Society of Forensic Toxicologists, Inc.

Volume 32, Issue 2
June 2008

TOXTALK

INSIDE THIS ISSUE:

REGISTER *NOW* FOR THE 2008 ANNUAL MEETING

The user friendly SOFT 2008 Meeting Website can be found at www.**soft2008.org** and contains the most up to date meeting information such as the current Preliminary Program and the detailed information on the twelve focus oriented workshops being offered at the 2008 meeting. In addition, the meeting website contains local "Arizona Activities" and information on the "all suites" resort, the Arizona Grand, home to the SOFT 2008 meeting. The website is updated routinely by meeting website developer, Douglas Kramer (a new SOFT member), and contains many links to related sites for this meeting. We highly recommend that you log in frequently and see "What's New".

Use this site to register and select desired workshops before they reach maximum capacity. Attending SOFT annual meetings is the best way to learn the latest scientific developments in forensic toxicology, plus enjoy the hospitality and culture of each distinctive host city.

S.O.F.T. STUDENT ENRICHMENT PROGRAM

Amanda Gallegos of the Phoenix Police Department Laboratory Services Bureau has joined Jeri Ropero-Miller of RTI International to coordinate the activities of the SSEP in Phoenix. While the SSEP targets college students in the regional area of the event, all interested college students can apply with equal consideration. Up to 100 students can attend SSEP. Students must provide their own travel to the Arizona Grand Resort and private sponsors and proceeds from prior SOFT Silent Auctions fund the entire day-long SSEP program. We are inviting these students to "Come Experience a Day-in-the-Life of a Forensic Toxicologist".

Amanda Gallegos

Jeri Ropero-Miller

DETAILS AT-A-GLANCE:

When: Monday, October 27, 2008

Who can Apply: College Students (undergraduate and graduate)

Application Period: May 1, 2008 through September 30, 2008

Applications Available from: http://www.soft2008.org/SSEP.html or jerimiller@rti.org

Acceptance Notification: October 10, 2008

SSEP Coordinators:

Jeri Ropero-Miller, Ph.D.
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919-485-5685; jerimiller@rti.org

Amanda Gallegos
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PRESIDENT'S MESSAGE
BY CHRISTINE MOORE, PH.D., DABCC

Greetings from Southern California….It's over 90°F today – life is good!

As time seems to go more quickly, the meeting planning for SOFT 2008 is well underway and as I write, the registration page has just "gone live". Please make sure you register early since the workshops being offered are of a very high quality and space will be limited – don't miss out.

The Plenary Session focuses on phenethylamines and the speaker is the world-famous Dr. Alexander Shulgin, who himself discovered many of these compounds. Dr. Shulgin has been active in the field of drug synthesis, co-authoring the books PiHKAL (Phenethylamines I Have Known and Loved: A Chemical Love Story) and TiHKAL (Tryptamines I Have Known and Loved: The Continuation), which discusses various aspects of psychoactive drugs….should be interesting!

The Editor for the Special Issue of the Journal of Analytical Toxicology 2008, Dan Anderson, reports that he received 33 submissions, all of which are currently under review/revision. That statistic alone shows the importance of our annual meeting, and leads into another SOFT Board initiative.

Each year the Special Issue is full of excellent research and the SOFT Board wanted to create an Award to reflect the best published article. The Award, to be entitled "***Experimental Design and Impact on Toxicology***" (EDIT), will recognize the first author of the paper that year, which, in the opinion of the judges, shows excellent scientific experimental design and has a wide impact on our field. The award will begin in 2009, and to ensure the Special Editor is not overwhelmed, three external judges will be asked to recommend the winner from accepted manuscripts prior to publication in the Special Issue. We are working with Tinsley Preston at JAT and Bruce Goldberger, Editor JAT to determine the precise logistics for 2009.

At the moment though, I am delighted to announce that the judges for the premier EDIT Award will be Drs. Edward Cone, Amanda Jenkins and Hans Maurer. I am sure you will agree that between them, these toxicologists have a vast knowledge of experimental design and extensive publication history. The SOFT Board is excited about the creation of this new initiative to recognize our scientists, and we encourage you to submit your research to our Special Issue.

Finally, just a reminder that we will be voting on increasing dues by $10 per year at the 2008 Business Meeting.

I thank you for your loyal membership and wish you all a very relaxing summer. Hope to see you in October.

S.O.F.T. 2008 ANNUAL MEETING
Phoenix, Arizona, October 27-31, 2008

Hosts: Vickie Watts / Norman Wade
Site: Arizona Grand Hotel (formerly the Pointe South Mountain)

PRELIMINARY PROGRAM

Sunday, October 26, 2008
- Registration Opens (9:00am-6:00pm)
- NLCP Training (2:00pm-6:00 pm)
- Dinner on your own

Monday, October 27, 2008
- Continental Breakfast (7:00am-8:30am)
- Registration (7:00am-6:00pm)
- SOFT Workshops (8:00am-5:00pm)
- SOFT Student Enrichment Program (8:00am-5:00pm)
- ABFT Exam Committee
- SOFT-AAFS Drugs and Driving Committee
- Tier-One Exhibitors Hospitality (6:30pm-8:30pm)

Tuesday, October 28, 2008
- Continental Breakfast (7:00am-8:30am)
- Registration (7:00am-6:00pm)
- SOFT Workshops (8:00am-5:00pm)
- SOFT Board Meeting (7:00am-noon)
- ABFT Exam
- ABFT Accreditation Committee
- ABFT Board Meeting
- Exhibits Setup (noon-5:00pm)
- Exhibits Open (6:30pm-8:00pm)
- Welcoming Reception (6:30pm-8:00pm)
- Elmer Gordon Forum (8:00pm-10:00pm)
- Nite Owl Reception (10:30pm-12:30am)

Wednesday, October 29, 2008
- Continental Breakfast (7:30am-9:00am)
- Registration (7:30am-5:00pm)
- AAFS Steering Committee (9:00am-10:00am)
- Exhibits open (7:30am-3:30pm)
- Opening Ceremonies Plenary Session
- Scientific Sessions (8:30am-noon)
- Lunch with Exhibitors (noon-1:15pm)
- DFSA Committee Meeting (noon-1:15pm)
- Scientific Sessions (1:15pm-5:00pm)
- Exhibitor's Happy Hour (5:00 pm-6:30pm)
- "Sunset at the Oasis" Poolside Reception (7:00 pm-10:00 pm)

Thursday, October 30, 2008
- SOFT Fun Run/Walk (6:30am-8:00am)
- Continental Breakfast (7:30am-9:00am)
- Registration (7:30am-5:00pm)
- Exhibits open (7:30am-1:30pm)
- Exhibitor Feedback Mtg (8:00am-9:30pm)
- Scientific Session (8:30am-noon)
- Lunch with Exhibitors (noon-1:15pm)
- Exhibits Breakdown (1:30pm-3:30pm)
- Scientific Session (1:15pm-2:30pm)
- SOFT Business Meeting (3:00pm-5:00pm)
- ABFT Certificate Reception Wine & Cheese (5:00pm-6:00pm)
- Presidents Banquet and Masquerade Ball (6:30pm-11:30pm)

Friday, October 31, 2008
- Continental Breakfast (7:30am-9:00am)
- Scientific Session (9:00am-noon)
- NSC Executive Board (1:00pm-3:30pm)

DRUGS IN THE NEWS

Submitted by Section Editor, Dwain Fuller, D-FTCB, TC-NRCC

Please send interesting "Drugs In The News" to Section Editor, Dwain Fuller at dwain.fuller@med.va.gov

RICIN

On February 14th, 2008, what seemed like a routine call to a Las Vegas 911 operator from a man suffering from respiratory problems became an incident of far-ranging significance. At last report, the man, now identified as Roger Von Bergendorff, a down-and-out graphic artist, was in critical condition in Spring Valley Hospital, and by at least one report, comatose.

The cause of his condition was not discovered until two weeks later, however. At around 2:30 p.m. on February 28th, Thomas Tholen, a cousin of Von Bergendorff, was cleaning out the Extended Stay America room that Von Bergendorff had rented, and from which he was now being evicted, when he came across a small vial. Mr. Tholen took the vial down to the motel's front desk which set in motion a massive police, hazmat and Homeland Security response. The content of the vial was determined to be ricin.

Ricin, as you may be familiar, is a toxin derived from castor beans. Its mechanism in the body is to inhibit protein synthesis. As a result of this inhibition, the cells are deprived of essential proteins and die. Thus ricin is a systemic poison affecting multiple systems of the entire body.

Castor Beans

Castor beans come from the castor bean plant (Ricinus communis) which has been cultivated for centuries, primarily for the oil produced by its seeds, or beans. The Egyptians burned castor oil in their lamps more than 4000 years ago. Castor oil, is perhaps best known to the lay person as a popular stimulant laxative of the early 20th century. As a child of the 60's, I have never experienced the joys of castor oil, but merely broaching the subject in my workplace initiated a torrent of stories of the horrors of taking castor oil. The aversion to the medicinal use of castor oil seems to lie in its taste rather than its effects.

There are many non-medical uses of castor oil as well. In the United States, castor oil has been used in aircraft lubricants, hydraulic fluids, synthesis of soaps, linoleum, printer's ink, nylon, varnishes, and in the manufacture of explosives. The seeds, stems from the pressing of castor oil contains about 5% ricin by weight. While it has been estimated that as few as four to eight castor beans would be toxic or even fatal to an adult human, the fact is that unless the seed coat is broken, such as by chewing, castor beans will likely pass through the body with no ill effects. Perhaps in testimony of its relatively benign nature, when not purposely abused some varieties of the castor bean plant are often grown as ornamentals. They are best adapted to the soils and climate of southeastern Kansas and Missouri, southern Illinois and Indiana, as well as Tennessee, Kentucky, and parts of Oklahoma and Texas.

While castor beans do not appear to pose too much of a threat, ricin itself, however, is a potent toxin. The estimated toxic dose for an adult is less than 1 milligram if inhaled or injected. There is no known antidote for ricin poisoning. Instead only symptomatic and supportive treatment is available and long-term organ damage is likely in survivors. The symptoms of ricin poisoning are dependent on the route of administration. If inhaled, the symptoms are: respiratory distress, fever, cough, nausea, and tightness in the chest. These are followed by pulmonary edema, hypoxia, cardiac arrhythmia, and death. If ingested, the symptoms are: vomiting, diarrhea, perhaps becoming bloody, followed by severe dehydration. Within several days the person's liver, spleen and kidneys may fail, leading to death.

Ricin was evaluated by the United States for its potential as a military weapon during World War I. At that time it was being considered for use either as a toxic dust or

Castor Bean Plant

DRUGS IN THE NEWS (CONTINUED)

as a coating for bullets or shrapnel. Due to the technological limitations of the time the dust-cloud concept could not be adequately developed and the bullet/shrapnel coating concept would be a violation of the Hague Convention of 1899. During World War II the United States and Canada studied the potential for the use of ricin in cluster bombs, but concluded that due to the necessity to aerosolize it as a dust, it was no more economical than phosgene.

Perhaps the most interesting chapter in the history of ricin is the incident of Georgi Ivan Markov. Georgi Markov was a Bulgarian-born novelist and playwright. Markov enjoyed a privileged existence in Bulgarian society even though his father was considered a "class enemy" of the communist party. This all ended, however, in 1969 when he defected to Italy after learning that his latest play had angered the government and put him at risk. After his defection he was accused and convicted by Bulgarian authorities, in absentia, of being a traitor. By 1971 Markov had immigrated to Britain where he became a broadcast journalist and commentator for the BBC. In, June of 1975, he began contributing programs to the CIA-funded, Radio Free Europe, where his weekly shows were sharply critical of Bulgarian bureaucrats and communist party officials, especially, party leader, Todor Zhivkov.

In early 1978, Markov began receiving death threats. In the last call in August of 1978, Markov was told that he would die of natural causes, killed by a poison the West could not detect nor treat. Two weeks later on September 7th, Markov parked his car in a parking lot on the south side of Waterloo Bridge in London. This was his usual parking spot where he would catch the bus to BBC headquarters. While waiting at the bus stop, Markov felt a sharp prick in the back of his right thigh. When he turned around he saw a gentleman bending over to pick up a dropped umbrella. The man said "I'm sorry" in a foreign accent, promptly hailed a cab, and left.

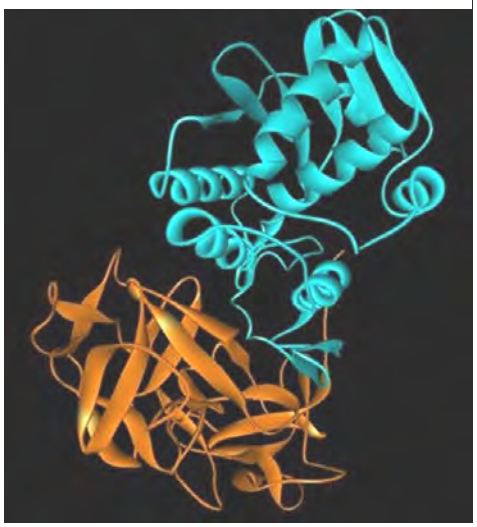
Markov, although in pain, continued on to work where he told his colleagues what had happened. On the back of his right thigh was a swollen pimple-like wound. That evening he developed a high fever and by the next day was having trouble talking. He was admitted to the hospital where he was initially treated for septicemia, but over the next few days Markov began to have bloody vomit and kidney failure. On September 11, 1978, his heart failed. Georgi Markov was dead.

At autopsy it was determined that Markov's lungs were full of fluid, his white blood cell count was extremely high, his liver was damaged, and his lymph nodes, intestines and heart were riddled with small hemorrhages. A large portion of tissue was removed from around the wound area on the back of Markov's right thigh. In this tissue examiners discovered a small pellet 1.52 mm in diameter composed of platinum and iridium. The pellet was eventually determined to be a watch bearing, but in this pellet were two 0.34 mm holes bored at right angles to each other, forming an X shaped well inside. Due to the extreme hardness of this material it was surmised that such precision machining could only have been done by a sophisticated laser process known as "spark erosion."

Although no poison was detected in the pellet, nor in Markov's body, it was surmised due to strong circumstantial evidence, based on Markov's symptoms, postmortem pathology, and intelligence information regarding the Soviet Union's involvement in research of the use of ricin as a weapon, that Markov did in fact die of ricin poisoning administered via the pellet propelled from a gas powered device disguised as an umbrella. Although the Soviet Union has denied any connection to the incident, KGB defectors, Oleg Kalugin and Oleg Gordievsky, have since confirmed the Soviet Union's involvement.

Ricin consists of two distinct protein chains with a molecular weight around 30 kDa each. Ricin A is an N-glycoside hydrolase that targets and depurinates an adenine base in the 28S rRNA molecule of the ribosome, resulting in an inhibition of protein synthesis. Ricin B is a lectin that binds galactosyl residues and is important in assisting ricin A's entry into a cell by binding the cell surface component. It is the presence of both of these chains that renders ricin so toxic. Many plants such as barley have the ricin A chain but not the ricin B chain. Since people do not get sick from eating even large quantities of such products, the ricin A chain is relatively harmless without the ricin B chain present.

Pellet recovered from Georgi Markov



Ricin A - blue, Ricin B - red

DRUGS IN THE NEWS (CONTINUED)

Ricins may have therapeutic use in the battle against cancer. It is theorized that Ricin could be linked to a monoclonal antibody to target malignant cells recognized by the antibody. It has also been postulated that one may be able to use the ricin B chain as a vehicle to effectively deliver antigens into cells thus greatly increasing their immunogenicity.

The fact that ricin is a complex protein of extremely high molecular weight presents an unusual problem for the forensic toxicologist. On one hand, these characteristics of ricin make it relatively well-suited for detection by a properly designed immunoassay, and many of these have been described in the literature. However due to its high molecular weight and complex structure, confirmation by electron-impact GC/MS or LC/MS, the standard tools of the forensic toxicologist, is not possible.

Although not fully explored in human subjects, the literature suggests that a biomarker of ricin exposure, ricinine, may be easily detected by standard solvent extraction and GC/MS or LC/MS technology. Ricinine is not a metabolite of ricin, but rather a biomarker that derives from the same source as ricin, the castor bean plant. Thus confirming the presence of ricinine in biological fluids that have screened positive for ricin by immunoassay greatly enhances evidence of ricin exposure or poisoning. Ricinine ($C_8H_8N_2O_2$) has a molecular weight of 164 daltons and thus is quite amenable to analysis by routine GC/MS or LC/MS technology.

Update:

Near the completion of this article it was reported that Roger Von Bergendorff had recovered and upon his release from the hospital on April 16th, 2008 was arrested and charged with possession of a deadly toxin. Mr. Von Bergendorff allegedly admitted making ricin in what he described as an "exotic idea" to harm his enemies. Found, among other clandestine items, at a self-storage rented by Von Bergendorff, was a safe containing a ricin contaminated mortar and a drawing made by Von Bergendorff of an injection device disguised as a pen.

References:

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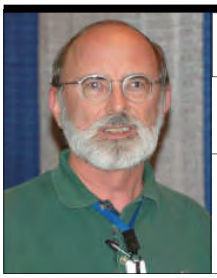
8. Johnson RC, Lemire SW, Woolfitt AR, Ospina M, Perry KP, Olson CT, Barr JR. Quantification of Ricinine in Rat and Human Urine: A Biomarker for Ricin Exposure. *J Anal Toxicol 2005;29:149-155*
9. Erin Alberty, Nevada man arrested after ricin found in W. Jordan storage unit. *The Salt Lake Tribune, April 17, 2008*

SUNSHINE / RIEDERS SILENT AUCTION

SOFT sponsored a memorial event during the 2006 Austin meeting to honor the recent passing of two illustrious leaders in forensic toxicology, Dr. Irving Sunshine, and Dr. Frederick Rieders. The event was named the "Sunshine / Rieders Silent Auction" and has since become an annual event that meeting attendees look forward to. A wide array of items are donated by exhibitors and individuals, then displayed with bid sheets, tracking names and bids. At a designated time, bidding closes and winners can pay and pick up their treasures. Not only is the auction a fun tradition, but complete proceeds benefit students interested in forensic toxicology through the SOFT Student Enrichment Program. Since Dr. Sunshine and Dr. Rieders focused their energy on academic encouragement in this field, it is thought to be an appropriate way to acknowledge their lifetime contributions and continue their legacy of promoting education in forensic toxicology.

SOFT Member, Laurie Tobler has generously volunteered to coordinate the 2008 Sunshine / Reiders Silent Auction. Anyone wishing to donate items, big or small, should contact:

Laurie Tobler at 801-573-2409
laurie.tobler@tandemlabs.com



CASE NOTES

Submitted by Section Editor, Matthew Barnhill, Ph.D., DABFT

Please send interesting "Case Notes" to Section Editor, Matthew Barnhill, Ph.D. at mbarnhilljr@worldnet.att.net

CASE NOTES #1: METHAQUALONE IN A STORED DUID CASE

Jeffery Hackett and Michael Coyer, Northern Tier Research, Mayfield, PA

Introduction:

In 2006, a sample of blood (along with several others) was submitted to the laboratory at Northern Tier Research for routine toxicological testing by a local enforcement agency. This testing included screening for drugs that may have impaired the ability of a driver to control his / her motor vehicle. The sample, at that time was tested by immunoassay after solid phase extraction using Clean Screen ™ DAU columns. The sample was also screened for drugs of abuse by gas chromatography-mass spectrometry on a HP 5890/5973 MSD (opiates, amphetamines, cocaine, cannabinoids) with negative results using the same type of solid phase columns for extraction of the sample. Analysis of the blood sample for prescription drugs by solid phase extraction and gas chromatography- mass spectrometry revealed the presence of butalbarbital, diazepam/nordiazepam and propoxyphene/ norpropoxyphene: (3.1 mg/L, 0.5 mg/ L, 0.7 mg/L, 0.29 mg/L, norpropoxyphene (positive), respectively). Following review of the analytical findings a report was filed.

In March 2008, as part of a method development project evaluating new solid phase/ liquid chromatography columns for the use in forensic toxicology, the blood sample was retrieved from long term storage where it had been kept in a refrigerated condition. The sample of blood was analyzed along with several others in a blind trial to see how both types of columns (solid phase and liquid chromatography) would perform using real sample types. Prior to analysis of the samples, no information regarding the drugs was known. In analyzing this particular sample, a benzodiazepine screen was performed in which nordiazepam and oxazepam were confirmed by LC-MSMS. A basic drug screen was also performed on the LC-MSMS in which the propoxyphene/ norpropoxyphene were also confirmed. In addition to those drugs, methaqua-lone was also detected. The MRM values for methaqualone (251.2-> 132.1, 251.2-> 91.0, respectively) were set up within the program prior to analysis. Confirmation was achieved by comparison with an unextracted sample of methaqualone.

Experimental (LC-MSMS):

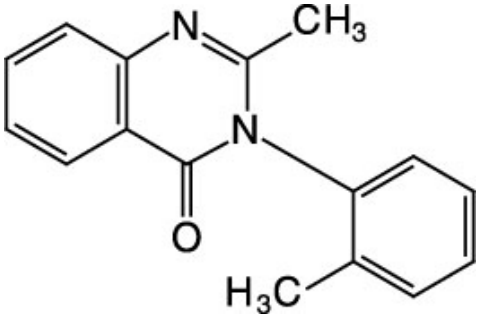
For screening purposes, 1 mL of sodium acetate buffer (1 M, pH 4.5) was spiked with propoxyphene-d11 and diazepam-d5, respectively as internal standards to which 1 mL of the sample blood was added. The mixture was diluted with a further 2 mL of the buffer, vortexed and centrifuged. The sample was applied to a SSCCXH solid phase column obtained from United Chemical Technologies, Inc. The column washed with distilled water and an acetonitrile/acetate buffer mix (5: 95 v/v), respectively then dried and eluted with 3 mL of a mixture of ethyl acetate/acetonitrile (78:20) containing 4% ammonium hydroxide by volume. The eluate was evaporated to dryness and the residue dissolved in methanol (100 µL) prior to analysis by LC-MSMS with a 5 µL injection volume. Separation of the analytes was performed using a Selectra™ Phenyl column (50 x 2.1 (3µm) using a gradient mobile phase program of acetonitrile / 0.1 % formic acid. Detection was carried out using an API2000 mass spectrometer in positive MRM mode.

For quantification of the methaqualone in the blood sample, the same extraction procedure was performed except for the replacement of diazepam-d5 by methaqualone- d7. Negative samples were spiked with methaqualone and methaqualone-d7.

Chromatogram of screen showing:

- Methaqualone/Norpropoxyphene/ Propoxyphene (Propoxyphene-d_{11}) (Upper trace)
- Methaqualone (MRM 251.2- >132.1) (Lower trace)

Structure of Methaqualone



Results and Discussion:

From the analysis of the sample, methaqualone was found to be < 0.001 mg/ L (1 ng/ mL). A search/ review of the stored gas chromatography-mass spectrometry computer file did not reveal the presence of the methaqualone in the earlier analysis,

CASE NOTES #1 (CONTINUED)

although the other reported drugs were present. A review of the other samples analyzed by solid phase extraction/ LC-MSMS before and after the present sample in the sequence list did not reveal the presence of methaqualone.

Methaqualone is a sedative and a hypnotic, first synthesized in 1951 and entered into the market place in 1956, but due to its misuse the drug was removed from use in the US in 1984[1]. Therapeutic levels of methaqualone are reported to be 0.4 to 5 mg/ L (plasma). [2] Toxic levels of methaqualone in post mortem blood have been reported to be in the range 5-42 mg/ L[2].

Several reports indicating the detection and quantification of methaqualone in driving cases have been published in the 1980's [3-4] but this is the first time that we have encountered this particular drug. The level of the methaqualone detected, although sub therapeutic, may have in combination with the other drugs had some impact upon the drivers ability to control the motor vehicle. Although some studies have been published regarding the stability of drugs stored in blood samples [5-6], it is not known by this particular laboratory exactly how methaqualone behaves when stored under refrigeration for a long period of time. It is thought that the diazepam originally present may have broken down to its metabolites during the time the blood sample was stored.

This use of LC-MSMS has shown that older samples may have contained drugs that were not detected previously.

References:

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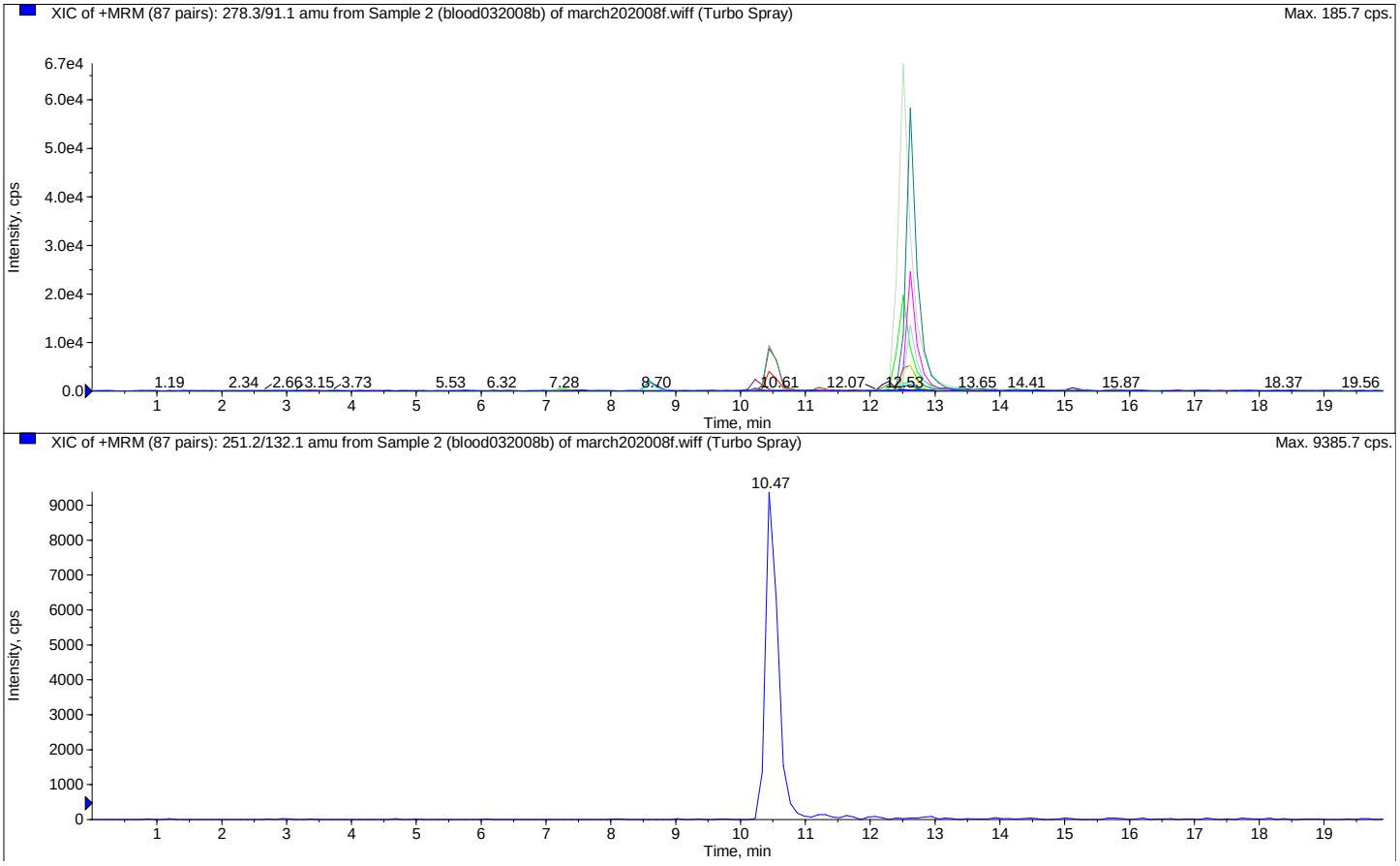


CASE NOTES #2: INHALANTS AND DRIVING IN SOUTH CAROLINA: A VOLATILE SITUATION

Tim Grambow and Laurie Shacker, Toxicology Department, SC Law Enforcement Division

Introduction:

The abuse of inhalants in order to achieve a "high" is not a new problem. Four major classes of abused inhalants have been identified. They are anesthetic gases (including nitrous oxide), industrial solvents (including hydrocarbons), aerosol propellants (including fluorocarbons) and organic nitrites (amyl or butyl).[1] Absorption of inhaled chemicals into the lungs leads to a quick distribution into the blood and subsequently the brain. Various mechanisms of action include oxygen displacement and interactions with ion channels. The user experiences intoxication and effects that are similar to those of alcohol including slurred speech, decreased motor skills, euphoria, and dizziness.[2] The quick onset of action combined with ease of obtaining these compounds makes inhalants popular alternatives to other drugs. As can be expected, using inhalants can impair one's ability to safely operate a motor vehicle.

Trends:

In 2007 the South Carolina Law Enforcement Division (SLED) received approximately 1,750 toxicology requests for DUI cases and 530 traffic fatalities. A volatile analysis was performed on submitted sample types which included blood

Figure 1: HS-GC/FID Chromatogram

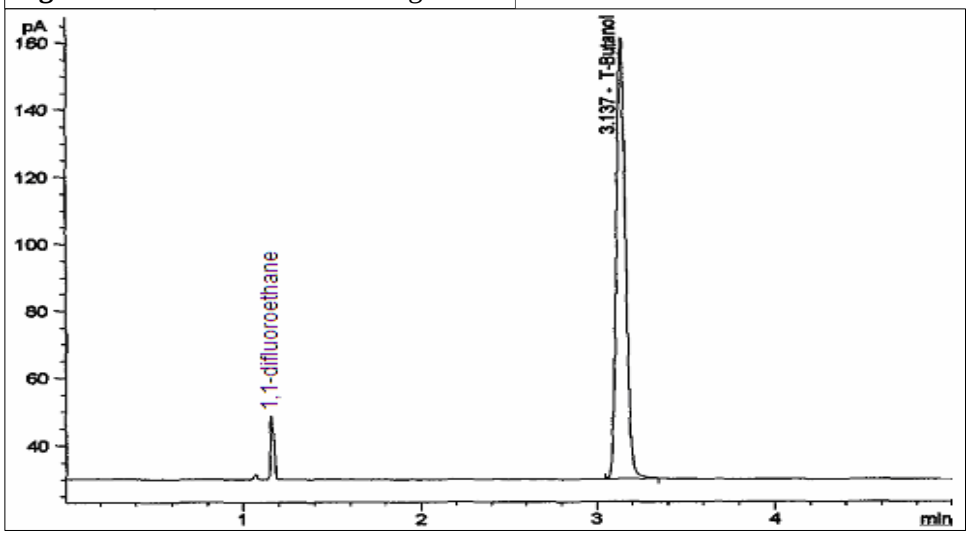


Figure 2: HS-GC/MS Chromatogram

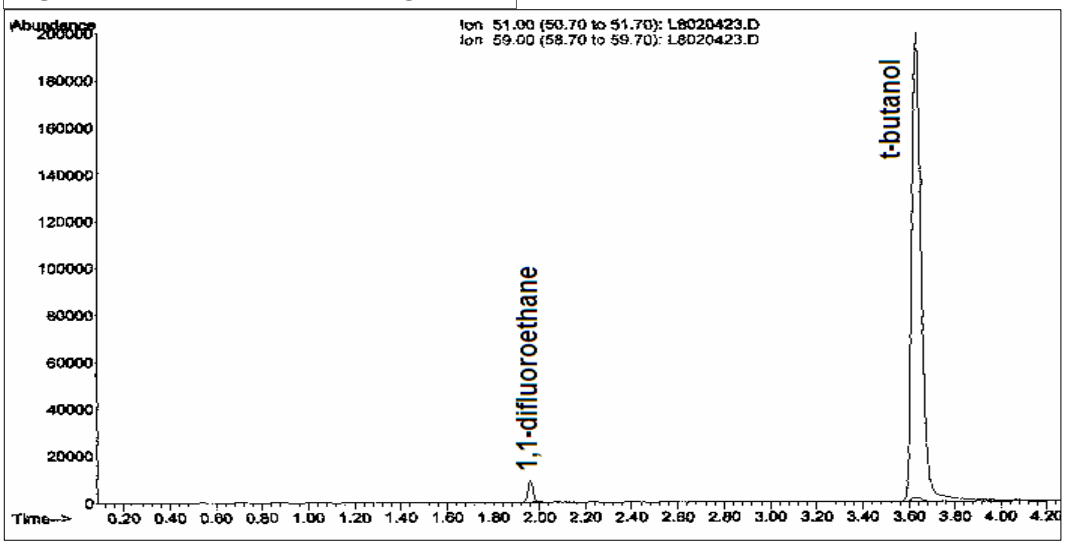
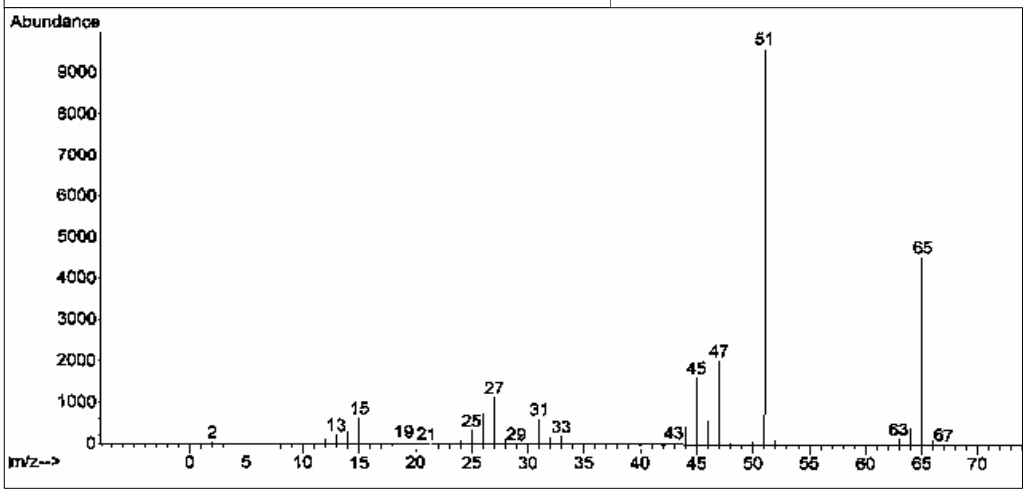


Figure 3: Mass Spectrum of 1,1-difluoroethane



CASE NOTES #2 CONTINUED:

and/or urine dependent on the individual case and South Carolina DUI law. No inhalants were reported in these cases until July. Two cases with inhalants were reported in July along with 1 case each in August, September and December. In all 5 of these cases the inhalant that was reported was 1,1-difluoroethane. This compound is most often found as an aerosol propellant, a component of canned air duster and is being phased out as a refrigerant (Freon-152a).

Six more instances of inhalants have been reported in DUI cases through April of 2008. In these cases 1,1-difluoroethane, isoflurane and toluene were detected. Isoflurane is a volatile anesthetic (Forane) while toluene is a petroleum based solvent. In 5 of the 8 cases involving 1,1-difluoroethane an indication of inhaling canned duster was given in the case history (several naming the brand suspected). In a single case, a canister was also submitted as evidence. No indication of inhalants was given for the cases involving isoflurane or toluene.

Detection:

All samples were initially analyzed using headspace gas chromatography with an FID detector. The HS-GC/FIDs used utilize either a Restek BAC-2 or a Carbowax-20M column depending on the individual instrument. A secondary analysis was run using HS-GC/MS with a Restek BAC-1 column. All results were qualitative only.

Discussion:

The effects of inhalants make their detection as important as other impairing drugs for DUI and traffic fatality cases. The detection of inhalants in 11 cases over the last 10 months compared to zero for the previous 6 months shows a large increase in usage by drivers. The two largest limiting factors in the detection of these inhalants is time of sample collection and instrumentation. First, the tissue solubility of various gases will affect the time window for collection. Nitrous oxide has a half-life of 5 minutes versus toluene which has an initial average half-life of 4.5 hours.[3] Very short half-lives require the sample to be collected soon after the supposed time of impairment otherwise the gas may be eliminated. Second, the specificity of the detector during the analysis affects the ability to identify various compounds. Mass spectrometry allows for explicit identification of a compound based on fragmentation patterns. The ability to reference a library of mass spectrums instead of unknown retention times allows proper standards to be used. This is advantageous since there is such a wide range of inhalants and often little or no case history is submitted.

References:

1. *Basic & Clinical Pharmacology*; Katzung, B., Ed. McGraw-Hill: New York, 2004.
2. *Inhalant Abuse*; National Institute on Drug Abuse Research Report (NIH Publication Number 05-3818). Revised March 2005.
3. R.C. Baselt. *Disposition of Toxic Drugs and Chemicals in Man*, 7th ed. Biomedical Publications, Foster City, CA 2004.

CASE NOTES #3: HOW MANY DRUGS DOES IT TAKE TO PROVE IMPAIRMENT?

Michele Glinn, Ph.D., Michigan State Police Toxicology Laboratory, Lansing, MI

Prosecutors in our state like their toxicology neat. They want to hear "The driver was definitely impaired. With that drug level, you are intoxicated. No question." They get irritated when they are told, "Maybe...it depends on the circumstances...it depends on the subject's medical history...I can't say that could never be true...." But sometimes you get a case where the toxicological findings seem to be pretty solid, the conclusions pretty straightforward, and you know your prosecutor is going to be happy. For instance, this one:

Scenario: A fatal crash occurred at 12:45 pm in a Detroit suburb. A driver in an SUV broadsided a van at an intersection, killing the van's elderly driver. Toxicological examination of the SUV driver's blood showed 566 ng/mL diazepam, 1081 ng/mL nordiazepam, 8 ng/mL hydrocodone, 135 ng/mL methadone, a methadone metabolite (not quantified) and 17 ng/mL 11-COOH-THC (no parent THC).

This driver had a lot prescription drugs on board, not to mention the cannabinoid. The prescriptions are all within therapeutic ranges, but the therapeutic ranges for diazepam and methadone are extremely broad (20 - 4000 ng/mL and 75 - 1100 ng/mL, respectively[1]); and, as any good toxicologist knows, "therapeutic range" does not equal "no effects." Even single doses of benzodiazepines can impair vigilance[2,3], and these levels of diazepam and nordiazepam are more reflective of multiple doses or chronic use. And then there's the cannabinoid: a moderate amount, in our lab's experience; and although no THC was found, THC resides in the brain after leaving the blood[4], where it presumably exerts its effects for some time further. The effects of THC can last up to 24 hours

CASE NOTES #3 CONTINUED:

depending on the ability tested[5], certainly past the time where it might no longer be detectable in blood.

The lab report raises the suspicion that the driver was impaired, probably seriously impaired. One might expect the rest of the circumstances of the case to support this opinion. Only they don't, exactly.

First, there are the observations of the witnesses. The crash occurred because the victim's van cut off the defendant's SUV at the intersection. The elderly driver of the van had a history of bad driving and had had his license suspended in the past because he had been involved in so many accidents. The defendant was not speeding and hit the brakes before the crash, as shown by the skid marks he left behind. No bad driving on his part was seen by anyone. Witnesses were unanimous that the crash was the fault of the victim, and the police report reflected as much.

The police and EMTs were called to the scene and transported both drivers to the hospital. The defendant was treated, observed and interviewed by a variety of trained personnel. None reported any apparent impairment. Hospital reports showed the defendant to be alert, conscious, and oriented. The police report specifically mentioned that no impairment was observed. No field sobriety tests were done, and it seems that none were thought necessary.

Blood was sent to the Michigan State Police for toxicological analysis as a matter of procedure. The officers were stunned by the results.

But when the defendant was shown a copy of the lab report, he admitted to all of it. He had prescriptions for Dolophine, Valium and Lorcet. He took them routinely, and had been doing so for at least six months. His normal dose was 3-4 tablets of Valium per day for muscle spasms. He had taken 2 Lorcet and 1/2 Dolophine early the day of the accident, and admitted to smoking marijuana about 11 pm the night before.

There was no obvious evidence of any impairment on the part of the defendant. Still, one could hypothesize that his divided attention abilities and reaction time could have been compromised with so many drugs in his system. He might have been able to react more quickly to an unexpected event if he hadn't been so medicated. But the defendant also admitted to being on his cell phone as he was driving. Foolish, maybe, but not illegal. If he was not able to respond quickly enough to an unexpected event, was it the drugs...or was it the distraction of the cell phone?

The drug levels seen here are consistent with the defendant's story. Steady-state blood levels of chronic diazepam and methadone users can reach 1500 ng/mL and 1000 ng/mL respectively[1,6]. Tolerance to the sedative effects of both drugs develops, and because of this, blood levels alone are not a good indication of impairment[7,8]. The levels of hydrocodone are also quite low, consistent with a dose several hours beforehand (therapeutic range, up to 250 ng/mL[1]); the acute effects would be expected to be in decline. And if the driver had smoked marijuana the evening before, most of the acute cannabis effects would have returned to baseline by the time of the accident[5]. Could there be residual effects? Possibly. Could there be a combination effect from everything? Probably. But there is no manifestation of such the toxicologist can point to and say, "That shows he was impaired." Bad driving? None. Cause of the crash? Victim's fault. Witness observations? Not impaired. Field sobriety testing? None. Reaction time deficit? Possible distraction from the cell phone. The only evidence for impairment is the lab report, and against that are the statements of several professional and lay witnesses that nothing in the driving or subject's behavior indicated impairment and that the victim's behavior, not the defendant's, was the cause of the accident.

It is possible that without the drugs on board, the defendant would have been able to react to the victim cutting him off, cell phone or no; however, this is mere speculation. The honest toxicologist cannot dispute the defense's likely positions that the driver is a tolerant user of the above prescribed medications, that acute effects of the cannabinoids had likely resolved by the time of the accident, and that trained medical witnesses would be expected to notice or document if there was evidence of impairment. In summary, there is no evidence besides the lab report that the driver was impaired, and a fair amount of circumstantial evidence that he wasn't.

I could not conclude that this driver was impaired, and the result was one more dissatisfied prosecutor. Beyond that, it was a lesson in not to jump to conclusions based on the lab report, and to consider all the circumstances of the case before formulating an opinion.

References:

1. Winek C. et. al. *Forensic Science International*.(2001):**122 (2)**:107-123.
2. Kozena L. et al., *Psychopharmacology* (1995):**119**:39-46.
3. Nakazono K. et al, *Yakugaku Zasshi (Pharmaceutical Soc. of Japan)*, (2005):**125(3)**:307-314.
4. Mura P. et al, *J. Analytical Toxicology* (2005): **29**:842 (letter).
5. Huestis M.A, *Forensic Science Reviews* (2002), 1**4(1/2)**:15-59.
6. Baselt R., *Disposition of Toxic Drugs and Chemicals in Man*, 6th Ed., 2002, Biomedical Publications, Foster City, CA.
7. Drummer O.H., *Forensic Science Reviews* (2002), **14(1/2)**:1-14.
8. Stout P.R., *Forensic Science Reviews* (2003), **15(1)**:29-60.

TOXICOLOGY - BITS & PIECES

Submitted by Section Editor, J. Robert Zettl, MPA

A.A.F.S. NEWS — TOXICOLOGY SECTION

The following is redacted from the May/June 2008 "Academy News" by Peter R. Stout, Ph.D., Section Chair:

The submission deadline for AAFS workshops and abstracts is August 1,2008. Those considering a workshop should contact Phil Kemp (p_kemp@ocmeokc.state. ok.us).

Ken Ferslew is Toxicology Section Program Chair for this year. If a member is interested in assisting with the February 2009 meeting in Denver, Colorado please contact Ken (ferslew@mail.etsu.edu).

The Section has formed an ad hoc committee consisting of Robert Bost, Henry Nipper, John Soper, Christopher Boden and Audra Brown that will be tasked to evaluate the Section's membership and work with AAFS to contact members who may be up for promotion and provide whatever assistance they may need.

The Section elected Marilyn Huestis to fill the seat vacated by Barry Logan on the AAFS Board of Directors. William Anderson, Diana Garside and Loralie Langman were elected to serve on the Awards and Scholarship Committee. Nikolas Lemos and Michael Corbett were elected to the Nominating Committee.

C.A.T. AT S.O.F.T. IN PHOENIX

The California Association of Toxicologists will meet Sunday, October 26, 2008 immediately preceding the S.O.F.T. 2008 meeting in Phoenix. Two half day workshops are scheduled; "Creating and Giving Quality Presentations" and "Street Drugs & Culture". Find details at the C.A.T. website (www.cal-tox.org).

A.A.F.S. / S.O.F.T. JOINT DRUGS & DRIVING COMMITTEE

Committee Chair, Dr. Sarah Kerrigan reports that a DUID website is under development. The site will contain a variety of DUID related resources for toxicologists, including but not limited to suggested reading, upcoming training events, government publications, DRE and legal information. A preliminary form of the website was presented for discussion purposes at the DUID committee meeting in Washington DC, and the committee hopes to have this project completed in October 2008 in time for the SOFT annual meeting in Phoenix.

The SOFT Continuing Education Committee and the SOFT/AAFS Drugs & Driving Committee recently concluded the "Interpretive DUID Course" in concert with the Palm Beach County Sheriff's Office in West Palm Beach, Florida on May 6-8th. The class was well attended with 25 attendees and six faculty. Sincere appreciation is extended to Tate Yeatman and Ann Marie Gordon for their time and commitment organizing this worthwhile workshop.

T.I.A.F.T. NEWS

La Martinique and the French West Indies welcomed visitors to the 46th annual meeting of the International Association of Forensic Toxicologists June 2-8, 2008. Two other organizations co-hosted this joint meeting; the French Society of Analytical Toxicology and the Society of Hair Testing.

TIAFT 2009 is scheduled August 23-27, 2009 in Geneva, Switzerland. Find details at the TIAFT website (www.tiaft.org).

NATIONAL SAFETY COUNCIL—COMMITTEE ON ALCOHOL AND OTHER DRUGS

The NSC/COAOD last met in February of 2008 in San Antonio, Texas. The committee officers for this year remain: Jerry Landau, Chair, Mack Cowan, Vice Chair, and Laura Liddicoat, Secretary; as well as other standing technical subcommittee chairs and co-chairs.

The next executive committee meeting of the NSC/COAOD will be held during the 2008 SOFT Conference in Phoenix. The agenda will include the committee's normal business and reports from its technical subcommittees. The meeting is open to anyone wishing to attend. Please refer to your program for the time and place.

At the Phoenix meeting the committee will announce a recipient to be awarded the Robert F. Borkenstein Award. This individual will be one who has a minimum tenure of 25 years of active service in the area of alcohol/drugs and traffic safety, has contributed to that field to a degree that their achievements are nationally recognized and has a minimum of 10 years of active and productive involvement as a volunteer with the National Safety Council. This year's awardee is -------? Please keep tuned as many of you long time SOFT members may want to attend that award banquet.

A.A.F.S. FUTURE MEETING SITES

2009 Denver, CO
2010 Seattle, WA
2011 Chicago, IL
2012 Atlanta, GA
2013 Washington, DC
2014 Seattle, WA

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ToxTalk is the official publication of the Society of Forensic Toxicologists, Inc., mailed quarterly (bulk mail) to its members. It is each member's responsibility to report changes of address to the SOFT Administrative Office. Non-members may receive ToxTalk for $15 per calendar year. Checks payable to SOFT may be mailed to the SOFT Administrative Office. To submit articles or address ToxTalk issues please email to ToxTalk@soft-tox.org.

ToxTalk **Deadlines** for Contributions

February 1 for March Issue
May 1 for June Issue
August 1 for September Issue
November 1 for December Issue

Future S.O.F.T. Meeting Info

2008: Phoenix, AZ……....Oct. 27-31, 2008…….Vickie Watts, Norman Wade

2009: Oklahoma City, OK……Oct. 18-23, 2009……...……...Phil Kemp

2010: Richmond, VA……Oct. 18-22, 2010…..…..Michelle Peace, Lisa Tarnai Moak

2011: San Francisco, CA……...Aug. 29-Sep. 2, 2011…….…….Nikolas Lemos

2012: Boston, MA…….June 30-July 6, 2012…..……..……...…Michael Wagner

Above: Downtown Phoenix

Below: Papago Buttes, Phoenix

NEED VOLUNTEERS

The S.O.F.T. 2008 meeting in Phoenix will be here before we know it! It takes the effort of many people to ensure all events proceed smoothly. Volunteers are always needed to help ensure that meeting attendees fully enjoy their stay in the host city.

Anyone able to offer a few hours of time during the meeting as a S.O.F.T. Volunteer should contact Deb Denson, the S.O.F.T. Volunteer Coordinator to schedule times and locations desirable to help with.

Thanks to both past and future volunteers for helping make S.O.F.T. meetings efficient and enjoyable for everyone!

Deb Denson, SOFT Volunteer Coordinator
Email: denson@rti.org Tele: 919-541-7265

2008 S.O.F.T. COMMITTEE CHAIRS

Committee	Committee Chair
Nominating	Diana Wilkins, Ph.D.
Membership	Sarah Kerrigan, Ph.D.
Strategic Planning	Bradford Hepler, Ph.D., DABFT
Budget, Finance, and Audit	Robert Turk, Ph.D., DABFT
ToxTalk Co-Editors	Yale Caplan, Ph.D., DABFT Vickie Watts, M.S.
ByLaws	Yale Caplan, Ph.D., DABFT
Publications (JAT Special Issue)	Dan Anderson, M.S., ABFT
Awards	Philip Kemp, Ph.D., DABFT
Drugs & Driving	Sarah Kerrigan, Ph.D.
Meeting Resource	Anthony Costantino, Ph.D., DABFT
Policy and Procedure	William Anderson, Ph.D.
SOFT Internet Web-Site	Bruce Goldberger, Ph.D., DABFT
Continuing Education	Ann Marie Gordon, M.S.
Laboratory Guidelines	W. Lee Hearn, Ph,D.
Ethics	Aaron Jacobs, Ph.D.
Drug Facilitated Rape & Sexual Assault	Marc LeBeau, Ph.D.
MS/MS Guidelines	John Cody, Ph.D.

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