

## Влияние загрязнения почвы тяжёлыми металлами на возникновение заболеваний нервной системы

Г.А. Батырова<sup>1</sup>, Г.А. Умарова<sup>1</sup>, С.Т. Уразаева<sup>1</sup>, У.К. Сарсембин<sup>2</sup>, А.Н. Исалдинова<sup>1</sup>, Г.Е. Таскожина<sup>1</sup>, Ж.Х. Исангужина<sup>1</sup>, Е.А. Умаров<sup>1</sup>

<sup>1</sup> Западно-Казахстанский медицинский университет им. Марата Оспанова, Актобе, Республика Казахстан;

<sup>2</sup> Актюбинский региональный университет им. К. Жубанова, Актобе, Республика Казахстан

### 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 о 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 функционирование, вызывая нейротоксические эффекты, вплоть до летальных исходов при острых отравлениях. Скрининговое выявление социальных групп повышенного риска отравления металлами и первичная профилактика в экологически неблагоприятных районах являются целесообразными мерами в борьбе с проблемой влияния загрязнённой тяжёлыми металлами почвы и их негативного влияния на организм.

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

### КАК ЦИТИРОВАТЬ:

Батырова Г.А., Умарова Г.А., Уразаева С.Т., Сарсембин У.К., Исалдинова А.Н., Таскожина Г.Е., Исангужина Ж.Х., Умаров Е.А. Влияние загрязнения почвы тяжёлыми металлами на возникновение заболеваний нервной системы // Экология человека. 2025. Т. 32, № 7. С. XX–XX. DOI: 10.17816/humeco643565 EDN: ADYJZU

Рукопись поступила: 30.12.2024

Рукопись одобрена: 11.08.2025

Опубликована online: 24.08.2025

Статья доступна по лицензии CC BY-NC-ND 4.0 International License

© Эко-Вектор, 2025

## The Effect of Soil Heavy Metal Contamination on the Occurrence of Diseases of the Nervous System

Gulnara Batyrova<sup>1</sup>, Gulmira Umarova<sup>1</sup>, Saltanat Urazayeva<sup>1</sup>, Umbetali Sarsembin<sup>2</sup>, Assel Issaldinova<sup>1</sup>, Gulaim Taskozhina<sup>1</sup>, Zhamilia Issanguzhina<sup>1</sup>, Yeskendir Umarov<sup>1</sup>

<sup>1</sup> West Kazakhstan Marat Ospanov Medical University, Aktobe, Republic of Kazakhstan;

<sup>2</sup> K. Zhubanov Aktobe Regional University, Aktobe, Republic of Kazakhstan

### ABSTRACT

Anthropogenic activities of industrialized countries lead to soil contamination with heavy metals, which accumulate in body tissues and have a high neurotoxicity. Given the acute environmental issue of accumulation of heavy metals in the soil and their toxicity to humans, the aim of the study was to analyse current scientific data on their pathological effects on the nervous tissue. To achieve this goal, evidence-based scientific articles from relevant open databases over the past five years have been processed. According to scientific data, cadmium, chromium, lead, and mercury are considered the most common metals that pollute the soil and have neurotoxicity. The toxicity of heavy metals in nervous tissue manifests through various mechanisms, such as disruption to the cell cycle and metabolic processes, as well as impairment to the integrity of the blood-brain barrier. These effects result in degenerative changes to the structures of the central nervous system. Cadmium, lead, mercury and chromium can interfere with the development and functioning of the nervous system, resulting in neurotoxic effects that can be fatal in cases of acute poisoning. Screening, identification of social groups at increased risk of metal poisoning and primary prevention in ecologically unfavourable areas are appropriate measures to combat the problem of the impact of soil contaminated with heavy metals and their negative impact on the body.

**Keywords:** trace elements; soil pollution; cadmium; neurotoxicity; mercury; chromium.

#### TO CITE THIS ARTICLE:

Batyrova G, Umarova G, Urazayeva S, Sarsembin U, Issaldinova A, Taskozhina G, Issanguzhina Zh, Umarov Ye. The Effect of Soil Heavy Metal Contamination on the Occurrence of Diseases of the Nervous System. *Ekologiya cheloveka* (Human Ecology). 2025;32(7):XX-XX. DOI: 10.17816/humeco643565 EDN: ADYJZU

Received: 30.12.2024

Accepted: 11.08.2025

Published online: 24.08.2025

The article can be used under the CC BY-NC-ND 4.0 International License

© Eco-Vector, 2025

## INTRODUCTION

Human economic and industrial activity has resulted in an ecological crisis, a significant manifestation of which is the adverse impact on the health of the population [1]. A significant environmental and global public health concern has been identified in numerous countries, pertaining to the contamination of ecosystems by toxic metals [2]. Consequently, anthropogenic alterations in the biosphere give rise to the prevailing concern of sustaining health and augmenting human life expectancy within contemporary contexts [3]. It is imperative to comprehend the aetiological causes and patterns of the pathological effects of chemical soil pollution with heavy metals on human health, in order to prevent potential negative processes that may result in specific syndromes and diseases.

The principal anthropogenic pollutants of the environment and soil are heavy metals, which poisoned the third most significant number of individuals after pesticide and nitrate poisoning, as indicated in the work by X. Shen et al. [4]. The anthropogenic accumulation of heavy metals can be attributed to various factors, including metallurgical and energy production, transport pollution, corrosion of technical structures, mining, and inefficient waste disposal [5]. The primary entry point of heavy metals into the environment is through dust and its deposits on the soil and leaves, that is, in the form of dry deposits.

The presence of heavy metals in the soil has a dual meaning: as trace elements, they are necessary for the course of physiological processes in the biosphere, but at the same time, they are toxic at elevated concentrations, which negatively affects the health of humans and animals [6]. The presence of heavy metal molecules has been observed at all levels of the ecological pyramid, thereby intensifying the problem of their impact on the body and emphasising the necessity for rapid identification of their aetiological interactions. This is particularly crucial with regard to the capacity for accumulation and the long-term consequences. Despite the natural presence of heavy metals in the earth's crust and soil, the anthropogenic activity of modern realities, according to Z. Rahman and V.P. Singh, for example, mining, electroplating, smelting, household and allied industries lead to abundant environmental pollution and human exposure to toxic metals [7]. Each type of heavy metal has its own unique characteristics in terms of its physiological effects; however, according to the research conducted by M. Zaynab et al., the presence of 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 contaminated soil into the human body accumulate in tissues and organs, gradually reaching a certain threshold level within the body. At this point, pathological disorders are initiated, which include changes in the activity of enzyme systems, metabolic processes, immunological reactions, and disruption of the activity of the main organocomplexes [9]. At the subsequent stage of influence on the body, symptoms of a specific disease emerge, unfolding into the clinical picture of the disease. Recent research by W. 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 the pathology of their functioning [10].

Chronic exposure to heavy metals has been demonstrated 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. Additionally, it is responsible for the control and conscious functions of the physical and psychoemotional sphere. Consequently, studying its tendency to the negative chronic influence of the soil is of paramount importance. The violation of the organs and structures of the central nervous system (CNS) has been demonstrated to result in a complex disruption of the endocrine, autonomic, metabolic and other areas of the body.

A number of epidemiological and experimental studies demonstrate a significant link between exposure to heavy metals in soil and neurotoxicity. Many neurodegenerative diseases, including Alzheimer's disease, Parkinson's 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]. Furthermore, heavy metals have been shown to disrupt protein folding mechanisms, thereby increasing the risk of neurodegenerative diseases in exposed populations. This process is increasingly recognised as a critical factor in the development and progression of neurodegenerative diseases such as Alzheimer's and Parkinson's [14]. Glial cells are particularly susceptible to metal-induced neurotoxicity. The accumulation of heavy metals in the brain contributes to the activation of microglia, triggering inflammatory responses that may coincide with other mechanisms of neurotoxicity, causing changes in synaptic transmission, cognitive impairment, and neuronal damage [15]. Furthermore, 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 exposure to individual heavy metals does [17].

Considering the acute environmental problem of soil pollution with heavy metals and their diverse negative impact on human health, the purpose of this work was to conduct a comprehensive analysis of the latest scientific data on the effect of heavy metals on the human nervous system.

## MATERIALS AND METHODS

In order to analyse modern scientific data on the relationship between soil contamination with heavy metals and their influence on possible complications in the work of organs and structures of the central and peripheral nervous system, a systematic analysis of scientific publications in the fields of ecology, geology, neurology, laboratory diagnostics, epidemiology, internal and social medicine is required. For the selection of data for the purpose of subsequent analysis and consecration, a number of publications were selected that were published by relevant and reliable periodicals with a high impact factor. The database of processed articles, statistical data, clinical recommendations and literature reviews was based on the principle of using advanced and evidence-based data that reflect the results of long-term studies and observations of various cases from practical medicine: for patients with chronic forms of complications as a result of heavy metal poisoning, with pathologies of the central or peripheral nervous systems due to chronic exposure to heavy metals, and patients with 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 in the projection from 3 to 5 years of follow-up with different options for complicating the functionality of the nervous system to analyse the features of chronicity or clearance of heavy metal accumulation processes in the body.

To study scientific data, the work includes medical publications for the period from 2019 to 2024 in specialized and relevant publications. The literature search was conducted using the keywords “heavy metals” and “heavy metal” and “cadmium” and “chromium” and “lead” and “mercury” and “soil pollution” and “neurotoxicity” and “nervous system” in PubMed, Scopus, Google Scholar, and Web of Science databases (Figure 1).

In the majority of cases, material from open databases of relevant scientific data was utilized. During the course of the work, the researcher-author of the work was identified, that is to say, an authorized entry and search on academic search platforms were used. The utilization of scientific databases facilitates the exclusion of results from the same scientists, related works, outdated data, and expedites the process of verifying the citation and impact factor of the work and the publication itself. In addition to the implementation of a filter for the robot by publication date, a number of keywords were also utilized during the search process. This approach enabled the exclusion of robots that addressed the influence of other elements of contaminated soil, or other target organs and body systems that were not pertinent to the subject of this work. Additionally, scientific works that addressed short-term medical observations were excluded. In addition to scientific articles, the work includes an analysis of the latest recommendations of the World Health Organization (WHO) and world associations in the field of prevention of the anthropogenic impact of hazardous elements on the human nervous system. It also includes a recommendation regarding the early detection, diagnosis, and prevention of heavy metal accumulation.

## RESULTS AND DISCUSSION

Given the prevalence of heavy metals in the biosphere, as a result of various anthropogenic activities in agricultural, household, medical, industrial and technological sectors, there is an urgent need to address the critical question of their pathological accumulation and impact on the state of various human organ systems. It has been established that the manifestation of high toxicity of heavy metals in the body is contingent on a number of factors, including the source of pollution and the patient's anamnestic data [18]. The factors to be considered include the chemical composition of the compound, the dosage, the duration of exposure, the route of exposure, and the amount of accumulated metal [19]. In the context of patient care, a number of factors have been identified as being of particular importance in determining the severity and sensitivity of pathological manifestations. These include age, gender, genetic predisposition, state and nutritional value, the work of biorhythms, the presence of toxic working conditions and background diseases. Heavy metals, including lead, tin, arsenic, cadmium, and mercury, are frequently employed in industrial processes. According to recent studies, cadmium, chromium, lead, and mercury are considered priority metals that are prevalent in contaminated soil and pose a direct threat to the human nervous system due to their proven toxicity [20]. The toxicity of these heavy metal compounds is measured by systemic toxicants in order to determine the degree of impact on the victim and to assess possible damage to organ systems.

According to S.C. Alvarez et al., heavy metal compounds do not undergo metabolic changes in body tissues, which leads to their direct accumulation in the course of chronic exposure, that is, bioaccumulation as a result of transdermal or parenteral intake into the body due to soil contamination [21]. As posited by L. 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 can result in the inhibition of vital enzymes present within the bloodstream, thus inducing a state of functional incapacitation that can ultimately culminate in death. Consequently, even negligible quantities of these substances can result in substantial physiological consequences. In addition to intensive bioaccumulation, heavy metals are characterised by the absence of biodegradation, that is, they can not be destroyed, neutralised or removed from the body in full [23]. The bioaccumulation of heavy metals in the tissues of the victim is exacerbated in cases of ingestion of animal products that have been exposed to and accumulated heavy metals in contaminated soil. Consequently, in such a scenario, the concentration of metals in foodstuffs may increase exponentially, reaching levels 100,000 times higher than those found in soil. This has been linked to the development of various pathologies, including those affecting the nervous system, as well as carcinogenesis [24].

### CADMIUM

Cadmium is utilised in various types of household batteries, plastic products, industrial pigments, and metal structures. It is also widely employed in electroplating, as referenced by M. Wang et al. [25]. Furthermore, 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 group of carcinogens of the first degree [26]. Fertilizers represent the primary source of soil contamination with cadmium, which is introduced into the composition of plants consumed by humans. It has been established that an additional source of cadmium in the soil is combustion products, with large forest fires being a significant contributor to this phenomenon. The content of cadmium in wood ash varies from 2 to 32 mg per kg, and in straw ash it is more than 9 mg per kg [27]. Given the predominance of alkaline properties in the ash, the cadmium present in its composition



is insoluble in water and does not effectively penetrate plant tissue. However, according to the studies of M. Rizwan et al., cadmium has been found to accumulate in the soil, and in the case of fermentation, it becomes available for absorption by plants [28]. It has been demonstrated that cadmium can gradually accumulate within the human body. Furthermore, cadmium, in conjunction with zinc, has been observed to permeate into seawater through a network of surface and ground soils. Despite the existence of reports concerning the reduction of cadmium emissions into the soil in various industrialised countries by J. Wu et al., the issue remains a source of concern, particularly with regard to its carcinogenic effects on workers in agriculture and metallurgy, as well as on individuals residing in areas with soil contaminated with cadmium [29].

For a considerable period, the impact of cadmium on the human body was confined to research investigating its accumulation in the nephrons of the kidneys, specifically in the epithelial cells of the proximal tubules and in the bone tissue, which was characterised by pathological alterations in the homeostasis of the mineralisation of lamellar bone tissue [30]. Recent scientific studies conducted on large groups of patients have demonstrated statistically significant effects of cadmium on nervous tissue in both central and peripheral nervous system structures. This conclusion is supported by the research of R. Zhou et al. [31]. Firstly, these are studies with an evidence-based process of cadmium accumulation in brain tissues, which is also associated with a violation of the blood-brain barrier (BBB) during the bioaccumulation of cadmium compounds in soft tissues. The pathology of the BBB during chronic accumulation of cadmium has been shown to be pathophysiologically associated with a violation of the oxidative-antioxidant homeostasis of the capillary system of the CNS, which leads to the development of oxidative stress [32]. Conversely, acute cadmium intoxication has been observed to exhibit its maximum concentrations in structures of the nervous system not encircled by the BBB, namely the epiphysis and meninges, as previously documented by J.J.V. Branca et al. [33]. The degree of BBB resistance to the penetration of cadmium and its compounds is also contingent on the patient's medical history, namely their age, comorbidities, and alcohol and smoking abuse [34].

According to current statistics, the concentration of cadmium in the blood and urine of residents of developed countries with a developed industrial infrastructure varies from 0.005 to 7.01  $\mu\text{g/L}$  and from 0.04 to 14  $\mu\text{g/g}$ , respectively [35]. The neurotoxic effects of cadmium involve the initiation of tissue oxidative stress (with a predominance of oxidants), affecting the activity of enzymes critical for neuronal function and intercellular homeostasis in the brain. This impacts the cell cycle and the programmed death of neurons and neuroglia [36, 37]. According to J.J. Branca et al. [38], cadmium in nervous tissue acts as an initiator of 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 increased expression of apoptotic factors. Recent studies by Y. Ge et al. demonstrate that cadmium can disrupt the formation of the neuronal cytoskeleton by inhibiting the expression of proteins responsible for assembling and organising cytoplasmic neurofilaments. These neurofilaments are marker organelles specific to neurons [39].

During cadmium-induced oxidative stress, elevated levels of malondialdehyde, nitric oxide and oxidised glutathione are detected. Another mechanism of cadmium's neurotoxic effects is explained by the impact of its compounds on the activity of calcium adenosine triphosphatase and calcium-magnesium adenosine triphosphatase [40]. The consequences of this deactivation are displayed by a decrease in the level of calcium ions, which play a role in synaptic communication in all types of synapse in the central nervous system (CNS). Thus, cadmium's effect on nervous tissue is manifested through various mechanisms that disrupt the cell cycle and intracellular metabolism, resulting in premature degenerative processes.

## LEAD

Diseases associated with lead accumulation are called saturnism. Lead is a highly toxic heavy metal with cumulative properties that mainly affect the human nervous system [41]. The metal content of fruits and vegetables contaminated with lead can increase by more than tenfold compared to the natural level in uncontaminated soil. Lead is present in small amounts in almost all plant crops, but its concentration is especially high when these crops are grown in lead-contaminated soil. Scientific data shows that high concentrations of lead in cereal grains, legumes and other food products are highly toxic to humans and negatively impact the yield of field crops. Lead itself worsens the physicochemical parameters and organisation of the soil's microbial environment [42]. The gastrointestinal and nervous systems are the primary target organs for exposure to lead in the home [43]. In terms of its effect on the central nervous system, lead exhibits pronounced neurotoxicity, which is manifested by impaired neurophysiological function and presents symptomatically as mental disorders and neurocognitive syndromes [42].

It has been proven that children's bodies are more susceptible to the neurotoxic effects of lead and its compounds than adults'. A study by V. I. Naranjo et al. on pediatric patients showed that children are still exposed to lead despite widespread community and health 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 administered to prevent negative consequences for the central nervous system (CNS). The N-methyl-D-aspartate receptor plays a role in the maturation of brain neurons and their functional plasticity, 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 blood-brain barrier (BBB), inhibiting the activity of endotheliocytes in the BBB system. Histocytological effects of lead disrupt normal nervous system development processes in both the prenatal and childhood periods. These disorders include disruption to signalling, growth and differentiation factors during the proliferation and differentiation of CNS multipolar neurons; impaired formation of synaptic connections due to reduced neurocyte production of sialic acid; and violations to the chronological sequence of glial cell differentiation. The pharmacological effects of lead poisoning are manifested by lead replacing calcium and initiating disturbances in calmodulin cascades [45]. Lead also blocks the secretion of neurotransmitters from the presynaptic membrane into the synaptic cleft, thereby disrupting the GABAergic, dopaminergic and cholinergic systems of the central nervous system (CNS). Inside the cytoplasm, lead blocks the release of calcium ions from both the cell's cytoplasmic contents and the mitochondria themselves. This leads to the accumulation of reactive oxygen species, the activation of mitochondrial lysis, and the initiation of programmed processes of apoptosis or necrosis [45].

## MERCURY

Mercury is present in almost all human foods, ranging from 1 to 50 micrograms per kilogram of body weight. Levels may be higher in marine products [46]. This metal is found in soil and water contaminated with heavy metals and can be converted into methylmercury by microorganisms. Methylmercury and mercuric chloride are highly carcinogenic factors, as evidenced by H. 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 mass entering the body settles in brain tissues, with smaller amounts settling in hepatocytes in the liver and epithelial cells in the nephrons of the kidneys. According to L. Yang et al., the classic symptoms of organic mercury poisoning include depressive disorder, headaches, limb tremors, memory problems, gastrointestinal disorders (e.g. diarrhea and nausea), skin rashes, weakness and high blood pressure. Anthropogenic activities directly or indirectly pollute the soil with three types of mercury: elemental, inorganic, and organic [48].

Methylmercury is extremely toxic to most bodily tissues and can easily penetrate three-dimensional cell membranes. This has been proven by many studies, including those by L.C. Abbott and F. Nigussie [49]. Biochemical manifestations of mercury toxicity include blocking sulfhydryl-containing enzymes of cellular metabolism, increased circulation of reactive oxygen species, oxidative stress and disruption to the intracellular functioning of calcium ions. The latter is similar to the action of lead inside the cells of target organs. As intracellular calcium ions perform many important functions in both synaptic transmission and neuronal function, disturbances to intracellular calcium levels are the main mechanism that explains mercury neurotoxicity. These changes include the inhibition of the ability of cells to use calcium from intracellular stores, violations of the physical properties of calcium penetration through specific transmembrane channels in the plasma membrane, and changes in protein phosphorylation processes. Oxidative stress due to mercury poisoning can affect the viability of nervous system cells directly or indirectly by disturbing intracellular calcium homeostasis.

Methylmercury has a high degree of similarity to thiol groups in body cells. Consequently, intoxication during the prenatal differentiation of neuroblasts and late neurulation leads to the aberrant migration of stem cells and the disorganisation of the developing brain's neocortex. According to scientific theories published by S. Yawei et al. [50] and S. T. Zulaikhah et al. [51] but not yet studied in humans, methylmercury disrupts genetic sequences that control the normal neurulation process in the first trimester of pregnancy. This alters cellular signalling factors for neuroblastic migration, leading to dysplasia and abnormal cortical and myeloarchitectonics. Among these signalling pathways, the Notch receptor stands out as being sensitive to the effects of mercury even at threshold concentrations, as proven in experimental animals [52]. J.G. Dórea's work shows that the neurotoxicity of methylmercury is associated with the

inhibition of cytoplasmic microtubule polymerisation, which in turn blocks cell migration and the cell cycle with division, since formation of the mitotic spindle for cell division is impossible [53].

## CHROMIUM

As M. Pavesi and J.C. Moreira point out [54], chromium occurs in the biosphere in various oxidation states, but 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 oxidisers, household fertilisers, chromium steel and the drilling of oil wells. The effect of chromium on the human body depends on the dose size, exposure route and duration of contact. Chromium compounds can act directly at the site of contact — 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 carcinogenesis and nervous system pathologies due to its neurotoxicity [55]. Some studies have shown that industrial, chronic exposure to chromium can lead to impaired olfactory function, an increased risk of motor neuron disease in cases of complex heavy metal poisoning, and the development of schizophrenia, particularly in individuals with a psychiatric history, as discussed by J. Ma et al. [56]. The authors point out that high levels of chromium in the initial stages of schizophrenia may exacerbate serotonin synthesis, thereby contributing to the disease burden [57].

According to the work of T. Pavesi and J.C. Moreira [54], trivalent chromium circulates in soil organic matter and in the form of oxides, hydroxides and sulphates. Studies of chromium workers, a social group at high risk of complications due to chromium exposure, indicate that they experience regular headaches, systemic dizziness and weakness. However, no information was found on 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 the metal in the form of potassium dichromate. These complications include cerebral oedema and necrotic lesions, which can result in death. Thus, contaminated soil poses a number of threats to human health based on the spectrum of heavy metals which exceed threshold norms. Given the wide range of ways in which heavy metals can enter the human body from contaminated soil and the increasing number of industrial and agricultural sources of soil contamination with metals, this problem remains relevant for the healthcare systems of developed countries worldwide. Table 1 summarises the main features of the heavy metals discussed above for the purpose of basic diagnostic differentiation of their effects on the human nervous system.

Despite some common features in the pathophysiological effects of heavy metals on the human nervous system — such as the development of oxidative stress in the haemocirculatory system and neuroglia, the disruption of calcium-dependent mechanisms in intracellular metabolism and the blocking of synaptic transmission in the multipolar central nervous system (CNS) — there are also specific features relating to a particular type of heavy metal, given its valency and the compounds it forms. Further research on this issue should include studying specific regions with contaminated soil, identifying patient groups vulnerable to metal intoxication and diagnosing background conditions that can lead to a complicated course of events.

## CONCLUSIONS

Detailed studies of the chain of influence and the pathogenetic cascades involved in the development of nervous system pathologies are important for developing appropriate preventive and protective measures against their chronic effects. Early exposure to toxic metal compounds can have negative neurological consequences for fetal and child development. The pathophysiological effects of heavy metals on cells and neuroglia in the human nervous system share certain properties. These include the development of oxidative stress in the haemocirculatory system and neurocytes, the inhibition of calcium-dependent mechanisms in intracellular metabolism, and the disruption of synaptic transmission in multipolar neuron systems. However, each has specific features that facilitate a differentiated diagnosis. Primary prevention and the identification of high-risk social groups are reasonable and cost-effective measures to combat the impact of soil contaminated with heavy metals on critical organs and structures of the nervous system from a public health perspective.

## ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

**Вклад авторов.** Г.А. Батырова — обзор литературы, сбор и анализ литературных источников, написание текста и редактирование статьи; Г.А. Умарова — обзор литературы, сбор и анализ литературных источников, подготовка и написание текста статьи; С.Т. Уразаева — сбор и анализ литературных источников, подготовка и написание текста статьи; А. Исалдинова — обзор литературы, сбор и анализ литературных источников, написание текста и редактирование статьи;

У. Сарсембин, Г.Е. Таскожина, Ж.Х. Исангузина, Е.А. Умаров — обзор литературы, сбор и анализ литературных источников, написание текста и редактирование статьи. Все авторы подтверждают соответствие своего авторства международным критериям ICMJE (все авторы внесли существенный вклад в разработку концепции, проведения исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией).

**Этическая экспертиза.** Неприменимо.

**Источники финансирования.** Данное исследование финансирует Комитет науки Министерства науки и высшего образования Республики Казахстан (ИРН № AP26199833).

**Раскрытие интересов.** Авторы заявляют об отсутствии отношений, деятельности и интересов за последние три года, связанных с третьими лицами (коммерческими и некоммерческими), интересы которых могут быть затронуты содержанием статьи.

**Оригинальность.** При создании настоящей работы авторы не использовали ранее опубликованные сведения (текст, иллюстрации, данные).

**Доступ к данным.** Редакционная политика в отношении совместного использования данных к настоящей работе не применима, новые данные не собирали и не создавали.

**Генеративный искусственный интеллект.** При создании настоящей статьи технологии генеративного искусственного интеллекта не использовали.

**Рассмотрение и рецензирование.** Настоящая работа подана в журнал в инициативном порядке и рассмотрена по обычной процедуре. В рецензировании участвовали два внешних рецензента, член редакционной коллегии и научный редактор издания.

## ADDITIONAL INFORMATION

**Author contributions:** G. Batyrova: literature review, collection and analysis of literary sources, writing the text and editing the article; G. Umarova: literature review, collection and analysis of literary sources, preparation and writing of the text of the article; S. Urazayeva: collection and analysis of literary sources, preparation and writing of the text of the article; A. Issaldinova: literature review, collection and analysis of literary sources, writing the text and editing the article; U. Sarsembin, G. Taskozhina, Zh. Issanguzhina, Ye. Umarov: literature review, collection and analysis of literary sources, writing the text and editing the article. All authors confirm that their authorship meets the ICMJE criteria (all authors made substantial contributions to the conceptualization, investigation, and manuscript preparation, and reviewed and approved the final version prior to publication).

**Ethics approval:** Not applicable.

**Funding sources:** This research was funded by the Science Committee of the Ministry of Science and Higher Education of the Republic of Kazakhstan (Grant No. AP26199833).

**Disclosure of interests:** The authors have no relationships, activities, or interests for the last three years related to for-profit or not-for-profit third parties whose interests may be affected by the content of the article.

**Statement of originality:** No previously published material (text, images, or data) was used in this work.

**Data availability statement:** The editorial policy regarding data sharing does not apply to this work, as no new data was collected or created.

**Generative AI:** No generative artificial intelligence technologies were used to prepare this article.

**Provenance and peer review:** This paper was submitted unsolicited and reviewed following the standard procedure. The peer review process involved two external reviewers, a member of the editorial board, and the in-house scientific editor.

## СПИСОК ЛИТЕРАТУРЫ | REFERENCES

1. Okereafor U, Makhatha M, Mekuto L, et al. Toxic metal implications on agricultural soils, plants, animals, aquatic life and human health. *Int J Environ Res Public Health*. 2020;17(7):2204. doi: 10.3390/ijerph17072204
2. Bhat SA, Hassan T, Majid S. Heavy metal toxicity and their harmful effects on living organisms — a review. *International Journal of Medical Science and Diagnosis Research*. 2019;3(1):106–122.
3. Mitra S, Chakraborty AJ, Tareq AM, et al. Impact of heavy metals on the environment and human health: Novel therapeutic insights to counter the toxicity. *Journal of King Saud University-Science*. 2022;34(3):101865. doi: 10.1016/j.jksus.2022.101865



4. Shen X, Dai M, Yang J, et al. A critical review on the phytoremediation of heavy metals from environment: performance and challenges. *Chemosphere*. 2022;291(Pt 3):132979. doi: 10.1016/j.chemosphere.2021.132979
5. Briffa J, Sinagra E, Blundell R. Heavy metal pollution in the environment and their toxicological effects on humans. *Heliyon*. 2020;6(9):e04691. doi: 10.1016/j.heliyon.2020.e04691
6. Sall ML, Diaw AKD, Gningue-Sall D, et al. Toxic heavy metals: impact on the environment and human health, and treatment with conducting organic polymers, a review. *Environ Sci Pollut Res Int*. 2020;27(24):29927–29942. doi: 10.1007/s11356-020-09354-3
7. Rahman Z, Singh VP. Bioremediation of toxic heavy metals (THMs) contaminated sites: concepts, applications and challenges. *Environ Sci Pollut Res Int*. 2020;27(22):27563–27581. doi: 10.1007/s11356-020-08903-0
8. Zaynab M, Al-Yahyai R, Ameen A, et al. Health and environmental effects of heavy metals. *Journal of King Saud University-Science*. 2021;34(1):101653. doi: 10.1016/j.jksus.2021.101653
9. Alengebaway A, Abdelkhalek ST, Qureshi SR, Wang MQ. Heavy metals and pesticides toxicity in agricultural soil and plants: ecological risks and human health implications. *Toxics*. 2021;9(3):42. doi: 10.3390/toxics9030042
10. Ahmad W, Alharthy RD, Zubair M, et al. Toxic and heavy metals contamination assessment in soil and water to evaluate human health risk. *Sci Rep*. 2021;11(1):17006. doi: 10.1038/s41598-021-94616-4
11. Ijomone OM, Ifenatuoha CW, Aluko OM, et al. The aging brain: impact of heavy metal neurotoxicity. *Crit Rev Toxicol*. 2020;50(9):801–814. doi: 10.1080/10408444.2020.1838441
12. Rehman Q, Rehman K, Akash MSH. Heavy metals and neurological disorders: from exposure to preventive interventions. In: MSH Akash, K Rehman, editors. *Environmental contaminants and neurological disorders. Emerging contaminants and associated treatment technologies*. Springer, Cham; 2021. doi: 10.1007/978-3-030-66376-6\_4
13. Mehta I, Verma M, Quasmi MN, et al. Emerging roles of histone modifications in environmental toxicants-induced neurotoxicity. *Toxicology*. 2025;515:154164. doi: 10.1016/j.tox.2025.154164
14. Kumar P. Heavy metal contamination causes protein misfolding, leading to neurodegenerative disorders. In: *Protein Misfolding in Neurodegenerative Diseases*. Academic Press; 2025. P. 463–492. ISBN: 978-0443187162
15. Ijomone OK, Ukwubile II, Aneke VO, et al. Glial perturbation in metal neurotoxicity: implications for brain disorders. *Neuroglia*. 2025;6(1):4. doi: 10.3390/neuroglia6010004
16. Althomali RH, Abbood MA, Saleh EAM, et al. Exposure to heavy metals and neurocognitive function in adults: a systematic review. *Environ Sci Eur*. 2024;36(1):18. doi: 10.1186/s12302-024-00843-7
17. Chen J, Chen J, Li M, et al. Probabilistic assessment of the cumulative risk from dietary heavy metal exposure in Chongqing, China using a hazard-driven approach. *Sci Rep*. 2025;15(1):2229. doi: 10.1038/s41598-024-83299-2
18. Fu Z, Xi S. The effects of heavy metals on human metabolism. *Toxicol Mech Methods*. 2020;30(3):167–176. doi: 10.1080/15376516.2019.1701594
19. Chen S, Zhao R, Sun X, et al. Toxicity and biocompatibility of liquid metals. *Adv Healthc Mater*. 2023;12(3):e2201924. doi: 10.1002/adhm.202201924
20. Prasad S, Yadav KK, Kumar S, et al. Chromium contamination and effect on environmental health and its remediation: a sustainable approaches. *J Environ Manage*. 2021;285:112174. doi: 10.1016/j.jenvman.2021.112174
21. Alvarez CC, Bravo Gómez ME, Hernández Zavala A. Hexavalent chromium: regulation and health effects. *J Trace Elem Med Biol*. 2021;65:126729. doi: 10.1016/j.jtemb.2021.126729
22. Chen L, Zhou M, Wang J, et al. A global meta-analysis of heavy metal(loid)s pollution in soils near copper mines: evaluation of pollution level and probabilistic health risks. *Sci Total Environ*. 2022;835:155441. doi: 10.1016/j.scitotenv.2022.155441
23. Long Z, Huang Y, Zhang W, et al. Effect of different industrial activities on soil heavy metal pollution, ecological risk, and health risk. *Environ Monit Assess*. 2021;193(1):20. doi: 10.1007/s10661-020-08807-z
24. Adimalla N, Chen J, Qian H. Spatial characteristics of heavy metal contamination and potential human health risk assessment of urban soils: a case study from an urban region of South India. *Ecotoxicol Environ Saf*. 2020;194:110406. doi: 10.1016/j.ecoenv.2020.110406
25. Wang M, Chen Z, Song W, et al. A review on cadmium exposure in the population and intervention strategies against cadmium toxicity. *Bull Environ Contam Toxicol*. 2021;106(1):65–74. doi: 10.1007/s00128-020-03088-1

26. WHO (2019). *Preventing disease through healthy environments: exposure to cadmium: a major public health concern*. World Health Organization; 2019. 6 p. URL: <https://iris.who.int/bitstream/handle/10665/329480/WHO-CED-PHE-EPE-19.4.3-eng.pdf>
27. Suhani I, Sahab S, Srivastava V, Singh RP. Impact of cadmium pollution on food safety and human health. *Current Opinion in Toxicology*. 2021;27:1–7. doi: 10.1016/j.cotox.2021.04.004
28. Rizwan M, Ali S, Rehman MZU, Maqbool A. A critical review on the effects of zinc at toxic levels of cadmium in plants. *Environ Sci Pollut Res Int*. 2019;26(7):6279–6289. doi: 10.1007/s11356-019-04174-6
29. Wu J, Mock HP, Giehl RFH, et al. Silicon decreases cadmium concentrations by modulating root endodermal suberin development in wheat plants. *J Hazard Mater*. 2019;364:581–590. doi: 10.1016/j.jhazmat.2018.10.052
30. Andjelkovic M, Buha Djordjevic A, Antonijevic E, et al. Toxic effect of acute cadmium and lead exposure in rat blood, liver, and kidney. *Int J Environ Res Public Health*. 2019;16(2):274. doi: 10.3390/ijerph16020274
31. Zhou R, Zhao J, Li D, et al. Combined exposure of lead and cadmium leads to the aggravated neurotoxicity through regulating the expression of histone deacetylase 2. *Chemosphere*. 2020;252:126589. doi: 10.1016/j.chemosphere.2020.126589
32. Bi SS, Talukder M, Sun XT, et al. Cerebellar injury induced by cadmium via disrupting the heat-shock response. *Environ Sci Pollut Res Int*. 2023;30(9):22550–22559. doi: 10.1007/s11356-022-23771-6
33. Branca JJV, Fiorillo C, Carrino D, et al. Cadmium-induced oxidative stress: focus on the central nervous system. *Antioxidants* (Basel). 2020;9(6):492. doi: 10.3390/antiox9060492
34. Chandravanshi L, Shiv K, Kumar S. Developmental toxicity of cadmium in infants and children: a review. *Environ Anal Health Toxicol*. 2021;36(1):e2021003-0. doi: 10.5620/eaht.2021003
35. Ruczaj A, Brzóska MM. Environmental exposure of the general population to cadmium as a risk factor of the damage to the nervous system: a critical review of current data. *J Appl Toxicol*. 2023;43(1):66–88. doi: 10.1002/jat.4322
36. Mubeena Mariyath PM, Shahi MH, Tayyab M, et al. Cadmium-induced neurodegeneration and activation of noncanonical sonic hedgehog pathway in rat cerebellum. *J Biochem Mol Toxicol*. 2019;33(4):e22274. doi: 10.1002/jbt.22274
37. Chouit Z, Djellal D, Haddad S, et al. Potentiation of the apoptotic signaling pathway in both the striatum and hippocampus and neurobehavioral impairment in rats exposed chronically to a low-dose of cadmium. *Environ Sci Pollut Res Int*. 2021;28(3):3307–3317. doi: 10.1007/s11356-020-10755-7
38. Branca JJV, Maresca M, Morucci G, et al. Effects of cadmium on ZO-1 tight junction integrity of the blood brain barrier. *Int J Mol Sci*. 2019;20(23):6010. doi: 10.3390/ijms20236010
39. Ge Y, Song X, Chen L, et al. Cadmium induces actin cytoskeleton alterations and dysfunction in Neuro-2a cells. *Environ Toxicol*. 2019;34(4):469–475. doi: 10.1002/tox.22700
40. Polykretis P, Cencetti F, Donati C, et al. Cadmium effects on superoxide dismutase 1 in human cells revealed by NMR. *Redox Biol*. 2019;21:101102. doi: 10.1016/j.redox.2019.101102
41. Kumar A, Kumar A, M M S CP, et al. Lead toxicity: health hazards, influence on food chain, and sustainable remediation approaches. *Int J Environ Res Public Health*. 2020;17(7):2179. doi: 10.3390/ijerph17072179
42. Chandrasekhar C, Ray JG. Lead accumulation, growth responses and biochemical changes of three plant species exposed to soil amended with different concentrations of lead nitrate. *Ecotoxicol Environ Saf*. 2019;171:26–36. doi: 10.1016/j.ecoenv.2018.12.058
43. Apte A, Bradford K, Dente C, Smith RN. Lead toxicity from retained bullet fragments: a systematic review and meta-analysis. *J Trauma Acute Care Surg*. 2019;87(3):707–716. doi: 10.1097/TA.0000000000002287
44. Naranjo VI, Hendricks M, Jones KS. Lead toxicity in children: an unrelenting public health problem. *Pediatr Neurol*. 2020;113:51–55. doi: 10.1016/j.pediatrneurol.2020.08.005
45. Sawicki K, Czajka M, Matysiak-Kucharek M, et al. Toxicity of metallic nanoparticles in the central nervous system. *Nanotechnology Reviews*. 2019;8(1):175–200. doi: 10.1515/ntrev-2019-0017
46. Pacyna JM. Recent advances in mercury research. *Sci Total Environ*. 2020;738:139955. doi: 10.1016/j.scitotenv.2020.139955
47. Kim H, Lee J, Woo HD, et al. Dietary mercury intake and colorectal cancer risk: a case-control study. *Clin Nutr*. 2020;39(7):2106–2113. doi: 10.1016/j.clnu.2019.08.025

48. Yang L, Zhang Y, Wang F, et al. Toxicity of mercury: Molecular evidence. *Chemosphere*. 2020;245:125586. doi: 10.1016/j.chemosphere.2019.125586
49. Abbott LC, Nigussie F. Mercury Toxicity and neurogenesis in the mammalian brain. *Int J Mol Sci*. 2021;22(14):7520. doi: 10.3390/ijms22147520
50. Yawei S, Jianhai L, Junxiu Z, et al. Epidemiology, clinical presentation, treatment, and follow-up of chronic mercury poisoning in China: a retrospective analysis. *BMC Pharmacol Toxicol*. 2021;22(1):25. doi: 10.1186/s40360-021-00493-y
51. Zulaikhah ST, Wahyuwibowo J, Pratama AA. Mercury and its effect on human health: a review of the literature. *Int J Public Health*. 2020;9(2):103–114. doi: 10.11591/ijphs.v9i2.20416
52. Du B, Yin R, Fu X, et al. Use of mercury isotopes to quantify sources of human inorganic mercury exposure and metabolic processes in the human body. *Environ Int*. 2021;147:106336. doi: 10.1016/j.envint.2020.106336
53. Dórea JG. Neurotoxic effects of combined exposures to aluminum and mercury in early life (infancy). *Environ Res*. 2020;188:109734. doi: 10.1016/j.envres.2020.109734
54. Pavesi T, Moreira JC. Mechanisms and individuality in chromium toxicity in humans. *J Appl Toxicol*. 2020;40(9):1183–1197. doi: 10.1002/jat.3965
55. Ukhurebor KE, Aigbe UO, Onyancha RB, et al. Effect of hexavalent chromium on the environment and removal techniques: a review. *J Environ Manage*. 2021;280:111809. doi: 10.1016/j.jenvman.2020.111809
56. Ma J, Yan L, Guo T, et al. Association of typical toxic heavy metals with schizophrenia. *Int J Environ Res Public Health*. 2019;16(21):4200. doi: 10.3390/ijerph16214200
57. Wise Jr JP, Young JL, Cai J, Cai L. Current understanding of hexavalent chromium [Cr(VI)] neurotoxicity and new perspectives. *Environ Int*. 2022;158:106877. doi: 10.1016/j.envint.2021.106877
58. Hossini H, Shafie B, Niri AD, et al. A comprehensive review on human health effects of chromium: insights on induced toxicity. *Environ Sci Pollut Res Int*. 2022;29(47):70686–70705. doi: 10.1007/s11356-022-22705-6

## ИНФОРМАЦИЯ ОБ АВТОРАХ / AUTHORS' INFO

* Автор, ответственный за переписку	* Corresponding author
* <b>Батырова Гульнара Арыстангалиевна</b> , PhD; адрес: Казахстан, 030019, Актөбе, ул. Маресьева, д. 68; ORCID: 0000-0001-7970-4059; eLibrary SPIN: 8584-5024; e-mail: g.batyrova@zkm.kz	* <b>Gulnara Batyrova</b> , PhD; address: 68 Maresyev st, Aktobe, Kazakhstan, 030019; ORCID: 0000-0001-7970-4059; eLibrary SPIN: 8584-5024; e-mail: g.batyrova@zkm.kz
<b>Умарова Гульмира Арыстангалиевна</b> , PhD; ORCID: 0000-0001-7637-113X; eLibrary SPIN: 9146-3959; e-mail: uga_80@mail.ru	<b>Gulmira Umarova</b> , PhD; ORCID: 0000-0001-7637-113X; eLibrary SPIN: 9146-3959; e-mail: uga_80@mail.ru
<b>Уразаева Салтанат Тураковна</b> , канд. мед. наук; ORCID: 0000-0002-4773-0807; e-mail: s.urazaeva@mail.ru	<b>Saltanat Urazayeva</b> , MD, Cand. Sci. (Medicine); ORCID: 0000-0002-4773-0807; e-mail: s.urazaeva@mail.ru
<b>Сарсембин Умбетали Куандыкович</b> , PhD; ORCID: 0000-0002-0796-3737; e-mail: umbetali_s.k@mail.ru	<b>Umbetali Sarsembin</b> , PhD; ORCID: 0000-0002-0796-3737; e-mail: umbetali_s.k@mail.ru
<b>Исалидинова Асель Нурлановна</b> , магистр образовательной программы; ORCID: 0000-0003-4843-5823; e-mail: aselisaldinova@gmail.com	<b>Assel Issaldinova</b> , Master of the program; ORCID: 0000-0003-4843-5823; e-mail: aselisaldinova@gmail.com
<b>Таскожина Гулайм Есенбаевна</b> , PhD докторант; ORCID: 0000-0003-3922-0054; e-mail: g.taskozhina@zkm.kz	<b>Gulaim Taskozhina</b> , PhD, Student; ORCID: 0000-0003-3922-0054; e-mail: g.taskozhina@zkm.kz
<b>Исангужина Жамиля Халимовна</b> , канд.	<b>Zhamilia Issanguzhina</b> , MD, Cand. Sci.

мед. наук; ORCID: 0000-0002-7557-8486; e-mail: gamilia0452@gmail.com	(Medicine); ORCID: 0000-0002-7557-8486; e-mail: gamilia0452@gmail.com
<b>Умаров Ескендир Арыстангалиевич</b> , магистр естественных наук; ORCID: 0000-0002-5661-4023; e-mail: eskendir.um@gmail.com	<b>Yeskendir Umarov</b> , Master of Natural Sciences; ORCID: 0000-0002-5661-4023; e-mail: eskendir.um@gmail.com

## ТАБЛИЦЫ

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

Characteristics of manifestation	Cadmium	Lead	Mercury	Chromium
Influence on the development of the nervous system prenatally	Possible	Proven	Proven	Possible
Impact on the children's body	Possible	Proven	Proven	Possible
Breach of the blood-brain barrier	Present	Present	Present	Not typical
Psychiatric disorders in metal poisoning	Present	Present	Not typical	Not typical
Disruption of synaptic transmission	Not typical	Characteristically	Characteristically	Characteristically
Neurological symptoms	Present	Present	Present	Present

Source: compiled by the authors.



## РИСУНКИ

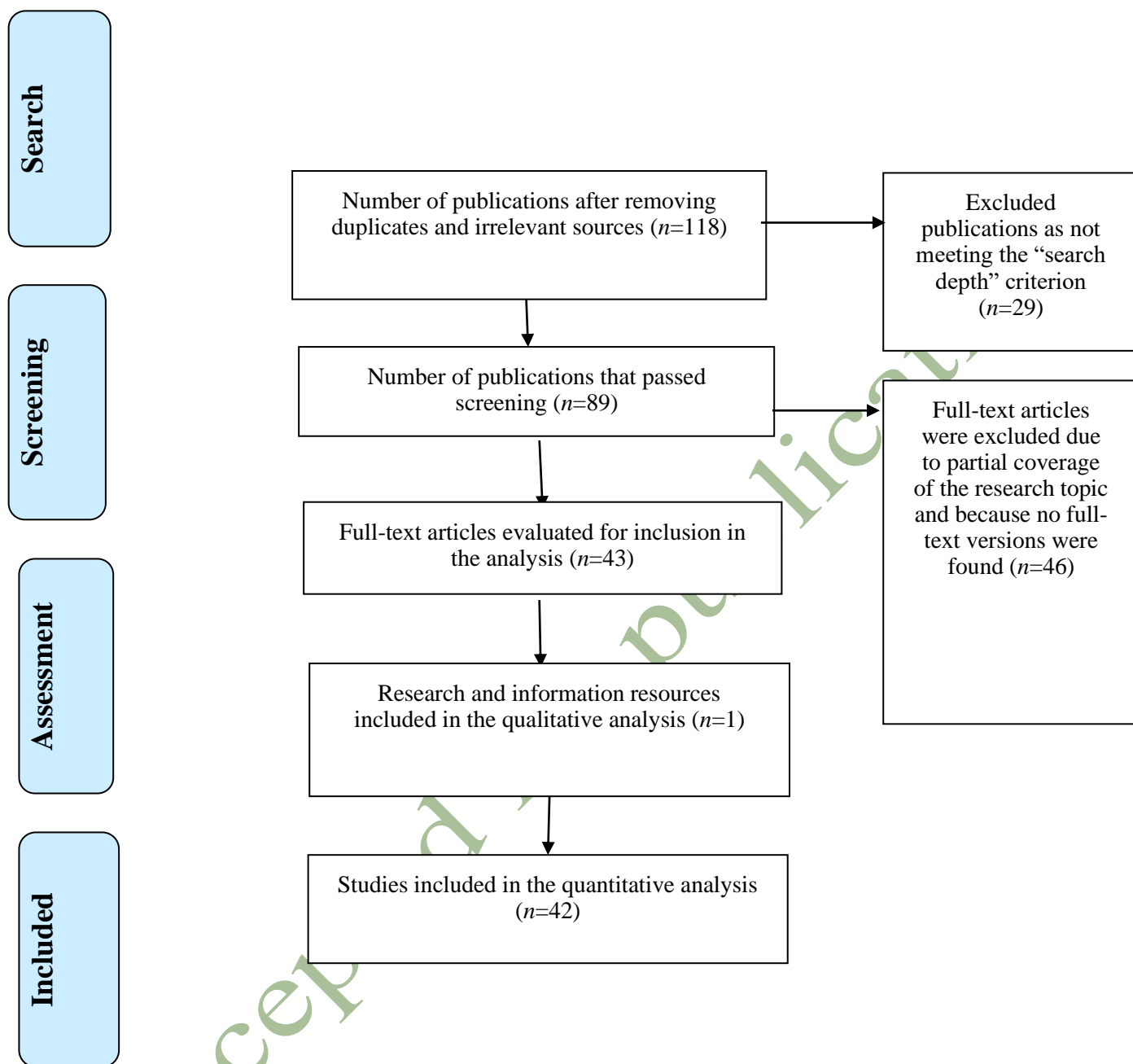


Fig. 1. Strategy for searching and selecting sources.