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American
Prosecutors
Research Institute

*Drug Toxicology
for Prosecutors*

Targeting Hardcore
Impaired Drivers

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Drug Toxicology for Prosecutors

Targeting Hardcore
Impaired Drivers

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INTRODUCTION: A HORSE OF A DIFFERENT COLOR

Drug impaired drivers kill and maim thousands of people each and every year in the United States. Unfortunately, prosecuting drug-impaired drivers is a daunting task. Jurors, who are very familiar with alcohol's effects, signs and symptoms, often know little or nothing about other drugs. Tainted by crime shows like *CSI: Miami*, they may have unrealistic expectations about the nature and quantum of available proof. Unlike alcohol, most states do not have “per se” limits for drugged driving.

To successfully explain the evidence and issues to jurors in Driving Under the Influence of Drugs (DUID) cases, prosecutors must understand the basics of drug toxicology. This publication is designed to provide prosecutors with a basic understanding of drug pharmacology and testing. The author, Dr. Sarah Kerrigan, is the former Toxicology Bureau Chief of the New Mexico Department of Health's Scientific Laboratory Division. Prior to this, she worked as a Forensic Toxicologist for the California Department of Justice. Originally trained at the Scotland Yard Forensic Laboratory in England, Dr. Kerrigan has worked closely with law enforcement officers and prosecutors in DUID (including Drug Recognition Expert) cases in New Mexico and California.

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DRUGS AND DRIVING FOR PROSECUTORS

The prosecution of drug impaired driving cases is more complex than alcohol-related DWI (driving while impaired) cases—both scientifically and legally. Impairment can be more difficult to discern and prove, thus making these cases more difficult to prosecute. Although alcohol is a drug, not all drugs can be considered in the same way. This means that a case involving a driver suspected of driving under the influence of drugs (DUID) may require special handling and evaluation. Good communication and effective integration of law enforcement and legal and scientific personnel are essential in these cases.

THE PREVALENCE OF DRUG-IMPAIRED DRIVING

In any given year, millions of Americans operate motor vehicles while impaired by alcohol or drugs. In 2003, over 32 million persons aged 12 or older drove under the influence of alcohol at least once during the previous year (1). An estimated 11 million persons reported driving under the influence of an illicit drug. In DWI cases, states have enacted *per se* laws, which prohibit driving with a blood or breath alcohol concentration (BAC) of 0.08 (2) regardless of actual impairment (3). In most states, there are no similar laws with regard to driving under the influence of drugs—even those commonly understood to impair driving.

There is a growing body of scientific evidence that driving under the influence of drugs has become a significant problem worldwide. Driving is a complex task which involves a variety of skills such as coordination, reaction time, tracking, judgment, attention and perception. Any drug which affects mental or physical processes has the *potential* to impair driving at sufficient dose. According to the 1996 National Household Survey on Drug Abuse, of the 9 million drivers who drove within two hours of drug use, the most commonly encountered drugs were marijuana and cocaine. Despite mounting evidence that driving under the influence of illegal drugs other than alcohol is common, drugged drivers are less frequently detected, prosecuted, or referred to treatment when compared with drunk drivers (4).

The 2003 National Survey on Drug Use and Health (NSDUH), conducted by the Substance Abuse and Mental Health Services Administration (SAMHSA), indicated that an estimated 19.5 million Americans (8.2% of the population aged 12 or older) had used an illicit drug during the previous month. Drug abuse, whether it involves controlled substances or the misuse of prescription drugs, has permeated almost every level of society to some degree:

- In 2003, an estimated 11 million people reported driving under the influence of an illicit drug during the past year (1).
- As many as 18% of 21 year-olds report drugged driving at least once during the past year (5).

- Drugs are used by approximately 10 - 22% of drivers involved in accidents, often in combination with alcohol (6).
- A study of fatally injured drivers from seven states showed that alcohol was present in more than 50% of the drivers; other drugs were present in 18% of the drivers (6).
- Positive drug findings in injured drivers who receive medical treatment range from less than 10% to as high as 40% (6).
- The incidence of drug-use among drivers arrested for motor vehicle offenses ranges between 15 - 50% (6).

Although it is well understood that drug use can be detrimental to safe driving, the extent to which drugs impair driving is often difficult to measure, predict or quantify. The degree of impairment depends upon a number of variables including the dose, drug history and time since drug use. Some drugs have the potential to impair driving performance for extended periods, while others may impair during the “crash” phase, during which time drug concentrations may be decreasing or very low. Drug-impaired driving is often under-reported and under-recognized. Although it is illegal to drive under the influence of drugs anywhere in the U.S., statutes vary widely. Toxicology testing is expensive, resources differ from state to state, and protocols vary between laboratories, further compounding the problem.

ALCOHOL VS. DRUG-RELATED DWI

Alcohol is a drug but not all drugs are alcohol.

Drug-related DWI is inherently more complex than alcohol-related DWI. Furthermore, the effect of alcohol on the body and on driving has been well characterized over several decades. Most people are familiar with the effects of alcohol and its ability to impair driving. However, that is not always true for other drugs. There are numerous illicit, prescription and over-the-counter drugs that have the potential to impair the mental and/or physical processes required for safe driving. Despite the ever-increasing existence of scientific literature on the impact of drugs on driving, many drugs have not yet been fully investigated. To complicate matters, drugs are often used in combination with alcohol or other drugs, requiring a case-by-case evaluation of the potential for interaction and possible impairment. Drug-impairment requires the jury to develop an understanding of the unique effects of specific substances and their complex potential to impair driving.

As of 2004, all 50 states have established a per se level of ethanol (0.08 g/100mL) in blood, but there are no widely accepted per se standards for drugs. Drug-impaired driving statutes typically approach the issue in one or more of three ways:

- Statutes that require the drug to render a driver incapable of driving safely;
- Statutes that require the drug to impair a driver's ability to operate a vehicle safely or require a driver to be under the influence, impaired or affected by an intoxicating drug; or
- Per se laws that make it a criminal offense to have a specified drug or drug by-product (metabolite) in the body while operating a vehicle. Some states' per se drug laws incorporate a "zero tolerance" standard in which any detectable level of a specified drug or metabolite constitutes a violation while a few states list actual drug concentrations at which a violation occurs.

States with zero tolerance drug statutes make the presence of any specified drug or metabolite in the blood or urine, obtained from a person who was operating a motor vehicle, a crime in and of itself—i.e., distinct from a charge of drug-impaired driving. Although these laws facilitate identification, prosecution and treatment of drivers who misuse drugs, they are typically used in conjunction with the aforementioned statutes that require evidence that the person was impaired, incapacitated or affected by the drug. Comparisons of drugged driving statutes between states are available elsewhere (7, 8).

PREVALENCE OF SPECIFIC DRUGS IN DWI

Marijuana, stimulants, depressants and opiates are among the most frequently encountered drugs in impaired drivers. However, prevalence varies with geographical location and emerging drug trends; for example, there may be increased methamphetamine use on the West coast, compared with increased oxycodone use on the East coast. Drugs of choice may vary by county and by socioeconomics. Table 1 lists some of the uses and examples of licit and illicit drugs within each class.

In 2003, marijuana was the most commonly used illicit drug in the United States (14.6 million users) followed by non-medical use of prescription drugs (6.3 million) (1). Of those persons who abused prescription drugs, an estimated 4.7 million abused pain relievers, 1.8 million abused tranquilizers, 1.2 million abused stimulants and 0.3 million abused sedative medications. In the same year, estimates for cocaine and hallucinogens were 2.3 million and 1 million, respectively.

Many abused substances have legitimate uses. In states with zero tolerance per se drug laws, a valid prescription may constitute a legitimate defense to the zero-tolerance portion of the statute. In these instances, prosecution must follow the “impaired” or “under the influence” statute instead, requiring proof that the person was “affected” to some degree.

Statistical evaluation of drug prevalence varies not only with geographical location but is also dependent on drug testing methodology and sample type (blood, urine, saliva, other). Drug testing methodology involves the use of analytical procedures which may have varying degrees of sophistication. For example, one toxicology laboratory may utilize different analytical procedures and instrumentation from another laboratory. Laboratories with state-of-the-art instrumentation and testing capabilities may demonstrate a higher percentage of positive findings than those laboratories with less-sophisticated equipment. The sample matrix, or type of biological evidence submitted for analysis, can also influence which drugs are most likely to be detected. This is because many drugs are present in a blood sample for a relatively short period of time compared with

urine. For example, cocaine continues to degrade or break down to benzoylecgonine, even after collection and preservation of a blood sample. However, cocaine may be present in other body fluids, such as urine, for a longer period of time. In this way, the choice of biological specimen may influence the outcome of a particular test.

The length of time that a drug or its metabolite is present in a given biological sample is often called its *detection time*. This may vary depending on the *dose* (amount), *route of administration* (injected, inhaled etc.) and *elimination rate* (how long it takes the body to get rid of the substance). The presence of a drug metabolite in a biological fluid may or may not reflect consumption of the drug recently enough to impair driving performance. For example, the presence of a marijuana metabolite in *urine* may not be, by itself, a reliable indicator of either driving impairment or of recent exposure to the drug. However, an elevated concentration of THC (the principal active component of marijuana) in *blood* may be consistent with recent use of the drug and related driving impairment. In addition to the analytical test results, supplemental information (including driving, performance on psychophysical tests, values obtained in physiological assessments, and unusual behaviors, statements or observations) often is necessary for an appropriate forensic toxicological interpretation of driving impairment.

Table 1.

Drug Class	Medicinal Uses	Examples
Depressants	Anticonvulsants Antidepressants Antihistamines Anxiolytics (anti-anxiety) Hypnotics (sleep inducers) Muscle relaxants Anticatatplectics (works to prevent a condition which results in sudden loss of muscle power following a stimulus like fright or shock) Sedatives (tranquilizers)	Alprazolam, Amitriptyline, Carbamazepine, Carisoprodol, Diazepam, Diphenhydramine, Gamma-Hydroxybutyrate (GHB), Meprobamate, Phenobarbital, Temazepam, Trazodone, Zolpidem
Stimulants	Anorectics (appetite stimulants) Attention Deficit Disorders (ADD) Narcoleptics (to prevent deep sleep attacks) Local anesthetics	Amphetamine, Cocaine, Methamphetamine, MDMA*
Opioids	Analgesics (pain relievers) Antitussives (cough suppressants) Codeine, Fentanyl,	Heroin* Hydrocodone, Methadone, Morphine, Oxycodone, Propoxyphene
Hallucinogens	Anesthetic adjuncts (assists in anesthesia) Appetite stimulants Antiemetics (to prevent vomiting)	Ketamine, Lysergic Acid Diethylamide* (LSD), MDMA*, Mescaline*, PCP*, Peyote*, Tetrahydrocannabinol (THC)

* Indicates no currently approved medicinal use in the United States.

COMMON DRUG EFFECTS: PHARMACOLOGY FOR PROSECUTORS

Many of the drugs that affect the central nervous system (CNS) produce characteristic effects. These similar effects provide the basis for most general drug classification schemes. Drug classes may include depressants, stimulants, opioids (narcotics) or hallucinogens. The classes themselves can be further subdivided, based upon the intended use of the drug (Table 1). The effects (signs and symptoms) of some commonly encountered drug classes are summarized in Table 2. Although many drugs within a class produce predictable effects, such as *ataxia* (inability to coordinate voluntary muscular movements), slow movements or slurred words following a sufficient dose of depressant drug, others are more complicated. Some substances are not easily classified because they have multiple characteristics. For example:

- Tetrahydrocannabinol (THC), the principal active component of marijuana, has both hallucinogenic and depressant effects.
- Methylenedioxymethamphetamine (MDMA) acts as both a stimulant and a hallucinogen.
- Phencyclidine (PCP) and ketamine have both depressant and hallucinogenic effects.

Although drug signs are determined to a large extent by the *pharmacology* (properties and reactions) of the drug, other factors such as dose, drug use history, mood, environment or setting, as well as the use of other substances, also help to determine the overall effect.

Table 2.

Drug	Signs and Symptoms
Depressants	Ataxia (uncoordinated movement), decreased blood pressure, decreased pulse, disorientation, decreased inhibitions, fumbling, horizontal gaze nystagmus (HGN), ptosis (droopy eyelids), slow pupillary reaction to light, sluggishness, slowed reflexes, sedation, slurred speech
Hallucinogens	Body tremors, dazed appearance, diaphoresis (excessive perspiration), dilated pupils, disorientation, dysarthria (difficulty in articulating words), elevated blood pressure, elevated pulse, memory loss, muscle rigidity, nausea, paranoia, poor coordination, poor time and distance perception, synesthesia (blending of the senses), visual/auditory disturbances
Marijuana	Ataxia (uncoordinated movement), body tremors, disorientation, elevated blood pressure, elevated pulse, eyelid tremors, increased appetite, lack of ocular convergence, poor time and distance perception, paranoia, reddened conjunctiva, reduced inhibitions, transient muscle rigidity
Opioids	Ataxia (uncoordinated movement), constipation, constricted pupils, decreased blood pressure, decreased pulse, dry mouth, dysphoria (state of unwellness or unhappiness), euphoria, facial itching, low and raspy voice, ptosis (droopy eyelids), puncture marks, mental clouding, muscle flaccidity, nausea, nodding off, sedation, slow or no pupillary reaction to light, slow reflexes, vomiting
PCP	Agitation, ataxia (uncoordinated movement), blank stare, confusion, cyclic behavior, diaphoresis (excessive perspiration), dissociative anesthesia (disconnected from pain and surroundings), dysarthria (difficulty in articulating words), elevated blood pressure, elevated pulse, hallucinations, HGN, “moon walking”, muscle rigidity, nystagmus, vertical gaze nystagmus (VGN)
Stimulants	Anxiety, body tremors, bruxism (teeth grinding), dilated pupils, dry mouth, excitation, eyelid tremors, euphoria, hyperreflexia (overactivity of physiological reflexes), hypervigilance (abnormal awareness of environmental stimuli), increased blood pressure, increased pulse, insomnia, irritability, jaw tightness, muscle rigidity, reduced appetite, rhinorrhea (runny nose), reddening of nasal mucosa, slow pupillary reaction to light, talkativeness

Many of the drugs encountered in impaired drivers are habit-forming or addictive. Drugs with high abuse-potential may produce chemical or psychological dependence that may also result in characteristic withdrawal effects (Table 3). These withdrawal effects may manifest as the exact opposite of the desired or expected effect of a particular class of drug. For example, during withdrawal or the “crash” phase following binge use of methamphetamine (a potent stimulant), an individual may experience profound lethargy, exhaustion and hypersomnolence. These effects are more consistent with those of a depressant drug.

Table 3.

Drug	Withdrawal Symptoms
Stimulants	Muscular aches, abdominal pain, tremors, anxiety, hypersomnolence (extreme fatigue), lack of energy, depression, suicidal thoughts, exhaustion
Opioids	Dilated pupils, watery eyes, rapid pulse, piloerection (erection/bristling of hairs), abdominal cramps, muscle spasms, vomiting, diarrhea, tremulousness, yawning, anxiety, rhinorrhea (runny nose), sweating, restlessness
Depressants	Trembling, insomnia, sweating, fever, anxiety, cardiovascular collapse, agitation, delirium, hallucinations, disorientation, convulsions, shock
Marijuana	Anorexia, nausea, insomnia, restlessness, irritability, anxiety, depression

To provide expert testimony, toxicologists look at the characteristic appearance, behavior or observable effects of the drug on the individual. Most toxicologists adopt a multi-strategy approach to interpretation. Again, the presence of a drug or drugs in a biological sample provides valuable insight, but more often than not, other factors will also be considered.

Pharmacology of a drug can be divided into two disciplines: *pharmacokinetics* and *pharmacodynamics*.

- *Pharmacokinetics* – How the drug (*pharmakon*) moves (*kinesis*) about the body. This helps answer questions like “When was the drug used?” and

“How much was taken?” A simple way to view pharmacokinetics is “what the body does to the drug.”

- *Pharmacodynamics* – How the drug interacts with receptors in the brain (how it affects the brain and consequently the person—mentally and physically). This helps answer questions like “What are the effects?” and “How long does it last?” A simple way to view pharmacodynamics is “what the drug does to the body.”

Pharmacokinetics

The human body recognizes a drug as a foreign substance or *xenobiotic*. When exposed, the body attempts to break down and eliminate these foreign substances. Pharmacokinetics involves *absorption* (getting the drug into the body), *distribution* (movement throughout the body), *metabolism* (breaking it down into other chemical components) and *elimination* (getting it out of the body). These processes largely determine the *efficacy* (the ability of the drug to produce a result) or *effectiveness* of the drug, its *concentration* at the active site (specific brain receptors), and the *duration* of the drug effect. Pharmacokinetic properties are used by pharmacologists, clinical researchers and toxicologists to develop new therapeutics, understand the factors that govern abuse, determine how drugs can be detected over time and interpret drug effects on human performance.

Route of Administration: How the drug gets into the system.

The onset of action, duration of effects, intensity and quality of the drug experience may vary depending upon the route of administration (Table 4). Intravenous drug administration provides maximum drug delivery and rapid onset of effects. However, this bypasses many of the body’s natural safeguards and may result in complications of intravenous drug use. For this reason, inhalation and smoking are popular alternatives. When a drug is smoked, it is rapidly absorbed in the lungs and transported to the brain via the arterial blood supply. Smoking is a preferred route of crack cocaine administration due to rapid onset, intensity and euphoria, even though pipes and smoking apparatus become hot and may burn the lips. In general, the efficiency and speed of drug delivery (the faster it is delivered to the brain) increases the potential for abuse and dependency.

Table 4.

Route	Drugs
Oral	Cannabinoids, opiates, LSD, mescaline, peyote, GHB, benzodiazepines
Inhalation	Solvents, gases, low-boiling-point alkaloids (usually colorless, complex, organic bases, like cocaine, that contain nitrogen and usually oxygen)
Intravenous	Opiates, cocaine, methamphetamine, PCP
Smoking	Marijuana, PCP, crack cocaine, methamphetamine
Intranasal	Cocaine, heroin, methamphetamine
Dermal	Fentanyl, nicotine

Absorption

For a drug to exert an effect, it must be absorbed into the bloodstream, traverse membranes, and activate specific receptors. This process is largely determined by the physical and chemical properties of the drug. Most drugs can be characterized as acidic, basic or neutral, and unlike alcohol, which is highly water-soluble, many drugs are also soluble in fat or lipids. The degree to which a particular drug is water-soluble or fat-soluble influences how it is *distributed* throughout the body.

Distribution

As soon as the drug is absorbed into the bloodstream, it is circulated to surrounding tissues and organs, and the distribution phase begins. Drugs that are lipid (fat) soluble are distributed more readily into the tissues, such as the heart, liver, kidney, brain and fat. THC is fat-soluble, which means it is distributed and stored in tissues and fat depots within the body, accounting for its gradual release and long *half-life* (time taken to eliminate half of the drug). The extent to which a drug is distributed in the body is given by its *volume of distribution* (V_d). Highly water-soluble (hydrophilic) drugs, like ethanol ($V_d = 0.5$ L/kg), are distributed mainly in the body water and have low volumes of distribution. Conversely, drugs with large volumes of distribution, like heroin ($V_d = 25$ L/kg), are widely distributed throughout the body, including the tissues (Table 5).

Metabolism

For most drugs, only relatively small amounts are excreted unchanged. To eliminate a drug, our bodies try to make the substance more soluble in water. This process makes it easier for us to eliminate the substance in our urine. Metabolism can affect pharmacological activity—i.e., the way the drug affects the body. For example, cocaine and THC are broken down in the body to benzoylecgonine and carboxy-THC respectively, both of which are *pharmacologically inactive* (having no effect on the nervous system). Alternatively, some drug metabolites may be *pharmacologically active*, therefore contributing to the overall effect, such as:

- Metabolism of diazepam to nordiazepam (an active metabolite of many benzodiazepines)
- Carisoprodol to meprobamate
- Codeine to morphine

There are a great many variables that can affect drug metabolism, including age, sex, genetic polymorphisms (common genetic mutations that may relate to specific genetic predispositions), health, disease and nutrition.

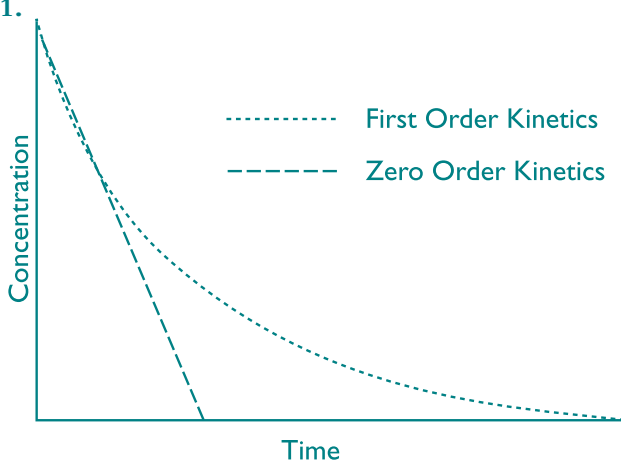
Elimination

Elimination is the pharmacokinetic process of getting the drug out of the body. Drugs are eliminated in two major ways—referred to as *zero order* and *first order* kinetics or elimination. Ethanol is eliminated at a *fixed* or *linear* rate which means that the body eliminates it at a relatively constant amount per unit of time (zero order kinetics). However, most drugs are eliminated using *first order* kinetics, which means that elimination is non-linear. Rather than referring to a steady elimination rate (e.g. 0.015 g/100mL/hour for ethanol), drug elimination is typically characterized by a variable *half-life* or $T_{1/2}$ (Table 5). When a drug is metabolized in a non-linear fashion, it is *generally* not possible to extrapolate backwards from some known drug concentration to some earlier time and concentration. This is true for the majority of drugs, including cocaine, methamphetamine or THC.

Figure 1 illustrates both *zero* and *first order* kinetics on a graph that plots drug concentration over time. The *zero order* line is straight, while the *first order* line curves over time, depending upon a drug's specific half-life.

Because the elimination rate is not constant, toxicologists cannot perform retrograde calculations for drugs as they might for alcohol.

Figure 1.



It is important to understand the overall dynamic nature of drug pharmacokinetics. The processes of *absorption*, *distribution*, *metabolism* and *elimination* do not occur in a discrete chronological fashion, one simply following completion of the other, but rather, they occur in combination with each other. Initially following drug administration, *absorption* will likely prevail; later, *absorption* wanes and *elimination* becomes the dominant process in the body. Corresponding drug and metabolite concentrations therefore represent the overall net effect of the pharmacokinetic processes at the time of sampling. Similarly, corresponding drug effects are also related to drug pharmacokinetics, or the *timeline* of drug use. For example, initial effects of methamphetamine may include intense euphoria, talkativeness and excitement, followed by dysphoria (unpleasant feelings), lethargy and anxiety several hours later. In addition to the relatively complex way in which many drugs are eliminated, the additional presence of active metabolites creates yet another level of consideration or complexity to the interpretation.

A simpler way to consider *elimination* is this analogy: a baseball dropped by a 10-year-old child sitting in a tree house, high above the ground, will fall straight down (alcohol *zero order* elimination). With practice, the child

might even be able to reasonably predict how long the ball takes to hit the ground (alcohol retrograde). If that same 10-year-old then drops a maple leaf attached to an acorn, it should hit the ground at about the same time as the baseball (other drugs with *zero order* elimination). However, what would happen if he dropped only the leaf without the acorn? It will drop much more slowly—as it is tossed and turned in the breeze—than the baseball or the leaf and acorn. The leaf’s size also changes during its descent as pieces break off in the wind (changing drug half-life); this also causes its rate of descent to slow. Eventually the leaf gets to the ground, but not in a straight line nor in a necessarily highly predictable time frame (drug *first order* elimination).

Pharmacodynamics: The dose-response relationship.

The *effect* of a drug is a result of the drug’s interaction at a given receptor site. Drugs that affect the central nervous system must reach and bind to specific receptors for their effects to be exhibited. These drugs act to either stimulate or depress certain areas of the brain to achieve a response, i.e. reduce pain, elevate mood, cause sedation, etc. Typically, an increase in the concentration of the drug modulates the receptor response and enhances the pharmacologic effect. A relationship exists between the amount of drug administered (dose) and the corresponding effect (response) on the body, including the extent to which it may “impair” normal function. This is the basis of the *dose-response relationship*.

The *duration* of a drug’s effects can be estimated, but these may vary with dose. Residual effects may exist long after the “acute” effects of the drug have been experienced (Table 5). The link between the amount of drug and its effect over time is the basis for establishing therapeutic and toxic drug concentrations. These ranges are widely published for clinical purposes, but there are no “therapeutic concentrations” for many illicit drugs.

Remember: A habitual drug user may develop a tolerance to the toxic effects of a drug, allowing him or her to withstand concentrations of drug that may be highly toxic or even fatal in a naïve (inexperienced) subject.

The pharmacologic effect can be intrinsically dependent on the time since the dose, rather than the concentration of drug in the blood. This phenomenon is referred to as *hysteresis*. For example, after consuming ethanol, a person tends to feel more excited and euphoric during the initial absorption phase than during the elimination phase, during which time they may feel more sedated and depressed (*Mellanby effect*). CNS stimulants like methamphetamine follow a similar cycle. A given methamphetamine concentration (say 0.2 mg/L) in blood may coincide with euphoria, exhilaration, restlessness and stimulation during the initial absorption phase. However, several hours later, the same drug concentration may coincide with confusion, depression, anxiety and exhaustion during the elimination phase. THC exhibits a counterclockwise *hysteresis*, indicating a slight delay between the effects and blood concentration. In other words, as the THC concentration decreases, the subject claims to maintain a subjective “high.”

The pharmacologic effect experienced by the user may be apparent from vital signs, involuntary reflexes or behavior. For the purpose of determining impairment, acute or chronic toxicity, blood is considered by most to be the preferred specimen. If a drug is in the blood, it is able to circulate and bind to receptors. While a number of laboratories across the country use urine samples with great success, the presence of the drug in *urine* is an indication of drug exposure over a period of hours, days or even weeks (evidence of *past use*). For this reason, additional information such as observations, behavior or clinical signs is very important to the toxicologist. The *presence* or *absence* of characteristic signs may be of interpretive value.

With the exception of ethanol, there is so far no widely accepted correlation between the drug concentration in blood and a corresponding level of driving impairment among the scientific community. What is more, factors such as tolerance can have a profound effect on the pharmacodynamic response in an individual. A quantity of cocaine sufficient to produce a mild “buzz” in a chronic user could be acutely cardiotoxic in a naïve (inexperienced) user, resulting in coma and death.

Remember: Vital signs, symptoms and behavioral response observed by clinicians and law enforcement personnel are highly relevant during toxicological interpretation.

Table 5. 9-11

Drug	Clinical Dose	Half-Life	Vd (L/kg)	Duration of Effect [#]	% Excreted unchanged in urine	Principal Metabolite(s)
Alprazolam	0.25-2mg	6-27h	0.9-1.3	4-8h	20%	alpha-hydroxyalprazolam ⁺
Carisoprodol	200-350mg	0.9-2.4h	-	4-6h	<1%	meprobamate ⁺
Cocaine	40-100mg	0.5-1h	1.6-2.7	1-2h	<10%	benzoylecgonine, ecgonine methyl ester
Diazepam	4-40mg	21-37h	0.7-2.6	4-8h	<1%	nordiazepam ⁺ , temazepam ⁺ , oxazepam ⁺
GHB	2-4g	0.3-1h	0.4	3-6h	-	-
MDMA	-	8h	5-8	1-4h	65%	methylenedioxyamphetamine (MDA) ⁺
Methadone	20-200mg	15-55h	5	12-24h	5-50%	2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine (EDDP)
Methamphetamine	2.5-15mg	6-15h	3-7	2-4h	10-20%	amphetamine ⁺
Morphine	5-30mg	1.3-6.7h	2-5	4-6h	<10%	conjugated morphine
Oxycodone	2.5-30mg	4-6h	1.8-3.7	4-5h	15%	oxymorphone ⁺ , conjugated oxycodone
THC	2.5-10mg	20-57h	4-14	3-6h	<1%	11-nor-9-carboxy-delta-9-THC (carboxy-THC)

*Half-life of drug metabolite(s) not indicated in this table

[#]Residual effects may last for extended periods

⁺Pharmacologically active metabolite

HOW CAN DRUGS IMPAIR DRIVING?

Drugs can impair driving by affecting some of the important skills necessary for safe operation of a vehicle (Table 6). In fact, drug manufacturers commonly issue warnings for prescription or over-the-counter drugs, indicating that the drug may impair mental or physical abilities required for performing hazardous tasks such as driving.

Coordination

Coordination and psychomotor control are essential because driving is a physical task. Drugs that affect nerves and muscles may impair braking, steering, acceleration and manipulation of the vehicle. Once a driver decides to brake, accelerate, swerve, etc., he or she must be able to effectively carry out the braking, accelerating, swerving. Braking too suddenly or too late, or using the wrong amount of force on the steering wheel and over- or under-correcting, can result from drug impairment.

Judgment / Decision-making

Drivers must process information and then make appropriate decisions. Some drugs affect cognition and have the potential to impair the ability to concentrate, detect, anticipate risk, avoid hazards or make emergency decisions. Mood-altering drugs have the potential to affect judgment. For example, stimulants like cocaine or methamphetamine can produce exhilaration, excitement and feelings of mental and physical power. This type of adrenergic response may in turn influence driving behavior—e.g., increased risk-taking.

Perception

The majority of information that a driver processes is visual. Drugs that can produce visual or auditory distortions, or drugs that can affect perception of time and distance (e.g., marijuana) have the potential to impair driving. A side effect of some depressant drugs and therapeutic

medications is blurred vision. Visual disturbances are also reported with other drugs, such as cocaine, which can cause flashes of light in peripheral vision, known as “snow lights.”

Tracking

Tracking is necessary in order to maintain position on the roadway. Depressant drugs and marijuana, as well as inhalants and PCP, can impair tracking ability. This is sometimes observed as weaving or the inability to maintain the vehicle within the lane (the constant minor over-corrections seen in an attempt to stay within the lane).

Reaction Time

A driver must not only receive information, but must also process it, make a decision, and then react. Slowed reaction times (reaction deficits), particularly with respect to braking and steering, may result in characteristic driving behavior, for example, striking a fixed object, rear-ending another vehicle, or failure to make an evasive maneuver. Several drugs can impair reaction time, in particular depressants.

Divided Attention and Multitasking

Driving requires divided attention, rather than focused attention. Divided attention involves the performance of multiple tasks, simultaneously –i.e., multitasking. Drivers must observe road signals and monitor pedestrians and other vehicles in addition to the environment. At the same time, they must effectively operate the gas, gears, braking and steering systems. While many of these functions are well learned, the driving task itself has a high demand for information processing. Ingestion of depressant drugs or marijuana may impair divided attention skills, as may stimulants, which may produce hypervigilance, preoccupation or distractibility.

Progressive symptoms and impairment of some commonly encountered drugs are summarized in Table 6. Differences between individuals as well as differences within the same individual at different times can produce different responses. For example, an individual with a headache takes two

aspirin and a short time later the headache is gone. A week later that same individual again has a headache, takes two aspirin, but the headache remains, although to a lesser degree. Another person never takes aspirin for headaches, only acetaminophen, because aspirin causes ringing in her ears and doesn't seem to make the headache go away.

The scientific evaluation of driving performance is technically and logistically complex. Various approaches have been taken. Although more than half (56%) (12) of people who reported driving after marijuana use claimed that the drug did not affect their ability to drive, it is highly questionable whether or not individuals can assess their own driving performance.

Table 6.⁹⁻¹¹

Drug	Progressive Symptoms	Impairment
Alprazolam*	Drowsiness, confusion, light-headedness, weakness, poor coordination, blurred vision, fatigue, irritability	Subjective sedation, impaired vision, reaction time, memory, tracking, vigilance, cognitive function, psychomotor function
Carisoprodol*/ Meprobamate*	Drowsiness, dizziness, ataxia, slurred speech, tremor, irritability, syncope, weakness	Attention, reaction time, subjective sedation, psychomotor function
Cocaine	Restlessness, euphoria, dizziness, mydriasis (dilated pupils), hyperactivity, irritability, dyskinesia (impairment of voluntary movements resulting in fragmented or jerky movements), anxiety, tremor, dysphoria (state of unwellness or unhappiness), insomnia, psychosis, fatigue, lethargy	Subjective confusion, perception, hallucinations, judgement

Table 6.⁹⁻¹¹ continued

Drug	Progressive Symptoms	Impairment
Diazepam*	Drowsiness, lethargy, ataxia (uncoordinated movement), dizziness, confusion	Vigilance, reaction time, memory, subjective sedation, attention, perception, anticipation of hazards, speed control, tracking, psychomotor function
GHB (Gamma Hydroxybutric Acid)	Drowsiness, lethargy, euphoria, confusion, disorientation, slurred speech, ataxia (uncoordinated movement), nausea, vomiting, mydriasis (dilated pupils), reduced inhibitions, dizziness, unconsciousness	Cognitive function, psychomotor function, loss of peripheral vision, visual disturbances
MDMA (Ecstasy) (Methylenedioxy-methamphetamine)	Sensory disturbances, nausea, dizziness, ataxia (uncoordinated movement), diaphoresis (excessive perspiration), muscular rigidity, restlessness, tremor	Subjective excitability, perception, cognitive function, attention, memory, psychomotor function
Methadone*	Drowsiness, dizziness, weakness, disorientation, miosis (constricted pupils), light-headedness, visual disturbances	Vision, reaction time, subjective sedation

Table 6.⁹⁻¹¹ continued

Drug	Progressive Symptoms	Impairment
Methamphetamine*	Restlessness, euphoria, dizziness, mydriasis (dilated pupils), dyskinesia (impairment of movements resulting in fragmented or jerky movements), tremor, dysphoria (state of unwellness or unhappiness), insomnia, irritability, nervousness, rapid speech, confusion, agitation, hyperactivity, psychosis, fatigue, somnolence, anxiety, delusions	Perception, judgment, attention, psychomotor function
Morphine*	Drowsiness, dizziness, lethargy, ataxia (uncoordinated movement), miosis (constricted pupils) visual disturbances, weakness, confusion	Subjective sedation, reaction time, psychomotor function, cognitive function
Oxycodone*	Drowsiness, dizziness, lethargy, miosis (constricted pupils), weakness, confusion	Psychomotor function, subjective sedation, reaction time
THC*	Ataxia (uncoordinated movements), confusion, dizziness, somnolence, euphoria, relaxation, hallucinations, speech difficulty, weakness, malaise, visual disturbances, paranoia	Perception, subjective sedation, reaction time, memory, vigilance, attention, emergency decision making, psychomotor function, cognitive function

* Indicates warning from manufacturer

MEASURING IMPAIRMENT

Although the scientific literature on the effects of drugs on driving skills is extensive and increasing, a great deal more work remains to be done. For ethical and safety reasons, on-the-road driving studies using “real-world” doses of drugs like cocaine and methamphetamine are not feasible. Therefore, a toxicologist must rely on a number of approaches, which may include:

- **Empirical Considerations:** What is the pharmacology of the drug? What effects does it produce? How long does it last?
- **Epidemiological Studies:** Retrospective studies that discuss drug use/driving behaviors in a given population of drivers.
- **Case Reports:** Actual published reports of impaired drivers in the literature.
- **Laboratory Studies:** Administer drug and evaluate psychophysical tests, for example, response time, motor control, divided attention, memory, vision, mood effects or subjective effects in a controlled setting.
- **Simulator Studies:** Administer drug and evaluate performance in a driving simulator, for example, lane position, speed, steering, reaction time, decision-making or vehicle manipulation.
- **Actual Driving Studies:** Administer drug and observe actual driving performance in a real-world setting, for example, highway driving or city streets.

There are advantages and disadvantages associated with each approach and these are summarized in Table 7. Collectively, these approaches can provide a toxicologist with a great deal of useful information. Taken together, the scientific literature helps determine whether the drug effects are compatible with safe driving, and specifically how they might impair a person’s ability to drive.

Drugs may affect normal behavior by enhancing or impairing human performance, such as cognition or psychomotor skills. The same drug may be capable of either enhancing or impairing performance, depending on the dose and pattern of drug use. For example, in laboratory stud-

ies, single low doses of amphetamine (5–15 mg) and methamphetamine (10–30 mg) have been shown to improve alertness and psychomotor performance in healthy and sleep-deprived individuals. Real-world doses of methamphetamine far exceed those used in the controlled studies. Epidemiological studies, as well as empirical knowledge of the drug effects at elevated dose, strongly suggest that methamphetamine can impair skills necessary for safe driving.

Individuals may claim their driving ability was enhanced through drug use, so be aware of study conditions and be able to explain the relative merits and caveats. In a similar manner, studies that evaluate drug combinations are readily misrepresented. For example, laboratory studies have shown that a single low dose of stimulant (methamphetamine) can offset sedation caused by a depressant (alcohol). This does not equate to a *reversal* of effects or a *zero net effect*. Alleviation of sedation in no way infers that a stimulant will reverse all of the impairing effects of alcohol (judgment, attention, psychomotor function), or vice versa.

Table 7.

Approach	Advantages	Disadvantages
Empirical Considerations	Readily supportable Extensively published Can draw inferences for similar drugs	Does not anticipate an atypical response Does not account for poly-drug use or drug interactions Not driving or individual specific Not environment or situation specific
Epidemiological Studies	Easily conducted May show differences between populations May display trends Large data pool	Largely descriptive and non-specific Inferences difficult to make May be time/location sensitive
Case Reports	Involves real-world doses Actual effects Relevant populations Personalized data First hand accounts	Anecdotal Lack of control data
Laboratory Studies	Isolates an individual task Controlled environment Moderately safe	Real-world doses may not be administered Does not simulate driving Learning effects may develop towards tests
Simulator Studies	Controlled environment May approximate driving task Moderately safe	Real-world doses may not be administered No consequences or real danger Not real driving Small sample size
Actual Driving	Realistic driving situation Potential for real consequences Closely approximates driving task	Real-world doses may not be administered Liability issues Ethical constraints Small sample size Infrequently conducted

TOXICOLOGY AND THE DRUG EVALUATION AND CLASSIFICATION (DEC) PROGRAM

The DEC Program provides specialized training and certification of law enforcement personnel commonly known as Drug Recognition Experts (DREs). The DEC process is a systematic, standardized, post-arrest procedure that can be used to determine whether a person is impaired by one or more categories of drugs. The evaluation is based upon a variety of observable signs and symptoms which are proven to be reliable indicators of drug impairment. For additional information, visit the NTLC website at www.ndaa-apri.org or contact the NTLC at **703-549-4253** or TrafficLaw@NDAA-APRI.org.

The observations and measurements that are made by a certified Drug Recognition Expert are extremely important to the toxicologist. DREs utilize a series of physiological and psychomotor tests to determine the category or categories of drug present: CNS stimulants, CNS depressants, narcotic analgesics, hallucinogens, PCP, cannabis or inhalants. Unlike the classification schemes that are often used by toxicologists, the categories used by the DEC program are not based on shared chemical structures, but rather on the “signs” (detectable by an observer, such as bloodshot eyes) and “symptoms” (the subjective experience of the user, such as nausea). It is the pattern of the effects, rather than a specific effect, that determines the DEC category.

A DRE’s ability to identify the category of drug is based upon his or her familiarity with the documented or known effects of the drug. The DRE evaluation itself is unique only from the standpoint that it provides a standardized and systematic approach to data collection. Clinical characteristics such as blood pressure, pulse, respiration, body temperature, nystagmus, ocular convergence (ability to cross eyes), pupil size and pupillary reaction to light can be useful indicators of drug use. A detailed summary of the effects associated with each drug class (DRE Matrix) is

available through the NTLC and the International Association of Chiefs of Police (IACP). Other observable effects, such as tremors, coordination, gait, muscle tone, perception, diaphoresis (extreme sweating), emesis (vomiting), lacrimation (excessive tearing) and appearance of the conjunctiva may also provide valuable insight (Table 2). As discussed earlier, abstinence or withdrawal syndromes resulting from chronic drug use produce effects that vary considerably from those caused by acute drug intoxication (Table 3).

DRUG DETECTION AND IMPLICATIONS FOR DRIVER IMPAIRMENT

The duration and the intensity of a given drug's effects depend on the dose administered, individual metabolism, frequency of drug use and the presence of other drugs. Because many of these factors are unknown, toxicological interpretation is often difficult. Questions regarding administration time can sometimes be answered using the pharmacokinetic principles, such as drug half-life. For a drug that is eliminated by *first order* kinetics, 99% of the drug is eliminated by seven half-lives, with less than 1% remaining in the body. By ten *half-lives*, 99.9% has been eliminated. Although detection times for different drugs can be estimated, these vary with dose, method of analysis and metabolic factors. Although the concentration of a particular drug in a blood sample provides important information, it should be considered in conjunction with reports of driving behavior, physiological signs and other data.

Blood and urine are the most frequently encountered biological fluids in DWI casework. However, DWI statutes in some states make provisions for alternative specimens, for example, saliva. The benefits and weaknesses of blood, urine and saliva samples are described below:

Blood

Advantages:

- A drug that is circulating in the blood may bind to receptors in the brain.
- Less-readily adulterated than urine due to method of collection.
- Quantitative, meaning the amount of drug in the blood may have some interpretive value.
- Detection times are much shorter than in urine. Therefore, a blood sample that contains a drug is more likely to indicate recent usage compared to a urine sample.

Disadvantages:

- Many drugs have a limited detection window in blood; it may be difficult to collect a sample in a short period of time (transporting individual from scene to collection site, etc.).
- There are complicated statutory regulations or protocols governing who is qualified to collect the sample and how it must be collected, processed and stored.
- Testing of blood is labor intensive and expensive compared to urine.

Urine

Advantages:

- Easily collected.
- Can be screened for drugs more readily (less laboratory time required as compared to blood testing).
- Longer detection times for most drugs or metabolites.

Disadvantages:

- Limited quantitative meaning for most drugs. In the absence of other information, a urinary metabolite reported as “present” may have limited significance when trying to determine whether the individual was impaired.
- More-readily adulterated, therefore requires careful collection procedures to prevent the sample from being compromised (e.g., diluted, replaced or manipulated by use of an additive or “masking agent”).
- Urinary detection times are even more difficult to predict due to differences in fluid intake, diuresis (excessive elimination of urine) and the effect of urinary pH on drug elimination. The relative acidity or alkalinity of the urine can determine how quickly a particular drug is eliminated from the urine. However, urine drug results may be useful in determining an approximate time frame during which drug exposure took place. For example, the heroin metabolite 6-acetylmorphine is detectable in urine for approximately 2–8 hours after ingestion.

Saliva

Advantages:

- Easily collected.
- Can be screened for drugs easily.

Disadvantages:

- Some pharmacological interpretation may be possible but there is limited reference data at present.
- Many drugs have limited detection window in saliva.
- Drugs partition into saliva from the blood to varying degrees; the degree to which a particular drug is present in saliva depends on many variables, including the pH of the saliva.
- Possibility of sample adulteration (by mouth).
- Relatively small volume available for analysis—this may prevent defendant from obtaining “independent test.”

Remember: Because the drug dose usually is unknown, it is generally not possible for a toxicologist to determine exactly when the drug was administered. However, the toxicologist may be able to infer an approximate *window of drug use*. For example, THC increases very rapidly during marijuana smoking, and upon cessation, is eliminated rapidly from the blood. As a result, elevated levels of THC in blood are a good indication of recent drug use. Cocaine has a short half-life and is relatively unstable. Therefore, the presence of elevated levels of cocaine in a blood sample may also indicate moderately recent use. The meaning of “recent” use will vary from one drug to the next.

The characterization of certain, specific concentrations of drugs in blood as therapeutic, toxic or lethal is often useful, but must be assigned with caution due to inter-individual differences. These ranges overlap for some drugs, making it difficult to classify the concentration in this way.

Remember: Human performance may be impaired at therapeutic concentrations.

A therapeutic level of a hypnotic or sedative drug can impair driving due to the central nervous system depressant effects. Even low or sub-clinical concentrations of some drugs in blood are associated with impaired driving. Following chronic use of a stimulant drug like methamphetamine or cocaine, an individual may experience extreme fatigue and exhaustion, consistent with the “crash” phase of drug use, sometimes called the “downside.” During this time, when an individual is experiencing the negative reinforcing effects, drug concentrations are much lower than during the acute or “high” phase, when positive reinforcing effects predominate. Thus, toxicological interpretation is usually based upon a combination of toxicological analyses, case information, and field observations made by law enforcement personnel or clinicians who may have had contact with the individual.

Multiple drug use can complicate interpretation, so drug combinations need to be examined in terms of their ability to interact with each other and produce additive, synergistic or antagonistic effects:

- *Additive effects* occur when a combination of drugs produce a total effect that is equal to the sum of the individual effects
- *Synergistic effects* occur when a combination of drugs produce a total effect that is greater than the sum of the individual effects
- *Antagonistic effects* occur when the effect of one drug is lessened due to the presence of another drug

A trained toxicologist will be familiar with the types of drugs that can have additive, synergistic or antagonistic effects.

Interpretation of toxicology results is compounded by a number of factors which includes, but is not limited to multiple drug use, history of drug use (chronic vs. naïve user), overall health, metabolism, individual sensitivity, individual response and withdrawal. The same dose of drug given to two individuals may possibly produce similar effects but with varying degrees of severity that elicits a different response. The presence of a drug *alone* in a person’s blood or urine does not necessarily mean that he or she was impaired. Other information, such as documentation of appearance, behavior, performance on standardized field sobriety tests,

DRE evaluation or driving behavior, is also important. Based on a combination of these factors (Figure 2) it is often possible for a toxicologist to provide expert testimony regarding the consistency of this information with driving impairment.

Figure 2.



TESTING METHODOLOGY IN THE FORENSIC TOXICOLOGY LABORATORY

Most forensic toxicology laboratories that routinely analyze DWI case samples for drugs utilize a two-tiered approach. Initially, samples are *screened* for common drugs or classes of drugs using an antibody-based test. Samples that screen positive are then re-tested using a second, more rigorous technique, usually called *confirmation*.

Screening Tests (Presumptive Tests) vs. Confirmatory Tests

Assume for a moment that you have in your hand a key ring with ten keys, all made of brass, all appearing to have the same cut. In front of you is a door with a lock. A few of those will fit in the lock (screening test with false positives since the keys are structurally similar to each other) but only one will actually turn and unlock the door (confirmation test). This holds true for drug testing, as well.

Screening Tests

An *immunoassay* test is the most common type of screening test for drugs of abuse. Using this type of test, a drug or metabolite in a biological sample can be tentatively identified using an anti-drug antibody. If a drug is present in the sample, the anti-drug antibody will bind to it; if no drug is present in the sample, the anti-drug antibody will not bind to the sample. Various methodologies and detection methods are utilized, giving rise to a number of immunoassays. These include enzyme linked immunosorbent assays (ELISA), enzyme multiplied immunoassay technique (EMIT), fluorescence polarization immunoassay (FPIA), cloned enzyme-donor immunoassay (CEDIA) and radioimmunoassay (RIA).

Immunoassay test results are considered *presumptive*, not conclusive, because the antibodies that are used may *cross-react* with other substances to varying degrees, resulting in false positive results. Analogs or substances that are structurally similar to the drug are most likely to produce a false positive. For example, common over-the-counter cold medicines that

contain pseudoephedrine may cause a false positive methamphetamine immunoassay result.

Most laboratories utilize screening tests only to determine which drugs or classes of drugs might be indicated. This allows confirmatory tests to be performed for the drugs indicated by the immunoassay. Since it is unfeasible to test every sample for every drug using confirmatory protocols, screening tests are used principally to determine where to focus analytical resources in the laboratory.

Cut-offs

The immunoassay test will have a *cut-off* value or threshold concentration, above which a sample is considered positive. Cut-off concentrations for urinary workplace drug testing are federally mandated by the Substance Abuse and Mental Health Services Administration (SAMHSA). These cut-off concentrations do not apply to forensic testing in DUID casework. The majority of state toxicology laboratories that perform drug tests in criminal casework set cut-off concentrations below the SAMHSA guidelines. This is because workplace drug testing cut-offs in urine are set so that inadvertent drug exposure (e.g. poppy seed ingestion) does not produce a positive drug test. As a result, the cut-offs are elevated so that workers who unintentionally expose themselves to drugs are not penalized. The forensic toxicology laboratory may utilize lower cut-off concentrations for blood samples compared with urine because of reduced detection times and concentrations in blood compared to urine. It is essential for law enforcement personnel to understand the implications of a negative laboratory result in this context.

Confirmatory Tests

The confirmatory test is more specific and usually more sensitive than the initial immunoassay test. The most frequently used confirmatory technique is gas chromatography–mass spectrometry (GC-MS or “GC-Mass Spec”) although others include high performance liquid chromatography (HPLC), liquid-chromatography–mass spectrometry (LC-MS) and others.

The increased specificity of the confirmatory technique allows the drug to be qualitatively identified, i.e. the ability to determine specifically which drug is present. For example, GC-MS can be used to distinguish structurally related drugs such as pseudoephedrine from methamphetamine. A quantitative analysis may be performed in blood samples, whereby the concentration of the drug is determined.

Unlike the screening tests described earlier, which are performed with little or no sample preparation, confirmatory drug tests usually require extensive sample preparation or “clean up.” In other words, the drug must be isolated from the biological sample prior to testing on the instrument. This is typically achieved using liquid-liquid extraction or solid phase extraction, whereby drugs in a complex mixture (e.g., blood, urine) are separated from the biological sample. Once the drugs are extracted from the sample, they can then be subjected to confirmatory analysis. For this reason, confirmatory tests are a great deal more labor-intensive than screening tests. Depending on the number of drugs that are present, it may take several days to complete the tests because each drug may require a different extraction and separate confirmatory analysis.

The basis for most confirmatory techniques is separation and positive identification. GC-MS is considered the “gold standard” for methods of confirmatory drug identification. In this method, individual components (drugs and metabolites) are first separated, based upon their chemical and physical properties, by the gas chromatograph (GC). The separated drug(s) then enters the mass spectrometer (MS), where it undergoes molecular fragmentation, resulting in a characteristic mass spectrum or fragmentation pattern. This “molecular fingerprint” of the drug, together with the characteristic retention time from the gas chromatograph allows the drug to be positively identified.

A helpful and widely used analogy for the GC-MS method is the following: inside the GC oven is a long, thin, coiled column; think of this column as a racetrack with different types of vehicles (drugs) traveling around it. Some cars are small and fast (methamphetamine), others big and slow (alprazolam); the road conditions (internal coating of the column) also dictate which cars travel faster—cars with special tires might

perform better in the snow, etc. As the cars travel around the track at different speeds they become separated and ultimately each crosses the finish line (the MS detector) and generates a unique “retention time.” At the finish line, each car is involved in a serious collision and is essentially blown apart by the MS; this generates pieces (molecular fragments) of the car, such as a bumper, hood, headlight, etc. These pieces are then compared with other cars of the same make, model and year (drug standards)—which allows for a near perfect overlay of car parts (unique drug fragmentation patterns) and finish times (retention times) for a positive drug identification. The GC-MS identification is based fundamentally upon how drugs are “put together” or arranged chemically, including molecular attractions which ultimately dictates how a molecule or drug will fragment or “blow up.”

CASE PREPARATION AND THE TOXICOLOGIST AS EXPERT WITNESS

Depending upon the evidentiary rules in your jurisdiction, a toxicologist may be necessary to testify at trial to establish the authenticity of the toxicology report, chain of custody and the implication and validity of the test results. Even if such testimony is not necessary to get the evidence admitted, prosecutors must consider carefully the additional benefits of having the toxicologist present to interpret the test results and provide expert testimony. Obviously, manpower concerns and costs associated with expert testimony likely limit the use of a toxicologist, but in some cases, expert testimony from a toxicologist might be essential. This is especially true in DUID cases, where the effects of drug or poly-drug consumption, and the meaning of drug concentrations, are not a matter of common knowledge to the layperson.

It is unlikely that a toxicologist will unequivocally state that all drivers who have a drug or metabolite in their blood or urine are impaired. Determination of impairment requires a case-by-case evaluation, so be sure to obtain the opinion of a toxicologist well before trial. Nothing is worse than having your own witness deliver an unexpected opinion to the jury. Since drug effects are complex, toxicologists may ask many questions before they can arrive at an opinion:

- How was the person driving?
- What was the reason for the traffic stop?
- Was there a crash?
- Was the person injured? What was the nature of the injuries?
- If so, were medications administered at the hospital?
- What is known about the overall health of the individual?
- Were field sobriety tests performed? If yes, what were the results?
- Was a DRE evaluation performed? What were the results?
- What signs, symptoms or behaviors were documented (motor skills, speech patterns, eye movements, etc)?

When the DRE Opinion Differs from the Toxicology Report

In most circumstances, the DRE opinion and the toxicology report agree. But toxicology results that do not agree may need to be addressed by a toxicologist who is familiar with the DRE evaluation process. For example, in the “crash” or “downside” phase of stimulant use, a person experiencing extreme fatigue and exhaustion may appear to be under the influence of a depressant or narcotic drug. Marijuana and stimulants increase blood pressure, increase pulse, and can produce eyelid or body tremors. Stimulants tend to speed up the internal clock and dilate pupils, and marijuana can distort pupil size and the internal clock. These similarities in the known effects of drugs at varying phases of ingestion or elimination can sometimes make it more difficult to identify the class of drug responsible. This is further complicated by poly-drug use, whereby the individual has ingested any number of substances, each of which exhibits certain characteristics on its own, but together these substances likely result in a whole host of contradictory signs and symptoms.

Laboratories cannot test for every known drug. Testing is both labor-intensive and expensive. Each laboratory will likely have a policy for drug testing in DUID cases that may limit the scope of the tests that are performed. Some laboratories may screen samples only for common classes of drugs to the exclusion of other, less common drugs, while other labs may conduct exhaustive toxicology. Be familiar with both testing protocols and policies governing how drug-related DWI casework is handled. Keep in mind as well that as newer drugs are developed a screening and confirmatory test may not yet exist. For these reasons, a negative toxicology report does not conclusively mean no drugs are in the person’s system. It may simply mean that the scope of the testing was too limited, the cutoff was too high, or a test for that particular drug was not available.

Witness Selection

Forensic toxicology can be divided into three main fields: post-mortem toxicology, workplace drug testing and human performance toxicology. *Human performance toxicology* is concerned with the mental and physical effects of drugs that may impair a person’s ability to safely operate a

motor vehicle. This is a challenging field, and an expert witness must be familiar with this sub-discipline.

Because of the breadth and scope of toxicology, it is important to determine that the “expert” has the necessary credentials. For example, a clinical toxicologist who performs drug tests in an emergency room or hospital may not be familiar with the effects of drugs on driving. Likewise, a toxicologist employed in the field of workplace or employee drug testing may not have expertise in *human performance toxicology*. The following questions may help identify the most appropriate witness for expert testimony (See Appendix for additional questions):

- What type of toxicologist are you?
- Are you familiar with the field of performance toxicology and in particular, the effects of drugs on driving?
- How did you gain this familiarity?
- What specialized training have you received in this area?
- Are you familiar with drug testing methodology and interpretation of the results?
- How many times have you been qualified as an expert witness on the effects of drugs on driving and in what courts?

CONCLUSION

DUID cases are both common in occurrence and complex to prosecute—legally, scientifically, and from a public policy standpoint. While prosecutors need not attain the depth of knowledge of a forensic toxicologist to do justice in these cases, it is essential to have a basic understanding of the scientific principles, together with effective channels of communication with the law enforcement officers and toxicologists who serve your jurisdiction.

CASE STUDIES

Case #1:

A 47-year-old female was apprehended for erratic driving. She was weaving, crossing the center line and striking the median. The officer noticed she had glassy eyes and slurred speech. During the Standardized Field Sobriety Tests (SFSTs), she had difficulty following instructions, maintaining balance and HGN was present. A breath alcohol test was negative, so a blood sample was drawn for drug testing. Toxicology tests indicated the following drugs: Chlordiazepoxide (1 mg/L), nordiazepam (0.6 mg/L), phenobarbital (8.8 mg/L), morphine (70 ng/mL) and codeine (less than 25 ng/mL).

CLAIM: I was only taking my prescribed medicine.

REALITY: The poor driving, observations, appearance and performance on SFSTs were well-documented. The toxicology report indicates several prescription depressant drugs and narcotic analgesics. The observations and driving behavior are consistent with someone who is under the influence of a central nervous system depressant. Having a valid prescription is not a legitimate defense in most states that adopt “affected by” DWI statutes.

Case #2:

A 39-year-old male was apprehended for an improper lane change. He was jittery, could not stand still, had rapid speech and spoke to himself during the SFSTs. He had difficulty balancing and had trouble concentrating. He held on to his trousers for support and balance. Eyelid and body tremors were noted. A blood sample was drawn. Toxicology tests indicated the following drugs: Methamphetamine (0.14 mg/L) and amphetamine (0.04 mg/L).

CLAIM: Methamphetamine has been shown to improve performance!

REALITY: The officer documented several characteristic indicators of a central nervous system stimulant. The toxicology confirmed the presence of a stimulant, methamphetamine and its metabolite, amphetamine. The observations are consistent with someone who is under the influence of a central nervous system stimulant drug. Small doses of stimulant drugs have been shown to improve mental alertness and motor performance in fatigued or sleep-deprived drivers. However, stimulants generally do not improve performance in otherwise normal individuals, particularly when they are used for illicit purposes and are taken in doses significantly higher than those used therapeutically.

Case #3:

A 53-year-old male was stopped for a broken tail light. The man performed poorly on the SFSTs and was arrested. No observations of appearance, demeanor or physical appearance were documented in the police report. A blood sample was drawn. Toxicology tests indicated a blood alcohol concentration of 0.05 g/100mL. Because the BAC was below the per se limit a drug test was performed. Benzoylcegonine (0.3 mg/L) was also present.

CLAIM: *I used cocaine yesterday.*

REALITY: A cocaine metabolite, benzoylcegonine was detected in the blood sample. It is not possible to reliably determine when the man used cocaine based upon the test result and the unknown dose. More importantly, there are no characteristic indicators of a stimulant drug in the police report. Based on this information, it is not possible to determine whether the driver was under the influence of a drug, although it is possible that the poor performance on the SFSTs might be attributed to the alcohol.

Case #4:

A 48-year-old man swerved into oncoming traffic, resulting in a near collision. The officer noticed that his speech was extremely slurred; he

had watery eyes, and was extremely unsteady on his feet. During the SFSTs the driver was unable to maintain balance and fell down. The tests were stopped for his own safety. HGN was present. He told the officer he drove onto the wrong side of the road because he dropped a tamale and was leaning over to pick it up. The driver stated he had medical problems including a back injury. A blood sample was drawn and sent for alcohol and drug testing. Toxicology tests revealed the following: Morphine (50 ng/mL), meprobamate (20 mg/L), carisoprodol (2 mg/L), oxycodone (130 ng/mL), hydrocodone (80 ng/mL), diazepam (0.3 mg/L) and nordiazepam (0.3 mg/L).

CLAIM: *I was distracted.*

REALITY: The poor driving, observations, appearance and performance on SFSTs were well documented. The toxicology report indicates several prescription depressant drugs and narcotic analgesics. The observations and driving behavior are consistent with someone who is under the influence of a central nervous system depressant. Many depressant drugs impair our ability to divide attention, so performing non-essential (distracting) tasks may further compromise our driving.

Case #5:

A 23-year-old female was apprehended for erratic driving. She crossed over the center line three times. The officer noticed the woman appeared relaxed, her eyes were red, and she appeared dazed or disoriented. During the SFSTs the woman was unable to remember the instructions and the test had to be restarted a number of times. She was unable to maintain balance. She admitted drinking two beers earlier in the afternoon. A blood sample was drawn and sent for alcohol and drug testing. Toxicology tests indicated the following results: Ethanol (0.05 g/100mL), THC (4 ng/mL), and carboxy-THC (53 ng/mL).

CLAIM: *I smoked marijuana yesterday.*

REALITY: The poor driving, observations, appearance and performance on SFSTs were well documented. The toxicology report indicates both

alcohol and THC. The observations and driving behavior are consistent with someone who is under the influence of alcohol and marijuana. THC disappears from the blood quickly so elevated concentrations in blood indicate recent smoking.

Case #6:

A 22-year-old male was apprehended for speeding. He had elevated blood pressure, elevated pulse, dilated pupils, and eyelid and body tremors. He had difficulty performing SFSTs due to poor coordination and mental alertness. He appeared extremely lethargic. The DRE officer believed the man to be under the influence of marijuana. A blood sample was drawn. Toxicology tests indicated the following drug: Methamphetamine (0.2 mg/L).

CLAIM: The DRE opinion is incorrect.

REALITY: The officer documented several characteristic indicators of a central nervous system stimulant. Lethargic behavior is consistent with the “down” side of methamphetamine use. Many of these observations are similar to the effects of marijuana, so it can sometimes be difficult to distinguish the two. The observations are consistent with someone who is under the influence of a central nervous system stimulant drug.

ENDNOTES

1. 2003 National Survey on Drug Use and Health: National Findings, Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Office of Applied Studies. Released September 2004 (available online at <http://www.DrugAbuseStatistics.samhsa.gov>).
2. For purposes of most DWI statutes, blood alcohol concentration is measured as a percentage by weight by volume (so a BAC of 0.08 is 0.08 percent by weight by volume) or number of grams per 210 liters of breath as indicated by a chemical test (so a BAC of 0.08 is 0.08 grams per 210 liters of breath). See, e.g., § 18.2-266, Code of Virginia (1950, as amended); § 9-30-5-1, Code of Indiana. For a compilation of DWI statutes, see, *Prior Convictions in DUI Prosecutions; A Prosecutor's Guide to Prove Out-of-State DUI/DWI Convictions*, Zenaida C. Cacnio, Ed., National Traffic Law Center, LEXIS/NEXIS (2003).
3. See, *Alcohol Toxicology for Prosecutors; Targeting Hardcore Impaired Drivers*, John Bobo, Ed., National Traffic Law Center, APRI (2003) (available online at http://www.ndaa.org/apri/programs/traffic/ntlc_home.html).
4. The Feasibility of Drugged Driving *Per Se* Legislation Consensus Report 2002, The Walsh Group, JM Walsh (available online at <http://www.walshgroup.org>).
5. The NSDUH Report: Drugged Driving, 2002 Update. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration (available online at <http://www.samhsa.gov>).
6. US Department of Transportation, National Highway and Traffic Safety Administration, Campaign Safe and Sober, Drug Impaired Driving (available online at <http://www.nhtsa.gov>).
7. Driving under the influence of drugs (DUID) legislation in the United States. The Walsh Group and the American Bar Association's Standing Committee on Substance Abuse. JM Walsh, G Danziger, LA Cangianelli and DB Koehler (2002) (available online at <http://www.walshgroup.org>).
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9. RC Baselt. *Drug Effects on Human Performance and Behavior*. Biomedical Publications, Foster City, CA (2001).
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 11. J Wilson. *Abused Drugs*. AACC Press, Washington DC (1994).
 12. *Driving After Drug or Alcohol Use Report*, 1996 National Household Survey on Drug Abuse, Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Office of Applied Studies (available online at <http://www.oas.samhsa.gov>).

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APPENDIX I: GLOSSARY

Additive Effects	The total effect is equal to the sum of the individual effects of those drugs.
Antagonistic Effects	The effect of one drug is lessened due to the presence of another.
Antidepressant	A substance that is used for the treatment of mental depression.
Ataxia	Inability to control voluntary muscular movement causing staggered or unsteady motion.
Cardiotoxic	A substance that has a toxic effect on the heart.
Central Nervous System	The part of the nervous system (brain and spinal cord) to which sensory impulses are transmitted and from which motor impulses pass out, and which supervises and coordinates the activity of the entire nervous system.
Concentration	The amount of a substance in a specified volume.
Depressant	A drug that causes slows down central nervous system function.
Detection Time	The length of time that a drug can be detected.
Dose	Amount or quantity of drug.

Drug	For the purposes of this article, “drug” is defined as any chemical that affects living processes and has the potential to impair those processes. This includes illicit drugs, prescription medicines, over-the-counter medicines, dietary supplements, herbals and botanicals.
First Order Elimination	Elimination of a substance in a concentration-dependent (non-linear) fashion.
Hallucinogen	A substance that alters perceptions, for example, visual images or sounds.
Hysteresis	The relationship between drug effects and time or the lagging of a physical effect on a body behind its cause.
Mellanby Effect	A form of acute tolerance whereby the perceived effects are more pronounced when the blood alcohol is rising rather than falling.
Metabolite	A byproduct of a drug, formed naturally by the body.
Opiate	A substance that contains or is derived from opium.
Opioid	Natural or synthetic derivatives of opium in addition to drugs that mimic the effects of morphine.
Pharmacokinetics	The manner in which a substance moves throughout the body. This involves absorption, distribution, metabolism and elimination.

Pharmacology	The study of the preparation, properties, uses and actions of drugs.
Route of Administration	The manner or process by which a substance, or drug, enters the body, i.e. intravenously, orally, etc.
Stimulant	An agent that increases the rate of activity.
Synergistic Effect	The total effect of multiple drugs that is greater than the sum of the individual effects of those drugs.
Volume of Distribution	A measure of how widely a drug is distributed throughout the body.
Zero Order Elimination	Elimination of a fixed amount of substance per unit time.

APPENDIX 2: PREDICATE QUESTIONS FOR TOXICOLOGIST

- What is your name, occupation?
- Where do you work?
- Is the laboratory certified or accredited?
- What is your current position?
- What are your job responsibilities or duties?
- How long have you worked at [current job] -previous employment if applicable?
- What is your academic background?
- What education, training or experience qualifies you as an expert on the effects of drugs on driving?
- What specialized training have you received in the effects of drugs on driving?
- Have you testified as an expert on the effects of drugs on driving?
- Did [lab] analyze [sample] of the person in question?
- How was the sample received, identified, packaged and sealed?
- What were you required to do with the sample?
- What was the sample analyzed for?
- What type of testing was used?
- Are these methods generally accepted by the scientific community?
- Were the tests performed in accordance with standard operating procedures?
- Was equipment in proper working order?
- What were the results of the tests?
- Were these results properly recorded [toxicology report]?
- What is [drug]?
- Are you familiar with the effects of [drug]? What are they?
- Is it possible for [drug] to affect driving? How?
- Can [drug(s)] affect a person's ability to drive safely?
- Were you provided with additional information in order to reach an opinion [police report, witness statements, DRE report]?
- Are driving behavior, SFST performance, signs and symptoms, etc. consistent with someone who is under the influence of the drug?
Consistent with impaired driving by the drug?



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