REVIEW ARTICLE / PREGLEDNI ČLANAK

DRINK SPIKING AND RELATED EMERGENCY DEPARTMENT VISITS: A NARRATIVE REVIEW

PODMETANJE TVARI U PIĆE I POVEZANI DOLASCI U HITNI BOLNIČKI PRIJAM: PREGLEDNI RAD

***Jasmin Hamzić[1], Bojana Radulović[1], Arnes Rešić[2,3], Andrijana Ščavnićar[4], Mila Lovrić[4], Lidija Vugrinec[5], Ivan Gornik[1]**

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Abstract

Background: Drink spiking the covert administration of psychoactive agents into an unsuspecting person's drink or food, remains a persistent public health and forensic challenge. Victims frequently present to emergency departments (EDs) with sudden central nervous system depression, anterograde amnesia, and physiologic abnormalities that complicate timely recognition.

Objective: To synthesise current evidence relevant to frontline emergency clinicians on the pharmacology, clinical recognition, toxicology, specimen handling, and acute management of drinkspiking presentations, and to propose ED-focused system measures to improve detection, patient care, and forensic preservation.

Methods: This narrative review integrates peer-reviewed literature, poison centre registry reports, forensic guidance, and recent reviews addressing commonly implicated agents. Emphasis was placed on pharmacokinetic, toxicodynamic features that determine detection windows, bedside recognition clues, prioritised ED investigations, specimenhandling imperatives, and agent-specific management strategies.

Conclusions: Drink spiking presents a complex intersection of acute medical risk, forensic urgency, and psychosocial trauma. Emergency clinicians should prioritise early recognition of agent-specific toxidromes, timely collection and preservation of specimens, trauma-informed care, and coordination with forensic and toxicology services. Strengthening ED workflows and regional analytic capacity will enhance detection, patient outcomes, and legal processes.

Key words: benzodiazepines; drink spiking; emergency department; forensic toxicology; gammahydroxybutyric acid; ketamine; poisoning.

Sažetak

Uvod: Dodavanje psihoaktivnih tvari u piće ili hranu bez pristanka druge osobe (engl. *drink spiking*) predstavlja rastući javnozdravstveni i forenzički problem. Žrtve se često javljaju u objedinjeni hitni bolnički prijam (OHBP) s iznenadnom depresijom središnjeg živčanog sustava, anterogradnom amnezijom i nespecifičnim fiziološkim poremećajima, što otežava pravodobno prepoznavanje i liječenje.

Cilj: Sinteza dostupnih dokaza važnih za liječnike hitne medicine o farmakologiji, kliničkom prepoznavanju, toksikologiji, rukovanju uzorcima i akutnom liječenju slučajeva „*drink spikinga*" te prijedlozi sustavnih mjera za poboljšanje otkrivanje, skrbi i forenzičke zaštite.

Metode: Narativni pregled literature obuhvatio je recenzirane radove, podatke iz registara centara za kontrolu trovanja, forenzičke smjernice i nedavne radove koji se odnose na najčešće agense. Poseban naglasak stavljen je na farmakokinetičke i toksikodinamičke značajke koje određuju prozore detekcije, kliničke znakove, pretrage u OHBPu, postupke očuvanja uzoraka i specifične mjere liječenja.

1 Emergency Department, University Hospital Center Zagreb, Zagreb, Croatia

2 Children's Hospital Zagreb, Zagreb, Croatia

3 University of Split Faculty of Health Sciences, Split, Croatia

4 Department of Laboratory Diagnostics, University Hospital Center Zagreb, Zagreb, Croatia

5 National Drug Information Unit and International Relations Department, Croatian Institute of Public Health, Zagreb, Croatia

***Corresponding author:**

Jasmin Hamzić, MD
Emergency Department, University Hospital Center Zagreb
Kišpatićeva 12, 10 000 Zagreb, Croatia
Phone: +385 98 255 219
E-mail: j_hamzic@hotmail.com

ORCID ID:

Hamzić Jasmin:
0000-0003-2726-4308

Bojana Radulović:
0000-0003-2355-8405

Arnes Rešić:
0000-0001-9082-7033

Andrijana Ščavnićar:
0009-0003-4518-726X

Mila Lovrić:
0000-0002-2086-643X

Ivan Gornik:
0000-0001-6146-1327

Zaključak: „*Drink spiking*“ predstavlja složen izazov za hitnu medicinu. Liječnici u OHBPu trebaju prepoznavati toksidrome specifične za agense, pravodobno prikupljati i čuvati uzorke, primjenjivati traumainformirani pristup te koordinirati rad s forenzičkim i toksikološkim službama. Jačanje procedura u OHBPu i regionalnih analitičkih kapaciteta poboljšat će detekciju, ishode pacijenata i pravne procese.

Ključne riječi: benzodiazepini; *drink spiking*; forenzička toksikologija; gamahidroksibutirat; hitni bolnički prijam; ketamin; trovanje

Introduction

Drink spiking, defined as the non-consensual administration of psychoactive agents into an individual's drink or food, has become an increasingly recognised cause of emergency department (ED) presentations (1). Although public awareness has grown, the true incidence remains difficult to quantify because of underreporting, inconsistent coding, and the short detection windows of many implicated agents (2). Victims often present with rapidonset central nervous system depression, anterograde amnesia, and physiologic abnormalities that are disproportionate to reported alcohol intake. These features complicate clinical assessment and delay recognition (2).

Drink spiking frequently presents to emergency departments with sudden central nervous system depression, anterograde amnesia, and clinical severity disproportionate to reported substance use.

Emergency physicians must rapidly differentiate voluntary intoxication from covert drug administration, initiate appropriate stabilisation, and preserve forensic evidence under trauma-informed protocols (3). This narrative review synthesises current knowledge on drink spiking with a focus on ED recognition, toxicological considerations, and acute management.

Methods

This narrative review was conducted by synthesising peer-reviewed articles, poison centre registry reports, forensic guidance documents, and recent reviews addressing drug-facilitated incapacitation. Sources were selected to emphasise the relevance of the emergency department, including pharmacokinetic and toxicodynamic data that inform detection windows, bedside recognition clues, recommended specimen types and preservation methods, and agent-specific management strategies. The review focuses on commonly implicated agents (gammahydroxybutyrate/gammabutyrolactone, GHB/GBL, benzodiazepines and designer analogues, ketamine, sedating antihistamines, alcohol, volatile inhalants, nitrites, and novel psychoactive substances) and on operational measures that EDs can implement to improve detection and forensic preservation. The references cited reflect the literature base informing the narrative synthesis.

Discussion

Drink spiking denotes the non-consensual introduction of psychoactive agents—commonly sedatives, dissociatives, hypnotics, or volatile compounds—into an individual's consumable item. Typical clinical hallmarks include rapid sedation, anterograde amnesia, and impaired psychomotor function in a person who does not recall ingesting a sedative. Agents most frequently implicated include gammahydroxybutyrate and its precursor gammabutyrolactone, benzodiazepines (including flunitrazepam and other prescription agents), ketamine, synthetic sedatives and designer benzodiazepines, sedating antihistamines (e.g., diphenhydramine), and alcohol, often used concurrently to potentiate effects. Less commonly, volatile inhalants and alkyl nitrites have been reported. Perpetrators favour agents that are colourless and odourless, act rapidly to incapacitate, produce amnesia, and in many cases have short detection windows that complicate forensic confirmation (48).

Gammahydroxybutyrate (GHB) and gamma-butyrolactone (GBL)

GHB and GBL are among the most frequently implicated agents in drug-facilitated incapacitation. Acting primarily as GABA-A receptor agonists, they produce profound sedation and anterograde amnesia within 10–20 minutes of ingestion, with peak effects at 30–60 minutes and a short elimination half-life of approximately 30–60 minutes (4-6). Clinically, GHB intoxication is characterised by a rapid *"switchoff"* loss of consciousness, shallow respiration, bradycardia, hypotension, and a striking inability to form new memories during the period of intoxication (5). Because of their short systemic persistence, analytical confirmation requires early specimen collection: obtain blood as soon as possible (preferably within the first few hours) and collect urine early; refrigerate urine and, when recommended, acidify it to minimise analyte loss (4,5,7). Emergency management is supportive: airway protection and ventilatory support are paramount in severe cases, aspiration precautions are essential, and ICU admission should be considered for prolonged depressed consciousness or respiratory compromise (5,8). There is no specific antidote for GHB (58).

Benzodiazepines and designer benzodiazepines

Benzodiazepines act as positive allosteric modulators at GAB-A receptors, producing sedation, anxiolysis, muscle relaxation, and anterograde amnesia. Classic agents such as flunitrazepam and lorazepam have variable onsets (15–60 minutes) and half-lives that permit detection in blood and urine for more extended periods than GHB; however, many designer benzodiazepines (novel analogues) are not detected by routine immunoassays and require targeted liquid chromatography–tandem mass spectrometry (LC–MS/MS) for confirmation. (9). Clinically, benzodiazepine intoxication presents with sedation, slurred speech, ataxia, and, in severe cases or with co-ingestion (e.g. alcohol, opioids), respiratory depression. Flumazenil can reverse benzodiazepine effects but may trigger seizures in mixed overdoses or in benzodiazepinedependent patients; reserve its use for carefully selected cases after toxicology advice (10). ED priorities include airway and respiratory support, monitoring for delayed respiratory depression when co-ingestants are suspected, and early specimen collection for chromatographic testing when clinical suspicion persists despite negative routine screen results (9).

Ketamine

Ketamine is an NMDA receptor antagonist that produces dissociation, analgesia, and amnesia. After oral or intranasal exposure, effects typically begin within 5–15 minutes and may include perceptual disturbances, derealisation, and horizontal nystagmus—features that help distinguish ketamine from pure sedatives. Cardiovascular stimulation (tachycardia, hypertension) is common (11). Ketamine typically produces dissociative effects and horizontal nystagmus; its metabolite, norketamine, prolongs the time during which chromatographic methods can detect exposure (11,12). ED management focuses on airway monitoring, benzodiazepines for severe agitation, and cardiovascular monitoring and treatment as indicated. Specimen collection for targeted chromatographic assays should be arranged early when ketamine exposure is suspected (11,12).

Volatile inhalants and solvents

Volatile solvents and aerosol propellants are less commonly used for drink spiking (they are more often inhaled), but they have been reported in some cases. These agents are highly lipophilic, produce rapid central nervous system depression, and can sensitise the myocardium to catecholamines, thereby increasing the risk of ventricular arrhythmias (13). Clinical clues include chemical odour on breath or clothing, conjunctival injection, dermatitis, and sudden CNS depression inconsistent with oral ingestion patterns. Management includes decontamination (removal of contaminated clothing and thorough skin washing), highflow oxygen, cardiac monitoring, and avoidance of catecholamine surges; specialised testing is rarely available in the ED, and detection windows are very short (13).

Sedating antihistamines (e.g., diphenhydramine)

Over-the-counter sedating antihistamines can be used opportunistically for spiking. They produce sedation and anticholinergic effects—dry mucous membranes, mydriasis, tachycardia, urinary retention, and, in severe cases, delirium. Onset is typically within 15–60 minutes, with duration of several hours. Routine toxicology screening may detect common antihistamines, but severe central anticholinergic toxicity may require physostigmine under toxicology guidance (13). ED care is supportive, with attention to airway protection, active cooling if hyperthermic, and cardiac monitoring.

Alcohol (co-ingestion)

Alcohol is frequently co-administered to potentiate the sedative effects of other agents, slow gastric emptying, and prolong unconsciousness. Ethanol is rapidly absorbed, with peak blood concentrations occurring within 30–90 minutes and exhibits zero-order elimination kinetics (14). Clinically, alcohol potentiates respiratory depression and sedation from other agents and complicates attribution of symptoms. ED clinicians should measure serum ethanol level, anticipate potentiation of respiratory compromise, and consider a more extended observation period or lower admission thresholds when alcohol co-ingestion is suspected (14).

Alkyl nitrites *("poppers")*

Alkyl nitrites are inhaled agents that produce vasodilation, transient hypotension, and, in some cases, methemoglobinemia. Clinical presentation includes flushing, headache, dizziness, syncope, and cyanosis with low pulse oximetry readings that do not correct with supplemental oxygen. Methemoglobinemia should be suspected when pulse oximetry readings are low despite a normal PaO_2. Measurement of methemoglobin level and treatment with intravenous methylene blue are indicated for symptomatic patients or for significant elevations (15). Alkyl nitrite exposure may lead to methemoglobinemia; symptomatic or clinically substantial cases should be treated with intravenous methylene blue using standard weight-based dosing per local protocol (15).

Novel psychoactive substances and designer sedatives

Novel psychoactive substances encompass a heterogeneous group of synthetic sedatives, cathinones, and other compounds that vary widely in potency and in the toxidrome they produce. Many NPS evade routine immunoassays and require LC-MS/MS or high-resolution mass spectrometry for detection (16-18). Clinical presentations range from profound sedation and amnesia to agitation, psychosis, and cardiovascular instability. When routine screening tests are negative but clinical features suggest exogenous intoxication, clinicians should preserve specimens and engage regional toxicology laboratories early (16-18).

Early recognition in the ED relies on integrating toxidromic patterns with contextual clues such as shared drinks, abrupt behavioural changes, or witness reports.

Clinical presentation and ED recognition clues

Patients present with a spectrum of findings. Neurological features commonly include sudden somnolence, slurred speech, ataxia, and anterograde amnesia; behavioural changes, such as delirium or dissociation, may be prominent with ketamine or specific NPS (4,7,11). Cardiovascular signs range from hypotension and bradycardia (notably with GHB) to tachycardia and hypertension (with ketamine or anticholinergic agents) (19,20). Respiratory depression and aspiration risk are central concerns in severe intoxication. Forensic clues—chemical odour on breath or clothing, reports of unusual taste, communal drink sharing, or witness accounts of abrupt behavioural change, should heighten clinical suspicion. Recognising suspected spiking depends on combining observed toxidromes with contextual information such as shared drinks or eyewitness accounts (4). Specific bedside signs can help differentiate agents: horizontal nystagmus and dissociation suggest ketamine; anticholinergic features point to antihistamines; diaphoresis and rapid recovery with amnesia are characteristic of GHB; low pulse oximetry readings with normal PaO_2 may indicate methemoglobinemia from nitrites (4,11,15).

Diagnostic methodologies and forensic specimen handling

Routine ED immunoassays often fail to detect GHB, designer benzodiazepines, and many NPS; definitive identification typically requires gas chromatography–mass spectrometry (GC-MS) or liquid chromatography–tandem mass spectrometry (16,18,21,22). Clinicians should collect forensic specimens early: obtain blood as soon as possible (preferably within the first few hours) and urine; refrigerate urine and, when recommended, acidify it to minimise analyte loss (4,7,23). Routine immunoassays do not detect many designer sedatives and novel compounds; definitive identification generally requires chromatographic-mass spectrometry techniques (16-18,22). Proper specimen handling—refrigerating samples, dual labelling, acidifying urine when indicated, and strict chainofcustody documentation—is essential to maintain evidentiary value (4,23). Early liaison with regional toxicology and forensic laboratories expedites appropriate testing and specimen transport (21,22).

Emergency department management: stabilisation and agent-specific care

Initial ED management follows ABC priorities: secure the airway, support breathing, and stabilise circulation. Continuous cardiac monitoring and pulse oximetry are mandatory in moderate-to-severe presentations. GHB and GBL have no specific antidote; care is supportive and focuses on airway protection and ventilatory support, with rapid respiratory depression or bradycardia sometimes requiring ICU admission for monitoring (2,8). For benzodiazepine exposures—including designer analogues—management is likewise supportive; flumazenil can reverse benzodiazepine effects but may precipitate seizures in mixed overdoses or in benzodiazepinedependent patients, so its use should be limited to carefully selected cases after toxicology consultation (10). Ketaminerelated presentations are managed by treating agitation with benzodiazepines when necessary, monitoring cardiovascular status and treating severe hypertension according to standard protocols, and observing patients for resolution of dissociative symptoms with psychiatric follow-up as indicated (11). Sedating antihistamine toxicity is treated supportively for anticholinergic effects, with physostigmine considered only in severe cases under toxicology guidance (13). For volatile inhalant exposures, remove contaminated clothing and decontaminate skin to prevent ongoing or secondary exposure, anticipate arrhythmias, and avoid interventions that provoke catecholamine surges (13). Alkyl nitrite inhalation can produce methemoglobinemia; clinicians should measure methemoglobin levels and treat symptomatic or clinically significant cases with intravenous methylene blue using standard weight-based dosing per local protocol while monitoring the response (15). Seizures are initially managed with benzodiazepines, and refractory seizures may require escalation to barbiturates or propofol in an intensive care setting. In all cases, clinicians should consider co-ingestions (notably alcohol and opioids), which increase the risk of respiratory depression and complicate antidote use. ED teams must also address sexual assault care needs, sexually transmitted infections (STI) prophylaxis, emergency contraception, HIV post-exposure prophylaxis and coordinate with forensic exam teams and victim advocates (19,21).

Forensic imperatives, documentation, and trauma-informed care

Forensic integrity depends on meticulous documentation and the preservation of evidence. Clinicians should record verbatim patient statements when possible, timestamp all findings and interventions, photograph injuries with consent, and preserve clothing and suspected containers in sealed evidence bags, following chain-of-custody protocols (19). Timely sampling, appropriate preservation measures (e.g., acidifying urine when indicated), and strict chain-of-custody documentation are critical for forensic validity (4,23). Trauma-informed communication explaining procedures, obtaining consent, minimising repeated recounting, and involving victim advocates and mental health professionals, improves patient cooperation and reduces re-traumatisation (20). Use trauma-informed approaches: explain interventions, seek consent, limit repeated retelling, and involve victim support and mental health services to improve cooperation and reduce retrauma (20).

Trauma-informed communication, coordinated forensic evidence handling, and clear documentation are essential components of ED care for suspected drug-facilitated incapacitation.

Disposition, follow-up, and psychosocial support

Admission is indicated for persistent respiratory depression, haemodynamic instability, significant arrhythmias, refractory seizures, or inability to protect the airway. Patients who return to baseline mental status within an observation period (commonly 6–8 hours), have typical vital signs, and have social supports may be discharged with clear instructions, referrals to sexual assault services, and follow-up for toxicology results and mentalhealth care. ED teams should offer STI prophylaxis, emergency contraception, and HIV post-exposure prophylaxis according to local protocols and ensure documented safety planning (21).

Surveillance, prevention, and system-level recommendations for EDs

EDs should implement practical tools to improve recognition and response: standardised order sets that bundle initial labs and specimen kits; EHR prompts that flag combinations of memory loss, communal drinking, and disproportionate sedation; specimentimelines; and simulation training that integrates medical stabilisation with forensic evidence preservation. Implementing system measures standardised ED order sets, specimentimeline checklists, EHR alerts, and ready access to regional toxicology—enhances detection and preserves evidentiary value (11,22). Regional access to advanced toxicology (GCMS, LCMS/MS) and 24/7 poison centre consultation enhances diagnostic yield and patient care (11,22). Embedding standardised registry codes for suspected non-consensual drug administration will enable surveillance and targeted prevention efforts (21, 24).

Standardised ED workflows including specimen timing checklists, order sets, and rapid access to toxicology consultation significantly improve diagnostic accuracy and forensic outcomes.

Prevention, harm reduction, and legal considerations

Prevention requires a multifaceted approach: public awareness campaigns, venue staff training, tamper-evident products, mobile safety applications, and policy incentives for venues that adopt best practices. Legal frameworks vary; explicit statutes criminalising non-consensual drug administration can streamline prosecutions and reduce evidentiary burdens, while confidential reporting pathways balance victim autonomy with public safety (25, 27).

Research gaps and future directions

Key research priorities include prospective ED-based cohort studies with standardised coding to establish incidence; validation of point-of-care assays for GHB and other agents; pharmacokinetic modelling of emerging sedatives to refine detection windows; evaluation of wearable biosensors and digital epidemiology tools for real-time cluster detection; and longitudinal studies of neurocognitive and psychosocial outcomes after drug-facilitated assault (28,33). Cost-effectiveness analyses of prevention programmes and trials of ED protocols will inform resource allocation and policy.

Conclusion

Drink spiking remains a clinically and forensically challenging presentation in emergency medicine. Early recognition of agent-specific toxidromes, prompt specimen collection with appropriate preservation, trauma-informed care, and coordination with forensic and toxicology services are essential to optimise patient outcomes and preserve legal evidence. Implementing ED protocols, improving access to advanced toxicology, and strengthening surveillance and prevention strategies will be critical to reducing the burden of drug-facilitated assaults.

References

1. Bendau A, Michnevich T, Petzold MB, Piest A, Schmolke R, Jakobson D et al. Spiking Versus Speculation? Perceived Prevalence, Probability, and Fear of Drink and Needle Spiking. J Drug Issues. 2023;55(1):89–103. doi:10.1177/00220426231197826
2. Davies EL, Piatkowski T, Frankovitch A, Cheneal Puljević, Barratt MJ, Barratt MJ et al. Exploring Experiences of Drink and Needle Spiking Incidents Among Global Drug Survey Respondents from 22 Countries. Journal of drug issues. 2024; https://doi.org/10.1177/00220426241248613
3. Pinchevsky GM, Wright EM, Fagan AA. Gender differences in the effects of exposure to violence on adolescent substance use. Violence Vict. 2013;28(1):122-44. doi: 10.1891/0886-6708.28.1.122.
4. Busardo F, Jones A. GHB Pharmacology and Toxicology: Acute Intoxication, Concentrations in Blood and Urine in Forensic Cases and Treatment of the Withdrawal Syndrome. Current Neuropharmacology (Internet). 2015;13(1):47–70. doi: 10.2174/1570159X13666141210215423
5. Schröck A, Hari Y, König S, Auwärter V, Schürch S, Weinmann W. Pharmacokinetics of GHB and detection window in serum and urine after single uptake of a low dose of GBL - an experiment with two volunteers. Drug Testing and Analysis. 2013;6(4):363-6. doi: 10.1002/dta.1498
6. Liechti ME, Quednow BB, Evangelia Liakoni, Dornbierer D, Robin von Rotz, María Salomé Gachet et al. Pharmacokinetics and pharmacodynamics of γ-hydroxybutyrate in healthy subjects. British Journal of Clinical Pharmacology (Internet). 2016;81(5):980–8. doi: 10.1111/bcp.12863
7. Voisin A, Solas-Chesneau C, Anne-Laure Pélissier-Alicot, Fabresse N. Biomarkers of Gamma-Hydroxybutyric Acid (GHB) Exposure: A Comprehensive Review of Analytical and Forensic Advances. Toxics (Internet). 2025;13(10):824–4. doi: 10.3390/toxics13100824
8. Skjelland D, Jørgenrud BM, Gundersen K, Bjørnaas MA, Brekke M, Dalaker VM et al. Gamma-hydroxybutyrate poisoning: clinical diagnosis versus laboratory findings. Clinical Toxicology. 2025;63(4):253–60. doi: 10.1080/15563650.2025.2463700
9. Elian AA. Detection of low levels of flunitrazepam and its metabolites in blood and bloodstains. Forensic Science International. 1999;101(2):107–11. doi: 10.1016/s0379-0738(99)00013-4

10. Penninga EI, Graudal N, Ladekarl MB, Jürgens G. Adverse Events Associated with Flumazenil Treatment for the Management of Suspected Benzodiazepine Intoxication - A Systematic Review with Meta-Analyses of Randomised Trials. Basic & Clinical Pharmacology & Toxicology. 2015;118(1):37–44. doi: 10.1111/bcpt.12434
11. Wang Y, Chang S, Chen D. Research trends and hotspots of ketamine from 2014 to 2023: a bibliometric analysis. Frontiers in Neuroscience. 2024;18. doi: 10.3389/fnins.2024.1407301
12. Zanos P, Moaddel R, Morris PJ, Riggs LM, Highland JN, Georgiou P, et al. Ketamine and Ketamine Metabolite Pharmacology: Insights into Therapeutic Mechanisms. Pharmacological reviews. 2018;70(3):621–60. doi: 10.1124/pr.117.015198
13. Leary AD, Schwartz MD, Kirk MA, Ignacio JS, Wencil EB, Cibulsky SM. Evidence-Based Patient Decontamination: An Integral Component of Mass Exposure Chemical Incident Planning and Response. Disaster Medicine and Public Health Preparedness (Internet). 2014;8(3):260–6. doi: 10.1017/dmp.2014.41
14. Helle AC, Wycoff AM, Griffin SA, Fleming M, Freeman LK, Vebares TJ et al. Co-use of medication and alcohol: The influence on subjective effects of intoxication and affect. Personality Disorders (Internet). 2022;13(1):75–83. doi: 10.1037/per0000480
15. Tutenges S. Nightlife tourism: A mixed methods study of young tourists at an international nightlife resort. Tourist Studies. 2012;12(2):131–50. doi: 10.1177/1468797612454250
16. Palamar JJ, Abukahok N, Acosta P, Walton SE, Stang B, Krotulski AJ. Exposures to synthetic cathinones, fentanyl, and xylazine among nightclub attendees in New York City, 2024. Drug and Alcohol Dependence. 2025;275:112792. doi: 10.1016/j.drugalcdep.2025.112792
17. Grafinger KE, Liechti ME, Liakoni E. Clinical value of analytical testing in patients presenting with new psychoactive substances intoxication. British Journal of Clinical Pharmacology. 2019 ;86(3):429–36. doi: 10.1111/bcp.14115
18. Taflaj B, La Maida N, Tittarelli R, Di Trana A, D'Acquarica I. New Psychoactive Substances Toxicity: A Systematic Review of Acute and Chronic Psychiatric Effects. Int J Mol Sci. 2024;25(17):9484. doi: 10.3390/ijms25179484.
19. Tozzo P, Ponzano E, Spigarolo G, Nespeca P, Caenazzo L. Collecting sexual assault history and forensic evidence from adult women in the emergency department: a retrospective study. BMC Health Services Research. 2018;18(1). doi: 10.1186/s12913-018-3205-8
20. Han HR, Miller HN, Nkimbeng M, Budhathoki C, Mikhael T, Rivers E et al. Trauma informed interventions: A systematic review. Sar V, editor. PloS one. 2021;16(6):1–28. doi: 10.1371/journal.pone.0252747
21. Hessler MR, Kacinko SL, Logan BK. Drug-facilitated crime: A review of findings between 2019 and 2023. Journal of Forensic Sciences. 2025; doi: 10.1111/1556-4029.70151
22. Adkins J, Bataineh A, Khalaf M. Identifying Persons of Interest in Digital Forensics Using NLP-Based AI. Future Internet (Internet). 2024;16(11):426. doi: 10.3390/fi16110426
23. Quest DW, Horsley J. Field-Test of a Date-Rape Drug Detection Device. Journal of Analytical Toxicology. 2007;31(6):354–7. doi: 10.1093/jat/31.6.354
24. García-Sánchez S, Somoza-Fernández B, de Lorenzo-Pinto A, Ortega-Navarro C, Herranz-Alonso A, Sanjurjo-Sáez M. Mobile Health Apps Providing Information on Drugs for Adult Emergency Care: Systematic Search on App Stores and Content Analysis (Preprint). JMIR mHealth and uHealth. 2021; doi: 10.2196/29985
25. Csete J, Kamarulzaman A, Kazatchkine M, Altice F, Balicki M, Buxton J, et al. Public health and international drug policy. The Lancet (Internet). 2016;387(10026):1427–80. doi: 10.1016/S0140-6736(16)00619-X
26. Weiss KG, Colyer CJ. Roofies, Mickies and Cautionary Tales: Examining the Persistence of the "Date-Rape Drug" Crime Narrative. Deviant Behavior. 2010;31(4):348–79. doi: 10.1080/01639620903004846
27. UNODC. Guidelines for the forensic analysis of drugs facilitating sexual assault and other criminal acts (Internet). 2011. Available from: https://www.unodc.org/unodc/en/scientists/guidelines-for-the-forensic-analysis-of-drugs-facilitating-sexual-assault-and-other-criminal-acts_new.html
28. DeVane CL. Clinical Pharmacokinetics and Pharmacodynamics of Anxiolytics and Sedative/Hypnotics. Applied Clinical Pharmacokinetics and Pharmacodynamics of Psychopharmacological Agents. 2016;247–66. doi: 10.1007/978-3-319-27883-4_10
29. Wu JY, Ching CTS, Wang HMD, Liao LD. Emerging Wearable Biosensor Technologies for Stress Monitoring and Their Real-World Applications. Biosensors. 2022;12(12):1097. doi: 10.3390/bios12121097
30. Xing E, Chen H, Xin X, Cui H, Dou Y, Song S. Recent Advances in Smart Phone-Based Biosensors for Various Applications. Chemosensors. 2025;13(7):221. doi: 10.3390/chemosensors13070221
31. Feltmann K, Elgán TH, Strandberg AK, Kvillemo P, Jayaram-Lindström N, Grabski M, et al. Illicit Drug Use and Associated Problems in the Nightlife Scene: A Potential Setting for Prevention. International Journal of Environmental Research and Public Health (Internet). 2021;18(9):4789. doi: 10.3390/ijerph18094789
32. O'Connell ME, Boat T, Warner KE, editors. Preventing Mental, Emotional, and Behavioral Disorders Among Young People: Progress and Possibilities. Washington (DC): National Academies Press (US); 2009. 9, Benefits and Costs of Prevention. Available from: https://www.ncbi.nlm.nih.gov/books/NBK32767/
33. Irantzu Recalde-Esnoz, Prego-Meleiro P, Montalvo G, Héctor Del Castillo. Drug-Facilitated Sexual Assault: A Systematic Review. Trauma, Violence, & Abuse. 2023;25(3). doi: https://doi.org/10.1177/15248380231195877