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Research Article

Methanol and Ethylene Glycol: Epidemiology of Poisoning, Biotransformation, Mechanism of Toxic Action

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Abstract

Background: Methanol and ethylene glycol are highly toxic alcohols that undergo biotransformation to metabolites more hazardous than the parent compounds, resulting in severe clinical outcomes including high mortality and disability.

Objective: To analyze scientific literature on the epidemiology and the pathogenetic mechanisms underlying the toxicity of acute methanol and ethylene glycol intoxication.

Materials and Methods: A systematic review of literature from PubMed, Scopus, and RSCI bibliographic databases was conducted. A systematic search was used.

Results: Evidence demonstrates that methanol and ethylene glycol remain significant causes of mass poisoning with severe sequelae in the twenty-first century. Methanol-related mass poisonings are documented in various countries worldwide on an almost annual basis and are typically characterized by high mortality rates. Ethylene glycol, employed by unscrupulous manufacturers in certain pharmaceutical formulations, has precipitated several mass poisonings among pediatric populations. This review presents legislative and regulatory frameworks from multiple countries that restrict the use of methanol and ethylene glycol in industrial and commercial product manufacturing. Toxicological profiles of methanol and ethylene glycol are provided, encompassing their toxicity, toxicokinetics, and biotransformation within the organism. The toxic mechanism of methanol is primarily attributed to its toxic metabolites-formaldehyde and formic acid. Similarly, the toxic effects of ethylene glycol are mediated by its metabolic products, including glycolaldehyde, glyoxal, glycolic acid, and glyoxylic acid. Metabolites of both methanol and ethylene glycol are capable of inducing cellular damage through mitochondrial dysfunction, activation of lipid peroxidation pathways, and depletion of the glutathione antioxidant system, culminating in dysregulation of multiple biochemical systems.

Conclusion: Methanol and ethylene glycol poisoning constitute a substantial proportion of substance-abuse-related and accidental exposures, particularly in countries with limited medical awareness. These poisonings cause significant morbidity and mortality, especially when antidote therapy is unavailable. Toxicity results from metabolite formation-formaldehyde and formic acid (methanol) and oxalates (ethylene glycol)-which precipitate metabolic acidosis and damage the optic nerves and kidneys. Prompt ethanol antidote therapy substantially reduces mortality.

Limitations: Incomplete data from certain regions, underreporting of cases, and lack of recent literature on the September 2025 Leningrad Region incident.

Keywords: *Methanol; Ethylene Glycol; Toxic Alcohols; Acute Poisoning; Alcohol Poisonings Epidemiology; Toxic Metabolites; Mechanisms of Toxic Action; Review*

Abbreviations

ADH: Alcohol Dehydrogenase; ALDH: Aldehyde Dehydrogenase; ATP: Adenosine Triphosphate; BAS: Biologically Active Substances; CNS: Central Nervous System; DNA: Deoxyribonucleic Acid; NAD: Nicotinamide Adenine Dinucleotide; RSCI: Russian Science Citation Index

Introduction

Alcohols comprise a large class of chemical compounds that are widely utilized in industry, medicine, and everyday life. This extensive use gives rise to a considerable risk of both occupational and accidental intoxication, as well as the development of dependence with regular consumption [21]. Among these, the toxic alcohols methanol and ethylene glycol are particularly hazardous, as they undergo biotransformation in the body to form metabolites that are considerably more toxic than the parent compounds. The key enzyme catalyzing these transformations is alcohol dehydrogenase (ADH) [22]. Poisoning by toxic alcohols typically occurs in group or mass outbreaks and is characterized by high mortality rates and a substantial risk of severe disability. Consequently, this issue constitutes one of the most significant medical and social challenges facing contemporary healthcare systems [21].

The primary approach to treating acute methanol and ethylene glycol intoxication is antidote therapy aimed at inhibiting toxicant metabolism. The most extensively studied and readily available antidote is ethanol, which competitively inhibits alcohol dehydrogenase (ADH), thereby preventing the formation of toxic metabolites [29].

Aim of the Study

The aim of this study is to analyze data from scientific literature on the prevalence and epidemiology of acute methanol and ethylene glycol intoxication, the pathogenetic mechanisms underlying their toxicity, and therapeutic approaches to these poisonings, with particular emphasis on antidote therapy using ethanol.

Materials and Methods

The material for the analysis was the literature sources reviewed in the bibliographic databases PubMed, Scopus, and RSCI. A systematic search was conducted using relevant keywords including “methanol poisoning”, “ethylene glycol poisoning”, “toxic alcohols”, “alcohol intoxication” and “alcohol poisoning epidemiology”. Articles were included if they reported clinical cases, epidemiological data, toxicological mechanisms, or therapeutic outcomes related to methanol or ethylene glycol poisoning. The review encompassed 41 publications from 1992 through 2025.

Results and Discussion

Prevalence and statistics of poisoning

Methyl alcohol poisoning remains a significant public health problem, affecting both developing and developed countries [13]. This issue is particularly pressing in countries where religious prohibitions restrict the consumption of alcoholic beverages, as this fosters the emergence of a clandestine market for alcoholic products of dubious origin, thereby elevating the risk of mass poisoning. Outbreaks of methanol poisoning have been repeatedly documented in Iran: in 2004, 62 individuals were affected; in 2013, 694 people [13]; and in 2018, 768 victims, with 76 fatalities [1].

The most substantial surge occurred in 2020, when, against the backdrop of the COVID-19 pandemic, false and misleading information was disseminated among the population claiming that ingestion or mouth-rinsing with alcohol-containing liquids, including methanol, could prevent SARS-CoV-2 infection. Consequently, approximately 2,200 poisoning cases were recorded, with 296 fatalities, including children and adolescents [20,33].

Mass episodes of methanol poisoning have also been documented in other Muslim-majority countries. In Malaysia, the first major outbreak in the region occurred in 2013, resulting in 27 deaths. A subsequent wave in 2018 claimed the lives of 64 individuals out of

90 victims, illustrating an exceptionally high mortality rate of 70% [31]. In 2020, 22 individuals in Turkey were affected following the consumption of methanol-contaminated alcohol [11], and in Tunisia in the same year, 65 individuals were affected [16].

Methanol intoxication also occurs with high frequency and regularity in European regions. The first outbreak in the new millennium occurred in Estonia in 2001, resulting in 68 deaths, and 43 cases involved severe consequences, including irreversible blindness and central nervous system (CNS) damage [26]. In 2012, more than 100 individuals were hospitalized in the Czech Republic following the consumption of counterfeit alcohol containing methanol, 38 of whom died [34]; in 2022, 19 fatal cases of poisoning were recorded in Poland [38].

Over the past thirty years, the number of individuals affected by consumption of methanol-contaminated alcohol in India has exceeded 2,000, a consequence of the proliferation of artisanal alcohol production and the absence of stringent regulatory measures [28]. Outbreaks of methanol poisoning are recorded with considerable frequency: in 1991 in Mumbai, 93 cases; in 2009 in Ahmedabad, 136 deaths related to surrogate alcohol poisoning; in 2011 in West Bengal, 172 cases; in 2015 in Mumbai, 102 cases; in 2016 in Bihar, 13 cases; and in 2019 in Uttarakhand, 99 deaths. In the same year, at least 114 individuals died in Assam, allegedly due to poisoning by counterfeit liquor [15,28]. In addition, it has been noted that in these cases the number of victims could have been higher, had it not been for the use of inpatient treatment with ethanol [15].

Methanol poisoning in Africa is characterized by a high mortality rate, attributable to limited access to antidote therapy and delayed diagnosis [32]. In Uganda, 15 cases of poisoning were reported in 2017, 12 of which were fatal. Ethanol was administered to 4 patients at the hospital stage, of whom 1 (25%) died, whereas among those who did not receive the antidote, 11 out of 11 (100%) succumbed [6]. Similar outbreaks were documented in Libya (2013, approximately 1,000 victims, 100 deaths) and Kenya (2014, 467 victims, 126 deaths), and both demonstrated a significant reduction in mortality following the timely administration of ethanol [32].

Cases of mass methanol poisoning have also been reported in the Russian Federation, the most extensive of which occurred in 2016 in Irkutsk, when more than 120 individuals suffered from the consumption of Hawthorn cosmetic liquid, of whom 78 died [41]. Among the victims, ages ranged from 18 to 77 years, with men accounting for 62% of cases and women for 38% [40].

Another high-profile case occurred in 2023 and affected several regions of the Russian Federation - the Ulyanovsk, Nizhny Novgorod, Penza, Samara, and Kurgan oblasts, as well as the republics of Udmurtia and Chuvashia. Mass poisoning from the beverage “Mr Cider,” which contained methanol, resulted in 30 deaths and hospitalization of several dozen additional victims, including minors and a pregnant woman [19].

In September 2025, another major poisoning incident occurred, not yet described in the scientific literature: in the Leningrad Region, 52 individuals were affected by the consumption of surrogate alcohol containing methanol, of whom 49 died.

Along with methanol, ethylene glycol is another toxicant that causes acute and often fatal intoxication. In recent years, the problem of glycol contamination of medicinal products has become acute, resulting in a number of disastrous consequences and demonstrating insufficient control over the quality of pharmaceutical products in several countries [27]. In 2022, numerous cases of acute renal failure were documented in Uzbekistan and adjacent regions of Tajikistan among children aged 6 months to 10 years who had consumed Dok-1 Max medicinal syrups manufactured by the Indian pharmaceutical company Marion Biotech [23]. The majority of affected children exhibited severe clinical and laboratory evidence of toxic injury, including anuria, increased potassium in the blood, and neurological disturbances. According to official data, 68 children died, while 18 sustained disabilities as a consequence of multiple organ failure [23,27].

Similar cases were observed in The Gambia, where 78 cases of poisoning among children were registered in 2022, of which 66 were fatal, corresponding to a mortality rate exceeding 80% [4]. Toxicological analysis of four pharmaceutical preparations - Kofexmalin,

Makoff, Magrip N, and Promethazine - demonstrated the presence of ethylene glycol and diethylene glycol at concentrations substantially exceeding permissible limits. These toxic substances were employed as solvents in place of the intended excipients, glycerol and propylene glycol [4,23]. Comparable tragedies had occurred previously: in Panama in 2006, 115 individuals died from diethylene glycol-contaminated pharmaceuticals, and in Nigeria in 2009, 84 children died following poisoning with glycols present in substandard medicines [27].

Ethylene glycol toxicity remains among the leading causes of death from chemical poisoning in industrialized nations. In the United States, more than 5,000 cases of ethylene glycol intoxication are reported annually, encompassing both accidental and intentional exposures [35]. In addition to individual intoxications, mass poisoning incidents also occur. In 2021, 11 soldiers stationed at Fort Bliss in Texas were hospitalized after ingesting an unmarked liquid that was identified as a cooling mixture containing 62% ethylene glycol. Owing to the prompt provision of medical care, including antidote therapy and hemodialysis, all affected individuals survived [35]. However, in Brazil in 2019, a comparable incident concluded more tragically: among 39 victims who consumed an alcoholic beverage contaminated with diethylene glycol, 9 individuals died. This outcome underscores the critical importance of timely detoxification measures, including the administration of antidotes [9].

Toxicological characteristics of methanol

Methyl alcohol (CH_3OH; methanol) is a monohydric alcohol that exists as a colorless, volatile liquid. Pure methanol was first isolated in 1661 by Robert Boyle through the dry distillation of boxwood, which led to its common name, wood alcohol [21].

Methanol poisoning primarily occurs via ingestion; however, inhalation and percutaneous (dermal) absorption also constitute viable routes of exposure [30]. One documented case occurred in the 1980s at a petrochemical facility in the port of Jubail, Saudi Arabia. An engineer, lacking personal protective equipment, spent 2 - 3 hours inside the confined space of a methanol storage tank and subsequently continued working on deck while wearing clothing saturated with methanol. Symptoms of acute toxicity were manifested 8 hours after exposure; however, owing to the prompt initiation of treatment with ethanol as an antidote, clinicians achieved complete recovery of the patient with no residual sequelae [7].

Methanol is rapidly absorbed following gastrointestinal administration; approximately 80 - 90% of an administered dose reaches the systemic circulation within 60 minutes [21]. The compound distributes uniformly within the body's aqueous compartments and does not bind to plasma proteins; its mean volume of distribution (Vd) is approximately 0.7 $L \cdot kg^{-1}$ [2]. The elimination half-life of methanol under physiological conditions ranges from approximately 12 to 24 hours; administration of antidotes such as ethanol or fomepizole prolongs the half-life, extending it to roughly 24 - 30 hours.

Biotransformation and mechanism of toxic action of methanol

Methanol can be absorbed via inhalation, ingestion, and percutaneous exposure; occupational poisoning cases provide evidence for these routes of entry.

Most methanol poisonings result from oral ingestion. Toxic effects may occur following ingestion of approximately 7 - 8 mL of pure methanol. Reported lethal doses of methanol vary widely, most commonly cited in the range of 30 - 100 mL [17,40]; nevertheless, fatalities have been reported following ingestion of as little as 5 mL, while survival has occurred after ingestion of 250 - 500 mL in some cases [25,36]. Toxic blood methanol concentrations are generally reported to be approximately 0.2 - 0.3 $mg \cdot mL^{-1}$, whereas concentrations of about 0.8 - 1.0 $mg \cdot mL^{-1}$ are associated with fatal outcomes [41].

Following oral consumption, methyl alcohol is absorbed relatively rapidly and attains peak blood concentrations within 30 - 60 minutes [12,41]. A portion of methanol is secreted by the gastric mucosa into the stomach lumen over the course of several days and subsequently undergoes reabsorption [41].

Methanol is not excreted unchanged in expired air or urine; rather, it is metabolized and distributed among organs and tissues. The highest concentrations accumulate in the liver and kidneys, with comparatively lower amounts in muscle, adipose tissue, and the brain. A portion of ingested methanol is secreted by the gastric mucosa into the stomach lumen over several days and subsequently undergoes reabsorption. The liver exhibits the greatest accumulation and metabolic activity toward methanol. Oxidation of methanol is catalyzed principally by alcohol dehydrogenase (ADH), with additional contributions from the hepatic microsomal system and the catalase-peroxidase system.

It is well established that a portion of absorbed methanol is excreted unchanged in expired air (~1%) and in urine (~5%), while the remainder undergoes relatively slow metabolism. Furthermore, absorbed methanol and its metabolites are secreted by the gastric mucosa into the stomach lumen over several days following poisoning and are subsequently reabsorbed in the intestine. Methanol metabolism occurs predominantly in the liver, which possesses the greatest oxidative capacity toward alcohols (accounting for up to 95% of oxidation). The principal oxidation products of methanol are formaldehyde and formic acid, and the toxic effects of methanol are primarily attributable to the actions of these metabolites [22].

Methanol is metabolized predominantly in the liver, where it is oxidized to formaldehyde by NAD^+-dependent alcohol dehydrogenase (ADH) [3,5]. A fraction of formaldehyde binds to blood proteins; however, the greater proportion is rapidly oxidized by aldehyde dehydrogenase to formic acid, which constitutes the principal toxic metabolite of methanol. Formic acid is either oxidized by formyltetrahydrofolate dehydrogenase to carbon dioxide and water or eliminated unchanged [39].

The role of folic acid in methanol metabolism is of particular importance in the development of methyl alcohol toxicity, as it serves as a cofactor for enzyme systems involved in methanol oxidation. Subsequent metabolism of methanol to its final oxidation products, carbon dioxide (CO_2) and water (H_2O), is completed via the Krebs citric acid cycle [22].

Methanol and its metabolites are regarded as potent neurovascular and protoplasmic poisons that disrupt oxidative phosphorylation, thereby inducing ATP deficiency, particularly in brain tissue and the retinal retina. These disturbances impair the local metabolism of biologically active substances (BAS) and ultimately lead to demyelination and subsequent atrophy of the optic nerve. Accumulation of organic acids (e.g. lactic acid, glucuronic acid) in the body precipitates metabolic acidosis, which is exacerbated by inhibition of oxidative processes resulting from the blocking effects of methanol and formic acid on cellular respiratory enzymes. Notably, metabolic acidosis itself further inhibits cellular respiration, creating a self-amplifying pathological cycle [22].

Toxicological characteristics of ethylene glycol

Ethylene glycol (1,2-dihydroxyethane [5]; 1,2-ethanediol; ethanediol-1,2), $HO\text{-}CH_2\text{-}CH_2\text{-}OH$, is an oxygen-containing organic compound belonging to the diol class and is a representative polyol (polyatomic alcohol). When purified, it is a clear, colorless liquid with a slightly viscous, oily consistency; it is odorless and has a sweet taste. The substance is toxic and presents fire and explosion hazards [22].

Ethylene glycol, a diol ($HO\text{-}CH_2\text{-}CH_2\text{-}OH$), is a constituent of numerous technical fluids, including antifreeze formulations used for cooling internal combustion engines, as well as brake fluids, shock-absorber fluids, and a range of hydraulic fluids [12]. Ethylene glycol is also present in detergents, paints, cosmetics, cooling solutions, and de-icing/defrosting reagents [14,24]. In certain commercial formulations, the dye sodium fluorescein is added [14].

This substance is rapidly absorbed from the gastrointestinal tract following oral ingestion and exhibits a volume of distribution of approximately 0.7 $L \cdot kg^{-1}$. At plasma concentrations below 250 $mg \cdot dL^{-1}$, ethylene glycol is eliminated by first-order kinetics, with a half-life of approximately 4 hours; at higher concentrations, elimination follows zero-order kinetics, resulting in a prolonged half-life.

In most cases, ethylene glycol (antifreeze) poisoning results from intentional ingestion for inebriation. Inhalation poisoning is not observed owing to the compound's low volatility. There is marked interindividual variability in sensitivity to ethylene glycol. Reported lethal doses range from 50 to 500 mL, with an average of approximately 100 mL [12].

Clinical manifestations typically progress through three distinct phases. Phase 1 (0.5 - 12 hours) is characterized by central nervous system (CNS) effects, including inebriation, ataxia, seizures, coma, and potentially death; gastrointestinal irritation may induce nausea and vomiting. Phase 2 (12 - 24 hours) involves accumulation of organic acids, resulting in a cardiopulmonary syndrome manifested by tachycardia, hypertension, tachypnea, and pulmonary edema. Phase 3 (24 - 72 hours) is characterized by acute renal failure secondary to osmotic disturbances and deposition of calcium oxalate crystals in the kidneys, while metabolic acidosis persists throughout the clinical course.

Furthermore, severe cases may exhibit a fourth phase characterized by neurological complications, including delayed cranial neuropathies, cerebral edema, seizures, elevated intracranial pressure, stroke-like manifestations, diaphragmatic paralysis, sensory radiculopathy, and autonomic nervous system dysfunction [8].

Biotransformation and mechanism of toxication of ethylene glycol

Ethylene glycol is rapidly absorbed from the stomach and intestines, attaining peak blood concentrations within the first 6 hours after ingestion; detectable circulating levels persist for approximately 48 hours. Only 20 - 30% of the absorbed dose is excreted unchanged by the kidneys [22]. Metabolism occurs predominantly in the liver and is mediated by alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH), enzymes that catalyze the reduction of NAD^+ to NADH [14,24]. Consequently, metabolism yields glycolic aldehyde, glycolic acid, and calcium oxalate, which initially increases the osmolar gap and is subsequently followed by development of an anion gap; these metabolic disturbances are accompanied by neurotoxicity and nephrotoxicity [24].

The efficiency of alcohol metabolism plays a critical role in the development of toxic effects; at the initial stage, ethylene glycol is oxidized to glycolaldehyde. The metabolic products - glycolic acid, glyoxylic acid, and their corresponding aldehydes - are substantially more toxic than the parent compound. During ethylene glycol oxidation, approximately 70 - 80% is metabolized in the liver to glycolaldehyde and organic acids, including glycolic acid, glyoxylic acid, and oxalic acid. Glycolaldehyde is subsequently converted to glycolic acid via enzymatic action, and, through the participation of lactate dehydrogenase or hydroxy acid oxidase (glycolate oxidase) 1, to glyoxylic acid.

Certain metabolites, such as glycolaldehyde, can be converted to glyoxal and subsequently to glyoxylate via both enzymatic and non-enzymatic pathways. The most toxic metabolites are glycolic acid and, particularly, glyoxylic acid, which induce severe metabolic acidosis that inhibits cellular respiration, protein synthesis, and DNA replication. Glyoxylic acid metabolism includes conversion to oxalic acid, formic acid, and glycine, as well as conjugation to form oxalomalate and related compounds. The formation of oxalic acid results in deposition of calcium oxalate crystals in brain tissue, blood vessels, lungs, and myocardium, leading to the development of hypocalcemia [22].

Patients exhibiting clinical evidence of toxic injury to target organs are at greater risk for adverse outcomes, including prolonged resuscitation intervals and mortality [10].

Conclusion

Methanol and ethylene glycol poisoning constitute a substantial proportion of substance-abuse-related and accidental exposures, particularly in countries with limited medical awareness. Their toxic mechanisms are mediated by the formation of metabolites-formaldehyde and formic acid in methanol poisoning, and oxalates in ethylene glycol poisoning-which precipitate metabolic acidosis and cause tissue injury, notably to the optic nerves and kidneys, and may result in death in the absence of timely treatment. Epidemiological data indicates high mortality rates for these poisonings, particularly when antidotal therapy and rapid medical intervention are unavailable.

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Authors' Contributions

Polyakov A.D.: Collection of material in bibliographic databases, processing and analysis of the material, writing the text; Strelova O.Yu.: Analysis of the material, editing; Grebenyuk A.N.: Concept and design of research, analysis of the material, editing. All co-authors are responsible for approving the final version of the article and ensuring the integrity of all parts of the article.

Conflict of Interests

The authors declare that there are no conflicts of interest.

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