

ORIGINAL ARTICLE

Cell biology and EMF safety standards

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Living cells react defensively and start to synthesize stress proteins when exposed to potentially harmful stimuli. Electromagnetic fields (EMF) are among the many different environmental stimuli that initiate stress protein synthesis. Although there is greater energy transfer and heating due to EMF at higher frequencies, there is no greater stress response. The cellular stress response is far more sensitive to EMF than to an increase in temperature. It should be obvious that an EMF safety standard should be based on the more sensitive, natural biological response.

Keywords

Cell biology, cellular stress response, DNA damage, DNA fractal antenna, EMF safety, EMF standards, thermal criterion

History

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Evaluating technology

The deliberations of expert panels developing safety standards for exposure to electromagnetic fields (EMF) bring to mind the old folk tale from India, of blind men describing an elephant. In the story, the blind men touch different parts of the elephant and describe the whole animal based on the particular part they happen to touch. You can imagine how different the descriptions are when based on touching the trunk, tail, tusk, ear, etc., and how far they all are from accurately describing an elephant.

I am sure we all agree that we need the most complete picture for an accurate description of a complex subject. However, we do not always recognize that we have a problem when we are caught up in the process. We certainly do not expect such a problem to occur in science, especially in an area that has important health ramifications. Yet, it is happening to us by neglecting established research in cell biology when evaluating the health risks of EMF exposure.

Electromagnetic fields (EMF) in the environment

The introduction of extremely low frequency (ELF) electric power, over a century ago, came with the recognized dangers of electric shock and electrocution, but the steady growth in the use of power has exposed people to higher and higher levels of EMF. This trend is now accelerating with the rapid spread of radiofrequency (RF)-based technology, e.g. cell

phones, WiFi, smart meters, etc. Epidemiological studies have raised the specter of EMF-generated health issues, and laboratory studies have shown biological consequences of exposure to EMF that support these suspicions and underscore the need for greater regulation.

It is clear that regulation of exposure requires measuring the fields and establishing limits. Measuring fields is the easy part for engineers generally involved in these activities. However, the setting of acceptable levels requires biological and medical information that is apparently much harder to obtain. In fact, the information appears to be beyond the abilities of those involved in the process. For example, Foster and Moulder (2013) claim, “The only unequivocal mechanism for bioeffects of RF energy at realistic exposure levels in the low-GHz frequency range involves heating of tissue.” They may believe that measuring an increase in temperature is the only way to assess bioeffects, but they are apparently unaware of the far more relevant biological measurements reviewed in Goodman and Blank (1998) and Blank and Goodman (2009). These biological data are based on the ways living cells protect themselves from many potentially harmful effects. Furthermore, the specialized biological mechanisms are in sharp contrast to the mechanisms that generate and dissipate heat that arises from ongoing biological functions associated with normal metabolism and muscle activity, and not directly related to potentially harmful environmental exposures.

Actually, the well-documented, cellular stress response is the primary protective reaction of cells to a wide variety of potentially harmful environmental stimuli. The response to EMF has been known for two decades, and it occurs long before a temperature rise activates the same cellular response. These studies indicate unequivocally that the cellular stress

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response is a far more sensitive measurement that can detect biological effects that are missed when only measuring the temperature rise. Other biological effects of EMF (Frey, 1962, 1993), such as “microwave hearing” and leakage of the blood–brain barrier, also occur long before a temperature rise can be detected. The exposure limits based on temperature rise are therefore unreliable as sentinels, and by ignoring biological effects that occur at much lower EMF levels, they have put the population at greater risk.

A biological standard

The cellular stress response is the biological guide to a variety of environmental dangers, and it indicates the levels at which natural defensive measures are activated. We should have realized that a thermal criterion based on energy absorbed is wrong when both ELF and RF evoke the same cellular stress response, despite the enormous difference in energy input by the two frequency ranges. We should actually use this information and make the cellular stress response the criterion for both ranges. This would correct the anomaly posed by the lowest RF frequencies, where action potential thresholds were introduced as the criterion because the thermal criterion that was acknowledged to be inadequate.

The same response in ELF and RF ranges could also have been suspected from the fact that the divisions of the electromagnetic spectrum are not based on biology and are essentially arbitrary. Cells respond to signals across the EM spectrum. This means that the safety of technologies based on all non-ionizing frequencies could be evaluated using the same biological criteria – including the additive effects of simultaneous and multiple exposures from across the EM spectrum.

Insight into a major biological mechanism

Another advantage in utilizing information from cell biology is that the mechanism of the cellular stress response has been studied and is known in considerable detail. The synthesis of any protein starts in the DNA of the cell nucleus that contains the genetic code for that protein. This was demonstrated for the stress protein, hsp70. The particular DNA segment that reacted with EMF to start the process was identified and attached to the DNA code of another protein (a reporter gene), and it was possible to activate synthesis of the other protein with the same EMF stimulus. This showed that the DNA segment functioned as both an EMF detector and an activator of an attached gene (Lin et al., 1999, 2001).

Studies of DNA structure also provide an explanation for the ability of DNA to interact with many different EMF frequencies (Blank and Goodman, 2011). Human DNA is a six-foot long chain packed into a micron size nucleus, so it is coiled many times and compacted in order to fit into this tiny structure. This results in a coiled-coil organization of DNA in the cell nucleus – a structure having the self-similarity that is characteristic of fractal structures. Each coil in this structure apparently can act like an antenna for a particular frequency range related to its size, and the result is that there are functional antennas of many sizes in the same nucleus. This structure can account for the reactivity of DNA to a wide range of frequencies, and it highlights the vulnerability of

nuclear DNA to the many EMF frequencies now present in the environment.

Considering the structure of DNA in greater detail, we can see why it can function as an antenna. The classic double helical configuration, with its two sugar phosphate polymer chains connected by the bases (that also function as a code), has mobile pi electrons all along the DNA. Many studies by the Barton group at Caltech (Hall and Barton, 1997; Hall et al., 1996; Wan et al., 1999, 2000) and others, have shown that these electrons can be made to move, i.e. to conduct electricity. The build-up of electrons at bends and constrictions in the DNA chain is a plausible mechanism for causing DNA damage. The breakup of a large molecule into its oligomers was demonstrated to result from an increase in the charge on hemoglobin (Blank and Soo, 1987). Both power and radio frequency EMF have been shown to damage DNA (Lai and Singh, 1995, 1996, 1997), and DNA damage is believed to be an important factor in the initiation of cancers. IARC, the International Association for Research on Cancer, declared both power frequency in 2002 and RF in 2011 to be possible carcinogens.

Electrons in DNA are not the only target of EMF. The fields have been shown to affect electron transfer reactions in other biological systems, e.g. Na, K-ATPase (Blank and Soo, 1996), cytochrome oxidase (Blank and Soo, 1998), Belousov–Jabotinsky reaction (Blank and Soo, 2003). In all the systems studied, the measured thresholds for the reactions are very low (at the level of a few mG’s), indicating an ability to easily affect biological oxidations.

EMF exposure associated with cancer

Cancer is not a single disease, and there is no proven mechanism to account for a connection between DNA damage and the many different cancers that are diagnosed many years after the damage occurs. However, there are many plausible mechanisms arising from studies of oxidative damage. In fact, the working hypothesis of biologists and physicians is that mutations that result from DNA damage can lead to the many different types of cancer, as well as other diseases. The stimulation of stress protein synthesis and the DNA chain breaks, stimulated by a wide range of ELF and RF frequencies, are valuable diagnostic criteria.

Cancer has generally been the focus of many studies of EMF safety, and most cancers have a long induction period before symptoms can be detected. The biological damage from short-term exposure to EMF is considered a likely first step in a plausible mechanism leading to cancer. There are other disease-related mechanisms due to biological damage by EMF that have been identified, such as inhibiting secretion of melatonin that affects circadian rhythm and molecular repair processes (Liburdy et al., 1993), as well as an effect on the capillaries of the blood–brain barrier causing leakage and damaging neurons in the brain (Frey, 1993). Like the DNA damage, these effects occur at exposures well below levels that are now considered safe, and are likely to also be caused by interaction with electrons.

The emphasis on disease in this discussion should not obscure the fact that the ability to affect biological processes is double-edged. It has been shown that stress proteins can

have protective and therapeutic effects when induced prior to other stresses. Di Carlo et al. (1998) have protected developing chick eggs and increased the percentage hatched, and George et al. (2008) have increased the survival rate following heart bypass surgery by inducing stress proteins with EMF prior to surgery. A particular advantage of the artificial induction of stress proteins with EMF is that it is a procedure that can be easily carried out non-invasively.

Perspective

The reluctance of EMF safety panels to be guided by biological evidence probably arises from a lack of familiarity with research in cell biology, coupled with a bias toward reliance on energy in an engineering context. When making judgments on safety, there is also a tendency to rely on previous panels. It must be easier to go along with these groups and not have to justify dismissing the research studies reviewed in the BioInitiative Report (2012). The scientific credentials of the Report are hard to dispute. It started as a symposium of the Bioelectromagnetics Society (BEMS) and included reviews by three former Presidents of that Society. Unlike the reports of many panels, the Report was written by scientists who actually did the relevant research.

There are now many research papers detailing the responses of living cells to potentially harmful EMF in the environment. A living cell is not just a sac of solution that heats up when exposed to EMF. The reactions of a cell and its many components have been studied, and the results tell us that EMF causes potentially harmful changes. These data should be used to estimate safe biologically based exposure levels. The thermal criterion is not specific to EMF and is not sensitive enough. The biological cellular stress response is sensitive, is applicable across a wide frequency range, and provides insight into mechanism.

Declaration of interest

The author declares no conflicts of interest. The author alone is responsible for the content and writing of this article.

References

BioInitiative Report. (2012). In: Sage, C., Carpenter, D., *A Scientific Perspective on Health Risk of Electromagnetic Fields*. Available from: <http://www.bioinitiative.org/report/index.htm> (accessed 31 Dec 2012).
 Blank, M., & Goodman, R. (2009). Electromagnetic fields stress living cells. *Pathophysiology*. 16:71–78.

Blank, M., & Goodman, R. (2011). DNA is a fractal antenna in electromagnetic fields (EMF). *Int. J. Radiat. Biol.* 87:409–415.
 Blank, M., & Soo, L. (1987). Surface free energy as the potential in oligomeric equilibria: Prediction of hemoglobin disaggregation constant. *Bioelectrochem. Bioenerg.* 17:349–360.
 Blank, M., & Soo, L. (1996). The threshold for Na, K-ATPase stimulation by electromagnetic fields. *Bioelectrochem. Bioenerg.* 40: 63–65.
 Blank, M., & Soo, L. (1998). Enhancement of cytochrome oxidase activity in 60Hz magnetic fields. *Bioelectrochem. Bioenerg.* 45: 253–259.
 Blank, M., & Soo, L. (2003). Electromagnetic acceleration of the Belousov-Zhabotinski reaction. *Bioelectrochemistry*. 61:93–97.
 Di Carlo, A., Farrell, J. M., & Litovitz, T. (1998). A simple experiment to study electromagnetic field effects: Protection induced by short-term exposures to 60Hz magnetic fields. *Bioelectromagnetics*. 19: 498–500.
 Frey, A. H. (1962). Human auditory systems response to modulated electromagnetic energy. *J. Appl. Physiol.* 17:689–692.
 Frey, A. H. (1993). Electromagnetic field interactions with biological systems. *FASEB J.* 7:272–281.
 Foster, K. R., & Moulder, J. E. (2013). Wi-Fi and health: Review of current status of research. *Health Phys.* 5:561–570.
 George, I., Geddis, M. S., Lill, Z., et al. (2008). Myocardial function improved by electromagnetic field induction of stress protein hsp70. *J. Cell. Physiol.* 216:816–823.
 Goodman, R., & Blank, M. (1998). Magnetic field stress induces expression of hsp70. *Cell Stress Chaperon.* 3:79–88.
 Hall, D. B., & Barton, J. K. (1997). Sensitivity of DNA-mediated electron transfer to the intervening pi-stack: A probe for the integrity of the DNA base stack. *J. Am. Chem. Soc.* 119:5045 (Abstract).
 Hall, D. B., Holmlin, R. E., & Barton, J. K. (1996). Oxidative DNA damage through long range electron transfer. *Nature*. 382:731–735.
 Lai, H., & Singh, N. P. (1995). Acute low-intensity microwave exposure increases DNA single strand breaks in rat brain cells. *Bioelectromagnetics*. 16:207–210.
 Lai, H., & Singh, N. P. (1996). Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. *Int. J. Radiat. Biol.* 69:513–521.
 Lai, H., & Singh, N. P. (1997). Acute exposure to a 60Hz Magnetic field increases DNA strand breaks in rat brain cells. *Bioelectromagnetics*. 18:156–165.
 Liburdy, R. P., Sloma, T. R., Sokolic, R., & Yaswen, P. (1993). ELF magnetic fields, breast cancer, and melatonin: 60Hz fields block melatonin's oncostatic action on ER+ breast cancer cell proliferation. *J. Pineal Res.* 14:89–97.
 Lin, H., Blank, M., Rossol-Haserath, K., & Goodman, R. (1999). A magnetic field responsive domain in the human HSP70 promoter. *J. Cell. Biochem.* 75:170–176.
 Lin, H., Blank, M., Rossol-Haserath, K., & Goodman, R. (2001). Regulating genes with electromagnetic response elements. *J. Cell. Biochem.* 81:143–148.
 Wan, C., Fiebig, T., Kelley, S. O., Treadway, C. R., & Barton, J. K. (1999). Femtosecond dynamics of DNA-mediated electron transfer. *Proc. Natl. Acad. Sci. USA.* 96:6014–6019.
 Wan, C., Fiebig, T., Schiemann, O., Barton, J. K., & Zewail, A. H. (2000). Femtosecond direct observation of charge transfer between bases in DNA. *Proc. Natl. Acad. Sci. USA* 97:14052–14055.