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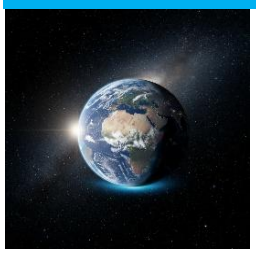

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Heavy Metal Exposure and Psychiatric Disorders: Biological Mechanisms and Age-Related Differences - A Narrative Review

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Abstract

Background:

Mental disorders are among the leading causes of disability worldwide. Beyond genetic and psychosocial determinants, environmental factors such as heavy metal exposure are increasingly recognized as potential contributors to psychiatric risk. Persistent toxic metals may adversely affect central nervous system function across the lifespan.

Aim:

The aim of this review was to analyze current evidence on the association between heavy metal exposure and psychiatric disorders, with emphasis on biological mechanisms and age-related differences.

Material and Methods:

A narrative review of epidemiological, experimental, and clinical studies published up to 2024 was conducted using major scientific databases. The analysis focused on lead, cadmium, mercury, arsenic, and manganese.

Results:

Evidence indicates that heavy metal exposure is associated with neurodevelopmental disorders, cognitive impairment, depressive and anxiety symptoms, and, less consistently, psychotic manifestations. Proposed mechanisms include oxidative stress, neuroinflammation, neurotransmitter dysregulation, hypothalamic–pituitary–adrenal axis disruption, impaired neuroplasticity, and epigenetic changes. Children are particularly vulnerable during critical developmental periods, whereas chronic exposure in adults is more often linked to affective disorders and cognitive decline.

Conclusions:

Heavy metal exposure may represent a modifiable environmental factor contributing to psychiatric vulnerability. Further longitudinal research and improved exposure assessment are required to clarify causal relationships and support preventive strategies.

Keywords: Heavy Metals; Environmental Exposure; Neurotoxicity; Mental Disorders; Child; Adults

1. Introduction

Environmental contamination with toxic metals constitutes a significant global health concern. Heavy metals released through industrial, agricultural, and mining activities may accumulate in biological systems and exert multisystem toxic effects, even at relatively low exposure levels. Increasing attention has therefore been directed toward the potential role of environmental toxicants as determinants of neurological and mental health outcomes [1,2].

Of particular concern is the ability of certain heavy metals to bioaccumulate in tissues and, in some cases, cross the blood–brain barrier, thereby exerting persistent neurotoxic effects [2,3]. Such exposure may disrupt cellular homeostasis and affect the central nervous system through

mechanisms including oxidative stress, interference with enzymatic and signaling pathways, and other forms of molecular injury [4].

Available evidence suggests that the neurodevelopmental and neuropsychiatric effects of heavy metal exposure are heterogeneous and depend on the timing and context of exposure [5,6]. The aim of this review is to summarize current evidence regarding the association between heavy metal exposure and psychiatric disorders, with particular emphasis on mechanistic pathways and age-related differences.

2. Neurotoxicity of Heavy Metals

2.1. Definition and Characteristics

The term *"heavy metals"* refers to a group of chemical elements with a well-documented toxic potential toward living organisms. They enter the natural environment primarily as a result of anthropogenic activities, including mining and smelting operations, industrial processes, and agricultural practices. Even low concentrations may induce cellular and molecular alterations, ultimately affecting the function of multiple organ systems [1,2].

Among heavy metals, those of greatest clinical and environmental relevance include lead (Pb), cadmium (Cd), mercury (Hg), and arsenic (As). These elements are not considered essential for normal human physiological function. However, their bioaccumulation may lead to significant multisystem toxicity, including well-documented neurotoxic effects, particularly in the case of lead [1,2,3].

The toxicity of heavy metals is mediated through multiple interconnected mechanisms, including modulation of gene expression critical for cellular development and homeostasis, structural alterations of biomolecules and proteins, disruption of enzymatic activity, and induction of oxidative stress. The latter plays a central role in the pathogenesis of neuronal injury and neurodegeneration [4].

2.2. Routes of Exposure

Exposure is typically chronic and involves prolonged contact with low concentrations, which significantly complicates clinical recognition. The primary routes of exposure include ingestion of contaminated food and water, inhalation of dust and metal-containing aerosols, and to a lesser

extent dermal contact. Occupational settings represent an additional important source of exposure, particularly in the metallurgical, chemical, and mining industries [1,2].

Heavy metals can cross both the placental barrier and the blood–brain barrier, which is of particular clinical relevance during the prenatal period and early childhood. During these critical stages of neurodevelopment, even low concentrations may lead to long-term alterations in brain structure and function, thereby increasing susceptibility to neuropsychiatric disorders later in life [3,5,6].

3. Biological Mechanisms

The association between heavy metal exposure and psychiatric disorders is mediated by a network of interrelated pathophysiological mechanisms affecting multiple levels of central nervous system organization. Heavy metals exert both direct neurotoxic effects and indirect disturbances of cellular and neuroendocrine regulation, leading to persistent structural,

neurochemical, and functional brain alterations [4,7]. The principal biological pathways implicated in psychiatric vulnerability are summarized in Table 1.

Table 1. Heavy metals, biological mechanisms, and associated psychiatric outcomes

Heavy Metal	Principal Biological Mechanisms	Pediatric Outcomes	Adult Outcomes
Lead (Pb)	Oxidative stress; NMDA receptor dysfunction; dopaminergic disruption; HPA axis dysregulation	ADHD; cognitive impairment; behavioral dysregulation	Depression; cognitive decline
Cadmium (Cd)	Oxidative stress; mitochondrial dysfunction; neuroinflammation	Behavioral problems; cognitive deficits	Depression; anxiety; cognitive impairment
Mercury (Hg)	Dopaminergic dysfunction; oxidative stress; neuroinflammation	Cognitive impairment; emotional disturbances	Mood disturbances; neuropsychiatric symptoms
Arsenic (As)	Oxidative stress; epigenetic changes	Neurodevelopmental impairment	Cognitive impairment
Manganese (Mn)	Dopaminergic system disruption; oxidative stress	Neurodevelopmental vulnerability	Neuropsychiatric symptoms

Source: Authors' own compilation based on references [4–16].

3.1. Oxidative Stress and Neuronal Damage

Induction of oxidative stress represents a core mechanism of heavy metal neurotoxicity. Toxic metals promote excessive generation of reactive oxygen and nitrogen species while impairing endogenous antioxidant defense systems [4]. As a result, lipid peroxidation, protein oxidation, and DNA damage may occur, compromising neuronal integrity.

Given their high metabolic demand and limited regenerative capacity, neurons are particularly susceptible to oxidative injury. Sustained oxidative stress may contribute to synaptic

dysfunction, mitochondrial impairment, and activation of apoptotic signaling pathways, which may underlie both cognitive and affective disturbances observed in exposed populations [4,8].

3.2. Neuroinflammation and Microglial Activation

Heavy metal exposure has been shown to induce chronic neuroinflammatory responses through activation of microglia and astrocytes [9]. Activated glial cells release proinflammatory cytokines, including interleukin-1β, interleukin-6, and tumor necrosis factor-α, which may disrupt synaptic transmission and neuronal homeostasis [9]. Persistent neuroinflammation may impair synaptic plasticity and neurogenesis, particularly within limbic structures such as the hippocampus, and may contribute to progressive neuronal dysfunction [9,11]. Moreover, inflammatory signaling can further enhance oxidative stress, reinforcing a self-perpetuating cycle of neurotoxicity [4,8]. Such mechanisms are increasingly discussed as plausible contributors to mood disorders and cognitive dysfunction [10].

3.3. Neurotransmission Disturbances

Disruption of neurotransmitter systems constitutes another important pathway linking heavy metal exposure to psychiatric manifestations. Toxic metals may interfere with neurotransmitter synthesis, receptor function, and synaptic signaling [4,7,8].

Lead exposure has been associated with alterations in glutamatergic transmission and NMDA receptor function, potentially contributing to executive dysfunction and behavioral abnormalities [12]. Mercury, in contrast, may affect dopaminergic pathways, thereby influencing emotional regulation and impulse control, with potential relevance to depressive, anxiety, and behavioral symptoms [13].

3.4. Dysregulation of the Hypothalamic–Pituitary–Adrenal Axis

Heavy metals may also interfere with neuroendocrine regulation, particularly the hypothalamic–pituitary–adrenal (HPA) axis, a central mediator of the stress response. Evidence suggests that exposure to toxic metals such as lead may disrupt HPA axis function and alter glucocorticoid (cortisol) regulation, supporting a role for stress pathway dysregulation in metal-related neuropsychiatric vulnerability [14].

Chronic HPA axis activation may adversely affect brain regions essential for emotional and cognitive regulation, including the hippocampus and prefrontal cortex. Neuroendocrine

dysregulation may further amplify inflammatory and oxidative pathways, thereby contributing to vulnerability to depressive and anxiety disorders [14,15].

3.5. Impairment of Neurogenesis and Brain Plasticity

Experimental and epidemiological findings indicate that heavy metals may impair neurogenesis and synaptic plasticity, processes essential for learning, memory, and emotional adaptation. Exposure may interfere with neuronal survival and plasticity-related processes, resulting in persistent structural and functional changes [5,8,13].

The hippocampus appears particularly sensitive to such toxic effects. Disruption of plasticity mechanisms may contribute both to early-life neurodevelopmental disturbances and to cognitive decline observed in chronically exposed adults [5,8].

3.6. Epigenetic and Gene–Environment Interactions

Increasingly robust data highlight the role of epigenetic modifications in mediating long-term effects of heavy metal exposure. Toxic metals may influence DNA methylation patterns, histone modifications, and microRNA expression, thereby altering gene regulation without changing the DNA sequence [16].

Such epigenetic alterations may persist over extended periods and may partly explain delayed onset of neuropsychiatric symptoms and interindividual variability in susceptibility. These mechanisms are particularly relevant during critical periods of brain development but may also modulate vulnerability across the lifespan [5,16].

Collectively, these interconnected mechanisms - summarized in Table 1 - illustrate that heavy metal exposure exerts multi-level effects on brain function. Oxidative imbalance, chronic neuroinflammation, neurotransmitter disruption, neuroendocrine dysregulation, impaired plasticity, and epigenetic modulation converge to shape long-term neural vulnerability. This integrative framework supports the hypothesis that heavy metal exposure may act as a

transdiagnostic risk factor contributing to affective, cognitive, and behavioral disturbances across different stages of life.

The clinical relevance of these mechanisms becomes particularly evident when comparing developmental vulnerability in children with cumulative exposure effects observed in adults.

4. Heavy Metal Exposure and Psychiatric Disorders in Children

Children and adolescents constitute a biologically sensitive population particularly vulnerable to the neurotoxic effects of heavy metals. This increased susceptibility is attributable to the immaturity of the blood–brain barrier, rapid neurodevelopmental processes, and proportionally greater exposure relative to body mass. Exposure during critical developmental windows may result in persistent structural and functional brain alterations, manifesting as neurodevelopmental, emotional, and behavioral disturbances [5,6].

4.1. Neurodevelopmental Disorders and ADHD

The most extensively documented association in pediatric populations concerns the relationship between lead exposure and *attention-deficit/hyperactivity disorder (ADHD).* Evidence suggests that even very low blood lead concentrations may be associated with increased impulsivity, hyperactivity, and attentional deficits in children [17].

The neurobiological plausibility of this association is supported by findings demonstrating impaired maturation of the prefrontal cortex, dysfunction of dopaminergic pathways, and alterations in glutamatergic neurotransmission following lead exposure [12,18]. These changes may underlie deficits in executive functioning and inhibitory control.

Timing of exposure appears critical. Meta-analytic evidence indicates that prenatal and early childhood lead exposure increases the risk of ADHD symptoms and their persistence into later developmental stages [19].

4.2. Cognitive Function and Intellectual Development

Exposure to heavy metals during childhood, particularly lead, is strongly and consistently associated with adverse neurodevelopmental outcomes, including impaired cognitive functioning [5,18]. Epidemiological studies indicate that elevated lead concentrations are associated with reduced IQ, attentional deficits, and poorer cognitive performance in children.

These neurocognitive effects are thought to reflect disrupted cortical maturation, especially within prefrontal regions, and altered neurotransmitter systems involved in executive control [18]. Importantly, the consequences may extend beyond psychometric outcomes and include broader developmental and functional difficulties relevant to school performance and everyday adaptation [20].

Available evidence further suggests that the effects of early-life exposure may persist into later developmental stages, indicating sustained neurodevelopmental consequences [5,20].

4.3. Emotional and Behavioral Disorders

An increasing body of evidence links childhood exposure to lead and other toxic metals with emotional and behavioral disturbances, including aggression, impaired emotional regulation, anxiety symptoms, and depressive features. Even low-level environmental exposure has been associated with measurable increases in internalizing and externalizing behaviors at the population level [20,21].

Mechanistically, these disturbances may reflect altered maturation of limbic–prefrontal circuits and dysregulated stress responsivity. Chronic low-grade neuroinflammation and HPA axis dysregulation, as discussed in Section 3, may further contribute to emotional dysregulation during sensitive developmental periods [9,14].

4.4. Long-Term Developmental Consequences

The consequences of childhood heavy metal exposure extend beyond immediate neurodevelopmental and emotional disturbances. Growing longitudinal evidence indicates that early heavy metal exposure may alter developmental trajectories and confer long-term psychiatric vulnerability [5,20].

These consequences include not only persistent psychiatric symptoms but also lower academic attainment, impaired social functioning, and reduced occupational prospects in adulthood [5,20]. These findings align with life-course models of mental health, in which early neurotoxic insults interact with later environmental stressors to amplify long-term psychiatric risk [5].

5. Heavy Metal Exposure and Psychiatric Disorders in Adults

In adults, heavy metal exposure is typically chronic and cumulative, arising from environmental, dietary, and occupational sources [1-3]. In contrast to pediatric exposure, adult exposure rarely results in classical neurodevelopmental disorders; instead, it more frequently manifests as affective disturbances, anxiety symptoms, and progressive cognitive decline [22-24].

Age-related differences in psychiatric manifestations associated with heavy metal exposure are presented in Table 2.

Table 2. Age-Related Differences in Psychiatric Manifestations of Heavy Metal Exposure

Domain	Children	Adults
Vulnerability	Immature blood–brain barrier; active neurodevelopment	Chronic cumulative exposure
Dominant Disorders	ADHD; neurodevelopmental disorders	Depressive and anxiety symptoms; cognitive impairment
Cognitive Impact	IQ reduction; attention deficits; impaired working memory	Memory impairment; executive dysfunction; cognitive decline
Biological Sensitivity	Critical developmental windows; altered brain maturation	Cumulative oxidative burden; neuroinflammation; neuroendocrine dysregulation
Long-Term Risk	Persistent neurodevelopment and psychosocial consequences	Progressive cognitive decline; long-term affective burden

Source: Authors' own compilation based on references [5,6,17–24].

5.1. Depressive Disorders

Among adults, the most extensively documented psychiatric association concerns depressive symptoms and depressive disorders. The strongest evidence implicates cadmium and lead exposure in relation to increased severity of depressive symptomatology. Population-based studies demonstrate that higher blood lead levels and elevated urinary cadmium concentrations are associated with increased depressive symptoms, even at environmentally low exposure levels [22].

Biological mechanisms underlying this association likely involve oxidative stress, neuroinflammation, HPA axis dysregulation, and alterations in neurotransmitter systems, as described previously [4,9,14]. Clinically, such associations may remain underrecognized, particularly when symptoms are subthreshold or nonspecific [22].

5.2. Anxiety Disorders

Epidemiological evidence suggests that heavy metal exposure in adults may also contribute to anxiety symptoms and anxiety disorders. Elevated levels of cadmium and mixed heavy metal exposure have been associated with increased anxiety, irritability, and sleep disturbances [23].

These associations are biologically plausible given the role of stress-regulatory pathways and inflammatory processes in anxiety pathophysiology [9,14]. However, these symptom patterns are often nonspecific and frequently overlap with depressive features, which complicates causal interpretation in clinical settings [23].

5.3. Cognitive Impairment and Brain Aging

Chronic heavy metal exposure in adulthood has been associated with impairments in memory, attention, and executive functioning. Population-based studies indicate that higher levels of lead and cadmium correlate with poorer neuropsychological performance and, in older individuals, accelerated cognitive decline. Cumulative exposure may contribute to processes resembling accelerated brain aging [24]. Mechanistically, oxidative imbalance, chronic neuroinflammation, and mitochondrial dysfunction are considered central contributors to these cognitive effects [4,8,9].

5.4. Psychotic Disorders

Compared with affective and cognitive outcomes, evidence linking heavy metal exposure to psychotic disorders remains limited [5]. Developmental neurotoxicity research suggests that lead exposure may influence dopaminergic and glutamatergic pathways relevant to psychosis vulnerability [5,12]. Clinical observations describe neuropsychiatric manifestations involving perceptual disturbances, thought disorganization, and behavioral dysregulation; however, these associations remain heterogeneous and do not currently support definitive causal conclusions

[5]. Further longitudinal studies are required to clarify the potential contribution of heavy metal exposure to severe psychiatric phenotypes.

6. Public Health Implications

The growing body of evidence linking heavy metal exposure to psychiatric outcomes highlights the importance of environmental determinants in mental health prevention strategies [5,10]. Although causality cannot yet be established definitively, converging epidemiological and experimental findings suggest that chronic exposure to toxic metals may contribute to psychiatric vulnerability at the population level [22,23].

Reducing environmental exposure to heavy metals remains a critical public health priority [1,2]. Regulatory policies aimed at controlling industrial emissions, improving air and water quality, and monitoring food contamination may have long-term benefits not only for somatic health but also for mental well-being. Particular attention should be directed toward vulnerable populations, including children, pregnant women, and individuals residing in highly industrialized or socioeconomically disadvantaged areas [5,6].

Incorporating environmental exposure assessment into mental health research and clinical practice may enhance early identification of at-risk individuals. Biomonitoring strategies and interdisciplinary collaboration among specialists in environmental medicine, psychiatry, and public health may improve risk stratification and support preventive interventions. Furthermore, longitudinal studies integrating environmental biomarkers with psychiatric outcomes are necessary to clarify temporal relationships and dose–response effects [10,24].

7. Conclusions

Current evidence suggests that heavy metal exposure may constitute a modifiable environmental determinant of mental health risk. Converging biological pathways provide a plausible mechanistic framework linking heavy metal exposure to alterations in brain structure and function.

Developmental timing appears critical. Early-life exposure is associated with long-term neurodevelopmental and behavioral consequences, whereas chronic exposure in adulthood is more frequently linked to affective symptoms and cognitive decline. Despite growing research

interest, heterogeneity in exposure assessment and study design limits definitive causal inference.

Future studies should prioritize longitudinal cohorts, standardized biomarker assessment, and integrative models incorporating environmental, genetic, and psychosocial determinants. Strengthening the evidence base may support prevention strategies and environmental health policies aimed at reducing the psychiatric burden associated with heavy metal exposure.

Disclosure

Supplementary Materials

Not applicable.

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