BIOMEDICINE AND CHEMICAL SCIENCES
AN INTERNATIONAL PEER-REVIEWED JOURNAL

BIOMEDICINE AND CHEMICAL SCIENCES
Website: https://journals.irapa.org/index.php/BCS
e-ISSN: 2790-296X | p-ISSN:2957-5826

Original Article

Pharmaceutical Importance and Applications of Glycols

Abdul Quiyoom*, Navneet Kumar Verma, Shekhar Singh, Ashish Pal, Vaishnavi Pandey, Ruchi Yadav

Suyash Institute of Pharmacy, Hakkabad, Gorakhpur, Uttar Pradesh, India

Corresponding Author:
Abdul Quiyoom

Article History:
Received on: Oct 04, 2025
Revised on: Nov 14, 2025
Accepted on: Nov 24, 2025
Published on: Dec 04, 2025

Citation: Abdul Quiyoom *et al.* Pharmaceutical Importance and Applications of Glycols. *Biomedicine and Chemical Sciences, 4(4): 1-8, 2025*

ABSTRACT

Glycols are aliphatic dihydric alcohols that contain two hydroxyl groups. They are sweet-tasting, colourless liquids with many industrial uses, especially as solvents and antifreeze agents [1]. Diethylene Glycol is a transparent, thick liquid that is an organic substance [4]. It tastes pleasant and is hygroscopic. When consumed, diethylene glycol, a very poisonous organic solvent, can result in acute renal failure and even death. Renal damage from DEG toxicity is caused by proximal tubular necrosis. In the chemical industry, ethylene glycol (EG) is a crucial reagent. Since it is widely used in many catalytic and non-catalytic chemical industries, including anti-freezing, liquid detergent, pharmaceuticals, explosives, hydraulic brake fluids, engine coolants, and hydrate inhibitors used for the recovery of subsea oil and gas, ethylene glycol (EG) is regarded as an extremely important organic compound.

Keywords: Ethylene Glycol (EG), Diethylene Glycol (DEG), Mode of Action, Pharmacokinetics and Pathophysiology.

INTRODUCTION

Ethylene Glycol (EG)

Glycols are aliphatic dihydric alcohols that contain two hydroxyl groups. They are sweet-tasting, colourless liquids with many industrial uses, especially as solvents and antifreeze agents [1]. In the pharmaceutical sector, syrups based on glycerol (GLY), sorbitol, or polyethylene glycol (PEG) may contain impurities such as ethylene glycol (EG) and diethylene glycol (DEG). Because they serve as organic solvents, humectants, antimicrobials, and preservatives, GLY, sorbitol, and PEG are frequently employed as excipients in pharmaceutical syrups [2]. Because of its extremely reactive chemical qualities, it is widely used in many different domains, such as the manufacturing of coolant and antifreeze, as well as in the textile, plastic, and coating industries [3].

Diethylene Glycol (DEG)

Diethylene Glycol is a transparent, thick liquid that is an organic substance [4]. It tastes pleasant and is hygroscopic. Water, lower alcohols, acetone, phenol, aniline, and chloroform are all soluble in it [5]. It dissolves sparingly in carbon tetrachloride, diethyl ether, and benzene. It dissolves poorly in vegetable and mineral oils [6]. Apart from being utilized in numerous industrial items, it has also been implicated in several notable mass poisonings since

1937. A thorough analysis has not yet been released, despite DEG's toxicity and the outbreaks of deadly poisonings that are linked to it [7].

Diethylene Glycol (DEG) is a chemical molecule that can be found in both household items and pharmaceuticals as a fake solvent. Acute kidney injury (AKI) and neurological aftereffects including diminished reflexes or facial and limb paralysis are the hallmarks of DEG poisonings. The dosage sensitivity of females to DEG toxicity is unknown, while prior research in male rats has shown that neurotoxic effects only appear when AKI is established [8].

For over a century, diethylene glycol dinitrate (EGDN), diethylene glycol dinitrate (DEGDN), and triethylene glycol dinitrate (TEGDN) have been utilized as pasticizers in propellant mixes. The crystal structures of all three of these compounds, which are liquids at room temperature, have not been established despite their lengthy history and industrial use [9].

When consumed, diethylene glycol, a very poisonous organic solvent, can result in acute renal failure and even death. When it was utilized in the US to make a sulphanilamide elixir in the 1930s, its toxicity became clear [10]. The United States Food, Drugs, and Cosmetics Act, which governs the testing and use of novel medications and foods, was passed in 1938 as a result of the at least 76 deaths caused by the consumption of this sulphanilamide elixir. Although it is rarely found in fatal amounts, diethylene glycol is nonetheless infrequently found in foods and medical treatments. The findings of examinations conducted in response to a widespread, initially inexplicable outbreak of acute renal failure caused by diethylene glycol poisoning are presented in this study [11].

Chemical Information of Impurities (Ethylene Glycol and Diethylene Glycol) Name: Ethylene Glycol (EG)
Chemical Name and Structure
Chemical Names: Ethane-1,2-diol; 1,2-ethanediol
Chemical Structure:
Molecular Formula and Molecular Weight Molecular Formula: C2H6O2
Molecular Weight: 62.07 g/mol Chemical Name and Structure
Chemical Names: 2,2′-Oxydiethanol; Ethylene diglycol; Diglycol.
Chemical Structure:
Molecular Formula and Molecular Weight Molecular Formula: C4H10O3
Molecular Weight: 106.12 g/mol [12].

Uses of Ethylene Glycol

In the chemical industry, ethylene glycol (EG) is a crucial reagent. Its primary uses include the synthesis of polymers, such as polyester and polyethylene terephthalate, as well as non-volatile braking and antifreeze fluids and anti-icing additives [13]. About 20 million metric tons of EG were produced and consumed worldwide in 2010, with an estimated 5–10% annual growth. As a result, there is a lot of interest in the concentration and purification of waste EG for its regeneration and reuse. Due to its extensive application in the production of chemicals, the transportation and textile sectors, and energy technologies, EG use is rising annually (Figure 1). The manufacturing of coolants or antifreezes for automobiles and personal computers is one of the most significant uses of EG. EG is also utilized as an anti -icing solution for windshields and airplanes because of its low freezing point. Polyester fibers and resins, such as polyethylene terephthalate (PET), are produced using EG in the plastics industry [14].

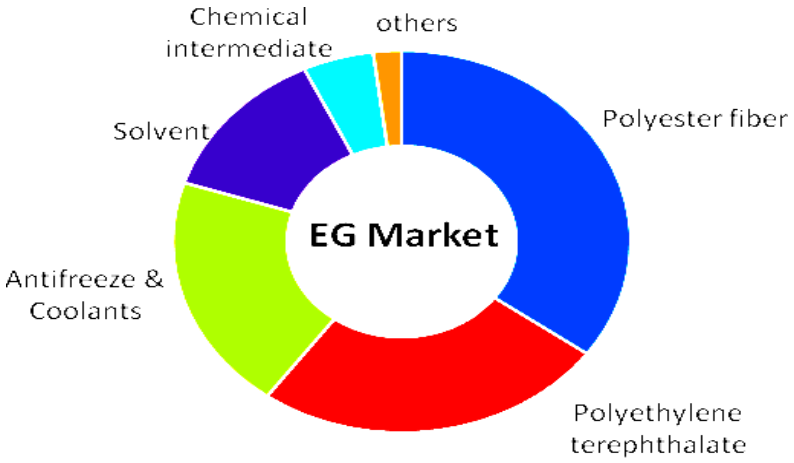

Figure: 1. Mono-Ethylene Glycol Market [14].

Since it is widely used in many catalytic and non-catalytic chemical industries, including anti-freezing, liquid detergent, pharmaceuticals, explosives, hydraulic brake fluids, engine coolants, and hydrate inhibitors used for the recovery of subsea oil and gas, ethylene glycol (EG) is regarded as an extremely important organic compound. Additionally, it is utilized as an intermediate compound in a variety of petrochemical industries, including the synthesis of resins, plastics, textiles, biodegradable polyester fibers, cosmetics, plasticizers, fabric, solvents for paints, and polyethylene terephthalat e (PET) resin, which is used to make plastic bottles for soft drinks and other downstream products [15].

Synthesis Of Ethylene Glycol

Naphtha to ethylene glycol (OEG), ethane to ethylene glycol (EEG), coal-based methanol to ethylene glycol (MEG), and coal-based syngas to ethylene glycol (CEG) are the primary feedstocks and production technological routes used to make ethylene glycols [16].

Nowadays, the majority of EG is made from fossil fuels through the hydration of ethylene oxide, with a tiny fraction coming from renewable resources through the dehydration of biobased ethanol. Additionally, a number of labs have thoroughly investigated the microbial synthesis of EG from plant-derived sugars, such as d-glucose and d-xylose. EG may now be produced selectively from CO2 using ethylene thanks to recent advancements in chemical and electrochemical methods. Additionally, syngas, lignocellulolytic biomass, and PET wastes are sources of EG that do not compete with human food supplies (Figure 2) [17].

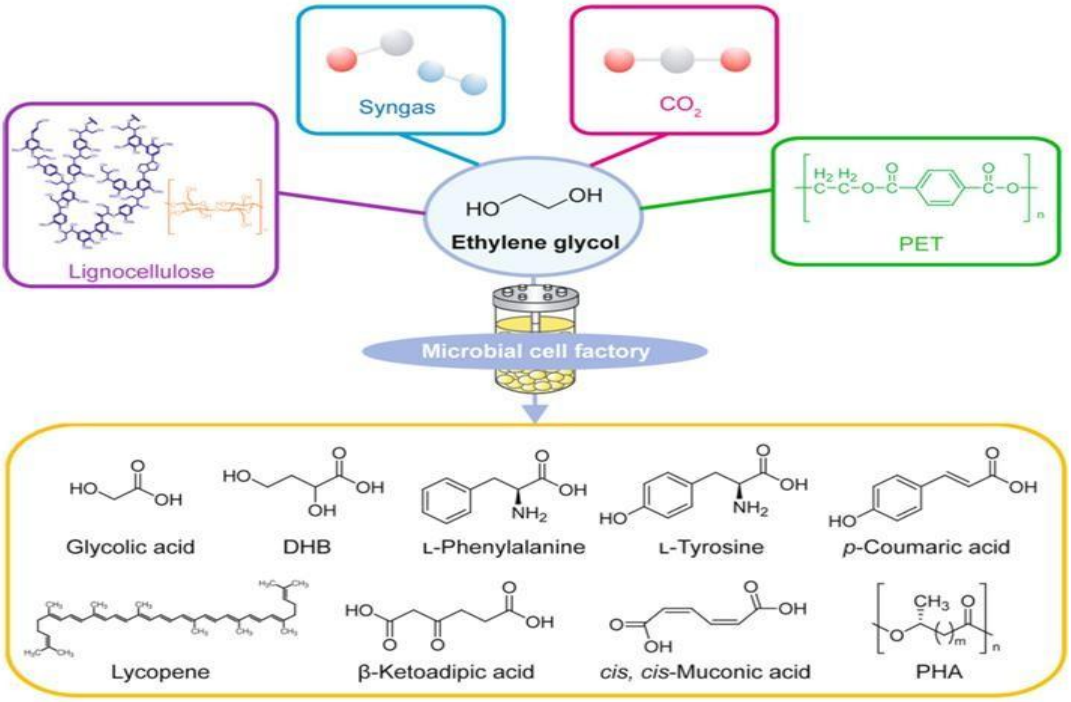

Figure: 2. Microbial Cell Factories For The Conversion of Ethylene Glycol To Value-Added Chemicals [17].

Toxicity Studies

There are several causes of acute renal failure in children. Numerous epidemics of severe renal failure, primarily affecting small newborns, have been linked to diethylene glycol (DEG) poisoning [18]. These children have displayed symptoms of gastrointestinal bleeding, seizures, liver failure, and renal failure. The poisoning is caused by either deliberate illegal use of DEG as a solvent or DEG contamination of pharmaceutical products. Over 300 children have died globally as a result of DEG poisoning [19]. The amount of DEG needed to cause morbidity and mortality in humans is unknown. Because they are mostly based on accounts from large poisoning epidemics, they might underestimate toxicity. The average estimated lethal dose for an adult is approximately 1 ml/kg of pure DEG [20].

Three of the twelve animals given DEG experienced AKI, as evidenced by elevated BUN and creatinine levels. those with AKI had higher levels of DGA in their brains and kidneys than those without AKI. Animals with AKI had a significantly higher total CSF protein level than both control and treated animals without AKI. Animals with AKI showed reductions in locomotor and rearing activity as well as forelimb grip strength when compared to control and non - AKI animals.

In a female animal, repeated DEG dosage resulted in nephrotoxic effects at a level comparable to those in males. Only females with AKI experienced a decline in motor performance and an increase in CSF protein. However, it is difficult to determine which consequence happens first because kidney and neurologic effects were only evaluated at the conclusion of the therapy. Global measurements of limb function and coordination were made, and more sensitive testing, like nerve conduction studies, may provide a thorough evaluation of the neurotoxicity of DEG's effects [21].

Clinical Features

The clinical effects of DEG poisoning can be divided into following three stages:

Preliminarily, the 1st phase consists of GIT symptoms such as nausea, vomiting, abdominal pain, and diarrhea. At this point, the patient may also have metabolic acidosis [22]. The 2nd phase started after 1 to 3 days DEG ingestion and is characterized by acute kidney injury (AKI), decreased urine output and increased anion gap metabolic acidosis. Liver injury has been reported in a few uncommon cases of elevated serum transaminase. Additionally, patients may experience hypertension and severe pancreatitis [23]. The 3rd phase starts after 1 to 2 weeks DEG ingestion if the patient survives the initial phases. Progressive and late neurological condition, defined by encephalopathy and polyneuropathies, such as bilateral facial nerve palsy, bulbar palsy, and widespread denervation of limb muscles, characterizes this phase [24]. Despite the description of this three-phase pattern, symptoms may overlap and be influenced by the amount consumed. The clinical manifestation of intoxication can also change when DEG is consumed in combination with other substances, such ethanol. In this instance, ethanol can postpone the development of symptoms for up to 48 hours by inhibiting DEG metabolism [25].

Mode of Action (MOA) – Renal Toxicity

Corley et al. (2005b) released the first evaluation of the MOA by which EG may cause kidney injury in people. In a nutshell, the MOA is thought to be as follows:

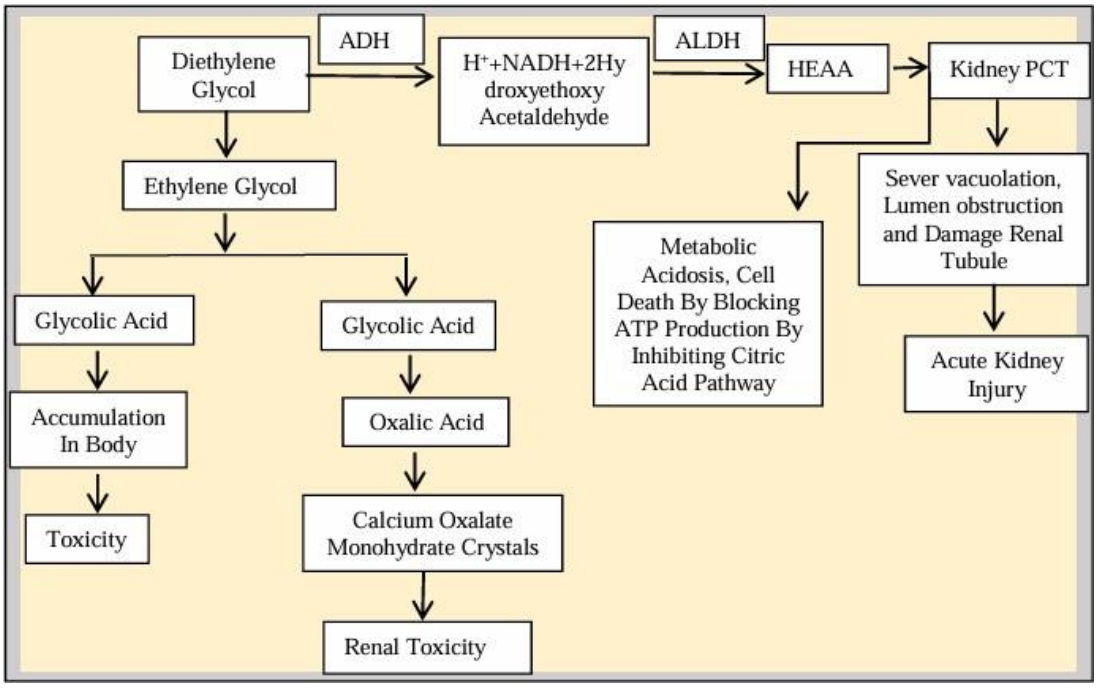

Figure 3. Mode of Action of Diethylene Glycol

ADH: Alcohol dehydrogenase, ALDH: Aldehyde dehydrogenase, HEAA: 2-Hydroxyethoxy acetic acid, PCT: Proximal Convulated Tubule

One of the metabolites of EG is oxalic acid, which can precipitate when it combines with calcium in the renal tubule epithelium. It is thought that renal tubule degeneration results from the calcium oxalate monohydrate crystals' ability to physically clog renal tubules and/or harm cells biochemically [26]. Renal damage from DEG toxicity is caused by proximal tubular necrosis. Severe vacuolation and epithelial cell enlargement cause the lumen to become obstructed. Kidney poisoning is caused by diglycolic acid and HEAA. After glomerular filtration, apical sodium dicarboxylate transporters-1 or organic anion transporters transfer diglycolic acid into the proximal tubular cells. There, it stops the production of adenosine triphosphate, which kills cells by inhibiting the citric acid cycle enzyme [27].

In Vivo Metabolism

There are two possible chemical routes for the metabolism of diethylene glycol dimethyl ether. Diethylene glycol dimethyl ether is primarily metabolized by O-demethylation following a single dosage, producing 2-(2-methoxyethoxy)ethanol [28]. Following repeated exposure to diethylene glycol dimethyl ether, enzymes are activated, boosting NADPH-dependent breakage of the central ether bond and producing two molecules of 2 -methoxy ethanol. Methoxyacetic acid is produced by oxidizing these. Male rats were given a single oral dose of diethylene glycol dimethyl ether; the predominant metabolite was found to be (2-methoxyethoxy)acetic acid, which accounted for 68% of the dose; the second metabolite, methoxyacetic acid, represented 6.2% of the dose. The same fraction of (2 -methoxyethoxy)acetic acid was found after multiple dosages, and 10% of it was eliminated as methoxyacetic acid (Figure 1) (Cheever et al. 1988) [29].

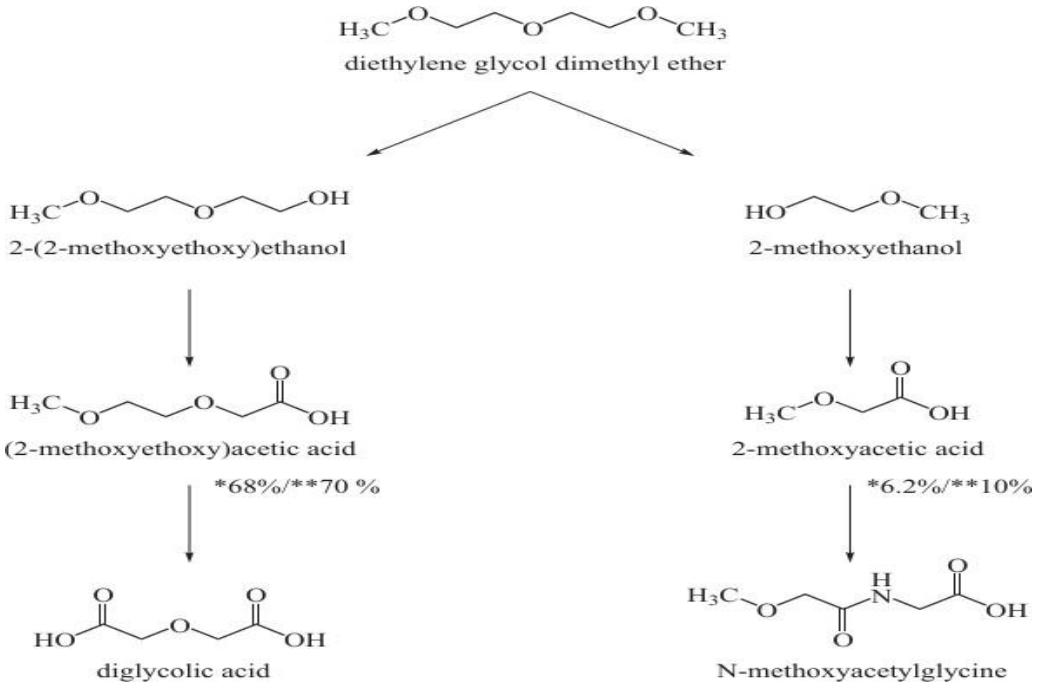

Figure: 1. Metabolism of Diethylene Glycol Methyl Ether in Male Rats After Oral Administration (According to Cheever et al. 1988) [29].

*% of the administered dose * after a single dose and ** after repeated dose*

Pharmacokinetics and Pathophysiology of Ethylene Glycol

In rodents, ethylene glycol has an oral bioavailability of 92–100% [30]. Intoxication symptoms and increased serum concentrations have been observed 20–30 minutes after intake, indicating that it is quickly absorbed from the human gastrointestinal tract [31, 32]. Its volume of distribution is somewhat small (0.5 –0.8 L/kg). Urine eliminates 20% of the parent molecule unaltered. Alcohol dehydrogenase converts the remaining 80% in the liver to glycoaldehyde, which is further converted by aldehyde dehydrogenase to a number of acid metabolites, including glycolic acid, glyoxylic acid, and oxalic acid. The anion gap metabolic acidosis, calcium oxalate crystals, and kidney damage associated with ethylene glycol poisoning are caused by these metabolites [33–38]. The only metabolite that significantly builds up in the blood is glycolic acid, which seems to be the main cause of the metabolic acidosis [36 –39]. Renal tubular cells are poisoned by glyoxylic acid, which is produced from glycolic acid [40].

Oxalic acid, which is produced when glyoxylic acid is metabolized, can interact with calcium to create calcium oxalate crystals, which can lead to hypocalcemia [31, 41–43]. Glyoxylic acid is converted by thiamine and pyridoxine to α-hydroxy-β-ketoadipic acid, glycine, and hippuric acid, which are thought to be less harmful than oxalate. The parent molecule has an elimination half-life of 3–8.5 hours [36,44,45]. Alcohol is the preferred substrate of alcohol dehydrogenase, while fomepizole (4-methylpyrazole) is an inhibitor. In patients with normal renal function, they will extend the first-order elimination half-life of ethylene glycol to roughly 17 hours [44, 45]. Seizures, coma, renal failure, and circulatory collapse are among the clinical signs of ethylene glycol poisoning [31,43,46]. Toxic intermediary metabolites, particularly aldehyde metabolites [34], glycolic acid, and to a lesser extent oxalate synthesis and excretion are the main cause of the central nervous system, metabolic, cardiac, and renal toxicities [33,47]. The first indications of intoxication, such as altered mental status, slurred speech, and ataxia, are believed to be caused by unaltered ethylene glycol [48]. Abdominal discomfort, nausea, and vomiting are common

gastrointestinal symptoms that appear shortly after consumption [46]. Infants may exhibit hypotonia, pallor, vomiting, lethargy, irritability, and poor feeding [49,50]. The prognosis is not good for a patient who has severe acidosis, hyperkalemia, coma, and seizures.

CONCLUSION

Serum ethylene glycol and, ideally, glycolic acid levels must be measured in order to make a definitive diagnosis and provide prompt, appropriate therapy for ethylene glycol poisoning, a very uncommon medical emergency. Despite the availability of precise, reliable, but technically demanding chromatographic techniques, few clinical laboratories now provide this testing since their analysis has historically been difficult. With the potential for severe morbidity and even death, ethylene glycol poisoning is still a major toxicological issue. The timing of intake affects the clinical presentation , and there are numerous differential diagnoses that need to be taken into account in addition to or instead of ethylene glycol poisoning. The main metabolites of ethylene glycol, not the parent substance, are responsible for serious toxicity

REFERENCES

1. Kumar P, Rastogi S, Saini PK, Sahoo S, Raghuvanshi RS, Jadaun GP. Minimizing the risk of ethylene glycol and diethylene glycol poisoning in medications: A regulatory and pharmacopoeial response. Regulatory Toxicology and Pharmacology. 2025 Jan 1;155:105741. https://doi.org/10.1016/j.yrtph.2024.105741 .
2. Altamimy MA, Alshehri YM, Aldawsari FS, Altalyan NH, AlShmmari SK, Alzaid SF, Al-Qahtani NA, Alsagoor TM, Al Shahrani MS. A Selective Gas Chromatography–Tandem Mass Spectrometry Method for Quantitation of Ethylene and Diethylene Glycol in Paediatric Syrups. Heliyon. 2024 Apr 15;10(7). https://doi.org/10.1016/j.heliyon.2024.e27559.
3. Wei JS, Ma SY, Cai YH, Xu CY, Liu JM, Jiang HT. A high-performance ethylene glycol sensor based on fibrous ErFeO3 prepared by electrostatic spinning. Ceramics International. 2023 Oct 15;49(20):32611-8. https://doi.org/10.1016/j.ceramint.2023.07.228 .
4. Jamison CN, Dayton RD, Latimer B, McKinney MP, Mitchell HG, McMartin KE. Diethylene glycol produces nephrotoxic and neurotoxic effects in female rats. Clinical toxicology. 2022 Mar 4;60(3):324 -31. https://doi.org/10.1080/15563650.2021.1953049 .
5.Schep LJ, Sla+ughter RJ, Temple WA, Beasley DM. Diethylene glycol poisoning. Clinical toxicology. 2009 Jul 1;47(6):525-35.
6.Murodov A, Xabibullaev S, Maxmudov N, Daminov T, Qarshiyev A. Creation of technology for desalination of saturated diethylene glycol substance in East Berdak gas complex preparation plant. InE3S Web of Conferences 2023 (Vol. 434, p. 02026). EDP Sciences. https://doi.org/10.1051/e3sconf/202343402026 .
7. Thakur S, Mandha M, Sharma S. Diethylene Glycol Toxicity In Children: Clinical Features, Mechanisms, And Global Outbreaks.
8. Jamison CN, Dayton RD, Latimer B, McKinney MP, Mitchell HG, McMartin KE. Neurotoxic effects of nephrotoxic compound diethylene glycol. Clinical Toxicology. 2021 Sep 2;59(9):810 -21.
9. Lechner JT, Neuer A, Bockmair V, Stierstorfer J, Klapötke TM. Crystal Structures of the Commonly Used Plasticizers Ethylene Glycol Dinitrate, Diethylene Glycol Dinitrate, and Triethylene Glycol Dinitrate— Characterization and Discussion of Their Structural Behavior. Crystal Growth & Design. 2022 Sep 7.
10. Duga AL, Fallah MP, Figueras A. Diethylene glycol: Unnoticed threat in the landscape of fixed-dose combination medications. Journal of Public Health in Africa. 2025 Apr 24;16(1):1271. 10.4102/jphia.v16i1.1271.
11. Hanif M, Mobarak MR, Ronan A, Rahman D, Donovan JJ, Bennish ML. Fatal renal failure caused by diethylene glycol in paracetamol elixir the Bangladesh epidemic. Bmj. 1995 Jul 8;311(6997):88 -91.
12. Naidu MN, Jakkan K, Sanjeeva P, Ramana PV. A Simple And Sensitive Validated Method For Quantitation Of Toxic Impurities-Ethylene Glycol And Diethylene Glycol In Pharmaceutical Ingredient-Propylene Glycol By Gas Chromatography. Frontiers in Health Informatics. 2025 Jan 1;14(1).
13. Arman BY, Legge I, Walsby-Tickle J, Bharucha T, Gabel J, Gnegel G, Deats M, Banerjee S, Stokes R, Merchant HA, Matousek P. Rapid screening of ethylene glycol and diethylene glycol in raw materials and medicinal syrups using low-cost field deployable assays. Scientific Reports. 2025 Dec 3;15(1):39737.
14. Rostovtseva V, Faykov I, Pulyalina A. A Review of Recent Developments of Pervaporation Membranes for Ethylene Glycol Purification. Membranes. 2022 Mar 10;12(3):312. https://doi.org/10.3390/membranes12030312 .
15. Shehata WM, Nady TG, Gad FK, Shoaib AM, Bhran AA. A comparative study of mono ethylene glycol economic production via different techniques. Scientific Reports. 2024 Nov 17;14(1):28375. https://doi.org/10.1038/s41598-024-77713-y.
16. Qian H, Song G, Lv L, Cheng Y, Zhang L, Chen J. The comparative life-cycle environmental effects of chemical feedstock change driven by energy system transition: A case study from China's ethylene glycol industry. Journal of Cleaner Production. 2022 Jun 25;355:131764. 10.1016/j.jclepro.2022.131764.

17. Shimizu T, Inui M. Novel aspects of ethylene glycol catabolism. Applied Microbiology and Biotechnology. 2024 Dec;108(1):369. https://doi.org/10.1007/s00253-024-13179-2.
18. Schier J, Chang A, Kapil V. Medication-associated diethylene glycol mass poisoning—a preventable cause of illness and death. The New England journal of medicine. 2023 Mar 2;388(13):1156. 10.1056/NEJMp2215840.
19. Alkahtani S, Sammons H, Choonara I. Epidemics of acute renal failure in children (diethylene glycol toxicity). Archives of disease in childhood. 2010 Dec 1;95(12):1062 -4.
20. Schep LJ, Slaughter RJ, Temple WA, Beasley DM. Diethylene glycol poisoning. Clinical toxicology. 2009 Jul 1;47(6):525-35.
21. Jamison CN, Dayton RD, Latimer B, McKinney MP, Mitchell HG, McMartin KE. Diethylene glycol produces nephrotoxic and neurotoxic effects in female rats. Clinical Toxicology. 2022 Mar 4;60(3):324 -31.
22. Unuma K, Isa S, Noritake K, Yamada A, Aki T, Tojo A. Ethylene glycol poisoning induces nephrolithiasis including complement-fibrinogen-related renal tubular injury in rats. Toxicology. 2026 Apr 15:154470. https://doi.org/10.1016/j.tox.2026.154470 .
23. Araujo SA, Faria BC, Vasconcelos JC, da Cruz AF, de Souza VS, Wanderley DC, e Silva AC. Renal Toxicity Caused by Diethylene Glycol: an Overview
24. Waheed N, Fakhrulddin A. Identification and quantification of ethylene glycol and diethylene glycol presented in pediatric cough and cold syrups in Iraqi local market. Journal of Research in Pharmacy. 2026 Nov 1;30(1):186 -97. https://doi.org/10.12991/jrespharm.1636170.
25. Snellings WM, Corley RA, McMartin KE, Kirman CR, Bobst SM. Oral Reference Dose for ethylene glycol based on oxalate crystal-induced renal tubule degeneration as the critical effect. Regulatory Toxicology and Pharmacology. 2013 Mar 1;65(2):229-41.
26. Aronson JK. When I use a word... Medicines regulation—diethylene glycol. bmj. 2024 Feb 9;384. https://doi.org/10.1136/bmj.q356.
27. Gopalakrishnan N, Kamarajan M, Balasubramaniyan T, Sakthirajan R, Dhanapriya J, Dineshkumar T. Diethylene glycol poisoning-induced acute kidney injury. Saudi Journal of Kidney Diseases and Transplantation. 2016 Nov 1;27(6):1276.
28. Kelsey JR, Cnubben NH, Bogaards JJ, Braakman RB, van Stee LL, Smet K. The urinary metabolic profile of diethylene glycol methyl ether and triethylene glycol methyl ether in Sprague-Dawley rats and the role of the metabolite methoxyacetic acid in their toxicity. Regulatory Toxicology and Pharmacology. 2020 Feb 1;110:104512. https://doi.org/10.1016/j.yrtph.2019.104512 .
29. Hartwig A, Arand M, MAK Commission. Diethylene glycol dimethyl ether. The MAK Collection for Occupational Health and Safety. 2023;8(4): Doc078. https://doi.org/10.34865/mb11196e8_4ad.
30. Frantz SW, Beskitt JL, Grosse CM, Tallant MJ, Dietz FK, Ballantyne B.Pharmacokinetics of ethylene glycol. I. Plasma disposition after singleintravenous, peroral, or percutaneous doses in female Sprague-Dawleyrats and CD-1 mice. Drug Metab Dispos 1996; 24:911 – 921.
31. Berman LB, Schreiner GE, Feys J. The nephrotic lesion of ethyleneglycol. Ann Intern Med 1957; 46:611 – 619.
32. Boyer EW, Mejia M, Woolf A, Shannon M. Severe ethylene glycolingestion treated without hemodialysis. Pediatrics 2001; 107:172–173.
33. Pons CA, Custer RP. Acute ethylene glycol poisoning: a clinico-pathologic report of eighteen fatal cases. Am J Med Sci 1946; 211:544 –552.
34. Parry MF, Wallach R. Ethylene glycol poisoning. Am J Med 1974;57:143 –150.
35. Gabow PA, Clay K, Sullivan JB, Lepoff R. Organic acids in ethyleneglycol intoxication. Ann Intern Med 1986; 105:16 – 20.
36. Jacobsen D, Hewlett TP, Webb R, Brown ST, McMartin KE. Ethyleneglycol intoxication: evaluation of kinetics and crystalluria. Am J Med1988; 84:145 – 152.
37. Moreau CL, Kerns W II, Tomaszewski CA, McMartin KE, Rose SR,Ford MD, Brent J. Glycolate kinetics and hemodialysis clearance inethylene glycol poisoning. META Study Group. J Toxicol ClinToxicol 1998; 36:659 – 666.
38. Porter WH, Rutter PW, Bush BA, Pappas AA, Dunnington JE. Ethyleneglycol toxicity: the role of serum glycolic acid in hemodialysis. J ToxicolClin Toxicol 2001; 39:607 – 615.
39. Jacobsen D, Ovrebo S, Ostborg J, Sejersted OM. Glycolate causes theacidosis in ethylene glycol poisoning and is effectively removed byhemodialysis. Acta Med Scand 1984; 216:409 – 416.
40. Poldelski V, Johnson A, Wright S, Rosa VD, Zager RA. Ethylene glycol-mediated tubular injury: identification of critical metabolites and injurypathways. Am J Kidney Dis 2001; 38:339 – 348.
41. Karlson-Stiber C, Persson H. Ethylene glycol poisoning: experiencesfrom an epidemic in Sweden. J Toxicol Clin Toxicol 1992; 30:565 – 574.
42. Jacobsen D, McMartin KE. Antidotes for methanol and ethylene glycolpoisoning. J Toxicol Clin Toxicol 1997; 35:127 – 143.
43. Barceloux DG, Krenzelok EP, Olson K, Watson W. American Academyof Clinical Toxicology practice guidelines on

the treatment of ethyleneglycol poisoning. Ad hoc Committee. J Toxicol Clin Toxicol 1999;37:537 – 560.
44. Peterson CD, Collins AJ, Himes JM, Bullock ML, Keane WF. Ethyleneglycol poisoning: pharmacokinetics during therapy with ethanol andhemodialysis. N Engl J Med 1981; 304:21 – 23.
45. Sivilotti ML, Burns MJ, McMartin KE, Brent J. Toxicokinetics ofethylene glycol during fomepizole therapy: implications for manage-ment. For the Methylpyrazole for Toxic Alcohols Study Group. AnnEmerg Med 2000; 36:114 – 125.
46. Moriarty RW, McDonald RH Jr. The spectrum of ethylene glycolpoisoning. Clin Toxicol 1974; 7:583 – 596.
47. Clay KL, Murphy RC. On the metabolic acidosis of ethylene glycolintoxication. Toxicol Appl Pharmacol 1977; 39:39 – 49.
48. Levy RI. Renal failure secondary to ethylene glycol intoxication. JAMA1960; 173:1210 – 1213.
49. Saladino R, Shannon M. Accidental and intentional poisonings withethylene glycol in infancy: diagnostic clues and management. PediatrEmerg Care 1991; 7:93 – 96.
50. Woolf AD, Wynshaw-Boris A, Rinaldo P, Levy HL. Intentional infantileethylene glycol poisoning presenting as an inherited metabolic disorder. J Pediatr 1992; 120:414 – 421.