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# Effects of 915 MHz Radiofrequency Identification Electromagnetic Field Exposure on Neuronal Precursor Cells in the Dentate Gyrus of Adult Rat Brains

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## Abstract

To explore the effects of radiofrequency electromagnetic field on the fate of neuronal cells, we investigated whether exposure to 915 MHz radiofrequency identification (RFID) caused morphological changes in neuronal cells in rat hippocampal dentate gyrus (DG). A reverberation chamber was used as a whole-body RFID exposure system. Rats were assigned to two groups: sham- and RFID-exposed groups. Rats in the RFID-exposed group were exposed to RFID at 4 W/kg specific absorption rate (SAR) for 8 hours daily, 5 days per week, for 2 weeks. Morphological evaluation of DG was performed using immunohistochemistry with doublecortin (DCX) as a neuronal precursor cell marker and neuronal nuclei (NeuN) as a mature neuronal cell marker. No significant morphological changes in DCX+ or NeuN+ cells in the DG of RFID-exposed rats were observed. These results suggest that RFID exposure induces no significant change in DCX+ neuronal precursor or NeuN+ neuronal cells in DG of rats.

**Key Words**: 915 MHz RFID, DCX, Dentate Gyrus, NeuN.

## I. Introduction

The huge increase in the use of telecommunication systems has caused concerns regarding the potential detrimental effects of exposure to radiofrequency electromagnetic fields (RF-EMFs). The potential effects of EMF on human health vary widely depending on the frequency and intensity of the field [1]. In the central nervous system, electrical signals play a crucial role in regulating neural activity [2]; therefore, RF-EMFs could affect neural activity [3, 4].

Adult neurogenesis, a delicate process of generating functional neurons from adult neuronal precursors, occurs throughout life in the two neurogenic zones: the subventricular zone (SVZ) and the subgranular zone of the hippocampal dentate gyrus (DG) in mammalian brains [5]. Neuronal progenitor cells within SGZ migrate into the granular cell layer, differentiate into the mature granule cells, and are integrated into the neuronal circuit [6]. Newly generated neu-

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rons are important for the maintenance of functional plasticity of the brain and contribute to learning and memory [5, 6]. Developing nervous cells can be altered by various factors such as disease, drug [7], diet [8], exercise [6], and aging [9].

The electromagnetic spectrum extends from below the low-frequencies used for radio communication to gamma radiation within the short-wavelength region. EMFs surround us and are present in two different forms, including extreme low frequency (ELF)-EMF and RF-EMF. ELF-EMFs (3 Hz to 3,000 kHz) are emitted from electrical power supplies and appliances; RF-EMFs (10 MHz to 300 GHz) are emitted from wireless devices, such as mobile phones and other communication devices, including radar.

Regarding the biological effects of ELF-EMFs on neuronal cells, they can alter post-natal neurogenesis [10, 11]. Recent *in vitro* studies have suggested that exposure of human bone marrow-derived mesenchymal stem cells to ELF-EMF decreases their proliferation rate instead of causing an increase in both osteogenic and neuronal differentiation [12-15]. To explore the effects of RF-EMFs on cultured neuronal cells, the growth and differentiation of a standard neuronal-like cell line was studied. It has also been reported that pulsed RF-EMFs can alter PC-6 [16]. Enhancement of neurite outgrowth has been observed after pulsed magnetic field (PMF) exposure to dorsal root ganglia explant cultures from 15-day-old Sprague-Dawley rats [17]. The authors of that study suggested that PMF may provide a noninvasive mechanism to promote nerve regeneration [17].

Regarding the biological effects of RF-EMFs on neuronal cells, exposure to such fields can induce a reduction in neurite generation and delay neurite outgrowth in the murine SN56 cholinergic cell line and rat primary cortical neurons [18]. In that study, morphological maturation was induced by continuous exposure to GSM-modulated 900 MHz EMF at 1 W/kg SAR [18]. A time delay in the process of neurite outgrowth may affect neurogenesis because neuronal activity is closely associated with the morphological maturation of neuronal cells [18]. According to our previous study with 848.5 MHz code division multiple access (CDMA) EMF, the differentiation of BrdU-positive cells in hippocampal DG and the SVZ were not affected after CDMA-EMF exposure at 2 W/kg SAR for 8 hours daily for 2 weeks in rats [19]. Previous studies could not demonstrate the influence of RF-EMFs on neurogenesis processes and on the fate of neuronal precursor cells in healthy adult rat brains. To evaluate the effects of 915 MHz RFID on neuronal cell fate, we explored whether morphological changes in the neuronal precursor cell occurred in the hippocampal DG based on immunohistochemical evaluation.

## II. Materials and Methods

A 915 MHz RFID exposure system for rats was established and its validation was completed before starting the animal studies [20]. A whole-body exposure system was designed for small animal experiments using a reverberation chamber; this system was validated under the supervision of the Korea Electromagnetic Institute Engineering Society (Fig. 1). A 915-MHz RFID signal, originating from an RFID source, was amplified using a pre-amplifier module (Kortcom, Anyang, Korea). Subsequently, the amplified signal was modulated for an input value control of a power amplifier (Kortcom) using a digital attenuator (Kortcom), followed by generation of modulated signals through an RFID antenna. The field distribution and field uniformity in the reverberation chamber were tested based on direct measurements using a three-axis isotropic probe (HI-6105; ETS-Lindgren, Cedar Park, TX, USA). The SAR distribution for a caged rat was calculated from the measured electric field strength using a commercial finite-difference time domain (FDTD) tool (XFDTD ver. 6.5; Remcom, State College, PA, USA) for the incident plane waves in six orthogonal directions with two polarizations, as described previously [21]. The calculated SAR values were then averaged and multiplied by the measured root mean square (RMS) electric field value to estimate real SAR values for a given input power. The electromagnetic waves in the experimental region of the reverberation chamber originated from all directions with equal amplitude. The rat model of the American Air Force Research Lab (AFRL, Dayton, OH, USA) was used for the simulation. For 4 W/kg of whole-body average SAR, the power output was controlled at 56 W for RFID. Then the amplified RFID signal was supplied to the chamber (Fig. 1). An 11-bit digital personal identification number diode attenuator (Model 349; General Microwave, Farmingdale, NY, USA) was used to control output power level.

Adult male Sprague–Dawley rats (6 weeks old, weight 200–250 g) were purchased from Dea-Han Biolink (Seoul, Korea) and used after 7 days of acclimation. They were housed in a conventional state under adequate temperature (22℃ ± 0.2℃) and a controlled 12/12 hour light/dark cycle with free access to water and food. All experimental procedures were in accordance with the Ethics Committee guidelines for animal experiments of Ajou University School of Medicine, Suwon, Korea.

To explore the effects of RFID exposure on neuronal cell maturation in the hippocampal DG, animals were divided into two groups (n = 4 in each group): the sham-exposed and


Fig. 1. A whole-body 915-MHz radiofrequency identification (RFID) exposure system. This system was designed for small animal experiments with a continuous wave 915-MHz RFID signal generation using a reverberation chamber.

RFID-exposed groups. Rats in the sham-exposed group were placed in the exposure system without exposure to RFID, while rats in the RFID exposed group were placed in the exposure system and exposed to RFID. There was no access to water or food during the time in the exposure system for either group. The rats were exposed at an SAR value of 4 W/kg at 915 MHz RFID for 8 hours daily, 5 days per week, for 2 weeks [20, 22]. Two days after the last RFID exposure, animals were anesthetized with 10% chloral hydrate 400 mg/kg (Sigma-Aldrich, St. Louis, MO, USA) and perfused transcardially with 0.9% saline followed by 4% paraformaldehyde (Sigma-Aldrich) in 0.1 M phosphate buffer (pH 7.2). The brains were removed and post-fixed overnight at 4℃. The brain tissues were cryoprotected by infiltration with 30% sucrose until they sank. Thereafter, six separate series of 40-μm coronal brain sections through the entire hippocampus (480 μm between sections) were obtained using a sliding microtome (Microm International, Walldorf, Germany) and stored in a solution at 4℃ until immunohistochemical analysis.

To obtain accurate data for immunohistochemistry, the free-floating sections were carefully processed under the same conditions. Immunohistochemistry was performed on a one-in-six series of equidistant (240 μm between sections) sections. The sections were sequentially treated with 0.3% hydrogen peroxide and 5% bovine serum albumin. Then they were incubated with diluted goat anti-DCX (1:100, Santacruz, Dallas, TX, USA) and mouse anti-NeuN (1:500, Millpore, Billerica, MA, USA) overnight at room temperature and subsequently incubated for 1 hour at room temperature with biotinylated anti-mouse and anti-goat IgG (1:200, Vector Laboratories, Burlingame, CA, USA), before being incubated for 1 hour in avidin/biotin substrate (ABC kit, Vector Laboratories). Then the sections were reacted with 3,3'-diaminobenzidine (DAB) solution (Vector Laboratories) and mounted on gelatin-coated slides.

Images of all DCX- and NeuN-positive structures were obtained from the hippocampal DG using BX54 bright-field microscopy (Olympus Optical, Tokyo, Japan). Bright-field images were obtained using the Picture Frame Application 2.3 software (Olympus Optical). The data shown here represent the mean ± SEM. Differences in the means among the groups were statistically analyzed using the Mann-Whitney *U* Test. Statistical significance was considered to be $p<0.05$.

## III. Results and Discussion

Photomicrographs show regions of DG stained with DCX marker in the sham- and RFID-exposed rat brains. DCX-positive cells in the hippocampal DG were observed using a microscope, and no significant morphological changes in DCX-positive cells in hippocampal DG caused by RFID exposure were observed in the sham-exposed group or the RFID-exposed group (n = 4 per group per trial) (Fig. 2). Magnifications of left- and right-side photomicrographs are 1× and 40×, respectively.

Photomicrographs show the region of DG stained with NeuN marker in the sham- and RFID-exposed rat brain. NeuN-positive cells in the hippocampal DG were observed using a microscope. No significant morphological changes in NeuN-positive cells in hippocampal DG caused by RFID exposure were observed in the sham-exposed group or the RFID-exposed group (n = 4 per group per trial) (Fig. 3). Magnifications of left-, middle-, and right-side photomicrographs are 4×, 10×, and 40×, respectively.

We examined the effects of 2 weeks of exposure to RFID signals on the morphological fate of neuronal precursor cells, such as the DCX-positive neuronal precursor cells and NeuN-positive mature neuronal cells, in the DG of healthy adult rat brain. Our preliminary results suggest that a 2-week exposure to 4 W/kg SAR did not induce significant morphological changes in DCX-positive cells or NeuN-positive cells in the DG of adult rat brain under our experimental conditions. These findings are of value because daily exposure to 8 hours of RFID (mimicking occupational exposure conditions) had no harmful effect on neuronal precursor cells in young healthy adult rat brain.

Few animal experimental reports have shown detrimental

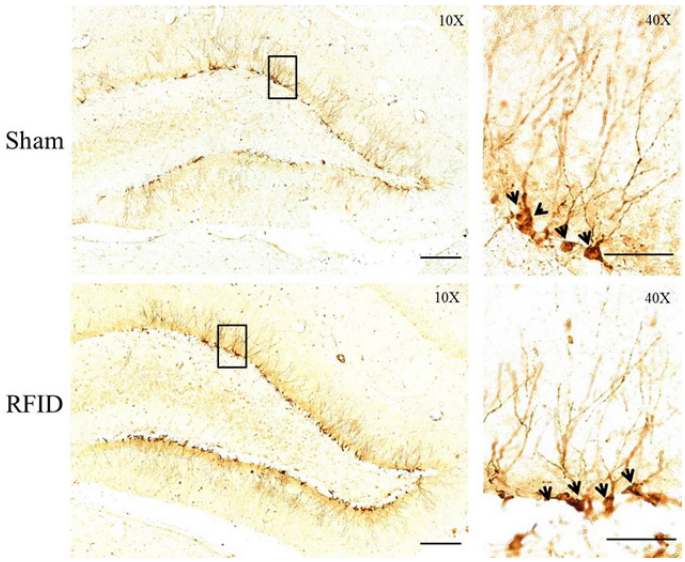


Fig. 2. Representative micrographs of the hippocampal dentate gyrus stained with DCX in the sham-exposed (upper panel) and RFID-exposed rats (lower panel). No significant RFID-exposure-induced morphological changes in DCX-positive cells were observed in the RFID-exposed rats (n = 4 per group per trial). Magnifications of the photomicrographs are 10× (left) and 40× (right). Scale bars = 200 μm and 50 μm, respectively. DCX = doublecortin, RFID = radiofrequency identification.

effects of RF-EMF on neuronal cells in the rat brain [23, 24]. In one study, it was shown that exposure to 900-MHz EMF induced significant apoptotic neuronal cell death in the cornu ammonis region of the rat brain [23]. In that study, 16-week-old female rats were exposed to 900-MHz continuous modulated EMF at an SAR of 2 W/kg for 1 hour daily over 28 days [23]. The authors suggested that the long duration of EMF exposure caused a significant reduction in the number of cerebellar Purkinje cells in female rats. However, they provided no definite evidence based on apoptotic cell death or immunohistochemical assays with neuronal cell markers. In that study, daily RF-EMF exposure duration was only 1 hour, which is significantly shorter than in other previous studies. Therefore, their experiments may need to be repeated to confirm that these findings are reproducible. In a second study, it was shown that widespread damage to rat hippocampal neurons was caused by only 2-hour EMF exposure [24]. In the experiments, rats were exposed to 915-MHz mobile GSM-modulated EMF at whole-body SARs of 2 mW/kg, 20 mW/kg, and 200 mW/kg, and were scarified 50 days after exposure to observe morphological changes in the hippocampal neurons. We suggest that this time delay may provide sufficient time for recovery from damage of neuronal cells induced by EMF exposure. If their exposure conditions were accurate, the effects of exposure to mobile GSM-modulated EMF could be serious. Therefore, this trial may also need to be repeated to confirm whether it is reproducible. Moreover, two previous studies that explored the effects of EMF exposure under the same conditions showed contradictory results [23, 25].

One observed no evidence of increased neuronal damage in the brain after acute exposure to pulsed 915-MHz EMF [25]. Rats were exposed to pulsed 915-MHz EMF at average whole-body SARs of 2 mW/kg, 20 mW/kg, and 200

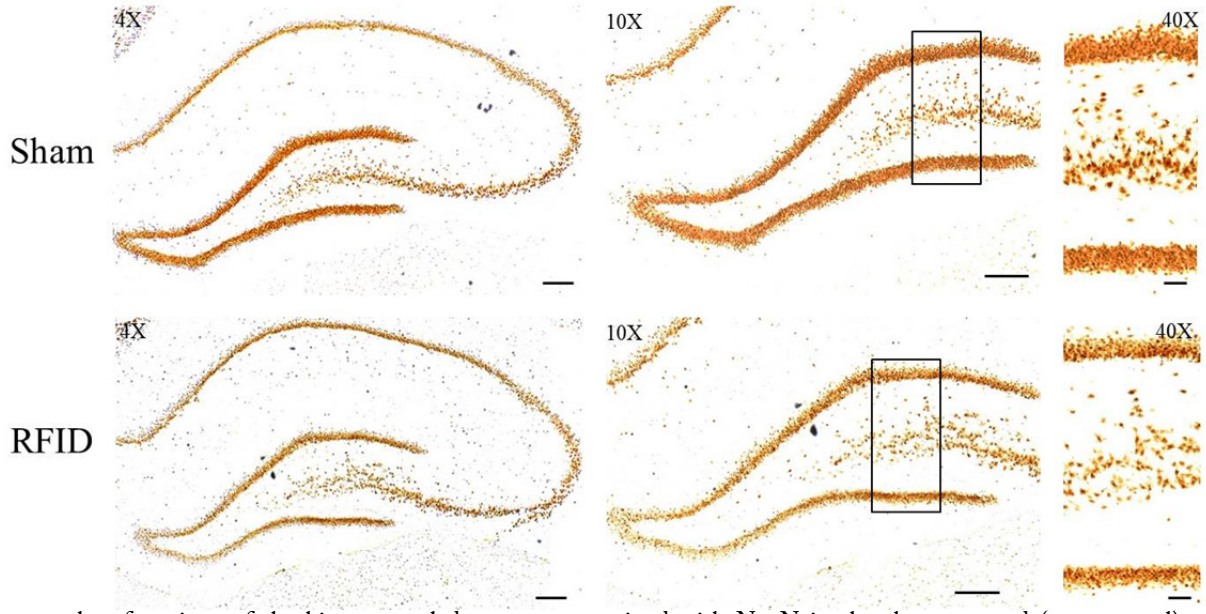


Fig. 3. Micrographs of sections of the hippocampal dentate gyrus stained with NeuN in the sham-exposed (upper panel) and RFID-exposed rats (lower panel). No significant RFID-exposure-induced morphological changes in NeuN-positive cells were observed in the RFID-exposed rats (n = 4 per group per trial). Magnifications of the photomicrographs are 4× (left), 10× (middle), and 40× (right). Scale bar = 200 μm, 200 μm, and 50 μm, respectively. NeuN = neuronal nuclei, RFID = radiofrequency identification.

mW/kg once for 2 hours [25]. The rats were sacrificed 50 days after exposure, and the daily duration of RF exposure and SAR values were the same as in the other, contradictory study [23]. In addition, a mouse study demonstrated that daily exposure to 900-MHz EMF did not induce neuronal degeneration in hippocampal DG [26]. In that study, the heads of C57BL mice were exposed to both 849-MHz or 1,763-MHz mobile-phone RF radiation at an SAR of 7.8 W/kg for 6 or 12 months [26]. Another study showed no change in NeuN-positive neuronal cells in the hippocampus of RF-exposed rats [27], which was similar to the results from the present study. Although that study was performed with a longer duration and stronger RF-EMF exposure than in the present study, no change was reported in neuronal cell fate. In our previous animal study with whole-body exposure to a 848.5-MHz CDMA signal at an SAR of 2 W/kg for 2 weeks, we observed no change in the numbers of BrdU-positive proliferating cells in the SVZ and DG [19]. Taken together with our previous report and the present study, although the experimental environments were different (local versus whole body, 1 day versus several weeks, and 915-MHz RFID versus 848.5-MHz CDMA), the RF-EMF exposure at an SAR of 2 W/kg had no harmful effects on neuronal cells in the adult rat brain.

The effects of EMF exposure on neuronal progenitor cells remain unclear. A previous report suggested that exposure to 50-MHz ELF-EMF induces significant neurogenesis in the brains of adult mice [28]. In that study, the mice were exposed for 1 to 7 hr/day for 7 days and were assessed based on immunohistochemical evaluation. The authors observed increased numbers of cells that were double-stained for BrdU and DCX [28]. Conversely, chronic exposure to 849-MHz and 1,763-MHz RF-EMF at an SAR of 7.8 W/kg induced no change in the numbers of proliferating cell nuclear antigen (PCNA)-positive neuronal progenitor cells in the hippocampus of RF-exposed rats [27]. PCNA can stain both neuronal progenitor and proliferating cells. In the present study, we observed no significant morphological changes in DCX-positive neuronal precursor cells in RFID-exposed rats. However, it is difficult to obtain a definitive conclusion with only a small number of trials. Therefore, further studies may be required under various exposure conditions (such as different RF frequencies, SAR values, and duration of RFID exposure, and animals of different sex, physical status, and age).

In most studies, the primary evidence of neurogenesis is obtained based on BrdU immunostaining. However, immunohistochemical identification of newly generated cells as neurons is accomplished based on double-staining for BrdU and NeuN [29]. DCX expression is specific to newborn healthy neurons, because nearly all DCX-positive cells express early neuronal markers such as PCNA, TUJ-1, and TUC-4. Thus, in this study, DCX was used as a marker of newborn neurons in the adult DG. In some previous reports, cresyl violet staining alone was performed to examine RF-EMF exposure-induced damage of neuronal cells [23, 24]. Because the brain is primarily composed of two broad classes of cells, neurons and glial cells [30], discrimination between neuronal cells and glial cells is essential to define the cell type damaged by RF-EMF exposure. Previous reports evaluated a collation between NeuN and cresyl violet staining to detect neurons [31]. Both neuronal cells and glial cells can easily be stained by cresyl violet; however, cresyl-violet-stained small neurons are indistinguishable from glia. NeuN is a neuron-specific marker because it selectively stains neuronal perikarya and nuclei [32]. Therefore, in this study, we used NeuN instead of the nonspecific marker cresyl violet, to distinguish small neurons from glia.

Regarding the limitations of this study, we summarized only morphological findings without stereological cell counting. Although adult neurogenesis can be assessed both in DG and SVG, we showed only results for DG in this report. To define the maturation of neuronal cells, immunohistochemical staining was performed in this study because the maturation degree of neuronal cells is difficult to define due to inevitable overlapping of the maturation stage. To evaluate the effects of RFID on neuronal maturation further, double immunohistochemistry staining with BrdU/DCX and DCX/NeuN in neuronal cells, as well as single immunohistochemistry staining with a stage-specific marker, may be required. Morphological evaluations are being performed for both stereological counting of cells and double-immunohistochemical staining.

## IV. Conclusion

In conclusion, 915-MHz RFID exposure may cause no significant morphological changes in DCX-positive neuronal precursor cells and NeuN-positive neuronal cells in the hippocampal DG of young, healthy adult male rats at 4 W/kg whole-body SAR.

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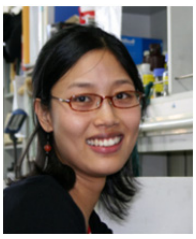

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