BIOLOGICAL EFFECTS OF LOW-INTENSITY RADIOFREQUENCY ELECTROMAGNETIC RADIATION – TIME FOR A PARADIGM SHIFT IN REGULATION OF PUBLIC EXPOSURE

Priyanka Bandara (1,2) PhD (Biochemistry & Molecular Genetics, UNSW) and Steve Weller (1) BSc (Biochemistry and Microbiology, Monash).

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ABSTRACT

This study focuses on man-made radiofrequency electromagnetic radiation (RF-EMR), which has increased exponentially around the globe over the last few decades due to a rapid expansion of mobile/wireless/satellite technologies. The WHO's IARC classified RF-EMR as a Group 2B possible human carcinogen in 2011. The scientific evidence emerged since, particularly epidemiological evidence linking mobile/cordless phone use to brain cancer and experimental evidence of genotoxicity and carcinogenicity, has led to calls for an update to this classification.

In many countries, including Australia, the current RF exposure regulation is based on the 1998 guidelines of the International Commission on Non-ionization Radiation Protection (ICNIRP). Several scientific organizations, including the US National Toxicology Program and EPA, and the American and European academies for environmental medicine, have raised concerns about the thermal basis of ICNIRP guidelines which only takes into account acute tissue heating effects. There is strong scientific evidence of non-thermal biological effects occurring in the absence of heating. These effects cannot be prevented by current thermally-based guidelines. The Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) has based its RF standard (RPS3) on the ICNIRP guidelines which inherit the same limitation – an inability to assure safety from chronic non-thermal effects. ARPANSA has been reluctant to accept potential health effects that may arise out of low-intensity (non-thermal) RF-EMR biological effects as ARPANSA claims a lack of an "established" mechanism other than heating. Our detailed study of the scientific literature challenges this paradigm. We present the experimental evidence of RF-EMR induced oxidative stress, a key non-thermal mechanism of biological effects at low intensity exposures.

In our recent review of the scientific literature, 216 out of 242 studies that investigated endpoints related to oxidative stress were found to have reported significant effects. Evaluation of the scientific literature by ARPANSA (TRS164 report) has failed to critically review the literature on oxidative stress and assess its potential impact on public health.

We present oxidative stress as a key central mechanism underlying adverse biological effects related to RF-EMR exposure, such as DNA damage. Considering the well-established role of oxidative stress in pathobiology of a wide array of chronic diseases, RF exposure standards require urgent reform.

1 Oceania Radiofrequency Scientific Advisory Association (ORSAA) Inc., Australia.
2 Environmental Health Trust, USA.

INTRODUCTION

The use of man-made non-ionizing electromagnetic radiation (EMR) has rapidly increased over the last few decades and, as a consequence, so has its presence in the environment. Radio frequency (RF) including microwaves emanating from modern wireless communication/surveillance systems and medical equipment, as well as extremely low frequency electromagnetic fields from power lines/electrical/medical appliances, have been investigated in scientific studies designed to assess the impact on human health. This paper focuses specifically on RF-EMR that has increased exponentially around the globe over the last few decades due to a rapid expansion of mobile/wireless/satellite communication technologies and medical applications of RF. From naturally occurring RF exposure that is found to in power densities below 10^{-15} W/m^2 [1], typical current public exposures have risen above 10^{-2} W/m^2 [2-5]. The WHO's IARC classified RF-EMR as a Group 2B possible human carcinogen in 2011 [6]. Scientific evidence emerged since, particularly epidemiological evidence linking mobile/cordless phone use to brain cancer [7-9], as well as experimental evidence of genotoxicity and carcinogenicity[10,11] has led to calls for an update to this classification[12].

In many countries including Australia, the current RF exposure regulation depends on the 1998 guidelines of the International Commission on Non-ionization Radiation Protection (ICNIRP) [13]). Several scientific organizations, including the US National Toxicology Program (NTP) and the Environmental Protection Agency (EPA), as well as the American and European academies for environmental medicine, have raised concerns about the thermal basis of ICNIRP guidelines which takes into account acute tissue heating effects only, and are therefore unable to protect against chronic non-thermal effects [14-16]. There is strong scientific evidence of non-thermal biological effects that cannot be prevented by the current thermal orientated guidelines[17]. The Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) has, by default, inherited the same limitation, as they have established the Australian RF standard (RPS3)[18] based on the ICNIRP 1998 guidelines. Therefore, it is not possible to assure safety from chronic non-thermal biological effects using the current RPS3 standard. ARPANSA has been unwilling to accept that potential health effects may arise out of low-intensity (non-thermal) RF-EMR biological effects because they claim that there is a lack of an "established" mechanism other than heating. Our detailed study of the scientific literature challenges this claim. In this paper, we present the experimental evidence and theoretical background of RF-EMR induced oxidative stress, a key non-thermal mechanism for biological effects that have been observed at low intensity exposures. The evidence of non-thermal biological effects is irrefutable at this stage given the clearly demonstrated effects on basic human biological functions, such as metabolism[19] caused by currently permitted non-thermal exposures.

Oxidative stress is a biochemical/physiological phenomenon of a stress state whereby the cellular pro-oxidant load generated mostly by reactive oxygen and nitrogen species (ROS and RNS) exceeds its anti-oxidant potential (contributed by enzyme and non-enzyme antioxidants) resulting in oxidative damage to cellular structural constituents such as DNA, lipids and proteins and also alteration of cellular communication processes (intra/inter cellular signal transduction). The evidence of the important role of ROS and RNS in signal transduction cascades controlling vital biological functions in growth, metabolism, hormonal and immune functions, etc. has emerged mostly over the last couple of decades[20]. Sustained elevation of the physiological levels of ROS and RNS causing oxidative stress and resultant disruption to cellular functions is likely to lead to chronic disease[21]. Oxidative stress is known to be implicated in the pathobiology of almost every chronic disease, notably cardiovascular disease, cancer and neurodegenerative diseases [22-24].

MATERIALS AND METHODS

The Oceania Radiofrequency Scientific Advisory Association (ORSAA) Inc., Australia, has constructed a customized database for storing and analysing published studies from the peer-reviewed scientific literature, captured from databases such as PubMed and EMF-Portal. There are over 2400 studies recorded in the ORSAA database[25]at present. The following selection criteria were applied when entering studies to the database: all studies reviewed by ARPANSA, as per the list of publications provided by ARPANSA on ORSAA's request were included; non-English papers with a published abstract in English with enough information on the exposure characteristics to evaluate the study methodology in peer-reviewed national journals in the country of origin were included; microwave ablation procedures used in medical applications due to high-intensity exposures involved (thermal effects) were excluded. For this study, a subset of

studies that investigated experimental endpoints related to oxidative stress with a cut-off date of 25th July 2017 has been analysed.

RESULTS

A subset of 242 studies which investigated experimental endpoints related to oxidative stress was identified. These are available for perusal on the ORSAA database and they are being further analysed at present. This extended set of oxidative stress studies further supports the previously published evidence for oxidative stress induced by RF-EMR exposure [26].

Only one study of the pooled 242 studies was published before the year 2000, 55 (23%) between 2005-2009, and 173 (72%) since 2010. These studies come from 29 different countries, while Australia has contributed minimally with a single study[27]. Of the 242 studies, most (216, 89%) found significant effects related to oxidative stress. Largely animal studies (*in vivo*) and cell culture studies (*in vitro*) have shown increased levels of endogenous oxidative stress markers and/or affected antioxidant levels in various tissue/cell types upon exposure to RF-EMR. Some studies have further demonstrated ameliorative effects upon supplementation with a range of antioxidants. These are complemented by limited human studies where RF exposure demonstrated oxidative stress and/or reduced antioxidant status. Further characterization of these studies is ongoing.

CONCLUSION

The weight of the scientific evidence, much of which has accumulated over the last decade, indicates low-intensity RF-EMR exposure as a cause of cellular oxidative stress. This is the most plausible mechanism for the biological effects that occur at the non-thermal levels of exposure currently permitted by existing thermally-based ARPANSA standards. Recently, renowned physical scientists have presented the experimental evidence and the theoretical explanations as to how weak fields of RF-EMR can generate oxidative stress in cells[28], strengthening the experimental evidence reported by biological scientists in these *in vivo* and *in vitro* studies analysed in this paper. The evidence is clear: nearly 90% of the studies have found oxidative stress to be induced by RF-EMR. This evidence is in stark contrast to the claim by the external expert that ARPANSA engaged to evaluate the *in vivo* and *in vitro* studies; i.e. "*the putative link between RF energy and altered ROS production remains tenuous*"[29]. This conclusion that has been endorsed by ARPANSA is not only false and misleading, but also puts public health at risk by influencing policy with regards to public exposure to RF-EMR and preventing the attention of the medical scientists to this area of research that is required to understand the mechanisms of potentially harmful RF biological effects.

The evidence presented here provides further support for the calls by independent scientists to abandon the thermally-based ICNIRP guidelines for RF-EMR exposure regulation, and to instead adopt a new more stringent biologically-based exposure guidelines[17]. This evidence also calls for a change to the controversial approach by the International EMF Project at the WHO[30]. Most of this evidence on oxidative stress has emerged since the IARC classification of RF-EMR as a Group 2B possible human carcinogen in 2011, when the "lack" of a biologically plausible mechanism prevented a higher classification. Therefore, an urgent review is warranted of the scientific evidence of RF-EMR by the IARC with respect to the Bradford Hill criteria for causation. This evidence clearly shows that there is a need for an evidence-based paradigm shift in non-ionizing radiation protection with regards to RF-EMR, in order to lessen the impact of EMR on public health. ARPANSA and other Australian stakeholders in radiation protection must adopt a precautionary approach and take immediate steps to reduce public exposure to RF-EMR from common wireless devices and infrastructure. In particular, minimising wireless use by children is important due to their increased vulnerability to RF absorption and biological effects[31], as well as potentially more serious cumulative effects due to longer life-time use.

FUTURE DIRECTIONS

The evidence presented here on oxidative stress suggests that ARPANSA needs to rectify the current state of affairs by assigning a team of cell biologists and clinicians experienced in oxidative stress research with extensive expertise in basic biochemistry/physiology as well as disease pathology to conduct an extensive review on *in vitro* and *in vivo* experimental studies. Relying on a single biophysicist with limited expertise is against the widely accepted methods of scientific review, such as expert panels assembled by the IARC and the Delphi method. This is essential for reaching scientific consensus from a vast body of scientific evidence and best possible outcomes for public health.

The evidence presented here provides further support for abandoning the thermally-based ICNIRP guidelines for RF-EMR exposure regulation and instead adopting more stringent biologically-based new exposure guidelines as per the calls by international scientists[17]. It also calls for a change to the controversial approach taken by the International EMF Project at the WHO[30] where the scientific evidence of low-intensity biological effects is not accurately addressed in favour of thermally-based ICNIRP guidelines. Most of the above evidence on oxidative stress has emerged since the IARC classification of RF-EMR as a Group 2B possible human carcinogen in 2011 when the "lack" of a biologically plausible mechanism prevented a higher classification. However, this is no longer the case; rather, the current knowledge-base warrants an urgent review of the scientific evidence of RF-EMR by the IARC with respect to the Bradford Hill criteria for causation. Overall, there is an urgent need for an evidence-based paradigm shift in non-ionizing radiation protection and a precautionary approach enacted with regards to RF-EMR in order to lessen the impact on public health.

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ARPS CONFERENCE SUPPORT POLICY

The ARPS Executive reviewed the Conference Support Policy and approved an updated version of the policy in August 2014.

An application form has also been created to assist with submission and approval of requests for support. The policy outlines eligibility and the level of support that will be offered. Applications must be submitted prior to the conference that a member wishes to attend.

The full policy and application form are available on the ARPS website:
http://www.arps.org.au/?q=content/arps-conference-support-policy-application-form