Electromagnetic Radiation and Health – March 2014 Keep


A conference presented by the British Society for Ecological Medicine (BSEM) in London on 7 March 2014, addressed the issues surrounding electromagnetic radiation (EMR) and chronic disease and electrosensitivity. It brought together national and international experts who shared their research and clinical experience in the subject. Although the conference considered the effects of EMR on all body systems, this summary highlights only those elements which are relevant to the cardiovascular (CV) system.

Introduction

The scene was set by Professor Denis Henshaw, from Bristol University, and Alasdair Philips of Powerwatch, who explained some of the basic issues involved in understanding electromagnetic radiation studies. Professor Henshaw described natural sources of EMR, such as geomagnetic storms arising from charged particles from the sun, which can increase blood pressure and affect heart rate variability, but noted that only 10-15% of the population seem affected. Alasdair Philips covered the man-made sources of EMR, such as electric wiring and power lines, any electrical appliance or equipment (including microwave ovens), computers, hospital scanners, airport scanners, smart meters, WiFi, DECT cordless phones and mobile phones.

Michael Bevington then talked about official reports on the effect of EMR on health, including the World Health Organisation (WHO) International Agency for Research on Cancer (IARC) 2011 report, which classified radiofrequency electromagnetic fields (EMFs) as possibly carcinogenic to humans, the International Commission on Non-Ionizing Radiation Protection (ICNIRP), which issued guidelines in 1998 for limiting exposure to EMFs up to 300GHz and the WHO International EMF Project to assess the scientific evidence of possible health effects of EMF in the frequency range from 0 to 300 GHz, which is still ongoing. Also influential was the BioInitiative Report 2012, written by 12 leading scientists, recommending precaution against EMF exposure at levels well below current international guidelines. Professor Henshaw and others pointed out that the effects of EMR have been shown to be unrelated to any thermal effect, yet all the international guidelines and safety precautions had been drawn up assuming that the only effects were thermal. The enlightened approach taken by Sweden in recognising electrosensitivity as a ‘functional disability’ was also highlighted. The studies of EMR and health

Rachel Nicoll talked about the overall findings from the many thousands of studies on EMR and health, pointing out that there was no clear association of exposure and acute or chronic health problems, with a broadly equal number of human and animal studies showing an effect and showing no effect, regardless of frequency, exposure time/dose, specific absorption rate or proximity to source. Among those showing an effect on CV function, a typical selection of studies showed decreased or increased blood pressure, increased heart rate variability, decreased cardiac epidermal growth factor, decreased myocyte mitochondria, deposition of collagen leading to cardiac fibrosis, lipid peroxidation,
dyslipidaemia\textsuperscript{14,15,16}, increased prothrombin time and decreased INR\textsuperscript{17}, altered ECG results\textsuperscript{18}, decreased stroke volume and tachycardia or bradycardia\textsuperscript{19}.

It is this inconclusive nature of the research findings, which prevents more attention being paid to the potential EMR sources of patients’ conditions; there are too many studies showing an adverse effect to be dismissed as chance findings, but not enough to provide conclusive evidence of an effect. This suggests that there is some hitherto unrecognised factor which determines whether EMR exposure will affect an individual or will have no effect. She quoted the typical review article conclusion:

‘Published papers show contradictions....the exact mechanism of electric and magnetic field interactions with biological structures is still unknown\textsuperscript{20}.

Further complicating the issue, an analysis of the animal studies had shown a number of reasons why an EMR study might show no positive association with disease, other than the true absence of an effect. These findings, which largely indicate that researchers did not fully understand the nature of EMR effects, included delayed effects not picked up in short studies, effects manifesting in an organ or biomarker not tested, tissue recovery at different time intervals (if rapid, the tissue damage may be missed in a long duration study), low dose exposure having effects absent in high dose exposure, age and gender interactions, the adaptive response and the fact that studies generally expect a linear dose/response effect whereas there may be a U-shaped response in cancer studies, while X-rays demonstrate a quadratic association.

Rachel Nicoll talked further about the adaptive response (known to cardiologists as ‘pre-conditioning’), the generally accepted definition of which is ‘a low dose pre-exposure which reduces or inhibits a pathological response to a later higher dose exposure (challenging dose)’. The animal studies reveal that this definition is inadequate, since the pre-exposure does not need to be of the same type of radiation and indeed may be provided by a heavy metal. The pre-exposure may also be transferred between animals by bone marrow injection, may be of a higher, rather than a lower, dose and may be administered after the challenging dose. It therefore seems that any magnitude of dose or toxin, applied at any time, can generate an adaptive response. It is hardly surprising that there is currently no agreed mechanism of action of the adaptive response, although it may be that it is unrelated to the applied toxin but instead merely downregulates some mechanism in the individual.

Alasdair Philips then discussed the possibilities of bias in human studies, as well study design problems, stemming from the failure of many researchers to understand the necessary determinants of radiation exposure. Although a number of studies showed no effect on human health, of note was the ECOLOG Report\textsuperscript{21}, produced in 2000, which, despite being funded by the mobile phone industry, concluded that low power densities may cause cancer development, damage the immune system, increase stress hormone secretion and impair cognitive function at exposures well below the current guidelines. After issuing a number of recommendations, it concluded that mobile phone use maximum SAR should be cut to a quarter of the existing permitted exposure. Furthermore, the 2005 Bamberg Report\textsuperscript{22} indicated that tachycardia, episodic hypertension and collapse, amongst other conditions, could be linked to home radiofrequency exposure, while the EU-funded REFLEX Report\textsuperscript{23} investigating the effect of EMR on cell lines concluded that extremely low frequency (ELF) and radiofrequency exposure had a genotoxic effect, as evidenced by increased DNA
strand breaks or micronuclei. Dr Erica Mallery-Blythe pointed out that 72% of research funded by the mobile phone industry showed no effect of EMFs, whereas only 33% of non-industry funded research showed no effect.

**Potential mechanisms**

Professor Martin Pall, of Washington State University, described his own biochemical research, which had been highlighted on the Global Medical Discovery website, investigating the puzzling finding in numerous studies that pulsed EMFs are far more biologically active than non-pulsed, continuous fields, which is inconsistent with the orthodox belief that effects are entirely thermal in nature. Instead he hypothesized that EMFs act to influence the voltage across plasma membranes of cells, thus activating voltage-gated calcium channels (VGCCs), mainly L-type, to allow influx of Ca2+ to the cells, thereby raising intracellular calcium, leading to the adverse biological effects of EMF exposure. As support for this hypothesis, he cited evidence showing that EMFs cause large increases in intracellular calcium and/or changes in calcium signalling, while adverse effects from exposure to low frequency (microwave) EMFs can be inhibited by calcium channel blockers, which block the VGCCs. Pilla has also shown that pulsed microwave frequency EMF exposures produce almost instantaneous increases in both intracellular Ca2+ and nitric oxide (NO) synthesis. Increased NO is relevant in this context because most of its pathological effects are mediated through its role as a precursor of peroxynitrite (ONOO-), leading to free radical generation and oxidative stress. There is considerable evidence that the single DNA strand breaks observed in microwave EMF exposure occur through oxidative stress, and furthermore they can be blocked by antioxidants. VGCCs are found in high quantity in cardiac myocytes, pacemaker cells and neuronal cell bodies, all likely to have a bearing on cardiac function.

Professor Pall went on to draw a parallel with a genetic mutation of the Cav1.2 channel, which causes it to be slow in closing, and hence hyperactive, allowing excess influx of Ca2+ to the pacemaker cells of the heart, altering electrical control of the heart and inducing tachycardia, arrhythmia, J-wave syndrome and sudden cardiac death, similar to some of the findings of EMF studies. He noted that since the sinoatrial node has an extremely high density of VGCCs of different types, it might be the case that EMF exposures acting on the sinoatrial node were responsible for much of the increase in these cardiac changes in the general population. There are a number of studies on dogs and excised hearts that show exactly this, while reports of arrhythmia have been more common among those living in close proximity to microwave towers. Support for this hypothesis was also seen in a review showing that both L-type and T-type calcium channel blockers are apparently helpful in the treatment of arrhythmias and a study showing that some electrosensitive patients exhibited instantaneous tachycardia on blinded exposure to microwave radiation.

The biochemist Dr John McLaren Howard described the necessary influx of small amounts of Ca2+ into cells via VGCCs for essential signalling to regulate contraction and relaxation in cardiac muscle and vascular smooth muscle cells, membrane integrity, as well as many other functions. Large amounts of Ca2+, however, can trigger apoptosis. His own research had shown that although there was no increase in intracellular calcium with EMFs in healthy subjects, in some patients with pre-
existing sensitivity (to benzoate in this experiment), EMF exposure further increased intracellular calcium. The relevance of using benzoate sensitive patients was that chemical sensitivity is very similar in its manifestations to electrosensitivity and patients are liable to have already elevated intracellular calcium. The two conditions often reinforce each other, as occurred in this instance, where EMF exposure amplifies chemical sensitivity, increasing the biochemical and clinical effects, which include disturbance of a major signal transduction pathway with potential for multiple adverse consequences. Whereas the large influx of Ca2+ should have triggered apoptosis, this may have been prevented by induction of calcium binding proteins, which maintain a damaged cell in circulation. Furthermore, the elevated calcium displaced essential magnesium on the actin fibril and could block the production of ATP, leading to fatigue and serious disease.

As well as the two biochemists, several other presenters addressed the issue of mechanisms. Professor Henshaw mentioned that low intensity magnetic fields generate ‘radical pairs’, inducing oxidative stress in the organism. He suggested that disruption by EMRs of circadian rhythms and decrease in melatonin, a powerful antioxidant produced in the pineal gland, may underlie some of the negative health effects found in many studies. Dr Erica Mallery-Blythe also touched on the fact that oxidative stress is implicated in many chronic conditions, including coronary heart disease, cardiac fibrosis, hypertension, ischaemia and myocardial infarction; one source may be EMF exposure, which can be counteracted by antioxidants. Rachel Nicoll also highlighted the fact that studies showed a clear association between exposure and oxidative stress and DNA/histone methylation.

Closing remarks

In closing, Rachel Nicoll stressed the need to share the information from the conference to make it more widely available to clinicians, researchers and the general public. There was an urgent need for researchers to determine a mechanism of effect, as this would clearly demonstrate the conditions under which EMR posed a threat to human health.

Correspondence to:
Rachel Nicoll
Department of Public Health and Clinical Medicine and Heart Centre
Umeå University
Sweden
rachelnicoll25@gmail.com

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