



3 March 2010

Tooele County Planning Commission

Re: Rocky Mountain Power's Application for Conditional Use Permit 2010-1

Tooele South East Bench Route 345kV high Power Transmission Line

Dear Sirs/Madams:

In the 1980s I served as the Executive Secretary of the New York State Powerlines Project, a state-funded study designed to determine whether there were adverse health effects from living near to powerlines. Our study confirmed earlier reports that children living near to powerlines which generate significant magnetic fields suffered from an elevated risk of leukemia. After the Powerlines Project was finished I became the spokesperson for New York State on the issue of health effects of electromagnetic fields. In the meantime there have been many additional studies demonstrating that exposure to magnetic fields increases risk of leukemia and probably brain tumors both in children and adults, and also increases risk of neurodegenerative disease (Alzheimer's and amyotrophic lateral sclerosis) in adults. Some other adverse health effect may result, but the evidence for other diseases is less well documented.

While all of us depend on electricity for everyday life, the evidence that human (especially child) exposure to magnetic fields from powerline poses a hazard to health is sufficiently strong that it is imperative that every reasonable effort be taken to reduce exposure to the greatest number of persons possible. This must be a critical consideration when routes for a new high voltage powerline are being considered. To the greatest degree possible every effort must be made to route new lines away from areas which impact residences, schools, churches and any other place where children are likely to spend significant amounts of time. This is complicated in some situations, but is not difficult in your community, where it would be easy to route the line outside of areas where a large number of people live, learn and worship. To fail to do so, given the strong evidence of harm to humans, is both irresponsible and invites later litigation.

I am a public health physician, whose responsibility it is to do what I can to reduce human disease from exposure to environmental agents. I am attaching two publications that provide detailed evidence for my recommendations, one of them coming from a presentation to the President's Cancer Panel a year ago. I am also attaching by *curriculum vitae* which gives my credentials. I urge you to disapprove any route for a high voltage power line that is not designed to minimize risk to the population. Thank you for your consideration.

Yours sincerely,

David O. Carpenter, M.D.

Director

Institute for Health and the Environment

University at Albany

RECEIVED

MAR 03 2010

From Toole City Concerned Citizens Gr

CURRICULUM VITAE

SUBMITTAL #: _____

Name: David O. Carpenter

Home Address: 2749 Old State Road
Schenectady, New York 12303

Position Held:
Director, Institute for Health and the Environment
University at Albany
Professor, Environmental Health Sciences
Professor, Biomedical Sciences
School of Public Health, University at Albany
5 University Place, A217, Rensselaer, NY 12144

Education: 1959 B.A., Harvard College, Cambridge, MA
1964 M.D., Harvard Medical School, Boston, MA

Positions Held:

- 9/61-6/62 Research Fellow, Department of Physiology, University of Goteborg, Sweden with Professor Anders Lundberg
- 7/64-6/65 Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
- 7/65-2/73 Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
- 2/73-3/80 Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
- 3/80-9/85 Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 9/85-1/98 Dean, School of Public Health, University at Albany
- 9/85-Pres. Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
- 9/85-7/98 Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 1/98-Pres. Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
- 2001-Pres. Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY
- 2005-Pres. Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New York

Editor-in-Chief: Cellular and Molecular Neurobiology
1981 - 1987

Editorial Advisor: Cellular and Molecular Neurobiology
1987 - Present

Editorial Board: Journal of Public Health Management and Practice
1995 - 2002
International Journal of Occupational Medicine & Environmental Health
1996 - Present
Journal of Alzheimer's Disease - Associate Editor
2007-2009

Reviews in Environmental Health; 2008-present
International Archives of Occupational and Environmental Health; 2009-present.
Journal of Environmental and Public Health, 2009-present.
Environmental Health Perspectives, 2010-present

National and International Committees:

1978, 1981 Physiology Study Section (Ad hoc member)
 1979-1985 NIH International Fellowship Study Section
 1974-1981 Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
 1981-1989 Member, USA National Committee for the International Brain Research Organization
 1985-1986 Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
 1986-1987 Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
 1987-1989 Member, Science Advisory Council of the American Paralysis Association
 1987-1990 Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
 1985-1993 Committee #79, National Council on Radiation Protection and Measurements
 1986-1997 Member, Legislative and Education Committees, Association of Schools of Public Health
 1989-1994 Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
 1994, 1995 Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
 1994-1997 Member, Legislative Committee of the Association of Schools of Public Health
 1997 Member, Executive Committee of the Association of Schools of Public Health
 1997-2000 National Advisory Environmental Health Sciences Council of the National Institutes of Health
 1998-Pres. Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
 2000-Pres. Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-Pres; Chair, 2004-2008
 2001-2008 United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
 2002-2003 Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
 2003-Pres. Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
 2003-Pres. Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.

State and Local Committees:

1980-1987 Executive Secretary, New York State Power Lines Project
 1985-1989 Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
 1986-1989 Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
 1991-1992 Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
 1991-1992 Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
 1991-1992 Member, State Task Force for the Reform of Middle Level Education in NY State
 1992-1993 Member, State Needs Task Force on Health Care and Education
 1987-1998 Delegate-at-Large, New York State Public Health Association
 1991-1995 Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
 1994 Chair, Council of Deans, University at Albany, SUNY
 1997-2008. Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
 2000-Pres. Member, Board of Directors, Healthy Schools Network, Inc.

2000-2003	Member, Medical Advisory Board, Hepatitis C Coalition, New York
2000-2004	Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
2001-2008	Member, Board of Directors, Environmental Advocates of New York
2004-2007	Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
2005-Pres.	Member, Schooling Chefs Curriculum Advisory Board
2005-2008	Member, Board of Directors, Citizens Environmental Coalition
2006-2009	Member, Board of Directors, Marine Environmental Research Institute
2007-2009	Member, New York State Renewable Energy Task Force

Honors, Awards And Fellowships:

1959	B.A. awarded <u>magna cum laude</u> . Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, <u>Ambystoma tigrinum</u> " (Thesis advisor, Professor George Wald) Elected to Phi Beta Kappa and to Sigma Xi
1964	M.D. awarded <u>cum laude</u> for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
1964	Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
1970	Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
1971	Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months
1982, 1986	Visiting Professor of Physiology, Department of Physiology, Kyushu University, Fukuoka, Japan, for a period of three months each
1987	
1989	Awarded Jacob Javits Neuroscience Investigator Award from the National Institute of Neurological and Communicative Diseases and Stroke
1999	Awarded Homer N. Calver Award from the American Public Health Association for studies in environmental health.
2001	Awarded 2001 Academic Laureate from the University at Albany Foundation.

Federal Grants Held: (Principal Investigator Only)

1980-1983	United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs", \$76,847 total direct costs.
1982-1988	National Institute of Health, "Mechanisms of Desensitization at Central Synapses", \$464,786 total direct costs.
1984-1986	Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs", \$330,504 total direct costs.
1986-1996	National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct costs.
1989-1993	National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total direct costs
1990-1995	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O. Carpenter, P.I. \$5,783,419 total direct costs.
1995-2001	Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. A Central/Eastern European Environ/Occup Training Program, D.O. Carpenter, P.I. \$657,520 total costs.

- 1995-2001 National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total direct costs.
- 1998-1999 Environmental Protection Agency, AIndoor Air Risk at Akwesasne - Pilot Project@, D.O. Carpenter, P.I. \$9,996 total costs.
- 2000-2002 Association Liaison Office for University Cooperation in Development, ACooperative Program in Environmental Health between the Institute of Public Health at Makerere University, Kampala, Uganda and the School of Public Health, University at Albany, USA@, D.O. Carpenter, P.I. \$96,432 total costs.
- 2001-2007 Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. AMultidisciplinary Environmental Health Training@, D.O. Carpenter, P.I. \$850,000 total costs.
- 2006-2010 Pakistan-US Science and Technology Cooperative Program (US National Academy of Sciences). "Association of particulate matter with daily morbidity in an urban population," D.O. Carpenter, P.I., \$391,104 total costs.

Research Interests:

Neural basis of human disease especially the dementias and ALS
 Neurotoxicology
 Ionizing and non-ionizing radiation biology
 Public health education
 Human disease caused by environmental pollutants

Other Professional Activities:

Host, The Public Radio Health Show (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001.

Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.
6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibibility of motoneurons of different sizes. J. Neurophysiol., 28:599-620, 1965.
7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. J. Neurophysiol., 28:560-580, 1965.
8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J. Pharmacol., 148:380-385, 1965.
9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. J. Neurophysiol., 28:958-965, 1965.

10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. Arch. Ital. Biol., 104:73-85, 1966.
11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. J. Neurophysiol., 29:353-368, 1966.
12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in Aplysia neurons. J. Gen. Physiol., 50:1469-1484, 1967.
13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv. Pharmacol., 6A:351-364, 1968.
14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na^+ pump to membrane potential in Aplysia neurons. J. Gen. Physiol., 52:1-21, 1968.
15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch. Neurol., 19:591-597, 1968.
16. Carpenter, D.O. Membrane potential produced directly by the Na^+ pump in Aplysia neurons. Comp. Biochem. Physiol., 35:371-385, 1970.
17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of Aplysia neurons upon Na^+ and Ca^{++} . J. Cell. Physiol., 75:121-127, 1970.
18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepinephrine in the presence of tetrodotoxin. J. Pharmacol. Exp. Therap., 73:416-421, 1970.
19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. Science, 169:597-598, 1970.
20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of Aplysia neurons and squid axon as determined by a new technique. Int. J. Neurosci., 2:35-48, 1971.
21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in Aplysia nervous and non-nervous tissues. Int. J. Neurosci., 2:49-56, 1971.
22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of Aplysia neuron somata. Science, 176:1329-1331, 1972.
23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. Science, 179:1336-1338, 1973.
24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. J. Physiol. (Lond.), 229:471-493, 1973.
25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. Aplysia acetylcholine receptors: Blockage by and binding of α -bungarotoxin. Brain Res., 72:225-240, 1974.
26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. Brain Res., 73:156-160, 1974.
27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in Aplysia suggests neurotransmitter function. Science, 185:364-365, 1974.
28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of Aplysia neurons. J. Cell. Physiol., 84:463-472, 1974.
29. Brownstein, M.J., Saavedra, J.M., Axelrod, J., Zeman, G.H. and Carpenter, D.O. Coexistence of several putative neurotransmitters in single identified neurons of Aplysia. Proc. Natl. Acad. Sci. (USA), 71:4662-4665, 1975.
30. Carpenter, D.O. and Gaubatz, G.L. Octopamine receptors on Aplysia neurons mediate hyperpolarization by increasing membrane conductance. Nature, 252:483-485, 1974.
31. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Afferent nerve fiber activity responding to temperature changes of the scrotal skin of the rat. J. Neurobiol., 38:601-612, 1975.

32. Carpenter, D.O. and Gaubatz, G.L. H₁ and H₂ histamine receptors on Aplysia neurons. Nature, 254:343-344, 1975.
33. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Resistivity of axoplasm. II. Internal resistivity of giant axons of squid and Myxicola. J. Gen. Physiol., 66:139-148, 1975.
34. Zeman, G.H. and Carpenter, D.O. Asymmetric distribution of aspartate in ganglia and single neurons of Aplysia. Comp. Biochem. Physiol., 52C:23-26, 1975.
35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents in vitro. Pflugers Arch., 359:349-356, 1975.
36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on Aplysia neurons. Nature, 258:751-754, 1975.
37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on Aplysia neurons. Science, 192:806-809, 1976.
38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. Biophys. J., 16:991-1001, 1976.
39. Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α -bungarotoxin with Aplysia acetylcholine receptors. Mol. Pharmacol., 12:999-1006, 1976.
40. Saavedra, J.M., Ribas, J., Swann, J. and Carpenter, D.O. Phenylethanolamine: A new putative neurotransmitter in Aplysia. Science, 195:1004-1006, 1977.
41. Carpenter, D.O., Swann, J.W. and Yarowsky, P.J. Effect of curare on responses to different putative neurotransmitters in Aplysia neurons. J. Neurobiol., 8:119-132, 1977.
42. Yarowsky, P.J. and Carpenter, D.O. GABA mediated excitatory responses on Aplysia neurons. Life Sci., 20:1441-1448, 1977.
43. Willis, J.A., Myers, P.R. and Carpenter, D.O. An ionophoretic module which controls electroosmosis. J. Electrophysiol. Tech., 6:34-41, 1977.
44. Yarowsky, P.J. and Carpenter, D.O. Receptors for gamma-aminobutyric acid (GABA) on Aplysia neurons. Brain Res., 144:75-94, 1978.
45. Carpenter, D.O., Gaubatz, G., Willis, J.A. and Severance, R. Effects of irradiation of Aplysia pacemaker neurons with 20 MeV electrons. Rad. Res., 76:32-47, 1978.
46. Yarowsky, P.J. and Carpenter, D.O. A comparison of similar ionic responses to gamma-aminobutyric acid and acetylcholine. J. Neurophysiol., 41:531-541, 1978.
47. Blum, B., Aufer, C.R. and Carpenter, D.O. A head holder and stereotaxic device for the rattlesnake. Brain Res. Bull., 3:271-274, 1978.
48. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Dopamine-induced muscle contractions and modulation of neuromuscular transmission in Aplysia. Brain Res., 157:167-172, 1978.
49. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Evidence for identified dopamine motor neurons to the gill of Aplysia. Neurosci. Lett., 10:275-280, 1978.
50. Keabian, P.R., Keabian, J.W. and Carpenter, D.O. Regulation of cyclic AMP in heart and gill of Aplysia by the putative neurotransmitters, dopamine and serotonin. Life Sci., 24:1757-1764, 1979.
51. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Res. Bull., 4:149-152, 1979.
52. Pellmar, T.C. and Carpenter, D.O. Voltage-dependent calcium current induced by serotonin. Nature, 277:483-484, 1979.
53. Ruben, P.C., Swann, J.W. and Carpenter, D.O. Neurotransmitter receptors on gill muscle fibers and the gill peripheral nerve plexus in Aplysia. Canad. J. Physiol. Pharmacol., 57:1088-1097, 1979.

54. Pellmar, T.C. and Carpenter, D.O. Serotonin induces a voltage-sensitive calcium current in neurons of Aplysia californica. J. Neurophysiol., 44:423-439, 1980.
55. Parver, L.M., Auker, C. and Carpenter, D.O. Choroidal blood flow as a heat dissipating mechanism in the macula. Am. J. Ophthalmol., 89:641-646, 1980.
56. Mell, L.D., Jr. and Carpenter, D.O. Fluorometric determination of octopamine in tissue homogenates by high-performance liquid chromatography. Neurochem. Res., 5:1089-1096, 1980.
57. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Thyrotropin-releasing hormone has multiple actions in cortex. Brain Res., 194:244-248, 1980.
58. Meszler, R.M., Auker, C.R. and Carpenter, D.O. Fine structure and organization of the infrared receptor relay, the lateral descending nucleus of the trigeminal nerve in pit vipers. J. Comp. Neurol., 196:571-584, 1981.
59. Auker, C.R., Parver, L.M., Doyle, T. and Carpenter, D.O. Choroidal blood flow: I. Ocular tissue temperature as a measure of flow. Arch. Ophthalmol., 100:1323-1326, 1982.
60. Parver, L.M., Auker, C., Carpenter, D.O. and Doyle, T. Choroidal blood flow: II. Reflexive control in the monkey. Arch. Ophthalmol., 100:1327-1330, 1982.
61. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Lateral olfactory tract transmitter: Glutamate, aspartate or neither? Cell. Mol. Neurobiol., 1:115-120, 1981.
62. Scappaticci, K.A., Dretchen, K.L., Carpenter, D.O. and Pellmar, T.C. Effects of furosemide on neural mechanisms in Aplysia. J. Neurobiol., 12:329-341, 1981.
63. Pellmar, T.C. and Carpenter, D.O. Cyclic AMP induces a voltage-dependent current in neurons of Aplysia californica. Neurosci. Lett., 22:151-157, 1981.
64. Parver, L., Auker, C. and Carpenter, D.O. Stabilization of macular temperature: The stabilizing effect of the choroidal circulation on the temperature environment of the macula. Retina, 2:117-120, 1982.
65. Green, R.W. and Carpenter, D.O. Biphasic responses to acetylcholine in mammalian reticulospinal neurons. Cell. Molec. Neurobiol., 1:401-405, 1981.
66. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Pharmacologic sensitivity of amino acid responses and synaptic activation of in vitro prepyriform neurons. J. Neurophysiol., 48:1289-1301, 1982.
67. Slater, N.T. and Carpenter, D.O. Blockade of acetylcholine-induced inward currents in Aplysia neurons by strychnine and desipramine: effect of membrane potential. Cell. Molec. Neurobiol., 2:53-58, 1982.
68. Swann, J.W., Sinback, C.N., Pierson, M.G. and Carpenter, D.O. Dopamine produces muscle contractions and modulates motoneuron-induced contractions in Aplysia gill. Cell. Molec. Neurobiol., 2:291-308, 1982.
69. Swann, J.W., Sinback, C.N., Keabian, P.R. and Carpenter, D.O. Motoneurons which may utilize dopamine as their neurotransmitter. Cell. Molec. Neurobiol., 2:309-324, 1982.
70. Auker, C.R., Meszler, R.M. and Carpenter, D.O. Apparent discrepancy between single unit activity and ¹⁴C-deoxyglucose labelling in the optic tectum of the rattlesnake. J. Neurophysiol., 49:1504-1516, 1983.
71. Slater, N.T., Carpenter, D.O., Freedman, J.E. and Snyder, S.H. Vipoxin both activates and antagonizes three types of acetylcholine response in Aplysia neurons. Brain Res., 278:266-270, 1983.
72. French-Mullen, J.M.H., Hori, N., Nakanishi, H., Slater, N.T. and Carpenter, D.O. Assymetric distribution of acetylcholine receptors and M channels on prepyriform neurons. Cell. Molec. Neurobiol., 3:163-182, 1983.
73. Carpenter, D.O., Briggs, D.B. and Strominger, N. Responses of neurons of canine area postrema to neurotransmitters and peptides. Cell. Molec. Neurobiol., 3:113-126, 1983.

74. Slater, N.T. and Carpenter, D.O. Blocking kinetics at excitatory acetylcholine responses on *Aplysia* neurons. Biophys. J., 45:24-25, 1984.
75. Chesnut, T.J. and Carpenter, D.O. Two-component desensitization of three types of responses to acetylcholine in *Aplysia*. Neurosci. Lett., 39:285-290, 1983.
76. Haas, H.L., Jeffreys, J.G.R., Slater, N.T. and Carpenter, D.O. Modulation of low calcium induced field bursts in the hippocampus by monoamines and cholinomimetics. Pflugers Arch., 400:28-33, 1984.
77. Parvar, L.M., Auker, C.R. and Carpenter, D.O. Choroidal blood flow. III. Reflexive control in human eyes. Arch. Ophthalmol., 101:1604-1606, 1983.
78. Slater, N.T., Haas, H.L. and Carpenter, D.O. Kinetics of acetylcholine-activated cation channel blockade by the calcium antagonist D-600 in *Aplysia* neurons. Cell. Molec. Neurobiol., 3:329-344, 1983.
79. McCreery, M.J. and Carpenter, D.O. Modulation of neuronal responses to L-glutamate in *Aplysia*. Cell. Molec. Neurobiol., 4:91-95, 1984.
80. Carpenter, D.O., Briggs, D.B. and Strominger, N. Peptide-induced emesis in dogs. Behav. Brain Res., 11:277-281, 1984.
81. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. N-methyl-D-aspartate and L-aspartate activate distinct receptors in prepyriform cortex. Cell. Molec. Neurobiol., 4:185-189, 1984.
82. Slater, N.T. and Carpenter, D.O. A study of the cholinergic actions of strychnine using the technique of concentration jump relaxation analysis. Cell Molec Neurobiol 4:263-271, 1984.
83. Slater, N.T., Hall, A.F. and Carpenter, D.O. Kinetic properties of cholinergic desensitization in *Aplysia* neurons. Proc. Roy. Soc. Lond. B, 223:63-78, 1984.
84. Akaike, N., Hattori, K., Oomura, Y. and Carpenter, D.O. Bicuculline and picrotoxin block gamma-aminobutyric acid-gated Cl⁻ conductance by different mechanisms. Experientia, 41:70-71, 1985.
85. Slater, N.T., Carpenter, D.O., Freedman, J.E. and Synder, S.H. Dual effects of the snake venom polypeptide vipoxin on receptors for acetylcholine and biogenic amines in *Aplysia* neurons. Neurosci., 14:723-733, 1985.
86. Mizuno, Y., Oomura, Y., Hori, N. and Carpenter, D.O. Action of vasopressin on CA1 pyramidal neurons in rat hippocampal slices. Brain Res., 309:241-246, 1984.
87. Slater, N.T., Hall, A.F. and Carpenter, D.O. Trifluoperazine and calcium antagonists accelerate cholinergic desensitization in *Aplysia* neurons. Brain Res., 329:275-279, 1985.
88. French-Mullen, J.M.H., Koller, K., Zaczek, R., Coyle, J.T., Hori, N. and Carpenter, D.O. N-acetylaspartylglutamate: Possible role as the neurotransmitter of the lateral olfactory tract. Proc. Nat. Acad. Sci., 82:3897-3900, 1985.
89. Greene, R.W. and Carpenter, D.O. Actions of neurotransmitters on pontine medial reticular formation neurons of the cat. J. Neurophysiol., 54:520-531, 1985.
90. Hori, N., French-Mullen, J.M.H. and Carpenter, D.O. Kainic acid responses and toxicity show pronounced Ca²⁺ dependence. Brain Res., 358:380-384, 1985.
91. Gaillard, W.D. and Carpenter, D.O. Spectra of neurotransmitter receptors and ionic responses on cerebral A and B neurons in *Aplysia californica*. Brain Res., 373:303-310, 1986.
92. Gaillard, W.D. and Carpenter, D.O. On the transmitter at the A-to-B cell in *Aplysia californica*. Brain Res., 373:311-315, 1986.
93. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. A comparison on the effects of quinolinate and N-methyl-aspartate on neurons in rat piriform cortex. Neurosci. Lett., 63:66-70, 1986.
94. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. Receptors for the excitatory amino acids on neurons in rat pyriform cortex. J. Neurophysiol., 55:1283-1294, 1986.

95. Slater, N.T., David, J.A. and Carpenter, D.O. Relaxation studies on the interaction of hexamethonium with acetylcholine-receptor channels in *Aplysia* neurons. Cell. Molec. Neurobiol., 6:191-211, 1986.
96. Leung, M.K., S.-Rozsa, K., Hall, A., Kuruvilla, S., Stefano, G.B. and Carpenter, D.O. Enkephalin-like substance in *Aplysia* nervous tissue and actions of leu-enkephalin on single neurons. Life Sci., 38:1529-34, 1986.
97. Slater, N.T., Filbert, M. and Carpenter, D.O. Multiple interactions of anticholinesterases with *Aplysia* acetylcholine responses. Brain Res., 375:407-412, 1986.
98. Carpenter, D.O. and Briggs, D.B. Insulin excites neurons of the area postrema and causes emesis. Neurosci. Lett., 68:85-89, 1986.
99. Carpenter, D.O., Briggs, D.B., Knox, A.P. and Strominger, N.L. Radiation-induced emesis in the dog: Effects of lesions and drugs. Rad. Res., 108:307-316, 1986.
100. Briggs, D.B. and Carpenter, D.O. Excitation of neurons in the canine area postrema by prostaglandins. Cell. Molec. Neurobiol., 6:421-426, 1986.
101. Chesnut, T.J., Carpenter, D.O. and Strichartz, G.R. Three effects of venom from *conus striatus* on the delayed rectifier potassium current of molluscan neurons. Toxicon, 25:267-278, 1987.
102. Yakushiji, T., Tokutomi, N., Akaike, N. and Carpenter, D.O. Agonists of GABA responses, studied using internally perfused frog dorsal root ganglion neurons. Neuroscience 22:1123-1133, 1987.
103. Akaike, N., Yakushiji, T., Tokutomi, N. and Carpenter, D.C. Multiple mechanisms of antagonism of GABA responses. Cell. Molec. Neurobiol., 7:97-103, 1987.
104. Hori, N., Galeno, T. and Carpenter, D.O. Responses of pyriform cortex neurons to excitatory amino acids: Voltage dependence, conductance changes and effects of divalent cations. Cell. Molec. Neurobiol., 7:73-90, 1987.
105. Oyama, Y., King, W.M. and Carpenter, D.O. Edrophonium-induced membrane current in single neurons physically isolated from *Aplysia californica*. Brain Res., 438:95-100, 1988.
106. Jahan-Parwar, B., S.-Rozsa, K., Salanki, J., Evans, M.L. and Carpenter, D.O. *In vivo* labeling of serotonin containing neurons by 5,7-dihydroxytryptamine in *Aplysia*. Brain Res., 426:173-178, 1987.
107. King, W.M. and Carpenter, D.O. Distinct GABA and glutamate receptors may share a common channel in *Aplysia* neurons. Neurosci. Lett., 82:343-348, 1987.
108. Carpenter, D.O., Briggs, D.B., Knox, A.P. and Strominger, N. Excitation of area postrema neurons by transmitters, peptides and cyclic nucleotides. J. Neurophysiol., 59:358-369, 1988.
109. Carpenter, D.O., Hall, A.F. and Rahmann, H. Exogenous gangliosides induce direct voltage and conductance changes on isolated neurons. Cell. Molec. Neurobiol., 8:245-250, 1988.
110. Hori, N., Carpenter, D.O. and Katsuda, N. Effect of acetylcholine on the pyramidal cell in the rat piriform cortex *in vitro*. Neurosciences, 13:172-174, 1987 (in Japanese).
111. Hori, N. and Carpenter, D.O. Excitatory amino acid receptors in piriform cortex do not show receptor desensitization. Brain Res., 457:350-354, 1988.
112. Allen, C.N., Brady, R., Swann, J., Hori, N. and Carpenter, D.O. N-methyl-D-aspartate (NMDA) receptors are inactivated by trypsin. Brain Res., 458:147-150, 1988.
113. Oyama, Y., Akaike, N. and Carpenter, D.O. Strychnine decreases the voltage-dependent Ca^{2+} current of both *Aplysia* and frog ganglion neurons. Cell. Molec. Neurobiol., 8:307-314, 1988.
114. Oyama, Y., King, W.M., Allen, C.N., Hori, N. and Carpenter, D.O. Characterization of an inward current elicited by edrophonium in physically isolated and internally perfused *Aplysia* neurons. Brain Res., 463:124-132, 1988.
115. Hori, N., Akaike, N. and Carpenter, D.O. Piriform cortex brain slices: Techniques for isolation of synaptic inputs. J. Neurosci. Methods, 25:197-208, 1988.

116. Oyama, Y., Evans, M.L., Akaike, N. and Carpenter, D.O. Electrophysiological detection of acetylcholinesterase activity using concentration clamp on physically isolated *Aplysia* neurons. Neuroscience Res., 6:174-180, 1988.
117. Tsuda, Y., Oyama, Y., Carpenter, D.O. and Akaike, N. Effects of Ca^{2+} on the transient outward current of single isolated *Helix* central neurones. Brit J. Pharmacol., 95:526-530, 1988.
118. Oyama, Y., Hori, N., Evans, M.L., Allen, C.N. and Carpenter, D.O. Electrophysiological estimation of the actions of acetylcholinesterase inhibitors on acetylcholine receptor and cholinesterase in physically isolated *Aplysia* neurones. Brit. J. Pharmacol., 96:573-582, 1989.
119. King, W.M. and Carpenter, D.O. Voltage-clamp characterization of Cl^- conductance gated by GABA and L-glutamate in single neurons of *Aplysia*. J. Neurophysiol., 61:892-899, 1989.
120. Evans, M.L. and Carpenter, D.O. Desensitization kinetics of a chloride acetylcholine response in *Aplysia*. Brain Res., 495:309-318, 1989.
121. Salanki, J., Evans, M.L. and Carpenter, D.O. Desensitization kinetics of a K^+ acetylcholine response in *Aplysia*. Brain Res., 495:298-308, 1989.
122. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Effects of exogenous ganglioside and cholesterol application on excitability of *Aplysia* neurons. Membrane Biochemistry, 8:19-26, 1989.
123. Carpenter, D. Neural mechanisms of emesis. Canad. J. Physiol. Pharmacol., 68:230-236, 1990.
124. Oyama, Y., Hori, N., Allen, C.N., and Carpenter, D.O. Influences of trypsin and collagenase on acetylcholine responses of physically-isolated single neurons of *Aplysia californica*. Cell. Molec. Neurobiol., 10:193-205, 1990.
125. Büsselberg, D., Evans, M.L., Rahmann, H., and Carpenter, D.O. Lead inhibits the voltage-activated calcium current of *Aplysia* neurons. Toxicol. Lett., 51:51-57, 1990.
126. Doi, N., Carpenter, D.O. and Hori, N. Differential effects of baclofen and GABA on rat piriform cortex pyramidal neurons *in vitro*. Cell. Molec. Neurobiol., 10: 559-564, 1991.
127. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Zn^{2+} blocks the voltage activated calcium current of *Aplysia* neurons. Neurosci. Letts., 117:117-122, 1990.
128. Büsselberg, D., Carpenter, D.O., Sugita, M., Araki, S., Satake, M. and Rahmann, H. Effects of exogenous lipid application on excitability of *Aplysia* neurons. Biomed. Res., 11:77-86, 1990.
129. Evans, M.L., Kadan, M.J., Hartig, P.R. and Carpenter, D.O. Correlation of ^{125}I -LSD autoradiographic labelling with serotonin voltage clamp responses in *Aplysia* neurones. Synapse, 8:22-29, 1991.
130. S.-Rozsa, K., Stefano, G., Salanki, J. and Carpenter, D.O. Characterization of responses to enkephalins and FMRFamide on B neurons of the cerebral ganglion of *Aplysia*. Comp. Biochem. Physiol., 99C:403-412, 1991.
131. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Lead and zinc block a voltage activated calcium channel of *Aplysia* neurons. J. Neurophysiol., 65:786-795, 1991.
132. Hori, N., Doi, N., Miyahara, S., Shinoda, Y. and Carpenter, D.O. Appearance of NMDA receptors triggered by anoxia independent of voltage *in vivo* and *in vitro*. Exp. Neurol., 112:304-311, 1991.
133. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Effects of inorganic and triethyl lead and inorganic mercury on the voltage activated calcium channel of *Aplysia* neurons. NeuroToxicology, 12:733-744, 1991.
134. Evans, M.L., Büsselberg, D. and Carpenter, D.O. Pb^{2+} blocks calcium currents of cultured dorsal root ganglion cells. Neurosci. Letts., 129:103-106, 1991.
135. Kemenes, G., S.-Rozsa, K., Stefano, G. and Carpenter, D.O. Distinct receptors for leu- and met-enkephalin on the metacerebral giant cell of *Aplysia*. Cell. Molec. Neurobiol., 12:107-119, 1992.

136. Ayrapetyan, S.N. and Carpenter, D.O. Very low concentrations of acetylcholine and GABA modulate transmitter responses. NeuroReport 2:563-565, 1991.
137. Carpenter, D.O. and Hori, N. Neurotransmitter and peptide receptors on medial vestibular nucleus neurons. Ann. NY Acad. Sci., 656:668-686, 1992.
138. Hernadi, L., S.-Rózsa, K., Jahan-Parwar, B. and Carpenter, D.O. A topography and ultrastructural characterization of *in vivo* 5,7-dihydroxytryptamine-labelled serotonin-containing neurons in the central nervous system of *Aplysia californica*. Cell. Molec. Neurobiol., 12:317-326, 1992.
139. Carpenter, D.O., Fejtl, M., Ayrapetyan, S., Szarowski, D. and Turner, J.N. Dynamic changes in neuronal volume resulting from osmotic and sodium transport manipulations. Acta Biologica Hungarica, 43:39-48, 1992.
140. Ayrapetyan, S.N. and Carpenter, D.O. On the modulating effect of ultralow transmitter concentrations on the functional activity of the neuron membrane. J. Evol. Biochem. Physiol., 27:110-116, 1991.
141. Büsselberg, D., Michael, D., Evans, M.L., Carpenter, D.O. and Haas, H.L. Zinc (Zn^{2+}) blocks voltage gated calcium channels in cultured rat dorsal root ganglion cells. Brain Res., 593:77-81, 1992.
142. Matthews, M.R., Parsons, P.J. and Carpenter, D.O. Solubility of lead as lead (II) chloride in HEPES-Ringer and artificial seawater (Ca-ASW) solutions. NeuroToxicology, 14:283-290, 1993.
143. Hori, N., Büsselberg, D., Matthews, R., Parsons, P.J. and Carpenter, D.O. Lead blocks LTP by an action not at NMDA receptors. Exp. Neurol., 119: 192-197, 1993.
144. Büsselberg, D., Evans, M.L., Haas, H.L. and Carpenter, D.O. Blockade of mammalian and invertebrate calcium channels by lead. NeuroToxicology, 14:249-258, 1993.
145. Riepe, M., Hori, N., Ludolph, A.C., Carpenter, D.O., Spencer, P.S. and Allen, C.N. Inhibition of energy metabolism by 3-nitropropionic acid activates ATP-sensitive potassium channels. Brain Res., 586:61-66, 1992.
146. Hori, N., Hirotsu, I., Davis, P.J. and Carpenter, D.O. Long-term potentiation is lost in aged rats but preserved by calorie restriction. NeuroReport, 3:1085-1088, 1992.
147. Knox, A.P., Strominger, N.L., Battles, A.H. and Carpenter, D.O. Behavioral studies of emetic sensitivity in the ferret. Brain Res. Bull., 31:477-484, 1993.
148. Allen, C.N., Spencer, P.S. and Carpenter, D.O. β -N-methylamino-L-alanine in the presence of bicarbonate is an agonist at non-N-methyl-D-aspartate-type receptors. Neuroscience 54:567-574, 1993.
149. Elekes, K., Stefano, G.B. and Carpenter, D.O. Enkephalin-like immunoreactive neurons in the central nervous system of gastropods (*Helix pomatia*, *Lymnaea stagnalis*, *Aplysia californica*): A comparative immunocytochemical study. Cell Tiss. Res. 272:329-41, 1993.
150. Büsselberg, D., Platt, B., Haas, H.L. and Carpenter, D.O. Voltage gated calcium channel currents of rat dorsal root ganglion (DRG) cells are blocked by Al^{3+} . Brain Res. 622:163-168, 1993.
151. Strominger, N.L., Knox, A.P. and Carpenter, D.O. The connectivity of the area postrema in the ferret. Brain Res. Bull., 33:33-47, 1994.
152. Knox, A.P., Strominger, N.L., Battles, A.H. and Carpenter, D.O. The central connections of the vagus nerve in the ferret. Brain Res. Bull., 33:49-63, 1994.
153. Lin, Y. and Carpenter, D.O. Medial vestibular neurons are endogenous pacemakers whose discharge is modulated by neurotransmitters. Cell. Molec. Neurobiol., 13:601-613, 1993.
154. Kemenes, G., S.-Rózsa, K. and Carpenter, D.O. Cyclic-AMP-mediated excitatory responses to leucine enkephalin in *Aplysia* neurones. J. Exp. Biol. 181: 321-328, 1993.
155. Büsselberg, D., Platt, B., Michael, D., Carpenter, D.O. and Haas, H.L. Mammalian voltage-activated calcium channel currents are blocked by Pb^{2+} , Zn^{2+} and Al^{3+} . J. Neurophysiol., 71:1491-1497, 1994.

156. Hori, N. and Carpenter, D.O. Transient ischemia causes a reduction of Mg^{2+} blockade of NMDA receptors. Neurosci. Letts., 173:75-78, 1994.
157. Riepe, M.W., Hori, N., Ludolph, A.C. and Carpenter, D.O. Failure of neuronal ion exchange, not potentiated excitation, causes excitotoxicity after inhibition of oxidative phosphorylation. Neuroscience, 64:91-97, 1995.
158. Hori, N. and Carpenter, D.O. Functional and morphological changes induced by transient *in vivo* ischemia. Exp. Neurol., 129:279-289, 1994.
159. Lin, Y. and Carpenter, D.O. Direct excitatory opiate effects mediated by non-synaptic actions on rat medial vestibular neurons. Eur. J. Pharmacol., 262:99-106, 1994.
160. Carpenter, D.O. Epidemiological evidence for an association between exposure to 50 and 60 Hz magnetic fields and cancer. James Bay Publication Series, Hydro-Electric Development: Environmental Impacts - Paper No. 6, pp. 2-31, 1994.
161. Carpenter, D.O. Communicating with the public on issues of science and public health. Environ. Health Perspect. 103:127-130, 1995.
162. Fejtli, M., Gyori, J. and Carpenter, D.O. Hg^{2+} increases the open probability of carbachol-activated Cl^- channels in *Aplysia* neurons. NeuroReport, 5:2317-2320, 1994.
163. Carpenter, D.O. The public health significance of metal neurotoxicity. Cell. Molec. Neurobiol., 14:591-597, 1994.
164. Gyori, J., Fejtli, M. and Carpenter, D.O. Effect of $HgCl_2$ on acetylcholine, carbachol and glutamate currents of *Aplysia* neurons. Cell. Molec. Neurobiol., 14:653-664, 1994.
165. Fejtli, M., Gyori, J. and Carpenter, D.O. Mercuric (II) chloride modulates single channel properties of carbachol activated Cl^- channels in cultured neurons of *Aplysia californica*. Cell. Molec. Neurobiol., 14:665-674, 1994.
166. Carpenter, D.O., Matthews, M.R., Parsons, P.J. and Hori, N. Long-term potentiation in piriform cortex is blocked by lead. Cell. Molec. Neurobiol., 14:723-733, 1994.
167. Salanki, J., Gyori, J. and Carpenter, D.O. Action of lead on glutamate-activated chloride currents in *Helix Pomatia L.* neurons. Cell. Molec. Neurobiol., 14:755-768, 1994.
168. Carpenter, D.O. How hazardous wastes affect human health. Cent. Eur. J. Publ. Hlth. 2:6-9, 1994.
169. Oyama, Y., Carpenter, D.O., Ueno, S., Hayashi, H. and Tomiyoshi, F. Methylmercury induces Ca^{2+} -dependent hyperpolarization of mouse thymocytes: A flow-cytometric study using fluorescent dyes. Eur. J. Pharmacol., 293:101-107, 1995.
170. Fejtli, M., Szarowski, D.H., Decker, D., Buttle, K., Carpenter, D.O. and Turner, J.N. Three-dimensional imaging and electrophysiology of live *Aplysia* neurons during volume perturbation: confocal light and high-voltage electron microscopy. JMSA 1(2):75-85, 1995.
171. Carpenter, D.O., Kemenes, G., Elekes, K., Leung, M., Stefano, G., S.-Rozsa, K. and Salanki, J. Opioid peptides in the nervous system of *Aplysia*: A combined biochemical immunocytochemical, and electrophysiological study. Cell. Molec. Neurobiol. 15:239-256, 1995.
172. Riepe, M. and Carpenter, D.O. Delayed increase of cell volume of single pyramidal cells in live hippocampal slices upon kainate application. Neurosci. Letts. 191:35-38, 1995.
173. Son, H. And Carpenter, D.O. Protein kinase C activation is necessary but not sufficient for induction of LTP at the synapse of mossy fiber-CA3 in the rat hippocampus. Neuroscience 72:1-13, 1996.
174. Iwase, T., Hori, N., Morioka, T. and Carpenter, D.O. Low power laser irradiation reduces ischemic damage in hippocampal slices *in vitro*. Lasers Surg. Med., 19:465-450, 1996.
175. Carpenter, D.O., King, W.M. and McCreery, M.J. The role of glutamate reuptake in regulation of glutamate responses in *Aplysia* neurons. Acta Biologica Hungaria 46:363-373, 1995.

176. Saghian, A.A., Ayrapetyan, S.N. and Carpenter, D.O. Low concentrations of ouabain stimulate Na/Ca exchange in neurons. Cell. Molec. Neurobiol., 16:489-498, 1996.
177. Platt, B., Carpenter, D.O., Büsselberg, D., Reymann, K.G. and Riedel, G. Aluminum impairs hippocampal long-term potentiation in rats *in vitro* and *in vivo*. Exp. Neurol., 134:73-86, 1995.
178. Rubakhin, S.S., Gyori, J., Carpenter, D.O. and Salanki, J. HgCl₂ potentiates GABA activated currents in *Lymnaea stagnalis* L. neurons. Acta Biologica Hungaria, 46:431-444, 1995.
179. Fejtli, M. and Carpenter, D.O. Neurite outgrowth is enhanced by conditioning factor(s) released from central ganglia of *Aplysia californica*. Neurosci. Letts., 199:33-36, 1995.
180. Riepe, M.W., Niemi, W.N., Megow, D., Ludolph, A.C. and Carpenter, D.O. Mitochondrial oxidation in rat hippocampus can be preconditioned by selective chemical inhibition of SDH. Exp. Neurol., 138:15-21, 1996.
181. Son, H. and Carpenter, D.O. Interactions among paired-pulse facilitation and post-tetanic and long-term potentiation in the mossy fiber-CA3 pathway in rat hippocampus. Synapse, 23:302-311, 1996.
182. Carpenter, D.O., Suk, W.A., Blaha, K. and Cikrt, M. Hazardous wastes in Eastern and Central Europe. Environ. Health Perspect., 104:244-248, 1996.
183. Son, H., Davis, P.J. and Carpenter, D.O. Time course and involvement of protein kinase C-mediated phosphorylation of F1/GAP-43 in area CA3 after the mossy fiber stimulation. Cell. Molec. Neurobiol., 17:171-194, 1997.
184. Dyatlov, V.A., Platoshin, A.V., Lawrence, D.A. and Carpenter, D.O. Mercury (Hg²⁺) enhances the depressant effect of kainate on Ca-inactivated potassium current in telencephalic cells derived from chick embryos. Toxicol. Appl. Pharmacol., 138:285-297, 1996.
185. Carpenter, D.O. and Conway, J.B. Optimizing professional education in public health. J. Public Health Management Practice, 2:66-72, 1996.
186. Carpenter, D.O. Great Lakes contaminants: A shift in human health outcomes. Health and Environment Digest, 10:17-19, 1996.
187. Boldyrev, A.A., Stvolinsky, S.L., Tyulina, O.V., Koshelev, V.B., Hori, N. and Carpenter, D.O. Biochemical and physiological evidence that carnosine is an endogenous neuroprotector against free radicals. Cell. Molec. Neurobiol., 17:259-271, 1997.
188. Szücs, A., Angiello, C., Salánki, J. and Carpenter, D.O. Effects of inorganic mercury and methylmercury on the ionic currents of cultured rat hippocampal neurons. Cell. Molec. Neurobiol., 17:273-288, 1997.
189. Niemi, W.D., Slivinski, K., Audi, J., Rej, R. and Carpenter, D.O. Propylthiouracil treatment reduces long-term potentiation in area CA1 of neonatal rat hippocampus. Neurosci. Letts., 210:127-129, 1996.
190. Son, H., Madelian, V. and Carpenter, D.O. The translocation and involvement of protein kinase C in mossy fiber-CA3 long-term potentiation in hippocampus of the rat brain. Brain Res., 739:282-292, 1997.
191. Oyama, Y., Carpenter, D.O., Chikahisa, L. and Okazaki, E. Flow-cytometric estimation on glutamate- and kainate-induced increases in intracellular Ca²⁺ of brain neurons. Brain Research, 728:121-124, 1996.
192. Carpenter, D.O., Stoner, C.R.T. and Lawrence, D.A. Flow cytometric measurements of neuronal death triggered by PCBs. NeuroToxicology, 18:507-514, 1997.
193. Azatian, K.V., Ayrapetyan, S.N. and Carpenter, D.O. Metabotropic GABA receptors regulate acetylcholine responses on snail neurons. Gen. Pharmacol., 29:67-72, 1997.
194. Carpenter, D.O., Stoner, C.T., Lawrence, D.A., Niemi, W.D., Shain, W. and Seegal, R. Multiple mechanisms of PCB neurotoxicity. Proceedings of the 1996 Pacific Basin Conference on Hazardous Waste, Kuala Lumpur, Malaysia, CONF-9611157, pp. 404-918.

195. Carpenter, D.O. New Dimensions in our understanding of the human health effects of environmental pollutants. Proceedings of the 1996 Pacific Basin Conference on Hazardous Waste, Kuala Lumpur, Malaysia, CONF-9611157, pp. 37-53.
196. Carpenter, D.O. Possible effects of electromagnetic fields on the nervous system and development. Men. Retard. Dev. Dis. Res. Rev. 3:270-274, 1997.
197. Chiarenzelli, J., Scrudato, R., Bush, B., Carpenter, D. and Bushart, S. Do large-scale remedial and dredging events have the potential to release significant amounts of semi-volatile compounds to the atmosphere? Environ. Hlth. Perspect., 106:47-49, 1998.
198. Dyatlov, V.A., Dyatlova O.M., Parsons, P.H., Lawrence, D.A. and Carpenter, D.O. Lipopolysaccharide and interleukin-6 enhance lead entry into cerebellar neurons: Application of a new and sensitive flow cytometric technique to measure intracellular lead and calcium concentrations. NeuroToxicology, 19:293-302, 1998.
199. Dyatlov, V.A., Platoshin, A.V., Lawrence, D.A. and Carpenter, D.O. Lead potentiates cytokine- and glutamate-mediated increases in permeability of the blood-brain barrier. NeuroToxicology, 19:283-292, 1998.
200. Niemi, W.D., Audi, J., Bush, B. and Carpenter, D.O. PCBs reduce long-term potentiation in the CA1 region of rat hippocampus. Exper. Neurol., 151:26-34, 1998.
201. Carpenter, D.O. Health effects of metals. Cent. Eur. J. Publ. Hlth., 6:160-163, 1998.
202. Carpenter, D.O., Bláha, K., Buekens, A., Cikrt, M., Damstra, T., Dellinger, B., Sarofim, A., Suk, W.A., Wykes, H. and Zejda, J. Remediation of hazardous wastes in Central and Eastern Europe: Technology and health effects. Cent. Eur. J. Publ. Hlth., 6:77-78, 1998.
203. Carpenter, D.O. Human health effects of environmental pollutants: New Insights. Environ. Monitor. Assess. J., 53:245-258, 1998.
204. Dyatlov, V.A., Makovetskaia, V.V., Leonhardt, R., Lawrence, D.A. and Carpenter, D.O. Vitamin E enhances Ca^{2+} -mediated vulnerability of immature cerebellar granule cells to ischemia. Free Rad. Biol. Med., 25: 793-802, 1998.
205. Fitzgerald, E.F., Schell, L.M., Marshall, E.G., Carpenter, D.O., Suk, W.A. and Zejda, J.E. Environmental pollution and child health in Central and Eastern Europe. Environ. Health Persp., 106:307-311, 1998.
206. Carpenter, D.O., Arcaro, K.F., Bush, B., Niemi, W.D., Pang, S. and Vakharia, D.D. Human health and chemical mixtures: An overview. Environ. Health Perspect., 106: 1263-1270, 1998.
207. Carpenter, D.O., Cikrt, M. and Suk, W.A. Hazardous wastes in Eastern and Central Europe: Technology and health effects. Environ. Health Perspect., 107: 3-4, 1999.
208. Carpenter, D.O. Polychlorinated biphenyls and human health. Int. J. Occup. Med. Environ. Hlth. 11: 291-303, 1998.
209. Boldyrev, A.A., Johnson, P., Yanzhang, W., Tan, Y. and Carpenter, D.O. Carnosine and taurine protect rat cerebellar granular cells from free radical damage. Neurosci. Letts., 263: 169-172, 1999.
210. Boldyrev, A.A., Carpenter, D.O., Huentelman, M.J., Peters, C.M. and Johnson, P. Sources of reactive oxygen species production in excitotoxin-stimulated neurons. Biophys. Biochem. Res. Commun., 256: 320-324, 1999.
211. Ayrapetyan, S.N., Ayrapetyan, G. and Carpenter, D.O. The electrogenic sodium pump activity in *Aplysia* neurons is not potential dependent. Acta Biologica Hungarica, 50: 27-34, 1999.
212. Boldyrev, A., Song, R., Lawrence, D. and Carpenter, D.O. Carnosine protects against excitotoxic cell death independently of effects on reactive oxygen species. Neuroscience, 94: 571-577, 1999.
213. Boldyrev, A., Song, R., Dyatlov, V.A., Lawrence, D.A. and Carpenter, D.O. Neuronal cell death and reactive oxygen species. Cell. Molec. Neurobiol., 20:433-450, 2000.
214. Gyori, J., Platoshyn, O., Carpenter, D.O. and Salanki, J. Effect of inorganic- and organic tin compounds on ACh- and voltage-activated Na currents. Cell. Molec. Neurobiol. 20:591-604, 2000.

215. Hussain, R.J., Gyori, J., DeCaprio, A.P. and Carpenter, D.O. *In vivo* and *in vitro* exposure to PCB 153 reduces long-term potentiation. Environ. Hlth. Perspect., 108 :827-831, 2000.
216. Negoita, S., Swamp, L., Kelley, B. and Carpenter, D.O. Chronic diseases surveillance of St. Regis Mohawk health service patients. J. Public Health Management Practice, 7:84-91, 2001.
217. Hussain, R.J., Parsons, P.J., Carpenter, D.O. Effects of lead on long-term potentiation in hippocampal CA3 vary with age. Dev. Brain Res., 121: 243-252, 2000.
218. Tanji, M., Katz, B.H., Spink, B.C. and Carpenter, D.O. Growth inhibition of MCF-7 cells by estrogen is dependent upon a serum factor. Anticancer Res., 20: 2779-2784, 2000.
219. Tanji, M. and Carpenter, D.O. A steroid-binding protein mediates estrogen-dependent inhibition of growth of MCF-7 breast cancer cells. Anticancer Res., 20:2785-2790, 2000.
220. Gyori, J., Hussain, R., Carpenter, D.O. Long-term potentiation in CA1 region of rat brain slices is blocked by PCB 153. Cent. Europ. J. Publ. Hlth., 8: 21-22, 2000.
221. Carpenter, D.O. Human health effects of polychlorinated biphenyls. Cent. Eur. J. Public Health, 8: 23-24, 2000.
- 221a. Sukdolova, V., Negoita, S., Hubicki, L., DeCaprio, A., and Carpenter, D.O. The assessment of risk to acquired hypothyroidism from exposure to PCBs: a study among Akwesasne Mohawk women. Cent. Eur. J. Public Health, 8: 167-168, 2000.
222. Carpenter, D.O., Chew, F.T., Damstra, T., Lam, L.H., Landrigan, P.J., Makalinao, I., Peralta, G.L. and Suk, W.A. Environmental threats to the health of children: The Asian perspective. Environ. Hlth. Perspect., 108: 989-992, 2000.
223. Boldyrev, A.A., Carpenter, D.O. and Johnson, P. Natural mechanisms of protection of neurons against oxidative stress. Recent Res. Devel. Comparative Biochem. & Physiol. 1: 91-103, 2000.
224. Strominger, N.L., Hori, N., Carpenter, D.O., Tan, Y. and Folger W.H. Effects of acetylcholine and GABA on neurons in the area postrema of *Suncus murinus* brainstem slices. Neurosci. Letts. 309: 77-80, 2001.
225. Strominger, N.L., Brady, R., Gullikson, G. and Carpenter, D.O. Imiquimod-elicited emesis is mediated by the area postrema, but not by direct neuronal activation. Brain Res. Bull. 55: 445-451, 2001.
226. Hori, N., Tan, Y., Strominger, N.L. and Carpenter, D.O. Intracellular activity of rat spinal cord motoneurons in slices. J. Neurosci. Meth. 112: 185-191, 2001.
227. Sukocheva, O.A., Abramov, A.Y., Levitskaya, J.O., Gagelgans, A.I. and Carpenter, D.O. Modulation of intracellular Ca concentration by vitamin B12 in rat thymocytes. Blood Cells. Mol. Dis. 27: 812-824, 2001.
228. Gilbertson, M., Carpenter, D. and Upshur, R. Methodology for assessing community health in Areas of Concern: Measuring the adverse effects on human health. Environ. Health Perspect. 109 (Suppl 6): 811-812, 2001.
229. Carpenter, D.O., Shen, Y., Nguyen, T., Le, L. and Lininger, L.L. Incidence of endocrine disease among residents of New York Areas of Concern. Environ. Health Perspect. 109: (Suppl 6) 845-851, 2001.
230. Suk, W.A., Carpenter, D.O., Cirk, M. and Smerhovsky, Z. Metals in Eastern and Central Europe: Health effects, sources of contamination and methods of remediation. Internat. J. Occup. Med. Environ. Health 14, 151-156, 2001.
231. Carpenter, D.O. Effects of metals on the nervous system of humans and animals. Internat. J. Occup. Med. Environ. Health 14: 209-218, 2001.
232. Carpenter, D.O., Arcaro, K. and Spink, D.C. Understanding the human health effects of chemical mixtures. Environ. Health Perspect. 110 (Suppl 1), 25-42, 2002.

233. Carpenter, D.O., Nguyen, T., Le, L., Kudryakov, R. and Lininger, L. Human disease in relation to residence near hazardous waste sites. Proceedings of The 10th Pacific Basin Conference on Hazardous Waste, Okayama, Japan, December 5-7, 2001.
234. Carpenter, D.O., Tarbell, A., Fitzgerald, E., Kadlec, M.J., O'Hehir, D.O. and Bush, B. University-community partnership for the study of environmental contamination at Akwesasne. In: Biomarkers of Environmentally Associated Disease, S.H. Wilson and W.A. Suk, editors, CRC Press/Lewis Publishers, 507-523, 2002.
235. Carpenter, D.O., Hussain, R.J., Berger, D.F., Lombardo, J.P., Park, H-Y. Electrophysiological and behavioral effects of perinatal and acute exposure of rats to lead and polychlorinated biphenyls. Environ. Health Perspect., 110: 377-386, 2002.
236. Hori, N., Tan, Y. King, M., Strominger, N.L. and Carpenter, D.O. Differential actions and excitotoxicity of glutamate agonists on motoneurons in adult mouse cervical spinal cord slices. Brain Res., 958: 434-438, 2002.
237. Laemle, L.K., Hori, N., Strominger, N.L., Tan, Y. and Carpenter, D.O. Physiological and anatomical properties of the suprachiasmatic nucleus of an anophthalmic mouse. Brain Res., 953: 73-81, 2002.
238. Hori, N., Tan, Y., Strominger, N.L. and Carpenter, D.O. Rat motoneuron cell death in development correlates with loss of N-methyl-D-aspartate receptors. Neurosci. Letts., 330:131-134, 2002.
239. Carpenter, D.O., Morris, D.L. and Legator, M. Initial attempts to profile health effects with types of exposure in Anniston, Alabama. FEB, 12: 191-195, 2003.
240. Carpenter, D.O., Nguyen, T., Le, L., Baibergenova, A. and Kudryakov, R. Profile of health effects related to proximity to PCB-contaminated hazardous waste sites in New York. FEB, 12: 173-180, 2003.
241. Hori, N., Carp, J.S., Carpenter, D.O. and Akaike, N. Corticospinal transmission to motoneurons in cervical spinal slices from adult rats. Life Sci., 72: 389-396, 2002.
242. Carpenter, D.O. and Hussain, R.J. Cell-to-cell communication of neurons is impaired by metals. Mat.-wiss. U. Werkstofftech. 34: 1-8, 2003.
243. Tan, Y., Hori, N. and Carpenter, D.O. The mechanism of presynaptic long-lasting-depression mediated by group 1 metabotropic glutamate receptors. Cell. Molec. Neurobiol., 23: 187-203, 2003.
244. Baibergenova, A., Kudryakov, R., Zdeb, M., and Carpenter, D.O. Low birth weight and residential proximity to PCB-contaminated waste sites. Environ. Health Perspect., 111: 1352-1357, 2003.
245. Nishizaki, Y., Oyama, Y., Sakai, Y., Hiram, S., Tomita, K., Nakao, H., Umebayashi, C., Ishida, S., Okano, Y. and Carpenter, D.O. PbCl₂-induced hyperpolarization of rat thymocytes: Involvement of charybdotoxin-sensitive K⁺ channels. Environ. Toxicol., 18(5): 321-326, 2003.
246. Hussain, R.J. and Carpenter, D.O. The effects of protein kinase C activity on synaptic transmission in two areas of rat hippocampus. Brain Res., 990: 28-37, 2003.
247. Suk, W.A., Ruchirawat, K., Balakrishnan, K., Berger, M., Carpenter, D., Damstra, T., Pronczuk de Garbino, J., Koh, D., Landrigan, P.J., Makalinao, I., Sly, P.D., Xu, Y. and Zheng, B.S. Environmental threats to children's health in Southeast Asia and the Western Pacific. Environ. Health Perspect. 111: 1340, 2003.
248. Carpenter, D.O. The need for global environmental health policy. New Solutions, 13(1): 53-59, 2003.
249. Tan, Y., Li, D., Song, R., Lawrence, D. and Carpenter, D.O. Ortho-substituted PCBs kill thymocytes. Toxicol. Sci., 76: 328-337, 2003.
250. Boldyrev, A., Bulygina, E., Carpenter, D.O. and Schoner, W. Glutamate receptors communicate with Na⁺/K⁺-ATPase in rat cerebellum granule cells: Demonstration of differences in the action of several metabotropic and ionotropic glutamate agonists on intracellular reactive oxygen species and the sodium pump. J. Molec. Neurosci., 21:213-222, 2003.

251. Hites, R.A., Foran, J.A., Carpenter, D.O., Hamilton, M.C., Knuth, B.A. and Schwager, S.J. Global assessment of organic contaminants in farmed salmon. Science 303: 226-229, 2004.
252. Sahdal, S., Yilmaz, B., Chen, C-H and Carpenter, D.O. Comparative effects of technical toxaphene, 2,5-dichloro-3-biphenylol and octabromodiphenylether on cell viability, $[Ca^{2+}]$ levels and membrane fluidity in mouse thymocytes. Toxicol. Letts., 151: 417-428, 2004.
253. Tan, Y., Chen, C-H., Lawrence, D. and Carpenter, D.O. Ortho-substituted PCBs kill cells by altering membrane structure. Toxicol. Sci., 80: 54-59, 2004.
254. Tan, Y., Song, R., Lawrence, D. and Carpenter, D.O. Ortho-substituted but not coplanar PCBs rapidly kill cerebellar granule cells. Toxicol. Sci., 79: 147-156, 2004.
255. Ozcan, M., Yilmaz, B., King, W.M. and Carpenter, D.O. Hippocampal long-term potentiation (LTP) is reduced by a coplanar PCB congener. NeuroToxicology, 25: 981-988, 2004.
256. Ssempebwa, J.C., Carpenter, D.O., Yilmaz, B., DeCaprio, A.P., O=Hehir, D.J. and Arcaro, K.F. Waste crankcase oil: an environmental contaminant with potential to modulate estrogenic responses. J. Toxicol. Environ. Hlth, Part A, 67: 1081-1094, 2004.
257. Foran, J.A., Hites, R.A., Carpenter, D.O., Hamilton, M.C., Mathews-Amos, A. and Schwager, S.J. A survey of metals in tissues of farmed Atlantic and wild Pacific salmon. Environ. Toxicol. Chem., 23: 2108-2110, 2004.
258. Oenga, G.N., Spink, D.C. and Carpenter, D.O. TCDD and PCBs inhibit breast cancer cell proliferation in vitro. Toxicol. In Vitro, 18: 811-819, 2004.
259. Hussain, R.J. and Carpenter, D.O. A comparison of the roles of protein kinase C in long-term potentiation in rat hippocampal areas CA1 and CA3. Cell. Molec. Neurobiol., 25: 649-661, 2005.
260. Hites, R.A., Foran, J.A., Schwager, S.J., Knuth, B.A., Hamilton, M.C. and Carpenter, D.O. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. Organohalogen Compounds, 66: 3826-3829, 2004.
261. Kudiyakov, R., Baibergenova, A., Zdeb, M. and Carpenter, D.O. Respiratory disease in relation to patient residence near to hazardous waste sites. Environ. Toxicol. Pharmacol., 18: 249-257, 2004.
262. Gilbertson, M. and Carpenter, D.O. An ecosystem approach to the health effects of mercury in the Great Lakes basin ecosystem. Environ. Res. 95: 240-246, 2004.
263. Hites, R.A., Foran, J.A., Schwager, S.J., Knuth, B.A., Hamilton, M.C. and Carpenter, D.O. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. Environ. Sci. Technol., 38: 4945-4949, 2004.
264. DeCaprio, A.P., Johnson, G.W., Tarbell, A.M., Carpenter, D.O. Chiarenzelli, J.R., Morse, G.S., Santiago-Rivera, A.L., Schymura, M.J., and the Akwesasne Task Force on the Environment. PCB exposure assessment by multivariate statistical analysis of serum congener profiles in an adult Native American population. Environ. Res., 98: 284-302, 2005.
265. Boldyrev, A.A., Kazey, V.I., Leinsoo, T.A., Mashkina, A.P., Tyulina O.V., Tuneva, J.O., Chittur, S. and Carpenter, D.O. Rodent lymphocytes express functionally active glutamate receptors. Biochem. Biophys. Res. Comm., 324: 133-139, 2004.
266. Boldyrev, A.A., Koudinov, A., Berezov, T. and Carpenter, D.O. Amyloid- β induced cell death is independent of free radicals. J. Alzheimer's Dis., 6: 633-638, 2004.
267. Neagu, B., Strominger, N.L. and Carpenter, D.O. Use of bipolar parallel electrodes for well-controlled microstimulation in a mouse hippocampal brain slice. J. Neurosci. Meth., 144: 153-163, 2005.
268. Suk, W.A., Avakian, M.D., Carpenter, D., Groopman, J.D., Scammell, M. and Wild, C.P. Human exposure monitoring and evaluation in the Arctic: The importance of understanding exposures to the development of public health policy. Environ. Health Perspect. 112: 113-120, 2004.

269. Neagu, B., Neagu, E.R., Strominger, N.L. and Carpenter, D.O. A new fast electro-physiological response measured extracellularly in a mouse hippocampal brain slice. Neurosci. Letts., 381: 179-184, 2005.
270. Sergeev, A.V. and Carpenter, D.O. Hospitalization rates for coronary heart disease in relation to residence near areas contaminated with POPs and other pollutants. Environ. Health Perspect., 113: 756-761, 2005.
271. Foran, J.A., Carpenter, D.O., Hamilton, M.C., Knuth, B.A. and Schwager, S.J. Risk-based consumption advice for farmed Atlantic and wild Pacific salmon contaminated with dioxins and dioxin-like compounds. Environ. Health Perspect. 113: 552-556, 2005.
272. Shaw, S.D., Bourakovsky, A., Brenner, D., Carpenter, D.O., Tao, L., Kannan, K. and Hong, C-S. Polybrominated diphenyl ethers (PBDEs) in farmed salmon from Maine and Eastern Canada. In: Proceedings of 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs (DIOXIN 2005), August 21-26, 2005, Toronto, Canada.
273. Carpenter, D.O., DeCaprio, A.P., O=Hehir, D., Akhtar, F., Johnson, G., Scrudato, R.J., Apatiki, L., Kava, J., Gologergen, J., Miller, P.K. and Eckstein, L. Polychlorinated biphenyls in serum of the Siberian Yupik people from St. Lawrence Island, Alaska. Int. J. Circumpolar Health, 64(4): 322-335, 2005.
274. Foran, J.A., Good, D.H., Carpenter, D.O., Hamilton, M.C., Knuth, B.A. and Schwager, S.J. Quantitative analysis of the benefits and risks of consuming farmed and wild salmon. J. Nutr 135: 2639-2643, 2005.
275. Huang, X., Hites, R.A., Foran, J.A., Hamilton, C., Knuth, B.A., Schwager, S.J. and Carpenter, D.O. Consumption advisories for salmon based on risk of cancer and non-cancer health effects. Environ. Res., 101: 263-274, 2006.
276. Shcherbatykh, I., Huang, X., Lessner, L. and Carpenter, D.O. Hazardous waste sites and stroke in New York State. Environ. Health, 4:18, 2005.
277. Hamilton, M.C., Hites, R.A., Schwager, S.J., Foran, J.A., Knuth, B.A. and Carpenter, D.O. Lipid composition and contaminants in farmed and wild salmon. Environ. Sci. Tech., 39: 8622-8629, 2005.
278. Yilmaz, B., Sandal, S., Chen, C-H. and Carpenter, D.O. Effects of PCB 52 and PCB 77 on cell viability, $[Ca^{2+}]_i$ levels and membrane fluidity in mouse thymocytes. Toxicology, 217: 184-193, 2006.
279. Tan, Y., Hori, N., and Carpenter, D.O. Electrophysiological effects of three groups of glutamate metabotropic receptors in rat piriform cortex. Cell. Molec. Neurobiol., 26: 915-924, 2006.
280. Boldyrev, A.A., Carpenter, D.O. and Johnson, P.A., Emerging evidence for a similar role of glutamate receptors in the nervous and immune systems. J. Neurochem., 95: 913-918, 2005.
281. Sandal, S., Yilmaz, B., Godekmerdan, A., Kelestimur, H. and Carpenter, D.O. Effects of PCBs 52 and 77 on Th1/Th2 balance in mouse thymocyte cell cultures. Immunopharmacol. Immunotoxicol. 27: 601-613, 2005.
282. Carpenter, D.O. Environmental contaminants and learning and memory. International Congress Series, 1287: 185-189, 2006.
283. Carpenter, D.O. Polychlorinated biphenyls (PCBs): Routes of exposure and effects on human health. Rev. Environ. Health, 21: 1-23, 2006.
284. Huang, X., Lessner, L. and Carpenter, D.O. Exposure to persistent organic pollutants and hypertensive disease. Environ. Res., 102: 101-106, 2006.
285. Carpenter, D.O., El-Qaderi, S., Fayzieva, D., Gilani, A., Hambartsumyan, A., Herz, K., Isobaev, M., Kasymov, O., Kudyakov, R., Majitova, Z., Mamadov, E., Nemer, L., Revich, B., Stege, P., Suk, W., Upshur, R., Yilmaz, B. and Zaineh K. Children's environmental health in Central Asia and the Middle East. Int. J. Occup. Environ. Health, 12: 362-368, 2006.
286. King, W.M., Sarup, V., Sauve, Y., Moreland, C.M., Carpenter, D.O. and Sharma, S.C. Expansion of visual receptive fields in experimental glaucoma. Visual Neurosci. 23: 137-142, 2006.

287. Tuneva, J., Chittur, S., Boldyrev, A.A., Birman, I. and Carpenter, D.O. Cerebellar granule cell death induced by aluminum. Neurotox. Res., 9: 297-304, 2006.
288. Trasande, L., Boscarino, J., Graber, N., Falk, R., Schechter, C., Dunkel, G., Geslani, J., Moline, J., Kaplan-Liss, E., Miller, R.K., Korfmacher, K., Carpenter, D., Balk, S.J., Laraque, D., Frumkin, H. and Landrigan, P.J. The environment in pediatric practice: A study of New York pediatricians' attitudes, beliefs, and practices towards children's environmental health. J. Urban Health, 2006, DOI: 10.1007/s11524-006-9071-4.
289. Surdu, S., Montoya, L.D., Tarbell, A. and Carpenter, D.O. Childhood asthma and indoor allergens in Native Americans in New York. Environ. Health: A Global Access Science Source, 5:22, 2006. DOI: 10.1186/1476-069X-5-22.
290. Ozcan M., Yilmaz, B. and Carpenter, D.O. Effects of melatonin on synaptic transmission and long term potentiation in two areas of mouse hippocampus. Brain Res., 1111: 90-94, 2006.
291. Shaw, S.D., Brenner, D., Berger, M.L., Pulser, E.L., Carpenter, D.O., Hong, C-W and Kannan K. PCBs, dioxin-like PCBs, dioxins, and organochlorine pesticides in farmed salmon (*Salmo salar*) from Maine and Eastern Canada. Environ. Sci. Technol. 40: 5347-5354, 2006.
292. Yilmaz, B., Ssempebwa J., Mackerer, C.R., Arcaro, K.F. and Carpenter, D.O. Effects of polycyclic aromatic hydrocarbon-containing oil mixtures on generation of reactive oxygen species and cell viability in MCF-7 breast cancer cells. J. Toxicol. Environ. Health, Part A: 70: 1-8, 2007.
293. Kouznetsova, M., Huang, X., Ma, J., Lessner, L. and Carpenter, D.O. Increased rate of hospitalization for diabetes and residential proximity of hazardous waste sites. Environ. Health Perspect., 115:75-79, 2007.
294. Yilmaz, Y., Seyran, A.D., Sandal, S., Aydin, M., Colakoglu, N., Kocer, M. and Carpenter, D.O. Modulatory effects of Aroclors 1221 and 1254 on bone turnover and vertebral histology in intact and ovariectomized rats. Toxicology Letts., 166: 276-294, 2006.
295. Shcherbatykh, I. and Carpenter, D.O. The role of metals in the etiology of Alzheimer's disease. J. Alzheimer's Dis., 11: 191-205, 2007.
296. Surdu S, Neamtiu I, Gurzau E, Kasler I and Carpenter D. Blood lead levels and hand lead contamination in children ages 4-6 in Copsa Mica, Romania. In: *Environmental Health in Central and Eastern Europe*. KC Donnelly and LH Cizmas, Eds. Springer Netherlands. pp. 123-134, 2007.
297. Carpenter D.O. The importance of the Great Lakes Water Quality Agreement. J Public Health Policy 28: 216-220, 2007.
298. Codru N, Schymura MJ, Negoita S, the Akwesasne Task Force on the Environment, Rej R and Carpenter DO. Diabetes in relation to serum levels of polychlorinated biphenyls (PCBs) and chlorinated pesticides in adult Native Americans. Environ Health Perspect. 115: 1442-1447, 2007.
299. Carpenter DO. Biomarcadores de efectos neuroconductuales. Acta Toxicol Argent 14 (Suplemento): 11-12, 2006.
300. Hennig B, Ormsbee L, Bachas L, Silverstone A, Milner J, Carpenter D, Thompson C and Suk WA . Introductory comments: nutrition, environmental toxins and implications in prevention and intervention of human diseases. J Nutrit Biochem 189: 161-163, 2007.
301. Arnold R, Armour MA, Barich J, Cebrian M, Cifuentes L, Kirk D, Koh D, Lewis ND, Ling B, Makalinalao I, Maiden T, Paz-y-Mino C, Peralta G, Singh K, Sly P, Suk W, Woodward A, Zheng B and Carpenter DO. Threats to human health and environmental sustainability in the Pacific Basin: The 11th International Conference of the Pacific Basin Consortium. Environ Health Perspect. 115: 1770-1775, 2007.
302. Parrish RR, Horstwood M, Arnason JG, Chenery S, Brewer T, Lloyd NS and Carpenter DO (2008) Depleted uranium contamination by inhalation exposure and its detection after approximately 25 years: Implications for health assessment. Sci Total Environ 390: 58-68.

303. Goncharov A, Haase RF, Santiago-Rivera A, Morse G, Akwesasne Task Force on the Environment, McCaffrey RJ, Rej R and Carpenter DO. High serum PCBs are associated with elevation of serum lipids and cardiovascular disease in a Native American population. *Environ Res.* 106: 226-239.
304. Ma J, Kouznetsova M, Lessner L and Carpenter DO. Asthma and infectious respiratory disease in children – correlation to residence near hazardous waste sites. *Paediatr Respir Rev* 8: 292-298, 2007.
305. Schell LM, Gallo MV, Denham M, Ravenscroft J, DeCaprio AP and Carpenter DO (2008) Relationship of thyroid hormone levels of polychlorinated biphenyls, lead, p,p'-DDE and other toxicants in Akwesasne Mohawk youth. *Environ Health Perspect.* 116: 806-813.
306. Ssempebwa J and Carpenter DO (2009) The generation, use and disposal of waste crankcase oil in developing countries: A case for Kampala District, Uganda. *J Hazard Materials* 161: 835-841.
307. Carpenter DO (2008) Environmental contaminants as risk factors for developing diabetes. *Rev Environ Health* 23: 59-74.
308. Shaw SD, Berger ML, Brenner D, Carpenter DO, Lao L, Hong CS and Kannan K (2008) Polybrominated diphenyl ethers (PBDEs) in farmed and wild salmon marketed in the Northeastern United States. *Chemosphere* 71: 1422-1431.
309. Suleyman S, Yilmaz B and Carpenter DO (2008) Genotoxic effects of PCB 52 and PCB 77 on cultured human peripheral lymphocytes. *Mutation Res.* 654: 88-92.
310. Carpenter DO and Sage C (2008) Setting prudent public health policy for electromagnetic field exposures. *Rev Environ Health* 23: 91-117.
311. Neagu B, Strominger NL and Carpenter DO (2008) Contribution of NMDA receptor-mediated component to the EPSP in mouse Schaffer collateral synapses under single pulse stimulation protocol. *Brain Res.* 1240: 54-61.
312. Holdren J, Tao S and Carpenter DO (2008) Environment and health in the 21st Century: Challenges and solutions. *Ann NY Acad Sci.* 1140:1-21.
313. Carpenter DO, Ma J and Lessner L (2008) Asthma and infectious respiratory disease in relation to residence near hazardous waste sites. *Ann NY Acad Sci.* 1140: 201-208.
314. Sandal S, Tuneva J, Yilmaz B and Carpenter DO (2009) Effects of cholesterol and docosahexaenoic acid on cell viability and (Ca²⁺)_i levels in acutely isolated mouse thymocytes. *Cell Biochem Funct* 27: 155-161.
315. Steele RE, de Leeuw, E and Carpenter DO (2009) A novel and effective treatment modality for medically unexplained symptoms. *J Pain Management* 1: 402-412
316. Sage C and Carpenter DO (2009) Public health implications of wireless technologies. *Pathophysiology* 16: 233-246.
317. Sly PD, Eskenazi B, Pronczuk J, Sram R, Diaz-Barriga F, Machin DG, Carpenter DO, Surdu S and Meslin EM (2009) Ethical issues in measuring biomarkers in children's environmental health. *Environ Health Perspect.* 117: 1185-1190.
318. Goncharov A, Rej R, Negoita S, Schymura M, Santiago-Rivera A, Morse G, Akwesasne Task Force on the Environment and Carpenter DO (2009) Lower serum testosterone associated with elevated polychlorinated biphenyl concentrations in Native American men. *Environ Health Perspect.* 117:1454-1460.
319. Tuneva JO, Karpova LV, Shittur SV, Carpenter DO, Johnson P and Boldyrev AA (2009) Amyloid- β and aluminum ions enhance neuronal damage mediated by NMDA-activated glutamate receptors. *Biochemistry (Moscow) Supplement Series A: Membrane and Cell Biology* 4: 466-471.
320. Carpenter DO and Nevin R (2009) Environmental causes of violence. *Physiol Behavior* 99: 260-268.

321. Goncharov A, Bloom MS, Pavuk M, Carpenter DO for the Anniston Environmental Health Research Consortium. (2009) Exposure to PCBs and hypertension in the Anniston Community Health Survey. *Organohal Comp* 71: 0-136.
322. Sergeev AV and Carpenter DO (2010) Exposure to persistent organic pollutants and first-time hospitalizations for myocardial infarction with comorbid diabetes mellitus: A 12-year population-based study. *Int J Occup Med Environ Health* In press.
323. Carpenter DO (2010) Electromagnetic fields and cancer: The cost of doing nothing. *Rev Environ Health* 25: 75-80.
324. Sergeev AV and Carpenter DO (2010) Exposure to persistent organic pollutants increases hospitalization rates for myocardial infarction with comorbid hypertension. *Primary Prevention Insights*. In press.

Books:

1. Cellular Pacemakers I: Mechanisms of Pacemaker Generation, David O. Carpenter, editor; John Wiley & Sons, New York, 1982.
2. Cellular Pacemakers II: Function in Normal and Disease States, David O. Carpenter, editor; John Wiley & Sons, New York 1982.
3. Biologic Effects of Electric and Magnetic Fields, Volume I: Sources and Mechanisms of Biologic Effects, David O. Carpenter and Sinerik Ayrapetyan, editors; Academic Press, California, 1994.
4. Biologic Effects of Electric and Magnetic Fields, Volume II: Beneficial and Harmful Effects, David O. Carpenter and Sinerik Ayrapetyan, editors; Academic Press, California, 1994.
5. Environmental Challenges in the Pacific Basin, David O. Carpenter, ed. New York Academy of Sciences, Vol 1140, 457 pp, 2008.

Reviews and Book Chapters:

1. Carpenter, D.O. Ionic mechanisms and models of endogenous discharge of *Aplysia* neurons. Proceedings of the Symposium on Neurobiology of Invertebrates: Mechanisms of Rhythm Regulation. Tihany, Hungary, August 2-5, 1971, Hungarian Academy of Sciences, pp. 35-58, 1973.
2. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Measurements of intracellular conductivity in *Aplysia* neurons: Evidence for organization of water and ions. *Ann. NY Acad. Sci.*, 204:502-533, 1973.
3. Carpenter, D.O., Hubbard, J.H., Humphrey, D.R., Thompson, H.K. and Marshall, W.H. CO₂ effects on nerve cell function. In: Topics in Environmental Physiology and Medicine: Carbon Dioxide and Metabolic Regulation. (Eds.: G. Nahas and K.A. Schaefer), Springer-Verlag, New York, pp. 49-62, 1974.
4. Parmentier, J. and Carpenter, D.O. Blocking action of snake venom neurotoxins at receptor sites to putative central nervous system transmitters. In: Animal, Plant and Microbial Toxins (Eds.: A. Ohaska, K. Hayashi, and Y. Sawai), Plenum Press, London, Vol. 2, pp. 179-191, 1976.
5. Pierau, Fr.-K. and Carpenter, D.O. Metabolic control of peripheral temperature receptors in the scrotal skin of the rat. *Israel J. Med. Sci.*, 12:1044-1046, 1976.
6. Carpenter, D.O. Membrane Excitability: In: Mammalian Cell Membranes Vol. 4, Membranes and Cellular Functions, (Eds.: G.A. Jamieson and D.M. Robinson), Butterworth & Co., London, pp. 184-206, 1977.
7. Carpenter, D.O., Myers, P.R., Shain, W., Sinback, C.N. and Swann, J.W. Interchangeable association of neurotransmitter receptors and ionophores in vertebrate and invertebrate cells. Proc. Symposium: "Iontophoresis and Transmitter Mechanisms in the Mammalian Central Nervous System", Cambridge, England, Raven Press, pp. 203-205, 1978.
8. Carpenter, D.O., McCreery, M.J., Woodbury, C.M. and Yarowsky, P.J. Modulation of endogenous discharge in neuron R-15 through specific receptors for several neurotransmitters. In: Abnormal

Neuronal Discharges, (Eds: N. Chalazonitis and M. Boisson), Raven Press, New York, pp. 189-203, 1978.

9. Tsien, R.W. and Carpenter, D.O. Ionic mechanisms of pacemaker activity in cardiac purkinje fibers. Fed. Proc., 37:2127-2131, 1978.
10. Keabian, P.R., Keabian, J.W. and Carpenter, D.O. Serotonin causes accumulation of cyclic AMP in *Aplysia* heart. The Proceedings of the Fourth International Catecholamine Symposium, (Eds: E. Usdin and I. Kopin), Pergamon Press, New York, pp. 1167-1169.
11. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Direct and modulatory actions of thyrotropin-releasing hormone (TRH) in sensorimotor cortex. Proc. EMBO Workshop on Drug Receptors in the Central Nervous System, Weizman Institute of Science, Rehovot, Israel, February 10-14, 1980.
12. Carpenter, D.O. Ionic and metabolic bases of neuronal thermosensitivity. Fed. Proc., 40:2808-2813, 1981.
13. Carpenter, D.O. and Reese, T.S. Chemistry and Physiology of Synaptic Transmissions. In: Basic Neurochemistry, 3rd Edition, (Eds.: Siegel, Albers, Agranoff and Katzman), Little, Brown and Company, pp. 161-168, 1981.
14. Shain, W. and Carpenter, D.O. Mechanisms of synaptic modulation. Intl. Rev. Neurobiol., 22:205-247, 1981.
15. Wiederhold, M.L. and Carpenter, D.O. Possible Role of Pacemaker Mechanisms in Sensory Systems. In: Cellular Pacemakers II: Function in Normal and Disease States, (Ed.: D.O. Carpenter), John Wiley & Sons, New York, pp. 27-58, 1982.
16. Carpenter, D.O. The generator potential mechanism in cold afferents may be an electrogenic sodium pump. Workshop on Mechanisms of Thermal Regulations. J. Therm. Biol., 387-390, 1983.
17. Carpenter, D.O. and Gregg, R.A. Functional significance of electrogenic pumps in neurons. In: Electrogenic transport: Fundamental Principles and Physiological Implications, (Eds.: M. Blaustein and M. Liebermann), Raven Press, pp. 253-270, 1984.
18. Carpenter, D.O., Briggs, D.B. and Strominger, N. Behavioral and electrophysiological studies of peptide-induced emesis in dogs. Fed. Proc., 43:16-18, 1984.
19. Coyle, J.T., Blakeley, R.D., Zaczeck, R., Ory-Lavallee, L., Koller, K., French-Mullen, J.M.H. and Carpenter, D.O. Acidic peptides in brain: Do they act at putative glutamatergic synapses. In: Excitatory Amino Acids and Epilepsy, (Eds.: Y. Ben-Ari and R. Schwarcz), Plenum Press, New York, pp. 375-384.
20. Carpenter, D.O., French-Mullen, J.M.H., Hori, N., Sinback, C.N. and Shain, W. Segregation of synaptic function on excitable cells. In: Neural Mechanisms of Conditioning, (Eds.: D. Alkon and C.D. Woody), Plenum Press, NY, pp. 355-369, 1985.
21. Carpenter, D.O. and Hall, A.F. Responses of *Aplysia* cerebral ganglion neurons to leucine enkephalin. In: Comparative Aspects of Opioid and Related Neuropeptide Mechanisms, (Eds.: M. Leung and G. Stefano), CRC Press, pp. 49-57.
22. Zaczeck, R., Koller, K., Carpenter, D.O., Fisher, R., French-Mullen, J.M.H. and Coyle, J.T. Interactions of acidic peptides: Excitatory amino acid receptors. In: Excitatory Amino Acids, (Ed.: P.J. Roberts), Macmillan, London, 1987.
23. Carpenter, D.O. Central nervous system mechanisms in deglutition and emesis. In: Handbook of Physiology, Section 6: The Gastrointestinal System. Vol. I, Motility and Circulation, (Ed.: J.D. Wood), American Physiological Society, Chapter 18, pp. 685-714, 1989.
24. Carpenter, D.O., Briggs, D.B. and Strominger, N. Mechanisms of radiation-induced emesis in the dog. Pharmacol. Ther., 39:367-371, 1988.
25. Carpenter, D.O. Comparative biology of neurotransmitter functions. Biology International, 15:2-9, 1987.

26. Carpenter, D.O. Electromagnetic Fields: Do We Know Enough to Act? In: Health and Environmental Digest, Vol. 2, pp. 3-4, 1988.
27. Carpenter, D.O. The New York State Power Lines Project: Summary and Conclusions. In: 20th Annual National Conference on Radiation Control, CRCPD Publication 88-6, Nashville, Tennessee, May 15-19, 1988, pp. 399-409.
28. S.-Rozsa, K., Carpenter, D.O., Stefano, G.B. and Salanki, J. Distinct responses to opiate peptides and FMRFamide on B-neurons of the Aplysia cerebral ganglia. In: Comparative Aspects of Neuropeptide Function, (Eds. E. Florey and G.B. Stefano), Manchester University Press, Chapter 6, pp. 73-86, 1991.
29. Carpenter, D.O. A common mechanism of excitation of area postrema neurons by several neuropeptides, hormones and monoamines. In: Comparative Aspects of Neuropeptide Function, (Eds. E. Florey and G.B. Stefano) Manchester University Press, Chapter 21, pp. 260-270, 1991.
30. Carpenter, D. O., Hirotsu, I., Katsuda, N. and Hori, N. The effects of acetylcholine and aging on electrical excitability of the central nervous system. In: Neuroregulatory Mechanisms in Aging, Pergamon Press LTD, pp. 5-23, 1993.
31. Turner, J.N., Swann, J.W., Szarowski, D.H., Smith, K.L., Shain, W., Carpenter, D.O. and Fejtl, M. Three-dimensional confocal light and electron microscopy of neurons: fluorescent and reflection stains. Methods in Cell Biology, 38:345-366, 1993.
32. Deno, D. and Carpenter, D.O. Sources and characteristics of electric and magnetic fields in the environment. In: Biologic Effects of Electric and Magnetic Fields, Volume I: Sources and Mechanisms of Biologic Effects, David O. Carpenter and Sinerik Ayrapetyan, editors, Academic Press, California, pp. 3-59, 1994.
33. Carpenter, D.O. The public health implications of magnetic field effects on biological systems. In: Biologic Effects of Electric and Magnetic Fields, Volume II: Beneficial and Harmful Effects, David O. Carpenter and Sinerik Ayrapetyan, editors, Academic Press, California, pp. 321-329, 1994.
34. Carpenter, D.O. Multidisciplinary study of hazardous wastes at a Great Lakes Superfund Site. Great Lakes Research Review, 1: 37-39, 1994.
35. Fejtl, M. and Carpenter, D.O. Single-channel studies in molluscan neurons. In: Ion Channels, Vol. 4, Toshio Narahashi, ed., Plenum Press, New York, pp. 333-376, 1996.
36. Turner, J.N., Swann, J.W., Szarowski, D.H., Smith, K.L., Shain, W., Carpenter, D.O. and Fejtl, M. Three-dimensional confocal light and electron microscopy of central nervous system tissue, and neurons and glia in culture. In: International Review of Experimental Pathology, V.J. Savin and T.B. Wiegmann, editors, Volume 36, Academic Press, pp. 53-72, 1996.
37. Boldyrev, A., Lawrence, D. and Carpenter, D. Effect of carnosine and its natural derivatives on apoptosis of neurons induced by excitotoxic compounds. In: Peptide Science-Present and Future, Y. Shimonishi, editor, Kluwer Academic Publishers, Great Britain, pp. 424-426, 1998.
38. Carpenter, D.O., Hussain, R., Tan, Y., Niemi, W. and Hori, N. Long-term potentiation and long-term depression: Relevance to learning and memory. In: Modern Problems of Cellular and Molecular Biophysics. S.N. Ayrapetyan and A.C.T. North, editors, Nayan Tapan, pp. 83-94, 2001.
39. Carpenter, D.O. NMDA receptors and molecular mechanisms of excitotoxicity. In: Oxidative Stress at Molecular, Cellular and Organ Levels, A. Boldyrev and P. Johnson, editors, Research Signpost, pp. 77-88, 2002.
40. Carpenter, D.O. Clearing the air: Asthma an indoor exposure. JNMA 96: 1-2, 2004.
41. Carpenter DO. Environmental contaminants and human health: The health effects of persistent toxic substances. Firat Tip Dergisi 10: ____: 2005.
42. Hermanson MH, Johnson GW and Carpenter DO. Routes of human exposure to PCBs in Anniston, Alabama. ACS Division of Environmental Chemistry, 232rd National Meeting, 46: 1117-1122, 2006

Other Publications:

1. Barker, J.L. and Carpenter, D.O. Neuronal thermosensitivity. Science, 172:1361-1362, 1971.
2. Carpenter, D.O. Cellular Pacemakers. Fed. Proc., 37:2125-2126, 1978.
3. Carpenter, D.O. Membrane biophysics and general neurobiology in Japan. ONR Tokyo Scientific Bulletin, 3:23-27, 1978.
4. Carpenter, D.O. Research on the primate nervous system in Japan. ONR Tokyo Scientific Bulletin, 3:28-32, 1978.
5. Carpenter, D.O. Report on the Sixth International Biophysics Congress, Kyoto, Japan. ONR Tokyo Scientific Bulletin, 3:38-40, 1978.
6. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Research Bulletin, 4:149-152, 1978.
7. Carpenter, D.O. and Ahlbom, A. Power lines and cancer: Public health and policy implications. Forum, 3:96-101, 1988.
8. Carpenter, D.O. Setting Health Policy When the Science and the Risk are Uncertain. In: The Scientific Basis of Health Policy in the 1990s, Proceedings of the School of Public Health's Fifth Anniversary Symposium, 54-63, 1990.
9. Carpenter, D.O. Integrating public health in professional education. Optometry and Vision Science, 70: 699-702, 1993.
10. Bowerman, W.W., Carey, J., Carpenter, D.O., Colborn, T., DeRosa, C., Fournier, M., Fox, G.A., Gibson, B.L., Gilbertson, M., Henshel, D., McMaster, S. and Upshur, R. Is it time for a Great Lakes Ecosystem Agreement separate from the Great Lakes Water Quality Agreement? J. Great Lakes Res. 25:237-238, 1999.
11. Carpenter, D.O. Editorial Comment of ΔPrimary hypoxic tolerance and chemical preconditioning during estrus cycle@. Stroke, 30:1262, 1999.
12. Carpenter, D.O. Bring environmental health back into public Health. J. Pub. Health Mgmt. Pract., 5:vii-viii, 1999.
13. Carpenter, D.O. Should children and women of childbearing age eat Great Lakes fish? Great Lakes Commission Advisor, 13: 8, 2000.
14. Hites, R.A., Foran, J.A., Schwager, S.J., Knuth, B.A., Hamilton, M.C. and Carpenter, D.O. Response to comment on ΔGlobal Assessment of Polybrominated Diphenyl Ethers in Farmed and Wild Salmon@. Environ. Sci. Technol. 39: 379-380.
15. Carpenter, D.O. Blood lead and IQ in older children. Letter to the editor. Environ. Health Perspect., 113: A581-A582, 2005.
16. Foran, J.A., Carpenter, D.O., Good, D.H., Hamilton, M.C., Hites, R.A., Knuth, B.A. and Schwager, S.J. Risks and benefits of seafood consumption. Letter to the editor. Am. J. Prev. Med. 30: 438-439, 2006.

Setting Prudent Public Health Policy for Electromagnetic Field Exposures

David O. Carpenter¹ and Cindy Sage²

¹*Institute for Health and the Environment, University at Albany, Rensselaer,
New York 12144;* ²*Sage Associates Santa Barbara, California; USA*

Abstract: Electromagnetic fields (EMF) permeate our environment, coming both from such natural sources as the sun and from manmade sources like electricity, communication technologies and medical devices. Although life on earth would not be possible without sunlight, increasing evidence indicates that exposures to the magnetic fields associated with electricity and to communication frequencies associated with radio, television, WiFi technology, and mobile cellular phones pose significant hazards to human health. The evidence is strongest for leukemia from electricity-frequency fields and for brain tumors from communication-frequency fields, yet evidence is emerging for an association with other diseases as well, including neurodegenerative diseases. Some uncertainty remains as to the mechanism(s) responsible for these biological effects, and as to which components of the fields are of greatest importance. Nevertheless, regardless of whether the associations are causal, the strengths of the associations are sufficiently strong that in the opinion of the authors, taking action to reduce exposures is imperative, especially for the fetus and children. Inaction is not compatible with the Precautionary Principle, as enunciated by the Rio Declaration. Because of ubiquitous exposure, the rapidly expanding development of new EMF technologies and the long latency for the development of such serious diseases as brain cancers, the failure to take immediate action risks epidemics of potentially fatal diseases in the future.

Keywords: leukemia, brain cancer, electricity, radiofrequency, cell phones, neurodegenerative diseases

Correspondence: David O. Carpenter, MD, 5 University Place, Rm A217, Rensselaer, NY 12144, USA; Fax (518) 525-2665; Telephone (518) 525-2660; email: carpent@uamail.albany.edu

TABLE OF CONTENTS

ABSTRACT

INTRODUCTION AND BACKGROUND

KEY FALLACIES AND ANSWERS IN THE DEBATE OVER ELECTROMAGNETIC FIELDS EVIDENCE

The evidence for elevated risk of childhood
leukemia from exposure to power-line frequency
EMF is weak and inconsistent

Only a small number of children are affected

The risk is low

The evidence that adult diseases are secondary to
50/60 Hz EMF exposure is insufficient

There is little evidence that low-intensity RF

fields pose human health hazards

There is no animal evidence

We do not know the mechanism

THE LEVELS OF PROOF AND STANDARDS OF
EVIDENCE FOR DECISION-MAKING DIFFER AMONG
PROFESSIONS

ELECTROMAGNETIC FIELDS AND PUBLIC HEALTH
STANDARDS OF EVIDENCE

DEFINING NEW EXPOSURE STANDARDS FOR ELF
AND RF

CONCLUSIONS

REFERENCES

INTRODUCTION AND BACKGROUND

Few issues have been as uncertain and divisive for so long a period as the question of whether exposure to electromagnetic fields (EMF) poses significant health hazards. The question of hazards from power line frequency EMF (50 Hz in much of the world, but 60 Hz in the United States (US), was first raised by the report of Wertheimer and Leeper /1/, who found elevated rates of childhood cancer in homes in Denver, Colorado that had elevated magnetic fields from neighborhood power lines. This initial report, greeted with significant skepticism, has been more-or-less replicated in most /2-4/ but not all /5-6/ succeeding studies. As everyone in the developed world is constantly exposed to electricity-derived EMFs, the question

of whether such exposures constitute a significant health hazard is of critical public health relevance.

The concerns, however, go way beyond just those exposures from power line-frequency EMFs. Figure 1 shows the electromagnetic spectrum, which goes from DC fields such as the magnetic field of the earth and the extremely low frequency (ELF) fields characteristic of electric power, to the very high frequency cosmic, gamma and X-ray EMFs, which have sufficient energy to break chemical bonds and are therefore are "ionizing" radiation. What is in between includes ultraviolet radiation, known to have significant adverse health effects /7/, visible light, which is essential for life, and the wide range of communication frequencies that are usually referred to as 'microwaves' or 'radiofrequency' (RF) fields.

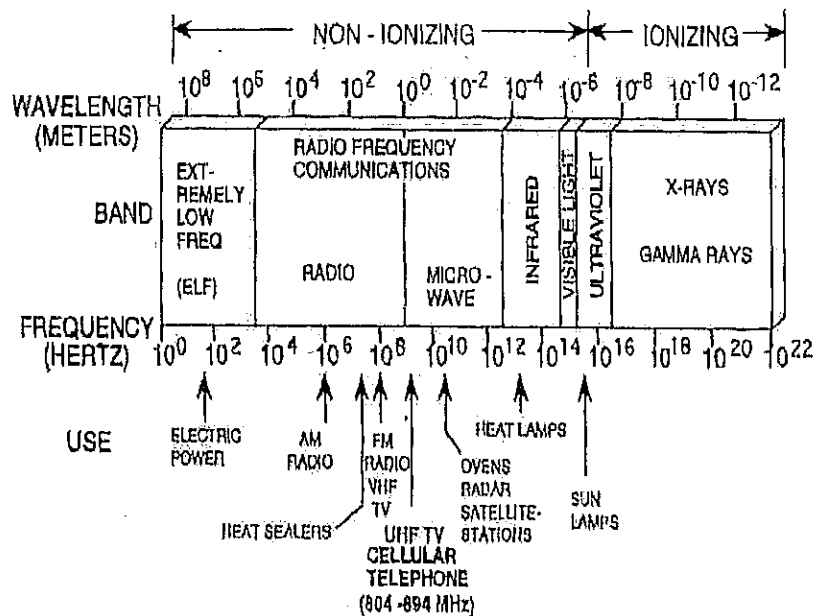


Fig. 1: The electromagnetic spectrum, showing the relations between ELF and RF fields, wavelength and frequency, and the ionizing and non-ionizing portions of the spectrum.

Public exposure to RF fields is increasing at a rapid rate. AM and FM radio and television stations broadcast signals that can be received almost everywhere in most countries. Most members of society now have and use cordless phones, cellular phones, and pagers. In addition, most populations are also exposed to antennas in communities designed to transmit wireless RF signals. Some developing countries have even given up running land lines because of the expense and their vulnerability, and because of easy access to cell phones. Long-term and cumulative exposure to such massively increased RF has no precedent in human history. Furthermore, the most pronounced change is for children, many of whom now routinely spend hours each day on the cell phone chatting or sending or receiving text messages. Everyone is exposed to a greater or lesser extent. No one can avoid exposure because even if living on a mountain-top without electricity, exposure to communication-frequency RF is likely. Vulnerable populations (pregnant women, very young children, elderly persons, the poor) are exposed to the same degree as the general population.

The energy within the EMF spectrum increases with the frequency; therefore, on the reasonable assumption that the relative health hazards are proportional to the energy, one would expect comparable RF exposures to be more hazardous than power-line frequency exposures. Although very little scientific investigation has been carried out on the health effects of RF fields until quite recently, the rapid profusion of WiFi (trade name for a high-frequency wireless local area network technology used in home networks, mobile phones, video games, and more), cell-phone towers, and cell-phone use in all segments of the population, including young children, makes it essential that risks to health be considered as technology advances.

This review was triggered by several reports /8-12/ and actions by governments and courts /13/ that, in the opinion of the authors, unjustifiably imply and/or conclude that EMF exposure does not

pose a significant health hazard to humans. These reviews and reports are important because they become the basis for regulatory standards. Each of these reports, however, presents evidence for the existence of human health hazards associated with EMFs, as well as discussions of the limitations in the overall understanding of the basis for such effects. The conservatism of their conclusions, in our view, fails to meet the standards of the European Commission Constitution Principle on Health (Section 3.1) /14/, European Union Treaties Article 174 /15/, the European Environmental Agency /16/, and other international statements on the "precautionary principle" as enunciated by the Rio Declaration of the United Nations /17/. The working definition used in the European Environmental Agency and that has been developed during the debates that followed the 2001 report, is explicit about specifying both uncertainty and ignorance as contexts for applying the principle, and in acknowledging that a case-specific sufficiency of scientific evidence is required to justify public policy action:

"The Precautionary Principle provides justification for public policy actions in situations of scientific complexity, uncertainty and ignorance, where there may be a need to act in order to avoid, or reduce, potentially serious or irreversible threats to health or the environment, using an appropriate level of scientific evidence, and taking into account the likely pros and cons of action and inaction" /16/.

We find that current standards in most countries are not protective of human health, and provide our reasoning for this important conclusion along with recommendation for standards that we feel to be appropriate based on current scientific evidence plus a consideration of the need for precaution. The issues surrounding EMF exposure are particularly important because of the exposure encountered by everyone to a greater or lesser extent. More

difficult is determining the degree of risk when no population is unexposed. Furthermore, the sources of EMF in the environment are such that exposure for any one person varies greatly throughout the day, depending upon where they are at any particular time. Exposure occurs at home from power lines in the street, household wiring, appliances, and wireless devices. Exposure will vary depending upon where one is in the house and what appliances or devices one is using or near. Exposures occur when walking down the street, while going to school or work, and during recreational activities. Each exposure is different in both frequency and intensity. Therefore, determining cumulative exposure over any significant period is exceptionally difficult. For all of these reasons it is likely that most studies, operating within these major limitations, have led to an underestimation of the true risk to human health. Therefore, considering ways in which to evaluate risk and reduce exposure is imperative. Good public health policy requires preventative action proportionate to the potential risk of harm and the public health consequence of taking no action.

KEY FALLACIES AND ANSWERS IN THE DEBATE OVER EMF EVIDENCE

Several arguments (false, in our view) have been presented by those who minimize the strength of the relation between exposure to both 50-60Hz ELF and RF EMFs. These arguments are as follows:

"Evidence for elevated risk of childhood leukemia from exposure to power line frequency EMF is weak and inconsistent"

The evidence reporting a relation between EMF exposure and childhood leukemia is neither weak nor inconsistent. The NRC (1997) report /8/ states,

"The link between wire-code rating and childhood leukemia is statistically significant

(unlikely to have arisen from chance) and is robust in the sense that eliminating any single study from the groups does to alter the conclusion that the associations exists."

In his introduction to the NIEHS EMF-RAPID program (1999) report /10/, Dr. Kenneth Olden, Director of the National Institute of Environmental Health Sciences, characterizes the state-of-the-art by the statement,

"The strongest evidence for health effects comes from associations observed in human populations with two forms of cancer: childhood leukemia and chronic lymphocytic leukemia in occupationally exposed adults. While the support from individual studies is weak, the epidemiological studies demonstrate, for some methods of measuring exposure, a fairly consistent pattern of a small, increased risk with increasing exposure that is somewhat weaker for chronic lymphocytic leukemia than for childhood leukemia."

Both reports then go on to minimize the observed relations based on the absence of knowledge about mechanisms explaining such relations. This is more directly stated in the 2007 WHO report /12/,

"Resolving the conflict between epidemiological data (which show an association between ELF magnetic field exposure and an increased risk of childhood leukemia) and experimental and mechanistic data (which do not support this association) is the highest research priority in this field."

Leaving aside the issue of mechanisms, which will be discussed later, it becomes apparent that all three reports have accepted the demonstration of a statistically significant relation between exposure to elevated magnetic power line fields and child-

hood leukemia. This conclusion is supported by at least three meta-analyses of the relation between childhood leukemia and EMFs. Wartenberg /18/ reported on 16 epidemiologic studies, considering reports using the Wertheimer and Leeper /1/ wire codes as well as measured fields, and concluded that *"the observed results identify a consistent risk that cannot be explained by random variations"*. Two more recent meta-analyses have been published. Greenland et al. /19/ reported a significantly elevated risk of 1.68 [95% Confidence Interval (CI) = 1.23-2.31] based on pooled results from 12 studies, using a time-weighted average of exposure greater than 3 mG (0.3 μ T). Ahlbom et al. /20/ reported on nine studies, and found a elevated risk of 2.0 (95% CI = 1.27-3.13) for exposures equal or greater than 4 mG (0.4 μ T) as compared with less than 1 mG (0.1 μ T).

These reports are important in that they show consistency of a clearly elevated risk of leukemia in children having EMF exposure from power-line fields in homes. These meta-analyses lead to the conclusion reflected in the WHO report that an association exists between childhood leukemia and exposure to elevated magnetic fields in homes.

In addition, several recent studies add to the conclusion that the exposure-leukemia relation is strong. Draper et al /6/ studied rates of leukemia in children in relation to proximity of their home to high-voltage power lines. The investigators found a dose-dependent relation, with relative risk being 1.69 (95% CI = 1.13-2.53) when comparing rates in children living within 200 m to those living > 600 m from the line, and the relative risk being 1.23 (95% CI = 1.02-1.49) for children living 200-600 m as compared with > 600 m. A significant ($P < .01$) trend was found in relation to closeness to the power line. In children with Down's Syndrome, Mejia-Arangure et al. /21/ found an OR of 3.7 (95% CI = 1.05-13.1) between spot measurements of magnetic fields greater than or equal to 6 mG (0.6 μ T) and leukemia. Foliart et al. /22/ examined the relation between magnetic field exposure and the survival of children with acute

lymphoblastic leukemia in the US and found a hazard ratio of 4.5 (95% CI = 1.5-13.8) for children exposed to greater than 3 mG (0.3 μ T) as compared with those having exposure to less than 1 mG (0.1 μ T). Svendsen et al. /23/ performed a similar study of German children with leukemia, and reported a hazard ratio of 2.6 (95% CI = 1.3-5.2) for the survival of children with acute lymphoblastic leukemia (ALL) exposed to 2 mG (0.2 μ T) during recovery as compared with those exposed to less than 1 mG (0.1 μ T).

Lowenthal et al. /24/ looked at adult lymphoproliferative and myeloproliferative diseases in relation to childhood residence within 300 m of a high-voltage power line during the first 15 years of life and found an OR of 3.23 (95% CI = 1.26-8.29). For those who lived within 300 m of a power line in the first 5 years of life, the increased risk was 4.74 (95% CI = 0.98-22.9), providing support for the hypothesis that younger children are more at risk, and that the resultant disease may occur many years later during adulthood. Infante-Rivard and Deadman /25/ showed that maternal exposure during pregnancy increased the risk of children 0-9 years of age developing leukemia (OR = 2.5, 95% CI = 1.2-3.0, for children of mothers in the highest 10% of exposure).

The observations of Lowenthal et al. /24/ and Infante-Rivard and Deadman /25/ are very important in that they demonstrate clearly that the fetus and young children are at greater risk than are adults, and that early life exposure may result in cancer many years later. This finding is consistent with a large body of information showing that the fetus and young child are more vulnerable than older persons are to chemicals /26/ and ionizing radiation /27/. This susceptibility may be why the evidence for the relation between magnetic field exposure and leukemia in children is stronger than that for adults. These considerations have led the US Environmental Protection Agency to propose a 10-fold risk adjustment for the first 2 years of life, and a 3-fold adjustment for years 3 to 5 /27/. Even these adjustments do not deal with fetal risk, which

is likely to be significantly greater because during this period of life, rapid organ development occurs.

In conclusion, the evidence for a relation between childhood exposures to magnetic fields, whether determined from residential wire codes or measured magnetic fields, and elevated rates of leukemia is consistent. Although the reported odds ratios are not particularly high, the limitations in the exposure assessment (consideration of only residential exposure from external power lines) are such that one would expect considerable underestimations of the actual risk.

"Only a small number of children are affected"

This argument is not correct because we do not know precisely how many children are affected with leukemia resulting from EMF exposure. In 1988, Carpenter and Ahlbom /28/ attempted to answer this question based on the results of the New York State Powerlines Project and the results of the study of Savitz et al. /2/, concluding that if the magnetic fields in homes in the US were similar to those in Denver, Colorado (where both the Wertheimer and Leeper /1/ and Savitz et al. /2/ studies were done), fully 10% to 15% of US childhood leukemia (about 1,000 cases) could be associated with residential magnetic field exposure from external power lines. The researchers then suggested that exposure to magnetic fields from non-residential sources (particularly appliances) must be at least equal in magnitude and that if so, then these two sources of exposure would account for 20% to 30% of all childhood leukemias. Other estimates are even higher /29/.

In the meta-analyses mentioned above, however, Greenland et al. /19/ calculated the attributable fraction of cases of childhood leukemia from residential magnetic field exposure in the US to be 3%. Kheifets et al. /30/ attempted to calculate the attributable fraction of worldwide childhood leukemia due to EMFs based on the meta-analyses of Ahlbom et al. /20/ and Greenland et al. /19/. The authors concluded that the attributable fraction of

leukemia was between < 1% to 4%. The recent WHO Environmental Health Criteria ELF Monograph #238 /12/ states,

"(A)ssuming that the association is causal, the number of cases of childhood leukaemia worldwide that might be attributable to exposure can be estimated to range from 100 to 2,400 cases per year. However this represents 0.2 to 4.9% of the total annual incidence of leukaemia cases, estimated to be 49,000 worldwide in 2000. Thus, in a global context, the impact on public health, if any, would be limited and uncertain."

We strongly disagree with the overall conclusion that these calculations indicate that the fraction of childhood leukemia attributable to EMFs is so small that it lacks serious public health implications. There are several reasons why the WHO ELF Environmental Health Criteria Monograph /12/ conclusions (as well as those of the earlier reports) are not justified. These studies all considered either only measured magnetic fields in homes or wire codes from power lines, ignoring exposure from appliances, wireless devices, and all exposures outside of the home. Thus, these metrics do not come close to accounting for any individual's cumulative exposure to EMFs. If residential magnetic fields cause cancer, then those from other sources will add to the risk, but only the Carpenter and Ahlbom /28/ analysis considered this factor. The failure to measure total EMF exposure would tend to obscure the relation and lead to significant underestimations of the true relation between exposure and disease.

A few reports have looked at childhood cancer specifically and solely in relation to appliance use. Savitz et al. /31/ reported weak associations between childhood leukemia and the use of both prenatal and postnatal electric blankets. Hatch et al. /32/ found statistically significant elevations in ALL in children whose mothers reported using an electric blanket or mattress pad during pregnancy

(OR = 1.59, 95% CI = 1.11-2.29). Children's use of electric blankets or mattress pads also showed a significant elevation in risk of ALL (OR = 2.75, 95% CI = 1.52-4.98). These reports clearly support the proposition that appliance use must be incorporated into the measurements of total exposure. None of the studies done to date has dealt with exposures at day care centers or schools, or at other places outside of the home where children spend time. Yet all such places are important in the consideration of cumulative exposure and risk.

Although the evidence for a relation between exposure and childhood leukemia may be considered to be definitive at exposure levels of 3 or 4 mG or higher; evidence from some (but not all) of the other studies indicates an elevated risk at levels not greater than 2 mG /2,33/. No evidence has been reported that exposures at lower levels are 'safe', as persons with such exposures usually serve as the 'control' group. Therefore, this WHO statement fails to acknowledge the true magnitude of the problem, even when considering only childhood leukemia. The global attributable risk of childhood leukemia resulting from exposure to EMFs must be significantly greater than that calculated from consideration of only residential 50/60 Hz magnetic fields in studies having no unexposed control.

"The risk is low"

This argument is incorrect because at present, determining the magnitude of the risk is not possible. Clearly as far as EMFs are concerned, no unexposed population exists. Therefore, one can only compare groups having different levels of exposure. We can perhaps say with confidence that the elevated risk of leukemia from residential exposure of children to magnetic fields is 'low' (meaning ORs in the range of 2-4), but this does not consider the child's exposure to appliances, exposure in automobiles and at daycare or school, exposures in playgrounds, and at all the other

places that a child spends time. Even if the risk to one individual is low, the societal impact when everyone is exposed may be very significant.

In addition, the exposure assessment is grossly inadequate, even in the best of studies. Most reports deal only with either characterization of the fields within residences or with job titles in occupational settings. Some studies attempt to quantify other sources of exposure, such as the frequency of cell-phone usage or the use of other appliances, but these studies almost always do not consider residential exposure from power lines or living, working, or going to school in a WiFi building. In no investigation has it been possible to follow the exposures of a large number of people over a number of years with an accurate monitoring of total exposure to EMFs. Such a task would of course be almost impossible to do for the very good reason that as a person moves through his or her environment, the exposures vary from place to place and from moment to moment. To truly and objectively determine the risk of exposure to EMFs, however, considering residential, occupational (or school) and recreational exposures to the full range of the electromagnetic spectrum, including appliances and wireless devices is essential. This coverage has not been accomplished in any study, and without such information, determining the overall magnitude of the risk is not possible. What is possible, indeed likely, is that upon consideration of both childhood and adult diseases that the risk is not low.

"Evidence that adult to 50/60 Hz EMF exposure is insufficient"

The level of evidence definitively proving an association between exposure to EMFs and adult cancer is less strong than the relation with childhood leukemia. Multiple studies, however, show statistically significant relations between occupational exposure and leukemia in adults despite major limitations in exposure assessment. Significant elevations in the rates of leukemia

following occupational exposure to elevated EMF have been reported in review articles /34/ and in a meta-analysis /35/. Kheifets et al. /35/ report an OR of 1.18 (95% CI = 1.12-1.24) for all leukemias based on data from 38 studies, with significant elevations for both acute myelogenous (AML) and chronic lymphocytic (CLL), but with non-significant elevations in acute lymphocytic (ALL) and chronic myelogenous (CML) leukemia. Although the reported ORs are somewhat lower than those in most childhood studies, this difference may be not remarkable given the greater variety of settings in which most adults spend time with all of accompanying difficulties in evaluating total exposures. Most important, the strongest evidence for a cancer is that the same cancer (leukemia) is significantly elevated in children. Yet, considering only occupational exposure without attention to residential and recreational exposures is certain to lead to inadequate exposure assessment.

Some recent studies report similar elevations, whereas others do not. Savitz and Loomis /36/ did not find any elevation in risk of leukemia in a study of 138,905 electric utility workers. Minder and Pfluger /37/ report elevated leukemia mortality among Swiss railway employees exposed to magnetic fields (OR = 2.4, 95% CI = 1.0-6.1), whereas Harrington et al. /38/ reported no elevated rates of leukemia among UK electricity generation and transmission workers when compared with the rest of the UK population. The failure to find a relation could of course reflect the healthy-worker effect. In a 1997 review, Miller et al. /39/ reported that of 124 studies reporting odds ratios for leukemia in relation to occupations associated with electricity, 41 showed a significant elevation, and 4 showed a dose-response relation. The studies concluded that there is a reasonable relation with occupational exposure, but that occupational EMF exposure alone cannot account for the majority of leukemia cases among working men.

Feychting et al. /40/ conducted an investigation of adult leukemia in relation to exposure to

magnetic fields with consideration of combined residential and occupational exposures. The investigators found no relation between residential exposure alone with either total leukemias or any of three specific types of leukemia, and only a non-significant elevation of risk of leukemia with occupational exposure alone. Nevertheless, when both residential and occupational exposures were considered, the authors reported a significant elevation of risk of all leukemias with an OR = 3.7 (95% CI = 1.5-9.4), and significant elevations in both AML and CML, but a non-significant elevation in CLL. This study convincingly demonstrates the importance of considering exposures in multiple settings, especially both residential and occupational.

In adults, some evidence has been found for a relation between magnetic field exposure and other kinds of cancer, which is strongest for brain cancer. Kheifets et al. /41/ performed a meta-analysis of 29 reports of brain cancer and EMFs and found an OR = 1.21 (95% CI = 1.11-1.33) for all electrical workers. The authors found significant elevations for electrical engineers, welders, and power station workers. Rodvall et al. /42/ investigated glioma and meningioma in central Sweden in relation to job title, and reported only non-significant elevations of both neoplasms in relation to measured magnetic fields. Villeneuve et al. /43/ also reported only non-significant elevations in rates of all brain cancers in relation to residential exposure to magnetic fields, but found a highly significant relation among men diagnosed with glioblastoma multiforme (OR = 5.36, 95% CI = 1.16-24.78).

The evidence for a relation between EMF exposure and breast cancer is relatively strong in men /44/, and some /45-46/ but by no means all /47-49/ studies show female breast cancer also to be significantly elevated with increased exposure. Peplonska et al. /50/ recently found increased risk of breast cancer in women occupationally exposed to elevated magnetic fields. Less evidence has been published on other cancers, but Charles et al. /51/

reported that workers in the highest 10% category for EMF exposure were twice as likely to die of prostate cancer as those exposed at lower levels (OR = 2.02, 95% CI = 1.34-3.04). Villeneuve et al. /52/ report statistically significant elevations of non-Hodgkin's lymphoma in electric utility workers in relation to EMF exposure, whereas Tynes et al. /53/ report elevated rates of malignant melanoma in persons living near to high voltage power lines. Although these observations need replication, they suggest a possible relation between exposure and cancer beyond leukemia and brain cancer in adults.

The evidence for an association between ELF-EMF exposure and the neurodegenerative diseases Alzheimer's and amyotrophic lateral sclerosis (ALS) is strong. For Alzheimer's disease, Qio et al. /54/ found ORs of 2.3 (95% CI = 1.0-5.1), Feychting et al. /55/ reported ORs of 2.3 (95% CI = 1.6-3.3), and Hakansson et al. /56/ found ORs of 4.0 (95% CI = 1.4-11.7). For ALS, Savitz et al. /57/ reported ORs of 3.1 (95% CI = 1.0-9.8) and Hakansson et al. /56/ found an OR of 2.2 (95% CI = 1.0-4.7). Roosli et al. /58/ looked at neurodegenerative diseases among Swiss railway employees and reported an elevated risk for train drivers as compared with a risk of 3.15 (95% CI = 0.90-11.04) for Alzheimer's disease in station masters. For every 10 μ T years of cumulative exposure the authors found Alzheimer's disease risk to increase by 9.4% (95% CI = 2.7-16.4). No elevated risk was found for Parkinson's disease or multiple sclerosis. Garcia et al. /59/ reported a meta-analysis of EMF exposure and Alzheimer's disease. From 14 different studies they found an OR of 2.03 (95% CI = 1.38-3.00 for case-control studies, and 1.62 (95% CI = 1.16-2.80) for cohort studies. These reports show a consistent pattern of elevated risk that cannot be ignored.

In total, the scientific evidence for adult disease, especially leukemia, brain cancer, Alzheimer's disease and ALS, associated with ELF-EMF exposure is sufficiently strong that preventive steps are not only appropriate but also called for. This

conclusion is despite all the difficulties with exposure assessment. Although many possible sources of false-positive results can be found in epidemiologic studies, even more possible reasons exist for false-negative results, depending on the sample size, exposure assessment, and a variety of other confounders. Discounting the positive studies just because not every investigation shows a positive result is inappropriate. Although further research is needed with better exposure assessment and control of confounders, the evidence for a relation between ELF-EMF exposure and adult cancers/neurodegenerative diseases is sufficiently strong at present to merit preventive actions to reduce EMF exposure.

"There is little evidence that low-intensity RF fields pose human health hazards"

The thermal effects of radiofrequency radiation, including microwaves, have been studied for many years (see review by Elder /60/), and are well known to cause serious harm if exposures result in tissue heating. The important question, however, is whether adverse health effects occur at RF intensities that do not cause heating. Present international standards for exposure to RF fields are based on limited information and the questionable assumption that there are no non-thermal hazardous effects of RF radiation. That non-thermal effects occur in biological systems is clear, but the degree to which these constitute a hazard is less clear /61/.

Current thermally based RF standards are designed only to protect against acute (not chronic) exposures and protect only against thermal damage based on a six-foot man model. Because size and shape are important determinants of RF exposure, the existing public safety standards are deficient in providing protection to children and smaller adults.

The literature for health hazards from RF fields is not as extensive as that for power lines frequencies. Yet, a large body of evidence reports elevations in cancer in relation to exposure.

Szmigielski /62/ investigated cancer morbidity in Polish military personnel and found that persons occupationally exposed to RF/microwave radiation had a greater than two-fold risk of any cancer than unexposed personnel. The relations were strongest for hematopoietic cancers, which were elevated between 5.8 and 13.9 fold. Grayson /63/ reported a significant 1.39-fold elevation (95% CI = 1.01-1.90) in brain tumors in US Air Force personnel exposed to RF radiation.

Several studies have reported elevated risk of leukemia resulting from exposure to RF fields from AM and FM radio communication frequencies. Dolk et al. /64/ reported a 1.3-fold elevation in leukemia among individuals living near an FM radio transmitter in England. Michelozzi et al. /65/ found a significant dose-dependent elevation in adult and childhood leukemia among residents living near to a high-powered radio station in Rome. Park et al. /66/ investigated cancer rates in Korea in individuals living near AM radio broadcasting towers, and found significant elevations in leukemia, especially in the young (standardized mortality ratio (MMR) = 2.29, 95% CI 1.05-5.98 for 0-14 years and MMR = 2.44, 95% CI = 1.07-5.24 for 15-29 years).

Ha et al. /67/ reported on an expanded cohort of 1,928 Korean children with leukemia, 956 children with brain cancer, and 3,082 age-matched controls with respiratory illnesses. The investigators found a significant elevation in risk of leukemia for children residing within 2 km of the nearest AM radio transmitter as compared with those residing more than 20 km away (OR = 2.15, 95% CI = 1.00-4.67), but no significant relation with brain cancer. This study is consistent with the hypothesis that radiofrequency EMFs increase risks of the same diseases reported for 50/60 Hz EMFs. Because radiofrequency EMFs have higher energy than do power line frequencies, one might expect that radiofrequency EMFs would be even more likely to cause disease.

Evidence is rapidly mounting that brain tumor risk is elevated with long-term cell-phone use.

Hardell et al. /68/ first reported that on multivariate analysis, the OR for ipsilateral temporal, occipital, or temporoparietal lobe brain tumors was 2.62 (95% CI = 1.02-6.71), whereas no elevation in risk was found for the contralateral brain (OR = 0.97, 95% CI = 0.36-2.59). Later, Hardell et al. /69/ found that individuals using analog cell phones had a greater than eightfold increased risk of developing brain tumors, and with cordless phone usage, the increased risk was more than fourfold. Additionally, Lonn et al. /70/ found an increased risk of acoustic neuroma (a form of brain cancer) among persons in Sweden who had been using a cell phone for 10 years or more.

Results are beginning to appear from the European INTERPHONE study, and although not complete as yet, both the German /71-72/ and the French /73/ preliminary reports present at least a suggestion of an elevation in rates of some forms of brain cancer and acoustic tumors among individuals who are the heaviest and longest duration users of cell phones. Schoemaker et al. /74/ reported on mobile phone use in a case-control study in five North European countries, and found that risk of acoustic neuroma on the same side of the head as reported phone use was raised for use for 10 years or longer (OR = 1.8, 95% CI = 1.1-3.1). Lahkola et al. /75/ reported on a similar study but focused on glioma. The authors report an OR of 1.39 (95% CI = 1.01-1.92, *p* for trend 0.04) for mobile phone use on the same side of the head, but no significant elevation in the contralateral hemisphere. In neither the Schoemaker /74/ nor the Lahkola /75/ studies was there a significant increase in overall risk of acoustic neuroma or glioma based simply on the use of a mobile phone. An Israeli component of the INTERPHONE study has reported a significant and dose-dependent elevation in the development of parotid gland tumors on the ipsilateral side (OR 1.58, 95% CI = 1.11-2.24), but no relation with contralateral tumors /76/. Other large studies, however, have not detected any relation between either brain cancer /77-78/ or acoustic neuroma and mobile phone use.

/77/. Some researchers who did not find a relation have noted that cell-phone usage is sufficiently recent such that concluding that long-term exposure is without hazard is not possible (cf. /77/).

Kundi et al. /79/ summarized the results of nine different human epidemiologic studies made in another recent review by ICNIPT et al. /80/, which points out that not all human studies are consistent, and that so many deficiencies are present in the studies conducted to date that one cannot rule out an association between exposure and cancer.

Recently a meta-analysis was published that focused on cell phone use and cancer. Hardell et al. /81/ examined 2 cohort and 16 case-control studies. Nine of the case-control studies were of cases with a latency period of greater than 10 years, but most of these included few cases. The risk of glioma was estimated to be 1.2 (95% CI = 0.8-1.9), and increased to 2.1 (95% CI = 1.2-3.4) for ipsilateral use. Acoustic neuroma risk was estimated to be 1.3 (95% CI = 0.6-2.8), increasing to 2.4 (95% CI = 1.1-5.3) for ipsilateral use. The enormous and very recent increase in the use of cell phones by children is particularly worrisome. Inadequate information is available at present concerning the long-term consequences of cell phone use, especially by children, but the reports cited above suggest that the risk of brain tumors and acoustic neurons is significant. Should further study confirm these relations, we may be facing an epidemic of disease resulting from cell-phone usage. Because the latency for developing such diseases is long, this situation is of particular concern, especially for children.

A number of human studies of biological effects other than cancer associated with RF fields have been reported, as well as a number of studies not finding such effects. Huber et al. /82/ showed that human exposure to digital radiotelephone handsets affects brain physiology in young healthy male subjects, modifying their electroencephalogram during subsequent sleep. Koivisto et al. /83/ reported that exposure to 902 MHz fields actually

accelerates simple reaction times in human participants. A number of other biological effects that are not believed to be secondary to thermal changes have been reported. Such effects include increase spontaneous abortion, shifts in red and white blood cell counts, increased mutations in lymphocytes (see /84/), and changes in brainwave activity /85-86/. Seitz et al. /87/ reviewed studies of electromagnetic hypersensitivity and subjective health complaints associated with EMF exposure and concluded that such effects are not proven, but that at present, long-term effects of impaired well-being also cannot be excluded. Three recent reports suggest a relation between cell-phone use and reduced male fertility /88-90/. Further studies are needed to determine whether significant effects of RF fields affect both nervous system function and fertility, but with careful exposure assessment and adequate concern for confounders.

Divan et al. /91/ reported that prenatal and postnatal exposure of children to cell-phone frequencies was associated with a significant increase in behavioral problems of emotion and hyperactivity around the age of school entry (OR = 1.80, 95% CI = 1.45-2.23). Although the results need replication, they point to an elevated susceptibility of the fetus and young children and suggest a variety of adverse effects of cell-phone frequencies beyond just cancer.

Although these studies do not provide the same level of proof found in the studies of power line frequencies, they most certainly **do not** allow one to conclude that RF exposures are safe.

"There is no animal evidence"

It is correct to say that no adequate animal model system is available that reproducibly demonstrates the development of cancer in response to exposure to EMFs at the various frequencies of concern. McCann et al. /92/ reviewed the animal studies, and whereas the authors found most studies to be negative, several

showed suggestive positive results. The investigators also clearly identified issues that must be improved in further animal carcinogenesis research. Kheifets et al. /93/, however, in a policy review noted that,

"...even consistent negative toxicological data cannot completely overcome consistent epidemiological studies. First, a good animal model for childhood leukemia has been lacking. Second, particularly for ELF, the complex exposures that humans encounter on a daily basis and a lack of understanding of the biologically relevant exposure calls into question the relevance of exposures applied in toxicology. Another limitation of toxicologic studies is that animals cannot be exposed to fields that are orders of magnitude more powerful than those encountered by humans, decreasing their power to detect small risks."

Further, they conclude that,

"(A)lthough the body of evidence is always considered as a whole, based on the weight of evidence approach and incorporating different lines of scientific enquiry, epidemiologic evidence, as most relevant, is given the greatest weight."

More striking is the report from Denver, Colorado, using the wire-code characterization originally developed by Wertheimer and Leeper /1/ showing that pet dogs living in homes that are characterized as having high or very high wire codes, as compared with those with low or very low wire codes or buried power lines, showed a OR of 1.8 (95% CI = 0.9-3.4) for developing lymphoma after adjustment for potential confounders, whereas dogs that lived in homes with very high wire codes had an OR of 6.8 (95% CI = 1.6-28.5) /94/. This study is impressive because the exposure of the dogs reflects the environment in

which exposure has been associated with elevated risk of human cancer in two independent investigations /1,2/.

One positive animal study is that by Rapacholi et al. /95/, who demonstrated that lymphoma-prone transgenic mice developed significantly more lymphomas after exposure to 900 MHz fields (lymphoma being the animal equivalent of human leukemia) than did unexposed animals. Utteridge et al. /96/, however, were not able to replicate this observation, although their exposures were not identical.

Salford et al. /97/ reported that low power RF fields, below that which caused thermal effects, increase the leakage of protein from the blood-brain barrier, and they later found that this resulted in direct damage to nerve cells from microwaves from a GSM mobile phone /98/. Tattersall et al. /99/ found that RF field applications below the level that causes heating resulted in changes in the electrical activity of brain slices, suggesting that such fields can alter nervous system function. Wang and Lai /100/ reported altered performance of rats in learning tasks exposed to 2450-MHz microwaves.

Curiously, in many legal situations the courts are reluctant to accept evidence that a chemical substance causes cancer in animals without corresponding evidence in humans. In the case of EMFs, we have strong evidence that magnetic fields cause cancer in humans, but much less evidence from animal models. The US Supreme Court /101/, in the case of *Daubert vs. Merrell Dow Pharmaceuticals*, effectively ruled that animal studies were not relevant to human health, and that the only admissible evidence must be from human epidemiologic studies! Although this is certainly not a justifiable conclusion, the situation with regard to EMF health effects is that we have strong evidence for human cancer from epidemiologic studies but do not have good evidence for cancer in experimental animals. Yet, humans are what we should be concerned about, not laboratory rats!

"We do not know a mechanism"

We do not know the mechanism of cancer in general, although we do know a lot about cancer. It came as a major surprise to most scientists when Lichtenstein et al. /102/ reported that genetic factors play a minor role in causing most types of cancer because it had been commonly assumed that genetics was the major cause. Yet, Lichtenstein et al. concluded from their study of identical twins that environmental factors were the initiating event in the majority of cancers. This finding does not of course mean that genetic susceptibility to environmental contaminants is unimportant, but rather that genetic factors alone do not result in cancer in most cases. We know the mechanisms of action for certain carcinogenic substances, but for most cancers, we know neither the environmental trigger nor the mechanism of action. Thus, there is no reason to negate the evidence that EMFs cause cancer just because we do not know a single mechanism to explain its mode of action. Whether magnetic fields actually cause childhood leukemia, or whether some other component in the electromagnetic environment is responsible for the association, is a subject of debate within the scientific community, but from a public health point of view, this controversy does not matter.

We do not know the mechanism or cause for the development of Alzheimer's disease or ALS. We do know that both are more common in individuals in certain occupations and that exposure to certain metals is associated with increased risk /103-104/. In the case of Alzheimer's disease, abnormalities of amyloid β and the tau protein have been found /105/, but the understanding of why or how they form is very limited. Neither the association with metals nor the presence of abnormal proteins constitutes a mechanism for the cause of these diseases. So, rather than discounting the relation between EMF exposure and neurodegenerative diseases, we should be using this information as a tool to better understand the etiology of these diseases.

Clear evidence has emerged from animal and cell culture studies that ELF and RFR have biological effects. Furthermore, such effects occur at intensities commonly experienced by humans. We know a number of ways in which EMFs alter cell physiology and function. Electromagnetic fields affect gene transcription /106-110/, induce the synthesis of stress proteins /111/, and cause breakage of DNA /112/, probably through the generation of reactive oxygen species /113-114/. Changes in the blood-brain-barrier and in calcium metabolism have been demonstrated for various RF frequencies (see review by Lai /115/), and such effects occur at exposures that do not cause significant heating. Any one of these actions might be responsible for the carcinogenic and/or neurodegenerative actions of EMFs. As with many environmental agents, however, assuming that only one target or mechanism of action exists would be a mistake. For example, it is unlikely that the mechanisms causing effects on the nervous system and behavior are secondary to the same as those leading to cancer. More likely is that multiple mechanisms of action are in force leading to disease. Yet, the lack of complete understanding of basic mechanisms does not alter the importance of the relations.

LEVELS OF PROOF AND STANDARDS OF EVIDENCE FOR DECISION-MAKING DIFFER AMONG PROFESSIONS

The levels of proof that are required for general acceptance vary among the disciplines. The level of proof that should trigger a public health response does not, and should not, require the same level of proof as that required for proof of a mathematical theorem or a basic principle in biology. The principal reason that the levels of proof are different in these situations is that in the case of public health, an enormous cost, in terms of human life lost, in doing nothing could be involved.

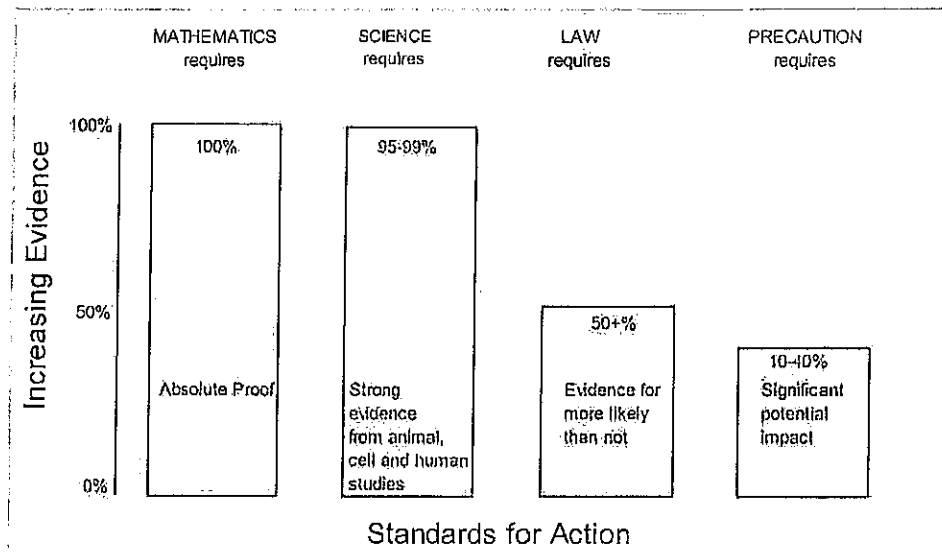


Fig. 2: Standards of evidence for decision-making, as used in mathematical proof, scientific investigations, legal and public health.

Evaluating the strengths of scientific evidence on public health and environmental hazards requires that these differences are recognized and transparent. Decision-makers and the public will be best served by a clear recognition that differing standards of evidence and levels of proof are expected and justified, and making them explicit in review processes is essential.

These differing standards reflect both the standards of the different professions and the training given to students of different disciplines. The consequences of these differences are of paramount importance in understanding why different 'experts' can arrive at apparently opposite conclusions when reviewing the same body of evidence. Such experts will differ in their judgments about when the evidence justifies drawing conclusions, what degree of evidentiary proof is sufficient to do so, and what actions might be justified at any point on that information continuum. This approach, however, creates confusion, during which different expert panels

reviewing the same body of evidence may well come to diametrically opposed opinions about whether sufficient information is available at a point in time to reasonably link cause to effect.

Figure 2 shows that at least four standards of evidence are accepted as levels of proof or requirements for action in different professions. The following discussion is presented to highlight several of the main differences in the professional approach and traditional ways of viewing and interpreting scientific evidence. The most rigorous is mathematical proof, which constitutes proof at 100% confidence. This level is the standard in mathematics, physics, and chemistry but is a level of proof that in almost every situation cannot be achieved in biology and medicine.

The level of proof used by the biology and medical scientific community is that the associations from experimental animal and cell studies and from human epidemiologic studies are established such that no more than a 5% possibility remains that the results could be due to chance.

This possibility is called the 95% confidence interval, or even better the 99% confidence interval, at which no more than a 1% possibility remains that the results are due to chance. This level is the accepted standard of proof of association (not necessarily of causation) in laboratory and epidemiologic studies, and when achieved, the results are concluded to be 'statistically significant'. We expect that all possible evidence (animal, cell, and epidemiologic studies, with replications) will show a high degree of consistency.

In human epidemiologic studies, the Hill Criteria are important factors for consideration. These Criteria were suggested by Sir Bradford Hill in a lecture in 1965 [116]. The Hill Criteria are important when one attempts to go beyond 'association' to 'causation'. Although some insist that each of the 'criteria' must be met to assign causation, understanding how Hill introduced these considerations is important. The Hill Criteria are listed in the sidebar, together with quotes from his article. Clearly, Hill did not believe that each consideration had to be met before concluding that a relation exists between exposure and disease. Rather, he meant these considerations to be the factors that are considered in determining the 'weight of evidence'. The concept of weight of evidence is very important, and is basically what the Hill Criteria are about—dealing with the strength of association, how similar the findings are from different studies, how strong the evidence is that more is worse, and how well the studies in different model systems provide consistent results. The Hill Criteria provide a framework for taking action when the weight of evidence indicates a relation between exposure and disease, even when some unknowns remain.

When evaluating the findings of statistically significant relations between EMF exposure and disease in relation to the considerations outlined by Hill, the evidence for leukemia in children is sufficiently strong to meet the criteria. The associations of disease with adult leukemia and brain tumors and for the neurodegenerative diseases

Alzheimer's disease and ALS is certainly less extensive, but still sufficient to meet most of the criteria. The evidence for the adverse effects of RF exposure, although growing rapidly, is not as complete but is still strongly suggestive. Thus, the question remains of how to deal with evidence that is incomplete, but for which the public health impact is potentially great.

The legal profession looks at the burden of proof and standards for judging the evidence in a far different way. The level of proof that is the standard applied in civil legal proceedings is 'more likely than not'. In other words, if there is a 50%+ likelihood of harm, then this level is taken as evidence for a relation, as shown in Figure 2. It is not necessary that the evidence of harm be conclusive, neither is some uncertainty of causation a reason to conclude that no relation exists between exposure and harm. In fact, a certain amount of uncertainty is allowable, even under the more stringent (criminal) standard of evidence, namely "beyond a reasonable doubt". No legal standard requires complete certainty of effect to make a defensible judgment on the evidence at hand. The level of certainty about an effect that is sufficient to take action (in this case to decide the admissibility of evidence or the outcome of a court trial) can be lower than a strictly scientific determination on causality. Important social issues must often be decided based on uncertain scientific evidence. This level of evidence has been more than reached for the association between prolonged and frequent use of cell phones and increased risk of ipsilateral brain tumors, acoustic neuromas, and parotid gland tumors.

Prudent public health policy requires yet a different approach to standards of evidence, based on precaution (far right bar in Figure 2). A large difference can be seen between what constitutes causal evidence for purposes of achieving scientific consensus, what constitutes "a more likely than not" case under the law, and what constitutes sufficient evidence for purposes of interim public health policy. The demonstration of a low level of proof of

The Hill Criteria, as presented by Sir Bradford Hill:

1. *Strength of the Association:* He indicates that a strong association is an important consideration, but comments "In thus putting emphasis upon the strength of an association we must, nevertheless, look at the obverse of the coin. We must not be too ready to dismiss a cause-and-effect hypothesis merely on the grounds that the observed association appears to be slight. There are many occasions in medicine when this is in truth so. Relatively few persons harboring the meningococcus fall sick of meningococcal meningitis. Relatively few persons occupationally exposed to rat's urine contract Weil's disease." Thus while strength of the association is an important consideration, it must be placed in context. With regard to health hazards from EMF it is true that in most studies the odd ratios are relatively low, often in the range of 1.5-3.0. But the consistency with which elevated and statistically significant ORs are found is the important consideration, particularly in light of the inadequacy of exposure assessment.
2. *Consistency:* This means that different studies get the same results. But again, Hill cautions "I would myself put a good deal of weight upon similar results reached in quite different ways, e.g., prospectively and retrospectively. Once again looking at the obverse of the coin there will be occasions when repetition is absent or impossible and yet we should not hesitate to draw conclusions." Thus, one does not need to demonstrate a statistically significant relation in every study, especially given the problem with exposure assessment.
3. *Specificity:* Specificity is to say that the effect is due to the specific exposure. He concludes, "We must also keep in mind that diseases may have more than one cause. It has always been possible to acquire a cancer of the scrotum without sweeping chimneys or taking to mule-spinning in Lancashire. One-to-one relations are not frequent. Indeed I believe that multi-causation is generally more likely than single causation though possibly if we knew all the answers we might get back to a single factor. In short, if specificity exists we may be able to draw conclusions without hesitation; if it is not apparent, we are not thereby necessarily left sitting irresolutely on the fence."
4. *Temporality:* Temporality refers to the time relation between exposure and disease. But this is often difficult to determine. Hill states, "This is a question which might be particularly relevant with diseases of slow development. Does a particular diet lead to disease or do the early stages of the disease lead to those peculiar dietetic habits?" The issue of brain tumors and acoustic neurons from cell phone use is a perfect example of the problem with diseases with a long latency.
5. *Dose-Response Relation* (which Hill calls "biological gradient"): Finding a dose-response relation is often considered a key factor in any toxicologic investigation. But Hill cautions, "Often the difficulty is to secure some satisfactory quantitative measure of the environment which will permit us to explore this dose-response. But we should invariably seek it." Thus, lack of a dose-response relation does not destroy a causal connection.
6. *Plausibility:* Hill notes, "It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day." This consideration is particularly relevant to EMF considerations.
7. *Coherence:* Coherence means that there should not be serious conflict between known facts of the disease under consideration. Hill discusses coherence in relation to smoking and lung cancer and says "Personally, I regard as greatly contributing to coherence the histopathological evidence from the bronchial epithelium of smokers and the isolation from cigarette smoke of factors carcinogenic for the skin of laboratory animals. Nevertheless, while such laboratory evidence can enormously strengthen the hypothesis and, indeed, may determine the actual causative agent, the lack of such evidence cannot nullify the epidemiological observations in man. Arsenic can undoubtedly cause cancer of the skin in man but it has never been possible to demonstrate such an effect on any other animal." Again, this consideration is directly relevant to the issue of a lack of an animal model for EMF-induced leukemia.

an environmental hazard, especially if there is a potential for a significant public health impact, should warrant preventative action and mitigation of impacts. Furthermore, the threshold for and the degree of action should vary with the magnitude of the potential impact on human health.

A central confusion in this debate has been whether prudent environmental policy and public health decisions necessarily require conclusive scientific evidence to first be demonstrated. We do not believe that this is the case. The state of the science needs to be presented in an understandable and scientifically accurate manner, but prudent public health actions do not and should not require proof of harm at any level described above. When some evidence for danger that may lead to significant harm is reported, taking preventative actions and implementing policies that are protective of public health, safety, and welfare rather than waiting for absolute scientific certainty may be essential.

ELECTROMAGNETIC FIELDS AND PUBLIC HEALTH STANDARDS OF EVIDENCE

In the case of EMF, where everyone is exposed, the societal implications may be huge if a real risk exists whose magnitude has simply not yet been clarified. For several of the major health effects discussed above (childhood and adult leukemia, Alzheimer's, and ALS) the degree of evidence of serious disease resulting from exposure is sufficient to merit action on the basis of traditional scientific criteria. For many other possible health outcomes (health effects of exposure to RF, EMF, electrosensitivity), the results are less certain. Public policies are needed to address the issue of decision-making in the face of this scientific uncertainty, especially when the potential for a significant impact on the health of the public is high. What should the public policy be when the level of certainty (10% to 40%) is relatively low? How should the lack of an unexposed population

be factored into the decision? What should policy be when one of the major concerns is the exposure of children, who currently often spend hours per day text messaging or chatting on a cell phone?

The landmark publication "Late Lessons from Early Warnings: The Precautionary Principle 1896-2000" /117/ has given a roadmap to those who wish to make more informed decisions about "when there is enough information to act" on environmental and health issues which, if ignored, could result in costly consequences. Future decision-makers have to balance the costs of being too restrictive with the costs of being too permissive. If problems are identified early, but questions still exist about possible risks, then identifying reasonable actions that are precautionary and proportionate is necessary. Choosing which actions to take depends upon the level of proof and on the size, nature, complexity, and distribution of the costs of being wrong (Figure 3).

"The level of proof depends on the size and nature of the potential harm, the claimed benefits, the available alternatives, and the potential costs of being wrong in both directions, i.e., of acting or not acting in the context of uncertainty, ignorance and high stakes." (page 193) "The goals of science and public policy-making on health and environmental hazards are different: science puts a greater priority on avoiding "false positives" by accepting only very high levels of proof of "causality", whereas public policy tries to prioritize the avoidance of "false negatives" on the basis of a sufficiency of evidence of potential harm."

The Precautionary Principle as encoded by the European Environmental Agency /117/ is a roadmap for decision-making. It describes how varying levels of scientific evidence (from scant to causal) can be interpreted in choosing appropriate levels of action that are based on the level of certainty or uncertainty involving risks. Both prevention and precaution are included as key principles in the European Treaty.

Table 1: Late lessons from early warnings: Table on evidence

Situation	State and dates of knowledge	Examples of action
Risk	'Known' impacts; 'known' probabilities; for example, asbestos	Prevention: action taken to reduce known hazards; for example, eliminating exposure to asbestos dust
Uncertainty	'Known' impacts; 'unknown' probabilities; for example, antibiotics in animal feed and associated human resistance to those antibiotics	Precautionary prevention: action taken to reduce exposure to potential hazards
Ignorance	'Unknown' impacts and therefore 'unknown' probabilities; for example, the 'surprises' of chlorofluorocarbons (CFCs), pre-1974.	Precaution: action taken to anticipate, identify and reduce the impact of 'surprises'

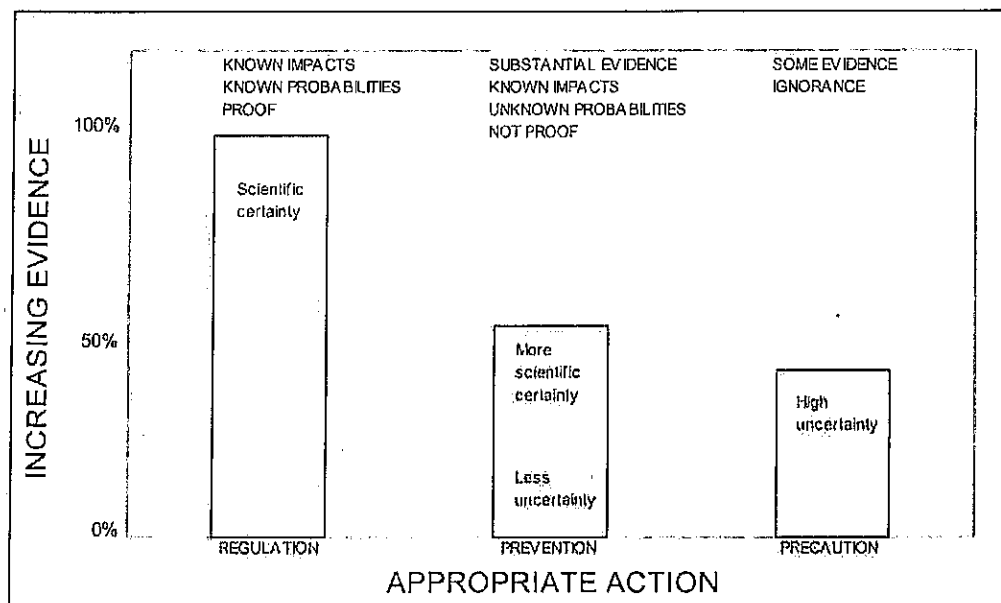
**Fig. 3:** Standards of evidence for precautionary/preventative action

Table 1 clarifies the basis on which these terms can be applied to take "appropriate precautionary actions to avoid serious threats to health or environments" /117/. How one determines a reasonable, proportionate, and defensible level of action depends on what evidence is available, how high the level of ignorance is about potential factors and outcomes, and what if anything, can be deduced about probabilities of risk. Factors that influence the level of precautionary or preventative action, or regulation, include the following:

- the costs (health, societal, economic, technological);
- the probable consequence of taking no action at all;
- how large an effect could occur;
- the populations potentially at risk;
- the nature, acceptability, and irreversibility of potential impacts; and
- the ethics of doing nothing in light of evidence of harm.

Preventative action is a clear and defensible choice for some level of action, when waiting for scientific proof might put millions at risk of a dread disease that could be avoided by simple education, by behavioral changes, or by the use of technology, as shown diagrammatically in Figure 3.

The Rio Declaration, coming from the 1992 Convention of the United Nations Environment Programme /17/ lists the following as Principle 15:

"In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation."

This concept was further developed at a Wingspread Conference in 1998 /118/, which

defined this principle as

"When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relations are not fully established scientifically. In this context the proponent of an activity, rather than the public, should bear the burden of proof. The process of applying the Precautionary Principle must be open, informed, and democratic and must include potentially affected parties. The Precautionary Principle must also involve an examination of the full range of alternatives, including no action."

The Precautionary Principle does not include a focus on economic factors, but rather implies that caution should be exercised in all decisions affecting human exposures. This principle is a formulation of the things your mother taught you—"An ounce of prevention is worth a pound of cure."

In the case of power-frequency and RF EMF, the sheer numbers of people who are at risk makes the wise handling of this issue a health policy imperative. In the face of inadequate evidence, the public health response should be proportional to the potential public health impact (Figure 4). It may be true that the risk to any one individual is not great, but from a societal perspective, the impact of exposure may be very large. Proof of harm should not be a pre-condition for taking action when the potential health impact is huge. What decision-makers need to address is what standard of evidence is appropriate now to guide them with respect to EMF exposures that are clearly of environmental and public health concern. The prudent approach from a public health point of view is to take preventive actions as if causation had been proven, while at the same time to continue to search for mechanisms of action. The fact that there are unknowns does not negate or override the ultimate public health responsibility, which is to protect the population from exposures that cause disease.

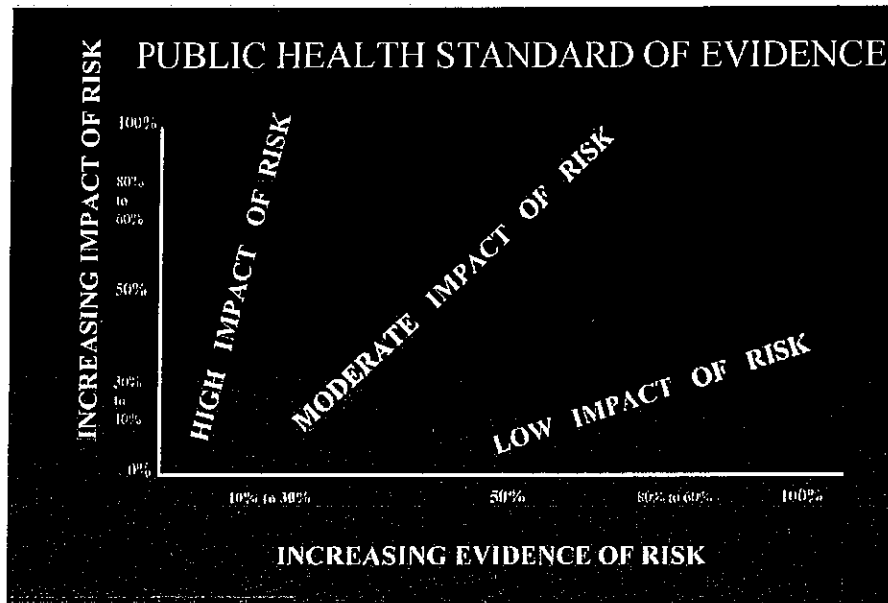


Fig. 4: A public health-based response must be relative to the magnitude of the potential impact of inaction. When the potential impact is high, action should be taken even when the evidence of risk is low.

DEFINING NEW EXPOSURE STANDARDS FOR ELF AND RF ELECTROMAGNETIC FIELDS BASED ON THE PRECAUTIONARY PRINCIPLE

The most contentious issue regarding public and occupational exposures to ELF involves the resolute adherence by many countries to the existing International Commission on Non-Ionizing Radiation Protection (ICNIRP) standards /119/ of 1,000 mG (100 μ T), in face of the growing scientific evidence of health risks at far lower levels. The basis on which most standard setting agencies justify their failure to set new safety limits for ELF and RF is nearly always that no certain proof of harm from exposure and no known mechanism of action have been presented. A demand for a causal level of evidence and scientific certainty is implicit in nearly all discussion on what are the appropriate safety standards for ELF and RF. This demand, however, runs counter to both the existing scientific evidence and good public health practice.

Two obvious factors work against governments

taking action to set exposure guidelines based on current scientific evidence of risk:

- Contemporary societies are very dependent upon electricity usage and RF communications, and anything that restricts current and future usage potentially has serious economic consequences.
- Power and communications industries have enormous political clout, and even provide support for a significant fraction of the research done on EMF.

This state of affairs results in legislation that protects the status quo and scientific publications whose conclusions are not always based only on the observations of the research. This situation also hinders wise public health policy actions and the implementation of prevention strategies because of the huge financial investments already made in these technologies. Huss et al. /120/ analyzed 59 studies of the health effects of cell phone use and found that studies funded exclusively by industry

were least likely to report a statistically significant result.

Substantial evidence indicates that ELF is carcinogenic at levels of exposure in the 2 mG to 5 mG (0.2-0.5 μ T) range and above. ICNIRP and other standards that place public exposure limits as high as 1,000 mG (100 μ T) are outdated and should be replaced, based on the evidence presented above. New standards are warranted now, based on the totality of scientific evidence, the risks of taking no-action, the large population at risk, the costs associated with ignoring the problem in new and upgraded site selection and construction, and the loss of public trust by ignoring the problem. New exposure limits must be developed for ELF-EMF based on the clear sufficiency of evidence for carcinogenicity to humans at levels that are routinely approved today for occupancy by children, pregnant women, and others. To wait any longer to adopt new public safety limits for ELF is not prudent public health policy. Such limits should reflect the exposures that are commonly associated with increased risk of childhood leukemia (in the 2 to 5 mG (0.2-0.5 μ T) range for all children, and over 1.4 mG (0.14 μ T) for children age 6 and younger.

Defining a new exposure standard for RF is complex, if we are to address properly new scientific results for chronic exposure to pulsed radiofrequency (for example from cell towers, cell phones, and other wireless technologies). Whereas the evidence of serious harm is strong, knowledge regarding the relation between cumulative exposure and risk of disease is inadequate. Uncertainty about how low such standards might have to go to be prudent from a public health standpoint should not prevent reasonable efforts to respond to the information at hand. No lower limit for bio-effects and adverse health effects from RF have been established, and no assertion of safety at any level of wireless exposure (chronic exposure) can be made at this time. A major concern is the exposure of children. We strongly recommend that wired alternatives to WI-FI be implemented particularly

in schools and libraries so that children will not be subjected to elevated RF levels until more is understood about possible health impacts.

The Bioinitiative Report /121/ presents a much more extensive and exhaustive discussion of the literature on health effects of both ELF and RF EMF than can be presented here. The Report contains a recommendation of an RF standard of 0.1 μ W/cm², but with the full knowledge that hazards may be associated with even lower exposures.

This review has focused on those diseases for which the evidence of increased risk with EMF exposure is the strongest. Other biological effects and potential health outcomes are presented in detail in the Bioinitiative Report /121/. The effects that drive the need for immediate action in lowering exposure are cancer and neurodegenerative diseases. Leukemia appears the cancer of greatest concern when the exposure to either ELF or RF is over the whole body, as is the case with most ELF exposures and exposure from RF towers. When exposure is focused on a part of the human body, such as is the case of the head in cell phone use, one sees cancers of the brain, acoustic nerve, or parotid gland. For these diseases, the evidence is clearly sufficient to warrant regulatory changes in public safety limits now, at levels that are widely reported to be associated with increased risk of childhood leukemia and brain tumors. Exposure limits against these diseases will also likely be protective for other less-well-defined health impacts. The BioInitiative Report /121/ provides additional justification for the adoption of these levels to prevent the health hazards resulting from exposure to ELF and RF.

CONCLUSIONS

The evidence for hazards to human health from both ELF and RF EMF is sufficiently strong as to merit immediate steps to reduce exposure. Such a reduction can best be achieved by setting exposure goals that are lower than levels known to be

associated with disease, even while understanding that these exposure goals are significantly lower than many current exposures. A reasonable approach would be a 1 mG (0.1 μ T) planning limit for structures adjacent to all new or upgraded power lines, and for occupied space that affects sensitive receptors (homes, schools, day-care, pre-school, etc), and targets not to exceed 2 mG (0.2 μ T) for all other occupied new construction. Although reconstructing all existing electrical distributions systems is not realistic, steps to reduce exposure from these existing systems should be encouraged. For RF EMF, setting a level with certainty is difficult. A precautionary action level would reasonably be 0.1 μ W/cm².

The proposals presented here reflect the evidence that a positive assertion of safety cannot be made with respect to chronic exposure to low-intensity levels of ELF and RF radiation.

As with many other standards for environmental exposures, even these proposed limits may not be completely protective, but more-stringent standards are not realistic at the present time.

REFERENCES

1. Wertheimer N and Leeper E. Electrical wiring configurations and childhood cancer. *Am J Epidemiol* 1979;109:273-84.
2. Savitz DA, Wachtel H, Barnes FA, John EM, Tvrdik JG. Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. *Am J Epidemiol* 1988;128:21-38.
3. London SJ, Thomas DC, Bowman JD, Sobel E, Cheng TC, Peters JM. Exposure to residential electric and magnetic fields and risk of childhood leukemia. *Am J Epidemiol* 1991;134:923-37.
4. Feychting M, Ahlbom A. Magnetic fields and cancer in children residing near Swedish high-voltage power lines. *Am J Epidemiol* 1993; 138: 467-81.
5. Linet MS, Hatch EE, Kleinerman RA, Robison LL, Kaune WT, Friedman DR et al. Residential exposure to magnetic fields and acute lymphoblastic leukemia in children. *N Engl J Med* 1997;337:1-7.
6. Draper G, Vincent T, Knoll ME, Swanson J. Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study. *BMJ* 2005;330:1290-3.
7. Clydesdale GJ, Dandie GW, Muller HK. Ultra-violet light induced injury: Immunological and inflammatory effects. *Immunol Cell Biol* 2001;79: 547-68.
8. US National Academy of Science, National Research Council. Possible health effects of exposure to residential electric and magnetic fields. Washington, DC, National Academy Press, 1997.
9. US National Academy of Sciences, National Research Council. Identification of research needs relating to potential biological or adverse health effects of wireless communication devices. Washington, D.C., National Academy Press, 2008.
10. National Institute of Environmental Health Sciences. Health effects from exposure to power-line frequency electric and magnetic fields, 1999.
11. European Commission, Health, and Consumer Protection. Scientific Committee on SCENIHR Report on emerging and newly identified health risks—possible effects of electromagnetic fields (EMF) on Human Health, 2007.
12. World Health Organization (WHO). Extremely low frequency fields. *Environmental Health Criteria*, Volume 238. Geneva: WHO, 20074.
13. US Supreme Court. Maria Gonzalez, individually and as mother and legal guardian of her daughters Tara Gonzalez (age 14) and Nicole Gonzalez (age 8). No. 06-175, 2006.
14. European Commission. Communication from the Commission on the Precautionary Principle; COM 1, Brussels, 2000.
15. European Commission. European Treaty 174, 2002. Available at: http://www.law.harvard.edu/library/services/research/guides/international/eu/eu_legal_research_treaties.php.
16. Gee D. Late lessons from early warnings: Toward realism and precaution with endocrine-disrupting substances. *Environ Health Perspect* 2006;114 (Suppl 1):152-160.
17. United Nations Environment Programme. The Rio Declaration on Environment and Development, 1992.
18. Wartenberg D. Residential magnetic fields and childhood leukemia: A meta-analysis. *Am J Public Health* 1998;88:1787-94.
19. Greenland S, Sheppard AR, Kaune WT, Poole C,

- Kelsh MA and the Childhood leukemia-EMF study group. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. *Epidemiology* 2000;11:624-34.
20. Ahlbom A, Day N, Feychting M, Roman E, Skinner J, Docterty J, et al. A pooled analysis of magnetic fields and childhood leukaemia. *Br J Cancer* 2000;83:692-8.
 21. Mejia-Arangure JM, Fajardo-Gutierrez A, Perez-Saldivar ML, Gorodezky C, Martinez-Avalos A, Romero-Guzman L, et al. Magnetic fields and acute leukaemia in children with Down syndrome. *Epidemiology* 2007;18:158-61.
 22. Foliat DE, Pollock BH, Mezei G, Iriye R, Silva JM, Epi KL, et al. Magnetic field exposure and long-term survival among children with leukemia. *Brit J Cancer* 2006;94:161-64.
 23. Svendsen AL, Weithkoph T, Kaatsch P, Schuz J. Exposure to magnetic fields and survival after diagnosis of childhood leukemia: a German cohort study. *Cancer Epidemiol Biomark Prev* 2007;16:1167-71.
 24. Lowenthal RM, Tuck DM, Bray IC. Residential exposure to electric power transmission lines and risk of lymphoproliferative and myeloproliferative disorders: a case-control study. *Int Med J* 2007; 37(9):614-9.
 25. Infante-Rivard C, Deadman JE. Maternal occupational exposure to extremely low frequency magnetic fields during pregnancy and childhood leukemia. *Epidemiology* 2003;14:437-41.
 26. Makri A, Goveia M, Balbus J, Parkin R. Children's susceptibility to chemicals: A review by developmental stage. *J Toxicol Environ Health B* 2004;7:417-35.
 27. Preston RJ. Children as a sensitive subpopulation for the risk assessment process. *Toxicol Appl Pharmacol* 2004;199:132-41.
 28. Carpenter DO, Ahlbom A. Power lines and cancer: Public health and policy implications. *Forum Appl Res Pub Policy*, 1988;Winter, 96-101.
 29. Milham S, Osslander EM. Historical evidence that residential electrification caused the emergence of the childhood leukemia peak. *Med Hypotheses* 2001;56:290-5.
 30. Kheifets L, Afifi AA, Shinkhada R. Public health impact of extremely low-frequency electromagnetic fields. *Environ Health Perspect* 2006; 114:1532-7.
 31. Savitz DA, John EM, Kleckner RC. Magnetic field exposure from electric appliances and childhood cancer. *Am J Epidemiol* 1990;131:763-73.
 32. Hatch EE, Linet MS, Kleinerman RA, Tarone RE, Severson RK, Hartsock CT, et al. Association between childhood acute lymphoblastic leukemia and use of electrical appliances during pregnancy and childhood. *Epidemiology* 1998;9:234-45.
 33. Green L. Childhood leukemia and EMF. *Cancer Causes Control* 1999;10:233-43.
 34. Savitz DA, Ahlbom A. Epidemiologic evidence on cancer in relation to residential and occupational exposure. In: Carpenter DO, Ayrapetyan A, eds. *Biological effects of electric and magnetic fields: beneficial and harmful effects*. New York, NY: Academic Press, 1994;233-61.
 35. Kheifets LJ, Afifi AA, Buffler PA, Zhang ZW, Mastkin CC. Occupational electric and magnetic field exposure and leukemia. *JOEM* 1997;39: 1075-1091.
 36. Savitz DA, Loomis DP. Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers. *Am J Epidemiol* 1995;141:123-34.
 37. Minder CE, Pfluger DH. Leukemia, brain tumors and exposure to extremely low frequency electromagnetic fields in Swiss railway employees. *Am J Epidemiol* 2001;153:825-35.
 38. Harrington JM, Nichols L, Sorahan T, van Tongeren M. Leukaemia mortality in relation to magnetic field exposure: findings from a study of United Kingdom electricity generation and transmission workers, 1973-97. *Occup Environ Med* 2001; 58:307-14.
 39. Miller RD, Neuberger JS, Gerald KB. Brain cancer and leukemia and exposure to power-frequency (50- to 60-HZ) electric and magnetic fields. *Epidemiol Rev* 1997;19:273-93.
 40. Feychting M, Forssen U, Floderus B. Occupational and residential magnetic field exposure and leukemia and central nervous system tumors. *Epidemiology* 1997;8:384-9.
 41. Kheifets LJ, Afifi AA, Buffler PA, Zhang ZW. Occupational electric and magnetic field exposure and brain cancer: A meta-analysis. *JOEM* 1995; 37:1327-1340.
 42. Rodvall Y, Ahlbom A, Stenlund C, Preston-Martin S, Lindh T, Spannare B. Occupational exposure to magnetic fields and brain tumours in Central Sweden. *Eur J Epidemiol* 1998;14:563-69.
 43. Villeneuve PJ, Agnew DA, Johnson KC, Mao Y,

- and the Canadian Cancer Registries Epidemiology Research Group. Brain cancer and occupational exposure to magnetic fields among men: results from a Canadian population-based case-control study. *Int J Epidemiol* 2002;31:210-17.
44. Erren TC. A meta-analysis of epidemiologic studies of electric and magnetic fields and breast cancer in women and men. *Bioelectromagnetics Supplement* 2001;5:S105-19.
 45. Zhu K, Hunter S, Payne-Wilks K, Roland CL, Forbes DG. Use of electric bedding devices and risk of breast cancer in African-American women. *Am J Epidemiol* 2003;158:798-806.
 46. Kliukiene J, Tynes T, Andersen A. Residential and occupational exposures to 50-Hz magnetic fields and breast cancer in women: A population-based study. *Am J Epidemiol* 2004;159:852-61.
 47. Davis S, Mirick DK, Stevens RG. Residential magnetic fields and the risk of breast cancer. *Am J Epidemiol* 2002;155:446-54.
 48. London SJ, Pogodda JM, Hwang KL, Langholz B, Monroe KR, Kolonel LN, et al. Residential magnetic field exposure and breast cancer risk: A nested case-control study from a multiethnic cohort in Los Angeles County, California. *Am J Epidemiol* 2003;158:969-80.
 49. Schoenfeld ER, O'Leary ES, Henderson K, Grimson R, Kabat GC, Ahnn S, et al. Electromagnetic fields and breast cancer on Long Island: A case-control study. *Am J Epidemiol* 2003;158:47-58.
 50. Peplonska B, Stewart P, Szeszenia-Dabrowska N, Resiecki J, Garcia-Closas M, Lissowska J, et al. Occupation and breast cancer risk in Polish women: a population-based case-control study. *Am J Ind Med* 2007;50:97-111.
 51. Charles LE, Loomis D, Shy CM, Newman B, Millikan R, Nylander-French LA, Couper D. Electromagnetic fields, polychlorinated biphenyls and prostate cancer mortality in electric utility workers. *Am J Epidemiol* 2003;157:683-91.
 52. Villeneuve PJ, Agnew DA, Miller AB, Corey PN. Non-Hodgkin's lymphoma among electric utility workers in Ontario: the evaluation of alternate indices of exposure to 60 Hz electric and magnetic fields. *Occup Environ Med* 2000;57:249-257.
 53. Tynes T, Klaeboe L, Haldorsen T. Residential and occupational exposure to 50 Hz magnetic fields and malignant melanoma: a population based study. *Occup Environ Med* 2003;60:343-7.
 54. Qui C, Fratiglioni L, Karp A, Winblad B, Bellander T. Occupational exposure to electromagnetic fields and risk of Alzheimer's Disease. *Epidemiology* 2004;15:687-94.
 55. Feychting M, Jonsson F, Pedersen NL, Ahlbom A. Occupational magnetic field exposure and neurodegenerative disease. *Epidemiology* 2003;14:413-19.
 56. Hakansson N, Gustavsson P, Johansen C, Floderus B. Neurodegenerative diseases in welders and other workers exposed to high levels of magnetic fields. *Epidemiology* 2003;14:420-6.
 57. Savitz DA, Checkoway H, Loomis DP. Magnetic field exposure and neurodegenerative disease mortality among electric utility workers. *Epidemiology* 1998;9:398-404.
 58. Roosli M, Lortscher M, Egger M, Pfluger D, Schreier N, Lortscher E, et al. Mortality from neurodegenerative disease and exposure to extremely low-frequency magnetic fields: 31 years of observations on Swiss railway employees. *Neuroepidemiology* 2007;28:197-206.
 59. Garcia AM, Sisternas A, Perez Hoyos S. Occupational exposure to extremely low frequency electric and magnetic fields and Alzheimer disease: a meta-analysis. *Int J Epidemiol* 2008;1-12.
 60. Elder JA. Thermal, cumulative, and life span effects and cancer in mammals exposed to radio-frequency radiation. In: Carpenter DO, Ayrapetyan S, eds. *Biological Effects of Electric and Magnetic Fields: Beneficial and harmful effects*. New York, NY: Academic Press, 1994; 279-95.
 61. Lai H. Biological effects of radiofrequency electromagnetic fields. In: Bowlin GL, Wnek G, eds. *Encyclopedia of biomaterials and biomedical engineering*. Taylor & Francis, 2005.
 62. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Sci Total Environ* 1996;180:9-17.
 63. Grayson JK. Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: A nested case-control study. *Am J Epidemiol* 1996;143:480-6.
 64. Doik H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt I, Elliott P. Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter. *Am J Epidemiol* 1997;145:1-9.
 65. Michelozzi P, Capon A, Kirchmayer U, Forastiere

- F, Biggeri A, Barca A, et al. Adult and childhood leukemia near a high-power radio station in Rome, Italy. *Am J Epidemiol* 2002;155:1096-103.
66. Park SK, Ha M, Im HJ. Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea. *Int Arch Occup Environ Health* 2004;77:387-94.
67. Ha M, Im H, Lee M, Kim HJ, Kim BC, Gimm YM, et al. Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. *Am J Epidemiol* 2007;166:270-9.
68. Hardell L, Nasman A, Pahlson A, Hallquist A. Case-control study of radiology work, medical X-ray investigations, and use of cellular telephones as risk factors for brain tumors. *MedGenMed* 2000;2:E2.
69. Hardell L, Mild KH, Carlberg M, Hallquist A. Cellular and cordless telephone use and the association with brain tumors in different age groups. *Arch Environ Health* 2004;59:132-7.
70. Lonn S, Ahlbom A, Hall P, Feychting M. Mobile phone use and the risk of acoustic neuroma. *Epidemiology* 2004;15:653-9.
71. Berg G, Spallek J, Schuz J, Schlehofer B, Bohler E, Schlaefer K, et al. Occupational exposure to radio frequency/microwave radiation and the risk of brain tumours: Interphone Study Group, Germany. *Am J Epidemiol* 2006;164:538-48.
72. Schuz J, Bohler E, Berg G, Schlehofer B, Hettinger I, Schlaefer K, et al. Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany). *Am J Epidemiol* 2006;163:512-520.
73. Hours M, Bernard M, Montestrucq L, Arslan M, Bergeret A, Deltour I, Cardis E. Cell phones and risk of brain and acoustic nerve tumours: the French INTERPHONE case-control study. *Rev Epidemiol Sante Publique*. 2007;55(5):321-32.
74. Schoemaker MJ, Sverdlow AJ, Ahlbom A, Auvinen A, Blaasaas G, Cardis E, et al. Mobile phone use and risk of acoustic neuroma: results of the Interphone case-control study in five North European countries. *Br J Cancer* 2005;93:842-8.
75. Lahkola A, Auvinen A, Raitanen J, Schoemaker MJ, Christensen HC, Feychting M, et al. Mobile phone use and risk of glioma in 5 North European countries. *Epidemiology* 2007;120:1769-75.
76. Sadetzki S, Chetrit A, Jarus-Hakak A, Cardis E, Deutch Y, Duvdevani S, et al. Cellular phone use and risk of benign and malignant parotid gland tumors. A nationwide case-control study. *Am J Epidemiol* 2008;167(4):457-67.
77. Inskip PD, Tarone RE, Hatch EE, Wilcosky TC, Shapiro WR, Selker RG, et al. Cellular-telephone use and brain tumours. *N Engl J Med* 2001;344:79-86.
78. Lonn S, Ahlbom A, Hall P, Feychting M, the Swedish Interphone Study Group. Long-term mobile phone use and brain tumor risk. *Am J Epidemiol* 2005;161:526-35.
79. Kundi M, Mild KH, Hardell L, Mattsson MO. Mobile telephones and cancer—A review of epidemiological evidence. *J Toxicol Environ Health B* 2004;7:351-384.
80. Ahlbom A, Green A, Kheifets L, Savitz D, Sverdlow A; ICNIRP (International Commission for Non-Ionizing Radiation Protection) Standing Committee on Epidemiology. Epidemiology of health effects of radiofrequency exposure. *Environ Health Perspect* 2004;112:1741-54.
81. Hardell L, Carlberg M, Soderqvist F, Mild KH, Morgan LL. Long-term use of cellular phones and brain tumours: increased risk associated with use for >10 years. *Occup Environ Med* 2007;64:626-32.
82. Huber R, Graf T, Cote KA, Wittman L, Galmann E, Matter D, et al. Exposure to pulsed high-frequency electromagnetic field during walking affects human sleep EEG. *NeuroReport* 2000;11:3321-5.
83. Koivisto M, Revonsuo A, Krause C, Haarala C, Sillanmaki L, Laine M, et al. Effects of 902 MHz electromagnetic field emitted by cellular telephones on response times in humans. *NeuroReport* 2000;11:413-15.
84. Goldsmith JR. Epidemiologic evidence relevant to radar (microwave) effects. *Environ Health Perspect* 1997;105(Suppl 6):1579-87.
85. Eulitz C, Ullsperger P, Freude G, Elbert T. Mobile phones modulate response patterns of human brain activity. *NeuroReport* 1998;9:3229-32.
86. Freude G, Ullsperger P, Eggert S, Ruppe I. Effects of microwaves emitted by cellular phones on human slow brain potentials. *Bioelectromagnetics* 1998;19:384-7.
87. Seitz H, Stinner D, Eikmann Th, Herr C, Moosli M. Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication—a literature review published between 2000 and 2004. *Sci Total Environ* 2005;349:45-55.

88. Agarwal A, Deepinder F, Sharma RK, Raniga G, Li J. Effect of cell phone usage on semen analysis in men attending infertility clinic: an observational study. *Fertil Steril* 2008;89:124-8.
89. Fejes I, Zavaczki Z, Szollosi J, Koloszar S, Daru J, Kovacs L, Pal A. Is there a relation between cell phone use and semen quality? *Arch Androl* 2005; 51:385-93.
90. Wdowiak A, Wdowiak L, Wiktor H. Evaluation of the effect of using mobile phones on male fertility. *Ann Agric Environ Med* 2007; 14:169-72.
91. Divan HA, Kheifets L, Obel C, Olsen J. Prenatal and postnatal exposure to cell phone use and behavioral problems in children. *Epidemiology* 2008; 2008 May 7. [Epub ahead of print]
92. McCann J, Kavel R, Rafferty CN. Testing electromagnetic fields for potential carcinogenic activity: A critical review of animal models. *Environ Health Perspect* 1997;105(Suppl 1):81-103.
93. Kheifets L, Shimkhada R. Childhood Leukemia and EMF: Review of the epidemiologic evidence. *Bioelectromagnetics* 2005;Suppl 7: S51-9.
94. Reif JS, Lower KS, Oglivie GK. Residential exposure to magnetic fields and risk of canine lymphoma. *Am J Epidemiol* 1995;141:352-9.
95. Repacholi MH, Basten A, Gebiski V, Noonan D, Finnie J, Harris AW. Lymphomas in Eμ-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. *Rad Res* 1997;147:631-40.
96. Utteridge TD, Gebiski V, Finnie JW, Vernon-Roberts B, Kuchel TR. Long term exposure of E-μ-Pim1 transgenic mice to 898.4 MHz microwaves does not increase lymphoma incidence. *Radiat Res* 2002;158:357-64.
97. Sanford LG, Brun AE, Stureson K, Eberhardt J, Persson B. 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.
98. Sanford LG, Brun AE, Eberhardt JL, Malmgren L, Persson BRR. Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones. *Environ Health Perspect* 2003; 111:881-3.
99. Tattersall JEH, Scott IR, Wood SJ, Nettell JJ, Bevir MK, Wang Z et al. Effects of low intensity radiofrequency electromagnetic fields on electrical activity in rat hippocampal slices. *Brain Res* 2001; 904:43-53.
100. Wang B, Lai H. Acute exposure to pulsed 2450-MHz microwaves affects water-maze performance of rats. *Bioelectromagnetics* 2000;21:52-6.
101. US Supreme Court. *Daubert v. Merrell Dow Pharmaceuticals, Inc.* 509 US 579, 1993.
102. Lichtenstein P, Holm NV, Verkasalo PK, Iliadou A, Kaprio J, Koskenvuo M, et al. Environmental and heritable factors in the causation of cancer: Analyses of cohorts of twins from Sweden, Denmark and Finland. *N Engl J Med* 2000;343: 78-85.
103. Kamel F, Umbach DM, Munsat TL, Shefner JM, Hu H, Sandler DP. Lead exposure and amyotrophic lateral sclerosis. *Epidemiology* 2002;13: 311-19.
104. Shcherbatykh I, Carpenter DO. The role of metals in the etiology of Alzheimer's disease. *J Alzheimer's Dis* 2007;11:191-205.
105. Goedert M, Spillantini MG. A century of Alzheimer's disease. *Science* 2006;314:777-84.
106. Leszczynski D, Joenvaara S, Reivinen J, Kuokka R. Non-thermal activation of the hsp27/p38 MAPK 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.
107. Leszczynski D, Nylund R, Joenvaara S, Reivinen J. Applicability of discovery science approach to determine biological effects of mobile phone radiation. *Proteomics* 2004; 4:426-31.
108. Olivares-Banuelos T, Navarro L, Gonzalez A, Drucker-Colin R. Differentiation of chromaffin cells elicited by ELF MF modifies gene expression pattern. *Cell Biol Int* 2004;28:273-9.
109. Lupke M, Frahm J, Lantow M, Maercker C, Remondini D, Bersani F, et al. Gene expression analysis of ELF-MF exposed human monocytes indicating the involvement of the alternative activation pathway. *Biochim Biophys Acta* 2006; 1763:402-12.
110. Zhao R, Zhang S, Xu Z, Ju L, Lu D, Yao G. Studying gene expression profile of rat neuron exposed to 1800 MHz radiofrequency electromagnetic fields with cDNA microassay. *Toxicology* 2007;235:167-75.
111. Goodman R, Blank M. Insights into electromagnetic interactions and mechanisms. *J Cell Physiol* 2002; 192:16-22.
112. Ivancsits S, Diem E, Jahn O, Rudiger HW. Intermittent extremely low frequency electromagnetic fields cause DNA damage in a dose-dependent way. *Int Arch Occup Environ Health* 2003;76: 431-36.
113. Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. *Bioelectromagnetics* 1995;16:513-21.

114. Lai H, Singh NP. Magnetic-field-induced DNA strand breaks in brain cells of the rat. *Environ Health Perspect* 2004;112:687-94.
115. Lai H. Biological effects of radiofrequency electromagnetic fields. In: Bowlin GL, Wnek G, eds. *Encyclopedia of biomaterials and biomedical engineering*. Taylor & Francis, 2005.
116. Hill BA. The environment and disease: association or causation? *Proc Royal Soc Med* 1965;58:295-300.
117. European Environmental Agency. Late lessons from early warnings. *The Precautionary Principle 1896-2000*. Copenhagen, Denmark, 2001.
118. Wingspread Conference on the Precautionary Principle. <http://www.sehn.org/wing/html>, 1998
119. International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz). *Health Physics* 1998; 4: April, 1998.
120. Huss A, Egger M, Hug K, Huwiler-Muntener K, Roosli 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.
121. Sage C, Carpenter D, eds. Bioinitiative Report. A rationale for a biologically-based public exposure standard for electromagnetic fields (ELF and RF). 2007. www.bioinitiative.org.

Electromagnetic Fields and Cancer: The Cost of Doing Nothing

President's Cancer Panel January 27, 2009

David O. Carpenter, M.D.

*Director, Institute for Health and the Environment, University at Albany, and Professor of
Environmental Health Sciences, School of Public Health University at Albany,
5 University Place, A217, Rensselaer, New York, NY 12144-3429, U.S.A.*

Abstract. Everyone is exposed to electromagnetic fields (EMFs) from electricity (extremely low frequency, ELF), communication frequencies, and wireless devices (radiofrequency, RF). Concern of health hazards from EMFs has increased as the use of cell phones and other wireless devices has grown in all segments of society, especially among children. While there has been strong evidence for an association between leukemia and residential or occupational exposure to ELF EMFs for many years, the standards in existence are not sufficiently stringent to protect from an increased risk of cancer. For RF EMFs, standards are set at levels designed to avoid tissue heating, in spite of convincing evidence of adverse biological effects at intensities too low to cause significant heating. Recent studies demonstrate elevations in rates of brain cancer and acoustic neuroma only on the side of the head where individuals used their cell phone. Individuals who begin exposure at younger ages are more vulnerable. These data indicate that the existing standards for radiofrequency exposure are not adequate. While there are many unanswered questions, the cost of doing nothing will result in an increasing number of people, many of them young, developing cancer.

INTRODUCTION

It has been known for many years that high frequency EMFs (X-rays, gamma rays, cosmic rays) have sufficient energy to directly break chemical bonds, causing damage to molecules ranging from water to DNA, leading to increased risk of cancer and birth defects /1/. Thus these forms of EMF are "ionizing". There is less consensus as to whether lower energy forms of EMFs, such as radiofrequency and ELF, can cause disease. In spite of strong documentation that exposure to non-ionizing EMFs is associated with an elevated risk of cancer, most national and international bodies have discounted this evidence, based on the belief that lower energy EMFs cannot possibly have adverse biological effects. This particular point of view is held by many in the physics and engineering communities, individuals not known for their

knowledge of biology or medicine /2/. There are legitimate concerns as to what mechanisms might explain the association between exposure and cancer. The purpose of this presentation is to provide an overview of the issues, explore both the associations between exposure and disease and the mechanisms that might explain them, and to propose biologically-based standards of exposure which, although difficult to achieve, would be more protective of human health. While there are a variety of diseases of possible concern, this review will focus on only cancer.

HEALTH EFFECTS OF ELF EMFS

There has been evidence that residential exposure to elevated magnetic fields results in an increased risk for childhood leukemia since the pioneering 1979 studies of Wertheimer and Leeper

/3/. Most subsequent studies have confirmed elevated risks of leukemia /4/, and several meta-analyses have shown significantly elevated odds ratios (ORs) whether exposure was determined through use of wire codes or measured magnetic fields /5/. In addition there is evidence that leukemia is elevated in adults employed in occupations that involve elevated exposure to EMFs from electricity /6/. Meta-analyses of occupational exposure have also reported elevated risks for leukemia, with less strong evidence for associations with other kinds of cancer /7/. A meta-analysis has also reported a significant elevation in rates of brain cancer among adults working in "electrical" occupations /8/. Thus the association between ELF exposure and cancer, especially leukemia, is very well documented in both children and adults and has been replicated in multiple investigations.

HUMAN DISEASE FROM EXPOSURE TO RF EMFS

Until recently there has been relatively little attention to RF exposures and human health. Older studies have reported elevations in both leukemia and brain tumors among individuals with occupational exposures to RF (see www.bioinitiative.org for references), but the results were not very consistent across studies. Recent reports have found elevated rates of leukemia among children who live near AM radio transmitter sites /12/. This is the same cancer elevated with exposure to power-line frequency EMFs, suggesting that leukemia is the cancer most likely to show elevated risk with whole body exposure to EMFs of any frequency. With the advent of enormous increases in the use of cell phones, we now have a situation in which a very large segment of society is regularly exposed to high levels of RF. In addition, the whole population has increased exposure through the placement of cell phone towers,

wireless buildings and even wireless cities. The strongest evidence for hazards has come from Europe, especially Scandinavia, where cell phones were initially manufactured and have been in wide use for a longer period of time than in other parts of the world. Long-term use of a cell phone is associated with an elevated risk of ipsilateral brain tumors and acoustic neuromas. A meta-analysis by Hardell et al. /13/, based on four studies, reported an OR of 2.0 (95% CL = 1.2-3.4) for glioma among adults who have used a cell phone for ten years or more, but only on the side of the head where the phone was used. There was also an OR of 2.4 (95% CL = 1.1-5.3) for acoustic neuroma among long-term users. Risks for meningioma were elevated, but not significantly so. Kundi /14/ has reported on 33 epidemiological studies, and finds that the combined ORs from these studies show an OR of 1.5 (95% CL = 1.2-1.8) for glioma. There was also a non-significant elevation in ORs for acoustic neuroma but no relationship with meningioma. Hopefully, additional information will come from the pooled results of the INTERPHONE study, a 13-nation investigation coordinated by the World Health Organization (WHO), which should be available in the near future. The Israeli component of this study has also found an elevated risk of ipsilateral parotid gland cancer with long-term cell phone use /15/. There is reason for particular concern about risks to children exposed to RF. Hardell et al. /16/ studied relative risk based on the age when a person began to use a cell phone. For use of either analog or cordless phones when assessed at >1 or >5 year latency, he found that individuals whose use began while they were in their 20s has higher ORs for brain cancer than those whose use began at an older age. Later Hardell reported at a meeting in London last September that children who began use of a cell phone prior to the age of 20 had an OR of developing glioma of 5.2 (95% CL = 2.2-12) after only one+ year of cell phone use, while for all ages the OR was 1.4 (95% CL = 1.1-1.7).

The same relative relationship was seen with use of a cordless phone, where use before the age of 20 years gave an OR of 4.4 (95% CL = 1.9-10), whereas for all ages the OR was 1.4 (95% CL = 1.1-1.8). These studies support the conclusion that use of cordless phones also increases risk, and that children are more vulnerable to risk of brain cancer than adults. The elevated risk to children poses a major concern given the current extensive use of cell phones by even young children.

WHY HAVE THESE RESULTS NOT BEEN REFLECTED IN NEW STANDARDS OF EXPOSURE?

In spite of this consistency in observations relating to ELF EMFs and leukemia, and the developing evidence for a relationship between cell phone use and elevated risk of brain cancer and acoustic neuroma, there has been a general failure of governments and international advisory bodies to accept the reported relationships as being cause and effect, and to follow through with standards designed to reduce exposure. This is a consequence of two major scientific problems, public support of wireless technologies and the political power of the industry. No single mechanism has been identified to be the basis for the development of cancer following exposure to EMFs. In addition, animal studies have not consistently demonstrated cancer as a result of exposure to ELF EMFs. This dilemma is captured well by a statement from the 2007 WHO report /17/: *"Resolving the conflict between epidemiological data (which show an association between ELF magnetic field exposure and an increased risk of childhood leukemia) and experimental and mechanistic data (which do not support this association) is the highest research priority in this field."* The central question in this issue is whether the statement that the experimental and mechanistic data "do not support this association" is correct. There is the widespread

but mistaken belief that all carcinogens act by causing direct DNA damage, as is the case with ionizing radiation. However, many proven human carcinogens do not cause direct DNA damage. These agents are identified as "non-mutagenic carcinogens" by the US Environmental Protection Agency and include such well-documented carcinogens as arsenic /18/ and dioxin /19/. Exact mechanisms are not known to explain the carcinogenicity of either. Thus the fact that ELF and RF EMFs are "non-ionizing" does not mean they are not carcinogens. Both ELF and RF EMFs are known to induce genes /20/, generate reactive oxygen species /21/, trigger formation of heat shock proteins /20,22/ and cause indirect DNA damage /21,23/, any one of which might lead to cancer (see www.bioinitiative.org for additional references and detailed discussion). Thus the argument that mechanistic data does not support a relationship between EMF exposure and cancer is simply wrong. The other argument for discounting the human health information is that animal models have not consistently demonstrated cancer as an outcome. While this is the case for most rodent laboratory studies, Reif et al. /24/ have demonstrated that dogs living in homes with very high wire codes (comparable to those associated with elevated risk of childhood cancer) showed a significant 6.8-fold elevated risk of developing lymphoma. EMFs differ significantly from chemical carcinogens, and it is not clear exactly what field parameters would be comparable to those levels resulting in cancer in humans. It is important to note that the US Supreme Court in the case of Daubert vs. Merrell Dow Pharmaceuticals /25/ effectively ruled that animal studies were not relevant to human health, and that the only admissible evidence must be from human studies. While this is certainly not a justifiable conclusion, in the case of EMFs we have strong evidence for a relationship between exposure and cancer in humans, but much weaker evidence from animal studies. For all of the above reasons lack of strong

evidence for cancer in animals is not a sufficient reason to disregard that strong evidence for a relationship between both ELF and RF exposure and cancer in humans.

PROPOSED EMF STANDARDS THAT ARE BASED ON STUDIES OF HUMAN HEALTH AFTER EXPOSURE

The Bioinitiative Report (www.bioinitiative.org) presents recommendations for standards of EMF exposure that are based on the epidemiological evidence in human populations. For ELF EMFs the proposed standard is 1 mG (0.1 μ T), to be compared with the current International Commission on Non-ionizing Radiation Protection standard of 1,000 mG (100 μ T). For RF radiation the proposed standard is 0.1 μ W/cm², to be compared with the US Federal Communications Commission standard of 583 μ W/cm² for 875 MHz cell phone frequency, and 1,000 μ W/cm² in the frequency range of 1,800-1,950 MHz. The differences between these numbers show the magnitude of the problem. There is no question that a sudden imposition of standards so drastically different from those existing would impose hardship. However, there is also no question that the human studies clearly indicate that the existing standards are not protective of human health. The benefits to society derived from electricity and wireless communications are significant, and certainly none of us is willing to return to the pre-electric age. However it is imperative that society at least acknowledge the disparities between current standards and current evidence of risk of cancer. Rigid and sudden imposition of the standards we propose is unrealistic, but these levels are appropriate goals that could at least be approached by a combination of development of new technology and changes in behaviors.

THE COSTS OF BEING WRONG

At present we do not know precisely to what degree the risk of cancer is increased by exposure to EMFs. Human studies are difficult under any circumstances, but those difficulties are even greater when studying the effects of EMFs. Levels of exposure for each of us vary over the course of every day as we move through our environment and use appliances, cell phones and other wireless devices. This makes exposure assessment extremely difficult. Given the long latency for development of cancer, one would expect that the actual risk of EMF-induced cancer is significantly greater than that indicated by studies with inadequate exposure assessment. There is considerable evidence that children are more vulnerable to many environmental insults than are adults /26/. The reality is that children are using cell phones at increasing rates and for long durations. Therefore, if the risks are real, and especially if children are more susceptible, we may be facing an epidemic of brain and other cancers. The concern is increased because to date, there has been little warning advising restrictions on use of cell phones, especially by children. While questions regarding mechanisms are not all answered, the evidence for a relationship between EMF exposure and cancer is sufficiently strong so as to demand action. The alternative may be significant increases in certain cancers, especially leukemia and brain cancer. It is not clear whether there is increased risk of other kinds of cancer following exposure because there has not been a study of, for example, the health hazards of wearing a cell phone on your belt and pelvic cancers. Fortunately, the rates of leukemia and brain cancer are not high, at least not at present.

There have recently been significant improvements in treatment of leukemia, especially among children. Kundi /14/ has hypothesized that use of cell phones may increase the rates of brain cancer by as much as 50%. Even if this is true, this

certainly does not mean that every exposed person will develop brain cancer. However an increase in brain cancer of 50% would still have a significant impact, not only on the individuals affected but also on society, especially given that much of this increase is likely to occur among young people. It is not appropriate to deny the well-documented relationship between EMF exposure and cancer only because the mechanistic details are uncertain. The evidence that we have at present is too convincing to be ignored. Our national and international standards are obsolete, and ignore evidence reported by many different investigators. The lack of certainty with regard to mechanisms and animal models is no reason to ignore studies of human health.

We need the electric and communications industries to be proactive in developing products that can be used with reduced exposures. We need governments and international organizations to set standards that are based on the evidence of hazard, not on a hypothesis that is not credible based on the evidence from animal, cellular and human studies. Most importantly, we need individuals to understand that personal decisions will significantly impact the level to which they are exposed to both ELF and RF EMFs.

REFERENCES

1. Little MP. Risks associated with ionizing radiation. *Brit. Med. Bull.* 2003;69 259-275
2. Adair RK. Static and low-frequency magnetic field effects: health risk and therapies. *Rep. Prog. Physics* 2000;63 415-454
3. Wertheimer N and Leeper E. Electrical wiring configurations and childhood cancer. *Am. J. Epidemiol.* 1979;109 273-284
4. Savitz DA, Wachtel H, Barnes FA, John EM, and Tvrdik JG. Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. *Am. J. Epidemiol.* 1988;128 21-38; London SJ, Thomas DC, Bowman JD, Sobel E, Cheng TC, Peters JM. Exposure to residential electric and magnetic fields and risk of childhood leukemia. *Am. J. Epidemiol.* 1991 134 923-937; Feychting M and Ahlbom A. Magnetic fields and cancer in children residing near Swedish high-voltage power lines. *Am. J. Epidemiol.* 1993;138 467-481
5. Wartenberg D. Residential magnetic fields and childhood leukemia: A meta-analysis. *Am. J. Public Health* 1998;88 1787-1794; Greenland S, Sheppard AR, Kaune WT, Poole C, Kelsh MA and the Childhood Leukemia-EMF Study Group. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. *Epidemiology* 2000; 11:624-634; Ahlbom A, Day N, Reychting M, Roman E, Skinner J, Doctery J et al. A pooled analysis of magnetic fields and childhood leukemia. *Br. J. Cancer* 2000;83 692-698
6. Savitz DA, Calle EE. Leukemia and occupational exposure to electromagnetic fields: Review of epidemiologic studies. *J. Occup. Med.* 1987;29 47-51
7. Kheifets LI, Afifi AA, Buffler PA, Zhang ZW, Mastkin CC. Occupational electric and magnetic field exposure and leukemia. *JOEM* 1997;29 1075-91
8. Kheifets LI, Afifi AA, Buffler PA, Zhang ZW. Occupational electric and magnetic field exposure and brain cancer: A meta-analysis. *JOEM* 1995;37 1327-40
9. Michelozzi P, Capon A, Kirchmayer U, Forastiere F, Biggeri A, Barca A et al. Adult and childhood leukemia near a high-power radio station in Rome, Italy. *Am. J. Epidemiol.* 2002;155:1096-103; Park SK, Ha M and Im HJ. Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea. *Int. Arch. Occup. Environ. Health* 2004;77 387-94; Ha M, Im H, Lee M, Kim HJ, Gimm YM et al. Radiofrequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. *Am. J. Epidemiol.* 2007;166:270-9
10. Hardell L, Carlberg M, Soderqvist F and Mild KH. Meta-analysis of long-term mobile phone use and the association with brain tumours. *Internat. J. Oncol.* 2008;32:1097-103
11. Kundi M. The controversy about a possible relationship between mobile phone use and cancer. *Environ. Health Perspect* 2000;117(3): 316-24.
12. Sadetzki S, Chetrit A, Jarus-Hakak A, Cardis E, Deutch Y, Duvdevani S et al. Cellular phone use

- and risk of benign and malignant parotid gland tumors. A nationwide case-control study. *Am. J. Epidemiol.* 2008;167:457-67.
13. Hardell L, Mild KH, Carlberg M, Hallquist A. Cellular and cordless telephone use and the association with brain tumors in different age groups. *Arch. Environ. Health* 2004;59:653-9.
 14. World Health Organization. Extremely Low Frequency Fields. Environmental Health Criteria, Volume 238. Geneva: WHO, 2007.
 15. Gebel T. Confounding variables in the environmental toxicology of arsenic *Toxicology* 2000; 144:155-62; Hughes MF. Arsenic toxicity and potential mechanisms of action. *Toxicol. Lett.* 2002;133:1-16.
 16. Nowak R. Dioxin: Environmental toxin, molecular probe. *J NIH Res* 1991;3:49-53.
 17. Goodman R, Blank M. Insights into 'electromagnetic interaction mechanisms. *J. Cell. Physiol.* 2002;192:16-22; Zhao R, Zhang S, Xu Z, Ju L, Lu D, Yao G. Studying gene expression profile of rat neuron exposed to 1800 MHz radiofrequency electromagnetic fields with cDNA microassay. *Toxicology* 2007;235:167-75.
 18. Lai H, Singh NP. Magnetic field-induced DNA strand breaks in brain cells of the rat. *Environ. Health Perspect.* 2004;112:687-94.
 19. Leszczynski D, Joenvaara S, Reivinen J, Kuokka R. Non-thermal activation of the hsp27p38MAPK 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.
 20. Svedenstal BM, Johanson KJ, Mattsson MO, Paulsson LE. DNA damage, cell kinetics and ODC activities studies in CBA mice exposed to electromagnetic fields generated by transmission lines. In *Vivo* 1999;13:507-14; Ivancsits S, Diem E, Jahn O, Rudiger HW. Intermittent extremely low frequency electromagnetic fields cause DNA damage in a dose-dependent way. *Int. Arch. Occup. Environ. Health* 2003;76:431-6.
 21. Reif JS, Lower KS, and Oglivie GK. Residential exposure to magnetic fields and risk of canine lymphoma. *Am. J. Epidemiol.* 1995;141:352-9.
 22. US Supreme Court. *Daubert v. Merrell Dow Pharmaceuticals, Inc.* 1993 509 US 579
 23. Ginsberg GL. Assessing cancer risks from short-term exposures in children. *Risk Anal.* 2003;23: 19-34; Landrigan PJ, Garg A. Children are not little adults. *Children's Health and the Environment. A Global Perspective.* J. Pronczuk-Garbino (ed) Geneva: World Health Organization, 2005;Chap. 1, pp 3-16.