COMMONWEALTH OF VIRGINIA

BEFORE THE

STATE CORPORATION COMMISSION

APPLICATION OF	)	
	)	
PATH ALLEGHENY VIRGINIA	)	
TRANSMISSION CORPORATION	)	CASE NO. PUE 2009-00043
	)	
For approval and certificate of electric	)	
Transmission facilities under Va. Code	)	
Sec. 56-46.1 and the Utilities Facilities Act,	)	
Va. Code sec. 65-265.1 et seq.	)	

DIRECT TESTIMONY AND EXHIBITS OF

PROFESSOR MARTIN BLANK

DEPARTMENT OF PHYSIOLOGY AND CELLULAR BIOPHYSICS

COLLEGE OF PHYSICIANS AND SURGEONS

COLUMBIA UNIVERSITY

SUBMITTED ON BEHALF OF RESPONDENTS

ALFRED T. GHIORZI AND IRENE A. GHIORZI

Q: Please state your name and business address.

Ans: My name is Martin Blank. I am a professor at Columbia University, as well as a consultant on scientific matters related to my research on electromagnetic fields (EMF). My consulting business address is 157 Columbus Drive, Tenafly, NJ 07670.

Q: Please summarize your educational and professional background.

Ans: I have PhD degrees from Columbia University and University of Cambridge. I am an Associate Professor in the Department of Physiology and Cellular Biophysics at Columbia University, College of Physicians and Surgeons, where I have been teaching and doing research for over 45 years. I have taught Medical Physiology to first year medical, dental and graduate students, including a year as Course Director in charge of 250 students. However, my primary responsibility has been to conduct research, and I have specialized in the effects of EMF on cell biochemistry and cell membrane function. My most recent research is on health related effects of electromagnetic fields (EMF), primarily on stress protein synthesis and enzyme function. I have lectured on my research around the world. In 2008, I was invited to address the Brazil Chamber of Deputies on biologically based EMF safety standards.

In my career, I have had appointments at Cambridge Univ (England), Weizmann Inst (Israel), UCal-Berkeley, Hebrew Univ (Israel), Monash Univ (Australia), Inst of Electrochemistry (USSR), Warsaw Univ (Poland), Tata Inst (India), Ben Gurion Univ (Israel), Univ of Victoria (Canada), Kyoto Univ (Japan). I have also held short term industrial research positions at California Research Corp, Esso Research, Unilever Research (2 labs in England; 1 in Netherlands), as well government appointments at the US Department of Defense, Office of Naval Research (in London and Arlington, Va). I have organized many scientific meetings, including two World

Congresses on Electricity and Magnetism in Biology and Medicine, four Erice (Italy) Courses on Bioelectrochemistry, and I started the Gordon Research Conference on Bioelectrochemistry. I have also served as Chairman of the Organic and Biological Division of the Electrochemical Society, President of the Bioelectrochemical Society, and President of the Bioelectromagnetics Society, as well as on the editorial boards of the *Journal of the Electrochemical Society* and *Bioelectrochemistry and Bioenergetics*. My publications include over 200 papers and reviews, as well as twelve edited books on electrical properties of biological systems, including the *Proceedings of the First World Congress on "Electricity and Magnetism in Biology and Medicine"* and *"Electromagnetic Fields: Biological Interactions and Mechanisms"*. I was one of the organizers of the Bioinitiative Report, as well as a contributor, and I edited the August 2009 special issue on EMF of the peer-reviewed journal *Pathophysiology*.

My many research and teaching roles, as well as professional activities with scientific societies and journals, have given me a broad perspective on scientific research related to EMF safety issues. The online Bioinitiative Report (2007) evaluating electromagnetic safety standards, to which I contributed a report on stress proteins, was cited by the European Parliament in their September 2008 decision to reconsider EMF safety standards. I recently edited a peer-reviewed update of the Bioinitiative Report in the scientific journal Pathophysiology. My Curriculum Vitae is Exhibit A.

Q: Have you testified in other proceedings involving EMF?

Ans: Yes. On January 10, 2006, I submitted testimony to the British Columbia Utilities Commission concerning a proposed 63 kV line wherein I stated that the EMF from that line would exceed the 3-4mG level within 70-80 feet of the line at typical power levels and even further at peak power levels. I testified that at 3-4mG the risk of leukemia in children is doubled.

I also testified that at 12mG, EMF blocks the inhibition of breast cancer cell growth by melatonin. A copy of that testimony is Exhibit B. New research since that testimony has added supporting evidence.

More recently, I submitted a letter to the Minister of the Environment of Poland regarding a planned power line when I was asked to summarize the health risk based on current scientific information. A copy of that letter is Exhibit C.

I have submitted prefiled testimony and plan to testify in the ongoing PSE&G Susquehanna-Roseland case.

Q: What is the purpose of your testimony?

Ans: The aim of my testimony is to discuss the medical and biological effects of exposure to EMF, and to show the scientific basis of our understanding of the health risks arising from exposure to the EMF associated with high voltage overhead transmission lines. Research on the biological processes affected by EMF and their consequences to human health has increased our understanding of biological mechanisms and underscored the need for greater protection of populations exposed to EMF, especially young children. Recent research has even provided a plausible biological explanation for the link between EMF and leukemia at the 3-4 milligauss (mG) level found in epidemiology studies.

I shall discuss published reports, primarily peer-reviewed epidemiological and laboratory studies, including my own research to show that:

- EMF affects many fundamental biochemical reactions in cells at field strengths in the range of observed epidemiology thresholds.
- Low levels of EMF stimulate stress protein synthesis (‘**the stress response’**), a

protective cellular mechanism that is activated by such established harmful stimuli as high temperature and acidity. **Activation of the stress response by low levels of EMF indicates that cells react to EMF as potentially harmful.**

- Since the code for synthesizing proteins is in the DNA structure, initiation of stress protein synthesis indicates that EMF interact with DNA. At relatively low intensities, EMF lead to DNA chain separation and protein synthesis. At higher intensities, EMF has been shown to damage DNA by causing strand breaks.
- It is generally agreed that cancer is associated with DNA damage (mutations). The link between EMF and DNA damage is seen in a study where children missing DNA repair genes have a much greater incidence of leukemia on exposure to EMF. This suggests that an inability to repair DNA damage caused by EMF can predispose a child to leukemia.
- EMF have also been associated with harmful biological effects in adults. EMF inhibit the secretion of melatonin which normally inhibits oxidation damage. Human breast cancer cells have been shown to grow faster in EMF, probably because of enhanced oxidation damage. EMF is also associated with an increase in the incidence of Alzheimer's disease and senile dementia, both of which increase linearly with the duration of EMF exposure. The large database of the study and the demonstrated dose-response of the data strengthen the reliability of the results, which described effects at relatively low EMF.
- There are plausible molecular mechanisms to account for the observed biological effects of low-level EMF. Biochemical studies have shown that EMF can accelerate electron transfer reactions, that electrons can be displaced in DNA, and that the resulting charging can cause DNA disaggregation. The physical properties (electron affinity, fluorescence depolarization) of particular bases (CTCT groups) in segments of DNA activated by EMF appear to contribute to these interactions.

In the past, EMF safety issues only considered the need to protect against acute large effects, such as electric shock, while chronic low level exposures were believed to be without consequence to health. Recent research has shown potentially harmful effects associated with chronic low level EMF (i.e. essentially AC magnetic fields) and a need for greater protection. Weak EMF pass easily into cells and constitute a significant risk, especially to children who spend many hours a day in schools and asleep at night in homes located near power lines. The magnetic fields cited in the report by J Michael Silva exceed biologically active levels at the edge of the right of way (ROW), as well as for some distance beyond the ROW, and they constitute a source of prolonged if not continuous exposure above biological thresholds. Since EMF increases with an increase in the electric power carried by the transmission line, addition of a 765 kV line would greatly increase the EMF exposure and the risk to health of people in the vicinity.

Q: Are living cells unusually sensitive to EMF (magnetic fields)?

Ans. Electric fields can make hair stand on end, but they do not effectively penetrate the skin or the membranes of cells, unless they are very high. (They are attenuated by a factor of over a million.) On the other hand, magnetic fields (EMF) pass easily into cells unattenuated and are therefore biologically far more active. Even weak EMF constitute a significant risk to living cells, especially to the rapidly growing cells in children. Low EMF thresholds in several biological systems have been published in peer-reviewed journals, and the first five values in the following Table are from our laboratory at Columbia University. The measured thresholds for changes in enzyme activity and in biosynthesis of stress proteins are in the range of the epidemiology threshold.

Biological EMF Thresholds (60Hz range)

Reactions:	Na,K-ATPase	2-3mG	Blank, Soo, Bioelectrochem 40:63-65, 1996
	Cytochrome C Oxidase	5-6mG	Blank, Soo, Bioelectrochem 45:253-259, 1998
	Malonic acid oxidation	1-2mG	Blank, Soo, Bioelectrochem 61:93-97, 2003
Stress response:	human cells (breast, HL60)	<8mG	Lin et al, J Cell Biochem 70:297-303, 1998
	human DNA + gene	<8mG	Lin et al, J Cell Biochem 81:143-148, 2001
Cancer cells:	Block inhibition by melatonin		
	(Breast cancer cells)	2-12mG	Liburdy et al, J Pineal Res 14:89-97, 1993
Epidemiology	leukemia/EMF	3-4mG	
ICNIRP	safety level	1000mG	

The above reactions are central to biological function. Electron transfer to cytochrome C oxidase is a critical step in converting foodstuff into ATP, the fuel used to power living cells. The Na,K-ATPase utilizes the ATP to drive the biological 'pump' that maintains the ionic composition of living cells. The stress response is a reaction to potentially harmful agents, such as high temperature, acidity, toxic metals, alcohol, etc, that leads to stimulation of DNA and the synthesis of stress proteins. Stimulation of the stress response by EMF shows that cells react to relatively low EMF levels as potentially harmful. EMF blockage of the inhibition of breast cancer cell growth by melatonin may be a significant factor in the mechanism of the disease.

Q: Please comment on the very high safety level set by ICNIRP.

Ans. From the above table of **EMF Thresholds,** it is clear that the safety level set by ICNIRP is approximately 3 orders of magnitude greater than the thresholds of the fundamental biological processes activated by EMF. ICNIRP interpret EMF safety as prevention of acute large effects,

such as electric shock, or large induced currents that can fire nerves. They believe that low level exposures, that they call non-thermal, do not cause biological effects and are without consequence to health. They may no longer be able to ignore the data showing that many fundamental biological processes are activated by low level EMF. The Parliament of the European Union voted in September 2008 to re-examine and readjust safety levels on the basis of measured biological effects. Activation of the stress response is certainly indicative of protective cellular responses to low level EMF.

Q: Describe the biological effects of EMF from your research on the stress response.

Ans. In 1994, my colleagues and I were the first to show that 60Hz EMF can activate the stress response, i.e., stimulate the synthesis of stress proteins, at low field strengths (reviewed in Goodman and Blank, Cell Stress and Chaperones 3:79-88, 2001). This observation has now been confirmed many times, in the RF as well as ELF ranges, and the research has provided insight into the biological mechanisms of EMF interactions. Following are some important conclusions based on this research:

- The stress response is a cellular reaction to potentially harmful environmental stimuli. The response should be seen as essentially the testimony of cells, in their own language, that EMF is potentially harmful.
- Activation in the non-thermal (i.e. low energy) ELF range as well as in the thermal RF range clearly indicates that non-thermal stimuli can activate biological effects similar to those activated by thermal stimuli. These data indicate the need to incorporate non-thermal biological effects in the evaluation of health risk and to revise recommended safe levels.
- The SAR, or specific absorption rate, is a measure of energy input that is used to estimate exposure at higher EMF frequencies. However, it is obviously unrelated to

biological thresholds and cannot serve as a measure of biological safety. The SAR should be replaced by a measure of biological function for purposes of risk assessment.

- Molecular studies of stress proteins have enabled us to determine the DNA segments that respond to EMF. We have transferred EMF responsive segments to artificial DNA constructs and activated other genes using EMF (Lin et al, J Cell Biochem 81:143-148, 2001). Columbia University holds the patent for this use of EMF responsive DNA as a magnetic trigger.
- The EMF responsive segments of DNA contain CTCT bases. The physical properties of the CTCT bases suggest a molecular mechanism for reacting to EMF and causing chain separation. The molecular insights from the research, together with related research on protein disaggregation reactions, have enabled us to account for stimulation of DNA by **relatively weak, environmental level EMF**. A plausible molecular mechanism has been proposed (Blank, Electromagnetic Biology and Medicine 27:3, 2008). See generally, Blank & Goodman, Electromagnetic fields stress living cells, Pathophysiology 16 (2009) 71-78. Exhibit D.

Q: Is there a plausible biological mechanism to link EMF with cancer?

Ans. Cancer is generally believed to result from DNA damage (mutations), and it is the ability of EMF to cause DNA damage by various mechanisms that suggests it as a possible cause. Cancer is actually many diseases and they generally take many years to develop. Childhood leukemia is atypical in appearing after only a few years, but children grow rapidly with active metabolisms that accentuate the molecular damage during protein and DNA synthesis as cells grow and divide. Natural repair mechanisms correct the errors that occur during normal growth, but they never correct 100% and in protein synthesis, at least one molecule is damaged for

every billion made. The errors build up and eventually damage a cell to the point where it cannot function properly.

For a long time it was thought that weak EMF do not have sufficient energy to activate, let alone damage, such large molecules as DNA. However, the stress response has shown activation at very low levels and DNA single strand breaks in the ELF range have shown the damage is possible. We now know that the charging of macromolecular complexes can lead to their disaggregation (Klug, Nobel lectures, 1982; Blank and Soo, Bioelectrochem Bioenerg 17:349-360.1987), and play a key role in membrane transport proteins, including ion channels. It is therefore possible for weak EMF to cause electron movements in DNA that redistribute charges and trigger large molecular rearrangements, e.g. DNA splitting. A possible sequence of steps may start with EMF displacing electrons and charging DNA segments, followed by disaggregation of DNA strands as a result of the charging.

It is interesting that the molecular properties of the CTCT bases on the EMF responsive DNA appear to be well suited for such a mechanism. The CTCT bases have low electron affinities, so electrons would be easily displaced by the EMF. Also, the CTCT (pyrimidine bases) and the GAGA (purine bases) on the complementary DNA strand result in a smaller area when they split, so disaggregation requires less energy and is more likely. Finally, a recent study of the lifetimes of excitation induced by ultraviolet (UV) light in different DNA structures [Schwalb and Temps, Science 322:243-245, 2008] has shown that CTCT bases dissipate energy 10 times faster than GAGA bases and therefore have a greater likelihood of reaching a breaking point, while the rest of the chain is a being held in place by the conjugate DNA strand. The CTCT bases are therefore likely to account for the ability of weak EMF in the extremely low frequency (ELF) range to stimulate protein synthesis. We know that at higher field strengths and higher

frequencies, EMF can cause dose dependent, single and double strand breaks in DNA (Lai and Singh, Bioelectromagnetics 18:156-165, 1997; and others in the online REFLEX Report, 2005). The relevance of DNA damage induced by EMF is reinforced in a recent epidemiology study where children missing the DNA repair genes were found to have a 4 fold greater incidence of leukemia from exposure to EMF as low as 1.4-1.8mG (Yang et al, Leukemia and Lymphoma 49: 2344-2350, 2008).

Q: Has EMF been linked to diseases other than leukemia?

Ans. EMF exposures have been shown to modify the tumor suppressing action of melatonin, an important natural secretion of the pineal gland in the brain that affects many natural processes, including sleep (Liburdy et al. J Pineal Res 14:89-97, 1993). Studies replicated in several labs show that low level EMF blocks the growth-inhibiting action of melatonin on human estrogen receptor-positive, breast cancer cells, as well as the near-complete blockage of the anticancer (chemotherapeutic) drug Tamoxifen. The threshold is between 2mG and 12mG.

A recent study from Switzerland (Hus et al, Amer J Epidemiology, 169:167-175, 2009) found that exposure to EMF from 220-380kV power lines is correlated with an increase in the incidence of Alzheimer's disease and senile dementia. The risk for those living within 50m compared to over 600m, increased with duration of exposure over 5, 10 and 15 years, with a doubling of the risk at 15 years. The fields were not measured in the study, but it is possible to estimate that the fields at 50m were in the range of 8-10mG, based on data published by the Bonneville Power Administration. The large population base in this study and the dose-response in the data make the association with EMF quite strong.

There are currently many studies of tumors in the head (gliomas, acoustic neuroma, parotid gland tumors) correlated with the use of cellphones. These are generally discussed in terms of

the radio frequency EMF that carries the cellphone signals, although there are low frequency components (12Hz, 217Hz) associated with the transmission that could be involved in interactions with DNA.

A recent study of the incidence of cancer in school teachers related the abnormally high incidence (odds ratio, OR = 9.8 vs expected OR = 2.78) to the radiofrequency noise on the 60Hz AC lines in the schoolrooms used by the teachers over a period of years. (Milham, Morgan Am. J. Ind. Med. 51:579-586, 2008). The unusually high cancer incidence was strongly associated with high frequency voltage transients, and it was suggested that this EMF may be a carcinogen similar to ionizing radiation. The data indicated that a single year of employment at this school increased a teacher's cancer risk by 21%.

The increasing frequency of reports of the association of diseases with EMF exposure along with a growing understanding of plausible mechanisms that involve changes in DNA have given greater credence to the links between EMF and disease. In the October 2009 issue of Medical Hypotheses, a paper by Sam Milham has demonstrated an association between 'diseases of civilization' (e.g. cardiovascular disease, malignant diseases) and electrification, by using 20th-century census data in the United States. He was able to examine this hypothesis because electrification proceeded at different rates in different states and there were large differences between rural and urban populations. This unusual retrospective epidemiology study is based on an unusually large volume of unbiased records that cover a long period of time. The data provide considerable support for links between EMF and disease.

Q: Is the 3-4mG level of EMF a 'safe' level?

Ans: The 3-4mG figure came from the epidemiology and laboratory research that led to the May 1999 National Institute of Environmental Health Sciences ("NIEHS") Report to the

Congress and their recommendation *"that the power industry continue its current practice of siting power lines to reduce exposures and continue to explore ways to reduce the creation of magnetic fields around transmission and distribution lines without creating new hazards."* The NIEHS-EMF review panel announced in June 1998 that magnetic fields should be considered a ***"****possible human carcinogen"*, and two pooled analyses (Greenland et al, Epidem 2000; Ahlbom et al, Brit J Cancer 2000), the first analyzing 15 major studies and the second 9 major studies, subsequently showed a statistically significant doubling of the risk of childhood leukemia at EMF exceeding 3-4mG. In addition to the epidemiological evidence**,** laboratory research (discussed earlier) provided evidence of biological reactions at this level.

The 3-4mG figure has been widely accepted as providing practical guidance for a reasonable level of safety, but it does not indicate safety below that level. A doubling of the risk at 3-4mG implies a lesser but still existing non-zero risk at levels just below that. Even when the median fields are below 3-4mG, the fields are higher half the time and lower half the time. Since there is no indication how high the values can go, they could exceed the thresholds of many additional biochemical reactions. There is no question that recent research has shown potentially harmful biological effects at low level EMF exposures for extended periods of time, and there is a need to reconsider EMF safety, as well as the 3-4mG level.

Q: How extensive is the health risk due to EMF levels near power lines?

Ans. The biochemical reactions listed in the Table of **Biological EMF Thresholds** (discussed earlier), and many other reactions that are inter-connected, would be stimulated within the ROW of existing and any added powerlines. This would cause changes in cellular energy production and utilization, as well as activation of DNA and the molecular damage associated with biosynthesis. There would also be increased risk of childhood leukemia, since the 3-4 mG level

set in the NIEHS report to Congress would be exceeded along the proposed powerline route where children would be exposed to an increased health risk. It is also clear that the added 765 kV line would raise the EMF well above the present level and extend its influence over a wider area, especially when the proposed 765 kV line would be constructed along side an existing 500 kV line and a 138 kV line. This is clearly not in line with the National Institute of Environmental Health Sciences recommendation *"that the power industry continue its current practice of siting power lines to reduce exposures and continue to explore ways to reduce the creation of magnetic fields around transmission and distribution lines without creating new hazards."*

There are related EMF issues that undoubtedly contribute to adverse biological effects.

- There is evidence that the radiofrequency RF 'noise' that accompanies a 60Hz signal (and that may become a feature of transmission lines with the addition of broadband) may cause such harmful effects as cancer and diabetes (Milham, Morgan, Am. J. Ind. Med. 51:579-586, 2008). A desirable optimal design would aim for as 'clean' a 60Hz sine wave as possible.
- The ground currents that account for a percentage of the AC return current are an indeterminate additional source of EMF exposure and depend on grounding, local circuitry, usage, etc.

Q: What is your opinion concerning the health effects of combining an overhead 765kV with an overhead 500kV AC line and an overhead 138kV AC line?

Ans: The health effects of EMF depend on the magnitude (and the direction) of the field, so the addition of a new line adds to the total EMF and the health risk of people exposed to the fields. The EMF along a power line can be minimized by dividing the current into three separate cables that transmit the current alongside each other 120° out of phase. I believe an additional line

would still result in a higher EMF. In any case, the estimated EMF are high, and among the highest I have seen proposed near residential areas. I believe that adding any HVAC lines to the existing 500 kV and 138 kV corridor would greatly increase the health risks of the exposed population.

Q: Please summarize your opinion of the health risk from EMF of the proposed PATH powerline.

Ans. There can be no doubt that the proposed addition of the 765kV line to the existing 500kV and 138kV lines would add to the level of EMF and thereby create an additional potential risk. This would be contrary to the recommendations of the May 1999 NIEHS Report to the Congress. The studies cited by J Michael Silva indicate a very high EMF at the edge of the ROW. Studies cited in my testimony indicate that even the weaker EMF beyond the ROW have the ability to cause significant changes in living cells by affecting fundamental biological processes, and predisposing them to the development of cancer and other diseases on prolonged exposures. It is therefore essential to minimize exposure of the population to EMF. While the errors in DNA that occur during cell division are most likely in rapidly growing children, recent research has shown that adults are also subject to the same risks on a somewhat longer timescale.

Because of the wide range of biological systems affected, the low response thresholds, the possibility of cumulative effects by repetitive stimulation and the inadequacy of exposure standards, it is urgent that the proposed powerline not be constructed as planned or that it be moved to a distance where the anticipated magnetic fields will not pose a hazard to the community. At the very least, **peak EMF levels should not exceed 3-4mG**. The recent study linking of the absence of DNA repair genes to EMF induced leukemia suggests that half that value, **1.4-1.8mG, would be a more prudent peak limit** to aim for.

Q. Have you read the direct testimony of Mr. Alfred T. Ghiorzi on the health effects of EMF's?

Ans: Yes

Q. If you have formed an opinion on it, what is that opinion?

Ans: Mr. Alfred T. Ghiorzi has presented an accurate, albeit, short summary of reports and studies dealing with the harmful effects of EMFs, including some of my own reports. While there are many more studies on EMFs and their harmful effects, he has reported the growing concern among many scientists who are engaged in research on EMF due to growing evidence of significant biological effects with health implications that occur at low levels of exposure.

Q: Does this conclude your testimony?

Ans: Yes.

PROFESSOR MARTIN BLANK

DEPARTMENT OF PHYSIOLOGY AND CELLULAR BIOPHYSICS

COLLEGE OF PHYSICIANS AND SURGEONS

COLUMBIA UNIVERSITY

Exhibit List

A. Curriculum Vitae

B. Testimony of Professor Martin Blank before the British Columbia Utilities Commission

C. Letter to the Ministry of the Environment, Poland concerning the proposed construction of power lines (2x400 Kv + 2 x200 Kv) near Kamionki

D. Electromagnetic Fields Stress Living Cells Martin Blank & Reba Goodman, Pathophysiology 16 (2009) 71-78

PROFESSOR MARTIN BLANK

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COLUMBIA UNIVERSITY

Exhibit A

CURRICULUM VITAE

CURRICULUM VITAE

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Columbia University, 630 West 168th Street, New York, NY 10032
(Tel: 212-305-3644; FAX: 212-305-5775; email: mb32@columbia.edu)

Personal

Born	February 28, 1933	New York, New York	
Married	Marion Sue Hersch	July 3, 1955	(3 children)

Education

1950-1954	City College of New York, BS Magna Cum Laude (Chemistry)
1954-1957	Columbia University, PhD (Physical Chemistry)
1957-1959	Cambridge University, England, PhD (Colloid Science)

Academic Appointments

1954-1955	Assistant in Chemistry, Columbia University
1955-1957	Research Fellow (Chemistry), Columbia University
1957-1959	Postdoctoral Research Fellow, Cambridge University, England
1959-1964	Instructor in Physiology, Columbia University
1964-1968	Assistant Professor of Physiology, Columbia University
1968-present	Associate Professor of Physiology and Cellular Biophysics, Columbia University

Other Appointments

Summer 1956	Chemist, California Research Corp, Richmond, CA.
Summer 1957	Chemist, Esso Research and Engineering Co, Linden, NJ.
Fall 1961	Research Fellow, Cambridge University, England
Summer 1964	Chemist, Unilever Research Lab, Cheshire, England
Summer 1966	Visiting Scientist, Polymer Dept, Weizmann Institute, Israel
Summer 1967	Chemist, Unilever Research Lab, Hertfordshire, England
Summer 1968	Visiting Scholar, Bioengineering Dept, University of California, Berkeley
Summer 1969	Research Chemist, Unilever Research Lab, Vlaardingen, Netherlands
1970	Visiting Professor, Pharmacology Dept, Hebrew University, Israel
1974-1975	Physiologist, Office of Naval Research, London, England
1982 (6 mo.)	Visiting Lecturer, Biochemistry Dept, Monash University, Australia
1984-1985	Biologist, Office of Naval Research, Arlington, VA
1986-1988	Part-time IPA Biologist, Office of Naval Research, Arlington, VA
1989 (May)	Visiting Professor, Acad Sci USSR, Inst Electrochemistry, Moscow, and Dept of Biophysics, Univ of Warsaw, Poland
1992 (Nov)	Visiting Professor, Tata Institute, Bombay, India
1995 (spring)	Visiting Professor, Dept of Chemistry, University of the Negev, Beersheba, Israel Visiting Scientist, Dept of Biology, University of Victoria, BC, Canada
2005 (July)	Visiting Professor, Dept of Theoretical Physics, Kyoto University, Japan

Honors

1953	Elected to Phi Beta Kappa, City College
1956	Elected to Sigma Xi, Columbia University
1955-1957	Consumers Union Research Fellowship, Columbia University
1957-1959	Postdoctoral Research Fellowship, National Heart Institute, Cambridge University
1960-1970	Research Career Development Award (USPHS), Columbia University
1975	Certificate of Appreciation, Office of Naval Research, London
1982 (June)	Distinguished Visiting Professor, Univ Western Australia
1984	Distinguished Lecturer in Physiology, Wayne State University
1985	Certificate of Commendation, Office Naval Research, Arlington
1987	Invited Lecturer, International Biophysics Congress, Jerusalem
1988	Invited Lecturer, Univ of Bologna, 900th Anniversary Symposium
1989 (May)	Visiting Professor, Acad Sci USSR, Institute of Electrochemistry, Moscow and Dept of Biophysics, University of Warsaw, Poland
1990	Certificate of Appreciation, The Electrochemical Society
	Yasuda Award, Bioelectrical Repair and Growth Society
1992	Invited Opening Speaker, First Congress of European Bioelectromagnetics Association, Brussels, Belgium
	(Nov) Visiting Professor, Tata Institute, Bombay, India
1992-1993	Editor-in Chief, Proceedings, First World Congress on "Electricity and Magnetism in Biology and Medicine"
1993-1999	American Editor, "Bioelectrochemistry and Bioenergetics"
	Certificate of Appreciation, American Chemical Society, Environment Division
1995 (spring)	Visiting Professor, Dept of Chemistry, University of Beersheba, Israel
	Visiting Scientist, Dept of Biology, University of Victoria, BC, Canada
1997	Plenary Lecturer, Second World Congress on "Electricity and Magnetism in Biology and Medicine", Bologna, Italy
2002	Plenary Lecturer, Bioelectromagnetics Society, Quebec, Canada.
2005 (July)	Visiting Professor, Dept of Theoretical Physics, Kyoto University, Japan
2005	Plenary Lecturer, Conference 'Biological Effects of Electromagnetic Fields', Kyoto, Japan
2007	Invited Lecturer, House of Deputies of Brazil on Biological Effects of EM Fields, Brazil

Areas of Research

General Experimental and Theoretical Areas:

Electromagnetic field effects on cells (stress response, enzyme reactions, DNA)
Membrane biophysics and transport mechanisms (active, passive, excitation mechanisms)
Biopolymers (surface and electrical properties of proteins, DNA)

Theoretical Models of Processes in Membranes and Biopolymers:

Electric and magnetic field effects on electron transfer reactions, enzymes, DNA
Ion fluxes in excitable membranes and ion gating in channels
Cooperative reactions in membranes, hemoglobin

Specific Biological Systems:

Electron transfer reactions: Belousov-Zhabotinski (oxidation of malonic acid), cytochrome oxidase
Enzymes: Na,K-ATPase, cytochrome oxidase, F_0F_1ATPase (effects of ions and EM fields)
Proteins: hemoglobin, red cell membrane, lung surfactant, Sciara salivary gland proteomics
Cells: red blood cells, sperm cells, HL60, Sciara salivary gland, E. coli
Membranes: red blood cells, sperm cells, membrane enzymes

Interfaces, Monolayers (proteins, lipids, ions), Bilayers:

Permeability (to water, gases, ions) and Rheology (elasticity, yield stress, flow)
Electrical effects: Adsorption, Electrode Noise, Surface Potential

Teaching

Faculty of Medicine - College of Physicians and Surgeons, Columbia University
- Medical Physiology - from 1961 to 1991
  - Lectures- physical biochemistry, membranes, transport.
  - Demonstrations- membrane properties, lung surfactant, analog computer.
  - Laboratory teaching including mammalian experiments.
- Course Director, 1989-1990
  - Computerized syllabus and administration (30 faculty, 310 students)
  - Introduced lab reports and new lab exercise
- Summer Science Teachers Program, 1995, 2000, 2004

Faculty of Pure Science - Graduate School of Arts and Sciences, Columbia University
- Basic Principles in Membrane Biophysics - Physical biochemistry (1970 - present)
  - membranes, electrical properties, ion transport
- Membrane Biophysics - Surfaces, membranes, channels, model systems.
- Graduate Seminar - Basic papers on membranes and transport.
- Control Mechanisms in Physiology - Lectures and lab on analog computer.
- Principles of Physiology - Lectures on biophysics (membranes, biopolymers)

Ettore Majorana Center, Erice, Italy-International School of Biophysics (Co-Director of 4 courses)
- 1981 Bioelectrochemistry I: Redox Processes
- 1984 Bioelectrochemistry II: Membrane Phenomena
- 1988 Bioelectrochemistry III: Charge Separation Across Biomembranes
- 1991 Bioelectrochemistry IV: Nerve-Muscle Function

National Medical School Review
- Lectures on Membranes, Nerve, Muscle

City University of New York (Graduate School)
- Surface Chemistry - Lectures on Surface Chemistry in Biology

Tata Institute, Bombay, India
- Course in Bioelectrochemistry

University of Beersheba (Department of Chemistry), Israel
- Course in Biophysics

Faculty Committees

Admissions, Faculty Council (and Executive Committee of the Faculty Council), By-Laws (Formulation of Stated Rules), First Year Faculty, Divisional Elections Commission, ad hoc tenure and department review committees.

Department of Physiology: Director of Seminar Program 1973-1984, Graduate Committee, Undergraduate Committee

Society Memberships

American Association for the Advancement of Science
Bioelectromagnetics Society
Bioelectrochemical Society
American Chemical Society (Colloid and Surface Chemistry Division)
Biophysical Society
Electrochemical Society (Organic and Biological Division)

Professional Activities

Editorial Boards

Bioelectrochemistry and Bioenergetics - Editorial Board, 1978 -1998;
Co-Editor, 1981 - 1987; North American Editor, 1993 - 1998
Journal of Electrochemical Society - Divisional Editor (Biology), 1978 -1991
Journal of Colloid and Interface Science - Advisory Board, 1978 -1981
Colloids and Surfaces (founded 1979) - Editorial Board, 1979 -1986
Pathophysiology, Guest Editor, special issue on Electromagnetic Fields, 2007-2009

Bioelectrochemical (BES) Society

Founding Member, March 1979; Vice President, 1979 - 1988; President, 1988 - 1992.
Co-organizer, 4th International Symposium, Woods Hole, MA, 1977.
Plenary Lecturer, Weimar, DDR, 1979.
Organizing Committee, Topical Lecturer, Jerusalem, 1981.
Scientific Committee, Invited Lecturer, Stuttgart, Germany, 1983; Bologna, Italy, 1985.
Liaison to Bioelectromagnetics Society Board, 1984-1996.
Organizing Committee, Invited Lecturer, Szeged, Hungary, 1987.
Honorary Committee, Invited Lecturer, Pont-a-Mousson, France, 1989; Bielefeld, Germany, 1992; Seville, Spain, 1994; Israel, 1996.
Organizer, Symposium on Biological Effects of Environmental EM Fields, Israel, 1996.
International Scientific Committee, Invited Lecturer, Denmark, 1998; Bratislava, Slovakia, 2001; Florence, Italy, 2003.

Bioelectromagnetics (BEMS) Society

Invited Lecturer, BEMS meetings, San Francisco, CA, 1985; Madison, WI, 1986; Stamford, CT, 1988; Quebec, Canada, 2002
Invited Speaker, BEMS Workshop on Cooperative Phenomena, Bethesda MD, 1988
Invited Speaker, BEMS Gene Workshop, Los Angeles, CA, 1993
Board of Directors, 1989-1992; liaison from BES 1985-1996.
President Elect, 1996; President, 1997-1998; Past President, 1998-1999
(Nominating Comm, Journal Comm, Public Affairs Comm)
Plenary Lecturer, Quebec, Canada, 2002
Symposium Organizer, Speaker (Bioelectromagnetic Mechanisms), Washington, DC, 2004
Symposium Organizer, Speaker (Precautionary Principle), Cancun, Mexico, 2006

BioInitiative Working Group (2005 - 2009)

An international group of scientists focused on EMF issues (including science, public policy and public health). **The BioInitiative Report,** entitled 'A Scientific Perspective on Health Risk of Electromagnetic Fields' was published on line on August 31, 2007. http://www.bioinitiative.org/report/index.htm
Author of Section 7, pp. 1-40. Evidence for Stress Response (Stress Proteins)
Peer- reviewed update of Report published in **Pathophysiology** 16 (2,3): 67-249, 2009.

World Congress on Electricity and Magnetism in Biology and Medicine

1992-3 Executive Committee, Site Selection Committee, Program Committee.
1992-3 Editor-in-Chief of Proceedings Volume, First World Congress
1994-7 Vice President, Executive Committee for Second World Congress
Chairman, Technical Program Committee, Second World Congress

International School of Biophysics, Erice, Italy; Co-Director and Lecturer in following:

Bioelectrochemistry I: Biological Redox Reactions and Energetics, 1981.
Bioelectrochemistry II: Membrane Phenomena, 1984.
Bioelectrochemistry III: Charge Separation Across Biomembranes, 1988.

Bioelectrochemistry IV: Nerve-Muscle Function, 1991.

Division of Colloid and Surface Chemistry, American Chemical Society

Symposium Chairman, "Surface Chemistry of Biological Systems", 1966; 1969.
VK LaMer Award Committee, 1971-1976, Chairman 1975-1976
Symposium Chairman, "Bioelectrochemistry", Miami, 1978; Cleveland, 1981; Washington, 1983; Denver, 1987.
Program Committee, Biology and Medicine, Chairman, 1979-1983.
Invited Lecturer, Colloid and Surface Science Symposium, Ann Arbor, 1987.
Invited Lecturer, Biological Interfacial Reactions Symposium, Atlanta, 1991.

Division of Organic and Biological Electrochemistry (Electrochemical Society)

Symposium Chairman, "Electrochemical Processes at Biological Membranes", Seattle, 1978
Officer: Secy-Treas 1979-1981; Vice Chair 1981-1983; Chair 1983-1985.
Board of Directors, Electrochemical Society, 1983-1985.
Symposium Chairman, "Electrical Double Layers in Biology", Toronto, 1985.
Invited Speaker, "Ion Transfer Across Interfaces", Boston, 1986.
Member, Interdivisional Committee on Chemical Sensors, 1984-1987.
Invited Speaker, "Redox and Interfacial Properties", Washington, 1991.

Gordon Research Conferences

Speaker, "Chemistry at Interfaces", 1963.
Speaker, "Sensory Transduction in Microorganisms", 1978.
Day Chairman and speaker, "Chemistry at Interfaces", 1974.
Organizing Chairman, First Conference "Bioelectrochemistry", 1980.
Day Chairman and speaker, "Bioelectrochemistry", 1982.
Speaker, "Bioelectrochemistry", 1984, 1986, 1988.
Speaker, "Protons and Membrane Reactions", 1985.
Speaker, "Physicochemical Aspects, Transport in Microvasculature", 1985.
Discussion Leader, "Bioelectrochemistry", 1990, 1992, 1994, 1996, 1998, 2000 (Oxford), 2002.
Speaker, "Bioelectrochemistry", 2004.

Selected Invitations to Meetings, Workshops, Panels (Departmental Seminars not listed)

Chairman and Lecturer, "Physical Chemistry of Interfacial Transport: Biological Interfaces - Flows and Exchanges" NY Heart Assoc, 1968
Chairman and Lecturer, "Transport and Rheology of Interfacial Layers", Internat Conf on Surface and Colloid Science, Jerusalem, Israel, 1981
Lecturer, "Structure and Function in Excitable Cells", Biophysical Congress Satellite Conf, Woods Hole, MA 1981
Lecturer, "Biophysics of Cell Surface", Arendsee, DDR, 1981
Guest Speaker, CIBA Foundation, Biological Effects of Electromagnetic Fields, London, 1984
Lecturer, "Electrochemical Growth Stimulation", International Society of Electrochemistry, Berkeley, CA, 1984
Lecturer, "Biophysics of Cell Surface", Heringsdorf, DDR, 1985
Lecturer, Bioelectrical Repair & Growth Soc, Utrecht, Netherlands, 1986
Lecturer, IEEE/Engineering in Biology and Medicine Soc, Fort Worth, TX, 1986
Lecturer, International Biophysics Congress, Jerusalem, Israel, 1987
Session Organizer, IEEE/Engineering in Biology and Medicine Soc, Boston, MA, 1987
Lecturer, Bioelectrical Repair & Growth Soc, Washington, DC, 1988
Lecturer, "Chemistry Physics of Electrified Interfaces", Bologna, Italy, 1988
Symposium Organizer, "Bioelectrochemistry", AIChE, Washington, DC, 1988
Speaker, BEMS Workshop on Cooperative Phenomena, Bethesda MD,1988

Speaker, National Research Council, "Health Effects of EM Fields", Washington, DC, 1989
Lecturer, "Electrobiology Today", Bologna, Italy, 1989

Speaker, California Department of Health Service Workshop on "ELF Field Exposure and Possible Health Effects", Berkeley, CA 1991
Speaker, FASEB Symposium on "Cancer, EM Fields and Biological Systems", Atlanta, GA 1991
Panelist, EPA- NYC Dept of Health Panel on Health Effects of EM Fields, New York, NY, 1991
Panelist, BEMS Workshop, Research Agenda, Health Effects of EM Fields, Milwaukee, WI, 1991
Opening Speaker, First Congress of European Bioelectromagnetics Association, Brussels, 1992
Speaker, EPRI Workshop on Neurobiology, Asilomar, CA, 1992
Speaker, FASEB Symposium, Biological Effects of Electromagnetic Fields, Anaheim, CA, 1992
Panelist, Molecular Electronics Symposium, First World Congress on Electricity and Magnetism in Biology & Medicine, Orlando, FL, 1992
Lectures (4) on Bioelectrochemistry of Proteins and Membranes, Tata Inst, Bombay, India, 1992
Plenary Lecture, Bioelectrochemical Society of India, Bombay, 1992
Speaker, Biophysical Society Public Policy Symposium on Biological Effects of Electromagnetic Fields, Washington, DC, 1993
Organizer, ACS Symp, Biological Effects of Environmental EM Fields, Denver, CO, 1993
Speaker, Helen Hayes Hospital, Haverstraw, NY, 1993
Speaker, Bell Labs (Series on EMF), Murray Hill, NJ 1993
Speaker, International Society of Molecular Electronics & Biocomputers, Gaithersberg, MD, 1993
Speaker, International Society of Toxicology, New Orleans, 1993
Speaker, ACS Conference on Chemical Health and Safety, Garden City, 1993
Panelist, Deadline Club, "Tension over High Tension", New York, 1993
Organizer and Speaker, Biophysical Society Workshop on Biological Effects of Environmental Electromagnetic Fields, New Orleans, LA, 1994
Speaker, ACS Conference on Environment, Hofstra University, NY, 1994
Lecturer, Hackensack Meadowlands Environment Center, Lyndhurst, NJ, 1994
Plenary Lecture, International Society of Electrochemistry, Portugal, 1994
Seminar Lecturer, Weizmann Institute, Rehovoth, Israel, 1995
Seminar Lecturer, Hebrew University-Hadassah Medical School, Jerusalem, Israel, 1995
Distinguished Lecturer, Wayne State University Medical School, Detroit, MI, 1995
Lecturer, Centre for Environmental Health, Victoria, BC, 1995
Lecturer, Victoria Cancer Clinic, Royal Jubilee Hospital, Victoria, BC, 1995
Speaker, First World Congress in Magnetotherapy, London, UK, 1996
Speaker, Applied Physics Division, CSIRO, Sydney, Australia, 1996
Speaker, Complementary Healing Conference, Baltimore, MD, 1996
Speaker, Vermont Law School Conference "Unplugged", Killington, VT, 1996
Speaker, 9th International Congress on Stress, Montreux, Switzerland, 1997
Speaker, Internat'l Comm Non-Ionizing Radiation Protection/ World Health Org (ICNIRP/WHO) Seminar, Bologna, Italy, 1997
Plenary Lecturer, Second World Congress on "Electricity and Magnetism in Biology and Medicine", Bologna, Italy, 1997
Speaker, EMF - Scientific and Legal Issues, Catania, Italy, 2002
Speaker, Chemistry and Biochemistry Departments, CUNY. 1998
Speaker, 10th International Congress on Stress, Montreux, Switzerland, 1999
Speaker, Electromed99, Norfolk, VA, 1999
Speaker, Tutorial on Magnetic Fields, Procter & Gamble, Cincinnati, 1999
Speaker, Potential Therapeutic Applications of Magnetic Fields, Vanderbilt Univ, 1999
Speaker, North American Academy of Magnetic Therapy, Los Angeles, 2000
Speaker, 3rd International Conference on Bioelectromagnetism, Slovenia, 2000
Speaker, Electromed2001, Portsmouth, VA, 2001

Speaker, European Bioelectromagnetics Association (EBEA) Conference on EMF. Helsinki, 2001
Plenary Lecturer, Bioelectromagnetics Society, Quebec, Canada, 2002
Speaker, XXVII URSI General Assembly, Maastricht, Netherlands, 2002
Speaker, EMF - Scientific and Legal Issues, Catania, Italy, 2002
Speaker, Physics Colloquium, University of South Florida, 2003
Speaker, RIFE Conference. Topic: Electromagnetic Fields and Living Cells. Seattle, 2004
Plenary Lecturer, Conference 'Biological Effects of Electromagnetic Fields', Kyoto, Japan, 2005
Speaker, Conference on EMF and the Precautionary Principle, Benevento, Italy, 2006
Keynote Speaker, Conference on Cell Towers and Wireless Technologies, Everett, MA, 2007
Invited lecturer on Biological Effects of EM Fields, Chamber of Deputies, Brasilia, Brazil, 2007
Invited lecturer, International Non-Ionizing Radiation and Health Workshop, Porto Alegre, Brazil, 2009

Grant Review Consultant

Office of Naval Research, Department of Defense
IPA Biologist, Manager of Membrane Electrochemistry ARI, 1986-1988
Chairman, Panel on Biological Sciences Div, August 1986
Member, Panel on Interdisciplinary Research, April 1979
Electric Power Research Institute, Palo Alto, CA
Member, Basic Sciences Advisory Committee, 1987-1991
National Institutes of Health
Radiation Study Section, 1991
(several ad hoc Study Sections and site visit committees)
National Science Foundation
US Army Research Office
US-Israel Binational Science Foundation
Petroleum Research Fund
Medical Research Council - Canada
Australian Research Grants Committee
Research Corporation (Providence, Rhode Island)
University and Polytechnic Grants Committee, Hong Kong
International Science Foundation (for Former Soviet Union), Washington, DC
Breast Cancer Research Program, University of California
US Army Medical Research and Materiel Command, Neurotoxin Exposure Program, AIBS
US Army Radiofrequency Radiation Research Program, AIBS

Consultant to various environmental groups on biological effects of electromagnetic fields (power frequency and radiofrequency)

PUBLICATIONS - Books, Reviews, Chapters

1. Blank M (1957) The Transfer of Monolayers through Surface Channels. **PhD Dissertation**, Chemistry Department, Columbia University, 54pp.
2. Blank M (1959) The Permeability of Monolayers to Carbon Dioxide and Oxygen. **PhD Dissertation**, Department of Colloid Science, Cambridge University, England, 105pp.
3. Blank M (1967) Editor, Symposium "Surface Chemistry of Biological Systems". **Journal of Colloid and Interface Science** 24:1-127.
4. Blank M and Britten JS (1970) Physical Principles in Monolayer and Membrane Permeation. in **"Physical Principles of Biological Membranes"**, edited by F Snell et al; Gordon & Breach, New York, pp 143-163.
5. Blank M (1970) Editor, **"Surface Chemistry of Biological Systems"**. Volume 7, "Advances in Experimental Medicine and Biology", Plenum Press, New York, 340pp.
6. Blank M (1972) The Measurement of Monolayer Permeability, in **"Techniques of Surface Chemistry and Physics"**, Volume I. Good, Stromberg, Patrick (eds); Marcel Dekker Inc., New York, pp 41-88
7. Blank M (1979) Monolayer Permeability. **Progress in Surface & Membrane Science** 13:87-139.
8. Blank M (1979) Surface Pharmacology: Drug Binding Equilibria and Ion Transport in Membrane Structures. **Pharmacology and Therapeutics** 7:313-328.
9. Blank M (1980) Editor, **"Bioelectrochemistry: Ions, Surfaces and Membranes"**, Advances in Chemistry, Volume 188, American Chem Soc, Washington, DC, 527pp.
10. Blank M (1981) Surface Pharmacology: Drug Binding Equilibria and Ion Transport in Membrane Structures, in **International Encyclopedia of Pharmacology and Therapeutics**, Inhibitors of Mitochondrial Functions, M Erecinska, DF Wilson (eds). Pergamon, New York, pp 19-34.
11. Milazzo G and Blank M (1983) Editors, **"Bioelectrochemistry I: Biological Redox Reactions"**, School of Biophysics, Erice, Italy. Plenum, New York, 348pp.
12. Blank M (1983) Transmembrane Potentials and Redox Reactions from the Physiological Point of View. in **"Bioelectrochemistry I: Biological Redox Reactions"**, G Milazzo, M Blank (eds), Plenum, New York, pp 227-247.
13. Blank M (1983) The Effects of Surface Compartments of Ion Transport Across Membranes. in **"Structure and Function in Excitable Cells"**, DC Chang, I Tasaki, WJ Adelman, HR Leuchtag (eds); Plenum, New York, pp. 435-449.
14. Blank M (1986) Editor, **"Electrical Double Layers in Biology"**, Plenum, NewYork, 319pp
15. Blank M (1987) The Surface Compartment Model: A Theory of Ion Transport Focused on Ionic Processes in the Electrical Double Layers at Membrane Protein Surfaces. **Biochimica et Biophysica Acta - Reviews on Biomembranes** 906:277-294.
16. Blank M and Findl E (1987) Editors, **"Mechanistic Approaches to the Interaction of Electric and Electromagnetic Fields with Living Systems"**, Plenum, New York, 439pp.
17. Milazzo G and Blank M (1987) Editors, **"Bioelectrochemistry II: Membrane Phenomena"**, International School of Biophysics, Erice, Italy. Plenum, New York, 543pp.
18. Blank M (1987) An Electrochemical Perspective on Excitable Membranes, Channels and Gating. in **"Bioelectrochemistry II: Membrane Phenomena"**, G Milazzo, M Blank (eds); Plenum, New York, pp. 431-456.
19. Blank M (1988) Recent Developments in the Theory of Ion Flow Across Membranes Under Imposed Electric Fields. In **"Modern Bioelectricity"**, AA Marino (ed); Dekker, New York, pp 345-364.
20. Markov M and Blank M (1988) Editors, **"Electromagnetic Fields and Biomembranes"**, Plenum, New York, 309pp.
21. Blank M (1990) Editor, **Syllabus for Human Physiology Course**, 13th Edition, Physiology Department, Columbia University, New York, 704pp.
22. Milazzo G and Blank M (1990) Editors, **"Bioelectrochemistry III: Charge Separation across Membranes"**, Plenum, New York, 337pp.
23. Blank M (1991) Membrane Transport: Insight from Colloid Science. in **"Interfacial Phenomena in**

Biological Systems", M Bender (ed). Dekker, New York, pp 337-366.

24. Blank M (1993) Electrochemistry of Nerve Excitation, **"Modern Aspects of Electrochemistry"** Number 24, edited by RE White et al, Plenum, New York, pp1-37.
25. Blank M (1993) Editor-in-Chief, Proceedings of First World Congress on **"Electricity and Magnetism in Biology and Medicine"**, San Francisco Press, 952pp.
26. Blank M and Vodyanoy I (1994) Editors, **"Biomembrane Electrochemistry"**, Advances in Chemistry Series of the American Chemical Society Press, 605pp.
27. Blank M (1994) An Electrochemical Model of Voltage Gated Channels. **Advances in Chemistry** 235:429-446.
28. Melandri BA, Milazzo G and Blank M (1994) Editors, **"Bioelectrochemistry IV: Nerve-Muscle Function"**. Life Sciences Volume 267, Plenum, New York, 376pp.
29. Blank M (1995) Editor, **"Electromagnetic Fields: Biological Interactions and Mechanisms"**, **Advances in Chemistry**, Volume 250, American Chemical Society Press, 512pp.
30. Blank M (1995) Biological Effects of Electromagnetic Fields: An Overview. **Advances in Chemistry** 250:3-12.
31. Blank M (1995) Electric Stimulation of Protein Synthesis in Muscle. **Advances in Chemistry** 250:143-153.
32. Blank M (1995) Electric and Magnetic Field Signal Transduction in the Membrane Na,K-ATPase. **Advances in Chemistry** 250:339-348.
33. Goodman R and Blank M (1995) The Biosynthetic Stress Response in Cells Exposed to Electromagnetic Fields. **Advances in Chemistry** 250:423-436.
34. Blank M (1997) Effects of Electromagnetic Fields on Cells as a Basis for Therapy. in **Proceedings of the First World Congress in Magnetotherapy**, pp. 151-156, London, May 1996.
35. Blank M (1997) Studies on the Mechanism of Electromagnetic Field Interactions with Cells: I-The Cellular Stress Response in Electromagnetic Fields; II-Electric and Magnetic Signal Transduction in a Membrane Protein. **Electric Power Res. Inst. Report TR-108947,** 99 pp.
36. Goodman R and Blank M (1998) Magnetic Field Induces Expression of hsp70. **Cell Stress and Chaperones** 3:79-88.
37. Goodman R and Blank M (2002) Insights into Electromagnetic Interaction Mechanisms. **Journal of Cellular Physiology** 192:16-22.
38. Blank M and Goodman R (2004) Initial Interactions in electromagnetic field-induced biosynthesis. **Journal of Cellular Physiology** 199:359-363.
39. Blank M (2008) Protein and DNA Reactions Stimulated by Electromagnrtic Fields. **Electromagnetic Biology and Medicine** 27: 3-23.
40. Blank M, editor (2009) Special issue of **Pathophysiology,** devoted to Electromagnetic Fields. Published on line, doi 10.1016/j.pathophys.2009. 10.02.002

PUBLICATIONS - Papers

1. LaMer VK and Blank M (1956) The Transfer of Surface Films through Surface Channels- Geometrical Factors. **Journal of Colloid Science** 11:608-616. 1956.
2. Blank M and LaMer VK (1957) The Mechanism of Transfer of Surface Films. Proceedings of the **Second International Congress on Surface Activity**, Vol II, pp 102-108.
3. Blank M and LaMer VK (1957) The Transfer of Monolayers through Surface Channels - II. Mechanism. **Journal of Physical Chemistry** 61:1611-1614.
4. Blank M and Roughton FJW (1960) The Permeability of Monolayers to Carbon Dioxide. **Transactions of the Faraday Society** 56:1832-1841.
5. Blank M (1961) The Effect of Vapors on Monolayer Permeability to Carbon Dioxide. **Journal of Physical Chemistry** 65:1698-1703.
6. Blank M and LaMer VK (1962) The Energy Barrier for Monolayer Penetration, in **"Retardation of Evaporation by Monolayers"**, edited by VK LaMer. Academic Press, New York, pp. 59-66.
7. Blank M (1962) The Permeability of Monolayers to Several Gases, in **"Retardation of Evaporation by Monolayers"**, edited by VK LaMer. Academic Press, New York, pp. 75-95.
8. Blank M and Rosano HL (1962) Surface Chemistry in a Biophysics Curriculum. **Journal of Chemical Education** 39:184-186.
9. Blank M (1962) Monolayer Permeability and the Properties of Natural Membranes. **Journal of Physical Chemistry** 66:1911-1918.
10. Blank M and Feig S (1963) Electric Fields across Water-Nitrobenzene Interfaces. **Science** 141:1173-1174.
11. Blank M and Ottewill RH (1964) Adsorption of Aromatic Vapors on Water Surfaces. **Journal of Physical Chemistry** 68:2206-2211.
12. Blank M (1964) An Approach to a Theory of Monolayer Permeation by Gases. **Journal of Physical Chemistry** 68:2793-2800.
13. Blank M and Britten JS (1965) Transport Properties of Condensed Monolayers. **Journal of Colloid Science** 20:789-800.
14. Blank M (1965) A Physical Interpretation of the Ionic Fluxes in Excitable Membranes. **Journal of Colloid Science** 20:933-949.
15. Blank M (1965) Some Effects due to the Flow of Current Across a Water Nitrobenzene Interface. **Journal of Colloid and Interface Science** 22:51-57.
16. Blank M (1966) Physical Models in Research on Biological Membranes. **Annals of the New York Academy of Sciences** 137:755-758.
17. Blank M and Essandoh SO (1967) The Surface Potential of a Di-Palmitoyl Lecithin Monolayer when Acetylcholine is in the Subphase. **Nature (London)** 215:286-287.
18. Blank M (1967) The Accumulation of Ions at Water Nitrobenzene Interfaces during Transference. in **"Physics and Physical Chemistry of Surface Active Substances"**, edited by Overbeek; Gordon and Breach, University Press Belfast, Vol II, pp 233-243.
19. Blank M (1967) The Process of Monolayer Permeation by Gases. in **"Physics and Physical Chemistry of Surface Active Substances"**, edited by Overbeek; Gordon and Breach, University Press, Belfast, Vol II, pp 969-979.
20. Blank M and Miller IR (1968) Transport of Ions Across Lipid Monolayers: Structure of Decylammonium Monolayers at the Polarized Mercury Water Interface. **Journal of Colloid and Interface Science** 26:26-33.
21. Miller IR and Blank M (1968) Transport of Ions Across Lipid Monolayers: Reduction of Polarographic Currents of Cu^{++} by Decylammonium Monolayers. **Journal of Colloid and Interface Science** 26:34-40.
22. Britten JS and Blank M (1968) Thallium Activation of the (Na^{+}-K^{+})-activated Adenosine Triphosphatase of Rabbit Kidney.**Biochimica Biophysica Acta** 159:160-166.
23. Blank M and Mussellwhite PR (1968) The Permeabilities of Adsorbed Monolayers to Water. **Journal**

of Colloid and Interface Science 27:188-192.

24. Blank M (1968) Introductory Remarks to New York Heart Association Symposium "Physical Chemistry of Interfacial Transport", **Journal of General Physiology** 52:187S-190S.
25. Blank M (1968) Monolayer, Interfacial Permeation. **Journal of General Physiology** 52:191S-208S.
26. Blank M, Goldstein AB and Lee BB (1968) Surface Properties of Lung Extract. **Journal of Colloid and Interface Science** 29:148-154.
27. Blank M (1969) Intermolecular Interactions in Newly Spread Serum Albumin Monolayers. **Journal of Colloid and Interface Science** 29:205-209.
28. Britten JS and Blank M (1969) The Action of Phloridzin and Sugars on the (Na+-K+)-Activated ATPase. **Journal of Membrane Biology** 1:238-247.
29. Blank M (1970) Transport Processes Across Liquid Interfaces and Monolayers. in **Permeability and Functions of Biological Membranes**, edited by L Bolis et al.; North Holland, Amsterdam, pp 177-184.
30. Blank M and Britten JS (1970) Determination of Yield Stress in Films of Lung Extract. **Journal of Colloid and Interface Science** 32:62-66.
31. Blank M and Britten JS (1970) Electron Flow at the Polarized Mercury-Water Interface in the Presence of Membrane Fragments Rich in Na^+-K^+-activated ATPase. **Journal of Membrane Biology** 2:1-16.
32. Blank M, Lucassen J and van den Tempel M (1970) The Elasticities of Spread Bovine Serum Albumin and Ovalbumin. **Journal of Colloid and Interface Science** 33:94-100.
33. Blank M and Lee BB (1971) Problems in the Study of Spread Films of Lung Extract. **Journal of Colloid and Interface Science** 36:151-152.
34. Werman R, Brookes N and Blank M (1971) The Stoichiometry of Transmitter-Receptor Interactions. **Experientia** 27:1120.
35. Blank M (1972) The Role of Surface Forces in Drug-Receptor Interactions. **Journal of Colloid and Interface Science** 38:470-476.
36. Blank M (1972) Cooperative Effects in Membrane Reactions. **Journal of Colloid and Interface Science** 41:97-104.
37. Miller IR, Britten JS and Blank M (1972) Polarographic Assay of p-Nitrophenyl Phosphatase Activity. **Analytical Biochemistry** 50:84-88.
38. Sweeney GD and Blank M (1973) Some Electrical Properties of Thin Lipid Films Formed from Cholesterol and Cetyl-trimethylammonium Bromide. **Journal of Colloid and Interface Science** 42:410-417.
39. Bach D, Britten JS and Blank M (1973) Polarographic Studies of Membrane Particles Containing Na-K ATPase, **Journal of Membrane Biology** 11:227-236.
40. Blank M and Britten JS (1973) Comments on the Molecular Basis of Fluidity in Membranes. **Chemistry and Physics of Lipids** 10:286-288.
41. Blank M, Lee BB and Britten JS (1973) The Effects of Cations on the Yield Stress of Ovalbumin Monolayers. **Journal of Colloid and Interface Science** 43:539-544.
42. Blank M (1973) The Oxygenation of Hemoglobin as a Problem in Surface Chemistry. **Journal of Colloid and Interface Science** 43:557-563.
43. Britten JS and Blank M (1973) Effects of Cations on Biologically Active Surfaces - Specific Binding Sites in the Na-K ATPase. **Journal of Colloid and Interface Science** 43:564-570.
44. Brookes N, Blank M and Werman R (1973) The Kinetics of the Conductance Increase Produced by GABA at the Membrane of Locust Muscle Fibers. **Molecular Pharmacology** 9:580-589.
45. Blank M (1974) "Physical Chemistry of Oscillatory Phenomena". **Faraday Symposium** 9:218.
46. Blank M, Soo L, and Britten JS (1974) Electrode Noise as a Source of Information on the Contact of Sperm Cells with Charged Surfaces. **Bioelectrochemistry and Bioenergetics** 1:293-300.
47. Blank M, Soo L, and Britten JS (1974) The Properties of Rabbit Sperm Membranes in Contact with Electrode Surfaces, **Journal of Membrane Biology** 18:351-364.
48. Blank M, Lee BB and Britten JS (1975) Adsorption Kinetics of Ovalbumin Monolayers. **Journal of Colloid and Interface Science** 50:215-222.
49. Blank M (1975) A Model for Calculating the Bohr Effect in Hemoglobin Equilibria. **Journal of Theoretical Biology** 51:127-134.

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50. Blank M and Britten JS (1975) Membrane Proteins and Membrane Models. **Biorheology** 12:271-274.
51. Blank M and Britten JS (1975) Effects of Cations on Biologically Active Surfaces - The Divalent Cation Selectivity of the Membrane Na-K Adenosine Triphosphatase. **Advances in Chemistry** 144:231-238.
52. Blank M (1975) Medicine for Physiologists. **The Physiologist** 18:525-528.
53. Miller IR, Britten JS and Blank M (1975) Binding of Ni^{++} to ATP: Polarographic Determination of Equilibrium and Rate Constants. **Bioelectrochemistry and Bioenergetics** 2:321-328.
54. Blank M (1975) Some Observations on Colloid Science and Molecular Biology. **Advances in Colloid and Interface Science** 5:277-279.
55. Blank M (1976) The Molecular Basis of Membrane Elasticity and Strength. in **"Membranes and Diseases"**, edited by L Bolis et al, North Holland Publ Co, Amsterdam, pp 81-88.
56. Blank M, Eisenberg W and Britten JS (1976) Ion Exhange Kinetics in Adsorbed Protein Film. **Bioelectrochemistry and Bioenergetics** 3:15-27.
57. Blank M (1976) Hemoglobin Reactions as Interfacial Phenomena. **Journal of the Electrochemical Society** 123:1653-1656.
58. Blank M and Soo L (1976) The Effect of Cholesterol on the Viscosity of Protein-Lipid Monolayers. **Chemistry and Physics of Lipids** 17:416-422.
59. Blank M (1976) Bioelectrochemistry and Biorheology - New Developments in Physiology. **The Physiologist** 19:477-483.
60. Blank M and Soo L (1976) The Adsorption of Serum Albumin on Rabbit Sperm Membranes. **Journal of Membrane Biology** 29:401-409.
61. Blank M and Lee BB (1976) Elasticities of Albumin Monolayers. in **Colloid and Surface Science**, Vol. V. Biocolloids, Polymers, Monolayers, Membranes and General Papers. Academic Press, New York, pp 239-249.
62. Britten JS and Blank M (1977) The Effect of Surface Charge on Interfacial Ion Transport. **Bioelectrochemistry and Bioenergetics** 4:209-216.
63. Blank M and Britten JS (1978) The Surface Compartment Model of the Steady State Excitable Membrane. **Bioelectrochemistry and Bioenergetics** 5:528-540.
64. Blank M, King RG, Soo L, Abbott RE and Chien S (1979) The Viscoelastic Properties of Monolayers of Red Cell Membrane Proteins. **Journal of Colloid and Interface Science** 69:67-73.
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PROFESSOR MARTIN BLANK

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COLLEGE OF PHYSICIANS AND SURGEONS

COLUMBIA UNIVERSITY

Exhibit B

TESTIMONY OF PROFESSOR MARTIN BLANK

BEFORE THE

BRITISH COLUMBIA UTILITIES COMMISSION

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January 10, 2006

Mr. Robert J. Pellatt,

Commission Secretary

British Columbia Utilities Commission

900 Howe Street, Box 250

Vancouver, BC V6Z 2N3 sent via Email: Commission.Secretary@bcuc.com

Dear Mr. Pellatt,

Re: **FortisBC Inc. Order No. G-114-05 / Project No. 3698407CPCN Application for Nk'Mip Substation and Transmission Line**

Mr. Hans Karow, Coalition to Reduce Electropollution (CORE), has asked me to provide testimony addressing the electromagnetic pollution issue associated with the proposed project cited above. As indicated in my CV (attached), I have spent many years studying the effects of low frequency electromagnetic fields (EMF) at both the cellular and molecular levels, and I have published extensively in peer reviewed journals.

Before addressing the main points, let me state that EMF from a 63kV power line will exceed the 3-4mG level within about 70-80 feet of the line, at typical power levels, based on the Bonneville Power Administration data. This level field will extend over an even wider range at peak power levels. Many biological systems are perturbed at relatively low EMF, but it has been shown that the risk of leukemia in children is doubled at the 3-4mG level. This field level is well within current safety limits, but the scientific basis of these limits is open to serious questions (Blank and Goodman, 2004) that challenge the capacity of these limits to be protective.

The main points I wish to emphasize are the following:

- recent epidemiological studies in the power frequency range suggest increased risk of leukemia associated with exposure to EMF

- current safety guidelines are not based on biological thresholds, and are many times above the levels that epidemiological studies have correlated with elevated risk of leukemia in children

- EMF thresholds of biological reactions are very low. Very low field strengths stimulate the stress response, the protective cellular reaction to potentially harmful stimuli

- the mechanism by which EMF cause changes in several well-documented biochemical systems involves interaction with electrons. Such a mechanism would affect many biological reactions, and possibly lead to cancer on interaction with DNA

Recent epidemiological studies indicate need for caution

Since the Wertheimer, Leeper paper of 1979, there have been many epidemiological studies of the effects of EMF in the power frequency range. Two recent meta analyses by groups of experts (Greenland et al, Epidem 2000; Ahlbom et al, Brit J Cancer 2000), of 15 and 9 major studies respectively, have shown a statistically significant doubling of the risk of childhood leukemia when exposures to low frequency EMF exceed 3-4mG. While the small number of cases of high exposure has resulted in a lack of statistical significance, the doubling of the risk of leukemia has persisted in many studies near the "significant" level. By pooling cases, it has been possible to demonstrate statistical significance. There is now quite general agreement that the epidemiological evidence indicates an association of EMF with childhood leukemia when exposures exceed 3-4mG.

Current safety guidelines are not based on biological mechanisms

In assessing the potential biological impact and risk of exposure, one would generally turn to the safety standards set by professional agencies such as ICNIRP and IEEE. However, the standards set by these agencies are unrelated to biological thresholds. They are based solely on the heating of tissue that results from the energy deposited by EMF. The energy deposition rate, the SAR, does not take into account many biological properties that change long before a change in the SAR can be detected. This fundamental flaw in the current standards makes them unreliable as a basis for safety:

- they assume that there are no biological reactions unless heating of cells occurs. The EMF thresholds discussed below, show that significant biological reactions occur in cells at very low EMF, in the absence of heating. These 'non-thermal' reactions raise an alarm regarding questions of safety.

- the standards were derived assuming that in EMF, the magnetic fields do not act directly, but only through the relatively weak electric fields they induce. This is not so. We have shown that both electric and magnetic fields can affect cells. In fact, magnetic fields penetrate cells far more effectively than electric fields at low frequency.

The SAR is a valid measure of energy deposition rate, but not of safety. It was derived at a time when all one could measure was temperature increase. Because of scientific advances, it is now possible to show many biological changes due to EMF that occur within the current safety guidelines. The current guidelines have been challenged by scientists, e.g., by an international commission that met in Catania, Italy in September 2002.

EM fields stimulate the cellular stress response

Regarding the question of safety, the most important observation is activation of stress protein synthesis in cells by EMF at both power and radio frequencies. The stress response occurs in reaction to a variety of potentially harmful influences in the environment, such as high temperature, toxic metal ions, alcohol, deviations of pH from neutrality, etc. For this reason, stimulation of the stress response by EMF can be seen as a direct answer by cells to the safety question. Cells react to EMF as a significant departure from a normal environment and as potentially harmful.

The stress proteins are the same whether stimulated by fields or by an increase in temperature, but the response to EMF requires much lower energy input. In Sciara salivary gland cells, the threshold energies of the EMF and thermal stimuli needed to evoke a stress response differ by 14 orders of magnitude, as shown in the Table below.

ENERGY to STIMULATE STRESS RESPONSE

Form of Energy	Stimulus	Energy Density (joules/m^3)
Magnetic	**8mG**	**2.6×10^{-7}**
Thermal	**5.5°C**	**$2.3 \times 10^{+7}$**

In addition to the stress response, many biological reactions, such as enzyme systems and electron transfer reactions, are affected by weak EMF. Low thresholds have been measured in several systems, and the values have been published in peer review journals. The Table below shows that the measured thresholds for changes in reaction rates of enzymes, the BZ reaction (oxidation of malonic acid), and reactions in DNA leading to biosynthesis of stress proteins, are in the range of cut-off thresholds in epidemiological studies. The table also has an entry for EMF needed to block the inhibition of breast cancer cell growth by melatonin. That study has been replicated in six labs, and it shows that a low EMF of 12mG blocks the growth-inhibiting action of melatonin on human estrogen receptor-positive, breast cancer cells, as well as the near complete blockage of the anticancer (chemotherapeutic) drug Tamoxifen. An EMF of 2mG has no effect, indicating that the threshold for an effect on these cancer cells lies between 2mG and 12mG.

Biological EM Field Thresholds (power frequency range)

Reactions:	**Na,K-ATPase**	**2-3mG**
	Cytochrome C Oxidase	**5-6mG**
	BZ (redox) reaction	**1-2mG**
DNA:	**Stress proteins (HL60 Cells)**	**<8mG**
	Stress proteins (Sciara Cells)	**<8mG**
Cells:	**Block inhibition by melatonin (Breast cancer cells)**	**2-12mG**
	Epidemiology threshold (leukemia)	**3-4mG**

Stimulation of the stress response by EMF shows that they activate DNA as the first step in protein synthesis. Several labs have shown that DNA can conduct electrons within its structure. Therefore, it appears possible for EMF to activate DNA by generating repulsive forces when interacting with electrons in DNA. We have shown that specific regions of DNA are associated with the response to EMF, and inactivating these sequences by removal or mutation eliminates the response to EMF. Inserting these DNA sequences into an artificial construct containing a gene makes the gene EMF-responsive. In brief, our understanding of mechanism has reached the point where we have identified an EMF sensitive DNA sequence, have transplanted it and have reactivated it with EMF. (We have obtained a patent for this process.) That experiment, together with the breast cancer cell study indicate that EMF can enter into health related mechanisms at very low field strengths.

Recommendation

Based on recent research on biological changes induced by EMF, it is wise and prudent to recommend minimizing exposure by all reasonable methods, especially of school age children, with the aim of being below 3-4mG at peak power levels. **ALARA** (As Low As Reasonably Achievable) has been a policy with regard to radiation safety, and the European Union has adopted a related measure, the **Precautionary Principle**, as a general approach to environmental issues. Italy and Austria have applied this approach to EMF, and I have organized a symposium on the Precautionary Principle for the next meeting of the Bioelectromagnetics Society.

On request, I am prepared to provide additional information and clarification. Please feel free to contact me via e-mail mb32@columbia.edu , or telephone provided above.

Martin Blank, PhD
Associate Professor of Physiology and Cellular Biophysics
Columbia University

Enclosure: Curriculum Vitae

CC: Mr. George Isherwood, Director Reg. Affairs, FortisBC George.Isherwood@fortisbc.com
Mr. Hans Karow, CORE hkarow@shaw.ca

PROFESSOR MARTIN BLANK

DEPARTMENT OF PHYSIOLOGY AND CELLULAR BIOPHYSICS

COLLEGE OF PHYSICIANS AND SURGEONS

COLUMBIA UNIVERSITY

Exhibit C

LETTER TO MINISTRY OF THE ENVIRONMENT, POLAND

CONCERNING THE PROPOSED CONSTRUCTION OF POWER LINES

(2X400Kv + 2 X200 Kv) NEAR KAMIONKI

Columbia University, College of Physicians and Surgeons

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New York, NY 10032 **EMAIL: mb32@columbia.ed**

June 18, 2009

Ministry of the Environment, Poland (sent to minister@mos.gov.pl, info@mos.gov.pl)

RE: the proposed construction of power lines (2x 400kV + 2x 220 kV) near Kamionki

Dear Minister,

I have been asked by Krzysztof Kuklinski of Kamionki to write to you regarding the health effects due to the electromagnetic fields (EMF) in the proposed project. After stating my credentials as a scientist, I shall summarize the latest scientific information regarding health risks due to exposure to EMF from power lines. It should become clear from the recent scientific studies that the project, as planned, would constitute a potential health hazard of considerable magnitude. The project should be changed to minimize these risks.

I am a scientist with research experience in bioelectromagnetics, and a professor of physiology and cellular biophysics.

- My degrees are: BS in chemistry, City College 1954; PhD in physical chemistry, Columbia Univ 1957; PhD in colloid science (an interdisciplinary mix of biology, physics, chemistry), Cambridge Univ 1959. I have published over 200 papers on EMF, cellular stress response, charge transport enzymes.
- I am Associate Professor of Physiology and Cellular Biophysics, at the College of Physicians and Surgeons, Columbia University, and have taught Graduate Physiology and Medical Physiology to medical, dental, graduate students. I was Course Director in both courses.
- My visiting appointments at other universities and research institutes include Cambridge-England; Weizmann Inst-Israel; UCalifornia-Berkeley; Monash Univ-Australia; Biophysics Dept, U Warsaw; Tata Inst-India; and Theoretical Physics Dept, Kyoto Univ, Japan.
- I was a Liaison scientist for the US Office of Naval Research (ONR) in London, and directed a research program for ONR out of Arlington, VA.
- I have done research in five different industrial laboratories in the US and Europe.
- I have been President of Bioelectromagnetics Soc, Bioelectrochemistry Soc; Chairman of the Biology Div (Electrochemistry Soc); editor of scientific journals; reviewer of scientific papers for publication and proposals for funding; evaluating research laboratories for US government agencies; testifying as expert witness. In 2008, I was invited to address the Brazil Chamber of Deputies on EMF safety.
- After evaluating the scientific evidence and potential health risks of exposure to EMF, I joined with other scientists in signing the **Benevento Resolution (2006)** and the related **Catania Resolution (2002)**.

- I was one of the organizers of the **BioInitiative Working Group (2006-2009),** an international group of scientists who reviewed the science and safety aspects of EMF in a report that was published online in August 2007 at http://www.bioinitiative.org/report/index.htm The recommendations of this report were cited by the European Parliament (September 2009) as a reason to reexamine EMF safety standards.

Let me start my summary of scientific information about health risks of EMF in 1998, after the National Institute of Environmental Health Sciences (NIEHS) NIEHS-EMF had conducted a year long review. In the Report to the US Congress (NIH publication No. 98-3981, 1998), the Director acknowledged the concerns about exposure to EMF in his statement that "...ELF-EMF exposure cannot be recognized at this time as entirely safe... passive regulatory action is warranted such as a **continued emphasis on educating both the public and the regulated community on means aimed at reducing exposures**." He specifically recommended limiting exposure. "The NIEHS suggests that the power industry continue its current practice of **siting power lines to reduce exposures and continue to explore ways to reduce the creation of magnetic fields around transmission and distribution lines without creating new hazards**." I have used bold print to emphasize the clear recommendation to reduce magnetic fields.

There can be no doubt that epidemiological evidence supports the increased health risk associated with EMF. Since the NIEHS review, two pooled analyses (Greenland et al, Epidemiology 11:624-634, 2000; Ahlbom et al, British J Cancer 83:692-698, 2000) have confirmed a statistically significant doubling of the risk of childhood leukemia when exposures exceed 3-4mG. The magnetic fields near Kamionki can be estimated to be comparable to the EMF levels cited by Bonneville Power Administration (1994) as about 58mG for a 230kV powerline and about 87mG for a 500kV powerline. The potentially harmful effects of fields of this magnitude would extend for a considerable distance and cause many residents, including children, to be exposed to EMF greatly in excess of 3-4mG.

In addition to the risk of leukemia in children, such fields have been shown to have adverse effects on the health of adults as well. A recent study from Switzerland (Hus et al., Amer J Epidemiology, January 2009) found that exposure to 220-380 kV powerlines is correlated with an increase in the incidence of Alzheimer's disease and senile dementia. The risk of those living within 50m compared to over 600m, increased with duration of exposure over 5, 10 and 15 years, with a doubling of the risk at 15 years. These fields were probably in the range of 8-10mG, based on the Bonneville Power Administration data.

Low level EMF have also been shown to contribute to cancer cell growth through inhibition of the tumor suppressing action of melatonin (normally secreted by the pineal gland in the brain). The original study (Liburdy et al, J Pineal Research 1993), now replicated in several labs, showed that 12mG blocks the growth-inhibiting action of melatonin on human breast cancer cells, as well as the near-complete blockage of the

anticancer (chemotherapeutic) drug Tamoxifen. A field strength of 2mG had no effect, indicating that the threshold for harmful effect lies between 2mG and 12mG.

People do not sense these low magnetic fields, so they are believed to be without any health effect. That is not so. There are significant biological effects of EMF on many important cellular systems even at very low fields. Below is a table of measured thresholds on a variety of cells and sub-cellular components from our published studies at Columbia University.

Biological EMF Thresholds

Biochemical Reactions	**EMF**	**Reference (authors, year)**
Enzyme Reaction Rates		
Na, K-ATPase	2 – 3 mG	Blank and Soo, 1996
cytochrome oxidase	5 – 6 mG	Blank and Soo, 1998
Oxidation-reduction reaction rate		
Belousov-Zhabotinski reaction	below 5mG	Blank and Soo, 2001
Stress response, stimulation of DNA		
HL60, Sciara, yeast cells	below 8mG	Goodman et al, 1994
Breast cells (HTB124, MCF7)	below 8mG	Lin et al, 1998

These biochemical reactions are central to biological function. Electron transfer to cytochrome oxidase is a critical step in converting foodstuff into ATP, the fuel used to power all living cells. The Na,K-ATPase utilizes the ATP to drive the biological 'pump' that maintains the ionic composition of all living cells.

In the above table, the ***'stress response'*** is a reaction to potentially harmful agents that is particularly important. Since it occurs in reaction to potentially harmful influences in the environment, such as high temperature, toxic metals, alcohol, etc, stimulation of the stress response by EMF can be seen as a direct answer by cells to the safety question. It shows that ***cells react to relatively low EMF levels as potentially harmful*** (Goodman and Blank, Cell Stress and Chaperones, 1998).

The ***stress response*** also demonstrates that EMF interacts with DNA at very low intensities. EMF has also been shown to cause damage to DNA at higher intensities. Since the stress response is initiated when the two strands of DNA come apart to start protein synthesis, it is clear that EMF leads to DNA chain separation. EMF interaction with DNA is important, because cancer is associated with changes in DNA (mutations) and a probable explanation of epidemiology results. The relevance of DNA damage induced by EMF is seen in a recent epidemiology study where children missing the genes needed to repair DNA were found to have a 4 fold greater incidence of leukemia from exposure to EMF as low as 1.4-1.8mG (Yang et al, 2008).

There are many recent studies showing effects of EMF on DNA, including the **REFLEX project (2004)** that is the work of 12 research laboratories in seven European countries. That report finds that both power and radio frequency EMF ***activate the stress***

response and cause DNA breaks at relatively low exposures. A summary of the final report can be found online at
http://www.verum-foundation.de/www2004/html/pdf/euprojekte01/REFLEX_ProgressSummary_231104.pdf
A more recent summary and review of research can be found in the Bioinitiative report that was published online at http://www.bioinitiative.org/report/index.htm
Scientific papers (e.g., Lai and Singh, 1995) point to links between EMF exposure and damage to DNA, so it is not unreasonable to see a connection between EMF and leukemia found in epidemiology studies. There is also research showing plausible mechanisms whereby weak EMF can stimulate DNA (Blank, Electromagnetic Biology and Medicine, 2008) and initiate the changes that can lead to disease.

From what we have learned about EMF interactions with cells, there is a strong scientific basis showing harmful effects on cells due to relatively low EMF. Therefore, there are good reasons for limiting exposure, especially of children. Because of the wide range of biological systems affected, the low response thresholds, the possibility of cumulative effects by repetitive stimulation and the inadequacy of exposure standards, it is urgent that the proposed powerline be moved to a distance where the anticipated magnetic fields will not pose a hazard to the community.

Sincerely yours,

Martin Blank, PhD
Associate Professor of Physiology and Cellular Biophysics
Bioelectromagnetics Society President, 1997-1998

PROFESSOR MARTIN BLANK

DEPARTMENT OF PHYSIOLOGY AND CELLULAR BIOPHYSICS

COLLEGE OF PHYSICIANS AND SURGEONS

COLUMBIA UNIVERSITY

Exhibit D

ELECTROMAGNETIC FIELDS STRESS LIVING CELLS

MARTIN BLANK & REBA GOODMAN,

PATHOPHYSIOLOGY 16 (2009) 71-78

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Electromagnetic fields stress living cells

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Received 30 January 2009; accepted 30 January 2009

Abstract

Electromagnetic fields (EMF), in both ELF (extremely low frequency) and radio frequency (RF) ranges, activate the cellular stress response, a protective mechanism that induces the expression of stress response genes, e.g., HSP70, and increased levels of stress proteins, e.g., hsp70. The 20 different stress protein families are evolutionarily conserved and act as 'chaperones' in the cell when they 'help' repair and refold damaged proteins and transport them across cell membranes. Induction of the stress response involves activation of DNA, and despite the large difference in energy between ELF and RF, the same cellular pathways respond in both frequency ranges. Specific DNA sequences on the promoter of the HSP70 stress gene are responsive to EMF, and studies with model biochemical systems suggest that EMF could interact directly with electrons in DNA. While low energy EMF interacts with DNA to induce the stress response, increasing EMF energy in the RF range can lead to breaks in DNA strands. It is clear that in order to protect living cells, EMF safety limits must be changed from the current thermal standard, based on energy, to one based on biological responses that occur long before the threshold for thermal changes.

Keywords: DNA; Biosynthesis; Electromagnetic fields; ELF; RF

1. Electromagnetic fields (EMF) alter protein synthesis

Until recently, genetic information stored in DNA was considered essentially invulnerable to change as it was passed on from parent to progeny. Mutations, such as those caused by cosmic radiation at the most energetic end of the EM spectrum, were thought to be relatively infrequent. The model of gene regulation was believed to be that the negatively charged DNA was tightly wrapped up in the nucleus with positively charged histones, and that most genes were 'turned off' most of the time. Of course, different regions of the DNA code are being read more or less all the time to replenish essential proteins that have broken down and those needed during cell division.

New insights into the structure and function of DNA have resulted from numerous, well-done laboratory studies. The demonstration that EMF induces gene expression and the synthesis of specific proteins [1,2] generated considerable controversy from power companies, government agencies, physicists, and most recently, cell phone companies. Physicists have insisted that the reported results were not possible because there was not enough energy in the power frequency range (ELF) to activate DNA. They were thinking solely of mechanical interaction with a large molecule and not of the large hydration energy tied up in protein and DNA structures that could be released by small changes in charge [3]. Of the biologists who accepted such results [4], most thought that the EMF interaction originated at, and was amplified by, the cell membrane and not with DNA.

It is now generally accepted that weak EMF in the power frequency range can activate DNA to synthesize proteins. An EMF reactive sequence in the DNA has been identified [5] and shown to be transferable to other gene promoters [6]. This DNA sequence acts as an EMF sensitive antenna

Abbreviations: EMF, electromagnetic fields; Hz, hertz; ELF, extremely low frequency; RF, radio frequency; MAPK, mitogen activated protein kinase; ERK1\2, extracellular signal regulated kinase; JNK, c-Jun-terminal kinase p38MAPK; SAPK, stress activated protein kinase; NADH, nicotinamide adenine dinucleotide dehydrogenase; ROS, reactive oxygen species.

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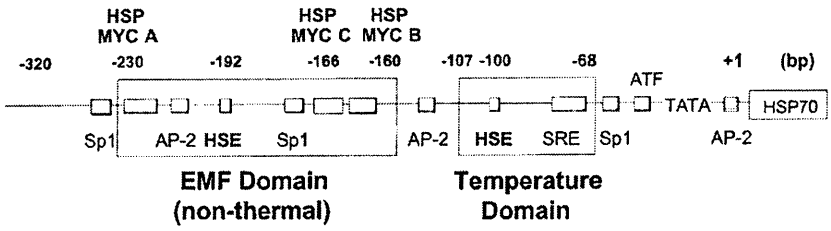

Fig. 1. Diagram of the HSP70 promoter showing the two different DNA sequences that have been identified as activated by EMF (non-thermal) and by thermal stimuli, respectively. The EMF domain contains three nCTCTn consensus sequences (electromagnetic response elements; EMRE), and differs from the consensus sequence (nGAAn) in the temperature or thermal domain.

that responds to EMF when transfected into reporter genes. Research at the more energetic levels of power frequency [7] and in the RF [8] ranges has shown that exposure to EMF can lead to breaks in the DNA strands. Therefore, DNA can no longer be considered unaffected by environmental EMF levels. It can be activated and damaged by EMF at levels that are considered safe [9]. The vulnerability of DNA to environmental influences and the possible dangers associated with EMF, had been underscored by discovery of EMF activation of the cellular stress response in the ELF range [10,11]. The cellular stress response is an unambiguous signal by the cell that EMF is potentially harmful.

2. Physiological stress and cellular stress

Discussions of physiological stress mechanisms usually describe responses of the body to pain, fear, 'oxygen debt' from muscle overexertion. These responses are mediated by organ systems. For example, the nervous system transmits action potentials along a network of nerves to cells, such as adrenal glands, that release rapidly acting agents such as epinephrine and norepinephrine and slower acting mineralocorticoids. These hormones are transported throughout the body by the circulatory system. They mobilize the defenses to cope with the adverse conditions and enable the body to 'fight or flee' from the noxious stimuli. The defensive actions include changes in heart rate, breathing rate, muscle activity, etc.

In addition to the responses of organ systems, there are protective mechanisms at the cellular level known as the cellular stress response. These mechanisms are activated by damage to cellular components such as DNA and protein [12], and the responses are characterized by increased levels of stress proteins [13] indicating that stress response genes have been upregulated in response to the stress.

The first stress response mechanism identified was the cellular reaction to sharp increases in temperature [14] and was referred to as 'heat shock', a term that is still retained in the nomenclature of the protective proteins, the hsps, heat shock proteins. Stress proteins are designated by the prefix 'hsp' followed by a number that gives the molecular weight in kilodaltons. There are about 20 different protein families ranging in molecular weight from a few kilodaltons to over 100 kD, with major groups of proteins around 30 kD, 70 kD and 90 kD.

Research on the 'heat shock' response has shown that hsp synthesis is activated by a variety of stresses that are potentially harmful to cells, including physical stimuli like pH and osmotic pressure changes, as well as chemicals such as alcohol and toxic metal ions like Cd^{2+}. EMF is a recent addition to the list of physical stimuli. It was initially shown in the power frequency (extremely low frequency, ELF) range [13], but shortly afterwards, radio frequency (RF) fields [15] and amplitude modulated RF fields [16] were shown to activate the same stress response.

Studies of stress protein stimulation by low frequency EMF have focused on a specific DNA sequence in the gene promoter that codes for hsp70, a major stress protein. Synthesis of this stress protein is initiated in a region of the promoter (see Fig. 1) where a transcription factor known as heat shock factor 1 (HSF-1) binds to a heat shock element (HSE). This EMF sensitive region on the HSP70 promoter is upstream from the thermal domain of the promoter and is not sensitive to increased temperature. The binding of HSF-1 to HSE occurs at −192 in the HSP70 promoter relative to the transcription initiation site. The EMF domain contains three nCTCTn myc-binding sites −230, −166 and −160 relative to the transcription initiation site and upstream of the binding sites for the heat shock (nGAAn) and serum responsive elements [5,6,17,18]. The electromagnetic response elements (EMREs) have also been identified on the c-*myc* promoter and are also responsive to EMF. The sensitivity of the DNA sequences, nCTCTn, to EMF exposures has been demonstrated by transfecting these sequences into CAT and Luciferase reporter genes [6]. Thus, the HSP70 promoter contains different DNA regions that are specifically sensitive to different stressors, thermal and non-thermal.

Induction of increased levels of the major stress protein, hsp70, by EMF is rapid, within 5 min. Also it occurs at extremely low levels of energy input, 14 orders of magnitude lower than with a thermal stimulus [10]. The far greater sensitivity to EMF than to temperature change in elevating the protective protein, hsp70, has been demonstrated to have potential clinical application, preventing injury from ischemia reperfusion [19–21]. George et al. [22] have shown the non-invasive use of EMF-induced stress proteins improved hemodynamic parameters during reperfusion

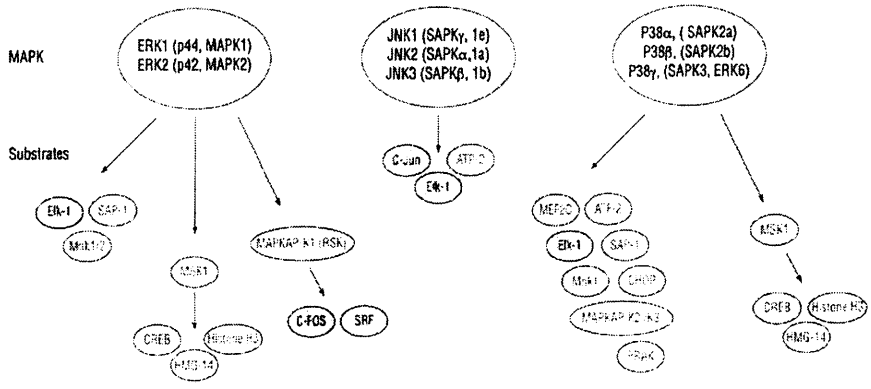

Fig. 2. The four mitogen activated protein kinase (MAPK) signaling cascades identified to date are: extracellular signal regulated kinase 1/2 (ERK), c-Jun-terminal kinase (JNK), p38MAPK and stress activated protein kinase (SAPK). Elements of the three MAPkinase pathways that have been identified as activated by EMF are shown as the shaded circles.

following ischemia. This effect occurred in the absence of measurable increased temperature.

3. EMF interaction with signaling pathways

EMF penetrate cells unattenuated and so can interact directly with the DNA in the cell nucleus, as well as other cell constituents. However, biological agents are impeded by membranes and require special mechanisms to gain access to the cell interior. Friedman et al. [23] have demonstrated that the initial step in transmitting extracellular information from the plasma membrane to the nucleus of the cell occurs when NADH oxidase rapidly generates reactive oxygen species (ROS). These ROS stimulate matrix metalloproteinases that allow them to cleave and release heparin binding epidermal growth factor. This secreted factor activates the epidermal growth receptor, which in turn activates the extracellular signal regulated kinase 1\2 (ERK) cascade. The ERK cascade is one of the four mitogen-activated protein kinase (MAPK) signaling cascades that regulate transcriptional activity in response to extracellular stimuli. The elements of the three MAPK signaling cascades implicated in exposures to ELF and RF are highlighted in Fig. 2.

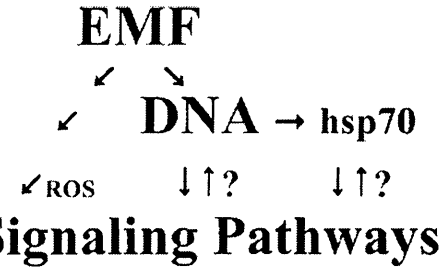

Fig. 3. The signaling pathways and the stress response are activated by EMF. The activation mechanisms discussed in the text are indicated by arrows. In the stress response, DNA activation leads to hsp synthesis and may be due to direct EMF interaction with DNA. The signaling pathways are activated by reactive oxygen species (ROS) that are probably generated by EMF. Possible interactions between the pathways, DNA and hsp are indicated with question marks. In any case, EMF leads to activation of all the processes shown.

The four MAPK cascades are: (1) ERK, (2) c-Jun-terminal kinase (JNK), (3) stress activated protein kinase (SAPK) and (4) p38SAPK. Each of the cascades is composed of three to six tiers of protein kinases, and their signals are transmitted by sequential phosphorylation and activation of the protein kinases in each of the tiers. The result is activation of a large number of regulatory proteins, which include a set of transcription factors, e.g., c-Jun, c-Fos, hsp27 and hsp70. Activation of the stress response is accompanied by activation of specific signal transduction cascades involved in regulating cell proliferation, differentiation and metabolism [24–26]. The MAPK pathways have been characterized in several cell types [24,27–30]. Exposure to non-thermal ELF as well as thermal RF affects the expression of many cellular proteins [23–25] (Fig. 3).

The elevated expression of these protein transcription factors participate in the induction of various cellular processes, including several that are affected by cell phones, e.g., replication and cell-cycle progression [25,31] and apoptosis [32]. RF fields have been shown to activate specific transcription factor binding that stimulate cell proliferation and induce stress proteins [25,33]. It has been reported [31] that within 10 min of cell phone exposures, two MAPKinase cascades, p38 and ERK1\2, are activated. Both ELF and RF activate the upregulation of the HSP70 gene and induction of elevated levels of the hsp70 protein. This effect on RNA transcription and protein stability is controlled by specific protein transcription factors that are elements of the mitogen MAPK cascade.

EMF also stimulate serum response factor which binds to the serum response element (SRE) through ERK MAPK activation and is associated with injury and repair *in vivo* and *in vitro*. The SRE site is on the promoter of an early response gene, c-fos, which under specific cellular circumstances has oncogenic properties. The c-fos promoter is EMF-sensitive; a 20 min exposure to 60 Hz 80mG fields significantly increases c-*fos* gene expression [34]. The SRE accessory protein,

Elk-1, contains a growth-regulated transcriptional activation domain. ERK phosphorylation potentiates Elk-1 and transactivation at the c-*fos* SRE [29].

During the past twenty years, the growing use of cellular phones has aroused great concern regarding the health effects of exposure of the brain to 900 MHz RF waves. Despite claims that the energy level is too low to induce changes in DNA and that the devices are safe, the non-thermal effects that have been demonstrated at both ELF and RF exposure levels can cause physiological changes in cells and tissues even at the level of DNA. Finally, it should be mentioned that some of the pathways described in this section also have roles in protein synthesis via RNA polymerase III, an enzyme in oncogenic pathways [35] and could, therefore, provide a mechanistic link between cancer and EMF exposure.

4. Cells affected by the stress response

Reviews on EMF and the stress response have appeared for the ELF range [13] and for the RF range [36]. The most recent review was published online in section 7 of the Bioinitiative Report [9], and it summarized both ELF and RF studies, mainly at frequencies 50 Hz, 60 Hz, 900 MHz and 1.8 GHz. The citations in that review were not exhaustive, but the different frequencies and biological systems represent the diversity of results on stimulation of DNA and stress protein synthesis in many different cells. It is clear that the stress response does not occur in reaction to EMF in all types of cells, and sometimes because of the use of tissue cultured cell lines, even the same cell line can give opposite results in the same laboratory [37].

Many different types of cells have been shown to respond to EMF, both *in vivo* and *in vitro*, including epithelial, endothelial and epidermal cells, cardiac muscle cells, fibroblasts, yeast, *E. coli*, developing chick eggs, and dipteran cells (see Bioinitiative Report [9], section 7). Tissue cultured cells are less likely to show an effect of EMF, probably because immortalized cells have been changed significantly to enable them to live indefinitely in unnatural laboratory conditions. This may also be true of cancer cells, although some (e.g., MCF7 breast cancer cells) have responded to EMF [38,39], and in HL60 cells, one cell line responds to EMF while another does not [24]. Czyz et al. [16] found that p53-deficient embryonic stem cells showed an increased EMF response, but the wild type did not.

A broad study of genotoxic effects (i.e., DNA damage) in different kinds of cells [40] found no effects with lymphocytes, monocytes and skeletal muscle cells, but did find effects with fibroblasts, melanocytes and rat granulosa cells. Other studies [41,42] have also found that the blood elements, such as lymphocytes and monocytes are natural cells that have not responded. Since mobile cells can easily move away from a stress, there would be little selective advantage and evolutionary pressure for developing the stress response. The lack of response by skeletal muscle cells is related to the need to desensitize the cells to excessive heating during activity. Unlike slow muscle fibers that do synthesize hsp70, cells containing fast muscle fibers do not synthesize hsp70 to protect them from over-reacting to the high temperatures reached a during activity.

Table 1
Biological thresholds in the ELF range.

Biological system	Threshold (μT)[a]	Reference
Acceleration of reaction rates		
Na,K-ATPase	0.2–0.3	Blank and Soo [49]
cytochrome oxidase	0.5–0.6	Blank and Soo [43]
ornithine decarboxylase	~2	Mullins et al. [58]
malonic acid oxidation	<0.5	Blank and Soo [59]
Biosynthesis of stress proteins		
HL60, Sciara, yeast,	<0.8	Goodman et al. [11]
breast (HTB124, MCF7)	<0.8	Lin et al. [39]
chick embryo (anoxia)	~2	DiCarlo et al. [60]
Breast cancer (MCF7) cell growth block melatonin inhibition	0.2 < 1.2	Liburdy et al. [38]
Leukemia epidemiology	0.3–4	Ahlbom et al. [61] Greenland et al. [62]

[a] The estimated values are for departures from the baseline, although Mullins et al. (1999) and DiCarlo et al. (2000) generally give inflection points in the dose–response curves. The leukemia epidemiology values are not experimental and are listed for comparison.

5. EMF–DNA interaction mechanisms: electron transfer

The biochemical compounds in living cells are composed of charges and dipoles that can interact with electric and magnetic fields by various mechanisms. An example discussed earlier is the generation of reactive oxygen species (ROS) in activation of the ERK signaling cascade. The cellular stress response leading to the synthesis of stress proteins is also activated by EMF. However, the specific reaction is not known, except that it is stimulated by very weak EMF. For this reason, our focus has been on molecular processes that are most sensitive to EMF and that could cause the DNA to come apart to initiate biosynthesis. We have suggested that direct EMF interaction with electrons in DNA is likely for the following reasons:

- The largest effects of EMF would be expected on electrons because of their high charge to mass ratio. At the sub-atomic level, one assumes that electrons respond instantaneously compared to protons and heavier atomic nuclei, as in the Born-Oppenheimer Approximation. The very low field strengths and durations that activate the stress response and other reactions (Table 1) suggest interaction with electrons, and make ion-based mechanisms unlikely.
- Weak ELF fields have been shown to affect the rates of electron transfer reactions [43,44]. A 10 μT magnetic field exerts a very small force of only $\sim 10^{-20}$ N on a unit charge,

but this force can move an isolated electron more than a bond length, ~1 nm, in ~1 nanosecond.
- There is a specific EMF responsive DNA sequence that is associated with the response to EMF (Fig. 1), and that retains this property when transfected
- Displacement of electrons in DNA would cause local charging that has been shown to lead to disaggregation of biopolymers [45].
- As the energy in an EMF stimulus increases, there is an increase in single strand breaks, followed by double strand breaks, suggesting an interaction with EMF at all energy levels [46].

Effects of EMF on electrons in chemical reactions were detected indirectly in studies on the Na,K-ATPase [47], a ubiquitous enzyme that establishes the normal Na and K ion gradients across cell membranes. Electric and magnetic fields, each accelerated the reaction only when the enzyme was relatively inactive. It is reasonable to assume that the threshold response occurs when the same charge is affected by the two fields, so the velocity (v) of the charge (q) could be calculated from these measurements and its nature determined. Assuming both fields exert the same force at the threshold, the electric (E) and the magnetic (B) forces should be equal.

$$F = qE = qvB. \quad (1)$$

From this $v = E/B$, the ratio of the threshold fields, and by substituting the measured thresholds [48,49], $E = 5 \times 10^{-4}$ V/m and $B = 5 \times 10^{-7}$T (0.5 μT), we obtain $v = 10^3 m/s$. This very rapid velocity, similar to that of electrons in DNA [50], indicated that electrons were probably involved in the ion transport mechanism of the Na,K-ATPase [47]. An electron moving at a velocity of 10^3 m/s crosses the enzyme (~10^{-8} m) before the ELF field has had a chance to change. This means that a low frequency sine wave signal is effectively a repeated DC pulse. This is true of all low frequency effects on fast moving electrons.

Studies of effects of EMF on electron transfer in cytochrome oxidase, ATP hydrolysis by the Na,K-ATPase, and the Belousov–Zhabotinski (BZ) redox reaction, have led to certain generalizations:

- EMF can accelerate reaction rates, including electron transfer rates
- EMF acts as a force that competes with the chemical forces in a reaction. The effect of EMF varies inversely with the intrinsic reaction rate, so EMF effects are only seen when intrinsic rates are low. (This is in keeping with the therapeutic efficacy of EMF on injured tissue, while there is usually little or no effect on normal tissue.)
- Experimentally determined thresholds are low (~0.5 μT) and comparable to levels found by epidemiology. See Table 1.
- Effects vary with frequency, with different optima for the reactions studied: The two enzymes showed broad frequency optima close to the reaction turnover numbers for Na,K-ATPase (60 Hz) and cytochrome oxidase (800 Hz), suggesting that EMF interacted optimally when in synchrony with the molecular kinetics. This is not true for EMF interactions with DNA, which are stimulated in both ELF and RF ranges and do not appear to involve electron transfer reactions with well-defined kinetics.

Probably the most convincing evidence for a frequency sensitive mechanism that involves stimulation of DNA is activation of protein synthesis in striated muscle. In this natural process, specific muscle proteins are synthesized by varying the rate of the (electrical) action potentials in the attached nerves [51]. The ionic currents of the action potentials that flow along and through the muscle membranes, also pass through the muscle cell nuclei that contain the DNA codes for the muscle proteins. Two frequencies were studied in muscle, high (100 Hz) and low (10 Hz) frequency, corresponding to the frequencies of the fast muscles and slow muscles that have different contraction rates and different muscle proteins. In the experiments, either the fast or slow muscle proteins were synthesized at the high or low frequency stimulation rates corresponding to the frequency of the action potentials. The clear dependence of the protein composition on the frequency of the action potentials indicates a relation between stimulation and activation of DNA in muscle physiology. The process is undoubtedly far more complicated and unlikely to be a simple electron transfer reaction as with cytochrome oxidase. It is more probable that an entire region of DNA coding for a group of related proteins is activated simultaneously.

A mechanism based on electron movement is in keeping with the mV/m electric field and μT magnetic field thresholds that affect the Na,K-ATPase. The very small force on a charge (~10^{-20} N) can affect an electron, but is unlikely to have a direct effect on much more massive ions and molecules, especially if they are hydrated. Ions are affected by the much larger DC electric fields of physiological membrane processes. The low EMF energy can move electrons, cause small changes in charge distribution and release the large hydration energy tied up in protein and DNA structures [3]. Electrons have been shown to move in DNA at great speed [50], and we have suggested that RF and ELF fields initiate the stress response by directly interacting and accelerating electrons moving within DNA [52,53].

A mechanism based on electron movement also provides insight into why the same stress response is stimulated by both ELF and RF even though the energies of the two stimuli differ by orders of magnitude. A typical ELF cycle at 10^2Hz lasts 10^{-2} s and a typical RF cycle at 10^{11} Hz lasts 10^{-11} s. Because the energy is spread over a different number of cycles/second in the two ranges, the energy/cycle is the same in both ELF and RF ranges. Since electron movement occurs much faster than the change of field, both frequencies are seen by rapidly moving electrons as essentially DC pulses. Each cycle contributes to electron movement at both

frequencies, but more rapidly at the higher frequency. The fluctuation of protons between water molecules in solution at a frequency of about 10^{12} Hz [54] gives an indication of the speed of electron movement, and may suggest an upper limit of the frequency in which sine wave EMF act as DC pulses.

6. DNA biology and the EM spectrum

Research on DNA and the stress response has shown that the same biology occurs across divisions of the EM spectrum, and that EMF safety standards based on cellular measures of potential harm should be much stricter. These data also raise questions about the utility of spectrum sub-divisions as the basis for properly assessing biological effects and setting separate safety standards for the different sub-divisions. The frequencies of the EM spectrum form a continuum, and division into frequency bands is only a convenience that makes it easier to assign and regulate different portions of the spectrum for practical uses, such as the different design requirements of devices for EMF generation and measurement. Except for the special case of the visual range, the frequency bands are not based on biology, and the separate bands now appear to be a poor way of dealing with biological responses needed for evaluating safety. The DNA studies indicate the need for an EMF safety standard rooted in biology and a rational basis for assessing health implications.

DNA responses to EMF can be used to create a single scale for evaluation of EMF dose because:

- The same biological responses are stimulated in ELF and RF ranges.
- The intensity of EMF interactions with DNA leads to greater effects on DNA as the energy increases with frequency. In the ELF range, the DNA is only activated to initiate protein synthesis, while single and double strand breaks occur in the more energetic RF and ionizing ranges.

A scale based on DNA biology also makes possible an approach to a quantitative relation between EMF dose and disease. This can be done by utilizing the data banks that have been kept for A-bomb exposure and victims of nuclear accidents, data that link exposure to ionizing radiation and subsequent development of cancer. Utilizing experimental studies of DNA breaks with ionizing radiation, it is possible in principle to relate cancer incidence to EMF exposures. It should be possible to determine single and double strand breaks in a standard preparation of DNA, caused by exposure to EMF for a specified duration, under standard conditions. Although many studies of DNA damage and repair rates under different conditions would be needed, this appears to be a possible experimental approach to assessing the relation between EMF exposure and disease.

7. The stress response and safety standards

Most scientists believe that basic research eventually pays off in practical ways. This has certainly been true of EMF research on the stress response, where EMF stimulated stress proteins have been used to minimize damage to ischemic tissues on reperfusion. However, more importantly, biological effects stimulated by both ELF and RF have shown that the standards used for developing safety guidelines are not protective of cells.

First and foremost, it is important to realize that the stress response occurs in reaction to a potentially harmful environmental influence. The stress response is an unambiguous indication that cells react to EMF as potentially harmful. It is therefore an indication of compromised cell safety, given by the cell, in the language of the cell. The low threshold level of the stress response shows that the current safety standards are much too high to be considered safe.

In general, cellular processes are unusually sensitive to fields in the environment. The biological thresholds in the ELF range (Table 1) are in the range of 0.5–1.0 μT—not very much higher than the ELF backgrounds of ~0.1 μT. The relatively low field strengths that can affect biochemical reactions is a further indication that cells are able to sense potential danger long before there is an increase in temperature.

EMF research has also shown that exposure durations do not have to be prolonged to have an effect. Litovitz et al. [55,56], working with the enzyme ornithine decarboxylase, showed an EMF response when cells were exposed for only 10 s to ELF or ELF modulated 915 MHz, providing that the exposure was continuous. Gaps in the sine wave resulted in a reduced response, and interference with the sine wave in the form of superimposed ELF noise also reduced the response [57]. The interfering effect of noise has been shown in the RF range by Lai and Singh [46], who reported that noise interferes with the ability of an RF signal to cause breaks in DNA strands. The decreased effect when noise is added to a signal is yet another indication that EMF energy is not the critical factor in causing a response. In fact, EMF noise appears to offer a technology for mitigating potentially harmful effects of EMF in the environment.

EMF research has shown that the thermal standard used by agencies to measure safety is at best incomplete, and in reality not protective of potentially harmful non-thermal fields. Non-thermal ELF mechanisms are as effective as thermal RF mechanisms in stimulating the stress response and other protective mechanisms. The current safety standard based on thermal response is fundamentally flawed, and not protective.

Finally, since both ELF and RF activate the same biology, simultaneous exposure to both is probably additive and total EMF exposure is important. Safety standards must consider total EMF exposure and not separate standards for ELF and RF ranges.

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