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## Dissociative Drugs of Abuse

### *Growing Family Includes Ketamine, Phencyclidine, and Their Analogs*

*By Matthew D. Krasowski, MD, PhD, and Kenichi Tamama, MD, PhD*

Ketamine and phencyclidine (PCP) are pharmacologically related drugs that produce dissociative effects on the central nervous system (CNS) characterized by feelings of detachment and sensory distortions. While PCP had only a brief run as a general anesthetic in the U.S. in the late 1950s and early 1960s before being abandoned on the clinical market, ketamine has been used as a clinical and veterinary general anesthetic since the 1970s.

Abuse of ketamine and PCP has been well-known since the 1960s and 1970s, with both drugs showing dramatic rises and falls in popularity in the subsequent decades. Recently, analogs of ketamine and PCP have appeared on the street and via Internet sources. Some of these analogs were synthesized and characterized decades ago as potential pharmaceuticals but have now emerged as "designer" dissociative drugs of abuse.

This article will first review ketamine and PCP as drugs of abuse and then discuss their analogs. The chemical structures of the compounds are shown in Figure 1 (ketamine and its analogs) and Figure 2 (PCP and its analogs). Table 1 summarizes compound names, classification, and controlled substance status.

#### Ketamine

Ketamine was first synthetized in 1962 and gained Food and Drug Administration (FDA) approval as a general anesthetic in 1970 (1). It has a short duration of action and produces dissociative anesthesia, a form of anesthesia characterized by analgesia (blockade of pain), amnesia (loss of memory), catalepsy (trance or seizure-like state with muscle rigidity), and catatonia (stupor and lack of movement).

One of the main clinical advantages of ketamine is its minimal effects on heart function, respiration, and airway reflexes, especially relative to other general anesthetics such as propofol or thiopental. This makes ketamine a good option for anesthesia in low resource or remote settings. It is used worldwide and is a core medication for intravenous anesthesia and sedation on the World Health Organization Model List of Essential Medicines (2). In clinical settings, it is usually administered intravenously or intramuscularly (1,3).

Reports of ketamine abuse first appeared in the 1970s with rising popularity in the club scene in the 1990s (4–6). Street names include special K, vitamin K, super K, and kit kat. Increasing abuse, including diversion from medical and veterinary supplies, led to its classification as a U.S. Drug Enforcement Administration (DEA) schedule III controlled substance in 1999 (4,6).

Ketamine is abused in the club setting and to enhance the sexual experience. Abusers often aim for what is known as the "k-hole," a state characterized by physical immobility and out-of-body experience (4). Abusers administer the drug by intravenous, intranasal, intramuscular, or smoking routes. It can be used alone or with other substances such as dextromethorphan (DXM; found in cough medications or via illicit sources) or gamma-hydroxybutyrate (GHB), another drug abused on the club scene.

Acute adverse effects include altered level of consciousness, amnesia, delirium, hallucinations, hyperthermia, impaired muscle tone, tachycardia, and hypertension (4). It can also cause perceptional alterations, including out-of-body experiences, altered sense of time, and color changes. Death is uncommon if ketamine alone is abused (7). There is currently no specific antidote for ketamine overdose.

Ketamine has been implicated in drug-

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## Inside...

# Dissociative Drug Analogs

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facilitated sexual assaults and other crimes that involve first incapacitating the victim (8). In a study in Ontario, Canada, ketamine was detected in 2.3% of suspected drug-facilitated sexual assault cases (9). Ketamine victims may appear conscious and even aware, complicating the ability to prove a lack of consent in legal cases. Ketamine's potent amnesiac properties can make it difficult or impossible for victims to recall events.

## Ketamine Testing

Immunoassays for ketamine screening in urine have recently been introduced (DRI Ketamine Assay, Thermo-Fisher Scientific; Ketamine Homogeneous Enzyme Immunoassay, Immunalysis) but are not yet FDA-approved for clinical use in the U.S. A microplate assay for urine and serum/plasma is also available for forensic toxicology applications (Micro-Plate EIA & Auto-Lyte, OraSure Technologies).

There is little published data on the performance of ketamine immunoassays except for a single report of two cases of suspected false positives on the Thermo-Fisher DRI Ketamine Assay caused by the antipsychotic drug quetiapine (10). Ketamine does not show much cross-reactivity with screening immunoassays for other drugs of abuse (11). The most common analytical methods for detection of ketamine are gas chromatography/mass spectrometry (GC-MS) and liquid chromatography/tandem mass spectrometry (LC-MS/MS) (12,13).

The current lack of any FDA-approved ketamine assay kits is a diagnostic challenge for clinical toxicology laboratories in the U.S. Ketamine abuse cases can thus be missed unless the level of suspicion is high enough to pursue specialized testing.

## Phencyclidine

PCP was first synthesized in 1926 but not used as a general anesthetic until 1956, marketed by Parke-Davis under the trade name of Sernyl. Clinical trials for PCP demonstrated short-term anesthetic properties in humans; however, psychological and physical reactions included delusions, delirium, hallucinations, muscle rigidity, and seizures (14). PCP was abandoned for clinical use in 1963, superseded by the pharmacologically similar but safer ketamine.

**Figure 1. Structure of Ketamine and Analogs**

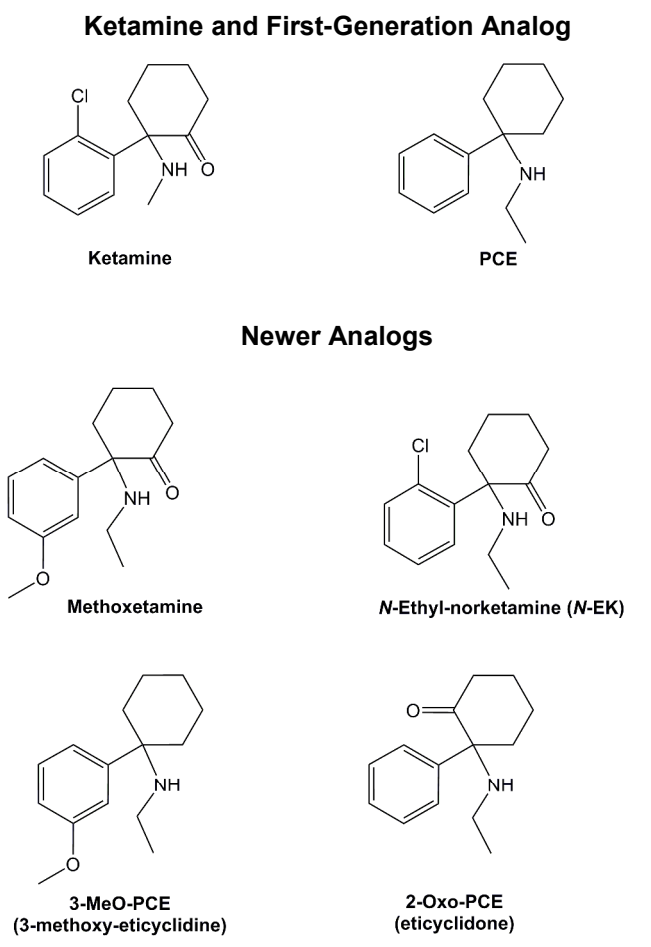
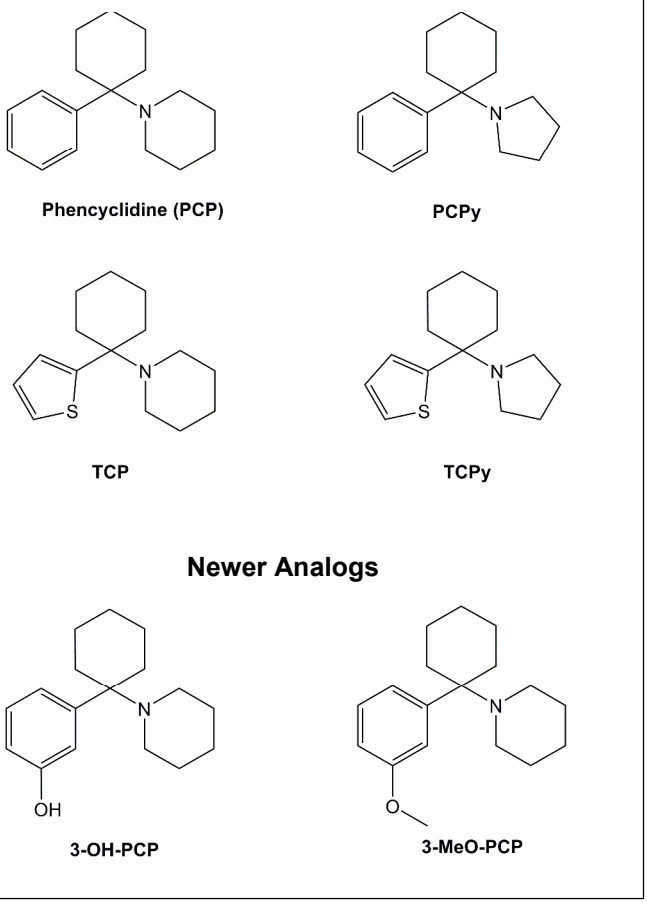



**Figure 2. Structure of Phencyclidine and Analogs**



Ketamine and PCP both act as antagonists of the *N*-methyl-D-aspartate (NMDA) glutamate receptor, an excitatory neurotransmitter receptor in the CNS (1).

PCP was a common drug of abuse in the late 1960s through 1980s. Its recreational use has since faded, but it still has regional popularity (1,15). PCP is currently classified as a Schedule II controlled substance in the U.S. It has no clinical indications and is not produced for medical use.

In the late 1960s, when PCP abuse emerged, the drug was known as the "peace pill" for its dissociative properties (14,16). It has many street names, including angel dust, crystal, hog, and embalming fluid. It can be combined with cannabis (to form krystal joint, supergrass, and killer weed) or cocaine (to form space base or tragic magic).

Smoking PCP causes peak blood levels in five to 20 minutes and peak effects after 15 to 30 minutes, with residual effects for four to six hours. Oral administration produces peak effects at one hour, with effects continuing for four to six hours.

PCP is known for its ability to induce euphoria as well as the illusion of omnipotence, superhuman strength, and sexual prowess (although some of these claims have been exaggerated by the media) (14,15). Although symptoms are not directly related to dose, low doses generally produce increased blood pressure, perspiration, tremors, facial grimacing, agitation, euphoria, disorganization of thought, and dissociation. Moderate doses (5 to 10 mg) can produce nystagmus, nausea and vomiting, repetitive motor movements, muscle rigidity, fever, insomnia, rage, amnesia, and hypnotic state. Acute PCP intoxication can produce a psychosis resembling acute schizophrenia or that produced by amphetamines or similar drugs. There is no specific treatment for PCP overdose.

## PCP Testing

Screening tests for PCP are mainly immunoassays, with confirmation by GC-MS or LC-MS/MS (17). The analyte measured is the unchanged parent drug PCP, although it accounts for only 30% to 50% of the dose excreted. The immunoassays have mostly been standardized to a 25 ng/mL cutoff, which is sufficiently sensitive to detect PCP use for several days if not longer. PCP immunoassay package inserts contain cross-reactivity data on some of the PCP analogs and metabolites; the cross-reactivity varies with kit manufacturer and analytical platform (11,18).

A clinically important feature of PCP immunoassays is cross-reactivity to structurally related compounds. A common cross-reactive compound is DXM, especially when present at high concentrations (19,20). Indeed, some clinical laboratories exploit this cross-reactivity to detect DXM abuse, because DXM can be abused as an inexpensive hallucinogen available by over-the-counter or Internet purchase (21). At high doses, DXM is an NMDA antagonist producing a similar toxidrome to that of PCP. Other compounds associated with false-positive PCP immunoassay

**Table 1. Ketamine, Phencyclidine, and Selected Analogs**

| Drug | Other names and abbreviations | U.S. DEA Schedule | Classification |
|---|---|---|---|
| Ketamine (Ketalar) | Special K, vitamin K, super K, lady K | III | Dissociative anesthetic |
| Phencyclidine (Sernyl) | PCP, angel dust, peace pills, rocket fuel | II | Dissociative anesthetic |
| PCE | Phenylcyclohexylethylamine, eticyclidine | I | First-generation ketamine analog |
| Methoxetamine | MXE, mexxy, MKET, rhino ket | Not scheduled[1,2] | Second-generation ketamine analog |
| *N*-Ethyl-norketamine | *N*-EK, ethyl-norketamine | Not scheduled[2] | Second-generation ketamine analog |
| 3-Methoxy-eticyclidine | 3-MeO-PCE | Not scheduled[2] | Second-generation ketamine analog |
| Eticyclidone | 2-Oxo-PCE | Not scheduled[2] | Second-generation ketamine analog |
| PCPy | Rolicyclidine | I | First-generation PCP analog |
| TCP | Tenocyclidine | I | First-generation PCP analog |
| TCPy | 1-[1-(Thiophen-2-yl)cyclohexyl]pyrrolidine | I | First-generation PCP analog |
| 3-Hydroxy-phencyclidine | 3-OH-PCP | Not scheduled[2] | Second-generation PCP analog |
| 3-Methoxy-phencyclidine | 3-MeO-PCP | Not scheduled[2] | Second-generation PCP analog |

[1] Not currently scheduled at federal level but schedule I in some U.S. states.
[2] May also be potentially prosecuted under the Federal Analogue Act.

results include metabolites of tramadol (22,23) and venlafaxine (24,25).

Confirmation with a GC-MS or LC-MS/MS assay is important. The highly variable and regionalized use of PCP makes identification of abuse challenging. When a PCP immunoassay is used in a population with low rates of PCP abuse, a high percentage (>80–90%) of the positive screens will be false positives caused by drugs such as DXM, tramadol, or venlafaxine (11,26). Low rates of PCP abuse have prompted many laboratories to discontinue PCP screening or to use PCP immunoassays primarily for the detection of cross-reactive compounds like DXM.

## First-Generation PCP Analogs

PCP is an arylcyclohexylamine with three key moieties: an aromatic ring, a substituted cyclohexane ring, and a basic amine substituent (Figure 2). Although PCP was the first of its chemical class to have medical applications, discovery of other arylcyclohexylamines started in the early 1900s, with the primary amine 1-(1-phenylcyclohexyl)amine (PCA) initially reported in 1907 (27). Several forerunners to PCP were synthesized in the 1950s, with some reported to have deep sedative effects; however, these compounds did not achieve clinical application.

First-generation analogs of PCP began circulating in the late 1960s and were distributed on the illicit market into the 1990s, often sold as PCP. These first-generation compounds were generally discovered through legitimate pharmaceutical research and not the work of clandestine chemists (27,28).

Figure 2 shows three first-generation PCP analogs: PCPy, 1-[1-(thiophen-2-yl)cyclohexyl] piperidine (TCP), and TCPy. The first-generation analogs are fairly simple derivatives of PCP that retain the cyclohexane ring. Modifications of the cyclohexane ring tend to reduce antagonist activity at the NMDA glutamate receptor (27). PCPy substitutes pyrrolidine for the piperidine ring of PCP, while TCP substitutes thiophene for the benzene ring.

The extent of distribution of the first-generation PCP analogs from the 1960s to 1990s has been hard to estimate, and media reports likely overestimated their use. Of the first-generation analogs, only three became prominent—*N*-ethyl-1-phenylcylohexylamine (phenylcyclohexylethylamine [PCE] or eticyclidine), PCPy, and TCP. The first-generation PCP analogs essentially disappeared from the illicit market after the 1990s (27).

## Second-Generation PCP Analogs

Since the 1990s, a newer generation of PCP analogs has emerged that are principally hydroxy (OH) and methoxy (MeO) derivatives of PCP and PCPy (27). Some of these compounds originated in legitimate research in the 1960s. However, unlike the first-generation analogs, the second-generation PCP analogs include multiple compounds first synthesized by clandestine chemists, with reports of their psychoactive effects appearing in online forums such as *The Hive* (which operated from 1987–2004).

Ease of chemical synthesis explains some of the patterns of clandestine drugs. For example, some structural modifications to PCP are relatively straightforward synthetically and yield analogs with similar potency and psychoactive effects; in contrast, some ketamine derivatives are more difficult to synthesize (29). The Internet allows the distribution of detailed synthetic routes for these second-generation analogs, with 4-MeO-PCP being the first of this newer wave of analogs to achieve significant illicit use in the late 1990s (27). 4-MeO-PCP has historic significance as the first dissociative anesthetic without prior medical or legitimate pharmaceutical research to appear on the illicit market. Sources sold this compound as a "research chemical" or other misleading categorization, typically via the Internet. Other second-generation PCP analogs include 3-OH-PCP and 3-MeO-PCP (Figure 2); these have been detected on the illegal market only in the past decade.

## Ketamine Analogs

Ketamine and its analogs lack the piperidine or pyrrolidine rings found in PCP or its analogs (Figure 1). Methoxetamine (MXE) is probably the best-known analog of ketamine and differs in two respects from the ketamine structure: replacement of the 2-chloro group by 3-methoxy and an *N*-ethylamino group instead of the *N*-methylamino group.

The first reports of MXE abuse appeared in 2010, with an expanding literature on the drug developing since then (27). MXE has been advertised as a safer and "legal" alternative to ketamine. With little controlled study of MXE, information on its effects is largely gleaned from user feedback on the Internet and case reports of patients with MXE toxicity. A number of sources have compiled information on MXE intoxication, including fatalities (30–33).

Since 2010, other ketamine derivatives have appeared as drugs of abuse, often sold through Internet sources (27). *N*-Ethyl-norketamine (*N*-EK) is a close analog of ketamine (Figure 1) that appeared in Europe following restrictions on MXE in 2012, including a temporary class drug control ban in the United Kingdom. *N*-EK has similar pharmacology to ketamine, although drug user reports on the Internet vary.

Other ketamine analogs are modifications of PCE (Figure 1). These include 3-MeO-PCE (3-methoxy-PCE; 3-methoxy-eticyclidine) and 2-oxo-PCE (eticyclidone). 3-MeO-PCE was the first novel ketamine analog to appear on the Internet market since MXE (27). This drug gained a reputation for a high likelihood of behavioral toxicity and psychosis. Insufflation of powdered 3-MeO-PCE can induce a dissociative state with analgesia, euphoria, and sensory enhancement lasting several hours.

2-Oxo-PCE was originally synthesized in the

1960s and was a promising pharmaceutical lead until ketamine became the clinically marketed dissociative anesthetic in this drug class. 2-Oxo-PCE was rediscovered and emerged on the Internet in 2009 along with other ketamine and PCP analogs. Recently, a wave of 2-oxo-PCE intoxications has been reported in East Asia (34,35).

**Regulatory Status of Analogs**

The regulation of drugs that are derivatives of existing controlled substances is complex and varies throughout the world (36,37). In the U.S., legislation is required to move drugs into controlled substance status. This can be done at the state or federal level. Options exist to temporarily schedule compounds pending permanent designation, as has been done with cathinone and fentanyl derivatives in recent years (38,39). The Controlled Substances Analogue Act of 1986 provides the legislative basis for controlling compounds that are "substantially similar" to existing controlled substances; however, the law is challenging to apply.

In general, the first-generation ketamine and PCP analogs are DEA schedule I in the U.S. (Table 1). The newer-generation analogs are either not currently scheduled or are scheduled only in some states. Some countries restrict these analogs, with regulations evolving over time as drug trends unfold (27,36,37).

**Detection of Ketamine and PCP Analogs**

Definitive identification of ketamine and PCP analogs is usually achieved with GC-MS or LC-MS/MS (27,40). In general, the metabolism and disposition of the analogs, particularly the second-generation ones, are incompletely understood (27). In some cases, as mentioned above, analogs circulating in the abuse community were evaluated as pharmaceuticals in the 1950s and 1960s; thus, pharmacokinetic data may be available in literature from that era.

Some of the PCP analogs can be detected by PCP immunoassays, although data is currently sparse. Given the propensity of many PCP immunoassays to cross-react with compounds such as DXM, meperidine, tramadol, and venlafaxine, closely related analogs such as 3-OH-PCP would also be predicted to cross-react. Package inserts for PCP immunoassays may contain information on cross-reactivity with first-generation PCP analogs such as PCPy, TCP, and TCPy (compounds known before the introduction of commercially marketed PCP immunoassays), as compiled in a study of immunoassay cross-reactivity published in 2009 (18).

In general, these first-generation analogs have similar or even greater cross-reactivity on PCP immunoassays than PCP itself. Based on their close structural similarity, second-generation PCP analogs would also be expected to cross-react with PCP immunoassays, although there is little experimental data published on this. Certainly, ingestion of a PCP analog should be considered in cases with clinical features similar to PCP intoxication with a positive PCP immunoassay screen but negative confirmation for PCP.

Commercial ketamine immunoassays have recently become available, but there is little data published on their cross-reactivity with ketamine analogs. One would predict that analogs that are minor modifications of the ketamine structure would be likely to cross-react with ketamine immunoassays. Confirmation of this hypothesis awaits further data.

**Discussion**

The growth in use of ketamine and PCP analogs illustrates that designer drugs of abuse can be either resurrections of pharmaceutical compounds abandoned decades ago or novel compounds synthesized by clandestine chemists. This pattern is also evolving with designer compounds in other drug classes such as benzodiazepines and opioids (28). The Internet has greatly enhanced the spread of information about novel compounds and aided their distribution throughout the world. As illustrated by PCP and ketamine analogs, the novel compounds are often simple modifications of the prototype drug, with a large number of combinations possible. Rapidly shifting patterns of compound availability make it challenging to stay on top of drug trends.

## Learning Objectives

After reading this article, the reader will be able to identify the toxicological properties and abuse potential of the dissociative drugs ketamine, phencyclidine, and their analogs. The reader will also be familiar with the analytical methodology and challenges in detecting these drugs as well as the drugs' regulatory status.

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The authors have nothing to disclose.

## FDA Issues Warning After Finding Heavy Metals in Kratom Products

The U.S. Food and Drug Administration has issued several warnings about the serious risks associated with the use of kratom, including high rates of contamination with salmonella that led to numerous illnesses. New tests have found that many kratom products are contaminated with heavy metals, including lead and nickel, at levels not safe for human consumption.

The Nov. 27 statement can be found here: https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm626738.htm

# 2018 IATDMCT Congress Featured Some "Top Science Down Under"

*By Kamisha L. Johnson-Davis, PhD*

The 2018 International Association of Therapeutic Drug Monitoring and Clinical Toxicology (IATDMCT) Congress was held in Brisbane, Australia, from Sept. 16 to 19.

The pre-congress symposium focused on drugs of use and misuse, pharmacogenetics, and pharmacojustice. The four speakers discussed a laboratory perspective on substance abuse harm reduction, substance abuse involvement in suicide, translational pharmacogenomics in the justice system, and the role of ethnicity and epigenetics in antiepileptic hypersensitivity syndrome.

The congress began with a welcome address from the co-chair of the organizing committee, Andrew McLachlan, PhD, of the University of Sydney, followed by a cultural dance from an Aborigine tribe of Brisbane.

Teun van Gelder, MD, PhD, of Erasmus Medical Center in Rotterdam, the Netherlands, delivered the presidential welcome address. Geoff Isbister, PhD, a clinician researcher at the University of Newcastle in New South Wales, opened the meeting by describing his research on using clinical toxicology to address the myths about patients poisoned by bites from snakes and spiders.

**A Variety of Topics**

The congress was filled with engaging topics in therapeutic drug monitoring and clinical toxicology. There were high-quality oral and poster presentations from scientists and physicians from around the world. The topics were proposed by scientific committees of IATDMCT: alternative sampling strategies, anti-infective drugs, clinical toxicology and drugs of abuse, immunosuppressive drugs, pharmacogenetics, pharmacometrics, TDM in oncology, and toxicology and environmental health.

Roger W. Jelliffe, MD, of the University of Southern California, received the prestigious C.E. Pippinger Award in recognition of his outstanding contributions to the field of therapeutic drug monitoring. Laure Elens, PhD, of the Catholic University of Louvain in Belgium, received the Victor Armstrong Young Investigator Award.

The congress closed with a plenary session by Jennifer Martin, PhD, director of the Griffith Institute for Drug Discovery at Griffith University in Queensland, on tackling antibiotic resistance by targeting virulence, not viability. Post-congress, two workshops featured hot topics: the medicalization and legalization of cannabis and Bayesian-based therapeutic drug monitoring concepts and applications.

There were 367 attendees at the congress from 41 different countries, with 140 people attending for the first time. The congress included four plenary sessions, 16 symposiums, and seven morning roundtable sessions. There were 94 symposium presenters, 55 oral presentations, and 150 poster presentations.

The 2019 congress will be held in Foz do Iguacu, Brazil, from Sept. 22 through 26.

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The author has nothing to disclose.

# Providing Expert Testimony

## *Seasoned Forensic Expert Witness Shares Tips Based on His Experience*

*By Dale Haak, PhD*

One of the more interesting opportunities that the forensic science profession offers is the chance to be an expert witness. Although there are any number of deliberative forums in which the forensic scientist can participate as an expert, the basic function remains essentially the same—to convey understanding of the scientific evidence pertinent to a question.

My 20 years of experience have been in drug testing of active-duty military service members and Department of Defense civilian employees. I have served as an expert witness in scores of hearings, including courts martial, administrative separation boards, merit system protection boards, and arbitration hearings. Despite the procedural differences of these settings, my role is always to testify concerning drug-test results and answer questions related to chain of custody, analytical methodology, and result interpretation. Along the way, I've gleaned some insight into what makes for effective expert witness testimony, which I've tried to distill into some helpful pointers.

*1. Make it your chief goal to provide the most accurate and truthful testimony you can.*

The fundamental aim of any deliberative proceeding is to assess available evidence to determine the truth of the matter in question. Inherent in this quest is continuous evaluation of the factual integrity of the evidence. As a general rule, certainty is not required for evidence to be useful. As long as the degree of uncertainty can be assessed, the finder(s) of fact can weigh consideration and reliance accordingly.

In witness testimony, factual integrity is primarily established by means of the testimonial oath, and

subsequently assumed until some reason arises that calls it into question. Although the legal principle of "falsus in uno, falsus in omnibus" (false at one point, false at all points) is not generally adhered to as much as in past times, it still holds sway, the case of expert witness testimony being one example. That is because the expert's primary role is educating legal professionals, who often admit to a poor understanding of the pertinent science. They usually therefore have little ability to assess the technical aspects of the expert's testimony and must trust what the witness says.

That being the case, it can be problematic if the expert's commitment to the truth is called into question on a matter that *can* be independently assessed. If you as an expert allow yourself to get into such a situation, you can at least expect the opposing legal counsel to argue vigorously for the exclusion of your entire testimony. Furthermore, any lapse of veracity will be in the procedural transcript and forever after available for discovery. You can then expect that any counsel doing due diligent research will require you to revisit the issue on the stand during any future testimony.

One might question the need to stress the obvious importance of telling the truth. But subtle pressures can compromise testimonial integrity. The remaining pointers touch on these pressures.

*2. Maintain a clear view of the limits of the expert's role in the judicial process.*

The legal system uses a familiar framework, with presumption of innocence being the default assumption. The task of the proceeding is to evaluate whether the evidence supports or refutes the presumption of innocence. I find it useful to frame the questions that I am asked as an expert in a similar manner.

A common scenario that I am asked to opine on is the plausibility of whether some environmental circumstances could have led to drug exposure that caused of a positive result. For example, a positive result for tetrahydrocannabinolic acid (marijuana metabolite) is often blamed on exposure to second-hand smoke from marijuana smokers.

Given the judicial presumption of innocence, my default assumption is that the cause of the result was as claimed, innocent exposure. I then apply my knowledge of the scientific literature along with my background in analytical science to consider whether or not the evidence makes the case for rejection of that default assumption. Depending on the circumstances of the case, the judgment can range from innocent ingestion being completely plausible to being ruled out with a high level of confidence. Controlled studies consistently show that positives from second-hand smoke are possible only in very particular environmental conditions: The exposure must be in a small space with no ventilation and must persist for an hour or more.

In this imperfect world, the answer often falls somewhere between these two happy places, with the pertinent factors indicating some degree of implausibility, but not enough to rule out the possibility. Therefore, my practice is to limit my answer to laying out the situation as best I can in order to impart to the decision maker(s) the clearest possible understanding of the science and what can and cannot be taken from it.

Of course, laying out the plausibility of an exposure scenario falls short of settling the question of guilt or innocence in the accused's case. It is up to the finder(s) of fact to make that determination. I cannot help in that regard because I was not at the scene. When the accused's story's plausibility cannot be ruled out scientifically, it generally comes down to whether or not they find the accused to be believable. Over the years I have seen essentially the same set of circumstances go either way.

*3. Stick to what you know and don't be afraid to recommend the use of other experts.*

On a number of occasions, I have run into the mistaken belief that because I have "PhD" after my name, my knowledge is inexhaustible. Although this notion can be flattering, the wise expert puts it to rest quickly. This issue normally comes up during the prehearing phase in consultation with counsel when I am asked a question outside the scope of my knowledge. I am usually aware of the field of expertise within which the question more appropriately falls. By pointing this out, I can still contribute to obtaining the best answers to questions that I cannot answer myself.

For example, a common exposure scenario is that the accused claims to have had a drink spiked in a public place without their knowledge. It is within my expertise to assess the plausibility that ingestion of a certain dose at a certain time prior to specimen collection could result in a positive result. But my opinion of the likelihood that a drink was actually spiked is no more expert than anyone else's. So when I'm asked this question, I suggest that a local narcotics detective who is more familiar with the drug-abusing habits of the local citizenry might be a better source.

*4. When providing testimony, always listen carefully to the question and answer it specifically.*

As the technical director of the laboratory, I don't physically test specimens myself. But I am responsible for our detailed written standard procedures, which provide the essential custodial and analytical history of each specimen. This documentation allows me to testify in defense of the results. This point is normally covered on direct exam in the witness qualification portion.

Once, the accused's representative began the

cross examination by asking, "So, it is the case that you have no personal knowledge of the testing of my client's specimen?" At this point I assumed he was covering the same ground as the direct exam in order to introduce his line of inquiry, as is often done. Further assuming that the point he was trying to make was that I had not myself physically performed the testing, I answered "yes."

To my surprise, he immediately addressed the judge, "Your honor, I move that this witness be excused because he has no personal knowledge of the testing of the specimen." He argued vigorously for my dismissal on the basis that I had no useful knowledge to offer the court. At that point it was necessary for the government counsel to "rehabilitate" me, which is a process in which a counsel re-interviews a witness who has misspoken to clear up testimonial discrepancies. The counsel had to establish that I'd misunderstood the question and that the correct answer was that, despite not having performed the testing myself, I did have knowledge of how the specimen was tested, and could testify concerning it by virtue of my direct responsibility for all the testing procedures in the laboratory. Ultimately the objections to my testimony were overruled, but the episode taught me this lesson the hard way. It also illustrates the point that an expert does not have to be perfect. Good faith errors can be recovered as long as they really are in good faith.

In conclusion, the role of the expert witness is certainly an interesting, but challenging, undertaking. Ultimately, the only thing that really prepares one for it is the experience of doing it, but I hope that young experts will find the above pointers useful.

## Learning Objectives

After reading this article, the reader will have a better understanding of the role of the expert witness in legal proceedings and the critical elements of effective expert witness testimony.

### Suggested Reading

1. Andrew L. Expert witness testimony: the ethics of being a medical expert witness. Emerg Med Clin N Am 2006; 24:715–31.
2. Cohen S. Expert witnessing and scientific testimony: surviving in the courtroom. Boca Raton, FL: CRC Press; 2008.

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The author has nothing to disclose.

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