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Special Issue

Fatal Intoxications with 25B-NBOMe and 25I-NBOMe in Indiana During 2014

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Over the last few years, NBOMe substances have been used either as a legal alternative to lysergic acid diethylamide (LSD) or sold surreptitiously as LSD to unknown users. These NBOMe substances have been detected in blotter papers, powders, capsules and liquids. We report the deaths of two teenage male subjects that were related to 25B-NBOMe and 25I-NBOMe in Indiana during 2014. Samples were extracted via a solvent protein precipitation with acetonitrile and analyzed via ultra-performance liquid chromatography with tandem mass spectrometry. For these two cases, we describe the NBOMe instrumental analysis, toxicological results for postmortem heart blood and urine specimens and the relevant case history and pathological findings at autopsy. In the first case, 25B-NBOMe was detected in postmortem heart blood at 1.59 ng/mL; in the second case, 25I-NBOMe was detected in postmortem heart blood at 19.8 ng/mL. We also review relevant published casework from clinical toxicology and postmortem toxicology in which analytically confirmed 25B-NBOMe and 25I-NBOMe were determined to be causative agents in intoxications or deaths.

Introduction

The NBOMe series of compounds were developed in the early 2000s during academic research for purposes of investigating the serotonin receptor (5-HT) system via acting as radiotracers for positron emission tomography (PET). 25B-NBOMe and 25I-NBOMe were first synthesized by Ralf Heim in 2003 at the Free University of Berlin and further studied a few years later by Dr David Nichols group at Purdue University. 25B-NBOMe is also known as 2-(4-bromo-2,5-dimethoxyphenyl)-*N*-[(2-methoxyphenyl)methyl]ethanamine and is a *N*-(2-methoxy)benzyl derivative of the phenethylamine substance, 2C-B. The molecular formula is $C_{18}H_{22}BrNO_3$. The carbon-11-labeled version of 25B-NBOMe is named Cimbi-36 and acts as a potent agonist at the 5-HT_{2A} receptor. 25I-NBOMe is also known as 2-(4-iodo-2,5-dimethoxyphenyl)-*N*-[(2-methoxyphenyl)methyl]ethanamine and is a *N*-(2-methoxy)benzyl derivative of the phenethylamine substance, 2C-I. The molecular formula is $C_{18}H_{22}INO_3$ and the molecular weight is 427.2 g/mol. The carbon-11 labeled version of 25I-NBOMe is named Cimbi-5 and also acts as an agonist at the 5-HT_{2A} receptor (1–3). The chemical structures of these two compounds are detailed in Figure 1.

NBOMe substances have been available over the Internet for the last several years and have been sold either as a replacement for lysergic acid diethylamide (LSD) or under the guise as LSD itself. They are often used sublingually or buccally on blotter paper, but it has been reported that the substances can be insufflated nasally or injected intravenously (4, 5). In November 2013, the United States Federal government used its emergency scheduling powers to place three NBOMe compounds (25B-NBOMe, 25C-NBOMe and 25I-NBOME) into Schedule I of the Controlled Substances Act (6).

We report two unrelated deaths associated with 25B-NBOMe and 25I-NBOMe in the state of Indiana during 2014 and discuss the detection of these compounds in postmortem blood casework in conjunction with pertinent case history and pathological findings at autopsy.

Testing protocol

During autopsy, the forensic pathologist collected blood specimens in the supplied polypropylene tubes and bottles which contained sodium fluoride and potassium oxalate. Urine was collected in a polypropylene container that did not contain any additives. The specimens were sent to AIT Laboratories' facility for toxicological analyses.

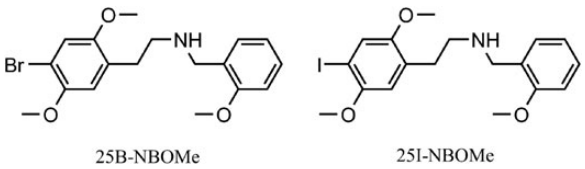
Standard screening analyses included an enzyme-linked immunosorbent assay (ELISA) screen for opiates/oxycodone and cannabinoids, a comprehensive screen (305 other substances) via liquid chromatography time of flight mass spectrometry (LC/ToF), and a headspace gas chromatography with flame ionization detection assay (GC-FID) for acetone, ethanol, isopropanol and methanol. All confirmatory assays were completed via liquid chromatography tandem mass spectrometry (LC–MS-MS).

Materials and methods

The 25B-NBOMe, 25I-NBOMe and 25I-NBOMe-d_3 reference standards were obtained from Cayman Chemical Company (Ann Arbor, MI). Acetonitrile, formic acid and methanol were obtained from Thermo Fisher Scientific (Pittsburgh, PA). Deionized (DI) water (18.2 MΩ cm) was obtained from the laboratory's water treatment system. All reference standards were diluted with methanol to working standard concentrations 10, 1, and 0.1 μg/mL. Negative whole blood was acquired from past postmortem casework and used for negative quality control matrix. Positive quality control specimens were fortified with analytes of interest at 1.5 and 6 ng/mL.

25B-NBOMe and 25I-NBOMe were analyzed via quick solvent protein precipitation extraction and ultra-performance liquid chromatography with tandem mass spectrometry. A 1 mL aliquot of acetonitrile held at −10°C containing 25I-NBOMe-d_3 (1.25 ng/mL) as an internal standard was added to 250 μL of specimen, vortex mixed for 2 min and centrifuged for 3 min at 3,000 rpm. A 500 μL portion of the organic supernatant was transferred to a new test tube and evaporated to dryness under nitrogen gas flow. The residue was reconstituted in 200 μL of DI water and transferred to a plastic auto sampler vial for instrumental analysis.

Instrumental analysis was completed via a Waters Acquity Ultra-Performance Liquid Chromatograph coupled to a Waters Tandem Quadrupole Detector (TQD) mass spectrometer. A gradient elution was performed using 0.1% formic acid in DI water



25B-NBOMe 25I-NBOMe

Figure 1. Chemical structures of 25B-NBOMe and 25I-NBOMe.

(mobile phase A) and 0.1% formic acid in acetonitrile (mobile phase B) as mobile phases at a flow rate of 0.5 mL/min. The initial gradient was 5.0% mobile phase B and was held for 0.5 min at which time it was then linearly increased to 40% at 4.0 min. From 4.0 to 4.7 min, mobile phase B was increased to 100%. At 4.7 min, it was returned to 5.0%. The total run time was 5.5 min. Ten microliters of specimen extract were injected onto a Waters Acquity BEH C18, 2.1 × 100 mm, 1.7 μm analytical column held at 50°C. The retention times for 25B-NBOMe, 25I-NBOMe and 25I-NBOMe-d_3 were 4.3, 4.5 and 4.5 min respectively.

Mass spectrometry was performed in positive electrospray ionization multiple reaction monitoring mode. Capillary and extractor voltages were 0.6 kV and 3 V respectively. Source temperature and desolvation temperature were 140 and 450°C, respectively. The desolvation gas flow and collision gas flow were 850 L/h and 0.3 mL/min, respectively. Two ion transitions were monitored for the analytes of interest and one ion transition was monitored for the internal standard. The transitions for 25B-NBOMe were 382.0 > 90.9 (cone voltage, 32 V; collision energy, 57 eV) and 382.0 > 121.0 (cone voltage, 32 V; collision energy, 18 eV). Transitions for 25I-NBOMe were 428.0 > 121.0 (cone voltage, 16 V; collision energy, 24 eV) and 428.0 > 91.0 (cone voltage, 16 V; collision energy, 58 eV). The transition for 25I-NBOMe-d3 was 431.1 > 92.1 (cone voltage, 30 V; collision energy, 60 eV). All dwell times were 0.02 s.

Method validation and results

The analytical method for 25B-NBOMe and 25I-NBOMe analysis in whole blood was validated as a quantitative assay. The overall method validation protocol and acceptance criteria used were the same as a previously published method validation procedure and is a standard protocol followed in our laboratory (7). Linearity, including limit of quantitation (LOQ) and limit of detection (LOD), accuracy and imprecision, ion suppression, carryover and exogenous drug interferences were assessed. The method validation results are detailed in Table I. In summary, the method was linear from 0.5 to 20 ng/mL for both 25B-NBOMe and 25I-NBOMe. Calibration curve points were 0.5, 1, 2, 5, 10 and 20 ng/mL. The LOQ for both analytes was 0.5 ng/mL and the LOD for both was 0.2 ng/mL. No lower concentrations of drug were analyzed for LOD determination. It was determined that the deuterated 25I-NBOMe internal standard did compensate for ion suppression (or enhancement) of the analyte signal. No carryover was detected in blank extracts injected immediately following a blood specimen fortified with 200 ng/mL of the analytes of interest. After analyzing 150 routine drugs and metabolites, no exogenous drug interferences were documented. A chromatographic representation of 25B-NBOMe, 25I-NBOMe and the internal standard ion transitions is shown in Figure 2.

Table I
Method Validation Results

Parameter	25B-NBOMe	25I-NBOMe
LOD	0.2 ng/mL	0.2 ng/mL
LOQ	0.5 ng/mL	0.5 ng/mL
Linearity	0.5–20 ng/mL	0.5–20 ng/mL
Coefficient of determination (R^2)	0.9980–0.9999	0.9987–0.9999
Accuracy		
1.5 ng/mL		
Intrarun	96.2–101.4%	94.1–100.2%
Interrun	98.1%	97.4%
6 ng/mL		
Intrarun	94.2–102.3%	95.9–102.0%
Interrun	99.3%	99.8%
Imprecision		
1.5 ng/mL		
Intrarun	0.8–5.6%	1.1–6.7%
Interrun	3.2%	4.6%
6 ng/mL		
Intrarun	1.1–6.6%	0.4–3.7%
Interrun	4.5%	2.5%
Ion suppression		
1.5 ng/mL		
Matrix effect	1.10 (5.6% CV)	1.05 (6.9% CV)
Response effect	1.05 (8.2% CV)	1.08 (9.4% CV)
10 ng/mL		
Matrix effect	0.98 (8.9% CV)	1.03 (6.9% CV)
Response effect	1.04 (4.4% CV)	1.00 (9.8% CV)
16 ng/mL		
Matrix effect	0.93 (5.1% CV)	1.04 (4.2% CV)
Response effect	0.98 (3.9% CV)	1.02 (7.7% CV)
Carryover	None detected at 200 ng/mL	None detected at 200 ng/mL
Exogenous interferences	None detected	None detected

Results

Case 1

An 18-year-old male who was with his brother and a friend ingested a drug they referred to as 'NBOMe'. According to scene investigators, the drug was bought on small pieces of paper that were ~2 cm^2 in size. The male allegedly ingested two of the squares. It was unknown if he consumed the paper sublingually/buccally or if he orally swallowed it. After ingestion, behavior became destructive as the individuals began breaking glass and other household objects and acting disruptive. After a short time the male was witnessed to collapse onto the kitchen floor and became unresponsive. Emergency medical personnel were called and transported the decedent to a local hospital where he was pronounced nonviable shortly after arrival.

An autopsy revealed external signs of superficial blunt force trauma including lacerations, contusions and abrasions to the body. Four superficial incised wounds were observed on the sole of the left foot. On internal examination, the right and left lungs weighed 1,160 and 1,060 g, respectively. Serial sectioning of the lungs revealed severe and diffuse pulmonary edema present within all five pulmonary lobes. There was aspiration of gastric contents present in the bronchi. The autopsy revealed no significant injuries or natural disease. Toxicological analyses on the postmortem heart blood were positive for 25B-NBOMe (1.59 ng/mL), Delta-9-tetrahydrocannabinol (2.4 ng/mL), 11-nor-9-carboxy-tetrahydrocannabinol (17.1 ng/mL) and caffeine (qualitative). Urine was positive for 25B-NBOMe (qualitative) and 11-nor-9-carboxy-tetrahydrocannabinol (361 ng/mL). No other substances were detected in the blood or urine.

Based on the combination of the scene investigation, autopsy findings and toxicology results, the cause of death was certified

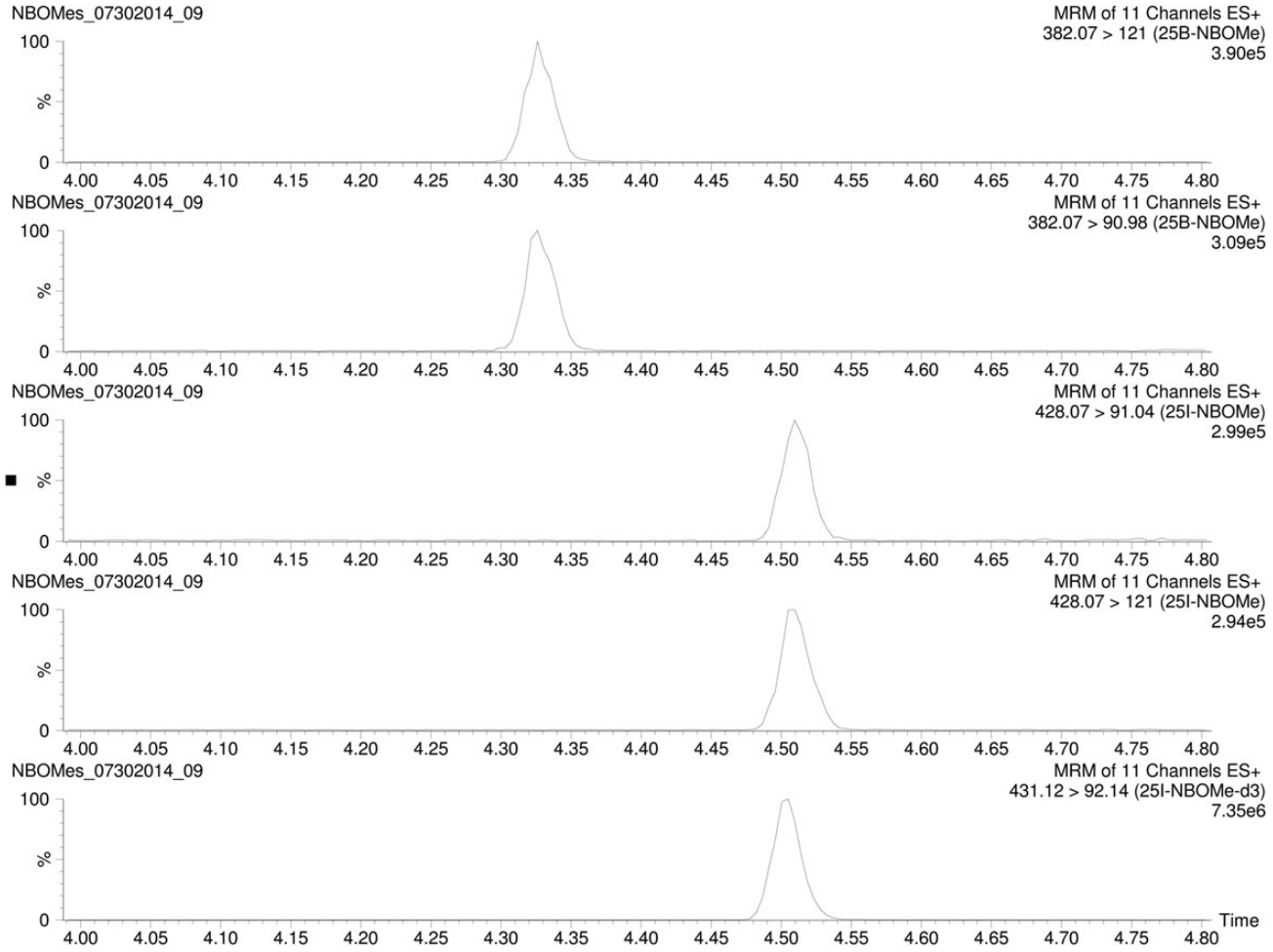


Figure 2. Chromatographic representation of 25B-NBOMe, 25I-NBOMe and the internal standard ion transitions.

as acute intoxication by 25B-NBOMe with the manner of death as accident.

Case 2

A 16-year-old male was partying with friends and used a drug that was spotted on blotter paper. The individual was found unresponsive in the morning by one of his friends. Drugs and a large amount of broken glass were present at the scene. Emergency medical personnel were called and the male was pronounced nonviable at the scene shortly after their arrival.

An autopsy revealed multiple red contusions and superficial abrasions present over the chest, arms, neck, bilateral knees and the bilateral shins. On internal examination, the right and left lungs weighed 810 and 660 g, respectively. Serial sectioning of the lungs revealed severe and diffuse pulmonary edema present within all five pulmonary lobes (right > left). The bronchial tree contained frothy edematous fluid. Mild cerebral edema was also observed. The autopsy revealed no significant injuries or natural disease. Toxicological analyses on the postmortem heart blood were positive for 25I-NBOMe (19.8 ng/mL). Urine was positive for the presence of 25I-NBOMe (qualitative). No other substances were detected in the blood or the urine. Based on the combination of the scene investigation, autopsy findings and toxicology results, the cause of death was listed as acute intoxication by 25I-NBOMe with the manner of death as accident.

Discussion

Including the two described cases, *analytically confirmed* 25B-NBOMe and 25I-NBOMe have been associated with many individual intoxications in published clinical toxicology case reports and five deaths in postmortem toxicology reports. Four of the postmortem reports have been associated with 25I-NBOMe. 25B-NBOMe had not previously been reported as a certified cause of death in postmortem toxicology casework until this case series.

In clinical toxicology casework, Kelly *et al.* reported a case series of four patients who presented to the emergency department after insufflation or oral ingestion of 25I-NBOMe. Tachycardia and psychomotor agitation were observed in all individuals. In three of the four patients, seizures were documented. One patient developed rhabdomyolysis and renal failure. The presence of 25I-NBOMe was detected in three of the patient's urine specimens at concentrations equal to 2, 28 and 36 ng/mL (8). Rose *et al.* described the intoxication of an 18-year-old male who presented to the emergency department with severe agitation and hallucinations. He had previously jumped out of a

moving vehicle. At the hospital he exhibited tachycardia and hypertension and was treated with intravenous lorazepam and ziprasidone, but due to his behavior, he required physical restraints. Over 2 days, he returned to baseline behavior. A serum specimen acquired at hospital admission was positive for 25I-NBOMe at 0.76 ng/mL (9). Stellpflug *et al.* described the case of an 18-year-old female who presented to the emergency department with agitation, confusion, hypertension, seizure and tachycardia. She was treated with intravenous fluids and benzodiazepines and was discharged several hours post-admission. An admission urine specimen was positive for 25I-NBOMe at 7.5 ng/mL (10). Poklis *et al.* described the severe intoxication of a 19-year-old male who was found unresponsive. He exhibited seizure activity and was admitted to the hospital. Hospital admission serum and urine specimens were positive for 25B-NBOMe at 0.18 ng/mL and 1.9 ng/mL, respectively (11). Tang *et al.* described the case of two males, 17 and 31 years of age, who both ingested substances labeled 'NBOMe' and 'Holland Film'. They experienced agitation, confusion, convulsions, deranged liver function, diaphoresis, hypertension, hyperthermia, mydriasis, rhabdomyolysis and tachycardia. Both individuals were treated with benzodiazepines. One of the individual's urine specimens was positive for 25B-NBOMe and the other individual's urine specimen was positive for 25B-NBOMe and 25C-NBOMe (12). Laskowski *et al.* reported a case in which a 15-year-old male reported use of 25I-NBOMe and subsequently developed bizarre behavior and experienced two generalized seizures along with persistent muscle rigidity. He was treated with benzodiazepines and discharged within 24 h of presentation at the hospital. A hospital admission serum specimen was positive for 25B-NBOMe (13). Hill *et al.* described seven cases of intoxication with 25I-NBOMe in the UK. The presence of 25I-NBOMe was confirmed positive in hospital admission specimens in all seven cases. The drug was administered in various routes including intravenous injection, nasal insufflation of powder and oral ingestion of powder and capsules. Common observed symptoms across the cases included aggression, agitation, hallucinations, hypertension, seizure and tachycardia (14). Poklis *et al.* briefly described emergency room presentations in which four patients exhibited agitation, hypertension, seizures and tachycardia. 25I-NBOMe was detected in three of urine specimens analyzed. One specimen was positive for 25I-NBOMe (<1 ng/mL). The second urine specimen was positive for 25B-NBOMe (1.7 ng/mL). The third specimen contained 25I-NBOMe (2.3 ng/mL) and 25C-NBOMe (<1 ng/mL). The fourth specimen was positive for 25I-NBOMe (1.2 ng/mL) and 25H-NBOMe (<1 ng/mL) (15).

Postmortem toxicology case reports for 25B-NBOMe and 25I-NBOMe associated deaths are limited. Poklis *et al.* reported a traumatic death of a 19-year-old male who ingested a blotter that was allegedly 'acid'. His friends witnessed paranoid and bizarre behavior which ultimately led to the male walking away from the group and he went missing. The friends visited his apartment in search of him and he was found unresponsive outside. After investigation, law enforcement concluded the decedent had suffered a fall from his apartment balcony. Peripheral blood, heart blood and urine were positive for 25I-NBOMe at 0.405, 0.410 and 2.86 ng/mL, respectively (16). Walterscheid *et al.* described two cases in which death was associated with 25I-NBOMe toxicity. In the first case, a 21-year-old male admitted to taking 'acid' and suddenly exhibited agitation and violence. He began to flail about and then became unresponsive. In the second case, a 15-year-old female was mingling outside a rave party when she became ill. She was transported to the hospital, but deteriorated en route. 25I-NBOMe was qualitatively confirmed in blood and urine samples in both cases (17).

In both of the presented Indiana cases, destructive behavior was noted through broken glass and destroyed household objects at the scenes, which is consistent with other reports of agitated, destructive or violent behavior described in other casework (9–10, 12–17). Other than the external physical trauma from the observed behavior, relatively nonspecific pathological findings of pulmonary edema were observed in both cases. NBOMe substances act as potent full and partial 5-HT_{2A} receptor agonists. While one of the effects of 5-HT_{2A} receptor agonism is smooth muscle vasoconstriction, which may lead to an increase in blood pressure (hypertension) and potential constriction of the bronchi causing edema, pulmonary edema is considered nonspecific because it can emanate from other causes, such as a cardiogenic etiology. Only three of the previously described cases reported quantitative blood or serum toxicology results for 25B-NBOMe and 25I-NBOMe. The postmortem blood concentration for 25B-NBOMe reported in Case 2 (19.8 ng/mL) is elevated when compared with the 0.18 ng/mL serum concentration reported by Poklis *et al.* (11). There are no other reported postmortem blood concentrations to which we can compare. The 25I-NBOMe postmortem blood concentration reported in Case 1 (1.59 ng/mL) is similar to the reported postmortem blood concentrations (0.405 and 0.410 ng/mL) in the fatality reported by Poklis *et al.* (16) and the serum concentrations (0.76 ng/mL) in the intoxication described by Rose *et al.* (9), but still two to four times greater. When interpreting a blood drug concentration in a postmortem setting from a central source such as from the heart or central cavity, postmortem redistribution (PMR) should always be a factor that is taken into consideration. Generally drugs with larger volumes of distribution ($V_D > 3$ L/kg) are more prone to PMR. The V_D for 25B-NBOMe and 25I-NBOMe are not known. Of the limited postmortem toxicology case reports, there is only one in which both heart and peripheral blood specimens were analyzed (16). In the specific case, Poklis *et al.* analyzed both heart and peripheral blood for 25I-NBOMe and the heart blood to peripheral blood drug concentration ratio was 1.01. No peripheral blood specimens were available for analysis in the two presented cases.

In conclusion, very little is known about NBOMe compounds and their pharmacology and toxicology in the human body. Over the last 3 years, an increasing number of case reports describing overall adverse serotonergic effects and symptoms of intoxication due to these compounds appeared in the peer-reviewed literature; however, postmortem toxicology case reports remain few. Combining information derived from the scene investigation, the pathological findings at autopsy and blood/urine toxicology results, we describe two unrelated cases in which intoxication with NBOMe compounds (25B-NBOMe and 25I-NBOMe) was ruled as cause of death. These case reports display that NBOMes are substances of significant toxicological consequence. While a forensic toxicology laboratory may not be able to include NBOMe compounds in a general screening analysis, we recommend that laboratories include the NBOMe compounds in their scope of test offerings.

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