



Health Matters

Microwave and RF Exposure-Induced Molecular and Genetic Alterations

■ James C. Lin 

Recent reports of studies on the effects of microwave and RF exposure on molecular and cytogenetic materials have drawn considerable attention. They suggest that RF exposure could induce DNA damage at levels considered safe by promulgated guidelines and regulatory standards.

In particular, a new study described the observation of genetic alterations in rat cancers resulting from lifetime exposure to low-level RF radiation [1]. For this retrospective study, rat tumor samples were derived from Sprague-Dawley rats exposed 19 h/day for two years at a whole-body specific absorption rate (SAR) of 0.1 W/kg [2]; see also [3]. This study analyzed the genetic makeup of rat brain and heart cancers that featured morphological and molecular similarities to those found in humans. Specifically, the research examined the genetic modifications in RF-induced rat tumor samples



through molecular characterization of cancer genes related to human brain gliomas. A targeted next-generation sequencing panel was constructed for rats based on human glioma-relevant genes. Single-nucleotide variants and small insertions and deletions were categorized in rat gliomas and cardiac schwannomas. The results indicate that rat gliomas induced by lifelong RF exposure histologically appear like low-grade human gliomas. Note that the threshold for safe RF effects is 4 W/Kg per current standards [4], [5], [6].

Furthermore, another investigation reported that whole-genome bisulfite sequencing immediately following RF exposure showed changes in deoxyribonucleic acid (DNA) methylation patterns and early differentially

methyated genes in human skin keratinocytes [7]. DNA methylation is an epigenetic process used by cells to regulate gene expression. It is dynamic and can be triggered in response to external stimuli. The investigation exposed cultured human keratinocytes to 900-MHz RF radiation for 1 h at a low SAR (<0.01 W/kg). The result highlights a possible epigenetic role in the cellular response to RF radiation. The report further suggested that the findings may potentially be developed as epigenetic biomarkers for immediate responses to RF exposure.

Beyond underlining a potential epigenetic role in the cellular response to low-level RF exposure, these results place a spotlight on an atypical event in which a scientific investigation from a U.S. military research laboratory reported a cytogenetic response [8]. Specifically, it suggested an epigenetic role in the cellular response to low-level RF exposure, potentially with major influences on gene activities.

These well-conducted studies lend further support to the results of earlier rat exposure studies showing RF radiation affecting DNA strand breaks.

In this context, a series of papers in the 1990s reporting on single- and

James C. Lin (lin@uic.edu) is with the University of Illinois Chicago, Chicago, IL 60607 USA.

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double-strand DNA breaks in rat brain cells after acute exposure to RF radiation is noteworthy [9], [10], [11]. The degree of DNA single-strand break was assayed in brain cells from rats acutely exposed to low-level 2,450-MHz microwaves using an alkaline microgel electrophoresis method. The single-cell gel electrophoresis assay (also known as a *comet assay*) is a technique used to detect DNA damage by measuring the migration of fragmented DNA from a cell under an electric field. Immediately after 2 h of exposure to pulsed microwaves (2- μ s width pulsed at 500 Hz), no significant effect was observed. However, at a whole-body SAR of 0.6 and 1.2 W/kg, increases in DNA single-strand breaks were found in brain cells of rats at 4 h postexposure. Furthermore, in rats exposed for 2 h to a spatially averaged power density of 20 W/m² and continuous-wave (CW) 2,450-MHz microwaves (a SAR of 1.2 W/kg), increases in brain cell DNA single-strand breaks were observed immediately as well as at 4 h postexposure. No significant difference was observed between the effects of CW or pulse forms of microwave radiation. It was suggested that these effects may have resulted from a direct effect of RF exposure on DNA molecules and/or impairment of DNA damage repair mechanisms in brain cells. Also, the finding that the opioid antagonist naltrexone and the free radical scavenger melatonin could partially block the DNA damage effects of RF exposure [11] could imply a potential role for free radicals or that changes in iron balance within cells might be involved as mechanisms in the observed DNA strand breaks.

Actually, changes in molecular, cytogenetic, and nucleic acid activities in rats for single and repeated short-term RF exposures were reported decades ago [12]. For example, a decrease in DNA and ribonucleic acid contents were reported in rats exposed to 10 GHz, 2 kW/m² for 30 min [13]. Repeated exposures to 3–10 GHz for 10–20 min daily for 21–22 days enhanced or maintained the changes observed after a single exposure.

Isolated DNA, dissolved, in vitro, and using a series of buffer conditions and temperatures, was exposed to 2,450 MHz at 94 W/kg (SAR) for 16 h [14]. There was no difference in the properties of isolated DNA between control and exposed preparations when the temperature of the exposed sample was identical to that of the control preparation during treatment. Even elevated temperatures (up to 50 °C) produced by microwaves (1 h at a SAR of 225 W/kg) did not cause a difference in the thermal denaturation profile of a DNA solution. The result was the same for solutions containing formaldehyde to enhance the sensitivity of the test to detect alterations in the pairing of the two strands of the DNA molecule when compared to a similar temperature elevation produced by conventional heating.

However, the older studies were controversial, and many are met with skepticism today. The subject investigations are multidisciplinary. It is often difficult to appreciate important facts or theories from another discipline. Many of the older studies employed RF exposure systems and measurement instrumentations that did not allow or were unable to ascertain the specific and relevant local exposure conditions of the biological preparations. Aside from the absence of a reported SAR and its differences and distributions, there is the possibility that localized variations in SAR distribution or some experimental artifact might be responsible for the experimental findings or lack thereof.

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