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In Vitro Exposure of Neuronal Networks to a GSM-1800 Signal

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The central nervous system is the most likely target of mobile telephony radiofrequency (RF) field exposure in terms of biological effects. Several electroencephalography (EEG) studies have reported variations in the alpha-band power spectrum during and/or after RF exposure, in resting EEG and during sleep. In this context, the observation of the spontaneous electrical activity of neuronal networks under RF exposure can be an efficient tool to detect the occurrence of low-level RF effects on the nervous system. Our research group has developed a dedicated experimental setup in the GHz range for the simultaneous exposure of neuronal networks and monitoring of electrical activity. A transverse electromagnetic (TEM) cell was used to expose the neuronal networks to GSM-1800 signals at a SAR level of 3.2 W/kg. Recording of the neuronal electrical activity and detection of the extracellular spikes and bursts under exposure were performed using microelectrode arrays (MEAs). This work provides the proof of feasibility and preliminary results of the integrated investigation regarding exposure setup, culture of the neuronal network, recording of the electrical activity, and analysis of the signals obtained under RF exposure. In this pilot study on 16 cultures, there was a 30% reversible decrease in firing rate (FR) and bursting rate (BR) during a 3 min exposure to RF. Additional experiments are needed to further characterize this effect. Bioelectromagnetics 34:571–578, 2013. © 2013 Wiley Periodicals, Inc.

Key words: feasibility study; GSM-1800 signal; neuronal networks; electrical activity; in vitro

INTRODUCTION

Evaluation of many potential mechanisms suggests that dielectric heating leading to temperature elevation in tissues is the dominant and possibly only mechanism for biological effects of the radiofrequency (RF) fields relevant to wireless communications [AGNIR, 2012]. Under typical exposure conditions of mobile telephony, the central nervous system is the most likely target of RF exposure in terms of biological effects, and several electroencephalography (EEG) studies have reported variations in the EEG power spectrum during and/or after RF exposure, in resting EEG and during sleep [Van Rongen et al., 2009; Croft et al., 2010; Schmid et al., 2012]. The most recent of these studies provides some evidence that RF exposure may directly influence brain functions. Therefore, it is crucial to clarify the mechanisms underlying these potential RF effects on the brain, both at cellular and network levels. In this context, the extracellular observation of the spontaneous electrical activity of neuronal networks under RF exposure can be an efficient tool to detect the occurrence of low-level RF effects on the nervous system.

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Our research group has developed a dedicated experimental setup in the GHz range for the simultaneous RF exposure of neuronal networks [Merla et al., 2011] and monitoring of electrical activity. A similar setup had been previously built for the same purpose, based on a rectangular waveguide [Koester et al., 2007]. Preliminary electrophysiological data were not included in the initial publication but in subsequent reports a correlation between the electrical activity and specific absorption rate (SAR) was reported [Gimsa, 2007; Sakowski and Gimsa, 2008]. In our work, a transverse electromagnetic (TEM) cell was used to expose the neuronal networks to Global System for Mobile Communications at 1800 MHz (GSM-1800) signals at an SAR level of 3.2 W/kg. The GSM signal was selected among the many wireless communications signals as it provides seven empty timeslots out of eight, which is favorable for recording the neuronal signals in the absence of RF fields. This work provides the proof of feasibility and preliminary results of the entire investigation regarding dosimetry of the exposure system, culture of the neuronal network, recording of the electrical activity, and analysis of the signals obtained under RF exposure.

MATERIALS AND METHODS

Acquisition System

The electrophysiological interface that we used was commercial microelectrode arrays (MEAs) from Qwane Biosciences (Qwane, Lausanne, Switzerland). These biochips are built on 15 mm × 15 mm glass substrates mounted on printed circuit boards (PCB; 50 mm × 50 mm) using standard microfabrication technologies. They provide 60 platinum electrodes (200 μm spaced with 40 μm diameter tips) and a 6 mm-high glass cylinder was used as the culture chamber and sealed with biocompatible silicone. For our application, the pre-amplifier (MEA1060-Inv, Multi Channel Systems (MCS), Reutlingen, Germany) had to be placed underneath the MEA to allow the insertion of the culture chamber inside the exposure system; we thus had custom MEAs built by Qwane Biosciences, in which the contact pads were placed on the lower side of the PCB (Figs. 1 and 2).

Exposure Setup

The MEA, hosting the neuronal network, was placed via a hole in the ground plane inside a TEM

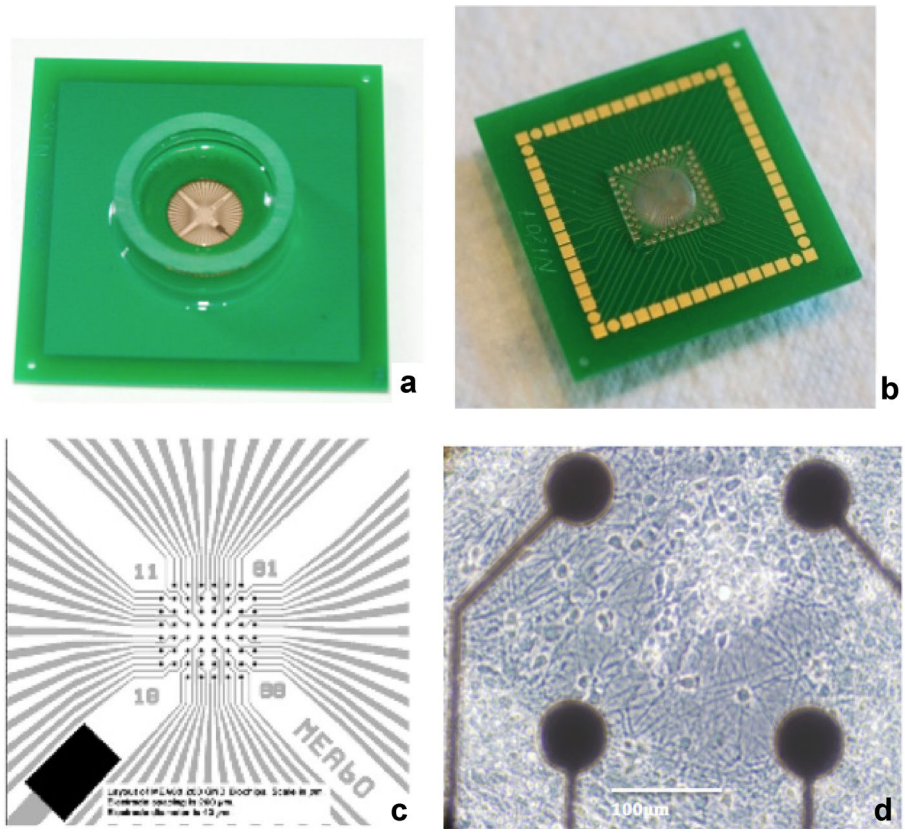

Fig.1. MEA views. **a**: Upper side of MEA with the culture chamber, and (**b**) lower side; (**c**) electrode layout grid; (**d**) zoom on electrode tips and neurons in culture.

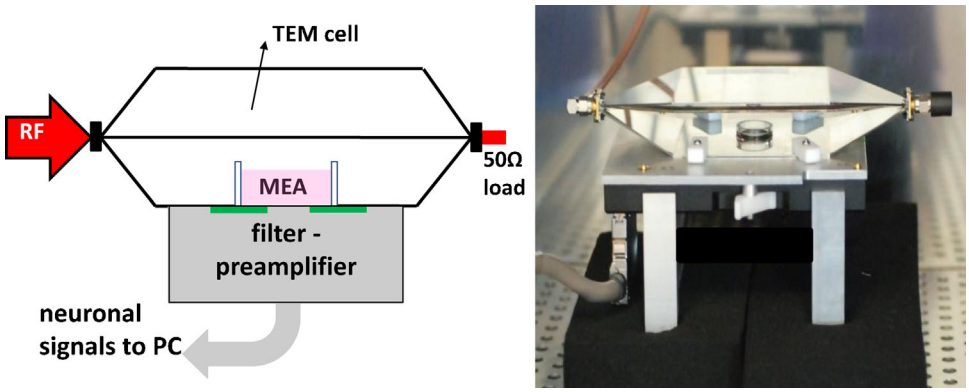

Fig. 2. Experimental setup. **Left**: schematic representation; **Right**: location inside the incubator.

cell where the GSM-1800 signal was propagating (Fig. 2). Dosimetric modeling of the exposure system has been published [Merla et al., 2011]. For an input power of 1 W, a SAR level of 3.2 W/kg was calculated, measured, and used in this work. This level is above the SAR level calculated in the human cortex during mobile phone use (around 0.15 W/kg for the GSM-1800 signal) and corresponds to a 0.06 °C temperature elevation in the culture medium.

Preparation of Cortical Neurons

Extracellular recordings of the electrical activity of cortical cell cultures were performed on the 60-channel planar MEAs described above. The MEAs were successively coated with polyethyleneimine and laminin (Sigma–Aldrich, St. Quentin-Fallavier, France). Primary neuronal cell cultures were obtained from the cortex of embryonic (E18) Sprague–Dawley rats (Charles River Laboratories, L'Arbresle, France). All chemicals quoted below were acquired from Fisher Scientific (Illkirch, France). Cortices were dissected in Dulbecco's Modified Eagle Medium (DMEM)–GlutaMAX and treated with trypsin for 25 min. The fragments were subjected to mechanical dissociation using Pasteur pipettes and briefly centrifuged. The supernatant was transferred into a new tube and centrifuged at 140*g* for 5 min. The pellet was then successively treated with trypsin, soybean trypsin inhibitor, DNase, and finally centrifuged at 140*g* for 5 min. Pellet-dissociated cortical cells were suspended in the culture medium (neurobasal medium supplemented with 2% B-27, 1% GlutaMAX, and 1% penicillin/streptomycin). Each MEA was plated with a suspension of 10^5 cells and kept in a 5% CO_2 incubator at 37 °C in a humidified atmosphere until recording. The culture medium was half-exchanged twice a week [Berdondini et al., 2006].

Acquisition of Electrical Activity

To maintain the proper cell culture conditions during the recordings, the experiments were carried out in a dry incubator (37 °C, 5% CO_2), which contained the pre-amplifier, the MEA, and the exposure system. A removable membrane of fluorinated ethylene-propylene (ALA Scientific Instruments, New York, NY) was used to seal the MEA culture chamber, preventing evaporation but allowing for gas exchange.

The pre-amplification gain was 1200 and a shielded cable allowed data transfer from the preamplifier inside the incubator to a personal computer equipped with an MCS-dedicated data acquisition board. Raw data were sampled at 25 kHz/channel. Signals were recorded and monitored using MC Rack software (MCS) for on-line visualization and raw data storage.

The neuronal networks were exposed between 15 days in vitro (DIV) and 21 DIV, when neuronal activity is optimal in terms of a balance between random spikes and bursts [Chiappalone et al., 2005; Van Pelt et al., 2005]. All recordings were carried out in three subsequent 3 min periods, that is, before, during, and after exposure. Sham exposures were also carried out on the day before, using the same exposure protocol but with the generator off (Fig. 3).

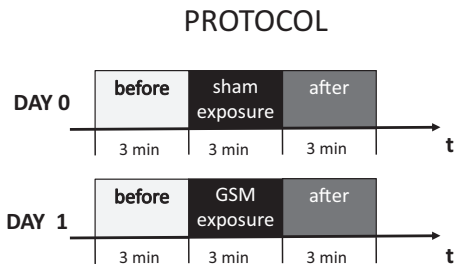

Fig. 3. Time profile of the test protocol.

DATA ANALYSIS

Signal Processing

Offline signal processing was performed using SPYCODE software [Bologna et al., 2010] developed in MATLAB (Mathworks, Natick, MA). SPYCODE is designed to work with multi-site extracellular recordings and can process MCS data files (.mcd) and convert them into MATLAB-format files. A second-order Butterworth high-pass filter with a cut-off frequency at 50 Hz was first applied to the raw data to eliminate slow variations in the signal baseline. During GSM exposure, we observed the presence of an artifact created by interference with the GSM RF signal, as shown in Figure 4 (right). The artifact amplitude never exceeded 1 mV, which guarantees that the pre-amplifier operated in its linear range (0–4 mV). We were thus able to apply a second-order linear filter in order to remove this artifact; the "GSM filter" consisted of a set of 30 band-stop Butterworth filters, each one centered on one of the GSM harmonic frequencies (217, 434, up to 6510 Hz), with a bandwidth of 4 Hz.

To ensure the reliability of our method, we tested the impact of this GSM filter on the number of detected spikes under two conditions: (i) recording of spontaneous activity in the absence of exposure for an electrode in one of the 16 cultures (reference signal); and (ii) a composite signal including exposure, obtained as the sum of the reference signal and a pure GSM artifact recording (Fig. 5, top). Using the GSM filter on this composite signal allowed us to remove the GSM interference (Fig. 5, bottom). This signal processing had a very small effect on spike detection in terms of the total number of spikes (Table 1). The same test was carried out using, as a reference signal, the spontaneous activity recorded of the six most active electrodes, and analogous results were obtained (see the Online Supplementary Material).

Spike and Burst Detection

Spikes were detected using an algorithm implemented in SPYCODE, which relies on a differential threshold (precision timing spike detection, PTSD) [Maccione et al., 2009] and detects a spike when the peak-to-peak amplitude of the signal exceeds eight times the standard deviation (SD) of the biological noise in a 2 ms sliding window (peak lifetime period = 2 ms, refractory period = 1 ms). The SD of the biological noise was evaluated for each recording channel in the pre-exposure phase.

Bursts were detected using the method described by Pasquale et al. [2010]. The algorithm is based on the computation of the logarithmic inter-spike interval (ISI) histogram and automatically detects the best threshold for distinguishing between inter- and intra-burst ISIs for each recording channel of the array.

Choice of Metrics

Electrodes were considered as active in terms of neuronal electrical activity if they had a spike rate of at least 0.1 Hz [Boehler et al., 2012]. For describing

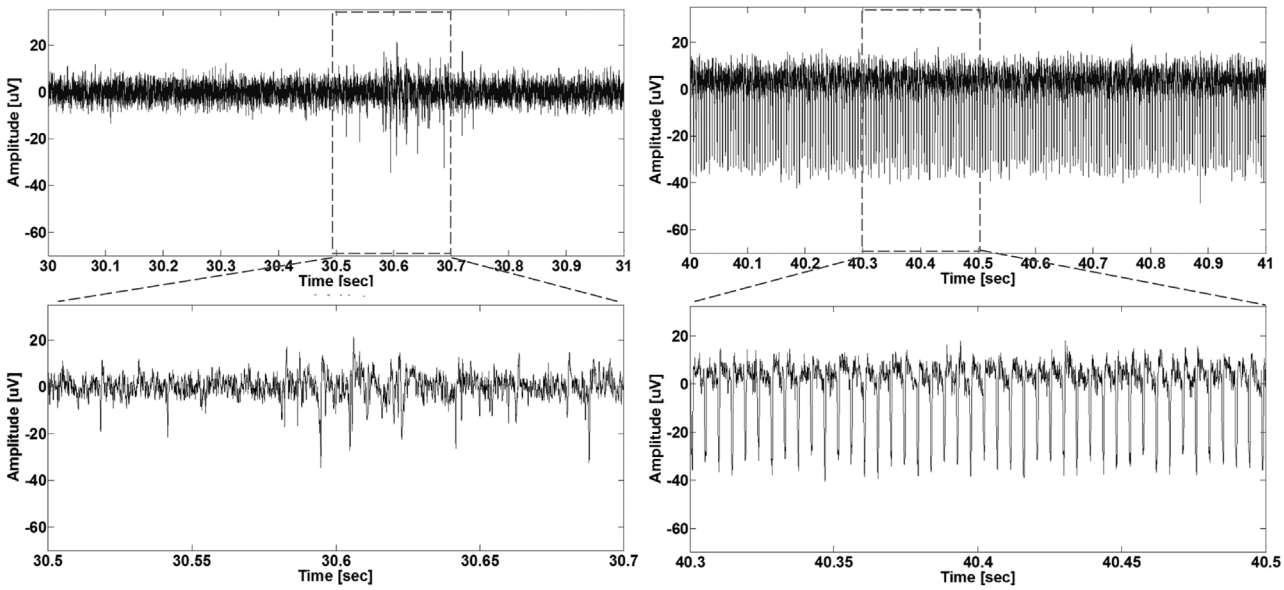

Fig. 4. Examples of recorded activity. **Left**: activity without GSM exposure; **Right**: activity during GSM exposure.

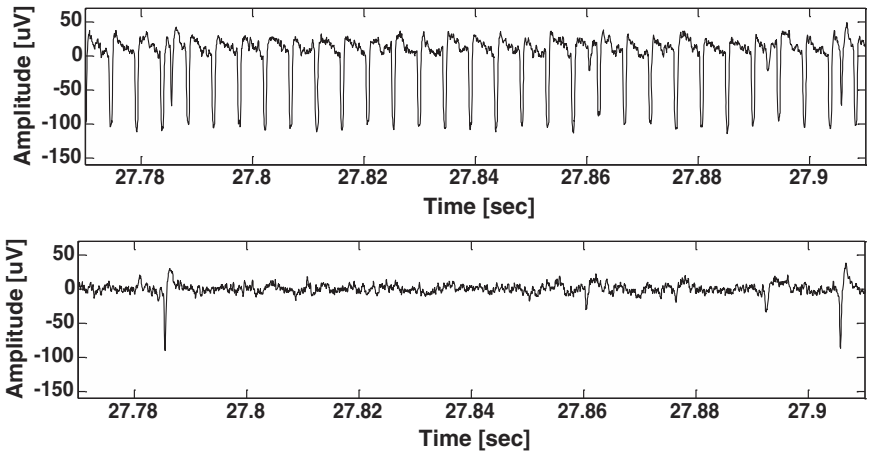

Fig. 5. Composite signal of electrical activity (μV) during exposure before (**top**) and after (**bottom**) the GSM filter.

electrical activity we used two parameters: the firing rate (FR), and the bursting rate (BR). The FR of a given culture is defined as the total number of spikes collected during 3 min for the six most active electrodes identified in the pre-exposure period. The BR is defined in terms of bursts/minute and is the total number of bursts for all burst-active electrodes over 3 min.

Statistical Analysis

As the sample size was small ($n = 16$ independent cultures), statistical analysis required the use of non-parametric tests. In the analysis of sham and exposure data, the Shapiro–Wilk test (Anastat software, Rilly sur Vienne, France) was used to process the ratios of FR and BR to detect a possible effect of GSM exposure on spontaneous activity. This test is used for analyzing the normality of a population but it can also assess the comparison with a fixed value, in a similar way as the Student's *t*-test but with a fewer number of independent samples.

RESULTS

During 2 consecutive days the cells were sham exposed and RF exposed according to the protocol described in Figure 3. Both sets of data were analyzed by testing two parameters related to spikes and bursts (FR and BR).

Pre- Versus Post-Exposure Data

In order to analyze the variations of each parameter during the different runs, before and after exposure, we evaluated the ratio $R_X = X_{\text{before}}/X_{\text{after}}$, where R_X is the ratio for parameter X (FR or BR) and X_{before} and X_{after} are the counts for parameter X before and after exposure, respectively. R_{FR} and R_{BR} were evaluated for both sets of exposures (sham and GSM). Table 2 shows that in all cases, R_{FR} and R_{BR} were not significantly different from unity ($P > 0.25$, Shapiro test), which indicates that the baseline of the FR and BR was stable over 9 min for both sham and real exposures.

TABLE 1. Impact of the GSM Filter on the Reference and Composite Signals in Terms of Number of Detected Spikes

Signal	Filter	# of detected spikes	% relative error
Reference signal	None	1536	—
	GSM filter	1514	−1%[a]
Composite signal = reference signal + GSM artifact	GSM filter	1611	+6%[b]

[a]Reference signal with no filter.
[b]Reference signal with GSM filter.

TABLE 2. Values of the Ratios for the Firing Rate (FR) and Bursting Rate (BR) Parameters and Corresponding *P* Values Based on the Shapiro–Wilk Test

	GSM	Sham
R_{FR}		
Mean	1.00	1.01
P-value	0.92	0.90
R_{BR}		
Mean	1.11	1.13
P-value	0.27	0.35

TABLE 3. Values of the Ratios for the FR and BR for Exposure and Sham Experiments, and Corresponding *P* Values Based on the Shapiro–Wilk Test

	GSM	Sham
R'_{FR}		
Mean	0.75	1.00
P-value	0.0002	0.95
R'_{BR}		
Mean	0.70	1.03
P-value	0.0001	0.72

Effects of Exposure

For each of these parameters, the ratio R'_X was calculated to express the changes observed during sham or GSM exposure with respect to the averaged counts before and after exposure, to account for any small slope in the signal baseline: $R'_X = X_{\text{exposure}}/((X_{\text{before}} + X_{\text{after}})/2)$, where X_{exposure} is the count for parameter X during exposure. Table 3 and Figure 6 show that FR and BR were not altered during sham exposure but were reduced by around 30% during GSM exposure. Moreover, these preliminary data suggest that the amplitude of the effect decreased with age when going from 15 to 21 DIV.

DISCUSSION AND CONCLUSION

The main aim of this work was to assess the feasibility of studying the electrical activity of neuronal networks under exposure to mobile-phone RF signals at 1800 MHz. The use of MEAs has already been successfully applied to pharmacological [Piet et al., 2011], toxicological [Scelfo et al., 2012], and patho-physiological investigations [Volmer et al., 2007; Dribben et al., 2010]. For our purpose, a RF system was built to expose the biological samples inside the MEAs and was well characterized in terms of dosimetry [Merla et al., 2011]. As described above, the GSM artifact was eliminated using a spectral filter but we also needed to exclude the possibility that this interference was the direct cause of the effect. The "GSM artifact", which was observed as an "apparent"

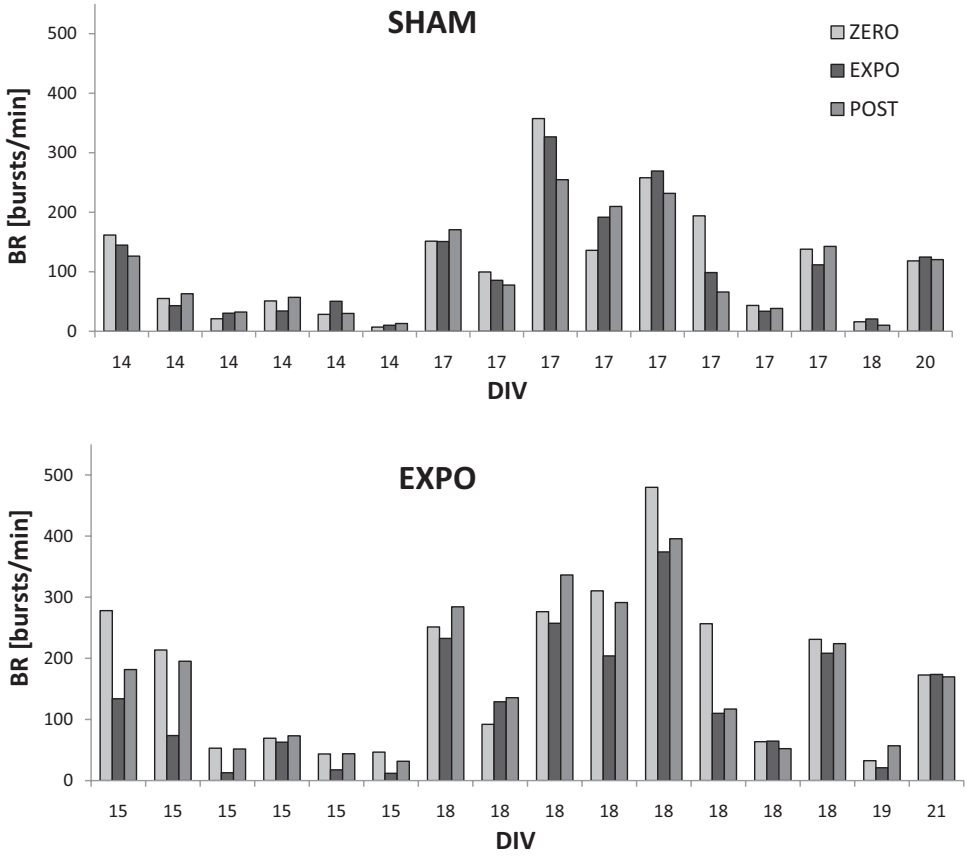

Fig. 6. Bursting Rate before, during, and after exposure for the 16 independent cultures. Age of the cultures is given in abscissa in days in vitro (DIV). Data for a given culture are shown for sham exposure (**top**) and GSM exposure (**bottom**).

induced voltage on the electrodes, was always much less than 1 mV in amplitude, and thus no electrical neuronal stimulation was expected as the threshold for such processes is known to be typically 1000-fold higher. Moreover, some preliminary tests showed that shielding the amplifier circuits using RF-absorbing material decreased the recorded amplitude of the artifact by 10-fold. Further evidence for a lack of contribution of the artifact in the elicitation of the effect came from the observation of a decrease in the amplitude of the observed effect with the age of the culture. In conclusion, there is evidence that the artifact corresponds to electromagnetic interference with the amplifier and not to an induced voltage at the electrodes in contact with the neurons.

The results of 16 independent experiments showed no alteration in electrical activity following cessation of 3 min RF exposures. However, a significant decrease in both the spontaneous spiking activity (FR) and bursting rate (BR) was observed during RF exposure. This effect was phasic and reversible as it lasted through the whole exposure period but ended with the exposure.

There are several stimuli that are known to elicit inhibitory effects on neuronal networks, such as pharmacological agents [McCabe et al., 2007; Piet et al., 2011], toxic agents such as the nickel ion [Gavazzo et al., 2011], or a decrease in temperature [Rubinsky et al., 2010]. However, none of those operate in a reversible manner as rapidly as observed in our work. Therefore, the inhibitory effect of GSM exposure seems to be specific at least for short exposure durations. The rapid onset of the effect and its reversibility are both in favor of a mechanism occurring at the neuronal membrane, where fast bioelectric phenomena can be generated with relatively little inertia.

The role of temperature elevation in the elicitation of the observed effect of GSM exposure must be discussed in terms of "thermal effects", that is, biological effects caused by temperature elevation in the tissues. To date, these are the only effects that have been well documented. In the investigations of the Gimsa group, performed using continuous wave (CW) and Universal Mobile Telecommunications System (UMTS) signals at up to 2.6 W/kg, there was a rise in temperature of up to 0.24 °C [Sakowski and Gimsa, 2008]. Around 33% of the evaluable neurons showed an increase in activity, which correlated with the power of the UMTS signal. No influence of the power control of the UMTS signal was found at 10 or 740 Hz. The conclusion of the authors was that the mechanism behind the increase in activity was of a thermal nature.

In our experimental system, the temperature elevation of the neurons had a time constant of 13 min and reached 0.3 °C at steady state for a SAR of 3.2 W/kg [Merla et al., 2011]. Under our exposure conditions, the temperature elevation at the end of the 3 min exposure was thus around 0.06 °C. This elevation is very small compared to the 0.24 °C temperature elevation measured in the experimental work of the Gimsa group described above, and is very unlikely to be sufficient to cause a reversible effect in a cellular system. However, when using the GSM signal, the energy is not deposited in a continuous manner since during the "on" timeslot, the power is eight times the average power. The only way to ascertain the role of this time domain multiple access (TDMA) amplitude modulation is to perform exposures using CW RF. The elicitation of the effect will be further studied systematically as a function of SAR, modulation, duration of exposure, age of the culture, and pharmacological stimulation.

Assuming that this effect of GSM exposure is validated, the consequences in terms of interpretation of the human EEG data that were obtained using GSM signals and not CW [Croft et al., 2010] are obviously difficult to assess at this time.

Experimental demonstration of the feasibility was achieved in this work and this opens new perspectives regarding the study of the effects of exposure to RF signals on neuronal tissue functioning.

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Supporting Information

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