



Implications of unexpectedly detectable methanol in patients presenting to an urban emergency department

Solana R. Archuleta, Jon B. Cole, Anita P. Wong & Travis D. Olives

To cite this article: Solana R. Archuleta, Jon B. Cole, Anita P. Wong & Travis D. Olives (19 Nov 2025): Implications of unexpectedly detectable methanol in patients presenting to an urban emergency department, Clinical Toxicology, DOI: [10.1080/15563650.2025.2563144](https://doi.org/10.1080/15563650.2025.2563144)

To link to this article: <https://doi.org/10.1080/15563650.2025.2563144>



Published online: 19 Nov 2025.



Submit your article to this journal [↗](#)



Article views: 31



View related articles [↗](#)



View Crossmark data [↗](#)

CLINICAL RESEARCH



Implications of unexpectedly detectable methanol in patients presenting to an urban emergency department

Solana R. Archuleta^a , Jon B. Cole^{a,b} , Anita P. Wong^c and Travis D. Olives^b 

^aDepartment of Emergency Medicine, Hennepin Healthcare, Minneapolis, MN, USA; ^bMinnesota Regional Poison Center, Hennepin Healthcare, Minneapolis, MN, USA; ^cDepartment of Pathology & Laboratory Services, Hennepin Healthcare, Minneapolis, MN, USA

ABSTRACT

Introduction: Methanol is ubiquitous, and early intoxication mimics other conditions. The prevalence of occult methanol intoxication is unknown. The goal of this study is to determine the frequency of unexpectedly positive methanol concentrations in patients at an urban safety net hospital with real-time testing capabilities, and to evaluate the clinical significance of these findings.

Methods: This was a retrospective cohort study of patients with detectable methanol concentrations presenting to an emergency department from 2015 to 2022. Dual column, dual flame ionization detection utilizing headspace sampling with gas chromatography is available 24 h/day. Ethanol and methanol concentrations result in tandem when either test is performed. For safety reasons, a positive methanol result triggers a reflex order to report the information in the electronic medical record. We reported descriptive statistics for cases of detectable methanol concentrations and compared expected and unexpected positive concentrations.

Results: Orders for 20,666 ethanol and 1,868 methanol concentrations were identified; methanol results were positive in 101 (6%) cases. Twenty-eight represented repeated testing during one patient's hospitalization. The median methanol concentration among the remaining 73 patients (30.1% female) was 60 mg/L (IQR: 50–70 mg/L; range 50–5,000 mg/L). There were 26 expected (median methanol concentration 80 mg/L [IQR: 60–290 mg/L]) and 47 unexpected (median methanol concentration 50 mg/L [IQR: 50–60 mg/L]) positive methanol assays during the study period (Mann–Whitney U-test, $P < 0.001$). No patients with incidentally positive methanol required directed therapies.

Discussion: Unexpectedly positive methanol concentrations in this study population were uncommon and without clinical significance, in that none required specific treatments or directed therapies.

Conclusions: Our data suggest that history and clinical suspicion are likely sufficient to detect clinically important methanol exposures. Occult methanol exposures of clinical significance appear unlikely when clinical suspicion is low.

ARTICLE HISTORY

Received 10 June 2025
Revised 2 September 2025
Accepted 12 September 2025

KEYWORDS

Ethanol; laboratory; methanol; poisoning; test availability

Introduction

Methanol is a volatile alcohol with a well-defined toxic profile in humans [1,2]. It is available in household, industrial, and clandestinely produced products [3–7] and is associated with significant morbidity and mortality if left untreated [8–10]. The most common route of poisoning is ingestion, although inhalational and dermal exposures are described [2,10,11]. Diagnosis requires a high degree of clinical suspicion, as concomitant intoxication with other substances may mask or mimic symptoms and findings [12–14], and methanol concentrations are infrequently available in real

time. Clinically significant methanol poisonings may thus be missed or diagnosed in a delayed fashion [15,16], increasing the risk of morbidity and mortality.

Although level A consensus recommendations for ethanol concentration availability within one hour are published [17], equivalent expediency has not been specifically recommended for methanol testing. Few hospitals maintain the capacity for real-time, onsite methanol testing, forcing clinicians to rely on surrogate laboratory and physical findings such as abnormalities in the osmol gap and anion gap, the development of a metabolic acidosis, and visual disturbances to diagnose methanol poisoning [18–20]. Still

other laboratory and clinical efforts to diagnose methanol and other toxic alcohol poisonings have met with less success [21,22]. The goal of this study is to assess the frequency of unexpectedly positive methanol concentrations in patients who have blood ethanol concentrations measured at a hospital with real-time testing capabilities, and to evaluate the clinical significance of these results compared to expected positive methanol concentrations.

Methods

This is a retrospective study of patients presenting to an urban safety net hospital from 2015 to 2022 with detectable methanol concentrations. We evaluated all adults for whom a volatile alcohol (ethanol or methanol) screen was reported. No additional exclusion criteria specific to unique patient populations were applied; repeated methanol concentrations from the same patient, taken during the same episode of care, were excluded. The local institutional review board approved this study.

Volatile alcohol laboratory results at the study site are available in real-time, 24 h/day (typical turn-around time: 120 min) via dual column, dual flame ionization detection utilizing headspace sampling with gas chromatography (Agilent GC 6890N with Agilent headspace analyzer 7697A, Santa Clara, CA). When a positive methanol analysis is detected in a sample analyzed solely for ethanol, a reflex methanol concentration order is placed to avoid overlooking occult methanol poisoning and subsequently populated in the patient's chart.

We queried the laboratory information system for all blood ethanol and methanol tests between 2015 and 2022 and further identified cases as unexpected positive or expected positive methanol results. We characterized a positive methanol concentration as expected if ordered by clinicians, and as unexpected if initiated reflexively within the laboratory from a clinician-ordered ethanol concentration. A methanol exposure was considered to be clinically significant if it was deemed to require directed therapies, including the administration of alcohol dehydrogenase inhibitors and/or leucovorin (folinic acid), or hemodialysis. Such therapies (alcohol dehydrogenase inhibitors, leucovorin) are generally recommended at serum methanol concentrations of >200 mg/L (6.24 mmol/L), or if there is suspicion of ingestion and there is an elevated anion gap, osmol gap, metabolic acidosis, or visual disturbance [23,24], while hemodialysis is recommended for cases of severe methanol poisoning [25]. Descriptive statistics describing methanol concentrations and

clinically significant data are reported. The Mann–Whitney U-test was applied to continuous variables as appropriate for comparison due to non-normal distribution of data.

Results

During the study period, 20,666 ethanol orders and 1,868 methanol orders were placed for patients in the hospital setting. Of these, 104 methanol results (6%) were positive. We excluded 28 representing repeated testing of individual patients during a single hospitalization, two that were erroneously reported positive initially but were later corrected, and one which was initially coded incorrectly. Seventy-three subjects thus remained for analysis (Table 1).

Among the remaining 73 results (30.1% female), the median methanol concentration was 60 mg/L (IQR: 50–70 mg/L; range 50–5,000 mg/L) [1.87 mmol/L; IQR: 1.56–2.18 mmol/L; range 1.56–156 mmol/L]. There were 26 expected positive methanol assays with a median methanol concentration of 80 mg/L (IQR: 60–290 mg/L; range 50–5,000 mg/L) [2.5 mmol/L; IQR: 1.87–9.05 mmol/L; range 1.56–156 mmol/L] and 47 unexpected positive methanol assays with a median methanol concentration of 50 mg/L (IQR: 50–60 mg/L; range 50–100 mg/L) [1.56 mmol/L; IQR: 1.56–1.87 mmol/L; range 1.56–3.12 mmol/L; $P < 0.001$]. The median ethanol concentration in the expectedly positive methanol group ($n = 18$) was 215 mg/L (IQR: 0–1,870 mg/L; range 0–3,320 mg/L) [4.67 mmol/L; IQR: 0–40.59 mmol/L; range 0–72.08 mmol/L], and in the unexpectedly positive methanol group ($n = 47$), the median ethanol concentration was 2,140 mg/L (IQR:

Table 1. Demographics of 73 patients with detectable methanol concentrations.

	Expected positive methanol ($n = 26$)	Unexpected positive methanol ($n = 47$)
Sex		
Male	19	34
Female	7	13
Age (years) (range)	41 (10–65)	48 (24–78)
Intention in expected positive group ^a		
Unintentional	7	
Intentional suicide attempt	5	
Source of methanol in expected positive group ^a		
Windshield wiper fluid	10	
Antifreeze	3	
Reason for ordering methanol concentration ^a		
Reported ingestion	13	Orders generated reflexively from hospital toxicology laboratory
Suicide attempt with other ingestion	1	
Drank denatured alcohol	1	
History of positive concentration	4	
Unknown/not documented	2	

^aIf known.

640–3,140 mg/L; range 0–4,780 mg/L) [46.4 mmol/L; IQR: 13.39–68.17 mmol/L; range 0–103.77 mmol/L]. The results are presented in Table 2.

Ten patients received directed treatment for methanol poisoning. All 10 patients received fomepizole, of whom five also received leucovorin, and three also underwent hemodialysis. All were known ingestions with expected positive methanol concentrations. For the remaining 16 patients with expected positive methanol concentrations, treatment was not clinically indicated. No patient with unexpectedly positive methanol concentrations received directed therapies as the identified methanol concentrations were deemed not clinically relevant by bedside providers based on medical history and ancillary laboratory testing (Table 2). There was no significant difference in patients with unexpectedly versus expectedly positive methanol assays in initially measured respiratory rate, anion gap, venous pH, bicarbonate concentration, or serum osmolality (Table 3).

Two deaths were identified, both in patients with unexpectedly positive methanol concentrations. The first presented with severe full-body, full-thickness burns resulting in death. The initial methanol concentration was 70 mg/L (2.19 mmol/L), the anion gap was 20, the serum lactate concentration was 13.8 mmol/L, the serum osmolality was 290 mOsm/kg (osmol gap 1 mOsm), and no ethanol was detectable. Repeated

methanol testing 9 h later was negative. The regional poison center was consulted, and it was determined that the identified methanol was unrelated to the presentation for severe, ultimately fatal, burns.

The second fatality presented with mixed ethanol and hypertriglyceridemia-induced pancreatitis and resultant abdominal compartment syndrome with delayed abdominal sepsis and shock liver, to which the patient eventually succumbed on hospital day 15. The initial methanol concentration was 50 mg/L (1.56 mmol/L), the anion gap 13, and the serum lactate concentration was >12 mmol/L; the serum osmolality was not evaluated. Serum triglycerides were 11,133 mg/dL (125.58 mmol/L), and serum lipase activity was 4,220 IU/L. Formate concentrations were not sent on either patient, though neither case was adjudicated as related to methanol toxicity.

Discussion

In the United States, both intentional and unintentional methanol exposures remain an important source of toxic morbidity and mortality [6,8]. Assessment of the potentially methanol-intoxicated patient includes laboratory evaluation to detect exposure. However, the availability of serum methanol testing across hospitals is poorly defined, and few have access to such testing in real time [26]. Thus, clinical suspicion and ancillary

Table 2. Characteristics of 73 patients with detectable methanol concentrations.

	Expected positive methanol (n = 26)		Unexpected positive methanol (n = 47)		P value
	n	Median (IQR)	n	Median (IQR)	
Methanol concentration (mg/L)	26	80 (60–290)	47	50 (50–60)	<0.001
Methanol concentration (mmol/L)		2.5 (1.87–9.05)		1.56 (1.56–1.87)	
Ethanol concentration (mg/L)	18	215 (0–1,870)	47	2,140 (640–3,140)	0.004
Ethanol concentration (mmol/L)		4.67 (0–40.59)		46.4 (13.89–68.17)	
Methanol concentration in patients treated for methanol poisoning (mg/L) ^a	10	345 (260–980)	0	–	–
Methanol concentration in patients treated for methanol poisoning (mmol/L)		10.8 (8.12–30.58)			

^aTreatments included fomepizole or leucovorin.

Table 3. Metabolic profiles of 73 patients with detectable methanol concentrations^a.

	Expected positive methanol (n = 26)		Unexpected positive methanol (n = 47)		P value
	n	Median (IQR)	n	Median (IQR)	
Initial respiratory rate (breaths/min)	24	18 (16–19)	45	18 (18–22)	0.49
Venous pH	5	7.42 (7.42–7.49)	15	7.41 (7.34–7.43)	0.17
Anion gap (mmol/L)	24	12.5 (11–17.5)	47	14 (11–16)	0.56
Serum bicarbonate concentration (mEq/L)	24	22.5 (16.5–25.5)	45	24 (20–25)	0.5
Serum osmolality (mmol/L)	7	313 (303–432)	3	296 (290–340)	0.21

^aIndividual counts (n) represent the total number of subjects for whom a given laboratory was ordered and resulted, or an initial respiratory rate was documented. For example, 24 of 26 subjects with expected positive methanol concentrations also had initial respiratory rates reported, as did 45 of 47 subjects with unexpectedly positive methanol concentrations.

testing, including serial metabolic blood chemistry and blood gas analyses, as well as assessments of developing visual complaints, are commonly employed to diagnose and to exclude significant methanol poisoning. When exposure to methanol is not suspected, the risk of clinically significant occult methanol exposures remains unclear. These data suggest that when methanol exposure is not suspected, incidentally detected, unexpectedly positive methanol concentrations are unlikely to be clinically significant or to warrant directed therapies.

Among the 10 patients in our study who received directed therapies, all had clinical histories that were clearly consistent with methanol ingestion at the time of presentation to the emergency department. All were among the 26 subjects with expectedly positive methanol concentrations. Five cases were reported as suicide attempts, and five as unintentional ingestions. In some instances, treatments were not based solely on methanol concentrations. For example, one patient received leucovorin after an elevated anion gap was detected in the context of ethanol intoxication (blood ethanol concentration 1,090 mg/L [23.7 mmol/L]), but the methanol concentration was only slightly above detectable concentrations, and a repeat test was negative. It is unclear why leucovorin, which was not clinically indicated, was administered in this case. Fomepizole was administered to another patient before they were transferred from a hospital lacking real-time methanol testing capabilities. Patients with unexpectedly positive methanol concentrations were determined not to require directed therapies by bedside providers, and thus received none. Specifically, metabolic parameters in the context of very low methanol concentrations did not warrant directed therapies, while those with metabolic derangements had clear alternate etiologies felt highly likely to be responsible for these derangements (e.g., low venous pH attributed to metabolic acidosis with hyperlactatemia in the setting of gastrointestinal bleed related to severe ethanol use disorder and withdrawal).

In this population of patients with unexpectedly detectable methanol concentrations, the origin of methanol exposure is unknown. It is likely that a portion of the unexpectedly positive results originate from the ingestion of commercially available distilled spirits and other fermented beverages, which may contain 300–3,000 mg methanol/L anhydrous ethanol [7,27,28]. For example, one study subject with multiple positive methanol results previously identified tequila as their only source of consumed ethanol [29]. Assuming an average serving size of 1.5 oz (44.36 mL) of distilled spirits, ingesting one serving of tequila

containing the maximal allowable methanol would be expected to result in a blood concentration that is detectable yet far below accepted toxic thresholds, even in a 10 kg child [24,30]. Detectable methanol concentrations might be expected from vigorous consumers of other commercial sources of ethanol, as well [28].

Evidence points to endogenous methanol production as a potential source of low-level methanol concentrations in humans [31], particularly in the context of concomitant ethanol administration [32]. This is presumably due to the accumulation of endogenously produced methanol in the presence of a preferred alcohol dehydrogenase substrate. Endogenously produced methanol has generally been felt to result in only low concentrations of detectable methanol, with average reported concentrations in humans of less than 5–10 mg/L (0.16–0.31 mmol/L) [33]. Other endogenous sources of methanol production are known, including the production of methanol via metabolism of excessive aspartame [34], as well as through the ingestion of foods containing pectin [35]. Given the very low reported blood concentrations of methanol in these cases, it seems unlikely that this mechanism was chiefly responsible for the detection of methanol in the cases we report, given that the median methanol concentration of 50 mg/L (range 50–100 mg/L) [1.56 mmol/L (range 1.56–3.12 mmol/L)] is well above those reported in this body of literature.

This study has several significant limitations that must be addressed. Our site is unique in that toxic alcohol testing is readily available, with results generally returned within 1–2 h. As most hospitals lack the capacity for real-time methanol testing, our results may not be generalizable. Additionally, it is plausible that very low but unexpectedly detectable methanol results arose from late testing of patients with earlier methanol ingestions, leading to a delay in diagnosis of clinically significant methanol toxicity. However, blood gas results from our study cohort (shown in Table 3) do not support this alternative hypothesis of late presentation, as more severe metabolic derangements, including acidosis, would be anticipated if this were the case. Additionally, the retrospective nature of this study is reflected in the incomplete laboratory data available on each included subject. Not all patients with expected positive methanol concentrations had concurrent ethanol concentrations assessed, and incomplete metabolic data similarly reflects provider ordering variations on a patient by patient basis. Similarly, as a retrospective study, the origin of methanol detected in the biological samples of these patients cannot be confirmed. Finally,

while this study speaks to the clinical insignificance of unexpectedly positive methanol concentrations, it does not address the same question with respect to other toxic alcohols, including ethylene glycol.

Conclusions

Obtaining an unexpectedly positive methanol concentration in this study population was rare, occurring in just 47 of 20,666 samples (0.23%) for which serum ethanol samples were resulted and accounting for only 2.5% of all resulted serum methanol concentrations over the same period. Unexpectedly positive serum methanol concentrations identified in this study population were not clinically significant and required no directed therapies based on bedside clinician assessments of methanol concentrations, metabolic parameters, and clinical contexts. None were >100 mg/L (3.13 mmol/L). Our data suggest that patient history and clinical suspicion are likely sufficient to detect clinically important methanol exposures, and that failing to identify occult incidental methanol exposures is unlikely to result in adverse outcomes. The data further suggest that testing for methanol exposure without clinical suspicion is unlikely to identify cases of occult methanol poisoning.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

The authors reported there is no funding associated with the work featured in this article.

ORCID

Solana R. Archuleta  <http://orcid.org/0000-0003-3264-4444>
Jon B. Cole  <http://orcid.org/0000-0002-7714-8826>
Travis D. Olives  <http://orcid.org/0000-0003-4330-1391>

Data availability statement

The deidentified data we analyzed are not publicly available, but requests to the corresponding author (TO) for the data will be considered on a case-by-case basis.

References

- [1] Kraut JA, Mullins ME. Toxic alcohols. *N Engl J Med*. 2018;378(3):270–280. doi:10.1056/NEJMr1615295.
- [2] Bennett IL, Cary FH, Mitchell GL, et al. Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. *Medicine*. 1953;32(4):431–463. doi:10.1097/00005792-195312000-00002.
- [3] Holstege CP, Ferguson JD, Wolf CE, et al. Analysis of moonshine for contaminants. *J Toxicol Clin Toxicol*. 2004;42(5):597–601. doi:10.1081/clt-200026976.
- [4] Minns AB, McIlvoy A, Clark A, et al. Examining the risk of methanol poisoning from methyl acetate-containing products. *Am J Emerg Med*. 2013;31(6):964–966. doi:10.1016/j.ajem.2013.02.038.
- [5] Konkel K, Burkhart K, Cheng C, et al. Methanol poisonings from contaminated hand sanitizers identified by the United States Food and Drug Administration. *Clin Toxicol*. 2023;61(12):1065–1067. doi:10.1080/15563650.2023.2288809.
- [6] Givens M, Kalbfleisch K, Bryson S. Comparison of methanol exposure routes reported to Texas Poison Control Centers. *West J Emerg Med*. 2008;9(3):150–153.
- [7] Lachenmeier DW, Sohnius E-M, Attig R, et al. Quantification of selected volatile constituents and anions in Mexican Agave spirits (Tequila, Mezcal, Sotol, Bacanora). *J Agric Food Chem*. 2006;54(11):3911–3915. doi:10.1021/jf060094h.
- [8] Yip L, Bixler D, Brooks DE, et al. Serious adverse health events, including death, associated with ingesting alcohol-based hand sanitizers containing methanol – Arizona and New Mexico, May–June 2020. *MMWR Morb Mortal Wkly Rep*. 2020;69(32):1070–1073. doi:10.15585/mmwr.mm6932e1.
- [9] Paasma R, Hovda KE, Tikkerberi A, et al. Methanol mass poisoning in Estonia: outbreak in 154 patients. *Clin Toxicol*. 2007;45(2):152–157. doi:10.1080/15563650600956329.
- [10] Rostrop M, Edwards JK, Abukalish M, et al. The methanol poisoning outbreaks in Libya 2013 and Kenya 2014. *PLOS One*. 2016;11(3):e0152676. doi:10.1371/journal.pone.0152676.
- [11] Ogawa Y, Takatsuki R, Uema T, et al. Acute optic neuropathy induced by thinner sniffing: inhalation of mixed organic solvent containing methyl alcohol and methyl acetate. *Ind Health*. 1988;26(4):239–244. doi:10.2486/indhealth.26.239.
- [12] Cohen ET, Su MK, Biary R, et al. Distinguishing between toxic alcohol ingestion vs alcoholic ketoacidosis: how can we tell the difference? *Clin Toxicol*. 2021;59(8):715–720.
- [13] Rastogi A, Itagaki B, Bajwa M, et al. Spurious elevation in serum creatinine caused by ingestion of nitromethane: implication for the diagnosis and treatment of methanol intoxication. *Am J Kidney Dis*. 2008;52(1):181–187. doi:10.1053/j.ajkd.2007.12.010.
- [14] Hegde AN, Mohan S, Lath N, et al. Differential diagnosis for bilateral abnormalities of the basal ganglia and thalamus. *Radiographics*. 2011;31(1):5–30. doi:10.1148/rg.311105041.
- [15] Hovda KE, Hunderi OH, Tafjord A-B, et al. Methanol outbreak in Norway 2002–2004: epidemiology, clinical features and prognostic signs. *J Intern Med*. 2005;258(2):181–190. doi:10.1111/j.1365-2796.2005.01521.x.
- [16] Birungi D, Eyu P, Okethwangu D, et al. Fatal methanol poisoning caused by drinking adulterated locally distilled alcohol: Wakiso District, Uganda. *J Environ Public Health*. 2020;58:5816162.
- [17] Wu AHB, McKay C, Broussard LA, et al. National academy of clinical biochemistry laboratory medicine practice guidelines: recommendations for the use of laboratory tests to support poisoned patients who present to

- the emergency department. *Clin Chem*. 2003;49(3):357–379. doi:10.1373/49.3.357.
- [18] Lynd LD, Richardson KJ, Pursell RA, et al. An evaluation of the osmole gap as a screening test for toxic alcohol poisoning. *BMC Emerg Med*. 2008;8(1):5. doi:10.1186/1471-227X-8-5.
- [19] Krahn J, Khajuria A. Osmolality gaps: diagnostic accuracy and long-term variability. *Clin Chem*. 2006;52(4):737–739. doi:10.1373/clinchem.2005.057695.
- [20] Krasowski MD, Wilcoxon RM, Miron J. A retrospective analysis of glycol and toxic alcohol ingestion: utility of anion and osmolal gaps. *BMC Clin Pathol*. 2012;12(1):1. (doi:10.1186/1472-6890-12-1.
- [21] Parsa T, Cunningham SJ, Wall SP, et al. The usefulness of urine fluorescence for suspected antifreeze ingestion in children. *Am J Emerg Med*. 2005;23(6):787–792. doi:10.1016/j.ajem.2005.02.051.
- [22] Shin JM, Sachs G, Kraut JA. Simple diagnostic tests to detect toxic alcohol intoxications. *Transl Res*. 2008;152(4):194–201. doi:10.1016/j.trsl.2008.07.002.
- [23] Barceloux DG, Bond GR, Krenzelok EP, et al. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. *J Toxicol Clin Toxicol*. 2002;40(4):415–446. doi:10.1081/clt-120006745.
- [24] Weiner SW, Nelson LS, Howland M, et al. Chapter 106: toxic alcohols. In: Goldfrank's toxicologic emergencies. 11th ed. McGraw-Hill Education; 2019.
- [25] Roberts DM, Yates C, Megarbane B, et al. Recommendations for the role of extracorporeal treatments in the management of acute methanol poisoning: a systematic review and consensus statement. *Crit Care Med*. 2015;43(2):461–472.
- [26] Church AS, Witting MD. Laboratory testing in ethanol, methanol, ethylene glycol, and isopropanol toxicities. *J Emerg Med*. 1997;15(5):687–692. doi:10.1016/s0736-4679(97)00150-9.
- [27] NORMA Oficial Mexicana NOM-006-SCFI-2012, Bebidas alcohólicas-Tequila-Especificaciones [Internet]. NOM-006-SCFI-2012 12;13; 2012. Available from: https://www.dof.gob.mx/nota_detalle.php?codigo=5282165&fecha=13/12/2012
- [28] Bindler F, Voges E, Laugel P. The problem of methanol concentration admissible in distilled fruit spirits. *Food Addit Contam*. 1988;5(3):343–351. doi:10.1080/02652038809373713.
- [29] Olives TD, Batdorff E, Love S. Tales of tequila tails: chronically elevated methanol levels in a chronic user of tequila. North American Congress of Clinical Toxicology (NACCT) Abstracts 2021. *Clin Toxicol*. 2021;59:1059–1060.
- [30] Kostic MA, Dart RC. Rethinking the toxic methanol level. *J Toxicol Clin Toxicol*. 2003;41(6):793–800. doi:10.1081/clt-120025344.
- [31] Jones AW. Clinical and forensic toxicology of methanol. *Forensic Sci Rev*. 2021;33(2):117–143.
- [32] Jones AW, Skagerberg S, Yonekura T, et al. Metabolic interaction between endogenous methanol and exogenous ethanol studied in human volunteers by analysis of breath. *Pharmacol Toxicol*. 1990;66(1):62–65. doi:10.1111/j.1600-0773.1990.tb00704.x.
- [33] Sarkola T, Eriksson CJP. Effect of 4-methylpyrazole on endogenous plasma ethanol and methanol levels in humans. *Alcohol Clin Exp Res*. 2001;25(4):513–516.
- [34] Stegink LD, Brummel MC, Filer LJJr., et al. Blood methanol concentrations in one-year-old infants administered graded doses of aspartame. *J Nutr*. 1983;113(8):1600–1606. doi:10.1093/jn/113.8.1600.
- [35] Casey JC, Self R, Swain T. Origin of methanol and dimethyl sulphide from cooked foods. *Nature*. 1963;200(4909):885–885. doi:10.1038/200885a0.