Postmortem Identification and Quantitation of 2,5-Dimethoxy-4-n-propylthiophenethylamine Using GC-MSD and GC-NPD*

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Abstract

2,5-Dimethoxy-4-n-propylthiophenethylamine (2C-T-7) has structural and pharmacodynamic similarities to methylenedioxymethamphetamine (MDMA). This compound was initially identified from a routine screening procedure in postmortem urine from a 20-year-old male that died in a local emergency room after reportedly insufflating 35 mg. This report describes the development of a quantitative method for 2C-T-7. A number of method parameters were studied including internal standard selection, liquid-liquid extraction scheme, and drug stability in preserved refrigerated blood. The adopted method for blood and urine involves the addition of trimethoxyamphetamine (TMA) as internal standard, alkalinization with ammonium hydroxide, and liquid-liquid extraction with n-chlorobutane. To facilitate recovery from liver, a 1:4 aqueous homogenate was pretreated with dilute perchloric acid, centrifuged, and the supernatant was extracted as previously described. In each case, 0.1% hydrochloric acid in methanol was added during the final concentration step to prevent loss of drug caused by evaporation. Samples were analyzed by gas chromatography with nitrogen-phosphorus detection (GC-NPD) and electron ionization GC-mass spectrometry (MS) utilizing selected ion monitoring. For the GC-MS analysis, the characteristic ions monitored for 2C-T-7 were m/z 226, 255, and 183 and for TMA, m/z 182. The limits of detection and quantitation in blood were 6.0 and 15.6 ng/mL, respectively, by both GC-NPD and GC-MS. The results from the postmortem case were as follows: heart blood, 57 ng/mL; femoral blood, 100 ng/mL; urine, 1120 ng/mL; and liver, 854 ng/g.

Introduction

The hallucinogen 2,5-dimethoxy-4-n-propylthiophenethylamine, also known as 2C-T-7 (Figure 1), has been sold in the United States under the names “Blue Mystic”, “Beautiful”, “Belladonna”, and “Tweetbird Mescaline” (1). First synthesized by Alexander Shulgin in January 1986 (2), 2C-T-7 has only recently appeared on the street, along with its cousins 2C-T-2 (2,5-dimethoxy-4-ethylthiophenethylamine, which was banned in the Netherlands in 2000) and paramethoxyamphetamine (PMA). 2C-T-7 is a phenethylamine derivative that has structural similarities to mescaline and methylenedioxymethamphetamine (MDMA). The naming system Shulgin used paralleled the Aleph series of amphetamine-based homologues he had synthesized earlier, in which the terminal number indicates the order in which the series was synthesized. In the 2C-T series, the last number is the same as the corresponding Aleph analogue. The designation 2C represents the 2-carbon aminoethane chain of phenethylamine, and T stands for the thio (sulfur) substitution. Thus, 2C-T-7 (4-n-propylthio) is the phenethylamine analogue to Aleph-7 (2,5-dimethoxy-4-n-propylthioamphetamine) (3). 2C-T-7 is a phenethylamine psychedelic with effects similar to mescaline and MDMA (4). Anecdotal information indicates that the effects of 2C-T-7, when taken orally, develop relatively slowly over a period of 1-2 h, even taking up to 3 h for full effects, and last 8-15 h. When “snorted”, the effects occur rapidly (5-30 min) and only last 4-8 h (2). The effects of psychedelic drugs are difficult to describe, and user reports are highly subjective. The subjective effects tend to be better described as a range of effects and how they compare between drugs than attempting to focus on specific aspects. 2C-T-7 trips involve strong visual effects for most people, which are described as realistic rather than abstract. The imagery is described as coherent and thematic. Anxiety, paranoia, delirium, and extreme panic attacks reportedly occur, particularly when insufflated and especially with novice hallucinogen users. Negative aspects are described as similar to MDMA, but milder with some reporting a temporary aphasia and impaired recall (2).

2C-T-7 has been associated with four deaths in Oklahoma, Tennessee, and Washington since January 2000 (personal communication, L. Kuykendall, Tennessee Bureau of Investigation,
August 2001). The toxicity, as well as the pharmacokinetics, of this drug has never been formally investigated. The appearance of 2C-T-7 on the street with the associated deaths creates an increased importance for characterizing the forensic aspects of its pharmacology including generating an analytical method for measuring postmortem 2C-T-7 fluid and tissue concentrations, elimination kinetics, and the potential for postmortem redistribution.

The purpose of this manuscript is to describe a postmortem case involving the ingestion of 2C-T-7 and the steps taken to initiate pharmacological studies of 2C-T-7 by developing a quantitative method in postmortem fluids and tissues.

Case History

A 20-year-old white male presented to a local emergency room in cardiac arrest and was subsequently pronounced dead. The young man was brought by acquaintances that gave this initial history. The decedent knocked at their door, appeared agitated, and told them he had taken a “designer” drug and was in distress. The decedent began convulsing and vomiting, at which point they brought him by car to the emergency room. Along the way, they noticed he had stopped breathing and attempted to clear the airway, but the decedent died in route. The acquaintances implied the possibility that the drug ingested was gamma-hydroxybutyrate (GHB), MDMA, or something similar.

Some time later, the decedent’s father called the medical examiners office with the information that his son may have taken a new designer drug. A fax from the local police department investigating the case stated the decedent might have taken GHB in combination with something called 2C-T-7. Further investigation revealed a different story of the circumstances occurring before the time of death. The decedent was at a private gathering at which he and two others used 2C-T-7. The decedent insufflated approximately 35 mg of the drug. Within 20 min, he became agitated, frightened, and cold. In 60–90 min, he experienced “mild vomiting”, but was otherwise “fine”. At approximately 90 min after snorting the drug, he began seizing, with extreme vomiting and bleeding from the nose. At this point, the others present put him in a car and headed for the hospital. The decedent subsequently died in route to the emergency room. The Office of the Chief Medical Examiner of Oklahoma took jurisdiction of the case as it met the statutory criteria of an unexpected drug-related death. During the investigation, literature was found at the decedent’s home on tryptamine derivative produced naturally by a species of toads.

Materials and Methods

The fluid specimens received for toxicological studies in our laboratory were preserved with sodium fluoride (NaF) and stored at 4°C. Tissues were stored at −20°C. Routine toxicology screens run on this case included headspace gas chromatography (GC) for volatiles, GC, GC–mass spectrometry (MS), color testing, and microplate enzyme immunoassay (MPEIA) (OraSure Technologies, Inc., Bethlehem, PA). MPEIA was performed on whole blood per the manufacturer’s instructions using serum-based kits. In addition, special screens for GHB by high-performance liquid chromatography (HPLC) and fumitrazepam by GC–MS were performed. Thin-layer chromatography (TLC) (ToxiLab®) was performed on urine per manufacturer instruction (5) to investigate 2C-T-7’s characteristics with this system. No testing was performed to identify MEO-DMT because of a lack of analytical data and standards.

The routine screen for basic drugs in our laboratory was performed on 2.5 mL postmortem whole blood. Full MS confirmation of 2C-T-7 in this case study required extracting 6.0 mL of heart blood and the increase of injection volume from 1.0 to 3.0 mL. Additionally, the purge valve opening was delayed from 0.30 to 0.50 min.

Materials

Trimethoxyphephinamine (TMA) was obtained from Sigma Chemical Corporation (St. Louis, MO). 2C-T-7 (Figure 2) was synthesized and provided by the United States Drug Enforcement Administration (DEA). Additional 2C-T-7 was obtained from Western Biosynthesis (Las Vegas, NV). Perchloric acid solution (6%, v/v) was prepared by the addition of 8.8 mL 70% perchloric acid (ACS grade, Fisher Scientific, Fairlawn, NJ) to 100 mL deionized water. All other reagents and chemicals were reagent grade or better.

Extraction

Internal standard selection. Based upon structural similarities, two compounds were chosen for possible internal standards for this assay. These were 3,4,5-TMA and methylenedioxypromethophene (MDPA). Comparison of these two internal standard candidates was accomplished by extracting three calibration curves on different days, consisting of five points each. Standards were prepared from serial dilutions in methanol and added to 1.0 mL of blank blood to yield 2C-T-7 concentrations of 4.0, 2.0, 1.0, 0.50, and 0.25 µg/mL. Both extraction schemes 1 and 4 (following) were used for this evaluation.

Blood extraction comparison. A comparison of several extraction schemes was performed in order to optimize recovery and sensitivity. Because it is common for sample volumes to be quite limited in forensic cases, the goal was detection of 20 ng/mL of 2C-T-7 using 1.0 mL of blood.

Four extraction procedures were compared using 1.0-mL
blood aliquots spiked with 800 ng 2C-T-7 and 200 ng TMA (internal standard). They were analyzed on an HP 5890 (Hewlett-Packard, Palo Alto, CA) GC equipped with a nitrogen-phosphorus selective detector (GC–NPD). The temperature program for all experiments is described in the Instrumentation subsection.

Scheme 1 was a modification of a previously published extraction (6,7) used for the initial screening for weak base drugs in our laboratory. Briefly, 0.5 mL concentrated ammonium hydroxide was added to 1.0 mL blood along with 7.5 mL n-chlorobutane. The samples were rotoextracted for 10 min and centrifuged for 5 min at 2500 rpm. The supernatant was transferred to a clean glass test tube, and 2.5 mL of 1.0N sulfuric acid was added. The tubes were capped, rotoextracted, and centrifuged as described previously. The organic solvent was removed to waste. Ammonium hydroxide (0.5 mL) and 2.5 mL n-chlorobutane were added. The tubes were rotoextracted and centrifuged. The supernatant was transferred to a 7.0-mL glass conical centrifuge tube and evaporated to residue in a waterbath (40°C) under a stream of nitrogen. The samples were then reconstituted in 50 µL of methanol.

Scheme 2 consisted of the extraction procedure in Scheme 1, with the substitution of dichloromethane for n-chlorobutane.

Scheme 3 consisted of an alkalinized single-stage partition into 2% isopropanol/pentane. Concentrated ammonium hydroxide (0.5 mL) was added to 1.0 mL blood along with 5.0 mL 2% isopropanol/pentane. The samples were rotoextracted and centrifuged. The supernatant was transferred to a 7.0-mL glass conical centrifuge tube and evaporated to residue, followed by reconstitution in 50 mL methanol. This scheme was selected because of the projected lipophilic character of the 4-propylthio group of 2C-T-7.

Scheme 4 consisted of an alkalinized partition into n-chlorobutane as in Scheme 1, but omitting the sulfuric acid back extraction. The extracts were then dried to residue and reconstituted in 50 µL of methanol.

Tissue recovery experiments. Initial testing using routine assays on the postmortem case indicated that recovery of 2C-T-7 from tissues would be difficult. A series of experiments were undertaken to address this issue. All recoveries in the following experiments were determined by analysis on GC–MS with direct peak-area comparison to unextracted 2C-T-7.

To investigate the effect of various solvent systems on recovery from tissues, the four extraction schemes described previously were performed on liver. In addition, 5% isoamyl alcohol in toluene and methyl-tert-butyl ether were substituted in scheme 4 for n-chlorobutane.

An initial hypothesis was that undissolved residue in the final reconstitution step was interfering with recovery. We therefore designed an experiment using a variety of solvents in the final reconstitution step. Homogenates of liver and brain (1:4, w/w, in water) were prepared. Liver homogenate (1.0 g) and 1.0 g brain homogenate were spiked with 400 ng 2C-T-7. These preparations were then extracted using scheme 4 (see the Extraction section). The dried extracts were reconstituted with hexane, acetone, isopropanol, methanol, or chloroform.

Finally, the hypothesis that sample pretreatment with perchloric acid would enable analysis of 2C-T-7 by releasing tissue-bound drug was tested. One milliliter of liver homogenate (1:4, w/w) was spiked with 200 ng 2C-T-7 (n = 2). Three milliliters of 6% (v/v) perchloric acid was added to each tube. The tubes were then mixed, centrifuged at 2500 rpm, and the supernatants were transferred to clean test tubes for extraction using extraction scheme 4.

The results from the internal standard selection, solvent scheme comparison, and tissue recovery experiments resulted in the adoption of the final procedure described next.

Standard curves over the linear range for 2C-T-7 were prepared from serial dilutions in methanol of a 1.0-mg/mL (in methanol) stock solution. Amounts of 100 µL of the serial dilutions were added to 1.0 mL of blank human blood in 15-mL screw-top test tubes. One milliliter of blood or urine or 1.0 g tissue homogenates (1:4, w/w, in water) was added to 15-mL screw-top test tubes in duplicate. To the standards and case specimens, 200 ng TMA was added as an internal standard. To the tissue homogenates was added 3.0 mL of 6% (v/v) perchloric acid, followed by a 15-min rotation on a rotary extractor. The tissues were centrifuged at 2500 rpm for 5 min, and then the supernatant was transferred to another 15-mL test tube. The pH of all tubes was adjusted to pH > 10 by addition of 0.5 mL concentrated ammonium hydroxide. Six milliliters n-chlorobutane was added, and the tubes were rotated on a rotary extractor for 10 min, then centrifuged at 2500 rpm for 5 min. The upper layer was transferred to a 5.0-mL glass conical centrifuge tube and placed in a 40°C water bath under a stream of nitrogen. Twenty microliters of 0.1% HCl in methanol was added when the chlorobutane was evaporated to approximately half volume. Evaporation was then continued to dryness. The extracts were then reconstituted in 50 µL of methanol and analyzed by GC.

A precision study on the final method was performed in blood at 2C-T-7 and TMA concentrations of 100 ng/mL. For intraday precision, 1.0-mL blood samples (n = 5) were extracted. For interday precision, 1.0-mL blood samples (n = 5) were extracted on three different days. In each case, the peak-height ratios between analyte and internal standard were used for calculating mean and standard deviation values.

Accuracy of the extraction was determined on GC–NPD with 10 blank blood samples spiked at a concentration of 62.5 ng/mL (the middle of the linear range).

The linearity of the assay was determined by linear regression analysis of duplicate standard curves run by GC–MS at concentrations of 2C-T-7 ranging from 15.6 to 250 ng/mL. The limit of detection (LOD) was defined as the lowest concentration of 2C-T-7 that could be identified with 3 ions. The limit of quantitation (LOQ) was defined as the lowest curve point of the linear range.

Stability studies. The decline of concentrations over time in stored specimens is a well-known problem for some drugs. In order to investigate this phenomenon for 2C-T-7, we took two different approaches. First, we prepared 100 ng/mL blood specimens at intervals of 27, 58, and 184 days before analysis (one specimen for each time point). They were preserved with sodium fluoride, stored at 4°C, and analyzed simultaneously by GC–MS. For the second study, we spiked two blood specimens at 200 ng/mL and stored them at 4°C with sodium fluoride.
preservative. They were analyzed at intervals of 18, 74, and 114 days after preparation. All analyses were performed in duplicate.

**Instrumentation**

The samples were analyzed using a Hewlett-Packard 6890/5973 GC-MS equipped with a 15-m × 0.25-mm × 0.25-mm HP-1MS capillary column and operated in electron ionization and selected ion monitoring modes. The characteristic ions monitored for 2C-T-7 were m/z 226, 255, and 183 and for TMA, m/z 182. The samples were run using splitless injection with a 0.3-min purge off time. The injector temperature was 250°C, and the transfer line was set at 300°C. The oven temperature program utilized an initial temperature of 120°C, which was increased to 300°C at a rate of 20°C/min.

A Hewlett-Packard 5890 GC with an NPD was also used in the method development. Separation was achieved with a 15-m × 0.25-mm × 0.25-mm HP-1 capillary column. The samples were run using splitless injection with a 0.5-min purge off time. The injector temperature was 250°C, and the detector temperature was set at 330°C. For the temperature program, the initial temperature was 120°C increased to 300°C at a rate of 20°C per min.

**Results and Discussion**

All routine toxicology tests on the postmortem blood from the case were negative. TLC on urine revealed spot migration and color profile similar to MDMA. Special screening for GHB and flunitrazepam in urine were negative. Routine analysis for basic drugs in urine revealed an unidentified peak at 11.27 min on GC-MSD. The full spectrum and total ion chromatogram of 2C-T-7 are noted in Figure 2. The principal electron impact mass spectra ions from highest to lowest abundance are m/z 226, 255, 183, 255, 169, 153, and 227. The m/z 255 ion is the molecular ion. Under our instrument conditions, there is a m/z 267 ion present in both case and standard spectra, which cannot be explained at this time.

The mass spectrum and GC relative retention times (RRT) for the 2C-T-7 provided by the DEA matched the data found in the decedent’s urine. No 2C-T-7 was detected in the decedent’s blood using the normal sample volume for analysis in our lab (2.5 mL). Increasing the sample volume to 6.0 mL allowed detection of the drug. Forensic blood specimens are often limited, however, so our method development targeted 1.0-mL sample volumes.

**Internal standard selection**

Internal standard methods are generally considered the most reliable means of quantitation. The internal standard is used to provide a way to account for interassay variability and, as such, ideally will exhibit the same chemical extraction characteristics as the analyte. Deuterated analytes currently provide the closest possible approach to this ideal, but have the negative aspects of a narrow range of available compounds and require mass spectral detection. Currently no commercial sources for deuterated 2C-T-7 have been located, except for the costly custom synthesis approach. The next best candidates are chemically related analogues. The closest analogues of 2C-T-7 are 2C-T-2 (differs by a 4-ethylthio group), 2C-T-4 (differs by a 4-isopropylthio group), and Aleph-7 (differs by a methyl group). The closest candidates available to us with similar functional groups were mescaline, 3,4,5-TMA, MDMA, and MDPA. 2C-T-7 elutes quite late for the phenethylamine/amphetamine drug group, and all these drugs eluted several minutes earlier. MDPA and TMA were selected as candidates because they eluted the latest of the four.

Comparison of these two candidates was accomplished by extracting three standard curves consisting of 5 points serially diluted and added to 1 mL blood to yield 2C-T-7 concentrations of 4, 2, 1, 0.5, and 0.25 μg/mL. Both extraction schemes 1 and 4 were used for this evaluation. In a comparison of linearity curves, TMA was deemed a better internal standard than MDPA. In both schemes 1 and 4, TMA gave consistently better linearity (r = 0.9999) over the range of 0.25–4.0 mg/mL of 2C-T-7. Extraction scheme 1 lost linearity for 2C-T-7 below 0.5

Figure 2. GC elution time and mass spectra of 2C-T-7 (11.27 min) from extract of 2.5 mL of postmortem urine. Oven program: 80–300°C (3-min hold) at 10°C/min.

Figure 3. Structure of internal standard TMA.
µg/mL. These data suggested that scheme 4 with TMA as the internal standard would be an acceptable method.

**Blood extraction comparison**

A liquid-liquid extraction of the analyte is one of the simplest most economical means of sample cleanup and preparation for GC analysis. With this in mind, a comparison of several extraction schemes was performed in order to optimize sensitivity. The case blood had required extraction of 6.0 mL in order to detect 2C-T-7. Because it is common for sample volumes to be quite limited in forensic cases, the goal set was detection of 20 ng/mL of 2C-T-7 using 1.0 mL of blood.

The results of the recovery experiments are found in Figures 4 and 5. In comparing the extraction efficiency of schemes 1 and 4, scheme 1 was found to yield a recovery 20% lower than scheme 4, as suggested by direct peak-area comparison (Figure 4). When compared with an unextracted standard (Figure 5), Scheme 4 yielded an extraction efficiency of 45%. The presence of undesirable sample matrix components is a concern with a single-stage extraction, but there does not appear to be any interference from blood components at 100 ng/mL 2C-T-7 when using GC–NPD. There were no interferences detected by GC–MS. The recoveries for schemes 2 and 3 (63% and 86.5% lower than scheme 4, respectively) were found to be significantly less efficient than schemes 1 and 4 and, thus, were eliminated from further consideration.

Precision experiments were performed for the final procedure at 100 ng/mL 2C-T-7. Intraday experiments yielded a mean peak-area ratio of 0.98 ± 0.02 [coefficient of variation (CV) = 2.1%]. Interday precision showed a mean peak-area ratio of 0.89 ± 0.09 (CV = 10%). The assay was determined by duplicate linear regression analysis to be linear over a range of 15.6–250 ng/mL (r = 0.999; y = 0.013x × 0.015). Multiple standard curves of varying ranges had been run; however, and these indicated that the upper limit of linearity was higher, up to 2.0 µg/mL.

The accuracy of the method was determined from 10 spiked blood samples at 62.5 ng/mL 2C-T-7. The mean value obtained from these assays was 63.4 ± 10.8 ng/mL.

The LODs and LOQs for the assay were determined to be 6.25 and 15.6 ng/mL, respectively. Figure 6 shows a total ion chromatogram of 6.25 ng/mL spiked in 1.0 mL blood.

**Tissue recovery experiments**

Several approaches were tried to enable the recovery of 2C-T-7 from tissues.

The first hypothesis was that tissue residue was interfering with the dissolution of the drug in the final reconstitution step. Hexane, methanol, isopropanol, acetone, and chloroform were tested with blank tissue and blood samples spiked with 400 ng/mL 2C-T-7. In the spiked blood samples, hexane yielded peaks for the internal standard only, whereas acetone yielded no peaks. Methanol, isopropanol, and chloroform showed both drug and internal standard peaks in the blood only. For liver and brain, only chloroform dissolved the entire visible residue in the extracts. However, no 2C-T-7 or TMA peaks were detected by GC–MS in these tissues. This would seem to indicate that the
problem with recovery is not excessive insoluble residues. There was a large variability seen with the tissue extraction solvent experiments. Using toluene with 5% isoamyl alcohol as an extraction solvent was successful in recovering drug from the tissues. The results, however, were not satisfactory because of loss of recovery caused by extremely dirty extracts. No other extraction scheme using various solvents including methyl tert-butyl ether resulted in any recovery from tissues. Any attempts to cleanup the extracts using back extractions or washes resulted in no recovery of drug. Pretreating spiked tissue extracts (n = 2) with dilute (6%, v/v) perchloric acid yielded some recovery of drug (33% and 24.3%, mean = 28.6%). Conversely, pretreating spiked blood with the perchloric acid greatly reduced recovery and was therefore not considered useful for this matrix.

Stability studies
The final analysis of the specimens from our postmortem case occurred after an extended period of time had elapsed because of the delay in obtaining drug standard. It is well known that certain drug concentrations may decline over time in stored specimens. In order to investigate this phenomenon for 2C-T-7, we took two different approaches. First, we prepared 100 ng/mL blood specimens at intervals of 27, 58, and 184 days before analysis. They were preserved with sodium fluoride, stored at 4°C, and analyzed simultaneously by GC–MS (Figure 7). We then spiked two blood specimens at 200 ng/mL and stored them at 4°C with sodium fluoride preservative. They were analyzed at intervals of 18, 74, and 114 days after preparation. Although there is a gap in the first set of data, the results agree that 2C-T-7 appears to be stable for approximately 70 days in sodium fluoride-preserved blood when stored at 4°C.

Postmortem case results
Final analysis of blood, urine, and liver from our postmortem case revealed the presence of 2C-T-7 at concentrations of 57 ng/mL in heart blood, 100 ng/mL in femoral blood, 1120 ng/mL in urine, and 854 ng/g in liver. These concentrations were based upon the adopted method with comparison with blood calibration curves up to 480 ng/mL. Initial analysis of the blood from our single postmortem case occurred approximately one year after the death of the decedent because primarily of the time to identify and obtain the drug standard. Although fluid specimens were preserved with sodium fluoride, our data suggests that 2C-T-7 levels begin to decline after approximately 70 days of storage at 4°C. Values at the time of death may have been higher. The liver from the postmortem case was stored at −8°C. The extended storage time between autopsy and analysis may have caused partial desiccation by the time recovery from tissues was accomplished at approximately 2.5 years from time of death. This, too, may have altered the concentration.

The relatively large concentration of 2C-T-7 in urine may indicate that a large percentage of 2C-T-7 is excreted intact in the urine. Further investigation for the analysis of brain and other fluids is ongoing, using both the postmortem case specimens and animal studies.

Conclusions
In this report, we have described the development of a new analytical method for the detection of the hallucinogen 2C-T-7 in postmortem blood and liver. Alkalinization with concentrated ammonium hydroxide, followed by a single-step extraction with n-chlorobutane and concentration in methanol provided sufficient recovery for the quantitation of this drug. Liver must be pretreated with perchloric acid to facilitate recovery.

The cause of death for this postmortem case was attributed to the toxicity associated with 2C-T-7 caused by the presence of this drug, as well as information obtained from field investigations and autopsy. More research is needed into this hallucinogen class of compounds. Animal studies are currently being conducted in our laboratory to further characterize the pharmacology of this drug. In addition, these studies will investigate postmortem redistribution of the drug under controlled conditions.

References