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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. PETRAEUS, 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

Defendants.

Case No. CV 09-0037-CW

**EXPERT REPORT OF
DANIEL E. FORD, M.D., M.P.H.**

1 **I. INTRODUCTION**

2 **A. Retention**

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

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

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

1 sources become available to me I reserve the right to supplement this report to incorporate that
2 information.

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

6 **B. Compensation**

7 5. I am being compensated for my work on this matter at my customary rate of \$400
8 per hour, plus expenses. I am being compensated for travel time at a rate of \$200 per hour up to a
9 daily maximum of \$1200. My compensation is not conditioned on the substance of my opinions,
10 testimony at deposition or trial, or the outcome of this matter.

11 **II. MY BACKGROUND AND QUALIFICATIONS**

12 6. I am currently Vice Dean for Clinical Investigation at the Johns Hopkins
13 University School of Medicine. In addition, I am the Director of the Institute for Clinical and
14 Translational Research at Johns Hopkins. I am a Professor in the Department of Medicine at the
15 Johns Hopkins University School of Medicine and hold joint appointments as a Professor in the
16 Department of Psychiatry and Behavioral Sciences (Johns Hopkins University School of
17 Medicine), Department of Epidemiology (Johns Hopkins University Bloomberg School of Public
18 Health), and Department of Health Policy and Management (Johns Hopkins University
19 Bloomberg School of Public Health). I am part of the active clinical staff at Johns Hopkins
20 Hospital. My expertise encompasses clinical research, clinical study design, clinical medicine,
21 psychiatry research, epidemiology, and public health.

22 7. I earned my Bachelor of Arts degree in 1978 from Cornell University. I completed
23 medical school at the State University of New York at Buffalo, Buffalo, New York, obtaining my
24 M.D. in 1982. My Master of Public Health degree (1986) is from the Johns Hopkins University
25 Bloomberg School of Public Health. Following medical school, I trained as an Osler Medical
26 Intern (1982-1983) and Osler Medical Resident (1983-1985) at Johns Hopkins Hospital. After
27 completing my residency training, I pursued further training and work in an Epidemiology
28 Training Fellowship in the U.S. Public Health Service from 1985 to 1988. During this time, I

1 was also a part-time Clinical Fellow in the Department of Medicine at Johns Hopkins Hospital
2 (1985-1988) and a Medical Staff Fellow, Primary Care Section, Clinical Services Research
3 Branch, Division of Biometry and Epidemiology, National Institute of Mental Health in
4 Rockville, Maryland (1985-1988). I have held multiple academic positions at the Johns Hopkins
5 University School of Medicine from 1988 to the present. I am board certified in internal
6 medicine and practice under a medical license from the state of Maryland.

7 8. A major focus of my work has been to instruct clinicians and other investigators
8 on how to perform clinical and epidemiological research and to promote the development of
9 clinical research projects at The Johns Hopkins University. For example, I have been serving as
10 the Principal Investigator of a \$19 million “Institutional Clinical and Translational Science
11 Award” from the National Institutes of Health (2007-2012). This grant supports clinical and
12 translational research¹ throughout Johns Hopkins and includes support for the education and
13 training of new translational investigators, facilities in which clinical research can take place, and
14 infrastructure support for patient recruitment, bioinformatics, biostatistics and translational core
15 centers. As part of my teaching responsibilities, I direct the Intensive Course in Clinical Research
16 Methods at Johns Hopkins. I also lecture in courses at Johns Hopkins on epidemiology and
17 outcomes assessment. I have served as a reviewer for numerous journals, including the *American*
18 *Journal of Epidemiology*, the *Journal of the American Medical Association*, *Epidemiology*, and
19 *Epidemiological Reviews*.

20 9. Psychiatry and psychology research has also been a major focus of my work.
21 Although I am not a board-certified psychiatrist, I am an expert on the design and execution of
22 psychiatry and psychology studies. I have served as Principal Investigator for many projects in
23 psychiatry and psychology, including, for example: “Development of Internet Intervention for
24 Depression” (National Institute of Mental Health, 2006-2008), “Evaluation of the Implementation
25 Phase of the Depression in Primary Care Program” (Robert Wood Johnson Foundation, 2003-

26
27 ¹ “Translational” research is research that helps to bring scientific discoveries into
28 practical use in a clinical setting.

1 2005), and “Quality Improvement for Depression” (National Institute of Mental Health, 1998-
2 2004). I am currently a Special Sections Editor for the journal, *General Hospital Psychiatry*, and
3 I have served as a reviewer for the *American Journal of Geriatric Psychiatry*, *Journal of Clinical*
4 *Psychiatry*, and *Archives of General Psychiatry*.

5 10. I have published more than 150 original research articles in peer-reviewed journals
6 as well as 8 book chapters. I have been invited to present my research work at numerous
7 professional meetings both in the United States and internationally. A current copy of my
8 *curriculum vitae* is attached hereto as Exhibit 1, which includes a complete list of my publications
9 to date.

10 11. I have not provided any expert testimony in either deposition or trial within the
11 past four years. I have not prepared any expert reports for a litigation matter in the past four
12 years.

13 **III. BASIS AND SCOPE OF MY OPINIONS**

14 12. I have been asked to review and assess various studies examining health outcomes
15 in test subjects who participated in various U.S. military testing programs. Moreover, I have been
16 asked to provide my opinion about the quality and methodology of these studies. I may testify
17 about any or all of these topics.

18 13. In arriving at my opinions, expressed in detail in this report, I have relied on my
19 personal and professional experience as well as various additional resources. I have relied upon
20 the types of information and resources that are normally relied upon by experts in my field, such
21 as articles in peer reviewed journals, treatises and similar scholarly works, and published reports
22 regarding the testing programs at issue. Among the documents and studies that I have reviewed
23 are several reports from the National Research Council (“NRC”) that focus primarily on the
24 testing programs conducted at Edgewood Arsenal.

25 14. I have also reviewed documents from various other sources which contain
26 contemporaneous reports and accounts of actual tests. These documents were helpful to my
27 understanding of the circumstances surrounding the experiments performed in the various testing
28 programs and example test protocols used.

1 15. With respect to the doses and pathways of exposure, I have reviewed data drawn
2 from several sources, including a database printout of Edgewood test subjects between 1955 and
3 1975, excerpted data from the Chem-Bio Database that was provided to me, and a book written
4 by one of the principal researchers at Edgewood Arsenal, Dr. James Ketchum.

5 16. These are some of the primary references I have reviewed and relied upon in
6 reaching my opinions; a complete list of documents I have consulted and considered is included
7 as Exhibit 2 to this report. Throughout my report I have cited specific documents, and portions of
8 those documents, to illustrate technical and historical points. These citations are only illustrative,
9 not exhaustive, and I may rely on other specific portions of these documents, as well as any of the
10 references listed in Exhibit 2 to support any of these points. Moreover, to the extent Defendants
11 provide an expert report responding to any of the points addressed in this report, I reserve the
12 right to consider, comment on, or rely on any documents referenced in any such report.

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

16 **IV. COMMON METHODOLOGICAL PROBLEMS IN STUDIES RELIED UPON BY**
17 **THE U.S. GOVERNMENT**

18 18. It is my understanding that the U.S. Government is relying on certain studies to
19 deny that long-term health effects have resulted from the participation by military test subjects in
20 U.S. chemical and biological warfare programs. I have reviewed these key studies and have
21 identified important study design flaws, methodological problems, and analytical issues that
22 impact more than one of these studies. Since these problems and issues impact more than one
23 study examined in this report, I present below a brief discussion as a way of introduction to these
24 topics. These issues will be discussed in greater detail within the individual sections of this
25 report.

26 **A. Retrospective Study Design**

27 19. Every study examining long-term health outcomes of veteran test subjects is
28 essentially retrospective in design. This is because the original U.S. government chemical and

1 biological warfare testing programs were neither designed nor intended to follow the long-term
2 health outcome of test subjects following exposure to chemical and biological weapons (see the
3 discuss in section V.C. below.). Retrospective study designs have the potential to introduce a
4 number of powerful systematic errors, or *biases*, that can reduce the ability of the study to detect
5 clinically important health outcomes. These biases, discussed further below, include selection
6 biases, recall biases, and retrospective biases.

7 **B. Lack of Appropriate Control (Comparison) Groups—The Healthy Soldier**
8 **and Healthy Test Subject Effect**

9 20. As the discussion below will show, the original investigators conducting the
10 chemical and biological warfare tests failed to include an appropriate control (or comparison)
11 group within their testing program. Later investigators, such as scientific panels assembled by the
12 National Research Council were therefore forced to come up with control groups of their own.
13 This proved to be an extremely challenging task (see, e.g., the discussion in section V.C.4.
14 below). The most obvious solution, making health comparisons between test subjects and the
15 general U.S. population of males, was not a good option because of at least two powerful
16 confounding factors. First, all of the test subjects were U.S. military personnel. Since entry into
17 the U.S. military requires meeting certain physical and psychological health requirements, men in
18 the military tend to be healthier than males in the general U.S. population. Since they are
19 healthier earlier in life, military men also tend to be healthier later in life. This can make any
20 adverse health reactions in military test subjects difficult to detect because their better overall
21 health would tend to “mask” any such adverse health reactions when the comparison is made to
22 the general U.S. population. This is called the “healthy soldier effect.”

23 21. In addition, test subjects were screened for physical and mental health before they
24 were allowed to participate in the chemical and biological warfare tests. The effect of this
25 screening was to produce a military test subject population that was healthier overall compared to
26 the general military population. Like the healthy soldier effect, this “healthy test subject effect”
27 also tended to make it more difficult to detect any long-term adverse health outcomes when the
28 comparisons were being made to the general U.S. population. Furthermore, the healthy test

1 subject effect also made it more difficult to detect any long-term adverse health outcomes when
2 the comparisons were being made to the general U.S. military population.

3 **C. Poor Documentation of Exposure**

4 22. One of the most important pieces of information required when trying to determine
5 whether an exposure to a test agent caused an adverse health outcome is documentation of the
6 exposure itself. By “exposure” I mean: 1) what substance was administered; 2) how much of the
7 substance was administered; 3) through what route (inhalation, skin application, etc.) was the
8 substance administered; 4) how frequently was the substance administered; and 5) was the
9 substance administered with any other substance in sequence or combination? These are key
10 pieces of information regarding exposure. Unfortunately, the government did not always keep
11 good records regarding exposure, and for some tests where protective equipment was being tested
12 (e.g., mustard gas studies), it is very difficult, if not impossible, to know precisely how much of
13 the test agent was administered to a particular subject. Without adequate exposure information, it
14 is difficult to properly assess health outcomes.

15 **D. Cross-Sectional Rather than Longitudinal Study Design**

16 23. The best way to assess long-term health outcomes is to follow individuals over
17 time. As the individuals go through life, an investigator could assess their health periodically and
18 record the results. This type of study design is called a longitudinal study. In contrast, a cross-
19 sectional study assesses health outcome at just one period in time. While a longitudinal study is
20 analogous to a movie that continuously follows an individual or group, a cross-sectional study is
21 like a single “snapshot” in time. Cross-sectional studies are often performed instead of
22 longitudinal studies because they can be performed as part of a retrospective study (a longitudinal
23 study requires preplanning—a “prospective” study design), generally require less time, labor, and
24 money, and are generally simpler to execute. Unfortunately, cross-sectional studies are not nearly
25 as good as longitudinal studies for detecting trends over time in health outcomes. While
26 longitudinal studies are more likely to produce valid and reliable results, cross-section studies are
27 often performed out of necessity and convenience.

28

1 **E. Poor Documentation of Outcome**

2 24. In order to compare health outcomes between two different groups, it is important
3 to document outcomes accurately and thoroughly. Ideally, health outcomes would be evaluated
4 repeatedly over time through direct interviews, physical examinations, and appropriate laboratory
5 and/or diagnostic testing. Unfortunately, such investigations tend to be very expensive and labor
6 intensive. As seen in the discussion below, the studies examining health outcomes in former test
7 subjects generally used simpler, less costly methods, such as one-time surveys (see section V.C.
8 below). Even if some studies employed physical examinations to assess health outcome, these
9 were usually cross-sectional studies that looked at health at one point in time. Without good
10 documentation of outcome, it is not possible to make meaningful comparisons between groups.

11 **F. Confusing □No Difference□with Demonstration of Equivalence**

12 25. One of the most statistically challenging tasks in clinical research is performing a
13 study proving that two groups share an equivalent outcome. Such a study is often called an
14 “equivalence study,” and equivalence studies tend to be among the largest, most expensive, and
15 best-designed studies in clinical medicine. The reason for this is that proving equivalence
16 requires ruling out other factors that can result in an erroneous finding of equivalence—e.g., small
17 sample size (lack of statistical power), poor documentation of exposure and outcome, the impact
18 of important confounding factors (e.g., the healthy soldier effect and healthy test subject effect),
19 and the lack of a proper comparison or control group. Finding equivalence is not the same thing
20 as finding “no difference” between two groups. Finding equivalence requires an exceptionally
21 well-designed, executed, and statistically powerful study. Finding “no difference” can be
22 accomplished using a poorly designed and executed study. Indeed, a finding of “no difference” is
23 often the ultimate result of poorly conceived studies. Unfortunately, even medical and scientific
24 professionals confuse the difference between a finding of “equivalence” and a finding of “no
25 difference.” As the discussion below will show, there are plenty of poor quality studies finding
26 “no difference” in the health outcomes between test subjects and a comparison group. However,
27 in my opinion, no study that I have reviewed or discussed here in this report has ever truly
28 demonstrated “equivalence” in health outcomes between test subjects and a comparison group.

1 **V. METHODOLOGICAL PROBLEMS IN SPECIFIC INDIVIDUAL STUDIES**

2 26. It is my understanding that the U.S. government has tested a very large number of
3 chemical and biological agents as part of their weapons programs.² I have had the opportunity to
4 review the major reports concerning the potential long-term health effects from exposure to these
5 agents. One important observation is that these reports together examine a relatively small
6 number of the agents tested by the U.S. government. For the vast majority of chemical and
7 biological agents tested by the U.S. government, there does not appear to have been any effort to
8 examine the long-term health effects resulting from exposure to those agents.

9 **A. National Research Council, Possible Long-Term Health Effects of Short-**
10 **Term Exposure to Chemical Agents, Volume I, Anticholinesterases and**
11 **Anticholinergics**

12 27. In response to a request from the Department of the Army, the Committee on
13 Toxicology of the National Research Council evaluated the possibility of long-term or delayed
14 adverse health effects of chemical agents tested on military volunteers during the 1960s and
15 1970s.³ The NRC Volume 1 report examined the anticholinergic and anticholinesterase
16 chemicals.⁴ Because of important methodological deficiencies in the study, I do not believe that
17 data presented in this study are sufficient to reach any firm conclusions regarding possible long-
18 term health effects of exposure to anticholinesterase and anticholinergic agents. In addition, I
19 believe methodological problems and the limited statistical power of the study design do not
20 allow any firm conclusions to be reached regarding any potential impact of anticholinesterase and
21 anticholinergic test exposure on mortality or morbidity rates.

22 ² Chemical Warfare Agent Experiments Among U.S. Service Members, Dept. of Veterans
23 Affairs, Washington, D.C., updated 2006 at VET001_015677; VBA Outreach Efforts to Veterans
24 Exposed to Chemical and Biological Substances, August 2008, at DVA003 010051-2.

25 ³ National Research Council, "Possible Long-Term Health Effects of Short-Term
26 Exposure to Chemical Agents, Volume I, Anticholinesterases and Anticholinergics," National
27 Academy Press, Washington, D.C. 1984 (hereinafter, "NRC Volume 1") at x.

28 ⁴ It is my understanding that a detailed description of these compounds will be presented
in another expert report, and so I will not discuss the properties of these compounds in any great
detail here. The panel discusses the properties of these compounds in some detail in Chapters 2
and 3 of NRC Volume 1.

1 28. The panel that authored the NRC Volume 1 report seemed to acknowledge the
2 limitations of their study, concluding that they were “unable to rule out the possibility that some
3 anti-ChE [anticholinesterase] agents produced long-term adverse health effects in some
4 individuals.”⁵ Similarly, while the panel claimed—prematurely in my opinion—that “[n]o firm
5 evidence has been seen that any of the anticholinergic test compounds surveyed produced long-
6 range adverse human health effects in the doses used at Edgewood Arsenal,” the panel also
7 acknowledged that “[m]ore intensive study is required to confirm this conclusion.”⁶

8 **1. Problems in the Design and Execution of the Original Edgewood**
9 **Testing Program**⁷

10 29. The panel that conducted the NRC Volume 1 study was charged in part to
11 determine whether “the data available are sufficient to estimate the likelihood that the test
12 chemicals have long-term health effects or delayed sequelae.”⁸ It is my opinion that the data
13 available were not sufficient to estimate the likelihood that the test chemicals have long-term
14 health effects or delayed sequelae. A major reason for this is the poor design and execution of the
15 original chemical warfare studies performed at Edgewood Arsenal. The quality of the data
16 available to the NRC panel was seriously impaired by the relatively poor quality of data collected
17 during the original chemical warfare studies. The original chemical warfare studies were not
18 designed or intended to examine the long-term health effects of exposure to chemical warfare
19 agents, but rather to assess the potential use and effects of these agents as military weapons.
20 Therefore, insufficient data exists from these studies to assess long-term health effects from
21 exposure to the test agents.

22
23 ⁵ NRC Volume 1 at xi.

24 ⁶ *Id.*

25 ⁷ The NRC studies discussed in this report focus on testing conducted on service members
26 at Edgewood Arsenal. I understand, however, that Defendants conducted testing at multiple other
locations as well, including prior to the 1950s.

27 ⁸ NRC Volume 1 at x.

1 30. The original studies performed by the U.S. military had serious methodological
2 problems that would be unacceptable in clinical research today.⁹ Problematic aspects of the
3 original testing program included, but were not limited to, potentially coercive test subject
4 recruitment, failure to properly use placebos, exposure to dangerously high doses of test agents,
5 poor quality and dose control of test agents, crude monitoring methods, and essentially non-
6 existent long-term follow up of test subjects.¹⁰

7 31. *Problematic Test Subject Recruitment and Consent.* The NRC panel states that by
8 1954, “the Chemical Corps (formerly CWS) had established a framework within which to
9 conduct human experimentation, but it lacked an adequate pool of volunteers.”¹¹ Considering
10 that the experimentation involved the use of test agents that could possibly be used for chemical
11 warfare, the lack of volunteers should not have been surprising. Ultimately, military investigators
12 settled on the “most practical source of volunteers,” namely “enlisted men stationed at Army
13 installations in the vicinity of Edgewood Arsenal.”¹² Military investigators supposedly
14 “emphasized that voluntary consent of each human subject was absolutely essential,”¹³ but it is
15 reasonable to believe that enlisted soldiers may have felt some coercive pressure to participate in
16 these chemical warfare tests, even with the offering of certain “incentives” such as a small
17 monetary allowance or a free weekend.¹⁴ In addition, because the U.S. military lacked sufficient

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21 t is also unclear whether substances were properly
22 accounted for, as illustrated by the sudden appearance and disappearance of thirty to forty pounds
23 of LSD from Dr. Ketchum’s office with “no comment from anyone, no receipt form and no other
24 paper work.” (James S. Ketchum, *Chemical Warfare Secrets Almost Forgotten*, Chapter 19
(ChemBooks Inc. 2006) at 203; Ketchum Depo. Transcript July 14, 2010 at 187:1–188:24.)

24 ¹⁰ See NRC Volume 1 at 1-3.

25 ¹¹ NRC Volume 1 at 1.

26 ¹² *Id.*

27 ¹³ *Id.*

28 ¹⁴ NRC Volume 1 at 2.

1 knowledge about the chemical agents that they planned to test, it was not possible to obtain true
2 informed consent where “subjects would be thoroughly informed of all procedures and of what
3 might be expected as a result of each test.”¹⁵ Importantly, it is not possible that the military
4 investigators knew enough about the chemical warfare agents to properly inform the test subjects
5 about potential long-term health effects from exposure to such agents. As determined in U.S.
6 Senate hearings that took place in 1975, “the consent information was inadequate by current
7 standards.”¹⁶

8 32. *Failure to Properly Use Placebos.* The original studies at Edgewood Arsenal
9 appear to have been conducted at times in an *ad hoc* or haphazard manner, the sort of clinical
10 study execution that would be unacceptable in modern clinical research. A good example of this
11 is the failure of military investigators to properly use placebos in their studies. Placebo groups
12 (where test subjects are given an inert or inactive substance that resembles the active test agent)
13 can be very important in assessing the effects of test agents because they provide an appropriate
14 comparison group. The original military investigators did not seem to adequately appreciate the
15 importance of placebo groups. As described by the NRC panel, placebos “were used in some
16 studies, but the cost with respect to subject confinement time, staff workload, and delay in
17 achieving estimate of potency made this impractical except in special cases (e.g., evaluation of
18 antagonists).”¹⁷ Use of the word “impractical” here suggests that it was not practically possible to
19 use placebos, which almost certainly was not the case. A better term would be “inconvenient,”
20 though inconvenience is not a legitimate reason for failing to use placebos. If an investigator is
21 using high doses of a test agent then there may be less need for placebos. However if one has a
22 reasonable concern for safety in human volunteers, investigators would start with small doses of
23 the test agent where placebos would be necessary to detect true effects. The failure to properly
24 use placebos made it much more difficult to assess the short-term and long-term health effects of

25 ¹⁵ NRC Volume 1 at 1.

26 ¹⁶ NRC Volume 1 at 2.

27 ¹⁷ NRC Volume 1 at 3.

1 exposure to test agents and is indicative of the poor planning, design, and execution of the
2 original chemical warfare studies performed at Edgewood Arsenal.

3 33. *Exposure to Dangerously High Doses of Test Agents.* There are indications that
4 some of the doses administered to test subjects at Edgewood Arsenal were high and unsafe. As
5 the NRC panel noted, “Signs of drug effects at all but the lowest doses were significant,”¹⁸
6 indicating that the doses administered were generally high enough to produce “significant”
7 clinical symptoms and/or signs in the test subjects. The NRC panel also reports that an
8 “incapacitating dose” for BZ, one of the anticholinergic test compounds, was determined by
9 Edgewood Arsenal investigators to be approximately 5.5 µg/kg, and that administered doses
10 sometimes exceeded 1.5 times this “incapacitating dose.”¹⁹ Any dose of a chemical agent that
11 can be described as “incapacitating” must be very high, and to exceed that dose, even rarely,
12 suggests that some test subjects were exposed to dangerous doses of agents like BZ. This is
13 confirmed by the NRC panel’s report that “one subject who had received BZ displayed
14 hyperthermia, tachycardia, and spastic movements for a few hours, and required vigorous
15 treatment.”²⁰ Another subject who received BZ “developed signs of decerebrate rigidity with
16 limb twitching” that was thought to represent “toxic encephalopathy” or “BZ delirium.”²¹
17 Furthermore, many test subjects received “multiple exposures” to test agents over a period of
18 “days or weeks,”²² increasing the likelihood that some, maybe many, test subjects received
19 dangerous doses of test agents. For example, hyperthermia is now known to be a well-
20 documented and potentially fatal adverse reaction to anticholinergic agents. All of this reinforces
21 my opinion that the studies performed at Edgewood Arsenal were poorly planned, designed, and
22 executed.

23 ¹⁸ *Id.*

24 ¹⁹ *Id.*

25 ²⁰ NRC Volume 1 at 65.

26 ²¹ *Id.*

27 ²² NRC Volume 1 at 3.

1 34. *Poor Quality and Dose Control of Test Agents.* While we know from some of the
2 severe acute reactions that some subjects received dangerous doses of the test agents, it is not
3 possible to precisely quantify the doses they received. One reason for this is our uncertainty
4 about the quality and purity of the test agents used. There is no indication that the original
5 investigators had strict quality control protocols to ensure the purity of the test agents. Indeed,
6 the NRC panel notes that subjects were given anticholinesterase agents of “unstated purity.” This
7 would not be surprising, since many of the test agents used—e.g., sarin and VX²³—were never
8 developed or intended to be marketed as commercially available pharmaceuticals. Precise control
9 and documentation of exposure to test agents is an essential feature of a well-designed and
10 executed clinical study. Poor quality and dose control of test agents make it even more difficult
11 to properly determine the potential adverse effects of test agents, since many adverse effects are
12 dose-dependent.

13 35. *Crude Monitoring Methods.* The original chemical warfare testing program was
14 conducted many decades ago when only crude monitoring methods were available. For example,
15 the original investigators used EEGs (electroencephalograms) as one of their monitoring tools for
16 adverse reactions to test agents.²⁴ While EEGs continue to be used clinically today, they are
17 relatively crude tools for monitoring adverse changes in brain function. Today, we have far more
18 sensitive and powerful tools for assessing adverse effects on brain function (e.g., functional MRI
19 brain scans). Modern advances are not limited to technologically advanced machines. Since the
20 time of the original testing program, the field of neuropsychology has developed very sensitive
21 clinical testing methods for detecting subtle cognitive impairments that may have been caused by
22 exposure to toxic substances. Because these modern technologies and techniques were not
23 available to the original Edgewood Arsenal investigators, only limited information from crude
24 monitoring techniques was available to NRC panel reviewing the data. As an illustrative
25 example, the NRC panel reported that one subject who had received the anticholinergic agent,

26 ²³ NRC Volume 1 at 37, Table 3.

27 ²⁴ E.g., NRC Volume 1 at 65.

1 BZ, had “developed signs of decerebrate rigidity with limb twitching,”²⁵ clinical signs that
2 suggested possibly significant brain injury. As evidence that the subject suffered no long-term
3 injuries, the NRC panel notes that an “EEG tracing 20 d after exposure was normal.”²⁶ An EEG
4 is hardly definitive evidence of normal brain functioning. It is certainly possible that a functional
5 MRI study or neuropsychological testing could have detected subtle impairments in brain
6 function and cognition that would have been easily missed on EEG. There are also more
7 advanced techniques available today for monitoring other types of injuries as well. Since modern
8 monitoring techniques were not available to the Edgewood Arsenal investigators, it is impossible
9 to rule out the possibility that some test subjects experienced significant brain (or other) injuries
10 that were not detectable by the monitoring techniques available when the original studies were
11 performed.

12 36. *Lack of Long-Term Follow-up of Test Subjects.* Test subjects were recruited and
13 “assigned for a 1- to 2- month period of temporary duty at Edgewood Arsenal.”²⁷ There was no
14 plan for systematic, long-term follow-up of these test subjects; the experiments were designed to
15 assess the acute effects of these test agents as potential military weapons, not to evaluate the long-
16 term health effects of acute exposure to these agents. As the NRC panel states in its discussion of
17 the testing of anticholinesterase agents at Edgewood Arsenal, the case summaries made available
18 to the panel were “brief and anecdotal” and with “the exception of one case, they deal only with
19 the period immediately after the test dose.”²⁸ The NRC panel concluded that the case summaries
20 “do not provide hard data that would allow the panel to address, in a definitive manner, the
21 question of whether or not there is a possibility of long-term or delayed effect.”²⁹ The panel
22 admitted that the “paucity of data in the medical records prevents further study in relation to the

23 ²⁵ NRC Volume 1 at 65.

24 ²⁶ *Id.*

25 ²⁷ NRC Volume 1 at 2.

26 ²⁸ NRC Volume 1 at 29.

27 ²⁹ NRC Volume 1 at 30.

1 goal” of the panel’s report, which was to investigate “the possibility of long-term or delayed
2 effects.”³⁰ I agree with these statements from the panel. The available follow-up data for the
3 anticholinergic agents were similarly limited. For example, the NRC panel states that their
4 “[u]nderstanding of the timecourse of effects [from BZ exposure] was confounded by erratic
5 written documentation, which at best was rather sparse”³¹ In summary, the NRC panel’s
6 mission of definitively evaluating the long-term or delayed effects from exposure to
7 anticholinesterase and anticholinergic agents was made extremely difficult, if not practically
8 impossible, by the absence of any long-term follow-up data from the original experiments
9 performed at Edgewood Arsenal.

10 2. Retrospective Design

11 37. Turning to the NRC panel’s own investigation, the most fundamental weakness of
12 the NRC Volume 1 study is its retrospective design. Unlike a pre-planned prospective study, a
13 retrospective study is designed after most of the key data (e.g., information in medical records)
14 have already been collected. As discussed above, the original studies at Edgewood Arsenal were
15 not designed or intended to assess the long-term health effects of acute exposure to the various
16 test agents used. Therefore, information on long-term health effects was not collected or
17 analyzed by the original investigators at Edgewood Arsenal. The NRC panel had to look
18 backwards at whatever information was available and their analysis was seriously limited by the
19 quantity and quality of the available medical records. A retrospective study design makes the
20 NRC Volume 1 study vulnerable to potential biases, including, but not limited to, selection
21 biases, retrospective biases, and poorly documented exposures, which I discuss further below.

22 38. *Potential Selection or Sampling Bias—Anticholinesterase Study.* Retrospective
23 studies are particularly vulnerable to selection or sampling bias, a type of error that can be
24 introduced into a study by using a non-random method to select individuals or cases for inclusion
25 in the retrospective analysis. The potential for sampling bias can be seen in the NRC Volume 1

26 ³⁰ *Id.* (emphasis in original).

27 ³¹ NRC Volume 1 at 63.

1 study. The panel reported that 16 anticholinesterases were tested on 1,406 subjects.³² For
2 reasons that were not explained, the NRC panel reviewed information from only about 15% (219)
3 of these cases.³³ While some of the cases were selected randomly based on the terminal digit of
4 the case number (i.e., those case numbers ending in 3), others were selected non-randomly “on
5 the basis of high dosage, repetitive exposure, or the presence of additional physiologic stress.”³⁴
6 Presumably, the non-random selection criteria were chosen to select those cases where the test
7 subjects received the highest exposure to test substances and, perhaps, exhibited the most adverse
8 effects. However, it is also possible that those who received the highest doses were the test
9 subjects initially deemed to be the most healthy and strong, and it may be possible that those test
10 subjects who received repetitive exposures to test agents were also those who were most tolerant
11 of their effects (e.g., one would not expect investigators, as a matter of good practice, to give
12 multiple doses of a test agent if the test subject did not tolerate the first dose very well). In
13 addition, the NRC panel also chose to include all 32 subjects tested with the V-series nerve agent,
14 EA 3148, in their analysis, because EA 3148 was considered the most potent anticholinesterase
15 agent.³⁵ While EA 3148 may have had the potential to produce the most severe adverse effects, it
16 is also possible that individuals received EA 3148 because they were deemed the healthiest,
17 strongest, or most tolerant test subjects. Because of the limited information available, we cannot
18 know how the non-random selection criteria may have biased the NRC study’s final results. In
19 my opinion, there would have been less potential for selection or sampling bias if the NRC panel
20 had selected cases using only random criteria or, even better, had reviewed and analyzed all 1,406
21 cases. Reviewing all of the cases would have been especially important for detecting rare or
22 uncommon adverse effects.

23
24 ³² NRC Volume 1 at 29 and 37, Table 3.

25 ³³ NRC Volume 1 at 29.

26 ³⁴ *Id.*

27 ³⁵ *Id.*

1 39. *Potential Selection or Sampling Bias—Anticholinergic Study.* Another example of
2 potential sampling bias may be seen in the NRC panel’s analysis of BZ cases. The NRC panel
3 chose to review and analyze the medical records of only 36 of the at least 354 test subjects who
4 were exposed to BZ.³⁶ There is no explanation regarding how these 36 cases were selected or
5 why the NRC panel chose not to review all 354 cases. Certainly, any process that eliminates
6 nearly 90% of all cases from the final analysis has the potential to introduce important selection
7 or sampling biases that could impact the final results of the study. These concerns are magnified
8 when the reported quality of the records was poor, as indicated by the NRC panel’s description of
9 “fragmentary data” and “erratic written documentation” of the time course of effects.³⁷

10 40. *Potential Retrospective Bias.* When planning and executing a retrospective study,
11 there is always the potential for retrospective bias, where the views, opinions, or biases of the
12 investigators can impact the way information is collected and analyzed, thereby influencing the
13 results of the study. As an example, the lead investigators of a retrospective clinical study may
14 already have a pre-formed opinion on whether a new therapy works, and may—intentionally or
15 not—design and execute the study in a way that favors a positive result for the new therapy. In
16 reviewing the limited information available on the methodology of the NRC Volume 1 study, I
17 cannot determine whether retrospective bias impacted the final results of the study. I can state
18 only that retrospective studies are particularly vulnerable to this type of bias and that this fact
19 should be kept in mind when reviewing and analyzing the results of any retrospective clinical
20 study.

21 **3. Problematic Use of General Population as Control Group—Healthy**
22 **Soldier and Healthy Test Subject Effect**

23 41. One of the key weaknesses of retrospective studies is that one cannot pre-plan data
24 collection for an appropriate or suitable control group. Appropriate control groups are necessary
25 to compare outcomes of interest with those of the test group. For example, in order to determine

26 ³⁶ NRC Volume 1 at 63.

27 ³⁷ *Id.*

1 whether exposure to anticholinesterase or anticholinergic agents has an effect on long-term
2 mortality rates, the test group exposed to these agents must be compared to another group of non-
3 exposed individuals who are similar with respect to key characteristics that may impact long-term
4 health (e.g., gender, age at the time of testing, baseline health, smoking history). Ideally, the non-
5 exposed control group would be drawn randomly from the same pool of individuals that provided
6 the exposed test subjects. Unfortunately, the original investigators at Edgewood Arsenal
7 abandoned the use of a placebo group, which could have served as an ideal control group, for
8 questionable and inappropriate reasons—“cost with respect to subject confinement time, staff
9 workload, and delay in achieving estimates of potency.”³⁸ In essence, the original Edgewood
10 Arsenal studies had no appropriate control group whatsoever, and the NRC panel was forced to
11 come up with their own control group.

12 42. The NRC panel ultimately decided to compare mortality data for the test subjects
13 exposed to anticholinesterase and anticholinergic agents to the mortality data for “the U.S.
14 population.”³⁹ It is not entirely clear whether the NRC panel is using both men and women from
15 the U.S. population or just women, since the report describes the control group as “the U.S.
16 population as a whole”⁴⁰ or simply “the U.S. population.”⁴¹ That particular detail is important,
17 since women apparently were not among the test subjects at Edgewood Arsenal and their
18 inclusion by the NRC panel in the control group would introduce a confounding factor that would
19 make the final results of the study difficult to interpret. In addition, using mortality data from the
20 general U.S. population would be highly problematic, since that would introduce two
21 confounding factors, the “healthy soldier effect” and the “healthy test subject effect,” that would
22 tend to bias the study’s results towards the null hypothesis—i.e., towards a finding of no
23

24 ³⁸ NRC Volume 1 at 3.

25 ³⁹ NRC Volume 1 at 30 and 68 .

26 ⁴⁰ NRC Volume 1 at 30.

27 ⁴¹ NRC Volume 1 at 68.

1 difference in mortality rates between the test subjects and the control group. I discuss the healthy
2 soldier effect and healthy test subject effect further below.

3 43. *Healthy Soldier Effect.* It has been well-established that there is a “healthy soldier
4 effect” that may result in an underestimation of the adverse effects of an exposure in studies that
5 use the general population as a comparison group.⁴² Military personnel in the United States are
6 required to undergo an initial physical screening in order to enter the armed services, and are
7 required to maintain a certain level of physical fitness in order to remain in the military. In
8 addition, military personnel are provided with health care services during their time in the
9 military. All of this results in a military population that is overall healthier than the general U.S.
10 population.⁴³ This “healthy soldier effect” is analogous to the “healthy worker effect” seen in
11 civilian populations, where employed individuals are generally healthier than the population as a
12 whole because a certain level of fitness and health is usually necessary to maintain employment.⁴⁴
13 Since all of the test subjects in NRC Volume 1 were active duty military personnel,⁴⁵ it is
14 reasonable to expect that this healthy soldier effect would impact the results of the NRC Volume
15 1 study since these soldiers were being compared by the NRC panel to the general U.S.
16 population. The expected impact of the healthy soldier effect would be to underestimate the
17 adverse effects of exposure to anticholinesterase and anticholinergic agents, minimizing any
18 differences seen in the long-term mortality rates between the exposed test subject population and
19 the general U.S. population control group. This is a reasonable conclusion since there is evidence
20 that the impact of the healthy soldier effect is apparent even after more than 30 years.⁴⁶ Indeed,
21 the NRC panel essentially used the healthy soldier effect in explaining why standardized

22 ⁴² See, e.g., M. Waller and A. McGuire, “Changes over time in the healthy soldier effect,”
23 *Population Health Metrics* 9:7 (2011), <http://www.pophealthmetrics.com/content/9/1/7>
(hereinafter, “Waller and McGuire”).

24 ⁴³ See *id.*

25 ⁴⁴ See *id.*

26 ⁴⁵ NRC Volume 1 at 2.

27 ⁴⁶ Waller and McGuire at p. 1 of 9.

1 mortality rates for test subjects was actually less than the rates expected for the U.S. population as
2 a whole.⁴⁷ As the NRC panel stated, this “presumably reflects the fact that those who enter the
3 military service do not have chronic diseases.”⁴⁸

4 44. *Healthy Test Subject Effect.* The test subjects were not only healthier, on average,
5 than the general U.S. population because they were soldiers (the healthy soldier effect), but were
6 also healthier than the average soldier because test subjects underwent further screening before
7 being selected for the experiments—what I would call the healthy test subject effect. As the NRC
8 panel outlines,⁴⁹ recruiters for chemical warfare testing at Edgewood Arsenal would visit Army
9 installations and give a presentation about the program to “a large number of enlisted men.” Up
10 to 20% of the audience would typically express interest in participating in the experiments. For
11 some period of time at Edgewood, those soldiers who expressed interest were then “asked to
12 complete a personal history, which included medical and psychologic items and the Minnesota
13 Multiphasic Personality Inventory (MMPI).” It was “not unusual for 400-600 men to request
14 assignment” to the testing program with “no more than 100” ultimately being selected. This
15 highly selective process produced a group of test subjects who were “above average in physical
16 and mental qualifications, with a mean IQ near 110, good behavior records, and ‘normal’ MMIPs
17 [sic]” The NRC panel recognized the existence of the healthy test subject effect in explaining
18 why the standardized mortality rate for test subjects exposed to anticholinergic agents was
19 significantly lower than that for the general U.S. population, stating that the result “probably
20 represents a selection artifact, inasmuch as volunteers for these studies were especially screened
21 for good health and thus would be expected to have lower than average mortality.”⁵⁰

22 45. The failure of the original Edgewood Arsenal investigators to use an appropriate
23 control group forced the NRC panel to choose its own control group. However, by choosing the

24 ⁴⁷ NRC Volume 1 at 30.

25 ⁴⁸ *Id.*

26 ⁴⁹ NRC Volume 1 at 2.

27 ⁵⁰ NRC Volume 1 at 68.

1 general U.S. population as the control group for their study, the NRC panel introduced two
2 powerful confounding factors—the healthy soldier effect and the healthy test subject effect. Both
3 confounding factors biased the study’s results towards the null hypothesis—i.e., towards a finding
4 of no difference in mortality rates between the test subjects and the control group. The existence
5 of both effects, essentially acknowledged by the NRC panel, made any comparisons to the
6 general U.S. population very problematic and difficult to interpret. As a result, I do not believe it
7 is possible to reach any definitive conclusions, based on the results presented in NRC Volume 1,
8 about whether the chemicals tested are likely to produce long-term adverse health effects or
9 delayed sequelae in the test subjects.

10 **4. Evidence of Long-Term and Delayed Adverse Effects**

11 46. The NRC panel discussed some “anecdotal” reports that clearly indicated that
12 some of the test agents could produce long-term or delayed adverse effects. For example, two
13 individuals who were accidentally exposed to the anticholinergic agent, EA 3167, displayed
14 evidence of impaired cognitive function up to 12 months following the exposure.⁵¹ The NRC
15 panel also discussed the case of a test subject who “experienced long-lasting psychologic
16 symptoms.”⁵² In addition, some test subjects were exposed to multiple doses of a test agent or
17 multiple agents: an average of 3.1 tests each and some test subjects participating in 10 or more
18 tests.⁵³ Multiple exposures to one or more test agents would presumably increase the risk of acute
19 adverse effects and possibly long-term or delayed adverse sequelae. In summary, despite the
20 limitations of the data available to the NRC panel, the panel still uncovered compelling evidence
21 that some test subjects may have experienced long-term or delayed adverse effects from their
22 exposure to test agents. Further evaluation of surviving test subjects may confirm this
23 possibility.⁵⁴

24 ⁵¹ NRC Volume 1 at 66.

25 ⁵² NRC Volume 1 at 30.

26 ⁵³ NRC Volume 1 at 3 and 77.

27 ⁵⁴ The most effective way to evaluate the test subjects for long-term adverse health effects
28 would be a comprehensive medical follow-up program. In addition, it is important that each test

(Footnote continues on next page.)

1 **5. Underpowered to Detect Small but Important Differences in Mortality**
2 **Rates**

3 47. It important to note that the NRC Volume 1 study had only enough statistical
4 power to detect relatively large differences in mortality rates between the test subjects and the
5 control group (the general U.S. population). Indeed, the NRC panel states that from “a statistical
6 point of view, the experience being studied is incapable of demonstrating risks of dying increased
7 less than three- or four-fold.”⁵⁵ In other words, the NRC Volume 1 study was able to detect an
8 increased risk of dying from exposure to test agents only if the risk was at least 300 to 400%
9 greater for test subjects compared to the control group. I believe this is an accurate assessment of
10 the statistical power of the NRC Volume 1 study. What this means is that smaller, but still very
11 important increases in the risk of death—e.g., a 50%, 100%, or 200% increase in the risk of
12 death—could not be detected by the NRC Volume 1 study and therefore cannot be ruled out.

13 **6. Conclusions**

14 48. The NRC panel stated two objectives for the NRC Volume 1 study: 1) to
15 determine whether the data available are sufficient to estimate the likelihood that the test
16 chemicals [i.e., anticholinesterases and anticholinergics] have long-term health effects or delayed
17 sequelae; and 2) to determine whether the involved chemicals, as tested, are likely to produce
18 long-term adverse health effects or delayed sequelae in the test subjects.⁵⁶ Since, for the reasons
19 stated above, the original studies performed at Edgewood Arsenal have deficiencies in design and
20 execution, in my opinion, data available from them alone are insufficient to conclude that the test
21 chemicals have no long-term health effects or delayed sequelae. Similarly, because of the
22 important limitations of the NRC panel’s own study, it is not possible to determine through the

23 (Footnote continued from previous page.)

24 subject is informed about what substance or substances he was exposed to, the dose, route of
25 administration, and any possible long-term adverse health effects from exposure to those
26 substances or from participation in the testing program. Having such information is necessary so
27 that the service member can obtain adequate ongoing health care and monitoring.

28 ⁵⁵ NRC Volume 1 at 80.

⁵⁶ NRC Volume 1 at x.

1 NRC Volume 1 study whether the involved chemicals, as tested, are likely to produce long-term
2 adverse health effects or delayed sequelae in test subjects.

3 **B. National Research Council, Possible Long-Term Health Effects of Short-**
4 **Term Exposure to Chemical Agents, Volume II, Cholinesterase Reactivators,**
5 **Psychochemicals, and Irritants and Vesicants⁵⁷**

6 **1. Cholinesterase Reactivators**

7 49. The mortality data for this class of test agents was presented in NRC Volume 1
8 and shared the same methodology and methodological problems discussed above for NRC
9 Volume 1.⁵⁸ Since the comparison group was the general U.S. population, and because the
10 mortality data analysis was subject to biases from both the healthy soldier effect and healthy test
11 subject effect, I do not believe any meaningful conclusions can be made from the study regarding
12 increased mortality from exposure to cholinesterase reactivators.

13 **a. Retrospective Design and Limitations of Available Data**

14 50. The original U.S. government studies using cholinesterase reactivators did not plan
15 for extended follow up of test subjects. Therefore, the quality of data available for assessing the
16 long-term health of test subjects was practically non-existent. As the NRC panel conceded, “the
17 lack of followup [sic] data on volunteers prevent certainty in predicting occurrence or absence of
18 delayed effects.”⁵⁹ The NRC panel also “found no data on the basis of which to determine or rule
19 out carcinogenicity, mutagenicity, teratogenicity, or reproductive effects of the four oximes and
20 therefore did not reach a conclusion in this area.”⁶⁰

21 ⁵⁷ National Research Council, “Possible Long-Term Health Effects of Short-Term
22 Exposure to Chemical Agents, Volume 2, Cholinesterase Reactivators, Psychochemicals, and
23 Irritants and Vesicants,” National Academy Press, Washington, D.C. 1984 (hereinafter, “NRC
24 Volume 2”). Among the general problems with this study is the possibility of a serious
25 retrospective bias (i.e., a bias that tends to favor a particular result or outcome). It is my
26 understanding that Dr. James S. Ketchum served as a technical consultant for the report. (NRC
27 Volume 2 at Panel of Cholinesterase Reactivator Chemicals member list.) And I also understand
28 that Dr. Ketchum played a major role in performing the tests at Edgewood Arsenal and that he
has previously testified that he wrote much of the NRC Volume 2 report. (Ketchum Deposition
Transcript 325:16 -327:5.)

⁵⁸ See NRC Volume 2 at 46.

⁵⁹ *Id.*

⁶⁰ *Id.*

1 51. The poor quality of data made available for review by the NRC panel is
2 exemplified by the fact that “reports of physicians’ examinations and physical findings were
3 generally not included.”⁶¹ Because of this paucity of data, it would have been difficult to assess
4 the *acute* effects of cholinesterase reactivators, much less the long-term effects of exposure to
5 these agents.

6 **b. Conclusions**

7 52. I believe the paucity of good quality data available from the original experiments
8 severely undercuts the conclusions of the NRC panel.

9 **2. Psychochemicals**

10 53. This retrospective NRC investigation looked primarily at phencyclidine (also,
11 “SNA”) and dibenzopyran.⁶²

12 **a. Problems with the Design and Execution of the Original Studies**

13 54. This retrospective NRC investigation, like those described in NRC Volume 1, is
14 limited by the quality of the original studies performed by the U.S. military. As noted by the
15 NRC panel, no placebo control groups were used in the original studies, making the evaluation of
16 the long-term health consequences of exposure to psychochemicals much more difficult.⁶³ I
17 strongly disagree with the NRC panel’s statement that placebo controls “were probably not
18 appropriate, given the goals of the research.”⁶⁴ Not only would it have been appropriate to use a
19 placebo control group, but also necessary if one were interested in assessing the acute and long-
20 term effects of the psychochemicals used.

21 55. One of the major problems with the original studies was the apparent “flexibility”
22 in the protocols used.⁶⁵ Ideally, a study protocol is established before any research participants

23 ⁶¹ NRC Volume 2 at 31.

24 ⁶² NRC Volume 2 at 47.

25 ⁶³ NRC Volume 2 at 52.

26 ⁶⁴ *Id.*

27 ⁶⁵ NRC Volume 2 at 51.

1 are actually enrolled, and the changes to the protocol are kept to a minimum. A rigid adherence
2 to protocol helps prevent the introduction of biases and other errors into the experiment, which
3 can skew or distort the results of the study. The NRC panel seems to recognize this problem and
4 states, “A critical and skeptical reviewer, in retrospect, might say that there was too great
5 emphasis on browsing and that the changes in protocol, with small groups tested under any single
6 protocol, precluded definitive conclusions.”⁶⁶

7 56. The NRC panel acknowledged the poor design of the original study, noting that in
8 the early 1960s, “optimal research strategy and design as we know them today, was truly in its
9 infancy.”⁶⁷ The fact remains, however, that the original studies had significant problems with
10 design and execution—including the lack of a placebo group and a constantly changing study
11 protocol.

12 **b. Screening Process—Healthy Soldier and Healthy Test Subject**
13 **Effect**

14 57. The screening process of test subjects in the original experiments appears to have
15 been especially rigorous, even compared to the other studies performed at Edgewood Arsenal.⁶⁸
16 The NRC panel acknowledges that this screening process used by the original investigators
17 “likely introduced a ‘healthy-test-subject-effect’ into the [original] study.”⁶⁹ This healthy test
18 subject effect was likely exacerbated by the fact that the original control group for the exposed
19 subjects were military personnel who had failed the screening tests for the study.⁷⁰ As the NRC
20 panel states, since “the exposed subjects were healthier at the start than the nonexposed subjects,
21 comparisons between these two groups may well yield results that understate the relative risk [of
22

23 ⁶⁶ *Id.*

24 ⁶⁷ NRC Volume 2 at 52.

25 ⁶⁸ NRC Volume 2 at 48.

26 ⁶⁹ *Id.*

27 ⁷⁰ *Id.*

1 the test agents] to the exposed subjects.”⁷¹ This problem arising from the healthy test subject
2 effect only reinforces the need for a placebo group in such experiments.

3 58. The healthy test subject effect may have been particularly powerful in the
4 psychochemical testing at Edgewood Arsenal because test subjects were divided into one of four
5 categories.⁷² Test subjects rated “A” were deemed suitable for any psychochemical testing, and
6 were presumably the healthiest of all the test subjects.⁷³ Test subjects rated “B” were deemed
7 suitable for only “low-dose” psychochemicals.⁷⁴ Those rated “C” were not deemed suitable for
8 psychochemical testing, and a rating of “D” was deemed suitable for equipment testing only.⁷⁵
9 Such tiered classification of test subjects make any meaningful comparisons with the general
10 military or civilian population difficult, if not impossible.

11 **c. New Testing Methods Now Available to Detect Subtle Brain**
12 **Injury**

13 59. Exposure to psychochemical agents will not necessarily result in marked changes
14 in mortality rates or obvious morbidities. Instead, many of the long-term adverse effects may
15 involve subtle changes in cognition or brain function that may be missed by the evaluation tools
16 (like EEG) used by the NRC panel. The NRC panel did not employ neuropsychological testing or
17 newer monitoring techniques like functional MRI that could have identified subtle, but
18 significant, changes in cognition and brain function.

19 **d. Conclusions**

20 60. Because of the absence of any suitable control group and design and execution
21 problems with the original studies, I do not believe any definitive conclusions can be reached
22

23 ⁷¹ *Id.*

24 ⁷² NRC Volume 2 at 50.

25 ⁷³ *Id.*

26 ⁷⁴ *Id.*

27 ⁷⁵ *Id.*

1 regarding the acute and long-term health effects of psychochemicals based on this retrospective
2 NRC panel report.

3 **3. Irritants and Vesicants**

4 **a. Retrospective Record Review**

5 61. The NCR panel’s discussion of irritants and vesicants is less a clinical study and
6 more a simple summary of findings from a review of the records of 147 human subjects who were
7 exposed to mustard gas (“H”)—most of them repeatedly—between 1955 and 1965.⁷⁶ There is no
8 real quantitative analysis and only basic descriptive data are presented.

9 **b. Lack of Follow-Up Data**

10 62. There is no indication that the NRC panel attempted to perform any follow-up
11 evaluations. The panel concludes that “[g]iven the absence of followup [sic] data, it is not
12 possible to predict long-term health effects, except scarring from acute injuries.”⁷⁷ I do not
13 entirely agree with this statement. While it is true that we cannot fully determine long-term
14 health effects without follow-up data, the severity of some of the reported acute injuries—e.g., at
15 least two hospitalizations, one for five days⁷⁸—allows us to confidently predict that at least some
16 of the subjects experienced chronic sequelae following severe acute injuries from mustard gas
17 exposure.

18 **c. Original Testing Priority—Eliciting Acute Effects Over Safety**

19 63. The mustard gas experiments at Edgewood Arsenal show that test subject safety
20 was not a high priority for the original investigators. Indeed, the study was designed to
21 repeatedly expose individuals to mustard gas until “dermal erythema indicated garment leakage.”
22 Other subjects were brought into direct contact with mustard gas through deliberate cutaneous
23 exposures to “test the effectiveness of antidotes or treated cloths.”⁷⁹ Most, if not all, of the

24 ⁷⁶ NRC Volume 2 at 124-128.

25 ⁷⁷ NRC Volume 2 at 127.

26 ⁷⁸ *Id.*

27 ⁷⁹ NRC Volume 2 at 124.

1 subjects underwent repeated exposures to mustard gas, up to 14 exposures in some cases.⁸⁰

2 Repeated exposures to a toxic substance will generally increase the risk of serious acute injuries,
3 and possibly chronic or permanent injuries as well. The experiments were designed first and
4 foremost to elicit the acute effects of mustard gas in human subjects, with safety a subordinated
5 priority.

6 64. Some of the subjects' acute injuries were severe. As the NRC panel notes,
7 blistering from mustard gas was seen in at least 11 men, with two of the men requiring
8 hospitalization. A few of the skin injuries "might have been severe enough to cause permanent
9 scarring."⁸¹ These severe acute reactions to mustard gas exposure certainly increased the risk for
10 long-term adverse health effects.

11 **d. Unwarranted Assumptions of Safety**

12 65. The NRC panel states that "[n]one of these subjects sustained ocular or respiratory
13 tract injury," concluding that this "indicates that the ocular and respiratory systems were
14 adequately protected during these tests."⁸² I believe these assumptions of safety are unwarranted.
15 Individuals developed erythema over a broad range of body parts, including the anterior trunk,
16 genitalia, and groin.⁸³ And it may have been the case that injuries to the eyes and the lungs were
17 more difficult to assess and detect than injuries to the surface of the skin.

18 66. The NRC panel properly notes that mustard gas "is not only a vesicant, but also a
19 systemic poison."⁸⁴ The systemic effects of mustard gas have been demonstrated "in bone
20 marrow, intestinal tract, and respiratory tract."⁸⁵ It is clear that the majority of test subjects

21
22 ⁸⁰ *Id.*

23 ⁸¹ NRC Volume 2 at 127.

24 ⁸² *Id.*

25 ⁸³ NRC Volume 2 at 126.

26 ⁸⁴ NRC Volume 2 at 127.

27 ⁸⁵ *Id.*

1 experienced multiple episodes of dermal contact with mustard gas.⁸⁶ The skin is a major route of
2 entry for chemical toxins, and it seems certain that many test subjects absorbed mustard gas
3 systemically through the skin. This seems to be confirmed in the panel's discussion of one group
4 of 11 subjects, with only 8 subjects having normal post-exposure blood counts and urinalyses.⁸⁷
5 Systemic injuries from a chemical toxin are often undetectable on a routine physical examination.
6 It seems very likely that at least some of the subjects exposed to mustard gas at Edgewood
7 Arsenal experienced systemic injuries from repeated dermal exposures to mustard gas.

8 **e. Conclusions**

9 67. The design of the mustard gas experiments at Edgewood Arsenal prioritized the
10 elicitation of acute mustard gas effects, markedly increasing the risk for both acute and long-term
11 adverse reactions.

12 68. It is very possible that some of the subjects exposed to mustard gas at Edgewood
13 Arsenal experienced systemic injuries from repeated dermal exposures to mustard gas.

14 **C. National Research Council, Possible Long-Term Health Effects of Short-**
15 **Term Exposure to Chemical Agents, Volume III, Final Report, Current**
16 **Health Status of Subjects**

16 69. This investigation was a retrospective survey.

17 70. The NRC panel preparing this report⁸⁸ was charged with two tasks. The first task
18 was to "prepare a final report for the series *Possible Long-Term Health Effects of Short-Term*
19 *Exposure to Chemical Agents* on the basis of results of a questionnaire regarding current health
20 status of test subjects.⁸⁹

23 ⁸⁶ NRC Volume 2 at 124-127.

24 ⁸⁷ NRC Volume 2 at 124.

25 ⁸⁸ National Research Council, "Possible Long-Term Health Effects of Short-Term
26 Exposure to Chemical Agents, Volume III, Final Report, Current Health Status of Subjects"
(hereinafter, "NRC Volume 3").

27 ⁸⁹ NRC Volume 3 at Executive Summary.

1 71. The second task of the NRC panel was to “evaluate the implications of findings
2 from the questionnaire for any of the conclusions reported in Volumes 1 and 2.”⁹⁰

3 **1. Analytic Problems Related to Retrospective Survey Design**

4 **a. Limitations of the Original Edgewood Arsenal Studies**

5 72. One problem with any retrospective investigation is that the analysis is limited by
6 the data (or lack of data) collected by the original investigators. As the NRC panel states, the
7 “main objective of [the Edgewood Arsenal] tests was to determine effects of various chemical
8 agents on the ability of test subjects to function effectively in a military situation.”⁹¹ It was not
9 the purpose of the Edgewood Arsenal tests to determine the long-term health effects from
10 exposure to these chemical agents, and “[i]t was not anticipated [by Edgewood Arsenal
11 investigators] that any late effects would occur.”⁹²

12 73. The NRC panel that prepared this report acknowledged that “the Edgewood tests
13 were intended for short-term and not long-term study and were therefore deficient in adequate
14 long-term controls.”⁹³ I agree with this statement. In my opinion, the limitations of the original
15 Edgewood Arsenal tests preclude any definitive statements regarding the long-term health of the
16 human test subjects used in those programs.

17 **b. Selection Biases**

18 74. The NRC panel’s investigation was also limited by problems in contacting the
19 approximately 6,720 subjects who participated in the original Army tests.⁹⁴ These problems
20 likely introduced substantial selection biases into the analysis, a common problem for studies that
21 did not plan explicitly for followup. Among the 6,720 subjects, 325 were already known to be
22

23 ⁹⁰ *Id.*

24 ⁹¹ NRC Volume 3 at 3.

25 ⁹² *Id.*

26 ⁹³ NRC Volume 3 at Executive Summary.

27 ⁹⁴ NRC Volume 3 at 2.

1 dead.⁹⁵ It is possible that the deceased subjects experienced the most health problems following
2 the Edgewood Arsenal tests, and their exclusion from the NRC panel’s investigation probably
3 resulted in an underestimate of long-term adverse health outcomes.

4 75. Among the approximately 6,395 test subjects presumed to be still living, another
5 1,399 subjects were lost from the analysis because the NRC panel could not obtain their current
6 mailing addresses.⁹⁶ The NRC panel stated that it “is not known whether this could be a serious
7 source of bias in the comparison of treatment groups,”⁹⁷ but I suspect that it was. This “lost”
8 group comprised nearly 22% of the remaining test subjects, and it is possible that many could not
9 be located because they were no longer living or were in poor health and no longer living
10 independently in their own housing. The potential bias introduced by their exclusion from the
11 analysis may have resulted in an underestimate of long-term adverse health outcomes.

12 76. The NRC panel also stated that the “911 men who received the questionnaire and
13 failed to respond were considered to constitute another potential source of bias, inasmuch as their
14 failure to respond could have resulted from an unhappy test experience.”⁹⁸ It is possible that an
15 “unhappy test experience” was the consequence of adverse reactions. If so, then at least some of
16 these 911 men were probably at higher risk for developing long-term adverse health effects due to
17 their participation in the tests at Edgewood Arsenal. If that is the case, then the NRC panel was
18 correct to consider the loss of that group as a potential source of bias—a selection bias that may
19 have resulted in an underestimate of long-term adverse health outcomes.

20 77. The NRC panel acknowledges the likelihood of selection bias, which they referred
21 to as “Response Bias,” because not all test subjects who were still assumed to be living had
22 participated in the survey.⁹⁹ For reasons that they explain in their report, the NRC panel

23 ⁹⁵ *Id.*

24 ⁹⁶ *Id.*

25 ⁹⁷ *Id.*

26 ⁹⁸ *Id.*

27 ⁹⁹ NRC Volume 3 at 15.

1 concludes that if a response bias exists, “it is in the direction of overestimation of current health
2 problems of the living volunteers.”¹⁰⁰ I disagree with this conclusion.

3 78. The NRC panel based their conclusion about the direction of selection bias on their
4 reported finding that the men who responded to the survey were hospitalized at a greater rate than
5 those who did not respond to the survey and those who could not be located.¹⁰¹ In my opinion,
6 the use of hospitalization rates—a necessity because of the paucity of other data available to the
7 NRC panel—is a poor way to estimate the frequency of health problems among the different
8 groups. There are multiple reasons, other than better health, that can explain the lower
9 hospitalization rate among those who did not respond to the survey or could not be located. First,
10 the NRC panel cannot even assume that all the non-located individuals are even alive. The NRC
11 panel used burial allowance claims received by the Veterans Administration to determine who is
12 alive or dead.¹⁰² This is not a particularly good way to separate the dead from the living. The
13 families of some deceased veterans may not have applied for a burial allowance (or even been
14 aware of this benefit). As another example, a homeless veteran¹⁰³ dying anonymously on the
15 streets of a major city is not likely to have a burial allowance claim filed on his behalf. So, some
16 of the non-located test subjects were possibly already dead, but assumed alive by the NRC panel.
17 Second, non-responding veterans and non-located veterans may not have been hospitalized
18 because they lacked access to health care services. After all, individuals in the United States who
19 have health insurance are more likely to utilize health care services than those who lack health
20 care insurance. Yet, the NRC panel seems to assume—incorrectly, in my opinion—that the non-
21 responding and non-located veterans had the same access to health care as the responding
22 veterans.¹⁰⁴ If the lower hospitalization rate among non-responding and non-located veterans was

23 ¹⁰⁰ NRC Volume 3 at 16.

24 ¹⁰¹ NRC Volume 3 at 15-16.

25 ¹⁰² NRC Volume 3 at 2.

26 ¹⁰³ Homelessness can certainly explain why some veterans were not locatable.

27 ¹⁰⁴ *See* NRC Volume 3 at 15-16.

1 indeed due, at least in part, to lack of access to health care, then the direction of selection bias
2 could be assumed to go towards an underestimation of current health problems, since people
3 without access to health care services tend to be less healthy overall compared to those who do
4 have access. In my opinion, the NRC panel was wrong and the direction of the selection bias
5 may very well be towards an underestimation of current health problems.

6 **c. Recall Bias**

7 79. This retrospective survey is also vulnerable to recall bias, where different veterans
8 may remember their medical history differently based on other factors. For example, those
9 veterans who had bad experiences during chemical testing may remember their subsequent
10 medical history in a different way compared to those whose experiences during chemical testing
11 was less negative. Recall biases are common for retrospective studies and make the interpretation
12 of retrospective survey results more difficult to interpret and validate.

13 **2. Underpowered Design Precludes Detection of Smaller Health Effects**

14 80. Limitations in the original Edgewood Arsenal study protocols and problems with
15 the NRC panel's own methods combined to make this study too underpowered to detect anything
16 but very large differences in clinical outcome. Smaller, but clinically important, differences in
17 outcome were likely to be missed entirely by the NRC panel's investigation. The NRC panel
18 admits this in their report:

19 The experimental methods and the available comparison groups
20 were such that only large effects were likely to be uncovered. The
21 large standard errors, the initial differences between the exposed
22 and nonexposed groups, the possibility that more than one
23 exposure might have led to the same adverse effect, and the self-
reporting nature of the questionnaire study all would tend to
obscure small differences.¹⁰⁵

24 81. The NRC panel performed power calculations to help determine the probability
25 that certain differences in outcome would be detected through the survey results.¹⁰⁶ The power

26 ¹⁰⁵ NRC Volume 3 at Executive Summary.

27 ¹⁰⁶ NRC Volume 3 at 5-6.

1 calculations are summarized in Tables 3 and 4.¹⁰⁷ These power calculations quantitatively
2 confirm the NRC panel’s qualitative assessment—i.e., that the statistical power of the survey
3 study for detecting small (but potentially clinically important) differences in outcome is low.
4 Unless an exposure caused a very large difference in clinical outcome, the difference in clinical
5 outcome was likely to be missed by this underpowered survey.

6 **3. Multiplicity of Chemical Exposures**

7 82. The NRC panel preparing NRC Volume 3 recognized and acknowledged a major
8 problem in the design and execution of the original chemical warfare testing program: the
9 multiplicity of chemical exposures.¹⁰⁸ As the NRC panel explained, “For the sake of efficiency,
10 many volunteers were used in two or more tests” involving exposure to multiple chemical
11 agents.¹⁰⁹ Whatever efficiency may or may not have been gained, the decision to expose many
12 subjects to multiple chemical warfare agents introduced potentially powerful confounding factors
13 that make it very challenging to ascribe long-term health effects to a specific test agent. The
14 NRC panel seemed to understand this, stating, “If a test substance produced detectable long-term
15 adverse effects in a man who was also exposed to another substance, it could be difficult to
16 ascribe the effect to the first substance alone, especially if many men were treated with both
17 substances.”¹¹⁰

18 83. In addition to the analytical challenges created by multiple exposures, it is also
19 important to recognize that exposure to multiple chemical warfare agents could possibly increase
20 the risk of long-term adverse health effects in an additive or synergistic manner. Therefore, while
21 exposure to multiple chemical agents can increase a test subject’s personal risk of developing
22 long-term adverse health effects, the study design flaw introduced by the original investigators
23

24 ¹⁰⁷ NRC Volume 3 at 35-36, Tables 3 and 4.

25 ¹⁰⁸ NRC Volume 3 at 3.

26 ¹⁰⁹ *Id.*

27 ¹¹⁰ *Id.*

1 made it much more challenging for the NRC panel to ascribe the cause of such adverse health
2 outcomes.

3 **4. Control Group—Healthy Soldier and Healthy Test Subject Effect**

4 84. The NRC panel recognized one of the main problems of the original chemical
5 warfare testing programs: the failure to include an appropriate control group.¹¹¹ They also
6 recognized the importance of the healthy soldier effect and the reasons why the U.S. general
7 population was an inappropriate control group for statistical comparisons. As the NRC panel
8 explained:

9 Men who are selected to serve in the Army are, in general, in better
10 physical and mental health than their peers. Because their later
11 health would also be expected to be better than average, it is
12 inappropriate to compare their health and life expectancies with
13 those of the general U.S. male population.¹¹²

12 85. The NRC panel also recognized and acknowledged the problem of the healthy test
13 subject effect, and explained why test subjects who did not receive any active test agent were an
14 unsuitable control group for those subjects who were exposed to active chemical agents:

15 The Volunteers received careful physical and mental screening
16 examinations for contraindications to the planned tests. The health
17 of a volunteer helped to determine the type of test in which he
18 participated. The more healthy men were exposed to the active
19 chemicals, and the less healthy were used as controls and in some
20 cases tested equipment without being exposed to chemicals. Such
21 selection bias means that the men not exposed to chemicals would
22 be expected to have more illness; therefore, the likelihood of
23 discovering effects in them (whether early or late) due to the
24 treatments would be smaller.¹¹³

21 86. The NRC panel was essentially left with no adequate control group for their
22 statistical analysis. The confounding biases introduced by the healthy soldier effect and healthy
23

24 ¹¹¹ *See id.*

25 ¹¹² *Id.*

26 ¹¹³ *Id.* The “selection bias” is the healthy test subject effect. In my opinion, it is
27 inappropriate to call the chemical warfare agents used, “treatments.”
28

1 test subject effect decreased the likelihood that the NRC panel would be able to detect long-term
2 adverse health outcome in the test subject population.

3 **5. Limitations of Cross-Sectional Study Design**

4 87. The NRC panel's survey is a cross-sectional investigation. In other words, the
5 NRC panel is taking a "snapshot" in time of the health of test subjects through their use of a
6 survey. Unlike longitudinal study designs where test subjects are followed over time, cross-
7 sectional studies are not nearly as good in identifying health trends over time within the test
8 subject population. The cross-sectional design of the NRC panel's survey made it less likely that
9 the NRC panel would be able to identify long-term adverse health outcomes attributable to
10 participation in chemical warfare testing programs.¹¹⁴

11 **6. Conclusions**

12 88. The NRC panel, recognizing the limitations of their retrospective survey, stated
13 that they "believed that the study might detect major effects if they were present and that the
14 limitations of the study could be appropriately described so its conclusions would not be
15 overinterpreted."¹¹⁵ In my opinion, any assertion that the NRC panel's findings represent a
16 definitive or conclusive study of long-term adverse health outcomes in chemical and warfare test
17 participants is an overinterpretation.

18 89. It is my opinion that the methodological problems in the original chemical warfare
19 testing program and the weaknesses of the NRC panel's own survey investigation preclude any
20 firm or definitive conclusions to be reached regarding the long-term health outcomes of test
21 subjects.

22 **D. Bullman and Kang, "A Fifty Year Mortality Follow-up Study of Veterans 23 Exposed to Low Level Chemical Warfare Agent, Mustard Gas"**

24 90. I have reviewed and analyzed a study published in 2000 by Tim Bullman and Han
25 Kang of the Environmental Epidemiology Service, Department of Veterans Affairs, titled "A

26 ¹¹⁴ NRC Volume 3 at 2.

27 ¹¹⁵ NRC Volume 3 at Executive Summary.

1 Fifty Year Mortality Follow-up Study of Veterans Exposed to Low Level Chemical Warfare
2 Agent, Mustard Gas.”¹¹⁶ The authors’ stated purpose was to “determine if there is an increased
3 risk of any cause specific mortality associated with low level mustard gas exposure among World
4 War II Navy veterans.” In their report, the authors found “no excess of any cause specific
5 mortality risks associated with varying levels of mustard gas exposures among Navy veterans
6 subject to the chamber tests.”¹¹⁷ As discussed below, I believe there are serious methodological
7 problems that prevent any firm conclusions to be drawn from the data presented in their report.

8 **1. Major Selection Bias in Excluding Important Veteran Populations**
9 **from the Study**

10 91. In discussing their methods, the authors note that mustard gas chamber tests were
11 performed at several sites, including the Naval Research Laboratory (“NRL”) in Washington,
12 D.C., the Edgewood Arsenal in Maryland, and the Great Lakes Training Center in Illinois.
13 Remarkably, Bullman and Kang simply note that since “NRL was the only site to maintain
14 accurate records of veterans who participated in chamber tests, NRL test participants were
15 selected as study subjects.”¹¹⁸ There is no explanation in the report discussing why the records of
16 veterans from Edgewood Arsenal and the Great Lakes Training Center were not deemed
17 “accurate,” or whether the differences in record keeping also reflected differences in how the
18 chamber tests were conducted at the various sites. The authors also fail to report how many
19 veterans were excluded from the study because of the decision to include only NRL veterans.
20 Just as importantly, there is no indication that the authors made any attempt to determine whether
21 the Edgewood Arsenal and Great Lakes Training Center veterans differed in any important way—
22 e.g., with respect to demographic characteristics, pre-existing conditions, or testing agent
23 exposure—compared to the NRL veterans, meaning there is no way to fully determine the types

24 ¹¹⁶ T. Bullman and H. Kang, “A Fifty Year Mortality Follow-up Study of Veterans
25 Exposed to Low Level Chemical Warfare Agent, Mustard Gas,” *Annals of Epidemiology*,
Vol. 10(5):333-338, 333 (2000) (hereinafter, “Bullman and Kang 2000”).

26 ¹¹⁷ Bullman and Kang 2000 at 333.

27 ¹¹⁸ Bullman and Kang 2000 at 334.

1 of biases and confounding factors that may have been introduced into the study by excluding
2 veterans from two of the three testing sites. The possible introduction of major selection biases
3 raises serious questions about the validity of the study and the generalizability of any reported
4 results to excluded veteran test subjects.

5 **2. Misclassification Bias Due to Non-Existent Documentation of Actual**
6 **Mustard Gas Exposure and the Use of Surrogate Exposure Markers**

7 92. According to the authors, the World War II era testing programs evaluated the
8 effectiveness of various protective clothing and equipment in preventing injury or incapacitation
9 due to mustard gas exposure.¹¹⁹ Because of the nature of the testing programs—evaluating
10 protective clothing and equipment—as well as possible poor record keeping, it is virtually
11 impossible to directly quantitate the physical exposure to mustard gas that veterans were
12 subjected to during the government’s testing programs. Bullman and Kang apparently recognized
13 this and instead used two surrogate measures of mustard gas exposure: a calculated “CT score”
14 and documented skin reactions to mustard gas. Exposure information is crucial for their analysis,
15 but the authors fail to identify where they obtained the data for these surrogate measures of
16 exposure and do not mention whether they performed any type of quality control assessment.¹²⁰
17 For the reasons that I discuss below, I believe the use of these questionable surrogate markers and
18 the non-existent documentation of actual mustard gas exposure probably resulted in substantial
19 misclassification bias that seriously undermines the results of the study.

20 **a. Misclassification Bias Due to the Use of the “CT score”**
21 **Surrogate Measure of Mustard Gas Exposure**

22 93. One of the surrogate measures of mustard gas employed by the authors was a “CT
23 score,” defined as the concentration of mustard gas in the chamber air, usually in milligrams per
24 liter, times length of exposure in minutes.¹²¹ The problems with using this surrogate measure of

25 ¹¹⁹ Bullman and Kang 2000 at 333.

26 ¹²⁰ See Bullman and Kang 2000 at 335 and Table 1.

27 ¹²¹ Bullman and Kang 2000 at 334.

1 exposure are obvious. Depending on the quality and effectiveness of the protective clothing and
2 equipment being tested, the concentration of mustard gas in the chamber air may have little
3 relation to the actual physical exposure of the test subject to the gas. For instance, a highly
4 effective protective suit may have prevented any physical exposure of a test subject to mustard
5 gas, even if testing was performed using a high concentration of mustard gas in the chamber air.
6 Alternatively, even a very low concentration of mustard gas for a very short period of time could
7 have resulted in a high physical exposure to mustard gas if the protective suit was defective or
8 ineffective. As illustrated in these examples, the use of CT score as a surrogate measure of
9 mustard gas exposure has the potential to either overestimate or underestimate the actual exposure
10 of test subjects to mustard gas. In Bullman and Kang’s study, this is a potentially important
11 source of error, as the authors used CT score to classify test subject exposure to mustard gas as
12 “high” or “low.”¹²² This type of bidirectional—sometimes called, non-differential—
13 *misclassification bias* (error) has the very strong potential to drive the results of an analysis
14 towards the null—i.e., towards a finding of no difference between exposure groups. The non-
15 differential misclassification bias introduced by the authors’ use of the CT score could very well
16 explain, in part, their study’s finding of no association between varying levels of mustard gas
17 exposure and an increased risk of any cause specific mortality.

18 94. Another source of misclassification bias with the use of the CT score surrogate
19 measure of mustard gas exposure is the authors’ seemingly arbitrary designation of CT scores of
20 100-120 as “low” exposure and scores of 121-960 as “high” exposure. The decision to include
21 CT scores of 120 in the “low” exposure group was especially crucial since more than half of all
22 test subjects (51.7%) had a CT score of 120.¹²³ The authors provide no explanation of how they
23 decided to use 120 as the cutoff for “low” exposure and there is no way to determine from their
24 report how the results of the study may have been different if they had used a different cutoff
25 number to differentiate between “low” and “high” exposures. This seemingly arbitrary method of

26 ¹²² *Id.*

27 ¹²³ Bullman and Kang 2000 at 335, Table 1.

1 classifying test subjects into “low” and “high” exposure groups is another important reason to
2 question the validity of the study’s results.

3 **3. Control Groups—Healthy Soldier and Healthy Test Subject Effects**

4 95. The authors claim that the “use of veterans as a referent group for other veterans
5 should minimize the effects of the so-called ‘healthy soldier effect.’”¹²⁴ While this may or may
6 not be true, the use of veterans stationed at the same location at the same time period would not
7 reduce any effects from a healthy test subject effect. There is no reason to expect that non-
8 exposed veterans based at the same location had the same general health risk profile as the
9 veterans who served as test subjects. It is very likely that test subjects were screened in some way
10 before being subjected to the mustard gas tests, likely resulting in a test subject population that
11 was healthier and otherwise different from the general population of military personnel at the
12 same location—i.e., a healthy test subject effect. With a better baseline health risk profile, one
13 would have expected the test subject group to have had better long-term health outcomes had they
14 not participated in the mustard gas experiments. We can infer that the test subject population was
15 very different from the general veteran population because the study authors had to exclude 956
16 veterans from a “random” sample of 3619 sailors (more than 26%) stationed at the same location
17 in order to obtain a control group that the authors perceived to be a match for the test subject
18 population.¹²⁵ Also indicating a difference between the test subjects and the control group is a
19 nearly three-year mean age difference between the groups,¹²⁶ suggesting that the test subjects
20 were younger and perhaps healthier than the control group at the time of entry. The authors
21 provide little information and virtually no statistical analyses comparing the demographic and
22 other characteristics of the test subject population and the control group, making it impossible to
23 fully assess whether the control population was an appropriate comparison group for the test
24 subject population. The paucity of information regarding the characteristics of the two groups

25 ¹²⁴ Bullman and Kang 2000 at 337.

26 ¹²⁵ Bullman and Kang 2000 at 334.

27 ¹²⁶ Bullman and Kang 2000 at 335.

28

1 also makes it impossible to know whether potential confounding factors (e.g., pre-existing
2 medical conditions, smoking habits, etc.) were evenly distributed between the two groups. These
3 problems lead me to question whether the control group used in this study was appropriate and
4 raise important concerns about the validity of the reported results.

5 **4. Underpowered to Detect Increases in Mortality Rate Under 100%**

6 96. The authors assert that their study has “substantial statistical power,” claiming
7 “over 95% statistical power to detect a 2-fold or more increased risk of deaths due to respiratory
8 cancers.”¹²⁷ Even if the authors’ claim is assumed to be true, this means the study was
9 sufficiently powered to detect a 100% increased risk of death from respiratory cancers, a very
10 large difference in mortality risk. Many investigators and most clinicians are likely to consider
11 even a 25% or 50% increased risk of death to be very important, but this study was underpowered
12 to detect such smaller, but important, differences in mortality rate. Furthermore, the authors
13 make no assertion about the power of the study for detecting increased risk of death from other
14 diseases, including skin cancers and chronic obstructive pulmonary disease. It is notable that the
15 authors reported an approximately 50% increase in the risk of death from chronic obstructive
16 pulmonary disease among test subjects with “high exposure” to mustard gas compared to all
17 Navy veterans with no exposure.¹²⁸ While the reported relative risk values (1.44 and 1.57) were
18 not deemed statistically significant,¹²⁹ the lack of statistical significance may reflect the
19 underpowered design of the study. In summary, while this study may have been sufficiently
20 powered to detect very large increases in risk of death, it was underpowered to detect smaller, but
21 still very clinically important, increases in mortality risk from exposure to mustard gas.

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25 ¹²⁷ Bullman and Kang 2000 at 337.

26 ¹²⁸ Bullman and Kang 2000 at 336, Table 3.

27 ¹²⁹ *Id.*

1 **5. Problematic Reliance on Death Certificates for Mortality Causation**
2 **Data**

3 97. The authors admit that “[r]eliance on death certificates for cause of death was a
4 potential weakness” of their study.¹³⁰ As they acknowledge, the accuracy of death certificates “in
5 recording cause-specific mortality, especially cancers, is somewhat variable.”¹³¹ There is no
6 indication in the report that the authors attempted to independently verify the cause(s) of death for
7 each deceased veteran. This is a weakness of the study, especially since the authors purport to
8 compare cause-specific mortality rates between the test subjects and control group.¹³² The
9 authors attempt to downplay this flaw by stating, “Even if lung cancer is underreported [in death
10 certificates], cancer rates would be equally underestimated for both exposed and unexposed
11 veterans.”¹³³ This reasoning is not reassuring, as underreporting in both groups would negatively
12 impact the power of the study for detecting differences in cause-specific mortality rates by
13 decreasing the overall number of reported cases in the test subject and control groups. In other
14 words, the reliance on death certificates for determining cause-specific mortality may bias the
15 study results towards the null hypothesis—i.e., towards a finding of no difference in relative
16 mortality risk between the test subject and control groups for specific causes of death.

17 **6. No Study of Morbidity**

18 98. This study specifically examines only mortality rates, and does not investigate
19 morbidity. Morbidity data are generally not available on death certificates, and at minimum,
20 obtaining morbidity data would require a detailed review of each veteran’s medical records.
21 Therefore, the study cannot rule out long-term adverse health effects that may impact the risk of
22 morbidity but may have a lesser, non-detectable impact on mortality rates.

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25 ¹³⁰ Bullman and Kang 2000 at 337.

26 ¹³¹ Bullman and Kang 2000 at 337.

27 ¹³² Bullman and Kang 2000 at 336, Tables 2 and 3.

28 ¹³³ Bullman and Kang 2000 at 337.

1 **7. Conclusions**

2 99. In my opinion, because of the methodological problems discussed above, no
3 definitive conclusions about relative mortality risks following exposure to mustard gas can be
4 drawn from the data presented by Bullman and Kang in this report.

5 **E. The 1980 LSD Follow-Up Study Report**

6 100. Between 1955 and 1967, the U.S. Army Chemical Corps and the U.S. Army
7 Intelligence Corps conducted a series of experiments on human test subjects using lysergic acid
8 diethylamide (“LSD”).¹³⁴ Most of this testing was performed at Edgewood Arsenal, although
9 other sites were used as well.¹³⁵

10 101. After the studies had concluded in 1967, the U.S. Army was notified that one of
11 the former LSD test subjects had developed temporal lobe epilepsy.¹³⁶ In addition, public and
12 Congressional interest in the LSD testing program had grown because of the public disclosure of
13 the suicide in 1953 of an Army mathematician shortly after being given LSD covertly by
14 government experimenters.¹³⁷ The LSD Follow-Up Study was intended to evaluate former LSD
15 test subjects for possible long-term adverse effects from their exposure to LSD and participation
16 in the Army’s testing program.¹³⁸ It is supposed to be the government’s most complete follow-up
17 study of former LSD test subjects.

18 **1. Inability to Obtain a Matched Control Group—Healthy Soldier and**
19 **Healthy Test Subject Effects**

20 102. The authors of the LSD Follow-Up Study stated that a “major and eventually
21 insuperable problem arose with regarding to the proposed study design; namely, it proved

22
23 ¹³⁴ U.S. Army Medical Department, “LSD Follow-Up Study Report,” October 1980
(hereinafter, “LSD Follow-Up Study”) at 1.

24 ¹³⁵ LSD Follow-Up Study at 1-2.

25 ¹³⁶ LSD Follow-Up Study at 2.

26 ¹³⁷ *Id.*

27 ¹³⁸ LSD Follow-Up Study at 2-3.

1 impossible to obtain matched controls for the LSD-exposed subjects.”¹³⁹ Whether it was truly
2 “impossible” to come up with a matched control group is debatable. It is at least clear that the
3 investigators failed to produce an adequate control group for the study. Instead of using a
4 matched control group for their study, the investigators compared the LSD test subject group with
5 age-similar males in the general U.S. population.¹⁴⁰ Even the authors acknowledged that using
6 the general U.S. population as a control group was a “much less satisfactory strategy.”¹⁴¹ I agree
7 and believe the control group used in this study was inappropriate and inadequate for the reasons
8 outlined below.

9 103. The major problem with using age-similar males in the general U.S. population as
10 a control group is that the analysis will be biased towards the null hypothesis (i.e., a finding of no
11 difference in health outcome) by two confounding factors: the healthy soldier effect and the
12 healthy test subject effect (see my discussion above regarding these two effects). Since LSD test
13 subjects had been U.S. military personnel, they were, as a group, more healthy on average than
14 the general U.S. population—the healthy soldier effect. As discussed earlier in this report, this is
15 because individuals must undergo physical and mental health evaluations before they are
16 permitted to serve in the U.S. military. Since the LSD test subjects were, on average, healthier
17 than the general U.S. population before they participated in the LSD experiments, any adverse
18 effects would tend to be masked by a comparison between the two groups.

19 104. In addition, the “LSD subjects were not in any sense a random cross-section of the
20 Army population.”¹⁴² For instance, the “average intelligence and level of education of the LSD-
21 treated group ... was considerably higher than that of the Army population in general.”¹⁴³
22 Reportedly, many of the LSD test subjects were Army Chemical Corps or Intelligence Corps

23 ¹³⁹ LSD Follow-Up Study at 4.

24 ¹⁴⁰ LSD Follow-Up Study at Executive Summary.

25 ¹⁴¹ *Id.*

26 ¹⁴² LSD Follow-Up Study at 4.

27 ¹⁴³ *Id.*

1 officers with advanced scientific degrees.¹⁴⁴ The likely effect of all this is that the LSD test
2 subject group was, on average, healthier and probably more adaptable (because of their
3 intelligence), than the general Army population—a healthy test subject effect. Since the LSD test
4 subjects were likely, on average, to be healthier than the general Army population, this would
5 again tend to bias the results of any comparisons with the U.S. general population towards the
6 null hypothesis (i.e., a finding of no difference in health outcomes).

7 105. Since there was likely a very powerful healthy soldier effect and healthy test
8 subject effect biasing the results of this study, I believe that any comparisons in the LSD Follow-
9 Up Study between the LSD test subjects and the general U.S. population of age-similar males to
10 be meaningless. Because the original LSD studies did not include an appropriate control group,
11 and because the investigators failed to put together an appropriate matched control group, the
12 LSD Follow-Up Study cannot be deemed to have any appropriate control group to which the LSD
13 test subject group may be compared.

14 2. Biases Arising from Retrospective Design

15 106. It is important to remember that the original LSD studies performed by the U.S.
16 Army at Edgewood Arsenal did not include any plan for long-term follow up of the test subjects.
17 The LSD Follow-Up Study is a retrospective study and is therefore vulnerable to all the biases
18 that can affect retrospective study designs.

19 a. Selection Biases

20 107. The investigators were only able to perform a follow-up examination or interview
21 with a portion of the original LSD study participants.¹⁴⁵ Among 741 LSD test subject identified,
22 only about 220 could be examined directly and another 100 questionnaires completed. Another
23 149 refused to participate and 193 others could not be located. Fifty-five LSD test subjects, for
24 reasons that are not clearly explained, were followed separately because they were now U.S. Air
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26 ¹⁴⁴ LSD Follow-Up Study at 4-5.

27 ¹⁴⁵ LSD Follow-Up Study at 11.

1 Force personnel.¹⁴⁶ There is no evaluation, qualitative or quantitative, presented in the report to
2 determine whether any of these excluded groups differed in any significant way from the 320
3 LSD test subjects who were eventually evaluated. Therefore, it is very possible that significant
4 biases were introduced into the study because of these exclusions.

5 108. Importantly, 24 of the 741 original LSD test subjects had already died and were
6 not included in the final analysis.¹⁴⁷ This has the potential to introduce a major selection bias into
7 the study since some of the deceased individuals may have been among those who had the most
8 severe adverse reactions to LSD exposure.

9 **b. Retrospective or Observer Bias**

10 109. As the authors acknowledge, the LSD Follow-Up Study was designed and
11 performed after the U.S. Army's LSD testing program had come under increased public
12 scrutiny.¹⁴⁸ This raises the possibility that some type of observer bias may have been introduced
13 into the study, since there may have been some pressure, whether intended or not, placed on the
14 investigators to minimize any damage to the reputation of the U.S. Army, which had conducted
15 the original LSD studies.

16 **c. Limitations of Cross-Sectional Design**

17 110. Since the original LSD testing program did not include any plans for long-term
18 follow-up of the test subjects, it was not possible to follow the health of the test subjects in a
19 longitudinal manner (i.e., over the course of time). Instead, partly because the LSD Follow-Up
20 Study is a retrospective study, it has a cross-sectional design, meaning that the individuals
21 assessed in the study are evaluated at just one "snapshot" in time. Since cross-sectional studies
22 evaluate individuals at just one point in time, rather than follow the individuals over a number of
23 years or decades, cross-sectional studies are not as good in identifying any trends (e.g., health

25 ¹⁴⁶ *Id.*

26 ¹⁴⁷ *Id.*

27 ¹⁴⁸ *See* LSD Follow-Up Study at 2.

1 trends) over time among the study population. This is a major limitation of the LSD Follow-Up
2 Study.

3 **3. Meaningful Data**

4 111. Since the LSD Follow-Up study does not have an appropriate control group, the
5 most meaningful data in the study is the survey of health problems reported by former LSD test
6 subjects. While quantitative comparisons with the U.S. general population are not meaningful, it
7 is still important that a substantial number of LSD test subjects report having adverse reactions—
8 e.g., persistent flashbacks, depression, and personality changes—that are probably related to their
9 exposure to LSD.¹⁴⁹ The investigators defined certain adverse reactions as a “probable LSD
10 effect” if it was reported to have initially occurred within 2 years of LSD exposure and was an
11 adverse reaction similar to known long-term effects of LSD or could conceivably have been
12 caused by LSD even if not previously reported.¹⁵⁰ I think this definition of “probable” LSD effect
13 may be too restrictive, since it is now known that certain long-term adverse reactions to LSD
14 exposure (e.g., flashbacks) may initially occur more than two years following the last dose of
15 LSD. The definition is also somewhat vague in defining adverse reactions that could conceivably
16 have been caused by LSD. Even so, this survey of reported long-term adverse reactions is the
17 most valuable data in the report and is clinically meaningful, despite the limitations of the LSD
18 Follow-Up Study’s design.

19 **4. Conclusions**

20 112. The lack of a proper control group and the retrospective design are major
21 weaknesses of the LSD Follow-Up Study.

22 113. Nevertheless, the LSD Follow-Up Study provides meaningful data through its
23 survey of reported long-term adverse reactions.

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26 ¹⁴⁹ LSD Follow-Up Study at 21-22.

27 ¹⁵⁰ LSD Follow-Up Study at 21.
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1 **F. William Page Institute of Medicine Study**

2 114. It is my understanding that this study¹⁵¹ will be discussed in detail in another
3 report. I give only some brief opinions here. I may testify more extensively and in greater detail
4 regarding this study.

5 115. This study has a number of methodological problems which I will briefly outline
6 here. First, it utilizes a cross-sectional design that provides only a “snapshot” in time of health
7 outcome in the responding group.¹⁵² A cross-sectional study is generally not as good as a
8 longitudinal study for determining health trends over time.

9 116. In addition, the study appears to have an important selection bias, with the
10 Nonrespondent group having important differences in key characteristics compared to the
11 Respondent Group. For example, the Nonrespondent group had, on average, significantly less
12 education beyond high school, a higher rate of hospital admissions between 1980 and 1985,
13 worse overall health, higher levels of beer and whiskey consumption, and higher use of heroin.¹⁵³
14 All of that suggests that the Nonrespondent group was less healthy than the Respondent group,
15 and that less healthy individuals were being systematically eliminated from the study. This can
16 only make it less likely that the study can detect long-term adverse health outcomes in former test
17 subjects.

18 117. The Page study used a survey method for gathering data¹⁵⁴, and therefore is subject
19 to the confounding effects of recall bias (errors in the way subjects recall their own medical and
20 personal history). This too makes the health outcome results of this study less trustworthy.

21 118. Also a source of selection bias is the decision to eliminate all known decedents
22 from the analysis. Obviously, decedents cannot complete surveys, but there was no attempt to

23 ¹⁵¹ W. Page, “Long-Term Health Effects of Exposure to Sarin and Other
24 Anticholinesterase Chemical Warfare Agents,” *Military Medicine* 168:239-245 (2003)
(hereinafter, “Page”).

25 ¹⁵² Page at 239-240.

26 ¹⁵³ Page at 241, Table II, 242-43.

27 ¹⁵⁴ Page at 239-240.

1 obtain detailed health histories of these dead former test subjects, who may have been among
2 those with the most adverse health reactions to their participation in the military tests.

3 119. The study also lacks an optimal control group.

4 120. In my opinion, the methodological problems of this study preclude any definitive
5 conclusions from being reached based on the study regarding the long-term health effects of
6 exposure to sarin and other anticholinesterase chemical warfare agents.

7 **VI. VETERANS OUTREACH LETTER, FACT SHEET, AND FREQUENTLY ASKED** 8 **QUESTIONS**

9 121. I have had the opportunity to review the Department of Veterans Affairs
10 Chem-Bio Outreach Letter (“Outreach Letter”), which I understand is a generic form letter that
11 was sent to some test subjects along with the Fact Sheet and Frequently Asked Questions sheet
12 discussed below. The Chem-Bio Outreach Letter is not particularized to reflect the circumstances
13 of any individual’s exposures or those of any discrete group. It does not identify what substances
14 a veteran receiving it was exposed to, and it does not disclose what sorts of long-term effects a
15 veteran might suffer as a result of their exposure.

16 **1. Fact Sheet from the Deployment Health Support Directorate**

17 122. I have reviewed a document with the header, “Edgewood Arsenal Chemical Agent
18 Exposure Studies: 1955-1975” (“Fact Sheet”). It is my understanding that the Fact Sheet was
19 attached to the Department of Veterans Affairs’ Outreach Letters sent to some of the veterans
20 who served as test subjects.

21 123. I have a few observations concerning certain statements in the Fact Sheet. The
22 Fact Sheet states that “The Institute of Medicine (IOM) published a three-volume study between
23 1982 and 1985 on the long-term health effects of exposure to the chemicals tested. The study did
24 not detect any significant long-term health effects in Edgewood Arsenal volunteers.” [citations
25 omitted]. First, this statement mischaracterizes the conclusion of the study. Second, as discussed
26 above, the design and quality of the study were inadequate to definitively determine whether there
27 are any significant long-term health effects in Edgewood Arsenal volunteers. This statement in
28 the Fact Sheet is therefore inaccurate because it fails to discuss the study’s many methodological

1 limitations, including those acknowledged by the authors, and its generally indeterminate findings
2 and conclusions.

3 124. The Fact Sheet also states that “The study investigators assured that the exposure
4 levels administered would not result in serious or life-threatening side effects.” There are
5 numerous examples, however, of serious acute side effects from exposure to the chemical and
6 biological agents used in these experiments. These serious side effects include, for example, the
7 initiation of a life-threatening grand mal seizure in one case and hallucinogenic flashbacks in
8 other cases. (See discussion above.)

9 2. **□Frequently Asked Questions: Edgewood Arsenal Chemical Agent
10 Exposure Studies: 1955-1975□**

11 125. I reviewed a document with the header, “Frequently Asked Questions” (“FAQs”).
12 It is my understanding that the FAQs were distributed with the Outreach Letters to some of the
13 veteran test subjects. The FAQs state that “The Army obtained the voluntary consent of
14 volunteers and provided them with study information.” True “voluntary consent” comprises
15 informed consent without coercion. It cannot be stated with certainty that coercion was not used
16 to recruit military test subjects for the various chemical agent tests.

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Respectfully submitted,

Dated: AUG 8, 2012

Daniel E. Ford, M.D., M.P.H.

Exhibit 1

Curriculum Vitae

Daniel E. Ford, M.D., M.P.H.

DEMOGRAPHIC INFORMATION

Current Appointments

- 2007- Director, Institute for Clinical and Translational Research
- 2005- Vice Dean for Clinical Investigation, Johns Hopkins University School of Medicine
- 2002- Professor, Johns Hopkins University School of Medicine, Department of Medicine, Division of General Internal Medicine, Welch Center for Prevention, Epidemiology and Clinical Research, Baltimore, Maryland
- 2002- Joint Appointment, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Maryland
- 1988- Joint Appointment, Department of Epidemiology, Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland
- 1988- Joint Appointment, Department of Health Policy and Management, Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland
- 1988- Active Full-time Medical Staff, Johns Hopkins Hospital, Baltimore, Maryland

Education and Training

- 1978 (BA) Cornell University, Ithaca, New York
- 1982 (MD) State University of New York at Buffalo, Buffalo, New York
- 1986 (MPH) Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland
- 1980 Summer Geriatric Research Fellowship, State University of New York at Buffalo School of Medicine. Effect of nonsteroidal anti-inflammatory drugs on urinary prostaglandins and renal function in the elderly.
- 1982-83 Osler Medical Intern, Johns Hopkins Hospital, Baltimore, Maryland
- 1983-85 Osler Medical Resident, Johns Hopkins Hospital, Baltimore, Maryland
- 1985-88 Part-time Clinical Fellow, Department of Medicine, Division of Internal Medicine, Johns Hopkins Hospital, Baltimore, Maryland
- 1985-88 Epidemiology Training Fellowship, U.S. Public Health Service
- 1985-88 Medical Staff Fellow, Primary Care Section, Clinical Services Research Branch, Division of Biometry and Epidemiology, National Institute of Mental Health, Rockville, Maryland
- 1990-91 Faculty Development, Johns Hopkins Bayview Medical Center, Primary Care, Internal Medicine, "Curriculum Development for Primary Care Internal Medicine"
- 1991-92 Faculty Development, Johns Hopkins Bayview Medical Center, Primary Care, Medicine, "Clinician-Teacher in Primary Care Internal Medicine"

Daniel E. Ford, M.D., M.P.H.

Professional Experience

- 1988-90 Instructor, Johns Hopkins University School of Medicine, Department of Medicine, Division of General Internal Medicine, Welch Center for Prevention, Epidemiology and Clinical Research, Baltimore, Maryland
- 1988- Joint Appointment, Johns Hopkins University Bloomberg School of Public Health, Department of Epidemiology, Baltimore, Maryland
- 1988- Joint Appointment, Johns Hopkins University Bloomberg School of Public Health, Department of Health Policy and Management, Baltimore, Maryland
- 1988- Active Full-time Medical Staff Appointment, Johns Hopkins Hospital, Baltimore, Maryland
- 1990-95 Assistant Professor, Johns Hopkins University School of Medicine, Department of Medicine, Division of General Internal Medicine, Welch Center for Prevention, Epidemiology and Clinical Research, Baltimore, Maryland
- 1995-2002 Associate Professor, Johns Hopkins University School of Medicine, Department of Medicine, Division of General Internal Medicine, Welch Center for Prevention, Epidemiology and Clinical Research, Baltimore, Maryland
- 2002- Professor, Johns Hopkins University School of Medicine, Department of Medicine, Division of General Internal Medicine, Welch Center for Prevention, Epidemiology and Clinical Research, Baltimore, Maryland
- 2003- Joint Appointment, Johns Hopkins Department of Psychiatry

RESEARCH ACTIVITIES

Publications – Peer-Reviewed Articles

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2. **Ford DE**, Kamerow DB and Thompson JW. Who talks to their physician about mental health and substance abuse problems? *J Gen Intern Med* 1988;3:363-369.
3. **Ford DE** and Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders: An opportunity for secondary prevention? *JAMA* 1989;262:1479-1484.
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Publications – Non-Peer Reviewed Articles

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5. McCauley JE, **Ford DE**, Jones CA and Kern D. Psychiatric distress as a predictor of patients initiating a weight reduction program. *The Bariatrician*, Fall 1992.

Publications – Book Chapters

1. Hayward RSA, Steinberg EP, **Ford DE**, Roizen MF, Roach K. Guidelines for periodic preventive care. In: Eddy EM (ed.) Common Screening Tests. Philadelphia, PA: American College of Physicians, 1991.
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Publications – Miscellaneous

- Letters to Editor: **Ford DE**, Sciamanna CN. Nutritional Counseling in Community Office Practices. *Ach Intern Med* 1997;157:361-362.
- Editorial: **Ford DE.** Managing Patients with Depression: Is Primary Care Up to the Challenge. *J Gen Intern Med* 2000;15(5):344-345.
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- Beach MC, **Ford DE.** Compassion and integrity in medical education. Review of: Ward Ethics: Dilemmas for medical students and doctors in training. *Yale Journal of Health Policy, Law and Ethics*. 2001;2(1):211-217.
- Editorship: **Ford DE.** Co-Editor with PK Whelton and Leon Gordis. Special Supplement of *Journal of General Internal Medicine*, September/October 1990. "Impact of the U.S. Preventive Services Task Force Report."
Associate Editor, *Journal of General Internal Medicine* 1999-2004

Inventions, Patents, Copyrights

None.

Extramural Sponsorship

Research Grant Participation – Current

Daniel E. Ford, Principal Investigator (25%) 09/30/07-09/29/12 5UL1RR025005-01A1 \$19,008,590
National Institute of Health
Institutional Clinical and Translational Science Award
This CTSA grant will support clinical and translational research throughout Johns Hopkins. It includes support for education and training of new translational investigators, facilities in which clinical research can take place and infrastructure support of patient recruitment, bioinformatics, biostatistics and translational core centers.

Daniel E. Ford, Principal Investigator 07/01/94-06/30/11 T32 HP10025-15 \$309,182
HRSA, Bureau of Health Professions
Institutional National Research Service Award-Primary Care Research
To identify general internists and support their development as creative and independent investigators in primary care research, determine the exact role for primary care in the health care delivery system and provide an opportunity for fellows to develop research skills in primary care.

University of Alabama Subcontract(Houston)04/01/2008-03-31/2013 \$34,719
National Institute of Health
QUIT – PRIMO Web Delivered Clinical Microsystem Intervention for Tobacco Control

Research Grant Participation – Pending

Daniel e. Ford, Principal Investigator 07/01/10-06/30/15 R01 \$448,166
National Institute of Mental Health
Health IT to Reduce Mental health Stigma in University Students
The objective of this study is to definitively test the efficacy of an innovative multi-level model of web-based continuous psycho-education (COPE) aimed to facilitate willingness to seek psychological help in university students.

Research Grant Participation – Previous

Daniel E. Ford, Co-Investigator National Institute of Aging Precursors of Premature Disease and Death To continue the longitudinal description of the onset of disability and death in a standardized fashion in this cohort now approaching retirement, to identify those genetic, physiologic and health behavioral and death prior to the age of 65 years; to describe the role of a variety of midlife health behaviors in the prediction of premature aging, to develop a midlife psychological profile of factors, and to measure specific life events which may act as stressors	09/01/93-02/28/10	R01 AG01760 (Klag)	\$350,888
Daniel E. Ford, Co-Investigator (15%) National Institute on Aging. (Michael J. Klag, P.I.) Precursors of Premature Disease and Death. To identify factors associated with premature disease, disability and death in former Johns Hopkins medical students.	09/30/03-08/31/08	1 R01 AG01760	\$1,820,808
Daniel E. Ford, Co-Investigator (10%) AHRQ (Lisa A. Cooper, P.I.) Patient-Centered Depression Care for African Americans To improve primary care physician's knowledge and communication skills as they relate to African-American patients with depression so that these patients are more likely to receive high quality care for depression.	09/30/03-08/31/08	1 R01 HS13645	\$2,132,297
Daniel E. Ford, Principal Investigator (25%) National Institute of Mental Health Development of Internet Intervention for Depression The goal of this project is to develop and evaluate the benefits of an internet-based depression support group for patients presenting to primary care with depression.	09/01/06-06/30/08	1 R34 MH073742	\$334,000
Daniel E. Ford, Principal Investigator (5%) HRSA Institutional National Research Service Award. To support and train six post-doctoral fellows annually in primary care research.	07/01/03-06/30/08	2 T32 HP10025	\$2,119,753
Daniel E. Ford, Co-Investigator (10%) National Institute of Mental Health (William W. Eaton, P.I.) Evolution of Psychopathology in the Population. To continue ten-year follow-up of the 1981-1982 Epidemiologic Catchment Area sample in East Baltimore; consequences of mental illness, such as mortality and cardiovascular disease.	07/01/02-06/30/07	1 R01 MH47447	\$3,723,120
Daniel E. Ford, Principal Investigator (25%) Robert Wood Johnson Foundation Implementation Evaluation of the Implementation Phase of the Depression in Primary Care Program. To implement an evaluation process at designated clinical and HMO sites assessing current support of treatment for depression.	02/01/03-01/31/05	045542	\$458,602
Daniel E. Ford, Co-Investigator (15%) NIMH (Thomas G. McGuire, P.I. Harvard, Daniel Ford, P.I. JH) Economics of Racial/Ethnic Disparities in Mental Health	03/01/04-02/28/05	Subcontract Subcontract	\$18,172
Daniel E. Ford, Principal Investigator (10%) National Cancer Institute. Smoking Cessation Coach: An Internet Tailoring Program. To explore potential methods of incorporating concepts of patient activation and social networking into an existing evidence-based primary care smoking cessation computer program; design and implement an Internet-based smoking cessation system and complete three pilot studies.	07/01/01-06/30/05	1-R01-CA-89011	\$200,000
Daniel E. Ford, Subcontract P.I. (10%)	03/15/00-02/28/04	1-R01-MH57852	

Daniel E. Ford, M.D., M.P.H. National Institute of Mental Health. (Joseph Gallo, P.I.) The Spectrum of Depression Late Life: A Primary Study. To systematically describe and validate a depressive syndrome, apathetic depression, that does not meet standard criteria for major depression in older primary care patients; assess how physical illness, cognitive impairment, anxiety, and hopelessness among older primary care patients alters the course of.	Subcontract Subcontract	\$94,648	
Daniel E. Ford, Principal Investigator on Major Component Subcontract (17.5%) CDC Funds (Association of Schools of Public Health Grant S1751-21/21) (Louise-Anne McNutt, P.I. Univ Albany; Daniel Ford, P.I. JH) Evaluation of an Intimate Partner Violence Intervention To decrease intimate partner violence, and improve health outcomes by training physicians and entire health care team in OB and GIM clinics, screening protocols, patient education, tailored approaches based on severity of abuse; flexible treatment options and active follow-up	Subcontract Subcontract	\$847,644	10/01/01-09/30/04
Daniel E. Ford, Principal Investigator (25%) National Institute of Mental Health. Quality Improvement for Depression. Randomized clinical trial to determine if implementation of AHCPR Guidelines for Treatment of Depression in Primary Care changes medical practice and improves patient outcomes.		\$959,012	09/15/98-11/30/04 2 R01 MH54443
Daniel E. Ford, Co-Investigator (10%) Aetna/U.S. HealthCare (Lisa A. Cooper, P.I.) Using Patient-Provider Communication Skills Training to Improve Depression Care for African Americans		\$249,995	01/01/02-12/31/04
Daniel E. Ford, Principal Investigator (25%) Robert Wood Johnson Foundation Evaluation Plan for the First Year of the Depression in Primary Care Program. To understand health care organizations In terms of the current support of treatment for depression, their potential to achieve change, models and process for change.		\$149,921	03/01/02-12/31/03 040688 Planning Phase
Daniel E. Ford, Co-Investigator (10%) National Institute on Alcohol Abuse and Alcoholism (Rosa M. Crum, P.I.) Sleep Disturbances and Risk for Alcohol Disorders		\$150,000	10/01/01-09/30/03 1-R21-AA13251
Daniel E. Ford, Co-Investigator (15%) National Institute on Aging. (Michael J. Klag, P.I.) Precursors of Premature Disease and Death.		\$1,199,683	09/01/98-08/31/03 1 R01 AG01760
Daniel E. Ford, Principal Investigator (5%) HRSA Institutional National Research Service Award.		\$1,382,755	07/01/98-06/30/03 2 T32 PE10025
Daniel E. Ford, Co-Investigator (10%) National Institute of Mental Health (William W. Eaton, P.I.) Evolution of Psychopathology in the Population.		\$1,296,038	03/01/97-06/30/02 1 R01 MH47447
Daniel E. Ford, Principal Investigator (15%) Aetna/U.S. Healthcare. Indicators of Quality of Care in Primary Care.		\$249,995	11/01/98-06/30/02
Daniel E. Ford, Principal Investigator (0%) John D. and Catherine MacArthur Foundation/RAND. Improving the Quality of Depression Treatment for Disadvantaged Populations		\$67,844	07/01/00-06/30/02 00-59301-A-HCD
Daniel E. Ford, Co-Investigator (0%) Bayer Institute for Health Care Communication. (Thomas Houston, P.I.) Electronic Mail: The Potential Impact on Physician-Patient Communication.		\$20,000	09/01/00-05/01/02 00-577
Daniel E. Ford, Principal Investigator (15%) John D. and Catherine MacArthur Foundation. Quality Improvement in Depression.		\$125,000	09/01/96-08/31/00 406
Daniel E. Ford, Principal Investigator (25%) National Institute of Drug Abuse. Cancer Risk and Preventive Health for Marijuana Users.		\$633,963	04/01/94-03/31/00 1 R01 DA08199

Daniel E. Ford, M.D., M.P.H. Daniel E. Ford, Principal Investigator (20%) 09/30/94-08/31/98 1 U01 MH54443 National Institute of Mental Health. Implementation of Depression Practice Guidelines.	\$1,020,051
Daniel E. Ford, Principal Investigator (0%) 07/01/94-06/30/98 1 T32 PE10025 Health Resources and Services Administration. National Service Research Award. .	\$540,233
Daniel E. Ford, Principal Investigator (15%) 1995-1998 Health Services Cost Review Commission of Maryland. In-Patient Smoking Cessation Program.	\$175,874
Daniel E. Ford, Co-Investigator (15%) 09/01/93-08/31/98 1 R01AG01760 National Institute on Aging. (Michael J. Klag, P.I.). Precursors of Premature Disease and Death.	\$1,343,999
Daniel E. Ford, Co-Investigator (10%) 04/01/92-02/28/97 1 R01 MH47447 National Institute of Mental Health (James C. Anthony and William W. Eaton, P.I.) Evolution of Psychopathology in the Population.	\$3,117,003
Daniel E. Ford, Co-Principal Investigator 07/01/94-06/30/97 Miles Institute for Health Care Communication. (Michael J. Klag, P.I.)	\$71,438
Daniel E. Ford, Co-Principal Investigator (10%) 10/01/93-09/30/96 H042SC The Pew Memorial Trust. (Paul K. Whelton, P.I.) Health of the Public.	\$200,000
Daniel E. Ford, Principal Investigator (50%) 08/01/90-01/31/95 1 R29 MH46967 National Institute of Mental Health. Sleep and Mental Disorders in General Medical Settings.	\$298,726
Daniel E. Ford, Principal Investigator 07/01/91-06/30/92 Johns Hopkins University Department of Medicine. Prevention Practice Project.	\$20,000
Daniel E. Ford, Co-Principal Investigator (25%) 10/01/87-09/20/92 T86-06020-003 The Pew Memorial Trust. (Leon Gordis, P.I.) Health of the Public.	\$1,000,000
Daniel E. Ford, Co-Investigator (10%) 03/01/89-02/28/91 Health Services Cost Review Commission of Maryland Illness Prevention Program. (Diane Becker, P.I.) Family and Community Heart Disease Prevention Program.	\$358,291

EDUCATIONAL ACTIVITIES

Teaching Activities – Academic – Johns Hopkins University School of Medicine

1989-92	Medicine Clerkship (Year III) – Regular participant in computer-based learning segment
1989-	Medical School (Year IV) – Developed and Directs Senior Elective in Clinical Preventive Medicine
1990-98	Clinical Epidemiology Course (Year I) – Lab Instructor
1990-95	Introduction to Clinical Skills Course (Year II) – Assistant Director Organized Module on Sexual History and Male Rectal-Genital Examination
1992-96	Physician and Society Course – (Year I and Year II) Developed and Direct Unit on Physician-Patient Communication
1992-94	Member of Research Team Evaluating New Curriculum for the Johns Hopkins University School of Medicine
1996-2005	Introduction to Medicine, Clinical Skills Course (Year II)
1998-2002	Continuing Medical Education Course -- Clinical Preventive Medicine – Director
2003-2008	Basic Pharmacology Course MS II – Smoking Cessation – Lecturer
2008-	Intensive Course in Clinical Research Methods - Director

Teaching Activities -- Academic -- Johns Hopkins University Bloomberg School of Public Health

- 1988-2002 Course Director "Fundamentals of Clinical Preventive Medicine" - Johns Hopkins University School of Hygiene and Public Health
1989-2008 Course Director "Principles of Clinical Epidemiology" - Johns Hopkins University School of Hygiene and Public Health
1991- Lecturer in Epidemiology and Prevention of Cardiovascular Disease Course
1997- Lecturer in Outcomes Assessment Course

Teaching Activities -- Clinical -- Johns Hopkins University School of Medicine

- 1990-93 Lecturer and Preceptor at Blalock 2 Ambulatory Medicine, Internal Medicine Residents' Clinic

Mentoring -- Advisees Pre- and Post-Doctoral

- 1989 Stefano Marino PhD; Visiting Post-doctoral Fellow
Research: Use of general medical and specialty mental health services by individuals with incident psychiatric disorders
1991 Frederick Brancati, MD MHS; MHS candidate
Research: Weight reduction services in general medical settings
1991 Luis Camacho MD; PhD Candidate-Epidemiology
Research: Reliability and validity of quality of care measures
1991 Rosa Crum, MD MHS; MHS candidate, Department of Epidemiology
Research: Detection of alcohol abusers in general medical settings
1991 Edward Ellerbeck MD MPH; Fellow, Division of Internal Medicine
Research: Involving patients to improve preventive care
1991 Karen Johnson MD MPH; Preventive Medicine Residency
Research: Injury prevention counseling by primary care providers
1991 Ellen Strahlman MD MHS; MHS candidate; Fellow, Department of Ophthalmology Research: Vision screening
1991 Fang Wang MD; PhD Dissertation, Department of Epidemiology
Research: Vision screening in a primary care setting
1992 Robert Hayward MD MPH; Fellow, Division of Internal Medicine
Research: Implementation of prevention guidelines
1992 Joseph Gallo, MD MPH; Department of Mental Hygiene
Research: Use of general medical services by individuals with mental disorders
1992 Melinda Midzenski; MHS, Department of Epidemiology
Research: Perception of risk and cancer prevention activities of oncology center employees
1992 Aaron Tokayer, MD; MHS, Department of Epidemiology
Research: Decision analysis of work-up for occult blood in the stool
1993 Yuriko Egami, MD MHS; Postdoctoral fellow, Department of Epidemiology
Research: Psychiatric diagnosis in self-reported child abuse
1993 Lisa Cooper MD; MPH; Fellow, Division of Internal Medicine
Research: Management of depressed patients in general medical settings
1993 Alison Schecter, MD; Resident in Medicine
Research: Preferences for invasive cardiac care for CCU patients
1994 Patricia Chang; Medical student
Research: Risk factors for depression in physicians
1994 Louis Franciscutti, MD PhD; Preventive Medicine Residency
Research: Preventive services in the emergency room setting
1994 Stephen D. Ryan, MD; MPH; Fellow, Division of Internal Medicine
Research: Implementation of clinical practice guidelines
1995 Fern Dickman, MPH
Research: Depression and ratings of functional status
1995 Christopher Sciamanna, MD, MPH; Fellow, Division of General Internal Medicine
Research: Smoking cessation counseling in primary care

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1995 Robin Ann Yurk, MD MPH; Fellow, Division of General Internal Medicine
Research: Quality of primary care in Medicaid patients

1996 Michael Weiner, MD MPH; Fellow, Division of General Internal Medicine
Research: Health informatics in geriatric patients

1996 Jeanne McCauley, MD; Fellow, Division of General Internal Medicine
Research: Interventions for domestic violence in primary care

1997 Jean-Paul Cretien; Medical student
Research: Effect of recognition of depression in primary care patients on patient outcomes

1997 Thomas P. Erlinger, MD MPH; Fellow, Division of General Internal Medicine
Research: Inflammatory markers in depression

1997 Naresh Punjabi, MD PhD; Department of Epidemiology
Research: Screening for sleep apnea in general medical patients

1998 Katherine E. Lucas, MHS; Department of Epidemiology
Research: Impact of low blood pressure on fatigue

1998 Joyce Chih-I Cheh, BA; post-doctoral fellow, Department of Epidemiology
Research: Cholesterol level and incident cancer

1998 Christine M. Meyer, MHS; Department of Epidemiology
Research: Incident hypertension associated with depression and anxiety in the Baltimore ECA study: risk and validity

1998 Akira Kobayashi; D.Phil candidate, Health Policy and Management
Research: Factors that affect physicians self-esteem: a longitudinal study

1998 Gail L. Daumit, MD MHS; Fellow, Medicine
Research: Integration of psychiatric and medical care for seriously mentally ill patients

1998 Thomas K. Houston, MD MPH; Fellow, Medicine
Research: Internet and patient education

1998 Mary Catherine Beach, MD MPH; Fellow, Medicine
Research: Physician Self-Disclosure in Patient-Physician Communication.

1999 Dario Torre, MD MPH; Fellow, Medicine
Research: Physician Education via the Internet

1999 Jane Koziol-McClain, RN PhD; Post-doc, Nursing
Research: Reducing Interpersonal Violence in Emergency Room Patients

1999 Leigh Ebony Boulware, MD MPH; Fellow, General Internal Medicine
Research: Patient assessment of quality of hypertension care

1999 Jonathan Darer, MD MPH; Fellow, General Internal Medicine
Research: Quality improvement in managing chronic diseases

2000 Benjamin Van Voorhees, MD MPH; Fellow, Department of Medicine
Research: Managed Care and Quality of Mental Health Care

2000 Mahdavi Reddy Patt, MD MPH; Fellow, Division of General Internal Medicine
Research: Physician-Patient E-mail Communication

2000 Gabrielle Bruegelman, MHS; Doctoral candidate, Department of Epidemiology
Research: Functional Status in Sleep Disordered Breathing

2000 Celia Wills, PhD; Michigan State University, K08 Mentor
Research: Patient decision-making about antidepressant use

2001 Yngvild Olsen, MD; Fellow, Division of General Internal Medicine
Research: Patterns of Use of Oxycontin

2001 Leonardo Tamariz, MD; Fellow, Division of General Internal Medicine
Research: Marijuana Use and Inflammatory Markers

2002 Jose Arbalaez, MD PhD; Doctoral candidate, Department of Epidemiology
Research: Depression, Stroke and Inflammation

2003 Vijay Singh, MD; Fellow, Masters of Public Health Candidate, Health Policy and Management
Research: Reporting and Concepts of Domestic Violence by Male Primary Care Patients

2003 Joshua Fogel; Post-doc Fellow, Department of Mental Health
Research: Minor depression

2004 Constantinos Tsilidis; Doctoral student, Department of Epidemiology
Research: Depression and myocardial infarction

2004 Jochen Schmitt, MD; MPH student
Research: Assessing quality of life in psoriasis patients via the Internet

2004 Hillary Bogner, MD, MHS; Assistant Professor Family Medicine, University of Pennsylvania
Research: Depression in patients with cardiovascular disease

2007 Mollie Davis, MD, MPH; Postdoctoral Fellow, General Internal Medicine Fellow

- Daniel E. Ford, M.D., M.P.H.
 Research: Preventive Care in Childhood Cancer Survivors
- 2007 Monique Tello, MD MPH: Postdoctoral Fellow, General Internal Medicine
 Research: Healthy Sexual Habits of Women
- 2007 Madhav Goyal, MD MPH: Postdoctoral Fellow, General Internal Medicine
 Research: Vipasanna Meditation and Migraine Headaches
- 2008 Henry Michtalik, MD MPH: Postdoctoral Fellow, General Internal Medicine
 Research: Delirium in Hospitalized Patient

Mentoring -- Thesis Committees

- 1995 Gregory Kirk (Role: Committee Member, Masters of Public Health, Epidemiology)
 Hepatitis B and C as Risk Factor for Hepatocellular Carcinoma
- 1996 Yi-Hsin (Sidney) Chen (Role: Committee Member, Master of Public Health, Epidemiology)
 Retinal Ischemia as Risk Factor for CVD
- 1996 Laurie Pratt (Role: Committee Member, Doctor of Philosophy, Epidemiology)
 Depression as Risk Factor for Myocardial Infarction
- 1998 Andrea Kopstein, MPH (Role: Committee Member, Doctor of Philosophy, Epidemiology)
 Motivational and Personality Factors Associated With Adolescent Alcohol, Tobacco and
 Marijuana Use
- 1998 Katherine E. Lucas (Role: Committee Member, Master of Health Sciences, Epidemiology)
 The Association Between Low Systolic Blood Pressure and Fatigue
- 1999 Joyce Chen (Role: Committee Member: Masters of Health Science, Epidemiology)
 Serum Cholesterol and Cancer Incidence in the Precursors Study Cohort
- 1999 Carolyn D.M. Furr-Holden (Role: Committee Member, Doctor of Philosophy, Epidemiology)
 The Epidemiology of Drug Dependence: A U.S.-U.K. Cross National Study
- 1999 Akira Kobayashi (Role: Committee Chairman, Doctor of Philosophy, Health Policy and
 Management) Precursors of Self-Esteem and Distress among Middle-Aged Male Physicians:
 A Longitudinal Study
- 1999 Noah Lechzin (Role: Committee Member, Masters of Health Sciences, Epidemiology)
 A Critical Review of Studies Characterizing Respiratory Muscle Involvement and Outcomes of
 Pulmonary Intervention in Patients with Amyotrophic Lateral Sclerosis
- 1999 Sandra Hochman Lesikar (Role: Committee Member, Doctor of Philosophy, Mental Hygiene)
 Health, Cognition, and Driving Behavior
- 2000 J. Gabrielle Breugelman, MS MPH (Role: Committee Member, Doctor of Public Health,
 Epidemiology) The Impact of Sleep Disordered Breathing on Quality of Life in Patients and their
 Bedroom Partners
- 2000 Sonya Singh (Role: Committee Member, Masters of Health Science, Epidemiology)
 Psychotropic Drug Use and the Risk of Fractures
- 2000 Gregory Stevens (Committee Member, Masters of Health Science, Health Policy &
 Management)
 Family Racial and Insurance Disparities in Primary Care Quality for Children
- 2001 Marsha F. Rosenberg (Role: Committee Chairman, Doctor of Philosophy, Mental Hygiene)
 Pharmacothanatology: An Epidemiological Investigation of Drug-Related Deaths
- 2001 Corey B. Smith (Role: Committee Member, Doctor of Philosophy, Mental Hygiene)
 Religiosity and Psychosocial Correlates of Psychopathology in a Community Sample of
 Bereaved Persons
- 2002 Chiadikaobi Uchendu Onyike (Role: Committee Member, Masters of Health Science,
 Epidemiology) Is Obesity Associated with Major Depression? Results from the Third National
 Health and Nutrition Examination Survey (HANES III)
- 2002 Lillian Ingster (Role: Committee Member, Doctor of Philosophy, Health Policy and
 Management) Long Term Trends in Hospitalizations for Ambulatory Care Sensitive Conditions
- 2003 Jose J. Arbelaez (Role: Committee Chairman, Doctor of Philosophy, Epidemiology) Change in
 Depressive Symptoms and Risk of Stroke: Inflammatory Markers as Potential Mediators
- 2003 Darryl R. Brown (Role: Committee Chairman, Doctor of Philosophy, Health Policy and
 Management) The Influence of Patient Satisfaction on Outcomes in After-Hours, Telephone-
 Based Primary Care
- 2003 Vaishali Patel (Role: Committee Member, Doctor of Philosophy, Health Policy and
 Management) Case Study Approach to Understanding How Outcomes Management Systems
 are Used Within Child and Adolescent Mental Health Treatment Settings

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- 2003 Efrat Shadmi (Role: Committee Member, Doctor of Philosophy, Health Policy and Management) Coordination of Primary-Specialty Care Interactions and its Impact on Referral Results
- 2004 Leigh Ann White (Role: Committee Chairman, Doctor of Philosophy, Health Policy and Management) Effects of Psychological Distress on Employment among Low-Income Women
- 2004 Erick Messias (Role: Committee Member, Doctoral, Department of Mental Hygiene) Treatment Needs and Services Utilization in Prevalent Mental Disorders
- 2004 Anne B. Woods (Role: Committee Member, Doctoral, Department of Nursing) Bio-Psycho-Immunologic Responses to Battering
- 2004 Ya-Pei Liu (Role: Committee Member, Doctoral) Predictors of Physical Decline and the Use of Compensatory Strategies in An Older Population

Mentoring -- Training Grant Participation

Daniel E. Ford, Core Faculty 07/01/00-06/30/04 T32MH20014 \$1,087,568
National Institute of Mental Health. Interdisciplinary Research Training on Violence.
(Jacqueline Campbell, PI)

Daniel E. Ford, Core Faculty 06/01/00-05/31/05 HL07024 \$2,421,444
National Heart, Lung and Blood Institute. Cardiovascular Epidemiology Institutional Training Program.
(Joseph Coresh, P.I.)

Editorial Activities

1999-2004 Associate Editor, Journal of General Internal Medicine
2005- General Hospital Psychiatry

Reviewer	American Journal of Epidemiology	Epidemiological Reviews
	American Journal of Geriatric Psychiatry	Journal of Clinical Psychiatry
	American Journal of Medicine	Journal of Psychosomatic Medicine
	American Journal of Preventive Medicine	Journal of the American College of Cardiology
	Annals of Internal Medicine	Journal of the American Medical Association
	Archives of Family Practice	Journal of General Internal Medicine
	Archives of General Psychiatry	Hypertension
	Epidemiology	Medical Care
	General Hospital Psychiatry	Medicine

CLINICAL ACTIVITIES

Certification

1982 Diplomate, American Board of Medical Examiners
1986 Diplomate, American Board of Internal Medicine
1986 State Medical License: Maryland

Service Responsibilities

Johns Hopkins Internal Medicine (Primary Care Physician, Hypertension), Attending Physician (10%)
University Health Service (Primary Care Physician), Director (10%) and Attending Physician (10%)

ORGANIZATIONAL ACTIVITIES

Organizational Activities -- Institutional Administrative Appointments

1993-2006 Director, University Health Service, Johns Hopkins University School of Medicine and School of Hygiene and Public Health

Organizational Activities -- Professional Society Memberships

1985- Society for General Internal Medicine
1985- American Public Health Association
1985- Johns Hopkins Medical and Surgical Association
1989- American College of Physicians/American Society of Internal Medicine
1995- Association for Health Services Research
2005- Clinical Research Forum
2008- Society for Clinical and Translational Science
2008- PRIM&R – Public Responsibility in Medicine & Research
2009- Association for Clinical Research Training
2009- Clinical Trials Transformation Initiative

Organizational Activities -- Conference Organizer

1999-2002 Clinical Preventive Medicine Course (2 days with CME credits)

Organizational Activities -- Advisory Committees

1992-93 Member, Abstract Selection Committee, Society for General Internal Medicine National Meeting
1998-99 Co-Chairman, Prevention Subcommittee on Abstract Selection, Society for General Internal Medicine National Committee
2002 NIMH Advisory Board to STAR*D Clinical Trial
2003 External Advisory Board, University of Pittsburgh Late Life Depression Center
2004 Advisory Board, University of Pennsylvania Advanced Center for Intervention Services Research
2009 External Advisory Board, Michigan Institute for Clinical and Health Research
2009 External Advisory Board – Cleveland CTSA
2009 CCTS External Advisory Board, University of Alabama
2011 CTSA External Advisory Board, University of Chicago

Organizational Activities -- National and State Committees

1992-99 Society of General Internal Medicine Representative to National Coordinating Committee on Clinical Preventive Services, Office Disease Prevention and Health Promotion, Assistant Secretary of Health and Human Services Office
1993-95 Maryland Cardiovascular Disease Prevention and Control Plan, Science Committee, State of Maryland, Department of Health and Mental Hygiene
1994- Foundation for Spirituality and Medicine, Board of Directors and Head of the Research Committee
1996-99 NIMH IRG Mental Health Services
1998-2004 Maryland State Advisory Council on High Blood Pressure and Related Cardiovascular Risk Factors, Office of the Governor, State of Maryland
1999-2002 NIMH IRG Interventions and Clinical Trials
2001 NIMH Committee to Develop Research Agenda for Mood Disorders
2002 Data and Safety Monitoring Board, PROSPECT study
2003 Chair, Data and Safety Monitoring Board, Treatment of Traumatic Grief
2004 Chair, Ad-Hoc Review Panel, NIMH
2007- Co-Chair, IT Roundtable, Clinical Research Forum
2008 Chair, Data and Safety Monitoring Board, TEAMcare Study (Wayne Katan, PI)
2008 Co-Director, CTSA Strategic Goal – Improving Clinical Research Management
2008- Faculty, Learning Sessions, National College Depression Partnership

Organizational Activities -- Johns Hopkins Committees

Daniel E. Ford, M.D., M.P.H.

- 1988- Johns Hopkins University School of Hygiene and Public Health: Standing Committee on Residencies
- 1989- General Clinical Research Center, Outpatient Center Research Protocol Review Committee
- 1989- General Clinical Research Center Advisory Committee
- 1992-99 Internship Selection Committee, Johns Hopkins Department of Medicine
- 1993- Johns Hopkins University Student Health Steering Committee
- 1996-99, 03 Medical School Admissions Committee, Johns Hopkins University School of Medicine
- 1998 Liaison Committee on Medical Education, Johns Hopkins University School of Medicine
- 1998- President's Council on Urban Health, Johns Hopkins University
- 2004 - Chair, Student Assistance Program Advisory Committee
- 2004 - Search Committee, Director of Cancer Prevention and Control
- 2003- Committee on Faculty Development and Gender
- 2004 - Chair, Johns Hopkins Health Information Management Group
- 2006 - Chair, Task Force on Processing and Storage of Biospecimens
- 2007 - Dean's Representative to Oncology Center Director Search Committee
- 2009 Search Committee, Welch Center Director
- 2009 Search Committee, Faculty Position in Health Informatics

Organizational Activities -- Consultantships

- 1998-2003 Medical Advisory Board, Merck-Medco Pharmaceutical Benefit Management Company
- 2001- 02 Primary Care Depression and Anxiety Advisory Board, Pfizer, Inc.
- 2001-02 Johns Hopkins Health Care Consulting. Epidemiology of Anxiety and Depressive Disorders. Bristol Myers Squibb.
- 2002-04 Health Resources and Services Administration, Bureau of Primary Health Care, Health Disparities Collaborative, Changing Practice, Changing Lives. Institute for Healthcare Improvement
- 2002-03 CMS Depression Screening in Cardiac Rehabilitation Settings
- 2005- Medical Advisory Board – Medco-Accredo Company
- 2010 RAND Corporation Technical Advisory Group (TAG)

RECOGNITION

Recognition -- Awards

State University of New York at Buffalo

- 1981 Student Award in Obstetrics-Gynecology
- 1982 Alpha Omega Alpha Honor Medical Society
- 1982 David K. Miller Prize in Medicine
- 1998 David Levine General Internal Medicine Fellows Appreciation Award
- 2006 Delta Omega Delta Honor Public Health Society

Recognition -- Invited Reviews

- 1987 National Institute of Alcohol Abuse Contract, "Development of Comprehensive Medical School Curriculum on Alcohol and Substance Abuse"
- 1988 National Institute of Alcohol Abuse Contract, "Development of Primary Care Medical School Curriculum on Alcohol and Substance Abuse"
- 1992 AHCPH Small Grants Program
- 1993 National Institute of Mental Health Ad Hoc Reviewer
- 1996-Bayer Institute for Health Care Communication Research Grants Review Committee

Recognition - Invited Talks, Panels

1. Which Patients Discuss Mental Health Problems With Non-Psychiatrists. University of Pittsburgh School of Medicine, Department of Psychiatry, Pittsburgh, PA (1987)
2. Implementation of Prevention Guidelines in Practice. Johns Hopkins Department of Medicine Annual Conference, Baltimore, MD (1989)
3. Screening for Alcohol or Psychiatric Conditions. Johns Hopkins Conference: Implementation of U.S. Preventive Services Task Force Guidelines, Baltimore, MD (1989)

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4. Treatment Adherence and Its Effects on Health Outcomes. Clinical Epidemiology Seminar. Wilmer Institute, Johns Hopkins Medical Institutions. Baltimore, MD (1990)
5. Prevention in the Office. Uniformed Health Services Medical School, General Internal Medicine Rounds, Bethesda, MD (1990)
6. Improving Prevention in the Office. Johns Hopkins Annual Obstetric-Gynecology Conference, Baltimore, MD (1991)
7. Assessment of Sleep Disturbances. Society of General Internal Medicine Annual Meeting Precourse, Seattle, WA (1991)
8. The Academic Medical Center Working with Communities. American Association of Medical Colleges Annual Meeting, Washington DC (1991)
9. Preventive Services and Physician Satisfaction. Johns Hopkins University Department of Medicine Research in Progress, Baltimore, MD (August 6, 1992)
10. Characteristics of Patients with Major Depression Who Received Care in General Medical and Specialty Mental Health Settings. Johns Hopkins University Department of Medicine Annual Retreat, Baltimore, MD (October 23-24, 1992)
11. Annual Health Exam. Johns Hopkins University School of Medicine, Grand Rounds, Baltimore, MD (November 14, 1992)
12. Models for Working with Communities. Prevention 92 Annual Meeting, Baltimore, MD (1992)
13. Assessment of Sleep Disturbances. American College of Physician National Conference, San Diego, CA (1992)
14. Collection of Prevention-Oriented Patient Data in Practice. Society of General Internal Medicine Annual Meeting, Washington, DC (1992)
15. Sleep Disturbances in General Medical Patients. Agency for Health Care Policy and Research. Third Primary Care Research Conference. Challenges in Practice-Based Research. U.S. Department of Health and Human Services, Bethesda, MD (January 10-12, 1993)
16. What factors influence job satisfaction among physicians? Health behaviors in physicians: less favorable in Blacks. American Medical Association, Physicians Health Foundation International Conference on Physician Health, Phoenix AZ (January 29-31, 1993)
17. Using Information Systems for Prevention Programs. Group Health Association of America, Prevention Programs: The Next Generation of Responsibilities and Initiatives in Managed Care, Colorado Springs CO (March 28-31, 1993)
18. Evaluation and Management of a Patient with a Sleep Disturbance. American College of Physicians, 74th Annual Session. Washington, DC (April 1, 1993)
19. What Prevention Topics Do Primary Care Patients Want to Talk About? Johns Hopkins University Department of Medicine Research in Progress, Baltimore, MD (August 5, 1993)
20. Chronic Diseases. American College of Preventive Medicine Review Course, Chicago, IL (August 29-31, 1993)
21. The Influence of Gender, Race and Education on Patient Preferences and Receipt of Invasive Cardiac Procedures Among Coronary Care Unit Patients. Johns Hopkins University Department of Medicine Annual Retreat, Baltimore, MD (October 22-23, 1993)
22. Screening for Colon Cancer. Johns Hopkins Medical Institutions. Topics in Ambulatory Medicine VI, Baltimore, MD (November 4, 1993)
23. How to Read and Write Clinical Literature. Risk of Cigarette Smoking and Smoking Cessation. Advance in Internal Medicine Evaluation and Prevention, Taiwan, R.O.C. (April 1994)
24. Expert testimony in the environmental on the epidemiologic risks for environmental tobacco smoke. Maryland Occupational Safety Health Hearing, Baltimore MD (May 1994)
25. Expert testimony on the epidemiologic risks for environmental tobacco smoke. Occupational Safety Health Administration, Washington DC (September 1994)
26. Practice Guidelines: The New Reality in Medicine. Maryland Association of Cardiovascular and Pulmonary Rehabilitation, Annapolis MD (November 4, 1994)
27. Can Outcomes Research Change the Way Doctors Practice? Johns Hopkins University School of Medicine, Baltimore, MD (November 10, 1994)
28. Diagnostic Tests. Critical Appraisal of Published Clinical Research, Baltimore, MD (March 1995)
29. Colon Cancer Screening. Johns Hopkins Medical Institutions, Grand Rounds, Baltimore, MD (April 29, 1995)
30. Putting Prevention into Practice. Harbor Hospital Medical Center, Grand Rounds, Baltimore, MD (June 23, 1995)
31. Controversies in the Treatment of Arterial Hypertension. Symposium on Cardiovascular Epidemiology and Treatment of Hypertension, Bucaramanga, Colombia (July 25-29, 1995)
32. Disease Prevention in the 1990's. American College of Physicians, Maryland Chapter,

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- Baltimore, MD (October 1995)
33. Primary Care: The ABC's of Screening for Malignancy. American Cancer Society. Eastern Shore Oncology Conference, Salisbury, MD (November 3, 1995)
 34. U.S. Preventive Health Services Task Force: Recent Update. Johns Hopkins Medical Institutions, Topics in Ambulatory Medicine VII, Baltimore, MD (November 15-17, 1995)
 35. Improving Outcomes Through Patient Participation. National Patient Empowerment Council. Washington, DC (December 8, 1995)
 36. Prevention in Medical Practice. Baltimore City Medical Society, Baltimore, MD (April 11, 1996)
 37. Health Communication in Managed Care Settings. American Academy of Physician and Patient Annual Meeting, (April 1996)
 38. Update on the U.S. Preventive Services Task Force. Johns Hopkins University School of Medicine, Topics in Internal Medicine, Baltimore, MD (May 13-17, 1996)
 39. Using Patient Outcomes to Improve the Quality of Health Care. National Institutes of Mental Health, Bethesda, MD (February 21, 1997)
 40. Provider-Patient Communication, Patient Satisfaction and Health Outcomes: Are They Linked? Case Management Society of America, Boston, MA (May 31, 1997)
 41. Religion and Medicine Course Survey. American Association of Medical Colleges, Washington, DC (November 4, 1997)
 42. Depression and Cardiovascular Disease. Johns Hopkins Bayview Medical Center, Geriatric Grand Rounds, Baltimore, MD (January 13, 1998)
 43. Recent Developments in Preventive Medicine. Harbor Hospital Medical Center, Baltimore, MD (January 30, 1998)
 44. Primary Care: Potential for Preventing Comorbidity. National Institute of Mental Health Workshop: Research Issues in the Prevention of Comorbidity, Bethesda, MD (June 22-23, 1998)
 45. The Treatment of Depression in Primary Care: What Have We Learned. Behavioral Pharmacology Research Unit at Bayview Medical Center, Baltimore MD. (February 10, 1999)
 46. Depression and Cardiovascular Disease. Georgetown University School of Medicine. Department of Psychiatry Grand Rounds (March 25, 1999)
 47. Treatment of Depression in Primary Care. Johns Hopkins Conjoint Clinic. (April 24, 1999)
 48. What About Me and You? Patient and Physician Preferences in Ambulatory Care. Coen Lectureship, Baystate Medical Center, Springfield MA (May 19, 1999)
 49. "Hot Topics" in Advanced Practice Nursing: Update on Hypertension. Institute for Johns Hopkins Nursing, Baltimore MD (September 16, 1999)
 50. Update on Preventive Medicine. Grand Rounds at Harbor Hospital Center, Baltimore MD. (September 17, 1999)
 51. Primary Care and Depression, Behavioral Pharmacology Research Unit at Bayview Medical Center, Baltimore MD (February 10, 2000)
 52. Generalism in the New Millennium: Exploring Career Opportunities in Internal Medicine (Panel Discussion), Medical Student Workshop. Mid-Atlantic Regional Meeting of the Society of General Internal Medicine. Baltimore MD (March 10, 2000)
 53. Depression in the Workplace. Aetna Academic Medicine and Managed Care, Washington DC (June 7-8, 2000)
 54. Clinical Guidelines for Prevention in Primary Care. American Academy of Family Practice, Ocean City MD (June 9, 2000)
 55. Challenges for the 21st Century: Mental Health Services Research. Depression in Primary Care: Diagnostic Instruments for Depression; Patient and Provider Factors Associated with Use of Sedative-Hypnotic Medications for Patients with Major Depression; Suicide, Depression and Panic Disorder in Primary Care (Paper Discussant); Washington DC (July 18-20, 2000)
 56. Benzodiazepines and Treatment of Depression, University of Pennsylvania Geriatric Psychiatry Rounds, Philadelphia PA (January 17, 2001)
 57. Depression and Coronary Artery Disease, Walter Reed Medical Center Medicine Grand Rounds (February 9, 2001)
 58. Integrating Depression into Cardiovascular and Diabetes Disease Management, Institute for Health Care Improvement, Bureau of Primary Care, HRSA Conference, Dallas TX (April 21, 2001)
 59. Improving Care for Depression through the Internet, National Institute of Mental Health, Grand Rounds, Bethesda, MD (May 18, 2001).
 60. Marijuana Use Is Not Associated With Head, Neck, or Lung Cancer in Adults Younger Than 55 Years: Results of a Case Cohort Study, National Institute of Drug Abuse, Washington DC (August 13-14, 2001)

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61. Surgeon General's Initiative "Bridging Mental Health and Primary Care: Crossing the Quality Chasm" Targeting Research, Practice and Financing Activities for Depression, Children and Adolescents and Serious Mental Illness. Discussion Group Participation. (August 21, 2001)
62. Clinical Practice Guidelines: Treating Tobacco Use and Dependence. Howard County General Hospital (CME Medical Staff Program) (December 5, 2001)
63. Depression as a Risk Factor for Cardiovascular Disease. Johns Hopkins Department of Medicine Grand Rounds, Baltimore MD. (March 22, 2002)
64. Smoking Cessation. Johns Hopkins Saturday Medicine, Baltimore MD. (March 23, 2002)
65. Depression and Risk for Cardiovascular Disease, Preventive Medicine Presentation, Welch Center Grand Rounds, Johns Hopkins Medical Institutions, Baltimore MD (March 27, 2002)
66. Evaluation Plan for Depression in Primary Care: Linking Clinical and System Strategies. Robert Wood Johnson Foundation Meeting, Pittsburgh PA (April 30 – May 2, 2002)
67. Antidepressant Selection Process: New Clinical Data Relevant to Medical Comorbidity. Mental Health in the Primary Care Setting: The Nurse Practitioner's Role in Diagnosing and Treating Mood and Anxiety Disorders. 17th Annual Conference Symposium. American Academy of Nurse Practitioners, Reno NV (June 19-23, 2002)
68. Epidemiology of Suicide in Physicians. Physician Suicide Workshop. American Foundation for Suicide Prevention, Philadelphia PA (October 5-7, 2002)
69. Depression and Risk for Cardiovascular Disease. NeuroScience, Inc., New York NY (December 13, 2002).
70. Depression and Cardiovascular Disease. Psychiatry Grand Rounds, Johns Hopkins University, Baltimore MD (December 23, 2002)
71. Integrating Behavioral Health and Medical Care. Improving Outcomes for Patients with Depression. Academy Health Meeting (June 26-27, 2003).
72. Depression and Risk for Cardiovascular Disease, Baltimore Medical Systems, Inc., Baltimore MD (July 31, 2003).
73. A Population-Based Approach to Treatment of Patients with Depression. Lecture to Pediatric Mental Health Trainees, Johns Hopkins, Baltimore MD (August 4, 2003).
74. Depression and Cardiovascular Disease, University of Pennsylvania, Department of Psychiatry, Philadelphia PA (September 22, 2003)
75. Modifiable Risk Factors: Marijuana and Head and Neck Cancers. Behavioral Science and Cancer: Relevance, Risk and Resilience. Howard University Cancer Center/Johns Hopkins Kimmel Cancer Center Partnership Symposium. Radisson Plaza, Baltimore, MD (November 19, 2003)
76. Depression and Coronary Artery Disease, University of Alabama Department of Medicine Grand Rounds (April, 2004) Birmingham, Alabama
77. Depression and Cardiovascular Disease: What is the link? Complementary and Alternative Medicine Rounds, Johns Hopkins Baltimore (Jan 4, 2005)
78. Depression and Coronary Artery Disease: What is the link? Department of Medicine Grand Rounds, MetroHealth Hospital, Cleveland (Jan 18, 2005)
79. Physician Health: What are the Risks? Department of Medicine Grand Rounds, St. Raphael's Hospital, New Haven, Connecticut (Feb 8, 2005)
80. Physician Health: What are the Risks? Department of Surgery Grand Rounds, St. Raphael's Hospital, New Haven, Connecticut (Feb 9, 2005)
81. Interrelationships between Depression, Schizophrenia and Cardiovascular Disease Department of Medicine Research Symposium Johns Hopkins, Baltimore MD (April 7, 2005)
82. Update on CTSA Activities: Workshop on Clinical Research Management, National Advisory Research Resource Council, Bethesda, MD (September 16, 2008)
83. Transforming Clinical and Translational Research at Johns Hopkins, International Symposium on Clinical Research and Translational Medicine, Fudan University, Shanghai, (September 24-29, 2008)
84. Community Engagement Green Group Regional Workshop, University of Pennsylvania (October 13, 2008)
85. National College Depression Partnership, Learning Sessions, Faculty Member (2008-)
86. Opportunities for IT in Clinical Research Support, American Medical Informatics Association Annual Symposium, Washington, DC (November 10, 2008)
87. Women in Clinical Trials, A Woman's Journey, Baltimore, MD (November 14, 2008)
88. Overview of Work Being Done in the Institute for Clinical and Translational Research, Blaustein Pain Grand Rounds, Baltimore, MD (December 2, 2008)
89. Resource Update – CTSA Implementation – Bayview Research Symposium, Baltimore, MD (December 23, 2008)

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90. Keeping Pace with the Economic & Political Environment: A Challenge to Clinical Research, Clinical Research Forum, Washington, DC (January 13-14, 2009)
91. Interdisciplinary Research, Panel Discussion, Southern Nursing Research Society, Baltimore, MD (February 12, 2009)
92. Opportunities: The NIH CTSA at Johns Hopkins – a Role for HPM? Health Policy and Management Retreat, Bethesda, MD (February 20, 2009)
93. New Opportunities for Clinical Research in Difficult Times, Clinical Research Forum IT Roundtable, Washington, DC (April 15-17, 2009)
94. Next Steps – Plans for Larger Future Study, CTSA Clinical Research Management Workshop, Bethesda, MD (June 22-23, 2009)
95. Strengthening Partnerships Between HRA Member Organizations and Academic Health Centers, Health Research Alliance Members' Meeting, Chevy Chase, MD (September 23-24, 2009)
96. Clinical Research Challenges and Opportunities, National eHealth Collaborative. Arlington, VA, (October 21, 2009)
97. Closed-Door Roundtable on *Comparative Effectiveness Research and Health Care Innovations* sponsored by the National Institute for Health Care Reform, the Center for Studying Health System Change (HSC), AcademyHealth and the Association of American Medical Colleges (AAMC), Washington, DC (February 1, 2010).
98. Rand Corp. Technical Advisory Group Meeting, Developing an Evaluation Design for the Primary and Behavioral Health Care Integration Grant Program, Washington, DC, (February 24, 2010)
99. Regulatory Affairs Professionals Society (RAPS), 2010 Horizons Conference opening keynote speaker. Baltimore, Maryland (March 25, 2010).
100. The Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins Leadership Retreat. "Enterprise-wide IT solutions to support translational research". Baltimore, MD (May 24, 2010).
101. Henrietta Lacks Memorial Symposium, Baltimore, MD. (October 2, 2010).

Publications – Selected Abstracts

1. **Ford DE** and Anthony JC: Performance of the general health questionnaire among different health care utilizers. Clin Res 35:740A, 1987.
2. **Ford DE** and Kamerow DB: Sleep disturbances: Longitudinal course and relationship to psychiatric disorders. Clin Res 36:711A, 1988.
3. Liss A, **Ford DE**, Wilder LB, Sigmund WR and Becker DM. Drug treatment for hyperlipidemia: Knowledge among medical interns and residents in ambulatory practice settings. American Heart Association, November, 1990.
4. Johnson K, **Ford DE**, Smith G: Prevention of residential fire deaths in a general medicine clinic. Society of General Internal Medicine, Annual Meeting, May, 1990.
5. **Ford DE**, Klag MJ, Mead LA, Appel LJ, Levine DM. What factors influence job satisfaction among physicians? Society of General Internal Medicine, Annual Meeting, May, 1990.
6. **Ford DE**, Klag MJ, Whelton PK: Physician's knowledge of the CAGE and its relationship to medical practice. Society of General Internal Medicine, Annual Meeting, May, 1990.
7. Ellerbeck EF, **Ford DE**, Becker DM, Liss AS, Sigmund WR. Improving cholesterol management by residents in an ambulatory care clinic. Society of General Internal Medicine, Annual Meeting, May, 1991. Clin Res 39, 616A, 1991.
8. Hayward RSA, **Ford DE**, Steinberg EP and Roizen MF. Prevention practice information tools for the clinician. Canadian Organization for Advancement of Computers in Health, 1991.
9. **Ford DE**, McCauley JM, Jones CA. Factors associated with obese patients beginning a weight reduction program. Clin Res 29, 635A, 1991.
10. Brancati FL, **Ford DE**, Klag MF, Appel LJ, Whelton PK. Patient and physician factors related to intensity of weight reduction care in a university medical clinic. American Heart Association National Meeting, November, 1991.
11. Crum R and **Ford DE**. Factors related to recognition of alcohol abusers in a primary care clinic. Fifth Annual National Institute of Mental Health International Research Conference, Washington, DC, September, 1991.
12. Girman-Ratan AM, Wilcox PM, Helzlsouer KJ and **Ford DE**. Evaluation of a breast examination educational unit for medical residents. American Academy of Cancer Education, Baltimore, MD, June, 1991.
13. Hayward RSA, Smittner JP, Meyers P, **Ford DE**, Roizen M, Steinberg E. Computer versus Interview Administered Preventive Care Questionnaire: Does Survey Medium Affect Patient

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- Response Reliability? Society of General Internal Medicine, Annual Meeting, May, 1992. Clin Res 40(2):608A, 1992.
14. Hayward RSA, **Ford DE**, Summerell D, Roizen MF, Steinberg EP. Randomized clinical trial of a patient-administered computerized preventive care system to implement adult practice guidelines. Society of General Internal Medicine, Annual Meeting, May, 1992. Clin Res 40(2):608A, 1992.
 15. Marino S, Gallo JJ, **Ford D**, Anthony JC. The pattern of health services use for individuals with incident mental disorder. 6th Annual NIMH International Research Conference on Primary Care Mental Health Research: Concepts, Methods, and Obstacles. October 18-20, 1992.
 16. **Ford D**, McCauley J, Kern D. Functional status of primary care patients with sleep disturbances: agreement between patient and significant others. 6th Annual NIMH International Research Conference on Primary Care Mental Health Research: Concepts, Methods, and Obstacles. October 18-20, 1992.
 17. Patrick LC, Crum R, **Ford D**. Characteristics of patients with major depression who received care in general medical and specialty mental health settings. 6th Annual NIMH International Research Conference on Primary Care Mental Health Research: Concepts, Methods, and Obstacles. October 18-20, 1992.
 18. Crum RM, Patrick LC, **Ford D**. Depressive symptoms in general medical patients: prevalence and one year outcome in the Epidemiologic Catchment Area Study. 6th Annual NIMH International Research Conference on Primary Care Mental Health Research: Concepts, Methods, and Obstacles. October 18-20, 1992.
 19. **Ford DE**, McCauley J, Kern D. Sleep disturbances in general medical patients. Agency for Health Care Policy and Research Third Primary Care Research Conference, Atlanta GA. January 10-12, 1993.
 20. Klag MJ, Mead LA, Thomas John, Thomas Johniene, **Ford DE**, Levine DM, Visco R, Pearson TA. Health behaviors in physicians: less favorable in Blacks. International Conference on Physician Health, Scottsdale AZ. January 29-31, 1993.
 21. **Ford DE**, Klag MJ, Mead LA, Appel LJ, Levine DM. What factors influence job satisfaction among physicians? International Conference on Physician Health, Scottsdale AZ. January 29-31, 1993.
 22. **Ford DE**, Hayward RSA, Ellis P, Roizen M, Steinberg E. Which prevention topics do patients want to talk about? Society of General Internal Medicine, Washington DC. March 5, 1993.
 23. Patrick LC and **Ford DE**. Identifying suicidal ideation in primary care patients. Society of General Internal Medicine, Washington DC. March 5, 1993.
 24. Patrick LC, **Ford DE**, Klag MJ, Mead L, Levine DM. Exercise and Mental Well-Being in Physicians. 33rd Annual Conference on Cardiovascular Disease Epidemiology, Sante Fe NM. March 17, 1993.
 25. Schecter AD, McKee G, Hoffeld D, Velez R, Myers M, Drayer T, Szych C, Chandra NG, Goldschmidt-Clermont PJ, **Ford DE**. Education level more than gender or race predicts patients' attitudes toward management in the coronary care unit. American College of Cardiology, 43rd Annual Scientific Session, March 16, 1994.
 26. Chang PP, **Ford DE**, Mead LA, Graves PL, Klag MJ. Predictors in male medical students for subsequent psychiatric distress and clinical depression. 17th Annual Meeting, Society of General Internal Medicine, April 27-29, 1994, Washington DC. J Gen Intern Med 1999;9(suppl 2):26.
 27. Chang PP, **Ford DE**, Mead LA, Klag MJ. Association of sleep patterns in young men with clinical depression and psychiatric distress: a prospective study. 17th Annual Meeting, Society of General Internal Medicine, April 27-29, 1994, Washington DC. J Gen Intern Med 1999;9(suppl 2):26.
 28. Cooper-Patrick L, **Ford DE**, Mead LA, Klag MJ. Exercise and Psychological Distress: A Prospective Study. 17th Annual Meeting, Society of General Internal Medicine, April 27-29, 1994, Washington DC. J Gen Intern Med 1999;9(suppl 2):28.
 29. **Ford DE**, McCauley J, Kern D. Association of Primary Care Patients and Physicians Ratings Concerning Whether a Mental Health Evaluation was Completed. 8th Annual NIMH International Research Conference on Mental Disorders in the General Health Care Sector, September 7-9, 1994, McLean VA.
 30. Mead LA, Levine DM, **Ford DE**, Brancati FL, Coresh J, Klag MJ. Family history of myocardial infarction as an independent risk factor for coronary heart disease (CHD): results from the Precursor's Study. American Heart Association, 67th Scientific Session. November 14-17, 1994.
 31. **Ford DE**, Mead LA, Chang PP, Levine DM, Klag MJ. Depression predicts cardiovascular disease in men: the Precursor's Study. American Heart Association, 67th Scientific session. November 14-17, 1994.
 32. Chang PP, **Ford DE**, Mead LC, Levine DM, Klag MJ. Anger in young men and risk of cardiovascular disease: results from the Precursors Study. American Heart Association, 67th Scientific Session. November 14-17, 1994.

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33. Schechter A, **Ford DE**, McKee G, Hoffeld D, Schulman S, Myers M, Chandra N, Goldschmidt-Clermont, P. The relationship of race, gender and education to CCU patient knowledge of modifiable cardiac risk factors: implications for patient education. American Heart Association, 67th Scientific Session. November 14-17, 1994.
34. **Ford DE**, Chang PP, Mead LA, Levine DM, Klag MJ. Serum cholesterol in young men and risk of subsequent depression. American Heart Association, 67th Scientific Session. November 14-17, 1994.
35. **Ford DE**, Mead LA, Wang N-Y, Chang PP, Cooper-Patrick L, Klag MJ. Anger in medical school predicts depression over 30 years of follow-up: the Precursors Study. 18th Annual Meeting, Society of General Internal Medicine, San Diego, May, 1995. *J Gen Intern Med* 1995;10(suppl 2):42.
36. Ryan S, Schechter A, Goldschmidt-Clermont P, Myers M, Hoffeld D, **Ford D**. Perceptions about health and preferences for invasive care and health locus of control in elderly cardiac care unit patients. 18th Annual Meeting, Society of General Internal Medicine, San Diego, May, 1995. *J Gen Intern Med* 1995;10(suppl 2):58.
37. Cooper-Patrick L, Crum RM, Pratt L, **Ford DE**. The Psychiatric and Socio-demographic Profile of Patients with Chronic Disease Who Do Not Receive Regular Medical Care. 18th Annual Meeting, Society of General Internal Medicine, San Diego, May, 1995. *J Gen Intern Med* 1995;10(suppl 2):64.
38. Dickman F, McCauley J, Kern D, **Ford DE**. Significant others rate primary care patients with psychiatric distress as having higher functional status than the patients themselves. National Institute of Mental Health Conference on Mental Health Services Research. Bethesda MD, September 11-12, 1995.
39. Klag MJ, Mead LA, Whelton PK, **Ford DE**. Prevalence of calcium channel blocker use among hypertensive physicians in the Precursors Study. American Heart Association. 36th Annual Conference on Cardiovascular Disease Epidemiology and Prevention. San Francisco, CA. March 13-16, 1996.
40. **Ford DE**, Butler J, Mead LA, Rardin K, Carroll JG, Klag MJ. Randomized trial of a physician-patient communication intervention in an HMO. 1996 Mid-Atlantic Regional Meeting, Society of General Internal Medicine, Baltimore, MD March 22, 1996
41. **Ford DE**, Mead LA, Butler J, Carroll JG, Klag MJ. Development of an instrument for patient rating of physicians' communication skills using factor analysis. 19th Annual Meeting, Society of General Internal Medicine, Washington DC, May 2-4, 1996. *J Gen Intern Med* 1996;11(suppl 1):133.
42. Cooper-Patrick L, Powe NR, Jenckes MW, Gonzales JJ, Levine DM, **Ford DE**. Using focus groups to identify patient attitudes and preferences regarding treatment of depression. 13th Annual Meeting of the Association for Health Services Research. Atlanta GA. June 9-11, 1996.
43. Cooper-Patrick L, Powe NR, Jenckes MW, Gonzales JJ, Levine DM, **Ford DE**. Ethnic and cultural experiences influencing patients' help-seeking and preferred treatment for depression. Picker-Commonwealth Meeting. Chicago IL. June 13-15, 1997.
44. Mueller BA, Mead LA, **Ford DE**, Brancati FL, Cooper-Patrick L, Klag MJ. Coffee drinking, cigarette smoking and risk of non-rheumatic atrial fibrillation in the Precursors Study. 4th International Conference on Preventive Cardiology. Montreal Canada. June 29-July 3, 1997.
45. Sciamanna CN, **Ford DE**, Stillman FA, Hoch JS. Validation of a simple method of measuring hospitalized smokers' motivation to quit. 21st Annual Meeting, Society of General Internal Medicine, Chicago IL. April 23-25, 1998. *J Gen Intern Med* 1998;13(suppl 1):107.
46. Sciamanna CN, Stillman FA, Hoch JS, **Ford DE**. Opportunities for improving inpatient smoking cessation program. 21st Annual Meeting, Society of General Internal Medicine, Chicago IL. April 23-25, 1998. *J Gen Intern Med* 1998;13(suppl 1):101.
47. Cooper-Patrick L, Gallo J, Gonzales JJ, Powe NR, Nelson C, **Ford DE**. African-American patients rate their physicians' decision-making styles as less participatory. 21st Annual Meeting, Society of General Internal Medicine, Chicago IL. April 23-25, 1998. *J Gen Intern Med* 1998;13(suppl 1):115.
48. Punjabi NM, O'Hearn DJ, Schwartz AR, Smith PL, Wise RA, **Ford DE**, Bandeen-Roche KL. Determinants of Multiple Sleep Latency Test (MSLT) in Patients with Obstructive Sleep Apnea (OSA). ALA/ATS International Conference, Chicago IL. April 24-29, 1998.
49. Punjabi NM, Bandeen-Roche KJ, O'Hearn DJ, Allen RP, Schwartz AR, Smith PL, Wise RA, **Ford DE**. Prediction of Obstructive Sleep Apnea (OSA) using Symptom Scores from the Hopkins Sleep Survey. ALA/ATS International Conference, Chicago IL. April 24-29, 1998.
50. Cooper-Patrick L, Brown C, Palenchar Dr, Gonzales JJ, **Ford DE**, Powe NR. Patients' Opinions Regarding the Importance of Various Aspects of Depression Treatment. 15th Annual Meeting, Association for Health Services Research. Washington, DC. June 21-23, 1998.

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51. Cooper-Patrick L, Gonzales JJ, Gallo JJ, Rost KM, **Ford DE**. Patient preferences for treatment of depression. 12th International Conference on Mental Health Problems in the General Health Care Sector, Bethesda MD. July 13-14, 1998. *The International Journal of Psychiatry in Medicine* 1998;28(4):422-423.
52. Punjabi NM, Sorkin JD, Katzel LI, Marx JJ, Schwartz AR, Smith PL, **Ford DE**. Insulin Resistance in an Independent Risk Factor for Sleep Apnea. ALA/ATS International Conference, San Diego, CA. April 23-28, 1999.
53. Punjabi NM, Chen CA, Allan LW, O'Hearn DJ, Bandeen-Roche KJ, Schwartz AR, Smith PL, **Ford DE**. Determinants of Quality of Life (QoL) Impairment with Sleep Apnea. ALA/ATS International Conference, San Diego, CA. April 23-28, 1999.
54. Houston T, Cooper-Patrick L, Kahn J, Vu H, **Ford D**. Identification of individuals with untreated depression through the internet. 22nd Annual Meeting, Society of General Internal Medicine, San Francisco CA. April 29-May 1, 1999. *J Gen Intern Med* 1999;14(suppl 2):40.
55. Houston T, Mead LA, **Ford DE**, Brancati F, Cooper-Patrick L, Levine DM, Klag MJ. Tennis, football and risk of cardiovascular disease. 22nd Annual Meeting, Society of General Internal Medicine, San Francisco CA. April 29-May 1, 1999. *J Gen Intern Med* 1999;14(suppl 2):40.
56. **Ford DE**, Vu H, Anthony J. Marijuana use and tobacco smoking persistence in young adults. 22nd Annual Meeting, Society of General Internal Medicine, San Francisco, CA. April 29-May 1, 1999. *J Gen Intern Med* 1999;14(suppl 2):29.
57. Sciamanna C, Flynn J, **Ford D**. Opportunities for Counseling Smokers to Quit. 22nd Annual Meeting, Society of General Internal Medicine, San Francisco, CA. April 29-May 1, 1999. *J Gen Intern Med* 1999;14(suppl 2):70.
58. **Ford DE**, Rost K, Meredith L, Duan N, Rubenstein L, Sherbourne C, Smith J, Vu H, Nutting P, Wells, K. Variation in the Quality of Primary Care Treatment Provided for Major Depression. July 12-13, 1999. 13th International Conference on Mental Health Problems in the General Health Care Sector.
59. Bajwa K, **Ford D**, Gallo J, Meredith L, Rubinstein L, Rost K, Nutting P, Gonzales J. Primary Care Physicians' Readiness to Change in Caring for Depression. July 12-13, 1999. 13th International Conference on Mental Health Problems in the General Health Care Sector.
60. **Ford DE**, Mead LA, Klag MJ. Low Socioeconomic status in childhood predicts coronary heart disease. 40th Conference on Cardiovascular Disease Epidemiology & Prevention, San Diego CA. March 3-5, 2000.
61. **Ford DE**, Vu HT, Hauer C, Helzlsouer KJ, Anthony JC. Marijuana use is not associated with head, neck or lung cancer in adults less than 55 years of age: results of case cohort study. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):66.
62. HoustonTK, **Ford DE**, Cooper-Patrick L. Ethnic differences in preferences for treatment among untreated depressed individuals. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):72.
63. Jenckes MW, Hebert R, O'Connor D, **Ford DE**, Cooper-Patrick L. Patients talk about spirituality and therapeutic relationships with physicians. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):74-75.
64. Beach Mc, Roter D, Frankel R, Levinson W, **Ford DE**. Patient response to office visits in which physician self-disclosure occurs. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):101.
65. Cooper-Patrick L, **Ford DE**, Vu HT, Powe NR, Steinwachs DM, Roter DL. Patient-physician race concordance and communication in primary care. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):106.
66. Daumit GL, Crum NR, **Ford DE**. National patterns of ambulatory medical care in the seriously mentally ill. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):107.
67. Daumit GL, Flynn JA, Powe NR, **Ford DE**. Primary care physicians' ratings of quality indicators in office practice. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):107.
68. Torre D, Wang N-Y, Mead LA, **Ford DE**, Young JH, Houston T, Kouzis A, Klag MJ. Mortality in a prospective study of physicians. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):150.
69. Hebert RS, Jenckes MW, O'Connor DR, **Ford DE**, Cooper-Patrick L. Patient preferences for discussing spirituality with physicians. 23rd Annual Meeting. Society of General Internal Medicine, Boston MA. May 4-6, 2000. *J Gen Intern Med* 2000;15(suppl 1):162-163.

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70. Alberg AJ, Landrigan JA, Vu H, Helzlsouer KJ, **Ford DE**. A case-control study of NAT2, GSTM1, and GSTT1 genotypes in relation to head and neck cancer. Society for Epidemiologic Research, Annual Meeting, Seattle WA. June 15-17, 2000.
71. **Ford DE**, Erlinger TP. Depression is Associated with Higher Levels of C-Reactive Protein in Men in the Third NHANE Survey. 24th Annual Meeting. Society of General Internal Medicine, San Diego CA. May 1-3, 2001. J Gen Intern Med 2001;16(suppl 1):132.
72. Houston, TK, Cooper LA, Vu HT, **Ford DE**, et.al. Screening the Public for Depression through the Internet. National Meeting, Society of General Internal Medicine, Apr 29-May 01, 1999 , San Francisco CA. Psychiatric Services, Mar 2001: 52(3), 362-367.
73. Patt MR, **Ford DE**, Sands DZ, Houston TK. Physician-Patient E-mail Communication: Benefits and Barriers. 24th Annual Meeting. Society of General Internal Medicine, San Diego CA. May 1-3, 2001. J Gen Intern Med 2001;16(suppl 1):231.
74. Torre DM, Wang N-Y, Meoni LA, Klag MJ, **Ford DE**. Is the Mortality Advantage of Physicians Due to Lower Rates of Smoking? 24th Annual Meeting. Society of General Internal Medicine, San Diego CA. May 1-3, 2001. J Gen Intern Med 2001;16(suppl 1):178.
75. Beach MC, Meredith L, Wells KB, **Ford DE**. Patient Loyalty, Societal Responsibility and Physician Career Satisfaction. J Gen Intern Med 2001;16(suppl 1):187
76. Houston T, Sands DZ, **Ford DE**, Patt M, Nash B. Experiences of Physicians Who Frequently Use E-Mail with Patients. J Gen Intern Med 2001;16(suppl 1):200.
77. VanVoorhees BW, Wang N-Y, **Ford DE**. Managed Care and Primary Care Physicians' Perception of Patient Access to High Quality Mental Health Services. 24th Annual Meeting. Society of General Internal Medicine, San Diego CA. May 1-3, 2001. J Gen Intern Med 2001;16(suppl 1):220.
78. Cooper LA, Roter DL, Rokeymore MB, Johnson RL, **Ford DE**. Are there racial differences in primary care patient-physician communication after depression? 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):155.
79. Beach MC, Roter D, Frankel RM, Levinson W, **Ford DE**. What do physicians tell patients about themselves? A qualitative analysis of physician self-disclosure. 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):171.
80. Beach MC, Meredith L, Halpern J, Wells KB, **Ford DE**. Physician conceptions of loyalty to patients and social justice in healthcare: relationship to professional satisfaction under managed care. 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):171.
81. Daumit GL, Cooper LA, **Ford DE**, Steinwachs DM. How do individuals with severe mental illness rate the participatory decision making styles of their primary care physicians? 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):189.
82. Daumit GL, **Ford DE**, Fahey M, Steinwachs DM. Perceptions of coordination of care between medical and mental health providers among individuals with severe mental illness. 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):189.
83. Daumit GL, Graham CM, Flynn JA, Jenckes M, **Ford DE**. Measuring structural components and quality in primary care office practices. 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):189.
84. VanVoorhees BW, **Ford DE**, Rost KM, Rubenstein LV, Meredith L. Are patients with depression in primary care more difficult to treat than those seen by mental health specialists? Results from a primary care clinic sample. 25th Annual Meeting. Society of General Internal Medicine, Atlanta GA. May 2-4, 2002. J Gen Intern Med 2002;17(suppl 1):215.
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87. Rubenstein LV, Swanson KA, Meredith L, Lee ML, **Ford DE**, Rost KM. Are discomfort with depression care and/or clinical uncertainty associated with burnout among primary care clinicians? 26th Annual Meeting. Society of General Internal Medicine, Vancouver, British Columbia, Canada. April 30-May 3, 2003. J Gen Intern Med 2003;18(suppl 1):254.
88. Clever SL, **Ford DR**, Rubenstein LV, Rost KM, Meredith L, Sherbourne CD, Dominici F, Cooper LA. Primary care patients' involvement in decision making is associated with improvement in

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- depression. 26th Annual Meeting. Society of General Internal Medicine, Vancouver, British Columbia, Canada. April 30-May 3, 2003. *J Gen Intern Med* 2003;18(suppl 1):281.
89. McCauley J, Campbell J, Abushomar H, McNutt LA, **Ford DE**. Impaired Health Function among Women Experiencing Current Intimate Partner Violence. 27th Annual Meeting. Society of General Internal Medicine. May 12-15, 2004. Chicago IL.
 90. McCauley J, Campbell J, McNutt LA, **Ford DE**. Attitudes and Experiences with Help-Seeking among Women Experiencing Intimate Partner Violence. 27th Annual Meeting. Society of General Internal Medicine. May 12-15, 2004. Chicago IL.
 91. Hepner KA, Rubenstein VL, Rowe M, **Ford DE**. The Importance of Primary Care Practitioners in Depression Quality of Care. 28th Annual Meeting. Society of General Internal Medicine May 11-14, 2005 New Orleans, LA. *J Gen Intern Med* 2005;20(Suppl1):181.
 92. Van Voorhees BW, Ellis J, **Ford DE**, Pilot Study of a Primary Care/Web-Based Depression Prevention Intervention for Young Adults (Project Catch-It). 28th Annual Meeting. Society of General Internal Medicine May 11-14, 2005 New Orleans, LA. *J Gen Intern Med* 2005;20(Suppl1):187.
 93. Houston T, **Ford DE**, Smoking Cessation Websites As an Approach to Increasing Physician Visits for Smoking Cessation. 28th Annual Meeting. Society of General Internal Medicine May 11-14, 2005 New Orleans, LA. *J Gen Intern Med* 2005;20(Suppl1):190.
 94. Rayburn NR, **Ford DE**, Sherbourne CD, Development of the Depression Prognosis Index (DPI) to Predict Short-Term Outcomes for Primary Care Patients with Depression. 28th Annual Meeting. Society of General Internal Medicine May 11-14, 2005 New Orleans, LA. *J Gen Intern Med* 2005;20(Suppl1):175.
 95. Hepner KA, Rubenstein LV, Rowe M, **Ford DE**, Does Adherence to Depression Guidelines in Primary Care Improve Outcomes? 28th Annual Meeting. Society of General Internal Medicine May 11-14, 2005 New Orleans, LA. *J Gen Intern Med* 2005;20(Suppl1):176.
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Exhibit 2

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Email from Mark Brown dated June 29, 2006	DVA052 000113-000114
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