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HEALTH RISKS TO THE POPULATION ASSOCIATED WITH POISONING BY NEUROTROPIC TOXICANTS (ANALYTICAL REVIEW)

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Introduction. Issues related to the epidemiologic aspects and clinical manifestations of poisoning by neurotoxicants, whose effects cause serious harm to the health of victims, are highly relevant. Acute and chronic poisoning can be manifested through diverse patterns; however, regardless of the type of neurotoxicant, all victims experience asthenovegetative and psychoorganic syndromes. These syndromes can develop during both toxicogenic and somatogenic phases of poisoning, manifesting in the period of its long-term consequences.
Objective. Scientific substantiation of the risks of poisoning by neurotoxicants, which pose a serious threat to public health due to their systemic toxic effects and the development of multiorgan pathology, including at the stage of long-term consequences of poisoning.
Discussion. In Russia, among various types of acute poisoning, intoxications by substances causing primary damage to the central nervous system rank first. The routes of entry of neurotoxicants into the body are indicated, and the forms of manifestation of neurotoxic processes are described. The pathogenesis of the toxic action of organic solvents, heavy metal salts, barbiturates, and carbamates is analyzed. Toxic neurotropic substances can adversely affect the nervous system both directly and indirectly, through damage to other organs and systems. Clinical cases of acute poisoning by neurotoxicants are described. After poisoning by representatives of the group of neurotropic toxicants, after a certain period of time, the victim develops long-term consequences with a highly varying clinical picture.
Conclusions. The presented data on poisoning exposures to neurotoxicants demonstrate the clinical and pathogenetic significance of their effects not only on the central nervous system but also on other organs and tissues, the development of systemic pathological processes and multiorgan pathologies. The identified features of the toxic action must be taken into account when analyzing the health risks to victims of poisoning with neurotropic toxicants. The most significant manifestations of the effects on other organs/tissues should be reflected in the protocols for diagnosing the severity of such poisoning accidents and their long-term consequences, as well as in the use of metabolic and cytoprotective agents for their treatment.

Keywords: neurotoxicity; neurotoxicant; poisoning; methanol; barbiturates; heavy metals; lead; carbamates; 1,4-butanediol

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РИСКИ ДЛЯ ЗДОРОВЬЯ НАСЕЛЕНИЯ, СВЯЗАННЫЕ С ОТРАВЛЕНИЯМИ НЕЙРОТРОПНЫМИ ТОКСИКАНТАМИ (АНАЛИТИЧЕСКИЙ ОБЗОР)

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Введение. Вопросы эпидемиологии и особенностей клинической картины отравлений нейротропными токсикантами, действие которых наносит серьезный вред здоровью пострадавших, являются актуальными и приоритетными. Картина острых и хронических отравлений может быть весьма разнообразна, однако есть ведущие синдромы, такие как астеновегетативный и психоорганический, которые наблюдаются у пострадавших независимо от вида нейротоксиканта. Данные синдромы могут развиваться как в токсикогенную фазу отравления, так и в соматогенную, проявляясь в периоде отдаленных последствий отравлений.
Цель. Научное обоснование рисков отравлений нейротоксикантами, представляющими серьезную угрозу для здоровья населения, связанную с их системным токсическим действием и формированием полиорганной патологии, в том числе на этапе отдаленных последствий отравлений.
Обсуждение. Отмечено, что в картине острых отравлений в Российской Федерации первое место занимают интоксикации веществами, вызывающими первичное поражение ЦНС. Показаны пути поступления нейротоксикантов в организм, описаны формы проявления нейротоксических процессов и патогенез токсического действия органических растворителей, солей тяжелых металлов, барбитуратов и карбаматов. Установлено, что токсичные нейротропные вещества могут оказывать негативное воздействие

на нервную систему как напрямую, так и опосредованно, через повреждение других органов и систем. Представлены описания клинических случаев острых отравлений нейротоксикантами. Выявлено, что после отравления представителями группы нейротропных токсикантов через определенный период времени у пострадавшего развиваются отдаленные последствия и клиническая картина их может быть весьма разнообразна.
Выводы. Представленные сведения по отравлениям нейротоксикантами показывают клиническую и патогенетическую значимость их действия не только на ЦНС, но и на другие органы и ткани, развитие системных патологических процессов и полиорганной патологии. Выявленные особенности токсического действия необходимо учитывать при анализе рисков здоровью пострадавших от отравления нейротропными токсикантами, а наиболее значимые проявления действия на другие органы/ткани должны найти отражение в протоколах диагностики степени тяжести таких отравлений и их отдаленных последствий, а также в применении средств метаболического и цитопротекторного действия для их лечения.

Ключевые слова: нейротоксичность; нейротоксикант; отравление; метанол; барбитураты; тяжелые металлы; свинец; карбаматы; 1,4-бутандиол

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INTRODUCTION

The rapid development of chemical science has been accompanied by uncovering fundamental patterns of chemical processes, developing chemical compounds through targeted synthesis, and establishing large-scale chemical production, thereby integrating chemistry into virtually all spheres of human life, including industry, agriculture, military affairs, and everyday life. The scientific and technological progress has undoubtedly enhanced the standards of living; however, it has also brought about new risks leading to incidents and even technological disasters of various scales. Virtually all types of emergencies are related to the potential adverse effects of chemical agents on human life, health, and the environment. Unfortunately, the frequency of accidents at chemically hazardous facilities resulting in casualties continues to demonstrate a persistent trend [1].

In Russia, for a number of years, mortality from external causes has been ranking third after oncological and cardiovascular diseases. In the structure of mortality from external causes, every fourth case of death has been caused by acute poisoning of chemical etiology. Acute poisoning remains a serious medical and social problem, as evidenced by its regular occurrence and the resulting consequences, including high rates of fatalities and disability among the victims [2].

According to poisoning statistics in the Russian Federation for the 2022–2024 period, the number of food poisoning incidents decreased; however, the number of victims of all other monitored toxicants showed a growing trend (Table 1). In 2023, Solonin et al. conducted a retrospective observational study to evaluate the results of chemical and toxicological analysis in patients admitted to the Department of Acute Poisonings and Somatopsychiatric Disorders of the Sklifosovsky Institute for Emergency Medicine (SIEM) in 2019–2021. Thus, during 2019–2021, 9590 patients sought specialized toxicological care at the SIEM, which corresponded to approximately one case of poisoning with neurotropic toxicants per 1000 of the population [3].

In the structure of acute poisoning in the Russian Federation, intoxications by substances that primarily cause damage to the nervous system rank first. The share of poisoning cases by neurotropic toxicants was about 65%, with more than a third of cases classified as severe and extremely severe intoxications [4, 5].

Neurotoxicity is the ability of a toxic substance to affect the central nervous system (CNS), leading to the destruction of its structure and/or disruption of its function [6]. The toxic process can cause such disorders in the body as changes in energy metabolism, neuromuscular transmission problems, damage to cell membranes and synapses. Neurotoxicity can be direct, i.e., caused by the action of a toxic substance directly on the nervous system, or indirect, when toxicity arises due to damage to other organs and systems [7].

Neurotoxic processes can manifest as impairments in motor, sensory, and cognitive functions, as well as changes in the emotional state [8]. Depending on the conditions of exposure, the structure of the toxicant, and its neurotoxic potential, the developing processes can be acute or chronic [6].

Table 1. Distribution of poisoning cases of chemical etiology by main monitored groups in the Russian Federation for 2022–2024

Toxicants groups	Analyzed period (year)					
	2022		2023		2024	
	total (people)	of which fatal (people)	total (people)	of which fatal (people)	total (people)	of which fatal (people)
Alcohol-containing products	30,917	9228	32,540	10,013	30,321	9313
Narcotic substances	22,054	7077	25,188	7909	23,289	6546
Medicinal products	20,940	662	23,748	710	24,610	761
Food products	1549	27	1536	18	1174	15
Other monitored species	25579	5981	29671	6197	30259	6287
Total	101,039	22,975	112,683	24,847	109,653	22,922

Table prepared by the authors based on data from the internet source[1]

The list of substances that can cause a chronic neurotoxic process is quite extensive, with the most common being:

- toxicants present in the external environment (derivatives of mercury, lead, arsenic, etc.);
- pharmaceuticals (antidepressants, antipsychotics, antiepileptic drugs, cytostatics, antibiotics, synthetic antimicrobial agents, etc.);
- organic solvents (benzene, toluene, acetone, ethyl, methyl, propyl alcohols, etc.);
- industrial chemicals and toxic compounds (organophosphorus and organochlorine pesticides, carbamates, pyrethroids, defoliants and other agrochemical agents, carbon monoxide, etc.) [9].

As a result of poisoning exposure to neurotoxicants, persistent impairments in mental and physical activity, emotional state, cognitive processes, sensitivity, as well as focal neurological disorders may occur. The available literature shows that while the initial stages of pathogenesis and the clinical development of acute poisoning have been extensively studied, the long-term consequences of poisoning by neurotoxicants require elucidation. In addition, data on the statistics of delayed nervous system disorders are lacking. CNS lesions developing in the long-term period after acute intoxication are usually not associated with these poisoning incidents[2].

In this work, our aim is to scientifically substantiate the risks of poisoning by neurotoxicants, which pose a serious threat to public health due to their systemic toxic effects and the development of multiorgan pathologies, including at the stage of long-term consequences of poisoning.

MATERIALS AND METHODS

A literature search was conducted in electronic bibliographic databases, including the Russian Science Citation Index (RSCI), Scopus, Web of Science (WoS), and PubMed. Search queries included the following keywords: neurotoxicity, intoxications by neurotropic toxicants, severe poisonings, long-term consequences. The review covers publications with an information search depth of no more than five years, as well as literature sources that are considered fundamental works on the subject under discussion, regardless of their publication year.

RESULTS AND DISCUSSION

In order to fulfill the research objectives, we carry out a detailed examination of representative substances from each group that can trigger a chronic neurotoxic process.

[1] Information on acute poisonings of chemical etiology by constituent entities of the Russian Federation. Federal Center for Hygiene and Epidemiology of the Federal Service for Surveillance on Consumer Rights Protection and Human Wellbeing. https://fcgie.ru/sgm.html (access date: 10.04.2025).
[2] Methodological Recommendations (MR) FMBA of Russia 12.05-18. Clinical Presentation, Diagnosis, Prevention and Treatment of Central Nervous System Lesions Following Severe Poisonings with Neurotoxicants. Moscow; 2018.

Environmental pollutants (heavy metals)

The term "heavy metals" (HMs) characterizes a broad group of substances that enter the environment primarily as a result of human activity [10, 11]. Sources of HM pollution include industrial and agricultural production, chemical plants, waste incineration facilities, and boiler houses. Additionally, there exist non-anthropogenic sources of HM pollution, such as volcanic eruptions [12]. Accumulation of HMs occurs in polluted air, water, soil, and consumer goods (e.g., cosmetic products) [13].

There are two primary routes of human exposure to heavy metals: oral and inhalation. In oral exposure, the main source is plant-based food, which accounts for 40–80% of heavy metal intake; atmospheric air and water contribute 20–40% of HMs. The second route is inhalation exposure, although it is less common [14].

According to the decision of the UN Economic Commission for Europe[3], the group of the most hazardous (and thus prioritized for monitoring, control, and regulation) HMs includes mercury, lead, cadmium, chromium, manganese, nickel, cobalt, vanadium, copper, iron, zinc, and antimony (as well as arsenic and selenium) [15].

Poisoning with HM salts occurs through ingestion into the digestive system, inhalation of vapors, or exposure via mucous membranes and skin. Multiple systems and organs — the CNS, kidneys, intestines, liver, endocrine organs, heart, and blood vessels — are affected. HM salts are capable of accumulating in the body, circulating for extended periods and gradually releasing into the bloodstream from their depots, rendering the process chronic [16].

Overall, the mechanisms of HM toxicity have been studied in detail. The impact of HM salts on the body is determined by numerous factors, including the nature of the metal, the type of compound, and its concentration. Metal ions are part of coenzymes. One of the primary mechanisms of HM toxicity consists in their competition with essential metals for binding sites in proteins. Furthermore, many protein macromolecules contain free sulfhydryl groups that can interact with ions of toxic metals, such as cadmium, lead, mercury, etc. This interaction subsequently leads to the loss of protein function and the development of toxic effects [11].

Lead poisoning (Pb). According to estimates presented in the updated release of the WHO publication "The impact of chemicals on public health: known and unknown for 2021," nearly half of the two million deaths caused by chemical substances in 2019 were due to lead exposure. Globally, the long-term health consequences of lead exposure through various routes of entry (oral, inhalation, transcutaneous) result in the loss of 21.7 million years of life due to disability and death (disability-adjusted life years, DALYs). Lead, as a contributing factor to disease development, accounts for up to 30% of all idiopathic intellectual disabilities, 4.6% of cardiovascular diseases, and approximately 3% of chronic kidney diseases.[4]

Lead poisoning, in the setting of oxidative stress, results in leukocytosis, lymphocytosis, a decrease in total protein, albumin, and globulin levels [17]. The genotoxic effect of lead was demonstrated experimentally by administering lead acetate in water (Pb) intraperitoneally to laboratory animals in small doses over six weeks. By the end of the observation period, the animals had developed moderate subchronic intoxication with signs characteristic of lead effects (caused by impaired heme formation and increased synthesis of cytochrome P450 in the liver) [18].

It was found that approximately 80% of lead entering the body accumulates in bone tissue, while the remaining 20% is distributed in adipose tissue, kidneys, and liver, binding to sulfhydryl groups of proteins and forming toxic compounds [11, 19, 20]. The primary target organs in lead poisoning are the hematopoietic system, CNS, and the kidneys. Lead poisoning is accompanied by changes in antioxidant status, ionic mimicry as a mechanism of molecular lead toxicity, alterations in the structure and function of intracellular organelles in neuronal cells, induction of autophagy through the PI3K/AKT/mTOR signaling pathway, effects on the cellular receptor apparatus, changes in synaptic plasticity, and impacts on the cellular genetic apparatus [21]. In the brain, diffuse edema of gray and white matter can be observed, as well as dystrophic changes in cortical and ganglionic neurons and demyelination of white matter [12].

The angiotoxic effect of lead is manifested through activation of mitogen-activated protein kinase signaling pathways, which triggers a cascade of reactions for the synthesis of pro-inflammatory proteins, leading to increased vascular resistance and blood pressure [22].

Cadmium poisoning (Cd). According to the WHO, cadmium ranks fifth among the most hazardous chemical substances affecting the human body. This metal is typically present in the environment in small quantities. However, due to human activities, cadmium levels are

[3] Protocol on Heavy Metals. UNECE; 1998.
[4] World Health Organization. URL: https://www.who.int/ru/news-room/fact-sheets/detail/lead-poisoning-and-health (access date: 05.04.2025).

gradually increasing each year [23]. Both in Russia and globally, the primary area of cadmium consumption is the production of nickel-cadmium batteries. Other applications of cadmium include anti-corrosion coatings, pigments, polyvinyl chloride stabilizers, and semiconductors for solar cells [24].

Cadmium can enter the human body through inhalation, via cigarette smoke (cadmium accumulates in tobacco leaves) or the air with accumulated road dust particles, including that generated from tire and brake pad wear. Orally, cadmium is ingested via its accumulating foods, such as mushrooms, various plants, and meat (pork or beef) [25]. Additionally, cadmium can also enter food products through packaging (typical of canned goods, particularly those made of materials prohibited for contact with food) [26]. Cadmium is chemically similar to zinc, capable of its replacement in biochemical reactions, acting as a pseudo-inducer or pseudo-inhibitor of zinc-containing enzymes [27].

The pathogenesis of the toxic effects of HMs on the body exhibits uniform features characteristic of many types of damage: activation of lipid peroxidation (LPO) and damaging effects on intracellular proteins [28]. Since liver cells do not absorb the cadmium-protein complex, it is transported from the gastrointestinal tract directly to the kidneys [10]. The most pronounced inhibitory effect of Cd is on the antioxidant system, leading to oxidative cell damage [29].

Cadmium poisoning is accompanied by disruptions in protein synthesis and nucleic acid metabolism. Cadmium also possesses carcinogenic and mutagenic properties; thus, experiments have confirmed its teratogenic effect, which is associated with cell damage during early stages of organogenesis [30].

Chronic cadmium intoxication leads to impaired functional state of the kidneys, characterized by significant changes in glomerular filtration rate and tubular water reabsorption. The nephrotoxic effect of cadmium alters electrolyte metabolism, marked by increased excretion of sodium and calcium and decreased excretion of potassium. In a situation of chronic cadmium poisoning, the concentrating function of the kidneys is impaired [31].

Arsenic poisoning (As). Arsenic, a natural component of more than 200 natural minerals, is included in the WHO list of 10 chemical elements that pose significant public health threats[5].

The mechanism of action of trivalent and pentavalent arsenic compounds differs. Trivalent arsenic acts by blocking the pyruvate dehydrogenase complex, which plays a key role in glycolytic processes. Trivalent arsenic reduces ATP resynthesis and the formation of oxaloacetic acid from pyruvate (disrupting pyruvate gluconeogenesis), ultimately leading to hypoglycemia. Trivalent arsenic also inhibits the activity of glutathione synthase, glucose-6-phosphate dehydrogenase, and glutathione reductase, resulting in glutathione deficiency in the liver and impaired arsenic detoxification processes. Due to disrupted glycolysis, acetylcholine synthesis is also impaired, which is a cause of peripheral neuropathy [32].

When entering the human body orally (through food or water) and via inhalation (atmospheric air, dust) in elevated quantities, arsenic can primarily cause liver dysfunction, allergic reactions, skin changes (hyperkeratosis, dermatitis), vascular damage (often in the lower extremities), hearing loss, immunosuppression, impaired hematopoiesis, and severe neurological disorders (increased CNS excitability, irritability, headaches). Chronic As intoxications lead to damage to peripheral nerve fibers, where demyelination is severely pronounced, even to the point of axonal destruction. Dermatological manifestations include the appearance of dark brown pigmentation in the form of isolated or merging spots on the skin, hyperkeratosis of the palms and soles, followed by the development of epidermoid carcinomas in these areas [33].

The main complications of acute As intoxication are the development of intravascular hemolysis, acute renal and hepatic failure, and cardiogenic shock. Long-term consequences of acute poisonings in children may include significant hearing loss. Damage to the nervous system manifests as toxic encephalopathy (impaired speech, coordination, epileptiform seizures, psychoses) [12].

Thallium poisoning (Tl). In the overall structure of HM poisonings, thallium compounds rank relatively low; however, the severity of their course, complex differential diagnosis, and challenges in treatment necessitate special attention to these intoxications[6].

A significant role in the mechanism of thallium toxic action is played by blocking of sulfhydryl groups and suppression of thiol-dependent enzymes that regulate mitochondrial permeability, leading to water influx and swelling. Thallium was established to disrupt glutathione metabolism, enhance lipid peroxidation processes, damags the membrane apparatus, and cause cell death [34, 35]. Thallium also delays protein synthesis by acting on ribosomes (particularly the 60S subunit), leading to impaired keratinization and alopecia. Thallium intoxication results in disrupted riboflavin metabolism by forming

[5] World Health Organization. URL: https://www.who.int/ru/news-room/fact-sheets/detail/lead-poisoning-and-health (access date: 05.04.2025).
[6] Livanov GA. Poisoning with Thallium Compounds (Clinical Presentation, Diagnosis and Treatment). A Guide for Physicians. Saint Petersburg; 2016.

an insoluble complex with riboflavin and leading to riboflavin deficiency and impaired cellular energy supply [36, 37]. Certain symptoms of thallium poisoning, such as peripheral neuropathy, alopecia, and dermatitis, confirm riboflavin deficiency (as demonstrated in animal models of riboflavin deficiency). All this leads to the blocking of active transport of alkali metal ions and causes disruptions in various functional systems, which accounts for diverse clinical patterns [38].

Given the above, one of the most potent and widespread forms of chemical pollution is HM contamination [10]. Lead is the most hazardous HM to human health, due to its ability to replace metal ions essential for biochemical processes and disrupt biological processes in cells [39]. It is important to note that the kinetics of such HMs as lead and cadmium are similar. Indeed, they lack their own transporters and enter the cells and blood using transport systems intended for normally present metals and trace elements. However, although the negative impact of cadmium on human health is beyond doubt, the relationship between the dose levels of cadmium intake and deterministic genotoxic effects remain to be understood. Such effects are likely to be related to the multifactorial nature of cadmium action resulting from cadmium ions binding to numerous cellular targets. Consequently, the development of approaches to reducing cadmium-induced toxicity is a priority direction in toxicological research [24].

Poisoning by medicinal neurotropic drugs

According to data from the World Association of Toxicological Centers (International Association of Forensic Toxicologists, TIAFT), acute poisonings with neurotropic drugs rank among the top positions in the structure of poisoning accidents [40]. Poisoning with barbiturates account for approximately 20–25% of all cases of acute poisonings for which patients are admitted to specialized toxicological hospitals [41].

Barbiturate poisoning. Recent years have seen a decline in barbiturate poisoning cases [42], attributed to both the reduced scope of application and distribution of barbituric acid derivatives, as well as the introduction of more modern/safer drugs, including benzodiazepines. Despite this, barbiturates remain among the top 15 classes of medications causing patient deaths from drug intake [43].

According to data from the Department of Clinical Toxicology of the Dzhanelidze Research Institute of Emergency Care, in 2015, out of 286 cases of barbiturate poisoning, 104 patients (36.3%) were over 60 years old, of whom 11 (3.8% of the elderly group) died. In 2016, out of 482 patients, 123 (25.5%) were of elderly and senile age, with 21 (17.1% of the elderly group) experiencing a fatal outcome. Poisoning by barbituric acid derivatives is most commonly provoked by the use of combined medications such as Corvalol or Valocordin®, which, in addition to phenobarbital, contain ethanol, ethyl ester of α-bromoisovaleric acid, and peppermint leaf oil. The severity of the patients' condition was determined by the development of toxic encephalopathy, clinically manifested by consciousness disorders of varying severity: from mild stupor to atonic coma [44].

Yesin et al. indicate that barbiturates are most frequently used in suicide attempts by individuals over 60 years old (27.6% of all suicidal poisonings in this age category), whereas among persons aged 18–59, suicidal barbiturate poisonings account for 11.8% [45].

For humans, a single oral intake of approximately 10 therapeutic doses of the drug is considered lethal. The lethal dose of phenobarbital ranges 2–10 g [46]. In high doses, barbiturates exert a depressant effect on the sensory areas of the cerebral cortex, thereby reducing motor activity and suppressing cerebral functions [42].

Clinically pronounced symptoms of toxic barbiturate poisoning may manifest only several hours after drug ingestion. Intoxication presents as nystagmus, ataxia, dizziness, headache, increasing psychomotor retardation, suppression or complete loss of reflexes, marked drowsiness or agitation, progressing to respiratory depression, pupillary constriction (alternating with paralytic dilation), increased or decreased heart rate while maintaining normal cardiac rhythm, and cyanosis. Pulmonary edema and coma development are among the complicated symptoms of barbiturate intoxication [46].

A detailed study of the clinical course of barbiturate intoxication conducted by Alexandrovsky et al. identified the main syndromes in 385 examined patients. These were comatose state and other neurological disorders (pupillary constriction, sensory disturbances, increased or decreased tendon and skin reflexes, etc.), respiratory and hemodynamic impairments [47].

Post-hypoxic brain injury is one of the most serious consequences of barbiturate poisoning. This complication is observed in 35% of patients with severe and extremely severe poisoning, manifested as reduced cognitive function, paralysis, paresis, and impaired functioning of internal organs. In some cases, the development of cerebral edema can lead to fatal outcomes or irreversible damage to the CNS [22].

Intoxication with organic solvents

Methanol poisoning. Methanol, also known as methyl or wood alcohol, is an organic chemical compound and the simplest aliphatic alcohol with the chemical formula of CH_3OH. It is a colorless, poisonous liquid widely used in the industrial sector as a solvent, in the synthesis of organic compounds, in the production of resins and dyes, and is a component of windshield washing fluids. Due to the active search for alternative energy sources and the consideration of alcohols as a replacement for hydrocarbon fuels, the use of methanol is forecasted to increase worldwide [48].

The problem of methanol poisoning is extremely acute. According to Rosstat data, poisoning with alcohol-containing products continues to be a leading cause of fatal outcomes due to chemical poisoning. The most tragic case of mass methanol poisoning occurred in Irkutsk (December 2016), where 77 people died, with the maximum number of fatalities in the working-age population [49]. Another highly publicized mass methanol poisoning in Russia occurred during the period from June 3 to 5, 2023, when residents of several regions consumed alcoholic products under the brand of Mr. Cider. As a result, 47 people died and at least 106 were affected. Loskutnikova et al., Yakovenko et al. have also noted a wave-like dynamics in mortality rates from methanol poisoning [50, 51].

According to the statistical data presented in the state report "On the State of Sanitary and Epidemiological Well-Being of the Population in the Russian Federation in 2021," 470,358 cases of acute poisoning with alcohol-containing products were registered in the Russian Federation in 2012–2021, with 124,813 of them being fatal (26.5%). The rate of acute poisoning with alcohol-containing products in 2021 was 21.19 cases per 100,000 population.[7]

Thus, the statistical data on poisonings in the Vologda Oblast shows that methanol poisoning consistently ranks second after the cases of ethanol poisoning (Table 2).

Acute methanol poisoning progresses through four stages: initial stage, stage of latent manifestations or false well-being, stage of pronounced clinical manifestations, and the period of poisoning consequences.[8]

The initial stage, lasting 1–12 h, is characterized by manifestations of ordinary alcohol intoxication caused by the narcotic effect of this alcohol on the CNS, with the degree of intoxication being less pronounced than that with comparable doses of ethyl alcohol[9]. The stage of false well-being follows the period of intoxication [53].

The stage of pronounced clinical manifestations is characterized by the development of toxic gastritis, toxic encephalopathy with headaches and dizziness, psychomotor agitation, stupor and confusion, loss of contact. At the culminating stage, symptoms of severe general

Table 2. Number of alcohol poisoning accidents in 2020–2022 (per 100,000 population) in the Vologda Oblast according to data from the Local Territorial Center for Hygiene and Epidemiology

Parameter	2020		2021		2022	
	total	of which fatal	total	of which fatal	total	of which fatal
Toxic effects of alcohol (T51) — total	6.4	3.7	4.7	1.8	4.5	1.4
Ethanol (T51.0)	4.4	2.7	3.5	1.4	3.2	0.8
Methanol (T51.1)	0.1	0.1	0.1	0.0	0.4	0.4
2-propanol (T51.2)	0.1	0.1	0.3	0.3	0.1	0.1
Fusel oil (T51.3)	0.2	0.2	0.0	0.0	0.0	0.0
Other alcohols (T51.8)	0.0	0.0	0.1	0.1	0.0	0.0
Unspecified alcohol (T51.9)	1.6	0.7	0.8	0.1	0.7	0.1

Table prepared by the authors according to the source [52]

[7] On the State of Sanitary and Epidemiological Well-Being of the Population in the Russian Federation in 2021: State Report. M.: Federal Service for Surveillance on Consumer Rights Protection and Human Wellbeing; 2022.

[8] Analysis of acute chemical etiology poisonings, including alcohol-related fatal cases for 2022. URL: https://fbuz35.ru/files.aspx?id=4038db6b560c4057b9be4e4358af0412 (access data: 10.04.2025).

[9] Clinical Guidelines "Toxic Effects of Methanol and Ethylene Glycol" (approved by the Ministry of Health of Russia). https://legalacts.ru/doc/klinicheskie-rekomendatsii-toksicheskoe-deistvie-metanola-i-etilenglikolja-odobreny-minzdravom/?ysclid=mdfwi7hjv1220621463 (access data: 10.04.2025).

intoxication develop in the form of acute respiratory and cardiovascular failure. Ophthalmological disorders are among the main consequences of methanol poisoning (in surviving patients). The main complications of methanol poisoning include toxic kidney damage, acute toxic myocarditis, and, in the long-term period, psychoneurological disorders in the form of memory impairment, emotional lability, disorders such as “body schema disturbance”.

A characteristic feature of methanol intoxication consists in its metabolism occuring according to the principle of “lethal” synthesis, resulting in the formation of more poisonous substances: formaldehyde, whose toxicity is 33 times higher than that of methanol itself, and formic acid. Part of the formaldehyde interacts with proteins, and another part is converted into formic acid. The metabolism of formaldehyde occurs through the two main ways: with the help of tetrahydrofolic acid and reduced glutathione. Formic acid is rapidly formed during the oxidation of formaldehyde, and its further metabolism proceeds slowly. The main metabolic transformations occur in the liver tissue, which has the greatest ability to oxidize formaldehyde. In addition, a significant part of methanol is excreted through the lungs.

Poisoning with diols (1,4-butanediol and related compounds). Among the most frequently encountered poisoning exposures to organic solvents, 1,4-butanediol (1,4-BD) holds a special place. It is a dihydric aliphatic alcohol widely used in industrial sectors as a solvent, an intermediate in organic synthesis, and the production of plastics[10]. In recent years, this compound has increasingly been used illegally to achieve a specific emotional state (euphoria). Thus, the prevalence of 1,4-butanediol use as a psychoactive substance (PAS) among the drug-dependent population ranges 22.3–43.7%; moreover, it is often consumed concurrently with other PAS (ethanol, amphetamine and its derivatives, etc.) [54, 55].

The toxicodynamics of oral 1,4-BD use includes rapid absorption by the stomach and upper small intestine into the blood from and passage through the blood–brain barrier. After oxidation by alcohol dehydrogenase (ADH) to gamma-hydroxybutyraldehyde, it is metabolized to GHB and its carboxyl metabolite (glucuronide) followed by metabolization in the liver to aldehyde metabolites under the influence of ADH [56]. Moreover, 1,4-butanediol is metabolized to GHB, on average, within 1 min [57]. At the same time, studies on volunteers have shown significant individual differences in the rate of metabolism of 1,4-butanediol to GHB, presumably associated with varying degrees of alcohol dehydrogenase activity [58]. The minimum toxic dose of 1,4-BD for humans is 5–20 g (88–300 mg/kg) [59].

The clinical pattern of acute 1,4-butanediol poisoning manifests as behavioral disorders in the form of psychomotor agitation accompanied by anxiety and aggression, hallucinations, disorientation, and delirium [59]. In severe 1,4-BD poisoning, loss of consciousness up to coma, neurological disorders, impaired respiratory function, cardiovascular activity, and various metabolic disturbances are observed [55].

A number of studies have presented illustrative clinical cases of acute oral poisonings with 1,4-BD. For instance, Sinchenko et al. described a clinical case of acute 1,4-BD poisoning combined with ethanol in a young man who had long used this psychoactive substance in small doses to enhance sexual arousal and physical endurance. Acute poisoning occurred after a single intake of an excessive dose of the psychoactive substance, leading to the development of a convulsive syndrome, depression of consciousness to the level of stage II coma, and the onset of toxic encephalopathy [55]. Livanov et al. described a clinical case of severe acute oral poisoning with 1,4-butanediol in a 12-year-old adolescent girl, accompanied by toxic-hypoxic encephalopathy and stage III coma, resulting from the accidental ingestion of 1,4-BD [59].

In the USA and EU, the issue of the narcotic use of gamma-hydroxybutyric acid (GHB) and its precursors (1,4-butanediol and gamma-butyrolactone) is related to the frequent use of these substances in the commission of sexual crimes [57].

Industrial chemicals and toxic compounds

Carbamate poisoning. Carbamates are derivatives of carbamic acid used in agriculture as insecticides and pesticides, as well as in pharmacology as medicinal products (tranquilizers, muscle relaxants, antidotes, etc.). The main routes of carbamate entry into the human body are the respiratory organs and intact skin, as well as the gastrointestinal tract[11].

Carbamate intoxication holds a significant position in the structure of poisoning caused by neurotoxic chemicals. These compounds can cause dysfunction of the cholinergic system by activating nicotinic and muscarinic receptors. A number of carbamic acid derivatives are highly toxic compounds, reversible (unlike organophosphates — OPs) inhibitors of cholinesterases, leading to the so-called “cholinergic crisis.” This condition is associated with the development of generalized convulsive syndrome, which results in coma and

[10] Great Russian Encyclopedia: Scientific and Educational Portal. https://bigenc.ru/c/1-4-butandiol-b997fe/?v=8031024 (access data: 05.04.2025).
[11] Gerunova L, Boyko T. Toksikologiya pestitsidov. Moscow: ID Nauchnaya biblioteka; 2021.

death in severe cases. Due to the rapid hydrolysis of the C=O bond (decarbamoylation of the enzyme) during acute carbamate intoxication, cholinesterase activity in survivors recovers within a few hours, with complete restoration of cholinesterase function observed within 24–48 h [60].

The clinical pattern of carbamate intoxication is determined by the accumulation of acetylcholinesterase at nerve endings. The symptoms of intoxication can be classified into the following groups [61]:

1. Muscarinic-like manifestations: increased bronchial secretion, profuse sweating and salivation, lacrimation, pupillary constriction, bronchospasm, abdominal cramps (vomiting and diarrhea), bradycardia.
2. Nicotinic-like manifestations: fasciculations of small muscles (in severe cases, also respiratory and diaphragmatic muscles), tachycardia.
3. Symptoms and signs of CNS damage: headache, dizziness, anxiety, memory loss, convulsions, coma.
4. Depression of the respiratory center.

All these symptoms manifest in various combinations and may vary in presentation and sequence depending on the substance, dose, and route of exposure. The duration of symptoms is generally shorter than with OP exposure [62]. In severe cases of poisoning, death may occur due to asphyxia from muscle spasm or acute heart failure [63]. Long-term consequences include significant impairments in cognitive and behavioral abilities [61].

Based on the analysis of the social status of individuals poisoned by carbamates, Reddy et al. revealed unexpected results: 23% of the victims were farmers, 27% were temporary workers, 21% were homemakers, 11% were permanent employees, and 8% were students [64]. Thus, in addition to the risk of poisoning in workplaces with hazardous conditions or during various terrorist incidents, accidental or intentional self-poisoning, as well as toxic effects from drug overdoses, are quite widespread [65]. According to the WHO, approximately one million cases of unintentional acute pesticide poisoning are registered worldwide each year.

CONCLUSION

Our study has reviewed the characteristics of the main representatives of neurotropic toxicant groups that pose threat to public health. It was revealed that although the central nervous system is the primary target organ for neurotropic toxicants, their toxicity adversely affects other organs. This explains the wide spectrum of pathologies developing as a result of exposure to these poisons (impairments of external respiratory function, cardiovascular activity, various metabolic disturbances up to multiorgan pathology). Signs of toxic action can manifest both immediately after intoxication and in the period of long-term consequences.

The health risk of poisonings by neurotropic toxicants is associated with the following aspects. First, such poisoning can occur not only in industrial but also in domestic settings. Second, poisoning by neurotropic toxicants leads to the development of chronic pathological processes in the victim's body. In addition, acute poisoning with some neurotropic toxicants includes a latent period lasting up to several days, which complicates the diagnosis and hinders the timely initiation of treatment. Third, although the main disorders in the acute period, manifested in the nervous, respiratory, and cardiovascular systems, can be managed through drug therapy, there is a risk of developing long-term consequences.

Future studies should address the formation mechanisms of long-term consequences in cases of intoxication with neurotropic toxicants, as well as the search for preventive and therapeutical measures to combat the arising pathological conditions.

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