Exposure to pulsed high-frequency electromagnetic field during waking affects human sleep EEG

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The aim of the study was to investigate whether the electromagnetic field (EMF) emitted by digital radiotelephone handsets affects brain physiology. Healthy, young male subjects were exposed for 30 min to EMF (900 MHz; spatial peak specific absorption rate 1 W/kg) during the waking period preceding sleep. Compared with the control condition with sham exposure, spectral power of the EEG in non-rapid eye movement sleep was increased. The maximum rise occurred in the 9.75–11.25 Hz and 12.5–13.25 Hz band during the initial part of sleep. These changes correspond to those obtained in a previous study where EMF was intermittently applied during sleep. Unilateral exposure induced no hemispheric asymmetry of EEG power. The present results demonstrate that exposure during waking modifies the EEG during subsequent sleep. Thus the changes of brain function induced by pulsed high-frequency EMF outlast the exposure period. *NeuroReport* 11:3321–3325

Key words: Electromagnetic exposure; Global system for mobile communication (GSM); Mobile phones; Non-REM sleep; Sleep spindles; Spectral analysis

INTRODUCTION

The extensive use of mobile phones has given rise to public debate about possible adverse effects on human health. A recent report of the Independent Expert Group on Mobile Phones established by the British government summarized the relevant studies on the biological effects of electromagnetic fields (EMF) [1]. They proposed that a precautionary approach be adopted until more robust scientific information becomes available.

In a previous study, we demonstrated that exposure to EMF during sleep reduced waking after sleep onset and affected the EEG in non-rapid eye movement (non-REM) sleep [2]. Spectral power was enhanced in the 7.25–14.25 Hz range in the first non-REM sleep episode. The maximum rise occurred in the 10–11 Hz and 13.5–14 Hz bands during the initial part of sleep and then subsided. A differential time course of the two bands was observed. The 10–11 Hz band showed a 15% increase in the first non-REM sleep episode only, whereas power in the 13.5–14 Hz band was increased in the first three non-REM sleep episodes and showed a declining trend.

In the present study, we investigated the effect of exposure to pulsed high-frequency EMF during waking on subsequent sleep. Fields similar to those emitted by mobile communications equipment of GSM type (global system for mobile communication) were applied. To simulate the real-life exposure conditions, the subjects were exposed unilaterally. The EMF was directed to either the right or left hemisphere for 30 min. The subsequent sleep episode was analyzed. As in a previous study in which mechanical stimulation of the right hand had been shown to induce unilateral changes in the sleep EEG [3] we anticipated hemispheric differences.

MATERIALS AND METHODS

Design of experiment, subjects and data recording: Sixteen healthy young right-handed men (mean age 22.3 years, range 20–25 years) participated in the study. The subjects were exposed to EMF for 30 min prior to a 3 h sleep episode. Sleep was scheduled in the late morning beginning at either 9.45 h or 10.15 h. The time between end of exposure and lights off was 10 min. To ensure a continuous daytime sleep episode, sleep in the preceding night was restricted to 4 h (beginning at either 22.45 h or 23.15 h). Subjects were under constant supervision between the two sleep episodes.

The experiment consisted of three sessions separated by 1 week intervals. EMF exposure was scheduled according

to a randomized, sham-exposure controlled, double blind, cross-over design. The three sessions involved right hemispheric exposure, left hemispheric exposure and sham exposure.

A screening night prior to the experiment served to exclude subjects with sleep apnea, nocturnal myoclonus and low sleep efficiency. All subjects were reported to be in good health and free of sleep complaints. They were instructed to abstain from caffeine and alcohol at least 3 days prior to the study and to maintain the habitual sleep–wake schedule (23.00–7.00 h) on the day prior to the study. Compliance was verified with wrist-worn activity monitors. The subjects gave their written informed consent, and the local ethical committee for research on human subjects approved the study protocol. During night-time and daytime sleep episodes the EEG (F3, C3, P3, O1, F4, C4, P4, O2, C_Z referenced to linked mastoid), the submental EMG, the EOG, (differential recording), and the electrocardiogram were recorded with a polygraphic amplifier [4]. Fifteen minutes after each sleep episode, subjective sleep variables and mood were assessed by visual analogue scales and questionnaires.

EMF exposure: Subjects were sitting on a chair with their heads positioned between two plates to ensure a well-defined position with respect to the planar antennas (Huber+Suhner SPA 920/65/9/0/V) mounted on both sides of the head. The two antennas were matched with respect to their input characteristics (deviation $< 0.5\%$).The distance between antenna and ear was 11 cm. The center of the antennas was 4.2 cm vertically above the ear canal (one-third of the distance between the ear canal and the top of the head). Either the right antenna, the left antenna or neither of the antennas (dummy load) was excited in a double blind fashion. The 900 MHz carrier signal was modulated as in the previous experiment [2]. The modulation pulse sequence simulated the main frames of the GSM system (2, 8, 217, 1736 Hz) with a duty cycle of 87.5%. The signal was split with a 3 dB power divider into two identical signals supplying the two exposure units, the antenna input power of which differed by $< 0.5\%$.

The simulation platform SEMCAD V1.0 served to optimize the exposure conditions and was used also for the detailed dosimetric analysis. The simulation was based on the MRI data set of the head of a healthy female subject (age 40) [5]. The 121 MRI slices were separated by 1 mm in the ear region and by 3 mm in the upper and lower head regions. A graded mesh was used with a minimum voxel size in the exposed head region of $1\,mm^3$, and 12 tissue types were distinguished. The spatial peak SAR averaged over 10 g was 0.5 W/kg normalized to an antenna input power of 1 W. The averaged SAR of the exposed brain hemisphere was 0.14 W/kg (s.d. ~70%). In order to achieve a relatively homogeneous exposure of the exposed brain hemisphere, the ratio of the averaged SAR values between the two hemispheres was only ~5.

The numerical dosimetry was verified using the near-field scanning system DASY3 (an improved version of the system described in [6]) and a generic twin phantom filled with tissue simulating liquid ($\sigma = 0.86\,Ohm^{-1}\,m^{-1}$, $\varepsilon_r = 41$). The correspondence between measurement and simulation using the same head phantom was better than 10%. Effects of the electrodes were experimentally assessed resulting in a reduction of the spatial peak SAR of ~10%. Hence, the targeted spatial peak SAR value of 1 W/kg was achieved by setting the time-averaged antenna input power to 2.2 W. The uncertainty of the dosimetry is estimated to be less than $\pm 30\%$ (including uncertainties due to dosimetric measurements and to different head anatomies).

Data analysis: Sleep stages were visually scored for 20 s epochs according to standard criteria [7]. Non-REM–REM sleep cycles were defined as in previous experiments [8]. Power spectra of consecutive 20 s epochs (FFT routine, Hanning window, averages of five 4 s epochs) were computed for the referential derivations C3 and C4 (referenced to linked mastoids). Artifacts were identified by visual inspection and on the basis of power in the 0.75–4.5 Hz and 20–40 Hz bands. Only 20 s epochs without artifacts were used for further analysis.

Statistical tests are described in the captions of Figures 1 and 2 and Table 1.

RESULTS

Exposure to EMF affected neither the sleep stages nor sleep latency (Table 1). A single subject who was awake for 63 min after the first solid non-REM sleep episode caused the higher value of waking after sleep onset following sham exposure. Sleep onset REM sleep episodes (i.e. occurrence of REM sleep within the first 15 min of sleep) were frequent (in nine subjects after sham exposure and left hemisphere exposure; in 10 subjects after right hemisphere exposure). The preceding 4 h night sleep episodes did not differ in their sleep stage distribution.

No significant effects of EMF exposure were observed for subjective assessment of waking after sleep onset, sleep latency and sleep quality.

The main effect of EMF exposure was the enhancement of EEG power density in the 9.75–11.25 Hz and in the 12.25–13.25 Hz range in the first 30 min of non-REM sleep (Fig. 1). This effect was also present when the left and right

Table 1. Sleep variables.

	Sham	Left + Right
Total sleep time	162.3 (3.3)	166.4 (2.2)
Sleep latency	5.7 (0.9)	4.6 (0.7)
REM sleep latency	27.7 (8.1)	21.9 (5.8)
Waking after sleep onset	12.3 (3.6)	8.8 (1.7)
Stage 1	13.3 (1.9)	13.6 (2.1)
Stage 2	88.3 (6.1)	84.9 (5.3)
Slow-wave sleep	18.7 (4.1)	20.7 (3.5)
REM sleep	42.1 (4.2)	47.3 (2.7)
Movement time	3.5 (0.4)	3.0 (0.3)

Sleep variables based on visual scoring of 3 h daytime sleep episode. Mean values (s.e.m. in parentheses; $n = 16$) in minutes for sham exposure (sham) and the averaged values of left and right hemisphere exposure (left+right). Sleep onset was defined as first occurrence of stage 2 or REM sleep. Sleep latency: Interval from lights off to sleep onset. REM sleep latency: Interval from sleep onset to the first occurrence of REM sleep. One-way ANOVA for repeated measures with the factor 'exposure' (left *vs* right hemisphere exposure) revealed no significant differences. The difference between left hemisphere exposure and sham exposure reached significance for REM sleep. Two-way ANOVA for repeated measures (within factor condition, between factor sequence and interaction condition × sequence) revealed no significant differences with the exception of a significant interaction for stage 1.

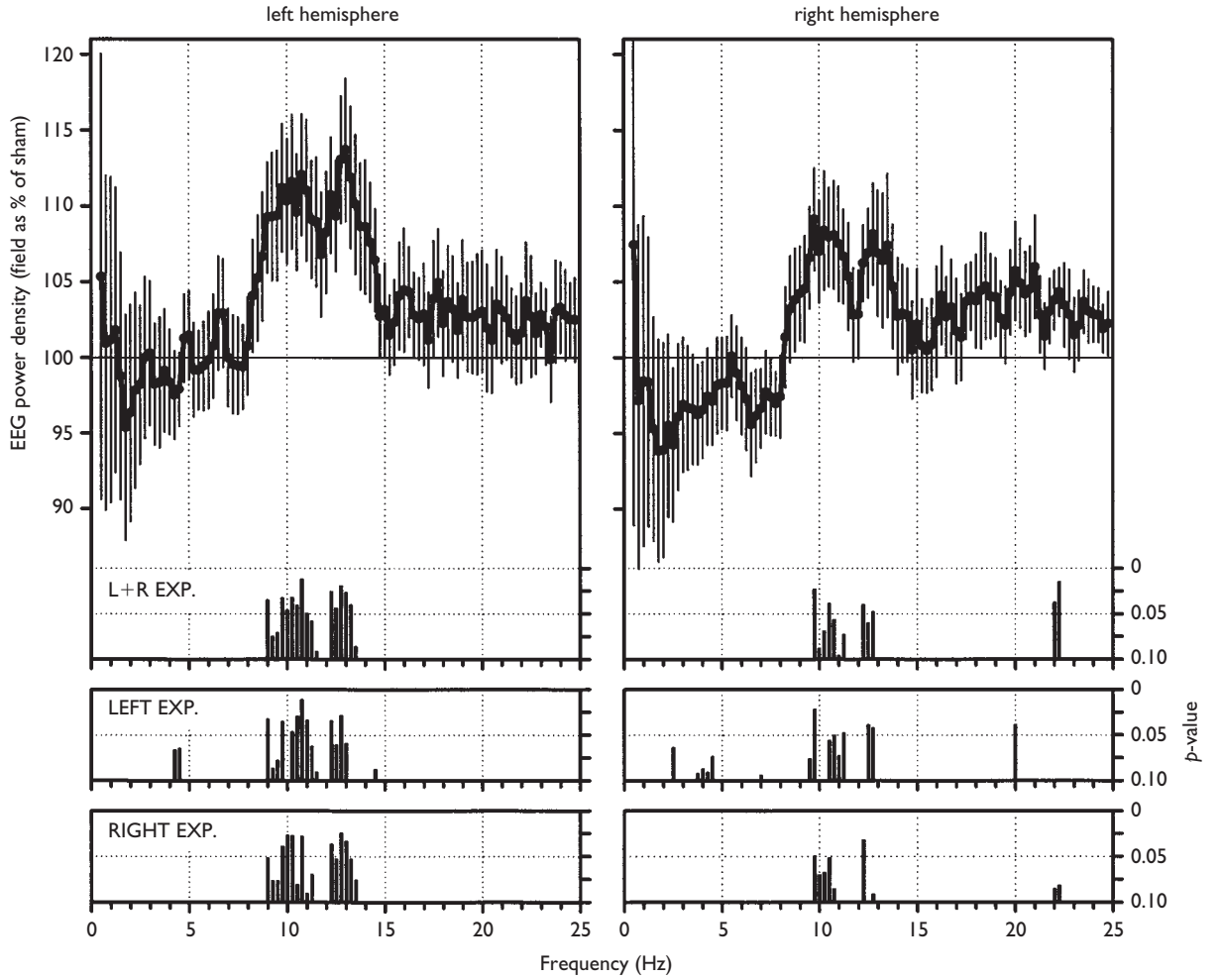

Fig. 1. Mean EEG power density spectra ($n = 16$) of daytime sleep following a 30 min EMF exposure. Average spectra of the first 30 min of non-REM sleep (stages 2, 3 and 4) for the central derivations of the left and right hemisphere (C3, C4 against linked mastoids) are illustrated. Each bin of the 30 min spectra was normalized with respect to the average power of non-REM sleep in the entire sleep episode in that bin. Spectra for left and right hemispheric exposure are averaged and expressed as a percentage of the corresponding value in the sham condition (mean ± s.e.m. for 0.25 Hz bins). Three different two-way ANOVAs for repeated measures were computed for the factors condition (sham *vs* average of left and right exposure or sham *vs* left exposure or sham *vs* right exposure) and sequence (sham exposure in the first session), and for their interaction (condition × sequence). The three bottom panels indicate p values for frequency bins in which power was enhanced (top panel, average of left and right hemispheric exposure (L + R); middle panel, left exposure; bottom panel, right exposure). p values (factor condition) are plotted from 0.1 to 0 in the reverse direction. Neither the factor sequence nor the interaction condition × sequence were significant. Note that two-tailed tests are presented, although one-tailed tests may be justified in view of the expectation based on the previous study [2].

exposure were analyzed separately (Fig. 1, lower two panels). The two hemispheres were similarly affected after left and right exposure, and therefore no lateralization effect was observed. A comparison within individuals showed that the spectral spindle peak frequency in the 10–15 Hz range was not shifted by the experimental condition. The REM sleep spectrum was not significantly affected.

Figure 2 depicts the temporal change of spectral power in the frequency band (12.75–13.25 Hz) that exhibited the largest increase. Mean power in the first and last 30 min period of non-REM sleep is illustrated. Under field conditions power decreased significantly more than in the sham condition. The values in the last 30 min period after field exposure tended to be below the sham exposure level ($p < 0.1$ in all four cases). In the lower frequency range (9.75–11.0 Hz) there was no evidence for a negative rebound.

The temporal position of the 30 min non-REM sleep periods varied somewhat due to differences in the occurrence of sleep onset, REM sleep and brief intermittent waking. However, the midpoints of these periods showed no significant differences between the conditions (first 30 min: left 38.8 (4.2), right 35.4 (4.5), sham 35.8 (3.1); last 30 min: left 147.5 (4.3), right 149.2 (3.3), sham 142.5 (4.4); in minutes after lights off (s.e.m.); $p > 0.3$, paired t-test). The sleep stage distribution of the 30 min period did not vary between conditions.

DISCUSSION

In this study we showed for the first time that exposure to EMF during waking affects the EEG during subsequent sleep. The changes of the spectra corresponded closely to those observed in a previous study in which sleeping

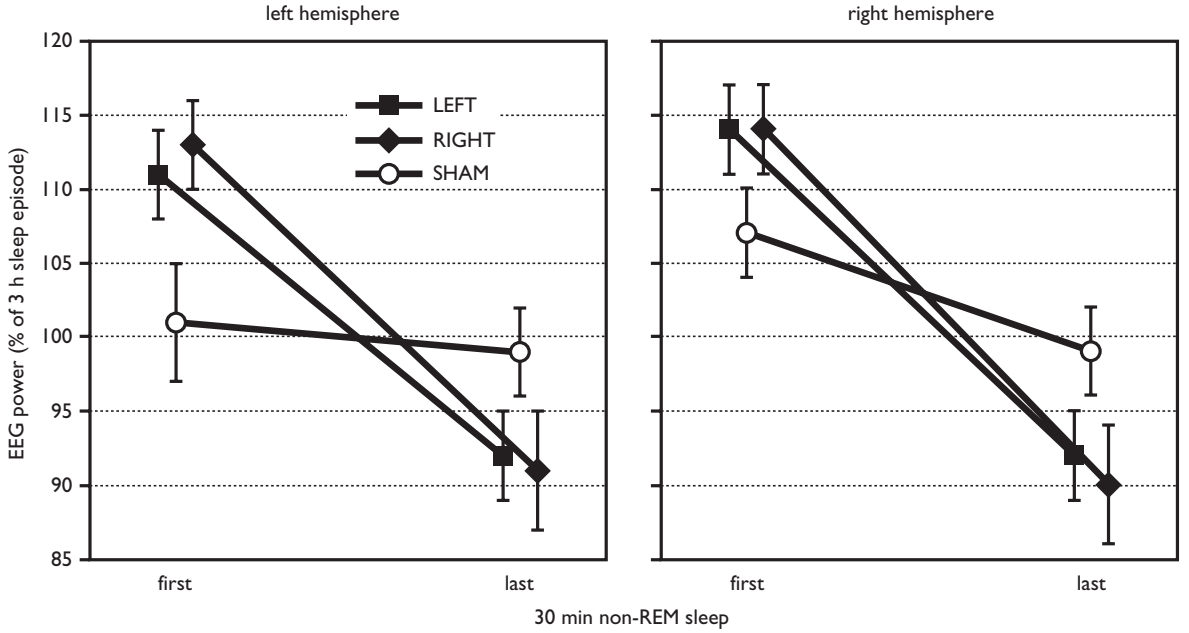

Fig. 2. EEG power in the 12.75–13.25 Hz band in the first and last 30 min period of non-REM sleep for the three experimental conditions (left hemispheric exposure, right hemispheric exposure, sham exposure). Power in each 30 min interval was normalized with respect to the average power of non-REM sleep in the entire sleep episode in this frequency range. Differences between the first and last 30 min period were tested by three-way ANOVAs for repeated measures with the factors condition (sham or field exposure), derivation (left or right hemisphere), sequence (sham exposure in the first session) and their interactions. Only the factor condition was significant ($p = 0.02$). Compared with sham exposure, power decreased significantly more from the first to the last 30 min period after EMF exposure (p values 0.011–0.034, based on two-tailed paired t-tests).

subjects had been intermittently exposed to EMF [2]. In both studies, power in non-REM sleep was enhanced in the alpha band and in the adjacent higher frequency range that is mainly determined by sleep spindles. Also the frequency of the two peaks in the relative spectrum and their height (~15% relative to sham-exposure) was similar.

The increase of power in the first 30 min of non-REM sleep was no longer present at the end of the 3 h sleep episode. This shows that the effect of EMF is transitory and restricted to the initial part of sleep. The decline of power below the sham-control level, even though not significant, may represent a negative rebound. Also in the previous study [2], the increase of power was limited to the first part of sleep despite the persistent intermittent exposure to EMF throughout the night. In view of the present results, the intermittent exposure may have influenced the EEG and prevented a negative rebound.

It is difficult to specify the onset of the EMF effect on the sleep EEG because it was manifest only during non-REM sleep. About 15 min elapsed between the end of exposure and sleep onset, and due to the occurrence of sleep onset REM sleep episodes, the mean onset time of non-REM sleep was delayed by another 5 min. In the previous study, a significant effect was present already after an exposure period of 15 min [2]. It is therefore probable that brain physiology was affected already prior to the onset of non-REM sleep.

In our previous study, the EMF was directed towards the top of the head to expose both hemispheres. In the present experiment, the field was aimed at the lateral part of the head to expose preferentially one hemisphere.

Contrary to our expectation, the change in EEG power was similar for both hemispheres and no asymmetry was detected. Two explanations may be considered. First, the SAR ratio of about 5 between the exposed and non-exposed hemisphere may have been too low to induce a differential effect or a ceiling effect may have been present (i.e. the lower field strength at the non-exposed hemisphere may have been sufficient for a maximal effect). Second, subcortical regions may contain the most sensitive structures to EMF and their bilateral cortical projection may explain the absence of a hemispheric asymmetry. Since the thalamus is centrally involved in the generation of sleep spindles [9], it represents a prime candidate for an EMF sensitive subcortical structure.

There was no EMF effect on sleep variables in contrast to the previous study where waking after sleep onset was reduced [2]. This mild sleep-promoting action of EMF was attributed to the counteraction of a slight sleep disturbance caused by the unfamiliar experimental setup. This factor was minimized in the present study, since the night-time and daytime sleep recordings were performed in close succession in the same environment and daytime sleep propensity was enhanced by the restriction of night-time sleep.

The present results lend support to previous reports on effects of EMF on physiological and psychological variables. These include sleep [10] and cognitive function [11–14] as well as blood pressure and heart rate [15]. However,

the present study is unique in having confirmed the results of an experiment performed under similar conditions. The other findings still need to be replicated or have not been reproduced [10,16].

CONCLUSION

Exposure to EMF emitted by digital radiotelephone handsets affects the sleep EEG by enhancing power in the 9.75-13.25 Hz range. Since power in this frequency range is largely determined by slow and fast sleep spindles [17], spindle generating mechanisms seem to be particularly susceptible to EMF exposure. The absence of hemispheric asymmetry despite unihemispheric exposure may suggest a high susceptibility of subcortical structures such as the thalamus. The changes in EEG power are manifested rapidly when exposure occurs during sleep [2]. They outlast exposure by 20–50 min when EMF is applied during waking prior to sleep. This study demonstrates that a short exposure to EMF emitted by mobile phones has an effect on brain physiology. Conclusions about possible adverse effects on human health are premature because the underlying mechanisms are unknown. Further studies are needed to delineate the time course of the changes, to specify field strength–response relationships, and to define the critical field parameters (e.g. modulation, frequency).

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