

UNITED STATES COURT OF APPEALS  
FOR THE DISTRICT OF COLUMBIA CIRCUIT

No. 21-1075

---

CHILDREN'S HEALTH DEFENSE, DR. ERICA ELLIOT, GINGER KESLER, ANGELA  
TSIANG, JONATHAN MIRIN  
PETITIONERS

V.

FEDERAL COMMUNICATIONS COMMISSION AND UNITED STATES OF AMERICA,  
RESPONDENTS

---

**APPENDUM TO EMERGENCY MOTION FOR STAY PENDING REVIEW  
OR IN THE ALTERNATIVE EXPEDITED REVIEW**

**VOLUME 2**

---

Robert F. Kennedy, Jr.  
Children's Health Defense  
48 Dewitt Mills Road  
1227 North Peachtree Pkwy, Suite 202  
Peachtree City, Georgia 30269  
NY Bar No. 1999994  
EMAIL: [rfk.fcc@childrenshealthdefense.org](mailto:rfk.fcc@childrenshealthdefense.org)  
TEL: 774.239.4768  
FAX: 512.692.2522

W. Scott McCollough  
McCollough Law Firm, P.C.  
2290 Gatlin Creek Rd.  
Dripping Springs, TX 78620  
Texas Bar No. 13434100  
EMAIL: [wsmc@dotlaw.biz](mailto:wsmc@dotlaw.biz)  
TEL: 512.888.1112  
FAX: 512.692.2522

*Counsel for all Petitioners*

## TABLE OF CONTENTS

Item ..... Tab

### VOLUME 1

Report and Order, *In the Matter of Updating the Commission's Rule for Over-the-Air Reception Devices*, FCC 21-10, WT Docket No. 19-71, \_\_  
FCC Rcd \_\_ (January 7, 2021) ..... A

Affidavit of Jonathan Mirin in Support of Motion for Stay ..... B

Affidavit of Dr. Erica Elliot in Support of Motion for Stay ..... C

Affidavit of Jennifer Baran in Support of Motion for Stay ..... D

Affidavit of Ginger Kesler in Support of Motion for Stay ..... E

Affidavit of Dr. David Hoffman in Support of Motion for Stay ..... F

Affidavit of Angela Tsiang in Support of Motion for Stay ..... G

Affidavit of Michele Hertz in Support of Motion for Stay ..... H

Affidavit of Dr. Toril Jelter in Support of Motion for Stay ..... I

Affidavit of Dr. Riina Bray in Support of Motion for Stay ..... J

### VOLUME 2

Affidavit of Dr. Beatrice Golomb in Support of Motion for Stay ..... K

Affidavit of Dafna Tachover in Support of Motion for Stay ..... L

Tab K

**AFFIDAVIT OF DR. BEATRICE GOLOMB IN SUPPORT OF STAY**

**UNITED STATES COURT OF APPEALS  
FOR THE DISTRICT OF COLUMBIA CIRCUIT**

Children's Health Defense, Dr. Erica  
Elliot, Ginger Kesler, Angela Tsiang,  
Jonathan Mirin  
Petitioners

USCA No. 21-1075

v.

Petition for Review of Order  
by the Federal Communications  
Commission  
(FCC 21-10)

Federal Communications Commission  
and United States of America,  
Respondents

**AFFIDAVIT OF DR. BEATRICE GOLOMB IN SUPPORT OF MOTION  
FOR STAY**

1. My name is DR. BEATRICE GOLOMB. I am a Professor at the University of California San Diego School of Medicine. My *curriculum vitae* is contained in Golomb Attachment 1.

2. I received a BS in physics, summa cum laude from the University of Southern California. After graduating, I worked at the Jet Propulsion Laboratory as an engineer. I have a PhD in biology with an emphasis on neurobiology as well as an MD from the University of California, San Diego. I focused on neural networks (now called "machine learning") in a post doctoral fellowship at the Salk Institute.

3. I served as a resident, and then Chief Resident in internal medicine, in the University of California Los Angeles (UCLA) medical system. I also received

training in research methods as a Robert Wood Johnson Clinical Scholar (RAND/UCLA), and served as a Health Consultant at RAND for some years.

4. I was a primary care physician for a panel of veteran patients in San Diego commencing in 1996 and was appointed to the faculty at the Department of Medicine at UC San Diego in 1998 where I am now a Professor of Medicine. I have over 15 years' experience as a primary care provider. I also have an active research lab.

5. My work in various areas has led to changes in US (and other nations') military policy, and drug regulation/labeling (e.g., FDA, Health Canada and the European Medicines Agency). A number of my research efforts have been featured in news and media, including the New York Times, Wall Street Journal, The Economist, TIME, Newsweek, BBC, NPR, CBS, NBC, and ABC among numerous other venues.

6. My research lab focuses on conditions and exposures that are tied to "Oxidative Stress" (OS) and impaired "mitochondrial" function that inhibits energy production in cells.

7. For the past eight years, this work has included research in injuries from exposure to pulsed and modulated radiofrequency ("RF") and Microwave ("MW") radiation used by wireless technology.

8. It was my work that originally determined that the “mystery illness” of US and Canadian diplomats in Cuba and China (and elsewhere) was, based on compelling evidence, caused by pulsed RF/MW. I was able to make this connection in part based on my research on RF/MW induced illness in adults and children who became sick after exposure to pulsed RF from wireless technology.

9. Attachment 2 concerns provides a detailed analysis of "Microwave Illness" (also known as “Electrosensitivity”). It provides the physiological mechanisms that lead to the characteristic symptoms and demonstrates they are caused by exposure to RF/MW radiation. These debilitating symptoms are engendered by underlying injuries for which a sound physiological basis is documented by hundreds, if not thousands, of peer-reviewed studies.

10. In an MRI study, brain injury was observed in persons with Microwave Illness. The authors of the study noted that “Over the years we have seen an increasing number of patients who had developed multi system complaints after long term repeated exposure to electromagnetic fields (EMFs). These complaints included headaches, intermittent cognitive and memory problems, intermittent disorientation, and also sensitivity to EMF exposure.”

11. These experts conducted functional MRIs of ten adult patients with “electrohypersensitivity” (sometimes known as “Microwave Illness”). Each of the ten “scans were abnormal with abnormalities which were consistent and similar.”

Their 2017 paper, “Functional brain MRI in patients complaining of electrohypersensitivity after long term exposure to electromagnetic fields” was published in Rev Environ Health 2017; 32(3): 291-299. It is contained in Golomb Attachment 2.

12. That study noted that brain imaging of individuals affected by Microwave Illness resembles traumatic brain injury, as was also reported for brain imaging studies of affected diplomats. The information and associated references are contained in my diplomat paper.

13. Other studies also affirm impaired blood flow to regions of the brain in individuals with Microwave Illness. As my paper demonstrates, there are other physiological manifestations in persons with Microwave Illness that can include permeability of the Blood-Brain-Barrier, depressed levels of melatonin, oxidative stress and auto-immune responses.

14. I was invited to speak to the National Academies of Sciences, Engineering, and Medicine Committee that was tasked by the State Department to examine the health problems experienced by US Foreign Services Personnel in Cuba. The National Academies’ report, published in December of 2020, came to the same conclusion that I had: the diplomats’ problems were likely caused by exposure to pulsed RF/MW. The report is contained in Golomb Attachment 4.

15. The predominant theory is that the RF/MW source that led to the diplomats' afflictions was an RF/MW weapon; such weapons are sometimes termed "neuroweapons." But it is clear the exposures to pulsed RF radiation lead to the same condition and injuries I, Dr. Heuser, Dr. Belpomme and many others have identified in many other individuals. Microwave Sickness or "electromagnetic sensitivity" can occur as the byproduct of an intentional assault through a neuroweapon or simply through supposedly benign longer term exposure to ambient RF/MW falling at or below FCC-authorized levels.

16. The fact is, some individuals at some point become intolerant to allowable levels of RF/MW. The intolerance can be minor at first. However, unless problematic exposure can be avoided, the condition then often worsens and, in many cases, devastating life consequences follow.

17. There is wide variation in both exposure type and response. Response can depend on factors like the RF/MW type or frequency, exposure to multiple frequencies of sources, on-off pulsation, sharp peaks and valleys, the modulation used, and/or chronic or long-term exposure.

18. Although it is a subset of the population that is or will become electrosensitive, the number of people affected is growing as the evolutionarily unprecedented exposure levels continue to rise.



19. Once affected, many individuals lose the ability to tolerate sources and levels of radiation which previously posed no problem to them. This was true for some of the affected diplomats, who now report they are unable to use a computer for more than a couple of hours a day.

20. These diplomats and civilians, who suffer from the same condition, feel abandoned by the system whose responsibility they believed it was to protect them. They report frustration and indeed are shocked by an apparent policy of feigned ignorance, dismissal, and denial of the problem.

21. One Foreign Services person (a senior CIA operative who had previously been shot at, several times) affected by a presumed RF/MW attack, stated about the devastating injury “I had rather been shot.”<sup>1</sup> Like many with RF/MW related illness, he described the distress from being disbelieved.

22. I have communicated with scores or hundreds of affected individuals. The suffering and anguish they experience is heart-wrenching. Like the Foreign Services person, they perceive they are under attack, and many describe the pain as torture.

---

<sup>1</sup> <https://www.archyde.com/a-secret-microwave-weapon-is-behind-the-attacks-on-cia-agents/>.

23. In all cases the only reliable “treatment” is exposure avoidance. Any other treatment is merely palliative. Avoidance reduces and, in some cases, may resolve the symptoms. But the condition is generally progressive. For many, re-exposure to a problematic source causes the symptoms to return. They may quickly regain their prior symptom severity, then continue to progress with potentially catastrophic or even deadly results.

24. We conducted a survey and found that half of those who were employed when they became affected lost or were forced to leave their jobs. Some spend months living in their car, no longer able to stay in their home, hunting for a safe place. Many are unable to work and exhaust all financial resources. They cannot find a home, have no recreation, social or family life, and cannot attend public events or religious gatherings. They must conduct a constant, increasingly difficult search for refuge that, even if found, is often soon taken away with the intrusion of some fresh emissions source. Thus, many live in dread that a new RF source will compel them to quickly flee again and begin their search anew, with all the consequent upheaval, uncertainties, burdens and costs.

25. This lifestyle poses significant risks in other ways. Often there are no basic amenities like heating, clean water, or restrooms for affected persons, living in their car or in the "wild"; and such individuals may be at risk from dishonorable or

predatory persons. For good reasons, these sufferers can descend into hopelessness and despair.

26. Some have chosen suicide, not due to traditional depression, but on account of their anguishing pain and debility, coupled with hopelessness about their plight.

27. A number of the patients we have studied in our lab, including those with Gulf War Illness or suffering side effects of fluoroquinolone antibiotics that produce chronic multi-symptom health problems in a vulnerable subset, have shared similar problems with disbelief and denial. A tragic difference in the case of RF-related illness is that affected individuals are subjected, without choice or recourse, to continued exposure to the inciting cause without meaningful ability to escape.

28. There is a surprisingly large and growing group of affected individuals with this condition. Their condition is not due to any personal fault or inadequacy. They must no longer be ignored, ridiculed, or rendered invisible or irrelevant. There must be some means to accommodate their situation and needs, to permit some place of refuge and grant them a measure of dignity. At minimum, they must be allowed to live in their own homes--for many, their sole sanctuary-- without being violated and driven out by some new, potentially unknown, and undisclosed emissions source. They need, and deserve, some place of refuge that does not itself become yet another place of torment.

29. This concludes my Affidavit.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on March 15, 2021

A handwritten signature in black ink, appearing to read "BeaT GM", written over a horizontal line.

Dr. Beatrice Golomb, MD, PhD

**GOLOMB Attachment 1**

**Curriculum Vitae****Beatrice Alexandra Golomb, MD, PhD**

Dept of Medicine 0995  
9500 Gilman Dr. La Jolla CA 92093-0995  
Phone: (858) 558-4950 x201  
Fax: (858) 558-4960  
Email: [bgolomb@ucsd.edu](mailto:bgolomb@ucsd.edu)

<http://medicine.ucsd.edu/SES/index.htm>

**CURRENT POSITION**

Professor of Medicine: July 2012 – present  
Division of General Internal Medicine  
University of California, San Diego School of Medicine

**EDUCATION AND TRAINING**

Robert Wood Johnson Clinical Scholar, UCLA: 1994 – 1996  
Chief Medical Resident, West Los Angeles VA Medical Center: 1993 – 1994  
Medical Resident, West Los Angeles VA Medical Center: 1990 – 1993  
Postdoctoral Fellow, Computational Neurobiology Laboratory, Salk Institute: 1989 – 1990  
MD, University of California, San Diego: June 1989  
PhD, Biology, University of California, San Diego: June 1988  
Medical Scientist Training Program, University of California, San Diego: June 1979  
BS, Physics, Summa Cum Laude (4.0 GPA at age 19), University of Southern California: 1979  
Physics graduate fellowship offers (declined), Harvard University and California Institute of Technology: 1979

**PAST APPOINTMENTS**

Professor of Family and Preventive Medicine: UC San Diego: July 2012 – July 2014  
Staff Physician, Department of Veterans Affairs, San Diego, 1996-2013  
Associate Professor of Medicine, UC San Diego: July 2004-2012  
Associate Professor of Family and Preventive Medicine, UC San Diego: July 2004-2012  
Health Consultant, RAND: Santa Monica, CA, August 1996 –2007.  
Research Associate Professor, Department of Psychology, Social Science Research Institute, University of Southern California, October 1998 – ?  
Robert Wood Johnson Generalist Physician Faculty Scholar: July 2003-2007  
Assistant Professor, Dept. of Family and Preventive Medicine, UC San Diego: July 2002-2004  
Assistant Professor of Medicine, UC San Diego: April 1998-July 2004  
Research Assistant Professor, Dept. of Psychology, USC: June 1995-1998  
Attending physician, Emergency Room, West Los Angeles VA Medical Center: 1994-1997

Teaching Assistant, Cell Biology, UCSD Department of Biology: 1987; Physiology of Sensation and Perception, UCSD Department of Psychology: 1987; Endocrinology, UCSD Department of Biology: 1986; Neurobiology, UCSD Department of Biology: 1985; Genetics, UCSD Department of Biology: 1983, 1984

Jet Propulsion Laboratory, Engineer I: 1979

Jet Propulsion Laboratory, Technical Aide A: 1978

**LICENSE** California, Issued October 8, 1991

**BOARD CERTIFICATION** American Board of Internal Medicine, September 1993

### **AWARDS, HONORS, FELLOWSHIPS**

Royal Society of Medicine, Overseas Fellow, May 2010

Robert Wood Johnson Generalist Physician Faculty Scholar Award: 2003 – 2007

Who's Who in America: 2000 – present

Fellow, AHA Council on Epidemiology and Prevention: 2000 – present

Fellow of the American Heart Association: July 2001

Invited Nominator, Edge of Computation Science Prize 2005

[http://www.edge.org/3rd\\_culture/prize05/prize05\\_index.html](http://www.edge.org/3rd_culture/prize05/prize05_index.html)

Associate Fellow, American Heart Association Council on Epidemiology and Prevention: elected March 25, 1999

Fellow, 23<sup>rd</sup> Annual American Heart Association 10-day Seminar on the Epidemiology and Prevention of Cardiovascular Diseases: 1997

Robert Wood Johnson Clinical Scholar: 1994-6 (listed also under Education and Training)

Solomon Scholar Research Award, UCLA: 1993

Solomon Scholar Research Award, UCLA: 1992

Emma Josephine Bradley Bovard Award (for graduating USC senior with best academic record): 1979

Summa cum laude graduate (4.0 GPA Physics, age 19)

Phi Kappa Phi: 1979

Phi Beta Kappa (Junior Inductee): 1978

### **PROFESSIONAL ACTIVITIES AND AFFILIATIONS (National/ International)**

Scientific Advisory board, We Are the Evidence, 2018-present

Scientific Advisory Board, Physicians for Safe Technology, 2018-present

Member, Cochrane Adverse Effects Methods Group, 2008 – present (International)

International Group for Reducing Inappropriate Medication Use and Polypharmacy (IGRIMUP): Invited as the US member, Dec 2012-present

Advisory Board, The Science Network (<http://www.tsntv.org/about/advisors.php>): 2004 – present

Department of Veterans Affairs Research Advisory Committee on Gulf War Veterans' Illnesses: Scientific Director Jan 2002 – Sep 2003; Chief Scientist: Sep 2003 – 2005; Member 2005-2015 (longest serving member)

Accompanied high-level mission to the Middle East, with Dr. Bernard Rostker (Assistant Secretary of Defense for Personnel and Manpower; then Undersecretary of the Navy), and several other DoD and Congress officials. Purpose: to brief officials from other nations regarding illness in Gulf War veterans and exposures in Persian

## PEER REVIEW

Peer Reviews for journals include:

*Major General Medicine Journals:* *New Engl J Med* (2001, 2003, 2006, 2010, 2017), *JAMA* (1999, 2008, 2013, 2014), *Lancet*, *Annals of Internal Med* (2005 x 2, 2006, 2007, 2008, 2011, 2012, 2014, 2015, **2018**), *Arch Int Med* now *JAMA Int Med* (2011, 2012, 2013x2, 2017), *BMJ* (1999, 2011, 2012, 2013x2, 2014x5, 2015x2, 2016x2, 2017x2, **2018x3, 2019, 2020**), *BMJ Open* (2012, 2013x3, 2014, 2015, 2017x2, **2019x2, 2020x2**), *Circulation*, *JACC* (2007 x 2, 2008, 2009, **2020x2**), *PLoS Medicine* (2014), *PLoS-ONE* (2008, 2011, 2013x2, 2014x2, 2015x3, 2017x2, **2018x3, 2019x2**).

*Other Journals:* *Adv Med Sci* (2011), *Afr J Agric Res* (2011), *Am J Cardiovascular Drugs* (2017), *Am J Clin Nutr* (2002, 2003), *Am J Epi*, *Am J Med* (1999, 2000), *AJMS* (**2019**), *Am J Kidney Disease* (2010), *Am J Preventive Med* (2010), *Am J Primatology*, *Ann Behav Med* (1999), *Ann Epi* (2006), *Ann Med* (2011), *Ann Surg* (2007), *Antibiotics* (**2019**) *Arch of Med and Health Sciences* (2015), *Arch Med Res* (2014), *Atherosclerosis*, *Biological Psychiatry* (2004, 2013), *BMC Cardiovascular Disorders* (2017x3, **2018x2**), *BMJ Cases* (2008 x 2), *BMJ Evidence* (**2020x2**), *BJCP* (**2018**), *Brain Sciences* (**2020**), *Br J Nutr* (2015x2), *Circulation: Cardiovascular Quality and Outcomes* (**2020**), *Clin Cardiol* (2012, 2013), *Clinical Infectious Diseases* (2017), *Clin Lipidology* (2015), *Complex Systems*, *Contemp Clin Trials* (2007x2, 2010), *Current Clin Pharm* (**2020**), *Current Drug Safety* (2011, **2020, 2021**), *Drug Safety* (2010, 2011), *EBioMedicine* (2015), *Eur J of Hospital Pharm* (**2020**), *EJON* (2017), *Eur J of Nutrition* (2015), *Eur J Pharmacol* (2007), *Evolutionary Anthropology*, *Expert Opinion on Drug Safety* (2017), *Expert Review of Cardiovascular Therapy* (2016), *Health Psychology* (2009, 2011, 2012x3), *Hypertension* (2013), *Infectious Disease and Therapy* (**2019**), *Int J Env Res Pub Health* (**2018, 2019x6, 2020x8, 2021x5**), *JAMA Ophthalmology* (2013), *J Affective Disorders* (2003, 2005, 2013), *J Applied Physiol* (2015), *J Clin Epi* (1999, 2000x2, 2001, 2003, 2004), *J Env Occup Sci* (**2018**), *J Gen Internal Med*, *J Health Psychol* (2014), *J Human Hypertension* (2012, 2013), *J of Int Med* (**2018**), *J Neuro and Neuro Med* (**2019**), *J Neurol Sci* (2012), *J Psychiatr Res* (2015x2, **2019**), *J Psychosom Res* (2013), *J Toxicology Env Health* (2009), *J Women's Health* (2007), *Marshall J of Med* (2017), *Medical Care*, *Med J Australia* (2012), *Military Med Res J* (**2020**), *Muscle and Nerve*, *Neural Computation* (**2020x2, 2021**), *NeuroImage: clinical* (2015), *Neuropsychopharmacology* (2006), *Neuroscience Letters*, *NeuroToxicology* (**2020**), *Neurotoxicology and Teratology* (2016x2, 2017x2), *Online Journal of Medicine and Medical Science Research* (2012), *Open Drug Safety* (2010, 2011), *Open Medicine* (2010), *Physiological Res* (2011, 2015), *Physiology & Behavior* (2000), *PLoS Comput Biol* (2013), *Psychiat Letter* (2005), *Psychiatric Services* (1999), *Psychiatry Research* (2005), *Psychosomatic Med* (1999, 2000, 2001x2, 2003), *Psychological Reports* (2000), *QJM* (2011, earlier), *Scientific Reports* (**2020**), *Social Science and Medicine* (2015x2), *SpringerPlus* (2016), *Ther Adv Drug Safety* (2011), *Tohoku J Exp Med* (2006, 2007), *Vaccines* (**2019, 2020**), *Webmed Central* (2011) others.

Peer Review quality: Received letters from *Annals of Internal Medicine* in three consecutive years stating they rate the quality of their reviews and that I was in the top 10% of reviewers by quality for the prior year (then the editor that provided these letters left). Received a letter again, 2016 after most recent review stating that my reviews were in the top “category” (unspecified) of review quality.

Peer Review participation, Books: Oxford University Press

Peer review, grants, national and international:

Grant Review, International Coenzyme Q10 Association, (2007)

National Institute for Health Research, Research for Patient Benefit Programme (UK), (2011)

DoD, Gulf War, Consortium Review panel (2012)

UCSD Clinical Translational Research Institute (CTRI) grant review (2017)

International Journal of Environmental Research and Public Health – Special Issue Editor (**2020**)

DoD Congressionally Directed Medical Research Program (CDMRP) grant review (**2020**)



Chair of a DoD Congressionally Directed Medical Research Program (CDMRP) grant review committee on Gulf War Illness (October 2020)

## EXPERT PANEL PARTICIPATION

Department of Veterans' Illnesses Affairs, Research Advisory Committee on Gulf War Illnesses: Jan 2002 – 2015.

National Lipid Association, Statin Adverse Effects meeting for position paper, Atlanta Oct 5-6 2013 (participated remotely). (I removed myself from the document which did not meet my standards for rigor or impartiality.)

Panelist, NIH Contract Review, Use of Biological Samples from WHI, May 2008.

Panelist, NIH (NHLBI) Program Project Review. February 2007.

Panelist, NIH (NHLBI) Review Panel on "Prevention of Cardiovascular Disease in Diabetes Mellitus: Clinical Center Network Proposals": June 1999.

Expert "panelist" for the Department of Defense in the Army After Next AMEDD Technical Workshop to advise strategies for troop health protection in the year 2025. MacLean, Virginia: June 13 1999 – June 18 1999.

Expert panelist for the Center for Health Policy Research/Health Care Financing Association "Normative Standards Project", pertaining to normative standards for home health care (requested; participation aborted due to date conflict with Department of Defense panel above): June 1999.

Panelist and speaker, Violence Prevention Coalition of Greater Los Angeles meeting entitled "How might interdisciplinary models of research guide us to a better understanding of violence?" Los Angeles, CA: Nov 1998.

## BRIEFINGS TO NATIONAL ACADEMIES GROUPS

"Diplomats' Mystery Illness and Pulsed Radiofrequency/Microwave Radiation", invited talk for Cuban Academy of Sciences, Havana, Cuba, March 2, 2020. <https://cnl.salk.edu/~terry/Beatrice-Havana/>

"Diplomats' Mystery Illness and Pulsed Radiofrequency/Microwave Radiation," invited talk for the U.S. National Academies of Sciences, Engineering, and Medicine's Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and their Families at Overseas Embassies, Washington, D.C., Feb 24<sup>th</sup>, 2020.

"Gulf War illness," Briefing to IOM Committee on Gulf War & Health, National Academy of Science Building, Washington, D.C., Jan 27, 2015 (invited briefing #2, given remotely)

"Gulf War illness," Briefing to IOM Committee on Gulf War & Health, National Academy of Science Building, Washington, D.C., Dec 3, 2014 (invited talk, given remotely)

Briefing to the Committee on Developing a Consensus Case Definition for Gulf War Illness, NAS Board Room, National Academy of Sciences Building, Washington, D.C. June 26, 2013 (remote)

"Coenzyme Q10 in Gulf War Illness: A Randomized Controlled Trial" invited talk for Institute of Medicine Committee on Gulf War and Health: Treatment of Chronic Multisymptom Illness, National Academies Beckman Center, Irvine, CA April 12, 2012.

"Pitfalls in the Application of Evidence." The National Academies seminar *What Can Be Learned from Public Health on the Role of Research for Policy Purposes?*, Invited lecture to Division of Behavioral and Social Sciences and Education Standing Committee on Social Science Evidence for Use, The National Academies, Irvine, CA: Oct 30, 2008.

## BRIEFINGS TO GOVERNMENT AGENCIES

"Recruitment of Gulf War veterans for research studies," Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC, Sept 23, 2014

- “Gulf War illness and mitochondrial dysfunction” Department of Veterans Affairs: Mitochondrial Disease meeting, June 12, 2014, Washington, D.C.
- “Treatment for Gulf War illness: Coenzyme Q10 Study Results”, briefing to House of Representative members and Staffers, Cannon House Office Building, Washington, D.C., 3:30 PM (invitation by Rep Kucinich), Feb 1, 2012.
- “Treatment for Gulf War illness: Coenzyme Q10 Study Results”, briefing to Senate staffers, 418 Russell Senate Office Building (invitation by Senator Bernie Sanders), 2:200 PM, Feb 1, 2012.
- “Treatment for Gulf War illness: Coenzyme Q10 Study Results”, briefing to Senate members, 332 Dirksen Senate Office Building, Washington, D.C., 5PM, Jan natio31, 2012 (invitation by Senator Bernie Sanders).
- “Coenzyme Q10 for Gulf War Veterans,” Invited talk to Department of Veteran Affairs Research Advisory Committee on Gulf War Veterans’ Illnesses, Washington, D.C., June 27, 2011.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: Sept 16, 2008.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: April 08, 2008.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: July 15, 2007.
- “Oxidative stress, mitochondria, and illness in Gulf War veterans: A hypothesis.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: April 24, 2007.
- “Statin Side Effects.” Invited discussion with Senate Finance Committee representatives. Dickson Building, Capitol Hill, Washington DC: June 9, 2006.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: May 16, 2006.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: April 8, 2005.
- “Gulf War Illnesses, Anthrax Vaccine.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: April 7, 2005.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses. Washington DC: June 29, 2004.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: Feb 23, 2004.
- “Gulf War Illnesses, Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: June 2003.
- “Birth Defects in Gulf War Veterans, Gulf War Veterans Research Update.” Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: June 2003.
- “Candidate Research Recommendations,” “Vaccines and illness in Gulf War Veterans”, Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: Feb 2003.
- “Gulf War Veterans Illnesses, Research Update focusing on Acetylcholinesterase inhibitors.” Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: Feb 2003.
- “Treatments for Gulf War Veterans: What has been tried, and what are candidate treatments?” “Vaccines and illness in Gulf War Veterans.” Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: Oct 2002.

"Vaccines and illness in Gulf War Veterans." Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: Oct 2002.

"Acetylcholinesterase inhibitors and illness in Gulf War veterans." Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: June 26, 2002.

"Treatments for ill Gulf War veterans." Research Advisory Committee on Gulf War Veterans Illnesses (Committee meeting open to the public). Washington DC: Feb 2002.

"Pyridostigmine Bromide: A Review of the Scientific Literature as it Pertain to Gulf War Illnesses." Testimony to Congress: House Veterans Affairs Committee, Health Subcommittee and Benefits Subcommittee. Washington DC: Nov 19, 1999. <http://www.rand.org/content/dam/rand/pubs/testimonies/2005/CT164.pdf>

Briefings to members of Senate subcommittees (Foreign Affairs and Veterans Affairs). Washington DC: Oct 19, 1999.

"Pyridostigmine Bromide: A Review of the Scientific Literature as it Pertain to Gulf War Illnesses." Briefing to representatives of multiple U.S. Veterans advocacy groups. Washington DC: Oct 19, 1999.

"Pyridostigmine Bromide: A Review of the Scientific Literature as it Pertain to Gulf War Illnesses." Press Briefing from the Pentagon. Washington DC: Oct 19, 1999.

"Pyridostigmine bromide and illness in Persian Gulf War veterans." Briefing to group consisting of: the Undersecretary of Health, the Undersecretary of the Army, the Surgeon General of the Army, the Principal Deputy for Health Affairs, representatives from the Surgeons General of the Navy and Air Force, several other generals, and the Directors of the Health and Defense Programs at RAND (The group convened for the exclusive purpose of hearing my briefing). Washington DC: Feb 3, 1998.

"RAND on PB and Immunizations." Presidential Special Oversight Board for Department of Defense Investigations of Gulf War Chemical and Biological Incidents. Arlington, VA: Oct 28, 1998.

"RAND Center for Military Health Policy Research: Gulf War Illness." (with Dr. Ross Anthony) Briefing to Major General James B. Peake, Commanding General/Commandant/ Installation Commander, US Army Medical Department Center and School. RAND Arroyo Center, Army Research Division. Santa Monica, CA: Oct 22, 1998.

"Health Effects of Service in the Gulf War." (with Dr. Ross Anthony) Briefing to DARPA and DSO (Defense Science Office. Arlington, VA: Sept 8, 1998.

"Health Effects of Service in the Gulf War." (with Dr. Ross Anthony) Briefing to Advisory Board of the government Defense Research Institute. RAND. Santa Monica, CA: May 5, 1998.

"Health Effects of Service in the Gulf War." (with Dr. Ross Anthony) Briefing to RAND Board of Trustees RAND's 50th birthday. Washington DC: April 9, 1998.

"Pyridostigmine bromide and illness in Persian Gulf War veterans." Briefing to representatives from DoD, VA, FDA, PAC, and Congress. RAND Washington, Washington DC: Nov 4, 1997.

"Pyridostigmine bromide." Briefing to Israeli Defense and Health personnel, as part of a mission to the Middle East with Dr. Bernard Rostker (Assistant Secretary of the Navy; now Undersecretary of the Army) and others from the DoD. Tel Aviv, Israel: Nov 1 1997 (Participated in other briefings in the Middle East - Kuwait, Saudi Arabia, and Egypt - during a ~2 week trip).

Other Research Advisory Committee on Gulf War Veterans Illnesses Meetings Attended: Numerous.

## ON-LINE TALKS/INTERVIEWS

Conflict of Interest, Salk Institute talk, 2008: <http://www.youtube.com/watch?v=nFtt-W3LROY>

Conflict of Interest, 2011 <http://vaccinesafetyconference.com/videos.html>

Interview Conflict of Interest (I did not name this talk):

<http://articles.mercola.com/sites/articles/archive/2010/06/12/beatrice-golomb-interview.aspx>

Interview Chocolate: [http://www.foodconsumer.org/newsite/Nutrition/Food/chocolate\\_082620120847.html](http://www.foodconsumer.org/newsite/Nutrition/Food/chocolate_082620120847.html)

Vaccine Safety Meeting: <http://www.youtube.com/watch?v=SZHyLODgUvs&feature=plcp>

Jon Stewart Daily Show (lampooned on):

<http://www.thedailyshow.com/watch/thu-october-21-1999/headlines---pills-bury-doughboys>

<http://thedailyshow.cc.com/videos/zgu22c/headlines---pills-bury-doughboys>

ABC (Australia Broadcasting Company) TV: <http://youtu.be/wAKaM330xzg>

EHS, radio interview with *Boil the Frog Slowly* with Sebastian Sanzotta

## **LECTURES, PRESENTATIONS (Includes invited presentations to National Academies groups)**

**(See also Briefings to Government Agencies; and Abstracts, many of which had accompanying presentations)**

“Evidence-based Diet,” lecture for Med 410 *From Principles to Practice*, UC San Diego, course director Ian Jenkins, La Jolla, CA, Feb 17, 2021.

“Diplomats’ Mystery Illness: Pulsed Radiofrequency/Microwave Radiation,” EMF Medical Conference 2021, January 28<sup>th</sup>, 2021. <https://emfconference2021.com/>

“EMF and Health: Lessons from Diplomats.” Building Biology Electromagnetic Radiation Specialists Meeting, Invited presentation and Q&A, January 12<sup>th</sup>, 2021.

“Bioenergetics in Veterans with Gulf War Illness Versus Healthy Controls: Replication and Expansion,” Gulf War Illness State of the Science Virtual Conference co-hosted by the Department of Veterans Affairs (VA) Office of Research & Development (ORD) and the Department of Defense (DoD) Congressionally Directed Medical Research Programs (CDMRP) Gulf War Illness Research Program (GWIRP), Washington, D.C., August 19<sup>th</sup>, 2020 (oral presentation).

“Nuclear and Mitochondrial Genetics Together Determine Gulf War Illness Severity and Symptom Profile,” Gulf War Illness State of the Science Virtual Conference co-hosted by the Department of Veterans Affairs (VA) Office of Research & Development (ORD) and the Department of Defense (DoD) Congressionally Directed Medical Research Programs (CDMRP) Gulf War Illness Research Program (GWIRP), Washington, D.C., August 18<sup>th</sup>, 2020 “poster presentation” (actually given as 10 minute taped talk). <http://va-eerc-ees.adobeconnect.com/plixyd0rh4ht/>

“When Good People Go Bad: Biological Risk Factors for Aggression,” Altman Clinical and Translational Research Institute seminar series at UCSD, La Jolla, CA, April 14<sup>th</sup>, 2020. ~~Pending~~ {Canceled due to Covid-19 pandemic}

“Diplomats’ Mystery Illness and Pulsed Radiofrequency/Microwave Radiation”, invited talk for Cuban Academy of Sciences, Havana, Cuba, March 2, 2020. <https://cnl.salk.edu/~terry/Beatrice-Havana/>

“Diplomats’ Mystery Illness and Pulsed Radiofrequency/Microwave Radiation,” invited talk for the U.S. National Academies of Sciences, Engineering, and Medicine’s Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and their Families at Overseas Embassies, Washington, D.C., Feb 24<sup>th</sup>, 2020.

“Evidence-based Diet,” lecture for Med 410 *From Principles to Practice*, UC San Diego, course director Ian Jenkins, La Jolla, CA, Feb 18, 2020

“5G: Are There Health Concerns?”, University of Southern California Entertainment Technology Center’s “5G: Planning and Piloting First M&E Services” Symposium, Los Angeles, CA, Dec 10, 2019

“Meta-analysis: Considerations and Limitations,” Graduate Biology Seminar, UC San Diego, course director Dr. Pamela Reinagel, La Jolla, CA May 28, 2019

“Diplomats’ Mystery Illness and Pulsed Radiofrequency/Microwave Radiation,” American Society of Safety Professionals, Jacobs Center for Innovation, San Diego, CA Mar 12, 2019

“Diplomats’ Mystery Illness and Pulsed Radiofrequency/Microwave Radiation,” UCSD Preventive Medicine fellows seminar, La Jolla, CA Mar 1, 2019

“Evidence-based Diet,” lecture for Med 410 *From Principles to Practice*, UC San Diego, course director Ian Jenkins, La Jolla, CA, Feb 12, 2019



- “Study Designs,” General Internal Medicine faculty seminar, UC San Diego, lecture series director Gerry Boss, La Jolla, CA (Jan 9, 2019).
- “Evaluation of Evidence and Inference,” UC San Diego General Internal Medicine faculty seminar, lecture series director Gerry Boss, La Jolla, CA Dec 8, 2018.
- “Diplomats’ Mystery Illness and Pulsed Radiofrequency/Microwave Radiation,” UC San Diego Marshall College Honors Seminar, Couse Director Leslie Carver, La Jolla, CA Nov 26, 2018
- “Gulf War illness – why it matters for the rest of us.” UCSD CTRI lecture series, Hillcrest, CA May 3, 2017,
- “Electrohypersensitivity: A ‘current’ and future problem”. at meeting *Cell Phones and Wireless Technologies—Should Safety Guidelines Be Strengthened to Protect Adults, Children and Vulnerable Populations?* Commonwealth Club, San Francisco (R), June 22, 2015. (invited talk)
- “Taking down the enemy: The industry (et al) playbook.” at *Royal Society* sponsored meeting *Science at the Crossroads: Scepticism vs Denial and Elitism vs Public Engagement*. Chicheley Hall, Milton Keynes, UK (R). June 15, 2015 (invited talk)
- “Gulf War illness,” Briefing to IOM Committee on Gulf War & Health, National Academy of Science Building, Washington, D.C (R), Jan 27, 2015 (invited briefing #2)
- “Stacking the Deck: Treatment Benefits and Risks” La Jolla Country Club Men’s Luncheon Club (retired executives and professionals), La Jolla, CA Jan 7, 2015. (invited talk)
- “Gulf War illness,” Briefing to IOM Committee on Gulf War & Health, National Academy of Science Building, Washington, D.C (R), Dec 3, 2014 (invited talk)
- “Gulf War illness and mitochondrial dysfunction “Department of Veterans Affairs: *Mitochondrial Disease* meeting, Washington, D.C. (R), June 12, 2014
- “Chocolate: My Favorite Vegetable.” Revelle College Honors Seminar, UC San Diego, La Jolla, CA, Feb 11. 2014.
- “Evidence Based Diet,” lecture for Med 410 *From Principles to Practice*, UC San Diego, La Jolla, CA, Feb 3, 2014
- “When Good People Go Bad: Biological Risk Factors for Aggression”. Naval Medical Center, Department of Psychiatry Grand Rounds, San Diego, CA Jan 31, 2014
- “The Angina Monologues: Statins in Women” (debate). American College of Cardiology - California, Beverly Hills, Nov 21, 2013.
- “Statin Effects and Risk Benefit.” Invited presentation, American College of Nutrition, San Diego, Nov 14, 2013
- “Distortions in Medical Information: Let me Count the Ways.” Science Studies, UCSD, La Jolla, Oct 14, 2013
- “The Older the Better?” International Association of Gerontology & Geriatrics 20<sup>th</sup> World Congress of Gerontology & Geriatrics in Seoul, South Korea. June 26, 2013 (with Marcella Evans).
- “Stop Medicating Beyond the Evidence. Guidelines for Guidelines on Preventive Treatments.” International Association of Gerontology & Geriatrics 20<sup>th</sup> World Congress of Gerontology & Geriatrics in Seoul, South Korea. June 24, 2013 (with Marcella Evans).
- “Stacking the deck: How conflict of interest advantages identification of benefit over harm.” Invited public lecture hosted by the Center for Values in Medicine, Science and Technology, fUT Dallas, Dallas, TX Apr 17, 2013.
- “Chocolate: My Favorite Vegetable.” Revelle College Honors Seminar, UCSD, San Diego, Feb 14, 2013.
- “Evidence Based Diet” lecture for Med 410 *From Principles to Practice*, UC San Diego, La Jolla, CA, Feb 12, 2013.
- Statin Roundtable, with Dr. Stephen Sinatra and Dr. David Perlmutter Dec 17, 2012
- “Statins in the Elderly,” Geriatric Pharmacology Seminar, UC San Diego, La Jolla, CA, Dec 05, 2012
- “Chocolate and Memory.” American Heart Association, Los Angeles, Nov 6, 2012.
- (“*Statins.*” Keynote talk, *Bringing Evidence to Frontline Clinicians*, Vancouver Nov 2, 2012 (*I had to cancel*))
- “Stacking the Deck: Conflict of Interest in Medicine.” Osher Lifelong Learning talk, UCSD, La Jolla, CA Oct 23, 2012.

“Statins: The Good, the Bad, the Recommendations, the Evidence.” Osher Lifelong Learning talk, UCSD, La Jolla, CA Oct 9, 2012.

“Chocolate: My favorite vegetable.” Biomedical library series, UCSD, La Jolla, Oct 4, 2012.

General Internal Medicine Grand Rounds, UC San Diego, La Jolla, CA Sept 26, 2012

“Gulf War illness.” briefing to regional Gulf War veterans, La Jolla, CA Sept 5, 2012.

“Placebos.” Skyped talk to Placebo meeting, University of British Columbia, Canada, May 23, 2012.

“Vaccines, Oxidative Stress, Autoimmunity, And Chronic Multisystem Health Outcomes” 8<sup>th</sup> International Congress on Autoimmunity. [www.kenes.com/Autoimmunity](http://www.kenes.com/Autoimmunity), Granada, Spain. May 11, 2012.

“Coenzyme Q10 in Gulf War Illness: A Randomized Controlled Trial” invited talk for Institute of Medicine Committee on Gulf War and Health: Treatment of Chronic Multisymptom Illness, National Academies Beckman Center, Irvine, CA April 12, 2012.

“Statins raise glucose preferentially among men who are older and at greater metabolic risk.” AHA Joint Conference - Nutrition, Physical Activity and Metabolism and Cardiovascular Disease Epidemiology and Prevention Scientific Sessions (oral presentation) San Diego. *Co-listed in Abstracts*. Mar 16, 2012.

“Evidence based diet.” lecture for Med 410 *From Principles to Practice*, UC San Diego, La Jolla, CA, Feb 14, 2012.

“Sound decisions about drugs. A call for improved drug safety science.” 11-20-2011, Washington, D.C.

“Q10 for Gulf War Veterans,” Invited talk to Department of Veteran Affairs Research Advisory Committee on Gulf War Veterans’ Illnesses, Washington, D.C., June 27, 2011. (Colisted under Briefings above).

“Conflict of interest: Stacking the deck in drug risks vs benefits.” Invited lecture, Dept of Medicine “B”, Sheba Medical Center affiliated with Tel Aviv University, Tel-Hashomer, Israel. June 16, 2011.

“Janus-faced predictors: And why randomized, double-blind, placebo-controlled trials are none of the above.” Conference in Honor of Halbert L. White, Jr. - Causality, Prediction, and Specification Analysis: Recent Advances and Future Directions. San Diego, CA May 6-7, 2011.

“Evidence Based Diet.” lecture for Med 410 *From Principles to Practice*, UCSD, La Jolla CA, Mar 15, 2011.

“Stacking the Deck: Drug Risks and Benefits.” Phil 26 Science, Society and Values: Good Bad and Junk Science. Course Director Professor Craig Callendar. UCSD, La Jolla, CA Feb 24, 2011.

“Research on drugs and vaccines, evidence vs truth: a call for formal study of drug harms.” Vaccine Safety conference, Montego Bay, Jamaica, Jan 5, 2011.

“Representation of drug benefits vs harms: the impact of conflict of interest.” Vaccine Safety conference, Montego Bay, Jamaica, Jan 4, 2011.

“Patient Reporting of Drug Adverse Effects.” International Society of Pharmacovigilance, Accra, Ghana, Nov 3-6, 2010 (Golomb = invited presenter and talk author; delivered by Marcella Evans whom I funded to attend).

“Conflict of Interest.” CREST (Clinical Research Enhancement Through Supplemental Training, (<http://crest.ucsd.edu>, La Jolla, CA, October 27 and 28, 2010.

“Statins and Exercise.” Invited lecture, American College of Sports Medicine, Southwest Chapter 2010 Annual Meeting, Mission Valley Marriott, San Diego, CA Oct 22, 2010.

“Statins, Q10 and Mitochondrial Function.” invited lecture, International Coenzyme Q10 Association Meeting, Brussels, Belgium May 29, 2010 (Golomb = invited presenter and talk author; delivered by Dr. Peter Langsjoen).

“Statin Effects and Adverse Effects.” Rockefeller University, New York, Mar 17, 2010.

“Evidence Based Diet.” UCSD School of Medicine SOM410, From Principles to Practice, La Jolla, CA, Feb 23, 2010.

- “Rectal (and Swallowed) Foreign Bodies.” Invited talk, *Ig Nobel Winners in San Diego*, San Diego Marriott, Feb 19, 2010, <http://improbable.com/airchives/mini2010/mini2010-02.htm>.
- “Stacking the Deck.” Invited Talk, PRIME-LC group (medical students), UC Irvine, Feb 9, 2010.
- “Stacking the Deck: Drug Benefits and Harms.” York University, Toronto, Canada, Jan 22, 2010.
- “Stacking the Deck.” ICES/CEU Conjoint Evaluative Sciences Rounds, University of Toronto, Jan 22, 2010.
- “Q10 for Gulf War Veterans.” Department of Defense research meeting, Hallmark Crowne Center, Kansas City, MO, Sep 2, 2009.
- “Stacking the Deck.” Phil 12: Logic and Decision Making, Philosophy of Science, UC San Diego, Warren Lecture Hall, San Diego, CA, Aug 27, 2009.
- “Evidence Based Diet.” Noon Conference, Veterans Affairs Medical Center, San Diego, CA, June 12, 2009.
- “Evidence Based Diet.” Hebrew University School of Medicine, Jerusalem, Israel, June 3, 2009.
- “Stacking the Deck: Drug Benefits and Harms.” The Technion School of Medicine, Haifa, Israel, June 2, 2009.
- “Statin Effects.” The Technion School of Medicine, Haifa, Israel, June 1, 2009.
- “Cholesterol and Behavior: From Case Reports to Population Data.” Invited presentation, seminar entitled *Modeling anti-social behavior: lessons from cholesterol biosynthesis*. Society for Biological Psychiatry 64<sup>th</sup> Annual Meeting, Vancouver: May 14-16, 2009.
- “Drug Risks and Benefits: Stacking the Deck.” Invited/guest lecture in Soc 40, Sociology of Healthcare Issues, UCSD, Course Director Tom J. Waidzun, PhD, La Jolla, CA May 18, 2009.
- “Statin Effects and Side Effects.” Cardiology Grand Rounds, San Diego Cardiac Center, Sharp Memorial Hospital, La Jolla, CA: April 17, 2009.
- “Gulf War Syndrome.” in Topics and Advances in Internal Medicine, University of California, San Diego School of Medicine, Hilton San Diego Resort, San Diego CA: March 9, 2009.
- “Evidence Based Diet.” UCSD School of Medicine SOM410, From Principles to Practice, La Jolla, CA: Feb.17, 2009.
- “Statin Side Effects.” Preventive Medicine seminar, UC San Diego, La Jolla, CA: Feb 10, 2009.
- “Aging.” San Diego Forum, La Jolla: CA: Jan 27, 2009.
- “Pitfalls in the Application of Evidence.” The National Academies seminar *What Can Be Learned from Public Health on the Role of Research for Policy Purposes?*, Invited lecture to Division of Behavioral and Social Sciences and Education Standing Committee on Social Science Evidence for Use, The National Academies, Irvine, CA: Oct 30, 2008.
- “Issues in the Identification and Communication of Drug Adverse Effects.” American Public Health Association, Invited lecture, San Diego, CA: Oct 27, 2008.
- “Conflict of Interest in Medicine.” *Beyond Belief: Candles in the Dark*, sponsored by *The Science Network* (tsntv.org), session entitled “This is Your Brain on Politics” Salk Institute, La Jolla, CA: Oct 5, 2008; <https://www.youtube.com/watch?v=nFtt-W3LROY>
- “Gulf War Syndrome.” General Internal Medicine Rounds, UC San Diego, La Jolla, CA: Aug 13, 2008.
- “Dissent in Medicine: Stacking the Deck.” *London School of Economics/UCSD Science Studies Program Dissent in Science: Origins and Outcomes* Workshop, La Jolla, CA: Mar 3 2008. (Sponsored by AHRC (UK) as part of the Contingency and Dissent in Science project at the CPNSS, London School of Economics).
- “Stacking the Deck: Drug Risks and Benefits.” Health Services Research & Development Scholarly Conference, Veterans Affairs San Diego Healthcare System, La Jolla CA: Feb 22, 2008.
- “Evidence Based Diet.” UCSD School of Medicine SOM 410, From Principles to Practice, La Jolla, CA: Feb.19, 2008.

“Stacking the Deck: Treatment Risks and Benefits.” General Internal Medicine Grand Rounds, UCSD School of Medicine, La Jolla, CA: Jan. 30, 2008.

“Simvastatin But Not Pravastatin Affects Sleep: Findings from the UCSD Statin Study.” American Heart Association annual meeting, oral presentation, Orlando, FL: Nov. 7, 2007.

“Statins: Risks and Benefits.” Grand Rounds, Scripps Green Hospital. La Jolla, CA: June 27, 2007.

“Statin side effects. A mitochondrial connection?” Invited talk, UC Irvine MitoMed group (mitochondrial medicine), Irvine, CA: June 18, 2007.

“Cholesterol, Heart Disease, and You.” UCSD Health and Wellness Series, Price Center, UCSD. La Jolla, CA: May 22, 2007.

“Statin Side Effects.” Invited Lecture, American College for Advancement in Medicine. Chicago, IL: May 13, 2007.

“Mitochondrial Dysfunction and Illness in Gulf War Veterans.” Research Advisory Committee on Gulf War Illness. Department of Veterans Affairs, Washington D.C.: April 24, 2007.

“Enhancing Post-marketing Drug Surveillance: A Response to Expressed Needs of Patients.” Robert Wood Johnson Foundation Generalist Physician Faculty Scholars Program Annual Meeting oral presentation, San Antonio, TX: Dec 1, 2006.

“Do Low Dose Statins Affect Cognition? Results of the UCSD Statin Study.” American Heart Association oral presentation, Chicago, IL: Nov. 15, 2006.

“Ethical Reasoning in Medicine – or the Contrary?” UCSD School of Medicine Biomedical Ethics Seminar: Sept 20, 2006.

“Stacking the Deck.” Health Services Research & Development, VA San Diego Healthcare Center. La Jolla, CA: July 20, 2006.

“Treatment risks vs benefits: Stacking the Deck.” International Relations course IRGN490, UCSD, April 19, 2006.

“Gulf War Illness.” VA San Diego Healthcare Center. La Jolla, CA: April 14, 2006.

“Do statins cause long term adverse effects?” Invited talk followed by invited panelist discussion, Panel: Controversies in Lipid Lowering Therapy, American College of Cardiology meeting. Atlanta, Georgia: March 12, 2006.

“Cholesterol and Violence.” Invited talk, San Diego Superior Court. San Diego, CA: Feb 7, 2006.

“Peer Review.” Invited talk, VA San Diego Healthcare center. La Jolla, CA: Dec 9 2005.

“Patient Targeted Adverse Event Surveillance: Use for Hypothesis Generation.” Robert Wood Johnson Generalist Physician Faculty Scholar meeting. Ft. Lauderdale, FL: Nov 10, 2005.

“Should statins be put in the water supply?” Distinguished Visitor Programme speaker, Biomedical Research Council, Agency for Science, Technology and Research (ASTAR). Singapore: Oct 25, 2005.

“Bridging the basic science/clinical science gap”. MSTP (Medical Scientist Training program – MD/PhD program) retreat roundtable presentation. Aug 27, 2005.

“Conflict of interest”; CREST (Clinical Research Enhancement Through Supplemental Training, <http://crest.ucsd.edu>), Patient Oriented Research II: Ethics and regulation of human research. San Diego, CA: Aug 17, 2005; La Jolla, CA: Aug 18, 2005.

“A Scientific Career.” McNair Summer Research Program, UCSD. La Jolla, CA: July 2005.

“Clinical follow-up after stopping statin treatment.” Fourth Conference of the International Coenzyme Q10 Association. Los Angeles, CA: April 2005.

“Lack of Physician Response Toward Perceived Statin Adverse Events.” 45th Annual Conference on Cardiovascular Disease Epidemiology and Prevention in association with the Council on Nutrition, Physical Activity and Metabolism. April 2005.



"Drug benefits and harms: Stacking the deck." Science Policy Analysis Roundtable series

(<http://acs.ucsd.edu/~spar/>), UCSD. La Jolla, CA: March 10, 2005.

"Adverse Drug Effects: The Case of Statins." UCSD Biomedical Ethics Seminar Series, UCSD. La Jolla: CA. Feb 16, 2005.

"Coenzyme Q10, Mitochondrial Function, Statins, and Aging." Stein Institute for Research on Aging Grand Rounds, UCSD. La Jolla, CA: Jan 10, 2005.

"Should Statins be Put in the Water Supply?" Dept of Medicine, Yale. Newhaven, CT: July 21, 2004.

"CNS effects of low cholesterol." UCSD Dept of Biology colloquium. La Jolla, CA: April 2004.

"Cholesterol and the brain". UCSD Dept of Psychology colloquium. La Jolla, CA: March 16, 2004.

"Should statins be put in the water supply?" CTF C 301, UCSD. La Jolla, CA: March 10, 2004.

"Cholesterol and the brain: Mood, violence, and cognition." Dept of Psychology, University of Southern California. Los Angeles, CA: Feb 11, 2004.

"Cardiovascular Prevention: Putting the Risk in Risk Benefit." Department of Preventive Medicine, SUNY. Stonybrook, NY: Jan 27, 2004.

"The Great Debate. Do benefits of statins as currently used exceed the risks?" (with debaters John H. Lehman, John Robin Crouse and Michael J. Davidson) American College of Toxicology Annual Meeting. Washington DC: Nov 4, 2003.

"Anthrax Vaccine: Is it safe? Is it effective?" VA Faculty Development Seminar Series. La Jolla, CA: June 15, 2003.

"The Anthrax Vaccine: Is it safe and effective?" Epidemiology conference, Family and Preventive Medicine. La Jolla, CA: April 7, 2003.

"Research Recommendations: Focus on Acetylcholinesterase Mechanisms." Presentation to the Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses. Washington DC: Feb 2003.

"Recent Gulf War Illnesses Research." Presentation to the Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses/ Washington DC: Feb 2003.

"Acetylcholinesterase Inhibitors and Gulf War Illnesses." Presentation to the Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses. Washington DC: Nov 2002.

"Gulf War Veterans Illnesses: Treatment issues." Presentation to the Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses. Washington DC: Nov 2002.

"Mitochondrial function and Gulf War Illnesses." Presentation to the Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses. Washington DC: Nov 2002.

"A new perspective on cholesterol, statins, and heart disease." Stein Institute for Aging public lecture (televised for UCSD TV), La Jolla, CA: Aug 21, 2002.

"Putting the risk in risk benefit analysis." Dept of Medicine, Stonybrook School of Medicine. Stonybrook, NY: July 16, 2002.

AHA Debate: "Controversies in Preventive Cardiology Debate: NCEP ATP III Guidelines have not gone far enough." Pro: Dr. John Robin Crouse. Con: Dr. Beatrice A. Golomb. American Heart Association Council on Epidemiology and Prevention, and American Society for Preventive Cardiology, 42nd Annual Conference on Cardiovascular Disease Epidemiology and Prevention. Honolulu, Hawaii: April 26, 2001.

"Cholesterol and Mood." UCSD Family & Preventive Medicine monthly conference, La Jolla, CA: Jan 15, 2002.

"The Angina Monologues." American Association of University Women: Oct 20, 2001.

"Restoring the risk to risk benefit analysis." Faculty Development series, VA San Diego Healthcare Center, La Jolla, CA: Oct 12, 2001.

- "Syndromes without objective findings." 3<sup>rd</sup> Annual Federal Workers' Compensation Conference, Chicago, IL: April 23, 2001.
- "Cholesterol and Violence." UCSD Medical Scientist Training Program conference series, La Jolla, CA: Aug 6, 2001.
- "Cholesterol." *Rancho Carlsbad Health Fair*, Carlsbad, CA: July 16, 2001.
- "Cholesterol." VA San Diego Healthcare Center morning conference series, La Jolla, CA: June 15, 2001.
- "Cholesterol and you." *Plan for Wellness II*, UCSD sponsored community lecture series, Mission Valley, La Jolla, CA: May 20, 2001.
- "Stroke." *Aging in the New Millenium*, Academic Geriatric Resource Center, UCSD School of Medicine, La Jolla, CA: May 19, 2001.
- "Cardiovascular Disease." *Aging in the New Millenium*, Academic Geriatric Resource Center, UCSD School of Medicine, La Jolla, CA: May 19, 2001.
- "Hyperlipidemia." VASDHS morning conference series, La Jolla, CA: April 18, 2001.
- "Statins and cognitive function." VASDHS morning conference, La Jolla, CA: March 16, 2001.
- "Cholesterol and lipids." lecture to first year UCSD medical students, La Jolla, CA: Dec. 4, 2000.
- "Putting the risk in risk-benefit analysis for cardiovascular disease." invited talk, American Heart Association, Annual Investigators Meeting, Research in Tobacco Related Illness, San Diego: Nov. 30, 2000.
- "Restoring the risk to risk benefit analysis for cardiovascular disease." UC Irvine, Irvine, CA: Oct. 5, 2000.
- "Gulf War Syndrome." UCSD Department of Medicine Grand Rounds, La Jolla, CA: Aug. 9, 2000.
- "Cardiovascular Prevention: Putting the Risk in Risk-Benefit." UCSD course (to international group of physicians), *Topics and Advances in Internal Medicine*, La Jolla, CA: Feb. 11, 2000.
- "Medical Care: Restoring the Risk to Risk-Benefit Analysis." University of Chicago Health Sciences group; Chicago, IL: Feb. 8, 2000.
- "Gulf War Illness." VASDHS Medicine Faculty Development series, La Jolla, CA: June 11, 1999.
- "Aspirin for Primary Prevention of Coronary Artery Disease." Debate vs Dr. Leda Felicio. Dept of Medicine quarterly conference series, "Why Do We Do It?." VA San Diego Healthcare System, La Jolla, CA: March 26, 1999.
- "Cholesterol and Violence: Where do we go from here?" Health Services Research and Development Seminar Series, San Diego VAMC/UC San Diego, La Jolla, CA: Feb. 11, 1999.
- "Cholesterol and Violence." Colloquium, Social Science Research Institute, USC, Los Angeles, CA: Nov. 19, 1998.
- "Cholesterol and Violence." Grand Rounds, Dept of Psychiatry, UC San Diego, La Jolla, CA: Nov 12, 1998.
- Invited speaker and panelist: "How might interdisciplinary models of research guide us to a better understanding of violence?" USC Interdisciplinary Perspectives for Understanding and Preventing Violence, co-sponsored by the Violence Prevention Coalition of Greater Los Angeles, Long Beach, CA: April 17, 1998.
- "Cholesterol and Violence." Dept of Psychology Colloquium, UCSD, La Jolla, CA: April 16, 1998.
- "Measurement and Management of Hyperlipidemia for the Primary Prevention of Coronary Heart Disease." with Dr. Michael H. Criqui. Primary Care Grand Rounds, UCSD Dept of Family and Preventive Medicine, La Jolla, CA: April 15, 1998.
- "The Cholesterol Controversy." VA San Diego Healthcare System quarterly "Why Do We Do It?" seminar, La Jolla, CA: March 29, 1997.
- "Cholesterol and Violence: The Serotonin Connection." Mc Donnell Pew Research Center, Warner Springs Ranch, CA: March 15, 1997.

- "Cholesterol and Violence." USC Dept. of Preventive Medicine, Los Angeles, CA: Feb 21, 1997.
- "The Cholesterol Controversy." San Diego VAMC, La Jolla, CA: Jan 10, 1997.
- "Hyperlipidemia Panel Discussion" Discussants: Alistair Fyfe, MD, PhD; B. Golomb, MD, PhD; David Heber, MD. UCLA Department of Medicine Grand Rounds, Los Angeles, CA: Aug 14, 1996.
- "Cholesterol and Violence: Is There a Connection?" RAND/UCLA Child and Adolescent Health Policy Seminar, RAND, Santa Monica, CA: July 9, 1996.
- "Cholesterol and Violence: The Serotonin Connection." Telluride Summer Research Center Public Lecture, Telluride CO: July 5, 1996.
- "Cholesterol and Violence: New Evidence." UCLA Health Services Research Seminar Series, Los Angeles, CA: June 21, 1996.
- "Cholesterol Reduction: When is it Indicated?" Cardiology Grand Rounds, Cedars Sinai Medical Center: June 18, 1996.
- "Cholesterol, Serotonin, and Violence: Is there a Connection?." The Helmholtz Society, UC Irvine, Irvine, CA: June 11, 1996.
- "Cholesterol Reduction in Primary Prevention: When is it Indicated?" Cedars Sinai Medical Center, Health Services Research Group: March 6, 1996.
- "Cholesterol Reduction: When and Who?" St. Johns Medical Center, Santa Monica, CA: Nov 2, 1995.
- "The Cholesterol Controversy." The Alzheimer Disease Research Consortium of Southern California, USC, Los Angeles, CA: April 21, 1995.
- "Cholesterol Reduction in Primary Prevention." Debate vs Dr. David Leaf. WLA VA Medical Center, Dept. of Medicine Grand Rounds, Los Angeles, CA: March 8, 1995.
- "The Cholesterol Controversy." UCLA Dept. of Medicine Grand Rounds, Los Angeles, CA: Jan 11, 1995.
- "Neural Networks Distinguish Demented Subjects from Elderly Controls based on EEGs." Neural Information Processing Systems conference, Neural Networks in Medicine workshop, Vail, CO: Dec. 3, 1994.
- "Cholesterol Reduction in Primary Prevention: Rethinking the Evidence." UCLA Dept. of Endocrinology Grand Rounds, Los Angeles, CA: Sept. 21, 1994.
- "Advance Directives." West Los Angeles VA Medical Center, Dept. of Medicine Conference, Los Angeles, CA: Sept. 16, 1994.
- "The Cholesterol Myth." Grand Rounds, West Los Angeles V. A. Medical Center, Dept. of Medicine, Los Angeles, CA: June 8, 1994.
- "Death by Hiccup." Reno V.A. Medical Center, Dept. of Medicine Conference, Reno NV: Feb. 1994.
- "Hiccups." V. A. West Los Angeles Medical Center, Dept. of Medicine Conference, Los Angeles, CA: Jan. 1994.
- "Neural Networks that Recognize Sex and Expressions from Faces." in international conference, *Facial Expression: Brain, Perception, and Development*, The Salk Institute, La Jolla, CA: April 6, 1991.
- "SexNet: A neural network that distinguishes sex from face." *Neural Information Processing Systems*, Spotlight presentation, Colorado: 1990.

## PUBLICATIONS

### RESEARCH PAPERS

**Golomb BA**, Koslik HJ, Han JH, Preger Guida AH, Hamilton G, Kelley RI. A Pilot Study of Bioenergetic Marker Relationships in Gulf War Illness: Phosphocreatine Recovery vs. Citric Acid Cycle Intermediates. *International Journal of Environmental Research and Public Health*. 2021; 18(4):1635.  
<https://doi.org/10.3390/ijerph18041635>

**Golomb BA**, Berg BK. Chocolate Consumption and Sex-Interest. *Cureus*. 2021;13(2): e13310.

doi:10.7759/cureus.13310

**Golomb BA**, Nguyen E, Dinkeloo E 2020. Radiation Exposure Predicts Reported Vaccine Adverse Effects in Veterans with Gulf War Illness. *International Journal of Environmental Research and Public Health* 17(19):7136,1-14. <https://www.mdpi.com/1660-4601/17/19/7136>

Magno H, **Golomb BA** 2020. Measuring the Benefits of Mass Vaccination Programs in the United States. *Vaccines* 8(4):561,1-13. <https://www.mdpi.com/2076-393X/8/4/561>

Tat J, Hays B, Teachworth M, Kuo A, Pilz RB, **Golomb BA**, Boss GR 2020. Assessment of High School Students' Participation in Blood Donation and Registration as an Organ Donor. *JAMA Network Open* 3(9):e2016377. <https://jamanetwork.com/journals/jamanetworkopen/fullarticle/2770732>

Naviaux RK, Naviaux JC, Li K, Wang L, Monk JM, Bright AT, Koslik HJ, Ritchie JB, **Golomb BA** 2019. Metabolic features of Gulf War illness. *PLoS ONE* 14(7): e0219531. <https://doi.org/10.1371/journal.pone.0219531>

**Golomb BA**, Koslik H, Christians U, Ritchie J, Wilson P, Elkins N, Klawitter J, Klawitter J, Smith D, Repine J 2019. Depressed prostaglandins and leukotrienes in veterans with Gulf War illness. *J Environ Sci Health, Part B*. <https://www.tandfonline.com/doi/full/10.1080/03601234.2019.1596001>

**Golomb BA** 2018. Diplomats' mystery illness and pulsed radiofrequency/ microwave radiation. *Neural Computation* 30(11): 1-104. [https://www.mitpressjournals.org/doi/abs/10.1162/neco\\_a\\_01133](https://www.mitpressjournals.org/doi/abs/10.1162/neco_a_01133)

**Golomb BA**, Verden A, Messner AK, Koslik HJ, Hoffman, KB 2018. Amyotrophic lateral sclerosis associated with statin use: A disproportionality analysis of the FDA's Adverse Event Reporting System. *Drug Safety* 41(4): 403-13. <http://doi.org/10.1007/s40264-017-0620-4>

Mangin D, Bahat G, **Golomb BA**, Mallery L, Moorhouse P, Onder G, Petrovic M, Garfinkel D 2018. International Group for Reducing Inappropriate Medication Use & Polypharmacy (IGRIMUP). Position Statement and Ten Recommendations for Action. *Drugs and Aging* 35 (7): 575-87. <https://www.ncbi.nlm.nih.gov/pubmed/30006810>

Koslik HJ, Meskimen AH, **Golomb BA** 2017. Physicians' Experiences as Patients with Statin Side Effects: A Case Series. *Drug Safety - Case Reports*. 4 (1):3. <https://link.springer.com/article/10.1007/s40800-017-0045-0>

White RF, Steele L, O'Callaghan JP, Sullivan K, Binns JH, **Golomb BA** et al 2016. Recent research on Gulf War illness and other health problems in veterans of the 1991 Gulf War: Effects of toxicant exposures during deployment. *Cortex*. 74: 449-75. <http://www.sciencedirect.com/science/article/pii/S0010945215003329>

**Golomb BA**, Dimsdale JE, Koslik HJ, Evans MA, Lu X, Rossi S, Mills PJ, White HL, Criqui MH 2015. Statin effects on aggression: Results from the UCSD Statin Study, a randomized controlled trial. *PLoS ONE*. 10 (7): e0124451 <http://www.ncbi.nlm.nih.gov/pubmed/26132393>  
<http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0124451>  
<http://www.plosone.org/article/fetchObject.action?uri=info:doi/10.1371/journal.pone.0124451&representation=PDF>

**Golomb BA**, Bui AK 2015 A fat to forget: Trans fat consumption and memory. *PLoS ONE*. 10 (6): e0128129. <http://www.ncbi.nlm.nih.gov/pubmed/26083739>  
<http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0128129>  
<http://www.plosone.org/article/fetchObject.action?uri=info:doi/10.1371/journal.pone.0128129&representation=PDF>

**Golomb BA**, Koslik HJ, Redd AJ 2015. Fluoroquinolone-induced serious, persistent, multi-symptom adverse effects. *BMJ Case Rep*. 1 Sept 2015. doi:10.1136/bcr-2015-209821  
<http://www.saferpills.org/wp-content/uploads/2014/10/FQ-induced-serious-persistent-multisx-adverse-effects-BMJ-Case-Reports.pdf>

Cham S, Koslik HJ, **Golomb BA** 2015. "Mood, Personality and Behavior Changes During Treatment with Statins: A Case Series". *Drug Safety – Case Reports*. 3(1): 1-13. doi: 10.1007/s40800-015-0024-2  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5005588/>



- Golomb BA**, Allison M, Koperski S, Koslik HJ, Devaraj S, Ritchie JB **2014**. Coenzyme Q10 benefits symptoms in Gulf War veterans: Results of a randomized double-blind study. *Neural Computation* 26(11):2594-4651. <http://www.ncbi.nlm.nih.gov/pubmed/?term=Coenzyme+Q10+Benefits+Symptoms+in+Gulf+War+Veterans%3A+Results+of+a+Randomized+Double-Blind+Study>
- Koslik HJ, Hamilton G, **Golomb BA** **2014**. Mitochondrial dysfunction in Gulf War illness revealed by <sup>31</sup>P-magnetic resonance spectroscopy: A case-control study. *PLoS ONE* 9(3) e92887. doi:10.1371/journal.pone.0092887 <https://journals.plos.org/plosone/article/file?id=10.1371/journal.pone.0092887&type=printable>
- Golomb BA**, Chan VT, Denenberg JO, Koperski S, Criqui MH **2014**. Risk marker associations with venous thromboembolism. A cross-sectional analysis. *BMJ Open* 4(3): e003208. doi:10.1136/bmjopen-2013-003208 <http://www.ncbi.nlm.nih.gov/pubmed/24657882>
- Golomb BA**, Erickson LC, Scott-Van Zeeland AA, Koperski SM, Haas RH, Wallace DC, Naviaux RK, Lincoln AL, Reiner GE, Hamilton G **2014**. Assessing Bioenergetic Compromise in Autism Spectrum Disorder with <sup>31</sup>P-MRS: Preliminary Report. *J Child Neurology* 20(2): 187-93. doi:10.1177/0883073813498466 <https://journals.sagepub.com/doi/abs/10.1177/0883073813498466>
- Erickson LC, Ritchie JB, Javors JM, **Golomb BA** **2013**. Recruiting a special sample with sparse resources: Lessons from a study of Gulf War veterans. *Clinical Trials* 10:481-90; doi: 10.1177/1740774512470040. [Email us for full text on an individual basis](#)
- Golomb BA**, Evans MA, Dimsdale JE, White HL **2012**. "Effects of statins on energy and fatigue with exertion: Results from a randomized controlled trial." *Arch Intern Med* 172 (15): 1180-2. doi:10.1001/archinternmed.2012.2171 <http://archinte.jamanetwork.com/article.aspx?doi=10.1001/archinternmed.2012.2171>
- Golomb BA**, Koperski S, White HL **2012**. "Association between more frequent chocolate consumption and lower body mass index." *Arch Intern Med* 172: 519-21. doi: 10.1001/archinternmed.2011.2100 <http://archinte.jamanetwork.com/article.aspx?articleid=1108800>
- Golomb BA**, Evans MA, White HL, Dimsdale JE **2012**. "Trans fat consumption and aggression." *PLoS ONE* 7(3): e32175; doi: 10.1371/journal.pone.0032175 <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0032175>
- Golomb BA**, Chan VT, Evans MA, Koperski S, White HL, Criqui MH **2012**. The older the better: Are elderly study participants more nonrepresentative? Analysis of observational and clinical trial samples. *BMJ Open* 2: e000833. doi:10.1136/bmjopen-2012-000833; <http://bmjopen.bmj.com/cgi/content/full/bmjopen-2012-000833>.
- Hoffman KB, Kraus C, Dimbil M, **Golomb BA** **2012**. A survey of the FDA's AERS database regarding muscle and tendon adverse events linked to the statin drug class. *PLoS ONE* 7(8): e42866. doi:10.1371/journal.pone.0042866 <http://www.plosone.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.0042866&representation=PDF>
- Erickson LC, Scott-Van Zeeland AA, Hamilton G, Lincoln A, **Golomb BA** **2012**. "Approaches to (31)P-MRS in Awake, Non-Sedated Children with and without Autism Spectrum Disorder." *J Autism Dev Disord* 42:1120-6. doi: 10.1007/s10803-011-1359-x <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3668346/>
- Reilly D, Cham S, **Golomb BA** **2011**. "First degree relatives with behavioural adverse effects on statins." *BMJ Case Reports*. doi:10.1136/bcr.09.2011.4758. [Email us for full text on an individual basis](#)
- Golomb BA**, Erickson LC, Koperski S, Sack D, Enkin M, Howick J **2010**. "What's in placebos: Who knows?" Analysis of randomized controlled trials." *Ann Intern Med* 153: 532-35. doi: 10.1059/0003-4819-153-8-201010190-00010 [Email us for full text on an individual basis](#)
- Rose N, Koperski S, **Golomb BA** **2010**. "Mood food: chocolate and depressive symptoms in a cross-sectional analysis." *Arch Intern Med* 170(8):699-703. doi:10.1001/archinternmed.2010.78 <http://archinte.jamanetwork.com/article.aspx?articleid=415834>

- Golomb BA**, Yaghmai R, Renvall MJ, Ramsdell JW 2010. "Electronic medical records and upper extremity symptoms: pain with the gain?" *Arch Intern Med* 170(7):655-657. doi: 10.1001/archinternmed.2010.55 <http://archinte.jamanetwork.com/article.aspx?articleid=486868>
- Cham S, Evans MA, Denenberg JO, **Golomb BA** 2010. "Statin-associated muscle-related adverse effects: a case series of 354 patients." *Pharmacotherapy* 30(6): 541-553. doi:10.1592/phco.30.6.541 <http://www.medscape.com/viewarticle/724842>
- MacGregor AJ, Shaffer RA, Dougherty AL, Galarneau MR, Raman R, Baker DG, Lindsay SP, **Golomb BA**, Corson KS 2010. "Prevalence and psychological correlates of traumatic brain injury in Operation Iraqi Freedom." *J Head Trauma Rehabil* 25(1):1-8. doi: 10.1097/HTR.0b013e3181c2993d <http://www.dtic.mil/dtic/tr/fulltext/u2/a541876.pdf>
- Cham S, Gill K, Koperski S, **Golomb BA** 2009. "Improvement in sleep apnoea with switch from simvastatin to pravastatin." *BMJ Case Reports*; doi:10.1136/bcr.05.2009.1875 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3029510/>
- MacGregor AJ, Corson KS, Larson GE, Shaffer RA, Dougherty AL, Galarneau MR, Raman R, Baker DG, Lindsay SP, **Golomb BA** 2009. "Injury-specific predictors of posttraumatic stress disorder." *Injury* 40(9):1004-10; doi: 10.1016/j.injury.2009.04.006 [http://www.researchgate.net/publication/26290686\\_Injury-specific\\_predictors\\_of\\_posttraumatic\\_stress\\_disorder/file/9fcfd511d493aded5f.pdf](http://www.researchgate.net/publication/26290686_Injury-specific_predictors_of_posttraumatic_stress_disorder/file/9fcfd511d493aded5f.pdf)
- Linares L, **Golomb BA**, Jaojoco J, Sikand H, Phillips PS 2009. "The Modern Spectrum of Rhabdomyolysis: Drug Toxicity Revealed by Creatine Kinase Screening." *Current Drug Safety* Sept 1. PMID: 19534642 E-pub ahead of print; doi: 10.2174/157488609789007010. [Email us for full text on an individual basis](#)
- Golomb BA**, Kwon E, Koperski SM, Evans MA 2009. "Amyotrophic lateral sclerosis-like conditions arising in possible association with cholesterol-lowering drugs." *Drug Safety* 32(8):649-651. doi: 10.2165/00002018-200932080-00004 [Email us for full text on an individual basis](#)
- Evans MA, **Golomb BA** 2009. "Statin associated cognitive problems reported by 171 subjects." *Pharmacotherapy* 29(7): 800-111. doi:10.1592/phco.29.7.800 [Email us for full text on an individual basis](#)
- MacGregor AJ, Shaffer R, Wade A, Galarneau M, Raman R, Baker D, Lindsay S, **Golomb BA**, Corson K 2009. "Psychological correlates of battle and nonbattle injury among Operation Iraqi Freedom veterans: Results from the Navy-Marine Corps Combat Trauma Registry." *Military Medicine* 174(3): 224-231. doi: 10.7205/MILMED-D-03-9107 <http://publications.amsus.org/doi/pdf/10.7205/MILMED-D-03-9107>
- Golomb BA**, Evans MA 2008. "Statin adverse effects: a review of the literature and evidence for a mitochondrial mechanism." *Am J Cardiovasc Drugs* 8:373-418. doi: 10.2165/0129784-200808060-00004 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2849981/>
- Golomb BA** 2008. "Acetylcholinesterase Inhibitors and Gulf War Illnesses." *Proceedings of the National Academy of Science* 105(11): 4295-4300. doi: 10.1073/pnas.0711986105 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2393741/>
- Golomb BA**, Dimsdale JE, White HL, Ritchie JB, Criqui MH 2008. "Reductions in blood pressure with statins: Results from the UCSD Statin Study, a randomized trial." *Archives of Internal Medicine* 168(7): 721-727. doi: 10.1001/archinte.168.7.721 <http://archinte.jamanetwork.com/article.aspx?articleid=414137>
- Golomb BA**, McGraw JJ, Evans, MA, Dimsdale, JE 2007. "Physician Response to Patient Reports of Drug Adverse Effects: Implications for Patient-Targeted Adverse Effect Surveillance." *Drug Safety* 30(8): 669-675. [Email us for full text on an individual basis](#)
- Golomb BA**, Cortez-Perez M, Jaworski BA, Mednick SA, Dimsdale, JE 2007. "Point subtraction aggression paradigm: validity of a brief schedule of use." *Violence & Victims* 22(1): 95-103. doi:10.1891/088667007780482829 [Email us for full text on an individual basis](#)
- Golomb BA**, Dang T, Criqui MH 2006. "Peripheral arterial disease: morbidity and mortality implications." *Circulation* 114: 688-699. doi: 10.1161/CIRCULATIONAHA.105.593442 <http://circ.ahajournals.org/content/114/7/688.long>

- Wang JC, Criqui MH, Denenberg JO, McDermott MM, **Golomb BA**, Fronck A 2005 "Exertional leg pain in patients with and without peripheral arterial disease." *Circulation* 112: 3501-8. doi: 10.1161/CIRCULATIONAHA.105.548099  
<http://circ.ahajournals.org/cgi/pmidlookup?view=long&pmid=16316971>
- Golomb BA**, Kane T, Dimsdale JE, 2004. "Severe irritability associated with statin cholesterol-lowering drugs." *Quarterly J Med* 97:229-235. doi: 10.1093/qjmed/hch035  
<http://qjmed.oxfordjournals.org/content/97/4/229.long>
- Golomb BA**, Criqui MH, White HL, Dimsdale JE 2004. "Study Design. The UCSD Statin Study: A randomized controlled trial assessing the impact of statins on noncardiac endpoints." *Controlled Clinical Trials* 25:178-202. doi: 10.1016/j.cct.2003.08.014  
<http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.5.4537&rep=rep1&type=pdf>
- Smythies J, **Golomb BA**, 2004. "Nerve gas antidotes." *Journal of the Royal Society of Medicine* 97:32. doi: 10.1258/jrsm.97.1.32 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1079267/>
- Golomb BA**, Criqui MH, White HL, Dimsdale JE 2004. "Conceptual Foundations of the UCSD Statin Study: A Randomized Controlled Trial Assessing the Impact of Statins on Cognition, Behavior, and Biochemistry." *Arch Intern Med* 164:153-162. doi: 10.1001/archinte.164.2.153  
<https://jamanetwork.com/journals/jamainternalmedicine/fullarticle/216553>
- Criqui MH, Jamosmos M, Froniek A, Denenberg JO, Langer RD, Bergan J, **Golomb BA** 2003 "Chronic venous disease in an ethnically diverse population: The San Diego Population Study." *Am J Epidemiol* 158:448-456. doi: 10.1093/aje/kwg166 <http://aje.oxfordjournals.org/cgi/pmidlookup?view=long&pmid=12936900>
- Golomb BA**, Tenkanen, L, Alikoski T, Niskanen T, Manninen V, Huttunen M, Mednick SA 2002. "Insulin sensitivity markers: Predictors of accidents and suicides in Helsinki Heart Study screenees." *Journal of Clinical Epidemiology* 55:1-7. doi: 10.1016/S0895-4356(02)00407-9  
[Email us for full text on an individual basis](#)
- Bagley SC, White HL, **Golomb BA** 2001. "Logistic regression in the medical literature: Standard for use and reporting, with particular attention to one medical domain." *Journal of Clinical Epidemiology* 54(10):979-85. doi: 10.1016/S0895-4356(01)00372-9 [http://www.aliquote.org/cours/2012\\_biomed/biblio/Bagley2001.pdf](http://www.aliquote.org/cours/2012_biomed/biblio/Bagley2001.pdf)
- Golomb BA**, Vickrey G, & Hays RD 2001. "A review of health-related quality-of-life measures in stroke." *Pharmacoeconomics* 19: 155-185. doi: 10.2165/00019053-200119020-00004  
[Email us for full text on an individual basis](#)
- Golomb BA**, Stattin H, Mednick SA 2000. "Low cholesterol and violent crime." *J Psychiatric Res* 34: 301-309. doi: 10.1016/S0022-3956(00)00024-8 [Email us for full text on an individual basis](#)
- Golomb BA**, Pyne JM, Jaworski BA, Wright B, Lohr JM, Bozzette SA 2000. "The Role of Psychiatrists in Primary Care of Patients With Severe Mental Illness" *Psychiatric Services* 51(6): 766-773. doi: 10.1176/appi.ps.51.6.766 <http://ps.psychiatryonline.org/article.aspx?articleid=84566>
- Golomb BA**, Criqui MH, Detrano RC, Wong ND, Bundens WP, Mattrey RF, Denenberg JO 1999. "Noninvasive testing to detect subclinical cardiovascular disease. What is its role?" *Preventive Cardiology* 2(4):42-50 (Fall supplement). [Email us for full text on an individual basis](#)
- Golomb BA** 1998. "Cholesterol and violence: Is there a connection?" *Annals of Internal Medicine* 128:478-487. doi: 10.7326/0003-4819-128-6-199803150-00009 [Email us for full text on an individual basis](#)
- Criqui MH, **Golomb BA** 1998. "Epidemiologic aspects of lipid abnormalities." *American Journal of Medicine* 105 (1A): 48S-57S. doi: 10.1016/S0002-9343(98)00212-5 [Email us for full text on an individual basis](#)
- Stewart-Bartlett M, Viola PA, Sejnowski TJ, **Golomb BA**, Larsen J, Hager JC and Ekman P 1996. "Classifying facial action." *Advances in Neural Information Processing Systems* 8: 823-829.  
<http://www.paulekman.com/wp-content/uploads/2013/07/Classifying-Facial-Action.pdf>
- Gray MS, Lawrence DT, **Golomb BA** and Sejnowski TJ 1995. "A perceptron reveals the face of sex." *Neural Computation* 7(6):1160-64. doi: 10.1162/neco.1995.7.6.1160



- Golomb BA**, Lawrence DT, and Sejnowski TJ 1991. "SexNet: A neural network that recognizes sex from human faces." *Advances in Neural Information Processing Systems* 3: 572-577.  
[http://papers.cnl.salk.edu/PDFs/Sexnet\\_%20A%20Neural%20Network%20Identifies%20Sex%20from%20Human%20Faces%201991-3594.pdf](http://papers.cnl.salk.edu/PDFs/Sexnet_%20A%20Neural%20Network%20Identifies%20Sex%20from%20Human%20Faces%201991-3594.pdf)
- Golomb BA**, Andersen RA, Nakayama K, MacLeod DIA, Wong A 1985. "Visual thresholds for shearing motion in monkey and man." *Vision Research* 25: 813-820. doi: 10.1016/0042-6989(85)90189-0  
<http://psy2.ucsd.edu/~dmacleod/publications/34GolombAndersonEtal1985.pdf>

### **EDITORIALS, INVITED PAPERS, OTHER**

- Golomb SW, Golomb BA** 2018. "A Career in Engineering." *IEEE Transactions on Information Theory* 64(4):2805-2838, invited article for a Special Issue in honor of my father, Solomon W. Golomb
- Golomb BA** 2017 "Effect Modification." Invited contribution to Edge.org, *What Scientific Term or Concept Ought to be More Widely Known?* <https://www.edge.org/response-detail/27223>
- Golomb BA** 2016 Gulf War illness Op-Ed (on Veterans day). "Renowned Gulf War Illness Researcher Urges Americans: Don't Forget the Men and Women of the "Forgotten War" on Veterans Day. *The Reno Dispatch*.  
<http://therenodispatch.blogspot.com/2016/11>.
- Golomb BA** 2015 "Will we recognize it when it happens?" Invited contribution to Edge.org, *What Do You Think About Machines that Think?* <https://edge.org/response-detail/26226>
- Golomb BA** 2014 "Statins linked to increased risk of diabetes: High potency agents look worse, though none are exempt. *BMJ*. (Invited Editorial) *Declared "excellent," accepted, proofs received -- then BMJ decided not to publish citing the controversy about Abramson and Malhotra papers in BMJ – BMJ was laboring under pressure to retract these two papers unflattering to statins, though they in no way met COPES criteria for retraction - exerted by Rory Collins (industry "3<sup>rd</sup> party partner" who ran the £96million Oxford Merck PCSK9 inhibitor study– and who later, as I understand, tried to get the BMJ editor fired for not acquiescing to his demands to retract).*
- Golomb BA** 2014 "Statins and activity: Proceed with caution" *Jama Internal Medicine*. 174:1270-2.  
<http://www.ncbi.nlm.nih.gov/pubmed/24912133>  
<http://archinte.jamanetwork.com/article.aspx?articleid=1878466>
- Golomb BA** 2014 "Psychogenic Illness" Invited contribution to Edge.org *What Scientific Idea is Ready for Retirement* <http://edge.org/response-detail/25334> (Invited Essay) - see also book chapters 2015
- Golomb BA** 2013 "The importance of monitoring adverse events in statins, and other, clinical trials." *Clinical Investigation* 3(10): 913-6. <http://www.future-science.com/doi/pdf/10.4155/cli.13.81> (Invited Editorial)
- Perlmutter D, **Golomb B**, Sinatra S, Campbell A.W. 2013. Appropriate clinical use of statins: a discussion of the evidence, scope, benefits, and risk. *Altern Ther Health Med* 19 Suppl 1. 14-25  
<http://www.drperlmutter.com/wp-content/uploads/2014/02/Appropriate-Clinical-Use-of-Statins.pdf> (Invited Roundtable)
- Golomb BA** 2012 "The Epidemic of Obesity, Diabetes and "Metabolic Syndrome: Cell Energy Adaptations in a Toxic World?" *What is your favorite deep, elegant or beautiful explanation?* Invited essay, Edge.org, <http://edge.org/response-detail/10515>. (Invited Essay)
- Golomb BA** 2012. "Oxidative stress and mitochondrial injury in chronic multisymptom conditions: From Gulf War illness to autism spectrum disorder: Available from *Nature Precedings*  
<http://hdl.handle.net/10101/npre.2012.6847.1>



- Golomb BA** 2011. "Do statins reduce the risk of infection? Observational evidence is now refuted by randomised trials." *BMJ* 343:d7134 doi: 10.1136/bmj.d7134. (Invited Editorial)  
<http://www.bmj.com/content/343/bmj.d7134.full.pdf+html>
- Golomb BA** 2011. "Are placebos inert or powerful? Vice versa." *Clinical Investigation* 1(11) 1471-3. doi: 10.4155/CLI.11.142 <http://www.future-science.com/doi/pdf/10.4155/cli.11.142> (Invited Editorial)
- Golomb BA** 2011. "The Dece(i)bo Effect." Invited contribution to Edge.org "What scientific concept would improve everybody's cognitive toolkit?" <http://edge.org/response-detail/11708> (Invited Essay)
- Golomb BA** 2011. "The starving cell: Metabolic syndrome as an adaptive process." *Nature Precedings*  
<http://precedings.nature.com/documents/6535/version/1>.
- Golomb BA, Koperski S** 2010. "Pondering the ponderous: Are the 'moral challenges' of bariatric surgery morally challenged?" *American Journal of Bioethics* 10 (12): 24-6. doi: 10.1080/15265161.2010.528522 (Invited Editorial) [Email us for full text on an individual basis](#)
- Golomb BA** 2009. "Doctoring the Evidence: The case against lying to patients about placebos." *American Journal of Bioethics*, 9:34-6. (Invited Editorial) [Email us for full text on an individual basis](#)
- Golomb BA** 2009. "Control theory: Placebo-controlled drug trials have problems. Active-controlled drug trials are not always the solution." *American Journal of Bioethics*, 9 (9):67-69. doi: 10.1080/15265160903098424 (Invited Editorial) [Email us for full text on an individual basis](#)
- Golomb BA** 2009. "Metabolic syndrome: Intima medial thickness and beyond." *J Am College Cardiol* 53: 2280-2. doi: 10.1016/j.jacc.2009.03.029 <http://content.onlinejacc.org/article.aspx?articleid=1139790> (Invited Editorial)
- Golomb BA, Parrish J, Broadwin JA** 2009. "Statins and Mortality." *On the Risk* 25: 66-71. <http://www.health-heart.org/GolombStatinAndMortality2009.pdf> (Invited article for Insurance Industry journal)
- Criqui MH, Golomb BA** 2008. "Lipid lowering: what and when to monitor." *Lancet* 2008;372(9638):516-7. doi: 10.1016/S0140-6736(08)61213-1 (Invited Editorial) [Email us for full text on an individual basis](#)
- Golomb BA** 2007. "Reasoning from evidence. A call for education." Invited contribution to: Edge.org "What have you changed your mind about?" <http://edge.org/response-detail/10203>
- Golomb BA** 2007. "Odile reminiscences." *Odile Crick Memorial Exhibition*, Edited by Becky Cohen, published by Salk Institute, p 23-24, 45.  
<http://www.amazon.com/Odile-Crick-Becky-curator-Cohen/dp/B00CC5KJCU>;  
<http://roger.ucsd.edu/search/?searchscope=9&searchtype=o&searcharg=180703216>
- Golomb BA** 2006. "Reforming scientific and medical publishing via the internet." Invited contribution to: Edge.org "What are you optimistic about?"  
(Comment: Mine was the first among the six Edge contributions (out of 158 contributions) to be highlighted on *Nature's* website – Nature.com.)  
Abbreviated version: [http://edge.org/q2007/q07\\_15.html#golomb](http://edge.org/q2007/q07_15.html#golomb)  
See also Book Chapters (abbreviated version later published)
- Golomb BA** 2005. "Statins and Blood Pressure a Probable Link?" <http://www.audiomedica.com/?p=52>. *Audio Journal of Cardiovascular Medicine*, 11:1.
- Golomb BA** 2005. "Implications of statin adverse events in the elderly." *Expert Opinion on Drug Safety* 4(3):389-397. doi: 10.1517/14740338.4.3.389 [Email us for full text on an individual basis](#)
- Criqui MH and Golomb BA** 2004. "Low and lowered cholesterol and total mortality." *J Am Coll Cardiol* 44(5): 1009-10. doi: 10.1016/j.jacc.2004.06.022 <http://content.onlinejacc.org/article.aspx?articleid=1135936>
- Golomb BA** 2004. "Statin Adverse Effects. Implications for the Elderly." *Geriatric Times* May/June, 18-20.  
<http://www.antibioticfailure.com/kb/alerts/drugs/statins.htm>
- Criqui MH and Golomb BA** 1999. "Should patients with diabetes drink to their health?" *Journal of the American Medical Association* 282 (3):279-80. doi: 10.1001/jama.282.3.279  
[Email us for full text on an individual basis](#)

**Golomb B** and Criqui M 1999. "Anti-hypertensives: Much ado about lipids." *Archives of Internal Medicine*, 159: 535-537. doi: 10.1001/archinte.159.6.535 [Email us for full text on an individual basis](#)

**Golomb BA** 1998. "Dietary fats and heart disease: dogma challenged." *Journal of Clinical Epidemiology* 51(6):461-4. [Email us for full text on an individual basis](#)

**Golomb BA** 1995. "Are placebos bearing false witness?" *Chemistry and Industry* 21: 900.  
[Email us for full text on an individual basis](#)

## **BOOKS**

**Golomb BA**. *A Review of the Scientific Literature as it Pertains to Gulf War Illnesses, Vol 3: Immunizations* RAND. Unreleased.

Cecchine G, Johnson D, Perry W, Anthony CR, **Golomb BA**, Hearn AC, Hilborne L, Sollinger J 2001. *Army Medical Support to the Army After Next*. RAND. 80 pages. [Ebook downloadable free from: \[http://www.rand.org/content/dam/rand/pubs/monograph\\\_reports/2007/MR1270.pdf\]\(http://www.rand.org/content/dam/rand/pubs/monograph\_reports/2007/MR1270.pdf\)](#)

Hilborne LH, **Golomb BA** 2001. *A Review of the Scientific Literature as it Pertains to Gulf War Illnesses, Vol 1: Infectious Diseases*. RAND, Santa Monica, CA. 119 pages. Peer reviewed book. [Ebook version downloadable free in chapters from: \[http://www.rand.org/pubs/monograph\\\_reports/MR1018z1.html\]\(http://www.rand.org/pubs/monograph\_reports/MR1018z1.html\)](#)

Cecchine G, **Golomb BA**, Hilborne LH, Spektor DM, Anthony RA 2000. *A Review of the Scientific Literature as it Pertains to Gulf War Illnesses, Vol 8: Pesticides* RAND, Santa Monica. 182 pages. Peer reviewed book. [Ebook version downloadable free in chapters from: \[http://www.rand.org/pubs/monograph\\\_reports/MR1018z8.html\]\(http://www.rand.org/pubs/monograph\_reports/MR1018z8.html\)](#)

**Golomb BA** 1999. *A Review of the Scientific Literature as it Pertains to Gulf War Illnesses, Vol 2: Pyridostigmine Bromide*. Washington, DC, RAND. 385 pages. Peer reviewed book. (Among top 10 best selling RAND books in 2000 list). [Ebook version downloadable free in chapters from: \[http://www.rand.org/pubs/monograph\\\_reports/MR1018z2.html\]\(http://www.rand.org/pubs/monograph\_reports/MR1018z2.html\)](#)

Executive summary: [http://www.rand.org/content/dam/rand/pubs/monograph\\_reports/2005/MR1018.2-1.pdf](http://www.rand.org/content/dam/rand/pubs/monograph_reports/2005/MR1018.2-1.pdf)

## **BOOK CHAPTERS**

**Golomb BA** 2017. "Effect Modification." In: John Brockman (Ed.) *This Idea is Brilliant: Lost, Overlooked, and Underappreciated Concepts Everyone Should Know*. Harper Perennial, New York, Pp 440-445  
<https://www.harpercollins.com/9780062698216/this-idea-is-brilliant/>

**Golomb BA** 2015. "Psychogenic illness." In: John Brockman (Ed.) *This Idea Must Die: Scientific Theories that are Blocking Progress* Harper Perennial, New York. Pp 511-514.  
<http://www.harpercollins.com/9780062374349/this-idea-must-die>

**Golomb BA** 2013. "Metabolic syndrome: Cell energy adaptations in a toxic world?". In: John Brockman (Ed.) *This Explains Everything. Deep, Beautiful, and Elegant Theories of How the World Works*. Harper Perennial, New York. p 359-63. <http://www.amazon.com/This-Explains-Everything-Beautiful-Theories/dp/0062230174>

Millen M, **Golomb BA** 2012. "Chocolate and mood." (Chapter 30) In: Dr. Ronald Watson et al. (Eds.) *Chocolate in Health and Nutrition*, in series Nutrition and Health (Adrienne Bendich, Series Editor), Humana Press Springer, pp 409-419. [http://www.springer.com/new+%26+forthcoming+titles+\(default\)/book/978-1-61779-802-3](http://www.springer.com/new+%26+forthcoming+titles+(default)/book/978-1-61779-802-3)

**Golomb BA** 2012. "The Dece(i)bo Effect." In: John Brockman (Ed.) *This Will Make You Smarter: New Scientific Concepts to Improve your Thinking*, Harper Perennial. pp 381-5. <http://www.amazon.com/This-Will-Change-Everything-Future/dp/0061899674>

**Golomb BA**, Criqui MH 2008. "Epidemiology of PAD." Chapter 1 In: MA Creager (Ed.), *Peripheral Arterial Disease* (2<sup>nd</sup> Edition). ReMedica Publishing, London. pp 1-22.

- Golomb BA** 2007. “Reforming Scientific and Medical Publishing Via the Internet.” In: *What Are You Optimistic About? Today's Leading Thinkers on Why Things Are Good and Getting Better*, John Brockman (Ed.), Harper Collins, New York. pp 346-350. <http://www.amazon.com/What-Are-You-Optimistic-About/dp/0061436933>
- Golomb BA**, Criqui MH, Bundens WP 2001. “Epidemiology of peripheral arterial disease.” In: MA Creager (Ed.), *Management of Peripheral Arterial Disease: Medical, Surgical and Interventional Aspects*. ReMedica Publishing, London. pp 1-18. <https://www.google.com/webhp?ssrp=1#q=peripheral+arterial+disease+-+Creager&tbm=shop&spd=7022393619389752917>
- Golomb BA**, Criqui MH, Bundens WP 2001. “Peripheral arterial disease.” In Hiatt, W.R., Hirsch, A.T., Regensteiner, J. (Eds.), *Peripheral Arterial Disease Handbook*. CRC Press, Boca Raton, pp. 57-80. <http://www.crcpress.com/product/isbn/9780849384134>
- Golomb BA** 2000. “Health and Medical Factors: Cholesterol.” In: *Violence in America - An Encyclopedia*, Ronald Gottesman, Ed; Charles Scribner’s Sons. [http://www.amazon.com/Violence-America-Encyclopedia-Three-Volume/product-reviews/0684804875/ref=dp\\_top\\_cm\\_cr\\_acr\\_txt?showViewpoints=1](http://www.amazon.com/Violence-America-Encyclopedia-Three-Volume/product-reviews/0684804875/ref=dp_top_cm_cr_acr_txt?showViewpoints=1)
- Golomb BA** 2000. “Cerebrovascular disease.” In Kerr EA, Asch SM, Hamilton EG, McGlynn EA (Eds.) *Quality of Care for Cardiopulmonary Conditions: A Review of the Literature and Quality Indicators*. RAND Health, Santa Monica: pp 69-90. [http://www.rand.org/content/dam/rand/pubs/monograph\\_reports/MR1282/mr1282.ch3.pdf](http://www.rand.org/content/dam/rand/pubs/monograph_reports/MR1282/mr1282.ch3.pdf)
- Golomb BA** 2000. “Chronic obstructive pulmonary disease.” In Kerr EA, Asch SM, Hamilton EG, McGlynn EA (Eds.) *Quality of Care for Cardiopulmonary Conditions: A Review of the Literature and Quality Indicators*. RAND Health, Santa Monica: pp 91-116. [http://www.rand.org/content/dam/rand/pubs/monograph\\_reports/MR1282/mr1282.ch4.pdf](http://www.rand.org/content/dam/rand/pubs/monograph_reports/MR1282/mr1282.ch4.pdf)
- Golomb BA** 2000. “Hyperlipidemia.” In Kerr EA, Asch SM, Hamilton EG, McGlynn EA (Eds.) *Quality of Care for Cardiopulmonary Conditions: A Review of the Literature and Quality Indicators*. RAND Health, Santa Monica: pp 201-216. [http://www.rand.org/content/dam/rand/pubs/monograph\\_reports/MR1282/mr1282.ch10.pdf](http://www.rand.org/content/dam/rand/pubs/monograph_reports/MR1282/mr1282.ch10.pdf)
- Golomb BA** 2000. “Dyspepsia and Peptic ulcer disease.” In Kerr EA, Asch SM, Hamilton EG, McGlynn EA (Eds.) *Quality of Care for General Medical Conditions A Review of the Literature and Quality Indicators*. RAND Health: pp 263-290. [http://www.rand.org/content/dam/rand/pubs/monograph\\_reports/MR1280/mr1280.ch18.pdf](http://www.rand.org/content/dam/rand/pubs/monograph_reports/MR1280/mr1280.ch18.pdf)
- Golomb BA** and Sejnowski TJ 1995. “Sex recognition from faces using neural networks.” In: AF Murray (Ed.), *Applications of Neural Networks*, Kluwer Academic Publishers. <https://papers.cnl.salk.edu/PDFs/Sex%20Recognition%20from%20Faces%20Using%20Neural%20Networks%201995-3416.pdf>

#### **TECHNICAL REPORTS, RAND REPORTS, GOVERNMENT REPORTS (see also Books)**

- Binns JH, Bloom FE, Bunker JA, Crawford F, **Golomb BA** et al 2014. Gulf War Illness and the Health of Gulf War Veterans: Research Update and Recommendations, 2009-2013 Washington, D.C.: US Government Printing Office;
- Binns JH, Barlow C, Bloom FE, Clauw DJ, **Golomb BA**, et al. 2008. Gulf War Illness and the Health of Gulf War Veterans. Scientific Findings and Recommendations. Research Advisory Committee on Gulf War Veterans Illnesses. US Government Printing Office, Washington, D.C. 454 pages. November 2008. [http://www.va.gov/rac-gwvi/docs/committee\\_documents/gwiandhealthofgwveterans\\_rac-gwvireport\\_2008.pdf](http://www.va.gov/rac-gwvi/docs/committee_documents/gwiandhealthofgwveterans_rac-gwvireport_2008.pdf)
- Binns JH, Cherry N, **Golomb BA**, et al 2004. Research Advisory Committee on Gulf War Veterans' Illnesses: Scientific Progress in Understanding Gulf War Veterans' Illnesses: Report and Recommendations. September 2004. [http://www.va.gov/rac-gwvi/docs/committee\\_documents/reportandrecommendations\\_scientificprogressinunderstandinggwvi\\_2004.pdf](http://www.va.gov/rac-gwvi/docs/committee_documents/reportandrecommendations_scientificprogressinunderstandinggwvi_2004.pdf)



**Golomb BA** 2003. *Research Recommendations*. Written brief for Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses.

**Golomb BA**, Pickett D 2003. *Surveillance for birth defects in ill Gulf War veterans*. Written brief for Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses.

**Golomb BA**. 2002. *Acetylcholinesterase inhibitor effects: Neurological and nonneurological mechanisms*. Written brief for Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses.

**Golomb BA**. 2002. *Mitochondrial dysfunction. A mechanism of illness in Gulf War veterans?* Written brief for Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses.

**Golomb BA**, Chadwick A. 2002. *Treatment considerations for ill Gulf War veterans*. Written brief for Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses.

**Golomb BA**, Haley R 2002. *Objective Markers: Means to Identify Mechanisms and Treatments in Gulf War veterans*. Written brief for Dept of Veterans Affairs Research Advisory Committee on Gulf War Illnesses.

**Golomb BA** 1999. *Assessing the Care of Vulnerable Elders. Potential Quality Indicators and Literature Review: Stroke*. RAND report. 113 pages.

**Golomb BA** 1999 Quality Indicators for The Management Of Stroke And Atrial Fibrillation For Vulnerable Older Persons. (The final quality indicators were subjected to an Expert Panel process, so do not match the evidence)

[http://www.rand.org/content/dam/rand/www/external/health/projects/acove/docs/acove\\_gistroke.pdf](http://www.rand.org/content/dam/rand/www/external/health/projects/acove/docs/acove_gistroke.pdf)

Perry W, Johnson D, Cecchine G, **Golomb BA**, Hilborn L, Hearn A, Anthony R 1999. *Issues and Insights from the Army Medical Technology Workshop, 1999*. Prepared for the US Army Training and Doctrine Command and the US Army Medical Department. RAND report PM-946-A, July 1999.

Hays RD, Vickrey BG, **Golomb BA** 1997. "Health related quality of life measures in studies of stroke." RAND report, July 1997.

Sherbourne CD, **Golomb BA**, Bystritsky A, Inkeles M, Marshall G. "Summary of Health-related Quality of Life Measures for Use in Anxiety Disorders." RAND report, July 1997.

Gray MS, Lawrence DT, **Golomb BA** and Sejnowski TJ 1993. "A perceptron reveals the face of sex." *Institute for Neural Computation Technical Report Series*, INC-9303.

<http://papers.cnl.salk.edu/PDFs/A%20Perceptron%20Reveals%20the%20Face%20of%20Sex%201993-717.pdf>

## **LETTERS TO THE EDITOR**

**Golomb BA** 2015. "Misinterpretation of trial evidence on statin adverse effects may harm patients." *Eur J Prev Cardiol* 22 (4): 492-3. <http://www.ncbi.nlm.nih.gov/pubmed/24770566>

**Golomb BA**, Brenner S, Chalfie M, Glashow SL, Glauber RJ, Greengard, P, Gross DJ, Hubel DH, Maskin ES, Roberts RJ, Tonegawa S, Wilczek FA, Brown EM, Sejnowski TJ 2013. "Chocolate habits of Nobel prizewinners." *Nature* 499(7459):409. doi: 10.1038/499409a (**11 Nobelist Coauthors**). See: <http://papers.cnl.salk.edu/PDFs/Chocolate%20habits%20of%20Nobel%20prizewinners%202013-4343.pdf>

**Golomb BA**, Koperski S 2013. "New statins also produce fatigue: spontaneous reporting as a complementary system to increase safety knowledge-reply" *JAMA-Int Med* 173(3): 247-8. doi: 10.1001/jamainternmed.2013.2113 [Email us for full text on an individual basis](#)

**Golomb BA** 2012. "Too sweet to be real?" *Arch Intern Med* 172 (16): 1270. doi: 10.1001/archinternmed.2012.3388 [Email us for full text on an individual basis](#)

**Golomb BA**, Koperski S, Rose, N 2010. "Chocolate consumption and effects on serotonin synthesis" (aka "Confection in coinfection"). *Arch Intern Med* 170(17): 1608-9 doi: 10.1001/archinternmed.2010.332 [Email us for full text on an individual basis](#)

**Golomb BA**, Koperski S, Evans MA 2010. "Statin adverse effects. A complementary perspective." *Drug Safety* 33(9): 803-4. doi: 10.2165/11538820-000000000-00000 [Email us for full text on an individual basis](#)

- Golomb BA**, Koperski S 2009. "Association Not Causation." *Arch Intern Med* 169:1079. doi: 10.1001/archinternmed.2009.156 [Email us for full text on an individual basis](#)
- Golomb BA**, Aranoff-Spencer E, Steadman MC, Wu W, Yan A 2009. "A ray of sunshine for the Vitamin D Heart hypothesis." *Arch Intern Med* 169(4): 416-7. doi: 10.1001/archinternmed.2008.607 [Email us for full text on an individual basis](#)
- Golomb BA**, Evans MA 2008. "Re: Statin therapy is associated with reduced neuropathologic changes of Alzheimer disease." *Neurology* 70(24): 2349-50. doi: 10.1212/01.wnl.0000317006.87071.b1 [Email us for full text on an individual basis](#)
- Golomb BA** 2008. Correction for Golomb, "Reply to Blazer et al.: Flawed challenges to 'Acetylcholinesterase inhibitors and Gulf War illnesses'." *Proc Natl Acad Sci U S A* 105(47): E94. doi: 10.1073/pnas.0809123105 (This was a correction of *their* error – the journal had made a change after the galleys were approved, that they later corrected.) <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2587534/>
- Golomb BA** 2008. "Reply to Blazer et al.: flawed challenges to 'Acetylcholinesterase inhibitors and Gulf War illnesses.'" *Proc Natl Acad Sci U S A* 105(33): E53. doi: 10.1073/pnas.0805246105 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2575265/pdf/zpqe53.pdf>
- Golomb BA**, Mednick SA, Tenkanen L 2007. "Suicide: A weighty matter?" *Arch Internal Med* 167(17): 1908. doi: 10.1001/archinte.167.17.1908-a <http://archinte.jamanetwork.com/article.aspx?articleid=413070>
- Golomb BA**, Evans MA 2007. "Potential link between HMG-CoA reductase inhibitor (statin) use and interstitial lung disease." *Med J Australia* 187(4): 253. [Email us for full text on an individual basis](#)
- Golomb BA**, Evans MA 2006. "Risk factors for rhabdomyolysis with simvastatin and atorvastatin." *Drug Safety* 29(12): 1191. doi: 10.2165/00002018-200629120-00009 [Email us for full text on an individual basis](#)
- Golomb BA** 2002. "When are medication side effects due to the nocebo phenomenon?" *JAMA* 287(19): 2502-3; discussion 2503-4. [Email us for full text on an individual basis](#)
- Kaplan RM, **Golomb BA** 2001. "Cost-effectiveness of statin medications." *Am Psychol* 56(4): 366-7. doi: 10.1037/0003-066X.56.4.366 [Email us for full text on an individual basis](#)
- Golomb BA** and Jaworski BA 2001. "Statins and Dementia." *Archives of Neurology* 58(7): 1169-70. [Email us for full text on an individual basis](#)
- Golomb BA** 1998. "Cholesterol and violence: Is there a connection?" *Annals of Internal Medicine* 129(8): 669-670. doi: 10.7326/0003-4819-128-6-199803150-00009 [Email us for full text on an individual basis](#)
- Golomb BA** 1996. "Low serum cholesterol and serotonin metabolism – other studies have been done in humans and monkeys." *BMJ* 312(7041):1299. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2351051/pdf/bmj00542-0065a.pdf>
- Golomb BA** 1996. "Using placebos." *Nature* 379: 765. doi: 10.1038/379765b0 [Email us for full text on an individual basis](#)
- Golomb BA** 1995. "Low cholesterol and violence." *Arch Intern Med* 155: 2485. doi: 10.1001/archinte.1995.00430220153019 [Email us for full text on an individual basis](#)
- Golomb BA** 1995. "Paradox of placebo effect." *Nature* 375: 530. doi: 10.1038/375530a0 [Email us for full text on an individual basis](#)
- Golomb BA** 1990. "Hiccup for hiccups." *Nature* 345: 774. doi: 10.1038/345774a0 [Email us for full text on an individual basis](#)
- Golomb BA** 1989. "Taking a byte out of time." *JAMA* 262: 3132. doi: 10.1001/jama.1989.03430220053023 [Email us for full text on an individual basis](#)

## **BOOK REVIEWS**

- Golomb BA** 1997. "Catalyst – For Change in Book Reviews," *Chemistry and Biology* 4: 651-652.

**Golomb BA** 1996. "Genius talk or moron babble? The complementarity principle," *Chemistry and Biology* 3: 813-814.

**Golomb BA** 1995. "Author's Imagination" (Review of *Nature's Imagination: The Frontiers of Scientific Vision*), *Chemistry and Biology* 2(10): 651-2. [http://ac.els-cdn.com/1074552195900268/1-s2.0-1074552195900268-main.pdf?\\_tid=032648f8-eb4c-11e3-84a0-00000aab0f26&acdnat=1401819925\\_61b248fd2950f1dc7a8d26bd0604afb5](http://ac.els-cdn.com/1074552195900268/1-s2.0-1074552195900268-main.pdf?_tid=032648f8-eb4c-11e3-84a0-00000aab0f26&acdnat=1401819925_61b248fd2950f1dc7a8d26bd0604afb5)

## **THESIS**

**Golomb BA** 1988. "Visual Motion Processing: Some Special Properties," PhD Thesis, Department of Biology, U.C. San Diego.

## **ABSTRACTS**

Fung A, Koslik H, Ritchie J, Dinkeloo E, **Golomb BA**. Bioenergetics in Veterans with Gulf War Illness Versus Healthy Controls: Replication and Expansion. Gulf War Illness State of the Science Virtual Conference. August 19, 2020, Washington, D.C.

Bui L, Nguyen E, Dinkeloo E, Ritchie J, **Golomb BA**. Nuclear and Mitochondrial Genetics Together Determine Gulf War Illness Severity and Symptom Profile. Gulf War Illness State of the Science Virtual Conference. August 18, 2020, Washington, D.C.

**Golomb BA**, Bui AK 2015. "Lesser LDL Drop on Statins Predicts Greater Glycemic Rise in Women." *Circulation* 131: AP219

Kamson C, Bui AK, **Golomb BA** 2015. "Wine Consumption and Cognitive Function." *Circulation* 131: AP032

**Golomb BA**, Bui AK 2014. "Fasting glucose positively predicts word memory performance in older men." American Heart Association 2014 Scientific Sessions, Nov 15-19, Chicago, IL. *Circulation* 130: A13365. [http://circ.ahajournals.org/content/130/Suppl\\_2/A13365.abstract?sid=6ab36869-d14b-4a9e-8c85-3e729104e972](http://circ.ahajournals.org/content/130/Suppl_2/A13365.abstract?sid=6ab36869-d14b-4a9e-8c85-3e729104e972)

**Golomb BA**, Bui AK 2014. "Trans fat consumption is adversely linked to memory in working-age adults." American Heart Association 2014 Scientific Sessions, Nov 15-19, Chicago, IL. *Circulation* 130: A15572. [http://circ.ahajournals.org/content/130/Suppl\\_2/A15572.abstract?sid=70b0d945-3f6d-4e41-9222-798c04226b4f](http://circ.ahajournals.org/content/130/Suppl_2/A15572.abstract?sid=70b0d945-3f6d-4e41-9222-798c04226b4f)

**Golomb BA**, Bui AK 2014. "Testosterone drop predicts glucose rise in men on statins." American Heart Association 2014 Scientific Sessions, Nov 15-19, Chicago, IL. *Circulation* A13926. [http://circ.ahajournals.org/content/130/Suppl\\_2/A13926.abstract?sid=6ab36869-d14b-4a9e-8c85-3e729104e972](http://circ.ahajournals.org/content/130/Suppl_2/A13926.abstract?sid=6ab36869-d14b-4a9e-8c85-3e729104e972)

**Golomb BA**, Koslik HJ, Bui AK 2014. "Sleep problems on simvastatin differentially predict weight change in men." American Heart Association 2014 Scientific Sessions, Nov 15-19, Chicago, IL. *Circulation* A13946. [http://circ.ahajournals.org/content/130/Suppl\\_2/A13946.short?rss=1](http://circ.ahajournals.org/content/130/Suppl_2/A13946.short?rss=1)

**Golomb BA**, Koslik H.J. 2014. "Trans Fats Consumption Linked to Higher BMI." Epidemiology and Prevention | Nutrition, Physical Activity and Metabolism 2014 Scientific Sessions. March 18-21, 2014, San Francisco, CA. *Circulation* 129: AP418. [http://circ.ahajournals.org/content/129/Suppl\\_1/AP418](http://circ.ahajournals.org/content/129/Suppl_1/AP418)

**Golomb BA**, Koperski S 2013. "Testosterone Change Relates to Lipid Change on Statins." Epidemiology and Prevention | Nutrition, Physical Activity and Metabolism 2013 Scientific Sessions. March 19-22, 2013, New Orleans, LA. *Circulation* 127: AMP 17. [http://circ.ahajournals.org/cgi/content/meeting\\_abstract/127/12\\_MeetingAbstracts/AMP17](http://circ.ahajournals.org/cgi/content/meeting_abstract/127/12_MeetingAbstracts/AMP17)

**Golomb BA** 2013. "Higher LDL and Lesser LDL-Drop Linked to More Muscle Problems in Men on Statins." Epidemiology and Prevention | Nutrition, Physical Activity and Metabolism 2013 Scientific Sessions. March 19-22, 2013, New Orleans, LA. *Circulation* 127: AP073. [http://circ.ahajournals.org/cgi/content/meeting\\_abstract/127/12\\_MeetingAbstracts/AP073](http://circ.ahajournals.org/cgi/content/meeting_abstract/127/12_MeetingAbstracts/AP073)

**Golomb BA**, Koperski S 2013. "Who Becomes Weak on Statins? Effect Modification Exposed in a RCT by Risk Factor Compounding." Epidemiology and Prevention | Nutrition, Physical Activity and Metabolism 2013

Scientific Sessions. March 19-22, 2013, New Orleans, LA. *Circulation* 127: AP072.

[http://circ.ahajournals.org/cgi/content/meeting\\_abstract/127/12\\_MeetingAbstracts/AP072](http://circ.ahajournals.org/cgi/content/meeting_abstract/127/12_MeetingAbstracts/AP072)

**Golomb BA** 2013. “Glucose Rise on Statins in Older Age: Adaptive Protection Against Fatigue?” Epidemiology and Prevention | Nutrition, Physical Activity and Metabolism 2013 Scientific Sessions. March 19-22, 2013, New Orleans, LA. *Circulation* 127: AP041.

[http://circ.ahajournals.org/cgi/content/meeting\\_abstract/127/12\\_MeetingAbstracts/AP041](http://circ.ahajournals.org/cgi/content/meeting_abstract/127/12_MeetingAbstracts/AP041)

**Golomb BA**, Evans MA 2013. “The Older the Better?” *Journal of Nutrition, Health & Aging* 17 (Supplement 1): S257. [Email us for abstract on an individual basis](#)

**Golomb BA**, Evans MA 2013. “Stop Medicating Beyond the Evidence: Guidelines for Guidelines on Preventive Treatments.” *Journal of Nutrition, Health & Aging* 17 (Supplement 1): S96.

[Email us for abstract on an individual basis](#)

Lai, Thai Hoang, **Golomb BA**. “Dispensing information on medicines prescribed: patients’ preferences and perceived rights” (abstract submitted with both authors names, listed with student’s name only) International Summit on GMP and GCP: USA, Europe, Japan, Asia Pacific. Dec 4, 2012, Philadelphia.

<http://www.omicsonline.org/gmp-gcp2012/scientific-programme.php?day=2&sid=200>.

[Email us for abstract on an individual basis](#)

**Golomb BA**, Vomberg Z, Huynh K, Meskimen AH 2012. More frequent chocolate consumption is linked to better word memory. AHA Scientific Sessions 2012, November 6, 2012, Los Angeles (oral presentation) *Circulation* 126: A16156.

[http://circ.ahajournals.org/cgi/content/meeting\\_abstract/126/21\\_MeetingAbstracts/A16156](http://circ.ahajournals.org/cgi/content/meeting_abstract/126/21_MeetingAbstracts/A16156)

**Golomb BA**, Meskimen AH 2012. Statins and tinnitus: an innovative analysis. AHA Scientific Sessions 2012, November 4, 2012, Los Angeles.

[http://circ.ahajournals.org/cgi/content/meeting\\_abstract/126/21\\_MeetingAbstracts/A19685](http://circ.ahajournals.org/cgi/content/meeting_abstract/126/21_MeetingAbstracts/A19685)

**Golomb BA**, Koperski S, White HL 2012. Statins raise glucose preferentially among men who are older and at greater metabolic risk. AHA Joint Conference - Nutrition, Physical Activity and Metabolism and Cardiovascular Disease Epidemiology and Prevention 2012 Scientific Sessions Mar 16, 2012, San Diego (oral presentation). [http://circ.ahajournals.org/cgi/content/meeting\\_abstract/125/10\\_MeetingAbstracts/A055](http://circ.ahajournals.org/cgi/content/meeting_abstract/125/10_MeetingAbstracts/A055)

**Golomb BA**, Koperski S, White HL 2011. More Frequent Chocolate Consumption is Associated with Lower Body Mass Index. AHA Joint Conference - Nutrition, Physical Activity and Metabolism and Cardiovascular Disease Epidemiology and Prevention 2011 Scientific Sessions Mar 22, 2011, Atlanta. Page 172, abstract #P50 [http://my.americanheart.org/idc/groups/ahamaphublic/@wcm/@sop/@scon/documents/downloadable/ucm\\_323597.pdf](http://my.americanheart.org/idc/groups/ahamaphublic/@wcm/@sop/@scon/documents/downloadable/ucm_323597.pdf)

**Golomb BA**, Koperski S, White HL 2011. Chocolate Consumption is Linked to Aggression. AHA Joint Conference - Nutrition, Physical Activity and Metabolism and Cardiovascular Disease Epidemiology and Prevention 2011 Scientific Sessions. Mar 22, 2011, Atlanta. Page 173, abstract #P53. [http://my.americanheart.org/idc/groups/ahamaph-public/@wcm/@sop/@scon/documents/downloadable/ucm\\_323597.pdf](http://my.americanheart.org/idc/groups/ahamaph-public/@wcm/@sop/@scon/documents/downloadable/ucm_323597.pdf)

Koperski S, Dimsdale J, White H, **Golomb BA** 2011. Sleep problems may mediate glucose elevations on statins: Results from the UCSD Statin Study. AHA Joint Conference - Nutrition, Physical Activity and Metabolism and Cardiovascular Disease Epidemiology and Prevention 2011 Scientific Sessions March 22-23, Atlanta. P160. [Email us for abstract on an individual basis](#)

**Golomb BA** 2010 “Patient reporting of drug adverse effects” *Drug Safety* 33(10): 952-3 From: ISOP meeting, Accra, Ghana. [Email us for abstract on an individual basis](#)

**Golomb BA**, Criqui MH, Dimsdale JE, Evans MA, Broadwin JA, White HL 2009 “Statin effects on energy: Results from the UCSD Statin Study, a randomized trial.” *Circulation* 119: e308. (Joint Conference – 49<sup>th</sup> Cardiovascular Disease Epidemiology and Prevention Annual Conference, and Nutrition, Physical Activity and Metabolism Conference. Abstract ID 837, March 11, Palm Harbor, Florida, presented 3-11-09).

[Email us for abstract on an individual basis](#)

**Golomb BA**, Broadwin JA, White HL, Criqui MH, Dimsdale JE 2009. “Statins reduce orgasm. Results from the UCSD Statin Study, a randomized trial.” American Psychosomatic Society, March 6, Chicago (presented 3-



- Golomb BA**, Evans MA, Dimsdale JE 2008. "Trans Fat Consumption Linked to Aggression." *Circulation* supplement; 117(11):e237. Presented at the American Heart Association 48<sup>th</sup> Cardiovascular Disease Epidemiology and Prevention Annual Conference, and Nutrition, Physical Activity and Metabolism Conference, Colorado Springs. [Email us for abstract on an individual basis](#)
- Golomb BA**, Dimsdale JE, Evans MA, Denenberg JO, White HL, Criqui MH 2008. "Effects of Statins on Aggression Differ by Gender: Results of a Double Blind Placebo Controlled Trial." *Circulation* supplement; 117(11):e268-269. Presented at the American Heart Association 48<sup>th</sup> Cardiovascular Disease Epidemiology and Prevention Annual Conference, and Nutrition, Physical Activity and Metabolism Conference, Colorado Springs. [Email us for abstract on an individual basis](#)
- Golomb BA**, Kwon EK, Criqui MH, Dimsdale JE 2007. "Simvastatin But Not Pravastatin Affects Sleep: Findings from the UCSD Statin Study." *Circulation suppl*; 116:II-847.  
[http://circ.ahajournals.org/cgi/content/meeting\\_abstract/116/16\\_MeetingAbstracts/II\\_847?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=3725&searchid=1&FIRSTINDEX=0&resourcetype=HWCIT](http://circ.ahajournals.org/cgi/content/meeting_abstract/116/16_MeetingAbstracts/II_847?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=3725&searchid=1&FIRSTINDEX=0&resourcetype=HWCIT)
- Yaghamai R; Renvall MJ; **Golomb BA**; Lenert LA; Ramsdell JW 2007. "An Epidemic Of Musculoskeletal Symptoms Among Physicians In Two Institutions Using EMR-Based Systems For Routine Care." Poster presentation/abstract, Society of General Internal Medicine meeting, Toronto, Canada: April 25-28, 2007. [Email us for abstract on an individual basis](#)
- Golomb BA**, Dimsdale JE, White HL, Criqui MH 2006. "Do Low Dose Statins Affect Cognition? Results of the UCSD Statin Study." *Circulation suppl*; 114(18): II-289.  
[http://circ.ahajournals.org/cgi/content/meeting\\_abstract/114/18\\_MeetingAbstracts/II\\_289-d](http://circ.ahajournals.org/cgi/content/meeting_abstract/114/18_MeetingAbstracts/II_289-d)
- Golomb BA**, McGraw J 2005. "Lack of Physician Response Toward Perceived Statin Adverse Events." *Circulation* supplement; 111(14):255. [Email us for abstract on an individual basis](#)
- Golomb BA**, Ritchie JB, Criqui MH, Dimsdale JE. 2004. Statins Lower Blood Pressure: Results from the UCSD Statin Study. *Circulation* (suppl 3) 110(17): 402. [Email us for abstract on an individual basis](#)
- Kordas K, Phillips P, **Golomb BA** 2004. Clinical Characteristics of 1053 Patients with Statin-Associated Muscle Complaints. *Arterioscler Thromb Vasc Biol*; 24:e51-136. [Email us for abstract on an individual basis](#)
- Golomb BA**, Yang E, Denenberg J, Criqui MH 2003. "Statin Associated Muscle Adverse Effects." Poster presentation/abstract, AHA Council on Epidemiology and Prevention meeting, Miami FL: March 7, 2002. *Circulation suppl*. [Email us for abstract on an individual basis](#)
- Yang E, Jaworski B, Denenberg J, Criqui MH, **Golomb BA** 2002. Muscle symptoms in patients on statins. American Heart Association Young Investigators Forum, San Diego, CA: Sept. 2002.
- Golomb, BA** 1999. "Could dysregulation of acetylcholine (from pyridostigmine bromide or other acetylcholinesterase inhibitors) contribute to illness in Persian Gulf War veterans?" Conference on Federally Sponsored Gulf War Veterans' Illnesses Research, June 23-25, 1999 (Abstract accepted; withdrawn for security reasons at request of DoD).
- Golomb BA** and Criqui MH 1998. "Put the risk back in risk-benefit," 1998 San Diego Biostatistics & Epidemiology Research Exchange Conference. La Jolla, CA (Platform presentation May 5, 1998).
- Bagley S and **Golomb BA** 1998. "Patient interest and response to genetic testing for cancer susceptibility: A systematic review of the literature," American Federation for Medical Research, Western Regional Meeting, Feb 4-7, 1998, Carmel, CA. *J Investigative Medicine* 46(1).
- Pyne J, **Golomb BA**, Schten E, Jaworski B, Bozzette S 1998. "Appropriate roles for psychiatrists delivering primary medical care," VA HSR&D meeting, Feb 1998
- Golomb BA** 1996. "Low cholesterol and violent crime," Proceedings of the Robert Wood Johnson Clinical Scholars Program 24th National Meeting, Ft. Lauderdale, FL. (Platform presentation: Nov 1996).



- Golomb BA** 1995. "Cholesterol, serotonin, and violence: Is there a connection?" Proceedings of the Robert Wood Johnson Clinical Scholars Program 23rd National Meeting, Ft. Lauderdale, FL. (Platform presentation: Nov 9, 1995).
- Golomb BA** and Leuchter AF 1993. "Neural Networks Distinguish Demented Patients from Elderly Controls Based on EEG Recordings," Solomon Scholar Research Award, UCLA. (Platform presentation: June 1993).
- Golomb BA**, Lawrence D, Sejnowski T, and Ekman P 1992. "Neural Networks Evaluate Facial Muscle Actions," Solomon Scholar Research Award, UCLA, 1992. (Platform presentation, June 1992).
- Golomb BA** 1990. "Cross adaptation to flow field motion," *Investigative Ophthalmology and Visual Science* 31 (4):522. Poster presentation, Association for Research in Vision and Ophthalmology, Sarasota, FL, April 1989.
- Golomb BA** 1988. "Motion Escape," *Investigative Ophthalmology and Visual Science* 29. Poster presentation, Association for Research in Vision and Ophthalmology, Sarasota, FL, April 1988.
- Golomb BA** 1986. "The Crankshaft Effect," *Investigative Ophthalmology and Visual Science* 27(3): 345. Poster presentation, Association for Research in Vision and Ophthalmology, Sarasota, FL, April 1986.
- Golomb BA**, Andersen, RA, Nakayama, K, MacLeod, DIA, Wong, A. 1984. "Thresholds for shearing motion detection in monkey and man," *Investigative Ophthalmology and Visual Science* 25(3):69. Poster presentation, Association for Research in Vision and Ophthalmology, Sarasota, FL, April 1984.

**MEDIA INTEREST** (Partial list). "x2" (or x3) generally means stories were run on 2 (or 3) different of our studies. 2020: U.S. News and World Report, BBC Future, Daily Mail (UK), GQ

2019: NCIS LA

2018: NY Times (Diplomat mystery illness), Newsweek, Nature, Men's Health, Daily Mail (UK), Vice.com

2017: Al Jazeera

2016: Scientific American Mind, Psychology Today, San Diego Union Tribune, MIT Press, Healthline, The Reno Dispatch, Star Tribune, Truthout.org, Daily Mail (UK)

2015: New York Times, Time magazine, Washington Post, BBC radio, CBS radio, CBS radio Detroit (diff topic), KPBS radio, WUFT public radio, Daily Mail (UK), Nature.com, Reuters Health, Medscape, The Daily Beast, Shape, The Heart.org, RadioMD, Consultant360, Motherboard Press, Chemistry and Biology, Pharmaceutical Journal, Pittsburg Triune Review, HowStuffWorks, Aging News, Revista Salude Brazilian Health mag, La Maison du 21 Siecle magazine, LiveScience.com, Neurology Today,

2014: Atlantic Monthly, CNN, CNN Money, Newsweek, Forbes, Fox News, San Diego Union Tribune, Time, USA Today, US News and World Report,

(Note: 2014 includes more obscure loci because we were sent a tracked list for one of our pieces in the news. For most other years we had no formal attempt to see where items were covered.)

A Breaking News, A Closer Look, (The) Age, ABP News, Aetna IntelliHealth, Air Force Times, Alexandria Daily Town Talk, American Heart Association, AniNews.in, Argentinastar, Argus Leader, Army Times, Article.wn.com, Asbury Park Press, Asian News International (ANI), AZCentral.com, Bazaar, Battle Creek Enquirer, Baxter Bulletin, Bazaar Magazine, Best Life, BioPortfolio, BioSpace, Boston Globe, Brisbane Times, Business Standard, Canberra Times, Capital Bay, CentralOhio.com, Channel4000, Chennaionline, Chillicothe Gazette, China Topix, Cincinnati Enquirer, Clarion Ledger, Clarksville, ClickOrlando, Clinical Advisor, ClinicaSpace, Counsel & Heal, Courier-Pos, tDaily Advertiser, Daily Journal, (The) Daily Meal, Daily Record, Daily News Journal, Daily Times, DailyRx, Des Moines Register, Deccan Chronicle, delhidailynews.com, Desert Sun, Detroit Free Press, Diabetes Care, DNA India, Doctors Lounge, EHE & me, eHealthy News You Can Use, EIN News, E! Science News, EurekAlert!, eWallstreeter, Examiner, Express.co.uk, FoodNavigaor-USA.com, FoodWorldNews.com, Fort Collins Coloradoan, Fremont News Messenger, Gary Null Show, Good Housekeeping, Good Medicine, Green Bay Press Gazette, Guam Pacific Daily News, (The) Gulf Today, Hattiesburg American, HCP Live, Headlines and Global News, Health.com, Health Finder, Health On the Net Foundation, Healthy Eats, HealingWell, Herald Times Reporter, Hindustan Times, Houston Style Magazine, Indianapolis Star, IOL, IOL (ZA), Iowa City Press-Citizen, Irish Health, Jackson Sun, Journal & Courier, Katie Couric Show, KAGS-TV, KARE-TV, KCCI-TV, KCRA-TV, KDVR-TV, KENS-TV, KEYT-TV, KESQ-TV, KETV-TV, KGNS-TV, KGW-TV, Khaleej Times, KHBS-

TV, KHOG-TV, KHOU-TV, KIFI-TV, KING-TV, KION-TV, KITV-TV, KMIZ-TV, KMOV-TV, KOAT-TV, KOCO-TV, KOTA-TV, KPRC-TV, KRCR-TV, KRDO-FM, KREM-TV, KSBW-TV, KSDK-TV, KSPR-TV, KTHV-TV, KTUU-TV, KTVB-TV, KTVM-TV, KTXS-TV, KUSA-TV, KVIA-TV, KVUE-TV, KWCH-TV, KWGN-TV, KXLY-TV, KXTV-TV, KYTV-TV, KYTX-TV, La Voz, Lancaster Eagle Gazette, Lansing State Journal, Legion Media Group, Le Quotidien de Medicin (France), Leaf Chronicle, Livingston County Daily Press and Argus, Louisville Courier Journal, KSL-TV online, LiveScience.com, Mail Online, Marine Corps Times, Mangalorean, Marion Star, Marshfield News Herald, medbroadcast.com, Medical Daily, Medical Express, Medical News Today, MedicineNet.com, MEDINDIA, MedlinePlus, Mens Health, Montgomery Advertiser, Monthly Prescribing Reference, MotherNature Network, MSN.com, my.news.yahoo.com, Navy Times, NDTV-India, NetIndia123.com, Newark Advocate, News 7 (Australia TV), Newkerala.com, News-Herald, News Journal, News Leader, News Press, News Star, Newsday, NewsMax, NorthWest Cable News (NWCN), Nutrition Horizon, Observer & Eccentric Newspapers, Oncology Nurse Advisor, Palladium-Item, Panorama.am, Pensacola News, Pharmacy Times, philly.com, Physicians Briefing, Port Huron Times Herald, Post-Crescent, Poughkeepsie Journal, Press & Sun Bulletin, Press-news.org, Redbook, RedOrbit, (The) Reporter, (The) Salinas Californian, Science Daily, Sciencecodex.com, Sheboygan Press, Sify.com, (The) Spectrum, Springfield News - Leader, State House News Service, Statesman Journal, stuff.co.nz, Summit Medical Group, Sydney Morning Herald, Tallahassee Democrat, Targeted News Service, Tennessean, (The) Times, Times of India, Times of Oman, (The) Verge, Virtual Strategy Magazine, Visalia Times-Delta, VOCM-AM, Walta Info, WAtoday.com.au, Wausau Daily Herald, WAPT-TV, WBAL-TV, WBIR-TV, WCNC-TV, WCSH-TV, WCTI-TV, WCVB-TV, WCYB-TV, WDIV-TV, WDJT-TV, WDSU-TV, webindia12.com, (The) Week, WESH-TV, WFAA-TV, WFMZ-TV, WGAL-TV, WHAS-TV, What's On Ningbo, WICU/WSEE-TV, Winnipeg Free Press, Wisconsin Rapids Daily Tribune, WISN-TV, WJXT-TV, WJXX-TV, WKBT-TV, WKYC-TV, WLBZ-TV, WLTX-TV, WLKY-TV, WLWT-TV, WMAZ-TV, WMBC-TV, WMTV-TV, WMUR-TV, WPBF-TV, WPTZ-TV, WREG-TV, WSBT-TV, WTAE-TV, WTSP-TV, WWLP-TV, WXIA-TV, WXII-TV, WZZM-TV, Yahoo! India, Yahoo! News UK and Ireland, Yuma News Now, Zanesville Times Recorder, ZeeNews.com

2013: Philadelphia Inquirer, Washington Post, Australian Broadcasting Company (ABC) TV, Chatelaine (Canada), Columbia Chronicle, Estadao (Brazil), Le Nouvel Observateur (France), Menta Magazine (Israel), Rodale.

2012: New York Times (x4, including most emailed story), Wall St Journal, CNN, NPR (x2, including most emailed story), Boston Globe (x2), Bloomberg News, ABC, CBC, BBC x 3, KPBS (radio, television), NBC, NBC Latino, CBS Radio, Radio New Zealand, Radio Scotland, Brazil Band News, CBC (Canadian Broadcasting System), News TV live, Daily Telegraph (UK), ABC Sydney, Al Jazeera (TV), Discovery News, CCTV (China), Globe and Mail (Canada), Huffington Post (x2), San Diego Union Tribune (x3), LA Times, Science News, NPR radio (x 2), NY Times Magazine, Time, Thomson Reuters (x2), UK Press association, Time, USA Today.

AARP Bulletin, About.com (NY Times), American Baby, American Medical News, Arthritis Today, Better Homes and Gardens, Bottom Line (x2), California Watch, Consumer Reports (x2), Dagbladet (Norway), Destination Sante (France), Doctor Oz You Beauty, EatDrinkBetter, El Mundo, Experience Life, First for Women, First Watch, Fitness (x2), Fitbe (Rodale), Fitness (x2), Food Network Magazine, Health, Healthday (x2), Healthy Woman, Hospitalist, The Internist (ACP), Istoe Magazine (Brazil), Journal Watch, Korea Radio "1013 Main Street", Korea news, La Vanguardia (Spain), Marie Claire (x 2), MedPage Today, Medscape (most emailed story), Mens Health, Mens Journal, Mercola (Skype interview), MyHealthNewsDaily, NewsMax, Now magazine, Pacific Standard OnLine, Parade, People's Pharmacy (radio interview), Postmedia News Canada, Prevention (x2), Revista Ciencia Hoje ("Science Today," from Brazilian Assn for Advancement of Science), Science News, Science & Vie (France), Scientific American Mind, Self (x2), Shape, Simply Nutrition, Sound Medicine, South China Morning, Spry, Tufts Health Letter, Post, La Vanguardia (Spain), Voice of Russia radio, WebMD (x2), Women's Health, WTIP Community Radio Roundtable, Yale Daily News, Yoga Journal.

Our article on chocolate and body mass index was the topic of the most emailed NY Times story that day, as well as the most emailed Medscape story. It was the leading news story from the University for that month (March), though it came out near the end of the month (Mar 26), besting the next biggest news story for the University that month by a factor of two; that story was also from our lab (on trans fats and aggression).

2011: ABC, NBC, Washington Post, Philadelphia enquirer, Slate, Boing Boing, Ivanhoe Broadcast

2010: *Time* magazine, *Scientific American*, *CNN*, *BBC News*, *Wall Street Journal*, *LA Times*, *Business Week* (both *News* and *Lifestyle* sections), *AOL*, *ABC News*, *CBS News*, *CANWorld*, *KNBC TV*, *Mens Health*, *Women's Health*, *FDA Reporter*, *The Globe and Mail* (Canada), *Radio New Zealand*, *UK Press Association*, *NPR*, *Reuters*, *Bloomberg News*, *CNN Health.com*, *CAN West news* (Canada), *LA Times* (x 2), *the Australian*, *Fox*, *Fox Business.com*, *Boston Global*, *Chicago Tribune*, *Denver Post*, *Discovery Health*, *Montreal Gazette*, *MSN*, *MSNBC*, *The Australian*, *The Daily Mail* (UK), *Time* magazine (again), *Times* (India), *Medscape*, *United Press International*, *WebMD*, *Xinhua News*, *Yahoo News*, many others.

Our April 2010 article on chocolate and depression in *Archives of Internal Medicine* (Google: chocolate depression Archives April 2010) led to hundreds of news stories extending to at least 50 foreign nations per Google News – the runaway big story for our University that month, and had only descended to the number 2 health story on CNN a month later (with stories still being run in major media).

2009: *NBC TV* (San Diego News Now!), *USA Today* (section A story), *CNN radio*, *60 minutes*, *Forbes*, *The Globe and Mail* (Canada), *WebMD*, *San Diego Union Tribune* (front page story).

2008: *BBC*, *Bloomberg News*, *Business Week*, *CNN*, *Daily Mail* (UK), *Daily Telegraph* (UK), *Discovery*, *Economist*, *Good Morning America* (ABC TV), *LA Times*, *New Scientist*, *New York Times*, *Reuters*, *The Australian* (Australia), *The Doctors* (TV show), *US News and World Report*, *Wall St Journal*, *Washington Post*.

Previous (selected).

Statin Adverse Effect website:

*Wall Street Journal*. “Researchers Ask Patients to Help Fill Gap in Data on Side Effects of Statins.” Tara Parker-Pope 10/3/2006

*United Press International*. “Statin Users Report Side Effects Online.” Leah Carliner. 9/22/2006; Channel 10 television news, San Diego, Carol LeBeau

*ABC7 News.com*. “Risks & Benefits of Statin Drugs.”

*Washington Post Online*, *Reuters Online*, *MSNBC Online*, *KPBS*, *American Heart Association*, *WebMD*, *Fox News*, *LA Times*. “Cholesterol-lowering drug linked to sleep disruptions.”

*The New York Times*. Dec 21, 2007.

*The New York Times*. Tara Parker-Pope. 2/13/2008.

Cholesterol; and Statin risk-benefit:

11-97 *KNBC TV*; 3-98 *New York Times* 5 column article, *CNN*, *NBC*, *ABC*, *CBS*, *LA Times*, *Reuters/AP*, *Science* magazine. Also, British newspapers.

*NBC Nightly News*, 8-24-01. *NBC Nightly News*, 11-13-01. *Wall Street Journal*, 4-25-02. *MSNBC* (print) 8-24-01. *Wall Street Journal*, 12-02. *San Diego Union Tribune* 5-03. *Los Angeles Times* 7-03. *Wall Street Journal* 1-04. *New York Times* 7-04. *CBS News with Dan Rather* 5-04. *Newsday* 7-04. *The San Diego Union Tribune* 7-04. *The Times* (London) 8-04.

Other: Multiple newspapers throughout US 7-00, 9-00; *LA Times*, 2000 & 2001. *San Diego Union Tribune*, 5-16-01 & 5-28-01 (front page article); *Philadelphia Enquirer* 8-27-01. *Sciences et Avenir* (France), 10-01. *WebMD* (peripheral neuropathy) 5-02. *Discover magazine*. *KPBS radio* 5-29-02 10-11 “These Days”; with Tom Fudge and Dr. Michael Criqui; *UCSD TV*, lecture 8-02. *Men's Health*, *San Francisco Chronicle* 1-05. *The Sunday Times-Britain* 3-05. *San Diego Union Tribune* 7-05. *Lifetime Fitness* 12-05. *Daily News* (UK) 6-06. *Daily Mail* (UK) 6-06. *BusinessWeek* 8-06. *Daily Mail* (UK) 1-07. *ABC7News.com* 2-07. *Smart Money*

*The New York Times*. “Great Drug, but Does It Prolong Life?” Tara Parker-Pope. 1/29/2008

*Ladies' Home Journal*. “Does Cholesterol Really Count?” Linda Marsa. Feb-08

*Business Week*

*The Wall Street Journal*. Melinda Beck. 2/12/2008.

*Good Morning America* 2-08

Israeli press (Hebrew symbols)

Gulf War illness:

Following Pentagon press briefing 10-99 (and interviews)

*NY Times* and *LA Times* (front page of each); *Washington Post*, *San Diego Union Tribune*, multiple other newspapers; *NPR*, other radio stations, national TV news on *ABC* and *CBS* (lead story), *NBC*; *CNN*, *CSPAN*; *policy.com*, *Yahoo* (lead news story), *The Daily Show with Jon Stewart* (lead “Headline News” story); science journals including *New Scientist*. *BBC radio* interview, *BBC television*, television news throughout Western Europe (e.g. Britain, Germany, other), Eastern Europe, Australia; newspapers and news magazines throughout same distribution (10-99) – e.g. *Le Monde* (France), Hungarian newspapers and news magazines, Danish periodicals, German, *BBC news*

Following Congressional testimony in 11-99: *CNN*.

Subsequent: *Science magazine* 4-01. *Science magazine* 4-02. *NPR Science Friday* (radio guest) 3-03.

On release of Committee report 2004: Front page article *NY Times* 10-15-04; Front page *San Diego Union Tribune* 10-15-04; *BBC radio* 10-15-04 (or 10-16); *London Times*; Others internationally.; *Science Magazine*; *BMJ* (news section); *BBC News* (and *BBC radio*) 10-04. *BMJ News Extra* 10-04.

“Study: Sarin at Root of Gulf War Syndrome.” Kelly Kennedy. *Army Times*. 5/25/2007

Following PNAS article on acetylcholinesterase inhibitors and illness in Gulf War veterans: *BBC radio* 3-08; *CNN radio* 3-08, *Economist* 3-08; other UK media/ press, *The Australian* (leading Australian daily), German radio interview, *LA Times*, *Reuters*, *Washington Post*, *San Diego Union Tribune*, *Bloomberg News*, many other venues

Following Gulf War RAC Committee Report: Nov 2008: most major news venues

Pyridostigmine FDA approval for Nerve Agent Protection 3-03: *Science magazine*, multiple regional papers (e.g. *Rocky Mountain Times*, *Orange County Register*)

Cholesterol and violent crime: *Web MD* 2000; *Clinical Pearls* 2001; *Crime Times* 2001

Alcohol and diabetes: re: *JAMA* article: 7-99: *Fox News*, *Science News*, other

Neural networks: pertaining to SexNet and ExpressionNet: 1990 *CNN*; 1990 Jim Jubek, *In the Image of the Brain* (featured in chapter one of lay book on neural network revolution); 1992, *The Machine that Changed the World, Episode 4: The Thinking Machine* (A PBS NOVA Documentary).

**GOLOMB Attachment 2**

**Diplomats' Mystery Illness and  
Pulsed Radiofrequency/ Microwave Radiation**

Beatrice Alexandra Golomb, MD, PhD

**Corresponding Author:**

Beatrice Alexandra Golomb, MD, PhD

Professor of Medicine

UC San Diego School of Medicine

858 558-4950 x201

[bgolomb@ucsd.edu](mailto:bgolomb@ucsd.edu)



**Abstract:**

**Importance:** A “mystery” illness striking US and Canadian diplomats to Cuba (and now China) “has confounded the FBI, the State Department and US intelligence agencies.” Sonic explanations for the so-called “health attacks” have long dominated media reports, propelled by peculiar sounds heard and auditory symptoms experienced. Sonic mediation was justly rejected by experts. We assessed whether pulsed radiofrequency/microwave radiation (RF/MW) exposure can accommodate facts - including unusual ones - reported in diplomats.

**Observations:** 1. Noises: Chirping, ringing or grinding noises were heard at night, during episodes reportedly triggering health problems, by many diplomats. Some reported that sounds were localized with laserlike precision; or said the sounds seemed to follow them (within the territory in which they were perceived). Pulsed RF/MW engenders just these apparent “sounds” via the “Frey effect.” Perceived “sounds” differ by head dimensions and pulse characteristics, and can be perceived as located behind, in or above the head. Ability to hear the “sounds” depends on high frequency hearing and low ambient noise. 2. Signs/symptoms: Hearing loss and tinnitus are prominent in affected diplomats – and in RF/MW-affected individuals. *Each* of protean symptoms that diplomats report, also affect persons reporting symptoms from RF/MW: Sleep problems, headaches, and cognitive problems dominate in both groups. Sensations of pressure or vibration figure in each. Both encompass vision, balance and speech problems, and nosebleeds. Brain injury and brain swelling are reported in both. 3. Mechanisms: Oxidative stress provides a documented mechanism of RF/MW injury compatible with reported signs and symptoms; sequelae of endothelial dysfunction (yielding blood flow compromise), membrane damage, blood brain barrier disruption, mitochondrial injury, apoptosis, and autoimmune triggering afford downstream mechanisms, of varying persistence, that merit investigation. 4. Of note, microwaving of the US embassy in Moscow is historically documented.

**Conclusions and Relevance:** Reported facts appear consistent with pulsed RF/MW as the source of injury in Cuba diplomats. Non-diplomats citing symptoms from RF/MW, often with an inciting pulsed-RF/MW

exposure, report compatible health conditions. Under the RF/MW hypothesis, lessons learned for diplomats

and for RF/MW-affected “civilians” may each aid the other.

**Introduction:**

More than two dozen American diplomats in Cuba<sup>1,2</sup>, and their families<sup>3</sup>, plus a smattering of Canadian diplomats in Cuba<sup>4,5</sup> and their families<sup>6</sup>, reportedly developed a “mystery” illness<sup>4,7-9</sup> that “has confounded the FBI, the state department and US intelligence agencies”<sup>9</sup>, “baffling US officials”<sup>10</sup>: “‘It’s just mystery after mystery after mystery’”<sup>10</sup>. Problems began in 2016, began to be widely reported in 2017, and as of January 2018, “‘We are not much further ahead than we were in finding out why this occurred,’ Undersecretary of State Steve Goldstein said”<sup>1</sup>. Similar problems first were recognized in China in April 2018, and “a number of diplomats at the US consulate in Guangzhou, China had been sent home with similar symptoms”<sup>2,11-13</sup> – by June’s end, “at least eight” from the consulate in Guangzhou, and “at least 11” from China more broadly<sup>14</sup>.

Media reports have long characterized these so-called “health attacks”<sup>15-17</sup> as “sonic attacks”<sup>2,7-10,18-20</sup>.

This characterization persisted despite rejection of sonic explanations by experts<sup>8-10,21,22</sup>. E.g. “No single, sonic gadget seems to explain such an odd, inconsistent array of physical responses”<sup>10</sup>. Per psychoacoustics expert Joseph Pompei: “‘Brain damage and concussions, it’s not possible.’...‘Somebody would have to submerge their head in powerful ultrasound transducers’”<sup>10</sup>. Some suggested a viral hypothesis<sup>1</sup>, but this fails to explain many features of these cases, including the strange noises associated with inciting events in some; and there isn’t a known viral illness with a compatible profile of symptoms. Though “officials told senators the US government knew of no weapon, sonic or otherwise, that could produce the effects seen in the Cuba patients”<sup>1</sup>, to this date, some media sources continue to reference sonic attacks<sup>2</sup>.

A different explanation is proposed that, it is suggested, better accommodates the facts – including the “odd, inconsistent array of physical responses”<sup>10</sup> and other “mysterious” and protean features reported. Reported features are assessed for compatibility to known effects of radiofrequency/ microwave radiation (RF/MW), particularly pulsed RF/MW. Symptoms and signs are assessed against symptoms and signs reported by people

that report health effects from RF/MW exposure – a condition that has been termed “radiofrequency sickness”<sup>23</sup>, “microwave syndrome”<sup>24</sup>, or to encompass people experiencing problems from exposures beyond a specific part of the electromagnetic spectrum, “electromagnetic hypersensitivity”<sup>25-29</sup>, “electrosensitivity”<sup>30-32</sup> or “electrohypersensitivity”<sup>33-38</sup>.

## **Methods:**

Features of diplomats’ “health attacks” – origins, symptoms, and findings are delineated, and examined in relation to evidence regarding symptoms from RF/MW.

Features to be examined for compatibility with an RF/MW-explanation include the following. Strange noises were heard by some diplomats during apparent inciting episodes<sup>5,11</sup>. The noises that were heard differed markedly for different diplomats<sup>5</sup>. The various descriptions included high pitched chirping similar to crickets or cicadas, ringing and grinding<sup>10</sup>. The noises were heard primarily at night<sup>10</sup>. Other diplomats heard no noises<sup>5</sup>, and were not aware of any inciting episodes – just onset of symptoms. In at least some cases, incidents (and noises) were confined to “parts of rooms with laser-like specificity”<sup>10</sup>. And, within the area in which a sound was perceived, it seemed to follow the person around the room<sup>11</sup>.

Auditory symptoms are a prominently reported and distinctive feature (though not present in all) and include hearing loss<sup>6,9,10,15,17,39</sup> and tinnitus<sup>5,6,9,40</sup>, and particularly during inciting episodes in some, ear pain<sup>40</sup>.

Other symptoms are protean and vary markedly from individual to individual -- “an odd, inconsistent array of physical symptoms”<sup>10</sup>. Sleep symptoms<sup>6,41,42</sup>, headache<sup>5,6,15</sup>, cognitive dysfunction<sup>6,10,40,41</sup>, and fatigue<sup>6,40</sup> are prominent among the “nonspecific” symptoms. In some, problems were temporary and apparently recovered with time away from the exposure<sup>9</sup>; others experienced persistent problems<sup>3,5</sup>.

Potentially objectively measurable problems with speech<sup>9,10</sup>, balance<sup>9,10,41,42</sup> and vision<sup>41,42</sup> as well as epistaxis (nosebleed)<sup>9</sup> are a feature in some. Peculiar sensory symptoms of pressure and vibration are reported<sup>41</sup>. Brain injury<sup>3,5,9,43</sup>, white matter abnormalities<sup>44</sup>, and brain swelling<sup>5,9</sup> have been reported.

To assess compatibility of symptoms in diplomats with those experiencing symptoms from RF/MW, we focus on those who are symptomatic in each group. “Only a minority of embassy staff were stricken”<sup>11</sup> and it is these who have been reported upon and studied. The appropriate comparator are the minority who are symptomatic from RF/MW exposures.

Peer reviewed publications are the primary source of information. However, the most authoritative source for information about symptoms and experiences of individuals is affected individuals themselves: peer review confers no benefit and has no power to adjudicate individuals’ reports. For this reason, peer reviewed literature to address issues of science is complemented by sources that have elicited and reported on symptoms and experiences of diplomats, or of RF/MW affected individuals, extending to encompass news reports, surveys, statements of affected individuals, or when applicable other “gray literature”. For diplomats, news/ media reports are complemented by a *Jama* report focused on neurological symptoms in diplomats<sup>41</sup>. Information that references “news,” rather than science, also cites media sources.

Mechanisms by which RF/MW may cause reported problems are cursorily addressed. Sources of RF/MW reported to affect the comparator group, and potential RF/MW sources of diplomats’ symptoms, are briefly reviewed.

## Results:

**Table 1** reviews characteristics of noises reported by diplomats in inciting episodes, and compatibility with RF/MW. Pulsed RF/MW in the 2.4-10,000MHz range produces perceived noises that resemble sounds “such

as a click, buzz, hiss, knock, or chirp” – just as diplomats report<sup>45</sup>. Ability to hear Frey “sounds” is reported to depend on high frequency hearing, and on low ambient noise<sup>45</sup> – through a phenomenon termed the “Frey effect.” (Synonyms include “microwave auditory effect,” “RF hearing” and variations of these.) This fits reports that noises were not universally perceived. The requirement for low ambient noise accounts for perception of “sounds” primarily at night<sup>10</sup>. The primary pitch perceived reportedly relates to head dimensions<sup>45</sup> – in addition to pulse waveform and other characteristics -- accounting for different “sounds” perceived by different diplomats. Sounds were localized with “laserlike” specificity in some cases, supposedly defying known physics<sup>10</sup>. This may defy the physics of sound, but not the physics of RF/MW: lasers *are* electromagnetic radiation (**EMR**). One diplomat reported that the sound seemed to follow him within the space in which it was heard<sup>11</sup>. Frey “sounds” are also reported to follow the person, often perceived as slightly behind the head, regardless of the body orientation relative to the source of radiation<sup>45-47</sup>. Of note, Frey induction is not governed by *average* radiation intensity, but the energy in a single pulse<sup>45</sup>. (Analogously, if a jackhammer hit each 2 minutes, the low time-averaged pressure would not explain the damage.)

**Table 2** reviews diplomats’ symptoms and signs, and compatibility of these with RF/MW.

Auditory symptoms, including tinnitus, hearing loss, and ear pain or pressure are prominent in diplomats<sup>41</sup> and in persons affected by RF/MW<sup>48-51</sup>. Symptoms are protean in both groups. Prevalent among non-auditory “nonspecific” symptoms are sleep problems, headaches, cognitive problems, and to a lesser degree dizziness and nausea<sup>9,10,40,41,48-51</sup>. Additional more specific symptoms that are in principle objectively measurable include problems with balance, speech, vision, and epistaxis, i.e. nosebleed<sup>9,10,41,48,49,51</sup>. Peculiar sensory symptoms are reported in both, including pressure and vibrations<sup>41,49</sup>. Reported brain findings have included brain swelling, problems consistent with traumatic brain injury, and white matter abnormalities. Each such feature is also observed in those with symptoms ascribed to RF/MW.

**Table 3** lists symptoms commonly reported in diplomats, together with percentages reporting each symptom, for symptoms assessed in the neurological appraisal of Cuba diplomats or mentioned in news reports<sup>9,10,40,41</sup>.



These symptoms (when elicited) are ranked by prevalence, in surveys of persons exposed to specific sources of RF/MW, or with symptoms ascribed to EMR exposure<sup>48-51</sup>. Fractions of symptomatic diplomats who report each symptom<sup>41</sup> appear similar to fractions of those symptomatic with EMR symptoms, who do so. Comparing rates in diplomats<sup>41</sup> to those in a peer reviewed study of EMR affected individuals<sup>50</sup> on symptoms tallied in both, symptom rates were: Headache 81%-vs-81%; Cognitive problems 81%-vs-81%; Sleep problems 86%-vs-76%; Irritability 67%-vs-56%; Nervousness/Anxiety 52%-vs-56%; Dizziness 67%-vs-64%; Tinnitus 57%-vs-63%<sup>41,50</sup>. Thus, rates conform closely.

The rates of symptoms reported for diplomats appear within reported variation, for studies of persons affected by RF/MW/EMR. Sleep problems were reported somewhat less frequently in EMR affected persons in the Kato study (76%), than in diplomats – but reported sleep problems, or their byproduct fatigue (for which prevalence was not recorded in the diplomat study), dominate the number one symptom position in studies of RF/MW affected persons (**Table 3**), and prevalence of sleep problems was higher than for diplomats in some other studies of RF/MW affected persons<sup>52</sup>. Of note, the Kato study was performed in Japan, where the traditional diet is rich in fish, which supply the long-chain omega-3 fatty acids that reportedly benefit sleep and reduce irritability<sup>53,54</sup> (the two symptoms that were >3% lower than in affected diplomats).

The protean character of symptoms in diplomats<sup>19</sup> (as for RF/MW-affected individuals) has led some to infer that a single cause cannot account for all. But a number of reports, in a number of nations and settings, tie RF/MW exposure (in vulnerable individuals) to each of the problems reported in diplomats. The coherence of findings in those citing affects of RF/MW, with findings in diplomats, supports a common cause within each group – and across the two groups. Of note, a protean suite of generally the same symptoms – but in a different distribution – is reported in other conditions that are tied to mitochondrial alteration and oxidative stress<sup>55-57</sup> (mechanisms which each promote the other<sup>58,59</sup>). RF/MW is tied to these mechanisms<sup>60-66</sup>. However the distinctive prominence of sleep and auditory symptoms, the peculiar somatic sensory experiences of pressure

and vibration, and the noises perceived during apparent inciting episodes, are relatively distinctive features – distinctive to diplomats’ reports, and to reported RF/MW problems.

**Table 4** reviews several mechanism considerations. Central to this is the critical role of oxidative stress, and the relevance of oxidative stress to potential auxiliary mechanisms, such as mitochondrial dysfunction, blood brain barrier disruption, membrane alterations, impaired blood flow, apoptosis, effects on voltage-gated calcium and anion channels, and triggering of autoimmune reactions. (In some cases effects are reciprocal – oxidative stress promotes mitochondrial dysfunction, calcium channel effects, inflammation, autoimmunity – which in turn can promote oxidative stress.) One analysis found that of 100 evaluated studies that examined the relationship of low level RF/MW to oxidative stress in biological systems, 93% supported a connection<sup>60</sup>. A role for oxidative stress in RF/MW/EMR affected persons is cemented by evidence that gene polymorphisms adverse to antioxidant defense are significantly more prevalent in persons experiencing symptoms from RF/MW/EMR<sup>67</sup>. Additionally, levels of a particular antioxidant – melatonin – known to be critical for RF/MW and broader EMR defense are consistently low in affected persons (assessed by a urinary metabolite)<sup>33</sup>. Oxidative stress has been tied to each of the symptoms and conditions reported in diplomats, and RF/MW affected persons.

Also noteworthy is the repudiation of psychogenic causation in the evaluation of diplomats<sup>11,41</sup>, which holds for RF/MW affected persons as well. Case narratives for those affected by RF/MW underscores that for many, symptoms developed and progressed when affected parties as yet had no knowledge that an RF/MW emitting device had been introduced, nor that one could cause problems<sup>49,52</sup>. A Swiss Telecom funded study found that sleep problems related to the electromagnetic field strength of the transmitter, and did not correlate with personality traits tied to worry about health<sup>48,68</sup>. The circumstance that some report being affected severely by levels of exposure that cause others no problem, is reviewed in the context of effect modification, variations in antioxidant defenses, and demonstrated variable involvement of secondary mechanisms such as autoimmune

activation<sup>33</sup>. In fact, analogous marked differences in harm or development of health effects are well known for other exposures, such as peanuts, penicillin, and pesticides. For EMR affected persons<sup>67</sup> – as for many other exposure-related illnesses – genetic influences on phase I or phase 2 detoxification, as well as factors that inhibit or compete for detoxification systems, play a documented role in who develops health effects<sup>69-74</sup>. (Phase 2 detoxification encompasses protections against oxidative damage.)

**Table 5** briefly addresses the range of RF/MW sources that have been presumptively tied to problems. It observes that RF/MW/microwave radiation is known to have been used on the US embassy in Moscow – there is precedent for use on diplomats<sup>75,76</sup>. That instance, though with presumably differing details of exposure, led to (disputed) reports of health effects in embassy staff, and shielding efforts by the US. Since the exposing device can be outside the building – and typically has been, for persons affected by RF/MW-emitting utility meters<sup>48</sup> – failure of the FBI to find devices in sweeps of diplomats’ rooms remains compatible with this explanation.

## **Discussion:**

### **Recap of Findings:**

Health effects reported by US and Canadian diplomats (and family members) in Cuba and China, and the circumstances surrounding inciting episodes, are consistent with effects of RF/MW. Reports of perceived sounds fit known characteristics reported for the Frey effect (RF hearing, microwave hearing): Sounds were heard by some but not other diplomats during inciting episodes, sounds differed in character from person to person, sounds included chirping, ringing and grinding, sounds were heard predominantly at night. Sounds were localized with “laserlike” specificity in some of the cases, and within that localization, seemed to follow people. Prominence of auditory symptoms, including hearing loss, tinnitus, and ear pain in diplomat reports, typify reports of injury from pulsed RF/MW. Presence of variable additional symptoms of protean character that differ markedly from person to person, with a relative emphasis on sleep disturbance, headaches, and

cognitive problems; plus presence in smaller subsets of vision, balance, and speech problems are also characteristic. Affected persons in both groups report sensory symptoms of pressure and vibrations. Persons in both groups show evidence of brain injury. Reports in both indicate that some persons had prior head injury, and brain injury may be a predisposing factor for, as well as a consequence of, RF/MW injury<sup>11,34</sup>. Both show varying rates of symptom persistence. How subsequent natural history will compare, for diplomat symptoms that *might* follow more intense discrete exposure (a more intense exposure may produce problems in persons who need not have relative vulnerability), vs follow repeated less intense ones (producing symptoms, evidence suggests, selectively in persons more vulnerable to free radical injury from RF/MW, at a level to which they will likely have subsequent exposure), is not known.

#### **Fit with Literature:**

Evidence for health effects of RF/MW is not new<sup>47,77-79</sup>. By the early 1930s, studies were citing compatible symptoms in radio amateurs and shipboard radio operators<sup>77</sup>. By 1971/2 a Naval report bearing over 2300 citations, many from Russia and Eastern Europe, documented health effects of microwave/RF/MW, emphasizing “non-ionizing radiation at these frequencies”<sup>80</sup>. Contrary to claims by industry-affiliated parties, copious evidence documents that radiation that is not “ionizing” can also cause health effects. Entire sections of the 1971/2 report were devoted to each of a number of the symptoms that diplomats are now reporting, including insomnia, headache, fatigue, cognitive problems, and dizziness<sup>80</sup>. Injury from nonionizing radiation occurs also without measurable heating – nonthermal radiation<sup>81-83</sup>. Indeed, oxidative stress, which mediates nonthermal effects, also mediates thermal effects; and melatonin, which defends against oxidative RF/MW injury, also defends against so-called thermal injury<sup>84-88</sup>. Moreover, other sources of heat do not produce the same so-called “thermal” damage that RF/MW does<sup>47</sup>: What are deemed thermal effects may be among the manifestations of oxidative injury. While a low percentage of individuals experience overt symptoms from usual RF/MW, the absolute number may be vast: the fraction with electrosensitivity/ electromagnetic illness has been estimated at between 1 and 5%, and apparently rising<sup>37,89-92</sup>.

**Limitations:**

Features of diplomats' experiences rely on media reports and one published neurological evaluation. We did not examine diplomats; however, in conditions with highly distinctive characteristics, the history is often the most important factor in the diagnosis, and diplomats' reports bear highly distinctive characteristics. The close matching of these distinctive characteristics to those of persons with health problems arising in apparent relation to pulsed RF/MW, provides a basis for concern that RF/MW exposures may underlie diplomats' symptoms and health conditions.

A tremendous number of physicians and scientists and entities and scientific studies and government reports, in many nations, over many decades, have identified that RF/MW causes symptoms consistent with the spectrum now described for diplomats. Scientific "skepticism" about RF/MW health effects is well represented in the literature, but is of the industry-fueled stripe (think tobacco): Effects of conflicts of interest on research results (as well as on funding, regulatory agencies, legislation and academics) vis a vis RF/MW, has been repeatedly documented and decried<sup>93-97</sup>, and evidence of this influence parallels evidence of potent impact of conflict of interest in medicine more generally<sup>98</sup>. In one illustrative analysis, studies of health effects of cell phones that were funded exclusively by industry were least likely to report a significant effect. Relative to studies funded exclusively by public agencies or charities, the odds ratio was 0.11 (95% CI 0.02-0.78)<sup>93</sup> – that is, the odds were ~a tenth as great for a significant finding in a study in purely industry funded studies. The finding was not materially altered when analysis was adjusted for factors like study quality. Richard Smith, then Editor in Chief of the BMJ (the British Medical Journal) penned an article "Conflicts of interest: How money clouds objectivity." Responding to evidence tying study results on a different lucrative product (tobacco) to conflicts of interest (often undisclosed), he suggested that "far from conflict of interest being unimportant in the objective and pure world of science where method and the quality of data is everything, it is the main factor determining the result of studies"<sup>99</sup>.

**Conclusions/Implications:**

Numerous highly specific features of diplomats' experiences and symptoms fit the hypothesis of RF/MW injury. To distinguish between sonic and microwave hypotheses, earplugs can be issued to diplomats for use in candidate episodes (e.g. strange noise plus ear pain): earplugs will mute sonic sources (caveat: a sound like crickets chirping may in fact be crickets chirping), but not microwave ones (which may even be intensified). Monitoring for culpable radiation sources must sensitively capture pulsed RF/MW, including that which may be used only on an intermittent basis. It should encompass the 2.4-10,000MHz range in which the Frey effect has been reported. Perhaps attention to diplomats' plight can ignite awareness of the many others affected by similar problems. Meanwhile, research already documenting compatible health effects of RF/MW in a subgroup, may inform those caring for diplomats, and those in pursuit of causative devices.



**Table 1. Features of Noises Reported by Diplomats during apparent inciting episodes.**

Though “sound” refers to air pressure waves, we will refer to what diplomats “heard” as (perceived) sound.

Diplomats’ Reports	Compatibility with RF/MW
<p>Strange noises were heard by many “of the 24 ‘medically confirmed’” affected US diplomats<sup>1</sup>, during what were perceived as inciting episodes<sup>10</sup>.</p>	<p>Sound ordinarily results from air-<i>pressure</i> waves (which are “longitudinal” waves – variation occurs along the direction of travel of the wave); whereas radiation arises from <i>electromagnetic</i> waves (which are transverse waves – variation occurs perpendicular to the direction of travel of the wave). In each case, a “frequency” is defined by the number of “cycles” of the wave (that pass, say, a given point) per second, for the respective wave type.</p> <p>Though electromagnetic signals are not themselves sound, RF/MW can lead to perceived noises via the so-called “Frey effect”<sup>45</sup> (aka microwave hearing, aka RF hearing).</p> <p>A 1976 Defense Intelligence Agency report stated “Sounds and possibly even words which appear to be originating intracranially can be induced by signal modulation at very low average-power densities”<sup>78</sup>.</p> <p>A 1994 Air Force Materiel Command report stated, based on knowledge at the time, that “Individuals exposed to pulsed RF/MW radiation have reported hearing a chirping, clicking or buzzing sound emanating from inside or behind the head. The auditory response has been observed only for pulsed modulated radiation emitted as a square-wave pulse train. The pulse width and pulse repetition rate are factors that appear to determine the type of sound perceived.... James Lin... reports that the sensation of hearing in humans occurs when the head is irradiated at an average incident power density level of about 0.1 mW/cm<sup>2</sup> and a peak intensity near 300 mW/cm<sup>2</sup>. Auditory responses have been observed for a frequency range of 200-3000 MHz and for pulse widths from 1-100 μs”<sup>47</sup>.</p> <p>The frequency range within which sounds can be heard was broadened by 2003: it was reported that sounds can be perceived by persons exposed to RF/MW in the 2.4-10,000MHz range<sup>45</sup>. It was noted that the same frequency did not produce the same sound, from person to person.</p>
<p>Not all diplomats heard noises<sup>10</sup>.</p>	<p>Ability to hear RF/MW-induced “sounds” (using the term to refer to the perception, not the stimulus) at all depends on individuals’ high frequency hearing<sup>45</sup>, as well as on low ambient noise<sup>45</sup>.</p>
<p>Among those who heard noises, the noises reported differed markedly for different diplomats<sup>5</sup>.</p>	<p>In RF hearing/ microwave hearing, the primary pitch heard (i.e. the perceived <i>sound</i> frequency), reportedly relates not to the <i>radiation</i> frequency (cycles/sec), but to head dimensions<sup>45</sup>. This comports with reports that different sounds were heard by different diplomats, even if they were exposed to the same frequency (or conceivably frequencies, plural) of radiation. Of note, whether sound is perceived from RF/MW is not governed by the <i>average</i> radiation level, but the energy in a single pulse. Injury to cells (in part through membrane damage) is also materially greater with pulsed radiation<sup>100,101</sup>. (Analogously, if a jackhammer hit very hard but very briefly at 2 minute intervals, the low time-averaged pressure would not explain the effects produced.) Pulses of comparatively high intensity (relative to typical exposures from technology) would have likely been necessary to produce the comparatively high prevalence of Frey-compatible sounds, and of health effects reported among US diplomats.</p> <p>The relatively high proportion of affected diplomats reporting Frey type noises, suggests the possibility of comparatively high intensity of pulses; and frequencies within the designated 2.4-10,000MHz range.</p>
<p>These noises included a high pitched “chirping,” ringing and “grinding”<sup>8, 10</sup>.</p>	<p>Frey “sounds” are “similar to other common sounds” “such as a click, buzz, hiss, knock, or chirp” – consistent with sounds that diplomats reported<sup>45</sup>.</p> <p>In a 2007 Dutch survey completed by 250 persons with electrosensitivity (ES), queries related to noise included buzzing (reported by n=96), hissing (n=80), strong low frequency sounds (n=55) and “sound of bells clanging” (n=28)<sup>102</sup>. The term “chirping” (if there is a Dutch equivalent) was not included among inquiries. Of note, the “strong low frequency sounds” are potentially consistent with the “blaring, grinding noise” reported by a diplomat, next section (“blaring” indicative of “strong,” and “grinding” consistent with low</p>

	<p>frequency); while the “sound of bells clanging” is consistent with reports of diplomats who awoke to hear ringing “and fumbled for their alarm clocks, only to discover the ringing {clanging} stopped when they moved away from their beds”<sup>5</sup>.</p> <p>In the Maine Smart Meter survey report<sup>49</sup>, comments by affected persons were included. Exemplars involving Frey noises included these: {After} “72 Itron AMI smart meters {were installed} near me in my townhome complex... I hear a constant buzzing that is driving me crazy. It keeps me awake and makes it hard to think. I am not sure if it is an actual sound, or if it is being generated inside my head, because when I put my fingers in my ears I still hear it... In addition, at about every 15 or 20 minutes, a more intense whine is added that lasts about 12-15 seconds, that hurts and gives me a mild headache which stops when the whine stops... When I go out into the state and regional parks around me where there are NO smart meters for miles, I no longer hear the buzzing and my heart doesn’t race” or in other cases “The noise I have in my head since smart meters is almost unbearable, sleep is at times impossible because it is so loud”<sup>49</sup>. “I became electrically sensitive almost immediately upon smart meter installation. My ears buzz, hum, and click constantly, pressure in the head and ears,... agitation and irritability all since the PLC smart meter was placed on my home... I was able to vacation where there was no smart meter installed and it felt as if a vice had been loosened from around my head”<sup>49</sup>. A post regarding a woman who removed her smart meter after becoming symptomatic repeated several times that the exposure caused her to hear “grinding”<sup>103</sup>, confirming this descriptor as among perceived RF/MW-hearing induced noises. Among those with ES who communicated with the UCSD ES Survey group; for instance, one stated that in proximity to “electrosmog producing devices, “I hear sounds like beehives and similar” (buzzing). Another stated: “The hissing in my ears is unbearable sometimes.” One wrote “annoying noise” was among other symptoms.</p>
The noises were heard primarily at night <sup>10</sup> .	Ability to hear RF/MW-induced sounds at all depends on low ambient noise <sup>45</sup> . Night is generally a time of low ambient noise.
A sound that has been recorded in Cuba and reported to be “similar” to some sounds heard is consistent with chirping of crickets or cicadas (Lederman & Weissenstein, 2017). Frey effect sounds should not be able to be recorded.	<p>Recorded sounds, if <i>similar</i> to what was “heard” by some, need not <i>be</i> what was “heard”. (Just as Frey sounds are “similar to other common sounds,” so those other common sounds can resemble the Frey sound.) The recorded sound does not cause symptoms in listeners. The sound does not fit reports by other diplomats of either the character of the sound; nor of strict sound localization (such as reports that when one moved from the bed, sound disappeared). Some diplomats had cited perceived sounds similar to crickets or cicadas: the recorded noises were reportedly very similar to the chirping of crickets or cicadas that are abundant along the Northern coast of Cuba<sup>104</sup>. Perhaps what was recorded was (or included) crickets or cicadas. Since Frey effects can sound like crickets chirping, presumably recordings of crickets chirping could resemble those Frey effect sounds.</p> <p>(Those deploying causative devices could of course capitalize on misguided sonic hypotheses to lead the US astray, by adding a recorded sound resembling Frey sounds; however there seems little need to postulate this.)</p>
There was apparent “laserlike” localization of sounds in some cases.	<p>For diplomats, “...at least some of the incidents were confined to specific rooms or even parts of rooms with laser-like specificity, baffling U.S. officials who say the facts and the physics don’t add up”<sup>10</sup>.</p> <p>One incident was described in media as follows: “The blaring, grinding noise jolted the U.S. diplomat from his bed in a Havana hotel. He moved just a few feet, and there was silence. He climbed back into bed. Inexplicably, the agonizing sound hit him again. It was as if he’d walked through some invisible wall cutting straight through his room. Soon came the hearing loss and speech problems...”<sup>10</sup>.</p> <p>In claims that “the facts and the physics don’t add up”<sup>5</sup>, it was the physics of <i>sonic</i> devices that are inconsistent. The physics of EMR is, to the contrary, compatible: lasers are themselves focused EMR. Tautologically, EMR can be focused in “laser-like” fashion.</p>
Within the room or parts of room where sounds were heard, the sound follows the listener <sup>11</sup> .	A diplomat reported that “a really odd loud noise that seemed to follow him in the room” <sup>11</sup> . Frey “sounds” are also reported to follow the person, often perceived as slightly behind the head, regardless of the body orientation relative to the source of radiation <sup>45-47</sup> . (In other cases “sounds” are perceived inside or above the head <sup>45,105,106</sup> .

**Table 2. Symptoms and Signs.**

<b>Diplomats' Symptoms and Signs</b>	<b>Compatibility with RF/MW</b>
<b>I. Auditory Symptoms are Distinctively Prominent</b>	<p>Auditory symptoms are prominent in reports of diplomats' experience, including ear pain or pressure<sup>41</sup>, sometimes within minutes of the perceived attack<sup>1</sup>, tinnitus<sup>5,6,9,10,40</sup> and hearing loss<sup>9,10,15,17,39,41, 42</sup>. This, coupled with the strange noises in diplomats' reports, likely launched the sonic theory. These idiosyncratic features are key to winnowing potential causes. Symptoms like headache and fatigue arise with many exposures and in many conditions. New onset of tinnitus and hearing loss is far more distinctive. (It is particularly so in the context of the spectrum of other reported symptoms and effects, and in the context of characteristics of instigating episodes.)</p> <p>These distinctive auditory problems are similarly prominent in people reporting symptoms from RF/MW<sup>48,51</sup>.</p> <p>Tinnitus and hearing loss were cited by 80% and 34% respectively in the UCSD survey of 202 individuals with current symptoms from EMR, with pulsed RF/MW causing symptoms in the vast majority<sup>52</sup>.</p> <p>"Initial" symptoms were reported to include tinnitus in 50%, ear pain in 30%, and hearing loss in 11%.</p> <p>Case descriptions shared by affected individuals underscore auditory effects. From the UCSD survey: "I bought a Kindle W-Fi. I charged it not realizing the default setting was 'on.' After 5-10 minutes exposure, I became nauseated, had a headache, loud tinnitus... and was dizzy. I turned the Wi-Fi off and the symptoms completely resolved in 5-10 minutes"<sup>52</sup>. A description by former educator Brinchman characterizes her abrupt development of headaches and hearing loss following introduction of pulsed RF/MW-emitting smart meters to her (and her neighbors') homes<sup>107</sup>.</p> <p>Similarly, physicians and physician groups that assessed patients with health effects from RF/MW and recognized the connection, also highlight effects on hearing. A psychotherapist in Germany with a longtime practice described a new group of patients with a physiological illness profile encompassing organic brain disease, with constellation of symptoms compatible with other reports of RF/MW injury. <i>She</i> was the one to discern the tie between patients' symptoms and their proximity to RF/MW sources (a connection that her patients had often missed – obviating nocebo effects as a source – see <b>Table 4</b>), and to note recovery with removal from those sources<sup>108</sup>. She describes "sudden hearing loss" as among the symptoms (in addition to sleep problems described as "almost ubiquitous," headache as extremely frequent, also noting fatigue, cognitive problems, tinnitus, etc)<sup>108</sup>.</p> <p>A group of 114 physicians, referencing their analysis of medical complaints of 356 people in Oberfranken, signed an Open Letter to the Prime Minister of Germany in 2004 (referred to as the Bamberg Appeal), stating "The pulsed high frequency electro magnetic fields (from mobile phone base stations, from cable-less DECT telephones, amongst others), led to a new, previously unknown pattern of illnesses with a characteristic symptom complex"<sup>109</sup>. Prominent and repeated mention is made of hearing loss: "People suffer from one, several or many of the following symptoms: Sleep disturbances, tiredness, disturbance in concentration, forgetfulness, problem with finding words, depressive mood, ear noises, <b>sudden loss of hearing, hearing loss</b>, giddiness, nose bleeds, visual disturbances, frequent infections, sinusitis, joint and limb pains, nerve and soft tissue pains, feeling of numbness, heart rhythm disturbances, increased blood pressure episodes, hormonal disturbances, night-time sweats, nausea... It is no way only a subjective sensitivity disturbance. Disturbances of rhythm, <b>hearing problems, sudden deafness, hearing loss</b>, loss of vision, increased blood pressure, hormonal disturbances, concentration impairments, and others can be proved using scientific objective measures"<sup>109</sup> (emphaes added). {Note also the mention of "ear noises" (the Frey Effect).}</p> <p>Some studies that experimentally examine effects of RF/MW on hearing show effects, though not all do. (See <b>Table 4</b> for discussion of "inconsistent" effects.) A material consideration is that evidence is consistent with a vulnerable subgroup.</p>

	<p>One experimental study in humans found that 60 minutes of close exposure to EMR from a mobile phone “had an immediate effect on HTL {hearing threshold limits} assessed by pure-tone audiogram and inner ear (assessed by DPOAE) in young human subjects. It also caused a number of other otologic symptoms”<sup>110</sup>.</p> <p>Of note, melatonin – which can be depressed by EMR (see <b>Table 4</b>) and is low in those with EHS<sup>33</sup> – protects against oxidative radiation injury (<b>Table 4</b>) – including to the inner ear<sup>111</sup>.</p> <p>Pulsed RF/MW (more than continuous) has been shown to increase tympanic temperature, even when, for instance, colonic temperature is not increased<sup>112</sup>. Since blood flow is critical for cooling, and oxidative stress leads to endothelial dysfunction and may compromise blood flow, affected individuals (see below, by hypothesis those with greater oxidative stress effects) may experience greater impairment in blood flow – so less cooling, and also, impaired delivery (via impaired bloodflow) of oxygen, glucose, other energy substrates as well as antioxidant defenses). The downstream effects of oxidative stress (e.g. apoptosis, inflammation etc – see below) and impaired cell energy/ mitochondrial dysfunction (cell dysfunction and death) may contribute to auditory pathology. In a study examining the histopathology of cochlear nuclei of rats “exposed continuously for 30days” to “a GSM-like 2100MHz EMF” “with a signal level (power) of 5.4dBm (3.47mW) to simulate the talk mode on a mobile phone,” compared to a control group of rats not similarly exposed, “an increase in neuronal degeneration and apoptosis in the auditory system” was observed in the RF/MW exposed group<sup>113</sup>. “The histopathologic analysis showed increased degeneration signs in the study group (p=0.007). In addition, immunohistochemical analysis revealed increased apoptotic index in the study group compared to that in the control group (p=0.002)”<sup>113</sup>. In another animal study, “a prominent effect of EMS {electromagnetic stimulation} was ... severe cochlear damage and permanent sensorimotor hearing loss in experimental animals”<sup>114</sup>.</p>
<b>II. Symptoms are Protean</b>	<p>Beyond the auditory symptoms, the profile of symptoms in diplomats varies from person to person: Different people report markedly different symptoms<sup>5</sup>. It was said that “The symptoms and circumstances reported have varied widely, making some hard to tie conclusively to the attacks”<sup>115</sup>; and “The cases vary deeply: different symptoms, different recollections of what happened. That’s what makes the puzzle so difficult to crack”<sup>5</sup>. Reported symptoms encompass sleep problems<sup>6,17,42</sup>, headaches<sup>6,10,15,42</sup>, cognitive problems<sup>10,42</sup>, nausea<sup>10</sup>, fatigue<sup>6</sup>, and dizziness<sup>10,15</sup>. Similar concerns had been raised with RF/MW injury. As noted by Dr. Aschermann (translated from German): “in the Deutsche Aerzteblatt (official journal of the German medical association – Bundesärztekammer) did an article ask the incredulous question: How could so many different symptoms possibly be attributed to one common underlying mechanism?”<sup>108</sup>.</p> <p>Despite the protean character of symptoms, multiple survey studies verify that a strikingly reproducible suite of protean symptoms <i>are</i> reported, in setting after setting, in people citing development of symptoms in response to EMR including RF/MW (<b>Table 3</b>). The profile of symptoms is strongly similar from study to study, with sleep/fatigue, headache, and cognitive problems commonly topping the list, auditory and visual symptoms, dizziness and nausea figuring in it.</p> <p>A similar primary list (sometimes augmented with a few additional symptoms – often including heart rhythm problems) is mentioned in other settings. The analyses of 65 patients by Dr. Aschermann cite symptoms of learning concentration and behavioral problems, headaches, insomnia, exhaustion, hearing loss, tinnitus, hearing loss, dizziness, nerve and soft tissue pain, “inner agitation”, as well as arrhythmia problems<sup>108</sup>. In the 2004 Bamberg appeal signed by 114 physicians to the then German Prime Minister, based on analysis of 356 patients: “The pulsed high frequency electro magnetic fields (from mobile phone base stations, from cable-less DECT telephones, amongst others), led to a new, previously unknown pattern of illnesses with a characteristic symptom complex. People suffer from one, several or many of the following symptoms: Sleep disturbances, tiredness, disturbance in concentration, forgetfulness, problem with finding words, depressive mood, ear noises, sudden loss of hearing, hearing loss, giddiness, nose bleeds, visual disturbances, frequent infections, sinusitis, joint and limb pains, nerve and soft tissue pains,” also nausea, and “feeling of numbness, heart rhythm disturbances, increased blood pressure episodes, hormonal disturbances,</p>

	<p>night-time sweats....The symptoms occur in temporal and spatial relationship to exposure. It is no way only a subjective sensitivity disturbance. Disturbances of rhythm, hearing problems, sudden deafness, hearing loss, loss of vision, increased blood pressure, hormonal disturbances, concentration impairments, and others can be proved using scientific objective measures”<sup>109</sup>.</p> <p>Among individuals participating in a physiological provocation study examining heart rate variability with RF/MW, among 25 patients 40% of whom believed themselves to be moderately or severely electrosensitive: “The most common symptoms of exposure to electrosmog, as identified by this group of participants, included poor short-term memory, difficulty concentrating, eye problems, sleep disorder, feeling unwell, headache, dizziness, tinnitus, chronic fatigue ...”<sup>116</sup>.</p> <p>Of note, the same symptoms also arise in the vulnerable subgroup of persons who develop health problems following <i>other</i> exposures that share a documented ability to cause mitochondrial impairment and oxidative stress<sup>55,57,117-119</sup>. However, the profile – which symptoms dominate – differs from exposure to exposure, based on factors such as what part (s) of the body the exposure may differentially reach, and whether additional mechanisms of injury are involved that potentiate damage to one domain.</p> <p>Sleep and auditory effects are clearly disproportionately represented, in diplomats and with RF/MW exposure, relative to their prevalence following other exposures that cause oxidative stress. The strong effects on sleep may relate to depressions in melatonin that can be produced with EMR/ RF/MW (see <b>Table 4</b>). Auditory effects are addressed above.</p> <p>A 1990 study commissioned in response to a petition by residents who cited adverse health experiences from a shortwave radio transmitter in their small town of Schwarzenburg, funded in part by Swiss Telecom, reported that sleep disruption in association with transmitters related directly to the EMR field strength of the transmitter, and affected 55% of those over age 45<sup>48,68</sup>. (There the denominator is <i>not</i> restricted to those who were symptomatic.)</p> <p>A 1994 Air Force Materiel Command reports that “Pulsed RF/MW radiation was reported to have an analeptic effect in animals. Experimental results presented by R.D. McAfee in 1971 showed that anesthetized animals could be awakened by irradiation from a pulsed 10 GHz RF/MW source...Experiments conducted on rats showed that these animals were aroused from states of deep sleep by irradiation”<sup>47</sup>.</p> <p>The prominence of auditory effects (see above for more on these symptoms) may relate in part to absence of a skull structure to protect the inner ear, producing an incident stimulus that is of greater effective intensity.</p> <p>The coherence of symptoms in response to RF/MW, with findings in Cuba (and China) diplomats, adds further support to the case for a common cause within each group – and across the two groups.</p>
<p><b>III. Symptoms include some that are (potentially) objectively measurable: Speech</b><sup>9,10,42</sup>, <b>Vision</b><sup>42</sup>, and <b>Balance</b><sup>10,42</sup>; and <b>Nose bleeds</b> in some<sup>9</sup>.</p>	<p>The symptoms reported in media and the Swanson article for diplomats – extending to the more specific, like dizziness/balance, vision and speech problems – are also reported in survey studies of those affected by RF/MW (<b>Table 3</b>).</p> <p><b>Speech</b> problems, mentioned in diplomats, were also among symptoms elicited and reported in a survey study examining effects of RF/MW following “smart meter” introduction in Australia<sup>48</sup>.</p> <p>Reported cases illustrate speech problems arising following RF/MW exposure. In a case referenced in the <i>LA Times</i>, a woman reported that if someone fails to turn off their cellphone on entering her home, she gets symptoms within 2 hours; ““After four hours I can’t speak anymore””<sup>32</sup>.</p> <p>In a case described in a 2015 Australian presentation on ES<sup>120</sup>: “Within hours, it felt as if someone had tied a thick rubber band around her head. Then came nausea, fatigue, ringing in her left ear – an onslaught of maladies all at once, and she had no idea why...A week or two into the job, whatever was affecting her wasn’t abating, and before long her speech became so jumbled that she couldn’t form a complete sentence in front of an audience...She went outside to inspect the place and found no fewer than 17 new ‘smart’ electricity meters strapped to the side of the building.”</p> <p>In a case reported to UCSD investigators, new onset right-sided ear pain and hearing loss attended the inciting episode (seated for six hours, unknowingly, directly across the wall from a bank of multiple smart meters for a building, slightly toward her right), along with vis-like</p>



headache, concentration problems, and two nights of no sleep (followed by chronic lesser sleep impairment), and, abating over months, continued to be triggered – always exclusively or predominantly on the right side – by previously tolerated RF/MW exposures thereafter. Many months later left ear predominant ear symptoms developed for the first time. A bank of smart meters was identified to the left of where she had sat hidden by plants so missed in an initial reconnaissance. That occasion, the only one with left predominant ear and hearing symptoms, was accompanied by speech difficulty, that resolved over about a week. In these two cases, aphasia was associated with left predominant ear symptoms. {Broca's area, damage of which leads to expressive aphasia, is left prefrontal.} It is an empirical question whether left-predominant auditory involvement will prove more often tied to affected speech.

**Balance** is multifactorial, involving e.g. vision, muscle strength, and vestibular function. In some media reports of diplomat health, the term vertigo is used<sup>40</sup>. Balance and vestibular testing were performed in diplomats<sup>41</sup>. Clinical examinations/objective measures raised concern for balance problems in 81% (higher than percent reporting subjective dizziness or balance problems)<sup>41</sup>.

Vestibular function involves the same (eighth) “cranial nerve” as hearing. Vertigo, hearing loss and tinnitus can arise (as adverse effects) as a triumvirate<sup>121,122</sup>. Dizziness more generally, in contrast to vertigo, is a nonspecific finding, that arises with many forms of brain insult – including brain hypoperfusion (low blood flow). Of note, cerebral hypoperfusion has been reported in persons with symptoms following RF/MW<sup>33</sup>.

In some surveys of RF/MW affected individuals, dizziness and balance are queried together<sup>48</sup>; other surveys only use the term dizziness. Individual reports of balance and dizziness problems were included among participant narrative reports in the Maine survey. E.g. “Balance problems have worsened since installation of the smart meter, leading to several falls”<sup>49</sup>. “I could not understand the dizziness which was scary. I actually thought I had a brain tumor all of a sudden”<sup>49</sup>. The Cuba diplomat study considered nausea as a vestibular symptom<sup>41</sup>. Though it need not necessarily be, it was linked to dizziness in some RF/MW/EMR affected cases: “Daily nausea and dizziness”<sup>49</sup>.

Loss of balance, with dizziness and disorientation, was identified as one of six clusters of symptoms seen in each of two smart meter surveys from different nations, with the clusters represented nearly in the same order (1. Sleep disruption; 2. Headache; 3. Ringing or buzzing in ears; 4. Fatigue; 5. Loss of concentration, memory or learning ability; 6. Disorientation, dizziness, or loss of balance)<sup>123</sup>.

**Vision:** Vision is affected by oxidative stress and mitochondrial impairment (see **Table 4**, mechanisms)<sup>124-129</sup>, not just to the eye but to cortical systems involved in vision<sup>130</sup>. Effects of these mechanisms include optic nerve damage<sup>129,131,132</sup>, “age” related macular degeneration<sup>125,128,133-137</sup>, retinal thinning<sup>138</sup>, and cataracts<sup>139-143</sup>. Where brain swelling ensues (see **Table 4**), this can affect the shape of the lens, affecting vision.

Effects of RF/MW on the eye and on vision have long been reported<sup>47,144-150</sup>. Particular attention has gone to effects on the lens and on cataracts. RF/MW, via oxidative mechanisms, promotes aging of the lens which can lead to cataracts. Cataracts have been a reported complication – sometimes in young people – among persons working with microwave radiation<sup>47, 144-147</sup>. A Swiss study (Hassig et al, 2012) documented increased cataracts in calves born near cell towers: “We examined and monitored a dairy farm in which a large number of calves were born with nuclear cataracts after a mobile phone base station had been erected in the vicinity of the barn. Calves showed a 3.5 times higher risk for heavy cataract if born there compared to Swiss average. All usual causes such as infection or poisoning common in Switzerland could be excluded.”

Vision problems are reported in RF/MW affected persons. In a study in Spain, in persons in proximity to two GSM (Global System of Mobile Communications) cell tower base stations, analysis of the closer group – with exposure in the range 0.25-1.29V/m2, in a model adjusted for age, sex, and distance, showed that vision problems were elevated with an odds ratio of 5.8 (95% CI 1.7-19.8, p= 0.005)<sup>151</sup>.

11% reported problems with eyes or vision in the Australian smart meter study; since this includes respondents who are unaffected, rates are lower than in purely symptomatic individuals<sup>48</sup>. 26% of survey participants reported eye/vision problems in the Halteman smart meter impacts survey<sup>51</sup>. Vision problems were reported by 17% as “severe and new,” by 38% as “moderate and new,” and by 12% as “severe and worsened” in the Maine smart meter survey<sup>49</sup>.

	<p>An assessment of neurological problems in US diplomats in Cuba underscores potential importance of eye movement dysfunction<sup>41</sup> – which is also tied to oxidative and mitochondrial mechanisms<sup>152-160</sup>.</p> <p><b>Epistaxis</b> (nosebleed): In a study in Selbitz, Bavaria, nosebleed was significantly more frequently reported (p=0.01) in those &lt;200m from a cell phone base station than 200-400m away<sup>161</sup>. Nosebleed was a reported symptom in each of several surveys of ES and symptoms associated with RF/MW, including in a study of smart meter symptoms<sup>48,49,51,52</sup> (<b>Table 3</b>). The Bamberg Appeal (on behalf of 114 physicians referencing assessment of medical complaints of 356 people with symptoms from cell tower base stations and DECT phones in their homes in Oberfranken) noted the more characteristic RF/MW symptoms (above) as well as nosebleed<sup>109</sup>.</p> <p>Comments from participants in survey studies include the following: “Severe headaches, gushing nosebleeds for the first time ever...They all went away when the smart meter was removed”<sup>49</sup>. “After the first day I was getting bloody noses and not understanding”<sup>49</sup>. Another stated “Nosebleeds, nausea, dizziness, ... ringing ears and intermittent strong agitation... When I am away from wireless devices the symptoms subside”<sup>49</sup>. And, another, “Had it not been for the severe nose bleeds I’m not sure I would ever have found out what was causing my health problems”<sup>49</sup>.</p>
<p><b>IV. Peculiar Sensory Symptoms of “Vibration” and “Pressure” Reported</b></p>	<p>“Associated sensory symptom” of “pressure” or “vibration” were reported in 43% and 14% respectively, in a neurological evaluation of diplomats<sup>41</sup>.</p> <p>The distinctive sensory symptoms of “pressure” and “vibration” are also reported by subsets of those who report symptoms from RF/MW. Neither were commonly elicited as symptoms in surveys. However, some surveys listed head pressure separately from headache, and in some cases it was more frequent. Eye pressure (Halteman, 2011) and ear pressure (Conrad &amp; Friedman, 2013) have also been reported in surveys of RF/MW/EMR affected persons. The UCSD ES survey did include “internal pressure,” which was reported as a symptom in 71% of participants who cite symptoms from EMR/RF/MW<sup>52</sup>.</p> <p>Spontaneous reports of vibration symptoms by different EMR/RF/MW affected persons, shared in a different survey study, include the following: “I experienced internal shaking and vibrating throughout my body” (along with sleep, mood, headache, head pressure, and other problems, after smart meter installation)<sup>49</sup>; “I can’t think clearly, or find words when speaking; my body feels like it is vibrating”<sup>49</sup>; and “have uncontrollable jelly-like quivering throughout whole body”<sup>49</sup>. In online comments posted in response to articles on related topics, in which persons describe their ES symptoms, statements include “vibration through my body”<sup>162</sup>; and “I have a smart meter on my house and I have been experiencing strange vibrations when I watch TV or use the computer”<sup>163</sup>. An email to us from an affected patient (9-2017) sharing her symptoms stated: “Which feels like my brain is vibrating and spinning at night – and my tinnitus gets much worse...”.</p>
<p><b>V. Brain Swelling is Reported in Some Diplomats<sup>5,9,19</sup>.</b></p>	<p>1. RF/MW may alter blood-brain barrier function via oxidative stress.</p> <ol style="list-style-type: none"> <li>An analysis reported that of 100 peer reviewed studies examining whether low intensity RF/MW causes oxidative stress, 93 found that it did<sup>60</sup>.</li> <li>Oxidative stress disrupts the “blood-brain barrier”<sup>164-176</sup>.</li> <li>Consistent with this, blood-brain barrier disruption has been shown in multiple studies with RF/MW<sup>172-174,176-179</sup>. Other studies have not shown blood brain barrier effects<sup>180-186</sup>. Studies vary in many respects (e.g. exposure duration, EMR exposure characteristics, model (in vivo vs in vitro, animal, age), delay between exposure and blood-brain barrier assessment, blood-brain barrier assessment used, etc. The blood-brain barrier is functional, and barrier function need not be affected for all substances equally.)</li> <li>Since genetics of oxidative stress management<sup>67</sup>, and levels of key antioxidants<sup>33</sup>, relate both to RF/MW injury and to oxidative stress, these factors – together with specifics of the RF/MW exposure – may guide blood-brain barrier disruption with RF/MW.</li> <li>A study that examined gene expression in the brain of rats exposed to GSM radiation, radiation that encompasses the multiple frequencies and pulsed waveforms present in GSM exposures, identified altered gene expression of a marker of blood-brain barrier function<sup>187</sup>.</li> </ol>

	<p>2. Altered blood brain barrier can lead to brain edema and “malignant brain edema”<sup>188,189</sup>. (Oxidative stress associated blood brain barrier disruption is, for instance, thought to underlie neuroleptic-induced cerebral edema<sup>190</sup>.)</p> <p>3. Perceived head pressure occurs with brain swelling, and is reported by many with ES. As also noted above in relation to the sensory symptom of “pressure,” some surveys collate head pressure separately from headache (which, in some studies, it surpasses)<sup>48,49,102</sup>; one survey included eye pressure<sup>51</sup>; and in one, several participants spontaneously reported ear pressure<sup>49</sup>. Communications to the UCSD ES study included write-in comment “brain feels like it’s swelling”<sup>52</sup>. One man with severe ES who communicated with the UCSD study group, who shared documentation of his approval for Social Security disability for his ES, reported that the severe brain swelling he experienced in response to EMR had led an eyeball to be pushed from the socket.</p>
<p><b>VI. Findings are Reported to be Compatible with Traumatic Brain Injury</b><sup>40,43,191-194</sup>.</p>	<p>1. Based on findings in an fMRI study of electrosensitive individuals it was stated: “the differential diagnosis for the abnormalities seen on the fMRI includes head injury”<sup>34</sup>.</p> <p>2. 6 of the 10 ES individuals assessed reported prior head injury<sup>34</sup>. However, four did not, and also showed evidence of brain injury. Moreover, prior head injury is reported to also be present in at least some, but an unstated fraction of, affected diplomats<sup>11</sup>.</p> <p>3. Head injury could predispose to ES: Head injury, like RF/MW, promotes oxidative stress and blood brain barrier disturbance – and melatonin (which is low in those with ES) protects from these effects in head injury<sup>195-198</sup>, as it protects against injury from radiation<sup>111,124,141,199-227</sup> – and from RF/MW<sup>228-240</sup>.</p> <p>4. One RF/MW affected who communicated with the UCSD study group indicated his ES was precipitated by a serious occupational head injury. (He also had occupational exposure to EMR, but until the head injury it had not bothered him.)</p> <p>5. Given findings consistent with low melatonin in those with ES<sup>33</sup>, this condition (and/or common cause) may also predispose to more significant damage from a given impact and character of head injury – so greater likelihood that a given head impact causes problems, and is remembered and reported as a head injury.</p> <p>6. The study did not report presence/absence of features indicative of greater severity of head injury – such as loss of consciousness, or symptoms or sequelae. Both because of this and point 5, there is not clarity about whether prior head impacts were in fact greater in number or intensity than in the general population. But as above, it might be expected that past head injury would be a risk factor.</p> <p>7. ES symptoms are sometimes experienced as similar to a head injury. For instance, an affected Rhode Island teacher likened effects experienced with RF/MW to a concussion<sup>241</sup>.</p> <p>Just as it is important to avoid even minor head trauma following traumatic concussion, until healing has occurred, so avoidance of RF/MW (or more generally EMR) aggravation may prove important following pulsed RF/MW injury: radiation injury may be cumulative and in addition to the intensity-duration profile, the interval between exposures may be important in the clinical course<sup>145</sup>.</p>
<p><b>VII. White Matter Abnormalities are Reported</b><sup>44</sup> in some diplomats.</p>	<p>In diplomats: “Medical testing has revealed that some embassy workers had apparent abnormalities in their white matter tracts that let different parts of the brain communicate”<sup>244</sup>.</p> <p>1. White matter changes were observed in some with ES, in the fMRI study of persons affected by RF/MW/EMR<sup>34</sup>.</p> <p>2. Oxidative stress and mitochondrial dysfunction (to which RF/MW can contribute, <b>Table 4</b>) are associated with white matter injury<sup>242-248</sup>. Among potential mechanisms, oxidative stress increases vulnerability of proteins (and lipids, DNA, RNA, etc) to autoimmune attack, which can include attacks on myelin<sup>249-258</sup>.</p> <p>Indeed, antibodies directed to O-myelin were reported in a subset of the 675 persons with ES that were included in a French study<sup>33</sup>, affirming one mechanism by which white matter changes might occur.</p> <p>3. Following GSM radiation exposure (study cited previously), examination of gene expression in rat brain showed alterations in myelin-related products (myelin-related glycoprotein)<sup>187</sup>.</p>

**Table 3. Symptoms in Diplomats: Comparison to Symptom Rankings in Survey Studies that report symptoms with EMR, or in those with ES.**

Percentages are given for diplomats (chosen for being symptomatic); and rankings for studies of persons reporting symptoms with EMR/RF/MW (not restricted to acute stage).

	<b>Cuba Diplomats</b>	<b>Australia</b> 2014	<b>US</b> 2011  Wireless Utility Meter Safety Impacts Survey	<b>US*</b> 2013  Maine Smart Meter Health Effects Survey & Report	<b>France</b> 2002	<b>Japan</b> 2012	<b>US*</b> 2015	<b>Nether- lands</b> 2007	<b>Sweden</b> 2006	<b>Finland</b> 2013	<b>Turkey</b> 2017
Citation	Study of diplomats <sup>41</sup>  News media	Lamech <sup>48</sup>	Halteman <sup>51</sup>	Conrad <sup>49</sup>	Santini <sup>259</sup>	Kato <sup>50</sup>	Golomb <sup>52</sup>	Schooneveld <sup>102</sup>	Johansson <sup>37</sup>  Cites Swedish Language article <sup>260</sup>	Hagstrom <sup>29</sup>	Durusoy <sup>261</sup>
EMR- or ES-related characteri- stic	N/A	Smart meter exposure	Smart meter exposure	Smart meter exposure	Proximity to cell phone base station	ES	ES	ES	ES, acute phase	ES, acute phase	Cell phone use – symptoms during
Sample characteri- stics	~24 US and 2 Canadian diplomats to Havana reporting symptoms attributed to “health at- tacks” in news; (24 US embassy community members with neurological findings often seen after mild traumatic brain injury/concussion <sup>41</sup> )	92 Residents of Victoria, Australia after exposure to smart meter radiation	318 US Responde nts from 28 states	210 Respond ents, 68% ES (142) †	530 People living near cellular phone base stations	75 Japanes e with ES or sensitiv e to EMF	202 Persons with current ES	250 Dutch responde nts with ES	22 with ES ranked symptoms. Most common were listed (not ranked)	194 with ES	2150 students in 26 high schools in Turkey.
<b>All have symptom</b>	Yes	No	No	No	No	Yes	Yes	Yes	Yes	Yes	No

<b>s</b>											
<b>Symptom Rankings</b>				Two rankings given: for severe <i>or</i> moderate and new/severe and new							
Sleep	(86%) 41  Also: <sup>6</sup>	#1	#1	#4/ #1	#3	#4  76%	#1  94%	#5	Yes	#2	#6
Headache	(81%) 41  Also: <sup>5,6,15</sup>  Also: (Lederman, Weissenstein, Lee et al., 2017; Panetta, 2017; Robles & Semple, 2017b)	#2	#3	#1/ #3  (pressure in head; headache is listed separately and would be #5/#5	#2	#2  81%	#2  88%	#7, #9, #10 (separated into 3 questions; #10 is pressure in head; #7 is numb feeling in head)	Yes	#4	#2
Cognitive	(81%) 41  Also: <sup>6,19,262</sup>	#5	#5	#2/#4	#4, #7	#3  81%	#3  85%	#2, #13	Yes	#7,#10	#4,#5
Stress anxiety irritability	67% irritability; 57% nervousness; 52% more emotional; 29% sadness  41	#11	#2	#8/#7  (agitation)	#6 (irritability)	#9 & #10, for “irritation” and “anxiety”  56% & 55%.	#6 in “initial symptoms”, irritability  45%				



Tinnitus	(57%) 41  Also: <sup>5,6</sup>	#3	#4	#3/ #2	Not queried (except as “hearing”)	#7  63%	#5;  80%	Not in main list, but by # affected in auditory list, #13		Not queried	
Fatigue	Not elicited ‡ 41  Mentioned in news media <sup>6,41</sup> )	#4	#6	#10/#9	#1	#1 (and possibly #5 “sluggish in the head”) 85%	“Exhaustion” was a write-in symptom (Not queried)	#1	Yes	#6	#1
Dizziness or balance	(67%)§ 41  Also: <sup>5,6,15</sup>	#7	#7	#7/#7	#14	#6  64%	#4 Initial  49%	#11	Yes	#12	#9
Vision problems	(76%) 41  Also: <sup>42</sup>	#12	#8	#10/#11	#12	---	#8 in Initial Symptoms 38%	#6	----	#13 (photosensitivity)	#10
Nausea	<sup>5,6,9</sup>	#9	#12	---	---	---	#9 “gastrointestinal symptoms” (64%) (Nausea not separately asked)	---	Yes (“symptoms from the gastrointestinal tract”)	#20	#15
Epistaxis (nose bleed)	not elicited 41  Mentioned in news/	#17	#13	#15 in symptoms that intensify	----	---	“Nosebleeds” as a write-in symptom	--- (#12 is “nose problems”	----	----	----

	media: <sup>9</sup>			d. New onset in several write-ins.			(not queried)	)			
Hearing loss	(43%) 41 Also: <sup>6,9,15,17,39</sup>	#18 (with ear pain)	---	---	#5	---	#11 34%	#3	---	---	#14
Speech problems	<b>Not elicited ¶</b> 41 Mentioned in: <sup>9</sup>	#30	---	---	---	---	**	---	---	---	---
Comment			††	‡‡	§§		¶¶				

---- = Not queried

Surveys in the smart meter era were prioritized for inclusion; proximity of emitting devices to homes may make these more comparable to diplomat experience. Studies of ES were also prioritized, as these focus on those who are symptomatic, providing symptom rates better suited for comparison to those in affected diplomats. Other studies on similar themes report similar findings.

(An exception is that older studies from Scandinavia that focused on exposure to video display terminals from that time, report high rates of skin problems.)

For instance, a 2007 study of 85 persons living nearby the first mobile phone station antenna in Menoufiya governorate, Egypt reported that “The prevalence of neuropsychiatric complaints as headache (23.5%), memory changes (28.2%), dizziness (18.8%), tremors (9.4%), depressive symptoms (21.7%), and sleep disturbance (23.5%) were significantly higher among exposed inhabitants than controls: (10%), (5%), (5%), (0%), (8.8%) and (10%), respectively (P < 0.05).” Sleep, headache and cognitive again topped the list in frequency<sup>263</sup>.

Some studies focus not on ranking, but dose-effect/distance relation. For instance, in Selbitz, Bavaria, those within 200m of a cell phone base station were compared on reported symptoms to those 200-400m away, and were found to report significantly more sleep problems, headache, concentration problems, “cerebral affections”, depression, auditory/vestibular problems, visual problems, GI problems, dizziness, and nosebleed – also cardiovascular problems, joint problems, infections and skin problems “(p = 0.01” for dizziness and nosebleed, “p=0.001” for the rest)<sup>161</sup>. A 2003 survey study of the “microwave syndrome” “in Murcia, Spain, in the vicinity of a Cellular Phone Base Station working in DCS-1800MHz,” reported that symptoms included fatigue, irritability, headache, nausea, insomnia, depression, discomfort, difficulty in concentration, memory loss, visual dysfunction, auditory dysfunction, dizziness (as well as several other symptoms)<sup>24</sup>. These were more prevalent within 150m of the station, relative to >250m, in most cases significantly so. It was noted that symptoms abated with removal from the RF/MW source<sup>24</sup>. A follow-on study examining rates of problems in relation to measured electric fields, and showed significance for 13 of 16 assessed symptoms, with symptom odds ratios as high as 59<sup>151</sup>.

Our rankings do not include as a symptom, “Onset of Electromagnetic Hypersensitivity Syndrome” or “Aggravation of Electromagnetic Hypersensitivity Syndrome”. We used the highest ranking if several cognitive queries were used (e.g. memory problems or concentration difficulties), or several head queries are used (e.g. headache, head pressure, heat or strange sensation in head), and exclude later exemplars of the category in ranking the lower ranked items.

\* There was no barrier to participation from outside the US, but participants are predominantly from the US

† 68% of participants had ES (N=142) of whom 63% felt certain their exposure to smart meter was responsible for initiating the ES. Of the 49 who were ES before smart meter exposure, all 49 (100%) stated that smart meter exposure made their ES not only worse but “much worse”

‡ Though fatigue was not elicited, it is noted that a number reported a “good day bad day” pattern in which mental or physical exertion on one day led to exacerbation for

several days.
§ Separates out balance (67%), dizziness (63%) and includes nausea (7%) in this category.
¶ Speech problems were not elicited but speech audiometry, speech therapy, speech pathology consultation are each mentioned totaling at least six references.
** Aphasia” was a write-in symptom (not queried).
†† 73% women; 93% over age 40; 43% over age 60; 78% from California; 49% characterize selves as EMF sensitive.
‡‡ The 1st number is severe <i>or</i> moderate and new; 2 <sup>nd</sup> number is severe and new. Pressure in head and headaches were queried separately. The overlap is uncertain. The higher ranking (pressure in head) was used. Concentration and memory were queried separately. The overlap is uncertain. The higher ranking (concentration problems) was used.
§§ Memory and concentration were queried separately, ranked #4 and #7 in the original. Combined might be higher. The higher ranking is used. This analysis provides values at different distances. Orderings for the closest distance are used. Ordering shifts slightly with longer distances but in general, the more frequently reported symptoms remain the more frequently reported.
¶¶ Ratings are based on (videotaped) Commonwealth Club slide presentation. Additional symptoms were elicited but not presented.
Notes buzzing ears, hissing sounds, loss of hearing, strong low frequency sounds, ear aches, and sound of bells clanging in 96, 80, 64, 545, 38, and 28 participants
This assesses acute symptoms. It also gives fractions who report those symptoms before the acute phase, but it is unclear whether someone who reports a symptom (say, headaches, dizziness) before exposure, had those symptoms only occasionally.

**Table 4. Mechanism Considerations.**

<b>Oxidative Stress – mediated by free radicals – is involved in RF/MW injury</b>	<p>Oxidative stress refers to a kind of injury against which “antioxidants” relatively protect, in which “reactive oxygen species” or “free radicals” produce changes/damage that can affect, for instance, lipids, proteins, DNA, and RNA.</p> <p>Mitochondria, which are the primary source of energy for cells (and regulate many other phenomena such as steroid hormone production and apoptosis) are a leading source and target of oxidative stress<sup>59,264-267</sup> – that is, mitochondrial injury not infrequently accompanies oxidative stress, and has been shown with RF/MW (see below).</p> <p>RF/MW produces oxidative stress. As above, in an analysis of 100 studies examining if low-level RF/MW produced oxidative injury, it was reported that ~93 found that it did<sup>60</sup>.</p> <p>Oxidative stress – and mitochondrial dysfunction are implicated in the symptoms and health effects that have been reported by diplomats (and RF/MW affected persons)<sup>127,138,139,268-300</sup>.</p> <p>For instance, oxidative stress is tied to tinnitus, antioxidants modestly alleviate it, and markers of oxidative stress in tinnitus are reported to be greater in jugular blood (near the ear) than the more commonly measured brachial blood<sup>269,270,301</sup>.</p> <p>Two findings substantially cement a role for oxidative stress in RF/MW health effects.</p> <p>First, persons who are “electrosensitive” (i.e. who experience symptoms at levels of radiation than many others tolerate) are significantly more likely to harbor gene variants that confer less-avid protection against oxidative injury<sup>67</sup>. This is an extremely important finding. People cannot manipulate their genes in response to suggestibility, and did not know their genes when they reported their sensitivity status. This powerfully supports a causal role for oxidative stress in the injury experienced.</p> <p>Second, a French study in electrically and chemically sensitive individuals (93% with ES), found <i>consistently</i> low levels of a urinary melatonin metabolite<sup>33</sup>. Since melatonin is an antioxidant that protects against damage to many toxins – but that has been shown in numerous studies to be <i>particularly</i> vital for defense specifically against oxidation injury <i>due to radiation</i> across the electromagnetic spectrum<sup>111,124,141,200-208,210,211,213,214,216-222,224,225,227,302,303</sup>, including due to RF/MW<sup>228-239,304</sup>, this dovetails with the aforementioned genetic data to compellingly support a role for oxidative stress – and to show that those with ES – those who experience <i>symptoms</i> with radiation that others tolerate – are also experiencing <i>greater cellular and subcellular injury</i> from this radiation.</p> <p>Many studies show the importance of antioxidant defenses – including but not limited to melatonin – in protection against RF/MW injury. For instance, melatonin and to a lesser degree caffeic acid protect against cell phone induced oxidative stress in rats – and melatonin increased activity of other endogenous antioxidant enzymes, superoxide dismutase (<b>SOD</b>), glutathione peroxidase (<b>GPx</b>) and catalase which were depressed with the cell phone radiation<sup>236</sup>. Melatonin protected against laryngotracheal oxidative injury from wireless (2.45 GHz) radiation in rats<sup>229</sup>. Melatonin protected against skin oxidative injury in an experimental mobile phone model in rats<sup>228</sup>. Melatonin protected against 900MHz microwave radiation induced lipid peroxidation in rats<sup>230</sup>. Melatonin reversed the oxidative damage of microwaves to rat testes – including protecting testosterone level, sperm count, and protecting against DNA fragmentation (a marker of cell death)<sup>232</sup>. Melatonin protected against oxidative damage from cell phone radiation to rat brain<sup>238</sup>. Melatonin protects against oxidative damage from Wi-Fi to lens of rats<sup>239</sup>. Vitamins E and C protect against “900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium”<sup>305</sup>. Ginkgo biloba protected against cell phones induced oxidative injury in rat brain<sup>306</sup>. And so on.</p> <p>Antioxidants work together, for instance, to recycle one another to the reduced form in which they are active as antioxidants.</p> <p>The importance of antioxidant defenses in protection against radiation injury from RF/MW, extends what is well known for injury from radiation throughout the electromagnetic spectrum, including so-called “ionizing radiation” (which includes gamma for instance, “A positive correlation was found between GPx activity, glutathione content and cell survival following ionizing irradiation”<sup>307</sup>. Glutathione depletion increased with gamma radiation induced DNA damage<sup>308</sup> and cell death<sup>309</sup>. Glutathione determined the survival “shoulder” for x-ray radiation in hypoxic cells<sup>310</sup>, and melatonin</p>
---	--

	<p>protected against x-ray induced lung injury<sup>217</sup>. Melatonin protected against radiation induced cataract<sup>141</sup> – and increased activity of other critical antioxidant enzymes, SOD and GPx. SOD protected against fractionated radiation induced esophagitis (and reduced the effect of that radiation on glutathione)<sup>311</sup>. Melatonin protected against UVB radiation-induced oxidative skin injury<sup>222,223</sup>; as did glutathione<sup>312</sup>, and chocolate, which is rich in antioxidant polyphenols<sup>313</sup>. Melatonin has specifically been reported to protect the inner ear against radiation injury, in rats exposed to “radiotherapy” at 4-6KHz<sup>111</sup>.</p> <p>A role for oxidative stress in radiation injury transcends labels of “ionizing” vs “nonionizing”, “thermal” vs “nonthermal” radiation. For this reason, those labels are of questionable utility in understanding radiation damage.</p>
<p><b>Radiation may depress melatonin – moreso in some – and in part through depressed melatonin, may depress other antioxidants</b></p>	<p>A number of studies report that EMR, including but not limited to RF/MW, can depress melatonin<sup>302,314-322</sup>. Evidence suggests that (like virtually all biological effects), a subgroup is more vulnerable<sup>323,324</sup>. {Note that sunlight, which provides EMR of a kind “expected” evolutionarily, is well recognized to govern (depress) melatonin, toward producing day-night and seasonal effects.}</p> <p>Light (a portion of the electromagnetic spectrum) inhibits melatonin as part of establishing circadian and seasonal rhythms<sup>325-327</sup>.. Evolution did not plan for man-made radiation sources, and one hypothesis is that, in some people, such radiation sources may induce similar effects.</p> <p>“EMF {electromagnetic fields} are known to affect Ca2+ homeostasis and suppress melatonin activity in a wide wavelength range. Ca2+ ions in pinealocytes are involved in regulation of cAMP synthesis that mediates conversion of serotonin into melatonin. Their leakage from pinealocytes results in a decrease of the cAMP level and thereby suppresses production of melatonin”<sup>328</sup>. Long-term radar workers reportedly have increased serotonin and depressed melatonin, consistent with this impaired conversion – and effects in the RF/MW frequency range<sup>329</sup>. Electronic repair workers have also been reported to have lower melatonin than controls, and more sleep problems<sup>330</sup>.</p> <p>Melatonin (and its derivatives) – though better known for effects on sleep – provide a critical antioxidant defense system that protects against toxicity of an extraordinary array of toxins and conditions<sup>274,331-355,356-382,383-416</sup>.</p> <p>For this reason, to the extent that EMR does depress melatonin, it is expected to potentiate the array of adverse health outcomes tied to these toxins, and other sources of injury.</p> <p>Melatonin specifically protects against radiation injury at frequencies across the electromagnetic spectrum<sup>111,200-202,205,206,208-210,212-214,216-220,223,225,226,230,233,238,239,304,417,418</sup>.</p> <p>A study examining gene expression in rat brain reported that brain expression of N-acetyltransferase-1, the rate limiting enzyme in melatonin production<sup>419</sup>, had significantly reduced expression following 915 MHz GSM-consistent RF/MW radiation (encompassing pulsed RF/MW) in rats, fold difference <math>0.48 \pm 0.13</math>, <math>p &lt; 0.0025</math><sup>187</sup>.</p> <p>Suppressed melatonin or sleep deprivation in turn increase damage to the pineal gland<sup>420</sup>, which produces most of the circulating melatonin. Thus, sufficiently depressed melatonin can beget still further depressed melatonin – and heightened vulnerability to injury from future EMR exposure.</p> <p>Ability to sustain adequate melatonin production in the face of EMR/RF/MW, <i>may</i> be a critical determinant of pineal vulnerability. The pineal gland has high antioxidant needs<sup>420,421</sup>, and in absence of such protections is vulnerable to involution<sup>422,423</sup>.</p> <p>Age-related involution of the pineal gland may help to explain why more middle-aged persons are reportedly affected by ES than younger people<sup>424</sup>, though presumably younger adults may be more exposed to technology. {Middle-aged persons may, however, have had more years of EMR exposure.}</p> <p>Melatonin supports levels and activity of other antioxidants, including in the setting of radiation exposures<sup>141,236,239</sup>. Modest exposure to oxidative stressors (including from radiation) in persons or animals or plants whose system is not overwhelmed, can lead to antioxidant upregulation – a phenomenon called oxidative preconditioning, seen with many sources of limited oxidative stress, including limited exposure to radiation<sup>425</sup>. In part because of this, the net effect of an oxidant exposure on antioxidant levels depends on factors like intensity and duration of exposure, other oxidative exposure (so, mitochondrial dysfunction state), and status of antioxidant defenses, as well as time from exposure to assessment. Some studies in some</p>

	<p>systems show antioxidant upregulation<sup>426</sup> or mixed direction effects on different antioxidants<sup>239</sup>, but many show depression of assessed antioxidants following EMR exposures<sup>222,223,427,428</sup> or specifically RF/MW exposure<sup>235,236,237,239,305,429-435,436</sup>. Such depressions, coupled with melatonin depressions, may enhance vulnerability to future EMR exposures – particularly where genetics provide for less effective variants of one or more antioxidants<sup>67</sup>. It is expected that mitochondrial impairment<sup>59,264,266,267</sup> or brain inflammation (sometimes itself a result of oxidative stress, amenable to reduction with melatonin<sup>225,437</sup>), since associated with greater production of free radicals and an expected less favorable balance of oxidative stress to antioxidant defenses, may be a risk factor for problems with the added oxidative stress from RF/MW, or from the depression in antioxidant defenses to which RF/MW may contribute.</p>
<b>RF/MW may depress xenobiotic protections</b>	<p>RF/MW has been reported to depress butyrylcholinesterase<sup>438</sup>, an important xenobiotic defense enzyme. Depressed activity of this enzyme is tied to higher cardiovascular and all-cause mortality<sup>439</sup>.</p>
<b>Oxidative Stress contributes to Auxiliary Mechanisms of radiation injury, such as Mitochondrial Dysfunction.</b>	<p>Oxidative stress contributes to multiple documented auxiliary mechanisms of RF/MW damage that likely contribute to health effects in subsets, including membrane alterations (cell membranes<sup>440</sup> and mitochondrial membranes<sup>441-444</sup>), blood brain barrier disruption<sup>166,168-170,172,173,445-449</sup>, effects on voltage gated calcium channels<sup>450</sup> (affected by and affecting oxidative stress<sup>451,452</sup>), but also on voltage gated anion channels that are an important part of the outer mitochondrial membrane<sup>453</sup> – potentially contributing to mitochondrial impairment and amplification of oxidative stress, EEG spiking<sup>233</sup>, impaired mitochondrial function<sup>240,454</sup> (bidirectionally related to oxidative stress<sup>58,455,456</sup> – and protected by melatonin<sup>457</sup>), impaired blood flow (e.g. via oxidative stress driven endothelial dysfunction)<sup>458-461</sup>, autoantibodies<sup>249,255,462-466</sup>, and apoptosis<sup>467-476</sup> (programmed cell death – which in turn triggers inflammation and coagulation activation<sup>477</sup>). Laboratory correlates for some of these were reported in ES participants in the French study: ~15% of those with ES had elevated markers of blood-brain barrier permeability; 29% in those with ES (23% in those with ES and multiple chemical sensitivity (MCS)) had antibodies to O-myelin<sup>33</sup>.</p>
<b>Melatonin considerations : RF/MW/EMR vs diplomats</b>	<p>While depressions in a melatonin metabolite were the norm in participants with ES in a French study<sup>33</sup>, this need not <i>necessarily</i> be the case for diplomats, even if a related cause (pulsed RF/MW) and related processes (e.g. tied to oxidative stress) are involved in symptom induction. In persons with “ES,” lowered defenses are needed, for nominally “modest” exposures to produce problems. But if exposures in affected diplomats were more intense or otherwise injurious, lowered defenses would not be required to produce injury. To assess this, it may be prudent to assess urine melatonin metabolites at the time diplomats are identified with symptoms.</p>
<b>Psychogenic illness has been dismissed</b>	<p>Psychogenic causation has been repeatedly suggested as the basis for diplomats’ symptoms<sup>13,14,478</sup>. This has been correctly dismissed, however, for the Cuba and China diplomats<sup>11,40,41</sup>. Psychogenic causation has similarly been suggested for symptoms from RF/MW<sup>479</sup> and has been similarly repudiated<sup>108,480</sup>. The Swiss Telecom funded study that documented a relation of sleep problems to transmitter field strength, also showed that symptoms were not related to a health-worrying personality<sup>48,68</sup>. The concordance of symptom profiles across studies, the emergence of RF/MW problems in people unaware of the exposure or its potential for problems, the concordance of symptoms and objective signs with known documented mechanisms of RF/MW injury, the presence of objective markers and ties to genetics that each cohere with known mechanisms of RF/MW injury<sup>33,67,116</sup> effectively preclude a psychogenic basis for the problem – were such a diagnosis meaningful. {See below, in the entry for study inconsistency, for provocation studies.} The notion that chronic symptoms can arise from psychogenic sources dates to Freud, who also pioneered the flaws associated with its application<sup>481</sup>. The foundation is substantially circular, a mechanism has never been physiologically defined or substantiated (much less documented to be operating in cases where the label is applied), and the label is deployed without the most basic scrutiny of the tacit assumptions<sup>482</sup>. Historically, many conditions that</p>



	were presumed psychogenic (such as ulcers, seizures) were recognized as organic as evidence emerged <sup>482</sup> .
<b>Not all are affected – a minority of embassy personnel R, and of RF/MW exposed</b>	<p>How might some people experience symptoms and signs of injury from what seem to be “low levels” of an exposure, seemingly well below levels that other people tolerate? For toxins we designate an “LD50”<sup>483-488</sup> (dose lethal in 50%) – or an LD5. This reflects the recognition that for each potentially toxic exposure, there is a range in which some will experience an outcome and others will not. One can also define an SD50 (symptoms in 50%) – or an SD25, or SD5. It would be surprising if a highly useful and lucrative technology were <i>not</i> pushed as far into this intensity range as possible. Genetic variations in a range of free radical detoxification systems, competition for those systems, alterations in gene expression based on prior exposures, differences in vulnerability of the tissue affected (via factors like mitochondrial “heteroplasmy,” past injury of that organ), and variations in secondary mechanisms triggered by oxidative stress, provide among the mechanisms by which variability is produced.</p> <p>The de facto intensity of the “same” exposure may differ radically (no pun intended) from person to person †. A further mode of variability arises from immune activation. Considering a more familiar “allergen,” one person can eat a jar of peanut butter without problem; while another is hospitalized for exposure to a crumb of peanut. As above, oxidative stress can modify substances in a fashion that makes them vulnerable to autoimmune attack. Immune/autoimmune activation is a documented feature in a subset of those citing symptoms from RF/MW/EMR<sup>33</sup>.</p>
<b>Effect modification</b>	<p>“Effect modification” refers to differences in effect in different individuals, and it is the rule rather than the exception in biology. Particular considerations are germane when the exposure has potential for prooxidant or antioxidant effects<sup>489</sup>. Many prooxidants can be antioxidant at low doses in some people (via “oxidative preconditioning” in which low level exposure to prooxidants may upregulate native antioxidant defenses; this can lead to net antioxidant effects in persons whose defenses are not already overwhelmed or maximally upregulated – as above). Conversely, many substances thought of as antioxidants are prooxidant in some settings, often including high dose<sup>490-498</sup>. So the same exposure can produce even opposite direction effects in different persons. Exemplifying the principle, statin cholesterol-lowering drugs are net antioxidant in many people (often tested in nonelderly males without metabolic syndrome factors), but are reproducibly prooxidant in a subset – and prooxidant dominance is tied to side effects<sup>499,500</sup>. These side effects (attended by net prooxidant effect<sup>499,500</sup>) arise disproportionately with higher doses, and in persons with conditions like older age and metabolic syndrome factors, that are statistically tied to mitochondrial impairment<sup>56</sup>. Side effects, too, occur disproportionately in women<sup>56</sup>. Women show higher rates of adverse effects from many drugs and environmental toxins (and many medical procedures); they are also more often affected by EMR<sup>91,102,259,424,501</sup>.</p> <p>There are many potential sources of effect modification from genetics (as has been documented<sup>67</sup>), level of exposure, and past and current environment that influence biology. Some exposures may cause mitochondrial injury or oxidative stress (competing for antioxidant defenses) or depress concentrations of antioxidants, boosting vulnerability. Other exposures may have protective effects.</p>
<b>Chemical exposures may serve as one source of effect modification</b>	<p>Many drugs and chemical exposures cause oxidative stress, cause mitochondrial injury (which also increases intracellular oxidative stress), depress antioxidant defenses, and/ or compete for or inhibit detoxification systems. Through these and other mechanisms, these exposures may magnify harm from RF/MW and vice versa. Preliminary evidence comparing chemical levels in Swedish persons with ES vs controls identifies higher levels of some organic pollutants in those with ES<sup>26</sup> – though larger studies are needed.</p> <p>Melatonin and glutathione (and other antioxidants) can be “radioprotective”<sup>204,307,502,503</sup> (here the root “radio” refers to radiation, not specifically to radiofrequency radiation). Other agents or conditions can be “radiosensitizing.”</p> <p>As might be expected, glutathione depletion can be radiosensitizing, though the status of other antioxidants may be important<sup>504-507</sup>. The tie between low melatonin (assessed by the principle metabolite) and ES in the French study<sup>33</sup> supports the expectation that melatonin depletion is radiosensitizing as well. Radiosensitization is used therapeutically, to enhance killing by radiation of tumor cells<sup>508</sup>, but its existence there is a reminder that chemicals interact with radiation to modify radiation effects. Radiation itself may be radiosensitizing – as potential effects on antioxidant systems, reviewed</p>

	<p>elsewhere, suggest – and reportedly ultra high frequency radiation is a particularly effective radiosensitizer<sup>509</sup>. Oxidative stress is an important, but not the only, means by which radiosensitization occurs<sup>510</sup>, consistent with multiple downstream mechanisms of injury.</p> <p>(Of note, because critical systems that are involved in radiation defense – like melatonin, glutathione, and other antioxidant systems – are also involved in defense against toxicity of chemicals and drugs<sup>511</sup>, and because factors that adversely affect antioxidant:oxidant balance may be adverse for oxidative stress mediated injury from either type of source, it is expected – as it is observed – that there will be overlap between chemical and electrical sensitivity<sup>33</sup>.)</p> <p>Two illustrations where we can <i>see</i> the radiosensitizing effect occur with ultraviolet (<b>uv</b>) light, since due to its high frequency, the effect is primarily on the skin. Photosensitizing agents and “radiation recall” are the illustrations.</p> <p>Photosensitizing or phototoxic or photoallergic agents are agents that magnify damage observed with uv radiation. (For simplicity we will use “photosensitizing” to encompass each of these.) In some cases, radiation breaks down a chemical to something toxic. Drugs may also photosensitize, for instance, by augmenting one of the mechanisms of radiation injury, such as oxidative stress or mitochondrial dysfunction<sup>512</sup>. Fluoroquinolone antibiotics, which can cause serious problems in a vulnerable subset through oxidative stress and mitochondrial dysfunction<sup>55</sup>, are strongly reported to photosensitize and to be phototoxic<sup>513-531</sup>. Fluoroquinolones have been tied to development of <i>persistent</i> phototoxicity (following withdrawal of the drug)<sup>532</sup> – i.e. ongoing higher vulnerability to this radiation – consistent with evidence that a vulnerable group experiences persistent damage from fluoroquinolones in which oxidative stress and mitochondrial injury play a role<sup>55</sup>. This “vulnerability” may be acquired, as mitochondrial injury can be cumulative, and a serious reaction sometimes follows a previous course of fluoroquinolones with a milder and time-limited reaction or none at all<sup>55</sup>. (Mitochondrial injury from radiation can also be cumulative<sup>533</sup>.) Fluoroquinolones have led to reported “photosensitivity” reactions to fluorescent lighting<sup>534</sup>. Statins, which as elsewhere are sometimes prooxidant<sup>499</sup> and sometimes mitochondrially toxic<sup>56</sup>, are also sometimes linked to photosensitivity<sup>535,536</sup>. (The below information about photosensitivity in Smith Lemli Opitz explains one reason that statins can be prooxidant, though they also have antioxidant mechanisms.)</p> <p>Given oxidative mechanisms of radiation injury that apply across the electromagnetic spectrum, it is expected that some agents that photosensitize may sensitize to other forms of radiation – potentially including RF/MW. Others have noted that photosensitizing drugs have played an apparent role in other radiation injury<sup>537</sup>. (Data we have presented, but not published, showed that past use of fluoroquinolones was significantly tied to development of ES; past adverse effects to fluoroquinolones, which signify oxidative-mitochondrial injury to a point producing symptoms (at least, they surpassed the symptom threshold for a time), showed a particularly strong connection<sup>52</sup>.)</p> <p>There are also disease conditions tied to magnified photosensitivity<sup>538</sup>. Where these are tied to depressed antioxidant defenses, or increased mitochondrial injury, they might be predicted to be tied to increased risk of ES development (accounting for radiation exposure). In Smith Lemli Opitz syndrome, which many studies have tied to photosensitivity, cholesterol levels are low<sup>539-550</sup>. Cholesterol transports critical fat soluble antioxidants<sup>56</sup>.</p> <p>In the phenomenon of “radiation recall,” injury to tissue initially caused by radiation can be made to reappear by another agent with shared mechanisms of injury, e.g. oxidative stress and mitochondrial injury – such as fluoroquinolone antibiotics – best recognized for skin reactions, since we are able to see these<sup>551-553</sup>.</p>
<b>Are provocation studies contributory?</b>	<p>Several so-called provocation studies have been conducted in persons with ES; some focus on symptoms, some on objective markers. In most of those that focus on symptoms, those with ES fail to reliably distinguish between blinded EMR “exposed” and “unexposed” settings<sup>554</sup>. Major flaws in the designs have been recognized and reviewed by others<sup>96, 102</sup>: for instance, studies assume that the details of exposure and time course do not need to be individualized, which is contrary to the evidence.</p> <p>But there are further problems. The most fundamental is the assumption that in ES, symptoms serve as a meter; this is invalid. Consider the analogy of sunburn – a form of radiation injury mediated by oxidative stress, that affects some but not others at usual exposure levels. Those who are affected “believe” sun exposure is responsible. They would be unlikely to discern when they are being exposed vs not to ultraviolet radiation. (It is their failure to</p>

know when significant injury is occurring, or has occurred that leaves them in the sun long enough to receive injury.) What is discerned is the inflammation (that follows the oxidative stress) that may only emerge late in exposure, or after the sun exposure has been “withdrawn.” A blinded sham-exposed study would likely also produce inability to discern sham from active treatment.

People do not sense the EMR, but the effects produced by it, and studies show that those with ES respond to different EMR sources. In RF/MW-affected persons, as in diplomats, the effects can arise after hours of exposure, or hours after a short exposure – oxidative stress can cause apoptosis and can then trigger inflammation<sup>477</sup>, or can cause blood brain barrier damage allowing brain swelling (see above) – progression of these mechanisms may not peak for hours or in some cases, even a couple of days. Recovery from effects can take still longer.

For such a study to have a chance to succeed, it would be essential to pretest and individualize both the control/negative exposure condition, and the active/positive exposure condition (including exposure and time course) in each individual, to define a condition that will be effective in that person – if such conditions can be successfully defined, and if cumulative effects don’t alter the condition from one trial to the next. For some people the background EMR at the facility, or its parking lot or lobby; or the exposure during transit to the facility may obviate ability to define a negative exposure condition for that individual. It would be better to bring the EMR exposure to a place where the affected party is stable and asymptomatic. And the specific EMR and timing must be individualized to produce a positive condition, in a suitable time course.

To be valid, such a study must also protect against the possibility of physiological conditioning effects. These are distinct from “nocebo” effects, and arise because the true stimulus produces actual physiological harm: It is known, for instance, that chemotherapy patients may vomit when they enter the room in which they have received chemotherapy. (Chemotherapy agents, like EMR, also cause toxicity via oxidative stress<sup>344, 555-557</sup> and mitochondrial injury<sup>558</sup>.) The fact that symptoms occur also with expectation of chemotherapy does not mean that the chemotherapy itself lacks toxicity (or that perceived adverse effects are due to a “nocebo” effect); rather, expectation produces symptoms *because* the exposure *is* toxic. Expectation of the noxious exposure may, via conditioning processes, produce symptoms ordinarily produced by the noxious exposure. (This is potentially evolutionarily adaptive – serving to encourage persons to avoid settings in which the toxic exposure is expected.) To ensure against conditioned effects arising with expectation, a set of negative exposure visits at the test site before (and between) each positive exposure visit may be required to ensure “extinction” of physiologically conditioned expectation effects. In essence, the setting that optimizes prospects to identify a real effect, if present, is that in which the participant believes there will *not* be an active exposure.

N-of-1 studies that focus on physiological effects of EMR have proven somewhat more able to identify EMR effects in those with ES, or subsets of them for which that physiological marker is affected. Just as symptoms vary, so physiological changes do so, so outcomes suited to one person may not apply for all. Physiological markers changed with blinded EMR exposure in a published study of a female physician with ES. She could not discern when the exposure was present or not, but measurable changes occurred and symptoms arose with the positive condition<sup>27</sup>. Symptoms were significantly more intense with pulsed (but not continuous) radiation than sham exposure<sup>27</sup>. An N-of-1 test was reportedly conducted in a former Miami organized crime prosecutor, who developed ES and chemical intolerance, with seizures an important part of his clinical profile, following a significant chemical exposure. An EEG was undertaken, turning on and off a TV, with the party blinded to the stimulus (blindfolded and with headphones to prevent him hearing when the TV was turned on or off). When the TV was shielded, no effect on the EEG was seen. With an unshielded television, EEG changes including seizure activity occurred when the television was turned on (and he experienced physical twitching)<sup>559</sup>. {This particular marker is unlikely to be generally useful, as seizure activity is not a usual part of the clinical profile in those affected by RF/MW.} A provocation study focused in a group of individuals showed changes in heart rate variability<sup>116</sup>, an index of autonomic function that is tied to hard outcomes like sudden death and coronary artery disease<sup>560,561</sup>. Moreover, three of the four participants who characterized their ES as “intense” (though only persons in this group) exhibited striking heart rate increase of between 45 and 90 beats per minute virtually immediately with the microwave exposure, associated with marked increase in sympathetic response. Declines in parasympathetic response with RF/MW exposure were seen for 23 of 25 tested people, in all groups (including,

	<p>though less so, those with no ES).</p> <p>In general, assessments of objectively measurable quantities of relevance, including both differences in affected vs unaffected persons irrespective of current exposure<sup>33,67</sup>, and changes occurring with exposure<sup>116</sup>, provide a more promising approach than real-time assessments of subjective outcomes for understanding this condition.</p>
<p><b>Financial conflict of interest is a major source of apparent disparities in results</b></p>	<p>One key source of disparities in study results is financial conflicts of interest. When present, financial conflicts strongly predict that study results will conform to the financial interests of authors or funders<sup>98,99,562-566</sup>. An analysis examined why some review articles on passive smoking concluded it was harmful while others concluded it was not: The <i>only</i> identified factor that predicted which conclusion, was industry conflict by authors – which was often undisclosed<sup>566</sup>. Richard Smith, the former Editor in Chief of the BMJ (the British Medical journal) observed that this suggested that “far from conflict of interest being unimportant in the objective and pure world of science where method and the quality of data is everything, it is the main factor determining the result of studies”<sup>99</sup>.</p> <p>Financial conflicts have been a concern specifically in relation to RF/MW, both for studies and for regulatory decisions<sup>93-96,567</sup>. In an analysis of studies looking at cell phone effects as a function of funding source, “Studies funded exclusively by industry reported the largest number of outcomes, but were least likely to report a statistically significant result” {So, they report everything that wasn’t affected?} “The odds ratio was 0.11 (95% confidence interval, 0.02–0.78), compared with studies funded by public agencies or charities. Analogous to findings for a relation of industry funding to failure to find tobacco related problems<sup>566</sup>, the finding was not materially altered in analyses adjusted for the number of outcomes reported, study quality, and other factors”<sup>93</sup>.</p> <p>It has been generally assumed that the disproportionately product-favorable results from industry-funded studies (including less evidence of product harm) arises by virtue of choices, selecting study design, exposure specifics, subjects, and outcomes to support the desired result. (See below, these can in fact influence outcomes.) But where harms of lucrative products are concerned, there is precedent for industry-funded studies going beyond those factors to hide even large and lethal harms, even for prespecified or primary outcomes – via means that have the appearance, at least, of fraud<sup>568,569</sup>. Special circumstances enabled the apparent shenanigans in those cases to be uncovered. Whether frank manipulation of data to hide harms of lucrative products is the rule or the exception in industry-funded studies is simply not known.</p> <p>Because of a robust body of evidence documents a strong relation of industry conflicts to outcomes, deliberations and standards should be based exclusively on studies in which such conflicts of interest are absent. (Industry funded-studies can be used for hypothesis generation.) This obviates one major source of apparent inconsistency in studies. But it eliminates inconsistencies due to this factor, only as far as it is possible to discern when financial conflicts are operating.</p>
<p><b>Study outcomes may appear different without “inconsistency”: Details matter, to see an effect</b></p>	<p>Design features can influence outcomes, and may be selected to do so.</p> <p>Details of RF/MW exposure that may influence outcomes include the following (some relevant features have doubtless been missed):</p> <ul style="list-style-type: none"> <li>- Radiation frequency or frequencies<sup>570-572</sup></li> <li>- Radiation intensity<sup>78</sup></li> <li>- Radiation waveform<sup>78</sup></li> <li>- Polarization<sup>571,573,574</sup></li> <li>- Pulsed vs continuous radiation<sup>574,575</sup></li> <li>- Pulse width<sup>100</sup></li> <li>- Time between pulses<sup>187</sup>/ repetition rate<sup>47</sup></li> <li>- Pulse waveform<sup>47,576</sup></li> <li>- Pulse intensity<sup>45</sup></li> <li>- Exposure duration<sup>577,578</sup>, and</li> </ul>

- Exposure intermittency<sup>579</sup> - on every time scale
- Environmental conditions – temperature, humidity, air currents<sup>78,580</sup>
- Concurrent (or preceding) exposures to other radiation<sup>78,97,581</sup> – which can cause synergistic effects<sup>78</sup>
- Concurrent (or preceding) chemical exposures or environment<sup>97,581</sup>
- State of health of the animal or subject<sup>78</sup>
- Species<sup>78</sup>
- Size of the subject relative to wavelength<sup>78</sup>
- Genetics of the animal<sup>67,571</sup>
- Antioxidant/ nutrient status of the animal or subject<sup>234,235,304,305,434,582-588</sup>
- Orientation of the animal or subject relative to the radiation source<sup>78</sup>
- Portion of the body irradiated<sup>78</sup>
- Time between exposure and assessment of effect<sup>571</sup>
- Effect measured
- Metric used to measure effect

Radiation that is pulsed, that is polarized, that is applied intermittently, that is more intense, and that is applied for a longer time, may be more likely to produce problems, for instance.

Even for studies nominally examining the “same” RF/MW exposure, different choices may be made. A range of choices are illustrated in this text: “There are 124 different channels/frequencies that are used in GSM900 mobile communication. They differ by 0.2 MHz in the frequency range between 890 and 915 MHz. The test mobile phone was programmed to use channel 124 with the frequency of 915 MHz. The signal included all standard GSM modulations. No voice modulation was applied. A GSM signal is produced as 577 ms pulses (time slots), with an interpulse waiting time of 4039 ms (seven time slots). The test phone was programmed to regulate output power in the pulses in the range of 0.02–2 W (13– 33 dBm). This power was kept constant during exposure at 33 dBm, as monitored online using a power meter (Bird 43, USA)”<sup>187</sup>.

Studies that examine symptoms as a function of distance from cell tower base stations suggest that in important, real world settings, more intense RF/MW exposure is generally a greater problem<sup>68,151,259,589</sup>) – though there may be an intensity range below which this ceases to be the case.

In some conditions, nonmonotonic effects of radiation have been reported<sup>574,590</sup>, and they are arguably expected for agents in the antioxidant-prooxidant spectrum (high dose antioxidants are often prooxidant, low dose prooxidants, via oxidative preconditioning, may be antioxidant). Opposite direction effects on a critical mechanism can produce opposite direction effects in a resulting outcome. Thus, lower doses of vitamin E fluidize, and higher concentrations stabilize membranes<sup>591</sup>; low vitamin E benefits and higher vitamin E harms vasodilatory function in cholesterol-fed rabbits<sup>592</sup>; “Low tocopherol concentrations have stronger antiinflammatory effects in PUVA-induced erythema than higher concentrations”<sup>593</sup>; low doses are tied to lower mortality in people, higher doses to higher all-cause mortality<sup>594</sup>, etc. For statins, an agent class that can produce prooxidant or antioxidant effects, bidirectional effects have been shown on many outcomes<sup>595</sup> – female sex and features tied to greater likelihood of mitochondrial problems are risk factors for harms – as is higher dose or use of a higher potency agent<sup>596</sup>. It is common that where a lower amount of something may be favorable (or neutral), a higher amount may be adverse – with a transition zone in which subject characteristics and covariables matter a lot in determining the direction. (There are instances in which this directionality is flipped<sup>596</sup> – for instance, sometimes a sufficient concentration leads an adaptive protection to be triggered.)

Beyond characteristics of the radiation, the subject may be exposed to it differently – e.g. in animal studies, there may be whole body radiation<sup>597</sup> or head-only exposure<sup>181,598</sup>, triggering a different spectrum of responses – and with in vitro exposure, even fewer of the variables that might contribute to



	<p>effects are present. The environment in which exposure occurs may differ in ways that influence toxicity of radiation, for instance differences in temperature may produce different effects<sup>580</sup>, or concurrent or background electromagnetic exposure<sup>581</sup> or chemical exposures<sup>97,599</sup>. Amphetamine use represents one exposure that has been reported to magnify problems with RF/MW<sup>47</sup>.</p> <p>Characteristics of the “subjects” may differ. In animal and in vitro studies, they may differ in species, strain, genetic features, cell type, cell preparation, and cell density<sup>571,599</sup>, for instance.</p> <p>“Effect modification” refers to the phenomenon by which effects, including adverse effects, are not equal in all subgroups. This is a major issue throughout biology, and particularly for exposures mediated by oxidative stress and cell energy impairment. Findings with statin cholesterol lowering drugs illustrate how massive the disparity may be as a function of participant group. Like RF/MW, these agents have potential for toxicity through prooxidant and mitochondrial adverse mechanisms<sup>56,499</sup>. RF/MW disproportionately affects sleep and hearing (through its special extra features) – but muscle and tendon problems are sometimes reported<sup>48,102,108</sup>. Fluoroquinolones disproportionately affect tendons through their extra mechanisms (statins can do so too, though more rarely<sup>600-603</sup>). Statins disproportionately affect muscle – the most feared muscle complication is rhabdomyolysis, massive breakdown of muscle that can overwhelm the kidneys and lead to kidney failure and death (which is also reported with fluoroquinolones though more rarely<sup>604-612</sup>).</p> <p>Statins were commonly hailed as so safe they should be put in the water supply<sup>613-616</sup>. But analysis of insurance claims data show that (focusing on the one adverse effect) while the rate of rhabdomyolysis was rare overall, it was frankly common in identifiable vulnerable subgroups. Hospitalized rhabdomyolysis, per year of treatment, occurred in fewer than 1 in 22,000 on statin monotherapy. However, the rate was far higher for older diabetics also on a fibrate (a second class of cholesterol lowering drug), and if they were on the statin agent whose clearance was most affected by fibrates, rhabdomyolysis occurred in about <i>1 in 10 per year of treatment</i><sup>617</sup>. So, depending on characteristics of the exposure, co-exposures <i>and the subject</i>, rates of a problem – and ability for science to show the problem – can vary widely. (The particular statin agent that caused the worst problems was pulled from the market, but the conceptual point stands.) Risks of harm with exposures are not distributed equally. A problem that appears very rare overall, or in one test group – often apparently not increased relative to unexposed – can be frankly common in another. If the groups most at risk are not studied, or their presence is seriously diluted, serious harms can be missed. Studies that fail to detect a harm do not invalidate those that show one – and are not of equal importance where a purpose is to establish that harms can occur.</p>
<b>Rates of problems</b>	<p>Though a minority of embassy personnel were reportedly affected<sup>11</sup>, it is unclear how many were exposed. The fraction of US diplomats in Cuba (and now China) reporting effects may be higher than the fraction of civilians citing similar severity problems with RF/MW exposure – though in neither group can the exposure of those affected be presumed to have been typical. <b>Table 3</b> suggests that once persons are symptomatic, the profile of symptoms is similar. The reportedly high prevalence of Frey-compatible effects, and what seem a comparatively large number of diplomats in Cuba affected, suggest exposures of a more intense or more damaging character – considering intensity, frequency, pulse waveform, pulse duration, duration, polarization, intercurrent exposures, and many other factors influence injury from RF/MW<sup>571</sup>.</p>
<b>Natural History</b>	<p>Both diplomats<sup>9</sup> and RF/MW affected individuals<sup>49,102</sup> have shown variable time course to onset of symptoms after apparent inciting exposure; and variable time course and completeness of recovery with time away from the exposure. Doctors submitting the Bamberg Appeal to the Prime Minister of Germany noted: “The symptoms occur in temporal and spatial relationship to exposure...Some of the health disturbance disappears immediately the exposure ceases (removal of DECT telephone, temporary moving away from home, permanently moving away, using shielding)”<sup>109</sup>. An “intervention study” from Japan, involving the “intervention” of removing a cellular phone base station on a condominium, affirms improvement with removal of the exposure. 107 of 122 inhabitants were interviewed and had medical examinations at two time points, while the base station was in operation, and three months after it was removed. “The health of these inhabitants was shown to improve after the removal of the antennas, and the researchers could identify no other factors that could explain this health improvement...The results of these examinations and interviews indicate a connection between adverse health effects and electromagnetic radiation from mobile phone base stations”<sup>618</sup>.</p>



	Natural history could differ for diplomats, who may have been exposed to a more intense stimulus or one with more injurious characteristics – suggested by what appear to be a comparatively high number affected, and high prevalence of Frey effects. With a powerful exposure, depressed defenses are not equally required to produce injury. There is not a basis to know if affected diplomats will have heightened vulnerability to “usual” RF/MW exposures going forward – though this bears assessing.
<p>† An illustration from a common drug, and a common food: “Grapefruit juice increased the mean peak serum concentration (<b>C<sub>max</sub></b>) of unchanged simvastatin about 9-fold (range, 5.1-fold to 31.4-fold; <math>P &lt; .01</math>) and the mean area under the serum simvastatin concentration-time curve {AUC (0-infinity)} 16-fold (range, 9.0-fold to 37.7-fold; <math>P &lt; .05</math>)”<sup>619</sup>. Thus, just one comparatively innocuous interacting factor – grapefruit juice (which inhibits an enzyme involved in simvastatin metabolism) – led some to have a 38-fold greater blood “amount” of a drug, than that same person would have had without the juice. Potential differences are magnified comparing <i>different</i> persons with/out juice; and moreso factoring in impact of other exposures. Other risk multiplying factors are tied to the individual: The same serum level can produce a radically different impact from person to person: relevant factors include genetic differences in muscle, and factors that reduce energy supply, or that increase energy demand to muscle<sup>56, 620-624</sup>. Thus, what is the “same” exposure before it hits two people, can become a radically different exposure once it interacts with individuals’ biology.</p>	

**Table 5. RF/MW Source Considerations.**

<b>What kinds of RF/MW sources affect civilians?</b>	<p>In the UCSD survey, smart meters were the dominant inciting trigger (~50% of those ~70% who recognized a triggering episode), with cell phones, Wi-Fi introduction or new routers, medical radiation and other factors also reported<sup>52</sup>. The range of apparent triggers has been vast, with RF/MW and particularly pulsed RF/MW commonly implicated. Considering those who have communicated with us, a couple from Scotland became affected several decades ago, after they moved to a rural area, but across from a radar factory. Though they moved away, both remain “electrosensitive” decades later. Others became affected when a cell tower was placed next to their home. Dr. Gro Harlem Brundtland reports becoming sensitized following exposure to a malfunctioning microwave oven (in an episode that also reportedly blinded her for a year)<sup>32,625</sup>. An Australian veteran reports that he became affected during his military service, working with radiofrequency radiation (radar workers in the military were among the first groups in whom such problems were recognized, many decades ago). One who communicated with us became sensitized in association with their job placing radio collars on wildlife. An architect who contacted us was sensitized after several months working closely with Bluetooth-enabled lighting devices. Parents reported to us the onset of ES in their children with Wi-Fi introduced to the school, accommodations were denied, forcing parents to remove their children from school and move elsewhere, and forcing some teachers from their job<sup>241,626</sup>. In Sweden and the UK, a controversial radio system called TETRA reportedly caused health problems in some police officers: severe insomnia in a Swedish officer resolved when the officer’s managers noted the connection, and placed the officer in a room without the exposure<sup>625</sup>. Some US firefighters were affected after municipalities placed cell towers on roofs of fire stations<sup>627</sup>: “Symptoms experienced by the firefighters have included neurological impairment including severe headache, confusion, inability to focus, lethargy, inability to sleep, and inability to wake up for 911 emergency calls. Firefighters have reported getting lost on 911 calls in the same community they grew up in, and one veteran medic forgot where he was in the midst of basic CPR on a cardiac victim and couldn’t recall how to start the procedure over again. Prior to the installation of the tower on his station, this medic had reportedly not made a single mistake in 20 years.”<sup>628</sup>. The International Association of Fire Fighters Division of Occupational Health, Safety and Medicine crafted a position paper<sup>627</sup>, and firefighters were exempted in the recent proposed California bill, SB-649<sup>628, 629</sup>, that sought to bypass local control in placing of 5G cell towers<sup>628</sup>.)</p> <p>These were not “nocebo” effects: many developed symptoms prior to identifying the source of the problem (or in some cases even being aware that the exposure existed at that time). Many had no idea the exposure had potential to produce problems. They were blindsided by onset of new problems. The causes were identified by their spatial and temporal relationship to onset, worsening and abatement.</p> <p>Reports of problems from commercial sources of RF/MW have emerged from many nations including (e.g.) Russia<sup>67,80</sup>, Korea<sup>630</sup>, Japan<sup>50</sup>, Taiwan<sup>631</sup>, Turkey<sup>261</sup>, Israel<sup>632</sup>, Australia<sup>48</sup>, New Zealand<sup>30</sup>, France<sup>33</sup>, England<sup>314,633</sup>, Ireland<sup>314,634,635</sup>, Spain<sup>24,151,636</sup>, Italy<sup>67,314</sup>, the Netherlands<sup>102</sup>, Switzerland<sup>68,92</sup>, Austria<sup>25,90,314,637</sup>, Germany<sup>314,638</sup>, Denmark<sup>314,639</sup>, Sweden<sup>36,424</sup> (where Ericsson designer Per Segerbäck was seriously affected<sup>640</sup>), Norway<sup>641</sup> (afflicting 3-time Prime Minister Gro Harlem Brundtland, as above), Finland<sup>29</sup> (reportedly affecting former Nokia Chief Technology Officer Matti Niemela<sup>642</sup>), the US<sup>32,34,38,91</sup>, where affected former Silicon Valley techies Peter Sullivan<sup>643</sup> and Jeromy Johnson<sup>644</sup> strive to bring attention to the problem; and Canada (where Frank Clegg, formerly head of Microsoft Canada, now of Canadians for Safe Technology – spearheads the effort toward recognition<sup>645</sup>).</p>
<b>Past RF/MW use and Diplomats</b>	<p>Exposure of diplomats to RF/MW is not a new phenomenon. The US embassy in Moscow was reportedly radiated with microwaves from 1953-1988 (other sources give earlier or later end dates), spawning efforts by the US to shield the embassy<sup>75,76</sup>. The Soviets claimed the purpose was to jam US listening devices<sup>75</sup>.</p> <p>Based on reports of past embassy staff, a number of personnel and their offspring developed health effects, some developed white blood cell count elevations, and a couple developed hematological malignancies<sup>76</sup>. Elevated white blood cell counts<sup>108</sup> (as well as depressed ones<sup>78</sup>) have elsewhere been reported in association with RF/MW, as have hematological malignancies<sup>646,647</sup>, including a recent report of an occupational relationship of RF/MW to “hemolymphatic” malignancies in the military setting: “The PF {percentage frequency} of HL {hemolymphatic} cancers in the case series was very high, at 40% with only 23% expected for the series age and gender profile, confidence interval CI95%: 26-56%, p&lt;0.01, 19 out of 47 patients had HL</p>

	<p>cancers. We also found high PF for multiple primaries. As for the three other cohort studies: In the Polish military sector, the PF of HL cancers was 36% in the exposed population as compared to 12% in the unexposed population, <math>p &lt; 0.001</math>. In a small group of employees exposed to RF/MW in Israeli defense industry, the PF of HL cancers was 60% versus 17% expected for the group age and gender profile, <math>p &lt; 0.05</math>. In Belgian radar battalions the HL PF was 8.3% versus 1.4% in the control battalions as shown in a causes of deaths study and HL cancer mortality rate ratio was 7.2 and statistically significant. Similar findings were reported on radio amateurs and Korean war technicians. Elevated risk ratios were previously reported in most of the above studies<sup>648</sup>. (There was a news report of a “blood disorder” in a Cuban diplomat, but its character was unspecified<sup>15</sup>.)</p> <p>A controversial Johns Hopkins study was commissioned to assess the health of Moscow embassy personnel, but was never published in peer reviewed literature. Staff from other Eastern European embassies were used as controls<sup>649</sup> – a problematic control group as these are the embassies most likely to have been subjected to similar exposures; indeed a FOIA request reportedly yielded claims of exposure from employees at other embassies<sup>649</sup>. A reanalysis asserted that Russian and Eastern European diplomats if combined, exhibited a significant increase, relative to expectation from the general US population, in three cancer types<sup>649,650</sup> that have each been associated with RF/MW exposure in other studies – hematological malignancy<sup>648</sup>, brain cancer<sup>651-654</sup>, and breast cancer<sup>655,656</sup>. Some complaints, such as vision problems, concentration problems, memory loss, depression and “other symptoms” were greater in the Moscow than the comparator group, in either men or women or, for vision and concentration problems, in each men and women. Given a presumed vulnerable subgroup, a problematic study design, and absence of a quality report, it is difficult to draw meaningful inferences – beyond that some diplomats were exposed, and some who were exposed reported health problems.</p>
<b>Current RF/MW Source Possibilities in Diplomats</b>	<p>The source of proposed EMR/RF/MW (probably pulsed) affecting diplomats is not a principal focus of this paper.</p> <p>For the diplomats in Cuba, causative RF/MW could in principle emanate from monitoring/surveillance devices (as has been speculated for microwaving of the US embassy in Moscow<sup>75</sup>); from efforts to jam our listening devices, as claimed by the Soviets<sup>75</sup>; from electronic weaponry, or conceivably from “innocent” communications sources of the type that affect some civilians (but presumably of higher typical intensity, or shorter pulse duration, or in the setting of other exposures that amplify oxidative stress, or with some other feature that amplifies the fraction affected).</p> <p>Surveillance-related efforts would seem perhaps the most likely, given the apparent preferential involvement of diplomats, in Cuba and China.</p>
<b>Room sweep by FBI yielded no devices<sup>10</sup></b>	<p>The source of the historical microwave exposure on the US embassy in Moscow was also outside the embassy building. It reportedly originated from the building next door, and later from the building across the street<sup>75</sup>.</p> <p>Smart meters (or banks of them) – outside the room – were the number one reported instigating cause of symptoms in the UCSD survey, with other causes including base stations or cell towers outside the home. Pulsed RF/MW producing devices thus need not be in the room. The exposure can be short term or intermittent – it need not be continuous. For this reason, devices in whatever their location need not remain present, after health effects have been produced.</p>

**Acknowledgments:** For kindly helping to retrieve articles for this, I thank Emily Nguyen, Hayley Koslik, Leeann Bui, Andrea Sember, Annabelle Amos, Karl Chen, Arthur Pavlovsky, and Aubrey Bunday. I thank Hayley Koslik for assistance with the submission process.

**References:**

1. Lederman J. US stands by claim workers attacked in Cuba, maybe by virus. Associated Press International 2018;Jan 10.
2. Perlez J, Myers L. China pledges to investigate fears of sonic attacks on U.S. diplomats. The New York Times 2018;June 7.
3. Lederman J, Lee M. Cuba tells Tillerson: No culpability. The Associated Press 2017;Sept 27.
4. Cochrane E. Mysterious health issues drove diplomats from Cuba. New York Times 2017;Aug 10.
5. Lederman J, Weissenstein M, Lee M, The Associated Press. Bizarre Cuba mystery: Did sonic weapon cause U.S. diplomats' brain injuries? Mercury News 2017;Sept 14.
6. Panetta A. Canada won't follow U.S. in reducing Cuba staff. The Canadian Press 2017;Sept 29.
7. Cuba's sonic attacks. Wall Street Journal 2017;Sep 26:A16.
8. The Associated Press. Bizarre Cuba mystery: Did sonic weapon cause U.S. diplomats brain injury. Mercury News 2017;Sept 14.
9. Associated Press in Washington. Mystery of sonic weapon attacks at US embassy in Cuba deepens. . The Guardian 2017;Sept 14.
10. Lederman J, Weissenstein M, Lee M. Cuba mystery grows: New details on what befell US diplomats. Associated Press News 2017;Sept 16.
11. Stone R. Reports of inner-ear damage deepen diplomat controversy: As mystery symptoms reported in Cuba spread to China, some blame an attack; others see "suggestion and paranoia". Science 2018;360:1281-2.
12. Harris G. Pompeo Says Mysterious Sickness Among Diplomats in Cuba Has Spread to China. New York Times 2018;May 23.
13. Buckley C, Harris G. First Cuba, now China? An American falls ill after 'abnormal' sounds. The New York Times 2018;May 23.
14. Myers SL. More Americans Evacuated From China Over Mysterious Ailments New York Times 2018;Jun 30.
15. Robles F, Semple K. 'Health attacks' on U.S. diplomats in Cuba baffle both countries. The New York Times 2017;October 4.
16. Robles F, Semple K. U.S. and Cuba baffled by 'health attacks' on American envoys in Cuba. The New York Times 2017;Aug 12.
17. Associated Press. Tillerson says diplomats in Havana suffered "health attacks". Los Angeles Times 2017;Aug 12.
18. Gearan A. State Department reports new instances of American diplomats harmed in Cuba. The Washington Post 2017;Sept 1.
19. Lederman J. 19 American diplomats in Cuba suffering health problems after 'attacks' blamed on secret sonic weapon. The Independent 2017;Sept 2.
20. Board TE. Cuba and the mystery of sonic weapons. The New York Times 2017;Oct 6.
21. Zimmer C. The 'sonic attack' that likely wasn't. The New York Times 2017;October 6.
22. Zimmer C. What's a science reporter to do when sound evidence isn't sound? The New York Times 2017;Oct 6.
23. Johnson Liakouris AG. Radiofrequency (RF) sickness in the Lilienfeld study: An effect of modulated microwaves? Arch Environ Health 1998;53:236-8.
24. Navarro A, Segura J, Portoles M, Gomez-Perretta C. The microwave syndrome: A preliminary study in Spain. Electromagnetic Biology and Medicine 2003;22:161-9.
25. Leitgeb N. Electromagnetic hypersensitivity. In: Leitgeb N, editor. Proceedings, International Workshop on Electromagnetic Fields and Non-specific Health Symptoms European Cooperation in the Field of Science and Technical Research; 1998; Graz, Austria; 1998. p. 8-16.
26. Hardell L, Carlberg M, Soderqvist F, et al. Increased concentrations of certain persistent organic pollutants in subjects with self-reported electromagnetic hypersensitivity--a pilot study. Electromagn Biol Med 2008;27:197-203.
27. McCarty DE, Carrubba S, Chesson AL, Frilot C, Gonzalez-Toledo E, Marino AA. Electromagnetic hypersensitivity: evidence for a novel neurological syndrome. The International journal of neuroscience 2011;121:670-6.
28. Genuis SJ, Lipp CT. Electromagnetic hypersensitivity: fact or fiction? Sci Total Environ 2012;414:103-12.
29. Hagstrom M, Auranen J, Ekman R. Electromagnetic hypersensitive Finns: Symptoms, perceived sources and treatments, a questionnaire study. Pathophysiology 2013;20:117-22.
30. [www.esnztrust](http://www.esnztrust) Electrosensitivity New Zealand.
31. [www.es-uk.info](http://www.es-uk.info). ElectroSensitivity UK.
32. Woolston C. Victims of electrosensitivity syndrome say EMFs caused symptoms. Los Angeles Times 2010; Feb 15: <http://articles.latimes.com/2010/feb/15/health/la-he-electromagnetic-syndrome1-feb15>.

33. Belpomme D, Campagnac C, Irigaray P. Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder. *Rev Environ Health* 2015;30:251-71.
34. Heuser G, Heuser SA. Functional brain MRI in patients complaining of electrohypersensitivity after long term exposure to electromagnetic fields. . *Rev Environ Health* 2017;Jul 5.
35. Redmayne M, Johansson O. Could myelin damage from radiofrequency electromagnetic field exposure help explain the functional impairment electrohypersensitivity? A review of the evidence. *J Toxicol Environ Health B Crit Rev* 2014;17:247-58.
36. Johansson O. Electrohypersensitivity: a functional impairment due to an inaccessible environment. *Rev Environ Health* 2015;30:311-21.
37. Johansson O. Electrohypersensitivity: state-of-the-art of a functional impairment. *Electromagn Biol Med* 2006;25:245-58.
38. Carpenter DO. Excessive exposure to radiofrequency electromagnetic fields may cause the development of electrohypersensitivity. *Altern Ther Health Med* 2014;20:40-2.
39. Wilkinson T. Cuban diplomats expelled from Washington over incident that harmed U.S. personnel in Havana, State Department says. *Los Angeles Times* 2017;Aug 8.
40. Harris G. U.S. to open inquiry over 24 Americans sickened in Cuba. *New York Times* 2018;Jan 10.
41. Swanson RL, Hampton S, Green-McKenzie J, et al. Neurological manifestations among US Government personnel reporting directional audible and sensory phenomena in Havana, Cuba. *Jama* 2018;Feb 15 published online.
42. Associated Press. Dangerous sound? What Americans heart in Cuba attacks. 2017;Oct 12.
43. Harris G. 16 Americans sickened after attack on embassy staff in Havana. *New York Times* 2017;Aug 24.
44. Weissenstein M. US senator says no evidence of 'sonic attacks' in Cuba. *Associated Press International* 2018;Jan 6.
45. Elder JA, Chou CK. Auditory response to pulsed radiofrequency energy. *Bioelectromagnetics* 2003;Suppl 6:S162-73.
46. Frey AH. Auditory system response to radio frequency energy. . *Aerosp Med* 1961;32:1140-2.
47. Bolen SM. Radiofrequency/microwave radiation biological effects and safety standards: a review: Rome Laboratory; 1988 June. Report No.: RL-TR-94-53.
48. Lamech F. Self-reporting of symptom development from exposure to radiofrequency fields of wireless smart meters in Victoria, Australia: A case series. *Altern Ther Health Med* 2014;20:28-39.
49. Conrad R, Friedman E. Smart Meter Health Effects Survey and Report, Exhibit D. ME Public Utilities Commission 2013;Docket 2011-00262: <http://www.mainecoalitiontostopsmartmeters.org/wp-content/uploads/2013/01/Exhibit-10-Smart-Meter-Health-Effects-Report-Survey2.pdf>.
50. Kato Y, Johansson O. Reported functional impairments of electrohypersensitive Japanese: A questionnaire survey. *Pathophysiology* 2012;19:95-100.
51. Halteman E. Wireless utility meter safety impacts survey: Final Results Summary. Sept 13 2011. <http://emfsafetynetwork.org/wp-content/uploads/2011/09/Wireless-Utility-Meter-Safety-Impacts-Survey-Results-Final.pdf>
52. Golomb BA. Electrosensitivity: A 'current' and future problem. Meeting: Cell Phones and Wireless Technologies—Should Safety Guidelines Be Strengthened to Protect Adults, Children and Vulnerable Populations? 2015;Commonwealth Club, San Francisco:June 22.
53. Conklin SM, Harris JI, Manuck SB, Yao JK, Hibbeln JR, Muldoon MF. Serum omega-3 fatty acids are associated with variation in mood, personality and behavior in hypercholesterolemic community volunteers. *Psychiatry Res* 2007;152:1-10.
54. Peet M, Horrobin DF. A dose-ranging study of the effects of ethyl-eicosapentaenoate in patients with ongoing depression despite apparently adequate treatment with standard drugs. *Arch Gen Psychiatry* 2002;59:913-9.
55. Golomb BA, Koslik HJ, Redd AJ. Fluoroquinolone-induced serious, persistent, multisymptom adverse effects. *BMJ Case Rep* 2015;2015.
56. Golomb BA, Evans MA. Statin adverse effects: a review of the literature and evidence for a mitochondrial mechanism. *Am J Cardiovasc Drugs* 2008;8:373-418.
57. Golomb BA, Allison M, Koperski S, Koslik HJ, Devaraj S, Ritchie JB. Coenzyme Q10 benefits symptoms in Gulf War veterans: results of a randomized double-blind study. *Neural Comput* 2014;26:2594-651.
58. Wei YH, Lee HC. Oxidative stress, mitochondrial DNA mutation, and impairment of antioxidant enzymes in aging. *Exp Biol Med (Maywood)* 2002;227:671-82.
59. Lee HC, Wei YH. Role of Mitochondria in Human Aging. *J Biomed Sci* 1997;4:319-26.



60. Yakymenko I, Tsybulin O, Sidorik E, Henshel D, Kyrylenko O, Kyrylenko S. Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation. *Electromagn Biol Med* 2015;35:186-202.
61. Yuksel M, Naziroglu M, Ozkaya MO. Long-term exposure to electromagnetic radiation from mobile phones and Wi-Fi devices decreases plasma prolactin, progesterone, and estrogen levels but increases uterine oxidative stress in pregnant rats and their offspring. *Endocrine* 2016;52:352-62.
62. Barnes F, Greenenbaum B. Some Effects of Weak Magnetic Fields on Biological Systems: RF fields can change radical concentrations and cancer cell growth rates. *IEEE Power Electronics Magazine* 2016;3:60-8.
63. Barnes FS, Greenebaum B. The effects of weak magnetic fields on radical pairs. *Bioelectromagnetics* 2015;36:45-54.
64. Gao XH, Hu HR, Ma XL, Chen J, Zhang GH. [Cellphone electromagnetic radiation damages the testicular ultrastructure of male rats]. *Zhonghua Nan Ke Xue* 2016;22:491-5.
65. Turedi S, Hanci H, Topal Z, et al. The effects of prenatal exposure to a 900-MHz electromagnetic field on the 21-day-old male rat heart. *Electromagn Biol Med* 2015;34:390-7.
66. Zhu W, Zhang W, Wang H, Xu J, Li Y, Lv S. Apoptosis induced by microwave radiation in pancreatic cancer JF305 cells. *Can J Physiol Pharmacol* 2014;92:324-9.
67. De Luca C, Chung Sheun Thai J, Raskovic D, et al. Metabolic and genetic screening of electromagnetic hypersensitive subjects as a feasible tool for diagnostics and intervention. *Mediators Inflamm* 2014;2014:924184.
68. Altpeter ES, Krebs T, Pfluger DH, et al. Study on Health Effects of the Shortwave Transmitter Station of Schwarzenburg, Berne, Switzerland. Berne, Switzerland: Federal Office of Energy 1995;Study 55.
69. Cherry N, Mackness M, Durrington P, et al. Paraoxonase (PON1) polymorphisms in farmers attributing ill health to sheep dip. *Lancet* 2002;359:763-4.
70. Steele L, Lockridge O, Gerkovich MM, Cook MR, Sastre A. Butyrylcholinesterase genotype and enzyme activity in relation to Gulf War illness: preliminary evidence of gene-exposure interaction from a case-control study of 1991 Gulf War veterans. *Environ Health* 2015;14:4.
71. Ishikawa C, Ozaki H, Nakajima T, et al. A frameshift variant of CYP2C8 was identified in a patient who suffered from rhabdomyolysis after administration of cerivastatin. *J Hum Genet* 2004;49:582-5.
72. Rowan C, Brinker AD, Nourjah P, et al. Rhabdomyolysis reports show interaction between simvastatin and CYP3A4 inhibitors. *Pharmacoepidemiol Drug Saf* 2009;18:301-9.
73. Page SR, Yee KC. Rhabdomyolysis in association with simvastatin and dosage increment in clarithromycin. *Intern Med J* 2014;44:690-3.
74. Molden E, Skovlund E, Braathen P. Risk management of simvastatin or atorvastatin interactions with CYP3A4 inhibitors. *Drug Saf* 2008;31:587-96.
75. Gwertzman B. Moscow rays linked to U.S. bugging. *New York Times* 1976;Feb 26.
76. Schumaker J. Moments in U.S. Diplomatic History. Microwaving Embassy Moscow - Another Perspective. . Association for Diplomat Studies and Training 2013;September:adst.org/2013/09/microwaving-embassy-moscow-another-perspective/#.WeOG0Dtrxfq.
77. Bergman W. The Effect of Microwaves on the Central Nervous System. Translated from the German for Research and Scientific Laboratory, Ford Motor Company, by the Technical Library Research Service 1965.
78. Adams RL, Williams RA. Biological effects of electromagnetic radiation (radiowaves and microwaves) - Eurasian Communist Countries: Defense Intelligence Agency; 1976 March.
79. Raines JK. Electromagnetic field interactions with the human body: Observed effects and theories. . NASA CR 166661 1981; Report Prepared for: National Aeronautics and Space Administration: <https://ntrs.nasa.gov/search.jsp?R=19810017132> 2018-04-13T20:33:40+00:00Z.
80. Glaser ZR. Bibliography of reported biological phenomena ('effects') and clinical manifestations attributed to microwave and radiofrequency radiation. Research Report. Second Printing, with Revisions, Corrections, and Additions: 20 April 1972. Bethesda, MD: Naval Medical Research Institute, National Naval Medical Center; 1972 Oct 4, 1971 Corrections and Additions April 1972. Report No.: AD750271 MF12.524.015-0004B. Supersedes AD No 734391.
81. Avendano C, Mata A, Sanchez Sarmiento CA, Doncel GF. Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation. *Fertil Steril* 2012;97:39-45 e2.
82. Markova E, Hillert L, Malmgren L, Persson BR, Belyaev IY. Microwaves from GSM mobile telephones affect 53BP1 and gamma-H2AX foci in human lymphocytes from hypersensitive and healthy persons. *Environ Health Perspect* 2005;113:1172-7.



83. Leszczynski D. Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: Molecular mechanism for cancer- and blood-brain barrier-related effects. *Differentiation* 2002;70:120-9.
84. Sener G, Sehirli AO, Satiroglu H, Keyer-Uysal M, Yegen BC. Melatonin prevents oxidative kidney damage in a rat model of thermal injury. *Life Sci* 2002;70:2977-85.
85. Sener G, Sehirli AO, Satiroglu H, Keyer-Uysal M, Yegen BC. Melatonin improves oxidative organ damage in a rat model of thermal injury. *Burns* 2002;28:419-25.
86. Tunali T, Sener G, Yarat A, Emekli N. Melatonin reduces oxidative damage to skin and normalizes blood coagulation in a rat model of thermal injury. *Life Sci* 2005;76:1259-65.
87. Maldonado MD, Murillo-Cabezas F, Calvo JR, et al. Melatonin as pharmacologic support in burn patients: a proposed solution to thermal injury-related lymphocytopenia and oxidative damage. *Crit Care Med* 2007;35:1177-85.
88. Bekyarova G, Tancheva S, Hristova M. Protective effect of melatonin against oxidative hepatic injury after experimental thermal trauma. *Methods Find Exp Clin Pharmacol* 2009;31:11-4.
89. Hillert L, Berglind N, Arnetz BB, Bellander T. Prevalence of self-reported hypersensitivity to electric or magnetic fields in a population-based questionnaire survey. *Scand J Work Environ Health* 2002;28:33-41.
90. Schröttner J, Leitgeb N. Sensitivity to electricity -- temporal changes in Austria. *BMC Public Health* 2008;8:310.
91. Levallois P, Neutra R, Lee G, Hristova L. Study of self-reported hypersensitivity to electromagnetic fields in California. *Environ Health Perspect* 2002;110 Suppl 4:619-23.
92. Schreier N, Huss A, Roosli M. The prevalence of symptoms attributed to electromagnetic field exposure: a cross-sectional representative survey in Switzerland. *Soz Präventivmed* 2006;51:202-9.
93. Huss A, Egger M, Hug K, Huwiler-Müntener K, Rössli M. Source of Funding and Results of Studies of Health Effects of Mobile Phone Use: Systematic Review of Experimental Studies. *Environ Health Perspect* 2007;115:1-4.
94. Alster N. Captured Agency: How the Federal Communications Commission is Dominated by the Industries it Presumably Regulates. Harvard University, Edmond J Safra Center for Ethics 2015; [www.harvard.ethics.edu](http://www.harvard.ethics.edu).
95. Hardell L. World Health Organization, radiofrequency radiation and health - a hard nut to crack (Review). *International Journal of Oncology* 2017;June 21.
96. Leszczynski D. Science and Conflict of Interest in Bioelectromagnetics. Keynote speech at Swiss association Gigahertz 2015;Mar 7: <http://bit.ly/1CMWkHq>.
97. Kostoff RN, Lau CGY. Chapter 4. Modified health effects of non-ionizing electromagnetic radiation combined with other agents reported in the biomedical literature. In: Geddes CD, ed. *Microwave Effects on DNA and Proteins*: Springer; 2017.
98. Golomb BA. Conflict of Interest in Medicine. Sponsored by The Science Network (tsntv.org), Salk Institute. La Jolla, CA. Oct 5; 2008. <http://thesciencenetwork.org/programs/beyond-belief-candles-in-the-dark/beatrice-golomb>
99. Smith R. Conflicts of interest: how money clouds objectivity. *J R Soc Med* 2006;99:292-7.
100. Bonnafous P, Vernhes M-C, Teissie J, Gabriel B. The generation of reactive-oxygen species associated with long-lasting pulse-induced electroporabilisation of mammalian cells is based on a non-destructive alteration of the plasma membrane. *Biochimica et Biophysica Acta (BBA) - Biomembranes* 1999;1461:123-34.
101. Shih P, Sanghvi H, et al. Enhancement of radiation cytotoxicity in murine cancer cells by electroporation: in vitro and in vivo studies. *J Environ Pathol Toxicol Oncol* 2005;24:291-8.
102. Schooneveld H, Kuiper J. Electrohypersensitivity (EHS) in the Netherlands--A Questionnaire Survey. Stichting EHS (Dutch EHS Foundation) 2007.
103. Smart meters or no power at all? Nevada Energy sends armed men to disconnect power.- just for opting out. <https://richardalanmillercom/newsblog/smart-meter-or-no-power-at-all-nevada-energy-sends-armed-men-to-disconnect-power-j> 2012;Sept 17:Download date 2018-05-01.
104. Weissenstein M, Rodriguez A. Cuba presents detailed defense against sonic attack charges. *The Associated Press* 2017;Oct 27.
105. Cain CA, Rissmann WJ. Mammalian auditory response to 3.0 GHz microwave pulses. *IEEE Trans Biomed Eng BME* 1978;25:288-93.
106. Ingalls CE. Sensation of hearing in electromagnetic fields. *NY State J Med* 1967;67:2992-7.
107. Brinchman S. Living Nightmare: How SDG&E led to headaches, hearing loss. [lamesapatchcome.com/blog\\_posts](http://lamesapatchcome.com/blog_posts) 2011;Aug 14.
108. Aschermann C. Observations from a Psychotherapy Practice on Mobile Telecommunications and DECT Telephones. Revised and Extended Version. Translated by Margaret E White 2009;June.

109. Waldman-Salsam C. Bamberg Appeal, on behalf of 114 physicians. Open Letter to Edmund Stoiber, Prime Minister, Germany 2004;Aug 3.
110. Alsanosi AA, Al-Momani MO, Hagr AA, Almomani FM, Shami IM, Al-Habeeb SF. The acute auditory effects of exposure for 60 minutes to mobile's electromagnetic field. *Saudi Med J* 2013;34:142-6.
111. Karaer I, Simsek G, Gul M, et al. Melatonin protects inner ear against radiation damage in rats. *Laryngoscope* 2015.
112. Frei M, Jauchem J, Heinmets F. Physiological effects of 2.8GHz radio-frequency radiation: a comparison of pulsed and continuous-wave radiation. *J Microw Power Electromagn Energy* 1988;23:85-93.
113. Celiker M, Özgür A, Tümkaya L, et al. Effects of exposure to 2100MHz GSM-like radiofrequency electromagnetic field on auditory system of rats. *Braz J Otorhinolaryngol* 2016;Nov 5:pil: S1808-8694(16)30222-1. doi: 10.1016/j.bjorl.2016.10.004.
114. Counter SA. Electromagnetic stimulation of the auditory system: effects and side-effects. *Scand Audiol Suppl* 1993;37:1-3.
115. Lederman J. Trump: Cuba 'is responsible' for attacks on US personnel. *The Associated Press* 2017;Oct 16.
116. Havas M, Marrongelle J, Pollner B, Kelley E, Rees CRG, Tully L. Provocation study using heart rate variability shows microwave radiation from 2.4 GHz cordless phone affects autonomic nervous system. *Eur J Oncol Library* 2010;5:273-300.
117. Koslik HJ, Hamilton G, Golomb BA. Mitochondrial Dysfunction in Gulf War Illness Revealed by <sup>31</sup>Phosphorus Magnetic Resonance Spectroscopy: A case-control study. *PLoS ONE* 2014;9:e92887.
118. Steele L. Prevalence and patterns of Gulf War illness in Kansas veterans: association of symptoms with characteristics of person, place, and time of military service. *American journal of epidemiology* 2000;152:992-1002.
119. Chen Y, Meyer JN, Hill HZ, et al. Role of mitochondrial DNA damage and dysfunction in veterans with Gulf War Illness. *PLoS One* 2017;12:e0184832.
120. Weller S. Electromagnetic Hypersensitivity. Electromagnetic Energy Reference Group (EMERG) committee meeting presentation 2015;May 20:electromagnetichealth.org/wp-content/uploads/2015/06/EHS-Presentation-Steven-Weller.pdf.
121. Porto Arceo JA. [Special features of NSAID intolerance in children]. *Allergol Immunopathol (Madr)* 2003;31:109-25.
122. Sepcic J, Bucuk M, Perkovic O, et al. Drug-induced aseptic meningitis, sensorineural hearing loss and vestibulopathy. *Coll Antropol* 2010;34:1101-4.
123. Powell RM. Symptoms after exposure to smart meter radiation. 2015;PMID: 25478801: <https://www.scribd.com/doc/289777267/Symptoms-after-Exposure-to-Smart-Meter-Radiation>.
124. Argun M, Tok L, Uguz AC, Celik O, Tok OY, Naziroglu M. Melatonin and amfenac modulate calcium entry, apoptosis, and oxidative stress in ARPE-19 cell culture exposed to blue light irradiation (405 nm). *Eye (Lond)* 2014;28:752-60.
125. Beatty S, Koh H, Phil M, Henson D, Boulton M. The role of oxidative stress in the pathogenesis of age-related macular degeneration. *Surv Ophthalmol* 2000;45:115-34.
126. King A, Gottlieb E, Brooks DG, Murphy MP, Dunaief JL. Mitochondria-derived reactive oxygen species mediate blue light-induced death of retinal pigment epithelial cells. *Photochem Photobiol* 2004;79:470-5.
127. Liang FQ, Green L, Wang C, Alssadi R, Godley BF. Melatonin protects human retinal pigment epithelial (RPE) cells against oxidative stress. *Exp Eye Res* 2004;78:1069-75.
128. Totan Y, Cekic O, Borazan M, Uz E, Sogut S, Akyol O. Plasma malondialdehyde and nitric oxide levels in age related macular degeneration. *Br J Ophthalmol* 2001;85:1426-8.
129. Javaheri M, Khurana RN, O'Hearn T M, Lai MM, Sadun AA. Linezolid-induced optic neuropathy: a mitochondrial disorder? *Br J Ophthalmol* 2007;91:111-5.
130. Pachalska M, DiMauro S, Forminska-Kapuscik M, et al. The course of vision disturbances in a patient with the MELAS syndrome. *Med Sci Monit* 2002;8:CS11-20.
131. Rucker JC, Hamilton SR, Bardenstein D, Isada CM, Lee MS. Linezolid-associated toxic optic neuropathy. *Neurology* 2006;66:595-8.
132. Qi X, Lewin AS, Sun L, Hauswirth WW, Guy J. Suppression of mitochondrial oxidative stress provides long-term neuroprotection in experimental optic neuritis. *Invest Ophthalmol Vis Sci* 2007;48:681-91.
133. Feher J, Kovacs B, Kovacs I, Schveoller M, Papale A, Balacco Gabrieli C. Improvement of visual functions and fundus alterations in early age-related macular degeneration treated with a combination of acetyl-L-carnitine, n-3 fatty acids, and coenzyme Q10. *Ophthalmologica* 2005;219:154-66.

134. Modi G, Heckman JM, Saffer D. Vitelliform macular degeneration associated with mitochondrial myopathy. *Br J Ophthalmol* 1992;76:58-60.
135. Yu J, Wu L, Lin X. [Preliminary study of mitochondrial DNA deletions in age-related macular degeneration]. *Yan Ke Xue Bao* 1997;13:52-5.
136. Liang FQ, Godley BF. Oxidative stress-induced mitochondrial DNA damage in human retinal pigment epithelial cells: a possible mechanism for RPE aging and age-related macular degeneration. *Exp Eye Res* 2003;76:397-403.
137. Feher J, Papale A, Mannino G, Gualdi L, Balacco Gabrieli C. Mitotropic compounds for the treatment of age-related macular degeneration. The metabolic approach and a pilot study. *Ophthalmologica* 2003;217:351-7.
138. Sandbach JM, Coscun PE, Grossniklaus HE, Kokoszka JE, Newman NJ, Wallace DC. Ocular pathology in mitochondrial superoxide dismutase (Sod2)-deficient mice. *Invest Ophthalmol Vis Sci* 2001;42:2173-8.
139. Ottonello S, Foroni C, Carta A, Petrucco S, Maraini G. Oxidative stress and age-related cataract. *Ophthalmologica* 2000;214:78-85.
140. Gul A, Rahman MA, Hasnain SN, Salim A, Simjee SU. Could oxidative stress associate with age products in cataractogenesis? *Curr Eye Res* 2008;33:669-75.
141. Karslioglu I, Ertekin MV, Taysi S, et al. Radioprotective effects of melatonin on radiation-induced cataract. *J Radiat Res (Tokyo)* 2005;46:277-82.
142. Taylor A, Jacques PF, Epstein EM. Relations among aging, antioxidant status, and cataract. *The American journal of clinical nutrition* 1995;62:1439S-47S.
143. Tarwadi K, Agte V. Linkages of antioxidant, micronutrient, and socioeconomic status with the degree of oxidative stress and lens opacity in indian cataract patients. *Nutrition* 2004;20:261-7.
144. Birenbaum L, Grosz GM, Rosenthal SWZ, M.M. Effect of microwaves on the eye. *IEEE Transactions on Biomedical Engineering* 1969;16:7-14.
145. Zaret MM. Microwave cataracts. *Medical Trial Technique Quarterly* 1973;19:246-52.
146. Cleary SF. Microwave cataractogenesis. *Proceeding IEEE* 1980;68:49-55.
147. McCally RL, Farrell RA, Barger CB, Kues HA, Hochheimer BF. Nonionizing radiation damage in the eye. *Johns Hopkins APL Technologies Digest* 1986;7:73-91.
148. Williams RJ, Finch ED. Examination of the cornea following exposure to microwave radiation. *Aerospace medicine* 1974;Apr:393-6.
149. Daily L, et al. The effects of microwave diathermy on the eye. *Am J Ophth* 1952;35:1001.
150. Cutz A. Effects of microwave radiation on the eye. The occupational health perspective. *Lens and Eye Toxicity Research* 1989;6:379-86.
151. Oberfeld G, Navarro AE, Portoles M, Maestu C, Gomez-Perretta C. The microwave syndrome - Further aspects of a Spanish study. WHO 3rd International Workshop on Biological Effects of Electromagnetic Fields, Kos, Greece, October 2004.
152. Smits BW, Westeneng HJ, van Hal MA, van Engelen BG, Overeem S. Sleep disturbances in chronic progressive external ophthalmoplegia. *Eur J Neurol* 2012;19:176-8.
153. Dodson RF, Patten BM, Hyman BM, Chu LW. Mitochondrial abnormalities in progressive ophthalmoplegia. *Cytobios* 1976;15:57-60.
154. Hyman BN, Patten BM, Dodson RF. Mitochondrial abnormalities in progressive external ophthalmoplegia. *Am J Ophthalmol* 1977;83:362-71.
155. Land JM, Hockaday JM, Hughes JT, Ross BD. Childhood mitochondrial myopathy with ophthalmoplegia. *J Neurol Sci* 1981;51:371-82.
156. Goto Y, Koga Y, Horai S, Nonaka I. Chronic progressive external ophthalmoplegia: a correlative study of mitochondrial DNA deletions and their phenotypic expression in muscle biopsies. *J Neurol Sci* 1990;100:63-9.
157. Kao KP. Mitochondrial disease with chronic progressive external ophthalmoplegia: clinical analysis of 19 cases. *Zhonghua Yi Xue Za Zhi (Taipei)* 1994;53:95-100.
158. Chen Q, Li X, Wu L, Qi Y, Wu X. Mitochondrial gene defect in patients with chronic progressive external ophthalmoplegia. *Chin Med J (Engl)* 1998;111:500-3.
159. Pineda M, Playan-Ariso A, Alcaine-Villarroya MJ, et al. [Familial chronic progressive external ophthalmoplegia of mitochondrial origin]. *Rev Neurol* 2004;38:1023-7.
160. Schaefer AM, Blakely EL, Griffiths PG, Turnbull DM, Taylor RW. Ophthalmoplegia due to mitochondrial DNA disease: the need for genetic diagnosis. *Muscle Nerve* 2005;32:104-7.
161. Eger H, Jahn M. Specific health symptoms and cell phone radiation in Selbitz (Bavaria, Germany) = Evidence of a dose-response relationship. *umwelt-medizin-gesellschaft* 2010;23.

162. Wallace F. Online comment in response to Blog post. Between a Rock and a Hard Place Science Blog on Mobile Phone Radiation and Health by Dariusz Leszczynski 2017;Nov 9: <https://betweenrockandhardplace.wordpress.com/2017/11/08/ehs-researchis-scientifically-worthless-for-two-reasons/>.
163. Wright K. Online comment in response to website article. <https://stopsmartmetersorg/direct-action/> 2013;May 31.
164. Takemori K, Murakami T, Kometani T, Ito H. Possible involvement of oxidative stress as a causative factor in blood-brain barrier dysfunction in stroke-prone spontaneously hypertensive rats. *Microvasc Res* 2013;90:169-72.
165. Enciu AM, Gherghiceanu M, Popescu BO. Triggers and effectors of oxidative stress at blood-brain barrier level: relevance for brain ageing and neurodegeneration. *Oxid Med Cell Longev* 2013;2013:297512.
166. Al Ahmad A, Gassmann M, Ogunshola OO. Involvement of oxidative stress in hypoxia-induced blood-brain barrier breakdown. *Microvasc Res* 2012;84:222-5.
167. Katsu M, Niizuma K, Yoshioka H, Okami N, Sakata H, Chan PH. Hemoglobin-induced oxidative stress contributes to matrix metalloproteinase activation and blood-brain barrier dysfunction in vivo. *J Cereb Blood Flow Metab* 2010;30:1939-50.
168. Hurst RD, Heales SJ, Dobbie MS, Barker JE, Clark JB. Decreased endothelial cell glutathione and increased sensitivity to oxidative stress in an in vitro blood-brain barrier model system. *Brain Res* 1998;802:232-40.
169. Lochhead JJ, McCaffrey G, Quigley CE, et al. Oxidative stress increases blood-brain barrier permeability and induces alterations in occludin during hypoxia-reoxygenation. *J Cereb Blood Flow Metab* 2010;30:1625-36.
170. Haorah J, Ramirez SH, Schall K, Smith D, Pandya R, Persidsky Y. Oxidative stress activates protein tyrosine kinase and matrix metalloproteinases leading to blood-brain barrier dysfunction. *J Neurochem* 2007;101:566-76.
171. Blasig IE, Mertsch K, Haseloff RF. Nitronyl nitroxides, a novel group of protective agents against oxidative stress in endothelial cells forming the blood-brain barrier. *Neuropharmacology* 2002;43:1006-14.
172. Nittby H, Brun A, Eberhardt J, Malmgren L, Persson BR, Salford LG. Increased blood-brain barrier permeability in mammalian brain 7 days after exposure to the radiation from a GSM-900 mobile phone. *Pathophysiology* 2009;16:103-12.
173. Salford LG, Brun A, Stureson K, Eberhardt JL, Persson BR. Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, and 200 Hz. *Microsc Res Tech* 1994;27:535-42.
174. Sirav B, Seyhan N. Blood-brain barrier disruption by continuous-wave radio frequency radiation. *Electromagn Biol Med* 2009;28:215-22.
175. Sirav B, Seyhan N. Effects of radiofrequency radiation exposure on blood-brain barrier permeability in male and female rats. *Electromagn Biol Med* 2011;30:253-60.
176. Tang J, Zhang Y, Yang L, et al. Exposure to 900 MHz electromagnetic fields activates the mmp-1/ERK pathway and causes blood-brain barrier damage and cognitive impairment in rats. *Brain Res* 2015;1601:92-101.
177. Soderqvist F, Carlberg M, Hardell L. Mobile and cordless telephones, serum transthyretin and the blood-cerebrospinal fluid barrier: a cross-sectional study. *Environ Health* 2009;8:19.
178. Soderqvist F, Carlberg M, Hansson Mild K, Hardell L. Exposure to an 890-MHz mobile phone-like signal and serum levels of S100B and transthyretin in volunteers. *Toxicol Lett* 2009;189:63-6.
179. Nittby H, Grafstrom G, Eberhardt JL, et al. Radiofrequency and extremely low-frequency electromagnetic field effects on the blood-brain barrier. *Electromagn Biol Med* 2008;27:103-26.
180. McQuade JM, Merritt JH, Miller SA, et al. xRadiofrequency-radiation exposure does not induce detectable leakage of albumin across the blood-brain barrier. *Radiat Res* 2009;171:615-21.
181. de Gannes FP, Billaudel B, Taxile M, et al. Effects of head-only exposure of rats to GSM-900 on blood-brain barrier permeability and neuronal degeneration X NO EFFECT. *Radiat Res* 2009;172:359-67.
182. Franke H, Streckert J, Bitz A, et al. xEffects of Universal Mobile Telecommunications System (UMTS) electromagnetic fields on the blood-brain barrier **in vitro**. *Radiat Res* 2005;164:258-69.
183. Finnie JW, Blumbergs PC, Manavis J, et al. xEffect of long-term mobile communication microwave exposure on vascular permeability in mouse brain. *Pathology* 2002;34:344-7.
184. Fritze K, Sommer C, Schmitz B, et al. xEffect of global system for mobile communication (GSM) microwave exposure on blood-brain barrier permeability in rat. *Acta Neuropathol* 1997;94:465-70.
185. Franke H, Ringelstein EB, Stogbauer F. xElectromagnetic fields (GSM 1800) do not alter blood-brain barrier permeability to sucrose in models in vitro with high barrier tightness. *Bioelectromagnetics* 2005;26:529-35.
186. Finnie JW, Blumbergs PC, Cai Z, Manavis J, Kuchel TR. xEffect of mobile telephony on blood-brain barrier permeability in the fetal mouse brain {X NO effect}. *Pathology* 2006;38:63-5.



187. Belyaev IY, Koch CB, Terenius O, et al. Exposure of rat brain to 915 MHz GSM microwaves induces changes in gene expression but not double stranded DNA breaks or effects on chromatin conformation. *Bioelectromagnetics* 2006;27:295-306.
188. Adair JC, Baldwin N, Kornfeld M, Rosenberg GA. Radiation-induced blood-brain barrier damage in astrocytoma: relation to elevated gelatinase B and urokinase. *J Neurooncol* 1999;44:283-9.
189. Witt KA, Mark KS, Sandoval KE, Davis TP. Reoxygenation stress on blood-brain barrier paracellular permeability and edema in the rat. *Microvasc Res* 2008;75:91-6.
190. Elmorsy E, Elzalabany LM, Elsheikha HM, Smith PA. Adverse effects of antipsychotics on micro-vascular endothelial cells of the human blood-brain barrier. *Brain Res* 2014.
191. Harris G. Tillerson says U.S. may close Cuba embassy over mystery ailments. *New York Times* 2017;Sept 17.
192. Harris G, Goldman A. Illnesses at U.S. embassy in Havana prompt evacuation of more diplomats. *New York Times* 2017;Sept 29.
193. Harris G, Goldman A. U.S. pares embassy in Cuba over mystery attack. *The New York Times* 2017;Sept 30.
194. Rogers A. Were US diplomats in Cuba victims of a sonic attack -- or something else? *Wired* 2017;Oct 5.
195. Dehghan F, Khaksari Hadad M, Asadikram G, Najafipour H, Shahrokhi N. Effect of melatonin on intracranial pressure and brain edema following traumatic brain injury: role of oxidative stresses. *Arch Med Res* 2013;44:251-8.
196. Ding K, Wang H, Xu J, et al. Melatonin stimulates antioxidant enzymes and reduces oxidative stress in experimental traumatic brain injury: the Nrf2-ARE signaling pathway as a potential mechanism. *Free Radic Biol Med* 2014;73:1-11.
197. Ozdemir D, Uysal N, Gonenc S, et al. Effect of melatonin on brain oxidative damage induced by traumatic brain injury in immature rats. *Physiol Res* 2005;54:631-7.
198. Senol N, Naziroglu M. Melatonin reduces traumatic brain injury-induced oxidative stress in the cerebral cortex and blood of rats. *Neural Regen Res* 2014;9:1112-6.
199. Sainz RM, Reiter RJ, Tan DX, et al. Critical role of glutathione in melatonin enhancement of tumor necrosis factor and ionizing radiation-induced apoptosis in prostate cancer cells in vitro. *J Pineal Res* 2008;45:258-70.
200. Sener G, Atasoy BM, Ersoy Y, Arbak S, Sengoz M, Yegen BC. Melatonin protects against ionizing radiation-induced oxidative damage in corpus cavernosum and urinary bladder in rats. *J Pineal Res* 2004;37:241-6.
201. Sener G, Jahovic N, Tosun O, Atasoy BM, Yegen BC. Melatonin ameliorates ionizing radiation-induced oxidative organ damage in rats. *Life Sci* 2003;74:563-72.
202. Sharma S, Halder C. Melatonin prevents X-ray irradiation induced oxidative damage in peripheral blood and spleen of the seasonally breeding rodent, *Funambulus pennanti* during reproductively active phase. *Int J Radiat Biol* 2006;82:411-9.
203. Shirazi A, Haddadi GH, Asadi-Amoli F, Sakhaee S, Ghazi-Khansari M, Avand A. Radioprotective effect of melatonin in reducing oxidative stress in rat lenses. *Cell J* 2011;13:79-82.
204. Shirazi A, Mihandoost E, Mohseni M, Ghazi-Khansari M, Rabie Mahdavi S. Radio-protective effects of melatonin against irradiation-induced oxidative damage in rat peripheral blood. *Phys Med* 2013;29:65-74.
205. Taysi S, Koc M, Buyukokuroglu ME, Altinkaynak K, Sahin YN. Melatonin reduces lipid peroxidation and nitric oxide during irradiation-induced oxidative injury in the rat liver. *J Pineal Res* 2003;34:173-7.
206. Taysi S, Memisogullari R, Koc M, et al. Melatonin reduces oxidative stress in the rat lens due to radiation-induced oxidative injury. *Int J Radiat Biol* 2008;84:803-8.
207. Vasin MV, Ushakov IB, Kovtun V, Komarova SN, Semenova LA, Galkin AA. [Comparative effectiveness of antioxidant melatonin and radioprotectors indralin and phenylephrine in local radiation injuries]. *Radiats Biol Radioecol* 2004;44:68-71.
208. Yilmaz S, Yilmaz E. Effects of melatonin and vitamin E on oxidative-antioxidative status in rats exposed to irradiation. *Toxicology* 2006;222:1-7.
209. Oliinyk EV, Meshchyshen IF. [Effect of melatonin and radiation on pro- and antioxidant state of the liver and blood of rats]. *Ukr Biokhim Zh* (1999) 2004;76:144-7.
210. Ortiz F, Acuna-Castroviejo D, Doerrier C, et al. Melatonin blunts the mitochondrial/NLRP3 connection and protects against radiation-induced oral mucositis. *J Pineal Res* 2015;58:34-49.
211. Naziroglu M, Tokat S, Demirci S. Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca<sup>2+</sup> signaling molecular pathways in breast cancer. *J Recept Signal Transduct Res* 2012;32:290-7.
212. Liu DD, Ren Z, Yang G, Zhao QR, Mei YA. Melatonin protects rat cerebellar granule cells against electromagnetic field-induced increases in Na(+) currents through intracellular Ca(2+) release. *J Cell Mol Med* 2014;18:1060-70.

213. Manda K, Anzai K, Kumari S, Bhatia AL. Melatonin attenuates radiation-induced learning deficit and brain oxidative stress in mice. *Acta Neurobiol Exp (Wars)* 2007;67:63-70.
214. Manda K, Reiter RJ. Melatonin maintains adult hippocampal neurogenesis and cognitive functions after irradiation. *Prog Neurobiol* 2010;90:60-8.
215. Manda K, Ueno M, Anzai K. AFMK, a melatonin metabolite, attenuates X-ray-induced oxidative damage to DNA, proteins and lipids in mice. *J Pineal Res* 2007;42:386-93.
216. Manda K, Ueno M, Anzai K. Melatonin mitigates oxidative damage and apoptosis in mouse cerebellum induced by high-LET <sup>56</sup>Fe particle irradiation. *J Pineal Res* 2008;44:189-96.
217. Jang SS, Kim HG, Lee JS, et al. Melatonin reduces X-ray radiation-induced lung injury in mice by modulating oxidative stress and cytokine expression. *Int J Radiat Biol* 2013;89:97-105.
218. Kim BC, Shon BS, Ryoo YW, Kim SP, Lee KS. Melatonin reduces X-ray irradiation-induced oxidative damages in cultured human skin fibroblasts. *J Dermatol Sci* 2001;26:194-200.
219. Koc M, Taysi S, Buyukokuroglu ME, Bakan N. Melatonin protects rat liver against irradiation-induced oxidative injury. *J Radiat Res* 2003;44:211-5.
220. Koc M, Taysi S, Emin Buyukokuroglu M, Bakan N. The effect of melatonin against oxidative damage during total-body irradiation in rats. *Radiat Res* 2003;160:251-5.
221. El-Missiry MA, Fayed TA, El-Sawy MR, El-Sayed AA. Ameliorative effect of melatonin against gamma-irradiation-induced oxidative stress and tissue injury. *Ecotoxicol Environ Saf* 2007;66:278-86.
222. Goswami S, Haldar C. UVB irradiation severely induces systemic tissue injury by augmenting oxidative load in a tropical rodent: efficacy of melatonin as an antioxidant. *J Photochem Photobiol B* 2014;141:84-92.
223. Goswami S, Haldar C. Melatonin improves ultraviolet B-induced oxidative damage and inflammatory conditions in cutaneous tissue of a diurnal Indian palm squirrel *Funambulus pennanti*. *Br J Dermatol* 2014;171:1147-55.
224. Goswami S, Sharma S, Haldar C. The oxidative damages caused by ultraviolet radiation type C (UVC) to a tropical rodent *Funambulus pennanti*: role of melatonin. *J Photochem Photobiol B* 2013;125:19-25.
225. Guney Y, Hicsonmez A, Uluoglu C, et al. Melatonin prevents inflammation and oxidative stress caused by abdominopelvic and total body irradiation of rat small intestine. *Braz J Med Biol Res* 2007;40:1305-14.
226. Bardak Y, Ozerturk Y, Ozguner F, Durmus M, Delibas N. Effect of melatonin against oxidative stress in ultraviolet-B exposed rat lens. *Curr Eye Res* 2000;20:225-30.
227. Bhatia AL, Manda K. Study on pre-treatment of melatonin against radiation-induced oxidative stress in mice. *Environ Toxicol Pharmacol* 2004;18:13-20.
228. Ayata A, Mollaoglu H, Yilmaz HR, Akturk O, Ozguner F, Altuntas I. Oxidative stress-mediated skin damage in an experimental mobile phone model can be prevented by melatonin. *J Dermatol* 2004;31:878-83.
229. Aynali G, Naziroglu M, Celik O, Dogan M, Yariktas M, Yasan H. Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin. *Eur Arch Otorhinolaryngol* 2013;270:1695-700.
230. Koylu H, Mollaoglu H, Ozguner F, Naziroglu M, Delibas N. Melatonin modulates 900 Mhz microwave-induced lipid peroxidation changes in rat brain. *Toxicol Ind Health* 2006;22:211-6.
231. Lai H, Singh NP. Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells. *Bioelectromagnetics* 1997;18:446-54.
232. Meena R, Kumari K, Kumar J, Rajamani P, Verma HN, Kesari KK. Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats. *Electromagn Biol Med* 2014;33:81-91.
233. Naziroglu M, Celik O, Ozgul C, et al. Melatonin modulates wireless (2.45 GHz)-induced oxidative injury through TRPM2 and voltage gated Ca(2+) channels in brain and dorsal root ganglion in rat. *Physiol Behav* 2012;105:683-92.
234. Oksay T, Naziroglu M, Dogan S, Guzel A, Gumral N, Kosar PA. Protective effects of melatonin against oxidative injury in rat testis induced by wireless (2.45 GHz) devices. *Andrologia* 2012.
235. Oktem F, Ozguner F, Mollaoglu H, Koyu A, Uz E. Oxidative damage in the kidney induced by 900-MHz-emitted mobile phone: protection by melatonin. *Arch Med Res* 2005;36:350-5.
236. Ozguner F, Bardak Y, Comlekci S. Protective effects of melatonin and caffeic acid phenethyl ester against retinal oxidative stress in long-term use of mobile phone: a comparative study. *Mol Cell Biochem* 2006;282:83-8.
237. Ozguner F, Oktem F, Armagan A, et al. Comparative analysis of the protective effects of melatonin and caffeic acid phenethyl ester (CAPE) on mobile phone-induced renal impairment in rat. *Mol Cell Biochem* 2005;276:31-7.
238. Sokolovic D, Djindjic B, Nikolic J, et al. Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain. *J Radiat Res* 2008;49:579-86.



239. Tok L, Naziroglu M, Dogan S, Kahya MC, Tok O. Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats. *Indian J Ophthalmol* 2014;62:12-5.
240. Xu S, Zhou Z, Zhang L, et al. Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons. *Brain Res* 2010;1311:189-96.
241. Math teacher raises concerns about WIFI comparing the effects to a concussion. North Kingston School Committee Meeting - Rhode Island USA May 13, 2014 2014; s://[www.youtube.com/watch?v=QbgIdyhAXfM](https://www.youtube.com/watch?v=QbgIdyhAXfM):Posted February 11, 2016 by Parents for Safe Technology. Download date Feb 14, 8.
242. Miyamoto N, Maki T, Pham LD, et al. Oxidative stress interferes with white matter renewal after prolonged cerebral hypoperfusion in mice. *Stroke* 2013;44:3516-21.
243. Rosenzweig S, Carmichael ST. Age-dependent exacerbation of white matter stroke outcomes: a role for oxidative damage and inflammatory mediators. *Stroke* 2013;44:2579-86.
244. Casta A, Quackenbush EJ, Houck CS, Korson MS. Perioperative white matter degeneration and death in a patient with a defect in mitochondrial oxidative phosphorylation. *Anesthesiology* 1997;87:420-5.
245. Back SA, Luo NL, Mallinson RA, et al. Selective vulnerability of preterm white matter to oxidative damage defined by F2-isoprostanes. *Ann Neurol* 2005;58:108-20.
246. Ikeda T, Choi BH, Yee S, Murata Y, Quilligan EJ. Oxidative stress, brain white matter damage and intrauterine asphyxia in fetal lambs. *Int J Dev Neurosci* 1999;17:1-14.
247. Miller VM, Lawrence DA, Mondal TK, Seegal RF. Reduced glutathione is highly expressed in white matter and neurons in the unperturbed mouse brain--implications for oxidative stress associated with neurodegeneration. *Brain Res* 2009;1276:22-30.
248. Munoz-Cortes M, Cabre C, Villa D, et al. Oxidative stress and other risk factors for white matter lesions in chronic hemodialysis patients. *Clin Nephrol* 2013;80:187-97.
249. Kumagai S, Jikimoto T, Saegusa J. [Pathological roles of oxidative stress in autoimmune diseases]. *Rinsho Byori* 2003;51:126-32.
250. Gelderman KA, Hultqvist M, Olsson LM, et al. Rheumatoid arthritis: the role of reactive oxygen species in disease development and therapeutic strategies. *Antioxid Redox Signal* 2007;9:1541-67.
251. Iborra A, Palacio JR, Martinez P. Oxidative stress and autoimmune response in the infertile woman. *Chem Immunol Allergy* 2005;88:150-62.
252. Iuchi Y, Kibe N, Tsunoda S, et al. Implication of oxidative stress as a cause of autoimmune hemolytic anemia in NZB mice. *Free Radic Biol Med* 2010;48:935-44.
253. Kalluri R, Cantley LG, Kerjaschki D, Neilson EG. Reactive oxygen species expose cryptic epitopes associated with autoimmune goodpasture syndrome. *J Biol Chem* 2000;275:20027-32.
254. Liu Y, Zhu B, Wang X, et al. Bilirubin as a potent antioxidant suppresses experimental autoimmune encephalomyelitis: implications for the role of oxidative stress in the development of multiple sclerosis. *J Neuroimmunol* 2003;139:27-35.
255. Maes M, Kubera M, Mihaylova I, et al. Increased autoimmune responses against auto-epitopes modified by oxidative and nitrosative damage in depression: implications for the pathways to chronic depression and neuroprogression. *J Affect Disord* 2013;149:23-9.
256. Profumo E, Buttari B, Rigano R. Oxidative stress in cardiovascular inflammation: its involvement in autoimmune responses. *Int J Inflam* 2011;2011:295705.
257. Shah AA, Sinha AA. Oxidative stress and autoimmune skin disease. *Eur J Dermatol* 2013;23:5-13.
258. Wang G, Cai P, Ansari GA, Khan MF. Oxidative and nitrosative stress in trichloroethene-mediated autoimmune response. *Toxicology* 2007;229:186-93.
259. Santini R, Santini P, Danze JM, Le Ruz P, Seigne M. [Investigation on the health of people living near mobile telephone relay stations: Incidence according to distance and sex]. *Pathol Biol (Paris)* 2002;50:369-73.
260. Holmboe G, Johansson O. Symptombeskrivning samt förekomst av IgE och positiv Phadiatop Combi hos personer med funktionsnedsättningen elöverkänslighet. [Description of symptoms as well as occurrence of IgE and positive Phadiatop Combi in persons with the physical impairment electrohypersensitivity}. *Medicinsk Access* 2005;1::58-63.
261. Durusoy R, Hassoy H, Ozkurt A, Karababa AO. Mobile phone use, school electromagnetic field levels and related symptoms: a cross-sectional survey among 2150 high school students in Izmir. *Environ Health* 2017;16:51.
262. The Associated Press. US Cuban diplomats to discuss health incidents. Sept 18 2017.
263. Abdel-Rassoul G, Abou El-Fateh O, Abou Salem M, et al. Neurobehavioral effects among inhabitants around mobile phone base stations. *NeuroToxicology* 2007;28.

264. Gruber J, Schaffer S, Halliwell B. The mitochondrial free radical theory of ageing--where do we stand? *Front Biosci* 2008;13:6554-79.
265. Kowald A. The mitochondrial theory of aging. *Biol Signals Recept* 2001;10:162-75.
266. Sastre J, Pallardo FV, Vina J. The role of mitochondrial oxidative stress in aging. *Free Radic Biol Med* 2003;35:1-8.
267. Wei YH. Oxidative stress and mitochondrial DNA mutations in human aging. *Proc Soc Exp Biol Med* 1998;217:53-63.
268. Fetoni AR, De Bartolo P, Eramo SLM, et al. Noise-Induced Hearing Loss (NIHL) as a Target of Oxidative Stress-Mediated Damage: Cochlear and Cortical Responses after an Increase in Antioxidant Defense. *The Journal of Neuroscience* 2013;33:4011-23.
269. Neri S, Signorelli S, Pulvirenti D, et al. Oxidative stress, nitric oxide, endothelial dysfunction and tinnitus. *Free Radic Res* 2006;40:615-8.
270. Savastano M, Brescia G, Marioni G. Antioxidant therapy in idiopathic tinnitus: preliminary outcomes. *Arch Med Res* 2007;38:456-9.
271. Vurucu S, Karaoglu A, Paksu MS, et al. Relationship between oxidative stress and chronic daily headache in children. *Human & Experimental Toxicology* 2013;32:113-9.
272. Adamczyk-Sowa M, Pierzchala K, Sowa P, et al. Melatonin acts as antioxidant and improves sleep in MS patients. *Neurochem Res* 2014;39:1585-93.
273. Sharma AK, Mehta AK, Rathor N, Chalawadi Hanumantappa MK, Khanna N, Bhattacharya SK. Melatonin attenuates cognitive dysfunction and reduces neural oxidative stress induced by phosphamidon. *Fundam Clin Pharmacol* 2013;27:146-51.
274. Zhang L, Zhang HQ, Liang XY, Zhang HF, Zhang T, Liu FE. Melatonin ameliorates cognitive impairment induced by sleep deprivation in rats: role of oxidative stress, BDNF and CaMKII. *Behav Brain Res* 2013;256:72-81.
275. Insel KC, Moore IM, Vidrine AN, Montgomery DW. Biomarkers for cognitive aging part II: oxidative stress, cognitive assessments, and medication adherence. *Biol Res Nurs* 2012;14:133-8.
276. Tiwari V, Chopra K. Resveratrol abrogates alcohol-induced cognitive deficits by attenuating oxidative-nitrosative stress and inflammatory cascade in the adult rat brain. *Neurochem Int* 2013;62:861-9.
277. Reynolds A, Laurie C, Mosley RL, Gendelman HE. Oxidative stress and the pathogenesis of neurodegenerative disorders. *Int Rev Neurobiol* 2007;82:297-325.
278. Fukui K, Omoi NO, Hayasaka T, et al. Cognitive impairment of rats caused by oxidative stress and aging, and its prevention by vitamin E. *Ann N Y Acad Sci* 2002;959:275-84.
279. Berr C, Balansard B, Arnaud J, Roussel AM, Alperovitch A. Cognitive decline is associated with systemic oxidative stress: the EVA study. *Etude du Vieillissement Arteriel. J Am Geriatr Soc* 2000;48:1285-91.
280. Kilic A, Selek S, Erel O, Aksoy N. Protective effects of melatonin on oxidative-antioxidative balance and cataract formation in rats. *Ann Ophthalmol (Skokie)* 2008;40:22-7.
281. Bonne C, Muller A. [Role of oxidative stress in age-related macular degeneration]. *J Fr Ophtalmol* 2000;23:835-40.
282. Zoric L, Kosanovic-Jakovic N, Colak E, Radosavljevic A, Jaksic V, Stevic S. [Oxidative stress in association with risk factors for the occurrence and development of age-related macular degeneration]. *Vojnosanit Pregl* 2008;65:313-8.
283. Seidman MD, Khan MJ, Bai U, Shirwany N, Quirk WS. Biologic activity of mitochondrial metabolites on aging and age-related hearing loss. *Am J Otol* 2000;21:161-7.
284. Jeyakumar A, Williamson ME, Brickman TM, Krakovitz P, Parikh S. Otolaryngologic manifestations of mitochondrial cytopathies. *Am J Otolaryngol* 2009;30:162-5.
285. Hoshino S, Tamaoka A, Ohkoshi N, Shoji S, Goto Y. [A case of mitochondrial encephalomyopathy showing ophthalmoplegia, diabetes mellitus and hearing loss associated with the A3243G mutation of mitochondrial DNA]. *Rinsho Shinkeigaku* 1997;37:326-30.
286. Someya S, Xu J, Kondo K, et al. Age-related hearing loss in C57BL/6J mice is mediated by Bak-dependent mitochondrial apoptosis. *Proc Natl Acad Sci U S A* 2009;106:19432-7.
287. Yamasoba T, Someya S, Yamada C, Weindruch R, Prolla TA, Tanokura M. Role of mitochondrial dysfunction and mitochondrial DNA mutations in age-related hearing loss. *Hear Res* 2007;226:185-93.
288. Manwaring N, Jones MM, Wang JJ, et al. Mitochondrial DNA haplogroups and age-related hearing loss. *Arch Otolaryngol Head Neck Surg* 2007;133:929-33.
289. Koga Y, Nataliya P. [Migraine headache and mitochondrial DNA abnormality]. *Nihon Rinsho* 2005;63:1720-6.
290. Rosen N. Headache and mitochondrial disorders. *Headache* 2008;48:733-4.

291. Koillinen H, Jaaskelainen S, Koski K. [Mitochondrial disorder underlying headache symptoms]. *Duodecim* 2009;125:297-300.
292. Ikeda-Douglas CJ, Zicker SC, Estrada J, Jewell DE, Milgram NW. Prior experience, antioxidants, and mitochondrial cofactors improve cognitive function in aged beagles. *Vet Ther* 2004;5:5-16.
293. Finsterer J. Cognitive decline as a manifestation of mitochondrial disorders (mitochondrial dementia). *J Neurol Sci* 2008;272:20-33.
294. Kuruppu DK, Matthews BR. Young-onset dementia. *Semin Neurol* 2013;33:365-85.
295. Wallace DC. Mitochondrial defects in neurodegenerative disease. *Ment Retard Dev Disabil Res Rev* 2001;7:158-66.
296. Carelli V, Ross-Cisneros FN, Sadun AA. Optic nerve degeneration and mitochondrial dysfunction: genetic and acquired optic neuropathies. *Neurochem Int* 2002;40:573-84.
297. Riordan-Eva P. Neuro-ophthalmology of mitochondrial diseases. *Curr Opin Ophthalmol* 2000;11:408-12.
298. Massin P, Guillausseau PJ, Vialettes B, et al. Macular pattern dystrophy associated with a mutation of mitochondrial DNA. *Am J Ophthalmol* 1995;120:247-8.
299. Brubaker JW, Mohny BG, Pulido JS. Cystoid macular edema in a patient with chronic progressive external ophthalmoplegia with mitochondrial myopathy. *Ophthalmic Genet* 2009;30:50-3.
300. Feng Z, Liu Z, Li X, et al. alpha-Tocopherol is an effective Phase II enzyme inducer: protective effects on acrolein-induced oxidative stress and mitochondrial dysfunction in human retinal pigment epithelial cells. *J Nutr Biochem* 2010;21:1222-31.
301. Van Campen LE, Murphy WJ, Franks JR, Mathias PI, Toraason MA. Oxidative DNA damage is associated with intense noise exposure in the rat. *Hear Res* 2002;164:29-38.
302. Griefahn B, Kunemund C, Blaszkewicz M, Lerchl A, Degen GH. Effects of electromagnetic radiation (bright light, extremely low-frequency magnetic fields, infrared radiation) on the circadian rhythm of melatonin synthesis, rectal temperature, and heart rate. *Ind Health* 2002;40:320-7.
303. Imaida K, Hagiwara A, Yoshino H, et al. Inhibitory effects of low doses of melatonin on induction of preneoplastic liver lesions in a medium-term liver bioassay in F344 rats: relation to the influence of electromagnetic near field exposure. *Cancer Lett* 2000;155:105-14.
304. Sokolovic D, Djordjevic B, Kocic G, et al. Melatonin protects rat thymus against oxidative stress caused by exposure to microwaves and modulates proliferation/apoptosis of thymocytes. *Gen Physiol Biophys* 2013;32:79-90.
305. Guney M, Ozguner F, Oral B, Karahan N, Mungan T. 900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium: protection by vitamins E and C. *Toxicol Ind Health* 2007;23:411-20.
306. Ilhan A, Gurel A, Armutcu F, et al. Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain. *Clin Chim Acta* 2004;340:153-62.
307. Bravard A, Ageron-Blanc A, Alvarez S, et al. Correlation between antioxidant status, tumorigenicity and radiosensitivity in sister rat cell lines. *Carcinogenesis* 2002;23:705-11.
308. Dutta A, Chakraborty A, Saha A, Ray S, Chatterjee A. Interaction of radiation- and bleomycin-induced lesions and influence of glutathione level on the interaction. *Mutagenesis* 2005;20:329-35.
309. Dethmers JK, Meister A. Glutathione export by human lymphoid cells: depletion of glutathione by inhibition of its synthesis decreases export and increases sensitivity to irradiation. *Proc Natl Acad Sci U S A* 1981;78:7492-6.
310. Evans JW, Taylor YC, Brown JM. The role of glutathione and DNA strand break repair in determining the shoulder of the radiation survival curve. *Br J Cancer Suppl* 1984;6:49-53.
311. Epperly MW, Kagan VE, Sikora CA, et al. Manganese superoxide dismutase-plasmid/liposome (MnSOD-PL) administration protects mice from esophagitis associated with fractionated radiation. *Int J Cancer* 2001;96:221-31.
312. Hanada K, Gange RW, Connor MJ. Effect of Glutathione Depletion on Sunburn Cell Formation in the Hairless Mouse. *Journal of Investigative Dermatology* 1990;96:838-40.
313. Williams S, Tamburic S, Lally C. Eating chocolate can significantly protect the skin from UV light. *J Cosmet Dermatol* 2009;8:169-73.
314. Bergqvist U, Vogel E, Aringer L, et al., eds. Possible health implications of subjective symptoms and electromagnetic fields. A report prepared by a European group of experts for the European Commission, DG V. Solna, Sweden: European Commission Directorate General V. Employment, Industrial Relations and Social Affairs, National Institute for Working Life, Sweden; 1997.
315. Halgamuge MN. Critical time delay of the pineal melatonin rhythm in humans due to weak electromagnetic exposure. *Indian J Biochem Biophys* 2013;50:259-65.
316. Qin F, Zhang J, Cao H, et al. Effects of 1800-MHz radiofrequency fields on circadian rhythm of plasma melatonin and testosterone in male rats. *J Toxicol Environ Health A* 2012;75:1120-8.

317. Fernie KJ, Bird DM, Petitcherc D. Effects of electromagnetic fields on photophasic circulating melatonin levels in American kestrels. *Environ Health Perspect* 1999;107:901-4.
318. Reiter RJ. Electromagnetic fields and melatonin production. *Biomed Pharmacother* 1993;47:439-44.
319. Burch JB, Reif JS, Yost MG. Geomagnetic activity and human melatonin metabolite excretion. *Neurosci Lett* 2008;438:76-9.
320. Weydahl A, Sothorn RB, Cornélissen G, Wetterberg L. Geomagnetic activity influences the melatonin secretion at latitude 70° N. *Biomedicine & Pharmacotherapy* 2000;55, Supplement 1:s57-s62.
321. Burch JB, Reif JS, Yost MG. Geomagnetic disturbances are associated with reduced nocturnal excretion of a melatonin metabolite in humans. *Neurosci Lett* 1999;266:209-12.
322. Reiter RJ. Melatonin suppression by static and extremely low frequency electromagnetic fields: relationship to the reported increased incidence of cancer. *Rev Environ Health* 1994;10:171-86.
323. Wood AW, Loughran SP, Stough C. Does evening exposure to mobile phone radiation affect subsequent melatonin production? *Int J Radiat Biol* 2006;82:69-76.
324. Parry BL, Meliska CJ, Sorenson DL, et al. Increased sensitivity to light-induced melatonin suppression in premenstrual dysphoric disorder. *Chronobiology international* 2010;27:1438-53.
325. Gammack JK. Light therapy for insomnia in older adults. *Clin Geriatr Med* 2008;24:139-49, viii.
326. Navara KJ, Nelson RJ. The dark side of light at night: physiological, epidemiological, and ecological consequences. *J Pineal Res* 2007;43:215-24.
327. Glickman G, Byrne B, Pineda C, Hauck WW, Brainard GC. Light therapy for seasonal affective disorder with blue narrow-band light-emitting diodes (LEDs). *Biol Psychiatry* 2006;59:502-7.
328. Rapoport SI, Breus TK. [Melatonin as a most important factor of natural electromagnetic fields impacting patients with hypertensive disease and coronary heart disease. Part 1]. *Klin Med (Mosk)* 2011;89:9-14.
329. Singh S, Mani KV, Kapoor N. Effect of occupational EMF exposure from radar at two different frequency bands on plasma melatonin and serotonin levels. *Int J Radiat Biol* 2015:1-9.
330. El-Helaly M, Abu-Hashem E. Oxidative stress, melatonin level, and sleep insufficiency among electronic equipment repairers. *Indian J Occup Environ Med* 2010;14:66-70.
331. Montilla PL, Vargas JF, Tunes IF, Munoz de Agueda MC, Valdevira ME, Cabrera ES. Oxidative stress in diabetic rats induced by streptozotocin: protective effects of melatonin. *J Pineal Res* 1998;25:94-100.
332. Melchiorri D, Reiter RJ, Attia AM, Hara M, Burgos A, Nistico G. Potent protective effect of melatonin on in vivo paraquat-induced oxidative damage in rats. *Life Sci* 1995;56:83-9.
333. Garcia-Rubio L, Matas P, Miguez MP. Protective effect of melatonin on paraquat-induced cytotoxicity in isolated rat hepatocytes. *Hum Exp Toxicol* 2005;24:475-80.
334. Bandyopadhyay D, Ghosh G, Bandyopadhyay A, Reiter RJ. Melatonin protects against piroxicam-induced gastric ulceration. *J Pineal Res* 2004;36:195-203.
335. Sousa SC, Castilho RF. Protective effect of melatonin on rotenone plus Ca<sup>2+</sup>-induced mitochondrial oxidative stress and PC12 cell death. *Antioxid Redox Signal* 2005;7:1110-6.
336. Thomas B, Mohanakumar KP. Melatonin protects against oxidative stress caused by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine in the mouse nigrostriatum. *J Pineal Res* 2004;36:25-32.
337. Chen LJ, Gao YQ, Li XJ, Shen DH, Sun FY. Melatonin protects against MPTP/MPP<sup>+</sup>-induced mitochondrial DNA oxidative damage in vivo and in vitro. *J Pineal Res* 2005;39:34-42.
338. Skaper SD, Floreani M, Ceccon M, Facci L, Giusti P. Excitotoxicity, oxidative stress, and the neuroprotective potential of melatonin. *Ann N Y Acad Sci* 1999;890:107-18.
339. Abdel Moneim AE, Ortiz F, Leonardo-Mendonca RC, et al. Protective effects of melatonin against oxidative damage induced by Egyptian cobra (*Naja haje*) crude venom in rats. *Acta Trop* 2015;143:58-65.
340. Othman AI, Edrees GM, El-Missiry MA, Ali DA, Aboel-Nour M, Dabdoub BR. Melatonin controlled apoptosis and protected the testes and sperm quality against bisphenol A-induced oxidative toxicity. *Toxicol Ind Health* 2014.
341. El-Missiry MA, Othman AI, Al-Abdan MA, El-Sayed AA. Melatonin ameliorates oxidative stress, modulates death receptor pathway proteins, and protects the rat cerebrum against bisphenol-A-induced apoptosis. *J Neurol Sci* 2014;347:251-6.
342. Souza LC, Wilhelm EA, Bortolatto CF, Nogueira CW, Boeira SP, Jesse CR. The protective effect of melatonin against brain oxidative stress and hyperlocomotion in a rat model of mania induced by ouabain. *Behav Brain Res* 2014;271:316-24.
343. Mehta KD, Mehta AK, Halder S, Khanna N, Tripathi AK, Sharma KK. Protective effect of melatonin on propoxur-induced impairment of memory and oxidative stress in rats. *Environ Toxicol* 2014;29:705-13.



344. Shokrzadeh M, Chabra A, Naghshvar F, Ahmadi A, Jafarnejhad M, Hasani-Nourian Y. Protective Effects of Melatonin against Cyclophosphamide-induced Oxidative Lung Toxicity in Mice. *Drug Res (Stuttg)* 2015;65:281-6.
345. Uygur R, Aktas C, Caglar V, Uygur E, Erdogan H, Ozen OA. Protective effects of melatonin against arsenic-induced apoptosis and oxidative stress in rat testes. *Toxicol Ind Health* 2013.
346. Chabra A, Shokrzadeh M, Naghshvar F, Salehi F, Ahmadi A. Melatonin ameliorates oxidative stress and reproductive toxicity induced by cyclophosphamide in male mice. *Hum Exp Toxicol* 2014;33:185-95.
347. Ebaid H, Bashandy SA, Alhazza IM, Rady A, El-Shehry S. Folic acid and melatonin ameliorate carbon tetrachloride-induced hepatic injury, oxidative stress and inflammation in rats. *Nutr Metab (Lond)* 2013;10:20.
348. Antunes Wilhelm E, Ricardo Jesse C, Folharini Bortolatto C, Wayne Nogueira C. Correlations between behavioural and oxidative parameters in a rat quinolinic acid model of Huntington's disease: protective effect of melatonin. *Eur J Pharmacol* 2013;701:65-72.
349. Korkmaz GG, Uzun H, Cakatay U, Aydin S. Melatonin ameliorates oxidative damage in hyperglycemia-induced liver injury. *Clin Invest Med* 2012;35:E370-7.
350. Baxi DB, Singh PK, Vachhrajani KD, Ramachandran AV. Melatonin supplementation in rat ameliorates ovariectomy-induced oxidative stress. *Climacteric* 2013;16:274-83.
351. Ochoa JJ, Diaz-Castro J, Kajarabille N, et al. Melatonin supplementation ameliorates oxidative stress and inflammatory signaling induced by strenuous exercise in adult human males. *J Pineal Res* 2011;51:373-80.
352. Jindal M, Garg GR, Mediratta PK, Fahim M. Protective role of melatonin in myocardial oxidative damage induced by mercury in murine model. *Hum Exp Toxicol* 2011;30:1489-500.
353. Laothong U, Pinlaor P, Hiraku Y, et al. Protective effect of melatonin against *Opisthorchis viverrini*-induced oxidative and nitrosative DNA damage and liver injury in hamsters. *J Pineal Res* 2010;49:271-82.
354. Fuentes-Broto L, Miana-Mena FJ, Piedrafita E, et al. Melatonin protects against tauro lithocholic-induced oxidative stress in rat liver. *J Cell Biochem* 2010;110:1219-25.
355. Xu SC, He MD, Zhong M, et al. Melatonin protects against Nickel-induced neurotoxicity in vitro by reducing oxidative stress and maintaining mitochondrial function. *J Pineal Res* 2010;49:86-94.
356. Aranda M, Albendea CD, Lostale F, et al. In vivo hepatic oxidative stress because of carbon tetrachloride toxicity: protection by melatonin and pinoline. *J Pineal Res* 2010;49:78-85.
357. El-Sokkary GH, Nafady AA, Shabash EH. Melatonin administration ameliorates cadmium-induced oxidative stress and morphological changes in the liver of rat. *Ecotoxicol Environ Saf* 2010;73:456-63.
358. Hu S, Yin S, Jiang X, Huang D, Shen G. Melatonin protects against alcoholic liver injury by attenuating oxidative stress, inflammatory response, and apoptosis. *Eur J Pharmacol* 2009;616:287-92.
359. Rao MV, Chhunchha B. Protective role of melatonin against the mercury induced oxidative stress in the rat thyroid. *Food Chem Toxicol* 2010;48:7-10.
360. Ozacmak VH, Barut F, Ozacmak HS. Melatonin provides neuroprotection by reducing oxidative stress and HSP70 expression during chronic cerebral hypoperfusion in ovariectomized rats. *J Pineal Res* 2009;47:156-63.
361. Fagundes DS, Gonzalo S, Arruebo MP, Plaza MA, Murillo MD. Melatonin and Trolox ameliorate duodenal LPS-induced disturbances and oxidative stress. *Dig Liver Dis* 2010;42:40-4.
362. Das A, Belagodu A, Reiter RJ, Ray SK, Banik NL. Cytoprotective effects of melatonin on C6 astroglial cells exposed to glutamate excitotoxicity and oxidative stress. *J Pineal Res* 2008;45:117-24.
363. Omurtag GZ, Tozan A, Sehirli AO, Sener G. Melatonin protects against endosulfan-induced oxidative tissue damage in rats. *J Pineal Res* 2008;44:432-8.
364. Saravanan KS, Sindhu KM, Mohanakumar KP. Melatonin protects against rotenone-induced oxidative stress in a hemiparkinsonian rat model. *J Pineal Res* 2007;42:247-53.
365. Sadir S, Deveci S, Korkmaz A, Oter S. Alpha-tocopherol, beta-carotene and melatonin administration protects cyclophosphamide-induced oxidative damage to bladder tissue in rats. *Cell Biochem Funct* 2007;25:521-6.
366. Suke SG, Kumar A, Ahmed RS, et al. Protective effect of melatonin against propoxur-induced oxidative stress and suppression of humoral immune response in rats. *Indian J Exp Biol* 2006;44:312-5.
367. Esrefoglu M, Gul M, Ates B, Selimoglu MA. Ultrastructural clues for the protective effect of melatonin against oxidative damage in cerulein-induced pancreatitis. *J Pineal Res* 2006;40:92-7.
368. Carrillo-Vico A, Lardone PJ, Naji L, et al. Beneficial pleiotropic actions of melatonin in an experimental model of septic shock in mice: regulation of pro-/anti-inflammatory cytokine network, protection against oxidative damage and anti-apoptotic effects. *J Pineal Res* 2005;39:400-8.
369. Rezzani R, Buffoli B, Rodella L, Stacchiotti A, Bianchi R. Protective role of melatonin in cyclosporine A-induced oxidative stress in rat liver. *Int Immunopharmacol* 2005;5:1397-405.

370. Wang H, Wei W, Wang NP, et al. Melatonin ameliorates carbon tetrachloride-induced hepatic fibrogenesis in rats via inhibition of oxidative stress. *Life Sci* 2005;77:1902-15.
371. Kerman M, Cirak B, Ozguner MF, et al. Does melatonin protect or treat brain damage from traumatic oxidative stress? *Exp Brain Res* 2005;163:406-10.
372. Esrefoglu M, Gul M, Emre MH, Polat A, Selimoglu MA. Protective effect of low dose of melatonin against cholestatic oxidative stress after common bile duct ligation in rats. *World J Gastroenterol* 2005;11:1951-6.
373. Kacmaz A, User EY, Sehirli AO, Tilki M, Ozkan S, Sener G. Protective effect of melatonin against ischemia/reperfusion-induced oxidative remote organ injury in the rat. *Surg Today* 2005;35:744-50.
374. Wang H, Wei W, Zhang SY, et al. Melatonin-selenium nanoparticles inhibit oxidative stress and protect against hepatic injury induced by Bacillus Calmette-Guerin/lipopolysaccharide in mice. *J Pineal Res* 2005;39:156-63.
375. Ozacmak VH, Sayan H, Arslan SO, Altaner S, Aktas RG. Protective effect of melatonin on contractile activity and oxidative injury induced by ischemia and reperfusion of rat ileum. *Life Sci* 2005;76:1575-88.
376. Sahna E, Parlakpınar H, Turkoz Y, Acet A. Protective effects of melatonin on myocardial ischemia/reperfusion induced infarct size and oxidative changes. *Physiol Res* 2005;54:491-5.
377. Sahna E, Parlakpınar H, Vardi N, Cigremis Y, Acet A. Efficacy of melatonin as protectant against oxidative stress and structural changes in liver tissue in pinealectomized rats. *Acta Histochem* 2004;106:331-6.
378. Watanabe K, Wakatsuki A, Shinohara K, Ikenoue N, Yokota K, Fukaya T. Maternally administered melatonin protects against ischemia and reperfusion-induced oxidative mitochondrial damage in premature fetal rat brain. *J Pineal Res* 2004;37:276-80.
379. Tunez I, Montilla P, Del Carmen Munoz M, Feijoo M, Salcedo M. Protective effect of melatonin on 3-nitropropionic acid-induced oxidative stress in synaptosomes in an animal model of Huntington's disease. *J Pineal Res* 2004;37:252-6.
380. Gupta YK, Gupta M, Kohli K. Neuroprotective role of melatonin in oxidative stress vulnerable brain. *Indian J Physiol Pharmacol* 2003;47:373-86.
381. Ozelcik N, Soyoz M, Kilinc I. Effects of ochratoxin a on oxidative damage in rat kidney: protective role of melatonin. *J Appl Toxicol* 2004;24:211-5.
382. Zavodnik IB, Lapshina EA, Zavodnik LB, Labieniec M, Bryszewska M, Reiter RJ. Hypochlorous acid-induced oxidative stress in Chinese hamster B14 cells: viability, DNA and protein damage and the protective action of melatonin. *Mutat Res* 2004;559:39-48.
383. Sener G, Paskaloglu K, Toklu H, et al. Melatonin ameliorates chronic renal failure-induced oxidative organ damage in rats. *J Pineal Res* 2004;36:232-41.
384. Sener G, Sehirli AO, Ayanoglu-Dulger G. Melatonin protects against mercury(II)-induced oxidative tissue damage in rats. *Pharmacol Toxicol* 2003;93:290-6.
385. Bruck R, Aeed H, Avni Y, et al. Melatonin inhibits nuclear factor kappa B activation and oxidative stress and protects against thioacetamide induced liver damage in rats. *J Hepatol* 2004;40:86-93.
386. Sener G, Kacmaz A, User Y, Ozkan S, Tilki M, Yegen BC. Melatonin ameliorates oxidative organ damage induced by acute intra-abdominal compartment syndrome in rats. *J Pineal Res* 2003;35:163-8.
387. Chen KB, Lin AM, Chiu TH. Oxidative injury to the locus coeruleus of rat brain: neuroprotection by melatonin. *J Pineal Res* 2003;35:109-17.
388. Mayo JC, Tan DX, Sainz RM, Lopez-Burillo S, Reiter RJ. Oxidative damage to catalase induced by peroxyl radicals: functional protection by melatonin and other antioxidants. *Free Radic Res* 2003;37:543-53.
389. Tunez I, Munoz Mdel C, Feijoo M, et al. Protective melatonin effect on oxidative stress induced by okadaic acid into rat brain. *J Pineal Res* 2003;34:265-8.
390. Mayo JC, Tan DX, Sainz RM, Natarajan M, Lopez-Burillo S, Reiter RJ. Protection against oxidative protein damage induced by metal-catalyzed reaction or alkylperoxyl radicals: comparative effects of melatonin and other antioxidants. *Biochim Biophys Acta* 2003;1620:139-50.
391. Abdel-Wahab MH, Arafa HM, El-Mahdy MA, Abdel-Naim AB. Potential protective effect of melatonin against dibromoacetonitrile-induced oxidative stress in mouse stomach. *Pharmacol Res* 2002;46:287-93.
392. Shen YX, Xu SY, Wei W, et al. The protective effects of melatonin from oxidative damage induced by amyloid beta-peptide 25-35 in middle-aged rats. *J Pineal Res* 2002;32:85-9.
393. Lankoff A, Banasik A, Nowak M. Protective effect of melatonin against nodularin-induced oxidative stress. *Arch Toxicol* 2002;76:158-65.



394. Tomas-Zapico C, Martinez-Fraga J, Rodriguez-Colunga MJ, Tolivia D, Hardeland R, Coto-Montes A. Melatonin protects against delta-aminolevulinic acid-induced oxidative damage in male Syrian hamster Harderian glands. *Int J Biochem Cell Biol* 2002;34:544-53.
395. Karbownik M, Reiter RJ. Melatonin protects against oxidative stress caused by delta-aminolevulinic acid: implications for cancer reduction. *Cancer Invest* 2002;20:276-86.
396. Herrera F, Sainz RM, Mayo JC, Martin V, Antolin I, Rodriguez C. Glutamate induces oxidative stress not mediated by glutamate receptors or cystine transporters: protective effect of melatonin and other antioxidants. *J Pineal Res* 2001;31:356-62.
397. Othman AI, El-Missiry MA, Amer MA. The protective action of melatonin on indomethacin-induced gastric and testicular oxidative stress in rats. *Redox Rep* 2001;6:173-7.
398. Bagchi M, Balmoori J, Ye X, Bagchi D, Ray SD, Stohs SJ. Protective effect of melatonin on naphthalene-induced oxidative stress and DNA damage in cultured macrophage J774A.1 cells. *Mol Cell Biochem* 2001;221:49-55.
399. Dabbeni-Sala F, Floreani M, Franceschini D, Skaper SD, Giusti P. Kainic acid induces selective mitochondrial oxidative phosphorylation enzyme dysfunction in cerebellar granule neurons: protective effects of melatonin and GSH ethyl ester. *FASEB J* 2001;15:1786-8.
400. El-Sokkary GH. Melatonin protects against oxidative stress induced by the kidney carcinogen KBrO(3). *Neuro Endocrinol Lett* 2000;21:461-8.
401. Behan WM, McDonald M, Darlington LG, Stone TW. Oxidative stress as a mechanism for quinolinic acid-induced hippocampal damage: protection by melatonin and deprenyl. *Br J Pharmacol* 1999;128:1754-60.
402. Cadenas S, Barja G. Resveratrol, melatonin, vitamin E, and PBN protect against renal oxidative DNA damage induced by the kidney carcinogen KBrO3. *Free Radic Biol Med* 1999;26:1531-7.
403. Montilla PL, Tunez IF, Munoz de Agueda C, Gascon FL, Soria JV. Protective role of melatonin and retinol palmitate in oxidative stress and hyperlipidemic nephropathy induced by adriamycin in rats. *J Pineal Res* 1998;25:86-93.
404. Princ FG, Maxit AG, Cardalda C, Batlle A, Juknat AA. In vivo protection by melatonin against delta-aminolevulinic acid-induced oxidative damage and its antioxidant effect on the activity of haem enzymes. *J Pineal Res* 1998;24:1-8.
405. Popov SS, Shulgin KK, Popova TN, Pashkov AN, Agarkov AA, de Carvalho MA. Effects of Melatonin-Aided Therapy on the Glutathione Antioxidant System Activity and Liver Protection. *J Biochem Mol Toxicol* 2015.
406. Spadoni G, Diamantini G, Bedini A, et al. Synthesis, antioxidant activity and structure-activity relationships for a new series of 2-(N-acylaminoethyl)indoles with melatonin-like cytoprotective activity. *J Pineal Res* 2006;40:259-69.
407. Soyoz M, Ozelik N, Kilinc I, Altuntas I. The effects of ochratoxin A on lipid peroxidation and antioxidant enzymes: a protective role of melatonin. *Cell Biol Toxicol* 2004;20:213-9.
408. Mor M, Spadoni G, Diamantini G, et al. Antioxidant and cytoprotective activity of indole derivatives related to melatonin. *Adv Exp Med Biol* 2003;527:567-75.
409. Martin V, Sainz RM, Antolin I, Mayo JC, Herrera F, Rodriguez C. Several antioxidant pathways are involved in astrocyte protection by melatonin. *J Pineal Res* 2002;33:204-12.
410. Ortega-Gutierrez S, Garcia JJ, Martinez-Ballarín E, et al. Melatonin improves deferoxamine antioxidant activity in protecting against lipid peroxidation caused by hydrogen peroxide in rat brain homogenates. *Neurosci Lett* 2002;323:55-9.
411. Hara M, Yoshida M, Nishijima H, et al. Melatonin, a pineal secretory product with antioxidant properties, protects against cisplatin-induced nephrotoxicity in rats. *J Pineal Res* 2001;30:129-38.
412. Shifow AA, Kumar KV, Naidu MU, Ratnakar KS. Melatonin, a pineal hormone with antioxidant property, protects against gentamicin-induced nephrotoxicity in rats. *Nephron* 2000;85:167-74.
413. Morishima I, Okumura K, Matsui H, et al. Zinc accumulation in adriamycin-induced cardiomyopathy in rats: effects of melatonin, a cardioprotective antioxidant. *J Pineal Res* 1999;26:204-10.
414. Morishima I, Matsui H, Mukawa H, et al. Melatonin, a pineal hormone with antioxidant property, protects against adriamycin cardiomyopathy in rats. *Life Sci* 1998;63:511-21.
415. Sutken E, Aral E, Ozdemir F, Uslu S, Alatas O, Colak O. Protective role of melatonin and coenzyme Q10 in ochratoxin A toxicity in rat liver and kidney. *Int J Toxicol* 2007;26:81-7.
416. Gazi S, Altun A, Erdogan O. Contrast-induced nephropathy: preventive and protective effects of melatonin. *J Pineal Res* 2006;41:53-7.
417. Cruz A, Padillo FJ, Granados J, et al. Effect of melatonin on cholestatic oxidative stress under constant light exposure. *Cell Biochem Funct* 2003;21:377-80.

418. Dogan MS, YavaÅŸ MC, GÃ¼nay A, et al. The protective effect of melatonin and Ganoderma lucidum against the negative effects of extremely low frequency electric and magnetic fields on pulp structure in rat teeth. *Biotechnology & Biotechnological Equipment* 2017;31:979-88.
419. Reiter RJ. Static and extremely low frequency electromagnetic field exposure: reported effects on the circadian production of melatonin. *J Cell Biochem* 1993;51:394-403.
420. Lan CT, Hsu JC, Ling EA. Influence of sleep deprivation coupled with administration of melatonin on the ultrastructure of rat pineal gland. *Brain Res* 2001;910:1-11.
421. Razygraev AV. [Pineal gland glutathione peroxidase activity in rats and its age-associated change]. *Adv Gerontol* 2010;23:392-5.
422. Lin'kova NS, Poliakova VO, Kvetnoi IM, Trofimov AV, Sevost'ianova NN. [Characteristics of the pineal gland and thymus relationship in aging]. *Adv Gerontol* 2011;24:38-42.
423. Polyakova VO, Linkova NS, Kvetnoy IM, Khavinson V. Functional unity of the thymus and pineal gland and study of the mechanisms of aging. *Bull Exp Biol Med* 2011;151:627-30.
424. Gruber MJ, Palmquist E, Nordin S. Characteristics of perceived electromagnetic hypersensitivity in the general population. *Scand J Psychol* 2018.
425. Chen YP. Microwave treatment of eight seconds protects cells of *Isatis indigotica* from enhanced UV-B radiation lesions. *Photochem Photobiol* 2006;82:503-7.
426. Irmak MK, Fadillioglu E, Gulec M, Erdogan H, Yagmurca M, Akyol O. Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits. *Cell Biochem Funct* 2002;20:279-83.
427. Martinez-Samano J, Torres-Duran PV, Juarez-Oropeza MA, Elias-Vinas D, Verdugo-Diaz L. Effects of acute electromagnetic field exposure and movement restraint on antioxidant system in liver, heart, kidney and plasma of Wistar rats: a preliminary report. *Int J Radiat Biol* 2010;86:1088-94.
428. Duan Y, Wang Z, Zhang H, et al. The preventive effect of lotus seedpod procyanidins on cognitive impairment and oxidative damage induced by extremely low frequency electromagnetic field exposure. *Food Funct* 2013;4:1252-62.
429. Yurekli AI, Ozkan M, Kalkan T, et al. GSM base station electromagnetic radiation and oxidative stress in rats. *Electromagn Biol Med* 2006;25:177-88.
430. Esmekaya MA, Ozer C, Seyhan N. 900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues. *Gen Physiol Biophys* 2011;30:84-9.
431. Akpinar D, Ozturk N, Ozen S, Agar A, Yargicoglu P. The effect of different strengths of extremely low-frequency electric fields on antioxidant status, lipid peroxidation, and visual evoked potentials. *Electromagn Biol Med* 2012;31:436-48.
432. Bahreymi Toossi MH, Sadeghnia HR, Mohammad Mahdizadeh Feyzabadi M, et al. Exposure to mobile phone (900-1800 MHz) during pregnancy: tissue oxidative stress after childbirth. *J Matern Fetal Neonatal Med* 2017;Apr 23 {Epub ahead of print}:1-6.
433. Megha K, Deshmukh PS, Banerjee BD, Tripathi AK, Ahmed R, Abegaonkar MP. Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain. *NeuroToxicology* 2015;51:158-65.
434. Ceyhan AM, Akkaya VB, Gulecol SC, Ceyhan BM, Ozguner F, Chen W. Protective effects of beta-glucan against oxidative injury induced by 2.45-GHz electromagnetic radiation in the skin tissue of rats. *Arch Dermatol Res* 2012;304:521-7.
435. Ozguner F, Oktem F, Ayata A, Koyu A, Yilmaz HR. A novel antioxidant agent caffeic acid phenethyl ester prevents long-term mobile phone exposure-induced renal impairment in rat. Prognostic value of malondialdehyde, N-acetyl-beta-D-glucosaminidase and nitric oxide determination. *Mol Cell Biochem* 2005;277:73-80.
436. Ozguner F, Altinbas A, Ozaydin M, et al. Mobile phone-induced myocardial oxidative stress: protection by a novel antioxidant agent caffeic acid phenethyl ester. *Toxicol Ind Health* 2005;21:223-30.
437. Halliday GM. Inflammation, gene mutation and photoimmunosuppression in response to UVR-induced oxidative damage contributes to photocarcinogenesis. *Mutat Res* 2005;571:107-20.
438. McRee DI. Soviet and Eastern European Research on Biological Effects of Microwave Radiation. *Proceeding IEEE* 1980;68:84-91.
439. Calderon-Margalit R, Adler B, Abramson JH, Gofin J, Kark JD. Butyrylcholinesterase activity, cardiovascular risk factors, and mortality in middle-aged and elderly men and women in Jerusalem. *Clin Chem* 2006;52:845-52.
440. Benderitter M, Vincent-Genod L, Pouget JP, Voisin P. The cell membrane as a biosensor of oxidative stress induced by radiation exposure: a multiparameter investigation. *Radiat Res* 2003;159:471-83.
441. Shonai T, Adachi M, Sakata K, et al. MEK/ERK pathway protects ionizing radiation-induced loss of mitochondrial membrane potential and cell death in lymphocytic leukemia cells. *Cell Death Differ* 2002;9:963-71.

442. Thomas SM, Gebicki JM, Dean RT. Radical initiated alpha-tocopherol depletion and lipid peroxidation in mitochondrial membranes. *Biochim Biophys Acta* 1989;1002:189-97.
443. Wang C, Cong J, Xian H, Cao X, Sun C, Wu K. [The effects of electromagnetic pulse on fluidity and lipid peroxidation of mitochondrial membrane]. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* 2002;20:266-8.
444. Vayssier-Taussat M, Kreps SE, Adrie C, Dall'Ava J, Christiani D, Polla BS. Mitochondrial membrane potential: a novel biomarker of oxidative environmental stress. *Environ Health Perspect* 2002;110:301-5.
445. Barichello T, Lemos JC, Generoso JS, et al. Oxidative Stress, Cytokine/Chemokine and Disruption of Blood-Brain Barrier in Neonate Rats After Meningitis by *Streptococcus agalactiae*. *Neurochem Res* 2011.
446. Gasche Y, Copin JC, Sugawara T, Fujimura M, Chan PH. Matrix metalloproteinase inhibition prevents oxidative stress-associated blood-brain barrier disruption after transient focal cerebral ischemia. *J Cereb Blood Flow Metab* 2001;21:1393-400.
447. Haorah J, Knipe B, Leibhart J, Ghorpade A, Persidsky Y. Alcohol-induced oxidative stress in brain endothelial cells causes blood-brain barrier dysfunction. *J Leukoc Biol* 2005;78:1223-32.
448. Zehendner CM, Librizzi L, Hedrich J, et al. Moderate hypoxia followed by reoxygenation results in blood-brain barrier breakdown via oxidative stress-dependent tight-junction protein disruption. *PLoS One* 2013;8:e82823.
449. Freeman LR, Keller JN. Oxidative stress and cerebral endothelial cells: regulation of the blood-brain-barrier and antioxidant based interventions. *Biochim Biophys Acta* 2012;1822:822-9.
450. Cui J, Zhong R, Chu E, et al. Correlation between oxidative stress and L-type calcium channel expression in the ventricular myocardia of selenium-deficient mice. *J Int Med Res* 2012;40:1677-87.
451. Pall ML. Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action. *Rev Environ Health* 2015;30:99-116.
452. Nishiyama A, Nakano D, Hitomi H. [Calcium antagonists: current and future applications based on new evidence. Effects of calcium channel blockers on oxidative stress]. *Clin Calcium* 2010;20:38-44.
453. Ferrer I. Altered mitochondria, energy metabolism, voltage-dependent anion channel, and lipid rafts converge to exhaust neurons in Alzheimer's disease. *J Bioenerg Biomembr* 2009;41:425-31.
454. Aitken RJ, Bennetts LE, Sawyer D, Wiklendt AM, King BV. Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline. *Int J Androl* 2005;28:171-9.
455. Houston BJ, Nixon B, King BV, De Iuliis GN, Aitken RJ. The effects of radiofrequency electromagnetic radiation on sperm function. *Reproduction* 2016;152:R263-R76.
456. Mancuso M, Coppede F, Migliore L, Siciliano G, Murri L. Mitochondrial dysfunction, oxidative stress and neurodegeneration. *J Alzheimers Dis* 2006;10:59-73.
457. Tan DX, Manchester LC, Qin L, Reiter RJ. Melatonin: A Mitochondrial Targeting Molecule Involving Mitochondrial Protection and Dynamics. *Int J Mol Sci* 2016;17.
458. Engin AB, Sepici-Dincel A, Gonul, II, Engin A. Oxidative stress-induced endothelial cell damage in thyroidectomized rat. *Exp Toxicol Pathol* 2012;64:481-5.
459. Indik JH, Goldman S, Gaballa MA. Oxidative stress contributes to vascular endothelial dysfunction in heart failure. *Am J Physiol Heart Circ Physiol* 2001;281:H1767-70.
460. Jarasuniene D, Simaitis A. [Oxidative stress and endothelial dysfunction]. *Medicina (Kaunas)* 2003;39:1151-7.
461. Loscalzo J. Oxidative stress in endothelial cell dysfunction and thrombosis. *Pathophysiol Haemost Thromb* 2002;32:359-60.
462. Ahsan H, Ali A, Ali R. Oxygen free radicals and systemic autoimmunity. *Clin Exp Immunol* 2003;131:398-404.
463. Fiorini A, Koudriavtseva T, Bucaj E, et al. Involvement of oxidative stress in occurrence of relapses in multiple sclerosis: the spectrum of oxidatively modified serum proteins detected by proteomics and redox proteomics analysis. *PLoS One* 2013;8:e65184.
464. Gilgun-Sherki Y, Melamed E, Offen D. The role of oxidative stress in the pathogenesis of multiple sclerosis: the need for effective antioxidant therapy. *Journal of neurology* 2004;251:261-8.
465. Kirkham PA, Caramori G, Casolari P, et al. Oxidative stress-induced antibodies to carbonyl-modified protein correlate with severity of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2011;184:796-802.
466. Ryan BJ, Nissim A, Winyard PG. Oxidative post-translational modifications and their involvement in the pathogenesis of autoimmune diseases. *Redox Biol* 2014;2:715-24.
467. Aoki M, Nata T, Morishita R, et al. Endothelial apoptosis induced by oxidative stress through activation of NF-kappaB: antiapoptotic effect of antioxidant agents on endothelial cells. *Hypertension* 2001;38:48-55.

468. Bresgen N, Karlhuber G, Krizbai I, Bauer H, Bauer HC, Eckl PM. Oxidative stress in cultured cerebral endothelial cells induces chromosomal aberrations, micronuclei, and apoptosis. *J Neurosci Res* 2003;72:327-33.
469. Espino J, Bejarano I, Ortiz A, et al. Melatonin as a potential tool against oxidative damage and apoptosis in ejaculated human spermatozoa. *Fertil Steril* 2010;94:1915-7.
470. Filomeni G, Cardaci S, Da Costa Ferreira AM, Rotilio G, Ciriolo MR. Metabolic oxidative stress elicited by the copper(II) complex [Cu(IIaepy)<sub>2</sub>] triggers apoptosis in SH-SY5Y cells through the induction of the AMP-activated protein kinase/p38MAPK/p53 signalling axis: evidence for a combined use with 3-bromopyruvate in neuroblastoma treatment. *Biochem J* 2011;437:443-53.
471. France-Lanord V, Brugg B, Michel PP, Agid Y, Ruberg M. Mitochondrial free radical signal in ceramide-dependent apoptosis: a putative mechanism for neuronal death in Parkinson's disease. *J Neurochem* 1997;69:1612-21.
472. Li J, Meng Z, Zhang G, et al. N-acetylcysteine relieves oxidative stress and protects hippocampus of rat from radiation-induced apoptosis by inhibiting caspase-3. *Biomed Pharmacother* 2015;70:1-6.
473. Li W, Lidebjer C, Yuan XM, et al. NK cell apoptosis in coronary artery disease: relation to oxidative stress. *Atherosclerosis* 2008;199:65-72.
474. Salido GM, Rosado JA. Apoptosis: Involvement of Oxidative Stress and Intracellular Ca<sup>2+</sup> Homeostasis. Springer 2009.
475. Yalcinkaya S, Unlucerci Y, Giris M, Olgac V, Dogru-Abbasoglu S, Uysal M. Oxidative and nitrosative stress and apoptosis in the liver of rats fed on high methionine diet: protective effect of taurine. *Nutrition* 2009;25:436-44.
476. Zhang Y, Zhang X, Rabbani ZN, Jackson IL, Vujaskovic Z. Oxidative stress mediates radiation lung injury by inducing apoptosis. *Int J Radiat Oncol Biol Phys* 2012;83:740-8.
477. Reutelingsperger CP, van Heerde WL. Annexin V, the regulator of phosphatidylserine-catalyzed inflammation and coagulation during apoptosis. *Cell Mol Life Sci* 1997;53:527-32.
478. Stone R. Stressful conditions, not 'sonic weapon,' sickened U.S diplomats, Cuba asserts. *Science* 2017;Dec 5: <http://www.sciencemag.org/news/2017/12/stressful-conditions-not-sonic-weapon-sickened-us-diplomats-cuba-panel-asserts>.
479. Maisch D. Smart meter health concerns: just a nocebo effect, or an emerging public health nightmare? *Australasian Coll Nutr Environ Med J* 2012;31:15-9.
480. Tressider A. Electrosensitivity – an Environmental illness, an Authentic Diagnosis, not a Delusional Disorder. To my Medical Colleagues, GPs, Psychiatrists, Neurologists and Others (letter) 2017.
481. Crews F. Freud. The Making of an Illusion. New York: Henry Holt and Company; 2017.
482. Golomb BA. Psychogenic Illness. In: John Brockman, ed. This Idea Must Die: Scientific Theories That are Blocking Progress. New York: Harper Perennial; 2015:511-4.
483. Shimoda K, Akahane K, Nomura M, Kato M. LD50 value, phototoxicity and convulsion induction test of the new quinolone antibacterial agent (S)-10-[(S)-(8-amino-6-azaspiro[3,4]octan-6-yl)]-9-fluoro-2, 3-dihydro-3-methyl-7-oxo-7H-pyrido[1,2,3-de][1,4]benzoxazine-6-carboxylic acid hemihydrate in laboratory animals. *Arzneimittelforschung* 1996;46:625-8.
484. Baiomy AA, Attia HF, Soliman MM, Makrum O. Protective effect of ginger and zinc chloride mixture on the liver and kidney alterations induced by malathion toxicity. *Int J Immunopathol Pharmacol* 2015;28:122-8.
485. Shafiee H, Mohammadi H, Rezayat SM, et al. Prevention of malathion-induced depletion of cardiac cells mitochondrial energy and free radical damage by a magnetic magnesium-carrying nanoparticle. *Toxicol Mech Methods* 2010;20:538-43.
486. Jagetia GC, Venkatesh P, Baliga MS. Fruit extract of Aegle marmelos protects mice against radiation-induced lethality. *Integr Cancer Ther* 2004;3:323-32.
487. Pal S, Chatterjee AK. Possible beneficial effects of melatonin supplementation on arsenic-induced oxidative stress in Wistar rats. *Drug Chem Toxicol* 2006;29:423-33.
488. Jagetia GC, Baliga MS. Treatment of mice with a herbal preparation (mentat) protects against radiation-induced mortality. *Phytother Res* 2003;17:876-81.
489. Golomb BA. Effect Modification. In: Brockman J, ed. This Idea is Brilliant: Lost, Overlooked, and Underappreciated Scientific Concepts Everyone Should Know. New York: Harper Perennial; 2018:440-3.
490. Bowry VW, Mohr D, Cleary J, Stocker R. Prevention of tocopherol-mediated peroxidation in ubiquinol-10-free human low density lipoprotein. *J Biol Chem* 1995;270:5756-63.
491. Hu ML, Chen YK, Lin YF. The antioxidant and prooxidant activity of some B vitamins and vitamin-like compounds. *Chem Biol Interact* 1995;97:63-73.



492. Kontush A, Finckh B, Karten B, Kohlschutter A, Beisiegel U. Antioxidant and prooxidant activity of alpha-tocopherol in human plasma and low density lipoprotein. *J Lipid Res* 1996;37:1436-48.
493. Palozza P, Luberto C, Calviello G, Ricci P, Bartoli GM. Antioxidant and prooxidant role of beta-carotene in murine normal and tumor thymocytes: effects of oxygen partial pressure. *Free Radic Biol Med* 1997;22:1065-73.
494. Gerster H. High-dose vitamin C: a risk for persons with high iron stores? *Int J Vitam Nutr Res* 1999;69:67-82.
495. Young AJ, Lowe GM. Antioxidant and prooxidant properties of carotenoids. *Arch Biochem Biophys* 2001;385:20-7.
496. Azam S, Hadi N, Khan NU, Hadi SM. Antioxidant and prooxidant properties of caffeine, theobromine and xanthine. *Med Sci Monit* 2003;9:BR325-30.
497. Hiramoto K, Ohkawa T, Oikawa N, Kikugawa K. Is nitric oxide (NO) an antioxidant or a prooxidant for lipid peroxidation? *Chem Pharm Bull (Tokyo)* 2003;51:1046-50.
498. Lee JC, Kim J, Park JK, Chung GH, Jang YS. The antioxidant, rather than prooxidant, activities of quercetin on normal cells: quercetin protects mouse thymocytes from glucose oxidase-mediated apoptosis. *Exp Cell Res* 2003;291:386-97.
499. Sinzinger H, Lupattelli G, Chehne F, Oguogho A, Furberg CD. Isoprostane 8-epi-PGF2alpha is frequently increased in patients with muscle pain and/or CK-elevation after HMG-Co-enzyme-A-reductase inhibitor therapy. *J Clin Pharm Ther* 2001;26:303-10.
500. Sinzinger H, Lupattelli G, Chehne F. Increased lipid peroxidation in a patient with CK-elevation and muscle pain during statin therapy. *Atherosclerosis* 2000;153:255-6.
501. Rösli M, Möser M, Baldinini Y, Meier M, Braun-Fahrlander C. Symptoms of ill health ascribed to electromagnetic field exposure - A questionnaire survey. *Int J Hyg Environ Health* 2004;207:141-50.
502. Simone G, Tamba M, Quintiliani M. Role of glutathione in affecting the radiosensitivity of molecular and cellular systems. *Radiat Environ Biophys* 1983;22:215-23.
503. Jensen GL, Meister A. Radioprotection of human lymphoid cells by exogenously supplied glutathione is mediated by gamma-glutamyl transpeptidase. *Proc Natl Acad Sci U S A* 1983;80:4714-7.
504. Koch CJ, Skov KA. Enhanced radiation-sensitivity by preincubation with nitroimidazoles: effect of glutathione depletion. *Int J Radiat Oncol Biol Phys* 1994;29:345-9.
505. Vallis KA. Glutathione deficiency and radiosensitivity in AIDS patients. *Lancet* 1991;337:918-9.
506. Vos O, van der Schans GP, Roos-Verheij WS. Reduction of intracellular glutathione content and radiosensitivity. *Int J Radiat Biol Relat Stud Phys Chem Med* 1986;50:155-65.
507. Hodgkiss RJ, Stratford MR, Watfa RR. The effect of alpha-tocopherol and alpha-tocopheryl quinone on the radiosensitivity of thiol-depleted mammalian cells. *Int J Radiat Oncol Biol Phys* 1989;16:1297-300.
508. Yi X, Ding L, Jin Y, Ni C, Wang W. The toxic effects, GSH depletion and radiosensitivity by BSO on retinoblastoma. *Int J Radiat Oncol Biol Phys* 1994;29:393-6.
509. Holt JA. Some characteristics of the glutathione cycle revealed by ionising and non-ionising electromagnetic radiation. *Med Hypotheses* 1995;45:345-68.
510. Park MT, Kim MJ, Kang YH, et al. Phytosphingosine in combination with ionizing radiation enhances apoptotic cell death in radiation-resistant cancer cells through ROS-dependent and -independent AIF release. *Blood* 2005;105:1724-33.
511. Mitchell JB, Russo A. The role of glutathione in radiation and drug induced cytotoxicity. *Br J Cancer Suppl* 1987;8:96-104.
512. Shea CR, Wimberly J, Hasan T. Mitochondrial phototoxicity sensitized by doxycycline in cultured human carcinoma cells. *J Invest Dermatol* 1986;87:338-42.
513. Oliveira HS, Goncalo M, Figueiredo AC. Photosensitivity to lomefloxacin. A clinical and photobiological study. *Photodermatol Photoimmunol Photomed* 2000;16:116-20.
514. Snyder RD, Cooper CS. Photogenotoxicity of fluoroquinolones in Chinese hamster V79 cells: dependency on active topoisomerase II. *Photochem Photobiol* 1999;69:288-93.
515. Bilski P, Martinez LJ, Koker EB, Chignell CF. Photosensitization by norfloxacin is a function of pH. *Photochem Photobiol* 1996;64:496-500.
516. Man I, Murphy J, Ferguson J. Fluoroquinolone phototoxicity: a comparison of moxifloxacin and lomefloxacin in normal volunteers. *J Antimicrob Chemother* 1999;43 Suppl B:77-82.
517. Trisciuglio D, Krasnowska E, Maggi A, Pozzi R, Parasassi T, Saporita O. Phototoxic effect of fluoroquinolones on two human cell lines. *Toxicol In Vitro* 2002;16:449-56.
518. Boccumini LE, Fowler CL, Campbell TA, Puertolas LF, Kaidbey KH. Photoreaction potential of orally administered levofloxacin in healthy subjects. *Ann Pharmacother* 2000;34:453-8.

519. Nedorost ST, Dijkstra JW, Handel DW. Drug-induced photosensitivity reaction. *Arch Dermatol* 1989;125:433-4.
520. Granowitz EV. Photosensitivity rash in a patient being treated with ciprofloxacin. *J Infect Dis* 1989;160:910-1.
521. Wagai N, Yamaguchi F, Sekiguchi M, Tawara K. Phototoxic potential of quinolone antibacterial agents in Balb/c mice. *Toxicol Lett* 1990;54:299-308.
522. Wagai N, Tawara K. Important role of oxygen metabolites in quinolone antibacterial agent-induced cutaneous phototoxicity in mice. *Arch Toxicol* 1991;65:495-9.
523. Ferguson J, Johnson BE. Clinical and laboratory studies of the photosensitizing potential of norfloxacin, a 4-quinolone broad-spectrum antibiotic. *Br J Dermatol* 1993;128:285-95.
524. Scheife RT, Cramer WR, Decker EL. Photosensitizing potential of ofloxacin. *Int J Dermatol* 1993;32:413-6.
525. Fujita H, Matsuo I. In vitro phototoxic activities of new quinolone antibacterial agents: lipid peroxidative potentials. *Photodermatol Photoimmunol Photomed* 1994;10:202-5.
526. Burdge DR, Nakielna EM, Rabin HR. Photosensitivity associated with ciprofloxacin use in adult patients with cystic fibrosis. *Antimicrob Agents Chemother* 1995;39:793.
527. Kimura M, Kawada A, Kobayashi T, Hiruma M, Ishibashi A. Photosensitivity induced by fleroxacin. *Clin Exp Dermatol* 1996;21:46-7.
528. Chetelat AA, Albertini S, Gocke E. The photomutagenicity of fluoroquinolones in tests for gene mutation, chromosomal aberration, gene conversion and DNA breakage (Comet assay). *Mutagenesis* 1996;11:497-504.
529. Agrawal N, Ray RS, Farooq M, Pant AB, Hans RK. Photosensitizing potential of ciprofloxacin at ambient level of UV radiation. *Photochem Photobiol* 2007;83:1226-36.
530. Ferguson J, Johnson BE. Ciprofloxacin-induced photosensitivity: in vitro and in vivo studies. *Br J Dermatol* 1990;123:9-20.
531. Akter U, Niwa M, Nose T, et al. Effects of several agents on UVB- and UVA plus systemic fluoroquinolone-induced erythema of guinea pig skin evaluated by reflectance colorimetry. *Free Radic Biol Med* 1998;24:1113-9.
532. Sailer E, Kamarachev J, Boehler A, et al. Persistent photodamage following drug photosensitization in a lung-transplant recipient. *Photodermatol Photoimmunol Photomed* 2011;27:213-5.
533. Prithivirajsingh S, Story MD, Bergh SA, et al. Accumulation of the common mitochondrial DNA deletion induced by ionizing radiation. *FEBS Lett* 2004;571:227-32.
534. Jaffe A, Bush A. If you can't stand the rash, get out of the kitchen: an unusual adverse reaction to ciprofloxacin. *Pediatr Pulmonol* 1999;28:449-50.
535. Thual N, Penven K, Chevallier JM, Dompormartin A, Leroy D. [Fluvastatin-induced dermatomyositis]. *Ann Dermatol Venereol* 2005;132:996-9.
536. Morimoto K, Kawada A, Hiruma M, Ishibashi A, Banba H. Photosensitivity to simvastatin with an unusual response to photopatch and photo tests. *Contact Dermatitis* 1995;33:274.
537. Dawson GA, Brown SI, Tellefsen L. A drug-related phototoxic reaction and its possible relationship to a radiation-induced skin reaction. *Oncologist* 2009;14:303-6.
538. Murphy GM. Diseases associated with photosensitivity. *J Photochem Photobiol B* 2001;64:93-8.
539. Martin JA, Taylor C, Trehan M, Baron ED, Anstey AV. Phototesting in patients with Smith-Lemli-Opitz syndrome confirms sensitivity to UV-A. *Arch Dermatol* 2006;142:647-8.
540. Anstey A. School in photodermatology: Smith-Lemli-Opitz syndrome. *Photodermatol Photoimmunol Photomed* 2006;22:200-4.
541. Anstey A. Photomedicine: lessons from the Smith-Lemli-Opitz syndrome. *J Photochem Photobiol B* 2001;62:123-7.
542. [A new congenital photosensitivity syndrome. Smith-Lemli-Opitz syndrome]. *Hautarzt* 1999;50:159.
543. Eapen BR. Photosensitivity in Smith-Lemli-Opitz syndrome: a flux balance analysis of altered metabolism. *Bioinformation* 2007;2:78-82.
544. Chignell CF, Kukienczak BM, Sik RH, Bilski PJ, He YY. Ultraviolet A sensitivity in Smith-Lemli-Opitz syndrome: Possible involvement of cholesta-5,7,9(11)-trien-3 beta-ol. *Free Radic Biol Med* 2006;41:339-46.
545. Charman CR, Ryan A, Tyrrell RM, et al. Photosensitivity associated with the Smith-Lemli-Opitz syndrome. *Br J Dermatol* 1998;138:885-8.
546. Azurdia RM, Anstey AV, Rhodes LE. Cholesterol supplementation objectively reduces photosensitivity in the Smith-Lemli-Opitz syndrome. *Br J Dermatol* 2001;144:143-5.
547. Anstey AV, Taylor CR. Photosensitivity in the Smith-Lemli-Opitz syndrome: the US experience of a new congenital photosensitivity syndrome. *J Am Acad Dermatol* 1999;41:121-3.
548. Anstey AV, Ryan A, Rhodes LE, et al. Characterization of photosensitivity in the Smith-Lemli-Opitz syndrome: a new congenital photosensitivity syndrome. *Br J Dermatol* 1999;141:406-14.



549. Anstey AV, Azurdia RM, Rhodes LE, Pearse AD, Bowden PE. Photosensitive Smith-Lemli-Opitz syndrome is not caused by a single gene mutation: analysis of the gene encoding 7-dehydrocholesterol reductase in five U.K. families. *Br J Dermatol* 2005;153:774-9.
550. Anstey AV. Photosensitivity in the Smith-Lemli-Opitz syndrome. *Photodermatol Photoimmunol Photomed* 1999;15:217-8.
551. Jain S, Agarwal J, Laskar S, Gupta T, Shrivastava S. Radiation recall dermatitis with gatifloxacin: a review of literature. *J Med Imaging Radiat Oncol* 2008;52:191-3.
552. Cho S, Breedlove JJ, Gunning ST. Radiation recall reaction induced by levofloxacin. *J Drugs Dermatol* 2008;7:64-7.
553. Wernicke AG, Swistel AJ, Parashar B, Myskowski PL. Levofloxacin-induced radiation recall dermatitis: a case report and a review of the literature. *Clin Breast Cancer* 2010;10:404-6.
554. Rubin GJ, Das Munshi J, Wessely S. Electromagnetic hypersensitivity: a systematic review of provocation studies. *Psychosom Med* 2005;67:224-32.
555. Abraham P, Kolli VK, Rabi S. Melatonin attenuates methotrexate-induced oxidative stress and renal damage in rats. *Cell Biochem Funct* 2010;28:426-33.
556. Brea-Calvo G, Rodriguez-Hernandez A, Fernandez-Ayala DJ, Navas P, Sanchez-Alcazar JA. Chemotherapy induces an increase in coenzyme Q10 levels in cancer cell lines. *Free Radic Biol Med* 2006;40:1293-302.
557. Husain K, Whitworth C, Somani SM, Rybak LP. Carboplatin-induced oxidative stress in rat cochlea. *Hear Res* 2001;159:14-22.
558. Nicolson GL, Conklin KA. Reversing mitochondrial dysfunction, fatigue and the adverse effects of chemotherapy of metastatic disease by molecular replacement therapy. *Clin Exp Metastasis* 2008;25:161-9.
559. Bell A. *Poisoned: How a Crime-Busting Prosecutor Turned his Medical Mystery Into a Crusade for Environmental Victims*. New York: Skyhorse Publishing 2017.
560. Hayano J. Decreased magnitude of heart rate spectral components in coronary artery disease. *Circulation* 1990;81:1217-24.
561. Singer DH, Martin GJ, Magid N, et al. Low heart rate variability and sudden cardiac death. *J Electrocardiol* 1988;21:S46-55.
562. Bero L, Oostvogel F, Bacchetti P, Lee K. Factors Associated with Findings of Published Trials of Drug-Drug Comparisons: Why Some Statins Appear More Efficacious than Others. *PLoS Med* 2007;4:e184.
563. Heres S, Davis J, Maino K, Jetzinger E, Kissling W, Leucht S. Why Olanzapine Beats Risperidone, Risperidone Beats Quetiapine, and Quetiapine Beats Olanzapine: An Exploratory Analysis of Head-to-Head Comparison Studies of Second-Generation Antipsychotics. *Am J Psychiatry* 2006;163:185-94.
564. Friedman LS, Richter ED. Relationship between conflicts of interest and research results. *J Gen Intern Med* 2004;19:51-6.
565. Smith R. Medical journals are an extension of the marketing arm of pharmaceutical companies. *PLoS Med* 2005;2:e138.
566. Barnes DE, Bero LA. Why review articles on the health effects of passive smoking reach different conclusions. *Jama* 1998;279:1566-70.
567. Adlkofer F, Richter K. *Radiation Protection in Conflict with Science: Competence Initiative for the Protection of Humanity, Environment and Democracy e.V.; 2011.*
568. Harris G. Caustic Government Report Deals Blow to Diabetes Drug. *New York Times* 2010;July 9, 2010.
569. Did GSK trial data mask Paxil suicide risk? *The New Scientist* 2008; 8 February 2008 12.
570. Gupta SK, Mesharam MK, Krishnamurthy S. Electromagnetic radiation 2450 MHz exposure causes cognition deficit with mitochondrial dysfunction and activation of intrinsic pathway of apoptosis in rats. *Journal of Biosciences* 2018;43:263-76.
571. Belyaev IY, Sheheglov VS, Alipov ED, Ushakov VD. Nonthermal Effects of Extremely High-Frequency Microwaves on Chromatin Conformation in Cells in vitro—Dependence on Physical, Physiological, and Genetic Factors. *IEEE Transactions on Microwave Theory and Techniques* 2000;48:2172-9.
572. Chen P, Yang YQ, Tao HH, Yang HC. [Effects of electromagnetic fields of different frequencies on proliferation and DNA damage of gallbladder cancer cells]. *Nan Fang Yi Ke Da Xue Xue Bao* 2006;26:328-30.
573. Panagopoulos DJ, Johansson O, Carlo GL. Polarization: A key difference between man-made and natural electromagnetic fields, in regard to biological activity. *Scientific Reports* 2015;October:1-10.
574. Pall M. Wi-Fi is an important threat to health. *Environmental Research* 2018;164:405-16.
575. Lai H, Horita A, Chou CK, Guy AW. Low-level microwave irradiations affect central cholinergic activity in the rat. *J Neurochem* 1987;48:40-5.

576. Wood AW, Armstrong SM, Sait ML, Devine L, Martin MJ. Changes in human plasma melatonin profiles in response to 50 Hz magnetic field exposure. *J Pineal Res* 1998;25:116-27.
577. Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. *Bioelectromagnetics* 1995;16:207-10.
578. Robison JG, Pendleton AR, Monson KO, Murray BK, O'Neill KL. Decreased DNA repair rates and protection from heat induced apoptosis mediated by electromagnetic field exposure. *Bioelectromagnetics* 2002;23:106-12.
579. Ivancsits S, Diem E, Pilger A, Rudiger HW, Jahn O. Induction of DNA strand breaks by intermittent exposure to extremely-low-frequency electromagnetic fields in human diploid fibroblasts. *Mutat Res* 2002;519:1-13.
580. Laszlo A, Davidson T, Harvey A, et al. Alterations in heat-induced radiosensitization accompanied by nuclear structure alterations in Chinese hamster cells. *Int J Hyperthermia* 2006;22:43-60.
581. Bua L, Tibaldi E, Falcioni L, et al. Results of lifespan exposure to continuous and intermittent extremely low frequency electromagnetic fields (ELFEMF) administered alone to Sprague Dawley rats. *Environ Res* 2018;164:271-9.
582. Gajski G, Garaj-Vrhovac V. Radioprotective effects of honeybee venom (*Apis mellifera*) against 915-MHz microwave radiation-induced DNA damage in wistar rat lymphocytes: in vitro study. *Int J Toxicol* 2009;28:88-98.
583. Li WH, Li YZ, Song DD, et al. Calreticulin protects rat microvascular endothelial cells against microwave radiation-induced injury by attenuating endoplasmic reticulum stress. *Microcirculation* 2014;21:506-15.
584. Zhang X, Gao Y, Dong J, et al. The compound Chinese medicine "Kang Fu Ling" protects against high power microwave-induced myocardial injury. *PLoS One* 2014;9:e101532.
585. Zhang J, Peng RY, Ren JH, et al. [The protective effects of Aduola Fuzhenglin on the heart injury induced by microwave exposure in rats]. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* 2011;29:367-70.
586. Oral B, Guney M, Ozguner F, et al. Endometrial apoptosis induced by a 900-MHz mobile phone: preventive effects of vitamins E and C. *Adv Ther* 2006;23:957-73.
587. Koyu A, Ozguner F, Yilmaz H, Uz E, Cesur G, Ozcelik N. The protective effect of caffeic acid phenethyl ester (CAPE) on oxidative stress in rat liver exposed to the 900 MHz electromagnetic field. *Toxicol Ind Health* 2009;25:429-34.
588. Gurler HS, Bilgici B, Akar AK, Tomak L, Bedir A. Increased DNA oxidation (8-OHdG) and protein oxidation (AOPP) by low level electromagnetic field (2.45 GHz) in rat brain and protective effect of garlic. *Int J Radiat Biol* 2014;90:892-6.
589. Navarro A, Sanchez Del Pino MJ, Gomez C, Peralta JL, Boveris A. Behavioral dysfunction, brain oxidative stress, and impaired mitochondrial electron transfer in aging mice. *Am J Physiol Regul Integr Comp Physiol* 2002;282:R985-92.
590. Chiang H, Yao GD, Fang QS, Wang KQ, Lu DZ, Zhou YK. Health Effects of Environmental Electromagnetic Fields. *Journal of Bioelectricity* 1989;8:127-31.
591. Packer L, Fuchs J, eds. *Vitamin E in Health and Disease*. New York: Marcel Dekker, Inc; 1993.
592. Keaney JJ, Gaziano J, Xu A, et al. Low-dose alpha-tocopherol improves and high-dose alpha-tocopherol worsens endothelial vasodilator function in cholesterol-fed rabbits. *Journal of Clinical Investigation*, 1994;93:844-5.
593. Fuchs J, Packer L. *Vitamin E in dermatological therapy*. In: Packer L, Fuchs J, eds. *Vitamin E in Health and Disease*. New York: Marcel Dekker, Inc; 1993.
594. Miller ER, 3rd, Pastor-Barriuso R, Dalal D, Riemersma RA, Appel LJ, Guallar E. Meta-analysis: high-dosage vitamin E supplementation may increase all-cause mortality. *Ann Intern Med* 2005;142:37-46.
595. Golomb BA. Misinterpretation of trial evidence on statin adverse effects may harm patients. *Eur J Prev Cardiol* 2015;22:492-3.
596. Au WW, Cantelli-Forti G, Hrelia P, Legator MS. Cytogenetic assays in genotoxic studies: Somatic cell effects of benzene and germinal cell effects of dibromochloropropane. *Teratogen Carcinogen Mutagen* 1990;10.
597. Bilgici B, Akar A, Avci B, Tuncel OK. Effect of 900 MHz radiofrequency radiation on oxidative stress in rat brain and serum. *Electromagn Biol Med* 2013;32:20-9.
598. Burdelya LG, Gleiberman AS, Tshkov I, et al. Toll-like receptor 5 agonist protects mice from dermatitis and oral mucositis caused by local radiation: implications for head-and-neck cancer radiotherapy. *Int J Radiat Oncol Biol Phys* 2012;83:228-34.
599. Del Vecchio G, Giuliani A, Fernandez M, et al. Effect of radiofrequency electromagnetic field exposure on in vitro models of neurodegenerative disease. *Bioelectromagnetics* 2009;30:564-72.
600. Marie I, Noblet C. [Drug-associated tendon disorders: after fluoroquinolones ... here are statins!]. *Rev Med Interne* 2009;30:307-10.

601. Tendon disorders due to statins. *Prescrire Int* 2010;19:73.
602. Esenkaya I, Unay K. Tendon, tendon healing, hyperlipidemia and statins. *Muscles Ligaments Tendons J* 2011;1:169-71.
603. Hoffman KB, Kraus C, Dimbil M, Golomb BA. A Survey of the FDA's AERS Database Regarding Muscle and Tendon Adverse Events Linked to the Statin Drug Class. *PLoS One* 2012;7:e42866.
604. Gupta A, Guron N, Harris M, Bell R. Levofloxacin-induced rhabdomyolysis in a hemodialysis patient. *Hemodial Int* 2012;16:101-3.
605. Khammassi N, Abdelhedi H, Mohsen D, Ben Sassi M, Cherif O. [Rhabdomyolysis and acute renal failure secondary to ciprofloxacin therapy]. *Therapie* 2012;67:67-8.
606. Qian Q, Nasr SH, Akogyeram CO, Sethi S. Myoglobin-associated acute kidney injury in the setting of ciprofloxacin administration. *Am J Kidney Dis* 2012;59:462-6.
607. Sanjith S, Raodeo A, Clerk A, Pandit R, Karnad DR. Moxifloxacin-induced rhabdomyolysis. *Intensive Care Med* 2012;38:725.
608. George P, Das J, Pawar B, Badyal D. Gatifloxacin-induced rhabdomyolysis. *J Postgrad Med* 2008;54:233-4.
609. Hsiao SH, Chang CM, Tsao CJ, Lee YY, Hsu MY, Wu TJ. Acute rhabdomyolysis associated with ofloxacin/levofloxacin therapy. *Ann Pharmacother* 2005;39:146-9.
610. Korzets A, Gaftor U, Dicker D, Herman M, Ori Y. Levofloxacin and rhabdomyolysis in a renal transplant patient. *Nephrol Dial Transplant* 2006;21:3304-5.
611. Eisele S, Garbe E, Zeitz M, Schneider T, Somasundaram R. Ciprofloxacin-related acute severe myalgia necessitating emergency care treatment: a case report and review of the literature. *Int J Clin Pharmacol Ther* 2009;47:165-8.
612. Petitjeans F, Nadaud J, Perez JP, et al. A case of rhabdomyolysis with fatal outcome after a treatment with levofloxacin. *Eur J Clin Pharmacol* 2003;59:779-80.
613. Roberts M. Statin-fortified drinking water? *BBC News* 2004;August 1.
614. Brown D. Heart drug far surpasses expectations. *Washington Post* 2001 May 19, 2001;Sect. A1.
615. Dales MJM. Statination. *Internal Medicine News* 2000;Feb 1:55.
616. Haney DQ. Cholesterol drug is very secret weapon. *San Diego Union Tribune* 1999 Aug 23, 1999;Sect. E2.
617. Graham DJ, Staffa JA, Shatin D, et al. Incidence of hospitalized rhabdomyolysis in patients treated with lipid-lowering drugs. *Jama* 2004;292:2585-90.
618. Shinjyo T, Shinjyo A. Significant decrease of clinical symptoms after mobile phone base station removed: An intervention study. 2014.
619. Lilja JJ, Kivisto KT, Neuvonen PJ. Grapefruit juice-simvastatin interaction: effect on serum concentrations of simvastatin, simvastatin acid, and HMG-CoA reductase inhibitors. *Clin Pharmacol Ther* 1998;64:477-83.
620. Oh J, Ban MR, Miskie BA, Pollex RL, Hegele RA. Genetic determinants of statin intolerance. *Lipids Health Dis* 2007;6:7.
621. Vladutiu GD, Simmons Z, Isackson PJ, et al. Genetic risk factors associated with lipid-lowering drug-induced myopathies. *Muscle Nerve* 2006;34:153-62.
622. Golomb BA. Statins and activity: proceed with caution. *JAMA Intern Med* 2014;174:1270-2.
623. Golomb BA, Koperski S. Who becomes weak on statins? Effect modification exposed in a RCT by risk factor compounding. *Circulation* 2013;127:AP072.
624. Sinzinger H, O'Grady J. Professional athletes suffering from familial hypercholesterolaemia rarely tolerate statin treatment because of muscular problems. *British Journal of Clinical Pharmacology* 2004;57:525-8.
625. [www.es-uk](http://www.es-uk). Gro Harlem Brundtland and EHS. Electrosensitivity UK newsletter, 2012.
626. Math teacher asks school to protect children from Wi-Fi. North Kingston School Committee Meeting - Rhode Island USA Feb 10, 2015 2015; <https://www.youtube.com/watch?v=UqrW4ZJb5Uc>:Posted Feb 11, 2016 by Parents for Safe Technology. Download date Feb 14, 8.
627. International Association of Fire Fighters Division of Occupational Health Safety and Medicine. Position on the health effects from radio frequency/ microwave (RF/MW) radiation in fire department facilities from base stations for antennas and towers for the conduction of cell phone transmissions. 2006.
628. Foster S. Health exemption for firefighters sends a message to the world. <http://betweenrockandhardplacewordpresscom> 2017;June 26.
629. State of California Senate Bill 649 (SB-649): Wireless Telecommunications Bill. Passed the Assembly Sept 13, 2017 Passed the Senate Sept 14, 2017 Vetoes by Governor Brown 2017.
630. Cho YM, Lim HJ, Jang H, et al. A follow-up study of the association between mobile phone use and symptoms of ill health. *Environ Health Toxicol* 2016;32:e2017001.

631. Tseng M-CM, Lin Y-P, Cheng t-j. Prevalence and psychiatric comorbidity of self-reported electromagnetic field sensitivity in Taiwan: a population-based study. J Formosan Medical Association 2011;110:634-41.
632. Tachover D. The Israeli Supreme Court ordered the Israeli Government to investigate the number of children currently suffering from EHS. EMFacts 2013;Jul 23: <https://www.emfacts.com/2013/07/the-israeli-supreme-court-ordered-the-israeli-government-to-investigate-the-number-of-children-currently-suffering-fr...>
633. Eltiti S, Wallace D, Zougkou K, et al. Development and evaluation of the electromagnetic hypersensitivity questionnaire. Bioelectromagnetics 2007;28:137-51.
634. IDEA The Irish Doctors' Environmental Association. IDEA position on electromagnetic radiation. . <http://www.idealireland.org/emr.htm> 2004.
635. [www.iervn.com](http://www.iervn.com) (Electrosensitivity organization in Ireland).
636. Bigorra D. Electromagnetic hypersensitivity is on the rise (Article from Spain; Google Translation with grammatical correction). <http://mieuxprevenirblogspotcom/2017/01/electrohypersensitivity-is-on-rise.html>  
[http://www.aracat.es/Hipersensibilidad-electromagnetica-trastorno-desconocido-alza\\_0\\_1688231329.html?utm\\_medium=social@utm\\_source=facebook&utm\\_campaign=ara](http://www.aracat.es/Hipersensibilidad-electromagnetica-trastorno-desconocido-alza_0_1688231329.html?utm_medium=social@utm_source=facebook&utm_campaign=ara) 2016;Nov 13.
637. Hutter HP, Moshhammer H, Wallner P, Kundi M. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. Occup Environ Med 2006;63:307-13.
638. Hensinger P, Wilke I. Wireless communication technologies: New study findings confirm risks of nonionizing radiation. Translated by Katharina Gustavs. . Umwelt-medizin-gesellschaft 2016;March: [www.diagnose-funk.org](http://www.diagnose-funk.org), [www.mobilfunkstudien.org](http://www.mobilfunkstudien.org).
639. EHS Foreningen (EHS Association). Hearing at the Danish Parliament on wireless radiation puts pressure on the National Board of Health. Press release 2018;Apr 10: <https://via.ritzau.dk/pressemeddelelse?publisherId=12609765&releaseId=76>.
640. Nordström G. The Invisible Disease. New York, USA: O Books; 2004.
641. [www.felo.no](http://www.felo.no). Foreningen for el-overfølsomme (Norwegian Electrosensitive Society).
642. Nikka A. Former Nokia Boss: Mobile-Phones wrecked my health. (Translated from Finnish by Henrik Eriksson). Satakunnan Kansa 2014; Translation posted on: <http://betweenrockandhardplace.wordpress.com/2014/10/18/former-nokia-technology-chief-mobile-phones-wrecked-my-health>.
643. Harkinson J. This former techie owes his fortune to electronic devices. Now he thinks they're dangerous. Mother Jones 2017;Jan 28.
644. Johnson J. <https://www.wemfanalysis.com/about/>; <https://www.youtube.com/watch?v=F0NEaPTu9oI>.
645. Clegg F. Electrohypersensitivity Is Real. The Huffington Post, Canada 2013;June 12, 2013.
646. Dolk H, Shaddick G, Walls P, et al. Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter. American journal of epidemiology 1997;145:1-9.
647. Hocking B, Gordon I. Decreased survival for childhood leukemia in proximity to television towers. Arch Environ Health 2003;58:560-4.
648. Peleg M, Nativ O, Richter ED. Radio frequency radiation-related cancer: assessing causation in the occupational/military setting. . Environ Res 2018;163:123-33.
649. Elwood JM. Microwaves in the cold war: The Moscow embassy study and its interpretation. Review of a retrospective cohort study. Environmental Health 2012;11:85-.
650. Goldsmith JR. Where the trail leads. . . Ethical problems arising when the trail of professional work lead to evidence of cover-up of serious risk and mis-representation of scientific judgement concerning human exposures to radar. Eubios J Asian Int Bioeth 1995;5:92-4.
651. Hardell L, Carlberg M. Mobile phone and cordless phone use and the risk for glioma: Analysis of pooled case-control studies in Sweden, 1997–2003 and 2007–2009. Pathophysiology 2015;22:1-13.
652. Hardell L, Carlberg M, Soderqvist F, Mild KH. Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997-2003 and 2007-2009 and use of mobile and cordless phones. Int J Oncol 2013;43:1036-44.
653. Hardell L, Carlberg M. Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones. Rev Environ Health 2013;28:97-106.
654. Hardell L, Carlberg M, Hansson Mild K. Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects. Int J Oncol 2011;38:1465-74.
655. Balekrouzou A, Yin P, Afewerky HK, et al. Behavioral risk factors of breast cancer in Bangui of Central Africa Republic: A retrospective case-control study. PLoS ONE 2017;12:e0171154.
656. West JG, Kapoor NS, Liao SY, Chen JW, Bailey L, Nagourney RA. Multifocal Breast Cancer in Young Women with Prolonged Contact between Their Breasts and Their Cellular Phones. Case Rep Med 2013;2013:354682.

**GOLOMB Attachment 3**



Gunnar Heuser\* and Sylvia A. Heuser

# Functional brain MRI in patients complaining of electrohypersensitivity after long term exposure to electromagnetic fields

DOI 10.1515/reveh-2017-0014

Received April 3, 2017; accepted May 25, 2017; previously published online July 5, 2017

## Abstract

**Introduction:** Ten adult patients with electromagnetic hypersensitivity underwent functional magnetic resonance imaging (fMRI) brain scans. All scans were abnormal with abnormalities which were consistent and similar. It is proposed that fMRI brain scans be used as a diagnostic aid for determining whether or not a patient has electromagnetic hypersensitivity. Over the years we have seen an increasing number of patients who had developed multi system complaints after long term repeated exposure to electromagnetic fields (EMFs). These complaints included headaches, intermittent cognitive and memory problems, intermittent disorientation, and also sensitivity to EMF exposure. Regular laboratory tests were within normal limits in these patients. The patients refused to be exposed to radioactivity. This of course ruled out positron emission tomography (PET) and single-photon emission computed tomography (SPECT) brain scanning. This is why we ordered fMRI brain scans on these patients. We hoped that we could document objective abnormalities in these patients who had often been labeled as psychiatric cases.

**Materials and methods:** Ten patients first underwent a regular magnetic resonance imaging (MRI) brain scan, using a 3 Tesla Siemens Verio MRI open system. A functional MRI study was then performed in the resting state using the following sequences:

1. A three-dimensional, T1-weighted, gradient-echo (MPRAGE)
2. Resting state network. The echo-planar imaging (EPI) sequences for this resting state blood oxygenation level

dependent (BOLD) scan were then post processed on a 3D workstation and the independent component analysis was performed separating out the various networks.

3. Arterial spin labeling.
4. Tractography and fractional anisotropy.

**Results:** All ten patients had abnormal functional MRI brain scans. The abnormality was often described as hyper connectivity of the anterior component of the default mode in the medial orbitofrontal area. Other abnormalities were usually found. Regular MRI studies of the brain were mostly unremarkable in these patients.

**Conclusion:** We propose that functional MRI studies should become a diagnostic aid when evaluating a patient who claims electrohypersensitivity (EHS) and has otherwise normal studies. Interestingly, the differential diagnosis for the abnormalities seen on the fMRI includes head injury. It turns out that many of our patients indeed had a history of head injury which was then followed sometime later by the development of EHS. Many of our patients also had a history of exposure to potentially neurotoxic chemicals, especially mold. Head injury and neurotoxic chemical exposure may make a patient more vulnerable to develop EHS.

**Keywords:** electrohypersensitivity (EHS); electromagnetic field (EMF); fMRI; multiple chemical sensitivity (MCS).

## Introduction

In the past the senior author (G.H.) practiced clinical toxicology and in that capacity saw more than 1000 patients who had suffered exposure to toxic chemicals. Their impairment was often neurologic with loss of memory function, headaches, intermittent confusion, problems with balance, and other symptoms. This impairment had persisted, at times for years after exposure to these toxic chemicals had ceased. Some of these patients had developed sensitivity to even small amounts of chemicals resulting in multiple chemical sensitivity (MCS). More than 60 patients were eventually studied and the results were published in a peer reviewed journal [1]. All of these patients had single-photon emission computed tomography

\*Corresponding author: Gunnar Heuser, MD, PhD, Formerly UCLA Medical Center, Department of Medicine, PO Box 5066, El Dorado Hills, Los Angeles, CA 95762, USA, Phone: +(310) 500-0041, E-mail: toxguns@netscape.net, Website: emfdoc.com; Emeritus Cedars Sinai Medical Center, Department of Medicine, Los Angeles, CA, USA; Former Member of Brain Research Institute, UCLA Medical Center, Los Angeles, CA, USA  
**Sylvia A. Heuser:** Environmental, Medical, Research and Information Center (EMRIC), Santa Barbara, CA, USA



(SPECT) brain scans all of which were abnormal. Additional studies were then performed and published [2, 3].

More recently we began to see patients who reported significant reactions to even small amounts of electromagnetic fields (EMFs). Some of these patients had in the past been seen for problems with chemical exposure and had now developed electrohypersensitivity (EHS). Some of these patients also gave a history of head injury.

Electrohypersensitivity has since been acknowledged by the medical profession since it demands more and more attention and evaluation [4–7]. The syndrome has now been called EHS.

Patients with EHS develop multi-system complaints on exposure to currents emitted by cell phones, cell phone towers, smart meters, power-lines and other sources of EMFs.

In this paper we present patients with EHS who had undergone functional brain MRI studies because of their complaints which were mostly neurological after exposure: memory and cognitive impairment, attention deficit disorder, changes in behavior, and other symptoms. They presented with a history of long term exposure to EMFs followed by development of EHS.

Every patient included in this paper reported significant symptomatology which served to arrive at a diagnosis of EHS. Symptoms developed upon exposure and usually diminished or disappeared when removed from EMF source. Multi-symptoms included headaches, impairment of cognitive function, tremors, weakness, and others. Multi-system complaints were triggered by exposure to cell phones, cell phone towers, smart meters, wi-fi, and other sources (see description of individual cases).

A careful and thorough lab evaluation ruled out diseases which often cause multi system complaints e.g. thyroid problems, diabetes, autoimmune disease, chronic infections and other conditions. Patients often provided pictures of nearby cell phone towers, smart meters and other sources of EMFs to document their claim.

## Methodology

All patients signed a release form allowing us to publish the results of their study. Our control population consisted of 60 volunteers of both sexes, ages 15–70. They were not on drugs, had no known diseases and were otherwise in perfect health. Our study group consisted of 10 patients all of whom experienced long term EMF exposure and developed EHS.

All patients had regular magnetic resonance imaging (MRI) brain scans using a 3 Tesla Siemens Verio MRI Open system. This was followed by an fMRI study, using the following sequences:

1. A three-dimensional, T1-weighted, gradient-echo (MPRAGE).
2. Resting state network. The echo-planar imaging (EPI) sequences for this resting state blood oxygenation level dependent (BOLD)

**Table 1:** Patient population.

Case number	Age	Gender	Head injury	Chem. Ex.
Case #1	60's	Female	Yes	Yes
Case#2	40's	Male	No	No
Case #3	60's	Male	Yes	Yes
Case #4	50's	Female	No	Yes
Case #5	50's	Female	No	Yes
Case #6	60's	Female	No	Yes
Case #7	70's	Male	No	Yes
Case #8	60's	Female	Yes	Yes
Case #9	60's	Female	Yes	Yes
Case #10	60's	Male	Yes	Yes

- scan were then post processed on a 3D workstation and the independent component analysis was performed separating out the various networks.
3. Arterial spin labeling.
  4. Tractography and fractional anisotropy (Table 1).

## Results

**Case 1.** This right handed patient was in her early sixties when she was evaluated. Whenever exposed to EMFs she developed cognitive and memory problems as well as a sense of malaise to the point of inability to function. Her EHS developed over the years. Her history includes multiple head injuries and also exposure to toxic chemicals, including mold, eventually resulting in multiple chemical sensitivity (MCS)

The fMRI showed severely abnormal default mode network (DMN) with hyper connectivity of the anterior component in the medial orbitofrontal area, also decrease of white matter tracts within the right frontal lobe, and finally decreased flow and/or metabolism within bifrontal lobes (Figure 1).

**Case 2.** This right handed patient was in his forties when evaluated. Several years before he had an abnormal SPECT brain scan and an abnormal neuropsychological test result suggesting attention deficit disorder (ADD). For approximately 11 years he had worked as an air conditioning expert, working on the roofs of many commercial buildings, thus being exposed to EMFs. He developed cognitive and memory problems, impaired coordination and balance, insomnia, chronic fatigue, and finally EHS. In addition he was diagnosed to have bi-lateral cataracts impairing his vision. His history is negative for head injury and toxic chemical exposure.

The fMRI showed abnormality of the DMN with increased hyperconnectivity of the anterior component.

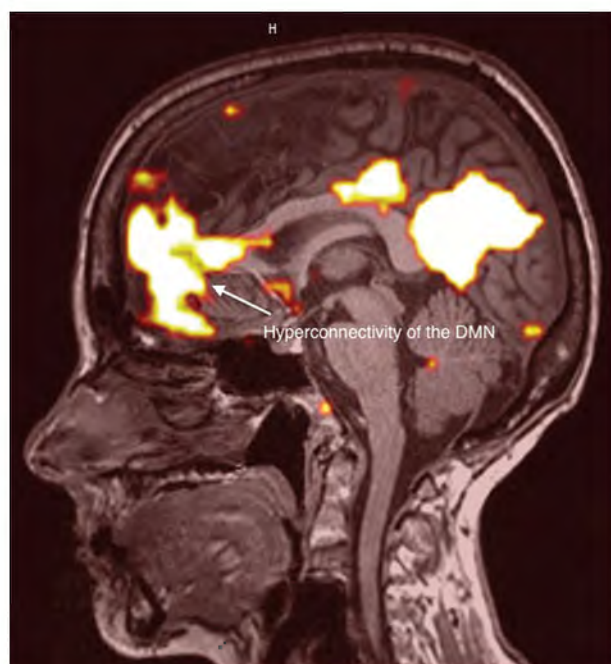


Figure 1: Lateral view of case no. 1.



Figure 2: Lateral view of case no. 2.

Fractional anisotropy was found to be decreased in the corpus callosum (Figure 2).

**Case 3.** This right handed patient was in his sixties when being evaluated. He had worked in a high voltage environment as a journeyman lineman for more than 30 years, evaluating and treating problems in that environment.



Figure 3: Lateral view of case no. 3.

Eventually, he developed a seizure disorder with loss of consciousness. His seizures subsided when he was off work and returned when he returned to work.

He had a concussion in his teenage years. He had not been exposed to mold.

The fMRI showed fragmentation of the anterior component of the DMN, also decreased fractional anisotropy, predominantly in the corpus callosum (Figure 3).

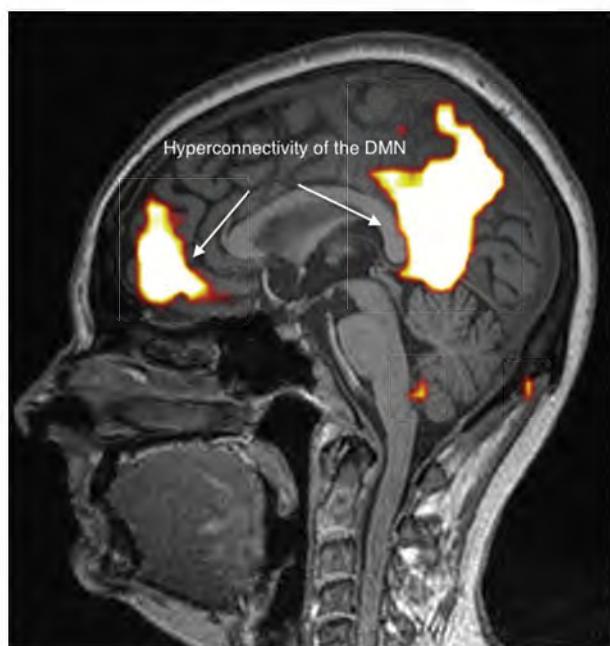
**Case 4.** This right handed patient was in her early fifties when tested. Exposure to chemicals started in childhood when she developed double pneumonia, allergies, and eventually asthma

For years before our testing she worked as an air traffic controller, surrounded by cell phone towers at work. In that setting she developed EHS.

Her history is negative for head injury. Her past history includes exposure to mold.

The fMRI showed abnormal DMN with hyper connectivity and fragmentation of the anterior component. There was also hyper connectivity of the posterior component. Finally there was decreased flow and/or metabolism in the bifrontal lobes (Figure 4).

**Case 5.** This left handed female was in her early fifties when tested. About 15 years earlier she was exposed to chemicals and eventually developed MCS. About 9 years earlier she got into the habit of holding her cell phone to her left ear in which she eventually developed pain, whenever she was using the phone. Additional symptoms



**Figure 4:** Lateral view of case no. 4.

eventually developed and included impaired cognitive function, intermittent confusion, headaches, nausea, and generalized weakness, all of these eventually developing into EHS. Her history is negative for head injury and for mold exposure.

The fMRI showed markedly hyperconnected medial orbitofrontal component in the resting state network. Diminished marked asymmetry of blood flow with

diminished flow in the right frontal temporal region was also found (Figure 5).

**Case 6.** This left handed patient was in her sixties when evaluated. Just a few years before, AT&T constructed a cell phone tower about 500 yards from her home. She developed impaired memory, speech pressure, insomnia, dry eyes and eventually EHS.

Her history is negative for head injury and positive for mold exposure.

The fMRI showed severely abnormal DMN with hyperconnectivity of the anterior component (Figure 6).

**Case 7.** This right handed male was in his seventies when tested. In his work area he had been exposed to EMFs, being surrounded by cell phone towers, many computers, and electrical equipment. He had developed memory and cognitive problems, together with headaches and eventually EHS. Years ago he had been exposed to diesel fumes and more recently to mold. The history is negative for head injury.

The fMRI showed abnormal DMN with fragmentation of the anterior component in the medial orbitofrontal area. It also showed decreased flow and/or metabolism within the bilateral posterior parietal lobes. Finally diffusely decreased white matter tracts in the left cerebral hemisphere were found (Figure 7).

**Case 8.** This right handed patient was in her sixties when testing was done. EMF exposure started around 2005 and



**Figure 5:** Lateral view of case no. 5.



**Figure 6:** Lateral view of case no. 6.



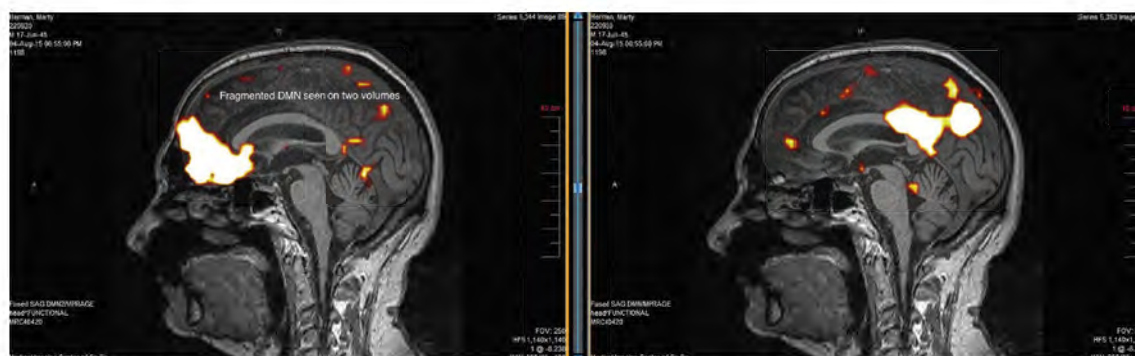


Figure 7: Lateral views of case no. 7.

continued ever since. This continual exposure eventually resulted in the development of EHS. Her complaints included headaches, insomnia, blurred vision, and impairment of speech. Her medical history includes allergies including asthma, mold exposure, and head injuries at ages 10 and 21.

The fMRI showed abnormal DMN with hyper connectivity of the anterior component of the medial orbitofrontal area. Also found was mildly decreased flow and/or metabolism in the bifrontal lobes (arterial spin labeling). Finally there was symmetric loss of white matter tracts in the left posterior parietal lobe (Figure 8).

**Case 9.** This right handed patient was in her early sixties when evaluated. For more than 20 years she had tested

and repaired batteries and was therefore exposed to toxic metals and EMFs on every working day. She was also exposed to a nearby cell phone tower at work.

She developed cognitive and memory impairment, numbness, and eventually EHS and a seizure disorder. Additional medical history included an industrial accident with “electrocution” and also exposure to mold, first at work then at home. Finally her history is positive for head injury.

The fMRI showed a hyperconnected anterior component of the DMN in the medial orbitofrontal area. Also found was decreased fractional anisotropy in the body of the corpus callosum. Finally there was diminished flow within the bifrontal lobes (Figure 9).

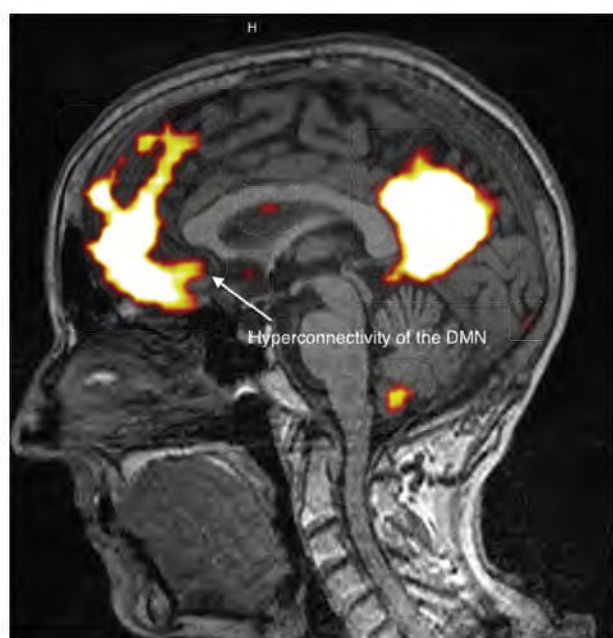


Figure 8: Lateral view of case no. 8.



Figure 9: Lateral view of case no. 9. (A) Sample MRI report for case no. 9. (B) Sample fMRI report for case no. 9.

**A****MRI OF THE BRAIN****HISTORY****Name:** DOB: 12/28/1954 Female**Exam Date:** 9/8/14**Referring Phys.:** GunnarHeuser,M.D.

This is a 59-year-old female with exposure to mold and mercury. The patient has symptoms of seizures, memory loss, and numbness in hands and left arm.

**PROCEDURE**

Using a 3 Tesla Siemens Verio MRI Open system, the following sequences were obtained:

- |                           |                                     |
|---------------------------|-------------------------------------|
| 1) Localizer.             | 4) DWI axial.                       |
| 2) T1 3D sagittal MPRAGE. | 5) SWI axial.                       |
| 3) T2 FLAIR sagittal.     | 6) T2 FLAIR axial. 7) T2 TSE axial. |

**FINDINGS**

No cavum septum pellucidum is seen. No blood products are visualized. There is no diffusion restriction.

There is no focal or global volume loss.

The posterior fossa is normal.

The sella and parasellar regions are normal. The flow voids of the vessels of the skull base are identified and normal.

The mastoid air cells, paranasal sinuses, and orbits are normal.

There are scattered foci of T2/FLAIR hyperintensity within the supratentorial subcortical white matter located in bifrontal lobes.

**IMPRESSION**

Scattered foci of T2/FLAIR hyperintensity in the supratentorial subcortical white matter representing gliosis. This can be seen in the setting of prior head trauma, chronic migraine headaches, less likely chronic small vessel ischemic disease given distribution.

**B****FUNCTIONAL MRI (fMRI) OF THE BRAIN****HISTORY DOB:** 12/28/1954 Female**Exam Date:** 9/8/14**Referring Phys.:** GunnarHeuser, M.D.

This is a 59-year-old female with exposure to mold and mercury, has symptoms of blackout, seizure, memory loss, head trauma to the left side of brain.

**PROCEDURE**

Using a 3 Tesla Siemens Verio MRI Open bore system, a functional MRI study was performed using the followings enquences:

- 1) MPRAGE.
- 2) Resting state network. The EPI sequences for this resting state BOLD scan were then post processed on a 3D workstation and the independent component analysis performed separating out the various networks.
- 3) Arterial spin labeling.
- 4) Tractography and fractional anisotropy.

**FINDINGS**

There is a markedly abnormal default mode network with hyperconnectivity of the anterior component of the default mode. This can be seen in the setting of OCD, chronicpain, post-traumatic brain injury and/or drug abuse.

The fractional anisotropy is low within the body of the corpus callosum with a minimal value of 0.22 in the anterior body .Diffusion tensor imaging is within normal.

The arterial spin labeling demonstrates symmetric diminished flow within bifrontal lobes.

**IMPRESSION**

1. Abnormal functional MRI with hyperconnected anterior component of the default mode network in the medial orbitofrontal area. This can be seen in the setting of traumatic brain injury, chronic pain, substance abuse and/or OCD.
2. Decreased fractional anisotropy within the body of the corpus callosum with a minimal value of 0.22 in th eanterior body. This is a sign of disorganization of fibers which can be seen in the setting of traumatic brain injury.
3. There is diminished flow within bifrontal regions which is consistent with hypometabolic activity. This can be seen in the setting of traumatic brain injury.

**Figure 9 (continued)**

**Case 10.** This right handed patient was in his sixties when testing was done. Over the years he had been exposed to pesticides and to mold. He had experienced a concussion about 10 years earlier. He gradually developed EHS.

The fMRI showed abnormal DMN with fragmentation and hyper connectivity of the anterior component of the default mode in the medial orbitofrontal area. There was borderline decreased flow and/or metabolism within bifrontal lobes (Figure 10).





Figure 10: Lateral views of case no. 10.

## Discussion

More than 30 years ago we became interested in the effects of toxic chemical exposure on the brain. We selected more than 70 patients who had developed multi system complaints after long-term exposure to potentially neurotoxic chemicals. All patients underwent functional brain scans of the SPECT type. They were all abnormal and statistically analyzed. The results were published [1] in a peer reviewed journal with the conclusion that neurotoxic chemical exposure can lead to long-term effects including abnormalities in brain function. The abnormalities seen were still present years after toxic exposure had ceased. The potentially neurotoxic effects of chemicals were further discussed in a German publication [3].

Since some of our patients had developed exquisite sensitivity to even small amounts of chemicals we decided to document brain function in these patients with multiple chemical sensitivity (MCS). For this study we chose positron emission tomography (PET) scanning of the brain and found definite objective abnormalities in these patients. The results were published with the conclusion that patients with MCS have objective abnormalities in brain function [2]. It should be noted that a number of PET studies were published by other authors but did not address MCS [4–7].

Of interest was the fact that PET brain scans in MCS patients showed increased activity in the amygdala [2]. This structure is known to control emotions. This is why some patients with MCS develop an emotional disorder which can be explained on the basis of a “hot” amygdala. In view of this past finding with our MCS patients we believe that EHS patients should be studied with PET scans so as to find out whether their amygdalae are also hyperactive.

MCS was further discussed in a consensus paper in which GH was a co-author [8].

In more recent years we started seeing patients who claimed exquisite sensitivity to electromagnetic fields (electrohypersensitivity, commonly recognized as EHS). It was very interesting to find that the complaints these patients had developed were similar to the complaints patients had who were exposed to and sensitive to chemicals: headaches, cognitive and memory problems, intermittent problems with balance, and intermittent tremor. This syndrome has been described by Carpenter and others [9–12].

The late Dr. Ross Adey [13] found that chemicals and EMFs can interact and aggravate each others’ effects. This became a finding we confirmed in our patients.

We suggested to some of our patients with EHS that they undergo a functional brain scan to document potential abnormalities. They agreed but did not want to have any radioactive material used for the scan. This of course excluded PET and SPECT scanning of the brain. Since these patients had significant complaints and were at times disabled we felt that it was important to obtain a functional MRI (fMRI) to document potential abnormalities of brain function. In the beginning this was considered as medically necessary and was not a research project and therefore not funded by any agency. Since insurance coverage is not yet available for fMRI, patients had to cover the cost of the whole evaluation. While fMRI studies look at functional connections between some brain areas, no studies in the literature specifically addressed functional connectivity after exposure to EMF. However, some studies in the literature refer to connections between specific areas of the brain by measuring EEG and related activities. None of these studies have addressed patients with EHS [14–16].

## Conclusion

All fMRI brain scans were abnormal in our patients. Their abnormalities were very similar in all of them.

The study of fMRI created a new language which had to be learned. We have included some references which help the reader to understand this new terminology [17–21].

The question now arises whether the same abnormalities seen in all 10 patients can become a diagnostic aid or biomarker for determining whether or not a patient has electrohypersensitivity (EHS). Some researchers have claimed that a diagnosis of EHS should really be a psychiatric diagnosis [22]. Other researchers have attempted to measure EHS [23]. The possible psychiatric aspects of EHS remind us of the history of medicine when epilepsy was considered to be a diagnosis of possession by the devil [24]. It was only the discovery of the EEG which helped to find epilepsy to be a real disease with objective findings. We hope that fMRI will play the role of the EEG in EHS.

In the context of the above paragraph we should mention the articles by Belpomme et al. [25] and De Luca et al. [26] which suggest a long list of biomarkers for the diagnosis of EHS. This list does not include functional brain scans as potential biomarkers.

The treatment of EHS is very difficult. It was reviewed in a recent publication [27]. We have found that hyperbaric oxygen is at times helpful [28]. We have proposed that every patient with EHS should be checked for mast cell disease since mast cell abnormalities were found by Gangi and Johansson [29]. We described mast cell disorder in patients with MCS [30]. Also we found mast cell disorder in some of our patients with EHS [31]. Finally we believe that mold and mold toxin (mycotoxin) exposure can trigger EHS. Mast cell disease and mold problems are treatable. Their treatment may decrease EHS. Otherwise treatment consists basically of avoidance.

A final point of interest is the fact that the abnormality seen on the fMRI can also be seen after head injury [32, 33]. Indeed, many of our patients complaining of EHS have a history of head injury (see our case reports).

Since neurotoxic chemicals as well as head injury and EMF exposure are known to impair the blood brain barrier, it is almost to be expected that singly or in combination they make a given patient more vulnerable to impaired brain function (including seizures).

In the past we studied patients who had developed multi system complaints after exposure to a number of neurotoxins. The only patients who developed a seizure disorder were the ones who gave a history of past head injury [31]. Two of our patients (#3 and 9) developed a well

documented seizure disorder. This can be understood as having been “kindled” by repeated exposures to EMFs.

Our study needs to be enlarged and also duplicated by others. The subject will be of increasing importance in our society in which we and our children are all exposed to more and more EMFs.

**Acknowledgements:** Medical Imaging of Southern California graciously made their studies (MRI and fMRI) available at a significant discount. We also acknowledge Brea Blevins who helped to retrieve and to display the images published in this paper. Most patients were either self referred or referred by their physician. Some were referred by The Peoples Initiative Foundation which was formed and is directed by Elizabeth Barris.

## Author Statement

**Research funding:** No funds for this study were available from any foundation or other financial entity for this study. **Partial payment was received for two patient studies by a charitable foundation (The Peoples Initiative Foundation).** Patients paid for their own consultations and testing. **No insurance reimbursements were available for the fMRI study.** **Conflict of interest:** Authors state no conflict of interest. **Informed consent:** Informed consent has been obtained from all individuals. **Ethical approval:** Ethical approval was not applicable.

## References

1. Heuser G, Mena I. Neurospect in neurotoxic chemical exposure. Demonstration of long-term functional abnormalities. *Toxicol Ind Health* 1998;14(6):813–27.
2. Heuser G, Wu JC. Deep subcortical (including limbic) hypermetabolism in patients with chemical intolerance: human PET studies. *Ann N Y Acad Sci* 2001;933:319–22.
3. Heuser G. Functional brain Imaging with SPECT and PET after neurotoxic exposure: two and three-dimensional displays. *Zeitschrift für Umweltmedizin* 1999;8:284–5.
4. Huber R, Treyer V, Schuderer J, Berthold T, Buck A, et al. [Exposure to pulse-modulated radio frequency electromagnetic fields affects regional cerebral blood flow.](#) *Eur J Neurosci* 2005;21(4):1000–6.
5. Aalto S, Haarala C, Brück A, Sipilä H, Hämäläinen H, et al. Mobile phone affects cerebral blood flow in humans. *J Cereb Blood Flow Metab* 2006;26(7):885–90.
6. Haarala C, Aalto S, Hautzel H, Julkunen L, Rinne JO, et al. Effects of a 902 MHz mobile phone on cerebral blood flow in humans: a PET study. *Neuroreport*. 2003;14(16):2019–23.
7. Huber R, Treyer V, Borbély AA, Schuderer J, Gottselig JM, et al. Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. *J Sleep Res* 2002;11(4):289–95.

8. Bartha L. Multiple chemical sensitivity: a 1999 consensus. *Arch Environ Health* 1999;54(3):147–9.
9. Carpenter DO. The microwave syndrome or electrohypersensitivity: historical background. *Rev Environ Health* 2015;30(4):217–22.
10. Hedendahl L, Carlberg M, Hardell L. Electromagnetic hypersensitivity – an increasing challenge to the medical profession. *Rev Environ Health* 2015;30(4):209–15.
11. Genuis SJ, Lipp CT. Electromagnetic hypersensitivity: fact or fiction? *Sci Total Environ* 2012;414:103–12.
12. McCarty DE, Carrubba S, Chesson AL, Frilot C, Gonzalez-Toledo E, et al. Electromagnetic hypersensitivity: evidence for a novel neurological syndrome. *Int J Neurosci* 2011;121(12):670–6.
13. Adey WR. Joint actions of environmental nonionizing electromagnetic fields and chemical pollution in cancer promotion. *Environ Health Perspect* 1990;86:297–305.
14. Haarala C, Takio F, Rintee T, Laine M, Koivisto M, et al. Pulsed and continuous wave mobile phone exposure over left versus right hemisphere: effects on human cognitive function. *Bioelectromagnetics* 2007;28(4):289–95.
15. Vecchio F, Babiloni C, Ferreri F, Curcio G, Fini R, et al. Mobile phone emission modulates interhemispheric functional coupling of EEG alpha rhythms. *Eur J Neurosci* 2007;25(6):1908–13.
16. Yang L, Chen Q, Lv B, Wu T. Long-Term evolution electromagnetic fields exposure modulates the resting state EEG on alpha and beta bands. *Clin EEG Neurosci* 2017;48(3):168–75.
17. van den Heuvel MP, Hulshoff Pol HE. Exploring the brain network: a review on resting-state fMRI functional connectivity. *Eur Neuropsychopharmacol* 2010;20(8):519–34.
18. Horn A, Dirk O, Reiser M, Blankenburg F. Default Mode Network. [revised 2016 August]. In: Wikipedia [Internet]. San Francisco, CA: Neurolmage; 2006 September. 10 pages. Available from: [www.wikipedia.com](http://www.wikipedia.com). DOI: 10.1016.
19. Liston C, Chen AC, Zebley BD, Drysdale AT, Gordon R, et al. Default mode network mechanisms of transcranial magnetic stimulation in depression. *Biol Psychiatry* 2014;76(7):517–26.
20. Smith SM, Vidaurre D, Beckmann CF, Glasser MF, Jenkinson M, et al. Functional connectomics from resting-state fMRI. *Trends Cogn Sci* 2013;17(12):666–82.
21. Lee MH, Smyser CD, Shimony JS. Resting-state fMRI: a review of methods and clinical applications. *AJNR Am J Neuroradiol* 2013;34(10):1866–72.
22. Rubin GJ, Hillert L, Nieto-Hernandez R, van Rongen E, Oftedal G. Do people with idiopathic environmental intolerance attributed to electromagnetic fields display physiological effects when exposed to electromagnetic fields? A systematic review of provocation studies. *Bioelectromagnetics* 2011;32(8):593–609.
23. Tuengler A, von Klitzing L. Hypothesis on how to measure electromagnetic hypersensitivity. *Electromagn Biol Med* 2013;32(3):281–90.
24. Espí Forcén C, Espí Forcén F. Demonic possessions and mental illness: discussion of selected cases in late medieval hagiographical literature. *Early Sci Med* 2014;19(3):258–79.
25. Belpomme D, Campagnac C, Irigaray P. Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder. *Rev Environ Health* 2015;30(4):251–71.
26. De Luca C, Chung Sheun Thai J, Raskovic D, Cesareo E, Caccamo D, et al. Metabolic and genetic screening of electromagnetic hypersensitive subjects as a feasible tool for diagnostics and intervention. *Mediators Inflamm* 2014;2014:924184.
27. Rubin GJ, Das Munshi J, Wessely S. A systematic review of treatments for electromagnetic hypersensitivity. *Psychother Psychosom* 2006;75(1):12–8.
28. Heuser G, Uszler JM. Hyperbaric oxygenation for cerebral palsy. *Lancet* 2001;357(9273):2053–4. Erratum in: *Lancet* 2001 Nov 24;358(9295):1820.
29. Gangi S, Johansson O. A theoretical model based upon mast cells and histamine to explain the recently proclaimed sensitivity to electric and/or magnetic fields in humans. *Med Hypotheses* 2000;54(4):663–71.
30. Heuser G. Mast cell disorder to be ruled out in MCS. *Arch Environ Health* 2000;55(4):284–5.
31. Unpublished observations by authors.
32. Mishra AM, Bai X, Sanganahalli BG, Waxman SG, Shatillo O, et al. Decreased resting functional connectivity after traumatic brain injury in the rat. *Plos One* 2014;09(4):e95280.
33. Zhou Y, Milham M, Lui Y, Zhou Y, Milham MP, et al. Default-mode network disruption in mild traumatic brain injury. *Radiology* 2012;265(3):882–92.

**GOLOMB Attachment 4**

This PDF is available at <http://nap.edu/25889>

SHARE



## An Assessment of Illness in U.S. Government Employees and Their Families at Overseas Embassies (2020)

### DETAILS

76 pages | 8.5 x 11 | PAPERBACK

ISBN 978-0-309-68137-7 | DOI 10.17226/25889

### CONTRIBUTORS

David A. Relman and Julie Pavlin, Editors; Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies; Health and Medicine Division; Division on Engineering and Physical Sciences; National Academies of Sciences, Engineering, and Medicine

### SUGGESTED CITATION

National Academies of Sciences, Engineering, and Medicine 2020. *An Assessment of Illness in U.S. Government Employees and Their Families at Overseas Embassies*. Washington, DC: The National Academies Press.  
<https://doi.org/10.17226/25889>.

GET THIS BOOK

FIND RELATED TITLES

Visit the National Academies Press at [NAP.edu](http://NAP.edu) and login or register to get:

- Access to free PDF downloads of thousands of scientific reports
- 10% off the price of print titles
- Email or social media notifications of new titles related to your interests
- Special offers and discounts



Distribution, posting, or copying of this PDF is strictly prohibited without written permission of the National Academies Press. (Request Permission) Unless otherwise indicated, all materials in this PDF are copyrighted by the National Academy of Sciences.

Copyright © National Academy of Sciences. All rights reserved.



# **An Assessment of Illness in U.S. Government Employees and Their Families at Overseas Embassies**

Standing Committee to Advise the Department of State  
on Unexplained Health Effects on U.S. Government  
Employees and Their Families at Overseas Embassies

David A. Relman and Julie A. Pavlin, *Editors*

Health and Medicine Division

Division on Engineering and Physical Sciences

**A Consensus Study Report of**  
*The National Academies of*  
**SCIENCES • ENGINEERING • MEDICINE**

THE NATIONAL ACADEMIES PRESS

*Washington, DC*

[www.nap.edu](http://www.nap.edu)

**THE NATIONAL ACADEMIES PRESS 500 Fifth Street, NW Washington, DC 20001**

This activity was supported by Contract 19AQMM19C0090 between the National Academy of Sciences and the Department of State. Any opinions, findings, conclusions, or recommendations expressed in this publication do not necessarily reflect the views of any organization or agency that provided support for the project.

International Standard Book Number-13: 978-0-309-XXXXX-X

International Standard Book Number-10: 0-309-XXXXX-X

Digital Object Identifier: <https://doi.org/10.17226/25889>

Additional copies of this publication are available from the National Academies Press, 500 Fifth Street, NW, Keck 360, Washington, DC 20001; (800) 624-6242 or (202) 334-3313; <http://www.nap.edu>.

Copyright 2020 by the National Academy of Sciences. All rights reserved.

Printed in the United States of America

Suggested citation: National Academies of Sciences, Engineering, and Medicine. 2020. *An assessment of illness in U.S. government employees and their families at overseas embassies*. Washington, DC: The National Academies Press. <https://doi.org/10.17226/25889>

*The National Academies of*  
**SCIENCES • ENGINEERING • MEDICINE**

The **National Academy of Sciences** was established in 1863 by an Act of Congress, signed by President Lincoln, as a private, nongovernmental institution to advise the nation on issues related to science and technology. Members are elected by their peers for outstanding contributions to research. Dr. Marcia McNutt is president.

The **National Academy of Engineering** was established in 1964 under the charter of the National Academy of Sciences to bring the practices of engineering to advising the nation. Members are elected by their peers for extraordinary contributions to engineering. Dr. John L. Anderson is president.

The **National Academy of Medicine** (formerly the Institute of Medicine) was established in 1970 under the charter of the National Academy of Sciences to advise the nation on medical and health issues. Members are elected by their peers for distinguished contributions to medicine and health. Dr. Victor J. Dzau is president.

The three Academies work together as the **National Academies of Sciences, Engineering, and Medicine** to provide independent, objective analysis and advice to the nation and conduct other activities to solve complex problems and inform public policy decisions. The National Academies also encourage education and research, recognize outstanding contributions to knowledge, and increase public understanding in matters of science, engineering, and medicine.

Learn more about the National Academies of Sciences, Engineering, and Medicine at [www.nationalacademies.org](http://www.nationalacademies.org).

*The National Academies of*  
**SCIENCES • ENGINEERING • MEDICINE**

**Consensus Study Reports** published by the National Academies of Sciences, Engineering, and Medicine document the evidence-based consensus on the study's statement of task by an authoring committee of experts. Reports typically include findings, conclusions, and recommendations based on information gathered by the committee and the committee's deliberations. Each report has been subjected to a rigorous and independent peer-review process and it represents the position of the National Academies on the statement of task.

**Proceedings** published by the National Academies of Sciences, Engineering, and Medicine chronicle the presentations and discussions at a workshop, symposium, or other event convened by the National Academies. The statements and opinions contained in proceedings are those of the participants and are not endorsed by other participants, the planning committee, or the National Academies.

For information about other products and activities of the National Academies, please visit [www.nationalacademies.org/about/whatwedo](http://www.nationalacademies.org/about/whatwedo).

## STANDING COMMITTEE TO ADVISE THE DEPARTMENT OF STATE ON UNEXPLAINED HEALTH EFFECTS ON U.S. GOVERNMENT EMPLOYEES AND THEIR FAMILIES AT OVERSEAS EMBASSIES

**DAVID A. RELMAN** (*Chair*), Thomas C. and Joan M. Merigan Professor, Department of Medicine, Department of Microbiology & Immunology; Senior Fellow, Freeman Spogli Institute for International Studies, Stanford University; Chief of Infectious Diseases, Veterans Affairs Palo Alto Health Care System

<sup>1</sup>**DORIS-EVA BAMIOU**, Professor of Neuroaudiology, Ear Institute, University College of London

**LINDA BIRNBAUM**, Director (*retired*), National Institute of Environmental Health Sciences, National Institutes of Health

**MICHAEL BONINGER**, Professor and Endowed Vice Chair for Research, Department of Physical Medicine and Rehabilitation, University of Pittsburgh School of Medicine

**RONALD BROOKMEYER**, Dean, Jonathon and Karin Fielding School of Public Health, University of California, Los Angeles

**CAROLINE BUCKEE**, Associate Professor of Epidemiology, Harvard T.H. Chan School of Public Health

**TIMOTHY J. BUCKLEY**, Exposure Methods and Measurements Division, National Exposure Research Laboratory, U.S. Environmental Protection Agency

**JOSEPH J. FINS**, E. William Davis, Jr., M.D. Professor of Medical Ethics; Chief, Division of Medical Ethics; Professor of Medicine, Professor of Medical Ethics in Neurology, Professor of Medical Ethics in Rehabilitation Medicine, Professor of Medicine in Psychiatry, Professor of Health Care Policy and Research, Division of Medical Ethics, Weill Cornell Medical College

**JOHN C. GORE**, Director and Hertha Ramsey Cress University Professor of Radiology and Radiological Sciences, Biomedical Engineering, Physics and Astronomy, and Molecular Physiology and Biophysics, Institute of Imaging Science, Vanderbilt University

**WALTER KOROSHETZ**, Director, National Institute of Neurological Disorders and Stroke, National Institutes of Health

**PAMELA LEIN**, Professor of Neurotoxicology and Department Chair, Department of Molecular Biosciences, School of Veterinary Medicine, University of California, Davis

**SAAFAN MALIK**, Director of Research and Acting Deputy Division Chief, Defense & Veterans Brain Injury Center, Research & Development Directorate J-9, Defense Health Agency, Department of Defense

**JEFFREY S. PALMER**, Group Leader, Human Health and Performance Systems Group, Lincoln Laboratory, Massachusetts Institute of Technology

**GREGORY B. SAATHOFF**, Professor of Emergency Medicine, Professor of Public Health Sciences, University of Virginia School of Medicine

**CLIFFORD B. SAPER**, James Jackson Putnam Professor and Department Chair, Department of Neurology, Harvard Medical School

**MARK J. SHELHAMER**, Professor of Otolaryngology, Johns Hopkins University School of Medicine

**JEFFREY P. STAAB**, Professor of Psychiatry, Director of the Fellowship in Consultation-Liaison Psychiatry, Department of Psychiatry and Psychology, Mayo Clinic, College of Medicine and Science; Consultant in the Departments of Psychiatry, Psychology and Otorhinolaryngology, Head and Neck Surgery at Mayo Clinic

**JONATHAN D. TROBE**, Professor, Ophthalmology and Visual Sciences, Department of Neurology; Co-Director, Kellogg Eye Center for International Ophthalmology, University of Michigan

**DAVID WHELAN**, Professor of the Practice, Electrical Engineering, University of California, San Diego

---

<sup>1</sup> Doris-Eva Bamiou is a member of the Standing Committee, but was unable to participate directly in the authoring of this report.



*Health and Medicine Division Project Staff*

**LIZA HAMILTON**, Program Officer

**CLAIRE MOERDER**, Research Assistant

**MARGARET MCFARLAND**, Senior Program Assistant

**JULIE PAVLIN**, Senior Director, Board on Global Health

*Department on Engineering and Physical Sciences Staff*

**ALAN SHAW**, Director, Intelligence Community Studies Board

**WILLIAM MILLONIG**, Director, Board on Army Research and Development

## Reviewers

This Consensus Study Report was reviewed in draft form by individuals chosen for their diverse perspectives and technical expertise. The purpose of this independent review is to provide candid and critical comments that will assist the National Academies of Sciences, Engineering, and Medicine in making each published report as sound as possible and to ensure that it meets the institutional standards for quality, objectivity, evidence, and responsiveness to the study charge. The review comments and draft manuscript remain confidential to protect the integrity of the deliberative process.

We thank the following individuals for their review of this report:

**Ellen Wright Clayton**, Center for Biomedical Ethics and Society

**Marion Ehrich**, Virginia Polytechnic Institute & State University

**Michael E. Goldberg**, Columbia University College of Physicians and Surgeons

**Joshua A. Gordon**, National Institute of Mental Health

**Suzet McKinney**, Illinois Medical District

**Aubrey K. Miller**, National Institute of Environmental Health Sciences

**Xin Qi**, Case Western Reserve University

**David A. Savitz**, Brown University

**Susan L. Whitney**, University of Pittsburgh

**Ross Zafonte**, Harvard University

Although the reviewers listed above provided many constructive comments and suggestions, they were not asked to endorse the conclusions or recommendations of this report nor did they see the final draft before its release. The review of this report was overseen by **Linda A. McCauley**, Emory University, and **Robert F. Sproull**, University of Massachusetts at Amherst. They were responsible for making certain that an independent examination of this report was carried out in accordance with the standards of the National Academies and that all review comments were carefully considered. Responsibility for the final content rests entirely with the authoring committee and the National Academies.

# Contents

<b>ACRONYMS AND ABBREVIATIONS</b>	<b>ix</b>
<b>PREFACE</b>	<b>xi</b>
<b>SUMMARY</b>	<b>1</b>
<b>SECTION 1 INTRODUCTION AND CHARGE TO THE COMMITTEE</b>	<b>5</b>
<b>SECTION 2 METHODS AND DATA</b>	<b>8</b>
<b>SECTION 3 CLINICAL FEATURES</b>	<b>10</b>
<b>SECTION 4 PLAUSIBLE MECHANISMS</b>	<b>17</b>
<b>SECTION 5 ACUTE TREATMENT AND REHABILITATION</b>	<b>34</b>
<b>SECTION 6 LOOKING TO THE FUTURE AND RECOMMENDATIONS</b>	<b>41</b>
<b>APPENDIXES</b>	
<b>A Committee Biographies</b>	<b>48</b>
<b>B Meeting Agendas</b>	<b>56</b>
<b>C Additional Comments on Directed Radio Frequency Energy</b>	<b>60</b>
<b>D Environmental Chemicals</b>	<b>62</b>

## Acronyms and Abbreviations

3-PBA	3-Phenoxybenzoic acid
3T	3 Tesla
ABIT	Acquired Brain Injury Tool
AChE	acetylcholinesterase
CDC	Centers for Disease Control and Prevention
DoD	Department of Defense
DOS	Department of State
DR2	Disaster Research Response
FDA	Food and Drug Administration
FSO	Foreign Service Officer
GAO	Government Accountability Office
GuLF STUDY	Gulf Long-term Follow-up Study
HHS	Department of Health and Human Services
HIPAA	Health Insurance Portability and Accountability Act
IEEE	Institute of Electrical and Electronics Engineers
IPM	Integrated Pest Management
IRB	Institutional Review Board
MED HART	Department of State, Bureau of Medical Services Health Alert Response Team
MPTP	1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine
MR	magnetic resonance
MRI	magnetic resonance imaging
mTBI	mild traumatic brain injury
NHANES	National Health and Nutrition Examination Survey
NIH	National Institutes of Health
NTP	National Toxicology Program
OCD	obsessive compulsive disorder
OGA	Office of Global Affairs
OP/Ops	organophosphate/organophosphates
PPPD	persistent postural-perceptual dizziness
RF	radio frequency
SARS-CoV-2	severe acute respiratory syndrome coronavirus 2
TMS	transcranial magnetic stimulation
U.S.S.R.	Union of Soviet Socialist Republics

## Preface

An individual assigned to the U.S. Embassy in Cuba was awakened one night at home in Havana in 2016 by severe pain and a sensation of intense pressure in the face, a loud piercing sound in one ear with directional features, and acute disequilibrium and nausea. Symptoms of vestibular and cognitive dysfunction ensued. A handful of other cases involving colleagues with similar features began that year, and others in the next. Few people were aware of these cases until spring 2017. In addition, the mechanisms and origins were mysterious, and for these and other reasons, there was a delay in recognizing an important cluster of unexplained illnesses, and an early failure to investigate them in a concerted, coordinated, rigorous, and interdisciplinary manner.

In some ways, the problem presented here is an age-old one; that is, how to detect and recognize important anomalies or signals, in a complicated, “noisy” background. Public health systems have grappled with this problem for centuries. In the 1990s, the Centers for Disease Control and Prevention (CDC) conducted population-based surveillance for “unexplained death and critical illness” in persons less than 50 years of age, with features suggestive of infectious cause, at four sites in the United States, and found a surprisingly high incidence of 0.5 cases per 100,000 per year (Hajjeh et al., 2002). The most common clinical presentation was neurologic; a known infectious cause was discovered for only a minority of them; and no obvious relationships among cases were uncovered (Nikkari et al., 2002). But the landscape that countries face today in which the cases in question arise, is an even more complicated one. Not only must governments consider a wide variety of evolving natural causes in a rapidly changing world, but also an increasing threat of disease of deliberate human origin, both accidental and purposeful.

The cases of the Department of State (DOS) employees in Cuba and China have attracted much attention. Among the reasons and ramifications, the clinical features were unusual; the circumstances have led to rampant speculation about the cause(s); and numerous studies, along with the charged political setting, have had consequences for international relations.

The committee was asked by DOS to review the cases, their clinical features and management, epidemiologic investigations, and scientific evidence in support of possible causes, and advise on approaches for the investigation of potential future cases. The committee faced a variety of challenges in responding to these requests (see Section 2). In particular, much of the detail and many of the investigations performed by others were not available to it, either because they are classified for reasons of national security or restricted for other reasons (e.g., internal department deliberations, protected health information, etc.). Thus, the committee had only limited amounts and kinds of information. Despite these challenges, the committee arrived at a number of observations and recommendations, after carefully reviewing the information that was available.

First, the committee found a constellation of acute clinical signs and symptoms with directional and location-specific features that was distinctive; to its knowledge, this constellation of clinical features is unlike any disorder in the neurological or general medical literature. From a neurologic standpoint, this combination of distinctive, acute, audio-vestibular symptoms and signs suggests localization of a disturbance to the labyrinth or the vestibulocochlear nerve or its brainstem connections. Yet, not all DOS cases shared these distinctive and acute signs and symptoms. In fact, the cases are highly heterogeneous. Some patients described only a set of nonspecific, chronic signs and symptoms indicative of disruption of vestibular processing and/or cognition and diffuse involvement of forebrain structures and function, raising the possibility of multiple causes or mechanisms among different patients, as well as for the same patient.



Second, after considering the information available to it and a set of possible mechanisms, the committee felt that many of the distinctive and acute signs, symptoms, and observations reported by DOS employees are consistent with the effects of directed, pulsed radio frequency (RF) energy. Studies published in the open literature more than a half century ago and over the subsequent decades by Western and Soviet sources provide circumstantial support for this possible mechanism. Other mechanisms may play reinforcing or additive effects, producing some of the nonspecific, chronic signs and symptoms, such as persistent postural-perceptual dizziness, a functional vestibular disorder, and psychological conditions.

The committee is left with a number of concerns. First, even though it was not in a position to assess or comment on how these DOS cases arose, such as a possible source of directed, pulsed RF energy and the exact circumstances of the putative exposures, the mere consideration of such a scenario raises grave concerns about a world with disinhibited malevolent actors and new tools for causing harm to others, as if the U.S. government does not have its hands full already with naturally occurring threats. Because the committee was not able to assess specific scenarios involving malevolent actors, one strong suggestion is that follow-up studies on this topic be undertaken by subject-matter experts with proper clearance, including those who work outside the U.S. government, with full access to all relevant information. Second, the committee was concerned about the possibility of future new cases among DOS or other U.S. government employees working overseas, either similar or dissimilar to these, and the ability of the U.S. government to recognize and respond to these cases in a coordinated and effective manner. The next event may be even more dispersed in time and place, and even more difficult to recognize quickly. Toward this end, the committee offers a number of observations, best practices, and recommendations for clinical management, surveillance, and a systematic response in anticipation of future health events. These observations and recommendations should be reviewed and acted on now. It is imperative that the United States recognize and quickly respond to future cases with a well-coordinated, multi-disciplinary, science-based investigation and effective interventions. Finally, the committee is concerned about how best to manage the continuing care of those already affected, and how to strengthen the nation's commitment to the health and well-being of those who serve the country overseas. Both of these priorities need and deserve additional attention and resources.

On a personal note, it was an honor and privilege to work with a wonderful committee and staff at the National Academies of Sciences, Engineering, and Medicine. Every person contributed unique and important insights and ideas. Finally, it was humbling to learn of the commitment and sacrifices made by those who work for DOS and the rest of the U.S. government in difficult and challenging circumstances overseas. It would behoove us all to consider how we can provide greater support.

David A. Relman, *Chair*

Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S.  
Government Employees and Their Families at Overseas Embassies

## REFERENCES

- Hajjeh, R. A., D. Relman, P. R. Cieslak, A. N. Sofair, D. Passaro, J. Flood, J. Johnson, J. K. Hacker, W-J Shieh, R. M. Hendry, S. Nikkari, S. Ladd-Wilson, J. Hadler, J. Rainbow, J. W. Tappero, C. W. Woods, L. Conn, S. Reagan, S. Zaki, and B. A. Perkins. 2002. Surveillance for unexplained deaths and critical illnesses due to possibly infectious causes, United States, 1995-1998. *Emerging Infectious Diseases* 8(2):145-153.
- Nikkari, S., F. A. Lopez, P. W. Lepp, P. R. Cieslak, S. Ladd-Wilson, D. Passaro, R. Danila, and D. A. Relman. 2002. Broad-range bacterial detection and the analysis of unexplained death and critical illness. *Emerging Infectious Diseases* 8(2):188-194.

## Summary

In late 2016, U.S. Embassy personnel in Havana, Cuba, began to report the development of an unusual set of symptoms and clinical signs. For some of these patients, their case began with the sudden onset of a loud noise, perceived to have directional features, and accompanied by pain in one or both ears or across a broad region of the head, and in some cases, a sensation of head pressure or vibration, dizziness, followed in some cases by tinnitus, visual problems, vertigo, and cognitive difficulties. Other personnel attached to the U.S. Consulate in Guangzhou, China, reported similar symptoms and signs to varying degrees, beginning in the following year. As of June 2020, many of these personnel continue to suffer from these and/or other health problems. Multiple hypotheses and mechanisms have been proposed to explain these clinical cases, but evidence has been lacking, no hypothesis has been proven, and the circumstances remain unclear. The Department of State (DOS), as part of its effort to inform government employees more effectively about health risks at posts abroad, ascertain potential causes of the illnesses, and determine best medical practices for screening, prevention, and treatment for both short and long-term health problems, asked the National Academies of Sciences, Engineering, and Medicine (the National Academies) to provide independent, expert guidance.

The Standing Committee to Advise the Department of States on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies faced several challenges in assessing these clinical cases, including lack of access to individual-level health and other information, evolving and changing clinical features over time, and a highly heterogeneous population in terms of the timing and type of clinical symptoms and signs, to include those whose symptoms were only acute, only chronic or both. However, the committee was able to identify distinctive clinical features, consider possible causes, evaluate plausible mechanisms and rehabilitation efforts, and offer recommendations for future planning and responses.

## CLINICAL FEATURES

A distinct set of unusual clinical manifestations occurred abruptly in some individuals at the onset of their illness, and the illness became chronic and debilitating for some, but not for all. The most distinctive clinical aspects of the illnesses were the nature of the onset and the initial features: the sudden onset of a perceived loud sound, a sensation of intense pressure or vibration in the head, and pain in the ear or more diffusely in the head. Most individuals reported that the sound or these other sensations seemed to originate from a particular direction and were perceived only when the individual was in a specific physical location. Some also reported sudden onset of tinnitus, hearing loss, dizziness, unsteady gait, and visual disturbances. From a neurologic standpoint, this combination of distinctive, acute, auditory-vestibular symptoms suggests an effect localized to the labyrinth or VIII cranial nerve or its brainstem connections.

Chronic symptoms suffered by many of those affected suggested problems with vestibular processing and cognition, as well as insomnia and headache; these manifestations are more consistent with diffuse involvement of forebrain structures and function, such as cerebral cortex or limbic structures. However, no consistent picture of brain injury emerged from laboratory-based tests of vestibular function. It is possible that these subsequent, more persistent symptoms were caused by sequelae of the same initial insult or that they occurred secondarily as an accommodative response. For those without reports of an acute initial phase, the symptoms

could be from a separate cause or a similar exposure that resulted more exclusively in forebrain dysfunction.

## PLAUSIBLE MECHANISMS

The committee found the unusual presentation of acute, directional or location-specific early phase signs, symptoms and observations reported by DOS employees to be consistent with the effects of directed, pulsed radio frequency (RF) energy. Many of the chronic, nonspecific symptoms are also consistent with known RF effects, such as dizziness, headache, fatigue, nausea, anxiety, cognitive deficits, and memory loss. Patient clinical heterogeneity could be due to variability of exposure dosage conditions, differences in interpretation of non-physiological vestibular stimuli, and anatomical differences that could influence individual exposure and/or response.

The committee also considered chemical exposures, infectious diseases and psychological issues as potential causes or aggravating factors. Although some reports suggested that exposure to organophosphates (OP) and/or pyrethroids from insecticide spraying in Havana could be a cause or contributing factor, the committee concluded that this mechanism was not likely because there was no convincing evidence of acute high-level exposures and the clinical histories of affected U.S. Embassy personnel were not consistent with acute OP poisoning. However, as insecticides can increase the risk or severity of adverse outcomes after exposure to a wide variety of physical or psychosocial stressors, the committee cannot rule out subacute or chronic OP and/or pyrethroid exposures as a possible contributing factor to nonspecific chronic symptoms.

Infectious agents known to be prevalent in Cuba at the time of the U.S. Embassy cases and capable of causing neurological manifestations most prominently include Zika, which was epidemic in Cuba in 2016-2017. However, after reviewing the medical and public health literature, the committee found it highly unlikely that Zika was the cause of the constellation of signs and symptoms reported among DOS personnel.

The acute initial, sudden-onset, distinctive, and unusual symptoms and signs are difficult to ascribe to psychological and social factors. However, the significant variability and clinical heterogeneity of the illnesses affecting DOS personnel leave open the possibility of multiple causal factors including psychological and social factors. These factors could exacerbate other causes of illness and cannot be ruled out as contributing to some of the cases, especially some of the chronic symptoms or later in the course of illness in some cases. Finally, the committee concurred with the diagnosis of persistent postural-perceptual dizziness (PPPD), a functional (not psychiatric) vestibular disorder that may be triggered by vestibular, neurologic, other medical and psychological conditions and may explain some chronic signs and symptoms in some patients.

Overall, directed pulsed RF energy, especially in those with the distinct early manifestations, appears to be the most plausible mechanism in explaining these cases among those that the committee considered, along with PPPD as a secondary reinforcing mechanism, as well as the possible additive effects of psychological conditions. The committee cannot rule out other possible mechanisms and considers it likely that a multiplicity of factors explains some cases and the differences between others. In particular, the committee could not be certain that the individuals with only the chronic set of signs and symptoms suffered from the same cause(s) and etiologic mechanisms as those who reported the initial, sudden onset set of signs and symptoms.

## REHABILITATION

The committee recommends early evaluation and treatment, a supportive environment, and an interdisciplinary approach for rehabilitation of chronic neurological conditions. Without information on patient-specific treatment approaches and responses, it was difficult for the committee to develop recommendations on specific neurologic rehabilitation alternatives. For those with chronic vestibular symptoms, a diagnosis of PPPD offers a potential avenue for rehabilitative interventions.

## FUTURE PREPAREDNESS

Part of the committee's task was to provide advice in anticipation of future threats to DOS personnel and their families' well-being. To that end, the committee proposes a number of recommendations in order to enhance future responses.

**Recommendation 1. The Department of State should expand its collection of baseline and longitudinal data and biological specimens from all personnel prior to and during overseas assignments.**

The committee believes that there should be routine data collection for all DOS employees on foreign assignments, including collection of whole blood, plasma, and urine, as well as general medical and neurological examinations, and local environmental assessments. The Acquired Brain Injury Tool (ABIT) is a clinical assessment tool currently used pre- and post-deployment to inventory the same neurological, vestibular and auditory symptoms that were identified in DOS personnel in Cuba. However, given that the nature of future events is unknown, it would be wise to revise it and include symptoms beyond those encountered in Cuba and China.

**Recommendation 2. The Department of State, with support from the U.S. government, should establish plans and protocols now to enable comprehensive, expeditious public health and research investigations in the future, should a cluster of new cases warrant investigation.**

The committee recommends that a response capability be prepared and authorized in advance of the next potential set of cases, so that the necessary collection of information for a proper public health investigation of U.S. embassy employees can be undertaken in a timely fashion and made available immediately.

**Recommendation 3. Following the identification of a possible new case cluster, the Department of State should ensure the collection of data critical for an effective investigation.**

The committee suggests that DOS utilize an expert panel described in Section 6 to provide advice on the collection of routine medical data. In addition to the collection of data pertaining to individual diplomats, it is critical that additional public health and epidemiological surveillance data be obtained to provide the temporal and geographic context for the health presentation of individuals.



**Recommendation 3-A. If research or assessments support the possibility of radio frequency (RF) energy as a cause of illness experienced by some of its employees, the Department of State should train and equip employees with the capability to measure and characterize their exposure to RF energy in real time should the need arise in the future.**

**Recommendation 3-B. The Department of State should develop a systematic approach for toxicological diagnoses, and a protocol that supports this approach.**

**Recommendation 4. The Department of State, with support from the U.S. government, should provide for appropriate personnel to identify public health emergencies and activate the necessary response.**

DOS should consider a change in policy so that it enables structured medical investigations of affected individuals in a manner that does not preclude, but is separate from private medical care. The National Institutes of Health Disaster Research Response (DR2) Program may serve as a valuable model for a coordinated system-wide research response to public health emergencies. In addition, to facilitate early identification of health threats to Embassy personnel, the committee suggests an expanded role for health attachés.

## Section 1

### Introduction and Charge to the Committee

In late 2016, U.S. Embassy personnel in Havana, Cuba, began to report the development of an unusual set of symptoms and clinical signs. For some of these patients, their case began with the sudden onset of a loud noise, perceived to have directional features, and accompanied by pain in one or both ears or across a broad region of the head, and in some cases, a sensation of head pressure or vibration, dizziness, followed in some cases by tinnitus, visual problems, vertigo, and cognitive difficulties. Other personnel attached to the U.S. Consulate in Guangzhou, China, reported similar symptoms and signs to varying degrees, beginning in the following year. As of June 2020, many of these personnel continue to suffer from these and/or other health problems. Multiple hypotheses and mechanisms have been proposed to explain these clinical cases, but evidence has been lacking, no hypothesis has been proven, and the circumstances remain unclear.

The Department of State (DOS), as part of its effort to inform government employees more effectively about health risks at posts abroad, ascertain potential causes of the illnesses, and determine best medical practices for screening, prevention, and treatment for both short and long-term health problems, asked the National Academies of Sciences, Engineering, and Medicine (the National Academies) to provide independent, expert guidance.

The task of the Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies, detailed in Box 1, included provision of advice to DOS on best practices in their approach to current patients and prevention or mitigation of potential future incidents. The committee's task was not to "solve" the mystery surrounding what caused the symptoms experienced by personnel in Cuba and China, but it did include the evaluation of proposed plausible mechanisms. Given the limited time available to the committee and the unavailability of relevant, detailed information about individual patients, the committee was not able to accomplish everything in the broad Statement of Task; however, it was able to address a number of critical issues.

The committee faced several challenges in assessing these clinical cases. Many of these challenges relate to the extreme variability in the cases. First, because of federal rules for protection of health and other information, the committee was not privy to health or other personal information about individuals, other than that which was voluntarily provided to the committee directly by a small number of affected DOS employees. Therefore, the committee could not link anonymized data about specific individuals from different clinical providers or clinical investigators. The Centers for Disease Control and Prevention (CDC) is the only U.S. federal agency with the authority to link health data from different sources about individual patients, and CDC did in fact undertake an investigation of these cases with the goal of establishing a case definition; however, CDC did not become involved until one year after the earliest events and only reviewed records rather than interviewing all of the affected individuals. The committee was not afforded access to CDC's final report of this investigation until near the end of the committee's term. Thus, the committee was blind to the different clinical tools and assessments used by different clinical providers or clinical investigators on the same patient.

A second challenge was that cases evolved over time and patients were evaluated by different clinicians and investigators after widely varying amounts of time following the onset of

their symptoms and signs, including up to several years later. Thus, the evolving and changing clinical features of these cases and the non-uniform timing of the clinical investigations created a second source of variability. Third, the patient population was highly heterogeneous in the timing and location of their overseas assignments; their roles and assignments while overseas; their ages, past medical and career histories and other demographic features; and in their clinical symptoms and signs. In general, the committee did not have access to individual-level information except for several instances where affected DOS employees agreed to tell their story before the committee. Furthermore, when viewed on their own, a number of these clinical signs and symptoms are nonspecific, i.e., they might be experienced by persons suffering from a variety of conditions. Despite these challenges, the committee did its best to collect, extract, and evaluate some shared or distinctive clinical features of these cases, evaluate some plausible mechanisms and efforts to treat some of the patients, and then offer recommendations for future management of these and potential new cases.

This report is organized into five subsequent sections:

- Section 2: Methods and Data
- Section 3: Clinical Features
- Section 4: Plausible Mechanisms
  - Directed Radio Frequency Energy
  - Chemicals
  - Infectious Agents
  - Psychological and Social Factors
- Section 5: Acute Treatment and Rehabilitation
- Section 6: Looking to the Future/Recommendations

Sections 3-5 are each organized according to Sources of Information; Assessment and Findings; Summary; and References.

**BOX 1**  
**Statement of Task**

To facilitate the Department of State (DOS) in implementing one of its responsibilities to protect U.S. government employees and their family members overseas, the Health and Medicine Division of the National Academies of Sciences, Engineering, and Medicine will form a Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies. This committee will collaborate with the DOS Bureau of Medical Services on best practices including but not limited to health monitoring, medical interventions, risk assessment, and exposure mitigation for overseas locations that may present a higher risk of adverse health effects. The standing committee will evaluate current DOS practices for pre- and post-assignment health screening, provide expertise on potential epidemiologic studies, help with characterizing and understanding the current cases of potential acoustic trauma, and develop a better understanding of possible causes of these cases and approaches for future incidents whether of an apparent acoustic nature or a different environmental or clinical presentation. The standing committee will also keep DOS abreast of any emerging concerns, interventions, and protective measures as these come available. The committee will organize ongoing

discussions, take on specific tasks and possibly issue classified and unclassified reports and recommendations on a variety of topics of importance to DOS in regards to health effects from potential exposures overseas.

The standing committee will provide a forum for discussion of scientific, technical, and social issues relevant to effective health management and protection of staff and family members assigned to overseas locations. The committee will consider relevant scientific, technical, and policy issues including but not limited to:

- Review of the current situation, to include discussions of epidemiologic investigations, case definitions, study methods, controls, and alternative hypotheses;
- Review the active research agenda, including defining what types of information ought to be collected and archived against possible future needs, and any potential additional studies needed;
- Assist in the optimization and deployment of screening protocols and assessment of treatment options, to include a review of currently available screening devices and technologies, appropriate level of baseline testing for a large number of personnel and policy needs;
- Review data, findings and conclusions generated by and for the U.S. government;
- Review scientific evidence of possible causes and approaches to addressing potential future incidents of unexplained clusters of medical symptoms;
- Determine the need for collection of relevant environmental data (e.g., biologic, acoustic, radiologic, chemical, toxicological) that might be useful in current and future situations; and
- Provide guidance on determining a clinical case definition.

## Section 2

### Methods and Data

The National Academies of Sciences, Engineering, and Medicine assembled a 19-member committee with expertise in clinical medicine to include neurology, neuro-ophthalmology, audiologic, and vestibular medicine, psychiatry, infectious diseases, and rehabilitative medicine, along with experts in epidemiology, environmental science and engineering, toxicology, neurobiology, neuroradiology, health effects of electromagnetic radiation and microwaves, exposure and risk assessment, and health monitoring. The committee members' biographical sketches are included in Appendix A.

The committee held two in-person meetings (December 18-19, 2019; February 24-25, 2020) and one virtual meeting (May 11-13, 2020); each of them included public sessions with external experts (see Appendix B). The committee reviewed the clinical data about the U.S. Embassy personnel published by clinical teams from the University of Miami (referred to here as "Miami") (Hoffer et al., 2019) and the University of Pennsylvania (referred to here as "Penn") (Swanson et al., 2018), as well as information about the U.S. Embassy personnel presented by Miami and Penn, as well as a clinical team from the National Institutes of Health (NIH) during the committee's meetings. During the February 2020 meeting, the committee heard from experts on the health effects of exposures to chemicals and to directed radio frequency (RF) energy. During the May 2020 meeting, the committee heard from three experts in the fields of mild traumatic brain injury (mTBI), and vestibular and cognitive rehabilitation.

During a closed session at each of the two in-person meetings, the committee met with former U.S. Embassy personnel from Cuba and China, who suffered from some of the clinical manifestations that are the subject of this report, and who volunteered to speak with the committee about their own cases. In order to protect their privacy and their personal health information, the committee omitted details from this report that might enable identification of these individuals.

The committee also reviewed studies of patients associated with the Canadian Embassy in Havana, published and presented by investigators from Dalhousie University in Halifax, Nova Scotia (referred to here as "Dalhousie") (Friedman et al., 2019).

The committee acquired access to the final report of the CUBA Unexplained Events Investigation conducted by the Centers for Disease Control and Prevention (CDC) on the U.S. Embassy Havana patients near the end of this study.<sup>2</sup> It is referred to here as "the CDC Report."

Throughout the course of this study, the committee received information from DOS that was germane to its tasks, and had multiple opportunities to speak directly with current DOS employees within the Bureau of Medical Services. For this, the committee is appreciative. The committee approached its reporting task by determining the topics and issues on which it felt sufficiently informed to be able to offer findings and conclusions, given the information provided to it. The committee then considered recommendations, supported by these findings, which might assist DOS in understanding and managing these cases, as well as in managing potential future health events. The committee also reviewed the information through the lens of

---

<sup>2</sup> Centers for Disease Control and Prevention. 2019. Cuba unexplained events investigation - final report. Received by the committee on April 28, 2020.



established procedures for investigating clusters of unknown health events (see Box 2). These procedures, among other things, emphasize the importance of standardized data collection.

It is the committee's view that the information made available to the committee on DOS patients from China is too sparse and fragmentary to be able to draw any substantive conclusions about these cases and their relationship to the cases from U.S. Embassy Havana. The committee's report therefore focuses on the personnel associated with the U.S. Embassy in Havana.

Of note, from a systems perspective, each agency and organization that has reviewed these cases during the past several years has had available to them different sets of data. There are myriad reasons, including different institutional responsibilities, approaches, investigative tools, timing of investigation, and access to classified information. Although the committee did not have the benefit of data and investigative tools that were available to others, the study may have benefitted from information that either was not obtained by others or was not available at the time that other investigations took place. As a result, experienced investigators from different organizations logically may reach different conclusions based on their own data sources and limitations.

#### BOX 2

##### Steps for Investigating Clusters of Health Events (CDC, 1990)

Stage 1 – Initial contact and response—collect information from the person or groups first reporting the issue

Stage 2 – Assessment—determine the likelihood that cases of illness or injury are above expected numbers or rates; verify the diagnosis or determine biologic plausibility; define the characteristics

Stage 3 – Feasibility study—examine the potential for an epidemiologic study to link the health event and a putative exposure

Stage 4 – Etiologic investigation—determine the potential disease or injury exposure relationship

#### REFERENCES

- CDC (Centers for Disease Control and Prevention). 1990. Guidelines for investigating clusters of health events. *Morbidity and Mortality Weekly Report* 29(RR-11):1-16.
- Friedman, A., C. Calkin, and C. Bowen. 2019. Havana syndrome: Neuroanatomical and neurofunctional assessment in acquired brain injury due to unknown etiology. <https://www.scribd.com/document/426438895/Etude-du-Centre-de-traitement-des-lesions-cerebrales-de-l-Universite-de-Dalhousie#download> (accessed July 7, 2020).
- Hoffer, M. E., B. E. Levin, H. Snapp, J. Buskirk, and C. Balaban. 2019. Acute findings in an acquired neurosensory dysfunction. *Laryngoscope Investigative Otolaryngology* 4(1):124-131.
- Swanson, R. L., 2nd, S. Hampton, J. Green-McKenzie, R. Diaz-Arrastia, M. S. Grady, R. Verma, R. Biester, D. Duda, R. L. Wolf, and D. H. Smith. 2018. Neurological manifestations among US government personnel reporting directional audible and sensory phenomena in Havana, Cuba. *JAMA* 319(11):1125-1133.

## Section 3

### Clinical Features

#### SOURCES OF INFORMATION

The clinical investigators presented data to the committee as aggregated summaries of patients' histories, physical examination findings, and results of laboratory testing and neuroimaging. These data were obtained from well-established methods of clinical assessment (Friedman et al., 2019; Hoffer et al., 2019; Swanson et al., 2018), as well as procedures that were investigative (i.e., experimental) in nature (Balaban et al., 2016; Verma et al., 2019). Experimental procedures included novel interpretations of results derived from well-established procedures (Friedman et al., 2019; Hoffer et al., 2019) and results obtained from newly developed, but not yet standardized, technologies (Balaban et al., 2016; Friedman et al., 2019; Verma et al., 2019). Individual patient-level data were not provided to the committee. DOS and the four clinical teams (Miami, Penn, National Institutes of Health [NIH], and Dalhousie) appropriately cited patient privacy and diplomatic and other security concerns in limiting data shared with the committee to the aggregate summaries only. The NIH team provided detailed multi-disciplinary clinical diagnoses of all patients that they evaluated, though this information, too, was provided to the committee in summary format. This made it impossible to link specific symptom constellations, physical examination results, and laboratory or imaging test findings within and between affected individuals for diagnostic purposes.

The committee was afforded an opportunity to speak directly with eight patients associated with U.S. Embassy Havana or with the U.S. Embassy or Consulates in China.

The CDC Report indicated substantial overlap in the populations of Havana patients included in the Miami, Penn, and NIH summative data. In fact, it was difficult for the committee to determine to what extent some individual patients may have been reported two or more times and whether the patients interviewed were among those included in the summative data from these three clinical sites. Importantly, the evaluations included in the summative data were generally separated from the original case events and from each other by considerable periods of time. Therefore, it was difficult to know whether differences in the reported signs and symptoms were due to changes from time of onset to the time of various evaluations, or because of the different evaluation procedures employed at the different sites, or because different subsets of patients were included in the different summaries.

#### ASSESSMENT AND FINDINGS

##### Clinical Features of Personnel Who Spent Time in Havana

The committee compiled signs and symptoms reported by DOS employees that spent time in Havana, based on information provided by the four clinical evaluation sites (Miami, Penn, NIH, Dalhousie) in presentations to the committee or in publications, as well as the signs and symptoms of affected employees interviewed by the committee in person. The committee included the sparse and fragmentary information on the China and Canadian patients here for the sake of a few comments.

The committee finds that the most distinctive and specific clinical features of these individuals occurred acutely at, or soon after the time of onset of their illness. In contrast, the chronic features that persisted for weeks, months, or years after the initial onset (in those individuals who reported an acute distinctive phase) were less specific to these DOS personnel and more common among general populations of patients with a variety of neurological or systemic conditions. The committee reasoned that the acute, more distinctive clinical features would be more informative about the possible cause(s) of the overall illnesses, rather than the chronic, less specific features.

The most common and distinctive features of the initial onset and acute phase of the illness in Havana personnel were the sudden onset of a perceived loud sound, sometimes described as screeching, chirping, clicking, or piercing, a sensation of intense pressure or vibration in the head, and pain in the ear or more diffusely in the head. Most individuals reported that the sound or these other sensations seemed to originate from a particular direction or that they perceived them only in certain physical locations. Individuals interviewed by the committee described alleviation of the symptoms by moving from their initial location to a different one, e.g., into a different room of the building in which they were located. According to data from Miami, 25 of 25 individuals perceived a loud sound, and according to data from Penn, 28 of 35 individuals from Havana, and 12 of 12 from China perceived a loud sound. Of the 35 Havana individuals assessed at Penn, 16 reported a sensation of pressure or vibration in the head and 18 described the sound or pressure as directional or as restricted spatially in their immediate environment. Variable numbers of individuals reported the accompanying sudden onset of tinnitus (8 of 25 reported this at Miami and 6 of 21 at Penn), ear pain (7 of 25 at Miami and 7 of 21 at Penn), hearing loss (8 of 25 at Miami and 9 of 21 at Penn), dizziness, unsteady gait (4 of 21 at Penn), and visual disturbances (14 of 21 at Penn).<sup>3</sup> Importantly, the committee finds this constellation of acute symptoms with directional and location-specific features to be very unusual, and to the best of its knowledge, unlike any disorder reported in the neurological or general medical literature.

Some of the acute signs and symptoms persisted or recurred and became chronic in some individuals, including dizziness (23 of 25 at the time they were examined at Miami and 13 of 21 at Penn), fatigue (10 of 21 at Penn), impaired balance (numbers not available), headache (6 of 25 at Miami and 16 of 21 at Penn), impaired concentration (5 of 8 at Miami and 8 of 21 at Penn) and memory (5 of 8 at Miami and 11 of 21 at Penn), depression (numbers not available), and insomnia (18 of 21 at Penn). These latter symptoms alone do not inform a specific etiologic diagnosis and can be due to a wide variety of common disorders (including viral and other inflammatory conditions, persistent postural-perceptual dizziness, chronic fatigue syndrome, traumatic brain injury, posttraumatic stress disorder, depression, and others). Most of the eight individuals that the committee interviewed described both early, acute onset and chronic clinical features, and continued to be debilitated.

The summary descriptions available to the committee of cases involving Canadian Embassy personnel from Havana failed to mention the perception of a loud sound, sensation of intense pressure or vibration, or ear pain, but did include impaired balance, headache, vertigo, tinnitus, and some of the same chronic clinical features as the U.S. Embassy personnel. The committee did not have sufficient information about U.S. Embassy personnel from China to be able to assess overall similarities or dissimilarities with cases from Havana.

---

<sup>3</sup> These numbers were extracted by the committee from publications and presentations by the clinical investigators, and not from the patient clinical records or from direct examination of the patients by the committee.

One problem with all of the data presented was that it lacked an appropriate control group (i.e., individuals who were present in the same environment as the U.S. Embassy employees who reported the acute phase of the illness, but were not exposed to whatever caused those distinctive signs and symptoms). It is noteworthy that the Canadian Embassy employees shared much of the environment of the U.S. Embassy employees, but generally lacked the acute signs and symptoms. Hence, it is possible that other exposures (viral illness, toxic chemicals, etc.) may have caused the chronic signs and symptoms shared by both the U.S. and Canadian personnel, while the acute signs and symptoms limited to the U.S. Embassy employees may have had a different cause.

The committee notes that the CDC Report also identified a biphasic onset of symptoms, with a set of early, sudden-onset symptoms and a set of later, more chronic, and less specific symptoms. The CDC Report defined a “presumptive” case as having components of each set.<sup>4</sup> Out of 95 records that CDC reviewed, they found 15 who met their case definition, along with 31 other “possible” cases. Out of the 15, over 2 years after the initial symptoms, six were still undergoing therapy with four unable to work and two needing modifications to work. Unlike CDC, the committee did not have the ability to link disparate findings from different clinical sites and times to the same individual. However, it is the committee’s impression that only a subset of individuals who suffered from the early set of signs and symptoms, also suffered from the later set of signs and symptoms. Conversely, only a subset of individuals who reported suffering from the late set of generally more common signs and symptoms, also described the more distinctive early set and in particular, the sudden onset of a directional or location-specific loud noise, pressure or pain. Because of these various aspects of case heterogeneity, the committee found it difficult to know with certainty that all cases were due to the same cause(s), and in particular, whether the individuals with only the chronic set of signs and symptoms suffered from the same cause(s) and etiologic mechanisms as those who reported the initial, sudden onset set of signs and symptoms.

### **Laboratory Test Results and Physical Examination Findings Reported for Embassy Personnel**

DOS personnel underwent physical examinations and different tests at different study sites, at different times during the course of their illness. The committee did not have access to primary reports or complete data. Nonetheless, it sought to identify and summarize pertinent test results and exam findings for which available data were adequate.

#### *Vestibular and Balance Assessments*

Patient questionnaires, physical examinations, office-based tests of balance performance, and vestibular and oculomotor laboratory tests were used to evaluate patients. The clinical teams from Miami and Penn selected tests based on each patient’s symptoms. Thus, patients did not undergo a consistent set of diagnostic evaluations at either site. Clinicians at NIH appeared to use a more consistent approach and set of test procedures.

Self-report questionnaires (e.g., Dizziness Handicap Inventory) and tests of balance performance (e.g., dynamic posturography) showed high rates of impairment and poor performance. The clinicians involved in these assessments interpreted these data as evidence of inner ear or brain injury (Swanson et al., 2018). However, self-report questionnaires and tests of

---

<sup>4</sup> The CDC case definition for a **presumptive case** required at least one of following in the initial phase (head pressure, disorientation, nausea, headache, vestibular disturbances, auditory symptoms, vision changes) and at least one of the following in a separate secondary phase (vestibular disturbances, cognitive deficits).

balance performance cannot be used properly to make specific diagnoses, as abnormal results may arise from structural, functional, or psychiatric disorders alone or in combination. Thus, these data indicate a high level of impairment in many patients at the time of testing, but do not provide any information about potential causative agents or specific mechanisms of injury.

Vestibular laboratory tests such as the video head impulse test, caloric test, vestibular evoked myogenic potentials, rotary chair test, and oculomotor examinations can provide information on the structural integrity of peripheral (inner ear) and central (brain) vestibular and oculomotor pathways. Test procedures vary across laboratories and several tests require cooperation and volitional effort on the part of patients to yield meaningful and consistent results. Consequently, findings from one center may not be directly comparable to findings from another center in the absence of descriptions of test procedures employed and thresholds for reporting normal versus abnormal results. Data published (Balaban et al., 2020; Hoffer et al., 2019) and presented by the clinical team at Miami were derived from a small number of established laboratory tests plus a battery of new assessment tools (i.e., experimental tests) developed by that group (Balaban et al., 2016). Data published (Swanson et al., 2018) and presented by the clinical team at Penn were derived predominantly from office-based tests. Only a portion of patients were evaluated with standard vestibular laboratory tests and only a portion of those results were published or presented. The Dalhousie team also used a small number of established vestibular tests (Friedman et al., 2019) but only examined Canadian patients. The NIH team employed an extensive battery of established tests, though their assessments were generally conducted later, from months to over a year, in the course of patients' illnesses and thus were less informative about potential early deficits. Some results were inconsistent across centers, although perhaps in part because they studied different individuals. For example, the Miami group reported high rates of absent or reduced-amplitude vestibular evoked myogenic potentials (Hoffer et al., 2019), whereas the Dalhousie group reported higher than normal mean amplitudes on those tests (Friedman et al., 2019).

The committee concluded that the aggregate data derived from the subset of well-established clinical laboratory diagnostic tests presented by the four clinical groups performed weeks, months, or years after the initial onset did not identify a common pattern of structural injuries to the labyrinths or brains of patients that could explain the reported vestibular symptoms. In the absence of patient-level data, the committee could not determine with certainty if any reported abnormalities coincided with key aspects of clinical histories for individual patients.

### *Neuropsychological and Psychological Assessments*

Patients underwent neuropsychological testing at Penn and NIH in various cases, weeks, months, or years after symptom onset. The Penn team presented aggregate results in a non-standard manner (i.e., the total number of patients with abnormal scores on each subtest of the test battery that they administered). The NIH team presented results in a more standardized clinical fashion, though still in aggregate. Neurobehavioral and cognitive evaluations in these situations are quite challenging. Standard assumptions and fact-finding methods used in normal clinical settings may be misleading, raising validity concerns (Lees-Haley, 1995). The committee concluded that no distinct pattern of clinically diagnosable cognitive deficits could be discerned from these data. A more comprehensive and uniform assessment approach to the entire group of patients earlier in the course of illness may have provided a better opportunity with regard to diagnosis and treatment issues.



The NIH team also presented aggregate data from psychological testing. The results showed psychological distress in some patients in a pattern that may be seen in those suffering from a variety of chronic medical conditions or somatic symptom disorders. These results indicated an increased burden of illness in patients with chronic symptoms but offered no insights into an initial cause.

### *Imaging Studies*

The committee reviewed the radiological studies from Penn and Dalhousie. The Penn group found essentially normal conventional structural magnetic resonance imaging (MRI) results months to years after initial symptoms. They subsequently reported that among 40 Havana patients compared to 48 healthy controls, there were small group differences in the average brain volumes in specific lobes, a decrease in mean diffusivity in the midline inferior cerebellum, and differences in functional connectivity in auditory and visuospatial networks (Verma et al., 2019). Difficulties in replicating results are common in studies of small patient groups using MR measures with low signal to noise ratios, and which involve a number of computational steps and algorithms that are known to perform imperfectly (based on frequent failures to replicate) (De Santis et al., 2014; Jonathan et al., 2007; Landman et al., 2007). Generally, studies of this type require a replication cohort (which was not available) to determine if the findings are reliable. The committee was not provided the results of efforts to correlate imaging findings to clinical findings, and most subjects examined did not show the pattern of imaging findings reported in the average, which further diminish the clinical value of the reported findings. Investigators at the NIH performed imaging on a small number of the Havana cohort at even later dates (i.e. years in some cases and did not find any differences from normal subjects, but their studies are ongoing).

The Dalhousie group also reported normal structural MRI findings, and reported changes in diffusion tensor imaging of the white matter tracts in the posterior part of the corpus callosum and the adjacent part of the fornix (Friedman et al., 2019). These types of changes are subject to the same caveats as the Penn findings (and a lack of congruity between the two studies is noted). However, the Dalhousie patients may not have had the same clinical disorder as DOS employees, as noted above.

In summary, the committee felt that none of the imaging studies performed so far were sufficient to serve as a basis for a case definition or for management of individual subjects.

### *General Comments About the Patient Evaluations*

The committee finds that as a routine, general approach to cases with neurological manifestations, patient evaluations should be performed as soon as possible after onset and should include a complete neurological examination, followed by a standard whole brain MR scan, with and without contrast, preferably performed at 3T, with diffusion weighted imaging and a T2\* sensitive sequence (susceptibility weighted imaging is preferred) to detect microbleeds. If the case involves auditory or vestibular symptoms, fine cuts might be added through the inner ear; for visual symptoms, fine cuts through the orbit; and for cases with somatosensory or motor phenomena, fine cuts through the cervical and thoracic spinal cord. Functional MRI is not well-suited to single subject studies. Similarly, diffusion tensor imaging for tractography is not reliable as an indicator of abnormalities in individuals.

In neuro-otology, there is no consensus about a standard test battery for auditory or vestibular symptoms. Evoked potentials are fairly simple, objective measures of slowing of

conduction in these sensory systems, due to a variety of causes, but are not uniformly accepted as a standard for evaluation.

Additional testing should be undertaken as clinically indicated. Blood should be collected as soon as possible after the onset of symptoms, and both plasma and whole blood frozen and stored for future toxicological, infectious, and other appropriate evaluations. All of these evaluations are routinely available in any modern large hospital, but may require plans in advance for referring Embassy personnel to such a facility as soon as possible after onset.

## SUMMARY

The most distinctive clinical aspects of the illnesses described in DOS Havana personnel are the nature of the onset and the initial features: the sudden onset of a perceived loud sound, a sensation of intense pressure or vibration in the head, and pain in the ear or more diffusely in the head. Most individuals reported that the sound or these other sensations seemed to originate from a particular direction and that they were perceived only when the individual was in a specific physical location. Different numbers of individuals also reported sudden onset of tinnitus, hearing loss, dizziness, unsteady gait and visual disturbances.

From a neurologic standpoint, this combination of distinctive, acute auditory-vestibular symptoms suggests an effect localized to the labyrinth or VIII cranial nerve or its brainstem connections. The committee found this constellation of acute symptoms with directional and location-specific features to be very unusual and, to the best of the committee's knowledge, unlike those associated with any disorder reported in the neurological or general medical literature. The sudden onset and immediate amelioration with change in location makes an infectious or toxic cause less likely, while the repeated testimony that the symptoms were experienced only in specific physical locations near windows or as originating or emanating from a particular direction raised the possibility that they were due to some physical force that could penetrate windows but not walls. As such, we considered in detail the possibility that these acute symptoms may have been caused by directed RF energy, as well as toxic, infectious, and psychological processes.

The more chronic (later phase) problems suffered by Havana personnel included mainly vestibular processing and cognitive problems as well as insomnia and headache. From a neurologic standpoint, these cognitive symptoms and insomnia are more consistent with diffuse involvement of forebrain structures and function, such as cerebral cortex or limbic structures. While these chronic symptoms are common complaints, it is quite possible that in those personnel with the dramatic initial phase auditory and vestibular symptoms, these subsequent, more persistent symptoms were caused by sequelae of the same initial insult or that they occurred secondarily as an accommodative response. In those personnel without reports of an acute initial phase, these chronic symptoms could suggest an exposure or cause distinct from those with an initial phase, or a similar exposure that resulted more exclusively in forebrain dysfunction.

A key problem in the committee's assessment was the lack of information collected in a systematic fashion from every affected individual from the initial onset of the first case, as well as from control individuals. By relying on routine clinical evaluation and management of these individuals within the U.S. commercial health care system, significant information was lost due to delays, differences in insurance coverage and consequent differences in clinical work-up of each case, and lack of a standardized approach. The information was also firewalled in distinct silos by Health Insurance Portability and Accountability (HIPAA) regulations, making it almost

impossible to put together a coherent picture. CDC is currently the only organization that has the ability to penetrate these firewalls, but they were not involved until many months after the primary investigations by Miami, Penn, and NIH. A plan that solves this issue would be of great benefit to DOS and its employees. Baseline visual, auditory, and vestibular data from each individual would have helped as well, given that deficits in these systems can often occur with aging.

## REFERENCES

- Balaban, C., M. E. Hoffer, M. Szczupak, H. Snapp, J. Crawford, S. Murphy, K. Marshall, C. Pelusso, S. Knowles, and A. Kiderman. 2016. Oculomotor, vestibular, and reaction time tests in mild traumatic brain injury. *PLoS One* 11(9):e0162168.
- Balaban, C. D., M. Szczupak, A. Kiderman, B. E. Levin, and M. E. Hoffer. 2020. Distinctive convergence eye movements in an acquired neurosensory dysfunction. *Frontiers in Neurology* 11:469.
- De Santis, S., M. Drakesmith, S. Bells, Y. Assaf, and D. K. Jones. 2014. Why diffusion tensor MRI does well only some of the time: Variance and covariance of white matter tissue microstructure attributes in the living human brain. *NeuroImage* 89(100):35-44.
- Friedman, A., C. Calkin, and C. Bowen. 2019. Havana syndrome: Neuroanatomical and neurofunctional assessment in acquired brain injury due to unknown etiology. <https://www.scribd.com/document/426438895/Etude-du-Centre-de-traitement-des-lesions-cerebrales-de-l-Universite-de-Dalhousie#download> (accessed July 7, 2020).
- Hoffer, M. E., B. E. Levin, H. Snapp, J. Buskirk, and C. Balaban. 2019. Acute findings in an acquired neurosensory dysfunction. *Laryngoscope Investigative Otolaryngology* 4(1):124-131.
- Jonathan, A. D., B. S. Farrell, A. Bennett, B. A. Landman, C. K. Jones, S. A. Smith, J. L. Prince, P. C. M. van Zijl, and S. Mori. 2007. Effects of signal-to-noise ratio on the accuracy and reproducibility of diffusion tensor imaging-derived fractional anisotropy, mean diffusivity, and principal eigenvector measurements at 1.5T. *Journal of Magnetic Resonance Imaging* 26(3):756-767.
- Landman, B. A., J. A. Farrell, C. K. Jones, S. A. Smith, J. L. Prince, and S. Mori. 2007. Effects of diffusion weighting schemes on the reproducibility of DTI-derived fractional anisotropy, mean diffusivity, and principal eigenvector measurements at 1.5T. *NeuroImage* 36(4):1123-1138.
- Lees-Haley, P. R. 1995. Neurobehavioral assessment in toxic injury evaluations. *Toxicology Letters* 82-83:197-202.
- Swanson, R. L., 2nd, S. Hampton, J. Green-McKenzie, R. Diaz-Arrastia, M. S. Grady, R. Verma, R. Biester, D. Duda, R. L. Wolf, and D. H. Smith. 2018. Neurological manifestations among US government personnel reporting directional audible and sensory phenomena in Havana, Cuba. *JAMA* 319(11):1125-1133.
- Verma, R., R. L. Swanson, D. Parker, A. A. Ould Ismail, R. T. Shinohara, J. A. Alappatt, J. Doshi, C. Davatzikos, M. Gallaway, D. Duda, H. I. Chen, J. J. Kim, R. C. Gur, R. L. Wolf, M. S. Grady, S. Hampton, R. Diaz-Arrastia, and D. H. Smith. 2019. Neuroimaging findings in US government personnel with possible exposure to directional phenomena in Havana, Cuba. *JAMA* 322(4):336.

## Section 4

### Plausible Mechanisms

Multiple kinds of mechanisms might contribute to the observed phenomena in the Department of State (DOS) personnel. The committee narrowed the investigation to four, based on their previous appearance in analogous outbreaks of paroxysmal symptoms, their presence in similar localized settings, information available from other investigators, and most notably the known constellation of medical effects (centering on neurologic findings of acute onset). As discussed in Section 3, the acute symptoms with directional dependence are highly unusual, and unlike any disorder reported in the neurological or general medical literature including those with known infectious, inflammatory, or toxic mechanism. The committee felt that these acute symptoms were more consistent with a directed radio frequency (RF) energy attack, and explored possible related mechanisms. At the same time, the chronic symptoms that were reported are often seen in patients after head trauma, as a result of chemical exposure, infectious diseases, or stress in a hostile environment. There did not appear to be any evidence for usual forms of traumatic injury, but the committee did evaluate possible chemical and infectious causes as well as psychosocial causes, for the chronic symptoms.

#### DIRECTED RADIO FREQUENCY ENERGY

##### Sources of Information

The committee relied on open source data from published literature as well as firsthand reports from clinicians, researchers, and affected DOS personnel shared in person at its December and February meetings, to evaluate the plausibility of directed RF energy exposure as a cause of both the acute and chronic clinical signs and symptoms discussed in Section 3 (Clinical Findings). While the committee did review the significant body of scientific literature on the potential therapeutic and palliative applications of electromagnetic energy (e.g., medical radiotherapies) (Citrin, 2017; Mohan et al., 2019; Saitz et al., 2019; Suh et al., 2020; Tsao et al., 2018) and the health risks of microwave radiation (e.g., cell phone emissions) (FDA, 2020; NTP, 2018a,b), this subsection primarily restricts its focus to RF biological effects that are consistent with the clinical and personal (by DOS patients) observations.

Observations from clinicians (including published summaries of symptoms and experiences) and DOS personnel were considered with respect to known biological effects of a wide variety of RF exposures (defined as 30KHz-300GHz, including microwave radiation as 300MHz-300GHz). The committee used these personal and clinician observations to identify known RF biological effects that should be either included or excluded from consideration in explaining the signs and symptoms in DOS personnel.

##### Assessment and Findings

Low-level RF exposures typically deposit energy below the threshold for significant heating (often called “nonthermal” effects), while high-level RF exposures can provide enough energy for significant heating (“thermal” effects) or even burns, and for stimulation of nervous and muscle tissues (“shock” effects) (IEEE, 2019). While much of the general public discussion

on RF biological effects has focused on cancer, there is a growing amount of data demonstrating a variety of non-cancer effects as well, in addition to those associated with thermal heating.

Based on a review of these information sources, the committee finds that many of the acute, early phase symptoms and observations reported by DOS employees are consistent with RF effects, including a perceived clicking sound within the head even when the ears were covered, a perceived force/pressure sensation within the head and on the face, perceived spatial localization and directionality of these perceived phenomena and other loud sounds, hearing loss, tinnitus, impaired gait and loss of balance, as well as the absence of heating sensation and absence of observed disruption of electronic devices in the immediate environment. In addition, many of the chronic, nonspecific symptoms are also consistent with known RF effects, such as dizziness, headache, fatigue, nausea, anxiety, cognitive deficits, and memory loss.

The absence of certain observed phenomena can also help to constrain potential RF source characteristics. For example, the absence of reporting of a heating sensation or internal thermal damage may exclude certain types of high-level RF energy.

There are multiple possible mechanisms for non-thermal RF biological effects, including apoptosis and cell oxidative stress (Barnes and Greenebaum, 2018; Ilhan et al., 2004; Salford et al., 2003; Steiner and Ulrich, 1989; Zhao et al., 2007). RF-induced, non-thermal cell membrane dysfunction (Ramundo-Orlando, 2010) can occur from coherent excitation (Fröhlich, 1988) above 1 GHz due to a variety of effects including electroporation, metabolic changes, pressure fluctuations, and voltage gated calcium channel disruption (Pall, 2013, 2016). However, many of the cognitive, vestibular, and auditory effects observed in DOS personnel are most consistent with modulated, or pulsed, RF biological effects.

There was significant research in Russia/USSR into the effects of pulsed, rather than continuous wave (CW) RF exposures because the reactions to pulsed and CW RF energy at equal time-averaged intensities yielded substantially different results (Pakhomov and Murphy, 2000). According to Pakhomov and Murphy, the Russian-language studies “indicated that pulsing may be an important (or even the most important) factor that determines the biological effects of low-intensity RF emissions” (Pakhomov and Murphy, 2000, p. 2). Military personnel (in Eurasian communist countries) exposed to non-thermal microwave radiation were said to have experienced headache, fatigue, dizziness, irritability, sleeplessness, depression, anxiety, forgetfulness, and lack of concentration, as well as internal sound perception for frequencies between 2.05-2.50 GHz (Adams and Williams, 1976). The review by Pakhomov and Murphy noted that many of the studies from the former Soviet Union were flawed in one or more ways, but that some were well done, replicated, and credible.

Pulsed RF effects on the nervous system can include changes to cognitive (D’Andrea, 1999; Lai, 1994; Tan et al., 2017), behavioral (D’Andrea and Cobb, 1987), vestibular (Lebovitz, 1973), EEG during sleep (Lustenberger et al., 2013), and auditory (Elder and Chou, 2003) function in animals and humans, though many RF exposure characteristics (carrier frequency, pulse repetition frequency, orientation, power densities, duration of exposure) complicate direct comparisons of different experiments (D’Andrea et al., 2003). Some animal studies have shown conflicting results, however, even when using the same exposure system. For example, researchers using the Transformer Energized Megavolt Pulsed Output (TEMPO) microwave pulse apparatus with high peak power RF energy but low specific absorption rate (SAR) values observed a negative effect on cognitive function in rats (time perception and discrimination tasks) (Raslear et al., 1993), but other researchers found no observable behavioral changes in rhesus monkeys (D’Andrea et al., 1989; Ziriak et al., 1999). It should be noted that the low SAR



values for both animal models were lower than whole-body SAR thresholds known to disrupt behavioral performance (D’Andrea, 1991; D’Andrea and de Lorge, 1990; de Lorge, 1984).

In 1961, Alan Frey identified a new, RF-induced auditory phenomenon in both normal and deaf humans that became known as the “Frey effect” (Frey, 1961) (see Appendix C). The areas near the ear were most sensitive to these RF exposures; modulating the RF energy could produce a variety of effects including the perception of “buffeting of the head” or pressure on the face/head without dizziness or nausea, a “pins and needles sensation,” and a sound described as a “buzz, clicking, hiss, or knocking” within the head for RF frequencies between 0.4-3 GHz, depending on pulse width, pulse-repetition frequency (PRF), and peak power density (Frey, 1962). These reported symptoms are consistent with some of the first-person accounts provided to the committee. Frey reported these symptoms with an RF source transmitting at 1.3 GHz (which provides the greatest absorption depth into cortical tissue) with a PRF of 244 Hz, 6  $\mu$ s pulse width, peak power density of 267 mW/cm<sup>2</sup>, and average power density of 0.4 mW/cm<sup>2</sup> (Frey, 1962). Others have demonstrated that GHz range, pulsed RF energy (~14 $\mu$ s pulse width) interacting with common materials can produce external sounds that are audible to nearby humans (Sharp et al., 1974). This is also consistent with potential smartphone microphone excitation from RF energy that would lead to an external, audible clicking sound from the phone. The ability for a pulsed RF source to create internal and external auditory stimuli simultaneously agrees with published and personal reports. Importantly, the Frey effect may be induced without causing identifiable structural injury to neural or labyrinthine tissues.

The potential for RF sources to stimulate the vestibular end organs via thermoelastic pressure waves (see Appendix C) or to excite central nervous system pathways via transduction akin to the Frey effect is not known. However, if these effects exist, this unusual form of vestibular stimulation could lead to very confusing perceptions, as central vestibular pathways do their best to resolve the non-physiological pattern of end organ stimulation resulting in perceptions of physically impossible motions, unexpected reflexive postural responses to them, and faulty inferences about external forces causing them. Affected individuals could report different sensations in response to the same external stimulus; thus, it is consistent with this scenario that the early phase reports of the perceptions of affected individuals varied from one individual to another, and may have been difficult for the individuals to describe. With regard to vestibular and balance systems, the functional vestibular disorder of persistent postural-perceptual dizziness (PPPD) may be triggered by any condition that causes symptoms of vertigo, unsteadiness, or dizziness, or disrupts balance function, even if transiently and without causing structural injury (Staab et al. 2017). The NIH team diagnosed PPPD in one-quarter of patients that they evaluated. Patients with PPPD commonly report problems with cognition and fatigue in addition to core symptoms of unsteadiness, dizziness and susceptibility to motion stimuli (Stone, 2016).

If a Frey-like effect can be induced on central nervous system tissue responsible for space and motion information processing, it likely would induce similarly idiosyncratic responses. More general neuropsychiatric effects from electromagnetic stimuli are well-known and are being used increasingly to treat psychiatric and neurologic disorders. In 2008, the Food and Drug Administration (FDA) approved transcranial magnetic stimulation (TMS) to treat major depression in adults who do not respond to antidepressant medications (Cook, 2018). Ten years later, the FDA approved office-based TMS as a treatment for obsessive compulsive disorder (OCD) (FDA, 2018) and portable TMS to treat migraine (Jeffrey, 2013).

The benefits derived from purposeful short-term exposures to therapeutic neuromodulation contrast with the adverse neurologic and neuropsychiatric symptoms described

by individuals exposed to electromagnetic fields (e.g., high tension electrical transmission cables) over longer periods of time (Pall, 2016) as summarized by Stein and Udasin (2020).

### **Summary**

The committee finds that many of the acute, sudden-onset, early phase signs, symptoms and observations reported by DOS employees are consistent with RF effects. In addition, many of the chronic, nonspecific symptoms are also consistent with known RF effects, such as dizziness, headache, fatigue, nausea, anxiety, cognitive deficits, and memory loss. It is not necessary for RF energy sources to produce gross structural damage to cause symptoms. Rather, as with the Frey effect or potential thermoelastic pressure waves, RF sources may trigger symptoms by transiently inducing alterations in brain functioning.

There are several types of data that would be helpful and could improve both the findings and their level of certainty. While there are several studies on the health effects of continuous wave and pulsed RF sources, there are insufficient data in the open literature on potential RF exposure/dosage characteristics and biological effects possible for DOS scenarios. Specific experiments would be needed with RF exposure and dosage characteristics (frequency, pulse repetition frequency, pulse width, incident angle between potential source and subject, duration of exposure, number of repeated exposures, etc.) to quantify the biological effects, but would be ethically difficult to justify. In the absence of such data, it is difficult to align specific biophysical effects within the potential RF exposure regime that could explain specific medical symptoms reported by DOS personnel and the variability in specific experiences and timelines of individuals. Patient clinical heterogeneity could be due to variability of exposure dosage conditions, differences in interpretation of non-physiological vestibular stimuli, and anatomical differences that could influence individual exposure and/or response.

## **CHEMICALS**

### **Sources of Information**

DOS asked the committee to consider the plausibility of organophosphate (OP) or pyrethroid insecticide exposure as a cause of the clinical signs/symptoms observed in U.S. Embassy personnel in Havana. This possible cause was raised by Canadian investigators who reported decreased cholinesterase activity, temephos (an OP), and pyrethroid metabolites in blood samples collected from some Canadian Embassy personnel and Canadian tourists who were in Havana during the same period as the affected U.S. Embassy personnel. Additionally, the timing of some cases in U.S. Embassy personnel coincided with widespread spraying of OP and pyrethroid insecticides in Cuba in 2016 to mitigate spread of Zika virus by mosquitos.

To address the plausibility of the OP/pyrethroid insecticide hypothesis, the committee examined five sources of information: (1) the Research Report, “Havana Syndrome: Neuroanatomical and Neurofunctional Assessment in Acquired Brain Injury Due to Unknown Etiology” (Friedman et al., 2019); (2) formal presentations to the committee by Claire Huson (DOS Office of Safety, Health, and Environmental Management), Cynthia Calkin and Alon Friedman (Dalhousie University Faculty of Medicine), Marion Ehrich (Virginia-Maryland College of Veterinary Medicine), and Nick Buckley (University of Sydney); (3) feedback provided during a question and answer session with DOS Bureau of Medical Services staff; (4) the National Toxicology Program publication, “Systematic review of long-term neurological effects following acute exposure to the organophosphorus nerve agent sarin,” (NTP, 2019); and (5) peer-reviewed scientific literature.

The committee considered three general issues: (1) What is the strength of the evidence that affected individuals were exposed to OP or pyrethroid insecticides?; (2) Were exposures at levels that might be expected to cause toxic effects?; and (3) How similar are the signs and symptoms of acute, subacute, or chronic exposures to OP or pyrethroid insecticides to the distinctive acute signs and symptoms and the less specific chronic signs and symptoms associated with cases from Havana?

### **Assessment and Findings**

With respect to the question of exposure, information presented by Claire Huson regarding the DOS Integrated Pest Management (IPM) program indicated that pyrethroids (lambda cyhalothrin, cyfluthrin, permethrin, and cypermethrin) were used in U.S. Embassy offices and residences in Havana; thus, the potential for exposure of U.S. Embassy personnel to these insecticides was quite high. OPs were not included in the IPM program and it is DOS IPM policy not to allow outside contractors to apply pesticides in U.S. Embassy offices or residences. Consistent with this information, the committee heard in a question and answer session with DOS medical staff that OPs were not detected in environmental samples collected from the residences of U.S. Embassy personnel some months after the incidence of unexplained illnesses. However, this information does not rule out the possibility that U.S. Embassy personnel were exposed to OPs in their residences proximal to the onset of symptoms because OPs are relatively short-lived in the environment (half-life of several days in the outdoor environment and weeks to months in the indoor environment depending on dust levels, light, and humidity). Moreover, information provided by presenters from Dalhousie University indicated widespread heavy spraying of OPs (including the OP chlorpyrifos) and pyrethroids throughout Cuba to prevent the spread of Zika virus by mosquitos. If the images of pesticide spraying shown in the formal presentations to the committee were reflective of actual conditions in Havana, it is highly likely that U.S. Embassy personnel were exposed to OPs either when they were in public spaces or via overspray that drifted from public spaces into U.S. Embassy offices and residences. As an aside, targeted exposures of individuals to OPs are also possible, as illustrated by the assassination of Kim Jong-nam, half-brother of North Korean leader Kim Jong-un, who died after two women allegedly applied OP nerve agent to his skin in the Kuala Lumpur airport on February 13, 2017, and by the attempted assassination of a former Russian spy and his daughter in Great Britain in 2018. However, these individuals showed acute symptoms of cholinergic poisoning associated with their exposure to OPs.

OP exposure is also monitored by measuring AChE activity in blood samples because OP insecticides inhibit AChE. AChE activity was not measured in blood from U.S. Embassy personnel. The Dalhousie University research team presented data they believed demonstrated significantly decreased AChE activity in at least a subset of Canadian Embassy personnel and Canadian tourists who were in Havana during the same time as affected U.S. Embassy personnel. Based on these data and targeted analysis of OPs and pyrethroid metabolites in serum samples that identified the OP temephos and the pyrethroid metabolite 3-PBA in blood from a subset of individuals (although the overlap between individuals with AChE inhibition and detectable OPs/pyrethroids is not clear), the Dalhousie University group developed a working hypothesis that neurological effects were due to chronic low level cholinesterase inhibitor toxicity. These data cannot, however, be considered supportive of this hypothesis. One reason, based on information presented to the committee, is that the Dalhousie group measured AChE activity in serum/plasma samples. However, AChE is a membrane-bound molecule found in blood only on erythrocytes; thus, whole blood samples, not serum or plasma, are required for accurate

determination of AChE activity in blood. Another concern with the Dalhousie measurements is that AChE levels should always be compared to the established reference values of the clinical laboratory in which the measurements are performed, rather than to the values of a specific and limited set of experimental controls, because laboratory reference values are generally based on many more samples and reflect a more realistic range of normal activities. The Dalhousie study relied instead on experimental controls. A second reason is that the number of Canadian personnel with detectable levels of temephos or 3-PBA was much smaller than the number of individuals with symptoms. A third reason is that Canadian personnel were not sampled at the time of initial signs and symptoms.

Absent data regarding the concentration of OPs or pyrethroids in relevant environmental samples collected proximal to the onset of symptoms or in samples from affected U.S. Embassy personnel at the time of initial signs and symptoms, it is not possible to determine whether exposures were at levels that might reasonably cause toxic effects, particularly in vulnerable individuals. This issue is complicated by the fact that there is growing evidence that at least some of the neurotoxic effects of OPs are mediated by mechanism(s) other than or in addition to AChE inhibition (Anger et al., 2020; Costa, 2006; Naughton and Terry, 2018; Pope, 1999).

With regards to the overlap of symptoms between chemical exposures and the Havana cases, epidemiologic and clinical studies have linked occupational or environmental chemical (including OP and pyrethroid insecticide) exposures to a subset of the distinctive early phase symptoms and many of the nonspecific chronic problems suffered by some of the U.S. Embassy Havana cases (see Appendix D).

Acute OP poisoning manifests as a clinical toxic syndrome known as cholinergic crisis, which includes parasympathomimetic symptoms (sweating, tears, rhinorrhea, salivation, urination, diarrhea, increased bronchial secretions and bronchoconstriction, and bradycardia), muscle fasciculation followed by flaccid paralysis, loss of consciousness and seizures (Eddleston et al., 2008; Hulse et al., 2014). Subacute and chronic OP exposures involving doses that do not cause significant AChE inhibition, do not cause cholinergic signs but can be associated with neurotoxic effects not only in individuals with occupational exposures, but also in the general public. OP-associated neurotoxic effects, which may or may not be associated with AChE inhibition in affected individuals, include hearing loss, tinnitus, dizziness, headache, fatigue, motor incoordination, nausea, insomnia, anxiety, memory deficits and inability to concentrate (Anger et al., 2020; Ashok Murthy and Visweswara Reddy, 2014; Choochouy et al., 2019; Crawford et al., 2008; Dassanayake et al., 2007, 2008, 2009; Dundar et al., 2016; Edwards and Tchounwou, 2005; London et al., 1998; Richter et al., 1992; Roldan-Tapia et al., 2006; Ross et al., 2013; Teixeira et al., 2002). Some of these effects were reported among affected DOS employees stationed in Havana.

There are significantly less epidemiologic and clinical data available regarding the neurotoxic effects of pyrethroids than there are for OPs, but published studies report associations between acute, subacute, and chronic pyrethroid exposures and hearing loss, visual disturbance, tinnitus, dizziness, headache, nausea, fatigue, and deficits in memory and concentration in occupational cohorts and in the general public (Campos et al., 2016; Chen et al., 1991; Lessenger, 1992; Müller-Mohnssen, 1999; Richardson et al., 2019; Teixeira et al., 2002; Xu et al., 2020; Zeigelboim et al., 2019). High dose acute pyrethroid exposures are also associated with tremors and seizures (Bal-Price et al., 2015).

**Summary**

In summary, the committee concludes that it is not likely that acute high-level exposure to OPs and/or pyrethroids contributed to the unexplained illnesses observed in the Havana cases because there is no convincing evidence of acute high-level exposures and the clinical history of affected U.S. Embassy personnel is not consistent with acute OP poisoning. It is also unlikely that subacute or chronic OP or pyrethroid exposures precipitated the onset of the distinctive acute symptoms associated with the Havana cases. However, given experimental data indicating that interactions between pesticides (particularly OPs) and psychosocial or physical stressors, the latter including noise and non-ionizing radiation, can increase risk and/or severity of adverse outcomes, the committee could not rule out the possibility, although slight, that exposure to insecticides, particularly OPs, increased susceptibility to the triggering factor(s) that caused the Embassy personnel cases. Alternatively, differential exposure to insecticides amongst affected individuals may have contributed to the clinical heterogeneity of the acute symptoms noted in Havana cases, since OP and pyrethroid exposures are associated with a subset of these acute symptoms (see Appendix D). The committee also finds it plausible that subacute or chronic OP and/or pyrethroid exposures contributed to the nonspecific chronic symptoms observed in affected U.S. Embassy personnel.

**INFECTIOUS AGENTS****Sources of Information**

The committee reviewed published medical and public health literature, including results of searches of PubMed for infectious diseases, Cuba, and neurological features.

**Assessment and Findings**

The committee considered endemic and epidemic infectious diseases known to have been present in Cuba during 2016-2018 and focused on those with known neurological manifestations. Some of these diseases could be excluded based on their dissimilar clinical features relative to the signs and symptoms reported by U.S. Embassy personnel in Havana, such as rabies or Guillain-Barré syndrome as a post-infectious complication of campylobacteriosis. Several mosquito-borne infections received further attention because of their prevalence and association with relevant, albeit rare, clinical features. These included dengue, chikungunya, and Zika virus infections. All three have been associated with encephalitis, Guillain-Barré syndrome, transverse myelitis, and neuro-ocular disease (Mehta et al., 2018). All of these complications are rare. For example, it has been estimated that approximately 0.1 percent of all chikungunya infections develop neurological disease (Economopoulou et al., 2009). Risk factors include underlying comorbidities, and the extremes of age. However, nearly all of these chikungunya cases with neurological complications also presented with typical acute systemic manifestations (i.e., fever, rash, arthralgia, and conjunctivitis). Although dengue has been the most commonly reported arboviral infection in Cuba (Guanche Garcell et al., 2020), the epidemiology and incidence of Zika in Cuba is particularly relevant to the timing of the DOS personnel health events.

Travel surveillance and genomic epidemiology revealed a large, unreported, and delayed Zika outbreak in Cuba that followed Zika outbreaks elsewhere in the Caribbean by about one year (Grubaugh et al., 2019). Zika disease began in Cuba in the latter half of 2016 and peaked in fall 2017. Genomic surveillance confirmed dengue disease in Cuba in 2014 and 2015 and a chikungunya outbreak in 2014, but little or none of these two diseases in 2016 and 2017. It is believed that implementation of an intensive mosquito control program based on insecticide use



beginning in February 2016 may have delayed the establishment of Zika virus and subsequent disease in Cuba until 2016-2017. (The committee considered the possible role of organophosphate and pyrethroid insecticide exposure in the DOS personnel illnesses—see Section 4, Chemicals.) Thus, the timing and relative prevalence of Zika in Cuba justify further comment on this infection as a possible cause of the DOS personnel cases.

A population-based observational study of Zika infection in the French West Indies in 2016 provides a valuable description of the neurologic complications of this disease (Lannuzel et al., 2019), and a reference against which the clinical features reported in DOS personnel (see Section 3) can be compared. In 2016, 66,600 persons in Guadeloupe and Martinique sought medical attention with manifestations of Zika infection. Of these, 87 presented to the major referral centers on the two islands with neurologic manifestations. Of the 87, 54 (62 percent) had peripheral nervous system (PNS) involvement, and of those 54, 40 were diagnosed with Guillain-Barré syndrome. Among the other 14 with PNS disease, 8 had cranial nerve palsies; in all 8, the facial nerve was involved, and in 4 of them, there was involvement of the vestibulocochlear nerve. Of the 87, 19 had central nervous system (CNS) involvement, with encephalitis the most common diagnosis. Of the 87, 14 had both PNS and CNS disease. Of 76 patients available for follow-up, 19 had residual disease at a median of 14 months after presentation.

Thus, the overall rate of Zika neurological complications seen in the main clinical referral centers during this epidemic year in the French West Indies was approximately 0.1-0.2 percent of those with known Zika infection (Lannuzel et al., 2019). This is similar to the rate for chikungunya on Reunion Island in 2005-2006. Approximately 5 percent of this small subpopulation with Zika neurologic complications shared a feature with the illnesses reported in the DOS Cuba patient cohort (i.e., vestibular disease, manifest as dizziness, nystagmus, and/or vertigo). A smaller number had hearing loss, memory difficulties, and visual loss. Thus, Zika virus can cause a few of the acute and chronic clinical features reported in the DOS Cuba patient cohort, but these features are quite rare with Zika and would not be expected to occur at all in a population of less than 1,000 people with Zika. None of the patients in the DOS Cuba cohort are said to have suffered from the much more common manifestations of Zika: rash, fever, arthralgia, myalgia, and conjunctivitis.

### Summary

In summary, the committee considered possible infectious etiologies that might explain the clinical features reported in DOS employees and focused on those infectious agents known to be prevalent in Cuba and capable of causing neurological manifestations. Among those agents, Zika infection received attention from the committee because it was epidemic in Cuba in 2016-2017 and is known to be able to produce relevant neurological findings. However, after reviewing the medical and public health literature, the committee found it highly unlikely that Zika was the cause of the constellation of signs and symptoms reported among DOS personnel, especially the acute, sudden onset, initial phase clinical features, for two major reasons. First, Zika is not known to cause an abrupt onset illness nor an illness with the collection of findings reported in the initial phase of the DOS employee illnesses—especially in the absence of rash, fever, arthralgia, myalgia and conjunctivitis. Second, the relevant neurological features of Zika are exceedingly rare and statistically would not be expected to occur in any DOS employee in Havana, and certainly not more than one.

The committee could not rule out the possibility that some employees were infected by Zika, and that it contributed in some fashion together with other causative factors to the chronic

clinical findings, especially during 2017. The committee is not aware of serological testing for Zika or any other infectious agents among DOS Embassy Havana affected personnel.

## PSYCHOLOGICAL AND SOCIAL FACTORS

### Sources of Information

As noted in Section 3, clinical investigators presented data to the committee as aggregated summaries of patients' histories, physical examination findings, and results of laboratory testing including neuropsychological assessments. Individual patient-level data were not provided to the committee, other than what the committee learned from direct interviews with a small number of affected DOS personnel.

### Assessment and Findings

#### *Acute Symptoms*

The committee carefully considered three possible roles for psychological and social factors in the morbidity experienced by DOS employees: (1) psychiatric disorders as primary causes of symptoms; (2) psychiatric disorders as secondary sequelae of other causes of illness; and (3) psychological and social factors co-existing with other causes of illness. As with all other potential causes and mechanisms reviewed by the committee, evidence was sought for and against specific associations between psychological and social factors and patients' symptoms. The committee did not regard psychological and social factors to be default explanations for enigmatic symptoms but endeavored to make a positive identification of their potential contributions to morbidity. These efforts were constrained by the limits of data collected and presented by the clinical teams from Miami, Penn, Dalhousie, and NIH, which offered an incomplete picture of the range of acute and chronic symptoms over space and time, and in particular about the course of illness of individual patients. Nevertheless, it appeared that a biphasic distribution of acute and chronic symptoms (see Section 2) offered coherence to the patterns of neuropsychiatric symptoms reported by the clinical teams and patients themselves.

In general, psychological factors may cause or contribute to emotional symptoms (sadness, frustration, irritability, anxiety, and anhedonia), vegetative symptoms (sleep, energy, and appetite changes), and cognitive symptoms (attention, concentration, and memory problems), as well as enigmatic somatic symptoms. At the milder end of the spectrum, these may fall short of fully diagnosable psychiatric disorders (i.e., transient and self-limited responses to life circumstances) or may represent adjustment disorders (i.e., periods of poor adjustment to stressors, including other illnesses). More severe or persistent symptoms may constitute major depressive or anxiety disorders, either as primary, secondary, or co-existing illnesses. In cases where individuals are exposed to potential threats to life or limb, acute and posttraumatic stress disorders may develop, manifesting with symptoms of re-experiencing, avoidance, hyperarousal, and negative mood and cognitions regarding the triggering event. The development of acute and posttraumatic stress disorders rests on the perception of threat by affected individuals. As such, reactions may vary considerably among exposed persons.

Potential threats attributed to human causes are more likely to trigger traumatic stress symptoms than threats attributed to natural causes, especially when the threat is thought to arise from the concerted efforts of an adversarial group (e.g., warfare) rather than isolated actions of individuals (e.g., unprovoked assaults) (Bromet et al., 2017; Staab et al., 1999). Environments that include incomplete, inconsistent, or erroneous information about potential threats may

exacerbate and perpetuate symptoms. After reviewing the nature of these disorders, the committee concluded that such reactions could not cause the initial sudden-onset distinct and unusual audio-vestibular symptoms and signs described in Section 3 or by CDC, but that psychological or psychiatric disorders could conceivably contribute to some of the other acute and chronic symptoms in some patients.

The committee then considered the possibility that acute auditory and vestibular symptoms described by DOS patients were hallucinations or delusions. Psychotic disorders may cause hallucinations involving any sensory modality. Auditory hallucinations are common, whereas vestibular and balance hallucinations are uncommon. However, auditory hallucinations caused by primary psychotic disorders usually take the form of human voices or other human sounds, less often other natural or mechanical sounds. Importantly, the committee received no evidence that any patients had psychiatric symptoms indicative of primary, secondary, or co-existing schizophrenic spectrum disorders, brief reactive psychoses, mood disorders with psychosis, psychoses related to substances of abuse, or psychoses associated with major cognitive disorders. Therefore, the committee found it very unlikely that any of the acute or chronic symptoms experienced by patients were caused by these conditions.

Patients with delusional disorders may describe a variety of sensory experiences that they relate to plausible (i.e., non-bizarre) but not factual causes. The most common delusions are paranoid in nature. Affected individuals are otherwise normal from a psychiatric perspective. Delusional disorders do not cause other emotional, vegetative, or cognitive symptoms. Infrequently, delusions may be shared by a few individuals close to the index case. The committee did not receive information about the psychiatric or psychological status of individual patients; therefore, it could not make a determination about the presence or absence of delusional disorder as a cause for the distinct acute symptoms in any affected persons. However, the committee did conclude that delusional disorders could not explain the full range of symptoms reported by the entire group of patients.

Reports in the medical literature (Bartholomew and Baloh, 2020) and mass media (Borger and Jaekl, 2017; Hurley, 2019) have opined that mass psychogenic illness (also known as mass hysteria or epidemic hysteria) was the cause of patients' symptoms. These reports cited the challenging political and security circumstances surrounding the diplomatic missions in Cuba and China, the frequent harassment experienced by DOS employees in their homes, the lack of evidence for a clear external cause for patients' symptoms, and the absence of a definitive pattern of structural deficits on medical examinations in support of this conclusion. The committee considered this possibility, while keeping in mind that the likelihood of mass psychogenic illness as an explanation for patients' symptoms had to be established from sufficient evidence. It could not be inferred merely by the absence of other causal mechanisms or the lack of definitive structural injuries.

Studies of mass psychogenic illnesses have found that social and environmental variables are important in triggering these events. Thereafter, social connections or exposure to developing cases either in person or vicariously through word of mouth or media, including social media, are necessary to sustain them (Bartholomew et al., 2012; Boss, 1997; Cole et al., 1990; Knight et al., 1965). However, adverse social circumstances are not required preconditions. Well-documented cases of mass psychogenic illnesses have occurred in groups that were under no identifiable external stress or internal strain at the onset of events (Bartholomew et al., 2012). Most individuals affected by mass psychogenic illnesses do not have pre-existing psychiatric disorders. Rather, in most events, index cases developed their initial symptoms in response to idiosyncratic interpersonal circumstances or after exposures to perceived or actual but benign

environmental stimuli (Bartholomew et al., 2012; Boss, 1997; Cole et al., 1990; Knight et al., 1965). However, it is important to recognize that mass psychogenic illness may follow index cases afflicted with serious medical conditions or exposed to harmful environmental agents, especially when potential causes of illnesses affecting index cases are unclear or misattributed to agents, actual or perceived, that may affect the larger group (Bartholomew et al., 2012). The term “mass” may be mistaken to imply that large numbers of individuals must be involved over a short period of time in these events. However, the medical literature shows that one-third of incidents since the 1970s involved fewer than 30 individuals and approximately 20 percent of events lasted longer than 30 days (Cole et al., 1990). For communities under chronic stress, resolution may take months (Bartholomew et al., 2012). Nonspecific dizziness, lightheadedness, and fatigue have been described commonly in mass psychogenic illnesses, but complaints similar to the directional audio-vestibular symptoms reported by affected individuals from Cuba have not (Bartholomew et al., 2012; Cole et al., 1990). Events of mass psychogenic illness end when the potential for social contagion is reduced (not necessarily eliminated) by separation of unaffected individuals from sources of contagion and when the majority of unaffected or previously affected individuals reach the conclusion that the inciting event was physically benign or no longer poses a risk (Bartholomew et al., 2012).

These key characteristics of mass psychogenic illness have strong parallels with outbreaks of infectious diseases and have been investigated successfully using similar rigorous epidemiologic methods since the 1960s (Knight et al., 1965), including detailed examinations of index cases and subsequently affected individuals, contact tracing, and careful investigation of the environments in which the events occurred (Bartholomew et al., 2012; Boss, 1997; Cole et al., 1990; Knight et al., 1965). As described in Section 3, the committee noted two constellations of signs and symptoms, one of them acute, occurring at the onset of some cases with more distinct and unusual features, and the other chronic, occurring later in the course of these cases or with subacute onset in other cases. However, in the absence of patient-level data, the committee could not identify index versus subsequent cases. Furthermore, the committee received no epidemiological evidence about patterns of social contacts that would permit a determination about possible social contagion. Without access to these data, a retrospective diagnosis of mass psychogenic illness is considered to be speculative at best and subject to necessary criticism (Jacobsen and Ebbelhøj, 2016, 2017; Jansen et al., 2016). Thus, the committee was not able to reach a conclusion about mass psychogenic illness as a possible cause of the events in Cuba or elsewhere.

### *Chronic Symptoms*

Despite extensive clinical evaluations, definitive causes of chronic symptoms have not been identified in most DOS personnel with ongoing morbidity. However, approximately one quarter of patients examined by the clinical team at NIH were diagnosed with persistent postural-perceptual dizziness (PPPD) and at least one patient received that diagnosis after a series of clinical examinations conducted elsewhere. PPPD is a functional (not psychiatric) vestibular disorder that may be triggered by vestibular, neurologic, other medical, and psychological conditions (Staab et al., 2017). Thus, its presence provides no information regarding the initial causality of patients’ symptoms. On the other hand, its presence may inform treatment as there are data from uncontrolled and small controlled investigations to support the use of vestibular (including visual) habituation exercises, cognitive behavioral therapy, and serotonergic antidepressants (selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors) for treating this condition (Popkirov et al., 2018; Staab, 2020). When symptoms of

PPPD occur in the setting of additional morbidity such as cognitive symptoms and psychological distress, expert opinion and clinical experience suggest that a broader array of treatments, including cognitive rehabilitation and third-wave psychotherapies (e.g., Mindfulness, Acceptance and Commitment Therapy) may be helpful.

### Summary

As stated previously, the committee sought positive evidence that psychological and social factors may have caused or contributed to symptoms reported by DOS personnel. The acute initial, sudden-onset, distinct and unusual symptoms and signs described in some affected DOS personnel (see Section 3 and CDC Report) cannot be ascribed to psychological and social factors in the absence of patient-level data. The significant variability and clinical heterogeneity of the illnesses affecting DOS personnel leave open the possibility of multiple causal factors over time and place, both for individual cases and for the population. Like other mechanisms reviewed by the committee, psychological and social factors could exacerbate other forms of pathology and have to be considered as contributors to morbidity in some of the cases, especially for individuals with chronic symptoms.

The chronic vestibular symptoms experienced by some DOS personnel are consistent with persistent postural-perceptual dizziness. This functional vestibular disorder may be triggered by vestibular, neurologic, other medical, and psychological conditions, and offers a potential avenue for rehabilitative interventions.

### REFERENCES

- Adams, R. L., and R. A. Williams. 1976. *Biological effects of electromagnetic radiation (radiowaves and microwaves)—Eurasian communist countries*. Defense Intelligence Agency.
- Anger, W. K., F. M. Farahat, P. J. Lein, M. R. Lasarev, J. R. Olson, T. M. Farahat, and D. S. Rohlman. 2020. Magnitude of behavioral deficits varies with job-related chlorpyrifos exposure levels among egyptian pesticide workers. *Neurotoxicology* 77:216-230.
- Ashok Murthy, V., and Y. J. Visweswara Reddy. 2014. Audiological assessment in organophosphorus compound poisoning. *Indian Journal of Otolaryngology and Head and Neck Surgery* 66(1):22-25.
- Bal-Price, A., K. M. Crofton, M. Sachana, T. J. Shafer, M. Behl, A. Forsby, A. Hargreaves, B. Landesmann, P. J. Lein, J. Louisse, F. Monnet-Tschudi, A. Paini, A. Rolaki, A. Schratzenholz, C. Sunol, C. van Thriel, M. Whelan, and E. Fritsche. 2015. Putative adverse outcome pathways relevant to neurotoxicity. *Critical Reviews in Toxicology* 45(1):83-91.
- Barnes, F., and B. Greenebaum. 2018. Role of radical pairs and feedback in weak radio frequency field effects on biological systems. *Environmental Research* 163:165-170.
- Bartholomew, R. E., and R. W. Baloh. 2020. Challenging the diagnosis of 'Havana syndrome' as a novel clinical entity. *Journal of the Royal Society of Medicine* 113(1):7-11.
- Bartholomew, R. E., S. Wessely, and G. J. Rubin. 2012. Mass psychogenic illness and the social network: Is it changing the pattern of outbreaks? *Journal of the Royal Society of Medicine* 105:509-512.
- Borger, J., and P. Jaekl. 2017. Mass hysteria may explain 'sonic attacks' in Cuba, say top neurologists. *The Guardian*. <https://www.theguardian.com/world/2017/oct/12/cuba-mass-hysteria-sonic-attacks-neurologists> (accessed July 18, 2020).
- Boss, L. P. 1997. Epidemic hysteria: A review of the published literature. *Epidemiologic Reviews* 19(2):243-253.
- Bromet, E. J., L. Atwoli, N. Kawakami, F. Navarro-Mateu, P. Piotrowski, A. J. King, S. Aguilar-Gaxiola, J. Alonso, B. Bunting, K. Demyttenaere, S. Florescu, G. de Girolamo, S. Gluzman, J. M. Haro, P. de Jonge, E. G. Karam, S. Lee, V. Kovess-Masfety, M. E. Medina-Mora, Z. Mneimneh, B. E. Pennell, J. Posada-Villa, D. Salmerón, T. Takeshima, and R. C. Kessler. 2017. Post-traumatic



- stress disorder associated with natural and human-made disasters in the world mental health surveys. *Psychological Medicine* 47(2):227-241.
- Campos, Y., V. Dos Santos Pinto da Silva, M. Sarpa Campos de Mello, and U. Barros Otero. 2016. Exposure to pesticides and mental disorders in a rural population of southern Brazil. *Neurotoxicology* 56:7-16.
- Chen, S. Y., Z. W. Zhang, F. S. He, P. P. Yao, Y. Q. Wu, J. X. Sun, L. H. Liu, and Q. G. Li. 1991. An epidemiological study on occupational acute pyrethroid poisoning in cotton farmers. *British Journal of Industrial Medicine* 48(2):77-81.
- Choochouy, N., P. Kongtip, S. Chantanakul, N. Nankongnab, D. Sujirarat, and S. R. Woskie. 2019. Hearing loss in agricultural workers exposed to pesticides and noise. *Annals of Work Exposure and Health* 63(7):707-718.
- Citrin, D. E. 2017. Recent developments in radiotherapy. *New England Journal of Medicine* 377(22):2200-2201.
- Cole, T. B., T. L. Chorbá, and J. Hora. 1990. Patterns of transmission of epidemic hysteria in a school. *Epidemiology* 1:212-218.
- Cook, I. A. 2018. Current FDA-cleared TMS systems and future innovations in TMS therapy. In *Transcranial magnetic stimulation: Clinical applications for psychiatric practice*, edited by R. A. Bermudes, K. Lanocha, and P. G. Janicak. Washington, DC: American Psychiatric Association Publishing. Pp. 173-198.
- Costa, L. G. 2006. Current issues in organophosphate toxicology. *Clinica Chimica Acta* 366(1-2):1-13.
- Crawford, J. M., J. A. Hoppin, M. C. Alavanja, A. Blair, D. P. Sandler, and F. Kamel. 2008. Hearing loss among licensed pesticide applicators in the agricultural health study. *Journal of Occupational and Environmental Medicine* 50(7):817-826.
- D'Andrea, J. A. 1991. MW radiation absorption: Behavioral effects. *Health Physics* 61:29-40.
- D'Andrea, J. A. 1999. Behavioral evaluation of microwave irradiation. *Bioelectromagnetics* Suppl 4:64-74.
- D'Andrea, J. A., and B. L. Cobb. 1987. High-peak-power microwave pulses at 1.3 GHz: Effects on fixed-interval and reaction-time performance in rats. Naval Aerospace Medical Research Laboratory Report #1337.
- D'Andrea, J. A., and J. O. de Lorge. 1990. Behavioral effects of electromagnetic fields. In *Biological Effects and Medical Applications of Electromagnetic Energy*, edited by O. P. Gandhi. Englewood Cliffs, NJ: Prentice Hall. Pp. 319-338.
- D'Andrea, J. A., B. L. Cobb, and J. O. de Lorge. 1989. Lack of behavioral effects in the rhesus monkey: High peak microwave pulses at 1.3 ghz. *Bioelectromagnetics* 10(1):65-76.
- D'Andrea, J. A., C. K. Chou, S. A. Johnston, and E. R. Adair. 2003. Microwave effects on the nervous system. *Bioelectromagnetics* Suppl 6:S107-S147.
- Dassanayake, T., V. Weerasinghe, U. Dangahadeniya, K. Kularatne, A. Dawson, L. Karalliedde, and N. Senanayake. 2007. Cognitive processing of visual stimuli in patients with organophosphate insecticide poisoning. *Neurology* 68(23):2027-2030.
- Dassanayake, T., V. Weerasinghe, U. Dangahadeniya, K. Kularatne, A. Dawson, L. Karalliedde, and N. Senanayake. 2008. Long-term event-related potential changes following organophosphorus insecticide poisoning. *Clinical Neurophysiology* 119(1):144-150.
- Dassanayake, T., I. B. Gawarammana, V. Weerasinghe, P. S. Dissanayake, S. Pragaash, A. Dawson, and N. Senanayake. 2009. Auditory event-related potential changes in chronic occupational exposure to organophosphate pesticides. *Clinical Neurophysiology* 120(9):1693-1698.
- de Lorge, J. O. 1984. Operant behavior and colonic temperature of *Macaca mulatta* exposed to radio-frequency fields at and above resonant frequencies. *Bioelectromagnetics* 5:233-246.
- Dundar, M. A., S. Derin, M. Aricigil, and M. A. Eryilmaz. 2016. Sudden bilateral hearing loss after organophosphate inhalation. *Turkish Journal of Emergency Medicine* 16(4):171-172.
- Economopoulou, A., M. Dominguez, B. Helynck, D. Sissoko, O. Wichmann, P. Quenel, P. Germonneau, and I. Quatresous. 2009. Atypical chikungunya virus infections: Clinical manifestations, mortality

- and risk factors for severe disease during the 2005-2006 outbreak on reunion. *Epidemiology & Infection* 137(4):534-541.
- Eddleston, M., N. A. Buckley, P. Eyer, and A. H. Dawson. 2008. Management of acute organophosphorus pesticide poisoning. *Lancet* 371(9612):597-607.
- Edwards, F. L., and P. B. Tchounwou. 2005. Environmental toxicology and health effects associated with methyl parathion exposure--a scientific review. *International Journal of Environmental Research and Public Health* 2(3-4):430-441.
- Elder, J. A., and C. K. Chou. 2003. Auditory response to pulsed radiofrequency energy. *Bioelectromagnetics* Suppl 6:S162-S173.
- FDA (Food and Drug Administration). 2018. FDA permits marketing of transcranial magnetic stimulation for treatment of obsessive compulsive disorder. <https://www.fda.gov/news-events/press-announcements/fda-permits-marketing-transcranial-magnetic-stimulation-treatment-obsessive-compulsive-disorder> (accessed July 16, 2020).
- FDA. 2020. Review of published literature between 2008 and 2018 of relevance to radiofrequency radiation and cancer. <https://www.fda.gov/media/135043/download> (accessed July 18, 2020).
- Frey, A. H. 1961. Auditory system response to radio frequency energy. Technical note. *Aerospace Medicine* 32:1140-1142.
- Frey, A. H. 1962. Human auditory system response to modulated electromagnetic energy. *Journal of Applied Physiology* 17:689-692.
- Friedman, A., C. Calkin, and C. Bowen. 2019. Havana syndrome: Neuroanatomical and neurofunctional assessment in acquired brain injury due to unknown etiology.. <https://www.scribd.com/document/426438895/Etude-du-Centre-de-traitement-des-lesions-cerebrales-de-l-Universite-de-Dalhousie#download> (accessed July 7, 2020).
- Fröhlich, H. 1988. Theoretical physics and biology. In *Biological coherence and response to external stimuli*, edited by H. Fröhlich. Berlin, Germany: Springer-Verlag. Pp. 1-24.
- Grubaugh, N. D., S. Saraf, K. Gangavarapu, A. Watts, A. L. Tan, R. J. Oidtman, J. T. Ladner, G. Oliveira, N. L. Matteson, M. U. G. Kraemer, C. B. F. Vogels, A. Hentoff, D. Bhatia, D. Stanek, B. Scott, V. Landis, I. Stryker, M. R. Cone, E. W. T. Kopp, A. C. Cannons, L. Heberlein-Larson, S. White, L. D. Gillis, M. J. Ricciardi, J. Kwal, P. K. Lichtenberger, D. M. Magnani, D. I. Watkins, G. Palacios, D. H. Hamer, G. S. Network, L. M. Gardner, T. A. Perkins, G. Baele, K. Khan, A. Morrison, S. Isern, S. F. Michael, and K. G. Andersen. 2019. Travel surveillance and genomics uncover a hidden Zika outbreak during the waning epidemic. *Cell* 178(5):1057-1072.
- Guanche Garcell, H., F. Gutiérrez García, M. Ramirez Nodal, A. Ruiz Lozano, C. R. Pérez Díaz, A. González Valdés, and L. Gonzalez Alvarez. 2020. Clinical relevance of Zika symptoms in the context of a Zika dengue epidemic. *Journal of Infection and Public Health* 13(2):173-176.
- Hulse, E. J., J. O. Davies, A. J. Simpson, A. M. Sciuto, and M. Eddleston. 2014. Respiratory complications of organophosphorus nerve agent and insecticide poisoning. Implications for respiratory and critical care. *American Journal of Respiratory and Critical Care Medicine* 190(12):1342-1354.
- Hurley, D. 2019. Was it an invisible attack on U.S. Diplomats, or something stranger? *The New York Times Magazine*. <https://www.nytimes.com/interactive/2019/05/15/magazine/diplomat-disorder.html> (accessed July 18, 2020).
- IEEE (Institute of Electrical and Electronics Engineers). 2019. IEEE standard for safety levels with respect to human exposure to electric, magnetic, and electromagnetic fields, 0 hz to 300 ghz. *IEEE Std C95.1-2019 (Revision of IEEE Std C95.1-2005/Incorporates IEEE Std C95.1-2019/Cor 1-2019)* 1-312.
- Ilhan, A., A. Gurel, F. Armutcu, S. Kamisli, M. Iraz, O. Akyol, and S. Ozen. 2004. Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain. *Clinica Chimica Acta* 340(1-2):153-162.
- Jacobsen P., and N. E. Ebbenhøj. 2016. Outbreak of mysterious illness in a hospital poisoning or iatrogenic induced mass psychogenic illness. *Journal of Emergency Medicine* 50:e47-e52.

- Jacobsen, P., and N. E. Ebbenhøj. 2017. Reply to Jansen et al. *Journal of Emergency Medicine* 52(4):581-583.
- Jansen, T., E. C. Jenson, U. B. Haastrup, K. Esperson. 2016. Comments on “Outbreak of mysterious illness in a hospital poisoning or iatrogenic induced mass psychogenic illness.” *Journal of Emergency Medicine* 52(4):581-583.
- Jeffrey, S. 2013. FDA approves first device to treat migraine pain. *Medscape Medical News*. <https://www.medscape.com/viewarticle/817831#:~:text=The%20US%20Food%20and%20Drug,by%20migraine%20headache%20with%20aura> (accessed July 18, 2020).
- Knight, J., T. Friedman, and J. Sulianti. 1965. Epidemic hysteria: A field study. *American Journal of Public Health* 55(6):858-865.
- Lai, H. 1994. Neurological effects of radio frequency electromagnetic radiation. In *Electromagnetic Fields in Living Systems*, Vol. 1, edited by J. C. Lin. New York: Plenum Press.
- Lannuzel, A., J. L. Fergé, Q. Lobjois, A. Signate, B. Rozé, B. Tressières, Y. Madec, P. Poullain, C. Herrmann, F. Najioullah, E. McGovern, A. C. Savidan, R. Valentino, S. Breurec, R. Césaire, E. Hirsch, P. M. Lledo, G. Thiery, A. Cabié, F. Lazarini, and E. Roze. 2019. Long-term outcome in neurozika: When biological diagnosis matters. *Neurology* 92(21):e2406-e2420.
- Lebovitz, R. M. 1973. Caloric vestibular stimulation via uhf-microwave irradiation. *IEEE Transactions on Biomedical Engineering* 20(2):119-126.
- Lessenger, J. E. 1992. Five office workers inadvertently exposed to cypermethrin. *Journal of Toxicology and Environmental Health* 35(4):261-267.
- London, L., V. Nell, M. L. Thompson, and J. E. Myers. 1998. Effects of long-term organophosphate exposures on neurological symptoms, vibration sense and tremor among South African farm workers. *Scandinavian Journal on Work, Environment, & Health* 24(1):18-29.
- Lustenberger, C., M. Murbach, R. Durr, M. R. Schmid, N. Kuster, P. Achermann, and R. Huber. 2013. Stimulation of the brain with radiofrequency electromagnetic field pulses affects sleep-dependent performance improvement. *Brain Stimulation* 6:805–811.
- Mehta, R., P. Gerardin, C. A. A. de Brito, C. N. Soares, M. L. B. Ferreira, and T. Solomon. 2018. The neurological complications of chikungunya virus: A systematic review. *Reviews in Medical Virology* 28(3):e1978.
- Mohan, G., T. P. Ayisha Hamna, A. J. Jijo, K. M. Saradha Devi, A. Narayanasamy, and B. Vellingiri. 2019. Recent advances in radiotherapy and its associated side effects in cancer—a review. *The Journal of Basic and Applied Zoology* 80(1):14.
- Müller-Mohnssen, H. 1999. Chronic sequelae and irreversible injuries following acute pyrethroid intoxication. *Toxicology Letters* 107(1-3):161-176.
- Naughton, S. X., and A. V. Terry, Jr. 2018. Neurotoxicity in acute and repeated organophosphate exposure. *Toxicology* 408:101-112.
- NTP (National Toxicology Program). 2018a. *Toxicology and carcinogenesis studies in B6C3F1/N mice exposed to whole-body radio frequency radiation at a frequency (1,900 mhz) and modulation (GSM and CDMA) used by cell phones*. National Institutes of Health. [https://ntp.niehs.nih.gov/ntp/about\\_ntp/trpanel/2018/march/tr596peerdraft.pdf](https://ntp.niehs.nih.gov/ntp/about_ntp/trpanel/2018/march/tr596peerdraft.pdf) (accessed July 27, 2020).
- NTP. 2018b. *Toxicology and carcinogenesis studies in HSD: Sprague Dawley SD rats exposed to whole-body radio frequency radiation at a frequency (900 mhz) and modulations (GSM and CDMA) used by cell phones*. National Institutes of Health. [https://ntp.niehs.nih.gov/ntp/htdocs/lt\\_rpts/tr595\\_508.pdf?utm\\_source=direct&utm\\_medium=prod&utm\\_campaign=ntpgolinks&utm\\_term=tr595](https://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr595_508.pdf?utm_source=direct&utm_medium=prod&utm_campaign=ntpgolinks&utm_term=tr595) (accessed July 27, 2020).
- NTP. 2019. *Systematic review of long-term neurological effects following acute exposure to the organophosphorus nerve agent sarin*. National Institutes of Health. [https://ntp.niehs.nih.gov/ntp/ohat/sarin/sarin\\_prepublication20190600\\_508.pdf](https://ntp.niehs.nih.gov/ntp/ohat/sarin/sarin_prepublication20190600_508.pdf) (accessed July 27, 2020).

- Pakhomov, A. G., and M. R. Murphy. 2000. A comprehensive review of the research on biological effects of pulsed radiofrequency radiation in Russia and the former Soviet Union. In *Electromagnetic Fields in Living Systems*, Vol. 3, edited by J. C. Lin. New York: Kluwer Academic/Plenum Publishers. Pp. 265-290.
- Pall, M. L. 2013. Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects. *Journal of Cellular and Molecular Medicine* 17(8):958-965.
- Pall, M. L. 2016. Microwave frequency electromagnetic fields (EMFs) produce widespread neuropsychiatric effects including depression. *Journal of Chemical Neuroanatomy* 75(Pt B):43-51.
- Pope, C. N. 1999. Organophosphorus pesticides: Do they all have the same mechanism of toxicity? *Journal of Toxicology and Environmental Health Part B: Critical Reviews* 2(2):161-181.
- Popkirov, S., J. Stone, and D. Holle-Lee. 2018. Treatment of persistent postural-perceptual dizziness (PPPD) and related disorders. *Current Treatment Options in Neurology* 20(12):50.
- Ramundo-Orlando, A. 2010. Effects of millimeter waves radiation on cell membrane—a brief review. *Journal of Infrared, Millimeter, and Terahertz Waves* 31(12):1400-1411.
- Raslear, T. G., Y. Akyel, F. Bates, M. Belt, and S. T. Lu. 1993. Temporal bisection in rats: The effects of high-peak-power pulsed microwave irradiation. *Bioelectromagnetics* 14:459-478.
- Richardson, J. R., V. Fitsanakis, R. H. S. Westerink, and A. G. Kanthasamy. 2019. Neurotoxicity of pesticides. *Acta Neuropathology* 138(3):343-362.
- Richter, E. D., P. Chuwers, Y. Levy, M. Gordon, F. Grauer, J. Marzouk, S. Levy, S. Barron, and N. Gruener. 1992. Health effects from exposure to organophosphate pesticides in workers and residents in Israel. *Israel Journal of Medical Sciences* 28(8-9):584-598.
- Roldan-Tapia, L., F. A. Nieto-Escamez, E. M. del Aguila, F. Laynez, T. Parron, and F. Sanchez-Santed. 2006. Neuropsychological sequelae from acute poisoning and long-term exposure to carbamate and organophosphate pesticides. *Neurotoxicology and Teratology* 28(6):694-703.
- Ross, S. M., I. C. McManus, V. Harrison, and O. Mason. 2013. Neurobehavioral problems following low-level exposure to organophosphate pesticides: A systematic and meta-analytic review. *Critical Reviews in Toxicology* 43(1):21-44.
- Saitz, T. R., M. J. Conlin, C. D. Tessier, and T. R. Hatch. 2019. The safety and efficacy of transurethral microwave therapy in high-risk catheter-dependent men. *Turkish Journal of Urology* 45(1):27-30.
- Salford, L. G., A. E. Brun, J. L. Eberhardt, L. Malmgren, and B. R. Persson. 2003. Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones. *Environmental Health Perspectives* 111(7):881-883; discussion A408.
- Sharp, J. C., H. M. Grove, and O. P. Ghandi. 1974. Generation of acoustic signals by pulsed microwave energy (letters). *IEEE Transactions on Microwave Theory and Techniques* 22:583-584.
- Staab, J. P. 2020. Persistent postural-perceptual dizziness. *Seminars in Neurology* 40(1):130-137.
- Staab, J. P., C. S. Fullerton, R. Ursano. 1999. A critical look at PTSD: Constructs, concepts, epidemiology, and implications. In *Response to Disaster: Psychosocial, Community, and Ecological Approaches* edited by R. Gist, and B. Lubin B.. Philadelphia, PA: Brunner/Mazel. Pp. 101-128.
- Staab, J. P., A. Eckhardt-Henn, A. Horii, R. Jacob, M. Strupp, T. Brandt, and A. Bronstein. 2017. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): Consensus document of the committee for the classification of vestibular disorders of the barany society. *Journal of Vestibular Research* 27(4):191-208.
- Stein, Y., and I. G. Udasin. 2020. Electromagnetic hypersensitivity (EHS, microwave syndrome)—review of mechanisms. *Environmental Research* 186:109445.
- Stone, J. 2016. Persistent posturo-perceptual dizziness (PPPD) (Functional Dizziness). [https://www.neurosymbols.org/download/i/mark\\_dl/u/4013612269/4634740784/Dizziness%20-%20PPPD%20-%20information%20sheet.pdf](https://www.neurosymbols.org/download/i/mark_dl/u/4013612269/4634740784/Dizziness%20-%20PPPD%20-%20information%20sheet.pdf) (accessed July 16, 2020).
- Steiner, U. E., and T. Ulrich. 1989. Magnetic field effects in chemical kinetics and related phenomena. *Chemical Reviews* 89(1):51-147.



- Suh, J. H., R. Kotecha, S. T. Chao, M. S. Ahluwalia, A. Sahgal, and E. L. Chang. 2020. Current approaches to the management of brain metastases. *Nature Reviews Clinical Oncology* 17(5):279-299.
- Tan, S., H. Wang, X. Xu, L. Zhao, J. Zhang, J. Dong, B. Yao, H. Wang, H. Zhou, Y. Gao, and R. Peng. 2017. Study on dose-dependent, frequency-dependent, and accumulative effects of 1.5 ghz and 2.856 ghz microwave on cognitive functions in wistar rats. *Scientific Reports* 7(1):10781.
- Teixeira, C. F., L. Giraldo Da Silva Augusto, and T. C. Morata. 2002. Occupational exposure to insecticides and their effects on the auditory system. *Noise Health* 4(14):31-39.
- Tsao, M. N., W. Xu, R. K. Wong, N. Lloyd, N. Laperriere, A. Sahgal, E. Rakovitch, and E. Chow. 2018. Whole brain radiotherapy for the treatment of newly diagnosed multiple brain metastases. *Cochrane Database of Systematic Reviews* 1:CD003869.
- Xu, H., Y. Mao, and B. Xu. 2020. Association between pyrethroid pesticide exposure and hearing loss in adolescents. *Environmental Research* 187:109640.
- Zeigelboim, B. S., J. S. Malisky, M. R. D. Rosa, A. B. M. Lacerda, P. S. Alcaraz, and V. R. Fonseca. 2019. The importance of otoneurological evaluation in Brazilian workers exposed to pesticides: A preliminary study. *International Archives of Otorhinolaryngology* 23(4):e389-e395.
- Zhao, T. Y., S. P. Zou, and P. E. Knapp. 2007. Exposure to cell phone radiation up-regulates apoptosis genes in primary cultures of neurons and astrocytes. *Neuroscience Letters* 412(1):34-38.
- Zirix, J. M., D. Hatcher, M. E. Belt, J. Roe, A. Thomas, P. Henry, M. Tovias, and J. A. D'Andrea. 1999. High peak power, low SAR effects on memory task performance in rhesus monkeys. In *Electricity and Magnetism in Biology and Medicine*, edited by F. Bersani. New York: Springer Science+Business Media. Pp. 621-624.



## Section 5

### Acute Treatment and Rehabilitation

#### SOURCES OF INFORMATION

The committee's Statement of Task requested an "assessment of treatment options." However, as discussed above, individual-specific data on treatment and clinical outcomes were not readily available to the committee. It was privy to anecdotal information about the treatment of some individuals, but this information was not standardized or systematic. The approach to initial treatment and rehabilitation was also complicated by a number of factors including remote locations and difficult access to specialized care, diverse clinical presentations by affected personnel, and a variety of reporting schemes at different times. It is beyond the scope of this report to assess the efficacy of specific treatments of individuals. It is important to note that in most injuries to the brain or vestibular system, poorly understood recovery or compensatory mechanisms lead to functional improvement over time. Non-pharmacologic therapies targeted at the person's deficit enhance or quicken the process of functional improvement.

#### Acute Treatment

The information available to the committee about the acute treatment of Department of State (DOS) personnel derived from direct testimony to it by affected personnel, summative reports in the literature, or information provided to it by the clinical investigators. In general, the personnel were treated acutely with rest and instructions to avoid the circumstances associated with the initial signs and symptoms. In the absence of recovery and out of an abundance of concern, some personnel were transferred to Miami where further evaluation was performed. Very limited information on treatment that occurred at that time was available to the committee.

#### Chronic Treatment

The data available to the committee on chronic treatment derived from direct testimony to it by several affected personnel, reports in the literature, and a presentation by Penn clinicians involved in the care of the affected DOS employees and their families. In general, the data were presented in summary form without specific details. For example, the granularity of the detail was at the level of general cognitive, neuro-optometric, vestibular and vocational rehabilitation interventions. Some more specific information is found on page 1130 of the publication by Swanson et al. (2018). The committee lacked specific information on patient-specific treatment approaches and responses, which would have helped in generating recommendations on potential alternatives. However, information made available to the committee suggested that affected individuals did improve after referral to vestibular or cognitive therapy.

Given these limitations, the committee focused its efforts instead on a review of the current state-of-the art in neurological, vestibular, and neuropsychological rehabilitation, with the goal of offering general guidance (this section) and recommendations (see Section 6) for treatment of patients with unexplained neurological or other medical manifestations like these in the future.

**ASSESSMENT AND FINDINGS**

The committee distinguishes observations related to care of the acute syndrome from those for longer-term care of the chronic syndrome associated with the Embassy employees in Havana.

**Acute Treatment**

In the case of acute symptoms that are thought to be caused by injury to the central nervous system, it is important that expert clinicians perform an evaluation as early as possible after the onset of the illness. This can include immediate care by on-site medical personnel, but it is unlikely that such personnel will be expert in the assessment of neurological injuries. In addition, although some level of care can and should be provided by telemedicine, it is very likely that parts of the neurological examination and much of the required early testing will not be feasible on-site. Hence, both to avoid further injury and to insure the most rapid access to expert care, there should be plans in place, as needed, for each embassy to remove the affected individuals to a protected site where this evaluation can be done. The actual indicated treatment will be specific to the type of injury that has been sustained. If removing personnel is required for evaluation, this evaluation should be completed as soon as possible and then personnel should be returned to whatever is the most supportive environment. Rehabilitation will also be guided by evidence of damage to specific parts of the nervous system.

**Chronic Treatment**

The absence of a known cause of the symptoms and signs in the affected DOS employees complicates initial treatment and early rehabilitation to some degree. However, it is not unusual for patients to develop chronic nonspecific symptoms (e.g., fatigue, dizziness) following acute medical events such as mild traumatic brain injuries, acute peripheral vestibulopathies, cardiovascular or cerebrovascular events, cancer treatments, and complex surgical procedures, which may differ among individuals (Donnell et al., 2012; Gunstad and Suhr, 2001; Julien et al., 2017; Pavawalla et al., 2013; Polusny et al., 2011; Voormolen et al., 2019) despite the same exposure and mechanism of injury (Collins et al., 2016; Gardner et al., 2019; Nelson et al., 2019; Si et al., 2018). Thus, even in the setting of an identified precipitant, it is not always clear that the acute event and the chronic symptoms are directly related. Nevertheless, the absence of a known cause or mechanism does not diminish the value or relevance of management guidelines. Accordingly, the committee offers general guidance below on the future management of patients with clinical presentations similar to those of DOS Embassy employees; for example, as commonly seen in individuals suffering from mild traumatic brain injury (Bomyea et al., 2019; Chen et al., 2019; Iverson et al., 2015; Kontos et al., 2018, 2019, 2020; Sweeney et al., 2020; Yue et al., 2019).

1. **Early evaluation and treatment are essential.** A significant body of literature demonstrates that the earlier an evaluation is undertaken and treatment initiated, the better the outcome (Belanger et al., 2015; Lacour and Bernard-Demanze, 2014; Lacour et al., 1976, 2020; Mittenberg et al., 1996; Ponsford et al., 2002). In general, early treatment can prevent development of chronic neurological conditions (Gil-Jardine et al., 2018; Mittenberg et al., 1996; Ponsford et al., 2002; Seabury et al., 2018; Snell et al., 2009; Twamley et al., 2015; Wade et al., 1998), which are much more difficult to treat (Hiploylee et al., 2017; Perry et al., 2016; Snell et al., 2009, 2019).

2. **Avoid removing personnel from supportive environments.** A supportive environment contributes to treatment success in rehabilitation of chronic symptoms that develop after brain injury or other acute medical events such as those listed above (Polich et al., 2020; Vanderploeg et al., 2018). Relocation of affected personnel to a site for chronic rehabilitation without providing for social supports, such as family or close colleagues, can result in social isolation and exacerbate anxiety. The committee heard accounts of such.
3. **Initial treatment should emphasize early assessment, education, and return to activity.** Proactive therapeutic interventions, sleep hygiene and exercise are simple but helpful measures.
4. **Chronic neurological syndromes require a multi-disciplinary approach.** Treatment of chronic syndromes may require more intensive treatment. The length of time to recovery may be months, and some residual symptoms may persist. Experience with persistent postural-perceptual dizziness (PPPD) provides useful lessons.

The diagnosis of PPPD in some Havana cases informs prognosis (Popkirov et al., 2018; Staab, 2020). With a well-integrated, multi-disciplinary treatment plan of physical and psychological therapies and medication provided over a course of 3-6 months, most patients with PPPD are able to achieve a reduction in symptoms to a level at which they are not impaired in their performance of routine daily activities inside or outside the home. Despite improvement, they may remain vulnerable to temporary (hours to days) exacerbations of symptoms on exposure to provocative stimuli such as extensive physical activity or motion-rich environments. Approximately 10-20 percent of patients in the general population who have had PPPD for more than 4 years remain work-disabled by their symptoms even after achieving their maximal response to currently available treatments (Schaaf and Hesse, 2015; Trinidad and Goebel, 2018).

As described previously, psychological disorders may develop as secondary complications of acute illnesses, regardless of cause. Data provided by the National Institutes of Health (NIH) team suggested that a considerable number of patients with ongoing physical symptoms may be experiencing clinically significant psychological distress. The most likely causes of these symptoms are secondary depressive, anxiety, traumatic stress, or somatic symptom disorders. The committee had no individual patient-level data with which to reach any conclusions about the presence of these conditions among patients with persistent symptoms. However, all of these are treatable conditions, which means that proper diagnosis and application of scientifically validated therapies could lead to a reduction in morbidity and improvements in functioning among affected individuals, regardless of the nature or cause of the initial illness or the presence of co-existing chronic conditions.

5. **Understand the phenotype.** Personal characteristics and situational and biological differences may all impact approach and response to treatment. In general, phenotypes that predict a more successful response to neurological rehabilitation include higher level of education, resilience in other settings, and job satisfaction. Negative prognostic features include stressful job and life situation, previous history of depression, and anxiety. Significant efforts are under way to identify genetic and other factors that may affect responsiveness to neurological rehabilitation. Further efforts to link mechanism of injury with clinical manifestations (phenotype), and

- selection of and response to different treatment modalities will be important. The committee anticipates that a telemedicine team (see Section 6) assembled to help locally available clinicians in situations like those faced by the affected DOS employees would understand this evolving literature and adapt their approaches to the latest interventions available.
6. **Testing and therapies without established validation should be for research purposes only.** The committee was asked to comment on the use of diffusion tensor imaging to establish brain injury and on visual rehabilitation for vestibular symptoms after brain injury in DOS personnel. As noted in Section 3, the findings with diffusion tensor imaging did not convince the committee of the validity of this approach as a diagnostic method, although it may have value as a research method in future events. Visual training therapy is widely practiced for patients with vestibular symptoms, but the evidence base for this type of treatment remains controversial (Barton and Ranalli, 2020). Hence, while there is not adequate evidence to recommend such therapies for routine treatment of patients suffering from traumatic brain injury (TBI) or symptoms similar to TBI, the committee concluded that if clinicians based on their judgment wanted to offer them to individual patients, that the patients should be informed that the therapy was not supported by adequate evidence, but that there were reports of it being helpful in some patients.
  7. **Interdisciplinary consultation.** The committee observed that a potential shortcoming of the medical evaluations and treatment received by DOS patients was that they were provided by teams focused on one area of clinical medicine (e.g., otology in Miami, brain injury at Penn). When symptoms are as enigmatic as those experienced by DOS patients, early involvement of a broader group of clinical specialists, paired with experts in epidemiology and environmental exposures (e.g., toxicologists, radio frequency engineers, and others as determined by the situation) would reduce the chances that early clues about causality would be missed or that premature conclusions would be drawn about potential diagnoses. These additional experts could contribute in person or virtually.

## SUMMARY

The committee lacked information on patient-specific treatment approaches and responses, which would have helped in generating recommendations on potential alternatives. In reviewing best practices in neurological rehabilitation, the committee finds that early evaluation and treatment are essential for optimal outcomes, a supportive environment is important, and an interdisciplinary approach for rehabilitation of chronic neurological conditions is best, as are appropriate and early education and realistic expectation-setting.

## REFERENCES

- Barton, J. J. S., and P. Ranalli. 2020. Vision therapy: Ocular motor training in mild traumatic brain injury. *Annals of Neurology*. doi.org/10.1002/ana.25820.
- Belanger, H. G., F. Barwick, M. A. Silva, T. Kretzmer, K. E. Kip, and R. D. Vanderploeg. 2015. Web-based psychoeducational intervention for postconcussion symptoms: A randomized trial. *Military Medicine* 180(2):192-200.
- Bomyea, J., L. A. Flashman, R. Zafonte, N. Andaluz, R. Coimbra, M. S. George, G. A. Grant, C. E. Marx, T. W. McAllister, L. Shutter, A. J. Lang, and M. B. Stein. 2019. Associations between

- neuropsychiatric and health status outcomes in individuals with probable mTBI. *Psychiatry Research* 272:531-539.
- Chen, J., B. Oddson, and H. C. Gilbert. 2019. Differential effect of recurrent concussions on symptom clusters in sport concussion assessment tool. *Journal of Sports Rehabilitation* 28(7):735-739.
- Collins, M. W., A. P. Kontos, D. O. Okonkwo, J. Almquist, J. Bailes, M. Barisa, J. Bazarian, O. J. Bloom, D. L. Brody, R. Cantu, J. Cardenas, J. Clugston, R. Cohen, R. Echemendia, R. J. Elbin, R. Ellenbogen, J. Fonseca, G. Gioia, K. Guskiewicz, R. Heyer, G. Hotz, G. L. Iverson, B. Jordan, G. Manley, J. Maroon, T. McAllister, M. McCrea, A. Mucha, E. Pieroth, K. Podell, M. Pombo, T. Shetty, A. Sills, G. Solomon, D. G. Thomas, T. C. Valovich McLeod, T. Yates, and R. Zafonte. 2016. Statements of agreement from the targeted evaluation and active management (TEAM) approaches to treating concussion meeting held in Pittsburgh, October 15-16, 2015. *Neurosurgery* 79(6):912-929.
- Donnell, A. J., M. S. Kim, M. A. Silva, and R. D. Vanderploeg. 2012. Incidence of postconcussion symptoms in psychiatric diagnostic groups, mild traumatic brain injury, and comorbid conditions. *Clinical Neuropsychology* 26(7):1092-1101.
- Gardner, R. C., J. Cheng, A. R. Ferguson, R. Boylan, J. Boscardin, R. D. Zafonte, G. T. Manley, and I. Citicoline Brain Injury Treatment Trial. 2019. Divergent six month functional recovery trajectories and predictors after traumatic brain injury: Novel insights from the citicoline brain injury treatment trial study. *Journal of Neurotrauma* 36(17):2521-2532.
- Gil-Jardine, C., S. Al Joboory, J. T. S. Jammes, G. Durand, R. Ribereau-Gayon, M. Galinski, L. R. Salmi, P. Revel, C. A. Regis, G. Valdenaire, E. Poulet, K. Tazarourte, and E. Lagarde. 2018. Prevention of post-concussion-like symptoms in patients presenting at the emergency room, early single eye movement desensitization, and reprocessing intervention versus usual care: Study protocol for a two-center randomized controlled trial. *Trials* 19(1):555.
- Gunstad, J., and J. A. Suhr. 2001. "Expectation as etiology" versus "the good old days": Postconcussion syndrome symptom reporting in athletes, headache sufferers, and depressed individuals. *Journal of the International Neuropsychology Society* 7(3):323-333.
- Hiploylee, C., P. A. Dufort, H. S. Davis, R. A. Wennberg, M. C. Tartaglia, D. Mikulis, L. N. Hazrati, and C. H. Tator. 2017. Longitudinal study of postconcussion syndrome: Not everyone recovers. *Journal of Neurotrauma* 34(8):1511-1523.
- Iverson, G. L., N. D. Silverberg, R. Mannix, B. A. Maxwell, J. E. Atkins, R. Zafonte, and P. D. Berkner. 2015. Factors associated with concussion-like symptom reporting in high school athletes. *JAMA Pediatrics* 169(12):1132-1140.
- Julien, J., S. Tinawi, K. Anderson, L. C. Frenette, H. Audrit, M. C. Ferland, M. Feyz, and E. De Guise. 2017. Highlighting the differences in post-traumatic symptoms between patients with complicated and uncomplicated mild traumatic brain injury and injured controls. *Brain Injury* 31(13-14):1846-1855.
- Kontos, A. P., M. W. Collins, C. L. Holland, V. L. Reeves, K. Edelman, S. Benso, W. Schneider, and D. Okonkwo. 2018. Preliminary evidence for improvement in symptoms, cognitive, vestibular, and oculomotor outcomes following targeted intervention with chronic mTBI patients. *Military Medicine* 183(Suppl\_1):333-338.
- Kontos, A. P., A. Sufrinko, N. Sandel, K. Emami, and M. W. Collins. 2019. Sport-related concussion clinical profiles: Clinical characteristics, targeted treatments, and preliminary evidence. *Current Sports Medicine Reports* 18(3):82-92.
- Kontos, A. P., R. J. Elbin, A. Trbovich, M. Womble, A. Said, V. F. Sumrok, J. French, N. Kegel, A. Puskas, N. Sherry, C. Holland, and M. Collins. 2020. Concussion clinical profiles screening (CP screen) tool: Preliminary evidence to inform a multidisciplinary approach. *Neurosurgery* 87(2):348-356.
- Lacour, M., and L. Bernard-Demanze. 2014. Interaction between vestibular compensation mechanisms and vestibular rehabilitation therapy: 10 recommendations for optimal functional recovery. *Frontiers in Neurology* 5:285.



- Lacour, M., J. P. Roll, and M. Appaix. 1976. Modifications and development of spinal reflexes in the alert baboon (*Papio papio*) following an unilateral vestibular neurotomy. *Brain Research* 113(2):255-269.
- Lacour, M., T. Laurent, and T. Alain. 2020. Rehabilitation of dynamic visual acuity in patients with unilateral vestibular hypofunction: earlier is better. *European Archives of Otorhinolaryngology* 277(1):103-113.
- Mittenberg, W., G. Tremont, R. E. Zielinski, S. Fichera, and K. R. Rayls. 1996. Cognitive-behavioral prevention of postconcussion syndrome. *Archives of Clinical Neuropsychology* 11(2):139-145.
- Nelson, L. D., N. R. Temkin, S. Dikmen, J. Barber, J. T. Giacino, E. Yuh, H. S. Levin, M. A. McCrea, M. B. Stein, P. Mukherjee, D. O. Okonkwo, R. Diaz-Arrastia, G. T. Manley, O. Adeoye, N. Badjatia, K. Boase, Y. Bodien, M. R. Bullock, R. Chesnut, J. D. Corrigan, K. Crawford, Mis, A. C. Duhaime, R. Ellenbogen, V. R. Feeser, A. Ferguson, B. Foreman, R. Gardner, E. Gaudette, L. Gonzalez, S. Gopinath, R. Gullapalli, J. C. Hemphill, G. Hotz, S. Jain, F. Korley, J. Kramer, N. Kreitzer, C. Lindsell, J. Machamer, C. Madden, A. Martin, T. McAllister, R. Merchant, F. Noel, E. Palacios, D. Perl, A. Puccio, M. Rabinowitz, C. S. Robertson, J. Rosand, A. Sander, G. Satris, D. Schnyer, S. Seabury, M. Sherer, S. Taylor, A. Toga, A. Valadka, M. J. Vassar, P. Vespa, K. Wang, J. K. Yue, and R. Zafonte. 2019. Recovery after mild traumatic brain injury in patients presenting to US level I trauma centers: A Transforming Research And Clinical Knowledge in Traumatic Brain injury (TRACK-TBI) study. *JAMA Neurology* 76(9):1049-1059.
- Pavawalla, S. P., R. Salazar, C. Cimino, H. G. Belanger, and R. D. Vanderploeg. 2013. An exploration of diagnosis threat and group identification following concussion injury. *Journal of the International Neuropsychology Society* 19(3):305-313.
- Perry, D. C., V. E. Sturm, M. J. Peterson, C. F. Pieper, T. Bullock, B. F. Boeve, B. L. Miller, K. M. Guskiewicz, M. S. Berger, J. H. Kramer, and K. A. Welsh-Bohmer. 2016. Association of traumatic brain injury with subsequent neurological and psychiatric disease: A meta-analysis. *Journal of Neurosurgery* 124(2):511-526.
- Polich, G., M. A. Iaccarino, T. J. Kaptchuk, L. Morales-Quezada, and R. Zafonte. 2020. Nocebo effects in concussion: Is all that is told beneficial? *American Journal of Physical Medicine & Rehabilitation* 99(1):71-80.
- Polusny, M. A., S. M. Kehle, N. W. Nelson, C. R. Erbes, P. A. Arbisi, and P. Thuras. 2011. Longitudinal effects of mild traumatic brain injury and posttraumatic stress disorder comorbidity on postdeployment outcomes in National Guard soldiers deployed to Iraq. *Archives of General Psychiatry* 68(1):79-89.
- Ponsford, J., C. Willmott, A. Rothwell, P. Cameron, A. M. Kelly, R. Nelms, and C. Curran. 2002. Impact of early intervention on outcome following mild head injury in adults. *Journal of Neurology, Neurosurgery and Psychiatry* 73(3):330-332.
- Popkirov, S., J. Stone, and D. Holle-Lee. 2018. Treatment of persistent postural-perceptual dizziness (PPPD) and related disorders. *Current Treatment Options in Neurology* 20(12):50.
- Schaaf, H., and G. Hesse. 2015. Patients with long-lasting dizziness: A follow-up after neurotological and psychotherapeutic inpatient treatment after a period of at least 1 year. *European Archives of Otorhinolaryngology* 272:1529-1535.
- Seabury, S. A., E. Gaudette, D. P. Goldman, A. J. Markowitz, J. Brooks, M. A. McCrea, D. O. Okonkwo, G. T. Manley, O. Adeoye, N. Badjatia, K. Boase, Y. Bodien, M. R. Bullock, R. Chesnut, J. D. Corrigan, K. Crawford, R. Diaz-Arrastia, S. Dikmen, A. C. Duhaime, R. Ellenbogen, V. R. Feeser, A. Ferguson, B. Foreman, R. Gardner, J. Giacino, L. Gonzalez, S. Gopinath, R. Gullapalli, J. C. Hemphill, G. Hotz, S. Jain, F. Korley, J. Kramer, N. Kreitzer, H. Levin, C. Lindsell, J. Machamer, C. Madden, A. Martin, T. McAllister, R. Merchant, P. Mukherjee, L. Nelson, F. Noel, E. Palacios, D. Perl, A. Puccio, M. Rabinowitz, C. Robertson, J. Rosand, A. Sander, G. Satris, D. Schnyer, M. Sherer, M. Stein, S. Taylor, N. Temkin, A. Toga, A. Valadka, M. Vassar, P. Vespa, K. Wang, J. Yue, E. Yuh, and R. Zafonte. 2018. Assessment of follow-up care after emergency department presentation for mild traumatic brain injury and concussion: Results from the TRACK-TBI study. *JAMA* 1(1):e180210.

- Si, B., G. Dumkrieger, T. Wu, R. Zafonte, A. B. Valadka, D. O. Okonkwo, G. T. Manley, L. Wang, D. W. Dodick, T. J. Schwedt, and J. Li. 2018. Sub-classifying patients with mild traumatic brain injury: A clustering approach based on baseline clinical characteristics and 90-day and 180-day outcomes. *PLoS One* 13(7):e0198741.
- Snell, D. L., L. J. Surgenor, E. J. Hay-Smith, and R. J. Siegert. 2009. A systematic review of psychological treatments for mild traumatic brain injury: An update on the evidence. *Journal of Clinical and Experimental Neuropsychology* 31(1):20-38.
- Staab, J. P. 2020. Persistent postural-perceptual dizziness. *Seminars in Neurology* 40(1):130-137.
- Stein, M. B., S. Jain, J. T. Giacino, H. Levin, S. Dikmen, L. D. Nelson, M. J. Vassar, D. O. Okonkwo, R. Diaz-Arrastia, C. S. Robertson, P. Mukherjee, M. McCrea, C. L. Mac Donald, J. K. Yue, E. Yuh, X. Sun, L. Campbell-Sills, N. Temkin, G. T. Manley, O. Adeoye, N. Badjatia, K. Boase, Y. Bodien, M. R. Bullock, R. Chesnut, J. D. Corrigan, K. Crawford, R. Diaz-Arrastia, S. Dikmen, A. C. Duhaime, R. Ellenbogen, V. R. Feeser, A. Ferguson, B. Foreman, R. Gardner, E. Gaudette, J. T. Giacino, L. Gonzalez, S. Gopinath, R. Gullapalli, J. C. Hemphill, G. Hotz, S. Jain, F. Korley, J. Kramer, N. Kreitzer, H. Levin, C. Lindsell, J. Machamer, C. Madden, A. Martin, T. McAllister, M. McCrea, R. Merchant, P. Mukherjee, L. D. Nelson, F. Noel, D. O. Okonkwo, E. Palacios, D. Perl, A. Puccio, M. Rabinowitz, C. S. Robertson, J. Rosand, A. Sander, G. Satris, D. Schnyer, S. Seabury, M. Sherer, M. B. Stein, S. Taylor, A. Toga, N. Temkin, A. Valadka, M. J. Vassar, P. Vespa, K. Wang, J. K. Yue, E. Yuh, and R. Zafonte. 2019. Risk of posttraumatic stress disorder and major depression in civilian patients after mild traumatic brain injury: A TRACK-TBI study. *JAMA Psychiatry* 76(3):249-258.
- Swanson, R. L., 2nd, S. Hampton, J. Green-McKenzie, R. Diaz-Arrastia, M. S. Grady, R. Verma, R. Biester, D. Duda, R. L. Wolf, and D. H. Smith. 2018. Neurological manifestations among US government personnel reporting directional audible and sensory phenomena in Havana, Cuba. *JAMA* 319(11):1125-1133.
- Sweeney, E. A., J. C. Wilson, M. N. Potter, K. S. Dahab, K. L. Denay, and D. R. Howell. 2020. Symptom profiles and postural control after concussion in female artistic athletes. *Brain Injury* 34(7):928-933.
- Trinidad, A., and J. A. Goebel. 2018. Persistent postural-perceptual dizziness—A systemic review of the literature for the balance specialist. *Otology and Neurotology* 39:1291-1303.
- Twamley, E. W., K. R. Thomas, A. M. Gregory, A. J. Jak, M. W. Bondi, D. C. Delis, and J. B. Lohr. 2015. Cogsmart compensatory cognitive training for traumatic brain injury: Effects over 1 year. *Journal of Head Trauma Rehabilitation* 30(6):391-401.
- Vanderploeg, R. D., D. B. Cooper, G. Curtiss, J. E. Kennedy, D. F. Tate, and A. O. Bowles. 2018. Predicting treatment response to cognitive rehabilitation in military service members with mild traumatic brain injury. *Rehabilitation Psychology* 63(2):194-204.
- Voormolen, D. C., M. C. Cnossen, S. Polinder, B. Y. Gravesteyn, N. Von Steinbuechel, R. G. L. Real, and J. A. Haagsma. 2019. Prevalence of post-concussion-like symptoms in the general population in Italy, the Netherlands and the United Kingdom. *Brain Injury* 33(8):1078-1086.
- Wade, D. T., N. S. King, F. J. Wenden, S. Crawford, and F. E. Caldwell. 1998. Routine follow up after head injury: A second randomised controlled trial. *Journal of Neurology, Neurosurgery and Psychiatry* 65(2):177-183.
- Yue, J. K., M. C. Cnossen, E. A. Winkler, H. Deng, R. R. L. Phelps, N. A. Coss, S. Sharma, C. K. Robinson, C. G. Suen, M. J. Vassar, D. M. Schnyer, A. M. Puccio, R. C. Gardner, E. L. Yuh, P. Mukherjee, A. B. Valadka, D. O. Okonkwo, H. F. Lingsma, G. T. Manley, S. R. Cooper, K. Dams-O'Connor, W. A. Gordon, A. J. Hricik, A. I. R. Maas, D. K. Menon, and D. J. Morabito. 2019. Pre-injury comorbidities are associated with functional impairment and post-concussive symptoms at 3- and 6-months after mild traumatic brain injury: A TRACK-TBI study. *Frontiers in Neurology* 10:343.

## Section 6

### Looking to the Future and Recommendations

The committee recognizes that Department of State (DOS) employees serve a vital role to the country and are vulnerable to a wide range of potential threats. Their health and well-being is a national imperative. The committee believes that DOS has a tripartite ethical obligation to safeguard the well-being of deployed personnel. This entails the prompt identification of threats, expeditious diagnosis and treatment, and the provision of rehabilitation and long-term care for service-related injuries. The committee believes that this is an enduring fiduciary responsibility of DOS much like that afforded to military service members and others who have sustained injuries or disabilities in the performance of their government duties.

Clarity about the nature of the illnesses that first began to affect DOS employees in Cuba in 2016 and subsequently in China, and the causative mechanism(s), remains elusive. What is clear is that a distinct set of unusual clinical manifestations occurred abruptly in some individuals at the onset of their illness, and that the illness became chronic and debilitating for some, but not for all individuals. It is also clear that there is significant heterogeneity among a larger population of affected employees; some did not experience the distinct set of manifestations at onset, and some have had only nonspecific common manifestations. This heterogeneity may reflect evolution of the illness over time, multiple mechanisms at play within and between individuals, and the varying methods used to investigate these individuals at different clinical study sites.

Among the plausible mechanisms that the committee considered, directed radio frequency (RF) energy, especially in those with the distinct early manifestations, appears most germane, along with persistent postural perceptual dizziness (PPPD) as a secondary reinforcing mechanism, as well as the additive effects of psychological conditions. The committee cannot rule out other possible mechanisms (see Section 4), and again, considers it likely that a multiplicity of factors explains some cases and the differences between others. Commencement of appropriate neurological rehabilitation methods early in these illnesses, even without a diagnosis, would have been helpful.

The committee recognizes the impossibility of going back in time to examine the affected individuals early in their illness, gather evidence for or against any of the possible mechanisms, and begin treatment. The committee and others are limited today in what can be pieced together about these cases. However, the committee believes that it would be useful, and even imperative, for actions to be taken now in anticipation of future cases. Although these future cases may resemble in some fashion those that began in Cuba and China in 2016-2018, they need not be similar. Early in a future “event,” cases may not be identifiable as such, and the existence of an event worthy of attention may not be initially obvious. Planning should accommodate all of these possibilities.

The committee’s purpose is both to respond to the needs of U.S. government employees in the wake of the experience in Cuba and China as well as to anticipate future threats to their well-being. While the committee clearly needs to understand these events in order to be able to respond to a recurrence, the larger issue is preparedness for new and unknown threats that might compromise the health and safety of U.S. diplomats serving abroad. It is not enough to design a plan that prepares DOS for the past. The emergence of the novel pathogen SARS-CoV-2 is a

stark reminder that DOS must be thoughtful and creative in its anticipation of future threats, both natural and human-made, and agile in its response. To that end, the committee proposes a number of recommendations in order to enhance future responses.

**Recommendation 1. The Department of State should expand its collection of baseline and longitudinal data and biological specimens from all personnel prior to and during overseas assignments.**

A major limitation to establishing associative or causal health effects among DOS personnel assigned to Cuba or China was the lack of pre-exposure or baseline health status. It is critical to identify changes in the health of embassy personnel from their baseline if a threat occurs. To make these determinations, medical staff who collect necessary information at the time of the incident must have access to baseline information on the affected individuals. Therefore, for surveillance, the committee believes that there should be routine data collection for all DOS employees on foreign assignments, including collection of whole blood, plasma, and urine, as well as general medical and neurological examinations. Given the nature of the symptoms of the Havana personnel, baseline visual and auditory examinations would be useful, at least for personnel assigned to locations where similar kinds of events might take place. Baselines should be updated regularly, and whenever a significant medical or environmental event occurs. For the affected cohort of DOS employees, DOS should establish ongoing registries to identify any late-onset symptomatology or illness attributable to exposures at post.

The Acquired Brain Injury Tool (ABIT) is a clinical assessment tool that is currently used by DOS pre- and post-deployment to inventory the same neurological, vestibular, and auditory symptoms that were identified in DOS personnel in Cuba and China. However, given that the committee does not know the nature of future events, it would be wise to revise it and include symptoms beyond those encountered in Cuba and China. Establishing a pre-deployment baseline is key.

In addition, to be of maximal value, the committee suggests that the ABIT—and other assessment tools—be modified to obtain relevant epidemiological data such as physical location and local environmental parameters at the time of symptom onset to identify potential sources of threat. In order to ensure the sensitivity and specificity of the evaluation of personnel overseas, the committee recommends that the ABIT be reviewed on a periodic basis by an expert panel of physicians and scientists who can keep the assessment tool forward-facing with respect to present and future threats. The committee suggests that this panel include specialists in general internal medicine, neurology, infectious disease, physiatry, tropical medicine, pharmacology, toxicology, biostatistics, epidemiology, environmental health, bioterrorism, and psychiatry, so as to gather information on natural and human-made threats that might affect embassy personnel. The committee recommends that revisions be based on evolving epidemiological patterns or knowledge of potential malign threats. In addition to the data currently collected, the committee strongly recommends collecting data on prior brain injury and additional assessment of prior and ongoing psychiatric symptoms that might be primary or secondary sequelae from a novel threat.

**Recommendation 2. The Department of State, with support from the U.S. government, should establish plans and protocols now to enable**



**comprehensive, expeditious public health and research investigations in the future, should a cluster of new cases warrant investigation.**

In considering needs for a response to future DOS cases, it is important to differentiate between personal medical care (which must remain private), research (which must remain voluntary), and the public health necessity for evaluating information on individuals in a way that may impinge on their privacy in order to protect the health of other embassy personnel or their future well-being should there be a public health threat. It appears that the Centers for Disease Control and Prevention (CDC) assumed this public health role with respect to the cases from Havana, but only did so beginning 1 year after discovery of the first case, when there was much less to be gained from their actions. The committee recommends that a similar response be prepared and authorized in advance of the next potential set of cases, so that the necessary collection of information for a proper public health investigation of U.S. Embassy employees can be undertaken in a timely fashion and made available immediately. It is critical that these protocols be developed in an open and transparent manner with the Foreign Service Officer (FSO) community in order to build and maintain trust. To assist with the aforementioned surveillance, DOS should receive increased resources for MED HART (DOS Bureau of Medical Services Health Alert Response Threat) in order to allow for more timely and agile responses to unexpected and novel threats to personnel. The committee notes that while MED HART was intended to provide operational medical support, it was not necessarily designed to perform epidemiological surveillance and analysis in an effort to identify new case clusters in real-time. An occupational health surveillance system that allows DOS to identify high-risk populations and worksites, emerging work-associated problems, hazardous conditions and exposures, and that can target and evaluate interventions, as outlined in the 2018 National Academies report on occupational safety and health, would benefit overseas DOS locations (NASEM, 2018). DOS should also be provided resources to create such an occupational health surveillance system that could provide ready access to information should an investigation need to be launched. The committee suggests that DOS utilize an expert panel to provide advice on the collection of routine medical data.

In addition to the information that may be necessary to counter a public health emergency involving Embassy personnel, a research investigation may be needed. Participation by Embassy personnel in this type of investigation should be subject to the same human subjects protection rules that apply to all human subjects research. In considering the lessons learned regarding the ethical conduct of environmental research following the *Deepwater Horizon* oil spill by the Gulf Long-term Follow-up Study (GuLF STUDY) (Resnik et al., 2015), the committee urges DOS to prepare in advance for the conduct of human subjects research that might pertain to an unexpected health hazard. The GuLF STUDY investigators recommended that investigators identify an Institutional Review Board (IRB) and be ready to engage in research when and if it became necessary. To that end, the expert panel described here could also inform the training of research teams that could be deployed and it could facilitate the engagement of the broader DOS community, whose trust will be necessary for the conduct of research. Protocols and consent forms could be developed in advance, and revised and modified to account for the specific populations, threats, and urgencies involved in a particular emergency. Such an approach would facilitate a rapid response in an emergency and the collection of data that might be unavailable if usual research approvals had to be initiated coincident with the emergence of a threat. Protocols should stipulate a longitudinal design so that subjects can be followed over time and clinical outcomes captured. The GuLF STUDY investigators strongly recommended this proactive



approach to “ensure adequate review by IRBs and other groups of complex ethical issues without jeopardizing rapid response to a public health emergency” (Resnik et al., 2015, p. A230). The committee believes such attention to anticipatory governance will enable the expeditious and systematic collection of data necessary to elucidate novel threats while ensuring human subjects protections.

**Recommendation 3. Following the identification of a possible new case cluster, the Department of State should ensure collection of data critical for an effective investigation.**

In addition to the collection of data pertaining to individual diplomats, it is critical that additional public health and epidemiological surveillance data be obtained to provide the temporal and geographic context for the health presentation of individuals. In this manner, patterns may emerge that will lead to the identification of clusters of individuals who have become ill, and inform possible causes. This will facilitate the early identification of threats, and also give credence to individuals who present with curious symptoms. When several patients present concurrently and these associations can be made quickly, it lends credibility to each patient's presentation whereas previously they might have been met with skepticism. This is a critical aspect of early threat identification and recognition of disease clusters. DOS might consider a mechanism for real-time, self-reporting by employees of concerning signs and symptoms. Continuous monitoring of these reports by a multi-disciplinary panel of medical and scientific experts might complement other approaches for health information gathering.

Medical surveillance provides a strategy for illuminating one (i.e., the medical) dimension of a potential health threat. The other critical and complementary strategies include surveillance of potential environmental factors. Routine environmental surveillance comes with the added potential advantage of detecting a threat early and before adverse effects have occurred. Tremendous advances are being made in sensor technology that provide the means for stationary, personal, or wearable devices that capture signal or material or both for evaluating the presence of chemical, biological, or physical agents. Such sensors could be randomly and routinely deployed or be available for response under circumstances when there is concern. One way that medical and environmental surveillance can be effectively integrated is with the collection and archiving of baseline biological and environmental specimens that are available for comparison to samples collected after event onset. It may be that the etiologic agent (metabolite or marker of its biological effect) is present within one or more biological materials (blood, serum, urine, hair, nails) or environmental samples. For example, in addressing the hypothesis put forth by the Canadians about possible etiologic agents in Havana, organophosphate insecticides or their clear biological effect (cholinesterase inhibition) would be apparent in appropriate biospecimens collected within a suitable timeframe of symptom onset and/or suspected exposure. Using appropriate chain-of-custody procedures, levels could be compared to those in baseline samples (preferable) and/or to normative population distribution values such as are available from CDC's National Health and Nutrition Examination Survey (NHANES).

**Recommendation 3-A. If research or assessments support the possibility of radio frequency (RF) energy as a cause of illness experienced by some of its employees, the Department of State should train and equip employees with**

**the capability to measure and characterize their exposure to RF energy in real time should the need arise in the future.**

Capturing a suspected exposure event in real time is critical to establishing cause and effect given the transient nature of the suspected exposure mechanism. It is within the state of the art of RF electronics engineering to measure incident RF energy power levels, frequency bands, pulse width, pulse repetition rate, and angle of arrival. A system could also record secondary effects on other electronics in the vicinity of the employees as well as their own notes on their actions to characterize the situation. Modern electronic measurement devices that are compact and user-friendly require only modest investment for development and deployment. Operationally, a systematic series of RF energy characterization measurements could be made by an embassy employee to map out the spatial energy incident at a particular location. The set of measurements would indicate the direction of incident RF energy, as well as the impact of physical barriers (e.g., walls, doors, etc.) on the transmission of the RF energy and the correlation with the perceived effect by the employee.

In addition to potential trigger and/or monitoring sensors that characterize the exposure characteristics, further experiments are required to demonstrate causal links between an RF exposure regime and biological dysfunction observed or experienced by DOS employees. This would enable an RF exposure diagnostic kit for simultaneously measuring exposure characteristics and estimating potential dosage levels for individuals. Capturing both sets of data (exposure characteristics and biological damage) would allow direct cause and effect to be established and help researchers develop mitigation techniques to counter future exposure events and provide immediate, appropriate medical care based on the exposure.

**Recommendation 3-B. The Department of State should develop a systematic approach for toxicological diagnoses, and a protocol that supports this approach.**

The absence of such an approach hampered the committee's assessment of toxicants as possible contributors to the illnesses in DOS employees from Havana. With respect to missing data for these employees, such a protocol might include more detailed records of pesticide and other potential chemical use (e.g., more extensive environmental sampling for OPs and pyrethroids, particularly proximal in time and space to the occurrence of symptoms in affected individuals), and the archiving of biological samples collected from affected individuals at the time of initial symptoms for subsequent targeted testing of environmental chemicals suspected of contributing to these illnesses. A valuable model for a coordinated system-wide research response to public health emergencies is provided by the National Institutes of Health (NIH) Disaster Research Response (DR2) Program.<sup>5</sup> This program was motivated by many of the same goals and has successfully addressed many of the same needs as the system the committee envisions for DOS in the future.

**Recommendation 4. The Department of State, with support from the U.S. government, should provide for appropriate personnel to identify public health emergencies and activate the necessary response.**

---

<sup>5</sup> See <http://dr2.nlm.nih.gov> (accessed July 24, 2020).

To facilitate early identification of health threats to Embassy personnel, the committee suggests an expanded role for health attachés. Health attachés are diplomats with specialized knowledge in health-related issues who engage in global health diplomacy to promote U.S. national interests, serve as liaisons with in-country counterparts, and provide technical assistance to Embassy personnel and local stakeholders. Health attachés are cross-trained in foreign affairs, international law, and other domains, as well as the public health disciplines. They are not present in every Embassy and in fact are posted sparingly around the globe. Most are on loan from the Department of Health and Human Services, CDC, the Food and Drug Administration, NIH, or from the Office of Global Affairs (OGA) in the Office of the Secretary of Health and Human Services. Others have hailed from the Department of Defense and the U.S. Agency for International Development. As of 2014, their work was coordinated with DOS's Office of Global Health Diplomacy (Brown et al., 2014). The committee believes that health attachés can serve as a critical nexus of timely information from in-country and cross-agency sources. As such, they would be well positioned to identify and respond to threats quickly, provide advice, and collect relevant data needed for informed responses. The committee urges increased budgetary support for health attachés posted in U.S. embassies. It suggests prioritization for their deployment based on perceived need and/or threat in order to utilize their interdisciplinary skills in an optimal fashion.

To remain responsive to the threat environment, DOS should engage in regular action reviews, or root cause analysis of sentinel events, to borrow a process from the Joint Commission on Accreditation of Healthcare Organizations (Joint Commission on Accreditation of Healthcare Organizations, 1998), in order to minimize delays in the identification of novel threats or in their communication from posts to Washington, DC. DOS should establish a system such that the intensity of an investigation into a new health threat rapidly escalates in real time, as needed. An established team with institutional knowledge that can quickly incorporate specific medical and environmental specialty expertise depending on the nature of the condition is best suited for such investigations. An associated advisory board might add expertise on relevant political, environmental, and other matters, in order to provide context for interpretation of unusual medical findings. As the committee already noted, DOS should consider a change in policy that enables structured medical investigations of affected individuals in a manner that does not preclude, but is separate from private medical care. Such medical investigations may be reserved for pre-specified circumstances in which there is concern that multiple DOS employees are the subject of a health attack. The committee urges additional specificity of response to the findings of the July 2018 Government Accountability Office Report in order to ensure that the proper information flow occurs between posts and Washington (GAO, 2018).

## REFERENCES

- Brown, M. D., T. K. Mackey, C. N. Shapiro, J. Kolker, E. Thomas, and T. E. Novotny. 2014. Bridging public health and foreign affairs: The tradecraft of global health diplomacy and the role of health attachés. *Science & Diplomacy* 3(3). <http://www.sciencediplomacy.org/article/2014/bridging-public-health-and-foreign-affairs> (accessed July 19, 2020).
- GAO (U.S. Government Accountability Office). 2018. *Reported injuries to U.S. personnel in Cuba: State should revise policies to ensure appropriate internal communication of relevant incidents*. <https://www.gao.gov/assets/700/693516.pdf> (accessed July 27, 2020).
- NASEM (National Academies of Sciences, Engineering, and Medicine). 2018. *A smarter national surveillance system for occupational safety and health in the 21st century*. Washington, DC: The National Academies Press.

- Resnik, D. B., A. K. Miller, R. K. Kwok, L. S. Engle, and D. P. Sandler. 2015. Ethical issues in environmental research related to public health contingencies: Reflections on the GuLF STUDY. *Environmental Health Perspectives* 123(9):A227-A231.
- Joint Commission on Accreditation of Healthcare Organizations. 1998. Sentinel events: Approaches to error reduction and prevention. *Joint Commission Journal on Quality Improvement* 24(4):175-186.

## A

## Committee Biographies

**David A. Relman, M.D. (Chair)**, is the Thomas C. and Joan M. Merigan Professor in Medicine, and Microbiology & Immunology at Stanford University and chief of infectious diseases at the Veterans Affairs Palo Alto Health Care System. He is also senior fellow at the Freeman Spogli Institute for International Studies (FSI), and served as science co-director at the Center for International Security and Cooperation (2013-2017) at Stanford University. He is currently director of a new Biosecurity Initiative at FSI. Dr. Relman trained at the Massachusetts Institute of Technology and then Harvard Medical School, followed by clinical training in internal medicine and infectious diseases at the Massachusetts General Hospital in Boston, and then a postdoctoral fellowship in microbiology at Stanford. Dr. Relman was an early pioneer in the modern study of the human microbiome. His recent work focuses on the features of human microbial community assembly and the basis for community stability and resilience. His previous work has included the development of methods for pathogen discovery and the identification of several historically important and novel microbial disease agents, as well as the use of genomic technologies for understanding human-microbe interactions. In the 1990s, he worked with the Centers for Disease Control and Prevention on its Unexplained Deaths and Critical Illnesses Project. Among policy-relevant activities in health and biological security, Dr. Relman served as vice-chair of the National Research Council committee that reviewed the science performed for the Federal Bureau of Investigation 2001 anthrax letters investigation, chair of the National Academies of Sciences, Engineering, and Medicine's Forum on Microbial Threats (2007-2017), and is currently a member of the Intelligence Community Studies Board (2016-present). He is an advisor to the Nuclear Threat Initiative and the Center for Strategic and International Studies. Dr. Relman was a founding member of the National Science Advisory Board on Biosecurity (2005-2014), a member of the Working Group on Biodefense for the President's Council of Advisors on Science and Technology (The White House) (2016), and served as president of the Infectious Diseases Society of America (2012-2013). He was a recipient of the National Institutes of Health Pioneer and Transformative Research Award and was elected to the National Academy of Medicine in 2011.

**Doris-Eva Bamiou, M.D., M.Sc., Ph.D.**, is professor of neuroaudiology at the University College of London (UCL) Ear Institute. She is also an honorary consultant in audiological medicine at the UCL Hospitals and Great Ormond Street Hospital. She has been director and organizer of the current trends in auditory processing disorders instructional courses (since 2001) and UCL master class in auditory processing disorders (since 2008) and program director of audio-vestibular medicine at UCL since 2010. She has served as secretary elect of the British Society of Audiology; chair of the Auditory Processing Disorders Specialist Interest Group (BSA); and editor of the Neuro-otology Module of the eBrain e-learning module (RCP and EFNS). She is also secretary of the International Association of Physicians in Audiology. Dr. Bamiou earned a Ph.D. in neurology from UCL.

**Linda Birnbaum, M.S., Ph.D.**, is the retired director of the National Institute of Environmental



Health Sciences of the National Institutes of Health. She is also director of the National Toxicology Program. In these roles, Dr. Birnbaum oversees federal funding for biomedical research to discover how the environment influences human health and disease. Her research focuses on the pharmacokinetic behavior of environmental chemicals, the mechanisms of action of toxicants—including endocrine disruptors and the linking of real-world exposures to health effects. Dr. Birnbaum earned a B.S. in biology from the University of Rochester and an M.S. and a Ph.D. in microbiology from the University of Illinois at Urbana-Champaign. She is a member of the National Academy of Medicine.

**Michael Boninger, M.D.**, is professor and endowed vice chair for research in the Department of Physical Medicine and Rehabilitation at the University of Pittsburgh School of Medicine. He has joint appointments in the Departments of Bioengineering, Rehabilitation Science and Technology, and the McGowan Institute of Regenerative Medicine. He is also physician researcher for the Department of Veterans Affairs and senior medical director for Post-Acute Care for the Health Services Division of the University of Pittsburgh Medical Center. Dr. Boninger has an extensive publication record of more than 250 peer-reviewed papers. His central research focus is on enabling increased function and participation for individuals with disabilities through development and application of assistive, rehabilitative, and regenerative technologies. Dr. Boninger also has extensive experience and publications related to training researchers and served as associate dean for medical student research in the University of Pittsburgh School of Medicine for a number of years. Dr. Boninger earned a B.S. in mechanical engineering and M.D. at The Ohio State University. He completed his residency in physical medicine and rehabilitation in Ann Arbor at the University of Michigan Medical Center. He is a member of the National Academy of Medicine.

**Ronald Brookmeyer, M.S., Ph.D.**, is professor in the Department of Biostatistics and interim dean of the Fielding School of Public Health at the University of California, Los Angeles. His research is at the interface of biostatistics, epidemiology, and public health. He uses the tools of the statistical and informational sciences to address global public health problems. A main theme concerns statistical and quantitative approaches for measuring and forecasting the health of populations. Dr. Brookmeyer has worked on the development of methods for tracking the course of the global HIV/AIDS epidemic and has also worked extensively on issues of biosecurity, such as anthrax. Dr. Brookmeyer has ongoing projects concerning the health problems of aging populations such as Alzheimer's disease. His research interests in biostatistical methodology include survival analysis, epidemic models, epidemiological methods, and clinical trials. Dr. Brookmeyer earned a B.S. in mathematics from Cooper Union for the Advancement of Science and Art and an M.S. and a Ph.D. in statistics from the University of Wisconsin. He is a member of the National Academy of Medicine.

**Caroline Buckee, D.Phil.**, is associate professor of epidemiology in the Harvard T.H. Chan School of Public Health. Dr. Buckee was also named associate director of the Center for Communicable Disease Dynamics. Her laboratory (the Buckee lab) uses mathematical models and data science to understand the mechanisms driving the spread of infectious diseases, particularly pathogens like malaria that effect vulnerable populations in low-income countries. Her focus is on the use of new technologies, including mobile phone data and pathogen genomics, to understand and control disease threats, and to prepare for, and forecast, epidemics. Her work led to an Omidyar Fellowship at the Santa Fe Institute, where she developed theoretical approaches to understanding malaria parasite evolution and ecology. After receiving a D.Phil. from the University of Oxford, Dr.

Buckee worked at the Kenya Medical Research Institute to analyze clinical and epidemiological aspects of malaria as a Sir Henry Wellcome Postdoctoral Fellow.

**Timothy J. Buckley, Ph.D.**, is director of the Exposure Methods and Measurements Division within the Environmental Protection Agency's National Exposure Research Laboratory. He previously spent 16 years within academia at the Johns Hopkins Bloomberg School of Public Health and The Ohio State University School of Public Health. His work is broad ranging and includes the development and application of exposure methods; measurements; and models to chemical, physical, and biological stressors within community and occupational settings. Exposure is treated comprehensively considering all relevant routes and pathways and typically includes biomonitoring to further inform this research. These studies have been applied in the context of health studies to evaluate environmental determinants of effects that are both salutogenic and adverse (e.g., cancer, neurotoxic, and respiratory). His work is strongly tied to the environmental interests and concerns of communities and has helped to identify and inform issues of environmental justice. His research has led to numerous funded grants and the publication of more than 75 peer-reviewed journal articles. Dr. Buckley has a Ph.D. in environmental science and exposure science.

**Joseph J. Fins, M.D.**, is the E. William Davis, Jr., M.D. Professor of Medical Ethics, professor of medicine, professor of medical ethics in neurology, professor of medical ethics in rehabilitation medicine, professor of medicine in psychiatry, professor of health care policy and research, and chief of the Division of Medical Ethics at Weill Cornell Medical College. His interests include ethical and policy issues related to the diagnosis and treatment of severe brain injury and disorders of consciousness. A member of the adjunct faculty at The Rockefeller University, he is also affiliated with the Yale Law School, where he is exploring the legal and ethical issues surrounding severe brain injury from a civil and disability rights perspective. He is also conducting research on ethical implications, including the diagnostic role of functional neuroimaging, neuroprosthetic devices used to promote functional communication (such as deep brain stimulation), the experiences of patients and surrogates touched by brain injury, and public policy for this population (civilian and military). As a board-certified internist physician and medical ethicist, his other interests include palliative care, research ethics in neurology and psychiatry, medical education, and methods of ethics case consultation—drawing on the American Pragmatic tradition. He earned an M.D. from Weill Cornell Medical College and is a graduate of Wesleyan University. He is a member of the National Academy of Medicine.

**John C. Gore, Ph.D.**, is director of the Institute of Imaging Science and the Hertha Ramsey Cress University Professor of radiology and radiological sciences, biomedical engineering, physics and astronomy, and molecular physiology and biophysics at Vanderbilt University. He has served formerly as a member of the National Advisory Council for Biomedical Imaging and Bioengineering at the National Institutes of Health. His research interests include the development and application of imaging methods for understanding tissue physiology and structure, molecular imaging, and functional brain imaging. He has published more than 700 original papers and contributions within the medical imaging field. He is fellow of the American Association for the Advancement of Science, the American Institute of Medical and Biological Engineering, the International Society for Magnetic Resonance in Medicine, the American Physical Society, and the Institute of Physics (United Kingdom). Dr. Gore obtained his Ph.D. in physics at the University of London in the United Kingdom and has been an active leader in imaging research and applications

for 40 years. He also holds a degree in law. Dr. Gore is a member of the National Academy of Engineering.

**Walter J. Koroshetz, M.D.**, is director at the National Institute of Neurological Disorders and Stroke (NINDS) within the National Institutes of Health (NIH). Previously, he served as deputy director of NINDS under Dr. Story Landis. Together, they directed program planning and budgeting and oversaw the scientific and administrative functions of NINDS. The mission of NINDS is to advance the fundamental knowledge about the brain and the nervous system and to use that knowledge to reduce the burden of neurological disorders. He has held leadership roles in a number of NIH and NINDS programs, including NIH's Brain Research through Advancing Innovative Neurotechnologies (BRAIN) Initiative; NIH Helping to End Addiction Longterm (HEAL) Initiative, the Traumatic Brain Injury Center collaborative effort between the NIH intramural program and the Uniformed Services University of the Health Sciences; and the establishment of the NIH Office of Emergency Care Research. Dr. Koroshetz earned a B.A. from Georgetown University and an M.D. from the University of Chicago Pritzker School of Medicine and completed residency training in internal medicine and neurology. Prior to coming to NIH, he was professor of neurology at Harvard Medical School, vice chair of neurology, and director of stroke and neurointensive care services at Massachusetts General Hospital. Dr. Koroshetz is a member of the National Academy of Medicine.

**Pamela Lein, M.S., Ph.D.**, is professor of neurotoxicology and chair of the Department of Molecular Biosciences in the School of Veterinary Medicine at the University of California, Davis. She is also a faculty member of the MIND Institute at the University of California, Davis. Research goals in her laboratory include identifying novel therapeutic approaches for preventing brain damage following exposure to chemicals that cause seizures; understanding the cellular and molecular mechanisms by which environmental factors interact with genetic factors to increase risk for neurodevelopmental disorders, such as autism spectrum disorders, and neurodegenerative diseases, such as Alzheimer's disease; and determining how pesticides alter communication between nerves and immune cells in the lung to cause airway hyperreactivity, a major symptom of asthma. This research leverages diverse model systems ranging from primary neuronal cell culture to zebrafish to rodent models and multiple techniques ranging from cellular and molecular techniques to in vivo imaging to behavioral studies. Professional societies of which she is a member include the Society of Toxicology, the Society for Neuroscience, the International Neurotoxicology Association, and the American Society for Pharmacology and Experimental Therapeutics. She earned a B.S. in biology from Cornell University, an M.S. in environmental health from East Tennessee State University, and a Ph.D. in pharmacology and toxicology from the University at Buffalo.

**Saafan Malik, M.D., M.B.A.**, is a physician-neuroscientist with more than 15 years of experience in the field of traumatic brain injury and neurological disorders. He served as director of the Research Branch at Defense and Veterans Brain Injury Center (DVBIC) at the Department of Defense from 2014-2019 and acting deputy division chief of DVBIC from 2019-2020. During his tenure at DVBIC, he directed an active research portfolio of 72 clinical research protocols at DVBIC headquarters and across 22 clinical/research network sites within the military health system (MHS). Currently he is acting deputy director of Research & Development Directorate/J9, Defense Health Agency (DHA). In his current role, he provides leadership, oversight, and management of all divisions within the R&D Directorate/J9, DHA. He serves on numerous

government scientific steering committees. Prior to DVBIC, he served as senior research investigator in the Department of Neurosurgery at the University of Pennsylvania Perelman School of Medicine and then at the Texas Tech University Health Sciences Center. He has authored several peer-reviewed publications and book chapters and given national and international presentations. His M.D. is from King Edward Medical University and neurosurgery training at the Cleveland Clinic Foundation. He held postdoctoral fellowships at the University of Pennsylvania and at the Carolinas Healthcare System. Dr. Malik also holds an M.B.A. in health care management from Western Governors University.

**Jeffrey S. Palmer, M.S., Ph.D.,** is group leader of the Human Health and Performance Systems Group at the Massachusetts Institute of Technology Lincoln Laboratory (MIT LL). He has expertise within the threat-relevant, biomedical disciplines associated with bioeffects and neurological damage mechanisms related to acoustic or directed energy exposures. He has worked in research laboratories in academia, industry (International Business Machines Corporation [IBM] and General Electric [GE]), and federally funded laboratories (Physical Sciences Laboratory, MIT LL). At MIT LL, he is the leader of the Human Health and Performance Systems Group, which focuses on objective, technology-based, human-centered solutions to measure, model, and modify cognitive and physiological function for enhancement, sustainment, or recovery. Dr. Palmer has been with MIT LL for 22 years, and some of his earlier work included directed energy research on modeling and testing of materials interaction effects. He currently oversees research in health monitoring, as well as the applied neurological, cognitive, and psychological technologies portfolio that includes neurocomputational damage modeling along with acoustic and auditory health research projects. These projects include measurement and modeling of the health effects of high power lasers and high power microwaves on biological tissue and phantoms. To support these and other activities, he initiated the creation of a new interdisciplinary laboratory to measure the interaction of biological materials with photonic, electromagnetic, acoustic, and mechanical sources. He has authored book chapters, technical articles, and given invited talks international conferences on DNA biometrics and forensics, biomechanics, cell biology, materials science, soldier nanotechnology, bio-chemical defense, polymer science, high-energy lasers, microelectronics packaging, wearable biomedical sensing, and neurocognitive technologies. He has served on editorial boards for journals in biomechanics, molecular science, biomedical informatics, and biosensors. He has chaired technical conferences for the National Science Foundation (NSF), the Department of Homeland Security, and the Institute of Electrical and Electronics Engineers (IEEE). Currently, he is vice chair and chair-elect of the IEEE Engineering in Medicine and Biology Society's (EMBS's) Technical Committee on Wearable Biomedical Sensors and Systems and the EMBS conference editorial board for tissue engineering and biomaterials. In addition, he has served as an advisor on senior military studies of enhancing health and performance; a North American Treaty Organization (NATO) human factors and medicine research technical group; and an NSF Nanosystems Center on Advanced Self-Powered Systems of Integrated Sensors and Technologies (ASSIST). Dr. Palmer received mechanical engineering degrees from New Mexico State University (B.S. with math minor), Rensselaer Polytechnic Institute (M.S.), and MIT (Ph.D. with bioengineering focus). His doctoral work focused on measuring and modeling biomechanical function and damage of protein networks from the molecular through tissue scales.

**Gregory B. Saathoff, M.D.,** is a board-certified psychiatrist who holds joint appointments as professor in the Departments of Emergency Medicine and Public Health Sciences at the University of Virginia (UVA) School of Medicine. He also serves as executive director of UVA's Critical



Incident Analysis Group (CIAG) and since 1996 has served as the Federal Bureau of Investigation's (FBI's) conflict resolution specialist. He continues to serve in this capacity as chief psychiatric consultant for the FBI's Behavioral Analysis Units and Crisis Negotiation Unit. Since 1992, he has taught medical students, residents, and fellows correctional psychiatry on-site at a men's maximum security prison. In his faculty role, he served as elected chair of UVA's General Faculty Council. From 1985 to 1994, Dr. Saathoff served as a major in the U.S. Army Reserves Psychiatry Medical Corps. He was called from Reserve Duty during Operation Desert Storm and deployed as a medical corps psychiatrist, earning the Army Commendation Medal in 1991. As a member of UVA's Kuwait Project, he studied societal trauma in Kuwait subsequent to the Iraqi occupation and has served on the faculty of the Saudi-U.S. Universities Project located at the King Faisal Specialist Hospital in Riyadh, Saudi Arabia. In addition to the Middle East, Dr. Saathoff's work has taken him to projects in the former Soviet Union, Western Europe, and Australia. In 2006, Dr. Saathoff was appointed to the Research Advisory Board of the FBI's National Center for the Analysis of Violent Crime. He has served as principal investigator on federal grants relating to public response to weapons of mass destruction and Internet radicalization. After receiving his undergraduate degree from the University of Notre Dame and his M.D. at the University of Missouri School of Medicine, Dr. Saathoff completed residency training in psychiatry at the UVA School of Medicine.

**Clifford B. Saper, M.S., M.D., Ph.D.,** is the James Jackson Putnam Professor of Neurology and Neuroscience and chair of the Beth Israel Deaconess Department of Neurology at Harvard Medical School. Dr. Saper earned a B.A. in biochemistry and M.S. in neurobiology from the University of Illinois, then his M.D. and Ph.D. in neuroscience from Washington University in St. Louis. After a residency in neurology at New York Hospital-Cornell University Medical Center, he then was on the faculty at Washington University and the University of Chicago, where he was the William D. Mabie Professor of Neurology and Neuroscience and chair of the Committee on Neurobiology, before taking his current position. The focus of Dr. Saper's research is on brain circuitry that controls basic functions like wake-sleep, circadian rhythms, body temperature regulation, and eating and drinking. He is a member of the National Academy of Medicine.

**Mark J. Shelhamer, M.S., Sc.D.,** is professor of otolaryngology at Johns Hopkins University. He was previously at the Massachusetts Institute of Technology (MIT) where he worked on sensorimotor physiology and modeling, including the study of astronaut adaptation to space flight. He then came to Johns Hopkins where he continued the study of sensorimotor adaptation with an emphasis on the vestibular and oculomotor systems. He has applied nonlinear dynamical analysis to the control of eye movements, including investigations of the functional implications of fractal activity in physiological behavior. In parallel with these activities, he has had support from the National Aeronautics and Space Administration (NASA) to study sensorimotor adaptation to space flight, amassing a fair amount of parabolic flight ("weightless") experience in the process. He also serves as advisor to the commercial spaceflight industry on the research potential of suborbital space flight. From 2013-2016 he was on leave from his academic position to serve as NASA's chief scientist for human research at the Johnson Space Center. He has a B.S. and M.S. in electrical engineering from Drexel University and a Ph.D. in biomedical engineering from MIT.

**Jeffrey P. Staab, M.D., M.S.,** is professor of psychiatry and director of the Fellowship in Consultation-Liaison Psychiatry in the Department of Psychiatry and Psychology at the Mayo Clinic College of Medicine and Science. He is also consultant in the Departments of Psychiatry and



Psychology and Otorhinolaryngology—Head and Neck Surgery at the Mayo Clinic. His research, which is funded by the National Institutes of Health, Department of Defense, and Mayo Clinic, covers a range of problems at the interface of psychiatry and medicine, including functional otologic and neurologic disorders and illness anxiety. He is best known for investigations of the differential diagnosis and treatment of chronic dizziness. He is author or co-author of more than 130 scientific articles, reviews, chapters, and abstracts. He serves on the editorial boards of six scientific journals in the fields of psychosomatic medicine and otorhinolaryngology. Dr. Staab received a B.S. in chemical engineering from Northwestern University, an M.S. in bioengineering from Carnegie Mellon University, and an M.D. from the University of Pittsburgh.

**Jonathan D. Trobe, M.D.**, is professor, ophthalmology and visual sciences; professor, Department of Neurology; and co-director, Kellogg Eye Center for International Ophthalmology at the University of Michigan. Dr. Trobe's research has covered a wide spectrum of neuro-ophthalmic entities, as well as studies of utilization of health care personnel and application of clinical trial data to medical practice. His research interests include neural visual pathway disorders, double vision, pupillary abnormalities, eyelid disorders, higher order disorders of visual integration, traumatic brain injury, and disorders of high and low intracranial pressure. In 2001, he was appointed editor of the *Journal of Neuro-Ophthalmology*, serving until 2009. He has written and taught widely around the world and authored nearly 200 peer-reviewed scholarly articles. For the American Academy of Ophthalmology, he authored *The Physician's Guide to Eye Care*, a widely used textbook. He has also authored *The Eyes Have It*, a web-based and mobile application designed to teach non-ophthalmologist providers and assess their ophthalmic knowledge. Dr. Trobe received his medical degree from Harvard University and completed residencies in ophthalmology at Wills Eye Hospital and neurology at the University of Miami. He completed a fellowship in neuro-ophthalmology at the Bascom Palmer Eye Institute.

**David Whelan, M.S., Ph.D.**, is professor of the practice in electrical and computer engineering at the University of California, San Diego. Dr. Whelan's expertise is in electromagnetic systems engineering for sensing, imaging, and communications, as well as in the management of science, technology, and innovation. He designs and engineers aircraft, RADAR and Light Detection and Ranging (LIDAR) systems, space-based communications and navigation systems, and diagnostic sensors for high-energy density physics experiments. His work has been used in space mission systems, airborne navigation, and surveillance systems. Dr. Whelan's 34-year career in the aerospace industry included science and engineering research positions and eventually executive research and development management as vice president and chief scientist of the Boeing Defense and Space Systems. He also served as office director for two of the Defense Advanced Research Projects Agency (DARPA) systems offices. While at DARPA, Dr. Whelan created many legacy joint programs with the Air Force, Navy, and Army, most notably, the Discoverer II Space Radar Program, the Army's Future Combat System, and the Unmanned Combat Air Vehicle for Navy and Air Force. He previously worked at the Hughes Aircraft Company as program manager and chief scientist for the B-2 Bomber Air-to-Air Radar Imaging Program. He also worked as a physicist for the Department of Energy's Lawrence Livermore National Laboratory on X-ray lasers and the Advanced Nuclear Weapons program. He started his career at Northrop where he was one of the key designers of the B-2 Stealth Bomber and contributed to the YF-23 Advanced Tactical Fighter. He has numerous publications on electromagnetic radiation, laser plasma phenomena, and defense systems. He holds more than 50 patents on navigation systems, radar systems, antenna, and low-observable technology. He is a fellow of the American Physical Society, the Institute of Electrical

and Electronics Engineers, and the American Institute of Aeronautics and Astronautics. He earned a B.A. in physics from the University of California, San Diego, and an M.S. and a Ph.D. in physics from the University of California, Los Angeles, where he studied Type III Radio Solar Bursts and Nonlinear Energy Flow. Dr. Whelan is a member of the National Academy of Engineering.

## **B**

# **Meeting Agendas**

### **First Meeting of the Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies**

**December 18, 2019**

National Academy of Sciences Building  
2101 Constitution Avenue, NW  
Washington, DC 20418  
Lecture Room

**Wednesday, December 18, 2019**

#### **DATA-GATHERING SESSION**

10:30 a.m. – 10:45 a.m.      **Greetings from Sponsor and Charge to Committee**  
**Mark Cohen**, Medical Director, Department of State

10:45 a.m. – 12:45 p.m.      **Presentations on Medical Investigations**

*Moderator* – **David Relman**, Professor of Medicine and Microbiology and Immunology, Stanford University

*Acute Presentation of an Acquired Neurosensory Syndrome*

**Michael Hoffer**, Professor, Department of Otolaryngology, University of Miami

**Carey Balaban**, Professor of Otolaryngology, Neurobiology, Communication Sciences and Disorders, and Bioengineering, University of Pittsburgh School of Medicine

*What Underlies Havana Syndrome*

**Douglas Smith**, Robert A. Groff Endowed Professor and Vice Chairman of Neurosurgery, University of Pennsylvania, and Director of University of Pennsylvania's Center for Brain Injury and Repair

*Multimodal Neuroimaging Reveals Neurotoxins as a Likely Underlying Cause of Illness Among Canadian Diplomats*

**Alon Friedman**, Professor of Neuroscience and Dennis Chair in Epilepsy Research, Dalhousie University

12:45 p.m.      **ADJOURN**

**Second Meeting of the Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies**

**February 24-25, 2020**

National Academy of Sciences Building  
2101 Constitution Avenue, NW  
Washington, DC 20418  
Lecture Room

**Monday, February 24, 2020**

**DATA-GATHERING SESSION – OPEN TO THE PUBLIC**

**Session III—Epidemiologic Investigations**

2:45 p.m. – 3:45 p.m. *Recommended Epidemiologic Investigations for Future Incidents*  
**Caroline Buckee**, Associate Professor of Epidemiology, Associate Director of the Center for Communicable Disease Dynamics, Harvard T.H. Chan School of Public Health

**Session IV—Possible Mechanisms of Injury—Radio Frequency Energy**

3:45 p.m. – 5:30 p.m. *Moderators*  
**Jeffrey Palmer**, Group Leader, Human Health and Performance Systems Group, Massachusetts Institute of Technology Lincoln Laboratory  
**David Whelan**, Professor of Practice, Jacobs School of Engineering, University of California, San Diego

*Neurologic Illness and Pulsed Radio Frequency/Microwave Radiation*  
**Beatrice Golomb**, Professor in Residence, Medicine, University of California, San Diego [via Zoom conference call]

5:30 p.m. – 6:00 p.m. **Committee Q&A**

6:00 p.m. **ADJOURN**

**Tuesday, February 25, 2020**

**DATA-GATHERING SESSION – OPEN TO THE PUBLIC**

**Session IV Continued—Possible Mechanisms of Injury—Radio Frequency Energy**

8:45 a.m. – 12:00 p.m. *Moderators*  
**Jeffrey Palmer**, Group Leader, Human Health and Performance Systems Group, Massachusetts Institute of Technology Lincoln Laboratory  
**David Whelan**, Professor of Practice, Jacobs School of Engineering, University of California, San Diego

*Correlation of Mild Traumatic Brain Injury and Biological Effects of Weak Electromagnetic Fields*

**Frank Barnes**, Distinguished Professor (Emeritus), Optics, Nanostructures and Bioengineering, University of Colorado

*Multi-disciplinary Analysis of Microwave Induced Sound and Pressure in Human Heads*

**James Lin**, Professor (Emeritus), Head of the Bioengineering Department, Director of the Robotics and Automation Laboratory, and Director of Special Projects in the College of Engineering, University of Illinois at Chicago

*Potential Adverse Effects Following Directed Energy Exposure*

**Stephanie Miller, Bennett Ibey, and Jason Payne**, Air Force Research Laboratory

12:00 p.m. – 1:00 p.m.

**Working Lunch**

**Session V—Possible Mechanisms of Injury—Chemicals and Toxicants**

*Moderator*

**Linda Birnbaum**, Former Director, National Institute for Environmental Health Sciences

1:00 p.m. – 1:30 p.m.

*Department of State’s Overseas Integrated Pest Management Program*

**Claire Huson**, Director, Policy, and Special Studies, Department of State’s Office of Safety, Health, and Environmental Management

1:30 p.m. – 2:00 p.m.

*Neurotoxic Agents and Routes of Exposure*

**Pamela Lein**, Professor, Department of Molecular Biosciences, School of Veterinary Medicine, University of California, Davis

2:00 p.m. – 2:45 p.m.

*How to Make a Toxicological Diagnosis*

**Marion Ehrich**, Professor, Virginia-Maryland College of Veterinary Medicine

2:45 p.m. – 3:00 p.m.

**Break**

3:00 p.m. – 3:45 p.m.

*Organophosphate Intoxication*

**Nick Buckley**, Professor of Clinical Pharmacology, Sydney Medical School, Australia [via Zoom conference call]

3:45 p.m. – 4:15 p.m.

**Committee Q&A**

4:15 p.m.

**ADJOURN**

**Third Meeting of the Standing Committee to Advise the Department of State on Unexplained Health Effects on U.S. Government Employees and Their Families at Overseas Embassies**



**May 12, 2020**

VIRTUAL MEETING (ZOOM)

**DATA-GATHERING SESSION – OPEN TO THE PUBLIC**

2:45 p.m. – 3:30 p.m.

**Preparation for Medical Emergencies**

**Aubrey Miller**, National Institute of Environmental Health Sciences' Chief Medical Officer and the Head of the National Institutes of Health Disaster Research Response Effort

**Rehabilitation Approach Should Similar Unexplained Health Effects Occur**

3:30 p.m. – 3:35 p.m.

*Introduction*

**Michael Boninger**, Professor and Endowed Vice Chair for Research, Department of Physical Medicine and Rehabilitation, University of Pittsburgh Medical Center

3:35 p.m. – 4:00 p.m.

*Brain Injury Rehabilitation*

**Ross Zafonte**, Earle P. and Ida S. Charlton Professor and Chair, Department of Physical Medicine and Rehabilitation, Harvard Medical School

4:00 p.m. – 4:25 p.m.

*Vestibular Rehabilitation*

**Susan Whitney**, Professor, Physical Therapy, University of Pittsburgh

4:25 p.m. – 4:50 p.m.

*Cognitive Rehabilitation and Cognitive Behavioral Therapies*

**Douglas B. Cooper**, Adjunct Associate Professor, Department of Psychiatry, UT Health–San Antonio

4:50 p.m. – 5:30 p.m.

**Discussion with Committee**

5:30 p.m.

**ADJOURN**

## C

**Additional Comments on Directed Radio Frequency Energy**

In order to create the Frey effect hearing and sensation of pressure within the head, there are four distinct steps involving the energy conversion from radio frequency (RF) to acoustic modalities. First, the RF energy penetrates the skull and couples to the neural tissue as a function of impedance matching and absorption in the tissue, with penetrations of 2-4 cm for frequencies of 915 MHz to 2.45 GHz (Brace, 2010). This coupling, in turn, creates a rapid oscillation of temperature changes that leads to a rapid, volumetric thermal expansion and contraction of local tissues (i.e., the increase in thermal energy causes an increase in kinetic energy of atoms, pushing against neighboring atoms to create an expansion or swelling in all directions). The oscillating tissue expansion and contraction launches a thermoelastic pressure wave (Lin and Wang, 2007; Yitzhak et al., 2009). If operated at the right pulse repetition frequency, the thermoelastic pressure wave can propagate to and excite the cochlea and vestibular organs at the resonance frequency of the cranium (Lenhardt, 2003; Yitzhak et al., 2014). Intracranial focusing is possible depending on the incident angle of the incoming RF radiation. Localization and intensity effects within a room can be achieved through nonlinear beat wave effects with careful design of the RF source and antenna. The absence, however, of electromagnetic disruption of other electronics within the immediate home/office environment suggests an upper bound to the RF energy, with implications for a potential RF system design. The average power densities associated with some of these effects (e.g., Frey effect hearing) are so low that they would not disrupt nearby electronics in a fashion similar to high-power microwaves (HPM) (Hoad, 2007; Jinshi et al., 2008). The lack of perceptual heating would also rule out other non-lethal HPM systems that have been developed for crowd control (e.g., Department of Defense's 95GHz Active Denial System that only penetrates the skin to 1/64 an inch but heats the skin to uncomfortable levels within seconds) (D'Andrea et al., 2008; DoD, 2020; Nelson et al., 2000).

It is well-known that the vestibular end organs and regions of the brain involved in processing of space and motion information may be excited by energy sources other than rotational or linear accelerations. External sonic, galvanic, and magnetic stimuli are used for diagnostic, experimental, and therapeutic purposes in neuro-otology and vestibular research such as generating vestibular evoked myogenic potentials (sonic), investigating vestibular response thresholds (galvanic), and as emerging therapies for chronic dizziness (transcranial magnetic and electrical stimulation) (Cha et al., 2013). Clinical observations also suggest that certain patients with vestibular disorders (e.g., Ménière's disease) may be susceptible to exacerbations of their symptoms in response to rapid changes in atmospheric pressure as occur with quickly moving weather fronts or changes in elevation during air or land travel (Gürkov et al., 2016). However, the potential for RF sources to stimulate the vestibular end organs via thermoelastic pressure waves or to excite central nervous system pathways via transduction akin to the Frey effect are not known. If these effects exist, then a few observations may be made about their potential manifestations. A thermoelastic pressure wave would be omnidirectional thereby stimulating the vestibular end organs in a non-physiological manner. This unusual form of vestibular stimulation could lead to very confusing percepts as central vestibular pathways do their best to resolve the non-physiological pattern of end organ stimulation resulting in sensations of physically impossible motions, unexpected reflexive postural responses to them, and faulty inferences about external

forces causing them. Affected individuals could report different sensations in response to the same external stimulus; thus, immediate reports of affected individuals may not be veridical and sensations may vary from one individual to another. If a Frey-like effect can be induced on central nervous system tissue responsible for space and motion information processing, it likely would induce similarly idiosyncratic responses.

## REFERENCES

- Brace, C. L. 2010. Microwave tissue ablation: Biophysics, technology, and applications. *Critical Reviews in Biomedical Engineering* 38(1):65-78.
- Cha, Y.-H., Y. Cui, and R. W. Baloh. 2013. Repetitive transcranial magnetic stimulation for mal de débarquement syndrome. *Otology & Neurotology* 34(1):175-179.
- D'Andrea, J., D. Cox, D., P. Henry, J. Ziriaux, D. Hatcher, and W. Hurt. 2008. Rhesus monkey aversion to 94-GHz facial exposure. Naval Health Research Center Detachment Directed Energy Bioeffects Laboratory, Technical Report–NHRC DEBL TR-2006-07.
- DoD (Department of Defense). 2020. *Active denial systems*. <https://jnlwp.defense.gov/Future-Intermediate-Force-Capabilities/Active-Denial-Technology> (accessed June 27, 2020).
- Gürkov, R., R. Strobl, N. Heinlin, E. Krause, B. Olzowy, C. Koppe, and E. Grill. 2016. Atmospheric pressure and onset of episodes of Ménière's disease. *PLoS One* 11(4):e0152714.
- Hoad, R. 2007. *The utility of electromagnetic attack detection to information security*. Ph.D. dissertation. University of Glamorgan, United Kingdom.
- Jinshi, X., L. Wenhua, Z. Shiyang, Z. Jinjua, and X. Changfeng. 2008. *Study of damage mechanism of high power microwave on electronic equipments*. Paper presented at the 2008 China-Japan Joint Microwave Conference, Shanghai.
- Lenhardt, M. L. 2003. Ultrasonic hearing in humans: Applications for tinnitus treatment. *The International Tinnitus Journal* 9(2):69-75.
- Lin, J. C., and Z. Wang. 2007. Hearing of microwave pulses by humans and animals: Effects, mechanism, and thresholds. *Health Physics* 92(6):621-628.
- Nelson, D. A., M. T. Nelson, T. J. Walters, and P. A. Mason. 2000. Skin heating effects of millimeter wave irradiation: Thermal modeling results. *IEEE Transactions on Microwave Theory and Techniques* 48:2111-2120.
- Yitzhak, N. M., R. Ruppín, and R. Hareuveny. 2009. Generalized model of the microwave auditory effect. *Physics in Medicine and Biology* 54(13):621-628.
- Yitzhak, N. M., R. Ruppín, and R. Hareuveny. 2014. Numerical simulation of pressure waves in the cochlea induced by a microwave pulse. *Bioelectromagnetics* 35(7):491-496.

**D****Environmental Chemicals**

Environmental chemicals reported to be associated with signs and symptoms similar to the chronic signs and symptoms observed in Havana patients (pesticides are in italics).

Symptoms	Chemicals Associated with Symptom
Ototoxicity, vestibulotoxicity, tinnitus, vertigo	<ul style="list-style-type: none"> <li>Organic solvents (benzene-based and aliphatic hydrocarbons, 1,2-dinitrobenzene, toluene, trichloroethylene, xylene)</li> <li>Nitriles, carbon disulfide, asphyxiants (CO), metals</li> <li><i>Organophosphorus pesticides (OPs)</i> and other phosphate-based chemicals (acute and chronic exposures)</li> <li><i>Acute pyrethroid exposure</i></li> <li>Quinine (chronic exposure)</li> </ul>
Sensorimotor function	<ul style="list-style-type: none"> <li>Bismuth</li> <li>Brevetoxins</li> <li><i>Pyrethroids</i></li> <li><i>Organophosphorus pesticides (OPs)</i></li> <li>Metals (Pb, Cd, thallium)</li> </ul>
Vision	<ul style="list-style-type: none"> <li><i>Acute OP exposures</i></li> <li>Chlordecone (kepone)</li> <li>Carbon tetrachloride, carbon disulfide, 2,5-hexanedione, methanol (formate)</li> <li>Quinine</li> </ul>
Motor dysfunction (incoordination, muscle weakness)	<ul style="list-style-type: none"> <li><i>Acute and chronic OP exposures</i></li> <li>B-N-methylamino-L-alanine (BMAA), domoic acid, tetanus toxin</li> <li>MPTP, ethanol, 3-nitropropionic acid</li> <li>Carbon monoxide, carbon disulfide, toluene</li> <li>Metals (Pb, Mn, Hg, As)</li> </ul>
Concentration/memory deficits	<ul style="list-style-type: none"> <li><i>Acute and chronic OP exposures</i></li> <li><i>Many pesticides that target neuronal signaling molecules</i></li> <li>Metals (Pb, Mn, Hg)</li> <li>Solvents</li> </ul>
Headaches, fatigue, insomnia	<ul style="list-style-type: none"> <li><i>OPs and pyrethroids</i></li> </ul>

SOURCES: Alcaras et al., 2013; Anger et al., 2020; Ashok Murthy and Visweswara Reddy, 2014; Campo et al., 2007; Chen et al., 1991; Choochouy et al., 2019; Crawford et al., 2008; Dassanayake et al., 2007, 2008, 2009; Dundar et al., 2016; Edwards and Tchounwou, 2005; Fuente and McPherson, 2012; London et al., 1998; Mont'Alverne et al., 2016; Müller-Mohnssen,

1999; Pham et al., 2016; Richter et al., 1992; Rohlman et al., 2011; Roldan-Tapia et al., 2006; Ross et al., 2013; Spencer et al., 2000; Teixeira et al., 2002; Zeigelboim et al., 2019.

## REFERENCES

- Alcaras, P. A., A. B. Larcera, and J. M. Marques. 2013. Study of evoked otoacoustic emissions and suppression effect on workers exposed to pesticides and noise. *Codas* 25(6):527-533.
- Anger, W. K., F. M. Farahat, P. J. Lein, M. R. Lasarev, J. R. Olson, T. M. Farahat, and D. S. Rohlman. 2020. Magnitude of behavioral deficits varies with job-related chlorpyrifos exposure levels among Egyptian pesticide workers. *Neurotoxicology* 77:216-230.
- Ashok Murthy, V., and Y. J. Visweswara Reddy. 2014. Audiological assessment in organophosphorus compound poisoning. *Indian Journal of Otolaryngology and Head and Neck Surgery* 66(1):22-25.
- Campo, P., K. Maguin, and R. Lataye. 2007. Effects of aromatic solvents on acoustic reflexes mediated by central auditory pathways. *Toxicological Sciences* 99(2):582-590.
- Chen, S. Y., Z. W. Zhang, F. S. He, P. P. Yao, Y. Q. Wu, J. X. Sun, L. H. Liu, and Q. G. Li. 1991. An epidemiological study on occupational acute pyrethroid poisoning in cotton farmers. *British Journal of Industrial Medicine* 48(2):77-81.
- Choochouy, N., P. Kongtip, S. Chantanakul, N. Nankongnab, D. Sujirarat, and S. R. Woskie. 2019. Hearing loss in agricultural workers exposed to pesticides and noise. *Annals of Work Exposure and Health* 63(7):707-718.
- Crawford, J. M., J. A. Hoppin, M. C. Alavanja, A. Blair, D. P. Sandler, and F. Kamel. 2008. Hearing loss among licensed pesticide applicators in the agricultural health study. *Journal of Occupational and Environmental Medicine* 50(7):817-826.
- Dassanayake, T., I. B. Gawarammana, V. Weerasinghe, P. S. Dissanayake, S. Pragaash, A. Dawson, and N. Senanayake. 2009. Auditory event-related potential changes in chronic occupational exposure to organophosphate pesticides. *Clinical Neurophysiology* 120(9):1693-1698.
- Dassanayake, T., V. Weerasinghe, U. Dangahadeniya, K. Kularatne, A. Dawson, L. Karalliedde, and N. Senanayake. 2007. Cognitive processing of visual stimuli in patients with organophosphate insecticide poisoning. *Neurology* 68(23):2027-2030.
- Dassanayake, T., V. Weerasinghe, U. Dangahadeniya, K. Kularatne, A. Dawson, L. Karalliedde, and N. Senanayake. 2008. Long-term event-related potential changes following organophosphorus insecticide poisoning. *Clinical Neurophysiology* 119(1):144-150.
- Dundar, M. A., S. Derin, M. Aricigil, and M. A. Eryilmaz. 2016. Sudden bilateral hearing loss after organophosphate inhalation. *Turkish Journal of Emergency Medicine* 16(4):171-172.
- Edwards, F. L., and P. B. Tchounwou. 2005. Environmental toxicology and health effects associated with methyl parathion exposure--a scientific review. *International Journal of Environmental Research and Public Health* 2(3-4):430-441.
- Fuente, A., and B. McPherson. 2012. Occupational chemical-induced hearing loss. In *Hearing loss*, edited by S. Naz. Intech Open. Pp. 171-190.
- London, L., V. Nell, M. L. Thompson, and J. E. Myers. 1998. Effects of long-term organophosphate exposures on neurological symptoms, vibration sense and tremor among South African farm workers. *Scandinavian Journal of Work, Environment, & Health* 24(1):18-29.
- Mont'Alverne, L. R., A. P. Corona, and M. A. Vasconcelos Rego. 2016. Hearing loss associated with organic solvent exposure: A systematic review. *Revista Brasileira de Saude Ocupacional* 41:e10.
- Müller-Mohnssen, H. 1999. Chronic sequelae and irreversible injuries following acute pyrethroid intoxication. *Toxicology Letters* 107(1-3):161-176.
- Pham, H., M. D. Lingao, A. Ganesh, J. E. Capasso, R. Keep, K. A. Sadagopan, and A. V. Levin. 2016. Organophosphate retinopathy. *Oman Journal of Ophthalmology* 9(1):49-51.



- Richter, E. D., P. Chuwers, Y. Levy, M. Gordon, F. Grauer, J. Marzouk, S. Levy, S. Barron, and N. Gruener. 1992. Health effects from exposure to organophosphate pesticides in workers and residents in Israel. *Israel Journal of Medical Sciences* 28(8-9):584-598.
- Rohlman, D. S., W. K. Anger, and P. J. Lein. 2011. Correlating neurobehavioral performance with biomarkers of organophosphorous pesticide exposure. *Neurotoxicology* 32(2):268-276.
- Roldan-Tapia, L., F. A. Nieto-Escamez, E. M. del Aguila, F. Laynez, T. Parron, and F. Sanchez-Santed. 2006. Neuropsychological sequelae from acute poisoning and long-term exposure to carbamate and organophosphate pesticides. *Neurotoxicology and Teratology* 28(6):694-703.
- Ross, S. M., I. C. McManus, V. Harrison, and O. Mason. 2013. Neurobehavioral problems following low-level exposure to organophosphate pesticides: A systematic and meta-analytic review. *Critical Reviews in Toxicology* 43(1):21-44.
- Spencer, P. S., H. H. Schaumburg, and A. C. Ludolph. 2000. *Experimental and clinical neurotoxicology*. 2nd ed. New York: Oxford University Press.
- Teixeira, C. F., L. Giraldo Da Silva Augusto, and T. C. Morata. 2002. Occupational exposure to insecticides and their effects on the auditory system. *Noise Health* 4(14):31-39.
- Zeigelboim, B. S., J. S. Malisky, M. R. D. Rosa, A. B. M. Lacerda, P. S. Alcaraz, and V. R. Fonseca. 2019. The importance of otoneurological evaluation in Brazilian workers exposed to pesticides: A preliminary study. *International Archives of Otorhinolaryngology* 23(4):e389-e395.

Tab L

**AFFIDAVIT OF DAFNA TACHOVER IN SUPPORT OF STAY**

**UNITED STATES COURT OF APPEALS  
FOR THE DISTRICT OF COLUMBIA CIRCUIT**

Children's Health Defense, Dr. Erica  
Elliot, Ginger Kesler, Angela Tsiang,  
and Jonathan Mirin,  
Petitioners

USCA No. 21-1075

v.

Petition for Review of Order  
by the Federal Communications  
Commission  
(FCC 21-10)

Federal Communications Commission  
and United States of America,  
Respondents

**AFFIDAVIT OF DAFNA TACHOVER IN SUPPORT OF STAY**

1. My name is Dafna Tachover. I am the Director of the Children's Health Defense 5G and Wireless Harms Project. I am also a member of Children's Health Defense.
2. I am a licensed attorney in New York and Israel. I have an MBA and a technology background. I was a telecommunications and computers officer in the Israeli Defense Forces, where I served as the commander of the military headquarters and operations center computer center and was responsible for all the systems and networks. I had a diverse legal and business career thereafter. In the US, I worked in senior executive positions for an international investment company headquartered in New York City.
3. I submit this Affidavit to demonstrate Children's Health Defense's organizational and representational standing in this matter. I also provide some

background on Radiation Sickness and other illnesses or conditions caused or exacerbated by wireless radiation, and the effect the rule change we are challenging will have if it goes into effect.

#### **About Children's Health Defense.**

4. Children's Health Defense ("CHD") is a non-profit organization. Its mission is to end the epidemic of children's chronic health conditions by working to eliminate harmful exposures to toxins, establish substantive and procedural protections for and on behalf of those who have already been injured and those who will be harmed in the future and prevent harm to others. CHD evaluates and educates about the harms of various toxins, provides advice, supports the injured and advocates on their behalf in educational and legal matters and gives referrals to medical and other professionals who may be able to help those who have been injured. We work to defend children's health, obtain justice for those already injured and ensure accountability. I will return to CHD organizational and representational interests below.

#### **For Some, RF is a Toxin**

5. Wireless technology uses Radiofrequencies (RFs) to carry wireless data. It is a form of radiation because it "radiates" energy in various ways. To encode the data being transferred over the RF carrier frequency, the signal is pulsed and modulated. This radiation, referred to hereinafter as "wireless radiation," can and

does harm adults and children. Wireless proliferation is significantly contributing to well-recognized but alarming and growing rates of sickness in children.<sup>1</sup> CHD's mission requires that it address wireless radiation's contribution to the overall levels of environmentally induced, toxicity-related children's sicknesses.

### **OTARD Rule Change**

6. The OTARD rule was originally adopted to protect individual “viewers” that want to use satellite-based television programming. It was limited to “receiving” devices. It has been expanded over the years to cover “transmitting” devices and to other wireless services, such as “wireless cable” services and then fixed wireless Internet access. The entire rule is about preemption. It overrules all state and local laws that attempt to regulate the placement and use of these devices by customers that desire to obtain the covered services for their own use, on their own property.

7. The OTARD rule limitation to “own use” and “own property” has always excluded from protection arrangements that extended fixed wireless communications to nearby and/or different properties. So, for example, if a fixed wireless provider arranges for one of its fixed-wireless customers to install a “hub” or “relay” and wirelessly extend voice/video/data service to new users over a wide

---

<sup>1</sup> [https://www.academicpedsjnl.net/article/S1876-2859\(10\)00250-0/fulltext](https://www.academicpedsjnl.net/article/S1876-2859(10)00250-0/fulltext).



are all local and state regulations (including zoning and permitting) and things like neighborhood association and deed restrictions still apply.

8. Certain Wireless Internet Service Providers petitioned for a further rule change that would, for the first time, bring “hub” and “relay” arrangements that facilitate extension to other properties and users within the OTARD rule’s coverage. The entire purpose was to overrule all, state laws, including zoning and homeowner association protections. The OTARD *Order* granted that request.

### **CHD Comments**

9. CHD filed comments in the FCC “OTARD” proceeding.<sup>2</sup> CHD extensively addressed the substantive and procedural problems arising from the adoption of the proposed rule amendment and the devastating effects it will have on the many adults and children who are sick from wireless radiation.<sup>3</sup> CHD observed that the rule would not just eliminate local zoning and preempt deed restrictive covenants: it would also preempt federal and state civil rights laws that protect the disabled and handicapped and eliminate current requirements for accommodations.

---

<sup>2</sup> <https://www.fcc.gov/ecfs/filing/104171342025759>.

<sup>3</sup> <https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20OTARD%20Objectors%20Declaration.pdf>.

10. The FCC was adamant about removing “barriers” and to the FCC it seems, the injured are a barrier that needs to be removed. Otherwise, why preempt disability accommodation rights?

11. CHD understood the implications, so it started a campaign to educate the public. Following a one-month campaign with our limited means, a record 15,090 people (1,988 of them are CHD members) joined CHD’s comments.<sup>4,5</sup> Of those who joined CHD’s submission, 6,231 (823 of whom are CHD members) declared that they and/or their children have become sick from wireless radiation.<sup>6</sup> Many added personal short comments explaining their personal experience and position on the issues. In general, they expressly objected to intrusions on their property and consider non-consensual irradiation to be a battery on them and their family and a form of child endangerment.<sup>7</sup>

12. Over 2,500 personal comments were included, many of them substantive. The few lines that were added in those comments often revealed heart-wrenching

---

<sup>4</sup> <https://www.fcc.gov/ecfs/filing/105191672708448>.

<sup>5</sup> <https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf>.

<sup>6</sup> <https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20OTARD%20Objectors%20Declaration.pdf>.

<sup>7</sup> <https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=690>.

stories of sickness and death. Many reported that exposure to wireless radiation has cause and/or aggravated their sickness including Radiation Sickness, neurodevelopmental conditions, cancer and epileptic seizures. Some reported the death of family members.

These commentators demanded that they not be subjected to emissions that will make them and/or their children or family members sicker and may even kill them. They expressed a plaintive but eminently reasonable desire to be safe in their homes, their only refuge.

### **Rule amendment effect**

13. The moment the rule change goes into effect, all state and local venues for people to obtain notice of activities that affect them and allow them to participate in any local proceedings and allow them object to the activity or seek an accommodation, will be preempted and must stop. This will remove vested substantive and procedural due process rights that many currently use and eliminate a valuable source of vital information. Mirin@27-39.

14. People will no longer know if their neighbor is about to bathe their property with harmful radiation. They will not be able to seek help and accommodations from local or state authorities. Similarly, those who purposefully bought property they thought would be relatively RF radiation-free or safer, and contracted with their neighbors to include deed restrictions relating to antennas and business

activity in residential areas will be stripped of their contractual rights. If someone in a neighborhood has a deed restriction that prohibits commercial activity and wireless systems that provide connectivity to unaffiliated persons on other properties, they will be free to ignore those restrictions, and the restriction is no longer enforceable through an action in local courts. Dr. Elliot and Ginger Kesler both joined in neighborhood associations with such restrictions, they did so on purpose, and now they will lose those rights when the rule amendment takes effect. Elliot@¶¶38-46, Kesler@¶¶6, 8, 43-44.

15. People will not be able to know when one of these new systems is turned on. Until they suddenly get sick again and some, including children, will experience life threatening symptoms. Dr. Elliot@¶23,47,55; Baran@¶9,27,34,46; Dr Hoffman@¶22,45,46; Dr. Jelter@¶35; Dr. Bray@¶14. They will have no option: they will be abruptly evicted from their home without notice and have nowhere to go. Mirin@¶7,22,23,26,48; Dr. Elliot 5,19,28,45,48,49,50,52,54; Baran@¶9,43-44,49,50; Kesler@¶48,49; Dr. Hoffman@¶38-41,44,46,48; Tsiang@¶; Hertz@¶39-43; Dr. Jelter@¶35; Dr. Bray@¶22; Dr. Golomb@¶27-28. With the OTARD amendment, people who suffer debilitating conditions and cannot be anywhere in the wireless world, even their home would no longer be a refuge.

16. The Commission summarily rejected CHD's comments (along with those of all of the people who joined in CHD's comments) in one brief paragraph that

mischaracterized CHD's positions and failed to meaningfully address the concerns raised by CHD and by those who support its position.<sup>8</sup>

### **What is Radiation Sickness?**

17. Radiation Sickness (also called Microwave Sickness; Electro-Sensitivity, Electro Hypersensitivity or EHS), is likely the most widespread sickness associated with exposure to wireless radiation. It describes a constellation of symptoms, mainly neurological (but not exclusively), that manifest with exposure to wireless radiation. Diagnosis guidelines exist.<sup>9, 10, 11</sup>

18. Radiation Sickness is a spectrum condition. People who develop the condition become intolerant and react to levels of wireless radiation they previously could tolerate. As with other toxins or allergens, eliminating exposure is

---

<sup>8</sup> Order ¶34, <https://ecfsapi.fcc.gov/file/01072222126137/FCC-21-10A1.pdf#page=19>.

<sup>9</sup><https://ecfsapi.fcc.gov/file/10052040910188/EUROPAEM%20EMF%20Guideline%202015-Belyaev%20et%20al%202015.pdf>.

<sup>10</sup>[https://ecfsapi.fcc.gov/file/1091546800918/Guideline%20of%20the%20Austrian%20Medical%20Association%20for%20the%20diagnosis%20and%20treatment%20of%20EMF%20related%20health%20problems%20and%20illnesses%20\(EMF%20syndrome\).pdf](https://ecfsapi.fcc.gov/file/1091546800918/Guideline%20of%20the%20Austrian%20Medical%20Association%20for%20the%20diagnosis%20and%20treatment%20of%20EMF%20related%20health%20problems%20and%20illnesses%20(EMF%20syndrome).pdf).

<sup>11</sup><https://ecfsapi.fcc.gov/file/109160003723483/Electrohypersensitivity%20as%20a%20Newly%20Identified%20and%20Characterized%20Neurologic%20Pathological%20Disorder%3B%20How%20to%20Diagnose%2C%20Treat%20and%20Prevent%20it-Belpomme%202020.pdf>.



the only way to avoid symptoms. The condition is progressive, so symptoms reappear with exposure.

19. Following reports of soldiers of symptoms from wireless systems, the US Navy studies the issue and in 1971 published a report referencing 2,311 studies showing RF radiation harms. It took 5 pages to list the various effects and symptoms associated with exposure.<sup>12</sup> Other US military agencies also reported the sickness including the US Air Force,<sup>13</sup> and NASA.<sup>14,15</sup>

20. In December 2020, the National Academy of Sciences, Engineering and Medicine (NAS) studied effects from pulsed RF (wireless radiation). Following the request of the US Department of State to advise them on the “mystery” sickness of the US diplomats, the NAS published a report “An Assessment of Illness in U.S.

---

<sup>12</sup><https://ecfsapi.fcc.gov/file/10914872405454/Naval%20Medical%20Research%20Institute%20Bibliography%20of%20reported%20biological%20effects...1972%20Full%20Report.pdf#page=10>.

<sup>13</sup>[https://ecfsapi.fcc.gov/file/109153103001086/RADIO%20FREQUENCY%20MICROWAVE%20RADIATION%20BIOLOGICAL%20EFFECTS%20AND%20SAFETY%20STANDARDS-A%20REVIEW%20\(Air%20Force%201994\).pdf](https://ecfsapi.fcc.gov/file/109153103001086/RADIO%20FREQUENCY%20MICROWAVE%20RADIATION%20BIOLOGICAL%20EFFECTS%20AND%20SAFETY%20STANDARDS-A%20REVIEW%20(Air%20Force%201994).pdf). See conclusions on p.18 “Experimental evidence has shown that exposure to low intensity radiation can have a profound effect on biological process.”

<sup>14</sup><https://ecfsapi.fcc.gov/file/1007064529654/AFFIDAVIT%20OF%20Susan%20D.%20Foster%2C%20MSW.pdf>.

<sup>15</sup><https://ecfsapi.fcc.gov/file/100518466598/FCC%20comments%20of%20Deborah%20Kopald%2014-177%2C%2015-256%2C%2010-112%2C%20and%2097-95.pdf>.

Government Employees and Their Families at Overseas Embassies.”<sup>16</sup> The report concluded that many of the observed symptoms, including brain damage, are consistent with the biological effects of pulsed RF exposure, and that it is likely the cause of the diplomats’ sickness. In other words, the diplomats suffer from Radiation Sickness, the same condition now experienced by a large and rapidly growing number of people.

21. The typical symptoms indicate severe physiological injuries associated with exposure to wireless radiation including damage to the Blood Brain Barrier (BBB), impaired brain blood flow (BBF) and adverse effects on the immune and hormonal systems.<sup>17, 18, 19</sup> There may be genetic predispositions.<sup>20</sup> A 2015 study on 675

---

<sup>16</sup> <https://www.nap.edu/read/25889/chapter/1>.

<sup>17</sup> <https://ecfsapi.fcc.gov/file/109160003723483/Electrohypersensitivity%20as%20a%20Newly%20Identified%20and%20Characterized%20Neurologic%20Pathological%20Disorder%3B%20How%20to%20Diagnose%2C%20Treat%20and%20Prevent%20it-Belpomme%202020.pdf>.

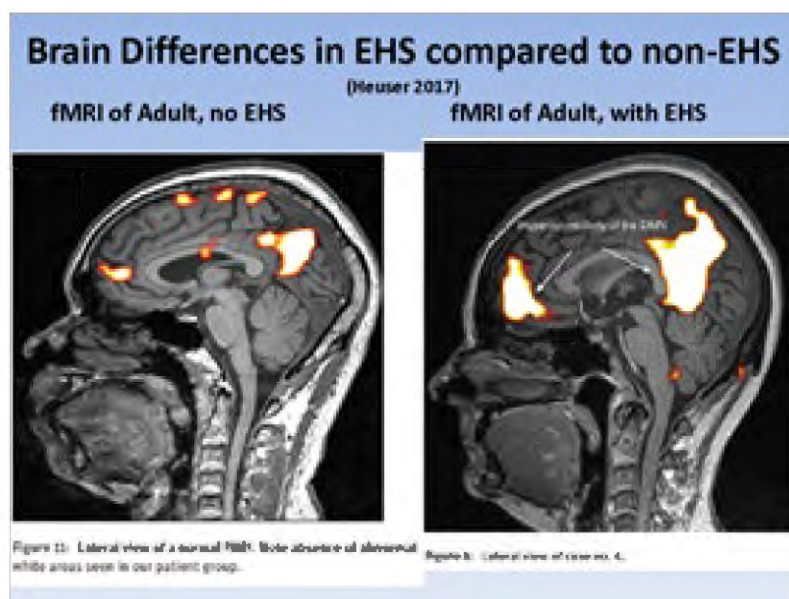
<sup>18</sup> <https://ecfsapi.fcc.gov/file/10912004686552/Reliable%20Disease%20Biomarkers%20Characterizing%20and%20Identifying%20EHS%20%26%20Multiple%20Chemical%20Sensitivity%20as%20%20Etiopathogenic%20Aspects%20of%20a%20Unique%20Pathological%20Disord.pdf>.

<sup>19</sup> <https://ecfsapi.fcc.gov/file/10052040910188/EUROPAEM%20EMF%20Guideline%202015-Belyaev%20et%20al%202015.pdf>.

<sup>20</sup> [https://ecfsapi.fcc.gov/file/1005862318785/Dr\\_Erica\\_Mallery-Blythe\\_EHS\\_A\\_Summary\\_Working\\_Draft\\_Version\\_1\\_Dec\\_2014\\_for\\_EESC\\_Brussels\\_\(3\)%20\(1\).pdf#page=41](https://ecfsapi.fcc.gov/file/1005862318785/Dr_Erica_Mallery-Blythe_EHS_A_Summary_Working_Draft_Version_1_Dec_2014_for_EESC_Brussels_(3)%20(1).pdf#page=41).

subjects who suffer from Radiation Sickness<sup>21</sup> found that 13-28% had Blood Brain Barrier (BBB) leakage. 40% had chronic inflammation. 23% had autoimmune antibodies and 100% had reduced levels of melatonin (the sleep hormone).

22. A study on 10 people who suffer from Radiation Sickness using functional MRI brain imaging, showed traumatic brain injury,<sup>22</sup> similar to what was discovered in several of the diplomats. The fMRI images below compare brain



<sup>21</sup><https://ecfsapi.fcc.gov/file/10912004686552/Reliable%20Disease%20Biomarkers%20Characterizing%20and%20Identifying%20EHS%20%26%20Multiple%20Chemical%20Sensitivity%20as%20%20Etiopathogenic%20Aspects%20of%20a%20Unique%20Pathological%20Disord.pdf>.

<sup>22</sup><https://ecfsapi.fcc.gov/file/10912254241147/Functional%20brain%20MRI%20in%20patients%20complaining%20of%20electrohypersensitivity%20after%20long%20term%20exposure%20to%20electromagnetic%20fields-Heuse-%202017.pdf>.

images of people with Radiation Sickness (right) to “normal” people (left).<sup>23</sup> The white areas represent impaired blood flow.

23. A causal mechanism of harm associated with many of the injuries suffered by people with Radiation Sickness is Oxidative Stress. Over 200 studies<sup>24</sup> showed that wireless radiation causes oxidative stress. For example, oxidative stress was found in Affiant Jennifer Baran’s children’s blood tests. Baran@¶30. Oxidative stress blood tests are used as biomarker for the diagnosis of Radiation Sickness.

24. In January 2021, the Swiss government expert advisory group on electromagnetic fields and non-ionizing radiation, BERENIS, published an extensive evaluation of the scientific literature on non-thermal RF/EMFs.<sup>25</sup> It concluded that exposure could cause or worsen several chronic illnesses, and that children and people with immune deficiencies or diseases are especially at risk. It

---

<sup>23</sup> Drs. Heuser Published a Corrigendum to explain the controls used in the study. <https://www.degruyter.com/document/doi/10.1515/reveh-2017-0027/html>. The controls were a composition fused fMRIs of a normal subjects in the 7 years prior to the 2017 study. Particular attention should be paid to the normal activity demonstrated in the frontal lobe in controls as opposed to the subjects.

<sup>24</sup> [https://ecfsapi.fcc.gov/file/10908969213114/Abstracts%20of%20research%20publications%20on%20radiofrequency%20radiation%20and%20free%20radical%20\(oxidative\)%20effects-Henry%20Lai.pdf](https://ecfsapi.fcc.gov/file/10908969213114/Abstracts%20of%20research%20publications%20on%20radiofrequency%20radiation%20and%20free%20radical%20(oxidative)%20effects-Henry%20Lai.pdf).

<sup>25</sup> <https://www.bafu.admin.ch/bafu/en/home/topics/electrosmog/newsletter-of-the-swiss-expert-group-on-electromagnetic-fields-a.html>.

acknowledged that oxidative stress from the chronic exposure is the underlying mechanism.

25. These findings explain why for example, in addition to Radiation Sickness Movant Ginger Kesler and I both developed a thyroid autoimmune (and hormonal) condition – Hashimoto, and Affiant Michele Hertz developed Graves' Disease following intense exposure to wireless radiation.<sup>26, 27, 28</sup>

### **Radiation Sickness Accommodation**

26. Various government agencies have recognized Radiation Sickness as a disability and have required accommodation. In 2002, the “Access Board,” the federal agency responsible for publishing ADA Accessibility Guidelines used by the Justice Department to enforce the ADA, recognized that “electromagnetic sensitivities may be considered disabilities under the ADA.”<sup>29</sup> The National Institute of Building Sciences (NIBS) was contracted by the Access Board to

---

<sup>26</sup><https://ecfsapi.fcc.gov/file/10908599004195/Public%20Health%20Implications%20of%20the%20Proposed%20Cell%20Phone%20Transmission%20Tower%20at%20Oakway%20Golf%20Course.pdf>, see pages 4 and 38.

<sup>27</sup><https://ecfsapi.fcc.gov/file/109111924311695/How%20does%20long%20term%20exposure%20to%20base%20stations%20and%20mobile%20phones%20affect%20human%20hormone%20profiles-eskander%202012.pdf>.

<sup>28</sup><https://ecfsapi.fcc.gov/file/100245258363/RFR%20Research%20Summary%20Henry%20Lai%202017.pdf>. See pages 234, 463, 602, 819.

<sup>29</sup> See Architectural and Transportation Barriers Compliance Board, ADA Accessibility Guidelines for Recreation Facilities, 68 FR 56351 (Sept. 3, 2002).



provide recommendations on how to accommodate people with Radiation

Sickness. Their 2005 report <sup>30</sup> concluded that wireless radiation is an “access

barrier” and can render buildings “inaccessible” to those with Radiation Sickness:

People with electromagnetic sensitivities can experience debilitating reactions... from electromagnetic fields emitted by computers, cell phones... The severity of sensitivities varies among people...

... public and commercial buildings are required to provide reasonable accommodations for those disabled by electromagnetic sensitivities.

27. The US. Department of Labor's Office of Disability Employment Policy

issued guidelines for accommodations in 2015. Tachover Attachment 1. The

guidelines state:

...the nature of electromagnetic sensitivity is such that even levels that are deemed safe for the general public can cause trigger symptoms for individuals who are hypersensitive...and therefore may need accommodation.

...

Individuals with electromagnetic sensitivity may experience ... fatigue, weakness, neurological issues, immunological issues, gastrointestinal issues, increased irritability, lack of ability to think clearly and quickly, sleep disturbance, overall malaise, and anxiety...Common workplace issues involve exposure to Wi-Fi, cell phones.”

General considerations include: ...Relocate workplace away from areas where symptoms are triggered...limiting certain types of devices in the vicinity of the employee's workstation... Provide wired telephones and network connections.

---

<sup>30</sup><https://ecfsapi.fcc.gov/file/1006784928637/IEQ%20Indoor%20Environmental%20Quality.pdf>.

28. The US Department of Education (“DOE”) has recognized that people with other conditions may also develop Radiation Sickness. In 2011, DOE issued a memorandum regarding accommodation of people with Multiple Chemical Sensitivities (“MCS”), including minimizing exposure to electromagnetic fields and radiation because it may trigger their symptoms. Tachover Attachment 2. The memo acknowledges the impact that Radiation Sickness can have on some people and the importance of their home as a refuge.

[I]ndividuals affected by MCS have created "sanctuaries" relatively free from chemical emissions and electromagnetic fields in their homes. Because of the serious impact of even an accidental unavoidable exposure, people often spend as much time at home as possible and often cannot participate fully in society. As a result, they may experience intense isolation, loss of self-esteem, and depression from not being able to have an active work, family, or social life. Supportive professional and peer counseling can help if available.

29. In 2019, the New-Hampshire legislature voted unanimously to establish a committee to learn the effects of 5G and wireless radiation. The committee was comprised of scientists, public representatives, and representatives of the wireless industry (through the CTIA, the wireless industry lobby association). The committee’s majority report published in October 2020 concluded that wireless radiation can be harmful. The New Hampshire committee report acknowledged

Radiation Sickness and the need to accommodate those who suffer from the condition.<sup>31</sup>

30. Almost 200 physicians participated in a recent medical conference about health effects associated with wireless radiation (January 2021).

### **Radiation Sickness prevalence**

31. Radiation Sickness is widespread and the OTARD amendment rule will lead to nationwide crisis. The exact rate of people who have developed the sickness is unknown. Various studies that conducted in European countries up to 2005, indicate a 10% rate.<sup>32, 33</sup>

32. The only available data from the US is a 2002 survey by the State of California's Department of Health Services.<sup>34</sup> The study reported an incidence of

---

<sup>31</sup><http://www.gencourt.state.nh.us/statstudcomm/committees/1474/reports/5G%20final%20report.pdf>.

<sup>32</sup> <https://ecfsapi.fcc.gov/file/109121913528616/Letter%20to%20the%20Editor-%20Will%20We%20All%20Become%20Electrosensitive-Hallberg%2C%20Oberfeld.pdf>.

<sup>33</sup> [https://ecfsapi.fcc.gov/file/1005862318785/Dr\\_Erica\\_Mallery-Blythe\\_EHS\\_A\\_Summary\\_Working\\_Draft\\_Version\\_1\\_Dec\\_2014\\_for\\_EESC\\_Brussels\\_\(3\)%20\(1\).pdf](https://ecfsapi.fcc.gov/file/1005862318785/Dr_Erica_Mallery-Blythe_EHS_A_Summary_Working_Draft_Version_1_Dec_2014_for_EESC_Brussels_(3)%20(1).pdf) pages 3 and 13.

<sup>34</sup> Levallois P, Neutra R, Lee G, Hristova L. Study of self-reported hypersensitivity to electromagnetic fields in California. Environ Health Perspect 2002;110 (Suppl. 4):619–23.

<https://ecfsapi.fcc.gov/file/100518466598/FCC%20comments%20of%20Deborah%20Kopald%2014-177%2C%2015-256%2C%2010-112%2C%20and%2097-95.pdf#page=2>.

3.2%. In California that would mean 1.2 million people and in the US 10.8 million.

This is far more than people who use wheelchairs or other walking assisting devices.<sup>35</sup>

33. However, these studies were done before the exponential increase in wireless radiation exposure. The current rates are likely higher. Further, as stated by Affiant Dr. Toril Jelter, and by Movant Dr. Erica Elliot, the lack of awareness among doctors leads to misdiagnosis. They attest that they are seeing an increase in the number of patients with the condition. A European Parliament resolution from 2012 stated that the problem is growing “exponentially.”<sup>36</sup>

**CHD’s evidence on prevalence and effect of the rule amendment.**

34. CHD’s comments indicate the sickness is indeed now widespread, and the effects of the rule will be devastating to individuals across the country. CHD’s campaign to inform the public about the amended OTARD rule to get people to join our submission to the FCC lasted only about a month. We sent a few emails about it and posted on social media. As a result of this short campaign with limited

---

<sup>35</sup> “Just over 6.8 million community-resident Americans use assistive devices to help them with mobility. This group comprises 1.7 million wheelchair or scooter riders and 6.1 million users of other mobility devices, such as canes, crutches, and walkers.” <https://www.disabled-world.com/disability/statistics/mobility-stats.php>.

<sup>36</sup><https://ecfsapi.fcc.gov/file/100518466598/FCC%20comments%20of%20Deborah%20Kopald%2014-177%2C%2015-256%2C%2010-112%2C%20and%2097-95.pdf#page=2>.

reach and resources, 15,090 people joined our submission. From these, a disturbing number of 6,231 reported that they and/or their children have been injured by exposure.

35. The affidavits included with the Motion also give a glimpse into the huge scale of those who will be affected by the order. Prof. Golomb reports being in touch with scores of hundreds of individuals who suffer from Radiation Sickness. Prof. Bray reports diagnosing 400 patients, including children, and a having a long waiting list. Her affidavit helps show the extent of the problem. Dr. Jelter is in north California has 100 patients including 20 children. Dr. Elliot resides in Santa-Fe, New-Mexico and has 50 patients. Michele Hertz founded an advocacy non-profit in New-York to help those suffer from Radiation Sickness and is in touch with hundreds of sufferers from New York. Dr. Hoffman reports that many of his patients suffer from Radiation Sickness symptoms. Jonathan Mirin reports personally knowing 15 people with radiation sickness who reside in his small rural area. I also know hundreds of adults and children who suffer from the condition and I am contacted by more and more every day.



36. According to a 2011 study funded by the Department of Health and Human Services (“DHHS”),<sup>37</sup> an estimated 43% of US children (32 million) currently have at least 1 of 20 chronic health conditions. Sickness has become the new normal.

37. The January 2021 Swiss government appointed expert committee report concluded that wireless radiation exposure can cause or worsen several chronic illnesses, and that children, especially those with immune deficiencies, or diseases are especially at risk.

38. ADHD rates have increased significantly both in children and adults over the last two decades.<sup>38</sup> A 2018 study shows that ADHD in U.S. children and adolescents has gone up from 6.1% in 1997 to 10.2% in 2016.<sup>39</sup> The evidence that exposure to wireless radiation prenatal and postnatal can lead to ADHD and behavioral problems

---

<sup>37</sup> [https://www.academicpedsjnl.net/article/S1876-2859\(10\)00250-0/fulltext](https://www.academicpedsjnl.net/article/S1876-2859(10)00250-0/fulltext).

<sup>38</sup> [http://jamanetwork.com/journals/jamanetworkopen/fullarticle/10.1001/jamanetworkopen.2019.14344?utm\\_source=For The Media&utm\\_medium=referral&utm\\_campaign=ftm\\_links&utm\\_term=110119](http://jamanetwork.com/journals/jamanetworkopen/fullarticle/10.1001/jamanetworkopen.2019.14344?utm_source=For%20The%20Media&utm_medium=referral&utm_campaign=ftm_links&utm_term=110119).

<sup>39</sup> <https://jamanetwork.com/journals/jamanetworkopen/fullarticle/2698633>.

was established in both animals and human studies.<sup>40,41,42,43</sup> If the amended rule goes into effect, many families like Jennifer Baran's family will be forced in their homes with radiation that is toxic for them.

39. Affiant Jennifer Baran's two sons were diagnosed with various neurodevelopmental problems including ADHD. Her younger son was also diagnosed with Autism and seizures. After she and her husband removed all wireless exposure, inside and outside their home, the doctor who initially diagnosed both her sons with ADHD removed the diagnosis a year later. Her younger son's Autism was improved as well as his seizures.

---

<sup>40</sup><https://ecfsapi.fcc.gov/file/109152869421632/Fetal%20Radiofrequency%20Radiation%20Exposure%20From%20800-1900%20Mhz-Rated%20Cellular%20Telephones%20affect%20neurodevelopment%20and%20behavior%20in%20mice-Aldad2012.pdf>.

<sup>41</sup><https://ecfsapi.fcc.gov/file/10911303968358/Cell%20phone%20use%20and%20behavioral%20problems%20in-young%20children-Divan-2010-UCLA%20Study.pdf>.

<sup>42</sup><https://ecfsapi.fcc.gov/file/1091233914433/Study%20Questions%20Safety%20Of%20Children%27s%20Exposure%20To%20Cell%20Phones%20During%20Prenatal%20And%20Early%20Childhood%20Period-Jonathan%20and%20Karin%20Fielding%20School%20of%20Public%20Health.pdf>.

<sup>43</sup><https://ecfsapi.fcc.gov/file/10911303968358/Cell%20Phone%20Use%20and%20Prenatal%20Exposure%20to%20Cell%20Phone%20Radiation%20May%20Cause%20Headache%20in%20Childre%E2%80%93Smart%26Safe%20EMF%20Solutions.pdf>.

40. According to the Centers for Disease Control and Prevention (“CDC”), the rates of Autism increased 2-fold in 10 years, and the current rate is 1 in 54 children, 1 in 40 in boys. Two studies from 2018, including a government funded study, indicate a higher rate of 1 in <sup>40, 44</sup> There is evidence of a possible association with increased exposure to RF/EMFs.<sup>45,46</sup>

41. While a direct causal link between wireless radiation and Autism has not been established, clinical evidence shows that limiting exposure can lead to dramatic improvement. Affiant Dr. Jelter is a pediatrician with 40 years of clinical experience. She provided 5 case studies showing the effects of removing exposure to wireless radiation have had on children with neurodevelopmental conditions. She reported that a non-verbal 10-year-old said his first sentence 3 days after the parents turned off all wireless devices at night. His violent behavior that led the parents to consider institutionalizing him, stopped as well.

42. The following comment was written by a parent who filed below with CHD:

---

<sup>44</sup> <https://www.webmd.com/brain/autism/news/20181126/report-autism-rate-rises-to-1-in-40-children>.

<sup>45</sup> <https://ecfsapi.fcc.gov/file/1002203764328/Autism%20and%20EMF%20Plausibility%20of%20a%20pathophysiological%20link%20%E2%80%93%20Part%20I.pdf>.

<sup>46</sup> <https://ecfsapi.fcc.gov/file/1002203764328/Autism%20and%20EMF%20Plausibility%20of%20a%20pathophysiological%20link%20%E2%80%93%20Part%20II.pdf>.

I have children with Autism, we have had to implement a lot of safety precautions regarding emfs in our home. Since we have, our children are finally sleeping through the night and my non-verbal child has begun speaking.<sup>47</sup>

43. Youth anxiety is widespread. According to the National Institutes of Health (“NIH”), nearly 1 in 3 of all adolescents ages 13 to 18 experience an anxiety disorder. These numbers have been rising steadily; between 2007 and 2012, in 5 years, anxiety disorders in children and teens went up 20%.<sup>48</sup> Interestingly, 2007 is the year that home Wi-Fi started to be widely available. Furthermore, typical anxiety symptoms (agitation, rapid heartbeat and/or heart palpitations, shortness of breath, chest pain, nausea, dizziness and tingling sensations) are similar to many of the physiological responses people with Radiation Sickness report when they are exposed to wireless radiation. The diplomats experienced “anxiety” as well.<sup>49</sup> Several experts believe many of these children are being misdiagnosed and in fact they suffer from Radiation Sickness.<sup>50</sup> Adults and parents of children with

---

<sup>47</sup> <https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=318>, line 6808.

<sup>48</sup> <https://www.childhealthdata.org/learn-about-the-nsch/NSCH>.

<sup>49</sup> <https://ecfsapi.fcc.gov/file/1091442657471/Cuban%20Embassy-Beatrice%20Golomb%20PhD-Microwave%20Attack.pdf#page=8>.

<sup>50</sup> [https://ecfsapi.fcc.gov/file/1005862318785/Dr\\_Erica\\_Mallery-Blythe\\_EHS\\_A\\_Summary\\_Working\\_Draft\\_Version\\_1\\_Dec\\_2014\\_for\\_EESC\\_Brussels\\_\(3\)%20\(1\).pdf](https://ecfsapi.fcc.gov/file/1005862318785/Dr_Erica_Mallery-Blythe_EHS_A_Summary_Working_Draft_Version_1_Dec_2014_for_EESC_Brussels_(3)%20(1).pdf).

“anxiety” report significant improvement of their anxiety symptoms when they remove exposure to wireless.<sup>51, 52, 53</sup>

44. RF affects sleep. According to a CDC report from 2011, 50-70 million US adults (20%) have a sleep disorder.<sup>54</sup> EEG studies in humans<sup>55, 56</sup> show effects of wireless radiation on sleep. Studies on the effects of cell towers show that one of the most common symptoms is sleep disturbances.<sup>57</sup> A 2015 study showed that 100% of 675 subjects who suffer from Radiation Sickness had decreased levels of melatonin, the sleep hormone.<sup>58</sup>

---

<sup>51</sup> <https://www.fcc.gov/ecfs/filing/10617257800411>.

<sup>52</sup> <https://www.fcc.gov/ecfs/filing/10603278568272>.

<sup>53</sup> <https://www.fcc.gov/ecfs/filing/10618099426968>.

<sup>54</sup> <https://sleepassociation.org/sleep-disorders/>.

<sup>55</sup> <https://ecfsapi.fcc.gov/file/109100958802926/Electromagnetic%20fields%2C%20such%20as%20those%20from%20mobile%20phones%2C%20alter%20regional%20cerebral%20blood%20flow%20and%20sleep%20and%20waking%20EEG-Huber%202002.pdf>.

<sup>56</sup> <https://ecfsapi.fcc.gov/file/10910894904877/Mobile%20phone%20%E2%80%98talk-mode%E2%80%99%20signal%20delays%20EEG-determined%20sleep%20onset-Hung%202007.pdf>.

<sup>57</sup> [https://ecfsapi.fcc.gov/file/100577665439/12b-%20Cell%20Tower%20Studies%20-%20\(Attachment%2012-%20900%2B%20Studies-%20General%20Opposition%20Statement%20-%20File%2013-0953\).pdf](https://ecfsapi.fcc.gov/file/100577665439/12b-%20Cell%20Tower%20Studies%20-%20(Attachment%2012-%20900%2B%20Studies-%20General%20Opposition%20Statement%20-%20File%2013-0953).pdf).

<sup>58</sup> <https://ecfsapi.fcc.gov/file/10912004686552/Reliable%20Disease%20Biomarkers%20Characterizing%20and%20Identifying%20EHS%20%26%20Multiple%20Chemical%20Sensitivity%20as%20%20Etiopathogenic%20Aspects%20of%20a%20Unique%20Pathological%20Disord.pdf>.

45. Suicides and ideation. According to the CDC, there has been a 57% increase in suicides among children and young people aged 10 to 24 between 2007 and 2018. Studies including US government reports acknowledge that RF/EMFs exposure can lead to depression and suicidal tendencies.<sup>59, 60</sup> Dr. Elliot's patient's depression and suicidal tendencies stopped when she removed wireless exposure. Dr. Elliot@¶18.

46. Doctors Golomb, Bray and Jelter all note that Radiation Sufferers begin to lose hope that they can ever find relief, and more people are contemplating suicide. These people do not suffer from a mental condition. It is just they cannot deal with the impossibility of their existence. The life of many who are sick from wireless radiation, has become a living hell and long ago stopped making any sense. The

---

<sup>59</sup><https://ecfsapi.fcc.gov/file/10914872405454/Naval%20Medical%20Research%20Institute-Bibliography%20of%20reported%20biological%20effects...%201972%20Partial%20Report-Symptom%20List.pdf#page=12>.

<sup>60</sup> The Defense Intelligence Agency warned its personnel of the risk from low-level microwaves including illnesses ranging from microwave sickness (flu like symptoms, depression, suicidal tendencies) to cancers and leukemia. Biological effects of electromagnetic radiation (radiowaves and microwaves) - Eurasian Communist Countries, Defense Intelligence Agency: DST-1810S-074-76, March (1976) available at <https://www.dia.mil/FOIA/FOIA-Electronic-Reading-Room/FOIA-Reading-Room-Nuclear-Biological-and-Chemical/FileId/39946/>.



impending OTARD amendment has already made many people feel even more desperate.

47. Numerous people filed comments through CHD's submission below<sup>61</sup> about their children's suffering from wireless radiation and the devastating effects OTARD amendment will have on them.

My special needs daughter is sensitive to this and it will directly effect her! We don't even have Wi-Fi in our home because of the health implications!<sup>62</sup>

My six year old gets migraine headaches from wireless radiation exposure please don't force this killing technology into our neighborhoods against our will and put my son in endless agony.<sup>63</sup>

Our daughter was having seizures while the smart meter was in use, but not anymore. God knows what 5G would do to her.<sup>64</sup>

My daughter is 100% disabled because of radio frequency radiation and it is life threatening. My daughter cannot go anywhere... With 5G she will not survive.<sup>65</sup>

48. Adults who have already been injured begged not to have more radiation forced on them and described their severe and even life-threatening symptoms.

---

<sup>61</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf>.

<sup>62</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=181>.

<sup>63</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=278>.

<sup>64</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=418>.

<sup>65</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=79>.

My wife is 100% disabled from radio frequency radiation. It is life threatening for my wife she will die.<sup>66</sup>

My nervous system is already sensitive to EMF! I am living with constant tinnitus, headaches, palpitation, anxiety...I cannot be subjected to anymore!<sup>67</sup>

I'm already feeling the ill effects of the 5G box that was just installed on a pole on my property.<sup>68</sup>

49. Many others filed comments directly to the FCC record reporting their sickness and describing the impact OTARD rule amendment will have on them.

Electro-sensitive people like myself, who are already unable to live near existing cell towers and antennas because of debilitating physical impacts, will have nowhere to go. Even if we find what we think is a safe place to live, we will not be able to control whether a neighbor installs a 5G antenna right next door. Possibly we will not know it has happened until we become sick. You must not take away community control...<sup>69</sup>

I already live like a hermit avoiding cell towers and meters and humans. I've spent most of the past 7 years sick and in bed 1/2 half of the time. It is hard enough to go to the food store and not be sick, the last thing I need is to have 5G on every street pole.<sup>70</sup>

I am microwave radiation sensitive, it gives me Ocular (eye) migraines where I lose sight then get a horrible migraine. Life was unbearable living across the street from a cell tower and I had to move.<sup>71</sup>

---

<sup>66</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=371>.

<sup>67</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=337>.

<sup>68</sup><https://ecfsapi.fcc.gov/file/105191672708448/Tachover%20Exhibit%202%20List%20of%20Objecting%20Persons.pdf#page=418>

<sup>69</sup><https://www.fcc.gov/ecfs/filing/1011037210617>.

<sup>70</sup><https://www.fcc.gov/ecfs/filing/10618119639263>.

<sup>71</sup><https://www.fcc.gov/ecfs/filing/10618079523172>.

50. Some reported suffering from both brain tumors and Radiation Sickness:

My wife is chronically ill, dealing with disabling effects of brain tumors and autoimmunity, and she is especially sensitive to EMF emissions. Your proposal could very likely cause severe health consequences for my wife and many others, and it is at odds with our Constitutionally protected rights to Life, Liberty and the pursuit of Happiness.<sup>72</sup>

I am a resident of Elburn with multiple brain tumors, severe migraine disorder, and vertigo. These symptoms are exacerbated greatly by EMF emissions from wireless devices to the point of being quite debilitating. The proposed rules would allow the proliferation of EMF-emitting wireless antennas without regulation, blocking the assertion of my rights and my due process. The installation of 5G on my property or in my town without my or my town's consent would be disabling to my health and infringes upon my rights as well as state's and town's rights.<sup>73</sup>

51. Given the prevalence of Radiation Sickness, the OTARD amendment will have vast implications on large and segments of the population and will certainly exacerbate this rapidly growing but outrageously ignored public health problem.

**CHD's organizational standing.**

52. I know first-hand about the torturing pain, the disabling symptoms, and the harsh reality of suffering sickness from wireless radiation. In 2009, like many others, I too developed Radiation Sickness. I was evaluated and diagnosed by three doctors. Professor William Meggs, MD, PhD, a Prof. of Emergency Medicine and Toxicology who blind tested me. I could reliably detect EMFs. The blinded test

---

<sup>72</sup> <https://www.fcc.gov/ecfs/filing/1061795629597>.

<sup>73</sup> <https://www.fcc.gov/ecfs/filing/10617547901248>.

also showed that my heart rate increased whenever I was exposed. Like many who suffer severe symptoms, it is increasingly challenging for me to spend time “in the world.” After one year of intense exposure to Wi-Fi from neighbors, because I could not find a safe house, I developed daily headaches, difficulty sleeping and heart palpitations and cognitive issues. I had blood tests done and they were shocking. My thyroid hormones are out of balance and in dangerous levels; the inflammation in the high-risk zone including for a heart attack. My sugar levels were diabetes borderline. I had gained a lot of weight but not due to diet. I was losing my sense of touch, a sign that my nervous system is shutting down.

53. I had to leave and eventually found a safer home. I now live in a rural area with barely tolerable RF levels but that could quickly change. Like many others who have Radiation Sickness, OTARD will have immediate and irreparable impact on my life. In fact, it already has. I have 4 neighbors who are about 150 feet away from me. If a hub/relay is installed by one of them or nearby, with no notice, I will learn about it by getting sick. I will have to leave and go back to living in my car. I live in daily fear of that happening. In anticipation of that day, knowing I will have to leave fast, I keep my possessions to a minimum, only what I can fit in my car. Of course, if I have nowhere to live, there is also a real likelihood that I will not be able to keep my job nor will I be able to find a new one. After being sick for already 10 years, I do not have financial resources and if I lose my job, I cannot

support myself financially. I do not know where to go from here and how I will be able to even just survived. I survived many years of inhumane existence with Radiation Sickness because I am practical and strong, But OTARD leaves me and many others with no way to exist.

54. I also experience the consequences of the constant increase in exposure every day in my work. I am contacted daily by people who became sick after antennas were installed near their homes or by those who realize that their illness is caused by their wireless devices. I am contacted by parents whose children are severely sick because of cell towers in or near their children's school. I am contacted by doctors who are becoming aware of the significant role wireless radiation has in the sicknesses they see in their clinics. I am contacted by parents who are looking for a doctor who is aware of the health effects of wireless technology in order to diagnose their child so they can ask for accommodation.

55. I am also contacted daily by people who are sick from wireless radiation, and the increased exposure is making it is impossible for them to continue to stay in their homes. Despite their efforts, they cannot find a safe place to live where they will not suffer every minute of their existence so they can sleep, think, and function. Very few such places now exist, mainly in rural areas. Some found a place. Others are on the roads looking, some for many months and even years.

Others just live in their cars, constantly. The rule amendment will cause more to be in this desperate situation.

56. For the past 4 years, I have been traveling around the country to educate federal, state and local public officials, doctors and communities. I have given many dozens of public lectures. I have presented to hundreds of elected officials to educate them on the impact of uncontrolled deployment of wireless on many of their constituents and help the injured in their efforts to get accommodation.

57. The motivation behind my work in the past 9 years has always been protecting children and those who have become sick by wireless radiation.

Therefore, I was grateful when CHD decided to address this grave public health issue. CHD has provided a good platform to work for recognition, change and a way to help those who suffer from radiation.

### **CHD's organizational standing and irreparable harm**

58. Children's Health Defense has Article III standing to bring and prosecute this case and motion on behalf of its members.

59. The decision under review has directly injured our members who developed Radiation Sickness or other sickness associated with wireless exposure or a condition which is aggravated by wireless exposure. 823 of our members filed to the docket together with CHD and declared that they and or their children are sick



from wireless radiation. Without a stay immediate and irreparable harm will be caused to them.

60. Children Health Defense participated below.<sup>74, 75</sup> CHD has Hobbs Act and Article III standing and grounds to pursue this matter on its own behalf and in a representative capacity for its members.

61. CHD is essential to the many more adults and children who are suffering as we speak, feeling anxious and hopeless. They are afraid of the devastating effects on their health and lives that no doubt will follow if the rule goes into effect.

62. The OTARD rule amendment will cause immediate and irreparable harm to CHD's ability to achieve our mission and our ability to assist our members and the public. These harms go far beyond, and are in addition to, the time and significant resources dedicated to prosecution of this petition for review and motion for stay.

63. A central part of our work is supporting those who need accommodation because of their wireless radiation related sickness. For example, we recently assisted Affiant Michele Hertz when she needed accommodation from exposure caused by a private company.

---

<sup>74</sup> <https://www.fcc.gov/ecfs/filing/104171342025759>.

<sup>75</sup> <https://www.fcc.gov/ecfs/filing/105191672708448>.

64. CHD has had to allocate substantial human and financial resources to address and mitigate the societal harms created or maintained by the FCC's rule amendment. CHD has already had to divert resources from other projects unrelated to wireless matters. For example, a paid legal intern was relocated from another project to assist.

65. The FCC rule amendment will require that CHD invest additional resources toward advocacy, counseling, referrals, education, and other legal actions related to its Wireless Harms Project. CHD has had to increase the amount of work hours of its managerial, professional, and other personnel to help maintain and update its website with campaigns, articles and answering a growing number of emails, phone calls and other requests for assistance. The additional workload has already required that I hire additional professionals that the organization did not budget.

66. To address the overwhelming implications of the OTARD amendment, we will have to intensify our efforts to educate the public about the harms that these hubs/relays may cause their sick neighbors. Hopefully, as a result, people will refuse to enter contracts with fixed wireless companies to install these devices on their homes. I have approached a graphic designer to help us create memes (images) for that effort.

67. The OTARD amendment will increase radiation levels in homes. As a result, it will increase the number of people who will approach us for advice on how to

shield their homes. We can refer people to EMF mitigation specialists. However, hiring an expert can be expensive and those who are sick many times cannot work and have no financial means so we must find alternatives. We will have to create more webinars, write more articles to provide people with advice and guidance on things they can do themselves.

68. To support our many members who are injured by the new rule, if a stay is not granted, we will try and budget to pay an attorney to help people file lawsuits and seek damages under the Tucker Act, the only recourse that will stay available to the injured.

69. The FCC's OTARD rule amendment will lead to increased and unnecessary wireless exposure and will impose more, higher, and even impossible hurdles to those who have already become sick. In turn, these people are coming to us for help in ever-growing numbers. We will have more demand for referrals, more demand for information, more demand for participation in educational events and more requests for mitigation advice. The rule amendment will exponentially increase these demands.

70. We get more desperate calls from people who are sick and feeling increasingly worse from growing radiation levels. They ask for advice as to where they can go and who can help them. OTARD will make this problem exponentially worse as many people will be forced out of their homes.

71. As part of our efforts to address the harms that will be created by the amended OTARD, we have been working with a group of other organizations to educate the public about the effects that will be caused by SpaceX deployment of 42,000 satellites and 1,000,000 on the ground antennas to offer a competitor service to existing internet providers. The OTARD rule amendment facilitates this massive satellite ground infrastructure. We have supported efforts for a protest, participated in a nationwide letter campaign, gave interviews on TV and radios and wrote articles on the subject. We will have to expand these efforts.

72. CHD's EMF Child Ambassador Project aims to encourage and support children to get involved and educate their peers about the science and how to reduce exposure. For example, we support children who decided to conduct their science projects on wireless harms. We provide them information, RF meters to measure exposure levels and offer access to experts who can assist them. We publish their work on our social media platforms. Through this project we also support those children whose parents are sick or who they are sick themselves.

73. The sick children, as the affidavits indicate, are experiencing severe pain and physiological injuries. In addition, they encounter an abusive system that denies and/or disregards their condition and rights. Just as in the case of Ginger Kessler and Angela Tsiang's children, many of these children are forced out of school, into isolation and lose social connections. Their home has become their whole world

and the only place they can exist with fewer symptoms scary pains. Their future looks rather bleak. Our Child Ambassador Project supports and aims to empower the sick children to speak up about their experience, have a voice and encourages them to act and work towards change. The OTARD amendment will require us to expand that program.

74. Youth suicidal ideation is increasing. OTARD will aggravate the psychological damage caused to the sick children from being ignored, dismissed, ridiculed, being regarded as an ‘obstacle’ by their government that now forces them out of their homes. More children will contemplate committing suicide. CHD will have to invest more efforts to support these children.

75. A decision by this Court to stay the FCC order would provide meaningful relief. It would give some small hope to all those who suffer every day, will allow them for now to stay in their homes and not lose their health and livelihood. It would allow them to maintain their status quo which is indeed dire but exponentially better than with OTARD amendment in effect. A stay will maintain some chance of recovery of and some hope for a tolerable existence. It would also validate their condition and remove the stigma flowing from the FCC’s disregard of their sickness and existence. It will give them some little hope there is and will always be a place for them in society. It may well prevent scores of suicides.

**CHD in representative capacity**

76. Several of the affidavits (Michele Hertz, Dr. David Hoffman's wife and daughter, Jennifer Baran and her two sons) included in the Motion are from CHD members who suffer from Radiation Sickness or have family members with the affliction. Two of them participated below. Michele Hertz filed independently to the record below and Dr. David Hoffman, joined CHD's filing. They describe their injuries in their affidavits. CHD is representing their interests, and the hundreds of others that also have the condition.

77. Our sick members and their children need to be protected. Instead, the OTARD rule amendment removes what little protection they presently have. It will require us to expend more resources trying to identify some means to help those who come to us and then taking what few avenues remain. We will have more to do, but fewer options, and those that still exist will cost more. Actions against governmental bodies and large private companies are complex and expensive.

**Effects on CHD Members**

78. Central to the OTARD rule amendment is extension of the communications link afforded by a fixed wireless arrangement to individual user end points using Wi-Fi or a similar wireless local area network topology that can now traverse property lines and irradiate people who have valid reasons to object to exposure and have little or no relationship to the person who sets up the arrangement.



79. A major cause of concern for our members and thousands like them is that OTARD hubs and switches will be located in close proximity to their homes and even their children's bedroom windows. The radiation will invade people's homes and the already pervasive radiation will become even more intense.

80. The rule also takes-away what little due process remains for those whose lives are ruined. One of the most concerning aspects is that under OTARD, "hubs" and "relay" antennas can be deployed without any duty to provide notice or duty to consider accommodation requests. This expansion of the preemption of local zoning procedures and homeowner association deed restrictions – in contrast to the situation where those procedures and restrictions still apply for common carrier personal wireless services – will undoubtedly lead to more unconstrained proliferation of wireless networks. Indeed, that is the stated purpose.

81. Planned Communities. Those who moved to a planned community, in order to avoid wireless radiation and to be somewhat protected because of bylaws that restrict antenna deployment especially for commercial purposes, will lose all their rights, and their plans and expectations will be crushed together with their health or the health of their children or spouses.

82. Safe Communities. I am aware of groups of people who are working to establish wireless-free communities. They worked hard to find and purchase or lease homes in these communities. The OTARD amendment will undo all these

efforts because homeowners' association rules, deed restrictions and all state-law based contracts are preempted and rendered unenforceable.

83. Effect on children. The effect the rule amendment will have on children is especially concerning. Growing numbers of children are becoming sickened by radiation sources at or near their schools. One major cause is wireless local area networks – the very thing facilitated by the rule amendment. I cannot keep count of the conversations I had with members' children and how this condition has impacted them. Many expressed being afraid that their peers will learn about it. Their lives have been ruined and their future have been robbed. They too are desperate and often hopeless. I know of a 15-year-old with Radiation Sickness who hanged herself from a tree.

84. Affected parents must remove their children from these schools and provide home schooling, but the growing forced exposure within people's own home is making it impossible for these children to get better and they are getting worse. Their home is their entire world: as cell towers have been installed in parks, playgrounds and even churches, the increasing levels of radiation outside their homes is intolerable to them. Their lives are very limited and isolated, but this rule amendment will eliminate even the safety within the home.

85. Financial implications. According to a survey conducted by Prof. Golomb, 50% of those who became affected by wireless radiation, had to leave their job.

Those who were able to keep their job because of the effects of the OTARD amendment may lose their livelihood and ability to financially sustain themselves. For those whose sickness already made it impossible to have a job, further deterioration to their health caused by wireless expansion that affects their home will put them in an even worse financial situation.

86. No Refuge. Many of those who have been injured will be forced (once again) to leave their homes in search of the next refuge, but there will be no refuge. The *Order* states that the rule change will enable significant deployment and therefore expand wireless broadband, especially in rural areas. I have no doubt this is true. The problem is that it will increase wireless radiation in the few remaining areas where it is still possible to find refuge from such radiation. Even in rural areas, a place that is safe one day could easily become unsafe the next.

87. The OTARD amendment will subject the injured to more radiation and take away the little control and due process rights that they currently have. The plain effect of the rule amendment is that people who are already injured or that will be injured from the new emissions source will suffer further irreparable harm to their health. They will not be able to seek and obtain accommodation under state disabilities laws. Nor – given the scope of claimed preemption of state tort laws – can the injured receive any compensation after the fact. Therefore, the rule

amendment will have intolerable and irreparable adverse effect on the health and finances of those who are injured.

88. CHD members and CHD have each and all suffered and will suffer concrete and particularized injuries traceable to the FCC's decision. These injuries are redressable by a stay from the court, thereby meeting Hobbs Act and Article III standing requirements.

**The balancing of interests undoubtedly and overwhelmingly supports a stay.**

89. Maintaining the status-quo until a final decision in the case is in the public's interest. Unbearable and irreparable harm will befall the Movants, Affiants, their children and their families. Immense and irreparable harm will also be caused to many all around the country including children. This rule harms those who are the most vulnerable and they are many. For those who suffer from Radiation Sickness and for others whose conditions may be exacerbated by wireless radiation with conditions like ADHD, Autism and seizures among others, the rule amendment takes away their right to exist even in their home.

90. The rule will cause irreparable harm to their health, to some the harm can be life threatening like to Movant Mirin's wife, Affiant Dr. Hoffman's wife and Affiant Baran whose son suffers radiation-induced seizures. It will cause emotional damage as was described by Movant Tsiang and her son. They will suffer unrecoverable financial costs for shielding their homes or from having to leave

their homes and losing jobs. But the worst, they will have to choose between staying in their homes and being extremely sick or leaving but having nowhere to go.

91. CHD members and CHD have suffered and will suffer concrete and particularized injuries traceable to the FCC's decision. These injuries are redressable by a stay from the Court, thereby meeting Hobbs Act and Article III standing requirements.

92. Every house in the US currently has access to some kind of broadband internet. No real harm will be caused to people who want it. Those who seek to have broadband internet access and currently do not have DSL, cables, or fiber optic, can still have access to "own property" fixed wireless including point to point wireless and satellite.

93. The only advantage the OTARD amendment will offer to users is competition and therefore possibly cheaper options. But of course, since people are unaware about the harm wireless radiation can cause, they do not take into consideration the potential costs of developing sickness, the burden on society or the tax and personal impacts.

94. The harm to those who are sick, its nature, scale and scope far outweighs any harm that may be caused to the providers of fixed wireless. The rule

amendment allows these companies to expand the services they offer, but they do not yet have a vested interest in these services. In fact, a stay is also in their best interest. If Petitioners prevail in the case and the court vacates the amended rule, these companies may incur losses. A stay is in the best interests of all concerned.

95. The harm that will be caused by the FCC's OTARD amendment is vast, devastating, and irreparable. The rule amendment will immediately and undoubtedly increase the size of an already-enormous though ignored and suppressed problem. The effects of this rule amendment are egregious and morally and legally unconscionable.

96. This concludes my Affidavit.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on March 18, 2021



---

Dafna Tachover



**TACHOVER Attachment 1**



# Effective Accommodation Practices (EAP) Series

## Job Accommodations for People with Electrical Sensitivity

Job Accommodation Network  
PO Box 6080  
Morgantown, WV 26506-6080  
(800)526-7234 (V)  
(877)781-9403 (TTY)  
[jan@askjan.org](mailto:jan@askjan.org)  
[askjan.org](http://askjan.org)



## JAN'S EAP SERIES

### JOB ACCOMMODATIONS FOR PEOPLE WITH ELECTRICAL SENSITIVITY

Electromagnetic sensitivity, also known as electromagnetic hypersensitivity, electrical sensitivity, electro-magnetic sensitivity, and idiopathic environmental illness (IEI), has been difficult for the environmental health and medical communities to define. Individuals with electromagnetic sensitivity may experience various non-specific symptoms including but not limited to fatigue, weakness, neurological issues, immunological issues, gastrointestinal issues, increased irritability, lack of ability to think clearly and quickly, sleep disturbance, overall malaise, and anxiety.

Individuals with electromagnetic sensitivity typically report managing symptoms by avoiding exposure to electromagnetic fields (EMFs) that trigger their symptoms. They often make modifications to their homes and daily routines to minimize exposure through avoidance of EMFs and reduce their overall long term exposure to EMFs. When it is not possible to avoid it, then limiting duration and strength of exposure and use of shielding may also be useful. Based on data from JAN calls, common workplace issues involve exposure to Wi-Fi, cell phones, and computer equipment such as CPUs and monitors.

According to a review of literature by Martin Rösli 2007<sup>1</sup>, a causal relationship between short term exposure to EMFs and elicitation of symptoms has been challenging to substantiate under laboratory conditions. However, population based studies involving longer term exposure have shown correlation between long term exposure and symptoms such as headache, cold hands or feet, and concentration difficulties. Research on this topic is ongoing.

The National Institute for Occupational Safety and Health (NIOSH) and the Centers for Disease Control and Prevention (CDC) have published guidelines for “safe” levels of human exposure in a publication called, Manual for Measuring Occupational Electric and Magnetic Field Exposures. However, the nature of electromagnetic sensitivity is such that even levels that are deemed safe for the general public can cause trigger symptoms for individuals who are hypersensitive. Individuals affected by electromagnetic sensitivity experience symptoms at far lower levels and therefore may need accommodations in the workplace beyond the safe levels of exposure indicated in the manual.

---

<sup>1</sup> Science Direct Environmental Research 107 (2008) 277–287 Radiofrequency electromagnetic field exposure and non-specific symptoms of ill health: A systematic review Martin Rösli Institute of Social and Preventive Medicine, Department of Social and Preventive Medicine, University of Bern, Finkenhubelweg 11, CH-3012 Bern, Switzerland Received 21 September 2007; received in revised form 4 February 2008; accepted 6 February 2008 Available online 21 March 2008 Retrieved 2/12/2015

Organizations such as the World Health Organization (WHO) and the United States Access Board, which offers technical assistance on the ADA Accessibility Guidelines, have issued statements and regulatory guidelines related to electrical sensitivity. The World Health Organization (WHO) held an international workshop on the issue in Prague, Czech Republic, in 2004. WHO recognizes that a significant number of people report symptoms after exposure to electromagnetic radiation that range from neurological and immunological to gastrointestinal issues (WHO, 2005). The Access Board addressed electromagnetic sensitivities as part of the IEQ Indoor Environmental Quality Project.

The following is a quick overview of some of the job accommodations that might be useful for people with electrical sensitivity. For a more in depth discussion, access JAN's publications at <http://AskJAN.org/media/atoz.htm>. To discuss an accommodation situation with a consultant, contact JAN directly.

### **General Accommodation Considerations**

- Allow communication via typewriter or handwritten notes rather than via computer or cover the computer with Plexiglas or other shielding material.
- Provide headset/handset extenders or alternate headsets to lengthen the distance between devices that trigger symptoms and the employee's body.
- Change the employee's shift to allow for less exposure to others' devices.
- Relocate workplace away from areas where symptoms are triggered. This may include limiting certain types of devices in the vicinity of the employee's workstation.
- Allow telework (Note: regarding work at home, unless the employee wants to work at home, other options should be explored first to keep the employee in the workplace).
- Allow the employee to meet with others in areas where triggers are minimized or allow remote access to meetings or activities that must take place in areas that trigger symptoms.
- Provide wired telephones and network connections.
- Provide building-wide and/or workspace shielding of equipment and devices, for example add filters to fluorescent lights and tape electrical cords.
- Individuals with electrical sensitivity may also experience limitations from fragrance sensitivity and/or photosensitivity.

Updated 04/28/15

This document was developed by the Job Accommodation Network (JAN). Preparation of this item was funded by the Office of Disability Employment Policy, U.S. Department of Labor, Grant Number OD-23442-12-75-4-54. This document does not necessarily reflect the views or policies of the Office of Disability Employment Policy, U.S. Department of Labor, nor does the mention of trade names, commercial products, or organizations imply endorsement by the U.S. Government.

**TACHOVER Attachment 2**



UNITED STATES DEPARTMENT OF EDUCATION  
OFFICE OF SPECIAL EDUCATION AND  
REHABILITATION SERVICES ADMINISTRATION  
REHABILITATION SERVICES ADMINISTRATION  
WASHINGTON, D.C. 20202

INFORMATION MEMORANDUM

RSA-IM-02-04

DATE: NOVEMBER 5, 2001

ADDRESSEES: STATE VOCATIONAL REHABILITATION AGENCIES (GENERAL)  
STATE VOCATIONAL REHABILITATION AGENCIES (BLIND)  
STATEWIDE INDEPENDENT LIVING COUNCILS  
CENTERS FOR INDEPENDENT LIVING  
CLIENT ASSISTANCE PROGRAMS  
PROTECTION & ADVOCACY OF INDIVIDUAL RIGHTS  
PROGRAMS  
REGIONAL REHABILITATION CONTINUING EDUCATION  
PROGRAMS  
AMERICAN INDIAN VOCATIONAL REHABILITATION  
PROGRAMS  
RSA SENIOR MANAGEMENT TEAM

SUBJECT: Multiple Chemical Sensitivity

CONTENT: In order to help RSA grantees and other service providers better understand and address the needs of persons with Multiple Chemical Sensitivity (MCS), RSA is distributing the attached document developed by the Arizona Technology Access Program, Institute for Human Development, Northern Arizona University. This document contains information on symptoms associated with MCS, potential causes of MCS, and suggestions on ways to accommodate individuals with MCS. The document also contains a listing of resources on MCS for consumers and professionals.

If you have any questions regarding this IM, please contact Dr. Thomas E. Finch at (202) 205-8292, or via email at [tom.finch@ed.gov](mailto:tom.finch@ed.gov).

---

Joanne Wilson  
Commissioner

Attachment

cc: NATIONAL COUNCIL ON INDEPENDENT LIVING  
COUNCIL OF STATE ADMINISTRATORS OF VOCATIONAL REHABILITATION  
NATIONAL ASSOCIATION OF PROTECTION AND ADVOCACY SYSTEMS  
NATIONAL REHABILITATION FACILITIES COALITION  
NATIONAL ORGANIZATION OF REHABILITATION PARTNERS

## Multiple Chemical Sensitivity

Chemical-based products are all around: in the clothes we wear, in the food we eat and in the air we breathe. It is not possible to escape exposure. Many people have become sensitized to the chemicals around them. It is estimated that as much as 15% of the population has become sensitized to common household and commercial products.

For some people the sensitization is not too serious a problem. They may have what appears to be a minor "allergy" to one or more chemicals. Other people are much more seriously affected. Such people have a condition known as Multiple Chemical Sensitivity or MCS.

### What is Multiple Chemical Sensitivity?

MCS is a disorder triggered by exposures to chemicals in the environment. Individuals with MCS can have symptoms from chemical exposures at concentrations far below the levels tolerated by most people. Symptoms typically occur in more than one area in the body, such as the nervous system and the lungs. Exposure may be from the air, from food or water, or through skin contact. The symptoms may come and go with exposures, though some individuals may have delayed reactions. As MCS gets worse, reactions become more severe and increasingly chronic, often significantly affecting bodily functions.

In the early stages, repeated exposure to the substance or substances that caused the initial health effects provokes a reaction. After a time, it takes less exposure to cause symptoms. An increasing number of chemical products may trigger a reaction, including some unrelated to the initial exposure.

Most frequently, MCS affects an individual's overall physical and emotional health. It typically impairs the nervous system. It may affect the digestive and respiratory systems as well. A chemically sensitive person may also have other pre-existing health conditions. Many affected people experience a number of symptoms with each chemical exposure.

### Symptoms of MCS

- asthma or other breathing problems
- autoimmune disorders
- behavioral problems
- bloating or other intestinal problems
- cardiovascular irregularities
- chronic exhaustion
- disorientation or becoming "lost"
- dizziness
- dystonia (paralysis)
- ear, nose and throat problems
- fatigue and depression
- flu-like symptoms
- food allergies and intolerances
- genitourinary problems

MCS may result from a single massive exposure to one or more toxic substance or repeated exposures to low doses. Some people become chemically sensitive following a toxic chemical spill at work or in their community, or after exposure to pesticides. Or, individuals may develop

this condition from spending time in a poorly ventilated building, where they breathe a combination of chemicals. MCS may be brought on by a wide array of chemicals found at home, at work, in hospitals, in parks, and at school.

### **People Diagnosed with MCS**

Studies have found that many people diagnosed with MCS were:

- industrial workers
- teachers, students, office and health care workers in sealed buildings
- chemical accident survivors
- people living near toxic waste sites
- people whose air or water is highly polluted
- people exposed to various chemicals in consumer products, food, and pharmaceuticals
- Gulf War and Vietnam Conflict veterans
- 

Not all people with MCS fit into these categories. For example, some have experienced a toxic exposure from flea and roach sprays, or from foam insulation (urea formaldehyde) in their home. Other people with MCS cannot readily identify situations where they have been exposed to chemical products.

People with MCS may become partially or totally disabled for several years or for life. They must make fundamental changes in lifestyle and at home. Their marriages and other relationships may end from the stress of coping with this disabling condition. They may drag themselves to work only to return home sicker and more exhausted each day. They may be forced to leave their jobs and deal with the devastating loss of income and health insurance. Some people eventually recover, but few return to complete health.

### **What Can Cause MCS?**

No one knows for sure what causes MCS. However, in non-industrial workplaces, a number of common products and processes have been identified as contributing to the onset of MCS. Some exposures that have been linked to this condition are:

- Agent Orange for Vietnam Veterans
- antibiotics and other medication
- carbonless paper, inks, copying machine and laser printer toner
- cleaning supplies
- DEET - an insect repellant which may have been a significant trigger for Gulf War Veterans
- formaldehyde in new clothes, books, and other products
- gas stoves
- house paints
- insecticides, synergists, piperonal butoxide
- new building materials and furnishings
- off gassing of new carpets (styrene butadiene latex in flooring adhesives and carpet backing)
- pesticides and wood preservatives
- second-hand tobacco smoke
- toxic chemicals used in art, photography, printing, etc.

- vehicle exhaust fumes

These substances contribute to indoor air pollution and are often contaminants in our air and water. Many of the chemicals that trigger MCS symptoms are known to be irritants or to be toxic to the nervous system. One especially harmful group of chemicals, known as "volatile organics," readily evaporates into the air at room temperature. Even low airborne levels of such contaminants can make ordinary people sick. The impact on health of long-term, low level exposure to most chemicals found in consumer products remains untested. The products and other chemicals that cause problems varies among affected individuals.

Commonly reported triggers include:

- anesthesia
- artificial colors, flavors, and preservatives in foods, drinks, and drugs
- detergents and other cleaners
- electromagnetic fields
- fluorescent lights
- perfumes and fragrances
- prescribed medications
- smoke from tobacco products
- solvents from dry cleaning, felt pens, etc.

When our bodies are assaulted with levels of toxic chemicals that cannot be safely processed, many of us become ill. For some, the outcome could be cancer or reproductive damage. Others may become hypersensitive or develop other chronic disorders, while some people may not experience any noticeable health effects. Even when high levels of exposure occur, only a small percentage of people become chemically sensitive. The threshold of toxic injury is not the same for everyone because the ability to detoxify varies greatly from individual to individual.

### **Treatments**

MCS can be difficult for physicians to define and diagnose. Physicians should take a complete patient history, which includes environmental and occupational exposures, carefully test for familial or exposure-related tendencies like porphyria, use brain and brain function scans, and act as diligent detectives in diagnosing this condition. After the onset of MCS, a person's health generally continues to deteriorate. It may only begin to improve once the chemical sensitivity condition is uncovered. While a number of treatments may help improve the health of some patients, there is currently no "cure." In almost all cases, avoidance of exposures must be practiced to alleviate symptoms. No single test for MCS currently exists.

Avoiding the exposures that may trigger reactions is essential, and may permit dramatic improvement. Yet the large number of new and untested synthetic chemicals we encounter in our daily lives makes this extremely difficult.

Individuals affected by MCS have created "sanctuaries" relatively free from chemical emissions and electromagnetic fields in their homes. Because of the serious impact of even an accidental unavoidable exposure, people often spend as much time at home as possible and often cannot participate fully in society. As a result, they may experience intense isolation, loss of self-esteem,

and depression from not being able to have an active work, family, or social life. Supportive professional and peer counseling can help if available.

### **MCS and the Medical Community**

Many conventional allergists and other physicians claim that there is not yet sufficient evidence that MCS "exists". Research regarding the mechanisms that cause MCS has been inadequate, and unfortunately is often financed by the industries, which benefit from chemical proliferation. Generally, medical doctors have not been trained to understand or seriously investigate conditions such as MCS. In fact, the vast majority of physicians receive very little training (four hours or less) in occupational and environmental medicine or in toxicology and nutrition.

Therefore, many affected individuals have to consult with a large number of specialists. People with MCS are sometimes misdiagnosed with serious degenerative diseases. Often, baffled doctors tell patients that their illness is psychosomatic...in their head. And many whose health is impaired by MCS have never heard of the condition. The lack of support from physicians, and the stress caused by having no explanation for symptoms, may contribute to a high level of anxiety and distress for people with MCS.

Conventional medicine offers very few medical treatments for MCS besides avoiding offending products. Unfortunately, medications and other conventional medical treatments offer little or no relief, and may even prompt new sets of symptoms. Treatment with anti-depressants may mask the underlying condition and can also cause other serious health problems.

Physicians who clearly recognize MCS include some occupational and environmental health specialists. A wide range of new or "alternative" treatments have been utilized by people with MCS with varying success. Some of these treatments are experimental and may include a combination of: nutritional programs, immunotherapy vaccines, food-allergy testing, detoxification regimens through exercise and saunas, chelation for heavy metals, and other healing treatments. Diagnosis may involve laboratory tests for chemical contaminants, such as total body burden of pesticides, or for porphyria, respiratory and brain function.

Unfortunately, these treatments and diagnostic workups are not often reimbursed by insurance plans. Few practitioners or medical insurance programs for people who are indigent support these alternative, yet sometimes productive approaches. Some disabled workers have won reimbursement for such care through Workers Compensation claims.

### **MCS Is Now Recognized as a Disability**

Both the US Department of Housing and Urban Development (HUD) and the Social Security Administration (SSA) have recognized MCS as a disabling condition. People with MCS have won several Workers Compensation cases. A recent human rights lawsuit in Pennsylvania established the right of an affected person to safe living space in subsidized housing. Both the Maryland State Legislature and New Jersey State Department of Health have commissioned studies of MCS. The NJ study provides an excellent overview of medical and legal issues related to MCS.



Just as physical barriers prevent wheelchair access, chemical use can prevent entry and use of public facilities to those with MCS. The Americans with Disabilities Act (ADA) protects people with disabilities from many types of discrimination, requiring reasonable accommodation for people with disabilities. Reasonable accommodations can enable people with MCS to enjoy access to work, public facilities and other settings. Whether an individual developed MCS at work or was already sensitized prior to employment, the right to a safe workplace must be established.

Injured workers who need Workers Compensation or Social Security Disability benefits should find a physician who can diagnose MCS and who will support legitimate claims. Establishing clear documentation is critical in awarding such a claim, as well as for gaining reasonable accommodation at work or for rental housing. If your employer is discriminating, do the following:

- get your condition diagnosed
- if you work in a unionized workplace, consult with your union about filing a grievance or taking legal action
- seek legal counsel
- join a support group

For further assistance, contact a worker health resource group or support group in your area, as well as other organizations listed at the end of this fact sheet. These cases can be difficult and take a long time, but can be resolved.

Similarly, if you have been injured on the job, find an attorney experienced with chemical exposure cases in the Workers Compensation system or personal injury claims. You will not need to pay your attorney up front. Your attorney receives a percentage from the settlement if you win. It typically costs you nothing if your case is unsuccessful. To find an attorney, consult your union or one of the organizations listed below to obtain referrals. Select your attorney carefully. Remember, you should trust and feel comfortable with him or her.

### **Accommodating Individuals with MCS in the Workplace**

These are some suggested ways to accommodate individuals with MCS at work. While not adequate in all cases, these measures will help prevent other workers from becoming similarly disabled, and contribute to the creation of a healthier work environment.

- windows that open to permit fresh air to circulate
- well ventilated space free of pollutants such as tobacco smoke, pesticides, toxic and fragrance-laden cleaning products, deodorizers
- selection of least toxic/allergenic building furnishings, flooring and supplies
- "least toxic" integrated pest management (IPM) using no sprayed or volatile pesticides in or around buildings
- pre-notification prior to painting, pesticide applications, and renovations, with provisions for alternative work arrangements
- education of co-workers, management, and other employers to avert stigma and harassment
- scheduling which permits people with MCS to work when fewer co-workers are present, when ventilation is at its peak, or where the work environment is least problematic

- allowing the option of working at home or off site
- minimizing exposure to electromagnetic fields from computers, fluorescent light ballasts, and other equipment

**MCS Is Preventable**

People with MCS are a driving force for improved indoor air quality and for the adoption of less toxic housekeeping and building maintenance practices. Good indoor air quality and substitution of less toxic materials boost morale and productivity. A healthy workplace lowers absenteeism and injuries. Complaints about indoor air problems must be taken seriously by employers, labor unions, regulatory agencies, and health care and social service providers.

## For Help and Information

### **National Center for Environmental Health Strategies (NCEHS)**

(609) 429-5358

Mary Lamielle, Director

1100 Rural Avenue

Voorhees, NJ 08043

Provides information, referral, and advocacy. Tracks scientific, legislative, legal, medical, and policy issues. Twice yearly newsletter, "The Delicate Balance." Information packets.

### **NY Coalition for Alternative Pesticides (NYCAP)**

(518) 426-8246

P.O. Box 6005

Albany, NY 12206-0005

Focuses on pesticide hazards and safer alternatives. Provides information, referral, workshops, and advocacy "NYCAP News" is its 40 page quarterly newsletter. Incident reporting project.

### **MCS Referral and Resources, Inc.**

Albert Donnay

508 Westgate Rd., Baltimore, MD 21229-2343

(410) 362-6400 Voice (410) 362-6401 Fax

Initiated by Grace Ziem, MD, DPH, to assist people with MCS, physicians, attorneys, and other professionals. Distributes articles and resources on prevention, diagnosis, accommodation.

Contact Albert Donnay online at: [donnaya@r.tk.net](mailto:donnaya@r.tk.net) or visit their homepage at [www.mcsrr.org](http://www.mcsrr.org).

### **The Environmental Health Network**

(415) 541-5075

P.O. Box 1155

Larkspur, CA 94977

Newsletter, "The New Reactor", MCS advocacy and survival primer, "The Good Old New Reactor," by Susan Molloy, is available for \$8.95 plus s/h.

### **Chemical Injury Information Network**

(406) 457-2255

Cynthia Wilson, Director

P.O. Box 301

White Sulphur Springs, MT 59645

"Our Toxic Times: monthly newsletter

### **Center for Safety in the Arts**

(212) 227-6220

5 Beekman Street, Suite 820

New York, NY 10038

Provides information, referral, workshops, and fact sheets on art hazards, safer substitutes and practices.

**National Coalition for the Chemically Injured**

(520) 536-4625

Susan Molloy, contact person in Arizona

Ste.C-501 HC-63 Box 7195

Snowflake, AZ 85937

**National Office of NCCI**

2400 Virginia Ave., NW

Washington, DC 10034

**The Labor Institute**

(212) 674-3322

853 Broadway, Room 2014

New York, NY 10012

Produced "Multiple Chemical Sensitivity: An Emerging Occupational Hazard" (28 minute video), and "Multiple Chemical Sensitivity at Work: A Training Workbook for Working People," (95 Pages). Order from APEX Press, Publication Office, P.O. Box 337, Croton-on-Hudson, NY 10952. (914) 271-6500.

**Human Ecology Action League (HEAL) of Southern Arizona**

(520) 577-9673

6655 E. Placita Alhaja

Tucson, AZ 85715-1251

**The Dispossessed Project**

(520) 636-2802

Rhonda Zwillinger

P.O. Box 402

Paulden, AZ 86334-0402

Graphically depicts the plight of people injured by toxic chemical exposure and who live with Multiple Chemical Sensitivities, through a collection of black and white photographs and biographical anecdotes. Provides a forum for people with MCS where they can accurately describe how they have lost their health and dignity.

**Electrical Sensitivity Network**

(520) 778-4637

Lucinda Grant, Director

P.O. Box 86302

Prescott, AZ 86302

Bi-monthly "Electrical Sensitivity News".

**American Academy of Environmental Medicine**

(215) 862-4544

10 E. Randolph St.

New Hope, PA 18938

Professional association of environmental and occupational physicians. Provides regional listings of member doctors.

**American Indian Environmental Illness Foundation**

(360) 665-3913

Terri Hansen, Director

P.O. Box 1039

Long Beach, WA 98631

**Government Agencies**

US Social Security Administration

Check your phone book under US Government Offices, Health and Human Services.

For general information, call 1-800-772-1213.

US Department of Housing and Urban Development (HUD)

Office of Fair Housing and Equal Opportunity

(602) 379-4461.

Request the MCS Information Packet, which includes citations and descriptions of helpful recent legal decisions regarding safe housing.

**Additional Reading****Staying Well in a Toxic World: Understanding Environmental Illness, Multiple Chemical Sensitivities, Chemical Injuries, and Sick Building Syndrome.**

By Lynn Lawson (1993)

\$18.95

P.O. Box 1732

Evanston, IL 60201

**Neurobiology of MCS.**

An interview by Cindy Duehring with Donald Dudley, M.D., Neuroscientist and President of the Washington Institute of Neurosciences in Gig Harbor, Washington.

April 1996 Issue of "Our Toxic Times."

Chemical Injury Information Network

P.O. Box 301

White Sulphur Springs, MT 59645.

\*\*\* Special thanks to Susan Molloy for her assistance in the creation of this fact sheet.

\*\*\* Portions of this fact sheet were reprinted with permission of the Multiple Chemical Sensitivity in the Workplace Task Force, NY Coalition for Alternatives to Pesticides, 353 Hamilton Street, Albany, NY 12210, (518) 426-8246.

For additional information on AzTAP please contact:

Arizona Technology Access Program Institute for Human Development Northern Arizona University

**FLAGSTAFF OFFICE:**

Pamela Alcala, Administrative Assistant  
Box 5630  
Flagstaff, AZ 86011-5630

(520) 523-5879 - Voice  
(520) 523-1695 - TTY  
(520) 523-9127 - Fax  
(800) 553-0714 - Toll Free

E-mail: Pamela.Alcala@nau.edu

**PHOENIX OFFICE:**

Jill Oberstein, Project Director  
2715 N. 3rd. Street, Suite 104  
Phoenix, AZ 85004

(602) 728-9532 - Voice  
(602) 728-9536 - TTY  
(602) 728-9535 - Fax  
(800) 477-9921 - Toll Free

E-mail: Jill.Oberstein@nau.edu



**CERTIFICATE OF SERVICE**

I hereby certify that, on March 18, 2021, I filed the foregoing in the United States Court of Appeals for the District of Columbia Circuit via the CM/ECF system. I further certify that all parties are registered CM/ECF users, and that service will be accomplished via electronic filing.

/s/ W. Scott McCollough

W. Scott McCollough