

# DEXTROMETHORPHAN- AN EMERGING DRUG OF ABUSE

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## Abstract

Dextromethorphan or DXM is an effective and harmless cough and cold suppressant available in many cold and cough preparations. At maximum dosage, it mimics hallucinogenic and psychoactive effects in human body and hence it is appealing for abuse. In this work, a detailed study of an antitussive agent i.e. Dextromethorphan which is a semi-synthetic opioid and a replacement of codeine and pholcodine in various cough linctuses has been described extensively. This review also revolves around the structured detailing of Dextromethorphan, its physical and chemical properties as well its pharmacokinetics in human body and also its effect on human brain and its relationship with NMDA receptors. This study also focuses on the emergence of Dextromethorphan as new drug of abuse due to its easy availability and as it possesses similar effects with LSD and other hallucinogens when taken in overdose in addition with the Toxicological analysis of Dextromethorphan from biological matrices using respective analytical methods.

**Keywords:** Abuse; Dextromethorphan; Hallucinogen; NMDA; Opioid.

## INTRODUCTION

Dextromethorphan or DXM is a type of cough suppressing drug which has been used widely for more than forty years. DXM (d-3-methoxymorphinan) is dextro-isomer form of levorphanol, in addition a codeine analogue and also a semi synthetic derivative of morphine. As it is obtained from levophenol which is a mu-opioid agonist, it doesn't possess full extent of CNS effects routine to opioid agonists, and it also doesn't produce classic opioid-like effects plus dependence. DXM also has some other uses too in medicine, varying from pain reliever to psychological applications. DXM is been sold in various forms like syrup, spray, tablet and lozenge forms. In its purest form, dextromethorphan materializes as white powder. Dextromethorphan or DXM is a type of semi-synthetic correspondent of codeine [1], it is widely used as cough suppressing agent among over-the-counter (OTC) cold and cough medicines [2] and is believed to be effective and safe when administered at prescribed doses.

Dextromethorphan is also referred to as "DXM" and is famous as "the poor man's PCP". In year 2015, in United States of America, all the Over-The-Counter drugs containing DXM covered 85–90% of all drugs containing an antitussive that were sold in 235 million packages of total [3]. There are several compounds that are active clinically classified as morphinans. At therapeutic dosage, dextromethorphan acts on the sigma receptors and activates its cough suppressing effects, whereas at high doses DXM is metabolized to dextrophan that is an active metabolite which is responsible for counteracting N - methyl-D-aspartate receptors [4]. DXM is synthesized and may be found in over 140 over-the-counter cold and flu medications [5], [6]. Although dextromethorphan is similar in composition to other opioids, it has minimum connection with opioid receptors. FDA granted approval for DXM before 3 December 1957. The IUPAC name of DXM is (1S, 9S, 10S)-4-methoxy-17-methyl-17-azatetracyclo [7.5.3.0.0] heptadeca-2(7), 3, 5 triene.

The cough suppressing effect of dextromethorphan is likely to be mediated by both the receptors i.e. NMDA and sigma-1 receptors [7]. The chemical structure of DXM is alike to that of codeine. Until recently, DXM was classed as a synthetic opioid. It is because the sigma-1 receptor is now no longer counted as an opioid receptor [8]. When DXM is used at therapeutic dosage, cough and flu products containing DXM causes minor adverse effects if any [9]. The most common cause of adverse repercussions is DXM misuse. Dextromethorphan consists of a large range of pharmaco-dynamic effects as a result of its activity on many receptors and channels. Dextromethorphan's popular effects of cough suppression are known to be the result

from its N-methyl-D-aspartate (NMDA) antagonism and stimulation of sigma-1 receptors ( $\sigma_1R$ ) [10]. Synthesis of DXM is done from benzyloquinoline using the process namely Grewe's cyclization, which provides communication with morphinan possessing a 3-D structure. Also, Dextromethorphan is a serotonergic drug which has a potential threat of serotonin syndrome [11], [12], and it enhances the anesthetic action of morphine along with some other  $\mu$ -receptor agonists. At therapeutic dosages, DXM generates minimal antitussive and anesthetic effects and has been employed for the sake of relief from coughs caused by lesser viral diseases of upper respiratory tract or breathed in irritants and is found to be best for an enduring, but nonproductive cough. Non-prescribed cough syrups, like DXM have been widely proposed by many medical and pharmaceutical professionals. Although, the therapeutic administration of dextromethorphan in children has been a questionable work since [13] DXM was accepted by the U.S. FDA in 1958 as OTC cough syrups. Since year 1970, it has been disapproved from the U.S. Act of Drug Enforcement Administration (DEA) Controlled Substance. Concerns about misuse of DXM are not entirely new as misuse of dextromethorphan was first believed more than 30 years ago [14]. When Dextromethorphan's authorized dose regimen is outmatched for illicit usage, a slew of mental problems might emerge. The effects caused by DXM depends on the administered dosages and varies from placid motor and cognitive damage to PCP like parapsychotic symptoms such as dissociative states, paranoia, visual hallucinations and delusions. Combination of such symptoms may lead to abrupt violent acts, like suicides, homicides or assaults [15]. Even though Dextromethorphan has been employed as an OTC medicine since year 1958, only short time ago it has been believed as potential emerging drug of abuse, including its pleasure giving usages becoming more recurring & far-flung back from the time of 1990s [16].

### ANTITUSSIVE PROPERTY OF DXM

Unlike most symptoms, cough comes under the category of voluntary control and also is connected to a sensation of irritation in air passage and an urge to cough and this entangle clinical trials on cough [17],[18]. Dextromethorphan is used as a cough suppressant in cold and cough medicines (and, in high doses, it acts as a dissociative hallucinogen), whereas levomethorphan, a levorotatory enantiomer and a pro-drug of levorphanol, is an active opioid anesthetic and is categorized as a schedule II drug in the United States [19], [20], [21]. Dextromethorphan is chemically close to Codeine, and is accepted widely as an effective and safest antitussive agent which is non-narcotic [22]. It is a non-aggressive receptor antagonist of the N-methyl-d-aspartate (NMDA) type of excitatory amino acid receptors that are involved in neuronal development and migration. Its adequacy has been validated in both clinical cough [23] and also in experimental studies of cough challenge [24]. Capon et al [25] tried to establish variances in experimental cough constraint by DXM in normal people as function of CYP2D6 phenotype. However, the study was indecisive because of inadequate statistical power. Pholcodine, Codeine, and DXM are believed to contain specific constraint actions over the areas of cough control of the brainstem, while anti-histamines like diphenhydramine have a common sedating action and feasible anti-cholinergic functioning in the brain [26].

### STRUCTURE OF DEXTROMETHORPHAN

DXM, also known as (3-methoxy-N-methylmorphinan), is the dextrorotatory [ $d$ - or  $(-)$ ] enantiomer of levomethorphan [ $l$ - or  $(+)$ ], and is the methyl ether compound of levorphanol and Dextrophan (3-hydroxy-N-methylmorphinan) respectively [27]. DXM is named according to IUPAC rules as -3-methoxy-17-methyl-9a, 13a, and 14a-morphinan. Methorphan is found in two isomeric forms, each with a different pharmacology and effect based on its two enantiomers. Molecular weight of DXM is 271.4 and is light yellowish to whitish in appearance. It doesn't have any odor i.e. it is odorless. Melting Point of DXM is around 109 °C to 111 °C whereas it is insoluble in water practically but freely soluble in Chloroform. Therefore, dextromethorphan chemically is a derivative of opium alkaloid though pharmacologically, Because it does not act on opioid receptors, it is not classified as an opioid and thus does not have anesthetic properties., respiratory depression, and euphoriant effects, observed in case of codeine and morphine [28].

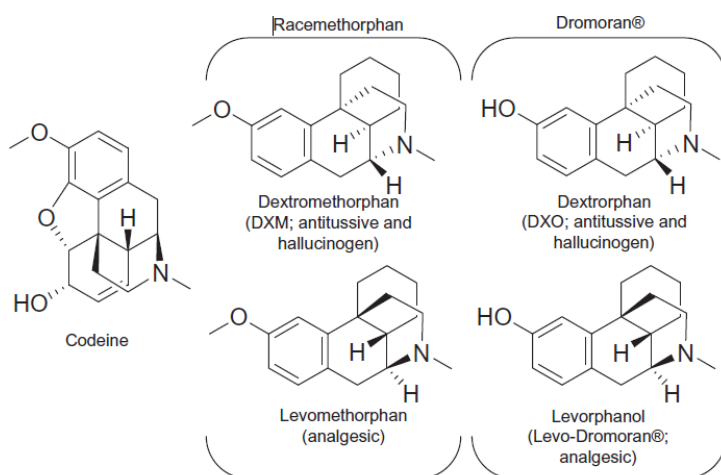


Figure 1: Chemical Structure of DXM and its analogues [29]

## FATE OF DXM IN HUMAN BODY

### 1) ABSORPTION

Dextromethorphan is absorbed rapidly in the GIT (gastrointestinal tract) and metabolized in about 20 minutes. Dextrophan is absorbed completely inside the GIT (gastrointestinal tract) and ratio of DXM's brain-to plasma measures from 25 to 500 [30]. Following absorption, DXM undergoes first-pass metabolism prior of being excreted via urine. The cytochrome P450 (CYP) 2D6 system plays an important role in metabolizing drug with a half-life of round about 2 hours [31]. The 3-O methylated major metabolite of Dextromethorphan i.e. dextrophan touched its highest peak point of plasma concentration in around Two hours [32].

### 2) DISTRIBUTION

DXM's plasma protein binding is approximately 60–70% [33] and after oral administration, DXM achieves peak serum concentration in 2–3 hours, whereas DXO attains concentration of peak serum in 1.5–3 hours, regardless of whether it is a tablet, liquid, or an extended-release formulation [34]. DXO's lower liposolubility compared to DXM may contribute to lessen cerebral bioavailability and effect of anti-NMDA neuromodulatory. DXM, despite likely crossing the barrier of placenta because of its low molecular weight, has no teratogenic effect [35].

### 3) METABOLISM & EXCRETION

Previous researches have revealed that dextromethorphan go through extensive and rapid hepatic metabolism & also the range of metabolism varies highly among the study's subjects [32]. The chief metabolic routes are O- and N- Methylation prior to subsequent conjugation. O-demethylation of DXM gives dextrophan whereas, dextromethorphan is N-demethylated to 3-methoxymorphinan and N, O-demethylation of DXM produces 3-hydroxymorphinan [36] [37]. Previous research studies have demonstrated that the metabolic conversion of dextromethorphan into 3-hydroxymorphinan is not only done by N-demethylation but also by O-demethylation of 3-methoxymorphinan [38]. DXM is rapidly and extensively O-demethylated by CYP2D6 to DXO; its primary active metabolite [39]. The plasma half-life of DXM is around two to four hours after administrating at therapeutic dose. Half-life of dextrophan is 3–5 h [40]. The metabolites of dextromethorphan along with parent drug get excreted out via urine in conjugated and unconjugated forms [36].

## EFFECTS OF DXM ON BRAIN

Due to the possibility of interactions, certain drugs should not be administered with Dextromethorphan, including SSRIs monoamine oxidase inhibitors (MAOIs), and the CYP2D6 inhibitors amiodarone, CNS depressants and quinidine [41]. Various studies showed that dextromethorphan and its major metabolite which is dextrophan, act as disharmonious NMDA receptor antagonists having low affinity, and it also suppresses glutamate-induced excitotoxicity in the Central Nervous System and regions of spinal cord. This action mechanism of DXM is especially important while treating some conditions, like amyotrophic lateral sclerosis (ALS) or methotrexate neurotoxicity [42]. Even if it is not used widely today as analgesics, dextromethorphan and levorphanol previously were believed to be pharmacological alternatives of morphine for managing pain [43]. Furthermore, it was demonstrated that DXM alone would be sufficient to produce this antidepressant-like effect, without the need for converting it to the metabolite DXO [44]. Symptoms have been averred toxicity is very rare and scantily described in the literature. Typically toxicity is evident at serum bromide levels more than 50 to 100 mg/dL [45].

## ABUSE OF DXM

It is worth noting that DXM misuse is consonant with the fact that hallucinogen usage peaks around the age of 19 years and swiftly declines after that [46]. Dextromethorphan was approved for OTC sale by the FDA in 1958, and its abuse pattern was not noted at the time [47]. This might be because these subjects were not m-agonist opioid addicts, and other assessed outcomes focused on its ability to elicit liking or euphoria, as well as if it possessed morphine-like properties. Its misuse has been seen to be more persistent and pervasive, particularly since the mid-1990s, due to the ease and quick distribution of information made available by the internet [46]. Only 15 occurrences of fatal outcomes utilizing DXM have been reported to the manufacturer in the last 30 years, with only one referring to therapeutic dosages in concurrent treatment with antidepressants [48]. Such accessibility and "on demand" purchasing may unintentionally lead to its safety assumption [29]. Sometimes recreational users report zero obvious physical addiction or withdrawal symptoms. Those who regularly take dextromethorphan with the goal of abusing it report minor withdrawal symptoms similar to opiate withdrawal. However, there is the possibility of resistance to dextromethorphan, which must be conquered in order to produce the desired "high." Dextromethorphan's exhilaration has been characterized as a PCP-kind of bliss, which has contributed to the street term "poor man's ecstasy" [49]. Street names for DXM include "skittles," "robos," "rojos," "velvet," "CCC," and "poor man's PCP," among others. Dextromethorphan intoxication is referred to as "robo-tripping," "skittling," and "dexing" in slang. Adolescents are the most likely to abuse dextromethorphan [50]. Dextromethorphan is most typically used orally as a recreational drug; however it can also be sniffed if taken in powdered form. DXM's desired effects are defined as early euphoria with less dextromethorphan consumption. Higher doses of dextromethorphan are used to treat vivid hallucinations and whole body analgesia. Complete dissociation is associated with higher amounts of DXM intake [51]. DXM can be used as recreational drug since it causes a psychotic symptoms experience or just an intoxicated effect at greater dosages [46].

## TOXICOLOGICAL ANALYSIS, RESULTS AND FORENSIC SIGNIFICANCE

DXM is not often considered in the toxicological testing of simple immunoassays. The increased blood concentrations of Dextromethorphan and Dextrophan, additionally the DSM-V psychiatric criteria, should be regarded in the diagnostic of a dissociative condition. Furthermore, because this analysis can fabricate false positive results for PCP, but also for opiates, confirmation and quantification using other specific techniques such as HPLC-MS (High Performance Liquid Chromatography coupled to mass spectrometry) and GC-MS (Gas Chromatography–Mass spectrometry), and less commonly electrophoresis, fluorimetry, ELISA, and spectrophotometry, are required. In addition, the usage of potentiometric sensors for dextromethorphan analysis has been examined [2] and various samples such as blood, oral fluid, and urine has been evaluated [52]. The International Association of Forensic Toxicologists' list of reference blood levels of dangerous and therapeutic compounds, as well as data from other comprehensive collections, report serum concentrations of 10–40 ng/mL [53]. Table- 1 represents the compiled data collected from various toxicological and analytical examinations carried out in last few years, studied and compiled by various authors.

Table 1 Details of various studies conducted on the analysis of cough syrups

Biological matrices	Extraction procedure	Instrumentation	Analytes concentration and detection limit	Ref.
<b>Skeletal Remains (Rats)</b>	Samples extracted using Filtration-Pass through Extraction protocol followed by methanolic extraction protocol in basic medium.	Ultra-Performance Liquid Chromatography quadrapole time-of-flight Mass Spectrometry (UPLC-qTOF-MS)	Limit of detection = 1ng/mL	[54]
<b>Blood</b>	Samples extracted using basic drug fractions, Liquid-Liquid & Liquid-Solid extraction protocols.	Enzyme Multiplied Immunoassay Technique (EMIT)	Toxic concentration of DXM - 0.15 -1.22 mg/L, in combination with Chlorpheniramine.	[15]
<b>Blood</b>			Toxic concentration of DXM- 0.19 -1.00 mg/L, in combination with Ethanol.	
<b>Heart Blood</b>			Lethal concentration of DXM - 3.23 mg/L, in combination with Cannabinoids. (17 years old)	
<b>Heart Blood</b>			Lethal concentration of DXM - 1.89 mg/L, in combination with Diphenhydramine and Cannabinoids (19 years old)	
<b>Iliac Blood</b>			Lethal concentration of DXM - 1.3 mg/L	
<b>Vitreous Humor</b>			Lethal concentration of DXM - 0.7 mg/L	
<b>Liver</b>			Lethal concentration of DXM - 19.0 mg/Kg	
<b>Urine</b>			Lethal concentration of DXM - >20.0 mg/L	
<b>Whole Blood</b>	Samples analyzed for Dextromethorphan (DXM) and its metabolite Dextrophan (DXO) and Terfenadine using Liquid-Liquid extraction in basic medium using Methanol as solvent.	Modified capillary Gas Chromatography coupled with Mass Spectrophotometer (GC-MS)	Lethal Concentration of DXM -5.09mg/L Lethal Concentration of DXO -1.40mg/L (22 years old, female)	[55]
<b>Urine</b>			Lethal Concentration of DXM -3.29mg/L Lethal Concentration of DXO -3.09mg/L (22 years old, female)	

<b>Bile</b>				Lethal Concentration of DXM -3.48mg/L Lethal Concentration of DXO -1.86mg/L (22 years old, female)
<b>Liver</b>				Lethal Concentration of DXM -10.74mg/L Lethal Concentration of DXO -4.81mg/L (22 years old, female)
<b>Heart</b>				Lethal Concentration of DXM -2.38mg/L Lethal Concentration of DXO -1.72mg/L (22 years old, female)
<b>Kidney</b>				Lethal Concentration of DXM -4.27mg/L Lethal Concentration of DXO -2.09mg/L (22 years old, female)
<b>Brain</b>				Lethal Concentration of DXM -3.47mg/L Lethal Concentration of DXO -1.58mg/L (22 years old, female)
<b>Femoral Blood</b>	<b>Vein</b>	Samples extracted in basic medium using Liquid-Liquid Solvent Extraction protocol by Chloroform.	GC-MS fused with Silica Capillary column and Nitrogen-Phosphorus sensitive detector	Lethal Concentration of DXM -9.2mg/g [56] Lethal Concentration of DXO -2.9mg/g (18 years old, female) Lethal Concentration of DXM -3.3mg/g Lethal Concentration of DXO -1.5mg/g (27 years old, male)
<b>Liver</b>				Lethal Concentration of DXM -31.2mg/g Lethal Concentration of DXO -11.5mg/g (18 years old, female) Lethal Concentration of DXM -23.0mg/g Lethal Concentration of DXO -29.2mg/g (27 years old, male)
<b>Blood</b>		Samples extracted using Liquid-Liquid solvent extraction protocol in basic pH medium with NaOH and was extracted 3 times with ethyl acetate	Gas Chromatography thermionic specific detection and GC-MS	Lethal Concentration of DXM -31.2mg/L in combination with Zipeprol (21 years old, Male) [57]  Lethal Concentration of DXM -1.2mg/L in combination with Zipeprol (20 years old, Male)

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Lethal Concentration of DXM -4.1mg/L in combination with Zipeprol (19 years old, female)

Lethal Concentration of DXM -1.8mg/L in combination with Zipeprol (21 years old, female)

Lethal Concentration of DXM -1.4mg/L in combination with Zipeprol (19 years old, female)

Lethal Concentration of DXM -1.8mg/L in combination with Zipeprol (29 years old, female)

Lethal Concentration of DXM -18.3mg/L in combination with Zipeprol (22 years old, female)

Lethal Concentration of DXM -2.9mg/L in combination with Zipeprol (21 years old, male)

Lethal Concentration of DXM -1.1mg/L in combination with Zipeprol (22 years old, male)

**Gastric contents**

Lethal Concentration of DXM -25.5mg/L in combination with Zipeprol (21 years old, Male)

Lethal Concentration of DXM -243.7mg/L in combination with Zipeprol (20 years old, Male)

Lethal Concentration of DXM -NA in combination with Zipeprol (19 years old, female)

Lethal Concentration of DXM -2.1mg/L in combination with Zipeprol (21 years old, female)

Lethal Concentration of DXM -3.4mg/L in combination with Zipeprol (19 years old, female)

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			Lethal Concentration of DXM -15.0mg/L in combination with Zipeprol (29 years old, female)
			Lethal Concentration of DXM -NA in combination with Zipeprol (22 years old, female)
			Lethal Concentration of DXM -NA in combination with Zipeprol (21 years old, male)
			Lethal Concentration of DXM -NA in combination with Zipeprol (22 years old, male)
<b>Blood from peripheral area</b>	Samples analyzed for the presence of alcohol and other volatile drugs. For this samples were extracted using acidic and basic Liquid-Liquid solvent extraction protocol.	Gas Chromatography, Enzyme Multiplied Immunoassay Technique (EMIT) And GC-MS	Lethal Concentration of DXM -4.74mg/L (31 years old, Male) [58]
<b>Blood</b>			Toxic Concentration of DXM -0.3mg/L (23 years old, Male)
<b>Blood</b>			Toxic Concentration of DXM -1.05mg/L in combination with Chlorpheniramine and Nor-9-Carboxy- $\Delta$ -TetraHydroCannabinoid (18 years old, Male)
<b>Blood from peripheral area</b>			Toxic Concentration of DXM -2.42mg/L in combination with pseudoephedrine, Chlorpheniramine, Sertraline, Caffeine, Cannabinoid and Desmethylsertraline (18 years old, Male)
<b>Blood from peripheral area</b>			Lethal Concentration of DXM -19.5mg/L in combination with Chlorpheniramine. (13 years old, Male)
<b>Blood</b>	Samples extracted using Liquid-Liquid Solvent Extraction Protocol in basic medium.	Enzyme Multiplied Immunoassay Technique (EMIT), Thin Layer Chromatography (TLC) and GC-HS	Lethal Concentration of DXM -0.5mg/Kg in combination with pseudoephedrine and Brompheniramine (Infant, 2months) [59]

<b>Liver</b>			Lethal Concentration of DXM -0.57mg/Kg in combination with pseudoephedrine and Brompheniramine (Infant, 2months)
<b>Blood collected from cavity</b>	Collected samples from the subjects extracted using Liquid-Liquid Solvent Extraction in basic medium.	GC-MS	Toxic concentration of DXM -0.55mg/L [60]
<b>Brain</b>			Toxic concentration of DXM -1.33mg/Kg
<b>Liver</b>			Toxic concentration of DXM -0.90mg/Kg in combination with Pseudoephedrine, Ephedrine, , Ethanol and Acetaminophen (8 months old)
<b>Urine</b>			Toxic concentration of DXM -2.9mg/L in combination with Pseudoephedrine, Ephedrine, , Ethanol and Acetaminophen (12 months old)
<b>Liver</b>			Lethal concentration of DXM -0.29mg/Kg in combination in combination with Ephedrine, Pseudoephedrine, Acetaminophen.
<b>Blood collected from heart</b>			Lethal concentration of DXM -0.04mg/L
<b>Blood collected from heart</b>			Toxic concentration of DXM -0.03mg/L in combination with Ephedrine, Pseudoephedrine, Acetaminophen. (3 months old)
<b>Blood collected from cavity</b>			Lethal concentration of DXM -0.09mg/L
<b>Liver</b>			Lethal concentration of DXM -0.55mg/Kg in combination with Ephedrine, Pseudoephedrine, Acetaminophen. (5 months old)
<b>Peripheral Blood</b>	Samples extracted using Liquid-Liquid Extraction Protocol for acidic and basic drugs.	HPLC	All of the individuals tested positive for opiates, 8 tested positive for cannabis, and 2 tested positive for benzodiazepine. One subject's gastric contents included 1.1gm/dL ethanol.(18-45 years old, 30 fatal cases) [61]

<b>Contents of</b>			Concentration of DXM -4.22 to 92.06mg/Kg	
<b>Liver gastric</b>			Concentration of DXM -9.9 to 349.6mg/L (17-45 years old, 7 fatal cases)	
<b>Plasma and Urine</b>	Using a convenient solvent extraction procedure along with its respective metabolites was separated out from the given matrices.	Cyano Column HPLC and Fluorescence Detector	LOD for 3 Hydroxymorphinan and 3-monoxymorphinan -0.5ng/mL LOD for DXM and DXO -1ng/mL	[62]
<b>Urine</b>		GC-MS and TLC	DXM -7.57mg/mL; DXO -3.12mg/mL (34 years old, Male)	[55]
	Sample of urine was added with 50µL Methanol solution and Lidocaine was used as internal standard. The pH of the medium was made basic by adding 6N NaOH (pH=11)		DXM -1.47mg/mL; DXO -1.66mg/mL (30 years old, Female) DXM -1.88mg/mL; DXO -0.68mg/mL (30 years old, Male) DXM -6.82mg/mL; DXO -16.67mg/mL (53 years old, Male) DXM -4.43mg/mL; DXO -23.75mg/mL (35 years old, Male) DXM -16.44mg/mL; DXO -13.48mg/mL (30 years old, Male) DXM -9.79mg/mL; DXO -7.12mg/mL (30 years old, Male) DXM -0.17mg/mL; DXO -0.41mg/mL (34 years old, Male) DXM -0.16mg/mL; DXO -1.12mg/mL (43 years old, Male) DXM -3.49mg/mL; DXO -1.37mg/mL (40 years old, Male)	

<b>Postmortem Blood</b>	Samples from 19 deceased subjects were collected and extracted using Chloroform in basic medium. The organic layer was separated out, dried and reconstituted with 100mL of Methanol	GC Coupled to inert Mass Selective Detector (MSD) with an inert source operating in Electron impact mode for Qualitative Analysis and RP-HPLC Method for Quantification.	LOD -0.50µg/mL and 1.5µg/mL Concentration of DXM -7.3mg/L to 41.7 mg/L (n= 19 specimens of postmortem blood along with Chlorpheniramine)
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## CONCLUSION

DXM is widely accessible, appearing in over hundreds of prescription and non-prescription cough and cold treatments, and it is widely renowned for its efficacy and safety at therapeutic dosages. Because dextromethorphan misuse is largely committed by adolescents, prompt treatment of dextromethorphan addiction is an essential component of avoiding broader drug abuse among these adolescents. It is critical that the general public is aware of this fact and that medical practitioners are capable of recognizing, treating, referring, and educating persons who exhibit problematic usage or who arrive inebriated from this growing substance of abuse. When DXM's intoxication occurs, the effects on the CNS are dominating, with additional significant symptoms such as tachycardia, nausea, hypertension, and vomiting arising. In addition, these patients should be evaluated for intoxication from other medicines that may have been co-ingested with DXM, like acetaminophen or antihistamines. Dextromethorphan is affordable, widely discussed among youngsters on a number of internet sites, widely considered as safe, and widely available through a variety of marketing channels. All of these elements contribute to the perception of dextromethorphan's assumed safety, strengthening its status as a novel and growing drug of abuse. As dextromethorphan becomes a new drug of abuse among the young generation, it is critical that the people be aware of this trend and that clinical and substance addiction experts recognize, cure, refer, and educate people at risk of dextromethorphan overuse. When all of these issues are considered together, they contribute to a variability in DXM toxicological and clinical aspects, and a better understanding of these may help demarcate strategic usage of this drug, contemplating its potential applications, allowing for a more efficient and safe use of Dextromethorphan. DXM's misuse is a potential future social concern, and the provision of DXM OTC to everyone age groups must be reconsidered.

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