NIDA
NATIONAL INSTITUTE
ON DRUG ABUSE

MDMA/Ecstasy Research:
Advances, Challenges, Future Directions

A SCIENTIFIC CONFERENCE

July 19-20, 2001
Natcher Auditorium
NIH Campus
Bethesda, Maryland

Sponsored by the
National Institute on Drug Abuse
National Institutes of Health

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service

National Institutes of Health
National Institute on Drug Abuse
Bethesda, Maryland 20892

July 19, 2001

Dear Colleagues:

On behalf of the National Institute on Drug Abuse (NIDA), I am pleased to welcome you to *MDMA/Ecstasy Research: Advances, Challenges, Future Directions.* A number of our best monitoring mechanisms are detecting alarming increases in the popularity of MDMA, particularly among today's youth. Unfortunately, myths abound about both the acute effects and long-term consequences of this drug. In response, we have convened a national and international cadre of experts to examine the latest scientific findings on MDMA and to identify areas requiring additional research. Specifically, this meeting will address patterns and trends of MDMA abuse, acute effects, long-term toxicity, and funtional consequences.

In addition to the plenary presentations conducted by some of the world's leading scientists, we have invited and equally impressive number of investigators to display poster presentations of their current research on MDMA. Please take some time during the meeting to visit the research poster gallery on display in the conference center and discuss these ongoing studies with the researchers.

Drug abuse research should not only be useful, it should be used. I hope that this conference provides researchers, practitioners, and policy makers with the scientific information needed to mount a comprehensive response to the individual and public health impact of MDMA abuse.

Sincerely,

Alan I. Leshner, Ph.D.
Director

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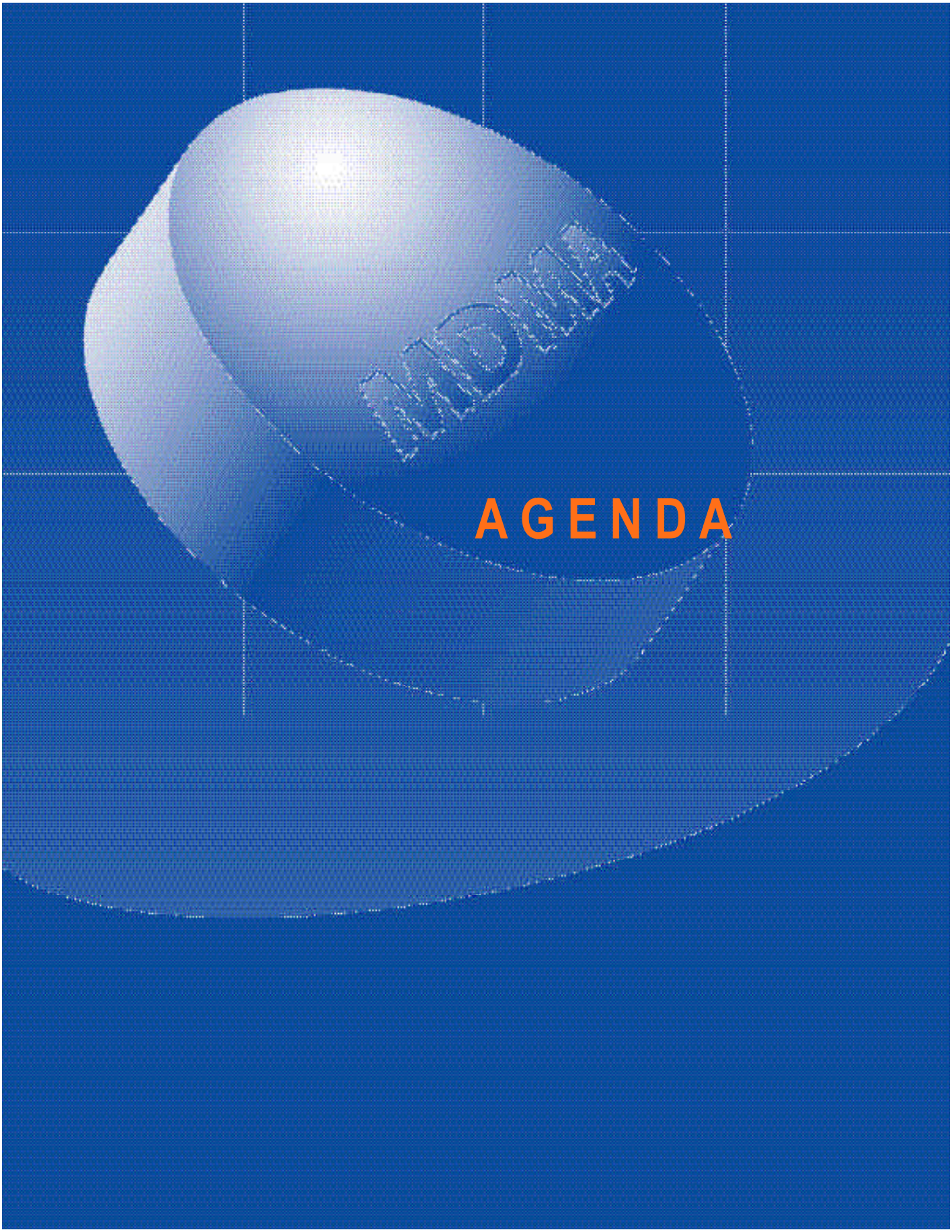

AGENDA

Thursday, July 19, 2001

8:30 a.m. - 8:45 a.m.

Welcome and Meeting Overview

Glen R. Hanson, D.D.S., Ph.D.
Director
Division of Neuroscience and Behavioral Research
National Institute on Drug Abuse

Alan I. Leshner, Ph.D.
Director
National Institute on Drug Abuse

8:45 a.m. - 9:00 a.m.

Keynote Addresses

Ruth L. Kirschstein, M.D.
Acting Director
National Institutes of Health

The Honorable Tommy G. Thompson
Secretary of Health and Human Services (invited)

SESSION I

Patterns and Trends of MDMA Abuse: Implications for Prevention

9:00 a.m. - 9:15 a.m.

Moderator: *Jacques L. Normand, Ph.D.*
Acting Chief
Epidemiology Research Branch
National Institute on Drug Abuse

9:15 a.m. - 9:35 a.m.

Public Health Perspective on MDMA
James N. Hall, B.A.
Executive Director
Up Front Drug Information Center
Miami, Florida

9:35 a.m. - 9:55 a.m.

Patterns of MDMA Use Among Men Who Have Sex With Men in Boston and New York City
Patricia Case, Sc.D.
Director
Program in Urban Health
Department of Social Medicine
Harvard Medical School
Boston, Massachusetts

9:55 a.m. - 10:15 a.m.

Patterns and Trends of MDMA Use and Sexual Risk Behavior in Central Ohio
Robert G. Carlson, Ph.D.
Professor
Center for Interventions, Treatment, and Addictions Research
Wright State University School of Medicine
Dayton, Ohio

10:15 a.m. - 10:40 a.m. **BREAK**

10:40 a.m. - 11:00 a.m. **The Diffusion of MDMA Use Among Urban Youth in Hartford, Connecticut; Implications for Drug and HIV Prevention in Club Drug Users and Their Networks**
Jean J. Schensul, Ph.D.
Executive Director
Institute for Community Research
Hartford, Connecticut

11:00 a.m. - 11:30 a.m. **A Burning Candle: Challenges in Ecstasy Research**
Claire E. Sterk, Ph.D., Discussant
Professor
Department of Behavioral Sciences and Health Education
Rollins School of Public Health
Emory University
Atlanta, Georgia

11:30 a.m. - 12 noon **Question and Answer Panel**
Moderator: Jacques L. Normand, Ph.D.
Acting Chief
Epidemiology Research Branch
National Institute on Drug Abuse

12 noon - 1:15 p.m. **LUNCH** (on your own)

SESSION II **Acute Effects**

1:15 p.m. - 1:30 p.m. *Moderator: Jean Lud Cadet, M.D.*
Chief
Molecular Neuropsychiatry Section
Clinical Director
Intramural Research Program
National Institute on Drug Abuse

1:30 p.m. - 2:00 p.m. **Neurochemical Mediators and Neurophysiological Consequences of Acute MDMA Exposure in Rats**
Bryan K. Yamamoto, Ph.D.
Professor of Psychiatry and Neurosciences
Director
Program in Basic and Clinical Neuroscience
University Hospitals of Cleveland and Case Western Reserve University
Cleveland, Ohio

2:00 pm. - 2:30 p.m. **The Acute Cardiovascular, Endocrine, and Pharmacokinetic Effects of MDMA in Humans**
John Mendelson, M.D.
Associate Clinical Professor of Psychiatry and Medicine
Drug Dependence Research Center
Langley Porter Psychiatric Institute
University of California at San Francisco
San Francisco, California

2:30 p.m. - 3:00 p.m.

Subjective, Reinforcing, and Discriminative Stimulus Effects of MDMA in Humans
Manuel E. Tancer, M.D.
Associate Professor
Departments of Psychiatry and Behavioral Neurosciences, and Pharmacology
Wayne State University School of Medicine
Detroit, Michigan

3:00 p.m. - 3:30 p.m.

BREAK

3:30 p.m. - 4:00 p.m.

"Ecstasy" Deaths - More Than Just MDMA: An Australian Perspective
Rodney J. Irvine, Ph.D.
Research Fellow
Department of Clinical and Experimental Pharmacology
University of Adelaide
Adelaide, Australia

4:00 p.m. - 4:30 p.m.

Acute Effects of MDMA: What Don't We Know?
Gantt P. Galloway, Pharm.D, Discussant
Chief of Pharmacological Research
Haight Ashbury Free Clinics, Inc.
San Francisco, California

4:30 p.m. - 5:00 p.m.

Question and Answer Panel
Moderator: Jean Lud Cadet, M.D.
Chief
Molecular Neuropsychiatry Section
Clinical Director
Intramural Research Program
National Institute on Drug Abuse

5:00 p.m. - 6:30 p.m.

POSTER PRESENTATIONS

Friday, July 20, 2001

SESSION III

Long-Term Toxicology

8:30 a.m. - 8:45 a.m.

Moderator: Steven Grant, Ph.D.
Program Administrator
Clinical Neurobiology Branch
National Institute on Drug Abuse

8:45 a.m. - 9:15 a.m.

Overview of MDMA-induced Persistent Neurotoxicity: Preclinical Perspective
Glen R. Hanson, D.D.S., Ph.D.
Director
Division of Neuroscience and Behavioral Research
National Institute on Drug Abuse

9:15 a.m. - 9:45 a.m.

Developmental Effects of MDMA
Charles V. Vorhees, Ph.D.
Professor
Division of Developmental Biology
Children's Hospital Medical Center
Cincinnati, Ohio

9:45 a.m. - 10:15 a.m.

MDMA-Induced Brain Serotonin Neurotoxicity: Preclinical Studies
George A. Ricaurte, M.D., Ph.D.
Associate Professor
Department of Neurology
Johns Hopkins University School of Medicine
Baltimore, Maryland

10:15 a.m. - 10:45 a.m.

BREAK

10:45 a.m. - 11:15 a.m.

Neuroimaging Studies in Chronic Effects of MDMA/Ecstasy Use
Linda Chang, M.D.
Scientist and Chair
Medical Department
Brookhaven National Laboratory
Upton, New York

11:15 a.m. - 11:45 a.m.

Discussion, and Degeneration in Brain Following Binge Stimulants: All Dopaminergics Induce Degeneration in Fasciculus Retroflexus, but MDMA Also Induces Degeneration in Oral Pontine Serotonin Terminals
Gaylord D. Ellison, Ph.D., Discussant
Professor of Psychology and Neuroscience
University of California at Los Angeles
Los Angeles, California

11:45 a.m. - 12:15 p.m.

Question and Answer Panel
Moderator: *Steven Grant, Ph.D.*
Program Administrator
Clinical Neurobiology Branch
National Institute on Drug Abuse

12:15 p.m. - 1:30 p.m.

LUNCH

SESSION IV

Long-Term Functional Consequences: Behavioral, Mood, Psychiatric, and Cognitive

1:30 p.m. - 1:45 p.m.

Moderator: *Minda R. Lynch, Ph.D.*
Acting Chief
Behavioral and Cognitive Science Research Branch
National Institute on Drug Abuse

1:45 p.m. - 2:15 p.m.

Neuropsychopathology Associated With MDMA
Andy C. Parrott, Ph.D.
Professor, Department of Psychology
Head of the Recreational Drugs Research Group
University of East London
London, United Kingdom

2:15 p.m. - 2:45 p.m.

MDMA-Induced Brain Serotonin Neurotoxicity: Clinical Studies
Una D. McCann, M.D.
Associate Professor
Behavioral Pharmacology Research Unit
Johns Hopkins School of Medicine
Baltimore, Maryland

2:45 p.m. - 3:15 p.m.

Long-Term Consequences of Ecstasy Use Upon Cognition
Euphrosyne Gouzoulis-Mayfrank, M.D.
Associate Professor
Department of Psychiatry and Psychotherapy
Medical Faculty of the University of Technology
Aachen, Germany

3:15 p.m. - 3:30 p.m.

BREAK

3:30 p.m. - 4:00 p.m.

Are the Psychological Problems Associated With Regular MDMA Use Reversed by Prolonged Abstinence?
Michael John Morgan, Ph.D.
Senior Lecturer
Department of Experimental Psychology
School of Biological Sciences
University of Sussex, Falmer
Brighton, United Kingdom

4:00 p.m. - 4:30 p.m.

Long-Term Functional Consequences of MDMA Abuse
H. Valerie Curran, Ph.D., Discussant
Professor of Psychopharmacology
Sub-Department of Clinical Health Psychology
University College London
London, United Kingdom

4:30 p.m. - 4:55 p.m.

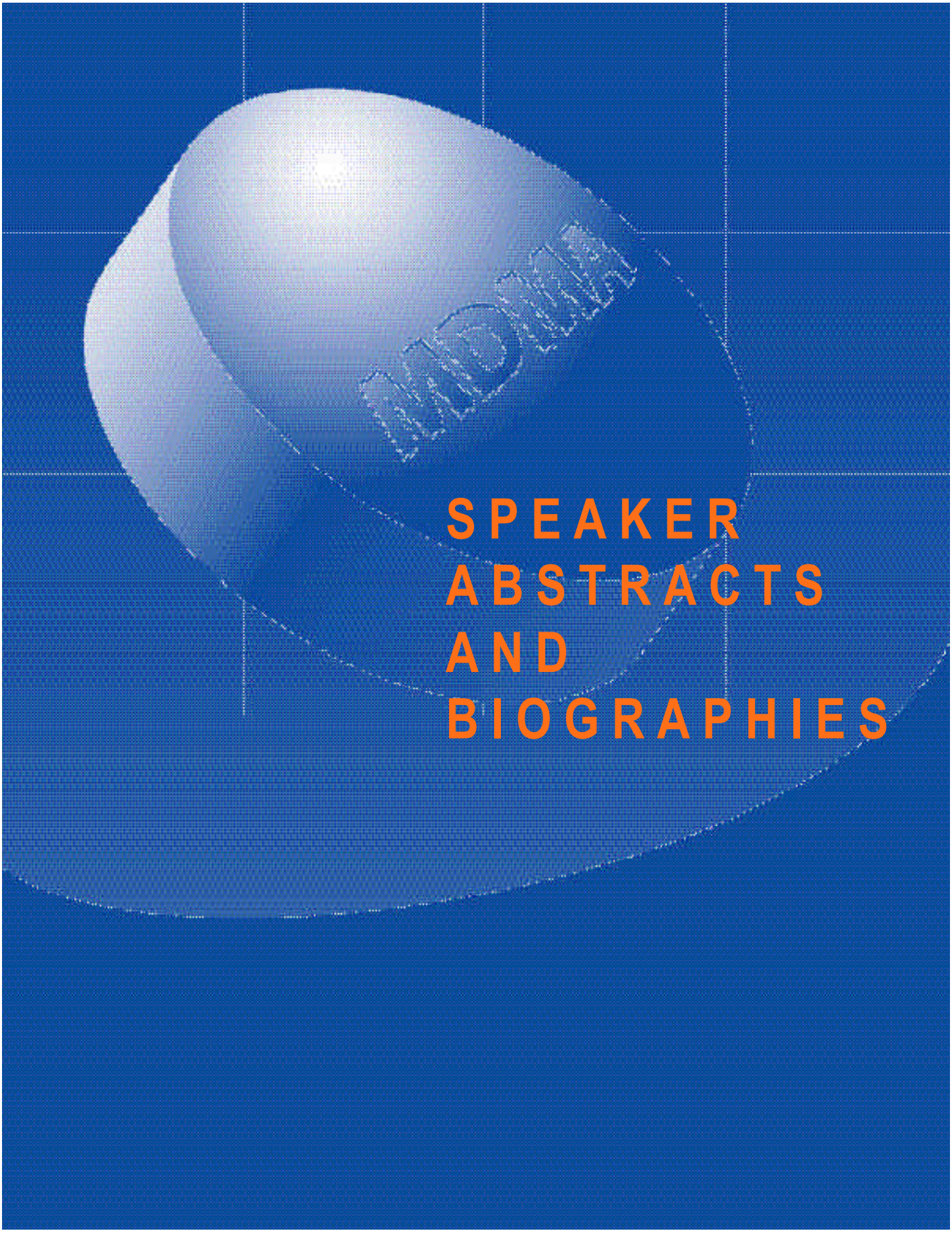
Question and Answer Panel
Moderator: Minda R. Lynch, Ph.D.
Acting Chief
Behavioral and Cognitive Science Research Branch
National Institute on Drug Abuse

4:55 p.m. - 5:00 p.m.

MDMA Research: What Next?
Jerry Frankenheim, Ph.D., Conference Chair
Pharmacologist and Program Official
Pharmacology, Integrative and Cellular Research Branch
National Institute on Drug Abuse

5:00 p.m.

ADJOURNMENT


SPEAKER
ABSTRACTS
AND
BIOGRAPHIES

Jean Lud Cadet, M.D.

Biography

Dr. Cadet graduated from Columbia University, College of Physicians and Surgeons, in 1979. He did a residency in neurology at Mount Sinai and in psychiatry at Columbia University in New York City. He subsequently did a fellowship in neuropsychiatry at the National Institute of Mental Health at the National Institutes of Health (NIH), after which he joined Columbia University as an assistant professor of neurology and psychiatry. He joined the National Institute on Drug Abuse (NIDA) in 1992. He is presently Chief, Molecular Neuropsychiatry Section, and Clinical Director of the NIH/NIDA Intramural Research Program. His research interests include (1) clinical neurobiology of drug abuse and addiction, (2) cellular and molecular neurotoxicology of drug abuse, (3) the involvement of free radicals in neurodegeneration, (4) the role of cell death-related genes in the toxicity of drug abuse, and (5) the participation of catecholamines in neurodegenerative disorders.

Advances

Recent qualitative research on patterns and trends of MDMA use in central Ohio has revealed different user groups, varying patterns of use, and increasingly diverse contexts of use. No longer limited to the "rave scene" or dance clubs, MDMA use has moved to concerts, parks, house parties, and a variety of other venues. MDMA is increasing in popularity among high school and college students as well as other young people not in school. Preliminary findings also indicate that some MDMA users engage in high-risk sexual behaviors while others do not; however, the characteristics of high-risk takers versus others are not clear.

Challenges

Participants emphasized a significant contradiction between messages they hear about the potential negative effects of MDMA use and those they hear about positive experiences of people who have used the drug. Although sporadic horror stories in the media about the negative effects of MDMA use impact some users, most people rationalize that "that won't happen to me." Convincing young people that significant health risks are associated with MDMA use is a major challenge to future prevention efforts.

Future Directions

National MDMA prevention efforts appear to be having limited impact. Because MDMA is often distributed and used in small groups, a peer-driven network approach to prevention may be appropriate. In addition, epidemiologic data on long-term patterns of MDMA and other drug use, sexual risk behavior, and health service needs among different user groups are urgently needed to develop effective prevention strategies.

References

Carlson RG. (2000) Shooting galleries, dope houses, and "doctors": Examining the social ecology of HIV risk behaviors among drug injectors in Dayton, Ohio. *Human Organization* 59(3):325-333.

Carlson RG, Falck RS, Siegal HA. (2000) Crack-cocaine injection in the heartland: An ethnographic perspective. *Medical Anthropology* 18:305-323.

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Biography

Dr. Carlson is a medical anthropologist and professor in the Center for Interventions, Treatment, and Addictions Research, Wright State University School of Medicine, Dayton, Ohio. He is principal investigator on a new study funded by NIDA entitled "MDMA/Club Drug Use and STD/HIV Sex Risk Behavior in Ohio," co-investigator on a natural history study of crack-cocaine users, and Project Administrator for the Ohio Substance Abuse Monitoring Network, a statewide epidemiologic surveillance system. Dr. Carlson received his Ph.D. degree from the University of Illinois at Urbana-Champaign in cultural anthropology and conducted his doctoral research on alcohol use among the Haya of Tanzania. For the past 12 years, he has conducted research on HIV risk behavior and health service use among injection drug and crack-cocaine users in Ohio. His research interests include psychoactive drug use, ethnographic methods, natural history research, AIDS prevention, and political economy. Dr. Carlson is past Chair of the AIDS and Anthropology Research Group and recipient of the 1996 Steven Polgar Award from the Society for Medical Anthropology.

Advances

Preliminary ethnographic findings suggest that, as has been reported for other population groups, men who have sex with men (MSM) in Boston and New York City commonly use MDMA. MDMA use is prevalent among diverse groups of MSM and in multiple contexts: dance parties, bars and clubs, house parties, sex clubs, and other venues in both cities. There are regional variations in the ways that MDMA is acquired and used; in New York City, there are well-established professional dealers and open sales of the drug, but this is less common in Boston. MSM rarely report using MDMA alone; instead, it is used in combination with other drugs as part of a "menu" of drugs selectively administered over a period of time or as an ingredient in mixtures of ketamine, cocaine, methamphetamine, Viagra, or other drugs. MSM report unprotected oral and anal sex in the context of MDMA use; however, unsafe sex is more often attributed to the use of other drugs such as ketamine and methamphetamine.

Challenges

Prevention messages centered on the risks of MDMA have had little effect in reducing the use of MDMA among MSM. An important challenge to prevention efforts is one of competing risks. In both cities, well-publicized overdose deaths from GHB in the MSM community have caused concern and reductions in use among MSM, but there are fewer reported problems associated with MDMA and, as a result, less concern about its use.

Future Directions

Epidemiological, ethnographic, clinical, and animal studies of emergent, complex patterns of MDMA use are needed to understand the health and medical consequences of MDMA use alone and in combination with other drugs. Interventions to reduce and prevent MDMA use should target specific risk groups and take into account the regional variations and complex patterns of MDMA administration.

References

Case P, Beckett GA, Jones TS. (1998) Access to syringes in Maine: Pharmacy practice after the 1993 repeal of the syringe prescription law. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology* 18 Suppl 1:S94-101.

Wolf RC, Case P, Pagano M. (1998) Estimation of the prevalence of injection drug use in greater Boston in 1993. *Journal of Psychoactive Drugs* 30:21-24.

Biography

Dr. Case is a social epidemiologist on the faculty of the Department of Social Medicine at Harvard Medical School. She is the Director of the Program in Urban Health and principal investigator of a NIDA-funded study, "HIV Risk and Club Drugs Among MSM: A Two City Comparison." She also serves as co-investigator of a NIDA-funded study," Social Course of Adherence to HAART in Active Users of Illegal Drugs." Dr. Case received her doctorate from the Harvard School of Public Health. Her research interests include emerging drug use patterns, infectious disease, HIV and drug policy, sexual minorities and patterns of drug use, and social network analysis.

Advances

3,4-Methylenedioxymethamphetamine (MDMA) is an illicit and popular drug that has been associated with serotonergic axonal degeneration in animals. Until recently, much less is known in humans regarding the neurotoxic effects of MDMA. In vivo neuroimaging studies have shown significant decreases in 5-HT transporters (McCann et al., 1998) and abnormalities in 5-HT receptors (Reneman et al., 2000a; Reneman et al., 2000b). Although resting cerebral blood flow (rCBF) (Chang et al., 2000) and brain activation (Gamma et al., 2001) do not differ between ecstasy users and controls, decreases in rCBF may be observed in individuals 2 weeks after MDMA is administered in a controlled setting (Chang et al., 2000). Furthermore, abstinent recreational users showed increased myoinositol, a glial marker, on magnetic resonance spectroscopy (Chang et al., 1999).

Challenges

Many MDMA users are polydrug users who tend to experiment also with hallucinogens, LSD, mushrooms, and cocaine. Therefore, extensive screening procedures are needed in order to recruit "pure" MDMA users; even these pure users may not be truthful about their drug use history. Another challenge is to assess and ensure the accurate duration since the drug was last used and when the imaging studies occurred. These problems are not unique to MDMA users but are common problems in all drug abuse research involving human subjects.

Future Directions

- What are the long-term effects of chronic MDMA abuse on brain function (cognition) sleep, and other physiological parameters and neurochemistry?
- Can these changes recover or improve?
- What, if any, are the interaction effects of other amphetamines with MDMA in terms of brain function and neurochemistry?
- Are there interaction effects with MDMA and HIV?

Studies were supported by NIDA, K-20 DA00280-05, and the GCRC MO1 000425.

References

Chang L, Ernst T, Grob CS, Poland RE. (1999) Cerebral ^{1}H MRS abnormalities in 3,4-methylenedioxy-methamphetamine (MDMA, "ecstasy") users. *Journal of Magnetic Resonance Imaging* 10(4):521-526.

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Gamma A, Buck A, Berthold T, Vollenweider F. (2001) No difference in brain activation during cognitive performance between ecstasy (3,4-methylenedioxymethamphetamine) users and control subjects: A [H2(15)O]-positron emission tomography study. *Journal of Clinical Psychopharmacology* 21(1):66-71.

McCann UD, Szabo Z, Scheffel U, Dannals RF, Ricaurte GA. (1998) Positron emission tomographic evidence of toxic effect of MDMA ("ecstasy") on brain serotonin neurons in human beings. *The Lancet* 352:1433-1437.

Reneman L, Booij J, Schmand B, van den Brink W, Gunning B. (2000a) Memory disturbances in "ecstasy" users are correlated with an altered brain serotonin neurotransmission. *Psychopharmacology* 148(3):322-324.

Reneman L, Habraken J, Majoie C, Booij J, den Heeten G. (2000b) MDMA ("ecstasy") and its association with cerebrovascular accidents: Preliminary findings. *American Journal of Neuroradiology* 21(6):1001-1007.

Biography

Dr. Chang was recently appointed Scientist and Chair of the Medical Department at Brookhaven National Laboratory. She received her M.D. and M.S. degrees in physiology and biophysics from Georgetown University and completed her internship in internal medicine at the University of Southern California and her neurology residency training at the University of California, Los Angeles (UCLA). She also completed two fellowships in electrophysiology and neuroimaging. While at UCLA, she applied physiological and functional MR techniques, as well as SPECT and PET, to evaluate brain injury associated with drug abuse (including cocaine, methamphetamine, and MDMA), HIV dementia, and opportunistic brain lesions in AIDS.

Where We Have Been

How far have we come in understanding the consequences of MDMA abuse on cognition function, mood, and behavior? Different strands of evidence from neurobiological, behavioral, and psychiatric studies with humans differ in the degree to which causal links can be drawn between MDMA use and functional consequences. Although research has looked at the effects of single doses administered to human volunteers, our understanding of the effects of longer term use relies on studies of people self-administering MDMA, and these are fraught with methodological difficulties (Hatzidimitriou et al., 1999). Nevertheless, there are some emerging consistencies. This discussion aims to draw together themes from the afternoon's talks and link these with the morning session on long-term toxicity.

Challenges

One central, practical challenge is overcoming methodological problems including how to verify drug use history, how to allow for multiple drug use, how to establish baseline (pre-morbid) levels of function, and how to determine the time course of effects.

Future Directions

Given that 5-HT neurotoxic effects persist in primates for several years (Curran, 2000), it is important to determine what happens to humans when they stop using MDMA. Some new data on this will be presented. Are there factors that predispose some to experience negative consequences of MDMA more than others? To what extent does this depend on extent of MDMA use, combined use with other drugs, and individual differences? What are the implications for treatment?

References

Curran HV. (2000) Is MDMA neurotoxic in humans? An overview of evidence and methodological problems in research. *Neuropsychobiology* 42:34-41.

Hatzidimitriou, McCann, and Ricaurte. (1999) Altered serotonin innervation patterns in the forebrain of monkeys treated with (+/-)3,4-methylenedioxymethamphetamine seven years previously: Factors influencing abnormal recovery. *J Neuroscience* 191:5096-5107.

Biography

Dr. Curran is Professor of Psychopharmacology in the Psychopharmacology Research Unit, Clinical Health Psychology, at University College London. Her research examines how psychotropic drugs affect memory, mood, and, more recently, the processing of emotional stimuli. Her studies are carried out in various settings with volunteers, psychiatric patients, and drug abusers. Recent research on abuse has included studies of benzodiazepines, ketamine, MDMA, and methadone.

Degeneration in Brain Following Binge Stimulants: All Dopaminergics Induce Degeneration in Fasciculus Retroflexus, but MDMA Also Includes Degeneration in Oral Pontine Serotonin Terminals

Gaylord D. Ellison, Ph.D.

Advances

MDMA has both common and unique properties. It shares, with other amphetamine-like drugs that potentiate dopamine (d-amphetamine, methamphetamine, cathinone), the characteristic of inducing degeneration in axons in fasciculus retroflexus, projecting from lateral habenula ventrally to target cells in SN, VTA, and raphe nuclei. As such, it destroys an important descending negative feedback system from forebrain projections onto the monoaminergic cells that innervate them. Some of these drugs also induce degeneration in other neuropil (for example, the amphetamines on dopamine terminals in caudate), but studies of degeneration also show that MDMA has other, unique long-lasting effects on brain. There is a very pronounced profile of degenerating terminals in ventral pons (presumably serotonergic terminals on the giant oral pontine cells) and also scattered axons in the trigeminal nerve.

Challenges

Why should this drug induce such pronounced degeneration in oral nuclei? This presumably underlies the oral syndromes and aftereffects of prolonged MDMA abuse.

Future Directions

Can other drugs that combine dopamine and serotonergic hyperactivity also induce this distinctive profile?

Biography

Dr. Ellison received his Ph.D. degree from Yale University in 1963 and then went, as the first Postdoctoral Scholar sent by the National Institute of Mental Health behind the “Iron Curtain,” to Warsaw, Poland, where he spent a year working with Jerzi Konorski, one of Pavlov’s most famous students. He then returned to Yale for a year in psychiatry and then moved to the Department of Psychology and the Brain Research Institute at the University of California, Los Angeles. Since that time, he has been the author of over 150 papers, chiefly in the area of psychopharmacology, including models of stimulant and PCP psychosis, tardive dyskinesia, and antidepressants. He first discovered the neurotoxic effects of amphetamines in caudate and the neurotoxic effects of all amphetamine-like substances on fasciculus retroflexus.

Biography

Dr. Frankenheim is a pharmacologist and program official in the Pharmacology, Integrative, and Cellular Neurobiology Research Branch, Division of Neuroscience and Behavioral Research, NIDA. He was a researcher and teacher in pharmacology at Downstate Medical Center (Brooklyn, NY) and the Universities of North Carolina (Chapel Hill), Western Australia (Perth), and Connecticut (Storrs). Just before joining NIDA, he did research at Pennwalt (Rochester, NY) to discover and develop novel antidepressants, anticonvulsants, cerebroprotectives (remacemide), and other drugs for psychiatric and neurologic indications. His interests at NIDA include neurotoxicology and neuro-HIV/AIDS; phencyclidine, ketamine, other glutamate antagonists, and synaptic plasticity, including pharmacologic models of psychosis; LSD-like “hallucinogens”; amphetamines; cannabinoids; and GHB. He chaired the organizing committee for this conference.

Biography

Dr. Galloway is Chief of Pharmacological Research at the Haight Ashbury Free Clinics in San Francisco. His research focuses on the consequences of drug abuse and pharmacotherapy for the treatment of addiction. He was a co-investigator on one of the first National Institute on Drug Abuse-funded sociologic studies of use of MDMA.

What We Know

Ecstasy (3,4-methylenedioxymethamphetamine = MDMA and related congeners) is a group of recreational drugs with neurotoxic effects upon central serotonergic systems in experimental animals. Recent evidence suggests that humans may also be susceptible to the neurotoxicity of ecstasy. So far, the most convincing evidence for long-term alterations of cerebral functions after ecstasy use derives from cognitive studies, which demonstrate relative memory impairment in regular ecstasy users.

Challenges

Most ecstasy users are polydrug users. Their lifestyle (e.g., regular attendance at all-night raves → sleep deprivation) makes it difficult to compare them with "control subjects," who do not belong to the drug and dance scene. In addition, the chemical composition and dosage of ecstasy tablets vary strongly. Therefore, the following question arises: Is the relative memory impairment of ecstasy users due to their ecstasy use? And, if yes: Is it due to the neurotoxic effects of ecstasy upon central serotonergic systems? However, irrespective of these difficulties, recent data do support the view that ecstasy use (possibly in conjunction with the use of other drugs) leads to a decline of memory functions. This relative deficit is likely to be related to the well-recognized neurotoxic potential of ecstasy upon brain serotonergic systems.

Future Directions

Future studies should include followup investigations of people with continued ecstasy use and former users after longer abstinence periods. In addition, morphological and functional neuroimaging studies should add to our understanding of how ecstasy may alter and impair neurocognitive functioning.

References

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Parrott AC. (2000) Human research on MDMA (3,4-methylenedioxymethamphetamine) neurotoxicity: Cognitive and behavioural indices of change. *Neuropsychobiology* 42:17-24.

Biography

Dr. Gouzoulis-Mayfrank is a neurologist and psychiatrist holding a clinical and research position in the Department of Psychiatry and Psychotherapy at the University of Technology in Germany. Her primary areas of scientific and clinical interest are the acute neurobiological effects of hallucinogens, stimulants, and ecstasy in humans, the long-term consequences of ecstasy use in humans, neurocognitive functioning in schizophrenia, and the treatment of dual diagnosis patients (schizophrenia and addiction).

Advances

The evolution of MDMA abuse as a public health issue is tracked over the past quarter century. Applying Golub and Johnson's model of drug abuse epidemics to patterns of MDMA abuse reveals an extended *incubation period* before 1996 among distinct groups. The MDMA epidemic's *expansion phase* has been fueled by dramatic increases in the drug's trafficking and its availability to younger populations. An update on MDMA abuse will include patterns reported at the June 2001 NIDA Community Epidemiology Work Group. A comparison with previous stimulant epidemics offers clues for reversing MDMA abuse.

Challenges and Opportunities

Tracking MDMA abuse reveals the classic pattern of a drug epidemic emerging among hidden populations. This particular epidemic has also introduced the need to identify and track hidden consequences of the drug's abuse. Problems related to MDMA are observed in the abuse of multiple drugs, thus revealing "ecstasy" use as a polydrug abuse phenomenon. The global nature of MDMA trafficking makes this a multinational problem mandating international research networks for its surveillance and prevention.

Future Directions

Technological advances and changes of the post-communist age have fueled the worldwide spread of MDMA abuse, foreshadowing how widespread future drug epidemics could be. Earlier detection, sharing of research findings, and faster responses to emerging epidemics are required for the modern drug abuse era. This conference will expose multiple determinants of MDMA abuse, which are the factors to target for its prevention. Linking supply-side intelligence with demand-side epidemiology assists development of both criminal justice and public health strategies.

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Biography

Mr. Hall has begun his 20th year as Executive Director of Miami's Up Front Drug Information Center. He has represented South Florida on NIDA's Community Epidemiology Work Group since 1985. Mr. Hall was the first to report in the public health literature on the abuse of the medication flunitrazepam (Rohypnol) in the United States. He also organized the Texas-Florida Rohypnol Response Group. Mr. Hall founded the Institute for the Prevention and Prosecution of Drug-Rape. He serves as a consultant to the Federal National Drug Intelligence Center and as Director of Research and Information for The Miami Coalition for a Safe and Drug-Free Community.

Advances

The potential neurotoxic properties of amphetamine-related drugs were suggested by observations in the rat of Gibb and Koda (*JPET* 185 [1973] 42) and Seiden et al. (*Drug Alcoh Depend* 1 [1976] 215) that high-dose methamphetamine (METH) treatment causes persistent deficits in the dopamine (DA) system associated with the basal ganglia. Gibb and Hotchkiss (*JPET* 214 [1980] 257) later reported that similar METH administrations also cause similar long-term declines in the serotonin (5HT) systems associated with the frontal cortex, striatum, and hippocampus. These findings suggested that heavy METH use can be neurotoxic to critical systems in the brain associated with memory, information processing, and motor functions. Because MDMA is a METH analogue, its effects on DA and 5HT systems have also been studied. Moderate-to-high doses of MDMA cause METH-like long-term deficits in brain 5HT, but not DA systems. These persistent serotonergic effects appear to be (1) at least partially mediated by MDMA-related stimulation of DA systems, (2) linked to production of free radicals, (3) dependent on the serotonin transporters, and (4) facilitated by hyperthermia. However, reactive MDMA metabolites do not appear to be necessary for this neurotoxic effect of MDMA. These preclinical findings in rat predict that, in humans, MDMA substantially enhances the activity of DA systems and is a potential neurotoxin to some 5HT systems.

Challenges

As with many animal models, the relevance of these findings to humans and real-life circumstances has been questioned. With new and more sophisticated methodologies, we are now able to determine whether our findings in the laboratory are predictive of the human experience.

Future Direction

If we are to effectively deal with persons who have experienced long-term brain consequences from using MDMA, we must identify more precisely how and why this drug damages the central nervous system. With such knowledge, we should be able to develop better strategies to prevent and treat MDMA-induced functional deficits.

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Biography

Dr. Hanson was appointed Director of the Division of Neuroscience and Behavioral Research at the National Institute on Drug Abuse (NIDA) in September 2000. This Division administers extramural research programs in basic neurobiological and behavioral sciences related to issues of drug abuse and addiction. Dr. Hanson is also a tenured full professor in pharmacology at the University of Utah. He is actively involved in research that until now has resulted in (1) approximately 150 papers in peer-reviewed scientific journals and (2) approximately 200 abstracts presented at scientific meetings around the world. Dr. Hanson is recognized as an expert on the psychostimulants and is particularly known for his work on the neurotoxic properties of ecstasy and the amphetamines and the role of brain peptides (small proteins) in psychiatric and neurological functions. Dr. Hanson has been supported by grants from NIDA and the National Institute of Mental Health since the early 1980s and in 1998 received a Senior Scientist Award from NIDA. He has served on several grant review committees for NIH and on the editorial board of the *Journal of Pharmacology and Experimental Therapeutics* and is a frequent reviewer for most of the major pharmacology and neuroscience journals.

What We Know

Acute adverse reactions after taking “ecstasy” can result in death. In nearly all cases, the symptoms include disruption of body temperature and cardiovascular regulation. Research on MDMA has identified mechanisms that explain some of these effects and support clinical observations suggesting that high ambient temperatures and high physical activity may contribute to the onset of these symptoms (Dafters, 1995). Unfortunately, this research still does not clearly explain why these adverse events are unpredictable and do not occur after every administration. It is likely there are many reasons, including genetic predisposition and drug interactions as well as the behavioral and environmental factors already mentioned.

Challenges

In many situations, ecstasy is not only MDMA but also includes a number of other drugs or drug combinations. This vastly complicates the pharmacology of ecstasy poisonings and makes the design of treatment problematic. For example, in Australia, p-methoxyamphetamine (PMA) is commonly sold as ecstasy and has resulted in deaths (Byard et al., 1998). Furthermore, our data suggest that PMA is the drug responsible for most of the ecstasy poisonings in our study population (Ling et al., 2001). Our animal studies also indicate that PMA has potent effects on thermoregulation and the cardiovascular system (Irvine et al., 2001).

Future Directions

In order to advance our understanding of acute toxic effects of ecstasy, a number of issues must be addressed. The adverse effects of agents such as MDMA and PMA in animal models need to be clearly characterized and the roles of fundamental variables such as gender, ambient temperature, and metabolism examined. These studies will allow examination of more complicated aspects of acute ecstasy toxicity such as drug interactions.

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Biography

Originally from Aberdeen, Scotland, Dr. Irvine is an in vivo pharmacologist undertaking primarily animal-based research on drugs of abuse. These projects include examination of the pharmacology of MDMA and PMA as well as opioids. He is particularly interested in the acute and long-term adverse effects of these drugs.
Dr. Irvine is also involved in human-based research conducted in a major teaching hospital.

Biography

Dr. Kirschstein was named Acting Director of the National Institutes of Health (NIH) on January 1, 2000. She served as the NIH Deputy Director between November 1993 and December 31, 1999. She also served as the Acting NIH Director between July 1993 and November 22, 1993. Prior to that, Dr. Kirschstein was the Director of the National Institute of General Medical Sciences (NIGMS), beginning that appointment on September 1, 1974. A native of Brooklyn, New York, she received her B.A. degree in 1947 from Long Island University and her M.D. in 1951 from Tulane University School of Medicine. Dr. Kirschstein has twice taken part in World Health Organization (WHO) deliberations in Geneva, Switzerland, in 1965 as a member of the WHO Expert Group on International Requirements for Biological Substances and in 1967 as a consultant on problems related to the use of live poliovirus oral vaccine. She has received many honors and awards, including the Presidential Meritorious Executive Rank Award, 1980; election to the Institute of Medicine, 1982; selection by the Office of Personnel Management as 1 of 10 outstanding executives and organizations for its first group of “Profiles in Excellence,” 1989; election as a fellow of the American Academy of Arts and Sciences, 1992; and the Public Service Award from the Federation of American Societies for Experimental Biology in 1993.

Biography

Dr. Leshner was appointed Director of the National Institute on Drug Abuse (NIDA) in February 1994. NIDA, one of the Institutes within the National Institutes of Health (NIH), supports more than 85 percent of the world's research on the health aspects of drug abuse and addiction. Before joining NIDA, Dr. Leshner had been with the National Institute of Mental Health (NIMH) since 1988, holding the position of Deputy Director and then Acting Director. He came to NIMH from the National Science Foundation (NSF), where he held a variety of senior positions focusing on basic research in the biological, behavioral, and social sciences, as well as on science education. Dr. Leshner joined the NSF after 10 years at Bucknell University, where he was a professor of psychology. His research has focused on the biological bases of behavior. He is the author of a major textbook on the relationship between hormones and behavior and numerous book chapters and papers in professional journals. Dr. Leshner received his undergraduate degree in psychology from Franklin and Marshall College and his master's and doctoral degrees in physiological psychology from Rutgers University. He also holds honorary Doctor of Science degrees from Franklin and Marshall College and the Pavlov Medical University in St. Petersburg, Russia. He has been elected a fellow of many professional societies, is a member of the Institute of Medicine of the National Academy of Sciences, and has received numerous awards from both professional and lay groups. In 1996, President Clinton conferred on Dr. Leshner the Presidential Distinguished Executive Rank Award, the highest award in Federal service. In 1998, Dr. Leshner was elected to membership in the Institute of Medicine of the National Academy of Sciences.

Biography

Dr. Lynch directed a program of preclinical investigation at the SUNY Health Science Center and Veterans Administration Medical Center in Syracuse, New York, for 13 years before joining the National Institute on Drug Abuse (NIDA) as a Program Administrator in 1998. Her multidisciplinary research program investigated the neurobiological substrates underlying (a) motivated behaviors (e.g., responses to incentive motivational stimuli) and (b) response patterns mimicking symptom profiles of psychiatric disease. As research faculty in the Graduate Neuroscience Program, she also served as course coordinator for *Cognitive and Behavioral Neuroscience* and was responsible for medical student instruction in *Neurotransmitters and Behavior* and *Pathophysiological Substrates of Psychiatric Disorders*. She is the Acting Chief of the Behavioral and Cognitive Science Research Branch at NIDA and Chair of the trans-institute Behavioral Working Group.

Advances

Studies indicating that MDMA is a potent brain serotonin (5-HT) neurotoxin in animals have raised concern that humans who use MDMA may also incur brain serotonergic neuronal injury. However, the paucity of methods available for assessing the status of brain serotonin neurons in living humans has been a significant obstacle for clinical studies of MDMA-induced 5-HT neurotoxicity. To date, two methods for detecting brain 5-HT neurotoxicity have been validated in nonhuman primates and subsequently used in human MDMA users. In particular, cerebrospinal fluid concentrations of 5-hydroxyindoleacetic acid (5-HIAA) have been shown to be reduced in primates with documented brain 5-HT neurotoxicity as well as abstinent human MDMA users. Similarly, positron emission tomography (PET) studies using a radioligand that binds to the serotonin transporter (SERT) show reductions in abstinent MDMA users similar to those seen in baboons with documented MDMA-induced neurotoxicity. In addition to validated measures of neurotoxicity, there is growing evidence that MDMA users have abnormalities in cognitive and neuroendocrine function, both of which are known to involve brain serotonin systems.

Challenges

Studies in MDMA users are limited by their retrospective nature and the possibility that other drugs of abuse or preexisting abnormalities play a role in the abnormal findings in this cohort.

Future Directions

Additional research should be focused on developing more sensitive and specific methods for detecting brain serotonin neurotoxicity in living humans as well as better defining its functional consequences. Studies are also needed to determine whether cognitive and neuroendocrine abnormalities in MDMA users are related to brain 5-HT deficits. Finally, longitudinal studies will be useful for determining whether MDMA users are more likely to develop neuropsychiatric problems with age.

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Biography

Dr. McCann is an associate professor of psychiatry at Johns Hopkins School of Medicine. Her research is in the area of drug abuse with a particular focus on the neurotoxic amphetamine analogues. Dr. McCann also serves as Associate Program Director of the Johns Hopkins Bayview General Clinical Research Center and is an attending physician at the Johns Hopkins Anxiety Disorders Program.

What We Know

MDMA is a sympathomimetic phenethylamine structurally related to the endogenous catecholamines such as epinephrine and dopamine, medications such as terbutaline and phenylephrine, and the hallucinogenic amphetamines such as mescaline. In addition to euphoria, MDMA produces robust cardiovascular stimulation and activates the hypothalamic-pituitary axis. MDMA increases heart rate, blood pressure, and myocardial oxygen consumption. In contrast with the cardiostimulatory beta agonist dobutamine, MDMA has little direct inotropic effect on the heart. The lack of inotropy may increase myocardial oxygen consumption more than expected on the basis of MDMA-induced increases in heart rate and blood pressure. MDMA increases plasma cortisol, prolactin, and dehydroepiandrosterone (DHEA). These hormonal changes may mediate some of the pleasurable effects of MDMA. The biodisposition of MDMA is both stereoselective and dose dependent. Therefore, plasma concentrations (with concomitant increases in toxicity) may rise dramatically when illicit users take multiple doses over brief time periods. In people, MDMA is probably metabolized by cytochrome p450 2D6. Increased plasma concentrations (and medical complications) are possible when MDMA is co-administered with other CYP 2D6 substrates such as dextromethorphan. Illicit MDMA ingestion has been associated with several poorly characterized adverse reactions (such as hyponatremia, rhabdomyolysis, seizures, and hyperthermia) that result in severe outcomes, including death. The frequency of occurrence and mechanisms of action causing these events are unknown but may be due to altered cardiovascular physiology, nonlinear pharmacokinetics, or inadvertent drug-drug interactions.

Challenges

MDMA use is increasing and idiosyncratic drug reactions are difficult to predict. A prime challenge will be delineating both the frequency of occurrence and the mechanism of these idiosyncratic drug reactions. For example, a very real issue is the substitution of dextromethorphan for MDMA by illicit dealers. The pharmacologic effects of this combination have received little study.

Directions

Studies of the effects of MDMA when co-administered with other CYP 2D6 substrates (such as dextromethorphan and methamphetamine) and the effects of MDMA on cardiac, hepatic, renal, and thermoregulatory physiology are needed.

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Biography

Dr. Mendelson is a practicing internist and Associate Clinical Professor of Psychiatry at the University of California, San Francisco. His research involves studies of the pharmacology of abused drugs in humans. He has conducted NIH-funded human laboratory-based experiments on the pharmacokinetics and dynamics of MDMA, the stereoisomers of methamphetamine, cocaine and ethanol interactions, and the combination of buprenorphine and naloxone.

Are the Psychological Problems Associated With Regular MDMA Use Reversed by Prolonged Abstinence?

Michael John Morgan, Ph.D.

What We Know

Chronic, regular recreational use of "ecstasy" (MDMA) is associated with elevated psychopathology (Morgan, 2000), behavioral impulsivity (Morgan, 1998), and persistent impairment of memory performance (Morgan, 1999). The aim of the present study was to investigate which of these sequelae persist after at least 6 months of abstinence from MDMA. Four groups of participants were compared: 18 current regular recreational MDMA users, 15 ex-regular MDMA users who had abstained from using the drug for an average of 2 years, 16 polydrug users who had never taken MDMA, and 15 drug-naive controls. Both current and ex-MDMA users exhibited elevated psychopathology and behavioral impulsivity compared with polydrug users and drug-naïve controls, and both groups also exhibited impaired working memory and recall performance compared with drug-naive controls, although only ex-users exhibited impaired delayed recall compared with polydrug users. Thus, the present data suggest that psychological problems associated with regular MDMA use are not reversed by prolonged abstinence.

Challenges

- Adequate sampling
- Verification of drug histories (including the purity and quantity of MDMA consumed)
- Polydrug use by MDMA users presenting difficulty in determining specific sequelae of MDMA use
- Determining the causality of these sequelae
- Converting media interest into sustained research funding

Future Directions

- Further investigation of the psychological sequelae of different patterns of consumption of MDMA and other substances
- Prospective longitudinal studies
- fMRI studies of the effects of MDMA, and tryptophan depletion, on brain activity and cognitive function in regular MDMA users

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Biography

Dr. Morgan spent 2 years as a visiting fellow in the neuroimaging laboratory at NIDA before returning to the United Kingdom in the early 1990s to develop his MDMA research at the University of Wales. He was one of the first investigators to report that regular MDMA use is specifically associated with particular persistent psychological deficits. He has recently moved to assume a senior position at the University of Sussex to develop his collaboration with Philip McGuire at the Institute of Psychiatry on fMRI studies of the effects of MDMA, and tryptophan depletion, on brain activity and cognitive function in regular MDMA users.

Biography

Dr. Normand is Acting Chief of the Epidemiology Research Branch in the Division of Epidemiology, Services and Prevention Research at the National Institute on Drug Abuse (NIDA), National Institutes of Health. As Branch Chief, he is responsible for planning, developing, and administering a national and international research program on the epidemiology and etiology of drug abuse and drug-related behavioral, social, and health consequences. Prior to his work at NIDA, he was a study director at the National Research Council and the Institute of Medicine of the National Academy of Sciences in Washington, D.C., where he directed the Panel on Needle Exchange and Bleach Distribution Programs and the Committee on Drug Use in the Workplace. His earlier professional experience included research positions in both the private and public sectors. He has published in various professional research journals and has spoken at numerous professional meetings on evaluation issues.

Advances

Recreational users of ecstasy/MDMA demonstrate a wide range of psychobiological problems that are consistent with impaired serotonergic functions. A number of surveys have assessed self-reported functioning while off drug. The psychobiological problems reported include altered sleep, changed appetite, and loss of sexual interest or pleasure. Psychiatric symptoms include depression, phobic anxiety, general anxiety, obsessive-compulsive disorder, impulsivity, and psychoticism. Interpersonal relationship difficulties, financial loss, and occupational problems have also been described. In studies involving non-drug user controls, psychiatric symptom rates are often significantly higher. But when other illicit drug user groups are included, they also tend to show high rates of psychobiological distress.

Challenges

Since most recreational ecstasy/MDMA users are heavy polydrug users, the main challenge is to tease out which neuropsychobiological problems are specific to MDMA and which are characteristic of illicit polydrug use in general.

Future Directions

Prospective studies are needed to monitor how the initial uptake of ecstasy and other specific drugs leads to changes in psychobiological functioning. Such studies could also be used to assess the effects of drug discontinuation. However, the prospects are not promising. We recently assessed a young former heavy user who had stopped using ecstasy 7 years ago. He demonstrated poor neurocognitive test performance and was still suffering from profound sleep disturbance, phobic anxiety, severe depression, and sexual impotence.

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Biography

After earning a Ph.D. degree at the University of Leeds, Dr. Parrott worked as a research fellow at the Human Psychopharmacology Research Unit, where he focused on benzodiazepines and second-generation antidepressants. At the Institute of Naval Medicine in Hampshire, he assessed the practical utility of transdermal scopolamine and other anti-motion sickness drugs in land and sea trials. At the University of East London, he assessed a wide variety of recreational drugs, including stimulants, anabolic steroids, LSD, ketamine, and cannabis (while based at Humboldt State University in California). At the University of East London, it has become increasingly apparent that nicotine dependency is psychobiologically damaging and leads to increased stress and depression. Dr. Parrott has also been investigating the acute and chronic effects of MDMA/ecstasy in humans for the past 7 years.

Advances

Animals treated with MDMA develop long-lasting depletions of brain serotonin (5-HT) axonal markers including 5-HT, 5-HIAA, 5-HT transporters (SERTs), and tryptophan hydroxylase (TPH). In addition, neocortical vesicular monoamine transporter (VMAT) density is reduced in MDMA-treated monkeys, and recent findings indicate that there is also a lasting impairment in 5-HT axonal transport. Further, brains of non-human primates treated with MDMA and evaluated 7 years later show evidence of "pruning," a phenomenon also seen following neurotoxic injury with various well-established monoaminergic neurotoxins, including 5,7-DHT. Doses of MDMA that damage brain serotonin neurons in animals overlap with those typically used by humans. Indeed, recent studies in squirrel monkeys and baboons using a dosing regimen of MDMA that closely parallels that used by humans attending "raves" reveal severe 5-HT neurotoxic injury 2 weeks after MDMA exposure.

Challenges

More data are needed on MDMA's effects in non-human primates, particularly data regarding the long-term fate of serotonin neurons with aging. In addition, there is a paucity of information regarding the functional consequences of MDMA-induced 5-HT damage in animals.

Future Directions

Additional research is needed to better characterize the long-term effects of MDMA on brain 5-HT neurons of non-human primates, and potential functional consequences. Studies in non-human primates should also be conducted to validate methods of detecting brain 5-HT injury suitable for use in humans. Finally, preclinical studies aimed at determining the mechanisms of MDMA-induced 5-HT neurotoxicity may have important scientific and clinical implications.

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Biography

Dr. Ricaurte is an associate professor in the Department of Neurology at Johns Hopkins Medical Institutions. He obtained his M.D. degree at Northwestern University School of Medicine and his Ph.D. degree at the University of Chicago. He trained in neurology at the Stanford University School of Medicine. At Johns Hopkins, he directs his neurotoxicology laboratory and heads the Movement Disorders Clinic at the Johns Hopkins Bayview Medical Center.

The Diffusion of MDMA Use Among Urban Youth in Hartford, Connecticut; Implications for Drug and HIV Prevention in Club Drug Users and Their Networks

Jean J. Schensul, Ph.D.

In spite of Federal and local efforts to inform the wider public about the dangers associated with MDMA, use has increased dramatically over the past 2 years. Once viewed as a "suburban" or "rave" drug, MDMA is now widely used among some networks of urban youth, who have limited access to accurate sources of information with consequent higher levels of exposure to risks associated with use, selling, and exposure to hard drug use. This paper combines data obtained through participant observation in urban party and club settings with network and survey data collected from urban youth between the ages of 16 and 24 to identify changing rates of diffusion of MDMA through street youth networks between 1999 and 2001, ways in which urban youth are introduced to MDMA and other dance drugs through attendance at "regular" and "after-hour and after-school clubs," the role of drugs in these settings, the means through which MDMA has diffused from these venues to urban street environments, and the changing role of media and other social influences in promoting and supporting MDMA use.

Research challenges in conducting ethnographic, epidemiologic, and network research with urban youth and in party settings include staffing, network recruitment, attrition, and confidentiality. Prevention strategies involving community media, youth networks, and local neighborhood economies and future research directions including longitudinal studies, intervention research, and ethnographic studies of drug diffusion and polydrug use will be discussed.

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Biography

Dr. Schensul is Executive Director of the Institute for Community Research and recipient of NIH grants on substance use and sexual risk and HIV exposure in urban youth and adults. She received her doctoral degree in anthropology from the University of Minnesota in 1974. Recent publications include the *Ethnographer's Toolkit* (7 books) (Altamira Press, 1999) and articles on ethnicity, social networks, and HIV risk in older drug users (*Advances in Medical Sociology*, Vol. 8, Oxford University Press, in press), high THC marijuana use in urban adolescents (*Medical Anthropology*, 2000), and action research for prevention and service learning with adolescents (*Service Learning Research*, Vol. 1, Information Age Publishers, 2001).

Advances

Data reveal ecstasy use to be worldwide. Its availability and use continue to increase in the United States, Western Europe, Australia, Southeast/East Asia, South and Western Africa, the Middle East, and South America. Globally, ecstasy use appears to be most common among adolescents and young adults from all socioeconomic strata. Ecstasy use continues to shift away from large dance events to more diffuse settings. Ecstasy is known to have positive as well as acute adverse effects. Less is known about its long-term impact.

Challenges

Ecstasy is perceived as a safe, nonaddictive drug, and the term refers to a wide range of phenethylamines that are classified as entactogens. A challenge is the uncertainty regarding the content of street samples of ecstasy. In addition, methodological challenges occur because most ecstasy users are polydrug users, making it difficult to determine the effects of ecstasy. Furthermore, the lack of baseline data on the functioning of ecstasy users makes it difficult to draw any conclusions about the causality of use-related adverse social and health consequences. Finally, no efficient treatment options have been identified.

Future Directions

Phenomenological research, in addition to continued clinical and neurological research, is needed to develop effective prevention interventions, including drug treatment. Such research also will provide insights into the developmental progression of ecstasy use, gender differences, and risk and protective factors. Longitudinal research is needed to investigate the long-term consequences of use. Finally, epidemiological and phenomenological trend data need to be collected worldwide using a shared methodology (e.g., for sampling and data collection).

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Biography

Dr. Sterk is a professor in the Department of Behavioral Sciences and Health Education at the Rollins School of Public Health of Emory University. She holds doctoral degrees in anthropology (University of Utrecht) and sociology (Erasmus University). Her research interests encompass substance abuse, including stages of use, emerging drug trends, and the consequences of use, women's health, mental health, and prevention interventions. She serves on NIDA's Community Epidemiology Working Group and was a member of the Institute of Medicine Panel on the Contributions of the Social and Behavioral Sciences to the Public's Health.

Advances
Administration of MDMA under controlled laboratory conditions in humans has opened up important research areas. Unlike animals, human volunteers can describe the subjective effects of a drug. These subjective effects may play a critical part in the drug experience and may be related to the pattern of drug use and abuse.

The overarching goal of the studies under way at Wayne State University is to understand the role of serotonin and dopamine systems in the subjective experience of MDMA ingestion.

Study 1 directly compares the subjective, physiological, and reinforcing effects of MDMA (1 and 2 mg/kg) with the serotonergic agent (metachlorophenylpiperazine [mCPP], 0.5 and 0.75 mg/kg), a dopaminergic drug (d-amphetamine, 10 mg and 20 mg), and placebo. MDMA was observed to have robust subjective effects (with features of both mCPP and d-amphetamine), to be highly reinforcing, and to have significant physiological effects.

Study 2 is a three-way drug discrimination study. Training drugs were mCPP, d-amphetamine, and placebo. MDMA was identified by some participants as mCPP and as d-amphetamine by others, confirming that both serotonergic and dopaminergic cues were being employed.

Challenges
Although the laboratory studies can help answer important questions concerning the pharmacology and acute effects of MDMA, the laboratory differs significantly from the conditions under which MDMA is consumed recreationally. Another challenge is to integrate findings from observational studies in MDMA users to develop hypotheses that can be more precisely tested under controlled conditions.

Future Directions
Future laboratory studies should employ multiple methods such as neuropsychological testing and functional brain imaging during MDMA administration.

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Biography
Dr. Tancer is a psychiatrist with extensive research and clinical experience. Trying to objectively describe the subjective experience of anxiety led him to develop a collaboration with Dr. Chris-Ellyn Johanson, a behavioral pharmacologist. This collaboration led to the submission of a training grant to look at serotonin/dopamine interactions in mediating stimulant drug effects. MDMA was selected as a mixed serotonin/dopamine drug. The explosion of MDMA use over the past few years has led to a new direction of inquiry, namely, examining the consequences of MDMA use.

Advances

Little is known about the effect of MDMA or methamphetamine on the developing brain. In animals, methamphetamine (given when granule cells are dividing in the hippocampus) causes impairments in spatial learning. Rats treated with MDMA during this same stage show spatial and sequential learning impairments. MDMA animals had no difficulty learning a cued version of the maze. Amphetamines suppress appetite and therefore growth. To determine whether this could explain the learning effects of MDMA, we treated four groups: (1) MDMA, litter size = 8, (2) S8 = saline, litter size = 8, (3) S16 = saline, litter size = 16, and (4) HC = no saline (handled), litter size = 8. Groups S8 and HC grew similarly, while S16 grew at the same reduced rate as the MDMA group. Only MDMA-treated animals showed impaired spatial learning. Therefore, spatial learning effects in MDMA offspring are not secondary to undernutrition. In adult brain, MDMA induces 5-HT release, blocks reuptake, and causes long-term depletion. In our rats, small changes were found in NE and 5-HT, but not in DA. These changes did not correlate with any measure of cognitive impairment.

Challenges

No data are available on earlier stages of brain development, i.e., during embryogenesis or early fetogenesis. No data exist on the developmental pharmacokinetics of MDMA. No information is available on the reversibility of the effects seen thus far or on the cellular changes accompanying these effects.

Future Directions

Human investigations are urgently needed. Animal studies are also needed in order to forecast effects, characterize the range of central nervous system changes, establish dose-response and critical period relationships, and determine the pathophysiology and molecular mechanisms of action of MDMA on the developing brain.

Reference

Broening HW, Morford LL, Inman-Wood SL, Fukumura M, Vorhees CV. (2001) 3,4-Methylenedioxymeth-amphetamine (ecstasy) induced learning and memory impairments in adult offspring depend on the age of exposure during early development. *J Neurosci* 21:3228-3235.

Biography

Dr. Vorhees is Professor of Pediatrics and Environmental Health in the Division of Developmental Biology at the Children's Hospital Research Foundation and University of Cincinnati College of Medicine. He is also the Director of the Graduate Program in Molecular and Developmental Biology and Editor in Chief of *Neurotoxicology and Teratology*. His research interests include effects of substituted amphetamines on brain development and behavior and understanding the biological basis of learning and memory using gene targeting of proteins such as PDE1B, PTPα, dopamine D1, DFF45, and c-fos.

Advances

The neurotoxicity of MDMA has been extensively characterized in animals. This is evidenced by the destruction of fine-diameter serotonergic fibers, loss of tryptophan hydroxylase and serotonin transporter activities, and a long-term depletion of serotonin content in tissue. Convergent lines of evidence point to the roles of oxidative damage and hyperthermia in mediating the toxicity to serotonin terminals (for reviews, see Fleckenstein et al., 2000; Huether et al., 1997; and Shankaran et al., 1999).

Challenges

Despite extensive descriptive evidence of MDMA-induced neurotoxicity in laboratory animals, the mechanistic underpinnings that mediate this damage to 5HT terminals remain to be defined. Since the amphetamines, in general, cause hyperthermia, sympathetic arousal, and increased metabolism, there is a reasonable possibility that high doses of MDMA also cause a metabolic stress that interacts with oxidative damage and hyperthermia to produce long-term damage to 5HT nerve terminals. To date, however, few studies have examined metabolic stress as a factor in MDMA neurotoxicity. In addition, there are few studies on the interaction between MDMA and environmental/psychological stressors as well as the impact of MDMA on normal physiological processes such as sleep/wakefulness activity. Recent data from our laboratory will provide evidence supportive of the role of metabolic stress and will indicate that an acute exposure to MDMA alters the reactivity to environmental stress and disrupts sleep/wakefulness physiology.

Future Directions

Future research directions highlighted by the above results include the precise mechanisms underlying a compromised bioenergetic state, changes in stress reactivity, and altered sleep/wakefulness physiology caused by MDMA that could provide insight into the development of effective therapeutic treatments for the long-term consequences of MDMA intoxication.

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Shankaran M, Yamamoto BK, Gudelsky GA. (1999) Effect of 3,4-methylenedioxymethamphetamine (MDMA) on hydroxyl radical formation in the striatum. *J Neurochemistry* 72:2516-2522.

Biography

Dr. Yamamoto received his B.A. degree from the University of California, Los Angeles, and his Ph.D. degree in psychobiology from Syracuse University. He was a postdoctoral fellow in pharmacology at the University of Colorado Health Sciences Center. Dr. Yamamoto has been at Case Western Reserve University in the Department of Psychiatry since 1990 and is currently Professor of Psychiatry and Neurosciences and Director of the Program in Basic and Clinical Neuroscience in the Department of Psychiatry. This fall, he will be a Professor of Pharmacology at the Boston University Medical School. Dr. Yamamoto has served on numerous NIH study sections and currently is a regular member of a NIDA study section. NIH has funded his research continuously for the past 15 years, and his work on the neurotoxicity of methamphetamine and MDMA has been funded by NIDA for the past 10 years.

POSTER ABSTRACTS

Significance of Serotonin Receptors in the Behavioral Effects of (+)-3,4-Methylenedioxymethamphetamine [(+)-MDMA, Ecstasy]

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The rapid rise in abuse of MDMA underscores the importance of understanding its mechanisms of action as a prelude to establishing therapeutic means to control overdose, toxicity, and overt psychological and physiological responsivity to MDMA. MDMA binds to the serotonin (5-HT) transporter (SERT) with high affinity and reverses SERT to result in 5-HT release from nerve terminals; in addition, MDMA is reported to bind to 5-HT_2 receptors (5-HT_2R). In the current series of experiments, pharmacological ligands specific for 5-HT_{1B}R, 5-HT_{2A}R, and 5-HT_{2C}R were employed to analyze the role of these receptors in the hyperactivity induced by acute or chronic administration of (+)-MDMA. Systemic administration of the 5-HT_{1B}R antagonist GR 127935 and the 5-HT_{2A}R antagonist M100907 effectively suppressed (+)-MDMA-induced activity. Thus, release of 5-HT consequent to (+)-MDMA binding to SERT results in a stimulation of 5-HT_{1B}R and 5-HT_{2A}R to result in hyperactivity. In contrast, the indirect stimulation of 5-HT_{2C}R serves to mask expression of (+)-MDMA-induced hyperactivity as evidenced by the observation that blockade of the 5-HT_{2C}R with SB 206553 or SB 242084 results in a dramatic increase in (+)-MDMA-evoked hypermotive effects. Following repeated, intermittent administration of (+)-MDMA, an enhancement of the acute hyperactive effects of (+)-MDMA was observed (termed “behavioral sensitization”). During withdrawal from this MDMA pretreatment regimen, enhanced hyperactivity and reduced hypomotility were observed upon administration of a 5-HT_{1B}R (RU 24969) or a 5-HT_{2C}R agonist (MK 212), respectively; these data support the hypothesis that chronic (+)-MDMA administration is associated with adaptational modifications in the function of 5-HT_{1B}R and 5-HT_{2C}R. In keeping with these results, withdrawal from a chronic treatment regimen of MK 212 resulted in sensitization to (+)-MDMA-induced hyperactivity, suggesting that adaptation of 5-HT_{2C}R function contributes to the modifications that underlie expression of sensitization. Thus, 5-HT_{1B}R, 5-HT_{2A}R, and 5-HT_{2C}R are important mediators of the acute and chronic effects of MDMA, and the role of these 5-HT receptors indicates a number of directions to pursue in our understanding of the clinical pharmacology of MDMA.

Research was supported by DA 06511, DA 00260, DA 13595, and DA 07287.

Acute Transcriptional Effects of MDMA on the Rat Cortex: Evidence From cDNA Analysis

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3,4-Methylenedioxymethamphetamine (MDMA) is an analogue of methamphetamine (METH). It is related to both amphetamines and hallucinogens. METH and MDMA are known toxins with a plethora of acute effects. Both the acute and long-term effects of MDMA appear to involve the production of oxygen-based reactive species since MDMA perturbs the activity of various antioxidant enzymes (Cu/Zn superoxide dismutase, catalase, glutathione peroxidase). Using cDNA array technique, we have further investigated the acute effects of MDMA on the rat cortex. Some of these data will be discussed in view of their support for the possible involvement of multiple systems in the effects of MDMA.

Visual Cortex Activation Using the fMRI Blood Oxygen Level-Dependent (BOLD) Method in Human MDMA (Ecstasy) Users

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The recreational club drug ecstasy (3,4-methylenedioxymethamphetamine, MDMA) is selectively toxic to the axons arising from the serotonergic dorsal raphe nucleus (DRN) in animals. Human studies using a variety of techniques also indicate that MDMA produces long-lasting alterations in serotonergic function. Because serotonin plays an important role in sensory function, with serotonin axons densely innervating visual cortex, we chose to use the functional magnetic resonance imaging (fMRI) BOLD method to study the effects of prior MDMA use on visual cortical activation during photic stimulation. Studies were performed on a small group of male and female MDMA users and controls using photic stimulation with red and blue light at 8 Hz with echoplanar imaging at 1.5 tesla.

A 3x8 pixel region of right and left visual cortex was analyzed for BOLD signal change in response to red or blue light at a frequency of 8 Hz. Linear regression methods were used to assess the statistical significance of the findings. A small cohort of males (6 MDMA users, 6 controls) and females (10 MDMA users, 5 controls) was studied. MDMA users and controls showed overlap in BOLD signal change in response to photic stimulation. Left-sided activation showed no clear trend in mean BOLD signal change to blue or red light in either males or females. However, right hemisphere mean BOLD signal change showed a nonsignificant trend for decreased activation to blue light in the male and female MDMA users, and right hemisphere activation to red light showed a similar trend for reduced activation in MDMA users, with significant reductions in mean activation in activated pixels for the male group ($p<.05$). These findings support the possibility that MDMA use can produce impairment in visual cortical activation in response to photic stimuli.

Control subjects and MDMA users reported similar history of at least one use of alcohol and marijuana, but MDMA users were generally heavier users of both substances. Compared with controls, MDMA users also reported more use of other drugs, including cocaine, opiates, amphetamine, sedative-hypnotic, or hallucinogen use. These preliminary data in a small sample that is unequally matched for other polydrug use suggest that MDMA use may lead to altered sensory processing as assayed by fMRI using a photic stimulation paradigm. A much larger sample, controlling for other drug use, duration of abstinence from MDMA, and degree of prior MDMA exposure, is necessary to confirm the specificity of these findings.

MDMA Produces Glycogenolysis and Increases the Extracellular Concentration of Glucose in the Rat Brain

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Oxidative and/or bioenergetic stress is thought to contribute to the mechanism of neurotoxicity of amphetamine derivatives, e.g., MDMA. In the present study, the effect of MDMA on brain energy regulation was investigated by examining the effect of MDMA on brain glycogen and glucose. A single injection of MDMA (10-40 mg/kg, sc) produced a dose-dependent decrease (40 percent) in brain glycogen, which persisted for at least 1 hour. MDMA (10 and 40 mg/kg, sc) also produced a significant and sustained increase in the extracellular concentration of glucose in the striatum. Subjecting rats to a cool ambient temperature of 17 ^{0}C significantly attenuated MDMA-induced hyperthermia and glycogenolysis. Amphetamine analogues, e.g., methamphetamine and parachloroamphetamine, which produce hyperthermia, also produced glycogenolysis, whereas fenfluramine, which does not produce hyperthermia, did not alter brain glycogen content. MDMA-induced glycogenolysis also was prevented by treatment of rats with the 5-HT$_2$ antagonists LY-53,857 (3 mg/kg), desipramine (10 mg/kg), and iprindole (10 mg/kg). LY-53,857 also attenuated the MDMA-induced increase in the extracellular concentration of glucose. These results support the view that MDMA promotes energy dysregulation and that hyperthermia may play an important role in MDMA-induced alterations in cellular energetics.

Research was supported by DA07427.

Discriminative Stimulus Properties of 4-MTA and the Optical Isomers of PMA

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1-(4-Methoxyphenyl)-2-aminopropane (PMA) and its sulfur analogue 1-(4-methylthiophenyl)-2-aminopropane (4-MTA) have been misrepresented as the controlled substance analogue MDMA (ecstasy). PMA is a Schedule I substance, and 4-MTA is currently under consideration for scheduling. Little is known about the stimulus properties of these phenylalkylamines. In general, phenylalkylamines with abuse potential can produce one or more of at least three distinct stimulus effects in animals: a stimulant or amphetamine-like effect, a hallucinogenic or DOM-like effect, and a third effect that is typified by N-methyl-1-(4-methoxyphenyl)-2-aminopropane (PMMA). PMMA is the N-methyl analogue of PMA. We previously demonstrated that PMA, but not PMMA, substitutes for (+)amphetamine. In the present investigation, we examined the optical isomers of PMA in PMMA-trained Sprague-Dawley rats. We also prepared 4-MTA and its N-methyl analogue 4-MTMA (i.e., the sulfur counterpart of PMMA) and examined them in rats trained to discriminate *S*(+)amphetamine (1 mg/kg) and PMMA (1.25 mg/kg) from vehicle. *R*(-)PMA (ED50 = 0.4 mg/kg) substituted for PMMA (ED50 = 0.4 mg/kg), whereas *S*(+)PMA produced a maximum of 72 percent PMMA-appropriate responding. 4-MTA (ED50 = 0.3 mg/kg) also substituted for PMMA. The PMMA stimulus failed to substitute for 4-MTMA (maximum 36 percent PMMA-appropriate responding). Administration of 4-MTA and 4-MTMA to (+)amphetamine-trained animals resulted only in partial generalization. Hence, *R*(-)PMA and 4-MTA are capable of producing PMMA stimulus effects in animals.

Research was supported in part by DA 01642.

Attachment Relationships and MDMA (Ecstasy) Use Among College Students

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Advances over the past 15 years indicate that MDMA is popular among college students. The psycho-emotional effects and context of use are described consistently as connectedness, oneness with others, increased empathy and communication, feelings of acceptance, and ease of emotive expression—a place that provides a sense of security, bonding, and attachment.

Advances in the family sciences indicate that Bowlby's Attachment Theory represents one potential familial factor for explaining the initiation of cigarette smoking, polydrug addiction, alcoholism, and opiate addiction. Theoretically, securely attached individuals have the ability to feel close to and supported by others, form satisfying relationships, and therefore are not likely to utilize maladaptive substitutes to satisfy attachment needs. The lack of positive internal working models inhibits the establishment of genuine, meaningful, interdependent relationships resulting in maladaptive strategies to induce a sense of attachment. The less securely attached may be particularly attracted to MDMA's effects of connectedness, bonding, or attachment and use it to satisfy attachment needs, although temporary and artificial.

Two major methodological challenges emerge in the investigation of attachment and MDMA use in college students. This high-functioning sample yields little variability of attachment score (93 percent moderately-highly attached). This contributes to the paucity of information provided by the attachment variable. Further, no standardized interpretive guidelines are available for this instrument, resulting in the inability to compare scores with norms.

If methodological challenges are overcome, attachment could emerge as a key risk/protective factor in the initiation of MDMA use. If so, "attachment work" may be a vital element in the prevention and treatment of MDMA abuse.

MDMA and Its Stereoisomers as Reinforcers in Rhesus Monkeys: Serotonergic Involvement

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3,4-Methylenedioxymethamphetamine (MDMA, "ecstasy") is a ring-substituted phenethylamine structurally related to both amphetamine and mescaline. While there have been two prior assessments of racemic MDMA in IV self-administration paradigms in primates, the reinforcing effects of the stereoisomers of MDMA have not been previously assessed. In this regard, four adult male rhesus monkeys were tested twice daily in 60-minute FR10TO1min sessions alternately maintained by IV infusions of (+/-)-MDMA, (+)-MDMA, (-)-MDMA, cocaine, methamphetamine, or equivolume saline. Racemic MDMA and both enantiomers generated inverted U-shaped functions across the dose ranges tested, although response rates were generally lower than those engendered by cocaine or methamphetamine. Subsequently, five MDMA-naive adult rhesus monkeys (three male, two female) were tested twice daily in 150-minute FR30TO45sec sessions alternately maintained by four discrete IV doses of cocaine, (+)-MDMA, (-)-MDMA, or equivolume saline, thus allowing rapid dose-effect curve determination. The effects of pretreatments with the 5-HT2 antagonists ketanserin and MDL100907 on MDMA self-administration were then assessed. Neither ketanserin nor MDL100907 pretreatment affected cocaine-maintained responding; however, both antagonists shifted the (+)-MDMA dose-effect curve and completely abolished responding for (-)-MDMA. Potential serotonergic contributions to MDMA reinforcement will be discussed.

Research was supported by USPHS grants DA-034948 and DA-05923.

Methylenedioxymethamphetamine Decreases Plasmalemmal and Vesicular Dopamine Transport

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Our laboratory has reported that multiple high-dose administrations of methamphetamine (METH) rapidly and profoundly decrease plasmalemmal and vesicular dopamine (DA) uptake, as assessed in synaptosomes and purified vesicles, respectively, prepared from the striata of treated rats. To determine whether these effects were common to other amphetamines of abuse, effects of methylenedioxymethamphetamine (MDMA) on the DA transporter (DAT) and vesicular monoamine transporter-2 (VMAT-2) were assessed. Results reveal that similar to the effects of METH, multiple high-dose administrations of MDMA rapidly (within 1 hour) decreased plasmalemmal DA uptake. Unlike effects of multiple METH injections, this deficit was reversed completely 24 hours after drug treatment. Also in contrast with the effects of multiple METH injections, neither prevention of hyperthermia nor prior depletion of DA prevented the MDMA-induced decrease in plasmalemmal DA uptake. However, a role for phosphorylation was suggested by findings that pretreatment with NPC15437, a protein kinase C inhibitor, attenuated the MDMA-induced deficit caused by MDMA application in an in vitro model system. In addition to affecting DAT function, MDMA rapidly decreased vesicular DA uptake. Unlike effects of multiple METH injections, this decrease in vesicular uptake partially recovered by 24 hours after drug treatment. Taken together, these results reveal several differences between the effects of MDMA and METH on DAT and VMAT-2, differences that may underlie the dissimilar DA neurotoxic potential of these agents.

Research was supported by NIDA grants DA00869, DA11389, and DA04222.

Comparative Effects of Substituted Amphetamines (PMA, MDMA, and METH) on Monoamines in Rat Caudate: A Microdialysis Study

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Paramethoxyamphetamine (PMA) is a methoxylated phenethylamine derivative that has been used illicitly in Australia since 1994. PMA is also becoming popular at rave parties in the United States. PMA raised a concern when a series of fatalities resulted after its use in South Australia, where it was marketed as “ecstasy,” which is the colloquial name for MDMA. In the present study, we evaluated the comparative neurotoxicity of substituted amphetamines (PMA, MDMA, and METH) in rats. Extracellular levels of dopamine (DA), 3,4-dihydroxy-phenylacetic acid (DOPAC), homovanillic acid (HVA), 5-hydroxyindoleacetic acid (5-HIAA), and serotonin (5-HT) were assayed in the caudate of freely moving rats using microdialysis and HPLC-EC. Dialysates were assayed every 20 minutes for 4 hours following an intraperitoneal (IP) injection of PMA (2.5, 5, 10, 20 mg/kg), MDMA (10, 20 mg/kg), or METH (2.5 mg/kg). METH produced a significant increase in extracellular DA (700 percent) and significant decreases in extracellular DOPAC and HVA (30 and 50 percent), with no detectable changes in either HIAA or 5-HT. MDMA produced significant increases in DA (700 percent at 10 mg/kg; 950 percent at 20 mg/kg) and decreases in DOPAC (15 percent for both 10 and 20 mg/kg) and HVA (50 percent at 10 mg/kg; 35 percent at 20 mg/kg). MDMA also increased 5-HT (350 percent at 10 mg/kg; 575 percent at 20 mg/kg) and decreased 5-HIAA to 60 percent for both dose levels. PMA produced no detectable increases in DA at dose levels of 2.5, 5, or 10 mg/kg but significantly increased DA (975 percent) at a dose of 20 mg/kg. However, PMA significantly decreased DOPAC at all dose levels (75 percent at 2.5 mg/kg; 40 percent at 5 mg/kg; 30 percent at 10 mg/kg; 10 percent at 20 mg/kg), with comparable decreases in HVA at all dose levels. PMA also produced significant increases in 5-HT at 10 and 20 mg/kg (350 percent for both dose levels), with no detectable changes in 5-HT at 2.5 or 5 mg/kg. All dose levels of PMA significantly decreased 5-HIAA (50 to 70 percent). These data suggest that PMA, like MDMA and METH, is capable of producing dopaminergic and serotonergic neurotoxicity.

Neurocognitive Function in Recreational Users of MDMA

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Ecstasy [(+/-) 3,4-methylenedioxymethamphetamine: MDMA] is an increasingly popular recreational drug. Animal research has provided evidence that the high levels of serotonin released during MDMA use result in the destruction of serotonergic (5-HT) brain cells. 5-HT modulates several psychological functions, including mood and memory. Clinical studies have reported memory impairment and dysphoric mood symptoms secondary to MDMA use, possibly because of long-term alterations in 5HT function. In this study, we examined affect and neurocognitive functioning in abstinent recreational users of MDMA (ages 18-35 years) compared with age-matched non-using controls and a second control group of alcohol abusers. The dose, frequency, and duration of MDMA use were assessed through self-report questionnaires, and structured clinical interviews were used to rule out other pathology. Participants also completed a comprehensive neuropsychological testing battery including measures of IQ, motor skill, working and recognition memory, planning, set-shifting, verbal and nonverbal skill, and attention. Self-report measures of affect and personality traits were obtained. Preliminary data analyses suggest that ecstasy use may not be as detrimental to cognitive function as has been previously reported. Findings will be discussed in relation to serotonin's modulation of cognition, emotion, and their interactions within frontostriatal networks.

Working Memory and MDMA Use Among Gay Circuit Party Attendees

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MDMA and HIV affect similar regions of prefrontal cortex and aspects of neurocognition. However, neurocognitive effects of MDMA have not been studied in men who have sex with men (MSM) who participate in the circuit party/dance club culture. This population is at high risk for HIV disease, and HIV-infected MSM who use MDMA may be at increased risk for neurocognitive deficits, which have serious implications for risk behavior and adherence to medication regimens.

In a preliminary study as part of a currently funded NIDA project, we studied working memory, an executive function mediated by prefrontal cortex and known to be defective in HIV-positive persons. We compared the performance of HIV-negative MSM with and without a history of MDMA use on a verbal working memory task with known sensitivity to HIV-related cognitive deficits in drug abusers.

MSM with a history of MDMA use showed significant defects in working memory compared with MSM with no history of MDMA use ($p<.05$). These preliminary data are encouraging in that working memory deficits can be detected in MSM users of MDMA. The next step is to determine whether MDMA users who are also HIV-positive are indeed at increased risk for cognitive defects.

Interactions of Acute and Chronic Stress With d-MDMA–Induced Dopaminergic Neurotoxicity in the Mouse

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There is a perception that stress may exacerbate toxic consequences of drug exposures, although this idea has not been extensively tested in an objective manner. We have examined the effects of a variety of forms of acute and chronic stress on the striatal dopaminergic neurotoxic effects of d-MDMA (15 mg/kg sc every 2 hours X 4) in mouse. End points used to assess neurotoxic damage include severe reduction of dopamine, associated metabolites, and tyrosine hydroxylase holoenzyme protein levels and marked elevation of striatal glial fibrillary acid protein, an index of astrogliosis. We found that acute stress induced in a variety of ways (including restraint, exposure to a 15 ^{0}C ambient temperature, or administration of ethanol) is protective against the dopaminergic neurotoxicity caused by d-MDMA, whereas repeated exposure to stress causes a loss of its protective properties. Acute stress appears to cause physiologic changes at the time of drug administration that are protective, since restraint stress applied before drug administration is not protective against neurotoxic consequences. We also have found that exposure to chronic elevations of circulating corticosterone at supraphysiologic levels causes an enhancement of the striatal damage caused by d-MDMA, although it does not cause damage in hippocampus, a brain area thought to be susceptible to glucocorticoid neuroendangerment. Together these studies indicate that the physiologic changes induced by acute stress can be protective of the mouse brain against the neurotoxic effects of MDMA, whereas chronic stress either is not protective or may enhance MDMA-induced brain damage.

Behavioral Psychopharmacology of MDMA and Related Compounds: A Review of Animal Studies

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Since being classified as a Schedule I controlled substance in 1985, MDMA (3,4-methylenedioxy-N-methylamphetamine, ecstasy) has been the subject of controversy regarding its potential therapeutic usage, mechanism of action, popularity with young people in the "rave" culture, and issues of potential neurotoxicity. While most attention has centered on issues of neurotoxicity and epidemiological aspects of MDMA use in humans, the basic behavioral pharmacology studies of MDMA have quietly continued to gather information regarding the mechanisms underlying the behavioral effects of MDMA in animals. This poster will review animal studies of the behavioral effects of MDMA and related compounds in locomotor activity and startle paradigms. MDMA and related compounds produce a unique behavioral profile in rodents that includes locomotor hyperactivity, reductions in exploratory behavior, and deficits in both habituation and prepulse inhibition of startle. These behavioral effects are attributable to the release of presynaptic serotonin and the consequent activation of multiple serotonin receptors. The precise sites and mechanisms of action for these behavioral effects are currently under investigation. Studies of MDMA and related compounds using animal subjects have provided, and will continue to provide, information that can be used to elucidate the complex mechanisms underlying drug abuse, cognition, arousal, and motor activity as well as mechanisms of neurotoxicity.

This work was supported by a grant from the National Institute on Drug Abuse (DA02925).

MDMA in Combination: "Trail Mix" and Other Powdered Drug Combinations

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Advances

Recent ethnographic research conducted in Boston and New York City on HIV risk behaviors among men who have sex with men (MSM) and use club drugs has revealed that MDMA users in this population rarely use MDMA alone. One emerging pattern is the intranasal use of MDMA in a powdered mix with other drugs. One such formulation is known by users as "trail mix," which is commonly understood to be a powdered combination of MDMA and ketamine with the addition of a stimulant (such as methamphetamine or cocaine) and sometimes ground Viagra or other drugs. Trail mix is not sold as such; rather, it is manufactured by users to achieve a variety of effects, such as enhancing sex by adding methamphetamine or Viagra. Accidental overdoses have occurred, which users attribute to poorly mixed combinations.

Challenges

As this is an emerging drug pattern, there is little information regarding the clinical implications of powdered combinations of MDMA and potentially dangerous interactions between the constituent drugs. Although MSM in Boston and New York City have reduced their use of drugs such as GHB in response to perceived danger, prevention efforts have had little effect in reducing the use of MDMA. In the absence of data regarding MDMA in combination with other drugs, there is a significant challenge in mounting appropriate prevention efforts.

Future Directions

In order to develop appropriate interventions, future research studies should identify the characteristics of high-risk groups and address HIV risk behavior patterns associated with specific combinations of MDMA and other drugs.

Reactive Oxygen, Nitrogen Species: Implications for Neurotoxicity

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Ecstasy Use and Sexual Risk Behaviors Among Urban Youth

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This poster focuses on the transition to ecstasy use among urban youth in a 15-month period and the different factors associated with it specifically related to sexual risk behavior and sexually transmitted disease (STD). It is based on 3 years of research on factors influencing pathways to high-risk drug abuse and other health risks in urban youth and young adults. Ecstasy use is recognized nationally as a growing problem among youth, and Connecticut has recently experienced an increase in the use of this substance.

Our study utilized a targeted sampling plan and a panel design (repeat interview) with two time points 15 months apart. We recruited 400 African American and Latino/Puerto Rican youth between the ages of 16 and 24. In this poster, we will utilize baseline data to report on factors associated with ecstasy use. Next we will present data from the second interview on those who made the transition to ecstasy use and the factors associated with it. We will review STDs associated with this transition.

Use of Antisense Technologies To Study the Role of Dopamine in MDMA Neurotoxicity

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Antisense (AS) regulation of protein expression represents a new and innovative approach to investigating the role of dopamine (DA) in the serotonergic neurotoxicity induced by 3,4-methylenedioxymethamphetamine (MDMA). Previous work from this laboratory has suggested a role for DA metabolism in the serotonergic toxicity induced by MDMA. The monoamine oxidase-B (MAO-B) inhibitor L-deprenyl protects against the serotonergic toxicity of MDMA. However, L-deprenyl possesses ancillary pharmacological actions (e.g., free-radical scavenging), which may account for the neuroprotection. In order to avoid these confounding effects, AS technology was utilized. Osmotic minipump administration of an AS targeted against the DA transporter (DAT) resulted in a 70 percent reduction in DAT levels and attenuated the serotonergic toxicity induced by MDMA (2 X 20 mg/kg, sc) 1 week later, in a region-specific manner. This protective effect is presumably due to a decrease in DA release following MDMA in the AS-treated animals. Furthermore, AS targeted against MAO-B resulted in a 40 percent reduction in MAO-B activity, which was comparable to the reduction in MAO-B produced by a single 2 mg/kg dose of L-deprenyl. We shall report the effects of the AS to MAO-B on MDMA (40 mg/kg, sc) neurotoxicity alone or in combination with L-deprenyl and the details of the results with the DAT AS. Neither AS reduced the hyperthermia induced by MDMA. Thus, AS technology represents a novel method for investigating MDMA neurotoxicity. The present results do provide further support for the role of DA in this neurotoxic process.

Psychobiologic Effects of 3,4-Methylenedioxymethamphetamine in Humans: A Pilot Study

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3,4-Methylenedioxymethamphetamine (MDMA) is a phenethylamine with potent effects on serotonergic neurotransmission that has been the object of controversy over its potential as a therapeutic adjunct versus its possible risk for causing neurotoxic injury. Methodological design and preliminary data of the first FDA-approved Phase 1 study prospectively evaluating the effects of MDMA administration in human subjects will be presented. Eighteen subjects with prior experience with MDMA were administered two different dosages of MDMA and an inactive placebo utilizing a randomized, double-blind methodological design. Dosages from 0.25 to 2.5 mg/kg were administered orally. Subjects tolerated the procedures without evidence of psychological distress or physical discomfort, although two subjects did experience transient hypertensive episodes. Modest elevation of temperature was observed at higher dosages. The threshold dose for the stimulation of ACTH, cortisol, and prolactin appeared to be between 0.5 and 0.75 mg/kg, with higher doses clearly stimulating neuroendocrine secretion. Baseline and followup neuropsychological measures did not reveal signs of cognitive dysfunction. Within the well-monitored, approved clinical research setting, the prospective administration of MDMA to experienced human subjects appears to be a safe procedure that may provide valuable data to further elucidate the effects of the drug.

Behavioral Sensitivity to Serotonergic Challenge in MDMA-Treated Rhesus Monkeys

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Rhesus monkeys (N = 3) were exposed to a high-dose, short-course regimen of (±)3,4-methylenedioxy-methamphetamine (MDMA) (4 days, 10 mg/kg i.m., b.i.d.). Following treatment, concentrations of 5-hydroxyindoleacetic acid (5-HIAA) in cerebrospinal fluid (CSF) were reduced by approximately 50 percent in comparison with both pretreatment and control (N = 3) values. Homovanillic acid concentrations in CSF were unchanged. We have previously reported (Taffe et al., *Neuropsychopharm 24:230-9, 2001)* that the MDMA regimen temporarily disrupts cognitive/behavioral performance on a variety of neuropsychological test battery measures; however, no persisting alterations in performance were observed during a 4-month period in which CSF 5-HIAA levels remained suppressed. In the present study, the monkeys' behavioral performance in several cognitive domains was challenged acutely with ketanserin (0.1-1.7 mg/kg, i.m.), mCPP (0.03-0.5 mg/kg, i.m.), and 8-OH-DPAT (0.032-0.1 mg/kg, i.m.). Each compound affected performance on one or more tasks in a dose-dependent manner and to an equivalent extent in each treatment group. Animals were then sacrificed and brain tissue from a number of regions was assayed for serotonin and 5-HIAA. The MDMA-treated animals exhibited fourfold to fivefold reductions in 5-HT in cortex 17 months after MDMA exposure. In total, the present observations indicate that a substantial reduction in cortical 5-HT content does not make rhesus monkeys' cognitive performance more susceptible to the disrupting effects of serotonergic agents.

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"Just a Little Pill": Patterns and Trends Among Young Adult Ecstasy Users in Atlanta, Georgia

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Advances

Little is known about the behaviors of ecstasy users or the social and health consequences of use. Findings from our own preliminary research show a typology of users, including experimental, occasional, and regular users. All users describe rapidly developing a tolerance for the drug as well as strategies to cope with this. Many view ecstasy as a harmless pill, and few ecstasy users anticipate the negative consequences of use. Typically, ecstasy is described as a "bonding" drug, with physical contact being limited to hugging and touching. However, some users also describe engaging in unsafe sex when high on ecstasy in a private setting.

Challenges

Behavioral and social scientists conducting ecstasy research encounter several major challenges. Drawing a representative sample of ecstasy users is hindered by their relatively hidden nature, especially of those who do not use at raves, clubs, or bars. Self-reported data on the amount and frequency of use may have limited validity. Self-reported data also reflect the user's limited knowledge of the pill's content. Finally, ecstasy use is often combined with other substances, thus complicating attempts to isolate its pharmacological and social consequences.

Future Directions

More research is needed on ecstasy users, the context of use, and use patterns, including the route of administration and polydrug use. Such studies should include cross-sectional phenomenological studies as well as more large-scale, longitudinal quantitative studies. The baseline information from such studies should be used to develop appropriate risk reduction interventions.

Neurocognitive Impairment in MDMA Users: A Meta-analytic Review

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Applicability and Reliability of Ecstasy Abuse and Dependence Criteria Among Persons Aged 12 to 17 Years

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As part of an ongoing NIDA study on the reliability and applicability of the computerized CIDI-SAM, 120 adolescents and young adults have been interviewed to date about illicit drug use and its consequences. The SAM includes DSM and ICD criteria for 10 classes of drugs and was recently revised to include club drugs. Respondents are being recruited from a local inpatient substance abuse program as well as through high school newspaper advertisements, flyers posted at college dormitories, raves, and chain referral methods. To date, nearly one-quarter (24 percent) reported more than five times use of club drugs lifetime, with all reporting at least ecstasy and several reporting two or three additional club drugs. This sample is 64 percent female, 16 percent non-white, and 32 percent between 12 and 15 years of age. Among the 25 users, reliability as well as rates of DSM-IV criteria were surprisingly high. Specifically, “continuing to use despite knowledge of harm” was the most prevalent criterion (64 percent), being reported with excellent 1-week retest agreement (kappa = .80). Withdrawal from use was endorsed by over one-half of the users (59 percent; kappa = .56). “Tolerance” to club drugs and “spending a great deal of time to use, obtain, or recover” from club drugs were each reported with high reliability (kappa = .70/.65) by over one-third of the users. Of note, 85 percent of users received a diagnosis of either abuse or dependence when DSM-IV criteria were applied to ecstasy; 40 percent met at least three criteria and thus received a diagnosis of dependent while 45 percent met criteria for abuse. This is the first effort to assess the reliability of club drug abuse and dependence. A larger sample that includes ethnically diverse users both in and out of treatment assessed with a club drug-specific assessment is needed.

In Utero Exposure to (±)-Methylenedioxymethamphetamine Enhances the Development and Metabolism of Serotonergic Neurons in Reaggregate Tissue Culture

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Methylenedioxymethamphetamine (MDMA, ecstasy) is a potent psychomotor stimulant with neurotoxic potential that is widely abused by females of childbearing age. This raises serious public health concerns in terms of exposure of the fetus to the drug. The current study was conducted utilizing the three-dimensional reaggregate tissue culture system as an approach to the assessment of risk to fetal monoaminergic neurons following exposure to MDMA during early to mid-gestation. In this culture system, the serotonergic and dopaminergic mesencephalic-striatal projections are reconstructed and develop with a time course similar to that observed in vivo. Pregnant C57Bl/6 mice were injected twice daily with 40 mg/kg (±)-MDMA or saline from gestational day 6 to 13. On gestational day 14, mesencephalic and striatal cells from MDMA and saline-exposed embryos were used to prepare reaggregate cultures. Levels of monoamines and their metabolites in the reaggregates and culture medium were assessed at 22 and 36 days of culture. There was a long-term enhancement of serotonergic development and metabolism by fetal exposure to MDMA as evidenced by increased reaggregate serotonin levels as well as the elevated production and release of 5-hydroxyindoleacetic acid in cultures prepared from MDMA-exposed embryos that persisted for up to 36 days of culture. Dopaminergic neurons in such cultures also exhibited increased transmitter turnover as indicated by elevated levels of dihydroxyphenylacetic acid in reaggregate tissue and culture medium. The data obtained suggest that exposure to MDMA in utero during early to mid-gestation may result in more active serotonergic and dopaminergic neurons.

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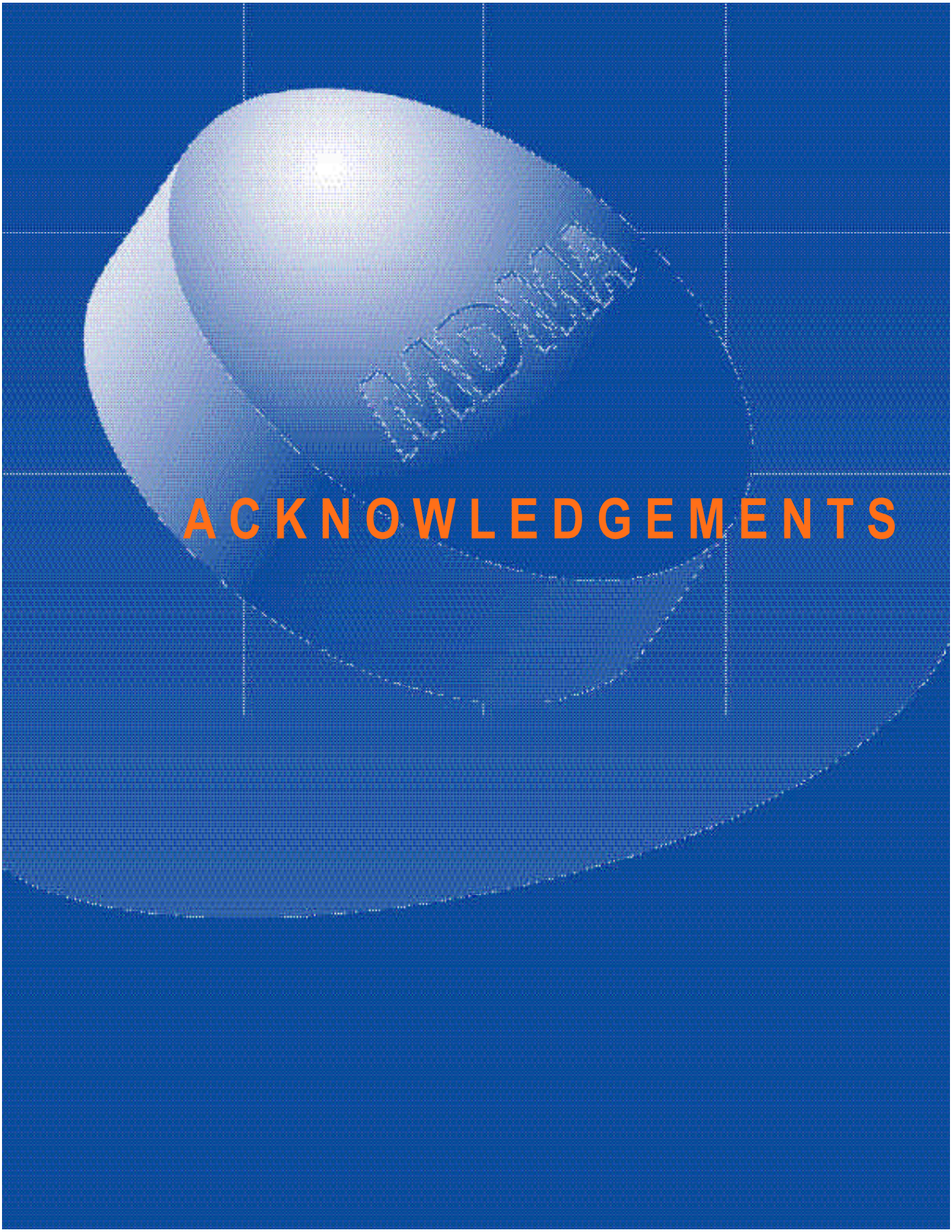

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