

DNA is a fractal antenna in electromagnetic fields

MARTIN BLANK¹ & REBA GOODMAN²

Departments of ¹Physiology, and ²Pathology, Columbia University, New York, USA

(Received 7 October 2010; Revised 29 October 2010; Accepted 2 November 2010)

Abstract

Purpose: To review the responses of deoxyribonucleic acid (DNA) to electromagnetic fields (EMF) in different frequency ranges, and characterise the properties of DNA as an antenna.

Materials and methods: We examined published reports of increased stress protein levels and DNA strand breaks due to EMF interactions, both of which are indicative of DNA damage. We also considered antenna properties such as electronic conduction within DNA and its compact structure in the nucleus.

Results: EMF interactions with DNA are similar over a range of non-ionising frequencies, i.e., extremely low frequency (ELF) and radio frequency (RF) ranges. There are similar effects in the ionising range, but the reactions are more complex. Conclusions: The wide frequency range of interaction with EMF is the functional characteristic of a fractal antenna, and DNA appears to possess the two structural characteristics of fractal antennas, electronic conduction and self symmetry. These properties contribute to greater reactivity of DNA with EMF in the environment, and the DNA damage could account for increases in cancer epidemiology, as well as variations in the rate of chemical evolution in early geologic history.

Keywords: DNA damage, DNA structure, non-ionising radiations, oxidative stress, stress, proteins, stress response genes

Introduction

The electromagnetic spectrum is divided into two broad classes, non-ionising and ionising radiation, with each being divided into several frequency ranges. The same physical laws apply to electric and magnetic fields across the spectrum, but the laws give rise to important differences in properties between the ranges that lead to different practical applications. In the non-ionising range, extremely low frequency (ELF) is used to transmit electric power and energise electrical appliances, and radiofrequency (RF) is used in communication technology. Because of the difference in frequency, there are qualitative differences in human exposure. In the ELF range the wavelength of the radiation can be many kilometres, so everything is in the near field of the source and the electric and magnetic fields can be measured separately. In the RF range, where wavelengths are much shorter and can be as low as a few centimetres, the near field is very close to the source and exposure is to a combined electromagnetic field. For example, the wavelength corresponding to an RF frequency of 1 GHz is 3 cm. For this

reason, exposure to a power line is very different from exposure to the radiation from a cell phone, where the head is in the near field of the radiation. However, the similarities in the biological effects indicate that these distinctions may not be as important for interactions with living cells.

Regarding the effects of electric versus magnetic fields in the ELF range, Blank (1995b) has shown that they have similar effects on the membrane ion transport enzyme, the Na,K-ATPase. ELF electric (Blank et al. 1992) and magnetic (Goodman and Blank 1998, Goodman et al. 1994, Goodman and Henderson, 1998) fields also stimulate protein synthesis in cells suspensions, a process that is stimulated by ELF electric fields in vivo to initiate protein synthesis in muscle tissue (Blank 1995a). Protein synthesis is also stimulated by combined electromagnetic fields in the RF range (dePomerai et al. 2000).

The muscle tissue studies mentioned above indicate in vivo mechanisms that activate deoxyribonucleic acid (DNA) when ionic currents pass through the DNA in muscle cell nuclei during the action potentials. The muscle proteins associated with fast muscles are stimulated at higher frequency

RIGHTS LINK()

 $(\sim 100 \text{ Hz})$ than the muscle proteins associated with slow muscles ($\sim 10-20$ Hz). Since different proteins can be induced by changing the frequency of stimulation, different parts of the DNA must be stimulated at different frequencies. The magnitude of the electric field driving the process in muscle, estimated at $\sim 10 \text{ V/m}$ (Blank and Goodman 2004), provides a large safety margin, since electric fields as low as 3mV/m stimulate stress protein synthesis in HL60 cells (Blank et al. 1992).

Materials and methods

Important similarities between electromagnetic field (EMF) interactions with DNA across different parts of the electromagnetic spectrum are pointed out. Most of the studies cited are in the non-ionising range, i.e., the ELF and RF ranges, and include both experimental and epidemiology studies. The experimental studies focus on the stress response and DNA strand breaks. The epidemiology studies relate the incidence of cancer to EMF exposure in each of the frequency ranges. Since cancer is believed to arise from mutations in DNA, such as those that occur in the ionising range as a result of oxidative damage, the similar effects of EMF may arise from electron transfer within the DNA of living cells. Electron transfer studies are presented to support the proposed mechanism for the observed effects of EMF on DNA.

The similarity of EMF interactions across a relatively wide range of frequencies suggests that the DNA acts as a fractal antenna, and an analysis of the structure of DNA in the cell nucleus indicates that there is a structural basis for this property. Similar data for the ionising range of the electromagnetic spectrum suggest that the fractal properties of DNA may extend beyond the ELF and RF ranges to higher frequencies.

The fractal properties of DNA have implications regarding the EMF interaction mechanism with cells, EMF safety standards, and may account for an effect on the rate of molecular evolution in the early history of the earth.

Results

Studies in the non-ionising range

There have been many biological studies in the low frequency range because of health effects associated with power lines and with the use of electrical appliances. In this range, experimental studies aimed at elucidating mechanisms have tended to use low field strengths to stimulate environmental exposures and also to characterise threshold events, i.e., the biological processes that are activated by low levels of

the field. Studies at low field strengths are desirable because pathways in cells are often interconnected and activation of one pathway usually affects others and complicates the analysis.

The discovery of the stress response activated by EMF indicated an interaction with DNA (Blank and Goodman 2004, Blank et al. 1994, Goodman et al. 1994, Goodman and Blank 1998, Kultz 2003). Since a wide range of genes is activated by EMF (Goodman and Henderson 1988), it appears that many parts of the DNA molecule interact. Also, since the stress response is activated by other stimuli that are potentially harmful to cells (Kultz 2003), it is clear that the stress response is a natural protective mechanism, and that cells respond to EMF as potentially harmful.

EMF interaction with DNA in the ELF range is also seen in reports of DNA strand breaks (Bioinitiative Report 2007, Lai and Singh 1997, Pathophysiology 2009, Reflex Project Report 2005). Singlestrand breaks occur at field strengths that are higher than the levels that stimulate the stress response. At still higher field strengths, there are also double strand breaks.

These experimental studies indicate changes in structure and molecular damage, and suggest a probable mechanism for the many epidemiology studies that have investigated health effects associated with chronic low-level exposures. The main focus of these studies has been on leukemia in children, and pooled analyses (Ahlbom et al. 2000, Greenland et al. 2000) of the best studies in the field were analysed to show an increase in risk at exposures of 0.3–0.4 μ T. More recent studies (Draper et al. 2005, Kabuto et al. 2006) have supported the linkage of leukemia with power lines exposure. The increase in risk may actually occur at fields as low as 0.18 μ T, as suggested recently from studies showing an association with damage to DNA repair genes (Yang et al. 2008).

In addition to childhood leukemia, studies have shown an increase in risk from power lines associated with adult diseases, such as leukemia (O'Carroll and Henshaw 2008), Alzheimer's and other neurodegenerative diseases (Garcia et al. 2008, Huss et al. 2009).

Milham (2009) has analysed a century of vital statistics in the US and correlated the incidence of several diseases, including cancer, heart disease and diabetes, in parallel with the introduction of (low frequency) electrification.

The initial biological reactions associated with DNA activation of the stress response in the ELF range also occur at higher frequency in the RF range (Bioinitiative Report 2007, DePomerai et al. 2000, Pathophysiology 2009, Reflex Project Report 2005). The same is true for the ability of EMF to cause



DNA strand breaks and damage to proteins in the RF range (Bioinitiative Report 2007, Lai and Singh 1997, Pathophysiology 2009, Reflex Project Report 2005). Following the pattern in the ELF frequency range, there are also epidemiology studies relating EMF exposure to the incidence of cancers in the RF range. Brain tumours (Hardell et al. 2009) and salivary gland tumours (Sadetzki et al. 2008) from the RF of cell phones have been reported. The results in the RF range appear to parallel those in the ELF range and suggest that the interaction of EMF with DNA is not limited to low frequency.

There are similar effects of EMF on DNA in the ionising range. However, here there are fewer studies and the effects are more complicated than in the non-ionising range. First of all, it is well known that ionising radiation can damage DNA directly by ionising the molecule. But, in addition, recent studies have shown that damage can also occur after exposure in nearby cells that were not directly irradiated (Mothersill and Seymour 2006). This 'bystander effect' raises the possibility that the biological damage in this range may not be a direct effect of the ionising radiation. There can be no doubt, however, that ionisation is due to transfer of an electron, and that the interaction of the higher frequency radiation with an electron is fundamentally related in that regard to the interactions in the non-ionising range.

DNA as a fractal antenna

An antenna receives and transmits electromagnetic radiation because it is made of a material that conducts electricity. In general, an antenna has an optimal frequency that depends upon its length. An antenna that can operate at many different frequencies simultaneously is unusual and is called a fractal antenna. This property is achieved by using self-similar design to maximise the length of conductor that can receive or transmit electromagnetic radiation within a given total surface area or volume. In self-similarity, all subdivisions have a geometry that is similar to the structure as a whole, i.e., different sections of the molecule resemble the shape of the entire molecule.

Since DNA can interact with EMF over a wide range of frequencies, and does not appear to be limited to an optimal frequency, it has the functional properties of a fractal antenna. The DNA molecule can conduct electrons within the double helix (Wan et al. 1999), and the DNA in the cell nucleus has the compacted structural properties of a fractal antenna (see Table I).

From the above analysis of the effect of EMF on the stress response, DNA strand breaks and cancer epidemiology, the fractal property of DNA is apparent in the ELF and RF ranges. The range of

Table I. Coiled structures of increasing size in nuclear DNA (coils get larger as more of the molecule is compacted).

DNA level	Diameter
Double helix	1 nm
Chromatin fiber	10 nm
Solenoid	30 nm
Hollow tube	200 nm

this fractal antenna is much greater than expected of an ordinary antenna, and it may even extend beyond the RF range. There are, in fact, cancer epidemiology studies that link DNA damage to radiation in the higher frequency ionising range. Well known examples are the incidence of cancer among atomic bomb survivors exposed to the effects of γ -rays (Gilbert 2009; Preston et al. 2004) and among children exposed to X-rays (Sadetzki et al. 2006). The cellular response to molecular damage, the stress response, is also found in the middle of the ultraviolet (UV) range (Spitz et al. 2004) which is in the ionising range.

Electron transfer is a plausible mechanism for EMF interactions with DNA at higher frequencies where higher energies are involved. The damage due to DNA strand breaks that occur at higher frequencies, including ionising radiation, is generally attributed to oxidation, another chemical name for electron transfer. Because of the greater energy at higher frequencies, reactive oxygen species, such as peroxides, contribute to the DNA damage. However, DNA strand breaks occur over a wide range of frequencies, and do not demonstrate frequency optima related to molecular reaction kinetics.

Regarding the connection between EMF and the incidence of cancer, the different EMF energy levels in the non-ionising and ionising ranges all involve interaction with and activation of DNA and induction of protein synthesis. The ability of EMF to cause DNA strand breaks and damage to proteins in the non-ionising range is similar to the destructive effects on DNA of the much more energetic X-rays and gamma rays in the ionising ranges, where it is generally acknowledged that the cancers are due to DNA damage. The recent epidemiology studies in the non-ionising range link EMF-caused changes in DNA with cancer. Additional support comes from the study showing that the absence of DNA repair genes is associated with a greater incidence of leukemia from exposure to low frequency EMF (Yang et al. 2008).

Discussion

EMF and electron transfer

Based on the low EMF energy at which DNA activation occurs in the stress response, it has been



proposed (Blank and Goodman 2002, 2008) that EMF probably interacts with the delocalised π electrons in the DNA bases. The electron has a single negative charge that can be accelerated in an electric or magnetic field. Because of its low mass, an electron experiences a relatively large displacement even at low field strengths. Furthermore, the π electrons in the DNA base pairs are delocalised and are able to move along the double helix (Wan et al. 1999). Conduction in DNA is a physical property that may turn out to be extremely useful in the future development of miniature electronic components (Bhalla et al. 2003).

Studies of the effects of EMF on electron transfer reactions have provided insight into the molecular mechanism of EMF interaction with DNA. The Na,K-ATPase (Blank 1995b), cytochrome oxidase (Blank and Soo 1998) and the Belousov-Zhabotinski (oxidation of malonic acid) reactions (Blank and Soo 2003) all show an acceleration due to EMF. The thresholds (field strengths) at 60 Hz for the reactions are: Na,K-ATPase (0.2–0.3 μ T), cytochrome oxidase (0.5–0.6 μT), Belousov-Zhabotinski (oxidation of malonic acid) reaction ($< 0.5 \mu T$). The threshold for stimulation of DNA to elicit protein synthesis is in the same range, $< 0.8 \mu T$ (Goodman and Henderson 1989). The low values are in the range of the epidemiological findings of a doubling in risk of childhood leukemia with EMF exposures above 0.3- $0.4 \mu T$, and they show that relatively little energy is needed to affect the reactions.

The two enzymes just mentioned show broad frequency optima that are close to the reaction turnover numbers (Na,K-ATPase, 60 Hz; cytochrome oxidase, 800 Hz), and this could indicate that the electromagnetic interactions are in synchrony with the molecular kinetics. The frequency optimum of 250 Hz for the Belousov-Zhabotinski reaction is probably the frequency of the rate limiting step in a reaction that involves many steps (Table II). These frequency optima relate to events at the active site of the enzyme, while the earlier frequency data on muscle protein synthesis were for activation of different promoter sites on DNA.

EMF interaction with electrons in DNA

Regarding possible EMF interaction mechanisms that activate cells, Friedman et al. (2007) have shown that interaction of RF with NADH oxidase, an enzyme present in the plasma membrane, can generate reactive oxygen species and activate signalling cascades. This is in line with the above mentioned research, where we have shown EMF interactions with enzymes that affect basic biological properties. The radical pair mechanism (Brocklehurst and McLauchlan 1996, Ritz et al. 2009),

Table II. Optimal frequency stimulating biological reaction.

Reaction stimulated	Optimal frequency	Reference
Na,K-ATPase	ELF (60 Hz peak)	Blank and Soo, 1996
Cytochrome C oxidase	ELF (800 Hz peak)	Blank and Soo, 1998
Belousov- Zhabotinsky	ELF (250 Hz peak)	Blank and Soo, 2003
DNA (biosynthesis)		
HL60, Sciara, yeast	ELF (no peak)	Goodman et al. 1994
C. elegans	RF (no peak)	dePomerai et al. 2000
Various cells	Ionising (no peak)	Spitz et al. 2004

involving a reaction with cryptochromes, appears to explain how the magnetic compass functions in birds. These and other mechanisms may be operative in EMF interactions with cells, but EMF may interact directly with DNA.

Given the low energy at which DNA activation occurs in the ELF range, the stress response probably involves EMF interaction with the delocalised π electrons in the DNA bases. Electrons have been described as moving along the DNA base pairs at relatively high speeds of about 300 cm/s (Wan et al. 1999). It has been suggested that the EMFinduced movement of electrons can result in local charging and the subsequent disaggregation of the DNA strands that precedes protein synthesis (Blank 2008). Since the force due to interaction of an electron with a magnetic field is determined by the strength of the field and the velocity of the electron, the largest force on an electron occurs when the magnetic field is changing and the electron is moving. A significant contribution to electron movement could also result from the normal 'flickering' of H-bonds in water molecules hydrating DNA (Fecko et al. 2003, McGuire and Shen 2006).

The synthesis of a stress protein is initiated at the promoter of the gene, and in EMF activation of the HSP70 gene, a specific nucleotide sequence has been linked to the response. The four base pair nucleotide sequence, nCTCTn, is essential for a response to EMF (Lin et al. 1999, Lin et al. 2001). The promoters of the HSP70 stress gene and the EMFactivated oncogene, c-myc, have multiple copies of this sequence. Artificial constructs containing these sequences can be stimulated by EMF to activate a reporter gene, and the EMF effect disappears when the sequences are mutated.

One can see why the nCTCTn sequences would respond readily to EMF. Their low electron affinities enable EMF to displace electrons easily, and when CTCT bases are excited by UV radiation, they lose EMF energy 10 times more rapidly than the GAGA bases on the complementary chain (Schwalb and Temps 2008). The difference in molecular motion indicated by the rate of energy loss would promote



separation of the two DNA strands at that site. When DNA comes apart with purines (GAGA bases) on one side and pyrimidines (CTCT bases) on the other, the result is a smaller area for interaction with water molecules that makes the energetics more favourable for separation. These properties increase the probability of a split at this sequence.

Possible effects on the rate of molecular evolution

The molecular properties of DNA and the way in which the double helix is compacted in the cell nucleus is a unique design that also facilitates interaction with EMF. The nCTCTn sequences that were shown to respond most efficiently to EMF would be expected to occur on average once every 256 base pairs and should be fairly common. The interactions that lead to a separation of two DNA strands must therefore be possible at many sites in the molecule. The particular part of the double helix in which a separation occurs is determined by the level of DNA structure that can best interact with the frequency of the EMF to which it is exposed. Some sites coding for stress proteins are 'bookmarked' (Xing et al. 2005), that is, they are in DNA segments that are not compacted and therefore more exposed and available for interaction.

EMF is believed to have been an important driving force in evolution. Because the physical arrangement of the DNA in a cell determines its properties as an antenna, the ability of DNA to act as a fractal antenna could account for the large difference in the rate of molecular evolution of prokaryotes and eukaryotes. There are major differences between the essentially circular prokaryotic DNA, which lacks the self-similarity of fractals, and the DNA of eukaryotes that have a fractal arrangement in the nucleus. Prokaryotes contain only a single loop of stable chromosomal DNA and are far less likely to respond to a variety of EMF frequencies.

It is generally believed that early evolution on Earth was driven by mutations caused by ionising radiation (e.g., UV, x-rays) from the Sun, as well as weak natural low frequency electrical discharges in the atmosphere, such as those that give rise to the Schumann resonances (in the 0–50 Hz range). Both prokaryotes and eukaryotes react to the frequencies that affect the DNA double helix, but the fractal DNA of eukaryotes can also react to the wider range of frequencies that affect the larger coiled structures in the nucleus. More changes in DNA increase the probability of mutations and the evolution of new species.

In the fossil record, prokaryotes appeared approximately 3.5 billion years ago, only about 1 billion years after the formation of the Earth's crust, while eukaryotes only appeared about 1.7 billion years ago.

Many factors undoubtedly affected the rate of evolution, but the great acceleration of the process shown in the fossil record is coincident with the appearance of fractal DNA. The record actually shows that many more new animal and plant species have evolved since the appearance of fractal DNA, in the same time span it took for the fractal structures to emerge from prokaryotic DNA.

The difference between fractal and non-fractal structures is probably also a factor to consider in the discussion on the relative roles of RNA versus DNA in early evolution. RNA structures contain loops but lack the self similarity of nuclear DNA, and would be less likely than DNA structures to react to EMF. It is possible that DNA may have had advantages in competition with RNA, because of this property.

Implications for safety standards

The many similarities in the interactions of EMF with DNA across a wide range of frequencies suggest greater caution in approaching questions of human health and safety. It should be obvious that safety standards in individual frequency ranges are not appropriate when the same biological processes are activated across the electromagnetic spectrum. It is the total exposure that should be considered, and EMF safety standards must be based on all biological responses.

In fact, a biologically-based safety standard is desperately needed. The existing 100 μ T ELF exposure limit set by ICNIRP (International Commission for Non-Ionizing Radiation Protection) is many times higher than the $0.4 \mu T$ where a doubling of childhood leukemia risk is widely acknowledged. It has also been pointed out that the specific absorption rate (SAR), the widely used thermal standard for EMF safety, does not relate at all to the biological thresholds of the stress response in the ELF and RF ranges, and that the threshold for the same biological process differs by many orders of magnitude in the two ranges (Blank and Goodman 2004).

If EMF interactions can lead to DNA damage, the great increase in the use of RF EMF for communication in the last few decades should accelerate the rate of interaction with DNA and cause more mutations. The proliferation of mobile phones, WiFi (wireless communication technology), etc. could lead to a large increase in mutations over a very short period of time. There have already been indications that proliferation of RF due to cell phone use may have contributed to an increase in brain cancer (Appendix 2 in Cardis 2010). It is difficult to estimate the effect on the rate of evolution because of the explosive nature of this change when considered on a geological time scale. The changes in DNA may come too fast and in such great numbers as to be



incapable of being tested by time for fitness and survival value. Recent reports indicating reduced sperm counts and sperm quality associated with cell phone use (Agarwal et al. 2008, Li et al. 2009) suggest that EMF may even affect evolution by interfering directly with reproduction. The expansion of the new EMF-based technologies without adequate testing of biological responses has indicated a need for better regulation of the environment (Bioinitiative Report 2007, Sage 2010), and the exercise of greater precaution before adding to the EMF burdens of daily life in modern society.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

- Agarwal A, Deepinder F, Sharma RK, Ranga G, Li J. 2008. Effect of cellphone usage on semen analysis in men attending in fertility clinic: an observational study. Fertility and Sterility 89:124-128
- Ahlbom H, Day N, Feychting M, Roman E, Skinner J, Dockerty J, Linet M, McBride M, Michaelis J, Olsen JH, Tynes T, Verkasalo PK. 2000. A pooled analysis of magnetic fields and childhood leukemia. British Journal of Cancer 83:692-698.
- Bhalla V, Ram BP, Bharadwaj LM. 2003. DNA electronics. EMBO reports 4:442-445. doi:10.1038/sj.embor.embor834.
- Bioinitiative Report (2007). A scientific perspective on health risk of electromagnetic fields. Edited by Sage C, Carpenter D, Published online 31August 2007 at: http://www.bioinitiative.org/report/index.htm
- Blank M. 1995a. Electric stimulation of protein synthesis in muscle. Advances in Chemistry 250:143-153.
- Blank M. 1995b. Electric and magnetic field signal transduction in the membrane Na,K-ATPase. Advances in Chemistry 250:339-348.
- Blank M. 2008. Protein and DNA interactions with electromagnetic fields. Electromagnetic Biology and Medicine 28:3-23.
- Blank M, Goodman R. 2002. Electromagnetic initiation of transcription at specific DNA sites. Journal of Cell Biochemistry 81:689-692.
- Blank M, Goodman R. 2004. Initial interactions in electromagnetic field-induced biosynthesis. Journal of Cellular Physiology 199:359-363.
- Blank M, Goodman R. 2007. A mechanism for stimulation of biosynthesis by electromagnetic fields: charge transfer in DNA and base pair separation. Journal of Cellular Physiology. Published online: 9 July 2007 doi: 10.1002/jcp.21198.
- Blank M, Soo L, Lin H, Henderson AS, Goodman R. 1992. Changes in transcription in HL-60 cells following exposure to alternating currents from electric fields. Bioelectrochemistry and Bioenergetics 28:301-309.
- Blank M, Khorkova O, Soo L, Lin H, Weisbrot D, Henderson AS, Goodman R. 1994. Increased levels of hsp70 transcripts are induced when cells are exposed to low frequency electromagnetic fields. Bioelectrochemistry and Bioenergetics 46:130-138.
- Blank M, Soo L. 1998. Frequency dependence of cytochrome oxidase activity in magnetic fields. Bioelectrochemistry and Bioenergetics 46:139-143.
- Blank M, Soo L. 2003. Electromagnetic acceleration of the Belousov-Zhabotinski reaction. Bioelectrochemistry 61: 93-97.

- Brocklehurst R. McLauchlan KA 1996. Free radical mechanism for the effects of environmental electromagnetic fields on biological systems. International Journal of Radiation Biology 69: 3-34.
- Cardis E (on behalf of the Interphone Study Group). 2010. Brain tumor risk in relation to mobile telephone use: results of the interphone international case-control study. International Journal of Epidemiology 2010:1-20.
- dePomerai DI, Daniells C, David H, Allan J, Duce I, Mutwakil M, Thomas D, Sewell P, Tattersall J, Jones D. 2000. Non-thermal heat-shock response to microwaves. Nature 6785:417–418.
- Draper G, Vincent T, Kroll ME, Swanson J. 2005. Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study. British Medical Journal 330:1290-1292A.
- Fecko CJ, Eaves JD, Loparo JJ, Tokmakoff A, Geissler PL. 2003. Ultrafast hydrogen-bond dynamics in infrared spectroscopy of water. Science 301:1698-1701.
- Friedman J, Kraus S, Hauptman Y, Schiff Y, Seger R. 2007. Mechanism of short-term ERK activation by electromagnetic fields at mobile phone frequencies. Biochemical Journal 405:559-568
- Garcia AM, Sisternas A, Hoyos SP. 2008. Occupational exposure to extremely low frequency electric and magnetic fields and Alzheimer's disease: a meta-analysis. International Journal of Epidemiology 37: 329-340.
- Gilbert ES. 2009. Ionizing radiation and cancer risk. What have we learned from epidemiology? International Journal of Radiation Biology 85:467.
- Goodman R, Blank M, Lin H, Khorkova O, Soo L, Weisbrot D, Henderson AS. 1994. Increased levels of hsp70 transcripts are induced when cells are exposed to low frequency electromagnetic fields. Bioelectrochemistry and Bioenergetics 33: 115-120.
- Goodman R, Blank M. 1998. Magnetic field stress induces expression of hsp70. Cell Stress and Chaperones 3:79-88.
- Goodman R, Henderson AS. 1988. Exposure of salivary gland cells to low frequency electromagnetic fields alters polypeptide synthesis. Proceedings of the National Academy of Science (US) 85:3928-3932.
- Greenland S, Sheppard AR, Kaune WT, Poole C, Kelsh MA. 2000. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Epidemiology 11:624-634.
- Hardell L, Carlberg M, Mild KH. 2009. Epidemiological evidence for an association between use of wireless phones and tumor diseases. Pathophysiology 16:113-122.
- Huss A, Spoerri A, Egger M, Roosli M. 2009. Residence near a power lines and mortality from neurodegenerative diseases: longitudinal study of the Swiss population. American Journal of Epidemiology 169:167-175.
- Kabuto M, Nitta H, Yamamoto S, Yamaguchi N, Akiba S, Honda Y, Hagihara J, Isaka K, Saito T, Ojima T, Nakamura Y, Mizoue T, Satoko I, Eboshida A, Yamazaki S, Sokejima S, Kurokawa Y, Kubo O. 2006. Childhood leukaemia and magnetic fields in Japan: a case control-study of childhood leukemia and residential power-frequency magnetic fields in Japan. International Journal of Cancer 119:643-650.
- Kultz D. 2003. Evolution of the cellular stress proteome: from monophyletic origin to ubiquitous function. Journal of Experimental Biology 206:3119-3124.
- Lai H, Singh NP. 1997. Acute exposure to a 60Hz magnetic field increases DNA strand breaks in rat brain cells. Bioelectromagnetics 18:156-165.
- Li DK, Yan B, Li Z, Gao E, Miao M, Gong D, Weng X, Ferber JR, Yuan W. 2010. Exposure to magnetic fields and risk a poor sperm quality. Reproductive Toxicology 29:86–92.
- Lin, H. Blank M. Head M. Goodman R. 1999. Magnetic fieldresponsive domain in the human HSP70 promoter. Journal of Cell Biochemistry 75:170-176.



- Lin H, Blank M, Rossol-Haseroth K, Goodman R. 2001. Regulating genes with electromagnetic response elements. Journal of Cell Biochemistry 81:143-148.
- McGuire JA, Shen YR. 2006. Ultrafast vibrational dynamics at water interfaces. Science 313:1945-1948.
- Milham, S. 2010. Historical evidence that electrification caused the 20th century epidemic of diseases of civilization. Medical Hypotheses 74:337-345.
- Mothersill C, Seymour CB. 2006. Radiation-induced bystander effects and the DNA paradigm: An "out of field" perspective. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis 597:5-10.
- O'Carroll MJ, Henshaw DL. 2008. Aggregating epidemiological evidence: comparing two seminal EMF reviews. Risk Analysis 28:225-234.
- Pathophysiology. 2009. Special issue on EMF. 16(2,3):67-248.
- Preston DL, Pierce DA, Shimizu Y, Collings HM, Fujita S, Funamoto S, Kodama K. 2004. Effect of recent changes in atomic bomb survivors dosimetry on cancer mortality risk estimates. Radiation Research 162:377-389.
- REFLEX Project Report. 2004. A summary of the final report can be found at http://www.verumfoundation.de/www2004/html/ pdf/euprojekte01/REFLEX ProgressSummary 231104.pdf
- Ritz T, Wiltschko R, Hore PJ, Rodgers CT, Stapput K, Thalau P, Timmel CR, Wiltschko W. 2009. Magnetic compass of birds is based on a molecule with optimal directional sensitivity. Biophysical Journal 96, 3451-3457. doi:10.1016/j.bpj.2008. 11.072.
- Sadetzki S, Chetrit A, Lubina A, Stovall M, Novikov I. 2010. Risk of thyroid cancer following childhood exposure to ionizing

- radiation for tinea capitis. Journal of Clinical Endocrinology and Metabolism 74:337-345.
- Sadetzki S, Chetrit A, Jarus-Hakak A, Cardis E, Deutch Y, Duvdevani S et al. 2008. Cellular phone use and risk of benign and malignant parotid gland tumors. A nationwide casecontrol study. American Journal of Epidemiology 167:457-67.
- Sage, C. 2010. Tragedy of the commons revisited: the new wireless commons. Reviews on Environmental Health 2011;26(1). © 2010 by Walter de Gruyter, Berlin, New York. DOI 10.1515/HMBCI.2010.000.
- Schwalb NK, Temps F. 2008. Base sequence and higher-order structure induce the complex excited-state dynamics in DNA. Science 322:243-245.
- Spitz DR, Azzam EI, Li JJ, Gius D. 2004. Metabolic oxidation/ reduction reactions and cellular responses to ionizing radiation: a unifying concept in stress response biology. Cancer and Metastasis Review 23:311-322.
- Wan C, Fiebig T, Kelley SO, Treadway CR, Barton JK. 1999. Femtosecond dynamics of DNA-mediated electron transfer. Proceedings of the National Academy of Sciences USA 96:6014-6019.
- Xing H, Wilkerson DC, Mayhew CN, Lubert EJ, Skaggs HS, Goodson ML, Hong Y, Park-Sarge OK, Sarge KD. 2005. Mechanism of HSP 70i gene bookmarking. Science 307:421-
- Yang Y, Jin X, Yan C, Tian Y, Tang J, Shen X. 2008. Case-only study of interactions between DNA repair genes (hMLH1, APEX1, MGMT, XRCC1 and XPD) and low-frequency electromagnetic fields in childhood acute leukemia. Leukemia and Lymphoma 49:2344-2350.

