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Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG

R. HUBER[1], V. TREYER[2], A. A. BORBÉLY[1], J. SCHUDERER[3], J. M. GOTTSELIG[1], H.-P. LANDOLT[1], E. WERTH[1], T. BERTHOLD[2], N. KUSTER[3], A. BUCK[2] and P. ACHERMANN[1]

[1]Institute of Pharmacology and Toxicology, University of Zurich, Zurich, Switzerland, [2]PET Center, Division of Nuclear Medicine, University Hospital, Zurich, Switzerland and [3]Foundation for Research on Information Technologies in Society (IT'IS), Zurich, Switzerland

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SUMMARY Usage of mobile phones is rapidly increasing, but there is limited data on the possible effects of electromagnetic field (EMF) exposure on brain physiology. We investigated the effect of EMF vs. sham control exposure on waking regional cerebral blood flow (rCBF) and on waking and sleep electroencephalogram (EEG) in humans. In Experiment 1, positron emission tomography (PET) scans were taken after unilateral head exposure to 30-min pulse-modulated 900 MHz electromagnetic field (pm-EMF). In Experiment 2, night-time sleep was polysomnographically recorded after EMF exposure. Pulse-modulated EMF exposure increased relative rCBF in the dorsolateral prefrontal cortex ipsilateral to exposure. Also, pm-EMF exposure enhanced EEG power in the alpha frequency range prior to sleep onset and in the spindle frequency range during stage 2 sleep. Exposure to EMF without pulse modulation did not enhance power in the waking or sleep EEG. We previously observed EMF effects on the sleep EEG (A. A. Borbély, R. Huber, T. Graf, B. Fuchs, E. Gallmann and P. Achermann. *Neurosci. Lett.*, 1999, 275: 207–210; R. Huber, T. Graf, K. A. Cote, L. Wittmann, E. Gallmann, D. Matter, J. Schuderer, N. Kuster, A. A. Borbély, and P. Achermann. *Neuroreport*, 2000, 11: 3321–3325), but the basis for these effects was unknown. The present results show for the first time that (1) pm-EMF alters waking rCBF and (2) pulse modulation of EMF is necessary to induce waking and sleep EEG changes. Pulse-modulated EMF exposure may provide a new, non-invasive method for modifying brain function for experimental, diagnostic and therapeutic purposes.

KEYWORDS sleep, cellular phone, positron emission tomography, electroencephalogram, electromagnetic fields, modulation

INTRODUCTION

The number of mobile telecommunication devices and duration of usage are increasing at a rapid rate. For the year 2005, 1.6 billion mobile phone users are predicted (WHO 2000). Consequently, a rising number of persons are exposed to electromagnetic fields (EMF) in the radio-frequency range. Because mobile phones are used in close proximity to the head, EMF emitted by mobile phones result in considerably higher brain tissue exposures than other EMF sources in the radio frequency band (Kuster *et al.* 1997). In contrast to these other sources, the signals of the most advanced telecommunication systems such as global system for mobile communication (GSM) and universal mobile telecommunications system (UMTS) include extremely low frequency (ELF) amplitude modulation or pulse modulation components. Given the immense number of mobile phone users, even small adverse health effects could have major public health implications (WHO 2000). A recent governmental report of the UK advises exercising caution in using mobile phones, because there is a lack of scientific data on their possible health effects (IEGMP 2000). To prevent potentially damaging thermal effects of high

Correspondence: Peter Achermann, PhD, Institute of Pharmacology and Toxicology, University of Zürich, Winterthurerstrasse 190, CH-8057 Zürich, Swtizerland. Tel.: +411 635 59 54; fax: +411 635 57 07; e-mail: acherman@pharma.unizh.ch

frequency EMF, international exposure limits have been established (ICNIRP 1998). Non-thermal effects are still a matter of debate.

In two previous studies we demonstrated that exposure to pulse-modulated-EMF (pm-EMF) during night-time sleep (Borbély *et al.* 1999) and prior to daytime sleep (Huber *et al.* 2000) affected the sleep EEG. Here, we addressed two new questions: (1) Does exposure to EMF modify regional cerebral blood flow (rCBF)?, (2) Is pulse modulation of the signal critical for the EEG effect?

METHODS

Subjects

Two groups of 16 healthy young male right-handed subjects (age range 20–25 years; mean age in positron emission tomography (PET) study 22.5 years, in sleep study 22.3 years) participated in the study. Written informed consent was obtained prior to the experiment. The local ethical committee for research on human subjects approved the study protocols. Subjects were instructed to abstain from caffeine, alcohol and medication, and to maintain their regular sleep–wake schedule (23:00–07:00 h) on the day prior to the experiment in the PET study, and during the 3 days prior to the experiment in the sleep study. Compliance was verified by activity monitors worn on the wrist. No mobile phone calls were allowed on the day of the experiment. The subjects participating in the sleep study reported no sleep complaints and were in good health. A screening night served to exclude subjects with sleep apnea, nocturnal myoclonus and low sleep efficiency.

Exposure to EMF

We report here on one type of EMF exposure in the PET study and two types in the sleep study with a common carrier frequency of 900 MHz. In both studies a pm-EMF, a 'handset-like' signal, approximating the spectral content emitted by GSM mobile phones (1 of eight slots active) was used and a sham exposure served as a control. In the sleep study, a non-modulated continuous-wave signal (cw-EMF) was applied as the second type of exposure. A 'base-station-like' signal approximating the signal emitted by a GSM base station (seven of eight slots of the basic frame active) was applied in our previous sleep studies (Borbély *et al.* 1999; Huber *et al.* 2000). The two pulse-modulated signals include the same ELF modulation components (2, 8, 217, 1736 Hz and the corresponding harmonics), but the spectral power of these components is considerably higher in the 'handset-like' signal.

During exposure the subjects sat on a chair with their heads positioned between two plates to ensure a well-defined location with respect to two planar antennas (Huber *et al.* 2000). In all conditions, the spatial peak specific absorption rate (SAR) averaged over 10 g was 1 W kg^{-1}. To control exposure conditions a detailed dosimetry was performed (Huber *et al.* 2000).

In both experiments, subjects and experimenters were unaware of the exposure condition and intervals between exposures were at least 1 week. A crossover design was used. For technical and logistical reasons, only 13 subjects completed the PET study and the order of conditions was not perfectly balanced. The factor order was included in our statistics, and there were no significant order effects or interactions involving order. In the EEG study, the design was double blind and the order of conditions was balanced.

PET data acquisition

The PET scanning was started 10 min after the end of the 30-min exposure to EMF of the left side of the head, on a whole-body scanner (Advance GE Medical Systems, Waukesha, WI, USA) in 3D mode. EMF exposure was scheduled between 08:00 and 14:00 h. During scanning the subjects were instructed to slowly count silently from one to 60 to ensure similar cognitive activity during all conditions. The three scans were performed at intervals of 10 min. For each scan, 300–350 mBq $H_2^{15}O$ was administered as a slow bolus using a remotely controlled injection device. The PET counts were recorded over 60 s after the arrival of the bolus in the brain. A 10-min transmission scan was performed at the end to correct for photon attenuation. Data were reconstructed into 35 image planes (slice thickness, 4.25 mm; matrix, 128 × 128; pixel size, 2.34 mm). The accumulated radioactivity counts over 60 s were taken as a measure for cerebral blood flow. Statistical parametric mapping (SPM) was performed as follows: First, head movements within and between data acquisition sessions (three scans per session, two separate sessions) were corrected using the least squares method implemented in the software SPM99 (Friston *et al.* 1995). Then the scans were spatially normalized into stereotactic space [Montreal Neurological Institute (MNI) coordinates as provided by SPM99] and smoothed with a 15-mm Gaussian filter to ameliorate residual interindividual anatomical and functional differences after spatial normalization. Statistical analysis was performed with pooled scans (three scans per condition) on globally normalized data. The difference between the conditions (pm-EMF minus sham) was evaluated voxel by voxel in a PET multisubject design. The results were corrected for multiple comparisons with a threshold of $P < 0.05$.

The region-of-interest analyses on the spatially and globally normalized images of each subject were performed with the medical image quantitation and kinetic modelling software PMOD (www.pmod.com) (Mikolajczyk *et al.* 1998). To outline the regions the T-map from the comparison pm-EMF minus sham was used. The regions-of-interest encompassed the activation cluster obtained from SPM at a cut-off level of $P = 0.05$ (corrected). Corresponding regions were placed on the contralateral side.

Polygraphic recordings

The experiments were performed in the sleep laboratory of the Institute of Pharmacology and Toxicology, University of

Zurich. Prior to the sleep recording (at approximately 22:20 h), the left side of the head was exposed to EMF for 30 min. The time between exposure and lights-off (approximately 23:00 h) was 10 min. Each of the three experimental nights was preceded by an adaptation night. During the 8-h night-time sleep episode EEG (C3A2 and C4A1 derivations), EOG (differential recording) and submental EMG recordings were performed. Sleep stages were visually scored for 20-s epochs according to standard criteria (Rechtschaffen and Kales 1968). For the two EEG derivations power spectra of consecutive 20-s epochs (FFT routine, Hanning window, averages of five 4-s epochs) were computed (Borbély *et al.* 1999; Huber *et al.* 2000). Visual and semiautomatic artifact removal were performed (Huber *et al.* 2000). For detection and further analysis of sleep spindles the EEG data were band-pass filtered between 12 and 15 Hz. The mean amplitude of the signal was then computed by calculating the standard deviation of consecutive 0.5-s epochs. A moving average over 80 0.5-s intervals of this new time series served as a threshold for sleep spindle detection.

RESULTS

In Experiment 1, the left side of the head was exposed to pm-EMF or sham-exposed for 30 min. Then rCBF was measured in three consecutive PET scans. Compared with the sham condition, pm-EMF exposure increased relative rCBF in the dorsolateral prefrontal cortex of the left hemisphere (Fig. 1). Three regions exhibited a significant rise only on the side of prior EMF exposure. A region-of-interest analysis revealed a hemispheric asymmetry that was significant for area B and showed a tendency for area A (Fig. 1).

In Experiment 2, the left side of the head was exposed to EMF for 30 min prior to a night-time sleep episode. We used a double-blind crossover design with three exposure conditions: (1) EMF with pulse modulation (pm-EMF), (2) EMF without pulse modulation (i.e. only with the carrier frequency, designated as cw-EMF and (3) sham control.

In the sham condition, subjects had a sleep latency of about 13 min which was not significantly affected by EMF exposure (Table 1). Spectral analysis of the EEG prior to sleep onset revealed that power in the alpha frequency range was increased in the pm-EMF conditions in comparison with the sham condition (Fig. 2a). This effect was not present for the cw-EMF condition (Fig. 2b).

The sleep EEG was also modified after pm-EMF exposure. In sleep stage 2, power in the 12.25–13.5 Hz range was increased relative to sham exposure (Fig. 2c). Power in this frequency range reflects sleep spindles, oscillatory EEG events that have a characteristic spindle shape and are abundant in stage 2 sleep (Dijk *et al.* 1993). The enhancement in this frequency range was not present after cw-EMF exposure (Fig. 2d). In fact, there tended to be a decrease, which was statistically significant in a single, slightly higher frequency bin (Fig. 2d).

Power in the frequency range of sleep spindles showed the typical increasing trend in the course of the night (Aeschbach and Borbély 1993; Dijk *et al.* 1993) (Fig. 3a). The well-known U-shaped pattern was apparent in the first four non-rapid eye movement (NREM) sleep episodes with peak values after sleep onset and before and after rapid eye movement (REM) sleep episodes. The enhancement of power in the spindle frequency range by pm-EMF exposure paralleled the general increasing trend of spindle frequency activity and was largest in the fourth and fifth NREM sleep episodes (Fig. 3b). The detailed representation of the time course (Fig. 3a) revealed that the differences between pm-EMF and cw-EMF were most pronounced near the peak values.

To ascertain whether observed changes were due to EMF exposure or incidental differences in sleep during the nights prior to exposure, stage 2 spectra were computed for the three adaptation nights preceding the experimental nights. Statistical analyses revealed no differences among the adaptation nights.

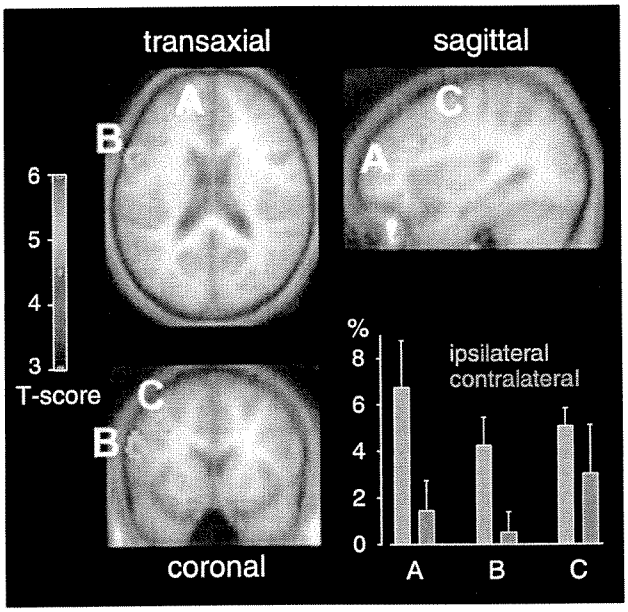

Figure 1. Increased regional cerebral blood flow after electromagnetic field (EMF) exposure. Regions showing higher relative regional cerebral blood flow (rCBF) after pulse modulated (pm) EMF exposure compared with sham exposure assessed by statistical parametric mapping ($n = 13$; sagittal, $x = -34$; coronal, $y = 12$; transaxial, $z = 20$; MNI coordinates in mm). The displayed results are corrected for multiple comparisons (region A: T = 5.40, $P_{corr} = 0.006$ at $x = -30$; $y = 32$ $z = 20$; B: T = 5.60, $P_{corr} = 0.003$ at $x = -56$; $y = 10$; $z = 20$; C: T = 5.78, $P_{corr} = 0.002$ at $x = -38$; $y = 8$; $z = 36$). In the bar graph the increase of rCBF following exposure relative to sham in the regions A, B and C is displayed on the exposed (left) side and on the homotopic areas on the right side. A significant increase of rCBF was observed in all three regions on the ipsilateral side ($P < 0.01$, two-tailed paired t-test) but not on the contralateral side. Region B showed a significant hemispheric difference ($P = 0.02$) and a trend was observed in region A ($P = 0.06$, paired t-tests after three-way ANOVA with factors 'order', 'region', 'hemisphere' revealed significance for factor 'hemisphere'). A small circumscribed reduction in relative rCBF was observed in the right parietal lobe (T = 5.01, $P_{corr} = 0.022$ at $x = 10$; $y = -60$; $z = 28$).

	Sham	*pm-EMF*	*cw-EMF*
Time in bed	480.0	480.0	480.0
Total sleep time	446.4 (3.0)	445.9 (3.5)	441.9 (3.9)
Sleep latency	12.9 (2.0)	12.8 (1.8)	15.4 (3.4)
REM sleep latency	71.4 (4.8)	70.7 (4.4)	75.3 (6.4)
Waking after sleep onset	7.5 (1.6)	8.4 (2.7)	8.5 (1.9)
Stage 2	224.2 (6.1)	222.0 (7.9)	223.8 (7.1)
Slow-wave sleep	80.0 (4.4)	86.8 (5.4)	80.5 (5.3)
REM sleep	110.0 (4.6)	105.5 (3.8)	105.2 (4.1)
Movement time	13.2 (0.9)	12.9 (0.8)	14.2 (0.9)

Table 1 Sleep variables after pulse-modulated (pm) and continuous-wave electromagnetic field (cw-EMF) exposure

Sleep variables based on visual scoring for the three experimental conditions sham, pulse modulation (pm) and continuous-wave (cw) EMF exposure. All night mean values in minutes (SEM in parenthesis; $n = 16$). Sleep latency: Interval from lights off to stage 2 sleep. REM sleep latency: Interval from sleep onset (stage 2) to the first occurrence of REM sleep. Slow-wave sleep: NREM sleep stages 3 and 4. Two-way ANOVA for repeated measures (between factor 'order', within factor 'condition' and interaction 'order*condition') revealed no significant differences.

Sleep spindles give rise to a distinct peak in the absolute power spectrum of the sleep EEG (Dijk *et al.* 1993). As determined for individual subjects, the peak frequency in the spindle range did not differ among the three experimental conditions. Furthermore, the effects of EMF exposure on single sleep spindles were analysed. Spindle amplitude was increased in the pm-EMF condition compared with the cw-EMF and sham conditions (5.4 ± 1.4 and 2.9 ± 1.5% increase, respectively; $P < 0.05$, two-tailed paired *t*-test). The duration and number of sleep spindles did not differ.

Although the NREM sleep EEG was modified, the REM sleep EEG was not affected and no differences were seen in sleep architecture, REM sleep latency (Table 1), or the duration of NREM–REM sleep cycles.

DISCUSSION

The PET results demonstrate for the first time that exposure to pm-EMF, as used in mobile phones, affects rCBF in the dorsolateral prefrontal cortex of the exposed hemisphere. This region plays a major role in working memory (Dade *et al.* 2001; Rypma *et al.* 1999; Wagner *et al.* 2001). Recent behavioral studies have shown changes in performance on working memory tasks when subjects were exposed to EMF emitted by GSM phones (Jech *et al.* 2001; Koivisto *et al.* 2000a,b). Therefore, EMF-induced changes in the activity of dorsolateral prefrontal cortex may underlie EMF effects on working memory. On the other hand, an interaction between the counting task and EMF exposure cannot be excluded due to the lack of a performance measure of the counting task. There is no reason to expect any systematic difference in subjects' counting from one condition to another, unless EMF exposure influences counting ability. If so, the regions of activation reflect the interaction between EMF exposure and counting rather than a main effect of EMF exposure. Nevertheless, the changes in brain physiology that we have observed following pulse-modulated EMF exposure are likely to have functional consequences.

The EEG results corroborate and extend our previous findings (Borbély *et al.* 1999; Huber *et al.* 2000). As in the previous studies, pm-EMF altered the EEG in the high spindle frequency range in NREM sleep. In addition, pm-EMF induced increased alpha activity in the waking EEG, supporting a prior preliminary report (von Klitzing 1995). Both sleep and waking EEG changes were associated only with pm-EMF. Thus, the EEG study provides an important novel contribution by demonstrating that pulse modulation is critical for EMF-induced changes in brain physiology. This observation may provide important new insights into the mechanisms of EMF influences on brain activity.

Because the time-averaged EMF exposure did not differ between the pm and cw conditions, our results cannot be attributed to a thermal action of EMF. The ELF modulation components resulting from the GSM signal shape were at 2, 8, 217, 1736 Hz and higher harmonics. Therefore, a single frequency component or a mixture of components may be responsible for the observed effects. In future studies, varying the modulation characteristics of the signal could help to specify the critical frequencies. Future studies may also examine dose–response relationships by varying the specific absorption rate, which was held constant at 1 W kg^{-1} in our studies.

Our results indicate that pm-EMF directly or indirectly modulates brain oscillations. The postexposure changes of rCBF in the cortex suggest altered cortical neuronal activity, which may in turn modify cortico-thalamo-cortical loops known to be involved in generating sleep spindles (Contreras *et al.* 1996; Steriade *et al.* 1993). A similar mechanism might account for enhancement of alpha activity in waking, as alpha-generating mechanisms seem to involve both thalamic and non-thalamic sources (Lopes da Silva 1991). Investigations of the mechanisms of cellular effects may provide additional understanding of macroscopic influences on the brain.

A striking aspect of the present experiment was the extended duration of pm-EMF-induced changes in the sleep EEG. Surprisingly, the changes appeared to increase in the course of the night. The EMF exposure that gave rise to the initial postexposure modification of cortical rCBF and to the increase in presleep alpha activity may have induced long-term effects on spindle generating mechanisms that became more apparent

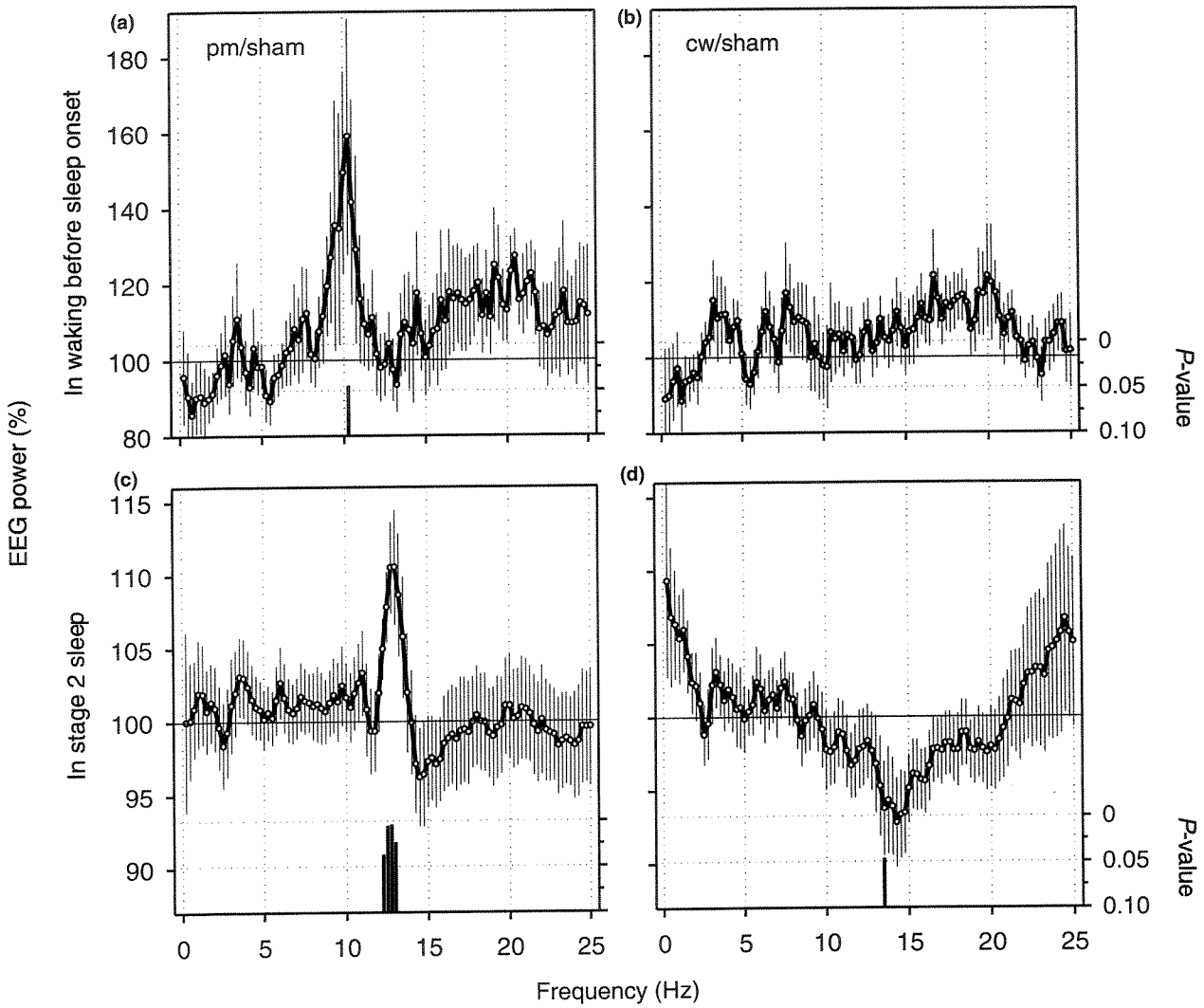

Figure 2. Changes in waking and sleep electroencephalogram (EEG) after electromagnetic field (EMF) exposure. Mean relative EEG power density spectra (C3A2 derivation) in waking before sleep onset (a, b; $n = 15$, a minimum criterion of 1 min of artifact free data was required, mean duration over all conditions was 9 ± 1 min), and in stage 2 sleep of the entire 8-h sleep episode (c, d; $n = 16$). The curves represent power after exposure to pulse-modulated (pm) EMF and continuous-wave (cw) EMF expressed as a percentage of the corresponding value after sham exposure (mean ± SEM for 0.25 Hz bins). Bottom bars indicate frequency bins for which power after EMF exposure was significantly different from sham EMF exposure (paired *t*-test for frequency bins where factor 'condition' reached significance in two-way ANOVAS including the between factor 'order'). For stage 2, values were expressed relative to the mean value of all three conditions prior to statistical analysis. Note that the scale of *P*-values is from top to bottom.

as spindle frequency activity increased in the course of the night. Alternatively, EMF exposure may have affected the phase of the circadian pacemaker, thereby altering the time course of spindle frequency activity. Modulation of sleep spindles by the circadian system is well documented (Aeschbach *et al.* 1997; Wei *et al.* 1999). In contrast to the present experiment, EMF exposure did not have a long-lasting effect on the EEG in our two previous studies. The different time course, as well as slight differences in the range of EEG frequencies affected, may be attributable to differences in the pulse structure of 'base-station-like' vs. 'handset-like' signals (see Methods). The latter provide higher spectral power of the 2 and 8 Hz modulation components and four times higher peak SAR while maintaining the same time-averaged SAR.

In conclusion, we demonstrated that exposure to pm-EMF significantly affects brain physiology as assessed by measures of cerebral blood flow and brain electrical activity. Furthermore, pulse modulation was shown to be critical for EMF-induced enhancement of EEG power in specific frequency bands during waking and sleep. In view of increasing popularity of mobile phones, EMF effects on the brain merit additional study. It should be emphasized that the observed effects of pm-EMF exposure were subtle. Despite modifications of the EEG, sleep latency and the sleep stage distribution

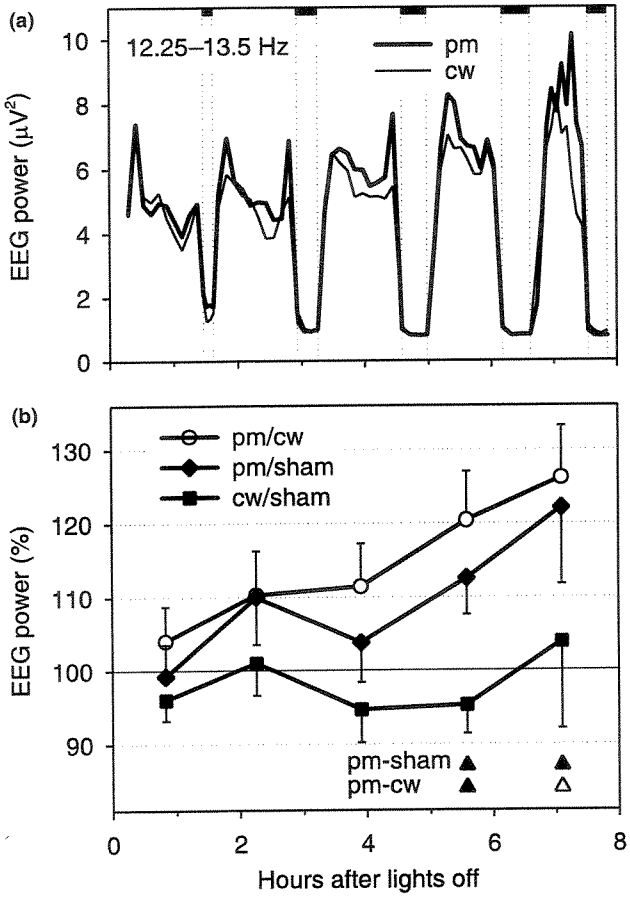

Figure 3. Long-lasting effect of electromagnetic field (EMF) exposure on the sleep electroencephalogram (EEG). Time course of EEG power (C3A2 derivation) in the 12.25–13.5 Hz band in the first five non-rapid eye movement–rapid eye movement (NREM–REM) sleep cycles. (a) Mean power after pulse-modulated (pm, thick line) and continuous-wave (cw, thin line) EMF exposure. Individual NREM sleep and REM sleep episodes were subdivided into equal intervals (NREM sleep, 10; REM sleep, 4) prior to averaging across subjects. The values are plotted with respect to the average timing. Horizontal black bars indicate REM sleep episodes. (b) Relative power (mean ± SEM) in stage 2 after EMF exposure is plotted for five consecutive NREM sleep episodes at episode mid-points. The values for pm-EMF are plotted both as a percentage of cw-EMF (open circles) and sham (solid diamonds). The values of cw-EMF (solid squares) are plotted as a percentage of sham. Statistics are indicated on bottom right for pm vs. cw and pm vs. sham (solid triangles, $P < 0.05$; open triangle, $P < 0.1$; two-tailed paired t-test, $n = 16$ except for cycle 4, $n = 15$ and cycle 5, $n = 10$). Differences were first assessed by three-way ANOVA for between factor 'order' (position of sham condition), within factor 'condition' (pm, cw, sham) and 'NREM episode', and their interactions performed on log-transformed absolute values. Factors 'condition' and 'episode' were significant ($P < 0.02$). Not all subjects had a fourth or fifth cycle.

were not affected. Based on the present results it would be premature to draw conclusions about health consequences of EMF exposure. Future studies should not only focus exclusively on possible harmful effects of EMF but also explore its potential as a non-invasive method for influencing the brain for experimental, diagnostic and therapeutic purposes.

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