Noise Magnetic Fields Abolish the Gap Junction Intercellular Communication Suppression Induced by 50 Hz Magnetic Fields

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Previously, we have reported that exposure to 50 Hz coherent sinusoidal magnetic fields (MF) for 24 h inhibits gap junction intercellular communication (GJIC) in mammalian cells at an intensity of 0.4 mT and enhances the inhibition effect of 12-*O*-tetradecanoylphorbol-13-acetate (TPA) at 0.2 mT. In the present study, we further explored the effects of incoherent noise MF on MF-induced GJIC inhibition. GJIC was determined by fluorescence recovery after photobleaching (FRAP) with a laser-scanning confocal microscope. The rate of fluorescence recovery (R) at 10 min after photobleaching was adopted as the functional index of GJIC. The R-value of NIH3T3 cells exposed to 50 Hz sinusoidal MF at 0.4 mT for 24 h was $30.85 \pm 14.70\%$, while the cells in sham exposure group had an R-value of $46.36 \pm 20.68\%$, demonstrating that the GJIC of NIH3T3 cells was significantly inhibited by MF exposure ($P < .05$). However, there were no significant differences in the R-values of the sham exposure, MF-plus-noise MF exposure (R: $49.58 \pm 19.38\%$), and noise MF exposure groups (R: $46.74 \pm 21.14\%$) ($P > .05$), indicating that the superposition of a noise MF alleviated the suppression of GJIC induced by the 50 Hz MF. In addition, although MF at an intensity of 0.2 mT synergistically enhanced TPA-induced GJIC inhibition (R: $24.90 \pm 13.50\%$ vs. $35.82 \pm 17.18\%$, $P < .05$), further imposition of a noise MF abolished the synergistic effect of coherent MF (R: $32.51 \pm 18.37\%$). Overall, the present data clearly showed that although noise MF itself had no effect on GJIC of NIH3T3 cells, its superposition onto a coherent sinusoidal MF at the same intensity abolished MF-induced GJIC suppression. This is the first report showing that noise MF neutralizes 50 Hz MF-induced biological effect by using a signaling component as the test endpoint. Bioelectromagnetics 27:1–6, 2006. © 2006 Wiley-Liss, Inc.

Key words: ELF magnetic fields; noise magnetic fields; gap junction intercellular communication

INTRODUCTION

Epidemiological studies suggest that exposure to extremely low frequency electromagnetic fields (ELF EMF) might increase the risk of certain types of cancer, particularly childhood leukemia [Wertheimer and Leeper, 1979; Lin and Lee, 1994]. Based on the potential association between childhood leukemia and residential exposure to ELF magnetic fields (MF), the National Institute of Environmental Health Sciences (NIEHS) (1998) and the International Agency for Research on Cancer (IARC) (2001) independently classify ELF MF as a possible human carcinogen. The cancer promotion possibility of ELF MF has led to numerous studies aiming to elucidate the underlying mechanisms at cellular and molecular levels. For example, low-intensity MF was reported to induce c-*myc* transcription [Phillips et al., 1992], increase HSP70 synthesis [Pipkin et al., 1999], and enhance ornithine decarboxylase (ODC) activity in L929 cells [Litovitz et al., 1991]. However, there are also reports showing MF does not affect these endpoints [Miyakoshi et al., 1996; Morehouse and Owen, 2000a,b]. Therefore,

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further studies are needed to solve the apparent inconsistency of these results.

Biophysical mechanisms concerning the initial interaction between ELF MF and biological systems are fundamental to understand MF-induced bioeffects. It is still unclear how cells respond to external EMF, which is 100–1000 times weaker than the random thermal noise fields generated by the thermal motion of the ions in and around the cells. To address this signal-to-noise puzzle, Litovitz et al. [1994] proposed that living cells distinguish thermal noise fields from external EMF because thermal noise fields are temporally and spatially incoherent while external EMF is spatially coherent. When external fields are both spatially and temporally coherent, they would induce biological effects. He deduced that the effects would be interfered with, by superposing a spatially coherent but random noise MF onto the spatially and temporally coherent MF.

This deduction was supported by a line of experimental evidence. The combined exposure of noise MF and MF abolished MF-induced bioeffects, such as cell growth in human epithelial amnion cells [Raskmark and Kwee, 1996], abnormalities of chicken embryos [Litovitz et al., 1994], increasing of c-*myc* transcript in HL60 cells [Lin and Goodman, 1995], reduction of 5′-nucleotidase activity in chicken embryos [Martin and Moses, 1995], enhancement of ornithine decarboxylase (ODC) activity in L929 murine cells [Farrell et al., 1998], and alteration of neurotransmitter synthesis (dopamine) in PC12 cells [Opler et al., 1997]. However, doubts exist about these studies, as the intensities of MF applied were very low (in the range of 0.0001–0.05 mT) and their corresponding effects were disputable.

It is believed that all biological effects require the involvement of one or more signaling pathways. Thus, it is conceivable that certain signaling pathways should be involved in the effects of MF on biological systems. Therefore, it will be valuable to explore the interference effects of noise MF on ELF MF-induced bioeffects by employing a cellular signaling element as a marker. Gap junction intercellular communication (GJIC), one component of the cell signaling system, is very important for cell homeostasis. The loss of GJIC is a key event in the conversion of normal cells into neoplastic ones [Ruch, 1994]. Thus, disruption of GJIC has been recognized as one of the non-genotoxic mechanisms of carcinogenesis. To study the possible health hazards of ELF MF, our laboratory had evaluated the effects of 50 Hz MF on GJIC using GJIC as a biomarker [Li et al., 1999; Hu et al., 2001, 2002; Zeng et al., 2003]. It was shown that 50 Hz MF-inhibited GJIC at a magnetic flux density threshold of 0.4 mT. In addition, although 0.2 mT MF did not have significant effect on GJIC, it enhanced the suppression effect of TPA on GJIC. We also found that the hyperphosphorylation and internalization of Connexin 43, a major component of gap junctions, contributed to ELF MF-induced GJIC inhibition. Thus, by using GJIC suppression as a testing endpoint, we investigated the interaction of an incoherent noise MF with a coherent MF to further evaluate Litovitz's hypothesis and found that the superposition of an incoherent noise MF could block ELF MF-induced GJIC inhibition.

MATERIALS AND METHODS

Exposure Systems

The sinusoidal MF exposure system used in this study is similar to the one reported previously [Li et al., 1999; Zeng et al., 2003], which consists of three groups of square copper coils (36 × 36 cm) placed inside a CO_2 incubator (Model 3164, Forma, Marietta, GA, USA). The upper, middle, and lower coils are connected in series and spaced 8 cm apart from each other. The coils are placed in an iron metal container with many ventilation holes to shield cells from stray MF. A 50 Hz sinusoidal MF was generated by feeding a line current to the coils, which is controlled by a power regulator. Magnetic flux densities were measured using a Model EFA-3 meter (Narda Safety Test Solutions, GmbH, Pfullingen, Germany). When energized, a very uniform MF can be generated in the center ($10 \times 10 \times 10\,cm^3$) of the coils, where cell culture dishes were placed. The flux density could be regulated from 0 to 0.8 mT.

To generate a noise MF, the above system is double-wrapped with two lines of copper wires. One of the double wires was provided with a 30–90 Hz white noise signal (generated through a software designed by Dr. Penafiel, Catholic University of America) after magnifying by a power amplifier (CROWN 1400CSL, Crown International Inc., Elkhart, IN, USA). The other wire was fed with sinusoidal 50 Hz current during combined MF exposure (MF + noise MF). The amplitude of the noise MF was adjusted to produce a MF strength (rms) equal to the sinusoidal fields used in the experiments.

The electromagnetic waveforms of different MF are monitored by oscillograph (Fig. 1). The AC background field in the incubator is 1–2 μT, and the total static MF is 18.5 μT with a 14.1 μT horizontal and 12.0 μT vertical components.

The sham exposure system is the same as exposure system but without current or signal input. Temperature in all the exposure systems was monitored with a thermocouple probe (sensitivity ±0.1 °C) near the

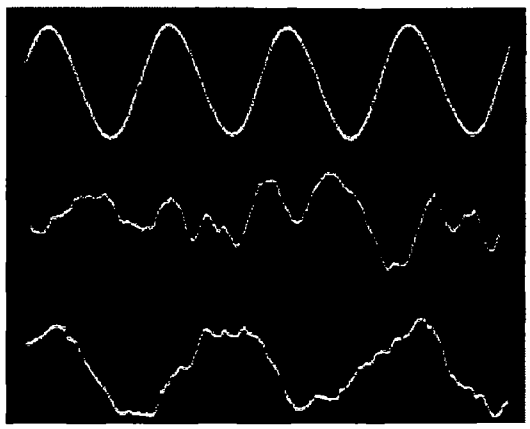

Fig. 1. The waveforms of MF and noise. The intensity of MF to noise MF is equal. **A:** Waveform of sinusoidal MF. **B:** Waveform of noise MF. **C:** Combined waveform of ELF MF and noise MF.

dishes and kept at 37 ± 0.2 °C throughout the entire experiment. The exposure system was turned on at least 2 h before experiment to achieve system stability and thermal equilibrium. After exposure, dishes were removed from the container while the system was still on.

Cell Culture

Mouse fibroblast cell line NIH3T3 (Institute of Biochemistry and Cell Biology, Shanghai institutes for Biological Sciences, Chinese Academy of Sciences) was cultured in RPMI 1640 medium (Invitrogen Corp., CA, USA) supplemented with 15% fetal calf serum (Hyclone, Logan, UT), L-glutamine (2 μM), HEPES (25 mM), penicillin G (50 IU/ml), streptomycin sulfate (50 μg/ml), and kanamycin sulfate (50 U/ml) at 37 °C in 5% humidified CO_2. Cells were seeded onto confocal special dishes (30 mm diameter, World Precision Instruments Ltd., Aston, UK) at a final density of 2×10^4 cells/cm^2. The culture dishes are custom-designed for a confocal microscope with a 0.2 mm thick glass bottom. After 48 h, the dishes were transferred to the exposure system.

Cell Exposure

The experiments were double-blindly conducted in eight treatment groups with four dishes in each group: (a) sham exposure, (b) 0.4 mT MF alone, (c) 0.4 mT MF combined with 0.4 mT noise MF, (d) 0.4 mT noise MF alone, (e) 0.2 mT MF plus TPA (5 ng /ml), (f) 0.2 mT MF plus TPA (5 ng /ml) and noise MF, (g) 0.2 mT noise MF alone, (h) 5 ng /ml TPA alone. The duration of MF and/or noise MF exposure was 24 h, and TPA was added to the corresponding dishes at the 23rd h. All the experiments were repeated at least three times. The dishes were placed coaxially with the center line in the central area of the coils.

Measurement of GJIC

Fluorescence recovery after photobleaching (FRAP) analysis was performed by employing a laser scanning confocal microscope (Leica TCS-SP, Heidelburg, Germany), mounted on a Leica inverted research biological microscope with a 20× objective. The fluorescence of 5, 6-carboxyfluorescein diacetate (5, 6-CFDA, Sigma-Aldrich, St. Louis, MO, USA) was excited at 488 nm by an argon ion laser, and captured through a grating at 530/30 nm.

After treatments, cells were rinsed twice with 1 ml of Hank's buffer and incubated with 5, 6-CFDA (10 ng/ml in DMEM) at 37 °C for 15 min. The cells were then rinsed five times with 2 ml of Hank's buffer to remove extracellular dye. Finally, 200 μl Hank's buffer was added to the dish and the cells were subjected to FRAP analysis. Clusters of five to six cells were selected under the microscope with the 20× objective lens. A cell contacted by four to five neighboring cells within a cluster was photobleached to 50–60% of the original fluorescence intensity using an argon laser beam. The bleached cell was then monitored for transfer of fluorescent dye from neighboring cells and examined for recovery of fluorescence by scanning at intervals of 60 s for a total period of 11 min. The data on the maximum intensity of recovered fluorescence (Ir) at the 10th min was collected as the functional index of GJIC. At least three such clusters were selected from each dish. Non-photobleached cells were monitored as controls for spontaneous photobleaching. The analyzed fluorescence recovery index is expressed as: $R = (\mathrm{Ir} - \mathrm{Ib})/(\mathrm{Ip} - \mathrm{Ib}) \times 100\%$, where Ib is the intensity of the photobleached fluorescence and Ip is the intensity of pre-bleached fluorescence. Fluorescence recovery was normalized with unbleached control to compensate fluorescence lost during the experiment.

Statistical analysis was performed by using the Variance (one-way ANOVA) programs available in SPSS software. A probability level (P-value) of less than .05 was considered to be significant. Data were presented as the mean ± standard deviation.

RESULTS

Noise MF Does Not Affect GJIC in NIH3T3 Cells

NIH3T3 cells were exposed to noise MF at intensities of 0, 0.2, or 0.4 mT for 24 h, and then subjected to FRAP analysis. As shown in Tables 1 and 2, there were no statistical differences in fluorescence

TABLE 1. The Rate of Fluorescence Recovery After Photobleaching NIH3T3 Cells Exposed to 0.4 mT MF, With or Without Noise MF

Treatment	*n*	Fluorescence recovery index (%) (mean ± SD)
Control	84	46.36 ± 20.68
0.4 mT noise MF	54	46.74 ± 21.14
0.4 mT MF	92	30.85 ± 14.70*
MF + noise MF (0.4 mT: 0.4 mT)	58	49.58 ± 19.38

n, number of tested cells, pooled from three independent experiments of four dishes each.
*$P < .05$ versus control group.

recovery index (*R*-value) among sham exposure, 0.2 mT noise MF, and 0.4 mT noise MF groups, suggesting that a spatially coherent but temporally incoherent noise MF did not affect GJIC function.

Noise MF Alleviates the GJIC Inhibition Induced by Sinusoidal MF Exposure

To evaluate the effect of noise MF on sinusoidal MF-induced GJIC inhibition, we exposed NIH3T3 cells to 0.4 mT MF with and without noise MF for 24 h, and then analyzed the GJIC functions. Table 1 showed that exposure to 0.4 mT 50 Hz sinusoidal MF yielded an *R*-value of 30.85 ± 14.70%, and the *R*-value for sham exposed cells was 46.36 ± 20.68%, demonstrating exposure to 0.4 mT MF-inhibited GJIC function by about 33%. When a noise MF with the same intensity was superimposed onto MF, the combined exposed cells had an *R*-value of 49.58 ± 19.38%, which was not statistically different from sham exposed and noise MF exposed cells (Table 1, $P > .05$). The data indicated superposing noise MF onto coherent sinusoidal MF mitigated the suppression of GJIC induced by the latter.

TABLE 2. The Rate of Fluorescence Recovery After Photobleaching NIH3T3 Cells Exposed to 0.2 mT MF, With or Without Noise MF and TPA

Treatment	*n*	Fluorescence recovery index (%) (mean ± SD)
Control	84	46.36 ± 20.38
0.2 mT noise MF	46	46.01 ± 18.64
0.2 mT MF	42	45.03 ± 19.85
5 ng/ml TPA	56	35.82 ± 17.18*
0.2 mT MF + 5 ng/ml TPA	58	24.90 ± 13.50* **
0.2 mT MF + 5 ng/ml TPA + 0.2 mT noise MF	46	32.51 ± 18.37*

n, number of tested cells, pooled as in Table 1.
*$P < .05$ versus control group.
**$P < .05$ versus TPA group.

Noise MF Interferes With the Synergistic Effect of MF on GJIC Inhibition by TPA

Although coherent MF at 0.2 mT did not inhibit GJIC in NIH3T3 cells, it synergistically enhanced TPA-induced GJIC inhibition (Table 2, *R*: 24.90 ± 13.50% vs. 35.82 ± 17.18%, $P < .05$). Interestingly, when a noise MF was superimposed onto the sinusoidal MF, the *R*-value returned to 32.51 ± 18.37%, which was almost the same as TPA-treated group. This result suggested that noise MF abolished the synergistic effect of coherent MF on GJIC inhibition by TPA in NIH3T3 cells.

DISCUSSION

Although noise MF was reported to interfere with the biological effects induced by ELF MF, its role is under debate due to the low intensities applied and the so-called uncertainties of detecting endpoints. GJIC has been established as a target of ELF MF action in cells, we therefore selected this marker to observe the possible interaction between noise MF and ELF MF at higher intensities (0.2 and 0.4 mT). The results clearly demonstrated that superposing an electromagnetic noise on ELF MF abolished MF-induced GJIC suppression, thus providing strong experimental evidence to support Litovitz's temporal-and-spatial coherence hypothesis regarding the biophysical mechanism of EMF-induced biological effects.

The bioeffects of EMF are hypothesized to be initiated at cell surface, propagated and amplified by signal transduction pathways, and ultimately lead to changes of cell behavior [Nie and Henderson, 2003]. Cellular membrane, as the boundary and the major signal sensing organelle of a cell, might be one of the primary interacting sites of ELF MF to accomplish the transition from a physical signal to a biological one, which eventually results in various biological responses. It was suggested that a significant number of receptors must be simultaneously and coherently activated to achieve this purpose [Litovitz et al., 1994; Lin and Goodman, 1995]. Indeed, our laboratory has shown that 50 Hz MF at 0.4 mT induces epidermal growth factor (EGF) and tumor necrosis factor (TNF) receptor clustering on the cell surface [Sun et al., 2003], providing a direct experimental proof for this hypothesis.

Receptor activation on the membrane will naturally lead to downstream intracellular signal transduction. Although there are quite a few reports demonstrating the activation of certain components in signal transduction pathways, including ERK [Nie and Henderson, 2003], SAPK/JNK [Sun et al., 2001],

p38 MAPK [Sun et al., 2002], Lyn [Uckun et al., 1995], Syk [Dibirdik et al., 1998], BTK [Kristupaitis et al., 1998], PLC-gamma2 [Uckun et al., 1995; Dibirdik et al., 1998; Kristupaitis et al., 1998], and protein kinase C (PKC) [Dibirdik et al., 1998; Kristupaitis et al., 1998; Sun et al., 2001, Uckun et al., 1995], gaps still exist in term of the linkage between the membrane receptors and intracellular signal transduction pathways. Available data suggested PKC activation may result in GJIC disruption. Our laboratory has found that hyperphosphorylation of connexin 43 is mediated by PKC [Hu et al., 2001; Chiang et al., 2002]. Therefore, it might be reasonable to conclude that ELF MF exposure induces the activation of membrane receptors, which leads to the activation of PKC through certain signal transduction pathway. Activated PKC subsequently hyperphosphorylates connexin 43 in the gap junctions of cells, which in turn causes internalization of connexin 43 and disruption of GJIC, that is GJIC inhibition.

As a membrane-located signaling channel, GJIC plays an important role in the regulation of cell growth, differentiation, and proliferation by exchanging small signal molecules between adjacent cells through gap junctions to fulfill inter-cell cooperation in response to various signals. GJIC inhibition will block the signal communications among neighboring cells, thus breaking this cooperation and causing aberrant cell behavior. It has been recognized that inhibition of GJIC is one of the many cellular changes seen in cells after exposure to cancer-promoting agents [Klaunig, 1991], and the function of GJIC was commonly used as an index for evaluation of suspected cancer promoters. Therefore, ELF MF-induced GJIC inhibition might indicate the possible health hazard of ELF MF and underline a possible molecular mechanism. The results reported here justify noise MF as a potential approach to prevent ELF MF-induced hazardous effects.

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