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Влияние загрязнения почвы тяжёлыми металлами на возникновение заболеваний нервной системы

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АННОТАЦИЯ

Антропогенная деятельность промышленно развитых стран приводит к загрязнению почвы тяжёлыми металлами, которые аккумулируются в тканях организма и обладают нейротоксическим действием. С учётом острой экологической проблемы накопления в почве тяжёлых металлов и их токсичности для человека целью исследования был анализ актуальных научных данных об их патологическом воздействии их на нервную ткань. Для достижения поставленной цели обработаны доказательные научные статьи релевантных открытых баз данных за последние пять лет. Согласно научным данным, кадмий, хром, свинец и ртуть считаются наиболее распространёнными металлами, которые загрязняют почву и оказывают нейротоксическое действие. Токсичность тяжёлых металлов в нервной ткани реализуется через множественные механизмы, включая нарушение клеточного цикла, метаболических процессов и целостности гематоэнцефалического барьера. Эти воздействия приводят к дегенеративным изменениям структур центральной нервной системы. Кадмий, свинец, ртуть и хром вмешиваются в развитие нервной системы и её функционирование, вызывая нейротоксические эффекты, вплоть до летальных исходов при острых отравлениях. В районах с неблагоприятной экологической обстановкой важно проводить скрининговые исследования для выявления групп населения, подверженных повышенному риску отравления металлами. Это необходимо для реализации первичных профилактических мер. Такие действия помогут снизить негативное влияние загрязнённой почвы, содержащей тяжёлые металлы, на здоровье людей.

Ключевые слова: микроэлементы; загрязнение почвы; кадмий; свинец; нейротоксичность; ртуть; хром.

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Effect of Soil Heavy Metal Contamination on Incidence of Nervous System Disorders

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ABSTRACT

Human activities in industrialized economies lead to soil contamination with highly neurotoxic heavy metals accumulating in body tissues. Given the urgent environmental issue of heavy metal accumulation in soil and their toxicity to humans, the aim of the study was to analyse the available scientific data on their neurotoxicity. To achieve this aim, evidence-based papers from the relevant open databases over the past five years have been reviewed. According to scientific evidence, cadmium, chromium, lead, and mercury are considered the most common neurotoxic metals that pollute the soil. The heavy metal toxicity to nervous tissue has various mechanisms, such as the impairment of the cell cycle and metabolism and blood-brain barrier disruption. This leads to structural degeneration of the central nervous system. Cadmium, lead, mercury, and chromium can affect the development and functions of the nervous system, resulting in neurotoxicity that can be fatal in cases of acute poisoning. Screening, identification of populations with the increased risk of metal poisoning, and primary prevention in environmentally unfavourable areas are the appropriate actions to solve the problem of heavy metal contamination of the soil and their adverse effect on the body.

Keywords: trace elements; soil contamination; cadmium; lead; neurotoxicity; mercury; chromium.

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土壤重金属污染对神经系统疾病发生的影响

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摘要

工业发达国家的人类活动导致土壤受到重金属污染，这些金属可在机体组织中累积并产生神经毒性作用。考虑到土壤中重金属累积所引发的严重生态问题及其对人体的毒性，本研究旨在分析现有科学数据，评估其对神经组织的病理性影响。为实现研究目的，对近五年公开数据库中的循证科学文献进行了整理和分析。 科学证据表明，镉、铬、铅和汞是最常见的污染土壤并产生神经毒性作用的金属。重金属在神经组织中的毒性通过多种机制介导，包括细胞周期紊乱、代谢过程破坏以及血脑屏障完整性受损。这些作用最终导致中枢神经系统结构的退行性改变。镉、铅、汞和铬干预神经系统的发育与功能，导致神经毒性效应，在急性中毒时可引起致死结局。对高风险社会群体开展金属中毒筛查，并在生态不利地区实施初级预防，是应对土壤重金属污染及其对人体不良影响的合理措施。

关键词：微量元素；土壤污染；镉；铅；神经毒性；汞；铬。

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INTRODUCTION

Human activities have resulted in an ecological crisis primarily manifested by the adverse effect on the health of the population [1]. A significant environmental and global public health concern pertaining to the toxic metal contamination of ecosystems has been identified in numerous countries [2]. Thus, anthropogenic biosphere alterations give rise to the urgent task of preserving health and extending human life expectancy in the contemporary contexts [3]. It is critical to understand the etiology and patterns of the adverse effects of chemical soil pollution with heavy metals on human health to prevent potential negative processes that may result in specific syndromes and diseases.

The most common anthropogenic pollutants of the environment and soil are heavy metals as they are the third most toxic substances after pesticides and nitrates as indicated by Shen *et al.* [4]. The anthropogenic accumulation of heavy metals can be caused by various factors, including metallurgical and energy production, transport pollution, structural corrosion, mining, and poor waste management [5]. The primary entry point of heavy metals into the environment is dust and its deposits on the soil and leaves, i.e. dry deposits.

Heavy metals play a dual role in the soil; as trace elements, they are essential for physiological processes in the biosphere; however, their high concentrations are toxic and affect the health of humans and animals [6]. The heavy metal molecules have been found at all levels of the ecological pyramid, intensifying the problem of their effect on the body and emphasizing the need for rapid identification of their etiological relationships. This is particularly crucial in terms of accumulation and long-term effects. According to Rahman and Singh, despite the presence of natural heavy metals in the Earth's crust and soil, the contemporary anthropogenic activities, including mining, electroplating, smelting, household, and related industries, lead to abundant environmental pollution and human exposure to toxic metals [7]. Each heavy metal is unique in terms of its physiological effects; however, according to the study by Zaynab *et al.*, high concentrations of these metals can have a deleterious effect on the gastrointestinal tract, cardiovascular, endocrine, nervous, and reproductive systems [8].

Initially, heavy metals released from the contaminated soil to the human body accumulate in tissues and organs, gradually reaching a certain threshold in the body. At this point, disorders are initiated, including the enzyme system disorders, metabolic disorders, immunologic reactions, and the main organ system dysfunctions [9]. The continued toxicity leads to the development of specific symptoms with further clinical manifestations of the disease. Recent research by Ahmad *et al.* revealed that the oxidative breakdown of biological macronutrients is associated with the binding of heavy metals to cellular components in the form of structural proteins, enzymes, and nucleic acids. This, in turn, is associated with their dysfunction [10].

Chronic exposure to heavy metals has been shown to have severe consequences, including carcinogenic induction of cell metaplasia and negative effects on the cardiovascular, central and peripheral nervous systems [11]. The nervous system plays a pivotal role in regulating the somatic and vegetative innervation of all internal organs, and physical and psychoemotional control and conscious functions. Thus, studying its tendency to the negative chronic effect on the soil is of paramount importance. Organic and structural disorders of the central nervous system (CNS) have been demonstrated to promote mixed disorders of the endocrine system, autonomic nervous system, metabolic, and other systems of the body.

Some epidemiological and experimental studies show a significant relationship between the exposure to heavy metals in soil and neurotoxicity. Many neurodegenerative diseases, including Alzheimer disease, Parkinson disease, amyotrophic lateral sclerosis, multiple sclerosis, and attention deficit hyperactivity disorder, are caused by heavy metal toxicity [12]. The neurotoxicity of heavy metals has been shown to be associated with epigenetic changes related to histone modifications. Heavy metals alter gene transcription, leading to neurological and neurodevelopmental disorders [13]. Moreover, heavy metals have been shown to disrupt protein folding mechanisms, increasing the risk of neurodegenerative diseases in the exposed populations. This process is increasingly recognized as a critical factor in the development and progression of neurodegenerative diseases, such as Alzheimer and Parkinson diseases [14]. Glial cells are particularly susceptible to metal-induced neurotoxicity. The accumulation of heavy metals in the brain contributes to the microglial activation, promoting inflammatory responses together with other types of neurotoxicity, impaired synaptic transmission, cognitive impairment, and neuronal damage [15]. In addition, exposure to heavy metals, such as cadmium and mercury, can lead to adverse neurocognitive outcomes in adults via various pathways [16]. Moreover, exposure to multiple heavy metals increases this risk more than the exposure to individual heavy metals [17].

Considering the acute environmental issue of soil pollution with heavy metals and their diverse adverse effect on human health, we conducted a comprehensive analysis of the latest evidence on the effect of heavy metals on the human nervous system.

METHODS

To analyze contemporary evidence on the relationship between the soil heavy metal contamination and possible organic and structural disorders of the central and peripheral nervous systems, a systematic review of ecology, geology, neurology, laboratory diagnosis, epidemiology, internal and social medicine papers is required. To select data for subsequent review, we used some high impact publications from the relevant and reliable journals. The database of the

reviewed articles, statistics, clinical guidelines, and literature reviews was created based on advanced evidence, including long-term studies and observations of various medical cases, such as patients with long-term effects of heavy metal poisoning, central, or peripheral nervous system disorders associated with chronic heavy metal exposure and patients with the industrial exposure to heavy metals (metal exposure as an occupational hazard). The latest meta-analyses have also been included, which covered large cohorts of patients from 3 to 5 years of follow-up with different nervous system disorders to analyse the chronicity or resolution of heavy metal accumulation in the body.

To study scientific evidence, the paper reviews the medical articles in special and relevant publications from 2019 to 2024. The literature search was conducted across PubMed, Scopus, Google Scholar, and Web of Science using the keywords *тяжелые металлы (heavy metals), тяжелый металл (heavy metal)*, *кадмий (cadmium), хром (chromium), свинец (lead), ртуть (mercury), загрязнение почвы (soil pollution), нейротоксичность (neurotoxicity)*, and *нервная система (nervous system)* (see Fig. 1).

In most cases, we used the relevant scientific evidence from open databases. During our study, we identified the researcher who wrote the paper by an authorized entry and search on academic search platforms. Scientific databases facilitate the exclusion of search results authored by the same researchers, related papers, and outdated evidence and verification of the citation and impact factor of the paper and the publication itself. In addition to a filter by the publication date, we used the keyword search. This approach enabled to exclude the papers on the effect of other soil contaminants or other target organs and body systems not related to the subject of this study. In addition, we excluded papers with short-term medical observations. In addition to scientific articles, the paper reviews the latest recommendations of the World Health Organization (WHO) and international organizations for the prevention of anthropogenic effects of hazardous elements on the human nervous system. It also includes a recommendation on the early detection, diagnosis, and prevention of heavy metal accumulation.

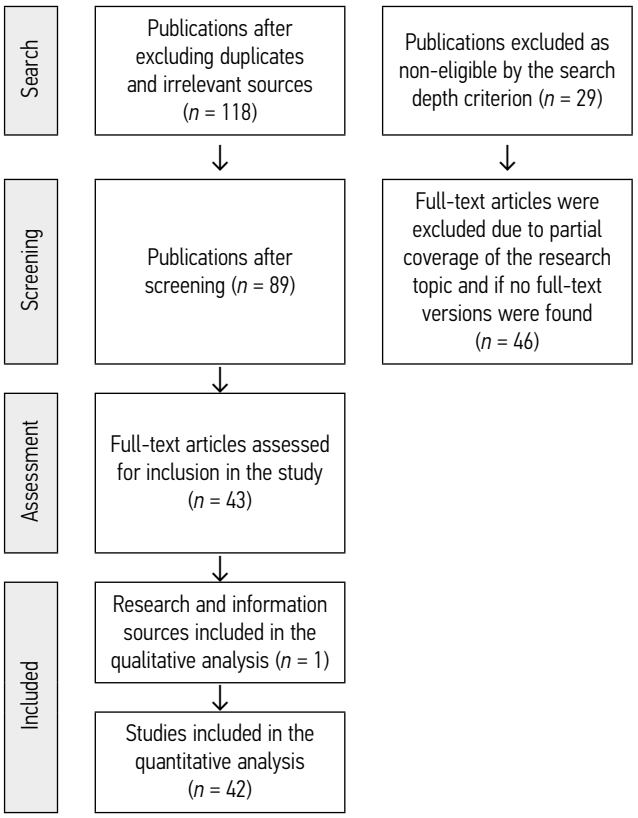

Fig. 1. Search and selection strategy.
Рис. 1. Стратегия поиска и отбора.

RESULTS AND DISCUSSION

Given the prevalence of heavy metals in the biosphere as a result of various anthropogenic activities, including agricultural, household, medical, industrial, and technology sectors, there is an urgent need to solve the critical issue of their abnormal uptake and effect on various human organ systems. It has been found that the manifestation of high toxicity of heavy metals in the body is related to some factors, including the source of pollution and the patient's history [18]. The analyzed factors include the chemical composition of the compound; the dosage, the duration, and route of exposure, and the amount of accumulated metal [19]. In the context of patient care, certain factors have been identified as particularly important for determining the severity and sensitivity of morbidities. These include age, sex, genetic predisposition, nutritional status and quality, biorhythms, toxic workplace environment, and co-morbidities. Heavy metals, including lead, tin, arsenic, cadmium, and mercury, are frequently used in industrial processes. According to recent studies, cadmium, chromium, lead, and mercury are considered the prevalent metals in contaminated soil and a direct hazard to the human nervous system due to their proven toxicity [20]. The toxicity of these heavy metal compounds is measured by systemic toxicants to determine the degree of their effect on the patient and to assess possible damage to organ systems.

According to Alvarez *et al.*, heavy metal compounds do not metabolize in body tissues, which leads to their direct uptake during chronic exposure, i.e. bioaccumulation as a result of transdermal or parenteral uptake due to soil contamination [21]. As posited by Chen *et al.*, the toxicity of heavy metals and their ions is also associated with their solubility in aqueous solutions [22]. The ingestion of contaminated soil containing water and heavy metals may inhibit vital enzymes in the blood, inducing possibly lethal functional incapacitation. Thus, even negligible amounts of these substances may have severe physiological effects. In addition to intensive bioaccumulation, heavy metals do not biodegrade, i.e. they cannot be destroyed, detoxified, or totally removed from the body [23]. The bioaccumulation of heavy metals in the tissues is exacerbated in cases of ingestion of animal products that have been exposed to and accumulated

heavy metals in the contaminated soil. Therefore, in such scenario, the concentration of metals in food may increase exponentially, reaching the concentrations 100,000 times higher than those found in soil. This has been linked to the development of various disorders, including nervous system disorders, and carcinogenesis [24].

Cadmium

Cadmium is used in various types of household batteries, plastic products, industrial pigments, and metal structures. According to Wang *et al.*, it is also widely used in electroplating [25]. Moreover, it has been determined that coal and mineral solutions in soils also contain cadmium. In the latest recommendations of the International Agency for Research on Cancer and WHO, cadmium compounds are classified as a human carcinogen [26]. Fertilizers is the primary source of soil contamination with cadmium, which is introduced in the content in plants consumed by humans. It has been found that an additional source of soil cadmium is combustion products with large forest fires being a major contributor. Cadmium content in wood ash ranges from 2 to 32 mg/kg and is more than 9 mg/kg in straw ash [27]. Given that the ash has mainly alkaline properties, the cadmium present in it is insoluble in water and does not effectively penetrate plant tissues. However, according to Rizwan *et al.*, cadmium has been found to accumulate in the soil and during fermentation, it becomes available for absorption by plants [28]. It has been shown that cadmium can gradually accumulate in the human body. Moreover, it has been observed that cadmium, in conjunction with zinc, enters seawater through a network of surface and ground soils. Despite the reports on the reduced cadmium contamination of the soil in various industrialized economies by Wu *et al.*, the issue is still concerning, particularly with regard to its carcinogenic effects on agriculture and metallurgy workers and individuals residing in areas with soil contaminated with cadmium [29].

For a considerable period, the effect of cadmium on the human body was limited to the research of its accumulation in the nephrons, specifically in the epithelial cells of the proximal tubules, and bone tissue, which was characterized by homeostasis disorders during the lamellar bone mineralization [30]. Recent studies of large groups of patients have demonstrated statistically significant structural effects of cadmium on nervous tissue in both central and peripheral nervous systems. This evidence is supported by the research by Zhou *et al.* [31]. First, these studies investigate the evidence-based cadmium accumulation in brain tissues, which is also associated with the blood-brain barrier (BBB) disruption during the bioaccumulation of cadmium compounds in soft tissues. The BBB disruption during chronic accumulation of cadmium has been shown to be pathophysiologically associated with the oxidative and antioxidant homeostasis disorder of the capillary system of the central nervous system, promoting oxidative stress [32]. Conversely, in acute cadmium intoxication, its highest levels have been observed in the nervous system structures not protected by the BBB, namely the pituitary gland and meninges, as previously documented by Branca *et al.* [33]. The degree of BBB resistance to the penetration of cadmium and its compounds is also related to the patient's medical history, including his or her age, comorbidities, and substance abuse [34].

According to current statistics, the blood and urine cadmium levels in the developed countries with a developed industrial infrastructure ranges from 0.005 to 7.01 μg/L and 0.04 to 14 μg/g, respectively [35]. Cadmium neurotoxicity involves the tissue oxidative stress (with a predominance of oxidants), affecting the activity of enzymes critical for neuronal function and intercellular homeostasis in the brain. This affects the cell cycle and the neuronal and neuroglial apoptosis [36, 37]. According to Branca *et al.* [38], cadmium in nervous tissue promotes neuronal cell cycle completion by blocking the proliferation of protoplasmic and fibrocytic astrocytes, causing apoptosis and necrosis of multipolar brain neurons. This effect on the cell cycle is evidenced by altered intracellular calcium ion levels; increased reactive oxygen species secretion; increased caspase immunoreactivity, and the increased expression of apoptotic factors. Recent studies by Ge *et al.* show that cadmium can disrupt the neuronal cytoskeleton development by inhibiting the expression of proteins involved in assembling and arranging cytoplasmic neurofilaments. These neurofilaments are marker organelles specific to neurons [39].

In cadmium-induced oxidative stress, high levels of malondialdehyde, nitric oxide, and oxidized glutathione are detected. Another mechanism of cadmium neurotoxicity is explained by the influence of its compounds on the activity of calcium adenosine triphosphatase and calcium/magnesium-dependent adenosine triphosphatase [40]. The effects of such deactivation are manifested by lower levels of calcium ions, which are critical for synaptic communication in all synapses of the central nervous system (CNS). Thus, cadmium affects nervous tissue through various mechanisms disrupting the cell cycle and intracellular metabolism, resulting in premature degeneration.

Lead

Diseases associated with lead accumulation are called lead poisoning, or saturnism. Lead is a highly toxic heavy metal with cumulative properties that mainly affect the human nervous system [41]. The metal content in fruits and vegetables contaminated with lead may increase by more than tenfold compared to the natural level in uncontaminated soil. Small amounts of lead are present in almost all plant crops. However, its level is especially high when these crops are grown in lead-contaminated soil. Scientific evidence shows that high levels of lead in cereal grains, legumes, and other foods are extremely toxic to humans and affect the yield of field crops. Lead itself affects the physicochemical

parameters and content of the soil microbial environment [42]. The gastrointestinal and nervous systems are the primary exposure targets of lead in households [43]. For its effect on the central nervous system, lead is highly neurotoxic, which is manifested by impaired neurophysiological function and symptoms of mental disorders and neurocognitive syndromes [42].

It has been proven that the child's body is more susceptible to the neurotoxic effects of lead and its compounds than adults. A study involving pediatric patients by Naranjo *et al.* showed that children are still exposed to lead despite widespread community and healthcare system awareness of its toxicity in different countries [44]. The authors demonstrate that, even in children with blood lead levels below the toxic threshold, specific therapy should be used to prevent negative effects on the CNS. The N-methyl-D-aspartate receptor is involved in the maturation of brain neurons and their functional plasticity, i.e. the processes that occur during the first three months of human prenatal development. Lead inhibits this receptor, leading to the interruption of the long-term potentiation of learned skills and memory abilities. Lead can also penetrate the BBB, inhibiting the endotheliocytes in the BBB system. Histocytological effects of lead disrupt both the prenatal and early nervous system development. These disorders include disruption to signalling, growth and differentiation factors during the proliferation and differentiation of CNS multipolar neurons; impaired synaptic connectivity due to reduced production of sialic acid by neurocytes, and impairment of the chronological sequence of glial cell differentiation. The pharmacological effects of lead poisoning include lead replacing calcium and disrupting calmodulin cascades [45]. Lead also blocks the secretion of neurotransmitters in the presynaptic membrane and their release to the synaptic cleft, disrupting the GABA-ergic, dopaminergic, and cholinergic systems of the CNS. In the cytoplasm, lead blocks the release of calcium ions from both the cell's cytoplasm and the mitochondria themselves. This leads to the accumulation of reactive oxygen species, the activation of mitochondrial lysis, and the initiation of apoptosis or necrosis [45].

Mercury

Mercury is present in almost all human foods, ranging from 1 to 50 μg/kg of body weight. Levels may be higher in seafood [46]. This metal is found in soil and water contaminated with heavy metals and can be converted to methylmercury by microorganisms. Methylmercury and mercury chloride are highly carcinogenic as evidenced by Kim *et al.* [47]. When mercury-contaminated food is consumed, mercury is easily absorbed by enterocytes and almost 100% of ingested mercury is deposited in cells and not excreted. The nervous system is sensitive to all types of mercury because of its high neurotoxicity. Once consumed, mercury first forms complexes with the sulfhydryl groups of blood plasma proteins and tissues before being transported through cell plasma membranes to target organs. More than 12% of the mercury entering the body is deposited in brain tissues with smaller amounts deposited in hepatocytes and epithelial cells in the nephrons. According to Yang *et al.*, the typical symptoms of organic mercury poisoning include depressive disorder, headache, limb tremor, memory disorders, gastrointestinal disorders (e.g. diarrhea and nausea), skin rash, fatigue, and high blood pressure. Human activities directly or indirectly lead to soil pollution with three types of mercury, including the elemental, inorganic, and organic [48].

Methylmercury is extremely toxic to most bodily tissues and can easily penetrate three-dimensional cell structures. This has been proven by many studies, including Abbott and Nigussie [49]. Biochemical manifestations of mercury toxicity include blocking sulfhydryl-containing enzymes involved in cellular metabolism; increased circulation of reactive oxygen species; oxidative stress, and impaired intracellular functioning of calcium ions. The latter is similar to the effect of lead in the cells of target organs. As intracellular calcium ions have many functions important for both synaptic transmission and neuronal function, changes in intracellular calcium levels is the main mechanism underlying mercury neurotoxicity. These changes include the inhibited ability of cells to utilize calcium from intracellular stores, changes in the physical properties of calcium penetration through specific transmembrane channels in the plasma membrane, and protein phosphorylation changes. Oxidative stress due to mercury poisoning can directly or indirectly affect the cellular viability of the nervous system by disturbing the intracellular calcium homeostasis.

Methylmercury is very similar to thiol groups in human cells. Thus, intoxication during the prenatal differentiation of neuroblasts and late neurulation leads to the aberrant migration of stem cells and the disorganization of the developing brain neocortex. According to hypotheses proposed by Yawei *et al.* [50] and Zulaikhah *et al.* [51] but not yet studied in humans, methylmercury disrupts genetic sequences that control the normal neurulation during the first trimester of pregnancy. This alters cellular signalling factors of neuroblastic migration, leading to dysplasia and abnormal cortical and myeloarchitectonics. Among these signalling pathways, the Notch receptor is distinguished as being sensitive to the effects of mercury even at threshold concentrations as proven in experimental animals [52]. Dórea's study shows that the methylmercury neurotoxicity is associated with the inhibition of cytoplasmic microtubule polymerization, which in turn blocks cell migration and the cell division cycle as the mitotic spindle formation for cell division is impossible [53].

Chromium

As Pavesi and Moreira point out [54], chromium is present in the biosphere in various oxidation states. However, it is trivalent and hexavalent chromium that is toxic to the human body. Sources of chromium pollution include the combustion of oil and coal, pigment oxidizers, household

fertilizers, chromium steel and the drilling of oil wells. The effect of chromium on the human body depends on the dosage, exposure route, and exposure duration. Chromium compounds can act directly at the contact site—this is particularly the case with the skin—or be transported and accumulated in other tissues. Hexavalent chromium is a global environmental pathogen that increases the risk of cancer and nervous system disorders due to its neurotoxicity [55]. Some studies have shown that chronic occupational exposure to chromium can impair the olfactory function, increase the risk of motor neuron disease in cases of complex heavy metal poisoning, and promote schizophrenia, particularly in individuals with a psychiatric history as discussed by Ma *et al.* [56]. The authors highlight that high levels of chromium at the initial stages of schizophrenia may exacerbate serotonin synthesis, thereby contributing to the disease burden [57].

According to a study by Pavesi and Moreira [54], trivalent chromium circulates in the organic soil matter and as oxides, hydroxides, and sulphates. Studies of chromium exposed workers, a social group with a high risk of complications due to chromium exposure, show that they suffer from regular headaches, vertigo, and fatigue. However, there is no evidence on its neurological effects [58]. There is evidence of acute neurological complications in people with acute chromium poisoning following the ingestion of more than 8 mg of potassium dichromate. These complications include potentially lethal cerebral edema and necrotic lesions. Thus, the contaminated soil involves certain health hazards based on the range of heavy metals which exceed threshold levels. Given the wide range of pathways of heavy metal accumulation in the human body from the contaminated soil and the increasing number of industrial and agricultural sources of soil contamination with metals, this issue is still relevant for the healthcare systems of the developed countries worldwide. Table 1 summarizes the basic properties of the heavy metals discussed above for the diagnostic differentiation of their effects on the human nervous system.

Despite some common pathophysiological effects of heavy metals on the human nervous system, such as oxidative stress in the circulatory system and neuroglia, the disruption of calcium-dependent intracellular metabolism, and synaptic transmission blocking in the multipolar CNS, particular heavy metals also have specific effects related to its valency and compounds. Further research on this issue should include studies of specific regions with contaminated soil, identification of the affected patient populations, and diagnosis of comorbidities that may lead to a complicated course of events.

CONCLUSION

Detailed studies of the chain of influence and the pathogenetic cascades involved in the development of nervous system disorders are important for developing the appropriate actions to prevent and protect from their chronic effects. Early exposure to toxic metal compounds can affect the fetal and early neurological development. The pathophysiological effects of heavy metals on cells and neuroglia in the human nervous system have some common properties, including the oxidative stress in the circulatory system and neurocytes; the inhibition of calcium-dependent intracellular metabolism, and the disruption of synaptic transmission in multipolar neuron systems. However, each metal has specific properties that facilitate a differentiated diagnosis. Primary prevention and the identification of high-risk populations are reasonable and cost-effective steps to prevent the effect of soil contaminated with heavy metals on critical organs and structures of the nervous system from a public health perspective.

ADDITIONAL INFO

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Table 1. Differentiated comparison of the main manifestations of soil heavy metals on the human nervous system

Таблица 1. Дифференцированное сравнение основных проявлений воздействия почвенных тяжелых металлов на нервную систему человека

Manifestations	Cadmium	Lead	Mercury	Chromium
Influence on the prenatal development of the nervous system	Possible	Proven	Proven	Possible
Effect on the child's body	Possible	Proven	Proven	Possible
Disruption of the blood-brain barrier	Yes	Yes	Yes	Not typical
Mental disorders in metal poisoning	Yes	Yes	Not typical	Not typical
Disruption of synaptic transmission	Not typical	Typical	Typical	Typical
Neurological symptoms	Yes	Yes	Yes	Yes

Source: compiled by the authors.

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