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Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram

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Abstract

To investigate whether the electromagnetic field (EMF) emitted by digital radiotelephone handsets affects the brain, healthy, young subjects were exposed during an entire night-time sleep episode to an intermittent radiation schedule (900 MHz; maximum specific absorption rate 1 W/kg) consisting of alternating 15-min on–15-min off intervals. Compared with a control night with sham exposure, the amount of waking after sleep onset was reduced from 18 to 12 min. Spectral power of the electroencephalogram in non-rapid eye movement sleep was increased. The maximum rise occurred in the 10–11 Hz and 13.5–14 Hz bands during the initial part of sleep and then subsided. The results demonstrate that pulsed high-frequency EMF in the range of radiotelephones may promote sleep and modify the sleep EEG. © 1999 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Global system for mobile communication; Mobile phones; Non-REM sleep; Specific absorption rate; Spectral analysis; Sleep spindles; Waking after sleep onset

An expert group of the European Commission has recently recommended increased research to establish possible health effects related to the use of radiotelephones [11]. In view of the very limited data related to the exposure to personal telecommunication devices, the need for human laboratory research on volunteers including sleep studies was recognized. Previous investigations of the effect of electromagnetic fields (EMF) on sleep yielded inconsistent results [10,14]. In the present study, pulsed high-frequency EMF corresponding to those emitted by radiotelephones of type GSM (global system for mobile communication) were applied to healthy young males to investigate the effect on sleep.

Two sound insulated and temperature controlled bedrooms were equipped for simultaneous EMF exposure or sham exposure of two subjects. Behind each bed an array of three $\lambda/2$ dipole antennas was mounted at a distance of 30 cm from the head of a recumbent subject. Absorber walls were placed around the bed. This set-up approximates an anechoic room and ensures a relatively uniform exposure even when the subject changes position in the bed. The determination of the specific absorption rate (SAR) was based on a homogeneous body phantom. The maximum exposure was set not to exceed 1 W/kg (averaged over 10 g; international exposure limit 2 W/kg [7]). The test signal was a pseudo GSM signal of 900 MHz (linear polarization perpendicular to the surface of bed) modulated with frequencies used by handsets and base stations (modulation frequency components: 2, 8, 217, 1736 Hz; 87.5% duty cycle as compared with 12.5% for cellular phones).

Nocturnal sleep (sleep episode: 23.00–07.00 h) of 24 healthy young right-handed men (mean age 22.6 years, range 20–25 years) was recorded during a night with exposure to EMF ('field exposure night') and a night without exposure ('sham exposure night'). A screening night served to exclude subjects with sleep apnea and nocturnal myoclonus. The subjects reported to be in good health and free of sleep complaints. They were instructed to abstain from caffeine and alcohol and to keep a regular sleep wake schedule for at least 3 days prior to the study. Informed written consent was obtained and the study protocol was approved by the local ethical committee for research on human subjects. The experiment consisted of two sessions at an interval of 1 week. On each session, an adaptation night was followed by an experimental night. EMF exposure was scheduled according to a randomized, placebo

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controlled, double-blind, cross-over design. During exposure nights, EMF was turned on and off automatically at 15-min intervals starting with the on condition at lights off. This protocol was chosen to detect immediate effects on sleep and the sleep EEG. The adaptation night was recorded in a different room than the experimental night and the absorber walls were mimicked by fabric. The EEG, the submental electromyogram (EMG), the electrooculogram (EOG, differential recording) and the electrocardiogram were recorded with a polygraphic amplifier [6]. Electromagnetic interference with the recording equipment had been eliminated by appropriate EMC technique. In the morning after awakening, subjective sleep variables and mood were assessed by visual analogue scales and questionnaires.

Sleep stages were visually scored for 20-s epochs according to standard criteria [12]. Non-REM-REM sleep cycles were defined as in previous experiments [2]. Power spectra of consecutive 20-s epochs (FFT routine, Hanning window, averages of five 4-s epochs) were computed for the referential derivation C3A2.

Spectral data were tested with an ANOVA for repeated measures with the between factor 'sequence' (exposure on first or second experimental night) and the within factor 'treatment' (field exposure or sham exposure). Tests were carried out with log-transformed standardized values (expressed relative to the mean value of both treatments) for each frequency bin separately. In addition, five traditional EEG frequency bands (δ, θ, α, σ, β) and the 10–11 Hz and 13.5–14 Hz bands were tested. ANOVA was also performed on the log transformed sleep variables and the subjective data, which was followed by post hoc Wilcoxon signed rank tests for paired samples.

Exposure to EMF reduced the duration of waking after sleep onset (sham 18.2 (2.9 SEM) min; field 12.1 (1.5) min; $n = 24$; $P < 0.01$). This effect was significant for the entire sleep episode as well as for the first and second 4-h intervals. Of the 24 subjects 17 showed a decrease in waking in the 8-h sleep episode. There was a sequence effect: the group that was sham exposed in the first experimental night showed a large decrease of waking (sham, 23.9 (4.7 SEM) min; field 13.4 (1.9) min; $P < 0.02$; $n = 12$) whereas in the group first exposed to the field there was no significant effect (sham, 12.6 (2.7) min; field 10.7 (2.3) min; $P = 0.31$; $n = 12$). Both the main effect ($P = 0.031$) and the sequence effect ($P = 0.046$) were confirmed by an ANOVA of log transformed data ($n = 24$). Compared with the adaptation night the group that was first sham exposed showed an increase in waking after sleep onset whereas the group that was first field exposed showed a decrease (sham, 8.3 (5.1) min; field −5.6 (2.6) min; $P = 0.02$; $n = 12$). The first adaptation nights did not differ (sham, 15.6 (1.3) min; field 16.3 (1.3) min; $P = 0.902$; $n = 12$). The subjective assessments of the duration of waking after sleep onset were similar to the objective data. In the group with sham exposure in the first night, self-rated waking showed a decreasing trend (sham, 20.0 (4.3) min; field, 10.5 (1.7) min; $P = 0.074$), whereas in the other group there was no difference (sham, 13.6 (2.1) min; field, 13.5 (3.5) min; $P = 0.75$). The objective and subjective values of waking after sleep onset were positively correlated ($r = 0.47$; $P = 0.02$; $n = 24$). Sleep latency and the sleep states did not differ between the sham and field exposure night.

This is the first report showing that exposure to EMF during sleep reduces waking after sleep onset. The result suggests that EMF exposure counteracts the mild sleep disturbance that may have been caused by the unfamiliar experimental set-up during the first experimental night.

Exposure to EMF affected the EEG in non-REM sleep. Analyzing the non-REM-REM sleep cycles separately revealed that in the first non-REM sleep episode spectral power was enhanced in the 7.25–14.25 Hz range (Fig. 1). The mean spectral ratio plot has two peaks (Fig. 1): one peak at 10–11 Hz is located within the frequency range of both alpha activity that occurs in conjunction with slow wave sleep ('alpha-delta pattern' [1]) and low-frequency sleep spindles [15]. The second peak at 13.5–14 Hz corresponds to high-frequency sleep spindles [15]. In contrast to waking after sleep onset there was no significant sequence effect. An intraindividual comparison of the spectral peak

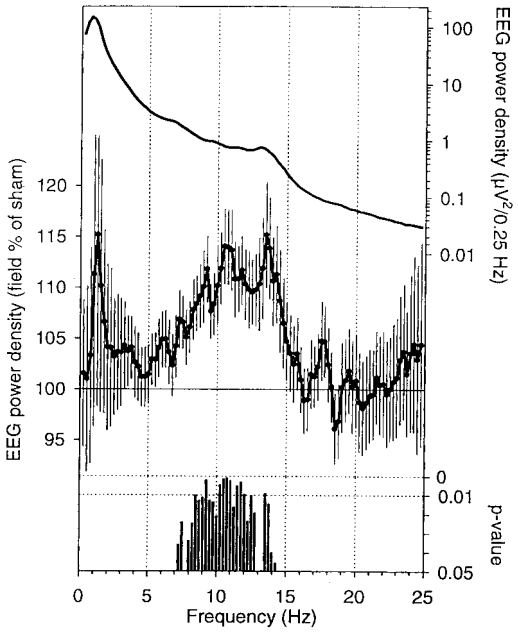


Fig. 1. Mean EEG power density spectrum ($n = 24$) in non-REM sleep (stages 2, 3 and 4) of the first non-REM sleep episode. The upper curve represents the absolute power spectrum of the sham exposure night. The lower curve depicts power in the field exposure night (mean ± SEM for 0.25-Hz bins) expressed as a percentage of the corresponding value of the sham exposure night. Bottom bars indicate frequency bins for which power in the field exposure night was significantly higher than in the sham exposure night (factor 'treatment' of two-way ANOVAs). Note that *P*-values are plotted in the reverse direction. Factor 'sequence' was significant only for a single frequency bin and no significant interaction 'treatment' × 'sequence' was observed.

position in the 10–15 Hz range in the two experimental nights revealed that the peak frequency was not affected by EMF exposure. Furthermore, testing power in five commonly used frequency bands revealed a significant increase in the α (8–12 Hz; $P < 0.005$) and σ band (12–15 Hz; $P < 0.05$). The REM sleep spectrum was not altered by exposure to EMF.

Fig. 2 depicts the time course of spectral power in the two frequency bands exhibiting the largest changes. Power in the 10–11 Hz band shows a rectangular pattern with high values in non-REM sleep and low values in REM sleep and a typical global declining trend (Fig. 2a) [2]. The effect of EMF exposure becomes evident after the initial peak in the first non-REM sleep episode when the two curves separate. The mean ratios per episode (Fig. 2c) show that the significant enhancement of power was limited to the first non-REM sleep episode. Power in the 13.5–14 Hz band exhibits a different pattern (Fig. 2b). It is characterized by peaks at the beginning and at the end of non-REM sleep episodes and by a rising trend during the night (Fig. 2b) [2]. While the increase of power in the exposure night was largest in the first non-REM sleep episode, the decline occurred gradually and a significant effect was still present in the third episode (Fig. 2d). A 15% increase in non-REM sleep power in the 13.5–14 Hz band was present already during the first 30-min interval after lights off, i.e. after 15 min of EMF exposure (data not shown; $P < 0.02$; $n = 22$ due to minimum criterion of 200 s non-REM sleep).

Power in this frequency band (reflecting the presence of 'fast spindles') is known to be readily affected by a variety of pharmacological [3,9] and physiological factors [4,5]. Its increase subsided more gradually than the rise in the 10–11 Hz band which was limited to the first non-REM sleep episode (Fig. 2). This suggests that EMF has a differential effect on brain circuits which generate or modulate different parts of the sleep EEG. A modulation by the 15-min on – 15-min off cycles of EMF was detected neither for waking after sleep onset nor for spectral power.

In the present setup, exposure to EMF as defined by the specific absorption rate (SAR) was in the range of the exposure during the use of mobile phones [8]. It is unlikely that the changes of sleep and the sleep EEG were due to thermal effects on the brain. Taking into account blood flow and conduction, the rise in brain temperature does not exceed 0.01–0.1°C [13].

Even though the processes underlying the present effects remain obscure, some information on their time course can be derived. The occurrence of EEG changes within the first non-REM sleep episode shows that an exposure time of 15–30 min is effective. For the enhancement of power in the frequency range of fast spindles a 15-min exposure is sufficient. The decline of the EEG changes in the course of sleep

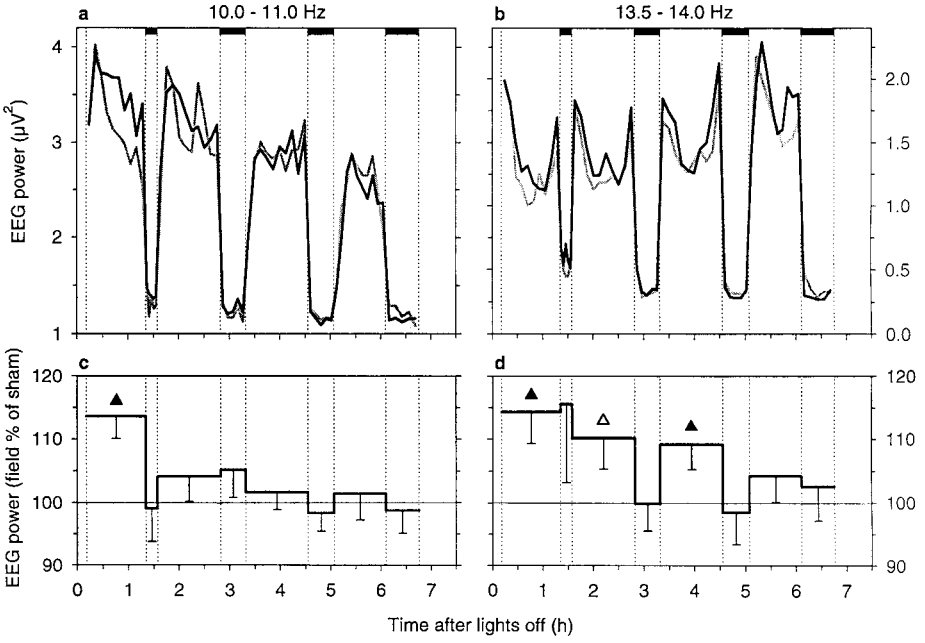


Fig. 2. Time course of EEG power in the 10.125–11.125 Hz (a,c) and the 13.375–13.875 Hz band (b,d) in the first four non-REM-REM sleep cycles ($n = 24$). (a,b) Power in the field exposure (black line) and sham exposure (gray line) night. The duration of non-REM and REM sleep episodes did not differ significantly between the two conditions. Individual non-REM sleep episodes were subdivided into ten and REM sleep episodes into five equal time intervals prior to averaging across subjects [9]. Data are plotted with respect to the average timing of both conditions. Horizontal black bars indicate REM sleep episodes. (c,d) Mean EEG power per non-REM and REM sleep episodes in the exposure night expressed as a percentage of the corresponding value in the sham exposure night (vertical bars represent 1 SEM). Filled triangles indicate significantly higher power ($P < 0.05$, factor 'treatment' of two-way ANOVAs) in the field exposure night compared with the sham exposure night and the open triangle a trend ($P < 0.1$). Factor 'sequence' and the interaction 'treatment' × 'sequence' were not significant. Frequency ranges indicated above panels were rounded to the nearest 0.5-Hz value.

points to an adaptation mechanism. The absence of a modulation of the EEG by the EMF on-off cycle suggests that field exposure triggers a chain of events rather than exerting a direct and immediate effect on sleep control. This is the first study showing that a short exposure to EMF in the range of mobile phones has an effect on the brain. Further studies are needed to examine the effects of a repeated field exposure, to investigate dose-response relations and to specify the frequency parameters of the signal that are critical for the biological effect.

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