UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

OAKLAND DIVISION

VIETNAM VETERANS OF AMERICA, a Non-Profit Corporation; SWORDS TO PLOWSHARES: VETERANS RIGHTS ORGANIZATION, a California Non-Profit Corporation; BRUCE PRICE; FRANKLIN D. ROCHELLE; LARRY MEIROW; ERIC P. MUTH; DAVID C. DUFRANE; WRAY C. FORREST; TIM MICHAEL JOSEPHS; and WILLIAM BLAZINSKI, individually, on behalf of themselves and all others similarly situated,

Plaintiffs,

v.

CENTRAL INTELLIGENCE AGENCY; DAVID H. PATRAEUS, Director of the Central Intelligence Agency; UNITED STATES DEPARTMENT OF DEFENSE; LEON PANETTA, Secretary of Defense; UNITED STATES DEPARTMENT OF THE ARMY; JOHN MCHUGH, United States Secretary of the Army; and ERIC K. SHINSEKI, UNITED STATES SECRETARY OF VETERANS AFFAIRS,

Defendants.

Case No. CV 09-0037-CW

EXPERT REPORT OF UNA D. MCCANN, M.D.

I. INTRODUCTION

A. Retention

1. I have been retained by Morrison & Foerster LLP on behalf its clients, plaintiffs in this matter, Vietnam Veterans of America, Swords to Plowshares: Veterans Rights Organization, Bruce Price, Franklin D. Rochelle, Larry Meirow, Eric P. Muth, David C. Dufrane, Wray C. Forrest, Tim Michael Josephs, and William Blazinski (collectively "Plaintiffs") to serve as a consultant and expert witness in the above captioned action.

2. I expect to testify at trial regarding the matters discussed in this expert report, and in any supplemental reports or declarations that I may prepare for this matter. I may also testify at trial regarding matters related to my opinions addressed by any expert or fact witness testifying on behalf of Plaintiffs or Defendants Central Intelligence Agency; David H. Patraeus, Director of the Central Intelligence Agency; United States Department of Defense; Leon Panetta, Secretary of Defense; United States Department of the Army; John McHugh, United States Secretary of the Army; United States Department of Veterans Affairs; and Eric K. Shinseki, United States Secretary of Veterans Affairs (collectively "Defendants"), including but not limited to any reports, testimony, exhibits, references, or demonstratives presented by Defendants.

3. I reserve the right to supplement or amend this report if additional facts and information that affect my opinions become available. It is my understanding that Plaintiffs have retained other experts and that Defendants may serve expert reports concerning one or more of the issues I address in this report. I reserve the right to testify concerning such other reports or testimony, and to respond to any such report from Defendants' expert(s) and to rebut at trial any opinions expressed in such a report. I also understand that depositions of additional fact witnesses may take place and that Defendants have just recently produced or will be producing additional documents that are still undergoing review. Furthermore, it is my understanding that Defendants have produced, and continue to produce, a substantial quantity of documents and other information in formats that are inaccessible or exceedingly difficult to access or evaluate properly, and that Plaintiffs' counsel is continuing to attempt to convert such information into a usable format. Should Plaintiffs' counsel's efforts be successful and information from these

sources becomes available to me, I reserve the right to supplement this report to incorporate that information.

4. The headings in this report have been added to create sections for ease of organization. I do not intend these headings to be in any way restrictive of the information contained in the respective sections.

B. Compensation

5. I am being compensated for my work on this matter at my customary rate of $600 per hour, plus expenses. I am being compensated for travel time at a rate of $250 per hour up to a daily maximum of $1500. My compensation is not conditioned on the substance of my opinions, testimony at deposition or trial, or the outcome of this matter.

II. MY BACKGROUND AND QUALIFICATIONS

6. I earned my Bachelor of Arts degree in psychology *summa cum laude* in 1980 from Princeton University, where I was a member of the Phi Beta Kappa and Sigma Xi honor societies. I completed medical school at the Duke University School of Medicine, obtaining my M.D. in 1984. Following medical school, I performed my internship (1984-1985) and residency (1985-1988) in psychiatry at Stanford University School of Medicine. During the last year of my psychiatry residency (1987-1988), I was also a Moos Clinical Research Fellow in psychiatry at Stanford University.

7. My military career began at Princeton University, where I served for four years in the U.S. Army ROTC Academic Scholarship program. After completing my clinical and research training in psychiatry, I served as an officer in the United States Army. From 1988 to 1990, I served as a Captain in the U.S. Army and as an Investigator in the Continuous Operations Branch, Department of Behavioral Biology, at the Walter Reed Army Institute of Research. I then served from 1990 to 1992 as a Major in the U.S. Army and as Chief of the Continuous Operations Branch, Department of Behavioral Biology, at Walter Reed Army Institute of Research.

8. From 1992 to 1999 I served as a Lieutenant Colonel in the United States Public Health Service. During this time, I was Chief of the Unit on Anxiety Disorders, Biological Psychiatry Branch, National Institute of Mental Health—Intramural Research Program.

9. In 1999, I began my academic career at Johns Hopkins University. From 1999 to 2008, I was an Associate Professor of Psychiatry and Behavioral Sciences at the Johns Hopkins University School of Medicine. Since 2008, I have been a Professor of Psychiatry and Behavioral Sciences at Johns Hopkins. During my tenure at Johns Hopkins University School of Medicine, I have also served as the Director of the Anxiety Disorders Program and Clinic (2005 to the present) as well as Co-Director of the Center for Interdisciplinary Sleep Medicine and Research. As a Professor of Psychiatry at Hopkins, I have regular duties as Attending Physician on the inpatient psychiatric unit and hospital consult service.

10. I am certified by the American Board of Psychiatry and Neurology with subspecialty certification in psychosomatics. I currently hold a Maryland medical license.

11. A major focus of my work has been treating and investigating the neurotoxicity of psychoactive compounds. I have served as the Principal Investigator or a Co-Investigator of research grants including the following grants: "PET Studies of Amphetamine Neurotoxicity in Adult ADHD" (2010-2015), "PET and Sleep Studies in Methamphetamine Users" (2010-2015), "Effects of Zolpidem Extended-Release on Withdrawal and Sleep in Cannabis Users" (2008-2010), "Sleep and Nocturnal Endocrine Function in MDMA Users" (2002-2010), "Structural Brain Correlates of MDMA Use" (2004-2010), "PET Imaging MDMA Neurotoxicity" (2002-2009), "MDMA Neurotoxicity in the Primate" (1990-2007), "MDMA Neurotoxicity in Humans: Occurrence and Consequences" (1992-2007), "ADHD Treatment and Amphetamine Neurotoxicity" (2005-2008), "Methamphetamine Neurotoxicity in Nonhuman Primates" (2001-2006), "Gene Expression and Methamphetamine Neurotoxicity" (2000-2005), "Safety Assessment of Fenfluramine and Phentermine in Humans" (1997-2004), "PET Studies of Methamphetamine Neurotoxicity in Humans" (1998-2005), "Studies of Substituted Amphetamine Neurotoxicity" (1994-2005), and "PET Imaging of Dopamine Neurotoxicity With [^{11}C]WIN-35,428" (1994-1999).

12. Another major focus of my work has been the treatment and investigation of posttraumatic stress disorder, or PTSD. My publications include: "Repetitive transcranial magnetic stimulation for PTSD: Two Case Reports" (*Archives of General Psychiatry*, 1998),

"Prospective and retrospective life-charting in posttraumatic stress disorder (the PTSD-LCM)" (*Journal of Traumatic Stress*, 2001), "Repetitive TMS combined with exposure therapy for PTSD: A preliminary study" (*J Anxiety Disord*, 2008), and "Anxiety and Anxiety Disorders," In: Principles of Ambulatory Medicine, 7th Edition (book chapter, 2006). I have treated patients, including patients who served in the U.S. Armed Forces, for anxiety disorders and PTSD. I am currently consulting with Tonix Pharmaceuticals in preparation of a research study that will evaluate the utility of a novel formulation of cyclobenzaprine in the treatment of sleep and daytime symptoms of PTSD.

13. My work has encompassed the evaluation and treatment of psychedelic agents and other substances of abuse. My publications include: "Psychedelic drugs." In: Neuropsychopharmacology: The Fifth Generation of Progress (book chapter, 2002), "PCP/Designer Drugs/MDMA" in, Substance Abuse, In: Substance Abuse: A Comprehensive Textbook, 5th Edition (book chapter, in press), "Neurological complications of drugs of abuse." *In: Neurology and General Medicine* (book chapter, 2001), and "Long-lasting effects of recreational drugs on the central nervous system" (*The Neuroscientist*, 1998).

14. In addition to publishing original research articles in peer reviewed journals, review articles, and book chapters, I have been invited to present my research work at numerous professional meetings both in the United States and internationally. A current copy of my *curriculum vitae* is attached hereto as Exhibit 1, which includes a complete list of my publications to date.

15. I have not testified as an expert witness in any matter in the last four years.

III. BASIS AND SCOPE OF MY OPINIONS

16. I have been asked to provide an overview of the subjects presented in this report. Below I discuss posttraumatic stress disorder ("PTSD") and the role of PTSD as a mediator of a broad range of physical ailments. I also discuss medical and scientific literature and other evidence demonstrating that the mere participation in chemical and biological warfare programs could result in the development of PTSD and subsequent physical ailments. I also discuss some of the psychogenic compounds used in the U.S. military's chemical warfare program, including

LSD and phencyclidine. I have been asked to discuss the potential long-term adverse health effects from acute exposure to those psychogenic compounds. It is my understanding that many other psychogenic compounds were tested by the U.S. government in various chemical warfare programs, and I do not intend to suggest that my expertise is limited only to LSD and phencyclidine. A comprehensive discussion of all the psychogenic compounds tested by the U.S. government would be impractical in a single report, and I am not precluding the offering of additional opinions if compounds not discussed in this report become a subject for litigation. I may testify about any or all of these topics.

17. In arriving at my opinions, expressed in detail in this report, I have relied on my personal and professional experience as well as various additional resources. I have relied upon the types of information and resources that are normally relied upon by experts in my field, such as articles in peer reviewed journals, treatises and similar scholarly works, and published reports regarding the testing programs at issue. In particular, I have reviewed the documents and other resources cited in this report, as well as other documents and materials provided to me by counsel.

18. I have reviewed documents from various other sources that contain reports and accounts of actual tests involving LSD and other psychogenic compounds. These documents were helpful to my understanding of the circumstances surrounding the experiments performed in the various testing programs and example test protocols used.

19. These are some of the primary references I have reviewed and relied upon in reaching my opinions; a complete list of documents I have consulted and considered is included as Exhibit A to this report. Throughout my report I have cited specific documents, and portions of those documents, to illustrate technical and historical points. These citations are only illustrative, not exhaustive, and I may rely on other specific portions of these documents, as well as any of the references listed in Exhibit B to support any of these points. Moreover, to the extent Defendants provide an expert report responding to any of the points addressed in this report, I reserve the right to consider, comment on, or rely on any documents referenced in any such report.

20. I reserve the right to provide further exhibits to be used as a summary of, or as support for, my opinions or testimony, including any testimony by experts or other witnesses at trial.

21. With respect to the doses and pathways of exposure, I have reviewed data drawn from several sources, including a database printout of Edgewood test subjects between 1955 and 1975, excerpted data from the Chem-Bio Database that was provided to me, and a book written by one of the principal researchers at Edgewood Arsenal, Dr. James Ketchum.

IV. POSTTRAUMATIC STRESS DISORDER IN MILITARY TEST SUBJECTS

22. Individuals who served as test subjects in the U.S. military's chemical and biological warfare programs may have been vulnerable to developing posttraumatic stress disorder ("PTSD") in several ways. Subjects may have experienced traumatic acute or chronic adverse health effects caused by their participation in the tests, leading to the development of PTSD. The mere participation in such tests could also result in the development of PTSD. PTSD can develop even in individuals who were never exposed to active test agents (e.g., those receiving placebos) if those individuals *perceive* that they were exposed to active agents.

23. The adverse health effects from PTSD are not limited to adverse mental health effects. It is firmly established that PTSD can serve as a mediator or cause of a broad range of physical ailments. Therefore, individuals who develop PTSD may require health care for physical ailments in addition to psychological ailments.

A. BACKGROUND

1. Posttraumatic Stress Disorder

24. PTSD is one of the accepted anxiety disorders defined by the Diagnostic and Statistical Manual of Mental Disorders (hereinafter, "DSM-IV-TR").[1] The "essential feature" of PTSD is the development of characteristic symptoms and other problems following exposure to an "extreme traumatic stressor." (DSM-IV-TR at 463.) These symptoms and other problems are

[1] *See* American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*, § 309.81, "Posttraumatic Stress Disorder." Washington, DC, American Psychiatric Association, 2000.

extremely varied, and may include, but not be limited to, the following: avoidance patterns that interfere with interpersonal relationships, marital conflict, loss of job, impaired affect (emotional) modulation, self-destructive and impulsive behavior, dissociative symptoms (e.g., memory loss, depression, distorted perception of reality, anxiety), somatic (physical) complaints, shame, despair, hopelessness, hostility, social withdrawal, and feeling constantly threatened. (DSM-IV-TR at 465.) In addition, PTSD is associated with increased rates of major depression and substance abuse. (*Id*.) Furthermore, many patients with PTSD suffer from chronic somatic (bodily) complaints.

25. PTSD can occur at any age, and while symptoms usually begin within the first 3 months after the trauma, "there may be a delay of months, or even years, before symptoms appear." (DSM-IV-TR at 466.) Therefore, PTSD can be broadly categorized by three different "specifiers" depending on the onset and duration of the symptoms: 1) Acute—duration of symptoms is less than 3 months; 2) Chronic—symptoms last 3 months or longer; and 3) With Delayed Onset—at least 6 months have passed between the traumatic event and the onset of the symptoms. (DSM-IV-TR at 465.) It is well-accepted that PTSD "may be especially severe or long lasting when the stressor is of human design." (DSM-IV-TR at 464.)

26. Detailed diagnostic criteria have been outlined for PTSD. (DSM-IV-TR at 467-68.) Briefly, the criteria include: 1) exposure to a traumatic event; 2) persistent reexperience of the traumatic event (e.g., as "flashback episodes"); 3) persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness; 4) persistent symptoms of increased arousal (e.g., difficulty sleeping, irritability, outbursts of anger, difficulty concentrating, hypervigilance, and an exaggerated startle response); 5) duration of the disturbance is more than 1 month; and 6) the disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. (*Id.*)

2. PTSD as a Mediator of Physical Ailments

27. While PTSD is classified as a type of anxiety disorder, it is crucial to understand that the adverse effects from PTSD may not be limited to mental and psychological disturbances. In particular, there is a growing body of evidence that PTSD can serve as a mediator or cause of

physical ailments, and this is a rapidly expanding area of contemporary research. For example, Dr. Steven S. Coughlin of the U.S. Department of Veterans Affairs recently published a review of the current evidence linking PTSD to cardiovascular and cerebrovascular disease.[2] As Dr. Coughlin states, individuals with PTSD have "an increased risk of hypertension [high blood pressure], hyperlipidemia [high cholesterol], obesity, and cardiovascular disease." (Coughlin at 164, 169.) The mechanisms underlying many of these associations are not clear, but are well documented.

28. There is evidence outlining the mechanistic link between PTSD and cardiovascular disease. Hypervigilance or hyper-arousal is a well-known manifestation of PTSD. (*See* DSM-IV-TR at 468 (listing persistent symptoms of increased arousal as one of the diagnostic criteria for PTSD); *see also* Coughlin at 164.) Chronic hypervigilance and hyper-arousal can result in disturbances of the endocrine (hormonal) system through abnormal regulation of the hypothalamic pituitary adrenal axis ("HPA") and the autonomic nervous system (i.e., the "involuntary" nervous system that partly controls such physiologic functions as heart rate and blood pressure). (*See* Coughlin at 165.) Studies in military veterans with PTSD have demonstrated increased circulating levels of norepinephrine (better known among laypersons as "noradrenaline", one of the "fight-or-flight" neurotransmitters). (Coughlin at 164.) Elevated levels of norepinephrine and similar neurotransmitters (known as catecholamines) can change the function of platelets—the small blood cells that are crucial for forming blood clots—by increasing platelet adhesion and aggregation. (Coughlin at 165.) More blood clots can mean more heart attacks and strokes. (*See* Coughlin at 165-68.) Chronic disturbances of the HPA and overstimulation of the autonomic nervous system may also cause changes in immune function that lead to elevated levels of interleukin 6 ("IL-6"), tumor necrosis factor, and C-reactive protein—all mediators of inflammation that have been reported to stimulate atherosclerosis in

[2]S. Coughlin, "Post-Traumatic Stress Disorder and Cardiovascular Disease," *The Open Cardiovascular Medicine Journal* 5:164-170 (2011) (hereinafter, "Coughlin").

blood vessels. (Coughlin at 168.) Robicsek *et al.*[3] note that patients with PTSD "develop a low grade systemic inflammatory state" that may culminate in "metabolic syndrome, elevated blood pressure, obesity, dyslipidemia, [and] diabetes," all major risk factors for coronary artery disease. (Robicsek at 548.) Finally, sleep disturbance, another manifestation of PTSD, may be responsible for a number of daytime symptoms of PTSD, including irritability and somatic complaints.

29. Although the evidence linking PTSD to physical ailments is probably strongest for cardiovascular disease, there is also evidence linking PTSD to other physical ailments. For example, Glaesmer *et al.*[4] performed a study of 1456 elderly individuals examining the relationship between PTSD and physical health. Confirming earlier reports, the investigators found a significantly elevated risk for cardiovascular disease (angina pectoris/coronary artery disease, congestive heart failure, and peripheral vascular disease) and cardiovascular risk factors (hypertension and elevated cholesterol) in patients with PTSD. (Glaesmer at 403-04.) However, the investigators also found an increased risk in PTSD patients for such physical ailments as cancer, asthma, hearing loss, thyroid disorders, osteoporosis, and stomach disorders. (Glaesmer at 401, 403-04.) These findings were consistent with "strong evidence from previous studies for the association of PTSD with cardiac, respiratory, and digestive diseases." (Glaesmer at 404.) The authors cited as explanations for these findings "autonomic dysfunction and changes in the hypothalamic pituitary axis," but more generally, "[b]iologic changes, poor health behavior, and dysfunctional coping." (Glaesmer at 405.)

30. The growing body of evidence that PTSD may be a mediator or cause of physical ailments highlights the importance of providing comprehensive health care to individuals suffering from PTSD. It is not enough to evaluate and treat PTSD patients for mental and

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[3]O. Robicsek *et al.*, "Hypercoagulation in Chronic Post-Traumatic Stress Disorder," *Israel Medical Association Journal* 13:548-552 (2011) (hereinafter "Robicsek").

[4]H. Glaesmer *et al.*, "The Association of Traumatic Experiences and Posttraumatic Stress Disorder with Physical Morbidity in Old Age: a German Population-Based Study," *Psychosomatic Medicine* 73(5):401-406 (2011) (hereinafter "Glaesmer").

psychological disturbances. It is also crucial that PTSD sufferers receive effective evaluation and treatment for the many physical ailments that may have been caused in whole or in part by PTSD.

3. Effective Treatments for PTSD

31. Although a comprehensive discussion of the available treatments for PTSD is beyond the scope of this report, it is essential to understand that many effective treatments are available for treating the mental and psychological disturbances caused by PTSD. As the U.S. Department of Veterans Affairs states on its website, "Effective treatments for PTSD exist."[5] Since effective treatments exist, it is imperative to provide access to such treatments (discussed briefly below) to those U.S. military veterans suffering from PTSD as a result of their participation in chemical and biological testing. Effective treatments for PTSD may be discussed in two broad categories: psychological therapies and pharmacological treatments.

a. Psychological Therapies for PTSD

32. EXPOSURE THERAPY. One of the most commonly employed psychological interventions for the treatment of PTSD is exposure therapy. In exposure therapy, the patient is "guided through a vivid remembering of the trauma" until "extinction" of the patient's conditioned responses to the traumatic event occurs.[6] In patients who developed PTSD following, for example, an automobile accident, exposure therapy may include returning to the scene of the accident in order to reduce avoidance of the "trauma cues"—e.g., the memories, thoughts, feelings, and situations—associated with the accident site and to promote psychological "mastery" over these cues that trigger and maintain the patient's PTSD. (Keane *et al.* at 178-79.) However, if it is not possible to physically revisit or recreate the site of the traumatic event, as would be the case for military veterans who participated in traumatic chemical and biological testing, exposure therapy can still be performed through "imaginal" exposure, where the patient is

[5]U.S. Department of Veterans Affairs website, "Treatment [for PTSD]," available at: http://www.ptsd.va.gov/public/pages/gen-treatment.asp (accessed March 14, 2012).

[6]T. Keane *et al.*, "Posttraumatic Stress Disorder: Etiology, Epidemiology, and Treatment Outcome," *Annual Review of Clinical Psychology* 2:161-197, at 178 (2006) (hereinafter, "Keane *et al.*").

asked to imagine and discuss the traumatic site and event. (Kean *et al.* at 179.) As with physical (or *in vivo*) exposure, the purpose of imaginal exposure is to reduce avoidance and promote mastery over the traumatic cues that trigger and maintain the patient's PTSD. There is strong and ample clinical evidence demonstrating the effectiveness of exposure therapy as a treatment for PTSD. (*See* Keane *et al.* at 180-83.) As the U.S. Department of Veterans Affairs states, exposure therapy "works for many people who have experienced trauma."[7]

33. ANXIETY MANAGEMENT TRAINING. Anxiety management training teaches patients an assortment of behavioral and cognitive skills that help them to manage the emotions associated with PTSD. (Keane *et al.* at 179.) Such skills may include relaxation training, breathing retraining, trauma education, guided self-dialogue, cognitive restructuring (a set of techniques for becoming more aware of one's thoughts and for modifying them when they become troubling), communications skills training, and anger management training. (*Id.*) As with exposure therapy, there is strong clinical evidence demonstrating the effectiveness of anxiety management training as a treatment for PTSD. (*See* Keane *et al.* at 180-83.)

34. COGNITIVE THERAPY. The term "cognitive therapy" encompasses a broad range of treatments for patients with PTSD. Cognitive therapy aims to modify certain "cognitive distortions" a patient may have regarding issues of safety, trust, power, control, self-esteem, and intimacy. (Keane *et al.* at 180.) Often combined with other treatment approaches as part of a combination therapy for PTSD (see below), cognitive therapy has been proven to be a highly effective treatment for PTSD. (*See* Keane *et al.* at 180-83.)

35. COMBINATION TREATMENTS. Today, many PTSD patients are treated with some form of combination treatment that combines some of the psychological interventions discussed above. One type of combination therapy is cognitive processing therapy, which combines elements of exposure therapy, anxiety management training, and cognitive therapy. (Keane *et al.* at 179.) The evidence supporting the effectiveness of cognitive processing therapy

[7]U.S. Department of Veterans Affairs website, "Prolonged Exposure Therapy," available at: http://www.ptsd.va.gov/public/pages/prolonged-exposure-therapy.asp (accessed March 14, 2012).

and other combination therapies is strong. (*See* Keane *et al.* at 180-83.) Indeed, the U.S. Department of Veterans Affairs utilizes a form of cognitive processing therapy, and describes it as "one of the most effective treatments for PTSD."[8]

b. Pharmacological Treatments for PTSD

36. Although psychological therapies remain the mainstay of treatment for PTSD, a growing understanding of the biology of PTSD has highlighted the importance of pharmacological treatments. Today, there is solid evidence supporting the use of a broad range of medications in the treatment of PTSD. The Department of Veterans Affairs sometimes combines pharmacological treatment, such as selective serotonin reuptake inhibitors (see below), with psychological therapies in their treatment of U.S. military veterans with PTSD.[9]

37. ANTIDEPRESSANTS. Antidepressant medications are the best studied pharmacologic agents for the treatment of PTSD. Particularly effective are selective serotonin reuptake inhibitors, such as sertraline (e.g., Zoloft®) and paroxetine (e.g., Paxil®), both approved by the Food and Drug Administration for the treatment of PTSD.[10] In addition to this newer class of antidepressant medications, older types of antidepressants, such as tricyclic antidepressants (e.g., imipramine and amitriptyline) and monoamine oxidase inhibitors (e.g., phenelzine and brofaromine) have also been effective in some patients, although fewer studies have been performed looking at these older agents. (Keane *et al.* at 184.)

38. ANTIADRENERGIC AGENTS. As discussed above, PTSD can result in the excessive release of certain hormones, including norepinephrine ("noradrenaline"), one of the "fight-or-flight" hormones of the "adrenergic" system. This understanding about the physiologic effects of PTSD led researchers to investigate the use of antiadrenergic agents to treat PTSD.

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[8]U.S. Department of Veterans Affairs website, "Cognitive Processing Therapy," available at: http://www.ptsd.va.gov/public/pages/cognitive_processing_therapy.asp (accessed March 14, 2012).

[9]*See* the booklet, "Understanding PTSD Treatment," National Center for PTSD, U.S. Department of Veterans Affairs, February 2011, at 2.

[10]*See, e.g.*, the July 2011 version of the prescribing label for Paxil® and the September 2011 version of the prescribing label for Zoloft®.

Prazosin (i.e., Minipress®) is an antiadrenergic agent that has been found to reduce nightmares, improve sleep, and reduce overall symptoms in some military veterans with PTSD. (Keane *et al.* at 185.) More research needs to be done using these agents, but this class of medications represents a promising subject of future study in the treatment of PTSD. (*See id.*)

39. OTHER MEDICATIONS. Anticonvulsants (i.e., anti-seizure medications) have been shown in some studies to be possibly effective in the treatment of PTSD, although these medications often have significant side effects. Antipsychotic medications have also been studied as potential treatments for PTSD. More research is warranted in the use of these agents for treating PTSD before any recommendation can be made for their widespread use. (*See* Keane *et al.* at 185.)

B. PTSD AND RESULTANT PHYSICAL AILMENTS CAUSED BY PARTICIPATION IN CHEMICAL AND BIOLOGICAL TESTING PROGRAMS

1. PTSD Among World War II Mustard Gas and Lewisite Test Subjects

40. There is evidence that mere participation in the U.S. government's chemical and biological testing programs can in many cases cause the development of PTSD, and subsequent physical ailments, in test subjects. One of the earliest studies to demonstrate this was conducted by Schnurr *et al.*[11] Two of the three authors of that study, Dr. Paula Schnurr and Dr. Matthew Friedman, were affiliated with the Department of Veterans Affairs. (Schnurr 1996 at 131.) Schnurr *et al.* evaluated 24 veterans who had participated in the U.S. military's mustard gas and Lewisite[12] program during World War II. (*Id.*) The primary objective of Schnurr *et al.*'s study was to "assess PTSD among participants in the mustard gas program." (Schnurr 1996 at 132.) A secondary objective was to "assess other psychological, psychosocial, and physical health outcomes" in these veterans. (*Id.*) The authors concluded that the "men who participated in WWII mustard gas test experiments had poor mental, physical, and functional health, relative to

[11] P. Schnurr *et al.*, "Post-Traumatic Stress Disorder among World War II Mustard Gas Test Participants," *Military Medicine* 3:131-136 (1996) (hereinafter, "Schnurr 1996").

[12] In their article, Schnurr *et al.* use the term "mustard gas" to mean both mustard gas and Lewisite. (Schnurr 1996 at 131.)

norms" (i.e., men of similar age). (Schnurr 1996 at 131, 135.) The investigators "observed PTSD related to the mustard gas tests and were able to distinguish this condition from PTSD due to other traumatic events." (Schnurr 1996 at 135.) Schnurr *et al.* scored the test subjects' PTSD symptoms on a quantitative scale and found that they were "similar to the scores of individuals in the community who discovered that they were living next to a toxic landfill, and slightly higher than those for survivors of the nuclear accident at Three Mile Island …." (Schnurr 1996 at 133.)

41. DESCRIPTION OF THE MUSTARD GAS/LEWISITE TESTS. Schnurr *et al.* described some of the mustard gas and Lewisite tests that these veterans had undergone. There were two main types of tests performed during the World War II program: chamber tests (called by military investigators, "man-break" tests) and field tests. (Schnurr 1996 at 131.) During chamber tests, the test subjects were provided with gas masks and special suits and required to enter a gas chamber and remain there from one to four hours. (*Id.*) In the chamber, the test subjects were exposed to mustard gas and/or Lewisite. One day after each chamber trial, the test subjects were examined for reddening of the skin (erythema), which indicated that the mustard gas and/or Lewisite had penetrated the suit and burned the skin. (*Id.*) The test subjects were then required to enter the chambers each day or every other day until they developed "moderate to intense" erythema. (*Id.*) During field tests, subjects were required to spend up to 72 hours traversing a field that had been bombed with mustard gas. (*Id.*) The men were further required to drop to the ground periodically so that they would have direct contact with surfaces contaminated with mustard gas. (*Id.*) Dropping to the ground also increased the test subjects' exposure to mustard gas vapor, which is heavier than air. (*Id.*) In both types of tests, subjects were sometimes required to participate with insufficient protection, forced to wear faulty equipment, or were not properly instructed in the use of the equipment. (*Id.*) The vast majority (83%) of test subjects experienced various physical symptoms following these experiments. (*Id.*) It is reasonable to assume that the physical injuries and symptoms caused by participation in these experiments contributed to the development of PTSD in many of these test subjects.

42. DOSE-RESPONSE RELATIONSHIP: Schnurr *et al.* were able to demonstrate a dose-response relationship between the number of exposures to mustard gas and/or Lewisite and

the subsequent diagnosis of PTSD. (Schnurr 1996 at 134-135.) Half of the test subjects evaluated eventually developed full PTSD (meeting all diagnostic criteria for PTSD in the *Diagnostic and Statistical Manual*) or subthreshold PTSD (having symptoms of PTSD and meeting most, but not all diagnostic criteria for PTSD). (Schnurr 1996 at 132-133.) Compared to test subjects who did not develop PTSD, the PTSD group had significantly more exposures to mustard gas and/or Lewisite: an average of 7.8 exposures versus 3.1 exposures. (Schnurr 1996 at 135.) This difference was statistically significant ($p = 0.04$). (*Id.*) Although the study by Schnurr *et al.* was relatively small, the investigators provided compelling quantitative evidence that the mere participation in mustard gas/Lewisite experiments could lead to the development of PTSD.

43. EFFECT OF THE SECRECY REQUIREMENT. About two-thirds of the test subjects evaluated by Schnurr *et al.* said they were required to keep their participation in the mustard gas experiments secret. (Schnurr 1996 at 133.) The investigators state that "not telling others about a traumatic event that one has experienced is related to increases in negative psychological and physical health outcomes." (Schnurr 1996 at 135.) Indeed, Schnurr *et al.* suggest that the secrecy requirement "may make mustard gas test participants more similar to survivors of childhood sexual and physical abuse and to adult rape victims, many of whom fear the stigma of disclosure or are threatened by perpetrators into remaining silent." (*Id.*) I agree with these statements and believe it is likely that the secrecy requirement contributed to the development of PTSD in at least some test subjects. Test subjects would have been forbidden to discuss the mustard gas experiments with family members, friends, and other individuals who form their personal support network, as well as physicians and psychologists who might have provided better care had they known of this history.

44. LACK OF INFORMED CONSENT. As noted by Schnurr *et al.*, only 22% of the test subjects understood that some danger was involved before participating in the experiments. (Schnurr 1996 at 131,133, Table I.) Full informed consent and a thorough understanding about the nature of the testing program and the agents being used might have mitigated some of the traumatic effects of the experiments for some of the test subjects. The development of PTSD

usually requires a traumatic event that creates a sense of "intense fear, helplessness, or horror." (DSM-IV-TR at 463.) Full informed consent and knowledge about a testing program and the agents being used may help alleviate any sense of fear, helplessness, and horror, and therefore help prevent the development of PTSD. In my opinion, a faulty or insufficient informed consent protocol could have contributed to the development of PTSD in test subjects.

45. PHYSICAL AILMENTS RELATED TO PTSD. Schnurr *et al.* observed that the former test subjects as a group "were less psychologically *and physically* healthy than expected for men of similar age." (Schnurr 1996 at 131 (emphasis added).) Compared to similar-aged males, the sample of former test subjects scored relatively poorly on the SF-36[13] patient health survey on general health perception, role dysfunction due to physical problems, energy/fatigue, and pain. (Schnurr 1996 at 133-34, Table III.) This finding is consistent with more recent research demonstrating that PTSD is a mediator of physical ailments.[14]

46. CONCLUSIONS. I agree with Schnurr *et al.*'s conclusion that "some men have PTSD due to their participation in the WWII mustard gas tests." (Schnurr 1996 at 135.) I also agree with their admonishment to "encourage recognition of the problem among the older veteran population." (*Id.*)

2. Confirming the Role of PTSD as a Mediator of Physical Ailments in World War II Mustard Gas and Lewisite Test Participants

47. Dr. Paula Schnurr and Dr. Matthew Friedman, while still affiliated with the Department of Veterans Affairs, performed a much larger follow-up study with several other investigators examining the development of PTSD and physical health problems in a group of 302 World War II veterans approximately 50 years after their participation in U.S. military mustard gas tests.[15] Their 2004 article was a follow-up to a preliminary 1997 report[16] in which

[13]A description of the SF-36 patient health survey is available at: http://www.sf-36.org/tools/SF36.shtml/ (accessed on August 2, 2012).

[14]See the discussion in section IV.A.2 *supra*.

[15]Ford *et al.*, "Posttraumatic Stress Disorder Symptoms, Physical Health, and Health Care Utilization 50 Years After Repeated Exposure to a Toxic Gas," *Journal of Traumatic Stress* 17(3):185-194 (2004) (hereinafter, "Schnurr 2004").

they had shown that "roughly one third of men who were exposed to mustard gas or Lewisite in secret tests during WWII developed PTSD." (Schnurr 1997 at 428.)

48. In the 2004 article, Schnurr and Friedman confirm that participation in mustard gas tests can lead to the development of PTSD and subsequent physical problems. (Schnurr 2004 at 189.) Importantly, they also demonstrated that the development of PTSD symptoms was significantly related to increased inpatient and outpatient health care utilization. (*Id.*) This much larger study confirmed their earlier findings that participation in mustard gas experiments can lead to the development of PTSD which in turn can lead to the development of physical ailments.

49. Schnurr and Friedman also offered a model that would explain the link between mustard gas and Lewisite test participation and the increased utilization of inpatient and outpatient health care services. (Schnurr 2004 at 187, Figure 1.) According to their model, the number of mustard gas/Lewisite test exposures and the development of immediate symptoms and signs (i.e., burns to the skin and eyes) are related to the development of PTSD symptoms. (Schnurr 2004 at 187, Figure 1, 189.) The development of PTSD symptoms is then related to the development of physical health problems and functional health status. (*Id.*) Physical health problems and functional health status are then related to the utilization of inpatient and outpatient health care services. (*Id.*) This model is no doubt a simplification of what actually happens in individual test subjects, but I believe it is a useful and accurate conceptual model for how PTSD resulting from participation in U.S. military chemical experiments can serve as a mediator of subsequent physical ailments and a resulting increase in the need for health care services.

3. Generalizability of the Findings of Schnurr and Friedman to Veterans Who Participated in Later Chemical Experiments

50. While the studies performed by Schnurr and Friedman examined U.S. military veterans who participated in mustard gas/Lewisite experiments during World War II, I believe their findings are applicable to veterans who participated in later experiments. One basis for this

[16]P. Schnurr *et al.*, "PTSD in WWII Mustard Gas Participants: A Preliminary Report," *Annals of the New York Academy of Sciences* 821:425-429 (1997) (hereinafter, "Schnurr 1997").

opinion is the similarity between some of the later studies and the testing programs performed during World War II. For example, the World War II veterans examined by Schnurr and Friedman were required to enter test chambers each day or every other day until they developed "moderate to intense" erythema. (Schnurr 1996 at 131.) In mustard gas tests performed at other sites, such as at Edgewood Arsenal between 1955 and 1965, test subjects "underwent up to 14 exposures to H [i.e., mustard gas] on different days and were removed from the tests when dermal erythema indicated garment leakage."[17] (NRC Volume 2 at 124.) As in the World War II experiments, many of the test subjects at Edgewood Arsenal experienced severe erythema, including to the genitalia, and some Edgewood Arsenal test subjects experienced vesication (blistering) of the skin. (NRC Volume 2 at 126-27.) Indeed, some of the skin injuries at Edgewood Arsenal "might have been severe enough to cause permanent scarring." (NRC Volume 2 at 127.) There is no question that the mustard gas tests must have been traumatic for some, and probably many, of the subjects. I would therefore conclude that some of the test subjects may have developed PTSD, and subsequent physical ailments, following their participating in mustard gas experiments.

51. Although Schnurr and Friedman focused their work on U.S. military veterans who participated in mustard gas and Lewisite experiments, I believe their findings are clearly applicable to veterans who participated in tests involving other chemical warfare agents as well as biological warfare agents. Experiments involving other test agents certainly had the potential to produce both psychological and physical trauma leading to the development of PTSD and subsequent physical ailments. For example, some test subjects at Edgewood Arsenal participated in inhalational studies involving the psychogenic compound, phencyclidine ("SNA"). (NRC Volume 2 at 71.) Some of these test subjects experienced "collapse and prostration," "incapacitation," nausea and vomiting, limb paresthesias, and memory impairment. (NRC

[17]National Research Council, "Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents: Volume 2, Cholinesterase Reactivators, Psychochemicals, and Irritants and Vesicants," National Academy Press (Washington, D.C. 1984) (hereinafter, "NRC Volume 2").

Volume 2 at 72.) Some test subjects "rapidly became noncommunicative." (*Id.*) It is reasonable to conclude that some of these test subjects found the experience to be traumatic and that some went on to develop PTSD. Other test subjects participated in experiments involving the hallucinogenic agent, LSD.[18] (LSD Follow-Up Study at 1.) Among the adverse reactions experienced by test subjects were "flashbacks," daily headaches for 6 months, "nervous exhaustion," weakness, depression, personality changes, anxiety, nightmares, paranoia, various neuroses, and even psychosis. (LSD Follow-Up Study at 48-61.) I understand, moreover, that Dr. Michael Kilpatrick of the Department of Defense testified that, based on his reading of Dr. Schnurr's "two studies in fairly small numbers of veterans who were test participants. . . . [it was] very clear if they didn't know what they were exposed to, believed that there was a requirement for secrecy, that those were predictors for subsequent PTSD."[19] (Kilpatrick Deposition Transcript July 6, 2011 at 154:19-24.) Again, it is reasonable to conclude that some of the test subjects found the experience with LSD to be traumatic and that some went on to develop PTSD. It is also reasonable to conclude that adverse reactions to other test agents (e.g., anticholinesterase or anticholinergic agents) could also lead to the development of PTSD and subsequent physical ailments if the test subjects found the experience to be traumatic.

52. Infectious organisms used in U.S. military biological testing programs can cause acute or long-term reactions that some test subjects may find traumatic. In addition, some infectious organisms have the ability to persist in the human body as chronic infections and this is well-known to the general public. Even perceived exposure to biological agents that can persist in the body could produce anxiety and fear among those who believe they were exposed to these

[18]U.S. Army Medical Department, "LSD Follow-Up Study Report," October 1980 (hereinafter, "LSD Follow-Up Study").

[19]I also understand that Dr. Kilpatrick described, based on his review of test records, one test subject "not [being] in touch with reality" and that "his being out of touch with reality could be called a psychotic breakdown." (Kilpatrick Transcript July 8, 2011 at 550:5–551:13.)

agents. I believe the following statement in a National Academies report[20] is particularly relevant for infectious organisms that may have been used in biological warfare experiments:

> Biological and chemical weapons function as more than simply agents of direct harm to life and bodily integrity. They are also psychological weapons. Their insidious mode of operation, and the lack of certainty about their presence and persistence, combine to create an unusual level of fear and stress among those believing themselves exposed to them. (Perceived Exposure to Biochemical Warfare Agents at 3.)

53. The National Academies has taken the position that even a *perceived* (not actual) exposure to a chemical or biological warfare agent could result in a psychogenic response like PTSD. (Perceived Exposure to Biochemical Warfare Agents at 10.) I agree with the position of the National Academies. There are some individuals, including those with anxiety disorders or obsessive-compulsive disorders, who may be particularly vulnerable to developing PTSD if they simply believe they have been exposed to a chemical or biological warfare agent. As the National Academies puts it, the "mechanism of PTSD's etiology is generally attributed to biologic as well [as] *cognitive* sources." (Perceived Exposure to Biochemical Warfare Agents at 12 (emphasis added). This means that even those subjects who did not know if they were actually exposed to any substances, or those individuals, if any, who were exposed only to placebos during biological or chemical testing, could develop PTSD and subsequent physical ailments, especially if those individuals had pre-existing risk factors that made them especially vulnerable to developing PTSD.

54. PTSD can be caused by any "traumatic stressor" and a very broad range of traumatic events have been reported as initializing events in the development of PTSD. (*See* DSM-IV-TR at 463-64, 467-68.) In my opinion, the findings of Schnurr and Friedman, as well as some of the statements issued by The National Academies, are generalizable to all veterans who served as test subjects in the U.S. military's chemical and biological warfare experiments.

[20]The National Academies, "Supplement: Health Effects of Perceived Exposure to Biochemical Warfare Agents," April 2004 (hereinafter, "Perceived Exposure to Biochemical Warfare Agents").

V. POTENTIAL LONG-TERM HEALTH EFFECTS FOLLOWING EXPOSURE TO PSYCHOGENIC COMPOUNDS

A. Potential Long-Term Health Effects of Lysergic Acid Diethylamide (LSD)

1. Background—Structure and Acute Physiologic, Psychological, and Sensory Effects of LSD

55. Lysergic acid diethylamide ("LSD", also known as LSD-25) is one of the most powerful hallucinogenic agents known and has no accepted legitimate medical use.[21] It is a synthetic hallucinogen that is manufactured from lysergic acid, a substance found in the fungus, ergot. LSD has a structure related to the neurotransmitter serotonin,[22] and exerts many of its effects through the serotonergic neuronal system. It is a very potent drug, with microgram quantities capable of producing significant psychogenic effects.

56. LSD can produce significant autonomic responses within an hour of ingestion. These commonly include pupillary dilation, diaphoresis (excessive sweating), piloerection (the "standing" of hair on the skin), tachycardia (elevated heart rate), bradycardia (decreased heart rate), respiratory stimulation, respiratory depressions, and nausea. (Eveloff at 369.)

57. The psychological and sensory responses to LSD have been well-described. Sensory responses may include distorted color perception and a fusion of sensory impressions ("synesthesia"—e.g., an auditory or tactile stimulus stimulating an experience of color). (*Id.*) Psychological responses may include depersonalization (i.e., feeling disembodied), pronounced mood fluctuations (feelings of euphoria or despair), "autistic withdrawal" and a preoccupation with one's own perceptions and thoughts, reduction of aggressive drives, and rage reactions. (*Id.*)

2. Background—Adverse Reactions to LSD

58. LSD has been used as a street drug since at least the 1960s, and so LSD's many adverse effects are well-known to the general public. Among the best known and well-

[21] For a general discussion of LSD and its illicit use, see the following DEA website: http://www.deadiversion.usdoj.gov/drugs_concern/lsd/lsd.htm (accessed August 4, 2012).

[22] H. Eveloff, "The LSD Syndrome: A Review," *California Medicine* 109(5):368-373, at 368-69 (1968) (hereinafter, "Eveloff").

documented adverse reactions are "flashbacks", unpredictable and unheralded episodes where an individual re-experiences the effects LSD without consumption of the drug, even if the last dose of LSD was taken months or even years before. (Eveloff at 369.) There are many case reports of LSD users having flashbacks or hallucinations more than ten years after their last dose of LSD, and in some instances changes in brain function have been demonstrated using modern techniques such as functional brain MRI.[23] Other well-documented adverse reactions include psychotic episodes, panic reactions, rage reactions, suicidal ideation[24], and actual suicides. (Eveloff at 370-72.)

3. Background—Importance of Expectation and Setting in Determining Individual Reactions to LSD

59. It has been known for decades that an individual's response to LSD administration can vary considerably depending on the information, if any, that the individual has been provided regarding LSD and its potential effects. The lack of any information prior to administration (i.e., a covert or involuntary administration of LSD) could result in a profoundly more disturbing experience with LSD (i.e., a particularly "bad trip"). In contrast, being fully informed about the effects of LSD can potentially mitigate most of the effects of the drug.

60. Also important is the setting in which LSD is administered. A setting that is friendly or safe (e.g., among friends, at home) can help to reduce any negative experiences, while a setting that is less comforting (e.g., as part of a military exercise or among strangers in a laboratory) could increase the risk of adverse reactions to LSD. The importance of expectation and setting should not be underestimated or ignored. A physician, Dr. Herbert Eveloff, nicely

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[23]E.g., G. Iaria *et al.*, "A case of persistent visual hallucinations of faces following LSD abuse: A functional Magnetic Resonance Imaging study," *Neurocase* 16(2):106-118 (2010) (hereinafter, "Iaria").

[24]E.g., G. Shoval *et al.*, "Substance Use, Suicidality, and Adolescent-Onset Schizophrenia: An Israeli 10-Year Retrospective Study," *Journal of Child and Adolescent Psychopharmacology* 16(6):767-775, 771 (2006) (suggesting that the association between LSD use and suicide attempts "may be related to its effect on depletion of serotonin, the neurotransmitter most associated with suicidal behavior and mood disorders" (citations omitted)).

summarized the importance of these factors, which he discussed as "The Effect of Expectation," in an article published in 1968:

> The form and intensity of the LSD experience is in large part shaped by the mental attitude ("set") of the subject and the setting in which the drug is taken. Anything that can influence the set or setting can be instrumental in determining the subjective experience that is reported—for instance, previous knowledge of expected response; comments by friends; surroundings at time of ingestion (that is, home, psychiatrist's office, research laboratory, party). The effect of subject expectation is so profound that it alone may suppress the entire LSD experience itself, including sensory responses if the subject is decidedly skeptical or otherwise unwilling to release himself to the drug. (Eveloff at 370.)

61. It was not just civilian physicians who recognized the impact of information and expectation in forming an individual's response to LSD. Dr. James S. Ketchum is a physician who administered and observed the effects of psychogenic compounds, including LSD, in U.S. military personnel at Edgewood Arsenal between 1961 and 1971.[25] (Ketchum Report at 31, Appendix C.) As Dr. Ketchum wrote, "When [LSD] is given in a favorable situation, in the presence of a reassuring individual, impulsive, undesirable behaviors and fear of insanity is less likely." (Ketchum Report at 14.) In contrast, Dr. Ketchum describes the risks and dangers of administering even "moderate" doses of LSD to an individual who is not adequately prepared or supported for the experience:

> Covert administration of LSD deprives the recipient of any protective set (i.e. psychological preparedness) to help him cope with subsequent aberrant ideas and impulses. Unexpected dosing is likely to cause severe anxiety and confusion. If aberrant behavior is precipitated, subsequent embarrassment and feelings of shame or guilt are predictable, especially in someone lacking prior experience with the drug.
>
> Thus, even moderate doses of LSD, if unknown to the recipient can be expected to lead to severe distress. (Ketchum Report at 14-15.)

62. While Dr. Ketchum's remarks concerned completely covert administration of LSD, in my opinion his comments apply to some extent to individuals who may have been

[25] J. Ketchum, "Psychiatric Evaluation of Wayne A. Ritchie for Sidney Bender, Esq.," December 5, 2001 (hereinafter, "Ketchum Report"). It is my understanding that Dr. Ketchum prepared this report in his role as an expert witness in a litigation matter.

insufficiently informed about the potential effects and adverse reactions of LSD or were required to receive LSD in relatively unsupportive or coercive settings. Such lack of informed consent or insufficient support could contribute to an individual's bad experience with LSD, possibly resulting in the subsequent development of PTSD and associated physical ailments.[26]

4. Background—Idiosyncratic Drug Reactions

63. Unusual or uncommon reactions to a drug are sometimes called *idiosyncratic* drug reactions, and it is now recognized that virtually any drug can cause idiosyncratic reactions in certain vulnerable individuals.

64. In the early years of street use of LSD as a "recreational" drug, it was thought by some researchers that some of the most serious adverse reactions to LSD (e.g., drug dependence, personality deterioration, psychoses, and suicide) were "suffered most frequently, *but not exclusively*, by those who have a preexisting emotional illness"—i.e., by those individuals who had some sort of pre-existing vulnerability to the adverse effects of LSD. (Eveloff at 372 (emphasis added).) However, just as important was the recognition that not all individuals who experienced these serious adverse reactions had some type of pre-existing vulnerability. Some individuals, for whatever reason (perhaps genetic), were prone to experience "bad trips" and other adverse reactions from LSD, even if given a relatively low dose in a friendly, supporting setting. Today, we might recognize these types of reactions as idiosyncratic adverse reactions to LSD.

65. In his LSD experiments on U.S. military personnel at Edgewood Arsenal, Dr. Ketchum also saw the great variability of individual responses to LSD at all dose levels. As Dr. Ketchum put it during testimony that he gave at a trial on April 8, 2005.[27]

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[26] It is my understanding that Dr. Ketchum testified in deposition that the Army did not disclose to soldiers that they were receiving LSD because the research was classified, and because the investigators did not want to bias the soldiers' reactions to LSD since the drug had been discussed in the popular press. *Ritchie v. United States*, United States District Court, Northern District of California, Case No. C00-3940, transcript of the August 5, 2003 deposition of James S. Ketchum (hereinafter, "Ketchum Deposition") at 23.

[27] *Ritchie v. United States*, Case No. C00-3940 MHP, United States District Court, Northern District of California, Transcript of Proceedings, April 8, 2005 (hereinafter, Ketchum Trial Testimony).

> There's a tremendous overlap between effects at various doses, as I pointed out by numerous authors. It can't be pinned down to 50 [micrograms of LSD] equals mild all the time or a hundred [micrograms of LSD] equals moderate all the time or 150 [micrograms of LSD] equals severe all the time ... the variation can be tremendous. As we observed, we got a great range of responses on our tests to every dose. (Ketchum Trial Testimony at 543.)

66. What this great and unpredictable variability in individual responses to LSD means is this: there is no such thing as a "safe" dose of LSD. An adult receiving a relatively high dose of LSD (e.g., 200 micrograms) might be able to tolerate the dose if well-informed in advance about LSD and its adverse effects and administered the drug in a supportive setting. Alternatively, an adult receiving a relatively small 50 microgram dose of LSD might have a very severe adverse reaction to the drug, even if the person had been fully informed about LSD and administered the drug in a supportive setting. Individual response to LSD is highly variable and unpredictable, and some individuals will have a severe, idiosyncratic adverse reaction to LSD even if a relatively small dose of the drug is administered in an optimal setting. Therefore, even the careful control of LSD dose and the use of optimal set (i.e., full informed consent) and setting in administering the hallucinogen cannot prevent all severe individual adverse reactions to the drug.

c. Conclusion

6. The 1980 LSD Follow-Up Study Report

74. The 1980 LSD Follow-Up Study Report prepared by the U.S. Army Medical Department is probably the most complete study of U.S. military test subjects who participated in LSD experiments at Edgewood Arsenal and at other sites between 1955 and 1967. (*See* LSD Follow-Up Study at 1.)

a. Lack of an Adequate Control Group

75. The study does have major methodological problems, including the lack of any matched control group. (LSD Follow-Up Study at Executive Summary and 4.) As the authors of the report note, the LSD test subjects "were not in any sense a random cross-section of the Army population." (*Id.*) The LSD test subjects were specially screened and were of above-average intelligence and more educated compared to the general Army population. (LSD Follow-Up Study at 4-5.) For some period of time at Edgewood, as Dr. Ketchum explained during his testimony that he gave at a trial on April 6, 2005:

> Even after they arrived, we further screened them after complete medical examinations including EKG, frequently EEG, electroencephalogram, total physical, complete battery of laboratory tests, psychiatric interview, then we graded them A, B, C and D. A was the top group, the astronaut group, you might say, whom we considered to be the most stable. And those were felt to be suitable for receiving large or medium doses of psychotropic and psychoactive drugs, such as LSD. Group B were suitable for low dose testing. Group C were approved for testing with other than psychoactive drugs? (sic) And group D were not considered suitable for testing with any drugs and only tested equipment." (Ketchum Trial Testimony at 223:21–224:9.)

76. It is reasonable to assume that these qualities discussed above made the LSD test subjects healthier and more psychologically fit as a group compared to the general Army population—what could be called a "healthy test subject effect." In addition, as I know from my personal and professional experience, individuals who are in military service tend to be physically healthier than the general U.S. population because military personnel undergo required health screening before entry into the military and must maintain a certain level of fitness to remain in the armed forces—a well-described phenomenon known as the "healthy soldier effect." Since an adequate matched control group could not be obtained, age-similar males in the general U.S.

population were used for comparisons with the LSD test subject group. (LSD Follow-Up Study at Executive Summary.) Because of the healthy soldier effect and healthy test subject effect, I believe that comparisons with the general U.S. population of males would not be meaningful.

77. The authors report that the frequency and type of medical illnesses and psychiatric illnesses were similar between the LSD test subject group and the general U.S. population of age-similar males. (*Id.*) For the reasons I stated above regarding the inadequacy of the control group used, in my opinion the comparisons are not clinically meaningful.

b. Use of Extremely High LSD Doses

78. One of the remarkable sets of numbers in the LSD Follow-Up Study is the reported range of LSD doses administered to test subjects. (LSD Follow-Up Study at 14.) In general, LSD doses of up to 1.5 micrograms per kg (approximately 100 micrograms total for the typical adult) or less can be considered a moderate to lower dose; a dose of around 2.0 micrograms per kg (approximately 150 micrograms total) can be considered a moderate dose; and a dose around 2.5 micrograms per kg (approximately 200 micrograms) could be considered a high, even incapacitating, dose. (*See* Ketchum Trial Testimony at 535.) In contrast, these doses administered to LSD test subjects ranged from 0.4 micrograms per kg (about 28 micrograms) to 75 micrograms per kg (about 5250 micrograms). (LSD Follow-Up Study at 14.) As discussed above, there is no such thing as a "safe" LSD dose, and such extremely high doses of LSD can only increase the risk of serious short-term and long-term adverse reactions in test subjects.

c. LSD Test Subjects Reporting Long-Term Health Effects

79. While I give little credence to the LSD Follow-Up Study's comparisons between the LSD test subject group and the general U.S. population of age-similar males, I do believe the study is worthwhile for its reports of long-term LSD-related adverse health effects. Among 320 LSD test subjects interviewed or examined, the authors found that fifty LSD test subjects (about 16%) had "probable" long-term LSD adverse effects. (LSD Follow-Up Study at 21.) The authors defined a "probable" LSD adverse effect as "one which was reported to have initially occurred within 2 years of LSD exposure and which is either similar to known long-term effects of LSD or could conceivably have been caused by LSD even if not previously reported." (*Id.*) Given the set

and setting of LSD exposure in military personnel, it is quite likely that many participants who were experiencing symptoms of PTSD did not report them in this time period. Similar phenomena have been seen in other populations (e.g., Vietnam veterans). In any case, it is notable that the authors acknowledge that so many LSD test subjects had adverse reactions that were probably attributable to LSD exposure in U.S. government tests from 1955 to 1967.

d. Flashbacks

80. The most common "probable" LSD adverse reaction was flashbacks, which the authors defined as the "spontaneous, transient occurrence of experiences reminiscent of the symptoms evoked by LSD exposure." (*Id.*) Twenty-four LSD test subjects reported having flashbacks, including 13 test subjects who reported their symptoms to be present up to the time they were interviewed for the LSD Follow-Up Study. (*Id.*) At least eleven LSD test subjects had reported the flashbacks to persist for an average of 18 years following their last exposure to LSD. While the authors claimed (in 1980) that the "generally accepted upper limit for the duration of flashbacks is about 2 years from the date of last exposure to LSD," (LSD Follow-Up Study at 49) this is no longer accepted today. Indeed, many cases of persistent flashbacks beyond two years have been reported, and the diagnosis of persistent flashbacks is now well-established as "Hallucinogen Persisting Perception Disorder."[29] It is also notable that most of the LSD test subjects reporting flashbacks as a long-term adverse reaction had received just a single dose of LSD. (LSD Follow-Up Study at 48.)

e. Somatic Complaints

81. The authors of the LSD Follow-Up Study reported that some LSD test subjects had somatic complaints, including headaches, "nervous exhaustion," and sexual impotence, following LSD exposure. (LSD Follow-Up Study Report at 49-51.) Those somatic complaints may be attributable to LSD exposure, but it is also possible that some LSD test subjects

[29]DSM-IV-TR at 253-254; *see also*, J. Halpern and H. Pope, "Hallucinogen persisting perception disorder: what do we know after 50 years?," *Drug and Alcohol Dependence* 69:109-119 (2003).

developed PTSD following their participation in government LSD studies and subsequently developed physical ailments caused by PTSD.[30]

f. Depression

82. Depression is a well-described and -documented adverse reaction to LSD exposure, and it is not surprising that this long-term health effect was observed in the LSD test group. (LSD Follow-Up Study at 21.) As the authors note, depression "is probably the most commonly reported prolonged reaction to LSD among normal research subjects." (LSD Follow-Up Study at 51.) Suicide is the most serious potential outcome from depression, and there were several cases among the LSD test subjects of suicide attempt, suicide gesture, or suicidal ideation. (LSD Follow-Up Study at 21.) It is interesting to note that two LSD test subjects were excluded from the analysis because they had died from non-combat duty gunshot wounds: one from a self-inflected gunshot wound and another who was shot "under unexplained circumstances." (LSD Follow-Up Study at 15.) It is possible that those two LSD test subjects experienced post-LSD depression.

g. Personality Changes

83. As the authors state, both "transient and long-term personality changes are frequently reported following LSD ingestion." (LSD Follow-Up Study at 54.) Among the potentially debilitating long-term personality changes reported by the LSD test subjects were social withdrawal, loss of interest in work, irritability, and aggressiveness. (LSD Follow-Up Study at 54-55.) One LSD test subject developed a tendency towards violent outbursts. (LSD Follow-Up Study at 55.) All of these symptoms are commonly seen in patients with PTSD.

[30] A National Research Council report briefly discussed results that seem to support these findings. The NRC panel observed "statistically significant increases in admissions to VA hospitals and Army hospitals for nervous system and sense organ disorders among men exposed to LSD." National Research Council, "Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents, Volume 3, Final Report: Current Health Status of Test Subjects" National Academy Press (Washington, D.C. 1984) (hereinafter, NRC Volume 3) at Executive Summary.

h. Other Long-Term Adverse Reactions to LSD

84. The authors reported a number of other long-term adverse reactions to LSD among the test subjects: anxiety, nightmares, paranoia, alcohol abuse, polydrug abuse[31], episodic withdrawal, acute confusional state, and seizure disorder. (LSD Follow-Up Study at 22.) It is worth observing that many of these reported adverse reactions could also be attributable to PTSD, which could have developed in test subjects who found their experience with LSD to be traumatic.

i. Conclusions

85. The LSD Follow-Up Study provides ample evidence that even a single exposure to LSD during a testing program can result in serious long-term adverse health effects, particularly if subjects were not appropriately informed regarding the substance to be tested and its potential effects. Furthermore, in my opinion, the data presented in the LSD Follow-Up Study provides evidence supporting the possibility that some test subjects developed PTSD, and possibly related physical ailments, following exposure to LSD. Because the U.S. general population of males was not a suitable or appropriate control group for the LSD test subjects, I do not believe the comparisons between the LSD test subject group and the general U.S. population are worthy of serious consideration.

B. Long-Term Health Effects of Other Psychoactive Compounds

86. It is my understanding that the U.S. government evaluated a variety of psychoactive compounds other than LSD (e.g., cannabinoids) as potential chemical warfare agents. I have focused my report on LSD since it is not practically possible to discuss every psychoactive compound in depth here in this report.

87. PHENCYCLIDINE. One of the psychoactive compounds assessed as a possible chemical warfare agent is phencyclidine. (NRC Volume 2 at 47.) A number of long-term or delayed effects of phencyclidine exposure have been reported, including in anesthesiology patients who may have had just one exposure to the drug. (*See* NRC Volume 2 at 67.) Among

[31] A NRC panel observed "increased use of LSD after the Edgewood tests" among test subjects. (NRC Volume 3 at Executive Summary.)

the potential long-term adverse reactions to phencyclidine exposure are psychotic reactions. I also agree with the NRC panel that subtle impairment of cognitive functioning or impairment of complex psychomotor skills cannot be ruled out. (NRC Volume 2 at 67, 70.)

C. Additive and Synergistic Adverse Effects From Exposure to Multiple Drugs

88. It is my understanding that some test subjects were exposed to multiple drugs, sometimes in sequence and sometimes in combination. It is well-known today that exposure to multiple drugs, whether in sequence or in combination, could lead to serious additive or synergistic adverse effects. Individual reactions to multiple drug exposures can be highly variable and unpredictable, and so it is often difficult to predict which drug combinations may be harmful to a particular individual. In general, exposure to multiple drugs in sequence or combination has the potential to increase an individual's risk for experiencing serious acute and long-term adverse health effects.

VI. COMMENTS ON THE DEPARTMENT OF VETERANS AFFAIRS OUTREACH LETTER

89. I have had the opportunity to review the Department of Veterans Affairs Outreach Letter dated June 30, 2006, as well as the accompanying Fact Sheet and frequently asked questions ("FAQs").

90. I believe the Fact Sheet is inaccurate with respect to LSD testing when it states that the "program evaluated the effects of low-dose exposures to chemical agents." As discussed above, some of the LSD doses tested by the U.S. military were extremely high (over 5,000 micrograms of LSD).

91. Similarly, I believe the Fact Sheet is inaccurate in stating that "study investigators assured that the exposure levels administered would not result in serious or life-threatening side effects." As discussed above, there is no "safe" dose of LSD. Furthermore, there could have been serious idiosyncratic adverse reactions to other drugs that were tested.

92. The FAQ states that although "the current medical literature indicates that such exposure [to hallucinogenic drugs like LSD] may have some long-lasting effects among some individuals, such as "flashbacks" (visual hallucinations without new drug exposure), the volunteer

records from the times of the Edgewood studies did not record these kinds of after effects among the Edgewood study volunteers." I understand, however, that during deposition, Dr. Kilpatrick of the DOD agreed that "the Army's own test results from Edgewood reported flashbacks among persons exposed to LSD." (Kilpatrick Transcript July 8, 2011 at 661:14-17.) Furthermore, as discussed above, the LSD Follow-Up Study did perform later interviews and examinations after "the times of the Edgewood studies" that clearly did demonstrate some long-lasting adverse effects from exposure to LSD.

VII. CONCLUSION

93. Subjects in the U.S. government's chemical and biological testing programs may have developed PTSD.

94. PTSD can serve as a mediator of physical ailments. Therefore, test subjects who

developed PTSD following their participation in the chemical and biological testing programs can subsequently develop physical ailments mediated by PTSD.

95. Subjects in the LSD testing programs may develop one of a number of well-documented and well-described long-term health effects from LSD exposure, including flashbacks and depression.

Respectfully submitted,

Dated: August 7, 2012

Una D. McCann, M.D.

Exhibit A

CURRICULUM VITAE FOR ACADEMIC PROMOTION

The Johns Hopkins University School of Medicine

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Una D. McCann Date of this version: 2-29-2012

DEMOGRAPHIC INFORMATION

Current Appointments

Department of Psychiatry and Behavioral Sciences
Johns Hopkins University School of Medicine
Johns Hopkins Bayview Medical Center
Johns Hopkins Hospital

Personal Data

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Department of Psychiatry and Behavioral Sciences
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Education and Training

A.B.	1976-1980	Princeton University (Psychology)
M.D.	1980-1984	Duke University School of Medicine
	1984 - 1985	Intern (Psychiatry), Stanford University School of Medicine
	1985 - 1988	Resident (Psychiatry), Stanford University School of Medicine
	1987 - 1988	Clinical Research Fellow, Stanford University School of Medicine

Professional Experience

1988 -1990	Captain, United States Army and Investigator Continuous Operations Branch, Dept. of Behavioral Biology Walter Reed Army Institute of Research
1990-1992	Major, United States Army and Chief Continuous Operations Branch, Dept. of Behavioral Biology Walter Reed Army Institute of Research
1992-1999	Lieutenant Colonel, Public Health Service and Chief Unit on Anxiety Disorders, Biological Psychiatry Branch, National Institute of Mental Health-Intramural Research Program
1999- 2/2008	Associate Professor of Psychiatry and Behavioral Sciences Johns Hopkins University School of Medicine
2/2008-present	Professor of Psychiatry and Behavioral Sciences Johns Hopkins University School of Medicine
2005-present	Director, Anxiety Disorders Program and Clinic Johns Hopkins University School of Medicine
2008-present	Co-Director, Center for Interdisciplinary Sleep Medicine and Research; Johns Hopkins School of Medicine

RESEARCH ACTIVITIES

Publications

- Peer-reviewed original research articles

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108. Mueller M, Yuan J, Maldonado Adrian C, **McCann UD**, Ricaurte GA. Inhibition of 3,4-methylenedioxymethamphetamine metabolism leads to marked decrease in 3,4-dihydroxymethamphetamine formation but no change in serotonin neurotoxicity: Implications for mechanisms of neurotoxicity. Synapse. 2011 Oct;65(10):983-90.

109. Kleykamp BA, Griffiths RR, **McCann UD**, Smith MT, Mintzer MZ. Acute effects of zolpidem extended-release on cognitive performance and sleep in healthy males after repeated nightly use. Exp Clin Psychopharmacol. 2012;20(1):28-39.

- **Review Articles**

1. Agras SW, **McCann UD**. The efficacy and role of antidepressants in the treatment of bulimia nervosa. *Annals of Behavioral Medicine* 1987; 9(4):18-21.

2. Steele T, **McCann UD**, Ricaurte G. 3,4-Methylenedioxymethamphetamine (MDMA, "Ecstasy"): Pharmacology and toxicology in animals and humans. *Addiction*, 1994; 89(5):539-51.

3. **McCann UD**, Ricaurte GA. On the neurotoxicity of MDMA and related amphetamine derivatives, *J Clinical Psychopharmacology*, 1995; 15:295-296.

4. Jobson KO, Davidson JR, Lydiard RB, **McCann UD** et al. Algorithm for the treatment of panic disorder with agoraphobia. *Psychopharmacol Bull*, 1995; 31(3):483-5.

5. **McCann, UD**. Sleep and Panic Disorder, *Anxiety Disorders Association of America Reporter*, 1995; VII (1):23-24.

6. **McCann UD**, Slate SO, Ricaurte GA. Adverse reactions with ((±)) 3,4-methylenedioxymethamphetamine (MDMA, "Ecstasy"), *Drug Safety*, 1996; 15(2):107-15.

7. **McCann UD**, Seiden LS, Rubin LJ, Ricaurte GA. Brain serotonin neurotoxicity and primary pulmonary hypertension: Potential adverse effects of fenfluramine (Pondemin) and dexfenfluramine (Redux), *Journal of the American Medical Association*, 1997; 278:666-672.

8. **McCann UD**, Lowe KA, Ricaurte GA. Long-lasting effects of recreational drugs on the central nervous system, *The Neuroscientist*, 1998; 3(6):399-411.

9. **McCann UD**, Eligulashvili V, Ricaurte G. Adverse neuropsychiatric events associated with dexfenfluramine and fenfluramine, *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 1998;22:1087-1102.

10. Post RM, Kimbrell T, **McCann UD**, Dunn R, Osuch E, Speer A, George M, Weiss SRB. Repetitive transcranial magnetic stimulation as a neuropsychiatric tool: Present status and future potential, *Journal of Electroconvulsive Therapy*, 1999; 15(1):39-59.

11. Ricaurte GA, Yuan J, **McCann UD**. (±) 3,4-Methylendioxymethamphetamine (MDMA, "Ecstasy")-induced serotonin neurotoxicity: Studies in animals. *Neuropsychobiology* 2000; 42(1): 5-10.

12. **McCann UD**, Ricaurte GA. Drug Abuse and Dependence: Hazards and consequences of heroin, cocaine and amphetamines. Current Opinion In Psychiatry, 2000; 13(3)321-325.

13. Bobes J, **McCann UD**. Developments in the treatment of drug dependence. *Current Opinion in Psychiatry*, 2000; 13(3): 333-338.

14. **McCann UD**, Ricaurte GA. Experimental studies on MDMA and its potential to damage brain serotonin neurons. *Neurotoxicity Research*, 2001; 3:85-89.

15. Shekhar A, **McCann UD**, Meaney MJ, Blanchard DC, Davis M, Frey KA, Liberzon I, Overall KL, Shear MK, Tecott LH, Winsky L. Summary of a National Institute of Mental Health workshop: developing animal models of anxiety disorders. *Psychopharmacology* (Berl). 2001; 157(4):327-39.

16. **McCann UD**, Stotland N. Toward optimal health: the experts provide a current perspective on anxiety in women. *J Womens Health* (Larchmt). 2003; 12(5):443-7.

17. **McCann UD**, Seiden L, Ricaurte GA. Amphetamine neurotoxicity: Accomplishments and remaining challenges. *Neuroscience and Biobehavioral Reviews*, 2004; 27(8):821-826.

18. Ricaurte GA, **McCann UD**. Recognition and management of complications of new recreational drug use. *Lancet*. 2005;365(9477):2137-45.

19. **McCann UD**, Fauerbach JA, Thombs BD. Anxiety and cardiac disease. *Primary Psychiatry*, 2005;12(3):47-50.

20. Thombs BD, Fauerbach JA, **McCann UD**. Stress disorders following traumatic injury: Assessment and treatment considerations. *Primary Psychiatry*, 2005;12(3):51-55.

21. Ator NA, **McCann UD**. New insights into the $GABA_A$ receptor. *CNS Spectrums*, 2005; 10(20):20-28.

22. Bush DE, Ziegelstein RC, Patel UV, Thombs BD, Ford DE, Fauerbach JA, **McCann UD**, Stewart KJ, Tsilidis KK, Patel AL, Feuerstein CJ, Bass EB. Post-myocardial infarction depression. *Evid Rep Technol Assess (Summ)*. 2005;(123):1-8.

23. Thombs BD, Magyar-Russell G, Bass EB, Stewart KJ, Tsilidis KK, Bush DE, Fauerbach JA, **McCann UD**, Ziegelstein RC. Performance characteristics of depression screening instruments in survivors of acute myocardial infarction: Review of the evidence. *Psychosomatics*, 2007;48(3):185-94.

24. **McCann UD**, Ricaurte GA. Effects of (+/-) 3,4-methylenedioxymethamphetamine (MDMA) on sleep and circadian rhythms. *ScientificWorldJournal*. 2007;7:231-8.

- **Editorials**

1. **McCann UD,** Ricaurte GA, Molliver ME. MDMA ("Ecstasy") and serotonin neurotoxicity: New findings raise more questions. *Arch Gen Psychiatry*, 2001; 58(10):907-8.

2. Ricaurte GA, **McCann UD**. Assessing long-term effects of MDMA (Ecstasy). *Lancet*. 2001; 358(9296):1831-2.

- **Book Chapters**

1. Krishnan RKR, **McCann UD**, France RD. Substance abuse in chronic pain patients: in *Chronic Pain* (France RD and Krishnan RKR, Eds) Washington DC, American Psychiatric Press, 1988; 220-227.

2. **McCann UD**, Ricaurte GA. Strategies for detecting partial monoamine depletions in humans. *NIDA Research Monograph*, 1993; 136:35-52.

3. Ricaurte GA, **McCann, UD**. Clinical management of opiate overdose and dependence. *Current Therapeutics and Neurologic Disease*. 4th Edition. Johnson D, Griffin J, eds. Mosby Yearbook Company, St. Louis, MO, 1994; 302-30.

4. **McCann UD**, Ricaurte GA. Use and abuse of ring-substituted amphetamines. In: *Amphetamine and its Analogs: Neuropsychopharmacology, Toxicology and Abuse*, Cho A and Segal D (eds), Academic Press, New York, 1994; 371-381.

5. **McCann UD**, Mertl MM, Ricaurte GA. Methylenedioxymethamphetamine (MDMA, "ecstasy"), In: *Sourcebook on Substance Abuse: Etiology, Methodology, and Intervention* (RE Tarter, RT Ammerman and PJ Ott, eds). Allyn & Bacon, New York, 1998; 567-577.

6. **McCann UD**, Ricaurte GA. *Aproximacion a la neurobiologia y neurotoxicidad comparada inducida por la MDMA.* In:Extasis (MDMA): Un abordaje comprehensivo (J Bobes Garcia, P Lorenzo Fernandez, PA Saiz Martinez, eds). Psiquiatria Medica, Masson, SA, Madrid, 1998; 89-97.

7. **McCann UD**, Langston JW, Ricaurte GA: Neurological complications of drugs of abuse. *In: Neurology and General Medicine,* Aminoff MJ (ed), Churchill Livingstone, N Y, (Revision/Update), 2001.

8. **McCann UD**, Anxiety and Anxiety Disorders, In: Principles of Ambulatory Medicine, 6th Edition (LR Barker, J Burton, PD Zieve, eds.), Lipincott Williams and Wilkins, Philadelphia, 2002.

9. **McCann UD**, Szabo Z, Ricaurte GA. Neuroimaging of MDMA-Induced Neurotoxicity, in: Neurotoxicology Handbook, Volume II; Neuroimaging Section. Totowa, NJ, Humana Press, Inc., 2002

10. Abraham HD, **McCann UD**, Ricaurte GA. Psychedelic drugs. In: Neuropsychopharmacology: The Fifth Generation of Progress (Davis KL. Charney D, Coyle JT, Nemeroff C, eds).Lipincott Williams and Wilkins, Philadelphia, 2002; 1545-1556.

11. **McCann UD**, Ricaurte GA: Imaging Studies of MDMA-induced Neurotoxicity, Monografia Drogas Recreativas Addicciones Vol: 15, Suppl. 2 2003; p. 111-120.

12. **McCann UD**, Bienvenu OJ. Anxiety and Anxiety Disorders, In: Principles of Ambulatory Medicine, 7th Edition (LR Barker, NH Fiebach, DE Kern, PA Thomas, RC Ziegelstein, eds), Lipincott Williams and Wilkins, Philadelphia, 2006.

13. Ricaurte GA, **McCann UD**. Drug Intoxications, In: Principles of Drug Therapy in Neurology, Second Edition (M.V. Johnston and R. A. Gross, eds), Oxford University Press, 2008.

14. **McCann UD**. Amphetamines, Methylphenidate and Excessive Sleepiness, In: Sleepiness: Causes, Consequences, Disorders and Treatment " (M Thorpy MD and M Billiard. Eds), Cambridge University Press, in press.

15. **McCann UD**. PCP/Designer Drugs/MDMA in, Substance Abuse, In: Substance Abuse: A Comprehensive Textbook, 5th Edition (Lowinson and Ruiz, eds), Lippincott, Williams and Wilkins, in press.

- **Letters**

1. **McCann UD**, Austin RK. Ballatrophobia: When clowns aren't funny, *Anxiety*,1996; 2:305

2. **McCann UD**, Szabo Z, Scheffel U, Dannals RF, Ricaurte GA. Positron emission tomography findings in heavy users of MDMA. *Lancet* 1999; 353: 592-593

3. **McCann UD**, Ricaurte GA. Caveat Emptor: Editors Beware. *Neuropsychopharmacology* 2001; 24(3):333-6.

4. Ricaurte GA, Yuan J, Hatzidimitriou G, Cord BJ, **McCann UD**. Response to O'Shea and Colado: the MDMA neurotoxicity profile might provide clues to mechanisms. *Trends Pharmacol Sci*. 2003 Jun; 24(6):275.

5. Ricaurte GA, Yuan J, Hatzidimitriou G, Cord BJ, **McCann UD**. MDMA ("Ecstasy") and Neurotoxicity. *Science* June 6, 2003; 1503.

Inventions, Patents, Copyrights

None

Extramural Sponsorship

- Current Grants

Dates: 1-1-10-12-31-15
Title: PET Studies of Amphetamine Neurotoxicity in Adult ADHD
Identification: pending
Sponsor: NIH/NIMH
Total Direct Costs: $1,909,799
Year 1 Direct Costs: $330,334
Principal Investigator: Ricaurte GA
Role: Co-Investigator
Percent Effort 30%

Dates: 1-1-10-12-31-15
Title: PET and Sleep Studies in Methamphetamine Users
Identification: pending
Sponsor: NIH/NIDA
Total Direct Costs: $1, 875,000
Year 1 Direct Costs: $320,000
Principal Investigator: McCann UD
Percent Effort: 30%

Dates: 9-1-07-6-30-12
Title: Sleep Disturbance, Central Pain Modulation and Clinical Pain in Osteoarthritis
Identification: R01 AR054871
Sponsor: NIH/NIDA
Total Direct Costs: $1,800.00
Year 1 Direct Costs: $414,434
Principal Investigator: Michael Smith, Ph.D.
Percent Effort: 5%

Dates: 12-1-08-11-30-10
Title: Effects of Zolpidem Extended-Release on Withdrawal and Sleep in Cannabis Users
Identification: R21 DA025794
Sponsor: NIH/NIDA
Total Direct Costs: $250,000
Year 1 Direct Costs: $125,000
Principal Investigator: Ryan Vandrey
Percent Effort: 5%

- Pending Grants:

Title: Genetic Studies of PTSD
Sponsor: MRMC
Total Direct Costs $6,250,000
Year 1 Direct Costs: $300,000

Title: Ascendin-4: A Novel Treatment for PTSD
Sponsor: MRMC
Total Direct Costs: $6,000,000
Year 1 Direct Costs: $550,000

- Previous Grants

Dates: 9-30-02 to 7-31-10
Title: Sleep and Nocturnal Endocrine Function in MDMA Users
Identification #: 1 R01 Hl071501
Sponsor: NIH/NHLBI/NIDA
Total Direct Costs: $1,612,940
Current Year Direct Costs: $250,000
Principal Investigator: McCann UD
Role: Principal Investigator
Percent Effort: 20%

Dates: 9-01-04 to 5-31-10
Title: Structural Brain Correlates of MDMA Use
Identification #: 1RO1 DA017231-01 A1
Sponsor: NIH/NIDA
Total Direct Costs: $1,000,000
Current Year Direct Costs: $250,000
Role: Co-Investigator
Percent Effort: 16%

Dates: 9-30-02 to 12-31-09
Title: PET Imaging MDMA Neurotoxicity
Identification #: 1 R01 DA010217
Sponsor: NIH/NIDA
Total Direct Costs: $1,503,090
Current Year Direct Costs: $293,635
Principal Investigator: McCann UD
Role: Principal Investigator
Percent Effort: 20%

Dates: 2-1-90 to 12-31-07
Title: MDMA Neurotoxicity in the Primate
Identification #: 2 RO1 DA05707
Sponsor: NIH/NIDA
Total Direct Costs: $1,476,084
Current Year Direct Costs: $300,025
Principal Investigator: Ricaurte GA
Role: Co- Investigator
Percent Effort: 5%

Dates: 3-1-92 to 2-28-07 (NCE)
Title: MDMA Neurotoxicity in Humans: Occurrence and Consequences
Identification #: 2 RO1 DA05938
Sponsor: NIH/NIDA
Total Direct Costs: $1,250,000
Current Year Direct Costs: $250,000
Principal Investigator: Ricaurte GA
Role: Co-Investigator
Percent Effort: 20%

Dates: 4-01-05 to 3-31-08
Title: ADHD Treatment and Amphetamine Neurotoxicity
Identification #: 1 R01 HD050202-01
Sponsor: NIH/NICHHD
Total Direct Costs: $736,781
Current Year Direct Costs: $237,000
Principal Investigator: Ricaurte GA
Role: Co-Investigator
Percent Effort: 20%

Dates: 10-01-01 to 9-30-06
Title: Methamphetamine Neurotoxicity in Nonhuman Primates
Identification #: 1 RO1 DA13946
Sponsor: NIH/NIDA
Total Direct Costs: $1,785,592
Role: Co- Investigator

Dates: 9-29-00 to 6-30-05
Title: Gene Expression and Methamphetamine Neurotoxicity
Identification #: 1 RO1 DA13790
Sponsor: NIH/NIDA
Total Direct Costs: $700,000
Role: Co- Investigator

Dates: 7-1-97 to 7-31-04
Title: Safety Assessment of Fenfluramine and Phentermine in Humans
Identification #: 1 RO1 DA11226
Sponsor: NIH/NIDA
Total Direct Costs: $1,633,785
Role: Co- Investigator

Dates: 8-1-98 to 8-31-05
Title: PET Studies of Methamphetamine Neurotoxicity in Humans
Identification #: 1 RO1 DA09487
Sponsor: NIH/NIDA
Total Direct Costs: $877,665
Role: Co- Investigator

Dates: 3-1-94 to 2-28-05
Title: Studies of Substituted Amphetamine Neurotoxicity
Identification #: KO2 DA00206
Sponsor: NIH/NIDA
Total Direct Costs: $468,843
Role: Co- Investigator

Dates: 10-1-94 to 9-30-99
Title: PET Imaging of Dopamine Neurotoxicity With [^{11}C]WIN-35,428
Identification #: 1RO1DA19487
Sponsor: NIH/NIDA
Total costs: $1,567, 606
Role: Co- Investigator

EDUCATIONAL ACTIVITIES

Teaching

- Classroom instruction

Introduction to Clinical Research Course; 2001-2003, Director
Introduction to Clinical Research Practicum, 2002-2003, lecturer
JHSOM Medical Students Psychiatry Rotation; 2004-present, lecturer
GIM Psychiatry Lecture Series; 2002-present, lecturer
Psychiatry PGY-II Lecture Series; 2004-present, lecturer
Psychiatry PGY-III Lecture Series; 2004-present, lecturer
Anxiety Disorders Seminar, 2004-present, Associate Director
Psychiatry PGY-III Cognitive Behavior Therapy Training 2004-present, faculty

- Clinical instruction

  Johns Hopkins Anxiety Disorders Clinic; 1999- July 2004, Attending Physician
  Johns Hopkins Anxiety Disorders Clinic; July 2004-present, Director
  Consultation Liaison Service, 1999-present, Attending Physician
  Acute Psychiatric Unit, 2006-present, Attending Physician

Mentoring

- Advisees

  Pre-Doctoral
  Victoria Eligulashvili, 1998-2000, BA under my mentorship, medical resident currently
  Audra de Ridder, 2003-2005, BA under my mentorship, currently holds BSN
  Maureen Flanagan 2001-2003, BA under my mentorship, currently a practicing orthoptist
  Branden Cord, 2000-2002, BS under my mentorship, currently obtaining an MD/PhD degree
  Peter Rosenblatt, 2003-2005, BA under my mentorship, currently obtaining an MPH
  Anna DuVal, 2003-2005, BA under my mentorship, currently obtaining an MPH
  Stephen Peterson 2004-2006, BA under my mentorship, currently in a MS pre-med program
  Rubyna Abbey 2005-2006, BA under my mentorship, currently obtaining an MPH

  Post-Doctoral
  Francis Sgambati, 2006-2007, BS and Masters, Sleep Programmer and Analyst
  Ryan Lanier, Ph.D., Post-doctoral fellow, BPRU, JHSOM
  Nancy Honeycutt, Ph.D., Assistant Professor of Psychiatry and Behavioral Sciences, JHSOM
  Michael Smith, Ph.D.; Assistant Professor of Psychiatry and Behavioral Sciences, JHSOM
  Emerson Wickwire, Ph.D.; Post-doctoral fellow, Behavioral Sleep Medicine Program, JHSOM
  Neda Gould, Ph.D.; Post-doctoral fellow, Anxiety Disorders Program
  Shawn Mason, Ph.D.; Post-doctoral fellow; Burn Psychology Program

- Thesis Committee

  John Anthony Brown, Ph.D. Candidate, Canberra, Australia; 2005-2006; Thesis Reviewer

- Training Grant Participation

  Behavioral Pharmacology Research Unit Post-Doctoral Training Grant;1999-present; Senior faculty
  Center for Mind-Body Research Training Grant; 2005-present; faculty

Editorial Activities

- Editorial Board Appointments

  Journal of Women's Health and Gender Based Medicine
  Women in Medicine
  Addiciones

- Journal peer review activities

Ad hoc reviewer-	Biological Psychiatry
Ad hoc reviewer-	Journal of Neuroscience
Ad hoc reviewer-	Psychopharmacology
Ad hoc reviewer-	Anxiety and Depression
Ad hoc reviewer-	Neuropsychiatry, Neuropsychology and Behavioral Neurology
Ad hoc reviewer-	Journal of Clinical Psychopharmacology
Ad hoc reviewer-	Human Psychopharmacology
Ad hoc reviewer-	Journal of the American Medical Association
Ad hoc reviewer-	The Lancet
Ad hoc reviewer-	Nature Pharmacogenomics
Ad hoc reviewer-	Journal of Psychopharmacology
Ad hoc reviewer-	Neuropsychopharmacology
Consulting Editor-	Health Psychology
Review Panelist-	Current Drugs

CLINICAL ACTIVITIES

Certification

- Medical

  Medical License, State of California, 1988-1992
  Medical License, State of Maryland, 1992-present

- Boards

  Diplomate National Board of Medical Examiners
  Certification, American Board of Psychiatry and Neurology
  Subspecialty: Psychosomoatics

Service Responsibilities

Johns Hopkins Anxiety Disorders Clinic, Director, weekly half-day clinic
Consultation Liaison Service, Johns Hopkins Bayview Medical Center, Attending, 6 weeks/yr
Behavioral Pharmacology Research Unit, Attending physician, three months/year
Acute Psychiatric Unit, Johns Hopkins Bayview Medical Center, Attending, 4 weekends/year
Second Call, Johns Hopkins Bayview Medical Center, Attending, 2-5 times per month

ORGANIZATIONAL ACTIVITIES

Institutional Administrative Committees

General Clinical Research Advisory Committee 1999-2001
Associate Program Director, JHBMC GCRC 2001-2004

Professional Societies/Memberships

Society for Neuroscience, 1988-present, member

Review Groups

Swiss National Research Grant Program, 1991, reviewer
NIMH Anxiety Disorder Education Disorder Program contracts, 1995, reviewer
NIMH Anxiety Disorders Education Program, 1996-1998, consultant
Alberta Heritage Foundation for Medical Research, 1997-1998, reviewer
NIDA RFA on Club Drugs and Trends, July 2001, reviewer
Scientific Review Group ZRG1 BDCN-2 (02), March 2003, reviewer
Welcome Trust Grant Program, 2004, reviewer
Scientific Review Group ZRG1 IFCN-C (03), November, 2004, reviewer
NMB Study Section, January, 2005, reviewer
NMB Study Section, June, 2005, reviewer
SEP for NMB Study Section, July, 2005, reviewer
Interventions Review Committee, Fall 2005, reviewer
Scientific Review Group U54 SNRP (NINDS/NIDA), March 2006, site visitor, reviewer
Scientific Review Group Learning and Memory Study Section, June 2006, reviewer
Scientific Review Group U54 SNRP (NINDS/NIDA), November 2006, reviewer
Scientific Review Group CDMRP, DoD PTSD/TBI Research Program, November 2007, Chair
Scientific Review Group I/START (NIDA), November 2007, reviewer
Scientific Review Group, MST Study Section, 2008
Scientific Review Group, ZDA1 MXG-S (10) 1, 2009
Scientific Review Group, VA MHBA Merit Review, 2009-present, Regular Member

Consultantships

NIMH Panic Disorder Education Program, 1994-1998, Spokesperson
NIMH Constituency Outreach and Education Program, 2000-present, expert panelist
ABC News, 2000-present, medical expert

RECOGNITION

Awards

Summa Cum Laude, 1980, Princeton University,
US Army Four-Year ROTC Academic Scholarship, 1976-1980
Phi Beta Kappa and Sigma Xi Societies, 1980
Sigma Xi Award in Psychology, 1980
Howard Hughes Foundation Student Scholar, Peter Bent Brigham Hospital, 1980
Moos Fellowship for Clinical Research in Psychiatry, Stanford University, 1987-1988
US Army Commissioned Officers Award, 1995

Invited Talks and Panels

1991 Yale University (New Haven V.A. Hospital), Neurobiology Grand Rounds, "Effects of AMPT in Humans"

1992 American Psychiatric Association; Washington DC; speaker, "Panic Disorder and Sleep" panel discussion

1996 Anxiety Disorders Education Program Launch; Washington, DC, "Overview of Anxiety Disorders"

1996 FAES Psychopharmacology Update, Bethesda Maryland, "Research on Panic Disorder"

1997 FAES Psychopharmacology Update, Bethesda Maryland, "Pre-clinical and Clinical Studies of MDMA Neurotoxicity"

1997 Anxiety Disorders Education Program Conference for Primary Care Physicians, Washington, DC,; Series of Lectures: "Panic Disorder", "Social Phobia", "Obsessive Compulsive Disorder"

1998 Annual Meeting on Women's Health, Hilton Head, South Carolina, , "Anxiety Disorders in Women"

1998 Wayne State University School of Medicine, Chairman's Grand Rounds. Detroit, Michigan, "Preclinical and Clinical Studies of MDMA Neurotoxicity"

1998 The Novartis Foundation, London, England, Ecstasy (MDMA): A Human Neurotoxin? "Clinical Studies of MDMA Neurotoxicity"

1999 Congreso Nacional de Psiquiatría. Oviedo, Spain, , "Research Update on Post Traumatic Stress Disorder"

2001 Canadian College of Neuropsychopharmacology/British Association for Pharmacology, Banff, Canada, symposium speaker "The Neuropharmacology of Ecstasy,"

2002 American Psychiatric Association, Philadelphia, panel speaker "MDMA-induced hyperthermia"

2001 First International Congress on MDMA Research, MDMA/Ecstasy Research: Advances, Challenges, and Future Directions, "MDMA-induced Neurotoxicity: Clinical studies"

2002 Brain Awareness Week, Vanderbilt University, Featured Speaker "MDMA Neurotoxicity: Preclinical and Clinical Studies"

2002 University of Utah, Psychiatry Grand Rounds,"MDMA Neurotoxicity"
DRADA Membership Meeting, Columbia, MD, 2003, "Anxiety Disorders"

2005 A Woman's Journey, Baltimore, MD 2003, "Anxiety Disorders"
Winter Conference on Brain Research, Breckenridge, CO, , Panel Organizer "What's Up With Ecstasy?"

2006 Winter Conference on Brain Research, Steamboat Springs, CO, Panelist "Stimulant Use in ADHD"

2006 Fountain House Luncheon and Fundraiser; New York City, Panelist "Anxiety Disorders"

2007 American Society for Pharmacology and Experimental Therapeutics, Washington DC, panelist; "Its all the Rave: Behavioral, Neuropharmacological and Toxic Effects of MDMA and Methamphetamine"

2007 The 52nd Annual Philip Tumulty Topics in Clinical Medicine at Johns Hopkins; Baltimore, MD, Lecturer; "Anxiety Disorders: Causes and Treatment"

2007 The Johns Hopkins Bayview Research Seminar; Baltimore, MD, November "MDMA Neurotoxicity: Occurrence and Consequences"

2007 Society for Neurosciences Minisymposium, November 2007, panelist "Understanding the Neurobiology of Drug Addiction by Studying Sleep Disturbances and Circadian Rhythms"

2007 Grand Rounds, Harbor Hospital
"Anxiety Disorders: Causes and Treatment"

2008 Winter Conference on Brain Research, Snowmass, Utah, Panel Chair
"Sleep and Drug Dependence: Some Stimulating Data"

2008 NIDA Research Seminar, February, Baltimore, MD
"Clinical Studies in MDMA Users"

2009 Winter Conference on Brain Research, Keystone, Colorado, Panel Member
"Role of Serotonin in Sleep"

2009 American Psychiatric Association Annual Meeting, Symposium Member
"Sleep in MDMA Users"

Exhibit B

American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*, Washington, DC, American Psychiatric Association, 2000.

Brown, M. email to Hyams, Salvatore, Van Diepen, Moore, Pringle, Wallick, and Abbot, "Re EDMS 352753 – Edgewood Arsenal Notification Letter – Expedite" dated June 29, 2006. (DVA052 000113-14.)

Coughlin, S., "Post-Traumatic Stress Disorder and Cardiovascular Disease," *The Open Cardiovascular Medicine Journal* 5:164-170 (2011).

Eveloff, H., "The LSD Syndrome: A Review," *California Medicine* 109(5):368-373 (1968).

Ford J., Schnurr, P., Friedman, M., Green, B., Adams, G., Jex, S., "Posttraumatic Stress Disorder Symptoms, Physical Health, and Health Care Utilization 50 Years After Repeated Exposure to a Toxic Gas," *Journal of Traumatic Stress* 17(3):185-194 (2004).

Glaesmer, H., Brahler, E., Gundel, H., Riedel-Heller, S., "The Association of Traumatic Experiences and Posttraumatic Stress Disorder with Physical Morbidity in Old Age: a German Population-Based Study," *Psychosomatic Medicine* 73(5):401-406 (2011).

Halpern, J., Pope, H., "Hallucinogen persisting perception disorder: what do we know after 50 years?," *Drug and Alcohol Dependence* 69:109-119 (2003).

Iaria, G., Fox, C., Scheel, M., Stoew, R., Barton, J., "A case of persistent visual hallucinations of faces following LSD abuse: A functional Magnetic Resonance Imaging study," *Neurocase* 16(2):106-118 (2010).

Keane, T., Marshall, A., Taft, C., "Posttraumatic Stress Disorder: Etiology, Epidemiology, and Treatment Outcome," *Annual Review of Clinical Psychology* 2:161-197 (2006).

Ketchum, J., *Chemical Warfare, Secrets Almost Forgotten*, Appendix, pp. 269-310 (ChemBooks Inc. 2006). (JK24 0028935-29026.)

The National Academies, "Supplement: Health Effects of Perceived Exposure to Biochemical Warfare Agents," April 2004. (Deposition Exhibit 354.)

National Research Council, "Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents: Volume 2, Cholinesterase Reactivators, Psychochemicals, and Irritants and Vesicants," National Academy Press (Washington, D.C. 1984). (CAR0000345-615, Deposition Exhibit 552.)

National Research Council, "Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents, Volume 3, Final Report: Current Health Status of Test Subjects," National Academy Press (Washington, D.C. 1985). (VET013-004999-5104.)

Ritchie v. United States, United States District Court, Northern District of California, Case No. C00-3940 MHP, Ketchum, J., "Psychiatric Evaluation of Wayne A. Ritchie for Sidney Bender, Esq.," December 5, 2001.

Ritchie v. United States, United States District Court, Northern District of California, Case No. C00-3940 MHP, Deposition Transcript of James S. Ketchum, M.D., August 5, 2003. (JK11 0017949-95.)

Ritchie v. United States, United States District Court, Northern District of California, Case No. C00-3940 MHP, Transcript of Proceedings, April 8, 2005.

Robicsek O., Makhoul, B., Klein, E. Brenner, B., Sarig, G., "Hypercoagulation in Chronic Post-Traumatic Stress Disorder," *Israel Medical Association Journal* 13:548-552 (2011).

Schnurr, P. Friedman, M., "Post-Traumatic Stress Disorder among World War II Mustard Gas Test Participants," Military Medicine 3:131-136 (1996).

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Chart of Chemical Dosage Ranges – Chem-Bio Database