**STATE OF MINNESOTA**

**OFFICE OF ADMINISTRATIVE HEARINGS**

**FOR THE PUBLIC UTILITIES COMMISSION**

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| IN THE MATTER OF THE ROUTE PERMIT APPLICATION BY GREAT RIVER ENERGY AND XCEL ENERGY FOR A 345 KV TRANSMISSION LINE FROM BROOKINGS COUNTY, SOUTH DAKOTA TO HAMPTON, MINNESOTA | PUC DOCKET NO. ET2/TL-08-1474<br>OAH DOCKET NO. 7-2500-20283-2 |

# TESTIMONY OF DAVID O. CARPENTER

On Behalf of
INTERVENORS
Robert and Patricia Johnson

Exhibit _____

# I. INTRODUCTION AND QUALIFICATIONS

**Q: Please state your name and your business address.**

A: My name is David O. Carpenter and my business address is at University at Albany, SUNY, Rensselaer, NY 12144I.

**Q: By whom are you employed and what is your position?**

A: I am a public health physician and Director of the Institute for Health and the Environment at the University at Albany, SUNY. My *Curriculum vitae* is attached as Schedule 1.

**Q: Please summarize your educational background.**

A: I received my Bachelors of Arts degree in 1959 *magna cum laude* from Harvard College, Cambridge, Massachusetts; and my Medical Degree in 1964 *cum laude* from Harvard Medical School, where I wrote a research thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems," and I was awarded the Leon Resnick Prize for the Medical School graduate showing promise in research.

**Q: Please summarize your professional research and experience, particularly as it pertains to the area of electric and magnetic fields ("EMF").**

A: My professional career and research has focused on basic neuroscience, the neural basis for human disease, neurotoxicology and human health effects of environmental exposures, including those from exposure to ionizing and non-ionizing radiation, toxic metals and organic contaminants such as polychlorinated biphenyls (PCBs), dioxins and chlorinated pesticides.

In my position as Director of the Institute for Health and the Environment I am responsible for promoting interdisciplinary research and grants relating to health and the environment. Our members come from other parts of the University at Albany, other universities and medical centers, and New York State agencies, and have research interests in environmental health, environmental sciences, environmental policy, environmental law, ecology, hazardous waste management, occupational health, risk assessment, risk management, risk communication and the social and psychological aspects of environmental pollution.

As an educator and Director, my educational mission, and that of the Institute is to promote interdisciplinary research focusing on factors that produce physical injury and illness, factors that lead to development blighted and contaminated areas, and policies that reverse the deterioration of these areas and improve the health of the residents. Greater detail on the Institute's mission and programs may be found at www.albany.edu/ihe/. I am also Professor of Environmental Health Sciences and Biomedical Sciences in the School of Public Health at the University at Albany, SUNY.

From March, 1980 until September, 1985, I was Director of the Wadsworth Center for Laboratories and Research for New York State's Department of Health in Albany, New York. Among other duties I was given the responsibility of administration of the New York State Power Lines Project, a 5 million dollar study of the health effects of power line-frequency electromagnetic fields (EMFs). One of the projects supported by this program confirmed an earlier observation and

demonstrated an elevation in rates of childhood leukemia among children living in homes with elevated magnetic fields (Savitz et al., 1988).

After completion of the New York State Power Lines Project I became the spokesperson for New York State on the issue of human health effects of EMFs. In this capacity I have served on several state and national committees on EMF issues. I have co-edited two books on EMFs and have served as the co-editor of the Bioinitiative Report (www.bioinitiative.org). In January 2008 I was invited to testify before the President's Cancer Panel on the issue of exposure to EMF and cancer.

## II. ANALYSIS OF IMPACTS OF EMF ON HUMAN HEALTH

**Q: Have you prepared a comprehensive report regarding the effects of EMF on human health?**

A: I published a comprehensive review of the literature pertaining to electromagnetic field exposures in the peer-reviewed journal, *Reviews on Environmental Health* in 2008, which is attached as Schedule 2. This journal article was not designed to meet the needs of any public or private client or to serve the outcome in any proceedings, but to summarize the current state of national and international research regarding electromagnetic field exposures and to recommend prudent public health policy. Many of the references discussed in my testimony are also referenced in this journal article.

**Q: Could you please summarize your professional opinion regarding the health effects of magnetic fields from high voltage power lines.**

A: My professional opinion as a public health physician, medical researcher and educator specializing in study of ionizing and non-ionizing radiation effects on biological systems is as follows:

1) There is strong scientific evidence that exposure to magnetic fields from power lines greater than 4 milligauss ("mG") is associated with an elevated risk of childhood leukemia.

2) Some studies have demonstrated significant elevations in childhood leukemia when comparing children living in homes with 2 mG exposure as compared to those in homes with 1 mG of exposure. There is sufficient scientific evidence to cause concern about leukemia risks at exposures above 2 mG.

3) There is some evidence that occupational and residential exposure to magnetic fields is associated with cancer in adults as well, particularly brain cancer. There is strong scientific evidence that lifetime exposure to magnetic fields in excess of 2 mG is associated with an increased risk of neurodegenerative diseases in adults, including Alzheimer's disease and amyotrophic lateral sclerosis (ALS).

4) While there is a debate as to which mechanisms are responsible, and there no specific animal model for the way in which magnetic fields cause cancer, there is a large body of evidence of ways in which magnetic fields affect tissue at a cellular level which may be the basis for the development of cancer and neurodegenerative disease.

5) There is no reliable evidence that power-line magnetic fields do not cause cancer, and a large body of evidence that power-line magnetic fields do cause adverse human health impacts, including cancer.

7) Prudent public health policy requires minimizing the effects of power line magnetic fields on human health.

**Q. Have you reviewed the testimony of Dr. Peter Valberg in this matter?**

A. I have reviewed both Dr. Valberg's written testimony and the report he prepared for the CapX2020 utilities in connection with these administrative proceedings.

**Q. What is your assessment of Dr. Valberg's opinions reflected in these documents?**

A. The arguments made by Dr. Valberg are not new. Similar arguments have been made by certain researchers and on behalf of industries involved in the generation of magnetic fields for many years. They are similar to the arguments which I addressed in the comprehensive review of literature published last year in *Reviews on Environmental Health*. I find these arguments to be false, inaccurate and not supported by a comprehensive and disinterested review of the scientific evidence.

**Q. Could you please explain the key arguments made to oppose a finding that magnetic fields increase health risks and the scientific evidence you believe is most pertinent to respond to these arguments?**

A. The first argument made is that evidence for elevated risk of childhood leukemia from exposure to power line EMF is "weak and inconsistent." Reports from leading research organizations do not support this argument.

1) The U.S. National Academy of Science, National Research Council report (1997) stated that the link between power line wire-code rating and childhood leukemia "is statistically significant (unlikely to have arisen from chance) and is robust" in the sense that removing a study from the group does not alter the conclusion that an association exists.

2) The introduction to the National Institute of Environmental Health Sciences EMF-RAPID program (1999) report summarized:

"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."

3) The 2007 World Health Organization report, while questioning the experimental evidence of mechanism, stated that the epidemiological data "show an association between ELF magnetic field exposure and an increased risk of childhood leukemia."

As explained above, all three reports have accepted the demonstration of a statistically significant relation between exposure to elevated magnetic power line fields and childhood leukemia. This conclusion is supported by at least three recent meta-analyses of the relation between childhood leukemia and EMFs.

**Q. What was found in these meta-analyses of the relation between childhood leukemia and magnetic fields?**

A. Wartenberg (1998) reported on 16 epidemiologic studies, considering reports using the Wertheimer and Leeper (1979) 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 other recent meta-analyses found a statistically significant elevated risk of childhood leukemia from exposure to magnetic fields. Greenland et al. (2000) reported a significantly elevated risk of 1.68 (68% increase in childhood leukemia) based on pooled results from 12 studies, using a time-weighted average of exposure greater than 3 mG (0.3 μT) and Ahlbom et al. (2000) reported on nine studies, and found a elevated risk of 2.0 (doubling of incidence) for exposures equal or greater than 4 mG (0.4 μT) as compared with less than 1 mG (0.1 μT).

These reports are important in that they show consistency of a clearly elevated risk of leukemia in children with magnetic field exposure from power-line fields in their homes. Several recent studies of a dose-dependent relationship between power line exposure and leukemia serve to confirm the likelihood of causation.

**Q. Why is it significant if studies show a dose-dependent relationship?**

A. In epidemiology, a dose-dependent relationship is usually considered the gold standard in determining whether an association reflects a causal relationship. If an effect becomes more pronounced as the dose increases -- whether the dose is

milligrams of a chemical or milligauss of a magnetic field – causation is much more likely.

Several recent studies add to the conclusion that the relationship between magnetic field exposure and leukemia is strong by demonstrating this type of dose dependence. Draper et al. (2005) studied rates of leukemia in children in relation to proximity of their homes to high-voltage power lines. The investigators found a dose-dependent relationship, with relative risk being 1.69 (69 % increase) for children living within 200 meters of a high voltage power line as compared to those living more than 600 meters from the line, and the relative risk being 1.23 (23% increase) for children living from 200 to 600 meters from the line as compared with those more than 600 meters away. The trend of increased risk based on closeness to the power line was statistically significant ($p < .01$).

Foliart et al. (2006) examined the relation between magnetic field exposure and the survival of children with acute lymphoblastic leukemia in the United States and found a hazard ratio of 4.5 (four and a half times the risk) for children exposed to greater than 3 mG (0.3 μT) magnetic fields as compared with those having exposure to less than 1 mG (0.1 μT). Svendsen et al. (2007) performed a similar study of German children with leukemia, and reported a hazard ratio of 2.6 (more than two and a half times the risk) for the survival of children with acute lymphoblastic leukemia exposed to 2 mG (0.2 μT) during recovery as compared with those exposed to less than 1 mG (0.1 μT).

**Q. Is there any scientific evidence that the fetus or child is at greater risk from magnetic fields than are adults?**

A. The scientific literature demonstrates 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. Lowenthal et al. (2007) compared incidence of adult lympho-proliferative and myeloproliferative cancers in relation to childhood residence and found an increased risk of 3.23 (more than three times the risk) for adults who lived within 300 meters of a high-voltage power line during the first 15 years of life. For those who lived within 300 meters of a power line in the first 5 years of life, the increased risk was 4.74 (nearly five times the risk), 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 (2003) showed that maternal exposure during pregnancy increased the risk of children 0-9 years of age developing leukemia, with a risk factor of 2.5 (two and a half times the risk) for children of mothers in the highest 10% of exposure.

These findings are consistent with a large body of information showing that the fetus and young child are more vulnerable than older persons are to chemicals and ionizing radiation. This susceptibility may be why the evidence for the relation between magnetic field exposure and leukemia in children is stronger than that for adults. 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. The limitations in the exposure assessment (consideration of only residential exposure from external power lines) are such that one would expect that studies have underestimated rather than overestimated the actual risk.

**Q. Is the public health impact of the risk from power line magnetic fields significant?**

A. Some commenters have suggested that only a small number of children are affected so that public health concerns are not substantial. This argument is not correct because we do not know precisely how many children are affected with leukemia resulting from of EMF exposure. In 1988, Carpenter and Ahlbom estimated that as much as 10% to 15% of US childhood leukemia (about 1,000 cases) could be associated with residential magnetic field exposure from external power lines. Some estimates are even higher (Milham and Ossiander, 2001). In the meta-analyses mentioned above, however, Greenland et al. (2000) calculated the attributable fraction of cases of childhood leukemia from residential magnetic field exposure in the US to be 3%. The recent WHO Environmental Health Criteria ELF Monograph #238 (2007) states, that if the association between childhood leukemia and exposure to power lines is causal, the number of cases of childhood leukemia worldwide that might be attributable to exposure can be estimated to range from 0.2 to 4.9% of the total annual incidence of leukemia cases. It should be noted that exposure to other household sources of magnetic fields also elevate the risk of childhood leukemia.

**Q. How high do magnetic fields need to be to increase the risk of childhood leukemia?**

A. In my professional opinion, the evidence for a relation between exposure and childhood leukemia may be considered to be definitive at exposure levels of 3 or 4 mG (milligauss) or higher. Evidence from some, but not all, of the scientific studies

indicates an elevated risk at levels greater than 2 mG (Savitz et al., 1988; Green, 1999). No evidence has been reported that exposures at lower levels are "safe," as persons with such exposures usually serve as the "control" group. I would suggest that magnetic fields of 2 mG raise some health concerns, although the evidence of risk is not conclusive.

**Q: Is it a concern that studies generally have used residential distance from power lines rather than measurements of actual magnetic fields?**

A. To completely determine the risk of exposure to magnetic fields, it would be desirable to study the full range of residential, occupational (or school) and recreational exposures to the full range of the electromagnetic spectrum, including electric devices as well as power lines. It is my opinion that, if such a study were constructed, the overall magnitude of the risk of magnetic fields to both children and adults would be higher, not lower, than what currently available research demonstrates.

**Q. Is there any evidence of adult disease associates with magnetic field exposures?**

A. The level of evidence definitively proving an association between exposure to magnetic fields and adult cancer is less strong than the relation with childhood leukemia. Several studies show statistically significant relations between occupational exposure to magnetic fields and leukemia in adults despite limitations in exposure assessment. Elevations in the rates of leukemia following occupational exposure to elevated EMF have been reported in review articles (Savitz and Ahlbom, 1994) and in a meta-analysis (Kheifets et al., 1997). Savitz and Loomis (1995) did not find any elevation in risk of leukemia in a study of 138,905 electric

utility workers. Minder and Pfluger (2001) report elevated leukemia mortality among Swiss railway employees exposed to magnetic fields, whereas Harrington et al. (2001) reported no elevated rates of leukemia among UK electricity generation and transmission workers when compared with the rest of the UK population.

In a review study, Miller et al. (1997) 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. Feychting et al. (1997) investigated adult leukemia in relation to magnetic field exposures in the home and at work. The investigators found no relation between residential exposure alone and only a non-significant elevation of risk of leukemia with occupational exposure alone. However, when both residential and occupational exposures were considered, the authors reported a significant elevation of risk of all leukemias with an odds ratio of 3.7 (more than 300 percent increase in risk).

In adults, the evidence for a relation between magnetic field exposure and cancers other than leukemia is strongest for brain cancer. Kheifets et al. (1995) performed a meta-analysis of 29 reports of brain cancer found statistically significant elevations for electrical engineers, welders, and power station workers. Rodvall et al. (1998) investigated glioma and meningioma in Sweden in relation to job title, and reported only non-significant elevations of both cancers in relation to measured magnetic fields. Villeneuve et al. (2002) also reported only non-significant elevations in rates of overall brain cancers in relation to residential exposure to magnetic fields, but found a highly significant relationship among men diagnosed with a specific brain cancer -- glioblastoma multiforme.

The evidence for a relation between magnetic field exposure and breast cancer is relatively strong in men (Erren, 2001), and some, (Zhu et al., 2003; Kliukiene et al., 2004) but by no means all (Schoenfeld et al., 2003) studies show female breast cancer also to be significantly elevated with increased exposure. Peplonska et al. (2007) recently found increased risk of breast cancer in women occupationally exposed to elevated magnetic fields.

Overall, although the evidence is less conclusive than for childhood leukemia, the weight of the scientific research suggests that it is likely that magnetic fields increase the risk of certain cancers in adults, as well as in children.

**Q. Are there any non-cancer health risks of magnetic fields to adults?**

A. There is strong evidence of an association between EMF exposure and the neurodegenerative diseases Alzheimer's and amyotrophic lateral sclerosis (ALS). For Alzheimer's disease, Qiu et al. (2004), Feychting et al. (2003) and Hakansson et al. (2003) found a statistically significant elevated risk with EMF exposure, approximately two or three times the incidence in a control population. For ALS, Savitz et al. (1998) and Hakansson et al. (2003) found a statistically significant increased risk, again approximately two to three times the incidence in a control population. Roosli et al. (2007) looked at neuro-degenerative diseases among Swiss railway employees exposed to magnetic fields. For every 10 μT years of cumulative exposure the authors found Alzheimer's disease risk to increase by 9.4%. No elevated risk was found for Parkinson's disease or multiple sclerosis. Garcia et al. (2008) analyzed 14 different studies of EMF exposure and Alzheimer's disease and found a consistent pattern of elevated risk.

**Q. How would you characterize the risk of magnetic fields to adults?**

A. In total, the scientific evidence for adult disease, especially leukemia, brain cancer, Alzheimer's disease and ALS, associated with EMF exposure is sufficiently strong that preventive steps are not only appropriate but also called for. I've reached this conclusion based on the epidemiologic evidence, understanding all of the limits in study design and exposure assessment. In epidemiology, discounting the positive studies just because not every investigation shows a positive result is inappropriate. Although further research should be conducted with better exposure assessment and controls, the evidence for a relation between EMF exposure and adult cancer and neurodegenerative diseases is sufficiently strong at present to merit preventive actions to reduce exposure.

**Q. How do you respond to the argument that there is no laboratory animal study that reliably demonstrates adverse health changes in response to electric power line magnetic field levels?**

A. 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. However, it should be pointed out, as Kheifets et al. (2005) did in their policy review, that there is no good animal model for childhood leukemia itself and that the limitations of laboratory studies make it difficult to detect risks. In this article, after describing some of the limitations of laboratory data, the authors concluded, based on the weight of evidence approach and incorporating different lines of scientific enquiry, that epidemiologic evidence, as most relevant, should be given the greatest weight. Interestingly, recent studies

have demonstrated that pet dogs living in homes characterized by high or very high wire codes as compared with those in homes with low wire codes or buried power lines have increased rates of lymphoma at a level that is statistically significant. (Reif et al.,1995).

**Q. How do you respond to the argument that scientists have not been able to establish a mechanistic model to explain how magnetic fields from power lines cause adverse health effects, such as cancer?**

A. As a physician and a scientist, the lack of mechanistic model is neither surprising nor significant in evaluating whether EMF exposures increase the risk of cancer. Although we know a lot about cancer, we do not know the mechanism of cancer in general. We know the mechanisms of action for certain carcinogenic substances, but for most cancers, we know neither the environmental trigger nor the precise mechanism of action.

It came as a major surprise to most scientists when Lichtenstein et al. (2000) concluded from their study of identical twins that environmental factors were the initiating event in the majority of cancers. One human study has demonstrated a genetic mechanism for childhood acute myeloid leukemia. Yang et al. (2008) found that children who live within 100 meters of a power line or transformer and have a certain gene (the XRCC! Ex9 + 16A allele of a DNA repair gene) have an increased risk 4.31 times greater (over 400 percent increase) of developing leukemia than children with the same exposure that did not have this gene. This is the first study demonstrating a genetic-environment interaction as a basis of human cancer from exposure to EMFs.

We also 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. In the case of Alzheimer's disease, abnormalities of amyloidβ and the tau protein have been found, but the understanding of why or how they form is very limited.

**Q. Are there any effects on biological tissue as a result of EMF at levels similar to power line exposures?**

A. Clear evidence has emerged that EMFs alter cell physiology and function. Electromagnetic fields (60 Hz) inhibit differentiation of an erythroleukemia cell line, affect gene transcription (Leszczynski et al., 2002; 2004; Olivares-Banuelos et al., 2004; Lupke et al., 2006; Zhao et al., 2007), induce the synthesis of stress proteins (Goodman and Blank, 2002; Tokalov and Gutzeit, 2004), and cause breakage of DNA (Svedenstal., et al., 1999; Ivanscits et al., 2003), probably through the generation of reactive oxygen species (Lai and Singh, 1995; 2004). Any one of these actions might be responsible for the carcinogenic and/or neuro-degenerative actions of EMFs. As with many environmental agents, however, assuming that only one mechanism of action exists would be a mistake, particularly where more than one disease is involved. It is more likely that multiple mechanisms of action would contribute to disease.

## III. RECOMMENDATIONS AND CONCLUSION

**Q. What significance does the information about a mechanism by which EMF could cause disease have for public health decisions?**

A. How, precisely, EMF or magnetic fields are responsible for increased risks of childhood leukemia or neurodegenerative disease may be a subject of debate within the scientific community, but from a public health point of view, this controversy does not matter. Persons responsible for the public health of communities need to take appropriate action and exercise appropriate caution to protect public health whether or not scientists can agree on the mechanisms causing the adverse effects observed in human beings.

**Q. What would you conclude is needed as public health measures to reduce the risks to human health associated with power lines?**

A. I would conclude that exposure for prolonged periods of time to magnetic fields above 2 to 4 mG from power lines is associated with increased risk of cancer, especially leukemia, and children are most vulnerable. In addition magnetic field exposure increases the risk of neurodegenerative diseases, including Alzheimer's Disease and amyotrophic lateral sclerosis. There is no known threshold below which magnetic fields should be assumed to have no adverse effects, and exercising reasonable precaution is appropriate in making public health decisions. From this perspective, I would recommend the following:

1) Information should be obtained for the specific power line and its various segments regarding the calculated magnetic field strength at various distances from the centerline, in particular the distances at which magnetic fields decline below 4 milligauss (mG) and 2 mG.

2) In my opinion any long-term exposure to magnetic fields above 2 mG is abnormally high and poses a risk to the health of residents. I believe that long-

term levels of power-line magnetic fields above 4 mG render a home unsafe and uninhabitable because such levels will increase risk of both cancer and neurodegenerative diseases. Power line routing should avoid exposures at these levels.

3) In addition to preventing residential exposure to magnetic fields above these levels, public health precaution suggests that high voltage lines be located as far from homes, schools and child care facilities as is possible. In areas where avoidance is not possible, mitigation of EMF by placing lines underground and placing lines where phase cancellation can reduce magnetic fields may also reduce human health impacts.

**Q. Do you have any additional comments?**

A. Electricity is an important part of our life. Beyond the scope of this particular power line proceeding, public decision-makers should consider what alternatives and technologies would reduce the exposure and intensity of magnetic fields.

**Q. Does this conclude your prefiled direct testimony?**

A. Yes.

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Savitz DA, Wachtel H, Barnes FA et al. (1988) Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. Am J Epidemiol 128: 21-38.

Schoenfeld ER, O'Leary ES, Henderson K, Grimson R, Kabat GC, Ahnn S, et al. (2003) Electro-magnetic fields and breast cancer on Long Island: A case-control study. Am J Epidemiol 158: 47-58.

Svedenstål BM, Johanson KJ, Mattsson MO, Paulsson LE (1999) DNA damage, cell kinetics and ODC activities studied in CBA mice exposed to electromagnetic fields generated by transmission lines. In Vivo 13:507-513.

Svendsen AL, Weihkoph T, Kaaatsch P, Schuz J (2007) Exposure to magnetic fields and survival after diagnosis of childhood leukemia: A German cohort study. Cancer Epidmiol Biomark Prev 16: 1167-1171.

Tokalov SV, Gutzeit HO (2004) Weak electromagnetic fields (50 Hz) elicit a stress response in human cells. Environ Res 94:145-151.

Tynes T, Klaeboe L, Haldorsen T (2003) Residential and occupational exposure to 50 Hz magnetic fields and malignant melanoma: a population based study. Occup Enviro Med 60:343-347.

US National Academy of Science, National Research Council (1997) Possible health effects of exposure to residential electric and magnetic fields. Washington, DC, National Academy Press.

Villeneuve PJ, Agnew DA, Johnson KC, Mao Y, and the Canadian Cancer Registries Epidemiology Research Group (2002) Brain cancer and occupational exposure to magnetic fields among men: results from a Canadian population-based case-control study. Int J Epidemiol 31:210-217.
Villeneuve PJ, Agnew DA, Miller AB, Corey PN (2000) 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 57: 249-257.

Wartenberg D (1998) Residential magnetic fields and childhood leukemia: A meta-analysis. Am J Public Health 88:1787-1794.

Wertheimer N and Leeper E (1979) Electrical wiring configurations and childhood cancer. Am J Epidemiol 109:273-84.

World Health Organization (WHO) (2007) Extremely low frequency fields. Environmental Health Criteria, Volume 238. Geneva: WHO.

Yang Y, Jin X, Yan C, Tian Y, Tang J, Shen X (2008) Case-only study of interactions between DNA repair genes (Hmlh1, APEX1, MGMT, XRCC1 and XPD) and low-frequency electromagnetic fields in childhood acute leukemia. Leukemia Lymphoma 49:2344-2350.

Zhao R, Zhang S, Xu Z, Ju L, Lu D, Yao G (2007) Studying gene expression profile of rat neuron exposed to 1800 MHz radiofrequency electromag-netic fields with cDNA microassay. Toxicology 235:167-175.

Zhu K, Hunter S, Payne-Wilks K, Roland CL, Forbes DG (2003) Use of electric bedding devices and risk of breast cancer in African-American women. Am J Epidemiol 158:798-806.

# *CURRICULUM VITAE*

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:

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

**Positions Held**:

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|---|---|
| 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.

**National and International Committees:**

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| 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:**

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| 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 |

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| 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-Pres. | 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-Pres. | Member, Board of Directors, Marine Environmental Research Institute |
| 2007-2009 | Member, New York State Renewable Energy Task Force |

**Honors, Awards And Fellowships:**

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| 1959 | B.A. awarded magna cum laude. Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, Ambystoma tigrinum" (Thesis advisor, Professor George Wald) |
| | Elected to Phi Beta Kappa and to Sigma Xi |
| 1964 | M.D. awarded cum laude 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 |
| 1987 | University, Fukuoka, Japan, for a period of three months each |
| 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)

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| 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. ACentral/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 inhibitibility 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 norepin-ephrine 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. Intl. 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 $\alpha$-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. $H_1$ and $H_2$ 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 restivity 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 $\alpha$-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., Auker, 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. Kebabian, P.R., Kebabian, 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. Ophthamol., 89:641-646, 1980.

56. Mell, L.D., Jr. and Carpenter, D.O. Fluorometric determination of octopamine in tissue homegenates 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. Opthal., 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. Opthal., 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., Kebabian, 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 $^{14}$C-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. ffrench-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.

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**REVIEWS ON ENVIRONMENTAL HEALTH** **VOLUME 23, NO. 2, 2008**

# Setting Prudent Public Health Policy for Electromagnetic Field Exposures

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***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

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**TABLE OF CONTENTS**

## 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.

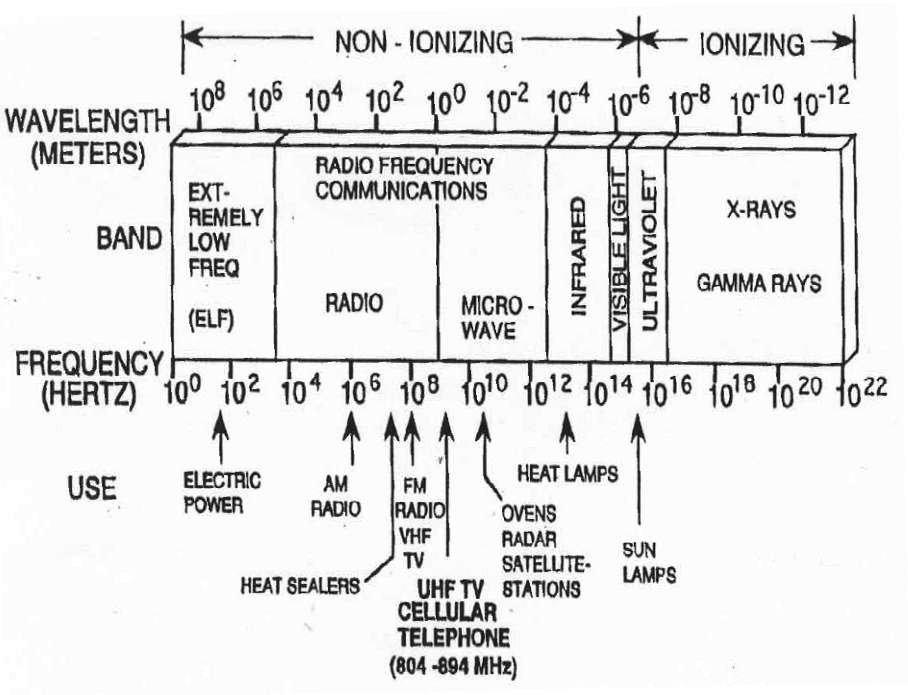


**Fig. 1:** The electromagnetic spectrum, showing the relation s between ELF and RF fields, wavelength and frequency, and the ionizing and non-ionizing portions of the spectrum.

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 μT). 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 μT) as compared with less than 1 mG (0.1 μT).

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 μT) 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 μT) as compared with those having exposure to less than 1 mG (0.1 μT). 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 μT) during recovery as compared with those exposed to less than 1 mG (0.1 μT).

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 of 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 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 = 1.12-1.124) 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 neuro-degenerative 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 μT 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 hematopoetic 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. Utteridege 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 it's 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 amyloidβ 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.

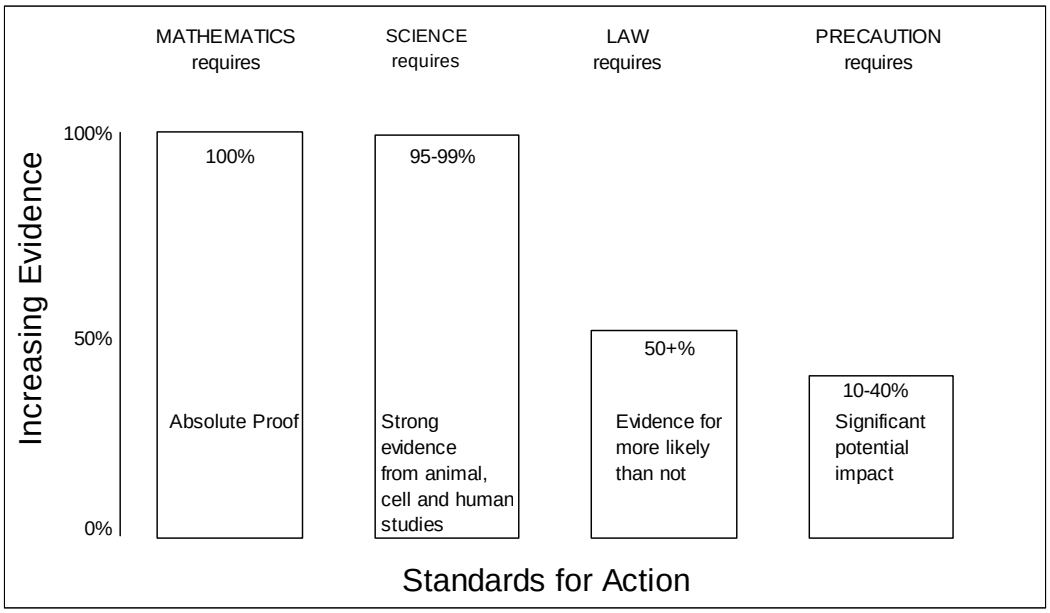


**Fig. 2:** Standards of evidence for decision-making, as used in mathematical proof, scientific investigations, legal and public health.

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 imple-menting 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' |

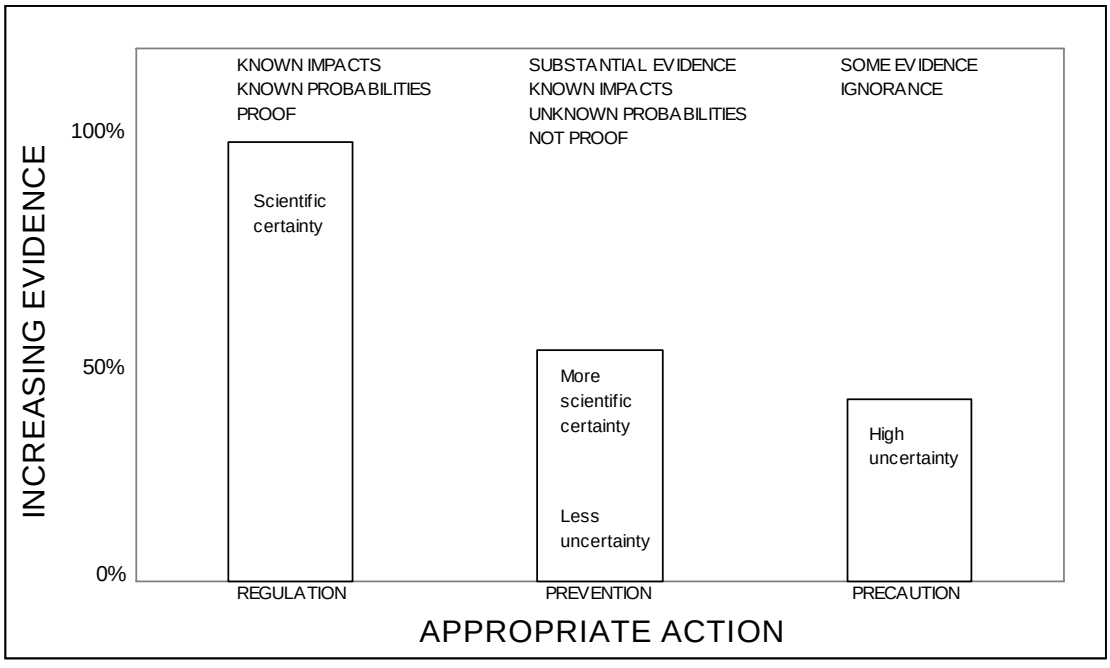


**Fig. 3:** Standards of evidence for precautionary/preventative action

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.

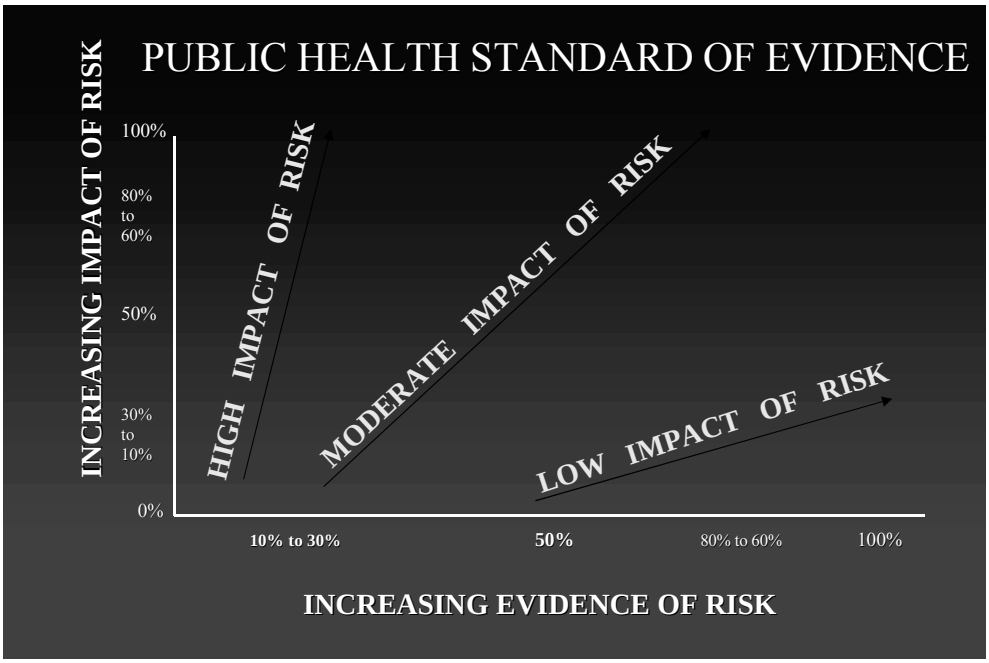


**Fig. 4:** A public health-based response must be relative to the magnitude of the potential impact of inaction. When the potential impact is high, action should be taken even when the evidence of risk is low.

## DEFINING NEW EXPOSURE STANDARDS FOR ELF AND RF ELECTROMAGNETIIC 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 µT), 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 safely 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 μT) range and above. ICNIRP and other standards that place public exposure limits as high as 1,000 mG (100 μT) 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 μT) range for all children, and over 1.4 mG (0.14 μT) 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 $\mu W/cm^2$, 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 μT) 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 μT) 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 μW/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.

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