Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects

Martin Pall writes:

One of the great puzzles about the action of electromagnetic fields is how can they influence the biology of our bodies? The reason that this is such a great puzzle is that these fields are comprised of low energy photons, with energies too low to influence the chemistry of our bodies. So how can they possibly influence our biology? Many have argued that the only thing that they can possibly do is to heat things, and yet it is very clear that levels of exposure that produce only the slightest heating have been repeatedly shown to produce substantial biological effects. Now this puzzle has been solved in a paper with the title of this email, published on line in the Journal of Cellular and Molecular Medicine, freely available on the publisher's web site:

http://onlinelibrary.wiley.com/doi/10.1111/jcmm.12088/pdf

That paper reviews 24 different studies in which EMF exposures produce biological effects that can be blocked by using calcium channel blockers, drugs that block the action of voltage-gated calcium channels (VGCCs). Most of these drug studies implicated L-type VGGCs, showing blockage by channel blockers specific for these L-type VGCCs; however three other classes of the voltage gated calcium channels were also implicated in some of these studies. What these and other studies show, is that EMF exposures act by partially depolarizing the electrical charge across the plasma membrane of cells, activating the VGCCs and it is the increased intracellular calcium levels that are responsible for the reaction to EMF exposure. These 24 studies implicate the VGCCs in responses to a variety of EMFs, including extremely low frequency EMFs such as 50 and 60 cycle fields produced by our alternating currents in our wiring, various microwave/radiofrequency EMFs and nanosecond electrical pulses. Static electrical fields also act via VGCCs, not surprisingly because they also influence the electrical charge across plasma membranes.

Perhaps more surprisingly, static magnetic fields also act via VGCCs. This is a bit surprising because static magnetic fields do not produce electrical changes in static objects. However as pointed out in the paper, living cells in the body are rarely static, often moving rapidly in such phenomena as cellular ruffling.

Having resolved this long-standing puzzle, the paper goes on to consider how VGCC activation can produce two well-documented responses to EMF exposure: stimulating of bone growth and the production of single stranded DNA breaks in EMF-exposed cells. EMF exposures have repeatedly been shown to produce increases in nitric oxide levels, in some cases almost instantaneously. These nitric oxide increases are produced through calcium stimulation of the action of the two nitric oxide synthases in the cell, iNOS and eNOS, which are both calcium-dependent enzymes. Nitric oxide in the cell, acts to produce most physiological effects, by stimulating the production of cycle GMP which stimulates, in turn the G-kinase (this is known as the NO/sGC/cGMP/G-kinase pathway). Most pathophysiological responses to nitric oxide to through another pathway, where nitric oxide acts as a precursor of peroxynitrite, a potent oxidant and reactive free radical precursor. The paper suggests that the EMF stimulation of bone growth, a very promising therapeutic response, goes through the first pathway. It also suggests that induction of single strand breaks in cellular DNA goes through the second pathway. It is possible that possible beneficial effects of EMFs go through the first pathway and adverse, pathophysiological effects go through the second pathway. Clearly we will need a lot of study to test mechanisms of EMF action.

This paper may be viewed in a practical setting as being very important in two ways:

1. There have been many claims that biological effects of EMF exposures cannot possibly exist because no plausible mechanism of action of such exposures could produce such effects. Clearly these claims are now defunct.

2. In studies aimed at understanding the mechanisms of action of EMF exposures we now know where to look. Such studies need to look at roles of VGCCs, intracellular calcium, nitric oxide and possibly cycle GMP or peroxynitrite. It can be argued, therefore, that this paper is very much a game changer, changing a situation where there has been substantial confusion, into one where, specific, targeted questions can be asked and answered experimentally.

Finally, this paper says nothing at all about EMF hypersensitivity (often abbreviated EHS), a condition where previous EMF exposure appears to induce high level sensitivity to some types of EMFs. EHS is similar to multiple chemical sensitivity (MCS), where previous chemical exposures produce high level chemical sensitivity. Chemicals act in MCS by indirectly activating the NMDA receptors and NMDA receptors have many similarities in their properties to those of the L-type VGCCs. You should expect, therefore, a future paper on a detailed proposed mechanism for EHS, with both many similarities and some apparent mechanism of MCS as well as some differences.

You may forward this message as you wish.

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