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The November Coalition

2 February 2001

U.S. Sentencing Commission
Michael Courlander, Public Affairs Officer
One Columbus Circle, N.E.
Washington, DC. 20002-8002

RE: Comments for the Sentencing Commission on Ecstasy emergency re-sentencing

Dear Mr. Courlander:

Congress has mandated an emergency review of Ecstasy sentencing provisions. The November Coalition is very concerned that the request of Congress, is yet another reactionary response to what congress perceives as an increase in dangerous drug use. Any increase of rates and length of incarceration on the federal level, will probably cause more harm to the enforcement targeted persons, Bureau of prisons staff morale and safety, and to society as a whole, than the use of these substances would ever effect.

We question the so-called emergency nature of this congressional request, there being little time for public comment, wherein physicians, chemists and other experts and professionals could have a true public discussion. Too many times since the war on drugs was declared over 30 years ago, we have seen the prison sentences increase with little input from the public and leaders alike.

In April 2000, Katherine Hawk, the director of the Federal Bureau of prisons testified before the Senate Subcommittee on Criminal Justice. We will include portions of her statement as our part of our comments on Ecstasy emergency re-sentencing:

“Overcrowding in BOP facilities is 34 percent over capacity system wide. At medium and high security facilities overcrowding levels are at even more dangerous proportions, 55 percent at medium security facilities and 51 percent at high security facilities. We must reduce overcrowding at those facilities for the security of staff, inmates, and the surrounding communities. With the resources Congress has already provided, we are making substantial progress with 22 new prisons funded. However, we need to do more . . . over the past 5 years we have had substantial

795 South Cedar • Colville, WA 99114

Phone & Fax (509) 684-1550

decreases in both inmate suicides and inmate misconduct, including assaults. However, such successes cannot be expected to continue in the face of the dramatic population increases and record setting overcrowding we project will occur in the next several years. Without the resources we have requested to bring additional bed space capacity on line, our record of service may be in jeopardy.”

There are over 95 federal penal institutions and contract confinement facilities at present. Increased penalties will require an overburdened prison system to grow more and more unstable.

We urge the Sentencing Commission to return to Congress and share Katherine Hawk’s recent comments once again. Bureau of Prison staff and prisoner safety “cannot be expected to continue in the face of the dramatic population increases and record setting overcrowding that is expect to occur in the next several years.”

The November Coalition is an organization of thousands upon thousands of prisoners, their loved ones and other concerned citizens nationwide. We know firsthand of mounting tension in our prisons—due to overcrowding, only one of the problems prisoners and staff must endure. Add to that tension, sentences that last decades with no hope of earned release, little hope of good outcome as family units unravel under the strain of incarceration and separation from those we love. These are not laws that serve society well. This is not the opinion of our organization—this statement embodies the many conclusions of numerous studies, some which have been sponsored by the federal government.

To recommend harsher penalties when we know that public education is less expensive and holds more promise of resolution than a rush to incarceration, is questionable from the start. We are the world’s leading jailer, certainly America can find better solutions to our social needs.

Prison cells built for one became two man cells last decade. Those one man cells are now being converted to house three men or women. Rooms previously used for education, recreation and rehabilitation are converted into “dorms”, temporary “units” and still the rush to imprison does not abate. Our leaders must find a better path in our society for our citizens—a better path than the dead end of prison. We know that these laws will target young people primarily. The emergency we see, is an overburdened federal prison system, not the use of a particular substance by a particular group of our society’s citizens.

The war on drugs is a failure, and to continue to wage it unchecked as this “emergency review” suggests, is immoral. This is not a time to rush to more imprisonment, but a time to reexamine every aspect of the war on drugs, and that includes the penalties for club drugs. De-incarceration should be our goal, and would serve justice—not the reverse.

Thank you for considering our hurried comments. Had there been more time, an appropriate time given citizens to respond, I am sure that you would have far more comments to consider.

Sincerely,



Nora Callahan
Executive Director

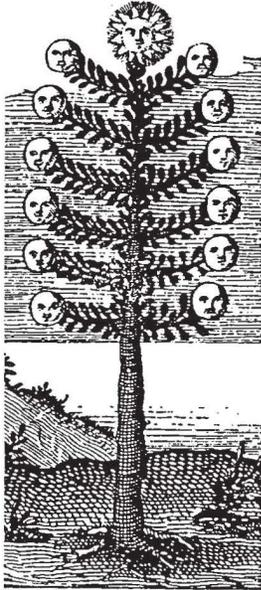
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THE
CENTER FOR COGNITIVE LIBERTY & ETHICS

JOURNAL OF COGNITIVE LIBERTIES

RICHARD GLEN BOIRE, ESQ.
EXECUTIVE DIRECTOR

MONDAY, FEBRUARY 05, 2001



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U.S. Sentencing Commission
Attention Michael Courlander
One Columbus Circle, N.E. Suite 2-500
Washington, D.C. 20002-8002

RE: Emergency Guidelines Amendments Regarding MDMA (Ecstasy)

Dear Commissioners:

The Center for Cognitive Liberty & Ethics submits the following comments and recommendations to the United States Sentencing Commission with respect to the Commission's notice inviting interested persons to submit comments concerning the Commission's proposed amendments to the federal sentencing guidelines. (See, 66(18) *Federal Register* pp. 7961-8017, January, 26, 2001.)

In particular, our comments, and those we hereby submit on behalf of other members of the public, pertain to the Commission's directive to determine the appropriate equivalencies under the sentencing guidelines for "Ecstasy" offenses.

I understand that the Commission's public hearing scheduled for March 19-20 is ostensibly limited to comments concerning the non-emergency amendments, which would preclude comments concerning MDMA ("Ecstasy"). Given that only 10 days were permitted for public comment under the emergency authority, it is my hope, and request, that the Commission will allocate some time at your March public hearing for comments concerning the appropriate punishment for "Ecstasy" offenses.

If you have any questions concerning the attached materials, please do not hesitate to contact me.

Sincerely,

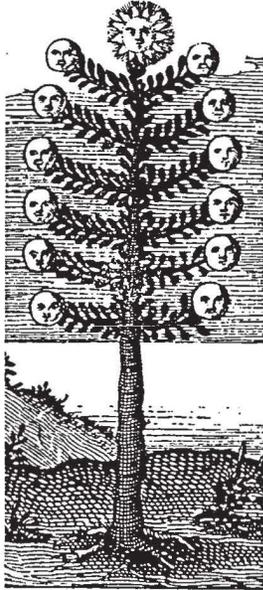
Richard Glen Boire, Esq.
Director, Center for Cognitive Liberty & Ethics

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JOURNAL OF COGNITIVE LIBERTIES

RICHARD GLEN BOIRE, J.D.
EXECUTIVE DIRECTOR

MONDAY, FEBRUARY 05, 2001



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THOMAS SZASZ, M.D.

U.S. Sentencing Commission
Attention Public Information
One Columbus Circle, N.E. Suite 2-500
Washington, D.C. 20002-8002

Comments & Recommendations from
the Center for Cognitive Liberty & Ethics,
Concerning Emergency Amendments Regarding MDMA ("Ecstasy")

Dear U.S. Sentencing Commission:

The Center for Cognitive Liberty & Ethics submits the following comments and recommendations in response to the Commission's invitation for comments concerning its proposed amendments to the federal sentencing guidelines. In particular, our comments and recommendations pertain to the proposed emergency amendments regarding MDMA, MDA, MDEA, and PMA.

1. THE COMMISSION IS NOT OBLIGATED TO INCREASE THE GUIDELINES' PUNISHMENT FOR MDMA, MDA, MDEA, OR PMA.

The United States Sentencing Commission (hereinafter "Commission") is an independent agency in the judicial branch of the government, vested with prescribing the appropriate form and severity of punishment for offenders of federal crimes. (*Mistretta v. United States* (1989) 488 U.S. 361.)

The Center for Cognitive Liberty & Ethics respectfully submits that the Commission is not obligated under section 3664 of Pub. L. 106-310, to *increase* the penalties for "Ecstasy" offenses, but is instead obligated under the Sentencing Reform Act provisions of the Comprehensive Crime Control Act of 1984, to design sentencing guidelines that: (1) incorporate the purposes of sentencing (*i.e.*, just punishment, deterrence, incapacitation, and rehabilitation); (2) provide certainty and fairness in meeting the purposes of sentencing by avoiding unwarranted disparity among offenders with similar characteristics convicted of similar criminal conduct, while permitting sufficient judicial flexibility to take into account relevant aggravating and mitigating factors; and (3) reflect, to the extent practicable, advancement in the knowledge of human behavior as it relates to the criminal justice process.

These three responsibilities, in conjunction with the fact that the Commission is an independent agency in the judicial branch, mandate that the Commission set the appropriate punishment for "Ecstasy" offenses, rather than yield to political pressure or act under an assumed (but not existent) obligation that it must increase the base offense level for "Ecstasy" offenses.

2. The Commission should hold a public hearing on the proposed Amendments regarding "Ecstasy" in order to receive and consider evidence concerning "Ecstasy" – evidence that is necessary to consider in order for the Commission to meet its obligations to set the appropriate penalty under Sentencing Reform Act provisions of the Comprehensive Crime Control Act of 1984.

Section 3662 of Pub. L. 106-310, incorporates Congress' "findings of fact." These summary findings, however, are insufficient for the Commission to determine the appropriate penalty for Ecstasy offenses. In particular, the "findings of fact" in section 3662 fail to acknowledge the findings and recommendations of DEA Administrative Law Judge Francis Young who concluded after a comprehensive hearing on MDMA, that: (1) MDMA has a safe and accepted medical use; and (2), that there was insufficient evidence to conclude that MDMA had a high potential for abuse. Based on these findings, Judge Young ruled that MDMA should be placed in Schedule III, and thus available for doctors to prescribe and use in therapy. (See *In the Matter of MDMA Scheduling*, United States Department of Justice, Drug Enforcement Administration, Docket No. 84-48 (1985-86) [Judge Young's Opinion is available online at: <http://www.mninter.net/~publish/mdma.htm>.]

The Center for Cognitive Liberty & Ethics respectfully submits and recommends that the Commission review and consider Judge Young's opinion, and, further, that the Commission hold public hearings for the purpose of learning more about MDMA, MDA, MDEA, and PMA, before determining the appropriate guidelines penalties for offenses involving these substances.

3. There is no evidence that MDMA use is presenting a significant harm to society.

Although evidence indicates that MDMA use has significantly increased in recent years there appears to be no evidence linking MDMA to disorderly, or violent conduct. Indeed, the evidence is to the contrary. The psychoactive effects of MDMA are almost universally experienced as communitarian, "heart-opening," and empathy eliciting. (See, Beck, J. & Rosenbaum, M., *Pursuit of Ecstasy: The MDMA Experience* (State Univ. of New York Press: NY, 1994.) Although MDMA is widely used (despite the prohibition), often in large gatherings of people, reports of violence or "anti-social" conduct by people who have ingested MDMA are virtually nonexistent.

The Center for Cognitive Liberty & Ethics respectfully submits and recommends that in light of the lack of evidence of public or social harm associated with MDMA use, it would be inappropriate for the Commission to increase the base offense level for MDMA offenses or to otherwise modify the guidelines such that harsher punishment would be imposed for MDMA offenses.

4. The proposed amendment, which would increase the base offense level for MDMA offenses, violates the moral right of adults to control their own mental processes without fear of government punishment.

Increasing the base offense level for MDMA offenses (which would result in lowering the amount of MDMA necessary for a federal prison sentence under the Guidelines), would unjustly and disproportionately punish otherwise law-abiding citizens who possess as little as 5 grams of MDMA, and/or ingest it responsibly without causing harm to others.

The Center for Cognitive Liberty & Ethics submits that increasing the criminal punishment for MDMA offenses violates the fundamental right of responsible adults to control their own consciousness. In the opinion of the Center for Cognitive Liberty & Ethics, any punishment related to MDMA should be strictly limited to punishing *conduct* that harms others, or which poses an immediate harm to others.

For example, in our opinion, using MDMA (or any other substance, including alcohol) to facilitate rape or another criminal offense should be a punished under the guidelines. However, in order to respect the moral rights of adults to autonomy over their own minds and cognitive processes, the Center for Cognitive Liberty & Ethics respectfully submits that any offense that involves merely the possession, manufacture,

importation, exportation, or distribution of MDMA should not be punished—certainly the existing penalties should not be increased.

The Center for Cognitive Liberty & Ethics submits that criminal prosecution and punishment of responsible users of psychoactive plants and chemicals is an ineffectual, immoral, unsophisticated, and socially harmful drug policy. The Center respectfully submits that the U.S. Sentencing Commission, as the body charged with determining the "appropriate punishment" for many of the federal offenses involving psychoactive plants and substances, should take the lead in considering alternatives to the failed "zero-tolerance" Prohibition/Punishment model of drug control.

Respectfully submitted,



Richard Glen Boire, Esq.
Center for Cognitive Liberty & Ethics

ABOUT THE CENTER FOR COGNITIVE LIBERTY & ETHICS

The Center for Cognitive Liberty & Ethics is a nonprofit law and policy center working in the public interest to protect fundamental civil liberties. The Center seeks to foster cognitive liberty – the basic human right to unrestrained independent thinking, including the right to control one's own mental processes and to experience the full spectrum of possible thought.

**THE FOLLOWING PUBLIC COMMENTS CONCERNING THE PROPOSED SENTENCING
AMENDMENT REGARDING MDMA, MDE, MDEA, AND PMA
WERE COLLECTED BY THE CENTER FOR COGNITIVE LIBERTY & ETHICS
AND ARE HEREBY SUBMITTED FOR CONSIDERATION BY THE SENTENCING COMMISSION.**

**THE FOLLOWING COMMENTS ARE THOSE OF THE WRITERS AND DO NOT NECESSARILY
REPRESENT THE OPINIONS OF THE CENTER FOR COGNITIVE LIBERTY & ETHICS**

specmind

From: <Csgrob@aol.com>
To: <rgb@cognitiveliberty.org>; <Gibravo@aol.com>
Sent: Monday, February 05, 2001 1:08 AM
Subject: MDMA Sentencing Commission Letter

February 4, 2001

U.S. Sentencing Commission

Dear Sirs/Madams:

As two psychiatrists who have conducted clinical research and have written extensively about the effects of MDMA (3,4-methylenedioxymethamphetamine), we would like to take this opportunity to express our strong opposition to the proposed new sentencing laws for MDMA.

It has become increasingly evident that raising penalties for MDMA offenses is an unfortunate perpetuation of ineffectual drug war legislation that will ultimately be counterproductive to the government's well-intentioned mission to stem the tide of MDMA use, and will only make a bad situation worse. Most MDMA users are in fact functional citizens, often young adults in our communities, and incarcerating them for longer periods of time would not only fail to be beneficial for society, but would regrettably inflict excessive degrees of punishment and injury to individuals caught within the web of illicit drug war activity, and to their families. We predict that the proposed sentencing laws would only result in targeting low-level dealers and users and not the high volume traffickers for which the laws are intended. Inevitably, the most pronounced consequence would be to push the world's MDMA supply increasingly into the hands of highly organized, unscrupulous, and profit-oriented crime syndicates.

The issue of MDMA has suffered from a persistent pattern of media misinformation. In fact, MDMA's potential for physical and psychological addiction is low. Relevant to the Sentencing Commission's inquiries, MDMA is more equivalent to mescaline in its behavioral and pharmacological effects than it is to heroin. Although there are a small proportion of users who have developed excessive use patterns, they were likely highly vulnerable individuals to begin with who under different circumstances would develop severe problems with other drugs and behaviors. We recognize and deplore the degree to which MDMA abuse does occur, and we readily acknowledge that there are other potentially dangerous adverse effects, particularly when the drug is used under high-risk conditions. However, this proposed change in sentencing will not remedy the situation. Indeed, we predict that the numbers of

[34]

2/5/2001

adverse events as well as fatalities will only increase subsequent to the enactment of the proposed change in sentencing law. Education and harm-reduction programs along with treatment on demand for problematic MDMA users will ultimately serve as far more effective and humane solutions for these problems.

To date, little public attention has been directed to MDMA's potential as a therapeutic medicine. It is our strong contention that MDMA's current placement as a Schedule 1 drug is highly inappropriate. Indeed, in 1986 the DEA's own administrative law judge recommended that the drug be placed in Schedule 3, which is for drugs with putative medical application. Entirely on political grounds, the DEA Director authorized that his own administrative law judge's recommendation be disregarded, and summarily placed MDMA in Schedule 1. It has remained there since, its legal status effectively preventing any approved clinical research from occurring. Well-controlled psychiatric research investigations on MDMA's potential safety and efficacy as an alternative treatment for conditions known to be refractory or non-responsive to conventional treatments, including individuals with end-stage cancer who have severe psychological distress and existential alienation and also for patients with chronic persistent post-traumatic stress disorder, need to be approved and funded.

Great alarm has been expressed about MDMA and its effect on the human brain. Unfortunately, debate has been stifled by the intrusion of a political agenda into the funding and reporting of neuroscience. Pivotal studies attesting to MDMA's "neurotoxic" nature have suffered from a pattern of serious flaws in methodology and problems in data interpretation. To an unfortunate degree, how studies were actually conducted was not honestly represented in formal publication. Ultimately, such practices not only erode scientific credibility, but also obfuscate our understanding of the true range of effects of MDMA.

One unintended result from the proposed sentencing changes will be the psychological, spiritual and material injury inflicted on the families of young people arrested for MDMA crimes. Most victims of these new sentencing guidelines will be young men and women, who will be caught providing friends and acquaintances with the drug. For the vast majority, these will have been activities of low entrepreneurial value.

For the reasons discussed above, we are concerned that the proposal to increase the MDMA sentencing laws will not only fail to reduce criminal activity, but will also lead to compounded degrees of injury to individuals, families and society. There are far more effective and responsible strategies available.

A complete list of our publications on MDMA in the medical literature is appended below.

We are happy to provide any further information or consultation if you so desire.

[35]

Sincerely,

Gary L. Bravo, M.D.
Staff Psychiatrist
Sonoma County Mental Health
3322 Chanate Road
Santa Rosa, CA 95404
(707) 565-4997

Charles S. Grob, M.D.
Director, Division of Child and Adolescent Psychiatry
Harbor-UCLA Medical Center
Professor of Psychiatry
UCLA School of Medicine
Box 498
1000 W. Carson St.
Torrance, CA 90509
(310) 222-3112

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PURDUE UNIVERSITY



SCHOOL OF PHARMACY AND
PHARMACAL SCIENCES

February 5, 2001

Michael Courlander
Public Affairs Officer
U.S. Sentencing Commission

Re: Sentencing guidelines for methylenedioxymethamphetamine (MDMA)

Dear Mr. Courlander:

I received word today that the Commission proposes to equate 1 g of MDMA, MDA, and MDEA to 1 kg of marijuana, rather than 35 g, 50 g and 30 g of marijuana, respectively. I also understand that 1 gram of mescaline is presently equated to 10 grams of marijuana, 1 gram of powder cocaine equated to 200 g of marijuana, and 1 gram of methamphetamine to 2 kg of marijuana.

One basis for this reconsideration is the assertion that "ecstasy..... is similar in its hallucinogenic effect on the user to mescaline." This statement is simply incorrect. Extensive literature published on this subject, some of it from my own laboratory and listed later, clearly shows that MDMA is in no way comparable to mescaline in its effect. MDMA does not act by the same pharmacological mechanism as does mescaline, nor does it have the ability to produce the profound sensory disruptions and hallucinations that are characteristic of mescaline. Similarly, while high doses of mescaline can provoke psychosis in labile individuals, there is no evidence that MDMA has similar potential. Whereas MDMA does have a stimulant effect, it is approximately only one-tenth that of methamphetamine, based on human dosage.

Based on reported human dosages I estimate that MDMA has roughly twice the psychoactive potency by weight of mescaline. Thus, based on human dosage, the equivalency for one gram of MDMA should then be equal to 20 g of marijuana.

MDMA has only about 1/25th (doses: 5 mg heroin vs 125 mg MDMA) the potency of heroin. There is no basis either through potency considerations or through risk assessment to equate the harm of one gram of MDMA with one gram of heroin.

In my professional opinion, based on my own 25 years of research into the action of psychoactive drugs of abuse, and extensive reading of the literature, one gram of MDMA can in no way be equated to one gram of heroin, either based on dosage, or upon the degree of harm that can result from use of these two very different substances. Heroin is highly addictive, and in overdose leads directly to death; MDMA is not. The degree of toxicity of heroin and MDMA is not comparable by any standard. Heroin is used by intravenous injection, accompanied by risks of infection with a variety of microorganisms and viruses, whereas MDMA is taken orally with none of those risks. One gram of pure heroin would be a sufficient quantity to lead to overdose death in perhaps as many as 20 drug-naïve individuals, whereas one gram of MDMA taken orally might be sufficient to cause fatality of one drug-naïve person.

[38]

Whereas I do understand the concern regarding large numbers of adolescents who are apparently abusing ecstasy, and support reasonable attempts to discourage this use, I am adamantly opposed to regulations that are not based on facts or science. If the guidelines are to be arbitrary, and based on political whims, then the sentencing commission should so state, but should not attempt to justify the guidelines by misrepresentation of the facts or the science so as to create the public impression that the dangers of MDMA are actually comparable to those of heroin when they demonstrably are not. That is, *there is absolutely no medical or scientific basis* upon which to support guidelines that would purport to equate one gram of MDMA with one gram of heroin.

Sincerely,



David E. Nichols, Ph.D.
Professor of Medicinal Chemistry
And Molecular Pharmacology

Relevant Scientific Literature Published by My Laboratory.

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CURRICULUM VITAE

Name and Title: David Earl Nichols
Professor of Medicinal Chemistry and
Professor of Pharmacology.

Addresses:**Home**

4740 North 225 West
W. Lafayette, Indiana 47906

Phone: 765-497-2320

Office

Department of Medicinal Chemistry and
Molecular Pharmacology
School of Pharmacy and Pharmacal Sciences
Purdue University
West Lafayette, Indiana 47907
Phone: (765)-494-1461; FAX (765)-494-1414
email: drdave@pharmacy.purdue.edu

Birthplace and Date: December 23, 1944, Covington, Kentucky

Degrees Held: B.S. Chemistry, University of Cincinnati, 1969
Ph.D. Medicinal Chemistry, University of Iowa, 1973
Postdoctoral Fellow, Pharmacology, Univ. of Iowa 1973, 1974

Appointments:

Founder and Co-chair, Scientific Advisory Board, DarPharma, Inc.
Interim Head, Dept. of Medicinal Chemistry and Molecular
Pharmacology, 7/1/96-10/1/96
Interim Head, Depts. of Medicinal Chemistry and Pharmacognosy,
and Pharmacology and Toxicology, 7/1/95-6/30/96
Interim Head, Dept. of Medicinal Chemistry and Pharmacognosy
9/1/94-6/30/95
President, Heffter Research Institute, Inc., 1993-present
Professor of Pharmacology
Purdue University 1985-present
Professor of Medicinal Chemistry,
Purdue University 1984-present
Associate Professor of Medicinal Chemistry,
Purdue University 1979-1984
Assistant Professor of Medicinal Chemistry,
Purdue University 1974-1979
Teaching Assistant, 1972, University of Iowa
NDEA IV Fellow, 1969-1972, University of Iowa

Memberships:

Sigma Xi Scientific Honorary
Phi Lambda Upsilon Chemistry Honorary
Rho Chi Pharmacy Honorary
American Chemical Society
Division of Medicinal Chemistry, ACS
American Association for the Advancement of Science
American Association of Pharmaceutical Scientists
Kappa Psi (Pharmacy fraternity)
Society for Neuroscience
American Society for Pharmacology and Experimental Therapeutics
Purdue University Neuroscience Program
American College of Neuropsychopharmacology, elected to membership 1996

Awards or Honors:

NDEA IV Predoctoral Fellow
Henry Heine Best Teacher Award, School of Pharmacy and Pharmacal Sciences,
Purdue University 1981.
Fellow, Academy of Pharmaceutical Sciences, 1985
Fellow, American Association of Pharmaceutical Scientists
Listed in Who's Who in America, Who's Who in Medicine and Healthcare, and Who's Who in the
Midwest
Keynote speaker, Annual MIKI Graduate Student Meeting, Iowa City, March 30, 1996

Refereed Publications:

1. F. Antun, J.R. Smythies, F. Benington, R.D. Morin, C.F. Barfknecht and D.E. Nichols, "Native Fluorescence and Hallucinogenic Potency of Some Amphetamines", *Experientia* **27**, 62-63 (1971).
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55. C.P. Lawler, V.J. Watts, J.H. Gilmore, S.B. Southerland, J.R. Atashi, H.P. Smith, C.A. Mathis, D.E. Nichols, and R.B. Mailman, "Dihydrexidine displays high potency and full efficacy at D₁ dopamine receptors in primate striatum," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.
56. M.H. Lewis, J.-L. Gariépy, P. Gendreau, M.A. Mayleben, D.E. Nichols, and R.B. Mailman, "D₁ dopamine receptor activation induces social reactivity in mice selectively bred for aggression," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.
57. D.H. Mooney, D.M. Mottola, S.B. Southerland, T.A. Knoerzer, D.E. Nichols, and R.B. Mailman, "Substitutions on the pendent phenyl ring of dihydrexidine alter D₁:D₂ selectivity," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.
58. T.J. Schmitt, S.B. Southerland, R.B. Mailman, D.E. Nichols, and M.H. Lewis, "Intracerebral injections of the full efficacy D₁ agonist dihydrexidine: Behavioral effects in the rat," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.

59. Q.D. Walker, M.H. Lewis, A.M. Perkins, D.A. Eckerman, D.E. Nichols, and R.B. Mailman, "Discriminative stimulus properties of the D₁ dopamine agonist, dihydrexidine," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.
60. V.J. Watts, D.M. Mottola, O. Civelli, R.A. Johnson, D.E. Nichols, and R.B. Mailman, "Dihydrexidine binds differently to human clonal and rat striatal D₁ dopamine receptors," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.
61. N.F. Nichols, P.J.K.D. Schreur, M.W. Smith, W.E. Hoffmann, D.E. Nichols, and M.F. Piercey, "Activation of postsynaptic but not presynaptic dopamine receptors by dihydrexidine, a potent D₁ and D₂ receptor ligand," 22nd Annual Meeting of the Society for Neuroscience, Anaheim, CA, October 1992.
62. D. Marona-Lewicka, D.E. Nichols, X. Huang, and M.P. Johnson, "Novel non-neurotoxic and selective serotonin releasing agents," XI Congress of the Polish Pharmacological Society, Gdansk, September 1992. *Polish J. Pharmacology and Pharmacy* (Supplement) 44:178 (1992).
63. X. Huang and D.E. Nichols, "Further Studies on 3,4-Methylenedioxyamphetamine (MDMA)-induced Serotonergic Neurotoxicity," Eleventh International Neurotoxicity Conference, Little Rock, Arkansas, September 26-30, 1993.
64. H.P. Smith, C.P. Lawler, A.M. Eaton, D.E. Nichols, and R.B. Mailman, "Vacuous chewing induced by N-n-propyl dihydrexidine: a D₂-like (D₃) selective dopamine agonist," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
65. J.H. Gilmore, V.J. Watts, C.P. Lawler, E.P. Noll, D.E. Nichols, and R.B. Mailman, "Efficacy of D₁ agonists in human caudate: relationship to parkinsonism," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
66. M.A. Mayleben, V.J. Watts, C.P. Lawler, D.K. Grandy, O. Civelli, D.E. Nichols, and R.B. Mailman, "Benzo[a]phenanthridines: novel ligands to study D₃ dopamine receptors," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
67. M.H. Lewis, L.L. Cook, S. Whatley, D.E. Nichols, and R.B. Mailman, "Activation of D₁ dopamine receptors in nucleus accumbens potentiates acoustic startle," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
68. Q.D. Walker, D.M. Black, D.A. Eckerman, D.E. Nichols, and R.B. Mailman, "Behavioral and pharmacological determinants of D₁ dopamine agonist substitution for the discriminative stimulus induced by dihydrexidine," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
69. R.B. Mailman, V.J. Watts, C.P. Lawler, Q.-Y. Zhou, O. Civelli, T. Knoerzer, and D.E. Nichols, "Benzo[a]phenanthridines: structurally rigid molecules to examine D₁ dopamine receptor activation," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
70. V.J. Watts, C.P. Lawler, A.J. Gonzales, Q.-Y. Zhou, O. Civelli, D.E. Nichols, and R.B. Mailman, "Efficacy of D₁ dopamine receptor agonists: the role of spare receptors," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
71. B.A. Heidenreich, F. Rehman, D.E. Nichols, R.B. Mailman, and T.C. Napier, "Partial and full dopamine D₁ agonists produce comparable excitations among ventral pallidal neurons," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
72. X. Huang and D.E. Nichols, "5-HT₂ Receptor-mediated potentiation of dopamine synthesis and central serotonergic deficits," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
73. D. Marona-Lewicka, R. Oberlender, and D.E. Nichols, "The effects of alpha₂-adrenoceptor agonists and antagonists on the stimulus properties of LSD," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.

74. R. Oberlender and D.E. Nichols, "Dextrofenfluramine Drug Discrimination," 23rd Annual Meeting of the Society for Neuroscience, Washington, D.C. November 1993.
75. K. Negash and D.E. Nichols, "Synthesis of some rigid analogues of β -phenyldopamine to evaluate dopamine receptor topography," 207th ACS National Meeting, San Diego, March 1994.
76. S. Vanveravong and D.E. Nichols, "Stereoselective synthesis of trans-2-(indol-3-yl)cyclopropylamines: rigid tryptamine analogues," 207th ACS National Meeting, San Diego, March 1994.
77. D.E. Nichols, "Hallucinogen Research in the decade of the brain: does medicinal chemistry tell us anything?" XIXth C.I.N.P. Congress, Washington, D.C. June 1994.
78. D.E. Nichols, A. Monte, X.-M. Huang, and D. Marona-Lewicka, "Stereoselective pharmacological effects of lysergic acid amides possessing chirality in the amide substituents," Third IUPHAR Satellite Meeting on Serotonin, Chicago, IL July 30-Aug 3, 1994.
79. D. Marona-Lewicka, J.E. Sprague, and D.E. Nichols, "Conditioned place preference effect of 5-methoxy-6-methyl-2-aminoindan (MMAI): A comparison study with entactogens and fenfluramine," 24th Annual Meeting of the Society for Neuroscience, Miami Beach, FL. November 1994.
80. B.L. Wiens, D.E. Nichols, R.B. Mailman, and K.A. Neve, "Multiple structural determinants of agonist efficacy at dopamine D2 receptors," 24th Annual Meeting of the Society for Neuroscience, Miami Beach, FL. November 1994.
81. V.J. Watts, C.P. Lawler, K.A. Neve, D.E. Nichols, and R.B. Mailman, "Binding and functional agonist properties of LSD and N(6)-alkyl analogs at D₁ dopamine receptors. 24th Annual Meeting of the Society for Neuroscience, Miami Beach, FL November 1994.
82. D.M. Black, C.P. Lawler, Q.D. Walker, V.J. Watts, D.E. Nichols and R.B. Mailman, "D₁ Receptors, cAMP synthesis, and the amphetamine discriminative stimulus," 24th Annual Meeting of the Society for Neuroscience, Miami Beach, FL November 1994.
83. J.K. Kilts, V.J. Watts, D.E. Nichols, K.A. Neve, and R.B. Mailman, "Inhibition of isoproterenol-stimulated cAMP accumulation in D_{2L} C-6 glioma cells by hexahydrobenzo[a]-phenanthridines," 24th Annual Meeting of the Society for Neuroscience, Miami Beach, FL November 1994.
84. M.A. Mayleben, V.J. Watts, C.P. Lawler, D.E. Nichols, K.A. Neve, and R.B. Mailman, "Desensitization of the D₁ dopamine receptor expressed in C-6 glioma cells parallels agonist intrinsic activity," 24th Annual Meeting of the Society for Neuroscience, Miami Beach, FL November 1994.
85. Q.D. Walker, D.M. Black, D.A. Eckerman, D.E. Nichols, and R.B. Mailman, "Potentiation of the dihydroxidine discriminative stimulus by phosphodiesterase inhibition and the effect of receptor reserve on D₁ agonist substitution," 24th Annual meeting of the Society for Neuroscience, Miami Beach, FL November 1994.
86. D. Ghosh, D.E. Nichols, V.J. Watts, and R.B. Mailman, "Synthesis and evaluation of novel 8-benzyl-2-aminotetralins as dopamine receptor (D1 and D2) probes," 210th National Meeting of the American Chemical Society, Chicago, IL, August 1995.
87. J.E. Sprague, S.T. Wasti, and D.E. Nichols, "Differential effects of MAOIs on [³H]paroxetine binding in the striatum, hippocampus, and frontal cortex following treatment with MDMA or PCA," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.
88. D. Marona-Lewicka and D.E. Nichols, "Drug discrimination studies of the interoceptive cues produced by selective serotonin uptake inhibitors and selective serotonin releasing agents," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.

89. M.A. Mayleben, A. Monte, W. Roeske, H. Yamamura, R.B. Mailman, and D.E. Nichols, "Methoxyphenethylamine-ergoline "hybrids": novel muscarinic ligands," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.
90. D.E. Nichols, V.J. Watts, C.P. Lawler, D. Ghosh, C. Striplin, and R.B. Mailman, "Dinapsoline: a novel full D1 agonist," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.
91. J.D. Kilts, M.A. Mayleben, D.E. Nichols, K.L. O'Malley, R.D. Todd, K. Suzuki, C.P. Lawler, and R.B. Mailman, "Binding and functional characterization of dihydrexidine and other hexahydrobenzo[a]phenanthridines in D₂- and D₄-transfected MN9D cells," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.
92. H.P. Smith, G.S. Oxford, D.E. Nichols, R.B. Mailman, and C.P. Lawler, "Dihydrexidine demonstrates functional selectivity at D2 dopamine receptor-mediated processes," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.
93. M.W. Lewis, D.E. Nichols, M.A. Ritter, J.S. Verbanac, and R.L. Commissaris, "Effects of acute treatment with indirect-acting serotonergic agonists on anxiety-like (conflict) behavior in maudsley rats," 25th Annual Meeting of the Society for Neuroscience, San Diego, CA, November 1995.
94. J.D. Kilts, C.P. Lawler, D.E. Nichols, K.L. O'Malley, R.d. Todd, and R.B. Mailman, "Functional selectivity: dihydrexidine, a D₂ agonist, acts as an antagonist at dopamine release-modulating D₂ receptors," 26th Annual Meeting of the Society for Neuroscience, November 1996.
95. H.P. Smith, G.S. Oxford, D.E. Nichols, R.B. Mailman, and C.P. Lawler, "Dihydrexidine activates D₂ dopamine receptors coupled to adenylyl cyclase, but not to potassium ion channels," 26th Annual Meeting of the Society for Neuroscience, November 1996.
96. M. Mayleben, C.D. Striplin, C.P. Lawler, S. Kongsamut, D.E. Nichols, and R.B. Mailman, "Differential responses in the rat after chronic administration of D₁ dopamine agonists having different intrinsic activity," 26th Annual Meeting of the Society for Neuroscience, November 1996.
97. P.R. Rau, R.B. Mailman, D.E. Nichols, and C.P. Lawler, "Dopamine D₁ receptor agonist-induced grooming in rats: an examination of the link to adenylylase," 26th Annual Meeting of the Society for Neuroscience, November 1996.
98. D. Marona-Lewicka and D.E. Nichols, "The effect of selective serotonin releasing agents in the chronic mild stress model of depression in rats," 26th Annual Meeting of the Society for Neuroscience, November 1996.
99. Ghosh, D., D.E. Nichols, C.R. Peck, and R.B. Mailman. 8-(Bromomethyl)benzyl-6,7-dihydroxy-2-aminotetralin derivatives as potential dopamine D2-like receptor alkylating agents. Amer. Chem. Soc. Abstracts 213, MEDI 190, 1997.
100. Andersson C., M.M. Lewis, C.R. Peck, D. Ghosh, D.E. Nichols, and R.B. Mailman. Dopamine-receptor inactivation by a novel D₂-like selective irreversible ligand. Soc. Neurosci. Abstr. 23: 1997.
101. Kilts J.D., D.E. Nichols, R.B. Mailman, and C.P. Lawler. The functionally selective agonist dihydrexidine inhibits adenylylase in rat striatum via the D₂ dopamine receptor. Soc. Neurosci. Abstr. 23: 1997.
102. Lewis M.M., C.P. Lawler, D.E. Nichols and R.B. Mailman. Drug-induced desensitization of the D_{1A} dopamine receptor dissociates from receptor occupancy and functional efficacy. Soc. Neurosci. Abstr. 23: 1997.
103. Sloan J.L., M.K.-H. Doll, C. Prioleau, C.P. Lawler, D.E. Nichols, R.B. Mailman. Characterization of novel quinpirole and quinolorane derivatives as D₁ ligands. Soc. Neurosci. Abstr. 23: 1997.

104. Blake B.L., J.L. Sloan, C.D. Striplin, M.M. Rasenick, D.E. Nichols, R.B. Mailman, and C.P. Lawler. Activation of G-proteins by full and partial D₂ dopamine receptor agonists in rat striatum. Soc. Neurosci. Abstr. 23: 1997.
105. Hoffman BT, JM Wilson, DE Nichols, SD Wyrick, and RB Mailman. A dibenzosuberane backbone in dopaminergic ligands may mediate selectivity for the D₁ vs. D₅ dopamine receptor. Soc. Neurosci. Abstr. 23: 1997.
106. Marona-Lewicka, D., J. Blair, D. M. Kurrasch, and D.E. Nichols. The effect of ring fluorination on the pharmacology of hallucinogenic tryptamines. Soc. Neurosci. Abstr. 23: 1997.
107. Marona-Lewicka, D. and D.E. Nichols. Structural requirements for amphetamine derivatives with high selectivity to release serotonin versus other neurotransmitters. Soc. Neurosci. Abstr. 24, Abs. 439.1, 1998.
108. Marona-Lewicka, D. M. Kowalska, I. Nalepa, J. Vetulani, and D.E. Nichols. Chronic treatment with the selective 5-HT releaser MMAI induced adaptive changes in rat brain. A comparative study with an SSRI. 2nd European Congress of Pharmacology, PT100, Budapest, July 3-7, 1999.
109. Marona-Lewicka, D. and D.E. Nichols. New therapeutic potentials of the potent and selective 5-HT releasing agent: 5-methoxy-6-methyl-2-aminoindan. 2nd European Congress of Pharmacology, PT101, Budapest, July 3-7, 1999.
110. E.G. Arrington, B.L. Blake, D.E. Nichols, R.B. Mailman and C.P. Lawler, "Functional selectivity of dihydrexidine at dopamine D₂ receptors is not predicted by binding to the high affinity receptor state or stimulation of GTP \square S binding." Society for Neuroscience, Miami, FL, October 1999.
111. M.M. Lewis, B. Hoffman, L.G. Henage, D.W. Miller, R.A. Nicholas, D.E. Nichols, A. Tropsha and R.B. Mailman, "Mutation of the D_{1A} dopamine (DA) receptor reveals specific residues involved in agonist recognition and receptor activation." Society for Neuroscience, Miami, FL, October 1999.
112. S.L. Oxendine, A.M. Qandil, D.E. Nichols and R.B. Mailman, "Dinapsoline (DNS) and its analogs: further insight into the pharmacophores for the D₁ and D₂ dopamine receptors." Society for Neuroscience, Miami, FL, October 1999.
113. J.P. Ryman, C.P. Lawler, D.E. Nichols and R.B. Mailman, "Tolerance induced by infusion of dopamine D₁ receptor full agonists in dopamine-denervated rats." Society for Neuroscience, Miami, FL, October 1999.
114. J.E. Sprague, T. Worst, A. Kanthasamy, D.E. Nichols and M.D. Kane, "Effects of antisense oligonucleotide alteration of MAO-B gene expression on MDMA-induced serotonergic neurotoxicity." Society for Neuroscience, Miami, FL, October 1999.
115. D. Marona-Lewicka, J.R. Selken and D.E. Nichols, "Effects of combined treatment with *d*-amphetamine and the selective 5-HT releasing agent, MMAI on behavior, thermoregulation, and body weight." Society for Neuroscience, Miami, FL, October 1999.
116. D.L. Roman, J.A. Nichols, D.R. Green, G.J. Rodrigues, F. Rahkshan, D.E. Nichols and E.L. Barker, "Cross-species chimeras identify domains involved with molecular recognition of amphetamines at the serotonin transporter." Society for Neuroscience, Miami, FL, October 1999.
117. D.M. Kurrasch-Orbaugh, J.J. Chambers, M. Parker and D.E. Nichols, "Effect of methoxy group orientation on the pharmacology of novel serotonin 2A and 2C receptor agonists." Society for Neuroscience, Miami, FL, October 1999.
118. A. Kanthasamy and D.E. Nichols, "The GABA transaminase inhibitor gamma-vinyl GABA attenuates MDMA-induced neurotoxicity in rats." Society for Neuroscience, Miami, FL, October 1999.

Invited Seminars:

- "Probing the Hallucinogen Receptor", Indiana University School of Medicine, Northwest Cancer for Medical Education, Gary, IN, March 24, 1977.
- "Progress in Understanding Hallucinogens", Center in Toxicology, Vanderbilt University School of Medicine, Nashville, TN, July 14, 1978.
- "Chemical Probes of Hallucinogen Receptors", Dept. of Biological Sciences, Chicago State University, Chicago, IL, October 16, 1980.
- "Chemical Probes of the Hallucinogen Receptor(s)", Dept. of Chemistry, IUPUI, Indianapolis, IN, January 12, 1981.
- "Structure-Activity Relationships of Hallucinogens", Dept. of Pharmaceutical Chemistry, Univ. California (S.F.) October 23, 1981.
- "Structure-Activity Relationships of Hallucinogens", Dept. of Chemistry, Indiana State University, Terre Haute, IN, March 15, 1982.
- "The Development of Novel Dopamine Agonists", Smith, Kline and French, Philadelphia, PA, August 23, 1982.
- "Chemistry and Structure-Activity Relationships of Hallucinogenic Drugs", Wabash College, Dept. of Chemistry, Crawfordsville, IN, January 24, 1984.
- "Structure-Activity Relationships of Hallucinogenic Drugs", University of Toledo, College of Pharmacy, Toledo, OH, May 11, 1984.
- "Structure-Activity Relationships of Hallucinogenic Drugs," Astra Pharmaceuticals, August 13, 1984.
- "Hallucinogens", Department of Pharmacology, University of Toronto, Toronto, Canada, August 16, 1984.
- "Use of Hallucinogen Analogs to Study Serotonin Receptors", Eli Lilly and Co., Indianapolis, IN, November 19, 1984.
- "Research and Social Impact of LSD", 360 House, Sponsored by Alcohol Issues Committee, Purdue University, West Lafayette, IN, November 20, 1984.
- "Principles of Medicinal Chemistry", Associated Colleges of the Chicago Area, Fall Short Course 1986, 2 Hr. Lecture, September 16, 1986.
- "Hallucinogens: A Medicinal Chemist's Approach", Annual Purdue University Neuroscience Retreat, May 15, 1987.
- "Studies Directed Toward the Development of Selective Dopamine D-1 Agonists and Antagonists," Abbott Laboratories, July 16, 1987.
- "Medicinal Chemistry Approaches to the Study of Psychedelics," Donner Laboratory, Berkeley, CA, August 7, 1987.
- "Hallucinogens and Serotonin Receptors: 14 Years Later," College of Pharmacy, University of Iowa, October 28, 1987.
- "Hallucinogens: Pandora's Box or Technology of the Next Century," *New York Academy of Sciences Seminar Series - Designer Drugs: Problems and Promises*, New York, March 14, 1988.
- "Structure-Activity Studies of MDMA-Like Substances," Dept. of Neuroscience, The Johns Hopkins University, School of Medicine, May 5, 1988.