

Local exposure of 849 MHz and 1763 MHz radiofrequency radiation to mouse heads does not induce cell death or cell proliferation in brain

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Abbreviations: SAR, specific absorption rate; RF, radiofrequency; CDMA, code domain multiple access; PCS, personal communications service; TUNEL, Terminal deoxynucleotidyl Transferase Biotin-dUTP Nick End Labeling; PCNA, proliferating cell nuclear antigen; GFAP, glial fibrillary acidic protein; FDTD, finite-difference time-domain; ANOVA, analysis of variance

Abstract

Even though there is no direct evidence to prove the cellular and molecular changes induced by radiofrequency (RF) radiation itself, we cannot completely exclude the possibility of any biological effect of mobile phone frequency radiation. We established a carousel-type exposure chamber for 849 MHz or 1763 MHz of mobile phone RF radiation to expose RF to the heads of C57BL mice. In this chamber, animals were irradiated intermittently at 7.8 W/kg for a maximum of 12 months. During this period, the body weights of 3 groups-sham, 849 MHz RF, and 1763 MHz RF-did not

show any differences between groups. The brain tissues were obtained from 3 groups at 6 months and 12 months to examine the differences in histology and cell proliferation between control and RF exposure groups, but we could not find any change upon RF radiation. Likewise, we could not find changes in the expression and distribution of NeuN and GFAP in hippocampus and cerebellum, or in cell death by TUNEL assay in RF exposure groups. From these data, we conclude that the chronic exposure to 849 MHz and 1763 MHz RF radiation at a 7.8 W/kg specific absorption rate (SAR) could not induce cellular alterations such as proliferation, death, and reactive gliosis.

Keywords: brain; cellular phone; radiation; radiation injuries; gliosis

Introduction

Although the energy level of RF radiation is not high enough to break covalent bonds, it can induce molecular responses, leading to cell proliferation or cell death, as reported in previous studies using *in vitro* or *in vivo* experiments (Moulder *et al.*, 1999). The possible mechanism for the biological effect of RF radiation might be due to the alteration of the intracellular signaling pathways through changes in ionic distribution or membrane fluidity (Hossmann and Hermann, 2003). Moreover, we cannot rule out the possibility of interactions between genes and RF radiation to lower the threshold of physiological changes, including diseases. For example, there have been many reports on the strong correlations between brain tumors and RF radiation in epidemiological studies (Ahlbom *et al.*, 2004).

The brain is especially the most important target tissue to study the biological effect of RF radiation in mobile phone users (Moulder *et al.*, 2005). Hardell *et al.* (1999) reported an increase in tumors ipsilateral to the side of the head on which the subjects recalled phone use, but this was not substantiated by other studies (Muscat *et al.*, 2000; Inskip *et al.*, 2001). In animal experiments, exposure to RF did not affect the incidence, malignancy, volume, multiplicity, latency, or fatality associated with any kind of neurogenic tumor, either (Zook and Simmens, 2006).

Alteration of cognitive and physiological function of brain upon exposure to mobile phone-frequency RF radiation has been reported by several electrophysiological studies (Hamblin *et al.*, 2004; Hinrichs and Heinze, 2004; Curcio *et al.*, 2005; Huber *et al.*, 2005). In these studies, RF exposures can induce measurable changes in human brain electrical activity, particularly in the alpha frequency band (8-13 Hz) over posterior regions of the scalp. In addition, rats exposed to RF showed neuronal damage in the cortex, hippocampus, and basal ganglia (Salford *et al.*, 2003), while it was not reproduced by others (Joubert *et al.*, 2007). However, there are a number of points to consider regarding whether RF radiation can affect the human brain and its subsequent output in the form of cognition and behavior.

We have established a carousel-type RF exposure system to irradiate heads of mice with mobile phone-frequency radiation. In this exposure device, C57BL mice were exposed at 7.8 W/kg for 1 h/day, 5 days/week, for 12 months to mimic the chronic effect of RF exposure. We used 2 frequencies for Korean mobile phone users, 849 MHz and 1763 MHz. After 6 and 12 months of exposure, we examined the cell proliferation, cell death, and distribution of neurons and glial cells by immunohistochemical staining. The study was conducted for 12 months to monitor the changes in body weight, cell proliferation and cell types under our exposure conditions.

Materials and Methods

Animals

Male and female C57BL/6N mice were used for this study. All animals were kept in specific pathogen-free (SPF) mouse chambers. Male mice were housed in isolated cages alone, and female mice were housed, 6 to a plastic normal cage, with hardwood chips (Andersons, Inc., Maumee, OH) for bedding. Animals were fed with pellet diets (Orient Co. Ltd., Seoul, Korea). The SPF mouse chamber was controlled automatically to maintain a temperature of $22 \pm 2^\circ\text{C}$, relative humidity of 40-70%, and a 12-h light/dark cycle. All animal experiments were performed after receiving approval of the Institutional Animal Care and Use Committee (IACUC) in Seoul National University College of Medicine, and National Research Council (NRC) guidelines for the care and use of laboratory animals were observed.

Experimental design and exposure condition

Six-week-old mice were adapted for one week, and

then trained to load in a mouse restrainer for one week before use. For the long-term animal exposure experiment, mice were exposed to 849MHz or 1763 MHz RF radiation at the brain average SAR of 7.8 W/kg. Exposure was conducted 1 h/day, 5 days/week, for 6 or 12 months with a carousel-type head exposure system. We started with 120 mice (60 male and 60 female) and divided them into six groups of 20 mice on each sex for sham-, 849 MHz- and 1,763 MHz-exposure. At six-month of exposure, we used 10 mice for autopsy in every group to have 10 mice in each group. During one year of exposure, we lost just two mice by accident without any pathological findings in autopsy.

Immunohistochemical analysis

Mice were sacrificed by cervical dislocation. Each brain was removed from the skull. Brains were fixed for 10 days at room temperature with 10% formaldehyde (Junsei Chemical, Tokyo, Japan) in PBS and embedded in paraffin. We used the DakoCytomation EnVision+ System-HRP kit (Cat no. K4011, DAKO, Carpinteria, CA) according to the manufacturer's instructions to detect PCNA, GFAP, and NeuN. Briefly, tissue sections (4 μm) were deparaffinized and rehydrated using xylene and passage through a series of graded ethanol. Antigen retrieval was performed by boiling specimens in a microwave oven 3 times for 5 min each time in 10 mM sodium citrate buffer (pH 6.0). Endogenous peroxidase was blocked with 0.03% hydrogen peroxide containing sodium azide for 5 min. After 3 washes in distilled water, sections were incubated for 1 h at room temperature applying PCNA (1 : 200; Santa Cruz Biotechnology, Santa Cruz, CA; sc-7907), GFAP (Cat no. MAB360, Chemicon, Temecula, CA), or NeuN (Cat no. MAB377) primary monoclonal antibody mixed with a modified biotinylated anti-mouse immunoglobulin for 15 min at room temperature, after which normal mouse serum was added to the mixture. After a 5 min incubation, the biotin-labeled primary antibody was applied to the specimen for 15 min at room temperature. After PBS washing, sections were incubated with streptavidin-peroxidase for 15 min, followed by reaction with diaminobenzidine/ hydrogen peroxidase as chromogen-substrate. For nuclear counterstaining, sections were immersed in a bath of aqueous Lillie's modified Mayer's hematoxylin (Cat no. S3309, DAKO) for 1 min at room temperature.

TUNEL assay

Apoptotic cells were identified by a TUNEL method

with Apoptag Peroxidase In Situ Apoptosis Detection Kit (Cat no. S7101, Chemicon). Paraffin-embedded tissues were deparaffinized, and proteins in tissue sections were digested by applying proteinase K (20 µg/ml) to specimens for 15 min at room temperature. After washing in distilled water, endogenous peroxidase was quenched in 3% hydrogen peroxide in PBS for 5 min. As described by the manufacturer, DNA strand breaks were labeled by attaching them to digoxigenin-conjugated nucleotides in a reaction catalyzed by exogenous terminal deoxynucleotidyl transferase (TdT), and immunodetected by binding an anti-digoxigenin antibody conjugated to a peroxidase reporter molecule. The immunoreaction product was visualized using 3,3'-diaminobenzidine chromogen solution. For counterstaining of nuclei, 0.5% (w : v) methyl green in 100 mM sodium acetate, pH 4.0 was used. Sections of rat mammary gland tissue (Cat no. S7115, Chemicon) were used as a positive control (Strange *et al.*, 1995).

Body weight measurement

In our exposure experiments, mice (n = 120) were randomly grouped by RF exposure types, sham (n = 40), 849MHz (n = 40) and 1763 MHz (n = 40) with equal numbers of male and female. All animals were numbered to trace growth rate individually. Body weight of experimental animals was measured every 4 weeks and presented as mean ± SD. Statistical analysis was performed using ANOVA.

Local exposure system for mobile phone frequency radiation

We have designed the exposure system consisting of real CDMA signal source module, trays, antennae, and chamber for shielding in analogy to previous studies (Burkhardt *et al.*, 1997; Fritze *et al.*, 1997; Moros *et al.*, 1999). The chamber was designed to expose 4 trays that are stacked on supporters inside the chamber. Each tray contains

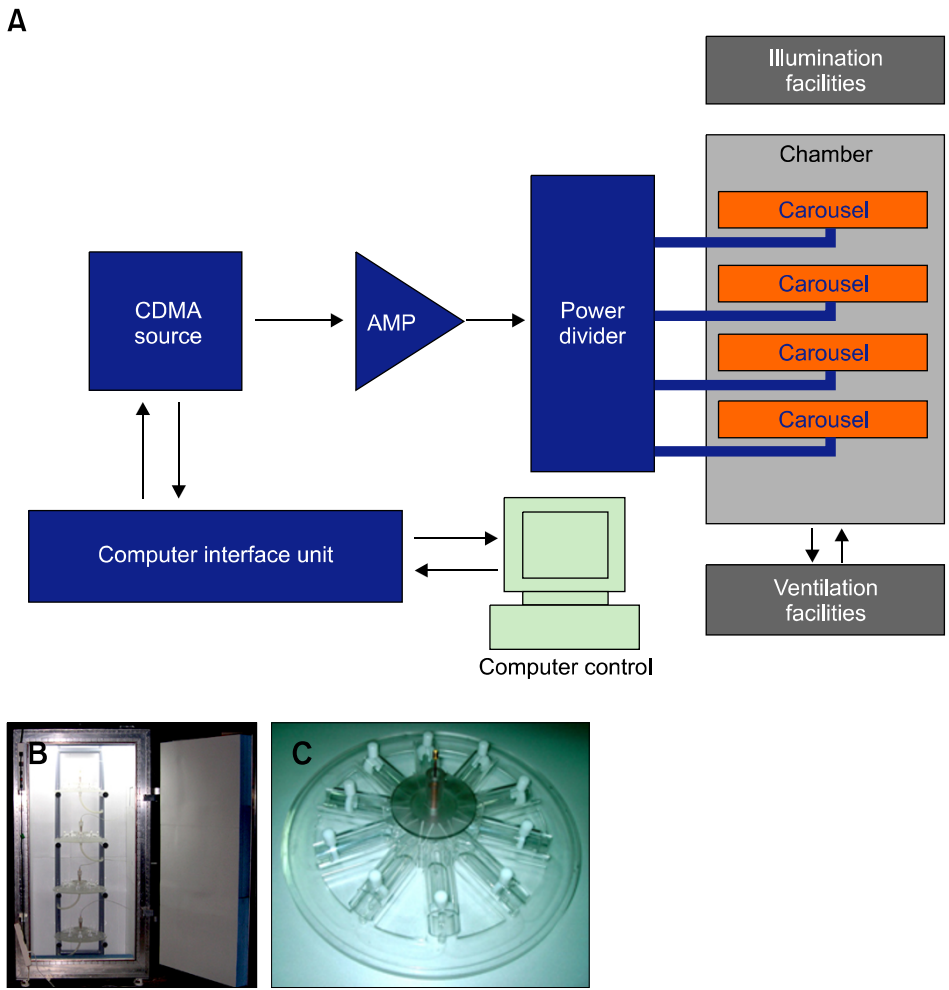


Figure 1. Carousel-type head-directed exposure system. The design for the exposure can be summarized in schematic diagram (A), and the overall view of system (B) and carousel with restrainer (C) can be visualized as pictures.

10 acrylic-tube restrainers, and thus 40 mice can be exposed at the same time (Figure 1). The chamber is made of aluminum, the external size of which is 745 mm × 1,060 mm × 1,980 mm. To reduce reflections from the walls, absorber foam with a thickness of 4.5 inches (AEL 4.5, AEMI. Inc.) is used inside the chamber, which reduces reflections at least 20 dB in the PCS frequency band. The inner diameter of the restrainer is 34 mm to match the girth of the imago mouse. The restrainers are tilted upwards about 3° for mouse waste excretion. Proximal acrylic wings that make angles of 50° with the tube's sagittal (shape of the arrow) plane are attached to the tips of restrainers. Each tray has a pipe to fix the antennae and to afford ventilation through the restrainers.

For the head-directed exposure, CDMA signals are supplied to the center of each tray by the sleeve dipole antennae (Motorola). The antenna for the exposure system is a half-wave sleeve dipole, and the vertical spacing between trays is 46

cm, which is sufficient to provide the necessary isolation between antennae. A CDMA test signal with the nominal bandwidth of 1.23 MHz is used, and the carrier frequency of the signal is 1762.5 MHz, which corresponds to one of the uplink PCS channels (channel number 250). Maximum power of the source is 30 W. The source power can be controlled from 1 W to 30 W by 1 W step. Real CDMA test signal is generated and applied to the chamber after amplification. Exposure levels and time schedules can be controlled using a

Table 1. Simulation of brain and whole body SAR using numerical mouse phantom.

Frequency	Brain average SAR (W/kg)	Whole body average SAR (W/kg)	Brain-average SAR/Whole body-average SAR (W/kg)
1,763 MHz	5.28	1.55	3.4

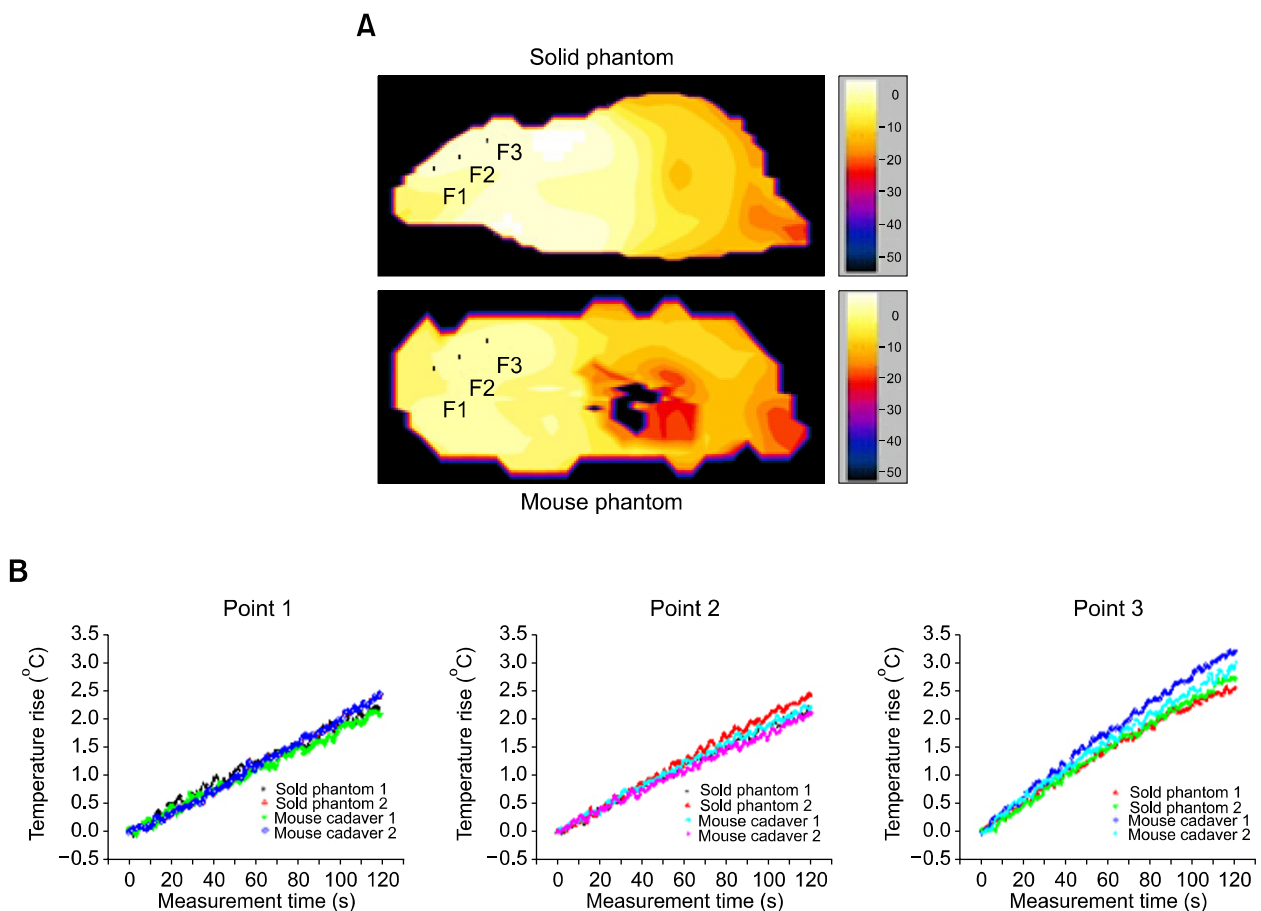


Figure 2. Simulation of SAR distribution and the thermal probe measurement at 1,763 MHz PCS band. SAR distribution in vertical cuts through the center of the mouse body was simulated (A). Temperature rises at P1, P2, and P3 in the heads of mouse cadavers and solid phantoms were measured using probes (B).

computer.

The FDTD method was used to compute SAR inside the mouse as described (Schonborn *et al.*,

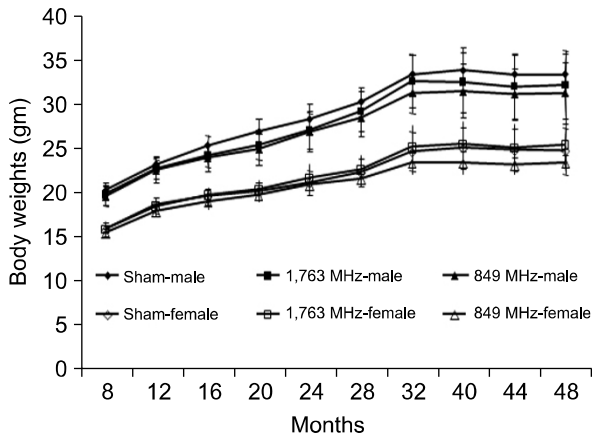


Figure 3. Changes in body weights of RF-exposed C57BL mice. The body weights of 10-20 mice in each group were measured and averaged to compare the weight gain at every 4 weeks. Error bars represent standard errors at each timepoint.

2004). The size of a mouse for simulation is 8 cm in length, 3.5 cm in height, and 3.5 cm in width. The mouse was modeled with 25 tissues, referring to the model of U.S. Air Force Research Laboratory. As summarized in Table 1, SAR inside the mouse head was measured using fluoro optic temperature probes (model 790, Luxtron Corp.). The probe was inserted into the head of an exposed mouse using a plastic catheter, and the probe was located at 3 locations in the mouse head or a brain-equivalent solid phantom (Figure 2A). The SAR distribution inside mice and solid phantoms were measured using an IR camera. In Figure 2B, we plotted the results of the temperature measurements at PCS band. In such an exposure system, the accurate prediction of SAR distribution inside mice as well as ideal environmental conditions, such as illumination and ventilation to minimize stress on mice, are very important. To supply fresh air, a twofold ventilation scheme was used, one for the chamber using fans, and the other through restrainers using blowers. Proper illumination was also provided by fluorescent lamps (150-300 Lux). The sound noise in the

A

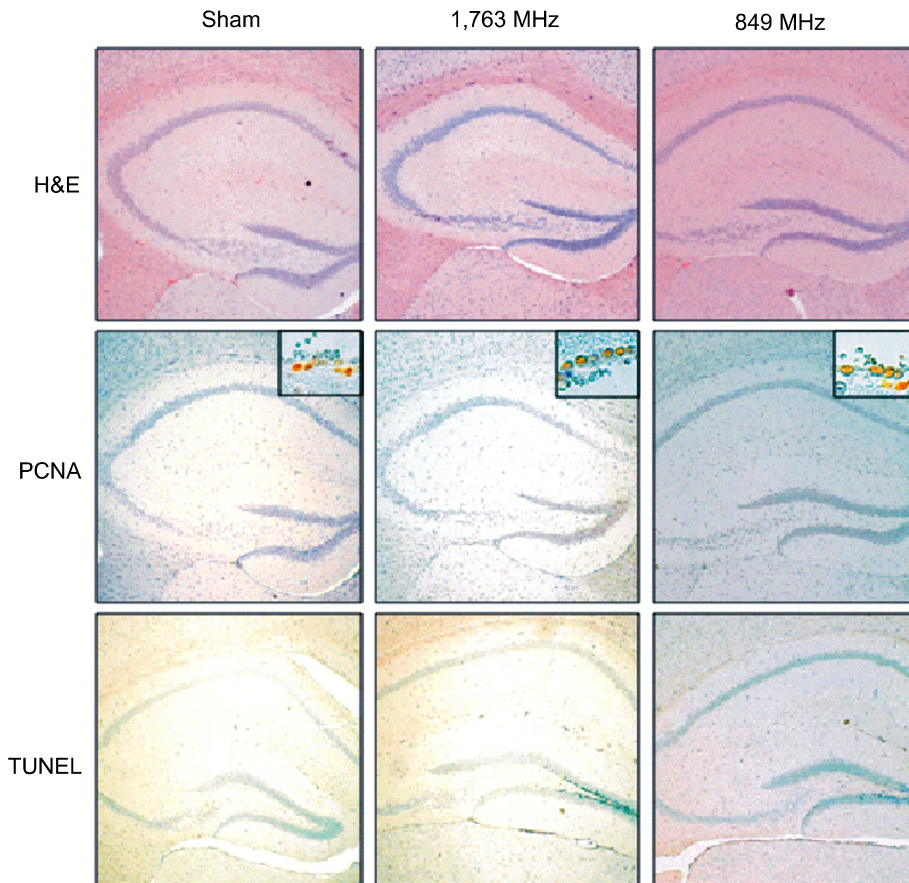


Figure 4. Effect of 1763 MHz RF exposure on cell proliferation and apoptosis in brain. Hippocampus of sham- and RF-exposed mouse were analyzed by H&E staining, immunohistochemical staining with anti-PCNA antibody, and TUNEL assay after 6 months (A) or 12 months (B). Cerebellum was also examined at 12 months after RF exposure (C). All the images were analyzed at $\times 40$, while we used higher magnification for the positive controls for the cell proliferation assay for the subventricular zone of the mouse brain ($\times 400$) and a positive control provided by manufacturer for TUNEL assay ($\times 200$).

chamber was sufficiently low (< 50 dB).

Results

Body weight changes upon RF radiation

In order to observe the effect of RF exposure to the head, C57BL mice were exposed to mobile phone frequency radiations of 849 MHz and 1763 MHz at a 7.8 W/kg SAR for 12 months, and the whole-body weight of male and female mice was measured every 4 week to check the effect on general conditions of the mice (Figure 3). The

average whole-body weight of 10-20 mice per group was calculated for each group in every four week, excluding two dead mice in 1,763 MHz-exposed female mice at the age of 44 weeks by accident. When we compared RF radiation-exposed groups with the sham-exposed group, mean body weights of surviving mice were not significantly different between groups by ANOVA in both males and females.

Cell proliferation and apoptosis after RF radiation

During RF radiation for 12 months, we could not

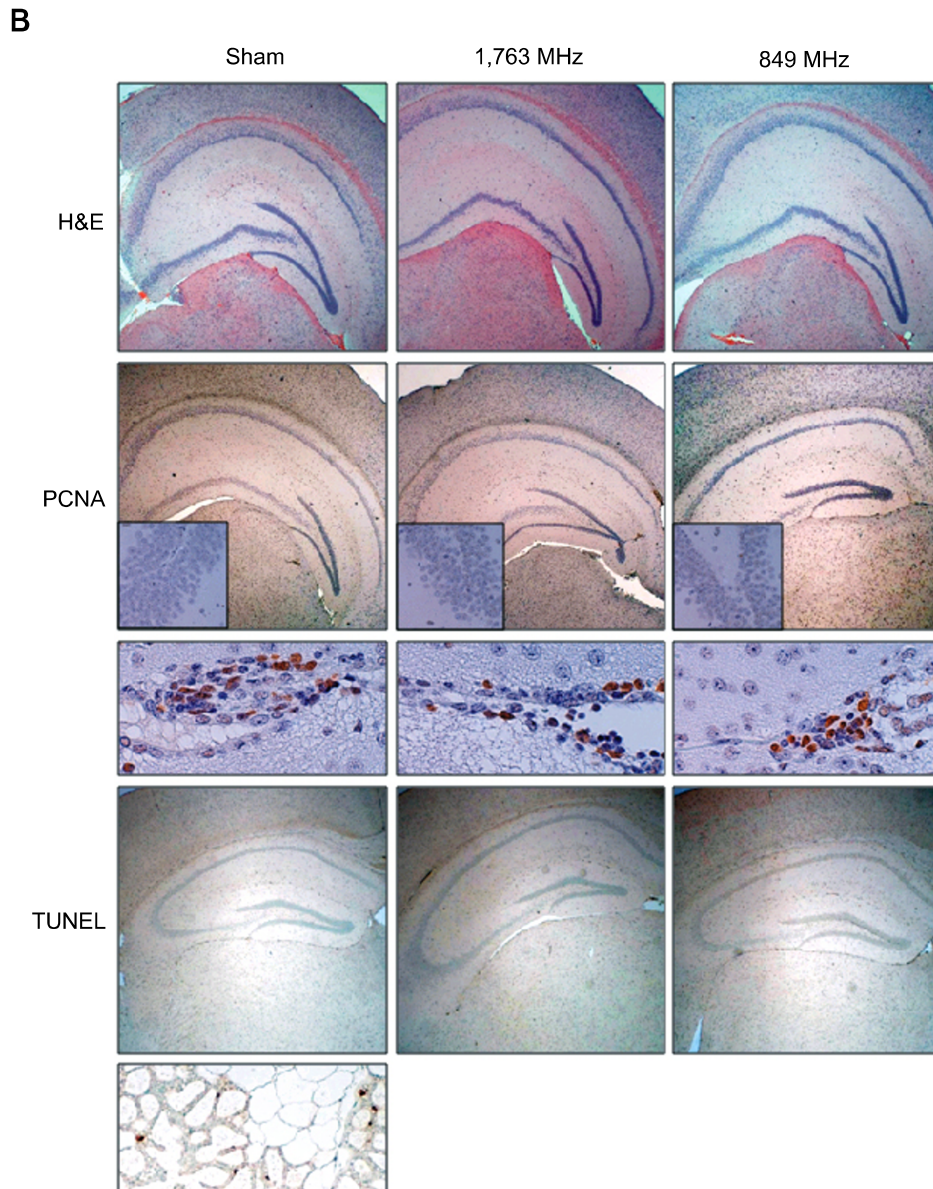


Figure 4. Continued.

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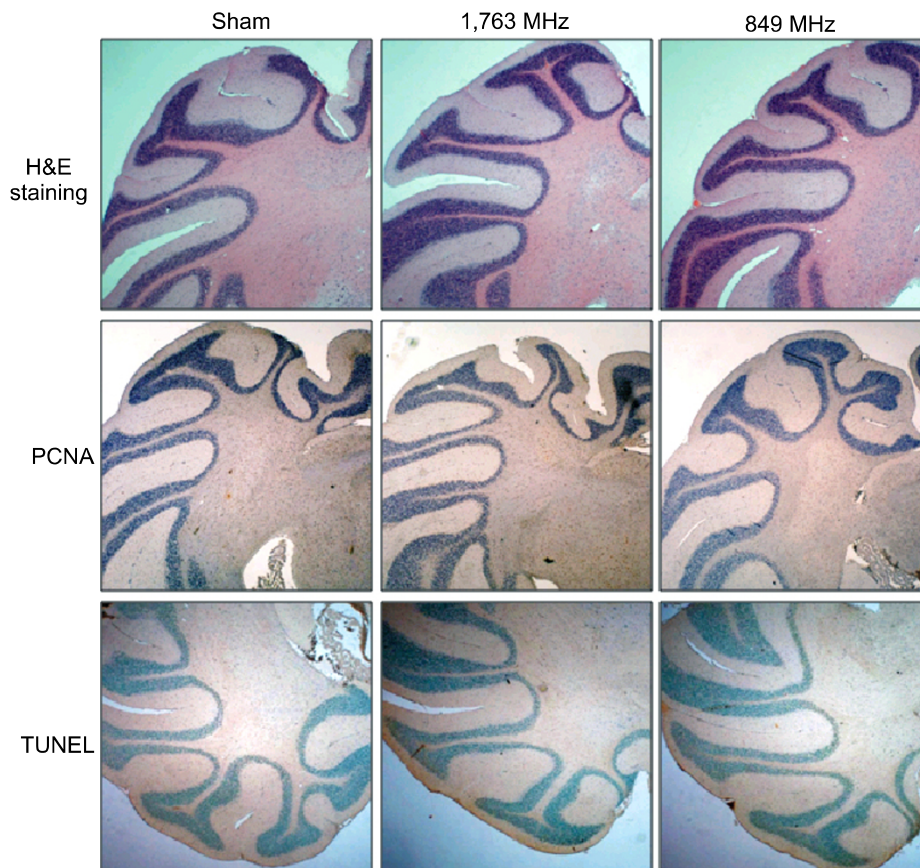


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find any gross changes in the head area or any behavioral disturbances. At 6 and 12 months of RF exposure, we sacrificed 10 mice from each time-point to examine the brain in more detail by histological analysis. As shown in Figure 4, we could not find any changes in the pattern of H&E staining in each group. We especially wanted to check the proliferation of cells upon RF radiation, which might lead to hyperplasia of target cells. To this end, we stained PCNA in the whole brain, and focused on the subgranular area in dentate gyrus of hippocampus and the subventricular area, where progenitor cells are enriched. However, we could not detect any signal in the cortex or midbrain area, and the number of PCNA-positive cells in the subgranular layer and subventricular area of RF-exposed mice were not different from that of sham-exposed mice. This finding was detected in both frequencies as well.

On the other hand, we also checked cell death upon RF radiation by TUNEL staining (Figure 4). We could not detect any positive signals in the examined tissues, while positive-control tissue sections provided by the manufacturer showed

strong signals.

Distribution of neuronal and glial cells in RF irradiated mouse brain

Overall findings under microscopic examination revealed no changes in cell proliferation potentials or cell death. Gross morphology did not show any changes, either. Although PCNA staining in Figure 4 did not show any difference in three groups, we wanted to check the distribution of neuronal or glial cells in brain. Because we started the RF exposure schedule in 8-week-old animals, their brain tissues were still under development, and might experience memory and education during the period of RF exposure. We checked the level and distribution of NeuN in neuronal cells and of GFAP in glial cells (Figure 5). At 6 and 12 months of exposure, we could not find any difference in hippocampus and cerebellum between the groups. Together with PCNA staining patterns in the progenitor cell area, we could exclude the possibility that RF radiation might interfere with development of the postnatal brain.

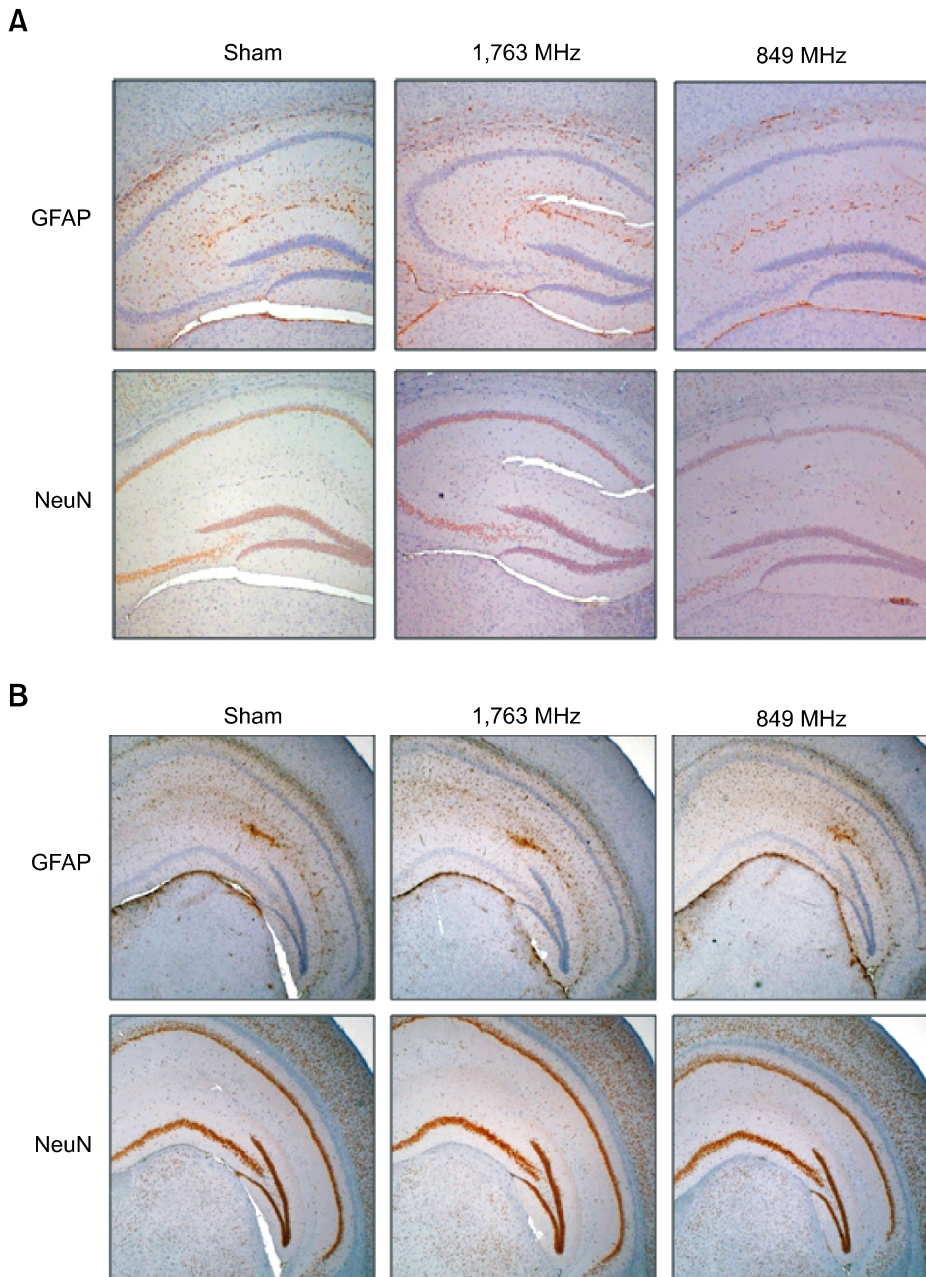


Figure 5. Influence of 1763 MHz RF exposure on glial and neuronal cells. The expressions of GFAP and NeuN were analyzed by immunohistochemical staining in hippocampus at 6 months (A) and 12 months (B) after RF exposure and in cerebellum at 12 month after RF exposure (C). Overall images were analyzed at $\times 40$ magnification, while insets were observed at $\times 400$.

Discussion

To explore the biological effect of RF radiation, the head of C57BL mice were intermittently irradiated with 849 MHz and 1,763 MHz frequency bands at 7.8 W/kg for 12 months. After 6 and 12 months of RF exposure, hippocampus and cerebellum of irradiated mouse brain were analyzed by immunohistochemical staining against PCNA, NeuN and GFAP as well as by TUNEL assay. In this study, we could not find any evidence supporting the

alterations in cell proliferation, apoptosis, reactive gliosis.

We started with 20 mice per group of each sex, and kept them for 12 months except two dead mice at the end of study. As shown in Figure 3, the body weights of all three groups in each sex were not significantly different each other. We have calculated the statistical power on the condition of alpha error = 0.05 and 10% differences in body weight change of 10 animal/group. The calculated statistical power was 66% (beta error = 0.96) to

C

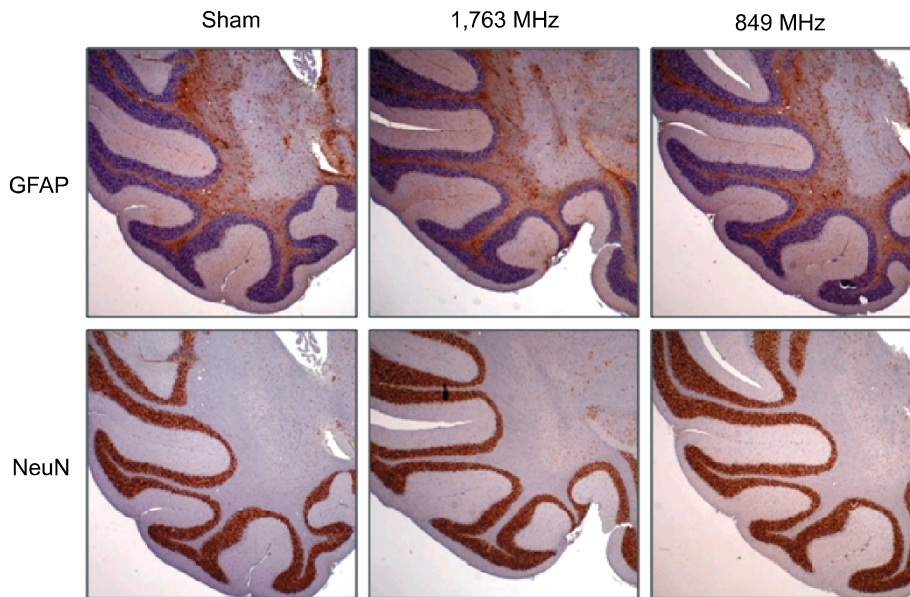


Figure 5. Continued.

detect 10% differences, which necessitates further study to confirm the reproducibility. However, we could hardly detect the differences in body weight change in three groups. To detect 1% of difference in body weight, the statistical power of this study was more than 90%.

In epidemiological studies, RF radiation might be related to brain tumors, such as acoustic neuroma (Hardell *et al.*, 1999), although it has not been proven by further studies (Hamblin *et al.*, 2004; Hinrichs and Heinze, 2004; Curcio *et al.*, 2005; Huber *et al.*, 2005) and animal experiments (Salford *et al.*, 2003; Zook and Simmens, 2006). If RF-exposed mice carried a certain tumor, we would expect changes in body weights. However, we could not detect any significant increase of body weight in RF-exposed groups of either sex. RF radiation might also induce cell death in the exposed area, but there was no decrease of body weight in RF-exposed mice. We would like to test whether RF radiation can induce brain tumors, but this hypothesis was excluded in our model.

As we observed in our gross examination, we could not find any evidence of tumorigenesis by RF radiation under microscopic examination. Increased expression of PCNA is one of the characteristics of cell proliferation, but it was not induced by RF radiation. In addition, PCNA can stain progenitor cells, such as neural stem cells, in localized areas. The patterns and the numbers of the distribution of these progenitor cells were not changed upon RF radiation, which indicate that neural differentiation was not affected by RF radiation, either. Because

the electric field might alter the neural differentiation *in vitro*, RF radiation might be involved in the developmental process (Rajnicek *et al.*, 2006). Although we could not rule out the possibility of the modulation of cellular differentiation by RF radiation, local exposure to heads *in vivo* did not affect the progenitor cells in our experiments.

Most studies that address the effects of RF exposure on cancer development or cell death have shown no significant change after long-term, low-level exposure using animal models (Frei *et al.*, 1998; Jauchem *et al.*, 2001). We have shown that 849 MHz and 1763 MHz RF radiation from a mobile phone source does not affect cell proliferation or apoptosis in brain. Although 2.4 GHz continuous wave RF radiation could reduce the life span of exposed mice (Liddle *et al.*, 1994), most studies on the effect of RF radiation concur that there is no significant increase or decrease in body weight following RF exposure.

In this study, we investigated the effect of local exposure of mobile phone frequency radiation to the mouse brain for 12 months. We could not find any evidence of increased cell proliferation or cell death. Reactive gliosis is a phenomenon describing endogenous CNS tissue responses to injury. Reactive gliosis specifically referred to the accumulation of enlarged glial cells, notably microglia and astrocytes, appearing immediately after CNS injury has occurred. The glial reactivity might be a largely passive response to injury (Norton *et al.*, 1992). To measure reactive gliosis, we stained glial cell markers such as GFAP, but we could not find

any increase of GFAP signal or any alteration of distribution of its signal. The data presented here suggest that exposure to 849 MHz and 1763 MHz of RF radiation from mobile phones at a 7.8 W/kg SAR does not change the pattern of neuronal and glial cells in hippocampus and cerebellum of C57BL mice.

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