb content. Pb concentrations in paints produced by four multinational Indian firms varied from 15 to 231 mg/kg. In contrast, in paints from small-sized enterprises, concentrations ranged from 4213 to 18,981 mg/kg. Unfortunately, Pb-based paints are still commonly accessible in the paint industry. Evaluation of 371 paint samples from seven Asian, three African, and two South American countries revealed that average Pb concentration ranged from 9688 (Singapore) to 31,960 mg/kg (Ecuador) [28]. The lowest number of samples (43.8 % and 43.9 %) containing Pb concentrations $\geq$ 90 mg/kg were from China and Singapore, while 32.8 % and 36.6 % of the samples contained Pb concentrations of $\geq$ 600 mg/kg. To evaluate the presence and concentration of Pb across 11 European nations, 236 road paint samples of various colors were analyzed. Energy-dispersive X-ray fluorescence spectrometry results revealed Pb concentrations exceeding 10 mg/kg in 148 samples, with levels as high as 17.2 % with white and yellow paints exhibiting the highest Pb concentrations exceeding 1000 mg/kg [32]. Developed countries generally enforce stricter environmental regulations for Pb-based paints than developing nations. The United States is regarded as a leader in implementing best practices to regulate Pb-based paints, with various regulations aimed at safeguarding homes and child-occupied facilities [3]. Historically, white house paints in the United States contained up to 50 % Pb, but this practice changed in 1971, when the federal government banned paints containing $>$ 1 % Pb. Permissible Pb concentration was further reduced to 600 mg/kg (0.06 %) in 1977, which was once again lowered to 90 mg/kg (0.009 %) in 2009. However, this regulation does not apply to paints used for bridges and in marine environments [33].

Africa merely accounted for 1 % of the paint trade in 2005 [34]. Pb-based paint manufactured in Africa is still a notable source of Pb exposure, even if its proportion vis-à-vis the global paint industry is relatively small. Approximately 96 % of home paints in Nigeria contain Pb levels exceeding 600 mg/kg [35]. In Portugal and other Eastern European countries, Pb concentration in newly applied paints has been a subject of recent investigations. Clark et al. estimated that the maximum Pb content was 53,000 mg/kg in many paint products in Russia [36]. Global paint use estimates in percentage are shown in Fig. 3.

3. Pb release into the environment

Pb-based paints have been a substantial source of environmental Pb contamination in urban areas and older structures. Weathering, abrasion, renovation activities, industrial processes (mining activities and Pb-based paint production), and improper disposal of paint debris contribute to release of Pb into the environment [35,36]. These processes pose environmental and public health challenges by contributing to dispersion of Pb particles into soils, sediments, and water systems [24, 37]. As for older buildings, Pb-based paints remain a notable contributor to environmental contamination. Historically, Pb-based paints and gasoline were considerable sources of environmental Pb, although their use has either been restricted or banned in the recent decades [38,37, 39]. Although already banned in 1978, Pb-based paints continue to be present in many houses in low-income areas, exposing children to Pb

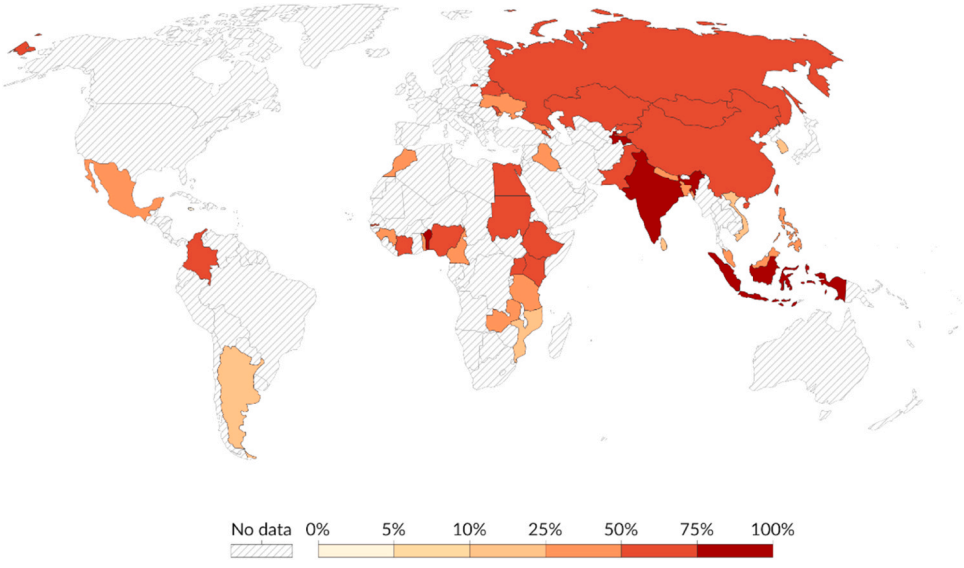

Fig. 2. Share of paints with lead levels greater than 90 mg/kg, 2021.
Data Source: International Pollutants Elimination Network (IPEN, 2021).

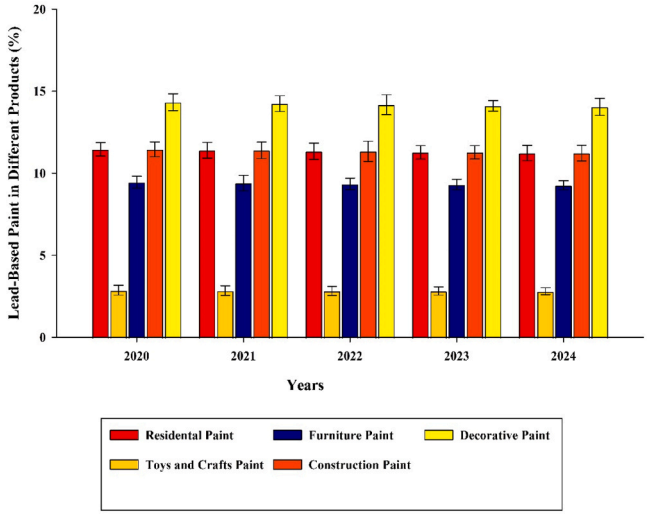

Fig. 3. Percentage of global paint use estimates.
Data Source: Annual Review: PPCJs, 2023 Global Coatings Market Update.

through dust production. As the paint cracks and peels with time, Pb dust is released into the air and soil; this Pb dust may be inhaled by children, resulting in severe health risks. Renovation and demolition of older buildings with Pb-based paint also results in release of Pb particles [40,41].

Weathering of exterior Pb-based paints upon exposure to rain, wind, and ultraviolet radiation leaches Pb into adjacent soil and water systems. Research indicates that Pb concentrations in soils adjacent to painted surfaces, including those in residences and bridges, are markedly higher versus background values, with Pb remaining in the environment for decades [42]. A previous study emphasized that decomposition of external Pb-based paints in residences constructed before the 1970s substantially increased soil Pb concentrations in adjacent regions. Rainwater runoff was recognized as a notable conduit to disseminating Pb particles into adjacent soils and aquatic environments, particularly in metropolitan areas [43]. Pb runoff from painted structures in an urban environment was assessed. High Pb levels were frequently observed, and age and condition of paint notably influenced Pb concentration. Notably, Pb concentrations from washes of older paints were notably greater than those from washes of newly painted surfaces. Surface washes contained Pb levels of 70 % or greater in particulate form, indicating release of Pb pigments from deteriorated paints [44].

Renovation activities, including sanding or scraping of painted surfaces, can aerosolize Pb particles, markedly elevating Pb levels indoors and outdoors [45]. A study measured levels of aerosolized Pb dust during renovation activities in a house built prior to the Pb paint ban. The result demonstrated markedly higher levels of airborne Pb, frequently surpassing the limits of residential and occupational exposure [46]. A study discovered that Pb dust from degrading exterior paints contributed to atmospheric resuspension during dry conditions, particularly in arid and semi-arid regions. Subsequently, the airborne Pb settles onto soil and interior surfaces [47]. Urban soil contamination due to deterioration of Pb-based paint was also investigated. A correlation was observed between elevated soil Pb levels and urban areas' older, painted housing stock. Emphasis was laid on Pb's persistence in urban soils and the role of exposure to environmental Pb in pediatric Pb poisoning [48].

Mining activities also substantially contribute to environmental Pb levels, often via generation of heavy metal-laden waste [39], which is frequently released without regulation [49], hence contaminating soil and water [50]. Pb acid batteries are also a primary source of Pb contamination, releasing notable amounts of Pb into the environment, particularly during raw material extraction and battery disposal [51]. Lin et al. (2009) identified that construction materials, soil, dust, and proximity to heavy traffic routes could introduce additional Pb from motor vehicle emissions into buildings, as observed in affected areas [52]. Pb is commonly released into the environment through corrosion of Pb-containing components in water distribution systems, such as service pipes, plumbing fixtures, and soldiers. This release leads to Pb contamination of drinking water, posing a notable exposure risk to households through consumption of Pb-contaminated water [53]. Pb contamination has been associated with low IQ, reduced mental health, and impaired cognitive abilities, even at lower blood Pb levels (BLLs) [54]. Therefore, various studies have highlighted the need for effective assessment and intervention [55]. Pb isotopes hold value in identifying the origins of Pb contamination in water, thereby providing helpful information for targeted cleanup operations [56]. Research in Georgia demonstrated that Pb isotopic analysis of environmental samples can be performed to trace Pb sources, including spices and dust, as notable contributors to elevated BLLs in children [57]. Moreover, long-term deposition of airborne pollutants responsible for influencing contemporary Pb pollution conditions in the soil, with estimates suggesting that approximately 4000 years of atmospheric pollution are still apparent in northern European soils [58].

These sources have contributed to global mobilization of Pb in the environment, raising concerns regarding environmental impact and implications for human health, particularly in children [59]. Pb release from Pb-based paints continues to be a major issue, particularly in areas with aging infrastructure. Regulatory measures have diminished new applications; however, the existing legacy burden highlights the necessity for continuous monitoring, remediation, and public health interventions [59].

4. Safety standards for Pb

Primary safety standards of Pb exposure should be assigned high priority so as to reduce the possibility for children to have BLLs > 0.05 mg/dL [60]. The Environmental Protection Agency set a standard of 0.15 $\mu g/m^3$ Pb level for ambient air quality. In contrast, the Occupational Safety and Health Administration emphasized occupational BLLs less than 0.04 mg/dL, with mandatory removal at 0.05 mg/dL [61,62]. In 2021, CDC reduced the levels to 0.0035 mg/dL for children because of their high susceptibility [63]. The European Union (EU) implemented extensive measures following the Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) regulation and occupational health directives. Accordingly, airborne Pb concentrations should be maintained to be < 0.5 $\mu g/m^3$ annually, and workers' blood Pb levels must not exceed 0.07 mg/dL. Guidelines for drinking water adhere to WHO standards, capping Pb levels at 0.01 mg/dL, demonstrating uniformity in health protection strategies [64]. The National Health and Medical Research Council, Australia advises interventions upon BLLs exceeding 0.005 mg/dL, consistent with the current guidelines. Guidelines for drinking water establish a Pb concentration limit of 0.01 mg/L, consistent with WHO recommendations [65]. China and India, two highly populated countries with developing economies, have air quality criteria of 1.0 and 0.5 $\mu g/m^3$, respectively. Both nations' drinking water standards align with the WHO's 0.01 mg/dL guideline. However, as per earlier guidelines, China's BLL action levels for children are still higher at 0.01 mg/dL [66,67].

Japan has established stringent regulations regarding Pb exposure, particularly those concerning air quality. The permissible Pb concentration in ambient air is set to be 0.1 $\mu g/m^3$, representing one of the most rigorous standards worldwide [68]. Regulations for Pb levels in drinking water are consistent with WHO guidelines [69]. Blood Pb levels for workers must be maintained to be < 0.04 mg/dL, with regular monitoring required. Stringent environmental and occupational Pb exposure regulations are enforced in the UK as well. The Control of Lead at Work Regulations (Health and Safety Executive) stipulates that blood

Pb levels in employees cannot exceed 0.06 mg/dL for men and 0.03 mg/dL for women of reproductive age [70]. In addition, ambient air Pb has been limited to 0.5 μg/m^3 per annum [71]. Furthermore, in Russia, the permissible ambient air Pb concentrations are set to be 0.3 μg/m^3 , which is more rigorous than the standards in many developed countries [72]. Safety standards set by different regulatory authorities are presented in Table 1.

In the late 20th century, various countries implemented numerous legislations to regulate or eradicate use of Pb in paints. In the 1970s, the United States established 600 mg/kg as an acceptable Pb level in residential paints, which was then lowered to 90 mg/kg by the Consumer Product Safety Improvement Act in 2008 [73]. The REACH law established within the EU comparable limitations consistent with US regulations at 90 mg/kg. Countries such as Canada, New Zealand, Australia, and South Korea have accepted the 90 mg/kg limit, although Japan and Brazil continue to maintain increased standards of 600 mg/kg [74,75]. Unfortunately, emerging economies and low-income regions frequently struggle to enforce stringent regulations because of constrained resources and inadequate regulatory frameworks.

Nevertheless, recent initiatives, including the Global Alliance to Eliminate Lead Paint by WHO and the United Nations Environment Program (UNEP), have expedited efforts in nations with historically elevated Pb thresholds, promoting more rigorous national and regional standards [76]. Permissible Pb limits are elevated in certain regions, particularly in parts of Asia and Latin America. For example, Japan enforces a Pb paint limit of 600 mg/kg under its Industrial Safety and Health Law, while Mexico and Brazil similarly permit up to 600 mg/kg in paints [77,78]. These elevated limits indicate difficulties in regulatory adaptation, with compromised enforcement capabilities and economic considerations possibly hindering the implementation of more rigorous standards. Table 2 presents laws and regulations for Pb in paints in different countries.

Regional organizations are critical to harmonizing Pb limits among their member nations. The East African Community, including Kenya, Tanzania, and Uganda, instituted a 90 mg/kg guideline, representing a substantial advancement for regions where Pb regulation had been

Table 1
Safety standards by different agencies.

Organization	Media	Unit	Lead levels	Year	Reference
World Health Organization (WHO)	Drinking Water	mg/L or μg/L	0.01 or 10	2017	[1]
Food Agriculture Organization (FAO)	Drinking Water	mg/L	0.01	2021	[2]
Food and Drug Administration (FDA)	Bottled water	μg/L	5	2012	[3]
United States Environmental Protection Agency (USEPA)	Drinking Water	μg/L	15	2015	[4]
Environmental Protection Agency (USEPA)	Soil (residential)	mg/kg	400	2001	[5]
Health Canada	Drinking Water	mg/L or (μg/L)	0.005 Or 5	2017	[6]
Agency for Toxic Substances and Disease Registry (ASTDR)	Soil	mg/kg	< 50	2023	[7]
National Health Commission of the People's Republic of China (NHC)	Drinking water	μg/L	10	2006	[8]
Ministry of Ecology and Environment (MEE)	Agricultural soil	mg/kg	80	2018	[9]

Table 2
Laws and regulations set by different countries for lead in paints.

Countries	Lead regulatory limits	Countries	Lead regulatory limits
Algeria	5000 mg/kg for the sale, production, and import of paint	Australia	1000 mg/kg for the production, sale, import, and export of paint
Cameron	90 mg/kg for paint additives	Bangladesh	90 mg/kg for decorative paints
Ethiopia	90 mg/kg for the sale, import, and export of paint	China	90 mg/kg for construction paints and 1000 mg/kg for industrial paints and cars
Kenya	90 mg/kg for the production, sale, import, and export of paint	India	90 mg/kg for decorative and household paints
United Republic of Tanzania	90 mg/kg or 100 mg/kg for lead in paints based on types	Nepal	90 mg/kg for the production and sale of paints
South Africa	600 mg/kg for the production, sale, import, and export of paint	New Zealand	1000 mg/kg for the production, sale, import, and export of paint
Iraq	90 mg/kg for all paints	Philippines	90 mg/kg for construction, decorative, household, and industrial paints
Jordan	90 mg/kg for the import and sale of decorative paints	Pakistan	100 mg/kg for interior and exterior enamel paints
Lebanon	Limited the use of lead in interior and exterior paints	Sri Lanka	90 mg/kg for internal and external emulsion paint and 600 mg/kg for floor and enamel paint
Oman	600 mg/kg for transport, use, import, and production	Thailand	100 mg/kg for all types of paints
Qatar	600 mg/kg for imported paints	Viet Nam	600 mg/kg for all paints and expected to be reduced to 90 mg/kg in 2025
Armenia	5000 mg/kg for household paints	31 countries in Europe Region	Restricted use
Argentina	600 mg/kg for use, import, and production with prohibited use of lead carbonate and lead sulfate	Israel	90 mg/kg for all paints
Brazil	600 mg/kg for transport, use, import and production	Kazakhstan	Limited use in interior building paints
Chile	600 mg/kg for the production, sale, import, and export of paint	Kyrgyzstan	Limited use in paints
Colombia	90 mg/kg for all paints	Monaco	Limited use in paints
Costa Rica	600 mg/kg for the production, sale, import, and export of paint	Montenegro	Limited use in paints
Cuba	20,000 mg/kg for all paints	North Macedonia	Limited use in paints
Dominica	600 mg/kg for all paints	Russian Federation	Restricted the use
Ecuador	100 mg/kg for common-use paints and 600 mg/kg for others	Serbia	Limited use in paints
Mexico	600 mg/kg for all paints	Switzerland	100 mg/kg in all paints

(*continued on next page*)

Table 2 (*continued*)

Countries	Lead regulatory limits	Countries	Lead regulatory limits
United States	90 mg/kg for production, sale, import, household and decorative paints	Panama	600 mg/kg for import and sale
Canada	90 mg/kg for production, sale, and import	Uruguay	600 mg/kg for production, sale, and import

Source: [10,11]

minimal [79]. Saudi Arabia, the United Arab of Emirates (UAE), and Qatar are part of the Gulf Cooperation Council, which exercises a 90 mg/kg norm through the Gulf Standardization Organization. The region benefits from international best practices and reduced cross-border commerce of Pb-contaminated paint products [80]. South Africa's National Regulator for Compulsory Specifications set a 90 mg/kg standard for Pb in paint, following WHO and UNEP's Global Alliance to Eliminate Lead Paint [81]. However, despite these advances, budget restrictions impede Pb standard enforcement in low- and middle-income nations. As more nations embrace the 90 mg/kg limit suggested by UNEP and WHO, the trend toward standardizing Pb rules is becoming increasingly apparent. The Global Alliance to Eliminate Lead Paint was created to assist countries in enacting policies and standards aimed at eradication of Pb-based paints, particularly in areas with weaker regulations or limited enforcement capabilities [76,82].

Despite advancements, particularly in high-income nations and specific regional collectives, substantial variability in Pb limits continues to persist worldwide. The challenges encompass the necessity for technical assistance, regulatory enforcement, and compliance-monitoring resources. As an illustration of rising commitment to public health despite economic constraints, nations with traditionally high Pb levels, such as China and India, have recently begun to enforce stricter Pb standards [27,28]. Pb is recognized for its effect on several biological systems, including the brain and kidneys, with prolonged exposure to it associated with behavioral issues, cognitive deficits, and decreased IQ in children. In light of these findings, global public health agencies promote reduction in and eventually eradication of Pb in consumer products, primarily paints [83].

5. Children's exposure to Pb

Children are particularly vulnerable to nonoccupational Pb exposure, which frequently occurs from playing with toys with Pb-based paints. Numerous toys contain Pb because of Pb-based paints or contamination from dust and soil. By ingesting, sucking, or licking such toys, children are at risk of Pb consumption [84]. Peng et al. (2019) observed considerable Pb levels in the soil and dust from playground equipment, endangering health of children [85]. Painted toys, particularly those composed of plastics, are crucial in Pb poisoning in children. Pb and cadmium, frequently found in plastic toys, are released when the material breaks down. Pb is released when autodigestion weakens the plastic structure [86]. Moreover, women of childbearing age may accumulate Pb in their bones, which may then leak into their bloodstream during pregnancy and affect the developing fetus [87].

According to Njati et al., between 2004 and 2010, >145 children under 18 years of age in the US tested positive for Pb exposure, with pica habits being a significant risk indicator for 36 % of the children tested [88]. In the UK, a 34-mon-old boy was recommended to a pediatric clinic by his family doctor because of symptoms of pallor and a pica habit. The boy used to ingest nonfood items such as wood, plaster, and paint. Based on his blood test reports, the BLL concentration was exceptionally high at 0.0346 mg/dL. He was living with his parents in a house built in the 1970s, where serious proof of the pica was present: the bathroom doorframe and portions of the walls were observed to be severely damaged. The boy was believed to have been exposed to Pb because he had bitten through more recent coats of paint into the older Pb-containing paint [89]. A multigoal study was conducted in Portugal; one of the goals was to assess childhood Pb exposure in children aged from 1 to 5 years who lived in the Oporto Historical Center. The results indicated that the average value of blood Pb concentration was 0.0139 mg/dL, with 85.8 % of the 240 children in the experiment having BLLs >0.01 mg/dL, showing a high prevalence of Pb exposure. Significant causes of Pb exposure in these children included father's line of work, mother's smoking habits, inadequate personal cleanliness, and pica practices linked to Pb paint and polluted soils [90]. History of pica habit was also determined as a critical factor of Pb exposure for children in South Africa, Malaysia, Pakistan, Morocco, and India [91–93].

6. Health impacts of Pb on children

Pb has no known biological function in the body of children. It is a robust and long-lasting neurotoxin mainly absorbed via ingestion and inhalation. It attaches to red blood cells after being absorbed into the body and circulates through the blood to adipose tissues such as those in the spleen, brain, lungs, heart, and muscles [94]. The remaining ingested Pb bioaccumulates in insoluble form in the hair, teeth, and bones, where it can persist for decades. Approximately 80 %–90 % of the Pb is eliminated [95]. Based on research, severe Pb poisoning can occur when chronic acidosis or disease results in Pb contained in bones to be released into the bloodstream [94]. Industrialized nations have gradually stopped using Pb-based paints in homes, due to its associated health risks. According to scientific discoveries, pregnant women and preschool-aged children are the groups most susceptible to the impacts of Pb and deficient BLLs [96].

The higher risk in the case of children is attributed to several factors. A study reported that exposure levels of Pb were low-moderate in infants (<1 year), moderate-high in toddlers (1–3 years), moderate in preschool (3–5 years) children, lower in school age (6–12 years) children, and lowest in adolescents (13–18 years). The study showed that the exposure levels significantly decreased with age [97]. Because of higher gastrointestinal adsorption rates in children, particularly those aged less than 6 years, they can adsorb up to 50 % of Pb ingested, which is higher versus the 10 % adsorption by adults. This increased Pb absorption into their bloodstream affects their developing bodies more significantly than adults. Furthermore, the blood–brain barrier (BBB), which protects the brain from toxic substances, is not fully developed in young children. Consequently, higher levels of Pb can pass into the brain, disrupting neuron growth and operations. The case is severe for children aged less than 6 years, wherein high levels can cause life-lasting cognitive deficiencies and behavioral and attention issues. However, the possibility of such health effects in adults is meager because of their BBB being fully developed, even if Pb can pass through BBBwhen exposed to higher levels [98,99].

Long-term storage of Pb in the bones may allow for it to re-enter the bloodstream during fast bone growth, such as that seen during puberty. This mobilization can increase the risk by exposing the body to the harmful consequences of Pb even years after first exposure [97,100]. However, the risk of remobilization is lower in adults because of the slow turnover rate of bones. The adverse outcomes of Pb exposure in adults may be noticed in the kidney and cardiovascular systems compared with the developing brain [101]. Furthermore, children, particularly toddlers, engage in hand-to-mouth behavior. They often place their hands and toys into their mouths and spend more time on floors, where Pb-contaminated dust can settle, resulting in higher Pb ingestion rates. Adults are not engaged in such behaviors and are typically exposed to Pb through occupational or environmental sources rather than direct ingestion [99].

Most of the Zn, Ca, and Fe in body tissues can be drastically replaced by Pb, impairing the essential functions of these ions in the human body [102]. Moreover, the potential of Pb to substitute calcium is believed to be a crucial factor in its capacity to cross the BBB and cause harm. In

contrast, absence of such a barrier is believed to contribute to newborns' vulnerability to Pb poisoning. Exposure to Pb during childhood can sometimes result in irreversible brain damage and subsequent intellectual disability because of blocking of glutamate receptors, a neurotransmitter essential for learning [96]. Noteworthily, bones can contribute to 45 %–55 % of BLLs, even in people not exposed to high amounts of Pb. Recent exposures are considered only in blood Pb evaluations. In contrast, mobilization of bone Pb is primarily associated with Pb accumulated upon previous exposures re-entering the blood [103]. During pregnancy, BLLs are mainly influenced by mobilization of Pb from bones. Because of Pb-induced impairment of heme biosynthesis and erythroid component maturation, elevated BLLs eventually cause anemia by reducing red blood cell counts and hemoglobin concentration [103] (Fig. 3).

However, studies have shown an unfavorable link between Pb exposure and intellectual function, even at blood Pb concentrations of < 0.01 mg/dL [104]. Pb poisoning can cause anemia, headaches, sleeplessness, faintness, anger, hallucinations, and kidney injury [105]. Within the US adult population, newly published population-based cohort research investigating the relationship between low-level Pb exposure and mortality observed that 20 % of the participants (14,289) had BLLs of 0.005 mg/dL. During an average monitoring spanning 19.3 years, a total of 4422 persons died, including 988 dying of ischemic heart disease and 1801 of cardiovascular disease. A correlation was observed between all-cause mortality and a rise in blood Pb content from 1.0 to 6.7 mg/dL. The population percentage for all-cause mortality corresponding to the blood Pb levels was 18.0 % or 4,12,000 deaths yearly. Fig. 4 shows the data on age-standardized deaths from all causes corresponding to Pb exposure per 1,00,000 people [106].

6.1. Nervous system

Pb poisoning in children primarily affects their neurological system. It affects both the central and peripheral neurological systems. In children, the peripheral nervous system is most affected, but the central nervous system is most affected [102]. In addition, the main signs of Pb exposure include irritation, dullness, headaches, tremors in muscles, hallucinations, and memory loss. Pb poisoning at extremely high levels can even cause ataxia, convulsions, delirium, paralysis, and coma. Pb poisoning in children is linked to cognitive impairment because it affects development of the nervous system [103]. Several studies reported an inverse correlation between BLL and cognitive disability.

According to a WHO assessment in 2001 on childhood Pb poisoning, early exposure to Pb causes irreversible and incurable brain damage, along with other consequences such as reduced intellect, attention deficit disorder, and behavioral disruption. Also documented was an association between BLL and results of four cognitive functioning tests on 4853 children in the US who were representative of the population: These results exhibited a decline in mean scores with each mg/dL increase in BLL. Another study involving 456 children aged 7 years indicated that BLL was significantly correlated with lower IQ scores, lower achievement, and more behavioral issues, such as attention deficits [107]. The hippocampus, a part of the brain, is crucial for learning and memory functions. Pb mostly interferes with learning because it destroys the cells in the hippocampus, particularly in children. In rats exposed to Pb, researchers observed structural damage, including irregularly shaped nuclei and denaturation of myelin [104].

Pb can also disrupt release of neurotransmitters [108], which are substances that neurons use to communicate with other cells. Communication between cells is disrupted because of this interference. Pb typically tampers with glutamate, a neurotransmitter critical to numerous processes, including learning [109]. It does so by blocking the N-methyl-D-aspartate (NMDA) receptors. Pb poisoning is believed to target the blocking of receptors. A study reported that Pb exposure inhibited the NMDA receptor and reduced the gene's abundance in a specific brain region. In animal research, Pb was also discovered to have a role in death of brain cells [110].

6.2. Hematopoietic system

Pb enters the bloodstream after absorption, with > 95 % binding to erythrocytes. Anemia is one of the clinical signs of Pb poisoning in children, and it is mainly caused by reduction in heme production and increase in rate at which erythrocytes are destroyed [111]. In the heme biosynthesis process, Pb has been linked with suppressing the activity of three essential enzymes: ferrochelatase, delta-aminolevulinic acid dehydratase (ALAD), and coproporphyrin oxidase [16]. The mitochondrial enzyme ferrochelatase converts ferric cation (Fe^{+3}) into ferrous cation (Fe^{+2}) and combines Fe^{+2} with protoporphyrin-IX to create heme. Because Pb poisoning prevents Fe^{3+} from being reduced into Fe^{2+}, less Fe^{+2} is transported into mitochondria, resulting in accumulation of protoporphyrin in erythrocytes and subsequent interaction with zinc to form zinc protoporphyrin (ZPP). Pb toxicity and iron deficiency can be detected by measuring the amount of ZPP in red blood cells [112].

In the heme biosynthesis pathway, ALAD catalyzes condensation of two aminolevulinic acid molecules to create a molecule of porphobilinogen. By interacting with the functional groups of the enzyme, Pb reduces the activities of ALAD, rising blood levels as well as urine levels of aminolevulinic acid. Hematological results of battery-manufacturing employees exposed to Pb at work for roughly 15 years demonstrated drop in erythrocyte counts and hemoglobin concentration, attributed to Pb intoxication-led disruptions in heme biosynthesis [112].

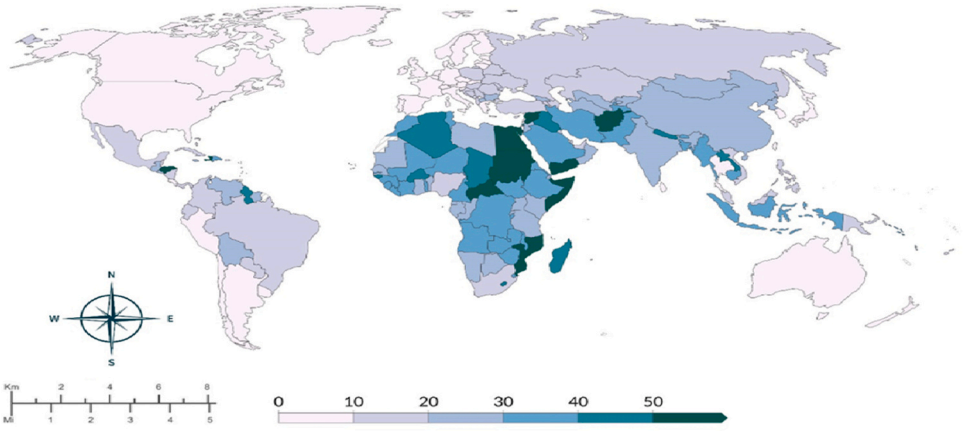

Fig. 4. The estimated number of age-standardized deaths from all causes attributed to lead exposure per 100,000 people.
Data Source: Global Burden of Disease; IMHE, 2024.

Furthermore, ZPP is formed when this enzyme is inhibited, replacing iron in the porphyrin ring. Consequently, ZPP concentration increases, which can also be utilized as a signal to track amount of Pb exposure [113]. The heme synthesis pathway prevents heme synthesis because of the combined inhibition of these three essential enzymes. Unfortunately, uncertainty surrounds the process that shortens the lives of erythrocytes. Basophilic stippling, or presence of thick particles in red blood cells, was one of the first hematological effects of Pb to be recognized. This condition is also a potential biomarker for diagnosis of Pb poisoning. These aggregates result from ribonucleic acid breakdown [114].

6.3. Cardiovascular system

The functional role of the cardiovascular system is determined by a complicated interaction between the vascular endothelium, smooth muscle, immune system, and neurological system. Most cardiovascular disease risk factors, such as chronic Pb exposure, diabetes, obesity, sedentary lifestyle, tobacco use, cholesterol, and hypertension, are preventable [115]. Peripheral vascular disease, cerebrovascular accidents, and ischemic coronary heart disease are severe conditions. Although there has been evidence of a causal link between Pb exposure and hypertension, this link only holds when Pb poisoning has adverse effects on the cardiovascular system [116]. Numerous studies demonstrated a link or association between ambient Pb exposure and occurrence of cardiovascular conditions, including peripheral artery disease [117], heart disease [118], stroke [119], hypertension [120], and cardiac rhythm alterations [115].

6.4. Renal system

Exposure to Pb at levels < 0.01 mg/dL has also been shown to cause harm. However, renal impairment is primarily associated with levels of > 0.06 mg/dL [121]. A correlation between Pb poisoning and kidney disorders in humans has been identified and established. Renal functioning abnormalities have two forms: acute nephropathy and chronic nephropathy. Physical manifestation of acute nephropathy includes degenerative alterations in the tubular epithelium and the occurrence of nuclear inclusion bodies containing Pb–protein complexes [122]. In addition, a defective tubular transport mechanism is a characteristic of acute nephropathy. Fanconi's syndrome, a combination of aberrant excretion of glucose, phosphates, and amino acids, can result from it, but it does not cause protein in urine. Contrarily, chronic nephropathy is considerably severer and can result in permanent morphological and functional abnormalities. Hypertension, hyperuricemia, and renal breakdown are the outcomes of glomerular and tubulointerstitial alterations [50].

6.5. Reproductive system

Both male and female reproductive health is negatively affected by Pb exposure in several ways [123]. Pb is regarded as one of the fetal toxic substances that can pass through the placenta in a pregnant woman and then exert a degenerative effect on the growing fetus. A study indicated that various unfavorable pregnancy outcomes, including early membrane rupture, low birth weight, early delivery, spontaneous abortion, hypertension throughout pregnancy, increase in number of prenatal deaths, and suppression of postnatal growth and development, were linked to Pb exposure [35]. In contrast, Destro et al., 2023 reported that Pb toxicity has men more prone to aberrant spermatogenesis, decreased libido, and poor prostatic function [124].

7. Mechanism of Pb toxicity

Pb toxicity in children primarily results from absorption through the digestive system and oral consumption. Pb absorption via the digestive system is influenced by factors such as Ca and Fe levels, fasting, age, and pregnancy, as well as the physical and chemical characteristics of the material consumed, for example, size of particles, solubility of minerals, and form of Pb [29]. Pb absorbed by the colon is then transported to soft tissues, such as those in the kidneys, liver, and bone tissue, where Pb gradually accumulates. The primary mechanism of transportation of Pb from the colon to various body organs involves red blood cells, wherein Pb binds to hemoglobin. Approximately 1 % of Pb in blood is found in the serum and plasma, whereas ~99 % is found in erythrocytes [125]. The amount of Pb in plasma, relative to that in whole blood significantly impacts the circulation of Pb in various organs such as the brain, renal, lungs, bones, teeth, and spleen [126]. The estimated half-life of Pb in soft tissue is 40 days, while the half-life of Pb in blood is believed to be 35 days. Pb can remain in the bone for up to 30 years, and the level of Pb in teeth and bone grows as people age. Children are believed to have a substantially higher biological half-time of Pb than adults [127]. Pb chemically interacts with protein thiol groups, and Pb poisoning is believed to impede enzymes, disrupting Ca, Mg, and Zn homeostasis [85]. Because Pb poisoning interferes with the pro-oxidant/antioxidant cell defense system, it results in Pb-induced oxidative stress. Antioxidant minerals, including Zn and Se, and vitamins E, C, B_6, and B-carotene, are believed to counteract Pb-induced oxidative stress [110].

Pb absorption is higher in children than in adults [128]. In addition, > 95 % of Pb deposits in skeletal bones are insoluble phosphate, and Pb absorption may increase during pregnancy [129]. As per autopsy reports, 90 %–95 % of the body's Pb accumulation is attributed to the teeth and cortical bone. In adults, the skeleton's overall Pb body accumulation ranges from 80 % to 95 %, while it is 73 % in children [29]. Pb can pass from mothers to the fetus and to babies while breastfeeding [128]. The human central nervous system is the primary target of Pb toxicity, and children who consume high levels of Pb from their surroundings, particularly when they are anemic, are associated with decreased IQ and compromised motor skills [130].

7.1. Oxidative stress

Superoxide Dismutase (SOD) is a Zn/Cu metalloenzyme that scavenges free radicals [105]. Several studies reported that Pb exposure dramatically decreased SOD levels. This may have resulted from a higher-than-usual quantity of Pb in the tissues and a potential interaction with this enzyme, decreasing the removal of superoxide radicals. Although it is a well-known Pb poisoning susceptibility, catalase is an effective H_2O_2 decomposer. Pb can reduce the activity of glutathione peroxidase in the brain. This may be attributed to (a) impaired functional groups such as glutathione and nicotinamide adenine dinucleotide phosphate or (b) selenium-mediated harmful metal detoxification. Meanwhile, glutathione S-transferase, an antioxidant enzyme, is known to offer defense against oxidative stress [107].

The Pb–oxyhemoglobin reaction generates superoxide radicals. When two S-aminolevulinic acid (ALA) molecules condense, 5-ALAd plays a role in the production process of heme precursor (porphobilinogen) [15]. Therefore, inhibition of ALAD results in impaired heme synthesis, which causes buildup of ALA. ALA accumulation causes auto-oxidation to be catalyzed by metal, which converts oxyhemoglobin into methemoglobin. This reaction generates reactive oxide species, such as superoxide and hydroperoxide [39].

7.2. Ionic mechanism of Pb toxicity

Pb's ionic mechanism of action mainly results from its capacity to replace other monovalent cations such as sodium (Na^+) and bivalent cations such as Ca^{2+}, Mg^{2+}, and Fe^{2+}, affecting several essential biological processes in the body [16]. In fact, many crucial physiological functions, including apoptosis, ionic transport, intra- and intercellular signaling, cell adhesion, protein folding and maturation, enzyme control, and release of neurotransmitters, have been discovered to be significantly affected [114]. The ionic pathway is primarily responsible

for neurological impairments, as Pb may substantially enter the BBB after replacing the Ca^{2+} ions. Pb accumulates in astroglial cells after crossing the BBB. The toxic effects are particularly noticeable in the developing nervous system, where young astrocyte cells without Pb-binding proteins are present, as shown in Fig. 5.

Pb readily harms immature astrocyte cells and prevents myelin sheath formation, two processes essential to establishing the BBB. Even picomolar concentrations of Pb can displace Ca [123]. This may affect notable neurotransmitters, including protein kinase C, which regulates long-term neural activation and memory consolidation. In addition, this affects the concentration of Na ions, which are necessary for many essential biological processes such as controlling the uptake and retention of Ca by synaptosomes, generating action potentials in excitatory tissues for cell-to-cell communication, and absorbing neurotransmitters (gamma-aminobutyric acid, dopamine, and choline). This Pb–Na interaction severely hampers normal operation of the aforementioned Na-dependent processes [124].

8. Future challenges

This review summarized different studies from across the world on Pb content in Pb-based paints and children's toys. In addition, it discussed the associated adverse health effects on the public. We suggest thoroughly grasping this review article's topic and stopping the current situation.

1. To prevent the sale of Pb-based paints and toys with high Pb content, suitable national, regional, and international laws and regulations must be passed and implemented, particularly for producing, importing, exporting, using, and selling of paints and toys.
2. To prevent Pb poisoning in future, there is an urgent need to raise public awareness of the dangerous consequences of Pb-containing paints and children's toys. It is devastating when parents are unaware of the risks their children are incurring while playing with such toys.
3. Manufacturers should place Pb warning signs and information on the amount of Pb present in products and tinned paints.
4. Innovations in Pb identification methods, such as employment of portable devices using X-ray fluorescence technology, is expected allow for more precise and extensive examinations of surfaces for presence of Pb.
5. Public health agencies should persist on allocation of resources to awareness initiatives, informing people about the dangers of Pb exposure and strategies for risk reduction.
6. Programs for screening of children, particularly in high-risk areas, should be extended. Early detection of elevated Pb levels in the bloodstream can result in appropriate measures and reduce the likelihood of long-term health consequences.
7. We strongly advise that the global community fully support the Global Alliance to Eliminate Lead Paint, led by UNEP and WHO, which aims to phase-out Pb-based paints worldwide by 2020.

9. Conclusion

There has been global apprehension regarding the ongoing use of Pb-containing paints in homes, decorations, and children's toys. The effect of Pb pollution is notable worldwide with respect to health concerns. The adverse health outcomes resulting from children's exposure to Pb are both profound and intricate, as they affect various functions, particularly that of the nervous system. Pb acts as a neurotoxin damaging brain structure and function via disruption of crucial developmental mechanisms. This can result in difficulties in learning, memory impairment, and issues with focus and attention. Evidence supports a correlation between BLL and decreased IQ levels in children. Moreover, Pb interferes with functioning of the blood system, heart health, kidney function, and reproductive processes, leading to conditions such as reduced red blood cell count, high blood pressure, renal impairment, and adverse pregnancy outcomes.

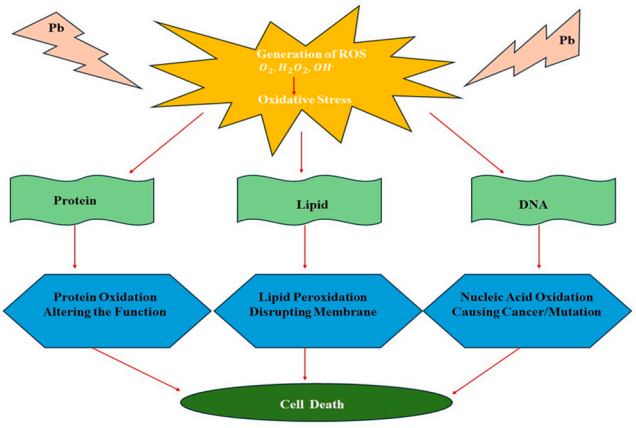

Fig. 5. Possible mechanisms of lead toxicity to cell death.

Pb is risky because it can mimic metals such as Ca, Fe, and Zn. This ability enables Pb to penetrate the BBB and disrupt processes. The negative effects of Pb exposure on health may persist over a prolonged period, particularly referring to long-term health problems later in life because of Pb accumulation both in bones and teeth. Reducing exposure to Pb is essential in the case of Pb-contaminated environments, as children absorb Pb faster than adults. Children are particularly vulnerable to Pb during their growth and developmental stages, which are crucial both for their immediate health and long-term well-being. They can be protected from Pb exposure via preventive actions such as prohibiting use of Pb-based paints, improving industrial emission standards, and raising public awareness.

Environmental implications

The use of lead in paint is of great concern. Although regulatory standards are set, the levels still exceed the safety standards in most countries. With time, the lead content finds its way into the environment and can lead to significant health concerns, especially in children. About 0.6 million children worldwide are estimated to suffer from moderate mental disorders due to lead exposure. The paper concluded that monitoring the health impacts imposed on the next generation exposed to lead prenatally or during their childhood is necessary.

CRediT authorship contribution statement

Muhammad Ubaid Ali: Conceptualization, Methodology, Visualization, Investigation, Validation, Writing – review & editing, Funding acquisition. **Muhammad Zeeshan Gulzar:** Visualization, Investigation, Writing – original draft. **Bisma Sattar:** Writing – review & editing. **Sajeela Sehar:** Software, Data curation. **Qumbar Abbas:** Writing- review & editing. **Muhammad Adnan:** Writing- review & editing. **Jingwei Sun:** Writing – review & editing. **Zhuanxi Luo:** Writing – review & editing. **Gongren Hu:** Resources Investigation. **Ruilian Yu:** Conceptualization, Methodology, Visualization, Investigation, Validation, Writing – review & editing, Funding acquisition. **Ming Hung Wong:** Conceptualization, Methodology, Validation, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

No data was used for the research described in the article.

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