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Effects of 835-MHz Radiation on the Intracellular Calcium, Reactive Oxygen Species, and F-actin Polymerization in Rat-2 Fibroblasts

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We investigated the effects of 835-MHz electromagnetic field (EMF), one of the most popular communication frequency band in Korean code-division multiple-access (CDMA) mobile phone system, on cellular signal transduction. For this, we examined the change of intracellular calcium ($[Ca^{2+}]_i$), reactive oxygen species (ROS) and F-actin polymerization after exposure to 835-MHz EMF followed by the treatment of agonists in Rat-2 fibroblast cells. Culture cells were pretreated with serum-free medium and concomitantly exposed to 835-MHz at specific absorption rate (SAR) of 4.0 W/kg for 24 hr in a specialized designed apparatus based on Transverse Electro Magnetics (TEM) wave theory. Intracellular Ca^{2+} responses to lysophosphatidic acid (LPA) and epidermal growth factor (EGF) in Rat-2 fibroblast after exposure to 835-MHz EMF were shown to be similar pattern as observed in normal cultured cells. However, the LPA-induced calcium spiking was slightly delayed to 7 sec and sustained thereafter to a little higher ground level under 835-MHz EMF radiation compared to unexposed cells. ROS production level by LPA in the exposed cells was not different from that in control. Furthermore, LPA induced the production of stress fibers with no significant difference in the exposed and unexposed cells. These results suggest that mobile phone radiation (835-MHz, SAR 4.0 W/kg) may not be directly related to signal transduction in Rat-2 fibroblasts except the slight effect of calcium spiking in LPA-induced cells but remain to be further elucidated for possible indirect intervention.

Key Words: Electromagnetic field; Specific absorption rate; Reactive oxygen species; Intracellular calcium concentration; Stress-Fiber formation

INTRODUCTION

The widespread uses of radiofrequency and microwave devices (300 kHz~300 GHz) in household electrical appliances, telecommunications and navigational equipments have increased the awareness of public health risk. Recently, the number of people subscribing to use portable hand-held mobile phone is exponentially increasing but little has been done about the epidemiological and biological investigations in mobile phone communication bands. Several contradictory evidences regarding the potential toxicity of EMF have been documented, leaving to be further studied in detail.

There are some studies of bio-safety at frequencies used in mobile phone communication. Malyapa and his colleages measured DNA damage of cultured cells U87MG and C3H 10T½ after exposure to 835.62- (frequency-division multiple-access, FDMA) and 847.74-MHz (code-division multiple-access, CDMA) at an SAR of 0.6 W/kg, but found no effect (Malyapa et al., 1997). In addition, neither 835.62-MHz FDMA nor 847.74-MHz CDMA had any significant effect at the same SAR indicated above on proto-oncogene expression during cellular proliferation (Goswami et al., 1999) as well as neoplastic transformation frequency (Roti Roti et al., 2001). Recently, Zeni group reported no correlation of genotoxic effects in human leucocytes exposed *in vitro* to

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900 MHz, a European mobile communication band (Zeni et al., 2003).

However, there are positive reports demonstrating the causative induction of DNA damage in rat brain cells after a 2-h *in vivo* exposure to 2450-MHz microwave radiation at 0.6 or 1.2 W/kg (Lai and Singh, 1996). Furthermore, exposure to a continuous radiation of 2450-MHz for 48 hr has been found to interact with Fas pathway between the receptor and caspase-3 activation and affect membrane proteins (Peinnequin et al., 2000). A 1.8-GHz wireless at 0.3 W/kg increased the permeability of the blood-brain barrier to sucrose (Schirmacher et al., 2000).

The discordance between previous studies may be due to different experimental conditions and methods exaggerating or overestimating small actual differences. According to our previous studies, 835 MHz EMF did not increase the mutation frequency or *in vitro* DNA degradation (Chang et al., 2005) but induced severly *in vivo* DNA damage after exposure combined with potent mutagen (Choi et al., 2004). EMFs may act as an extracellular signal affecting cell metabolism, making it likely to influence one or more steps in cellular signal transduction pathway. Exposure to extremely low-frequency magnetic fields in Jurkat E6-1 cells decreased significantly cytosolic calcium (McCleary et al., 2002). Contrast to this, the application of a 1 or 10 Hz electric field to human hepatoma cells induced a fourfold increase in in tracellular calcium concentration (Cho et al., 1999). In addition, the 500-V/m electrical field increased intracellular ROS, but not $[Ca^{2+}]_i$ in cardiomyocyte differentiation of embryonic stem cells (Sauer et al., 1999) and induced production of ROS paralleled with increased transient $[Ca^{2+}]_i$ in cancer spheroids (Watenberg et al., 1997).

To our knowledge, there are few report regarding the direct effect of mobile phone radiation on the change of signaling molecule such $[Ca^{2+}]_i$, ROS and F-actin polymerization. In the present study, we chose Rat-2 fibroblast cell, which is a well-established system for LPA and EGF-induced signaling transduction system (Lee et al., 1998, 2000; Shin et al., 1999).

MATERIALS AND METHODS

1. Exposure facility setup

Culture cells were exposed to an 835-MHz EMF in a specially designed apparatus based on the TEM wave theory, in which Wave Exposure V10 (Model CTI835, Patent #0311892, Republic of Korea) irradiated cultivating cells in a CO_2 gas incubator with an 835-MHz CDMA wave (Chang et al., 2005; Choi et al., 2004). The characteristics of Wave Exposure V10 are summarized in Table 1. The TEM cell was positioned horizontally to allow uniform radiation to all plates and was placed inside CO_2 incubator with constant temperature throughout the radiation. There are two selectable modes-for blood, conductivity $\delta = 1.19$, relative dielectric constant $\varepsilon_r = 74$ and for skin, $\delta = 0.92$, $\varepsilon_r = 57$-by a rotary switch on the front panel. The power level at each mode is controlled by an SAR-level switch on the control panel: each mode has four SAR levels (1.6, 2.0, 3.0 and 4.0 W/kg). Two petri-dishes, 10 cm in diameter and 2 cm high, can be layered in the TEM cell apparatus by mounting them in a jig fixture (Fig. 1). The space in the TEM cell was maintained under controlled conditions similar to those in an incubator; i.e. an atmosphere of 5% CO_2 and 95% air and a temperature of 37℃. To test the increase of temperature during the radiation of 835-MHz at 4.0 W/kg, preliminary measurements were carried out with digital thermometer every one hour. The temperature change con-

Table 1. Characteristics of wave exposure V10

Component	Specific Description
Manufacturer	Dept. of IT Eng. Soonchunhyang Univ. Rep. of Korea
Cell model	CTI835
Patent	No. 0311892, Rep. of Korea
Frequency	CDMA 835-MHz
RF power	1.7, 2.1, 3.2, 4.2 W for blood mode 2.2, 2.7, 4.2, 5.4 W for skin mode
Selectable SAR	1.6, 2.0, 3.0, 4.0 W/kg
Max. size of Petri-dish	10 cm (diameter) × 2 cm (height)
Main power	220V AC, 6A, 60Hz
CO_2 incubator	
Main control	VFD display, micro processor controller
Temperature range	+5 to +60℃
CO_2 range	0 to 20%, ±0.1% at 5% Digital PID controller, IR sensor
Humidification	up to 96% RH
Programmability	10 steps per 10 memories
Capacity	130 liter
Water jacket	65 liter
Dimension	445 × 470 × 620 mm, inside 582 × 580 × 015 mm, outside

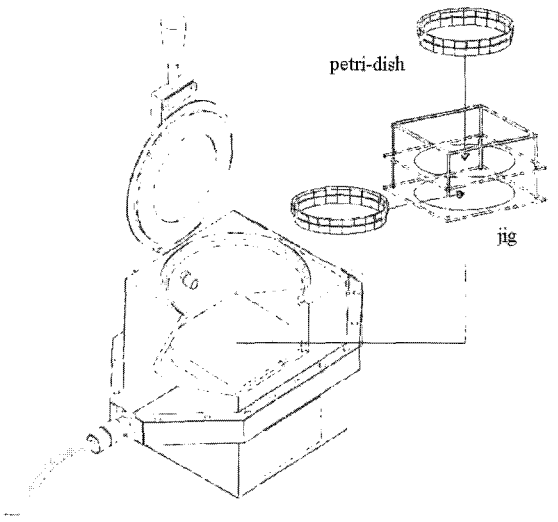

Fig. 1. Sketch of the TEM cell. Cultivated cells on two-Petri-dishes stacked in the jig fixture are exposed to uniform TEM-mode EMF in the apparatus.

stantly resulted in 36.9±0.1℃ throughout the 24-hr radiation in both exposed and unexposed groups (data not shown).

2. Pretreatment of culture cells prior to the measurement of intracellular signaling molecules

Rat-2 fibroblasts (ATCC CCL 92) were maintained at 37℃ in Dulbecco's modified Eagle's medium (Bibco-BRL, Gaitherburg, MD) supplemented with 25 mM HEPES (pH 7.4), 10% (v/v) fetal bovine serum, 100 units/ml penicillin and 100 μg/ml streptomycin in an atmosphere of 95% air and 5% CO_2 (culture medium). For experiments, cells were cultured on round coverslips in 12-well plates and then serum starved for 24 hr with Dulbecco's modified Eagle's medium supplemented with 5 μg/ml apotransferrin, 1 mg/ml bovine serum albumin, 25 mM HEPES (pH 7.4), 2 mM glutamine, 100 units/ml penicillin and 100 μg/ml streptomycin (serum-free medium).

3. Analysis of $[Ca^{2+}]_i$ in Rat-2 fibroblast after exposure to 835-MHz EMF

The changes in $[Ca^{2+}]_i$ were monitored using a laser scanning confocal microscope (Carl Zeiss LSM 410) as described previously (Lee et al., 1998). Briefly, cells were grown on round coverslips in multiple-well culture plates, serum starved for 24 hr and incubated with 3 μM fluo-4-AM (Molecular Probes, Eugene, OR) in serum-free medium for 40 min. Each round coverslip containing stained cells was mounted on a perfusion chamber (Model MPS-2000, Patent #0272302, Republic of Korea) equipped with a laser scanning confocal microscope. Subsequently, each sample was scanned every second with a 488-nm excitation Ar laser and a 515-nm long-pass emission filter. All images obtained from the scanning (about 200 images) were processed to analyze changes in $[Ca^{2+}]_i$ at a single-cell level.

4. Analysis of intracellular ROS production in Rat-2 fibroblast after exposure to 835-MHz EMF

The amount of intracellular ROS was measured as described previously (Koo et al., 1999). Briefly, serum-starved cells on round cover-slips were stabilized in serum-free medium without phenol red for at least 30 min and then stimulated with 5 μg/ml LPA, 100 μM arachidonic acid (AA) or 50 ng/ml EGF for various durations. For the last 5 min of stimulation, 5 μM H_2DCFDA (a cell-permeable ROS-sensitive fluorophore, purchased from Molecular Probes) was added to measure intracellular ROS. The cells were then immediately observed using a laser scanning confocal microscope. The samples were excited by a 488-nm Ar laser and their fluorescence filtered by a 515-nm long-pass filter. About 30 cells were randomly selected from three separate experiments and the H_2DCFDA fluorescence intensities of treated cells were compared with those of unstimulated control cells.

5. Observation of F-actin polymerization in Rat-2 fibroblast after exposure to 835-MHz EMF

F-actin was observed as described previously (Jung et al., 1997). Briefly, cells were grown on round coverslips in a 12-well culture plate and serum starved for 24 hr. After being stabilized in fresh serum-free medium for 1 hr the cells were incubated with 5 μg/ml LPA for 30 min and fixed with 3.7% (v/v) formaldehyde in Dulbecco's phosphate-buffered saline (DPBS) (1.2 mM KH_2PO_4, 8.1 mM Na_2HPO_4, 0.9 mM $CaCl_2$, 2.7 mM KCl, 0.5 mM $MgCl_2$ and 138 mM NaCl, pH 7.5) for 30 min. The cells were then permeabilized with 0.2% (v/v) Triton X-100 in DPBS for 15 min and stained with 0.5 units/ml rhodamine-phalloidin (Molecular Probes, Eugene, OR) for 30 min at room temperature. Stained cells were mounted on slide glasses with gelvatol and observed with a laser scanning confocal microscope. Gelvatol was prepared by mixing 100 ml of 0.23% polyvinyl alcohol in DPBS with 50 ml glycerol. Samples were exci-

ted by a 543-nm HeNe laser and their fluorescence filtered by a 560-nm long-pass filter. Three-dimensional images were constructed from 5~10 serial images (each 1 μm thick) obtained by automatic optical sectioning.

RESULTS

1. Effects of 835-MHz EMF on the increase in $[Ca^{2+}]_i$ induced by LPA and EGF

It has been reported that $[Ca^{2+}]_i$ is increased by LPA (Perkins et al., 1994). Hence we investigated whether $[Ca^{2+}]_i$

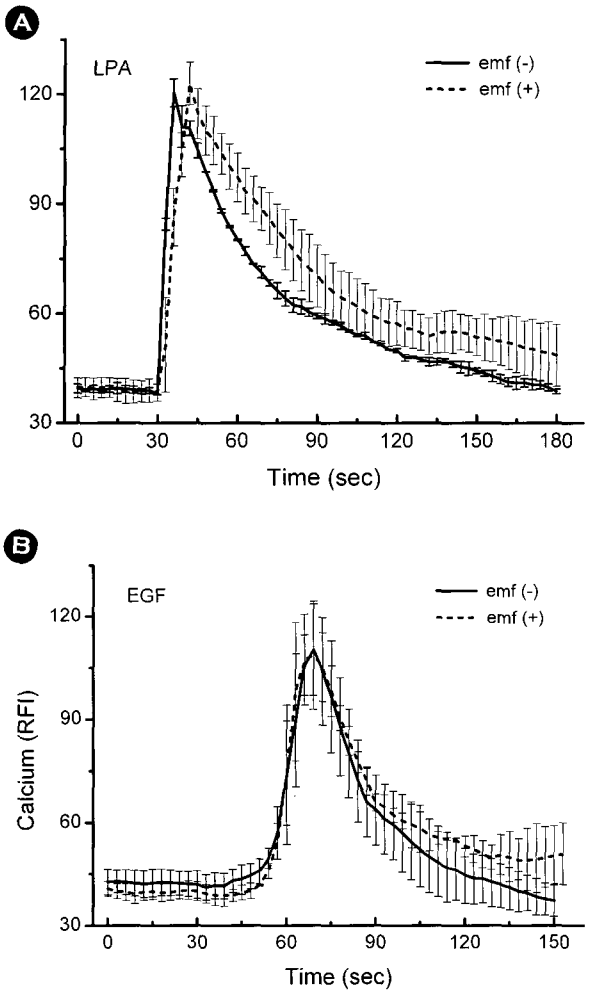

Fig. 2. Comparison of changes in $[Ca^{2+}]_i$ induced by LPA and EGF in 835-MHz-exposed and unexposed Rat-2 fibroblasts. Serum-starved (solid line) and 835-MHz EMF-exposed (dotted line) Rat-2 cells were loaded with 3 μM fluo-4-AM for 40 min and treated with 1 μg/ml LPA **(A)** or 50 ng/ml EGF **(B)** at the indicated time. $[Ca^{2+}]_i$ was then monitored using a confocal microscope as described in the Materials and Methods. Results are expressed as the relative fluorescence intensity (RFI). Each trace represents the mean data from at least 30 cells from at least three separate experiments.

was also essentially affected by mobile phone radiation. The intracellular concentration of Ca^{2+} was monitored using a confocal microscope in 835-MHz-exposed cells in the presence of LPA and EGF (Fig. 2). LPA and EGF induced a rapid and transient increase in $[Ca^{2+}]_i$, showing peaks at 0.5 and 1 min, respectively. Thereafter, the increased Ca^{2+} level was rapidly decreased over 3 min. We also compared the changes of $[Ca^{2+}]_i$ induced by LPA and EGF in 835-MHz EMF-exposed cells with those in unexposed control. The Ca^{2+} response to EGF in 835-MHz EMF-exposed cells was similarly observed as that in normal cells. When Rat-2 fibroblast cells were induced by LPA after exposure to 835-MHz EMF, the peaking time of $[Ca^{2+}]_i$ was about 7 sec delayed and the level of decline after peaking was observed slightly higher than the control. However, the overall change of intracellular calcium seems that intracellular Ca^{2+} levels of Rat-2 fibroblasts are not significantly affected by an 835-MHz EMF.

2. Production of intracellular ROS in 835-MHz expo- sed cells by LPA, AA and EGF

In order to investigate the possible regulation of intracellular ROS by 835-MHz EMF, we initially determined the changes in intracellular ROS in response to LPA in Rat-2 cells using H_2DCFDA, which is widely used to determine the level of intracellular ROS. LPA induced a significant increase in intracellular ROS above the control level (Fig. 3) and this was blocked by incubation with ROS scavengers, N-acetyl cysteine and *A. niger* catalase (data not shown). Cells on the cover-slip were exposed to an 835-MHz EMF at 4.0 W/kg for 24 hr. ROS production by LPA in 835-MHz EMF-exposed cells was not different from that in unexposed cells. The cells were also incubated with AA and EGF for various durations and the changes in intracellular ROS were examined. AA and EGF induced a rapid increase in intracellular ROS (Fig. 3). The increase was maximal at 5 min and, thereafter, the level was slowly decreased over 30 min (data not shown). These results indicate that an 835-MHz EMF has no effect on LPA, AA or EGF-induced intracellular ROS production in Rat-2 fibroblasts.

3. Effects of 835-MHz EMF on stress-fiber formation by LPA

Since there was a report that ROS production by LPA

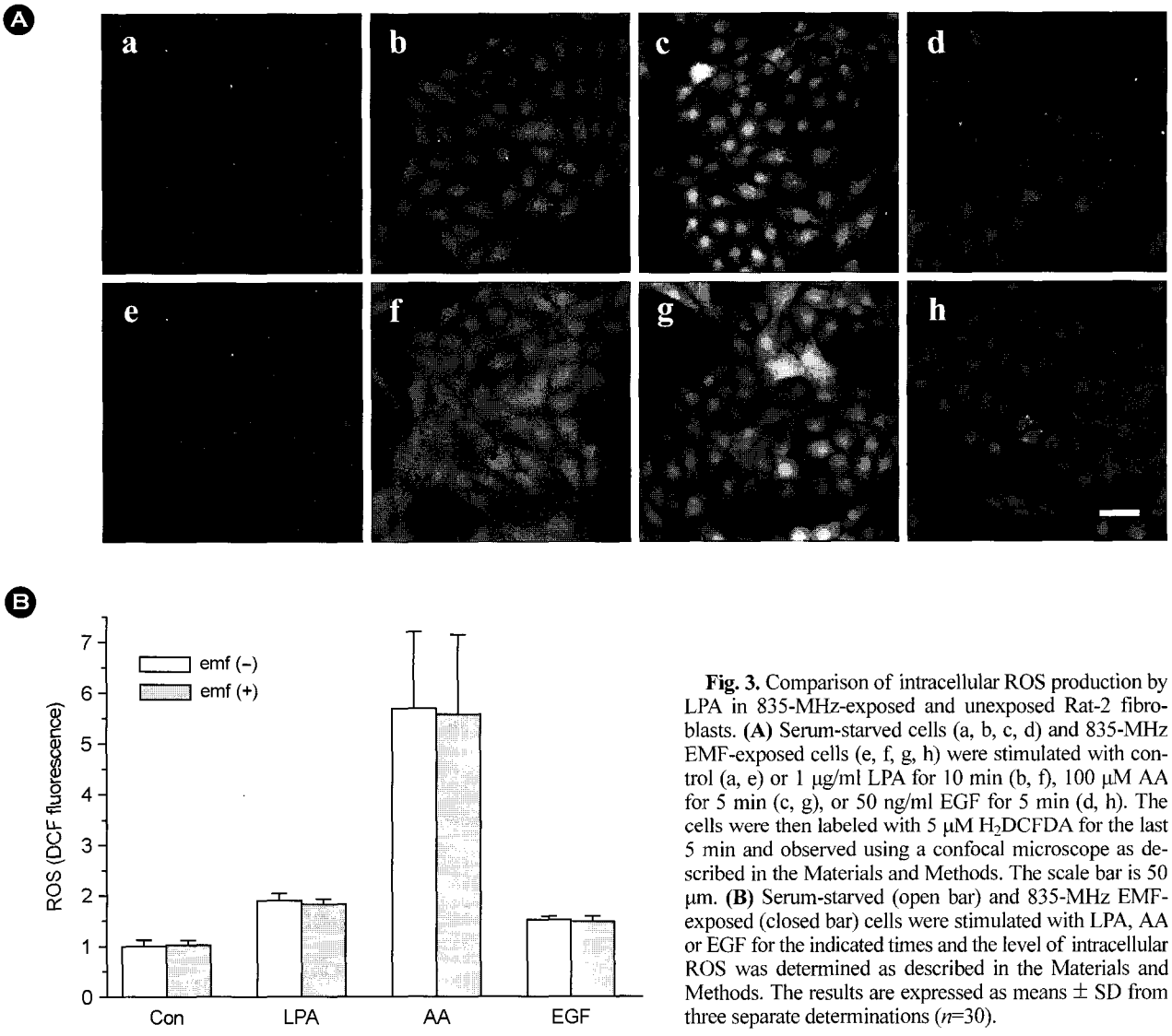

Fig. 3. Comparison of intracellular ROS production by LPA in 835-MHz-exposed and unexposed Rat-2 fibroblasts. **(A)** Serum-starved cells (a, b, c, d) and 835-MHz EMF-exposed cells (e, f, g, h) were stimulated with control (a, e) or 1 μg/ml LPA for 10 min (b, f), 100 μM AA for 5 min (c, g), or 50 ng/ml EGF for 5 min (d, h). The cells were then labeled with 5 μM H_2DCFDA for the last 5 min and observed using a confocal microscope as described in the Materials and Methods. The scale bar is 50 μm. **(B)** Serum-starved (open bar) and 835-MHz EMF-exposed (closed bar) cells were stimulated with LPA, AA or EGF for the indicated times and the level of intracellular ROS was determined as described in the Materials and Methods. The results are expressed as means ± SD from three separate determinations (n=30).

resulted in stress-fiber formation (Shin et al., 1999), we investigated the possible interference of ROS downstream signaling by a mobile phone frequency radiation. Rat-2 fibroblast cells were exposed to 835-MHz EMF for 24 hr and subsequently, stress-fiber formation as an outcome phenotype of accumulated F-actin polymerization was observed and compared with unexposed control. As expected, LPA induced the production of microscopic stress-fibers to the same extent in both exposed and unexposed cells (Fig. 4). Thus, it is likely that 835-MHz radiation has no effects on stress-fiber formation induced by LPA in Rat-2 fibroblasts.

DISCUSSION

The epidemiological association studies of extremely low-frequency (1~300 kHz) electromagnetic fields from power lines have been focused on the public health risk, which is in turn contentiously issued in the scientific community. This issue largely dates from 1979 when Wertheimer and Leeper (1979) reported an epidemiological link between power-line magnetic fields and childhood leukemia in Denver and Colorado (Wertheimer, 1980). Since then they have reported a statistically significant correlation between magnetic-field exposure and cancer (Wertheimer, 1979; Wertheimer and Leeper, 1979, 1982). More recently, the widespread use of mobile communication devices in the relatively shorter range of 0.8~2 GHz has stimulated many researchers to describe the biological effects from the radio-frequency (RF) electromagnetic fields.

As an initial attempt, we challenged to examine whether

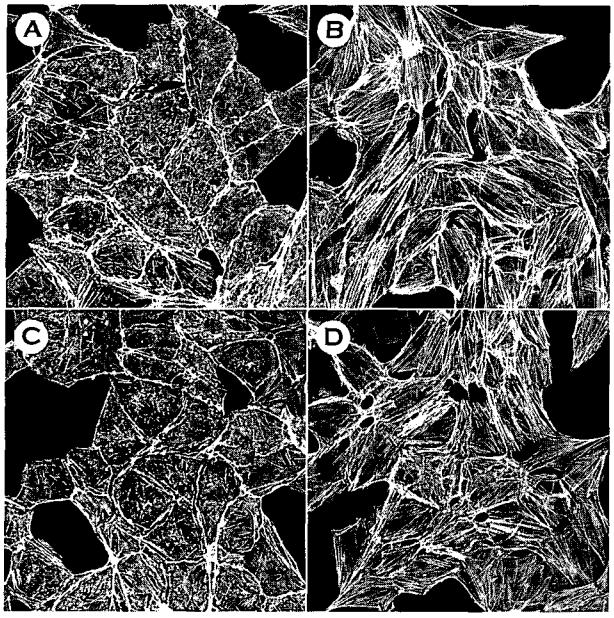

Fig. 4. Comparison of 835-MHz EMF effect on stress-fiber formation by LPA. Serum-starved **(A, B)** and 835-MHz EMF-exposed **(C, D)** cells were stimulated with control **(A, C)** or 1 μg/ml LPA **(B, D)** for 30 min. The cells were then stained with 0.5 units/ml rhodamine-phalloidin and observed using a laser scanning confocal microscope as described in the Materials and Methods. Representative images in each group are shown. The scale bar is 30 μm.

RF-EMF radiation generated by mobile phone perturbs the signal transduction pathways in culture cell system. Self-designed RF-EMF generator was set at 835-MHz, one of the most popular communication frequency CDMA bands in Korea. Here, we applied SAR at 4.0 W/kg, which the value is beyond the prescribed safe limit as 1.6 W/kg for population exposure in America by ANSI/IEEE and 2.0 W/kg in European Union by ICNIRP guidelines (Gandhi and Kang, 2002).

First of all, we measured temperature change throughout the radiation to evaluate the possible thermal effect, in which turned out to be constant in either presence or absence of 835-MHz EMF radiation. Thus, we can exclude the possibility that the change of temperature is attributable to biological effects as generally accepted in EMF radiation (Zmsylony, 2000). In the present study, we conducted to monitor the real-time changes of $[Ca^{2+}]_i$, ROS and F-actin by agonists in Rat-2 fibroblast after the presence or absence of RF-EMF radiation. As shown in Fig. 2, the exposure to 835-MHz radiation by itself did not affect the basal concentration of $[Ca^{2+}]_i$. Furthermore, no significant difference of the changes of $[Ca^{2+}]_i$ was observed by EGF in 835-MHz-exposed and unexposed cells. These results are consistent with the previous literature, in which 915-MHz radiation at SAR 2 W/kg is not associated with any changes in calcium levels or calcium signaling in Jurkat cells (Cranfield et al., 2001). However, when LPA was pretreated to Rat-2 fibroblasts after exposure to 835-MHz EMF, peaking time was 7 sec delayed and, thereafter, the decline level of $[Ca^{2+}]_i$ was slightly sustained higher than the unexposed control (Fig. 2A). This suggests that 835-MHz EMF affect calcium influx and/or the following desensitization step. Since the change of intracellular calcium concentration is involved with differential routes of Ca^{2+} influx (Miyakawa and Kojima, 1998; Carpenter, 1999), there remains to be elucidated LPA-induced calcium signaling in details.

We and other investigators (Sauer et al., 2001) have reported that ROS function as second messengers in the control of cell proliferation and differentiation in reference to the intracellular ROS levels. When 835-MHz radiation was applied to Rat-2 fibroblasts, we could not find any difference of intracellular ROS production mediated by LPA (Fig. 3). There are reports that F-actin can be used in cell proliferation, gene expression, and various signal transduction pathways and that LPA activates stress-fiber formation in fibroblasts (Kawamura et al., 2003; Kim et al., 2002). In general, the application to external alternating current electic fields is known to induce formation of cell protrusion such as actin polymerization (Popov and Margolis, 1988) and change of intracellular calcium ion concentration (Graziana et al., 1990). However, exposure to 835-MHz EMF for 24 hr had no effect on either the intensity or pattern of stress-fiber formation activated by LPA in Rat-2 cells (Fig. 4). We confirmed these negative results by 835-MHz EMF in other cell line, C6 glioma cells (data not given). From our preliminary experiments, it is not clear whether the measurements of Ca^{2+}, ROS and F-actin are sufficiently specific and sensitive to detect the influence of mobile phone radiation. Thus, we cannot exclude the possible indirect intervention of signal transduction pathways. Recently the mutagenic and co-mutagenic effects of 835-MHz radiation were examined at a SAR 4.0 W/kg, which resulted in no detectable increase in mutagenicity except for *Salmonella typhimuirum* TA102 (Chang et al., 2005). However, the exposure to CDMA-mobile phone radiation at 835-MHz frequency potentiates dramatically DNA strand breaks of mouse thymic lymphoma L5178Y $Tk^{+/-}$ cells at SAR 4.0 W/kg for 1 or 2

days (Choi et al., 2004).

In conclusion, our results suggest that exposure to 835-MHz radiation at an SAR of 4.0 W/kg likely to be encountered in Korean mobile phone CDMA-frequency band did not influence the distinct changes in $[Ca^{2+}]_i$ induced by EGF and LPA-albeit a slight delay of peaking followed by increase of decline level of $[Ca^{2+}]_i$. In addition, such an 835-MHz radiation also had no effect on the changes in intracellular ROS induced by LPA and stress-fiber formation by LPA in Rat-2 fibroblasts.

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