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Attorneys for Plaintiffs

IN THE UNITED STATES DISTRICT COURT
DISTRICT OF OREGON
(Medford Division)

**THE CHURCH OF THE HOLY LIGHT OF
THE QUEEN**, a/k/a The Santo Daime Church,
an Oregon religious corporation, on its own
behalf and on behalf of all of its members,
JONATHAN GOLDMAN, individually and as
Spiritual Leader of the "Santo Daime Church,"
**JACQUELYN PRESTIDGE, MARY ROW,
M.D., MIRIAM RAMSEY, ALEXANDRA
BLISS YEAGER and SCOTT FERGUSON**,
members of the Santo Daime Church,

Plaintiffs,

v.

Civil No. 08-cv-03095-PA

**AMENDED EXPERT WITNESS
STATEMENT OF NICHOLAS V.
COZZI, Ph.D.**

MICHAEL B. MUKASEY, Attorney General
of the United States; **KARIN J. IMMERGUT**,
United States Attorney, District of Oregon;
HENRY M. PAULSON, Secretary of the U.S.
Department of the Treasury,

Defendants.

My name is Nicholas V. Cozzi, Ph.D.

PHARMACOLOGICAL, TOXICOLOGICAL, AND DRUG POLICY ISSUES SURROUNDING THE SOUTH AMERICAN DAIME

I have been requested to provide my expert opinion on the pharmacology of "Daime", a sacramental tea used by the Santo Daime Church. Daime is prepared from the bark of *Banisteriopsis caapi* and the leaves of *Psychotria viridis*, two plants indigenous to the Amazon region of South America. Daime is also known as "ayahuasca" and "hoasca" outside the Santo Daime Church. I have also been asked to provide an assessment of any toxic effects that may arise from ingesting Daime. Finally, I have been asked to comment on drug policy issues related to Daime.

I hold a B.S. degree in Pharmacology and Toxicology from the University of Wisconsin and a Ph.D. degree in Pharmacology, also from the University of Wisconsin. Pharmacology is commonly defined as the unified study of the properties of chemicals and living organisms and all aspects of their interactions. Toxicology is a subdivision of Pharmacology that is concerned with the adverse or toxic effects of chemicals on biological systems. My education and experience in these disciplines is detailed in my curriculum vitae. I currently hold the positions of Faculty Associate and Senior Scientist in the Department of Pharmacology at the University of Wisconsin School of Medicine and Public Health, Madison, WI. I am both the course director and an instructor in the Medical Pharmacology course taken by all second-year medical students as well as graduate students in the Molecular and Cellular Pharmacology program at the University of Wisconsin. I also direct a research program focused on the pharmacological effects of various psychoactive substances, including the substances present in Daime. My knowledge of the pharmacology and toxicology of Daime comes from my reading of the medical and scientific literature, my participation in professional conferences, discussions with colleagues, and my own research over more than twenty-five years.

In preparation for writing this Declaration, I have reviewed numerous scientific journal articles, the expert reports of Dr. John Halpern, and the report submitted by Dr. David Nichols for the "O Centro case" involving the União do Vegetal (Joint Appendix to the Supreme Court at 337).. I will testify to my agreement with all of Dr. Nichol's statements.

Introduction

The earliest published reports in the Western literature of ayahuasca (Daime) use were written by Richard Spruce, an English botanist and explorer. Spruce described the use of ayahuasca by several tribes living in the upper Amazon River region of South America. These accounts date to the 1850's, but it is evident that the sacramental tea has been used by native South American peoples from much earlier times, possibly thousands of years. The tea has been used for both religious and ceremonial purposes by these people. In eastern Peru, for example, the tea is ingested by shamans to diagnose and treat diseases, while in Colombia and Brazil, Daime is employed in religious ceremonies that are rooted in tribal mythology. In more recent times, the religious use of ayahuasca has been organized under the auspices of the Santo Daime Church (who call it Daime) and the União do Vegetal (who call it hoasca tea). Both of these religions are syncretic religions. That is, they combine preexisting local beliefs with the Christian beliefs of European people with whom they have intermingled. The state of consciousness elicited by Daime is said to be deeply religious and is discussed in other reports. Similar states of consciousness, often referred to as "hallucinogenic", are produced by various plants and synthetic compounds.

The Molecular Pharmacology of Daime

Studies of the plants used to prepare Daime reveal that these plants contain pharmacologically active compounds. One of the plants used to prepare Daime, *Psychotria viridis*, contains the indole alkaloid *N,N*-dimethyltryptamine (DMT). DMT is not unique to *Psychotria viridis* but is found in hundreds of plants around the world. DMT is a potent psychoactive drug in its synthetic form and when injected; however, it is not normally active when consumed by mouth because it is rapidly destroyed by enzymes in the gut. In order for DMT to be orally active, the enzymes that destroy DMT in the gut must first be neutralized. *Banisteriopsis caapi*, the other main component of Daime, in fact contains the substances that neutralize the enzymes that would normally destroy DMT. Thus, it is the combination of both *Psychotria viridis* and *Banisteriopsis caapi* together that allows the Daime tea to produce its psychoactive effects.

The psychoactive properties of Daime tea are due to the abilities of the substances in the tea (DMT and other compounds) to act on certain receptor proteins found in nerve cells. These receptors normally serve to convey the chemical (neurotransmitter) signals released by one cell into a response in another cell. It is the release of chemical neurotransmitters, the binding of these neurotransmitters to receptors, and the generation of a response in another cell that is the biochemical basis for cell-to-cell communication in the brain. In other words, neurons "talk" to each other by releasing chemicals that bind to receptors on other cells, and it is the neurons "talking" to each other that underlies various moods, feelings, and thoughts. By acting on receptor proteins, DMT and the other compounds present in Daime alter the cell-to-cell communication, thereby changing the cellular "dialogue". This is the primary molecular mechanism which underlies the changes in thinking, feeling, and perception that occur after ingesting Daime.

Is Daime Toxic?

There are no credible reports in the medical literature of overt toxicity resulting from Daime use. Also, from a toxicological viewpoint, there is nothing particularly remarkable about the chemical structures of DMT or the other compounds found in Daime that would suggest that these molecules would be chemically damaging to cells. In fact, in a study conducted in the Brazilian Amazon in which Daime users were compared to matched controls, the Daime users revealed a high functional status with no evidence of personality or cognitive deterioration (Human Psychopharmacology Of Hoasca, A Plant Hallucinogen Used In Ritual Context In Brazil. CS Grob; DJ McKenna; JC Callaway; GS Brito; ES Neves; G Oberlaender; OL Saide; E Labigalini; C Tacla; CT Miranda; RJ Strassman; KB Boone. *J. Nerv. Ment. Dis.*, 184, 86-94 [1996]). In a meta-analysis of nine earlier studies on the psychological effects of chronic hallucinogen use, (including LSD, psilocybin, and mescaline, but not Daime), it was concluded that few, if any, long-term neuropsychological deficits could be attributed to the use of these substances (Do Hallucinogens Cause Residual Neuropsychological Toxicity? JH Halpern; HG Pope. *Drug Alcohol Depen.*, 53, 247-256 [1999]). Nevertheless, any substance can be toxic in sufficiently high amounts. The potential to consume a toxic amount of Daime tea is, however, practically-nonexistent for the following reason: Daime typically produces nausea and vomiting in the amounts consumed for religious purposes. This naturally limits the amount of Daime that can be ingested; the purging effect makes it virtually impossible for a human being to consume enough Daime tea to experience toxicity. This is in contrast to most other substances taken by mouth, including over-the-counter and prescription drugs, where such a fail-safe mechanism does not exist. As an aside, the vomiting produced by Daime tea is not considered to be an undesirable effect by Daime users. This is because the religious users of Daime consider the vomiting to be a purging of toxins from the body.

As is true for many pharmacologically active substances, there may be interactions between Daime and other medications. In particular, a person who has been prescribed certain antidepressants (including tricyclic antidepressants, selective serotonin reuptake inhibitors, and monoamine oxidase inhibitors) or who uses certain over-the-counter cold remedies (especially those containing phenylephrine or pseudoephedrine) should not ingest Daime, lest a possibly serious drug-drug interaction occur. This interaction can trigger a so-called "hypertensive crisis" characterized by severe headache, elevated blood pressure, shortness of breath, and anxiety. In addition, other prescription drugs (e.g. some asthma medications) and some foods (e.g. avocados, aged cheese, sherry) contain compounds that are inactivated by the same enzymes that are neutralized by the substances in Daime. It is possible that, in the presence of Daime, these compounds could reach levels in the body that would produce too great of an effect. I am informed that Santo Daime Church leaders are carefully trained to obtain information from prospective church members before administering Daime to prevent such drug interactions from occurring. As noted by the Brazilian Drug Council's report, there have been no physical problems reported as a result of taking Daime, suggesting that the screening procedure is effective. I have provided a list of contraindicated pharmaceuticals to assist in reviewing the medical status of persons to determine whether they should avoid drinking the tea.

Drug Control Policy

According to Federal regulations (TITLE 21 - FOOD AND DRUGS CHAPTER 13 - DRUG ABUSE PREVENTION AND CONTROL SUBCHAPTER I - CONTROL AND ENFORCEMENT Part B - SECTION 812; 21 USC Sec. 812 01/22/02), drugs are restricted in their availability according to three criteria: medical use, ability to produce physical or psychological dependence, and abuse potential. Drugs are assigned to one of five categories (Schedules) based on various combinations of these three criteria. Regarding the first criterion, the use of Daime in a religious context is not primarily intended as a "medical" use (i.e. it is *non-drug* use, although there is indeed clinical evidence that religious experiences and spirituality have beneficial effects on a person's health). With respect to physical or psychological dependence, there are neither anecdotal reports nor reports in the medical literature that Daime tea produces either condition. That is, there are no indications that Daime users feel compelled to continue use either because of psychological craving or physical need. What can be said of Daime in terms of abuse potential? Certainly, it is a drink that does not seem to lend itself well to casual use. It is not particularly convenient to prepare nor is the drink palatable. As noted earlier, it often causes nausea and vomiting. Because of its purgative effect, the amount that can be consumed is limited. Together, these considerations suggest that Daime has little or no abuse potential.

The real nature of Daime is only appreciated when its *intended use* is recognized. The intended use of a substance, especially for religious use in a structured setting with the goal of enriching one's spiritual being, has special significance. Religious beliefs are among the most personal and potentially life-improving forces in human culture. It is apparent that Santo Daime Church members are absolutely sincere in their religious beliefs and that Daime tea is for these people a means to achieve greater spiritual fulfillment. Furthermore, Daime is used under responsible, controlled conditions. For these reasons, the use of Daime by the Santo Daime Church should be held in the same high esteem and afforded the same legal protections that are provided to other religions. The *non-drug* use of alcohol (wine) in religious ceremonies such as the Catholic Mass is not considered abuse; indeed, it is protected. Likewise, while peyote is classified as a drug of abuse under the Controlled Substances Act of the United States, the *non-drug* use of peyote in Native American Church ceremonies is recognized as a special situation and is not considered abuse; like the religious use of alcohol, it is also protected by law. Clearly, the use of Daime tea in religious ceremonies conducted by the Santo Daime Church falls into the same category as the religious use of alcohol or peyote: it is *non-drug* use. It, too, should be legally protected.

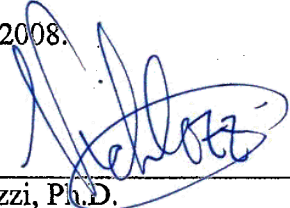
At the request of plaintiffs' counsel, I have reviewed the two-page document marked Plaintiffs' Exhibit 4, which is the report analyzing the tea seized from plaintiff Goldman in May 1999. The document contains reports of the analysis of several exhibits containing dimethyltryptamine (DMT). The concentration of DMT in each exhibit is expressed in mg/mL, and each exhibit contains different concentrations, ranging from 0.27 mg/mL to 0.84 mg/mL. The total amount of DMT in each exhibit (AMT. OF PURE DRUG, column 32) is obtained by multiplying the concentration by the total volume of the respective exhibit. For example, on Exhibit 4's second page, the concentration of DMT in Exhibit 4A is reported as 0.27 mg/mL (columns 29 and 30). The total volume of Exhibit 4A is given as 13.40 L (= 13400mL; column 33). Thus, $0.27 \text{ mg/mL} \times 13400 \text{ mL} = 3618 \text{ mg} = 3.618 \text{ g}$; this is rounded to 3.6 g (column 32).

The amount of DMT required for psychoactive effects is approximately 50-100 mg (see for example, <http://www.usdoj.gov/dea/pubs/abuse/8-hallu.htm#Psilocybin>). At the concentrations reported in Plaintiffs' Exhibit 4, one would need to consume 100-150 mL (roughly ½ cup) to produce psychoactive effects.

I have not testified in the past four years in court or by way of deposition. I was paid approximately \$2,000 for consultation and report writing services.

The statements set forth in this document are my own and are based on my education and experience. Pursuant to 28 USC § 1746, I declare under penalty of perjury that the foregoing is true and correct.

DATED this 10 day of November, 2008.



Nicholas V. Cozzi, Ph.D.

Exhibit A

to

Amended Expert Witness Statement

of

Nicholas V. Cozzi, Ph.D.

Nicholas V. Cozzi, Ph.D.
Department of Pharmacology
University of Wisconsin School of Medicine and Public Health
Madison, WI 53706
608-262-7596 cozzi@wisc.edu

CURRICULUM VITAE

2006-present	Faculty Associate/Senior Scientist	Dr. Arnold Ruoho, Chair
1996-1998	Senior Research Associate	Dept. of Pharmacology School of Medicine and Public Health University of Wisconsin-Madison Madison, WI 53706
2005-2007	Professor of Pharmacology	Dr. Jane Hopp, Dean Dept. of Natural and Health Sciences Carroll College Waukesha, WI 53186
2004-2006	Director of Pharmacology	John Seman, CEO PhysioGenix, Inc. 10437 Innovation Dr. Wauwatosa, WI 53226
1998-2004	Assistant Professor of Pharmacology and Toxicology	Dr. David Taylor, Chair Dept. of Pharmacology and Toxicology Brody School of Medicine East Carolina University Greenville, NC 27858
1995-1996	Postdoctoral Fellow	Dr. Cynthia Czajkowski Dept. of Physiology School of Medicine and Public Health University of Wisconsin-Madison Madison, WI 53706
1994	Ph.D., Pharmacology Thesis: <i>Pharmacological Studies of Some Psychoactive Phenylalkylamines: Entactogens, Hallucinogens, and Anorectics.</i>	Dr. Thomas Rudy Dept. of Pharmacology School of Pharmacy University of Wisconsin-Madison Madison, WI 53706
1992-1993	Research Associate	Dr. David Nichols Dept. of Medicinal Chemistry School of Pharmacy and Pharmacal Sciences Purdue University West Lafayette, IN 47907
1989-1991	Research Associate	Dr. Molly Weiler Dept. of Pharmacology School of Pharmacy University of Wisconsin-Madison Madison, WI 53706
1988	B.S., Pharmacology and Toxicology	School of Pharmacy University of Wisconsin-Madison Madison, WI 53706

Teaching

- 2008-present Course Director, Instructor: Physiology in Pharmacology (graduate course). Dept. of Pharmacology, University of Wisconsin School of Medicine and Public Health, Madison, WI. Topics: homeostasis, cell structure, movement across membranes, neuronal signaling, sensory physiology, brain, muscle, endocrine, reproduction, cardiovascular system, pulmonary, renal, gastrointestinal, metabolism, immunology.
- 2007-present Course Director, Instructor: Medical Pharmacology (professional/graduate course). Dept. of Pharmacology, University of Wisconsin School of Medicine and Public Health, Madison, WI. Topics: general principles, drug disposition, autonomic and neuromuscular drugs, cardiovascular and blood drugs, central nervous system agents, gastrointestinal drugs, chemotherapeutics, drug therapy of inflammation, endocrine drugs, botanicals, toxicology, gene therapy, immunosuppressant agents, drug interactions.
- 2005-2007 Course Director, Instructor: Pharmacology (professional course). Dept. of Natural and Health Sciences, Carroll University, Waukesha, WI. Topics: general principles, drug disposition, autonomic and neuromuscular drugs, cardiovascular and blood drugs, central nervous system drugs, gastrointestinal drugs, chemotherapeutics, drug therapy of inflammation, endocrine drugs, botanicals, toxicology, drug interactions.
- 2003-2004 Instructor: Physiological Proteogenomics (graduate course). Dept. of Physiology, Brody School of Medicine, East Carolina University, Greenville, NC. Course coordinator: Dr. Alexander Murashov. Topic: applications of proteome analysis to drug development and toxicology.
- 2001-2004 Course Director, Instructor: Pharmacology Seminar (graduate course). Dept. of Pharmacology and Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC.
- 2000-2004 Instructor: Molecular Pharmacology (graduate course). Dept. of Pharmacology and Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC. Course coordinator: Dr. Tatyana Nikolova. Topics: receptor kinases, neurotransmitter transporters, cell adhesion molecules, site-directed mutagenesis, chimeras, positron emission tomography.
- 2000-2004 Course Director, Instructor: Laboratory Research Techniques (graduate course). Dept. of Pharmacology and Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC. Topics: neurotransmitter transporter assays, cell culture techniques, polymerase chain reaction.
- 1999-2004 Instructor: Central Nervous System Pharmacology (graduate course). Dept. of Pharmacology and Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC. Course coordinator: Dr. Brian McMillen. Topics: neurotransmitter receptors, gene knockouts, synaptic vesicle storage and release mechanisms.
- 1999-2004 Instructor: Cellular and Molecular Neuroscience (graduate course). Neuroscience Program, East Carolina University, Greenville, NC. Course coordinator: Dr. Edward Lieberman. Topics: neurotransmitter receptors, neurotransmitter transporters.
- 1998-2004 Instructor: Medical Pharmacology (professional/graduate course). Dept. of Pharmacology and Toxicology, Brody School of Medicine, East Carolina University, Greenville, NC. Course coordinator: Dr. Donald Barnes. Topics: general principles, receptor mechanisms, dose-response relationships, absorption, distribution, metabolism, excretion, pharmacokinetics, drug interactions.
- 1986-present Visiting Professor: Neuropharmacology (undergraduate course). Dept. of Learning, Development, and Special Education, Northern Illinois University, DeKalb, IL. Course coordinator: Dr. Thomas Roberts. Topic: CNS agents.
- 1995-1998 Instructor: Pharmacology (professional course). Dept. of Pharmacology, University of Wisconsin School of Pharmacy, Madison, WI. Course coordinator: Dr. Thomas Rudy. Topic: cardiovascular pharmacology.
- 1994-1998 Instructor: Environmental Toxicology (graduate course). Center for Environmental Toxicology, University of Wisconsin-Madison, Madison, WI. Course coordinator: Dr. E. Burt Olson, Jr. Topics: neurotoxicology, gastrointestinal toxicology.

1989-1994 Teaching Assistant: Pharmacy curriculum (various undergraduate Pharmacy courses). University of Wisconsin School of Pharmacy, Madison, WI. Course coordinator: Dr. William Mellon.

Graduate Students

Dr. Kevin Foley; received Ph.D. 2002

Dr. Kevin DeSanty; received Ph.D. 2002

Dr. LuAnn Cuthbertson-Lucas; received Ph.D. 2001

Ms. Jessica Gaskey

Service

Educational Policy Council, University of Wisconsin School of Medicine and Public Health

Year 2 Curriculum Steering Committee, University of Wisconsin School of Medicine and Public Health

Consulting Editor, *Journal of Drug Education and Awareness*

Manuscript Reviewer: *Journal of Neurochemistry*, *Journal of Neural Transmission*, *Archives of Toxicology*, *CNS Neuroscience & Therapeutics*

Research Proposal Reviewer, Department of Veterans Affairs, Office of External Reviews, Neurobiology-D

Telemedicine Distance Learning Committee, Brody School of Medicine, East Carolina University

Neuroscience Steering Committee, East Carolina University

Neuroscience Symposium Organizing Committee, East Carolina University

Neuroscience Doctoral Program Curriculum Committee, East Carolina University

Judge, Doctoral Student Research Day, Brody School of Medicine, East Carolina University

Honors/Awards

University of Wisconsin Professional Development Grant. Awarded 2008-2009

National Institute on Drug Abuse Grant DA017675: *Amphetamine-Related Photoaffinity Probes*. Awarded 2004-2007

Brody Basic Science Incentive Award, Brody School of Medicine, East Carolina University. Awarded 2001

Excellence in Teaching Award, Brody School of Medicine, East Carolina University. Awarded 2000

Young Investigator Award, National Alliance for Research on Schizophrenia and Depression (NARSAD): *Molecular Structure of the Antidepressant Binding Site on the Serotonin Reuptake Transporter*. Awarded 1997-2000

Presentations and Scholarly Activities

2007 UW School of Medicine and Public Health Team Member, Enhancing the Professional Culture of Schools of Medicine: Relationship-Centered Care Initiative Immersion Conference II, Indiana University School of Medicine and Regenstrief Institute, Indianapolis, IN. Host: Dr. Thomas Inui

2004 Participant, NIH Summit Workshop on Predictive Toxicology, NIH Campus, Bethesda MD.

2003 PhysioGenix, Milwaukee, WI. Topic: *New Ways to Skin a Cat*. Host: Dr. Howard Jacob.

2003 Discovery Channel Unsolved History series: Salem Witch Trials. Topic: *Stability of ergot alkaloids under conditions of extreme heat*. Producer: Sue Houghton. First national airing 10/22/03.

2003 Discovery Channel Unsolved History series: Death of Marilyn Monroe. Topic: *Pharmacokinetics of pentobarbital absorption*. Producer: Dr. James Younger. First national airing 10/01/03.

2003 Department of Pharmaceutical Sciences, University of Wisconsin School of Pharmacy, Madison, WI. Topic: *Another Way To Skin A Cat(hinone)*. Host: Dr. Thomas Rudy.

2002 Department of Cellular and Molecular Pharmacology, Chicago Medical School, Finch University of Health Sciences, North Chicago, IL. Topic: *Novel Monoaminergic Agents*. Host: Dr. Francis White.

2002 Department of Chemistry, East Carolina University, Greenville, NC. Topic: *Novel Monoaminergic Agents*. Host: Dr. Chia-yu Li.

2001 Department of Physiology, East Carolina University, Greenville, NC. Topic: *Probing Monoamine Transporters with Aminopropiophenones*. Host: Dr. Alexander Murashov.

2000 Department of Medicinal Chemistry and Department of Pharmacology and Toxicology, Virginia Commonwealth University, Richmond, VA. Topic: *Mapping the Serotonin Reuptake Transporter*. Host: Dr. William Devane.

1995 Invited Speaker, The Rainforest Pharmacy. Massachusetts College of Pharmacy, Boston, MA. Topic: *Drugs of the Rainforest: A Pharmacological Sampler*. Host: Dr. June Riedlinger.

1991 Panel Member, The Bridge Conference. Stanford University, Palo Alto, CA. Topic: *Drug Education at the College Level*. Host: Dr. Thomas Roberts.

1991 University of Wisconsin Medical School, Madison, WI. Topic: *Nerve Gases: Mechanisms of Toxicity, Physiological Effects, and Antidotes*. Host: Pre-Medical Student Association.

Publications

Refereed

D Fontanilla, M Johannessen, AR Hajipour, **NV Cozzi**, MB Jackson, AE Ruoho. The hallucinogen *N,N*-dimethyltryptamine (DMT) is an endogenous sigma-1 receptor regulator. *Science (in press)*

SD Brandt, SS Tirunaranapuram, S Freeman, N Dempster, SA Barker, PF Daley, **NV Cozzi**, CPB Martins. Microwave-accelerated synthesis of psychoactive deuterated *N,N*-dialkylated-[$\alpha,\alpha,\beta,\beta$ -d₄]-tryptamines. *J. Labelled Cpd. Radiopharm., (in press)*

JM Dorsey, MG Miranda, **NV Cozzi**, KG Pinney. Synthesis and biological evaluation of 2-(4-fluorophenoxy)-2-phenylethyl piperazines as serotonin-selective reuptake inhibitors with a potentially improved adverse reaction profile. *Bioorg. Med. Chem., 12*, 1483-1491 (2004)

NV Cozzi, KF Foley. Methcathinone is a substrate for the serotonin uptake transporter. *Pharmacol. Toxicol., 93*, 219-225 (2003)

KF Foley, **NV Cozzi**. Novel aminopropiophenones as potential antidepressants. *Drug Dev. Res., 60*, 252-260 (2003)

KF Foley, ME Van Dort, MK Sievert, AE Ruoho, **NV Cozzi**. Stereospecific inhibition of monoamine uptake transporters by *meta*-hydroxyephedrine isomers. *J. Neural Transm., 109*, 1229-1240 (2002)

KF Foley, **NV Cozzi**. Inhibition of transport function and desipramine binding at the human noradrenaline transporter by *N*-ethylmaleimide and protection by substrate analogs. *Naunyn-Schmiedeberg's Arch. Pharmacol., 365*, 457-461 (2002)

NV Cozzi, KF Foley. Rapid and efficient method for suspending cells for neurotransmitter uptake assays. *Biotechniques, 32*, 486-492 (2002)

RA Sewell, **NV Cozzi**. More about Parkinsonism after taking Ecstasy. *N. Engl. J. Med., 341*, 1400 (1999)

NV Cozzi, MK Sievert, AT Shulgin, P Jacob III, AE Ruoho. Inhibition of plasma membrane monoamine transporters by β -ketoamphetamines. *Eur. J. Pharmacol., 381*, 63-69 (1999)

NV Cozzi, AE Ruoho. Radiosynthesis of [³H]methcathinone, an inhibitor of monoamine reuptake transporters. *J. Labelled Cpd. Radiopharm., XLI*, 927-933 (1998)

NV Cozzi, S Frescas, D Marona-Lewika, X Huang, DE Nichols. Indan analogs of fenfluramine and norfenfluramine have reduced neurotoxic potential. *Pharmacol. Biochem. Behav., 59*, 709-715 (1998)

NV Cozzi, DE Nichols. 5-HT_{2A} receptor antagonists inhibit potassium-stimulated gamma-aminobutyric acid release in rat frontal cortex. *Eur. J. Pharmacol., 309*, 25-31 (1996)

AP Monte, D Marona-Lewicka, **NV Cozzi**, DL Nelson, DE Nichols. Conformationally restricted tetrahydro-1-benzoxepin analogs of hallucinogenic phenethylamines. *Med. Chem. Res., 5*, 651-663 (1995)

AP Monte, D Marona-Lewika, **NV Cozzi**, DE Nichols. Synthesis and pharmacological examination of benzofuran, indan, and tetralin analogs of 3,4-(methylenedioxy)amphetamine. *J. Med. Chem.*, 36, 3700-3706 (1993)

H-J Lee, **NV Cozzi**, MH Weiler. Age-related differences in the effects of some muscarinic agents on acetylcholine release from rat neostriatal slices. *J. Pharmacol. Exp. Ther.*, 258, 496-501 (1991)

Abstracts

NV Cozzi, AT Shulgin, PF Daley, A Gopalakrishnan, LL Anderson, JT Feih, AE Ruoho. Psychoactive *N,N*-dialkyltryptamines modulate serotonin transport by at least two mechanisms. *Soc. Neurosci. Abs.*, 34, 536.17 (2008)

D Fontanilla, M Johannessen, AR Hajipour, A Pal, **NV Cozzi**, MB Jackson, AE Ruoho. *N,N*-dimethyltryptamine (DMT) as an endogenous ligand candidate for the sigma receptor. *Soc. Neurosci. Abs.*, 34, 660.10 (2008)

NV Cozzi, A Gopalakrishnan, LL Anderson, JT Feih, AE Ruoho. *N,N*-dialkyltryptamines inhibit plasma membrane and vesicular serotonin transport. *FASEB J.*, 22, 714.10 (2008)

NV Cozzi, KF Foley, D Fontanilla, A Gopalakrishnan, AE Ruoho. A novel amphetamine-related photoaffinity probe. *FASEB J.*, 21, 715.2 (2007)

DM Raffel, W Chen, YW Jung, DL Gildersleeve, **NV Cozzi**. [¹¹C]-Phenethylguanidines: transport kinetics and binding affinities for the human norepinephrine transporter. *J. Nucl. Med.*, 47, 72P (2006)

RJ Roman, **NV Cozzi**, SH Nye, AJ Dahly-Vernon, JF Baye, DL Evans, YA Evrard, SK Korb, LH Lapczynski, JA O'Connor, HJ Vernon, AL Wittenburg, HJ Jacob. Combinatorial rat panels for predictive toxicology. *Toxicol. Pathol.*, 34, P27 (2006)

AL Wittenburg, JA O'Connor, DL Evans, YA Evrard, LH Lapczynski, SK Korb, HJ Vernon, SH Nye, **NV Cozzi**, AJ Dahly-Vernon, HJ Jacob, RJ Roman. Acceleration of diabetic nephropathy in the T2DN rat. *Amer. Diabetes Assoc. Abs.*, 66th Annual Scientific Sessions, 2186-PO (2006)

JA O'Connor, LH Lapczynski, AJ Dahly-Vernon, YA Evrard, DL Evans, AL Wittenburg, SK Korb, SH Nye, HJ Vernon, **NV Cozzi**, HJ Jacob, RJ Roman. Delay in the progression of diabetic nephropathy in the T2DN rat. *Amer. Diabetes Assoc. Abs.*, 66th Annual Scientific Sessions, 2185-PO (2006)

JA O'Connor, AL Wittenburg, MF Perrine, LH Lapczynski, DL Evans, YA Evrard, SK Korb, AJ Dahly-Vernon, HJ Vernon, SH Nye, **NV Cozzi**, HJ Jacob, RJ Roman. Diabetes-induced nephropathy in the T2DN rat. *Amer. Diabetes Assoc. 66th Annual Scientific Sessions*, 773-P, (2006)

YA Harrington, SK Korb, **NV Cozzi**, SH Nye, AL Wittenburg, HJ Vernon, DL Evans, JA O'Connor, LH Lapczynski, AJ Dahly-Vernon, JF Baye, RJ Roman, HJ Jacob. Using PharmGenix rats to detect tacrine hepatotoxicity. *Clin. Pharmacol. Ther.*, 79, P82 (2006)

NV Cozzi, D Marona-Lewicka, DE Nichols, A Gokin, KF Foley. Novel aminopropiophenones as antiobesity agents. *Soc. Neurosci. Abs.*, 31, 533.5 (2005)

SH Nye, **NV Cozzi**, JF Baye, DL Evans, YA Evrard, SK Korb, JA O'Connor, HJ Vernon, AL Wittenburg, M Hessner, X Wang, HJ Jacob, RJ Roman. Does the lack of genetic diversity in animal models currently used for safety testing put the public at risk? *FDA Science Forum Abs.*, 305 (2005)

KF Foley, RA Galbraith, A Gokin, **NV Cozzi**. Novel aminopropiophenones as antiobesity agents. *Exp. Biol. 2005 Abs.*, 7290 (2005)

SH Nye, **NV Cozzi**, JF Baye, AL Wittenburg, SK Korb, YA Evrard, RJ Roman, HJ Jacob. Improved rat models for predictive toxicology. *Soc. Toxicol. Abs.*, 44, 1897 (2005)

SH Nye, **NV Cozzi**, JF Baye, AL Wittenburg, SK Korb, MJ Guy, RJ Roman, HJ Jacob. Pharmgenix: a combinatorial rat panel for predictive toxicology. *Toxicol. Pathol.*, 33, P53 (2005)

DM Raffel, W Chen, DL Gildersleeve, YW Jung, **NV Cozzi**. Transport of [¹¹C]meta-hydroxyephedrine, [¹¹C]-epinephrine, and biogenic amines by the human norepinephrine transporter. *J. Nucl. Med.*, 45, 216P (2004)

NV Cozzi, KF Foley. Stimulant and antidepressant-like effects of novel arylketones. *Soc. Neurosci. Abs.*, 29, 757.11 (2003)

NV Cozzi, KF Foley. Methcathinone is a substrate for the serotonin uptake transporter. *Soc. Neurosci. Abs.*, 27, 814.10 (2001)

KF Foley, ME Van Dort, **NV Cozzi**. Stereochemical effects of *meta*-hydroxyephedrine isomers at monoamine uptake transporters: a structure-activity study. *Soc. Neurosci. Abs.*, 26, 819.20 (2000)

NV Cozzi, KF Foley. Bromine substitution at the ring 3- or 4-position of methcathinone enhances potency at serotonin uptake transporters. *Soc. Neurosci. Abs.*, 25, 678.17 (1999)

NV Cozzi, MK Sievert, AT Shulgin, P Jacob III, AE Ruoho. Methcathinone and 2-methylamino-1-(3,4-methylenedioxyphenyl)propan-1-one (methylon) selectively inhibit plasma membrane catecholamine reuptake transporters. *Soc. Neurosci. Abs.*, 24, 341.8 (1998)

NV Cozzi, AT Shulgin, AE Ruoho. Methcathinone (MCAT) and 2-methylamino-1-(3,4-methylenedioxyphenyl)propan-1-one (MDMCAT) inhibit [³H]serotonin uptake into human platelets. *Amer. Chem. Soc. Div. Med. Chem. Abs.*, 215, 152 (1998)

A Boileau, **NV Cozzi**, P Chen, C Czajkowski. Identification of GABA- and diazepam-responsive regions of the GABA_A receptor using γ 2/ α 1 subunit chimeras. *Soc. Neurosci. Abs.*, 22, 509.3 (1996)

MH Weiler, D Katz, H.-J Lee, **NV Cozzi**, K Das Gupta. Inositol phosphate (IP) accumulation in rat neostriatal slices under conditions used to monitor neurotransmitter release. *Soc. Neurosci. Abs.*, 17, 173.9 (1991)

Invited Reviews and Other Publications

Book review: *Psychedelic Medicine: New Evidence for Hallucinogens as Treatments*. TB Roberts, MJ Winkelman, Eds. Praeger/Greenwood, Westport, CT (2007). ISBN: 0-275-99023-0

Contributing Editor: *Psychedelics in Alterations of Consciousness: An Empirical Analysis for Social Scientists*, I Baruss. American Psychological Association Books, Washington, DC (2003). ISBN: 1-557-98993-1

NV Cozzi. SB-207266, an orally active 5-HT₄ receptor antagonist for the treatment of irritable bowel syndrome. *Curr. Res. Serotonin*, 3, 115-118 (1998)

Contributing Editor: *Peyote and the Native American Church in Peyote*, N Ross-Flanigan. Enslow Publishers, Inc., New Jersey (1997). ISBN: 0894908510

Contributing Editor: *Toxicity of Ecstasy in Ecstasy Reconsidered*, N Saunders. Turnaround Press, London (1997). ISBN: 0953006506

NV Cozzi. SDZ-HTF-919, a 5-HT₄ receptor partial agonist for the treatment of gastrointestinal motility disorder and irritable bowel syndrome. *Curr. Drugs Serotonin ID Res. Alert, SDZ-HTF-919*, ISSN 1361 6285 (1997)

NV Cozzi. A review of the chemistry and pharmacology of CV-5197, a 5-HT₂ receptor antagonist. *Curr. Drugs Serotonin ID Res. Alert, CV-5197*, ISSN 1361 6285 (1997)

NV Cozzi. Effects of water filtration on marijuana smoke: a literature review. *Multidisc. Assoc. Psychedelic Studies*, 4, (2), 4-6 (1993)

Professional Affiliations

American Chemical Society (Division of Medicinal Chemistry)

American Society for Pharmacology and Experimental Therapeutics (Division of Neuropharmacology)

Society for Neuroscience