

Is the brain influenced by a phone call? An EEG study of resting wakefulness

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Abstract

We recorded the resting electroencephalogram of 20 healthy subjects in order to investigate the effect of electromagnetic field (EMF) exposure on EEG waking activity and its temporal development. The subjects were randomly assigned to two groups and exposed, in double-blind conditions, to a typical mobile phone signal (902.40 MHz, modulated at 217 Hz, with an average power of 0.25 W) *before* or *during* the EEG recording session. The results show that, under real exposure as compared to baseline and sham conditions, EEG spectral power was influenced in some bins of the alpha band. This effect was greater when the EMF was on *during* the EEG recording session than *before* it. The present data lend further support to the idea that pulsed high-frequency electromagnetic fields can affect normal brain functioning, also if no conclusions can be drawn about the possible health effects.

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1. Introduction

The increasing number of telecommunication devices available and the length of time spent using mobile telephones has aroused interest for possible interactions between humans and radio-frequency (RF) radiations (Hyland, 2000; Repacholi, 1998). Under a health perspective, an independent expert group on mobile phones has even proposed that a precautionary principle should be adopted, particularly for children (IEGMP, 2000) even if, at present, there is no convincing scientific data to recommend such a limited use of mobile phones (e.g. Van Rongen et al., 2004).

In view of the proximity between radio-frequency source (i.e. mobile phones) and the human brain, several studies have investigated the effects of EMFs on resting cerebral

activity, but results have often been contradictory (for a review, see Cook et al., 2002; Hamblin and Wood, 2002). Some of the available data, in fact (Reiser et al., 1995; Croft et al., 2002), have shown an influence of EMFs limited to some EEG rhythms, especially on the alpha band (8–13 Hz). Similar findings have also been observed in the spectral content of EEG recorded both prior to sleep onset (Huber et al., 2002) and during sleep (Borbély et al., 1999). However, other studies have detected no influence on human EEG after exposure to RF radiations (Hietanen et al., 2000; Röschke and Mann, 1997).

The aim of the present study was to test: (a) whether a global system for mobile communication (GSM) signal affects human resting EEG, analyzed with a high EEG frequency resolution (i.e. Hz-by-Hz), and (b) when this influence is most evident on EEG spectral power, i.e. whether this is restricted to the period of real exposure or if it continues after exposure cessation.

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2. Materials and methods

2.1. Subjects

Twenty healthy, right-handed subjects (10 males and 10 females; mean age 26.4 ± 2.86 years, range 22–31) took part in the study and gave their written informed consent. All of them were regular mobile phone users, but none used their phone in the 10 h prior to the recording sessions (carried out at 10.00 a.m.). Any neurological or psychiatric history and medication or drug intake was ruled out by means of a questionnaire and a clinical interview. The study was approved by the local ethics committee.

2.2. Exposure characteristics

The mobile phone used was a Motorola *Timeport 260*, set by a test card to transmit a typical GSM RF-ON signal with a carrier frequency of 902.40 MHz (pulsed with a frequency of 217 Hz) at its maximum electromagnetic power emission of 2 W (equivalent to an average power of 0.25 W). An appropriate helmet was used to hold the phone in the usual position of use (the aerial oriented to the temporo-parietal areas and the microphone directed towards the mouth) at a distance of 1.5 cm from the left ear. Exposure to EMF was previously measured by using a head phantom filled with a semi-liquid muscle equivalent material. The phone was positioned 1.5 cm away from the ear and switched-on at its maximum power. In accordance with the standards of compliance (IEC, 2002), the specific absorption rate (SAR) was evaluated inside the phantom: a maximum SAR of 0.5 W/kg was measured (more details in Curcio et al., 2004).

2.3. Procedure

The subjects were randomly assigned to one of two experimental groups and, as indicated in the paradigm shown in Fig. 1, they were submitted to three different conditions, in randomized order: a baseline (BSL), a real exposure (EMF-ON), and a sham exposure session (EMF-OFF), with at least 48 h between each session, according to a double-blind protocol. The BSL session was recorded to obtain a steady level of brain activity, avoiding any possible uncontrolled effect due to the presence of the mobile phone. During this session, subjects wore only the helmet, whereas during the EMF-ON and EMF-OFF conditions, they wore both helmet and mobile phone (turned, respectively, on and off).

Moreover, Group 1 (10 Ss, five females) was exposed to the signal for 45 min *before* the resting EEG recording, while Group 2 (10 Ss, five females) was exposed to the field for an equivalent period of time, but the EEG recording session occurred *during* the final 7 min of the exposure period (see Fig. 1): however, the total amount of RF exposure was the same for both groups, and a same quantity of absorption occurred in all the subjects. During the 45 min periods, the subjects of Group 1 were engaged in their

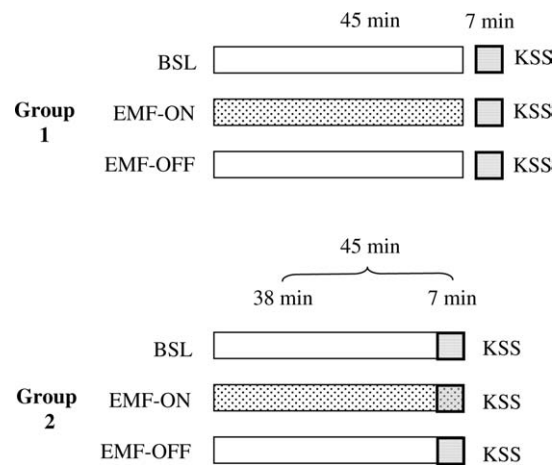


Fig. 1. Schematic representation of the experimental paradigm. Shaded squares indicate the 7 min period of EEG recording repeated in all the three conditions: baseline (BSL), real exposure (EMF-ON, dotted area), and sham exposure (EMF-OFF). The two groups were differentiated by the moment of EEG recording: after the end of exposure in Group 1, and during the last 7 min of exposure for Group 2.

favourite activities, such as reading, talking, listening to music, etc., similar activities were permitted to the subjects of Group 2, but limitedly to the first 38 min, before the EEG recording. On the other hand, during each of the 7 min EEG recording sessions, the subjects were asked to remain seated in a comfortable armchair, fully relaxed and with eyes closed. Immediately after the end of EEG recording, the subjects filled in a subjective scale of sleepiness (Karolinska sleepiness scale (KSS), Åkerstedt and Gillberg, 1990).

Finally, to exclude the possibility that the effect on the waking EEG could be influenced by uncontrolled auditory signals possibly emitted by the active mobile phone, as previously suggested (Haarala et al., 2003), white noise was delivered by means of a loudspeaker, positioned in front of the subjects, during all the experimental sessions. It should be stressed that subjects did not hear any voices or sounds from the phone.

2.4. EEG recordings

An Esaote Biomedica VEGA 24 polygraph set at a paper speed of 10 mm/s was used for polygraphic recordings. EEG signals were high-pass filtered with a time constant of 0.3 s and low-pass filtered at 30 Hz (30 dB/octave); five unipolar EEG channels (Fz-A1, Cz-A1, Pz-A1, T3-A2, T4-A1) were applied using the international 10–20 system. Both the electromyogram (EMG) and electrooculogram (EOG) were recorded, in order to identify any epochs contaminated by artifacts and to thus reject them from the analysis. The EMG was recorded from electrodes placed over the submental muscles, with a time constant of 0.03 s. The EOG was recorded by means of bipolar electrodes located about 2 cm above and below the right eye pupil: vertical eye movements were recorded with a time constant of 1 s. Electrode impedance was kept below 5 K Ω . EEG analogical data were digitalized online (sampling

rate, 128 Hz) and stored on a personal computer. During an offline analysis, each EEG recording was divided into 2 s epochs and, through a visual inspection, any epochs with ocular and/or muscular artifacts were eliminated. The 2 s artifact-free epochs were then submitted to the fast Fourier transform (FFT) algorithm: power values were calculated across a 1–24 Hz frequency range in a 1 Hz resolution. Absolute power was then log-transformed before statistical analyses.

2.5. Statistical analyses

A mixed design of analysis of variance (ANOVA) group (*exposure before, exposure during*) $\times$ condition (BSL, EMF-ON, EMF-OFF) $\times$ frequency (1, 2, ..., 24 Hz) was carried out on resting EEG power values recorded from each derivation. The Bonferroni correction was applied to correct for multiple comparisons: considering the mean correlation between the variables ($r = .45$), the alpha level was then adjusted to ≤ 0.003 . A mixed ANOVA group (*exposure before, exposure during*) $\times$ condition (BSL; EMF-ON; EMF-OFF) was carried out on the KSS scores.

3. Results

The ANOVA on the Cz lead showed a significant condition $\times$ frequency interaction ($F_{58, 1044} = 1.650$;

$p = .002$; Fig. 2, left panel). Post hoc comparisons (*Scheffé test*) indicated a significant difference between EMF-ON (EEG power mean $\pm$ S.E. = 0.77 ± 0.07) and, respectively, BSL (EEG power = 0.65 ± 0.06 ; $p = .02$) and EMF-OFF conditions (EEG power = 0.68 ± 0.09 ; $p = .004$) at the 9 Hz bin. Similar significant differences were found at the 10 Hz bin by comparing EMF-ON (EEG power = 0.87 ± 0.08) and, respectively, BSL (EEG power = 0.79 ± 0.09 ; $p = .02$) and EMF-OFF conditions (EEG power = 0.81 ± 0.09 ; $p = .04$). In both cases, the EMF-ON power values were higher than the baseline and sham exposure power values, which did not differ between them. No other main effect or interaction was significant.

On the T3 derivation, the condition $\times$ frequency interaction only approached significance ($F_{58, 1044} = 1.448$; $p = .018$) at the 9 Hz bin, while EEG power recorded from the Fz and T4 derivations showed no significant main effects or interactions (Fig. 2, right panels).

The ANOVA on the EEG spectral power of the parietal derivation (Pz) showed a significant three-level interaction group $\times$ condition $\times$ frequency ($F_{58, 1044} = 1.887$; $p = .0001$), pointing to a difference between the two groups solely for the EMF-ON condition and for the 11 Hz bin (post hoc comparison with the *Scheffé test*; $p < .00001$). EEG power at this frequency bin was higher in Group 2 (EEG power mean $\pm$ S.E. = 1.15 ± 0.07), exposed to the EMF during the recording session, as compared to Group 1 (EEG

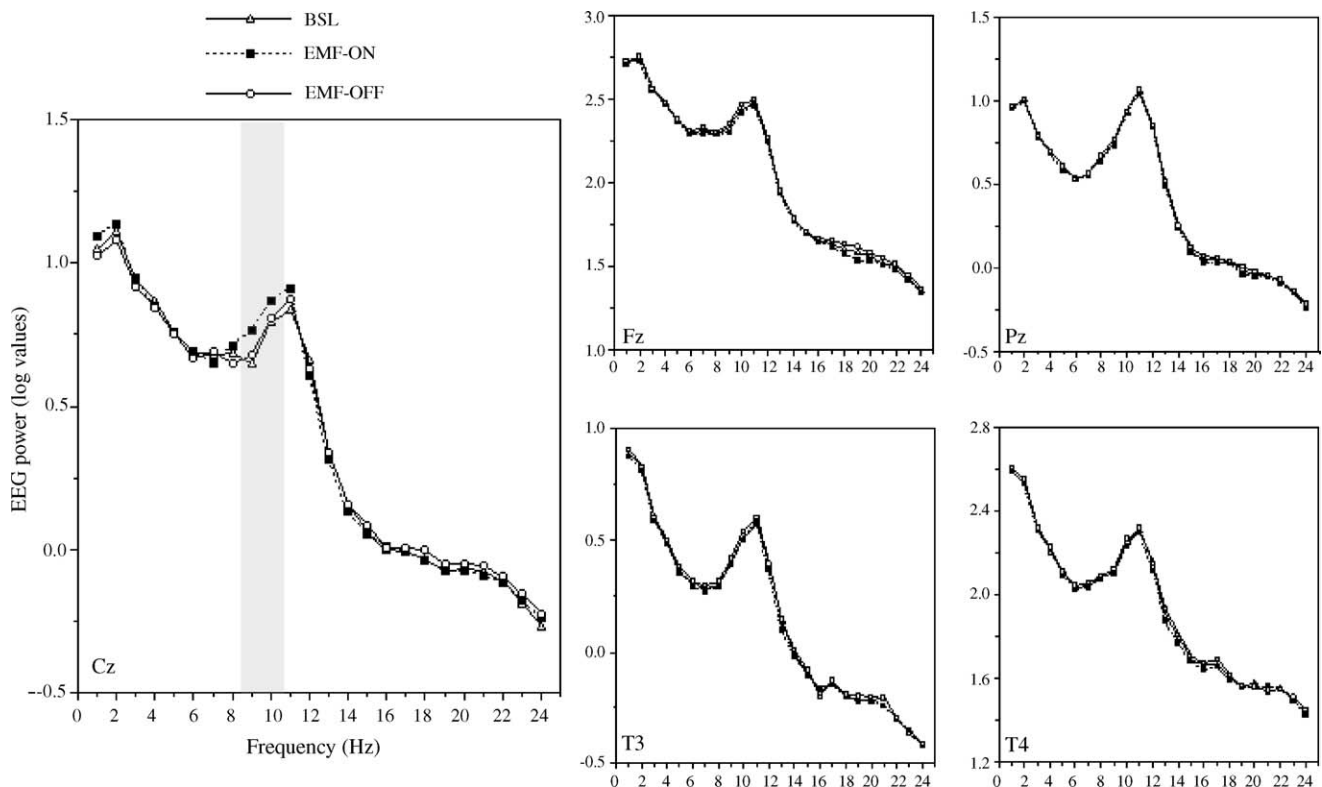


Fig. 2. Mean EEG power for single-Hz bins during resting wakefulness (*condition $\times$ bins interaction*), recorded at Cz (main panel, on the left) and Fz, Pz, T3, and T4 (small panels, on the right). Shading indicates bins in which spectral EEG power significantly increased under real exposure (EMF-ON) with respect to sham (EMF-OFF) and baseline (BSL) conditions (*condition $\times$ frequency interaction*, for more details see the Section 3).

power = 0.96 ± 0.08), exposed *before* the recording session. No other main effect or interactions were significant.

Also, no significant main effects or interactions were observed on the self-rated sleepiness as assessed by the KSS scores.

4. Discussion

As shown in Fig. 2, real RF radiation exposure (EMF-ON) does affect the EEG. The GSM signal influences a small range of EEG frequencies on a specific cortical site, that is the 9–10 Hz bins on the central derivation. This result supports previous studies (Croft et al., 2002) showing that a pulsed RF radiation affects EEG activity also during resting and not only during mental performance (e.g. Krause et al., 2000). A similar effect was also found at Pz, where the 11 Hz EEG power increased in the EMF-ON condition as compared to the BSL and EMF-OFF conditions. Moreover, over the parietal lead, a temporal correspondence between EMF exposure and its effect on the EEG may also be observed, since subjects exposed *during* the recording session (Group 2) showed higher EEG power than subjects exposed *before* the EEG recording (Group 1). These data suggest that the more affected scalp locations seem to be those closest to the aerial, where the greatest absorption should be present. Similar observations were made on the basis of computational approaches (e.g. Bernardi et al., 2000). Nevertheless, it should be born in mind that the activity recorded from midline derivations arises from bilateral sources, as indicated by MEG recordings (e.g. Verkint et al., 1995). As a result, it could be hypothesized that the current resting EEG recorded on Cz and Pz, are the result of a composite activity coming from bilateral areas, and that the possible effect of EMF exposure could be induced by a kind of spreading of RF power across the brain.

A note of caution is needed regarding the strength of the present data, particularly with respect to the sample size and to the effect size. Nevertheless, the current effect size is very similar to those reported by other studies, which showed (e.g. Huber et al., 2002) very slight effects of the GSM signal on the EEG. Finally, the absence of significant effects on sleepiness self-evaluations, indicate that the vigilance level was almost constant across groups and conditions, allowing us to exclude vigilance-induced influences.

The actual electromagnetic field exposure (EMF-ON) affected resting EEG activity in the alpha frequency range, commonly known as the dominant rhythm of the human scalp EEG of adults. The alpha rhythm “works” as an oscillatory component of brain activity and, consequently, as a basic form of information transmission in the brain (for a review Klimesch, 1999). Several studies of event-related activity have indeed shown a positive correlation between alpha frequency and the speed of information processing, and between this rhythm and a good cognitive performance (e.g. Krause et al., 2000; Klimesch, 1999). On the other

hand, under resting conditions, the alpha rhythm is quite widely seen as reflecting a cortical de-activation: a higher power value is often correlated to a situation in which the cortex “has nothing to do”, while a blocking of alpha rhythm is observed in attentive processing to external stimuli or mental operations with high cognitive load (e.g. Laufs et al., 2003). According to this interpretation, EMFs from mobile phones have been seen to cause an increase in EEG power in some alpha frequency bins during wake before sleep onset (Huber et al., 2002). This effect is explained by authors as an altered cortical neuronal activity, probably mediated by changes in thalamic functioning. The current results of an increase in some bins of alpha power seem to point to a reduced cortical activation as a direct consequence of EMF exposure. Nonetheless, this effect may also reflect the difficulty in reproducing an effective situation of resting wakefulness, in which subjects are truly able to do and think nothing (Laufs et al., 2003). Since alpha bins seem to be the most sensitive to signal exposure, future experiments should be addressed to the direct manipulation of the subjects’ cognitive state, by comparing resting versus different levels of cognitive activation, also focusing on posterior derivations (i.e. occipital sites), not investigated in the present study. Moreover, by means of PET scans, an increase in cerebral metabolism immediately after the electromagnetic irradiation over some exposed regions of the scalp has also been reported: this effect was still interpreted as a consequence of an altered cortical activity induced by EMF exposure (Huber et al., 2002), confirming that an increase in cerebral blood flow does not necessarily show a negative correlation with alpha activity (Laufs et al., 2003).

If we look at the temporal development of EMF-mediated effects, some results in line with the current ones were previously reported on avian brain functioning, where neuronal responses started immediately after the exposure to an EMF and lasted for a limited period of time (Beason and Semm, 2002). Moreover, studies on rat hippocampus slices showed that acute exposure to a field in the range of RF promptly causes significant changes in both evoked and spontaneous electrical activity (Tattersall et al., 2001): in this case the neuronal firing varied as a function of field intensity. Finally, an immediate increase in alpha activity was recorded in human scalp EEG as a result of short and repeated exposures to an EMF (15 min ON and 15 min OFF intervals) during a whole night of sleep (Borbély et al., 1999).

In short, we propose that such an altered cerebral activity, reflected by a modified activity at cell, electroencephalographic and metabolic levels, may be due to an alteration of cortical excitability instead of a local heating. In fact, it is well known that in a normal cerebral cortex, the high blood perfusion rate provides an efficient thermoregulating mechanism (Wainwright, 2000), and that in the presence of clearly altered electrical activity, no detectable increases in temperature are observed on hippocampal slices (Tattersall et al., 2001). Similar inconsistencies in the relationship between behavioral performances and thermal increases were

found on the tympanic temperature of human subjects exposed to an active mobile phone emitting a typical GSM signal (Curcio et al., 2004). Taken together, these data suggest the existence of an action mechanism not necessarily mediated by temperature variations. As a result, alternative mechanisms to the thermal one should be considered, such as the modulation of membrane ionic channels like those of Na^+ and K^+ (Cleary, 1995), the altered Ca^{++} efflux from brain tissue (Adey, 1981) or the existence of a specific way for converting RF radiation into a lower frequency modulation, able to influence cortical excitability via cell membrane changes (Stuchly, 1979).

Variations of cortical excitability can also be induced by transcranial magnetic stimulation (TMS), with which a low-frequency stimulation causes inhibition, while a faster stimulation causes cortical activation (Lemon, 2002). Of course, GSM signal and TMS stimulation are very different, since they are respectively an electromagnetic signal and a pure magnetic field (B) that induces an electric field (E). Also, induced power in the brain, depth of penetration and stimulated volume are very different (Carpenter and Ayrappayan, 1994). Nonetheless, they could be mediated by the same mechanism of local membrane depolarization, which can lead to a neural activation (Ruohonen and Ilmoniemi, 2002).

Finally, a recent study observed a structural and functional effect on acetylcholinesterase activity (Barteri et al., 2005): the consequent altered cell functioning may in turn modulate the physiological brain oscillations and, consequently, behaviour (Repacholi, 1998). As an indirect demonstration, recent PET studies during both sleep (Huber et al., 2002) and waking (Huber et al., 2005) indicated that just these low-frequency modulations to the carrier signal are critical to highlight EEG alterations.

5. Conclusions

In view of the present data, we may conclude that EMFs like the ones emitted by mobile phones influence normal brain physiology, probably by means of changes in cortical excitability. Nonetheless, investigations on cell mechanisms are needed to better understand the macroscopic cerebral influences. Also, further studies on humans should be carried out to directly manipulate the amount of exposure in order to show possible dose-related effects. However, conclusions about possible health consequences are premature, particularly with respect to chronic and/or repeated exposures.

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