Comments on Notice of Inquiry, ET Docket No. 13-84

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I am responding to the FCC's request for information relevant to its current review of the thermal (tissue-heating) model of harm as the basis for radiofrequency (RF) radiation safety policies. I have studied the effects of electromagnetic fields (EMF) on biological cells for several decades, and what follows is a brief summary of biological evidence demonstrating the unsuitability of the thermal model as a basis for safety standards of human RF exposures.

Using an increase in temperature as a gauge of damage has an intuitive appeal; many of us recall from childhood our parents using a thermometer to determine if we were ill. And, of course, it is true that an increase in body temperature is an indication that we are ill. At the same time, we can be ill with little or no perceptible temperature rise.

Just as the human body can be ill without a perceptible rise in temperature, EMF damage to human cells can occur long before changes in temperature can be detected. And, indeed, research studies have repeatedly demonstrated that damaging cellular reactions to EMF occur long before any measurable rise in temperature. Biological measures of cellular harm are far more sensitive than temperature increase,

Cellular damage occurs in response to RF exposures even in the absence of a thermal response.

The thermal model is based on the understanding that, when the temperature of a cell is raised several degrees, the cell begins to synthesize stress proteins, in a process that was first termed 'heat shock'. It was later found that the same stress proteins were produced when cells were exposed to other harmful agents, e.g., low oxygen, changes

in pH, alcohol, etc. EMF was also shown to evoke the cellular stress response. (An early review: Goodman R, Blank M. 1998. Magnetic Field Induces Expression of hsp70. Cell Stress and Chaperones 3:79-88.)

It is widely accepted that these stress proteins, and the cellular stress response, are indicators of cellular damage. It is notable, then, that the stress response occurs in cells exposed to EMF, even in the absence of a discernible rise in temperature.

The genetic code for stress proteins is in the DNA; EMF exposure triggers the synthesis of stress proteins. We have identified a particular segment of DNA that reacts with EMF; we demonstrated that, when this particular segment of DNA was attached to another protein, we then could activate the resultant protein with EMF. This study explains that EMF interaction with DNA is the mechanism of the cellular response. (Lin H, Blank M, Goodman R. 1999. Magnetic Field-Responsive Domain in the Human HSP70 Promoter. J Cellular Biochemistry 75:170-176.) Other biological mechanisms, such as EMF effects on melatonin secretion, blood brain barrier permeability, have also been demonstrated.

It has been shown that a wide range of EMF frequencies (including the power frequency range) can trigger the cellular stress response at levels of exposure insufficient to increase tissue temperature. That such a breadth of frequencies in the EM spectrum can trigger this response, at such low levels of exposure, suggests that the double-helix DNA possesses the properties of a fractal antenna. (The coiled–coil structure endows DNA with the property of self-similarity, the essential characteristic of fractal structures.) Fractal antennas can react to a variety of EMF frequencies, and we have suggested that this property of DNA accounts for its ability to react to many EMF frequencies. (Blank M, Goodman R. 2011. DNA is a fractal antenna in electromagnetic fields, EMF. Int. J. Radiation Biol 87: 409-15.)

And thus,

1) The cellular stress response results from EMF exposures insufficient to create a

perceptible rise in temperature.

2) Cellular damage occurs at levels that are considered safe according to the thermal model.
3) The fractal antenna nature of DNA enhances the receptivity and sensitivity of the cells to EMF exposures.

For these reasons, the thermal model is both misleading and inadequate as the basis for determining radiofrequency safety standards. The thermal model of harm is an invalid basis on which to approach safety standards. The thermal model should be replaced by a more appropriate biological model, such as the one proposed by myself and Dr. Goodman (Blank M, Goodman R. 2012. Electromagnetic fields and health: DNA-based dosimetry.

Further, that DNA appears to react to EMF as a fractal antenna strongly suggests that the FCC must devise a safety regimen that considers the cumulative effects of simultaneous exposures across many frequencies, including ELF from appliances as well as RF from cell phones and WiFi from an increasing number of sources.

Citations

Blank M, Goodman R. 2012. Electromagnetic fields and health: DNA-based dosimetry. Electromagnetic Biology and Medicine 31(4):243-249.

Goodman R, Blank M. 1998. Magnetic Field Induces Expression of hsp70. Cell Stress and Chaperones 3:79-88.

Lin H, Blank M, Goodman R. 1999. Magnetic Field-Responsive Domain in the Human HSP70 Promoter. J Cellular Biochemistry 75:170-176.

Blank M, Goodman R. 2011. DNA is a fractal antenna in electromagnetic fields, EMF. Int. J. Radiation Biol 87: 409-15.