Carisoprodol — Effects on Human Performance and Behavior

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ABSTRACT: Carisoprodol, a commonly prescribed muscle relaxant, has adverse effects on human performance and is gaining recognition as a factor in driver impairment and accident causation. Carisoprodol is a centrally acting skeletal muscle relaxant indicated for the relief of musculoskeletal pain. Carisoprodol and its major metabolite meprobamate have central nervous system (CNS) sedating effects similar to benzodiazepines or alcohol. Following the ingestion of carisoprodol or meprobamate symptoms such as drowsiness, confusion, poor balance, and coordination are well documented in drivers, all of which are detrimental to human performance and driving ability. Although identified as a drug capable of producing decreased human performance, the full extent of carisoprodol and meprobamate's involvement in motor vehicle accidents and effect on driving skills may not be fully appreciated. This is due in part to the common co-administration of other CNS depressants, hypnotics, or narcotic drugs and the lack of routine testing for carisoprodol and meprobamate in the human performance toxicology laboratory.

KEY WORDS: Behavior, carisoprodol, driving, human performance, impairment, meprobamate.

INTRODUCTION

Carisoprodol, a synthetic compound first synthesized in 1959, is related structurally to meprobamate, mebutamate, and tybamate (**Figure 1**). Not currently a federally scheduled compound in the United States, carisoprodol is marketed as a muscle relaxant and dispensed by prescription under the trade names of Soma® tablets, Soma® compound, Soma® with codeine, or Sodol® Compound. Carisoprodol is indicated for the relief of pain associated with acute musculoskeletal conditions and in the treatment of acute muscular spasm [9,23,29]. Carisoprodol is frequently prescribed as adjunct therapy in combination with other medications such as benzodiazepines, opiates, and analgesics.

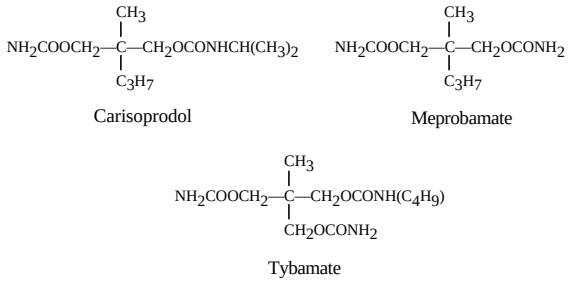

Figure 1. Chemical structures of carisoprodol, meprobamate, and tybamate.

Once absorbed, carisoprodol is rapidly metabolized to meprobamate, a pharmacologically active compound. Meprobamate, a schedule IV drug in the United States, is prescribed under the trade names Miltown®, Equanil®, Neuramate®, and Meprospan®. Meprobamate is a central nervous system (CNS) depressant with sedative hypnotic properties and is indicated for the treatment of anxiety [23]. The pharmacological effects of carisoprodol appear to be due to the combination of the effects of carisoprodol and meprobamate. In addition to the desired skeletal muscle-relaxing effects, carisoprodol and meprobamate also produce weak anticholinergic, antipyretic, and analgesic effects [11].

Like benzodiazepines and opiates, the subjective mood-altering properties of carisoprodol and meprobamate suggest this compound may be susceptible to abuse and misuse. Abusers of carisoprodol become habituated to the pleasurable effects such as relaxation, euphoria, and mood alteration during the time they are being treated for the musculoskeletal condition. These users subsequently continue taking carisoprodol after the symptoms for which it was prescribed have subsided, and often increase the dose above that prescribed for symptomatic control [8,17,21,24,25]. Of interest, anecdotal evidence suggests that the population misusing and abusing carisoprodol does not fit the "typical" drug-abuser profile — i.e., the young adolescent male, recreationally using during evenings and weekends. Marinetti-Sheff and Ludwig reported that the majority of carisoprodol-related driving under the influence (DUI) cases in the Detroit/Flint region of Michigan were middle-aged males and the driving offenses usually occurred during the daylight hours rather than the evening hours [20].

The effects of carisoprodol and meprobamate, which include sedation, loss of balance, confusion, and increased reaction time are similar to those of alcohol, benzodiaz-

epines, and other CNS depressants and are well documented to decrease human performance and adversely affect driving safety. Interestingly when prescribed, both carisoprodol and meprobamate are labeled with warnings regarding their potential effects on complex tasks such as driving and operating hazardous machinery [23], suggesting their effect on human performance is appreciated.

In addition to the effects produced by carisoprodol, drug interactions are of particular concern. Carisoprodol is often prescribed in combination with other CNS depressants such as codeine, hydrocodone, and benzodiazepines. A user may also ingest drugs such as alcohol and sedative antihistamines and drugs of abuse such as cocaine [25].

Carisoprodol is not yet routinely analyzed for in the human performance toxicology laboratory. The magnitude of the effects of carisoprodol either alone or in combination with co-ingested central nervous system depressants, stimulants, or sedative hypnotic drugs is largely unknown. At this stage it can only be inferred from a review of the literature, its pharmacology, and the few publications relating to carisoprodol and human performance.

I. CHEMISTRY

Carisoprodol (*N*-isopropoyl-2-methyl-2-propyl-1,3-propanediol dicarbamate) is a synthetic carbamate derivative. Carisoprodol is *N*-isopropyl meprobamate and hence is chemically, structurally, and pharmacologically related to meprobamate (*see* Figure 1). The molecular weight of carisoprodol is 260.3 ($C_{12}H_{24}N_2O_4$). A white crystalline powder with a bitter taste and a mild, characteristic odor, carisoprodol is present as a racemic mixture [23].

II. METHODS OF ANALYSIS

The analysis of carisoprodol should include the simultaneous identification and quantification of meprobamate.

A. Specimen Requirements

1. Whole Blood or Plasma (Serum)

For issues concerning human performance and behavior, whole blood or plasma are the recommended specimens of choice. Quantitation of both carisoprodol and meprobamate are required in order for the investigator to have some ability to correlate concentration with effect. Plasma to whole blood concentration ratios have not been established for either carisoprodol or meprobamate; therefore, when consulting the literature database for interpretation of carisoprodol or meprobamate concentrations, choose data that compare to the specimen type that was analyzed. The stability of carisoprodol and meprobamate has not been investigated. General recommendations for the collection and preservation of whole blood or plasma specimens should be followed. Specimens should be collected in fluoridated tubes and stored refrigerated or frozen. Caution should be taken when freezing specimens in glass due to the possibility of glass tubes cracking. Concentrations of carisoprodol and meprobamate are typically in the mg/L range in whole blood or plasma, hence sensitivity is not usually an analytical problem. As a result, specimen extraction volumes of 1 mL or less are usually adequate.

2. Urine

For the determination of use in the prior 72 hours, or patient compliance, urine is typically the specimen of choice. Reliable interpretation of effect on human performance cannot be determined from a urine concentration alone.

3. Tissues

In those cases in which a determination of the cause and manner of death is required, liver, kidney, and other solid tissues can easily be examined following homogenization.

B. Extraction Techniques

The extraction of carisoprodol and meprobamate from liquid and solid specimens is relatively simple and does not require extensive or complex procedures. The extraction is usually performed using acid/neutral conditions with both carisoprodol and meprobamate co-extracting in the same fraction. These extraction conditions will also extract other acid/neutral drugs such as barbiturates, phenytoin, and carbamazepine. An evaluation of potential interferences of these and other co-extracted compounds would have to be performed.

Depending on the extraction conditions, pH, and the amount of carisoprodol and/or meprobamate present in the specimen, both drugs can also be extracted in the basic fraction along with other basic drugs. This would not be an ideal situation for a quantitative analysis but would be acceptable for qualitative identification. If a basic extract is chosen to screen specimens for carisoprodol and meprobamate, ensure that the conditions of the extract are such that pharmacologically active concentrations of both carisoprodol and meprobamate can be reliably detected (*see* further discussion in Section II-C-5).

C. Instrumental Methods of Analysis

1. Immunoassay

A carisoprodol immunoassay kit has recently been developed by Immunalysis Corporation. Specificity and sensitivity in whole blood, serum, and urine are adequate with relatively small sample sizes required. The kit has a lower limit of detection of 25 ng/mL for carisoprodol and a 19% cross-reactivity for meprobamate at 2600 ng/mL [14].

2. Colorimetric Analysis

Colorimetric analysis, particularly Toxi-Lab® thin layer chromatography for carbamates, is a nonspecific screening analysis that works well on urine and stomach content specimens. If a carbamate is detected, additional identification and quantification analyses will need to be performed to identify the specific carbamate and to determine its concentration.

3. High Performance Liquid Chromatography

HPLC-UV is not a practical technique due to the lack of a suitable chromophore. Other HPLC combination techniques such as mass spectrometry have not been described in the literature.

4. Gas Chromatography

Gas chromatography is the separation technique most frequently used for the analysis of carisoprodol and meprobamate. Procedures using gas chromatography with both nitrogen-phosphorus detection (NPD) and flame ionization detection (FID) have been described [15,18].

5. Gas Chromatography/Mass Spectroscopy

Gas chromatography with mass spectrometric analysis (GC/MS) offers the most specific degree of analysis for carisoprodol and meprobamate. The determination of carisoprodol and meprobamate can be accomplished in a single analysis [2,18].

The meprobamate artifact (base peak 84) can be used as an indicator of the possible presence of carisoprodol and/or meprobamate, to help ensure that a GC/MS drug screen does not miss either drug due to co-elution of other compounds.

III. PHARMACOLOGY

A. Administration

Carisoprodol is indicated in patients with acute muscular pain. Carisoprodol is typically prescribed as 350 mg tablets (Soma®), with aspirin (Soma® Compound: 200 mg carisoprodol/325 mg aspirin) or with both aspirin and codeine (Soma® Compound with codeine: 200 mg carisoprodol/325 mg aspirin/16 mg codeine). During treatment, the recommended dose of carisoprodol is one tablet (350 mg) taken three times daily and at bedtime (1400 mg/day). When indicated in the treatment of anxiety, meprobamate may be given alone and is usually given in daily divided doses of up to 2400 mg [23].

B. Pharmacokinetics

1. Absorption

Carisoprodol is rapidly absorbed from the gastrointestinal tract and has a pKa of 4.2, facilitating absorption from the stomach and small intestine.

2. Distribution

Following absorption, carisoprodol is rapidly distributed to the CNS. Protein binding is approximately 60% and 25% for carisoprodol and meprobamate, respectively. The volume of distribution in humans has not been documented for carisoprodol. Meprobamate's volume of distribution is 0.7 L/kg.

3. Elimination

Carisoprodol is rapidly and predominantly dealkylated to meprobamate, an active metabolite, and to a lesser extent hydroxylated to hydroxycarisoprodol and hydroxymeprobamate followed by conjugation and excretion (**Figure 2**). These metabolic processes primarily occur in the liver. The half-life of carisoprodol is approximately 100 minutes [7,22] and that of meprobamate is many times longer, between 6 and 17 hours. The isoenzyme P450 2C19 is responsible for the conversion of carisoprodol to meprobamate and hence any interference or polymorphism of this enzyme may lead to increased half-life of carisoprodol and increased elimination time for meprobamate [7].

4. Plasma Concentrations

Plasma concentrations of carisoprodol in 18 subjects following a single oral dose of 350 mg reached an average peak concentration of 2.1 mg/L at 1 hour, was 1.1 mg/L at 3 hours, and had decreased to 0.24 mg/L by 6 hours[16]. Steady state concentrations have not been described. When meprobamate is prescribed for the treatment of anxiety, the meprobamate concentrations in blood are typically within the range of 5 to 20 mg/L [23]. As a result of the significantly longer half-life of meprobamate relative to carisoprodol, accumulation of meprobamate during chronic therapy may occur.

In another study, 700 mg of carisoprodol was given orally, leading to a peak serum concentration of 3.5 ± 0.94

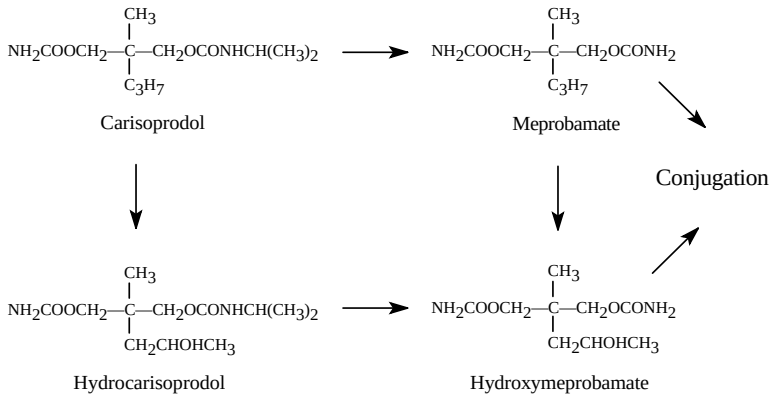

Figure 2. Metabolism of carisoprodol and meprobamate. (Redrawn from Baselt RC, Cravey RH: *Disposition of Toxic Drugs and Chemicals in Man*, 4th ed; Chemical Toxicology Institute: Foster City, CA; 1995.)

mg/L at 45 minutes and a peak meprobamate concentration of 4.01 ± 0.59 mg/L after 220 minutes [22]. In another group of subjects also administered 700 mg orally, the peak plasma concentration of carisoprodol was 3.1 ± 1.0 mg/L at 96 minutes and the peak meprobamate concentration of 4.8 ± 0.44 mg/L occurred at 336 minutes [7].

C. Pharmacodynamics

Although not well understood, the mechanism of action of carisoprodol in humans may be related to its sedative properties, either directly or indirectly, via the effects of meprobamate. Carisoprodol does not appear to directly relax tense skeletal muscles in man [23]. In animals, carisoprodol produces muscle relaxation by blocking interneuronal activity in the descending reticular formation and spinal cord; however, it is unknown if this mechanism of action is also present in humans [23]. There is some evidence linking carisoprodol and meprobamate activity to GABA receptors. Logan et al. suggested that meprobamate has barbiturate-like activity at $GABA_A$ receptors [18]. Additional support for interactions at the $GABA_A$ receptors was shown by Roberge et al., who found evidence that carisoprodol may be a $GABA_A$ receptor indirect agonist with central nervous system chloride ion channel conductance effects similar to the benzodiazepines [26]. This was demonstrated by using the benzodiazepine antagonist flumazenil in a case of carisoprodol intoxication. A $GABA_A$ interaction would explain some of the documented sedative effects that are similar to the benzodiazepine family of drugs. However, it is unclear whether the $GABA_A$ receptor-like effects are from carisoprodol itself or its metabolite meprobamate.

D. Dependence/Tolerance

Meprobamate is known to produce both physical and psychological dependence. Abuse begins after chronic treatment with carisoprodol. Tolerance occurs, requiring higher doses to achieve the desired effects and avoid the withdrawal syndrome. The withdrawal syndrome consists of anxiety, tremors, insomnia, and occasionally seizures and hallucinations. There have been many reports of the development of abuse and dependence involving carisoprodol and meprobamate [8,17,21,23,24,28]. Reeves and Carter [25] surveyed 20 substance abusers who had used prescription carisoprodol for 3 months or longer. Thirteen of the subjects admitted using carisoprodol in some manner other than that prescribed by their physician and 3 admitted using carisoprodol to modify the effect of another drug. Subjects have admitted using carisoprodol to augment the effect of ethanol or alprazolam and to "take the edge off the jittery feeling" from cocaine use. Carisoprodol has also been shown to produce cross-tolerance to barbiturates [27].

E. Relationship Between Blood Concentration and Effect

Following ingestion, the pharmacological effects of carisoprodol begin within 30 minutes and last for up to 4 to 6 hours [23]. It has been documented that there is a general direct relationship between blood concentration of carisoprodol and meprobamate and CNS depressant effects. Maddock and Bloomer showed that plasma meprobamate concentrations exceeding 100 mg/L were associated with deep coma, between 60 mg/L and 120 mg/L

were associated with light coma, and below 50 mg/L patients were generally conscious [19]. The Physicians' Desk Reference provides the following information on blood concentration and effect: a concentration of 30 to 100 mg/L is characterized by mild to moderate impairment such as stupor or light coma, a concentration of 100 to 200 mg/L produces effects consistent with a deeper coma, and concentrations exceeding 200 mg/L result in fatalities more often than survivals [23]. Bailey and Shaw also showed a statistically significant relationship between blood concentrations of meprobamate and the consciousness of patients [3]. In 104 motor vehicle drivers impaired by multiple drugs including carisoprodol and meprobamate, only 21 drivers had carisoprodol or meprobamate as the only drug(s) detected. In these 21 drivers Logan et al. observed that symptoms of impairment began at blood concentrations as low as 1 mg/L of meprobamate. The most severe driving impairment and the most overt symptoms of intoxication occurred in 16 out of these 21 drivers whose combined carisoprodol and meprobamate blood concentrations were greater than 10 mg/L [18]. Although these studies suggest a predictable relationship between blood concentration and effect, individual variation is significant and like other drugs, variations in concentration/response relationships will occur based on the subject's physiology and experience with the specific drug(s). These variations may be related to tolerance, cross-tolerance, level of fatigue, age, health status, and the presence of other drugs.

IV. CLINICAL TOXICOLOGY

The side effects of carisoprodol and meprobamate are consistent with those of other compounds with sedative hypnotic and CNS depressant properties such as sedative antihistamines, alcohol, and the benzodiazepine family of drugs. Side effects associated with carisoprodol therapy include, but are not limited to, agitation, depression, dizziness, drowsiness, facial flushing, headache, sleep disturbance, loss of coordination, and tachycardia.

As concentrations increase, nystagmus on lateral gaze becomes more evident and individuals may become obtunded and comatosed. A 19-year-old female survived the ingestion of 14.7 grams of carisoprodol. She experienced convulsions for 17 hours and loss of consciousness for 33 hours. She was tachycardic throughout [6]. In the 21 carisoprodol/meprobamate only cases described by Logan et al., drivers routinely exhibited clinical signs of impairment when their combined carisoprodol and meprobamate concentrations exceeded 10 mg/L [18].

Carisoprodol has been implicated in death, both directly and indirectly. Following the acute ingestion of 3.5 grams of carisoprodol, a 4-year-old boy became stuporous and semi-comatosed. He died of cardiac arrest 36 hours after admission, following episodes of vomiting and developing bilateral diffuse infiltrates. Prior to death his blood count, urinalysis, serum glucose, electrolytes, and blood gases were within normal limits. His serum concentration of carisoprodol and meprobamate was 36.4 mg/L and 15 mg/L, respectively, 4.5 hours after ingestion [1]. In another case of carisoprodol ingestion leading to death, a 39-year-old female ingested up to 30 carisoprodol tablets and was found dead in bed. Postmortem heart blood concentrations and femoral blood concentrations of carisoprodol were approximately 40 mg/L. Meprobamate concentrations were 40.1 mg/L and 51.9 mg/L in heart and femoral blood, respectively, and in the urine concentrations of carisoprodol and meprobamate were 12.6 mg/L and 61 mg/L, respectively [2]. A study of 78 medical examiner cases involving the ingestion of carisoprodol and meprobamate revealed that the subject was usually found dead, was often a white male or a black female with an average age of 40 ± 6 years, involved multiple drug use with 6 ± 2 additional drugs present, and the average carisoprodol/meprobamate concentrations were 17.3 and 19.8 mg/L, respectively. The most common additional drugs listed from the most frequently encountered to the least encountered were codeine, *N*-desmethyldiazepam, diazepam, morphine, propoxyphene, methadone, acetaminophen, norpropoxyphene, and ethanol [13].

V. EFFECT OF CARISOPRODOL ON DRIVING

There are few peer-reviewed articles describing the effects of carisoprodol and meprobamate on driving. Marinetti-Sheff and Ludwig documented 117 carisoprodol-related DUI cases in the Lower Peninsula of Michigan including Detroit and surrounding suburbs [20]. The authors observed that in recent years the incidence of accidents and DUI cases involving carisoprodol has increased. Interestingly, they observed that the majority of the incidents occurred between the hours of 9 am – 9 pm and were fairly evenly distributed across the days of the week, with Saturday showing the lowest number of incidents. Codeine and diazepam were the most frequently detected drugs in combination with carisoprodol. Unfortunately, clinical observations by arresting officers were not well documented. Observations in a Norwegian study were consistent with those of Marinetti-Sheff and Ludwig. Bramness et al. observed that in suspected drugged drivers, the frequency of blood samples testing positive for carisoprodol and meprobamate in Norway has increased in recent years [5].

In a comprehensive study by Logan et al., 104 drivers involved in accidents or arrested for impaired driving in Washington State between January 1996 and July 1998 tested positive for meprobamate and/or carisoprodol [18]. Meprobamate was detected in the blood specimens of all 104 drivers. The median concentrations for carisoprodol and meprobamate were 4.30 mg/L (range 0–25.1 mg/L) and 11.65 mg/L (range 1–77.6 mg/L), respectively. In the majority of cases, alcohol and/or other drugs including benzodiazepines, narcotic analgesics, and barbiturates were present, making the correlation between driving behavior and the presence of carisoprodol and/or meprobamate difficult. Of the 104 cases described by Logan et al., 21 contained only carisoprodol and /or meprobamate. In this population of 21 drivers, the median carisoprodol and meprobamate concentrations were 4.85 mg/L (range 0.00–15.20 mg/L) and 11.00 mg/L (range 1.00–35.60 mg/L), respectively. In the majority of the 21 cases, the concentrations of carisoprodol were elevated above those expected following a single therapeutic dose ingested for muscular pain, approximately 3.5 mg/L.

Twelve of the 21 meprobamate/carisoprodol-only drivers were involved in accidents. In all cases, the driver was at fault for the accident. Observed driver behavior included extreme lane travel and weaving, striking other vehicles and fixed objects, slow speed, hit-and-run accidents where the subject appeared unaware they had hit another vehicle, and in one case the individual was driving the wrong way on a freeway. Symptoms included depressed reflexes, slowed movements, confusion, impairment in balance and coordination, disorientation to place and time, slurred or thick speech, and dazed and groggy appearance. These drivers invariably demonstrated horizontal gaze nystagmus. Some were unable to understand instructions or communicate, and it is of interest that no subject was combative.

In another study, Finkle described the driving behavior of 11 drivers with meprobamate in their blood. In these cases, the typical meprobamate concentration was 30 mg/L and above. The driving behavior was described as erratic and their clinical symptoms of intoxication were similar to those described by Logan et al. [10]. Most drivers had alcohol or some other sedative hypnotic drug present, making the interpretation of the contribution of meprobamate difficult.

The symptoms associated with carisoprodol/meprobamate use in these drivers are consistent with CNS depression, and hence the effects on complicated tasks such as driving resemble those of alcohol and other CNS depressants. Tasks requiring divided attention become difficult to complete, tracking becomes poor and lane weaving common, coordination is negatively affected, reaction time is increased, judgment is compromised — all of which has a detrimental effect on decision-making together with other skills essential to driving. Logan et al. went on to conclude that when the combined carisoprodol and meprobamate concentrations were above 10 mg/L, the most pronounced evidence of psychomotor impairment was present [18].

A drug recognition expert (DRE) evaluation of a driver under the influence of carisoprodol/meprobamate would include signs and symptoms of depressant-type intoxication such as ataxia, slurred speech, stupor, and drowsiness. Mydriasis, nystagmus, clouded thinking, and memory impairment have also been observed [12]. However, since carisoprodol and/or meprobamate are so commonly encountered in combination with other drugs, the DRE evaluation could vary significantly from that stated above depending on the type of drug co-ingested and its influence in the combined effects of all the drugs present.

CONCLUSION

Carisoprodol and meprobamate, widely prescribed and frequently abused, produce a multitude of CNS depressant effects detrimental to human performance and impairing to complex tasks such as driving. The symptoms are similar to other CNS depressants such as alcohol and benzodiazepines. Although frequently co-ingested with other CNS depressants, severe impairment has been consistently demonstrated at a combined blood concentration of carisoprodol and meprobamate greater than 10 mg/L. Unfortunately, carisoprodol is not yet routinely analyzed for in the human performance toxicology laboratory; therefore, the magnitude of the problem can only be approximated.

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Dr. Robertson has authored several peer-reviewed papers and book chapters in recognized journals and has made numerous presentations to professional and civic groups regarding various aspects of forensic toxicology such as: drugs and driving, postmortem stability, redistribution and bioconversion of drugs, rave drugs and drug-facilitated sexual assault. He has also lectured at numerous universities on topics such as pharmacology, general toxicology, and forensic toxicology. Dr. Robertson currently holds membership in the International Society of Forensic Toxicologists (TIAFT) and the Society of Forensic Toxicologists (SOFT), and is a provisional member of

the American Academy of Forensic Sciences (AAFS). He is also an affiliate of the California Association of Toxicologists (CAT) and a member of the SOFT Drugs and Driving Committee and Drug Facilitated Sexual Assault Committee.

Laureen J. Marinetti received her B.S. and M.S. degrees in forensic science from Michigan State University (East Lansing, MI) in 1983 and 1991, respectively. Laureen is currently a Ph.D. candidate beginning her fourth year of her doctoral program at Wayne State University (WSU: Detroit, MI) in the Department of Pharmaceutical Sciences, College of Pharmacy and Allied Health Professions. Under the direction of Bradford Hepler, Ph.D., and Dan Isenschmid, Ph.D., toxicologists of the Wayne County Medical Examiner's Office (Detroit, MI), Laureen consults in postmortem forensic toxicology in the areas of method development, quality assurance, and drug abuse demographics, especially concerning the drugs carisoprodol and γ-hydroxybutyric acid (GHB).

In 1987, Laureen began her career with the Michigan State Police Crime Laboratory in the Toxicology Subunit working in the areas of human performance and postmortem drug testing as well as method development and court testimony. She then took a deferred retirement in September 1998 to pursue her doctorate at WSU. Laureen's research interests include all aspects of GHB and its analogs, γ-butyrolactone (GBL) and 1,4-butanediol (1,4BD). She is currently gathering data on the behavioral pharmacology of GHB alone and in combination with ethanol and other drugs using rats. She has written a chapter on the pharmacology, toxicology, and detection of GHB, GBL, and 1,4BD for the book *Benzodiazepines and GHB: Detection and Pharmacology*, edited by S. J. Salamone, (Humana Press, September 2001) and co-authored a GHB monograph with Fiona Couper, Ph.D., which appeared in volume 14 (1/2) (January 2002) of *Forensic Science Review*. She has also made several presentations on various toxicology topics, the majority of which are on GHB and its analogs and rave drugs such as MDMA, PMMA, and ketamine.

Laureen has been qualified as an expert witness in analytical and interpretive forensic toxicology more than 150 times at both district and circuit court in over 40 counties in Michigan and also in the state of Florida. Laureen is a member of the Society of Forensic Toxicologists and co-chairman of the Drug Facilitated Sexual Assault Committee. She is also a fellow of the Toxicology Section of the American Academy of Forensic Sciences and a member and the secretary of the Midwest Association for Toxicology and Therapeutic Drug Monitoring as well as a member of The International Association of Forensic Toxicologists.

In December 1998, Laureen was awarded a Forensic Toxicology Fellowship from the Wayne County Medical Examiner's Office. In June 1999 and June 2000, she received Substance Abuse Educator Awards from WSU for her presentations to various organizations on GHB and its analogs. Most recently, in October 2000 and October 2002, Laureen received the Education Research Award from the Society of Forensic Toxicologists for her research on GHB.