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Individual Differences in the Effects of Mobile Phone Exposure on Human Sleep: Rethinking the Problem

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Mobile phone exposure-related effects on the human electroencephalogram (EEG) have been shown during both waking and sleep states, albeit with slight differences in the frequency affected. This discrepancy, combined with studies that failed to find effects, has led many to conclude that no consistent effects exist. We hypothesised that these differences might partly be due to individual variability in response, and that mobile phone emissions may in fact have large but differential effects on human brain activity. Twenty volunteers from our previous study underwent an adaptation night followed by two experimental nights in which they were randomly exposed to two conditions (Active and Sham), followed by a full-night sleep episode. The EEG spectral power was increased in the sleep spindle frequency range in the first 30 min of non-rapid eye movement (non-REM) sleep following Active exposure. This increase was more prominent in the participants that showed an increase in the original study. These results confirm previous findings of mobile phone-like emissions affecting the EEG during non-REM sleep. Importantly, this low-level effect was also shown to be sensitive to individual variability. Furthermore, this indicates that previous negative results are not strong evidence for a lack of an effect and, given the far-reaching implications of mobile phone research, we may need to rethink the interpretation of results and the manner in which research is conducted in this field. Bioelectromagnetics 33:86–93, 2012. © 2011 Wiley Periodicals, Inc.

Key words: radiofrequency electromagnetic fields; electroencephalogram (EEG); non-REM sleep; individual differences; spectral analysis

INTRODUCTION

Mobile phone use has become a universal part of everyday life and recent figures show that there are now over five billion users of the Global System for Mobile Communications (GSM) network worldwide [GSMWorld, 2010]. This widespread use, and the fact that the radiofrequency (RF) electromagnetic fields (EMF) emitted by mobile phones are partly absorbed by the human head, has led to an increasing demand for scientific research on potential health effects related to this. Numerous studies have been conducted over the last 20 years addressing this question, with endpoints ranging from effects on brain electrical activity, cognition, and sleep, to more subjective endpoints such as personal well-being [van Rongen et al., 2009; Valentini et al., 2010]. However, despite this abundance of research, results appear contradictory, which has led to widespread debate regarding whether mobile phones have effects on human physiology or health.

Sleep disturbances and fatigue are among the most commonly reported health complaints attributed to mobile phones [Röösli et al., 2004; Hillert et al.,

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2008]. Research regarding the effects of mobile phone-like emissions on the sleep electroencephalogram (EEG) has arguably been the most consistent to date, with a number of studies reporting increases in EEG spectral power in the alpha and spindle frequency ranges during non-rapid eye movement (non-REM) sleep [Borbély et al., 1999; Huber et al., 2000, 2002; Loughran et al., 2005; Regel et al., 2007]. However, overall conclusions regarding the presence of an effect remain difficult due to a similar number of studies failing to find any effects on the EEG during sleep [Mann et al., 1998; Wagner et al., 1998, 2000; Hinrichs et al., 2005; Fritzer et al., 2007].

There are a number of potential reasons why previous findings are inconsistent. Large differences in exposure parameters, such as exposure duration and specific absorption rates (SAR) of the applied signals, are important factors that could be partly responsible for the lack of consistent results [Kuster et al., 2004]. Adding to this, the majority of studies have suffered from small sample sizes and it is not clear that statistical analysis techniques have been appropriate. Arguing for this mobile phone-related sleep EEG effect being real and not artefactual, with the methodological improvements that have occurred since the first research on this issue, mobile phone-related changes to sleep EEG are now being reported more consistently [Huber et al., 2003; Loughran et al., 2005; Regel et al., 2007].

However, participant differences across (and within) studies may pose a greater difficulty for mobile phone-induced effects on the sleep EEG, and mobile phone bioeffects research more generally. Indeed, closer inspection of the data from our previous study revealed that although there was an overall increase in EEG power following RF EMF exposure, this increase was not present in all participants, suggesting that mobile phone emissions may have different effects on different individuals [Loughran et al., 2005]. If this is the case, then this could have important ramifications for not only mobile phone sleep research, but all human RF bioeffects research. That is, if there are individual differences in response to mobile phones, then previous research results could not only have been strongly influenced by the particular individuals tested, but also result in substantially reduced statistical power, and the incorrect conclusion in many studies that mobile phone emissions have no effect. Follow-up or replication studies in which the same individuals are re-tested would control for these design issues and result in methodologically and statistically stronger results.

Similarly, although the reported mobile phone-related changes to sleep EEG have not induced changes in overall sleep quality, it remains to be seen whether these changes are also dependent on the individual, and if so, whether sleep quality is affected in these so-called 'responsive' individuals. Thus, the present study aimed to not only replicate our previous results of an enhancement of EEG power in the 11.5–12.25 Hz frequency range, but also to determine for the first time whether mobile phone emissions have different effects on both the sleep EEG and sleep quality of different individuals by re-testing a subset of participants from our previous study [Loughran et al., 2005].

MATERIALS AND METHODS

Participants

Twenty healthy volunteers (7 males, 13 females) aged between 20 and 51 years (mean = 27.9, SD = 6.5) who were participants in our previous study [Loughran et al., 2005] consented to be re-tested in the current follow-up study. This represents the volunteers from the previous study, which was comprised of 50 participants (27 males, 23 females), who were able to be contacted and were willing to participate again. All participants were mobile phone users, with 90% reporting daily usage. Participants were instructed to maintain a regular sleep–wake schedule in line with their scheduled experimental sleep times in the week prior to participation, and were also required to abstain from caffeine, alcohol and the use of mobile phones on the adaptation and experimental nights. Compliance was verified with a sleep diary and self-report questionnaires. No participant reported to be suffering from any sleep complaints, neurological or psychological disorders, and polysomnographic recordings from the adaptation night confirmed that none of the participants suffered from sleep apnoea or other polysomnographically measured sleep-related problems. The Human Research Ethics Committees of Swinburne University of Technology (Melbourne, Australia) and the Austin Hospital (Melbourne, Australia) approved the study protocols, and written informed consent was obtained from all volunteers prior to participation.

Procedure

A double-blind, counterbalanced, crossover design was employed in which participants spent three consecutive nights (approximately 10:00 pm–

6:00 am, including exposure) in the sleep laboratory. The first night was an adaptation night designed to help participants acclimate to the laboratory conditions and also to rule out the presence of respiratory-related sleep disorders. The following two nights served as the experimental nights in which participants were randomly exposed to the two exposure conditions (Active and Sham). Lights-off and lights-on occurred at the same time for each participant on all three nights spent in the laboratory.

At the beginning of each night, participants were required to fill out a demographics questionnaire and the Karolinska Sleepiness Scale (KSS) [Akerstedt and Gillberg, 1990], which was also completed each morning upon awakening. In addition, each participant completed a sleep diary for one week, which included the four nights prior to their participation and the three nights spent in the laboratory.

On the experimental nights the participants sat comfortably in a chair and were randomly exposed for 30 min to either the Active or Sham exposure prior to a full night-time sleep episode. Participants sat still and relaxed, and were constantly monitored by the experimenter to ensure that they did not fall asleep while undergoing exposure. During sleep, EEG (C3-A2 and C4-A1 derivations), electrocardiogram, electrooculogram, submental electromyogram and SaO2 (arterial oxygen saturation) were monitored, along with nasal pressure, thoracic respiration, abdominal respiration and leg movements on the adaptation night (for exclusion measures only), using the Compumedics S-series polysomnography system (Compumedics, Abbotsford, Victoria, Australia). Electrodes were attached following exposure, which ensures that there is no possible interference in the EEG of the RF EMF emitted from the mobile phone. There was approximately 20 min between the end of exposure and lights-off. The EEG signals were sampled at 250 Hz, high-pass filtered at 0.3 Hz and low-pass filtered at 30 Hz. All EEG electrode impedances were below 5 KΩ at the start of each recording.

Exposure Set-Up

The exposure was generated using a modified Nokia 6110 GSM handset (Nokia, Espoo, Finland) that was set via a laptop and manufacturer software to continuously transmit at a peak power of 2 W, resulting in a mean power output of 0.25 W and, therefore, not including the lower frequency components associated with discontinuous transmission (DTX) and adaptive power control (APC). The signal emitted by the antenna was an 894.6 MHz RF field pulsed at a frequency of 217 Hz with a duty cycle of 0.125, resulting in a pulse width of 576 ms (26th frame not idle).

A detailed dosimetric analysis of the exposure configuration was performed inside a Specific Anthropomorphic Mannequin (SAM) phantom using the precision RF near-field Dosimetric Assessment System (DASY4; Schmid & Partner Engineering AG (SPEAG), Zurich, Switzerland). The SAR of the exposed hemisphere averaged over 10 g was 0.11 W/kg, and the resulting maximum peak spatial SAR averaged over 10 g was determined to be 0.674 W/kg (touch position) [Loughran et al., 2005; Hamblin et al., 2007]. Further modelling to compute the distribution of the SAR revealed a very localised exposure of the upper cheek and inner ear regions, which was concentrated on a limited area of the middle temporal gyrus just above the ear (for full details, see Boutry et al. [2008]).

On the experimental nights participants sat comfortably and were fitted with an adjustable head cradle to which the mobile phone was attached. The phone was positioned over the right temporal region and aligned toward the corner of the mouth, comparable to normal use.

The audio circuits of the phone were disconnected and padding was placed between the handset and its cover to ensure that both the researcher and participants were not given acoustic cues revealing the operational status of the phone. The padding also served to eliminate any heat being felt by the participant that may have been generated from extended battery operation. Additionally, an earplug was placed in the right ear of the participant in order to mask any residual sound from the handset's operation. Participants were asked at the end of each exposure whether they were able to perceive a field but none reported being able to do so.

Data Analysis

Sleep stages were visually scored by an experienced sleep technician for each 30-s epoch according to the standard criteria of Rechtschaffen and Kales [1968]. The sleep technician was unaware of the experimental conditions. Sleep onset was defined as the first occurrence of stage 2 sleep. Using Neuroscan Edit software (Compumedics), the C3-A2 and C4-A1 EEG derivations were first averaged together and analysed to provide power spectral density estimates for each consecutive epoch (fast Fourier transform routine, Hanning window, averages over 4-s epochs) for the first 30 min of each participant's initial non-REM sleep period. Artefact removal was performed by visual inspection and only artefact-free epochs were used for further analysis.

The sleep scoring of the polysomnographic recordings also resulted in conventional sleep parameters that were used for exploratory analysis.

Statistical Analyses

Participants were divided into two groups based on the results of our previous experiment [Loughran et al., 2005]: an 'Increasers' group and a 'Decreasers' group. Increasers were defined as those participants who had shown an increase in spectral power in the 11.5–12.25 Hz frequency range during non-REM sleep, and Decreasers as those who showed a decrease in spectral power in the 11.5–12.25 Hz frequency range during non-REM sleep.

Hypothesis driven. As all previous effects on the sleep EEG have been an increase in spectral power [Borbély et al., 1999; Huber et al., 2000, 2002; Loughran et al., 2005; Regel et al., 2007], a directional repeated measures *t*-test was employed to test for an overall increase in the 11.5–12.25 Hz band in the Active condition. Based on our previous results [Loughran et al., 2005], a directional independent measures *t*-test was also employed on the Active/Sham ratio to test for more of an increase in the 11.5–12.25 Hz band in the Increasers than the Decreasers. Note that the Active/Sham ratio was normalised using natural logarithms.

Exploratory. Similar analyses to the hypothesis-driven set were employed to test for effects of the mobile phone on: (1) each of the 12.25–13.5 Hz and 13.5–14 Hz frequency ranges (which were affected in Huber et al. [2002] and Borbély et al. [1999], respectively, but not in our previous study [Loughran et al., 2005]); and (2) a number of other variables that have not been consistently reported to be affected in the literature (sleep latency, REM latency, sleep duration, sleep efficiency, number of arousals and KSS score for the morning after exposure). These differed only in that they were non-directional. Additionally, independent measures analyses were performed for REM latency and number of arousals because these variables were previously reported to be inconsistently affected by exposure [Borbély et al., 1999; Huber et al., 2000, 2002; Loughran et al., 2005; Regel et al., 2007]. However, rather than grouping participants according to their 11.5–12.25 Hz change, participants were grouped based on their change in REM latency and number of arousals [Loughran et al., 2005]. Note that the 12.25–13.5 Hz and 13.5–14 Hz Active/Sham ratios were normalised using natural logarithms, and sleep latency, REM latency and number of arousals were transformed using square root transformation.

For any significant results, an exploratory independent *t*-test was performed to determine whether the Active/Sham ratio score was related to gender (male, female), and a Pearson's *r* calculated to determine whether there was a significant relationship between the Active/Sham ratio and age.

RESULTS

Hypothesis Driven

Spectral analysis of the sleep EEG revealed an overall increase in power in the 11.5–12.25 Hz frequency range (Fig. 1) in the Active exposure compared to Sham, in the first 30 min of the first non-REM sleep period ($t(19) = 1.77$, $P = 0.046$). Furthermore, there was more of an increase in EEG power in the Increasers group than the Decreasers group ($t(18) = 1.89$, $P = 0.038$). Individual responses to exposure for the 11.5–12.25 Hz frequency range are shown in Figure 2.

Exploratory

An exploratory analysis was performed to investigate whether there was an effect of exposure on other frequency ranges previously reported to be affected. No significant change in power was observed between the Active and Sham exposure conditions in the 12.25–13.5 Hz and 13.5–14 Hz frequency ranges (Fig. 1), either overall ($P > 0.29$), or between Increasers and Decreasers ($P > 0.34$).

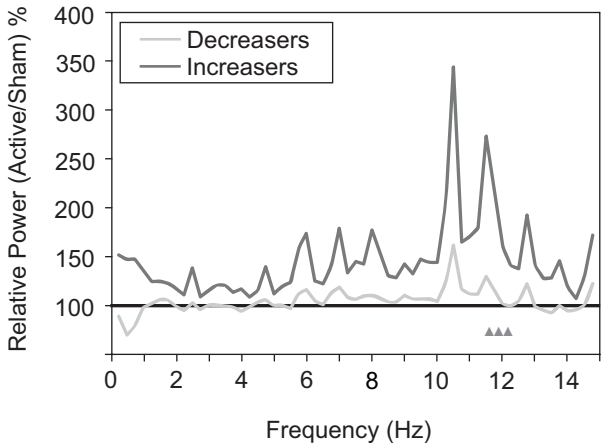

Fig. 1. Mean relative EEG power density spectrum for the first 30 min of the first non-REM sleep episode (average of C3-A2 and C4-A1 derivations; $n = 20$ participants, 8 Increasers and 12 Decreasers from Loughran et al. [2005]), expressed as a percentage of the corresponding value from the Sham condition. Significant frequency bins are indicated with grey triangles.

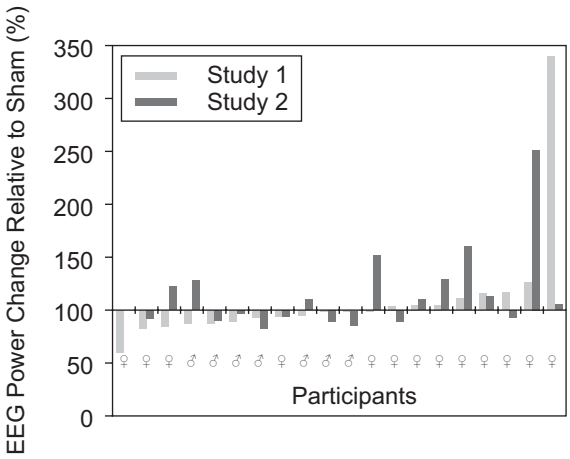

Fig. 2. Individual mean relative EEG power density change for the first 30 min of the first non-REM sleep episode (average of C3-A2 and C4-A1 derivations; $n = 20$ participants) expressed as a percentage of the corresponding value from the Sham condition in each study based on the 11.5–12.25 Hz frequency range.

A Pearson's correlation showed that the significant effect observed in the 11.5–12.25 Hz Active/Sham ratio did not correlate with age ($r(20) < 0.01$; $P = 0.970$). However, this ratio was found to be related to gender (Fig. 3), with females responding more than males overall ($t(18) = 2.28$; $P = 0.035$).

Independent samples *t*-tests on visually scored sleep variables and subjective sleepiness revealed no effect of exposure. Specifically, no significant change was observed between the Active and Sham exposure conditions for either sleep latency, REM latency, sleep duration, sleep efficiency, number of arousals or KSS score ($P > 0.18$; Table 1). Additionally, no evidence of a differential response between the Increasers and Decreasers was found for either of the previously mentioned variables ($P > 0.27$). When grouped based on the change in Loughran et al., [2005], there was also no significant change observed between exposure conditions for REM latency ($P = 0.97$) or number of arousals ($P = 0.63$).

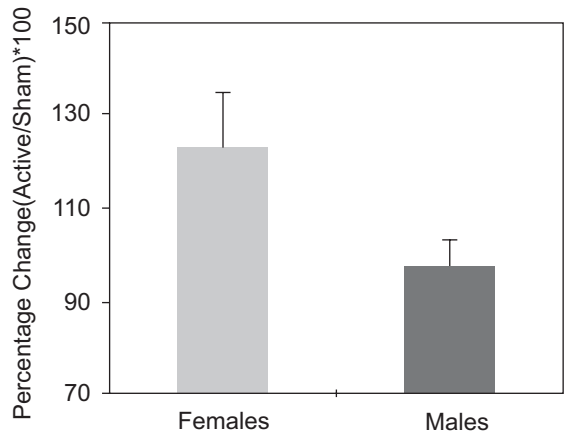

Fig. 3. Percentage EEG power density change (+SEM) for the 11.5–12.25 Hz frequency range for the first 30 min of the first non-REM sleep episode ($n = 20$ participants, 7 males). An independent samples *t*-test revealed that overall, females responded significantly more than males ($P = 0.035$).

TABLE 1. Effect of RF EMF on Visually Scored Sleep Variables

	Sham	Active	*P*-values
Total sleep time (min)	384.5 (5.5)	375.0 (6.0)	0.526
Sleep latency (min)	23.1 (3.0)	26.1 (3.1)	0.853
REM sleep latency (min)	95.7 (16.0)	106.5 (15.0)	0.416
Arousal index (per h)	11.5 (1.2)	11.0 (0.8)	0.658
Sleep efficiency (%)	88.1 (0.9)	87.9 (1.0)	0.470
KSS	5.6 (0.4)	5.4 (0.5)	0.656

Sleep variable mean values (SEM in parentheses; $n = 20$ for all variables except KSS, where $n = 19$ due to missing data) based on visual scoring of all night sleep recordings for the two exposure conditions (Active and Sham). *Sleep latency*: Interval from lights-out until onset of stage 2 sleep. *REM latency*: Interval between sleep onset and the onset of the first REM period. *Arousal index*: Number of wakings per hour. *Sleep efficiency*: Total sleep time as a percentage of total time in bed.

DISCUSSION

Employing a strong methodology, the current results support previous reports of enhanced EEG power during non-REM sleep following mobile phone exposure [Borbély et al., 1999; Huber et al., 2000, 2002; Loughran et al., 2005; Regel et al., 2007]. Consistent with our previous study, this effect was present during the first 30 min of the first non-REM sleep period and was only found in the 11.5–12.25 Hz frequency range and not the other two frequency ranges tested (12.25–13.5 Hz and 13.5–14 Hz). Furthermore, this enhancement was more prominent in those participants whose EEG power increased in our previous study. This provides strong evidence that the effects of exposure to mobile phone-type emissions are different for different people as the effect was again shown to be differential but remained similar within an individual. No significant effects on visually scored sleep variables such as sleep latency, arousals or sleep efficiency were observed.

Interestingly, although there was still an overall significant mobile phone-induced increase in EEG power, the effect was found to be more prominent in females, providing further support for individual variability in response to RF EMF exposure. Given

that previous research regarding effects on the sleep EEG has included only male participants (excluding our own previous study), this is the first time that a gender-related exposure effect has been reported in the sleep EEG, which therefore requires verification before further conclusions can be drawn.

Consistent with the majority of previous research, the small changes observed in EEG power were not related to any measurable changes in conventional sleep parameters or overall sleep architecture. Furthermore, the exploratory results from our previous study suggesting a decrease in REM sleep latency could not be verified by the current results, providing further evidence that changes in EEG power induced by mobile phone RF EMF exposure are not related to changes in overall sleep quality. This was further supported by the lack of change in subjective sleepiness on the following morning.

It should also be noted that participants generally responded in the same direction as in the first study, although there were a few participants who showed the opposite response following exposure in the current study. This adds further complexity to our knowledge of the effects of RF EMF on the EEG and may suggest that changes in the EEG spectral power are due to a non-specific response to exposure that leads to a subsequent change in brain activity. This change in brain activity may in turn be dependent on other factors, for example, particular stage of sleep or prior sleep–wake activity, providing another possible explanation for differential responses to exposure on different occasions. Furthermore, prior sleep–wake activity is known to influence the subsequent sleep EEG, and therefore subtle differences in this parameter may also represent a potential confound between the two studies. However, verification of prior sleep–wake activity, including the use of a laboratory-controlled adaptation night (as was used in the present study), would help to minimise this possibility.

The findings of the current study add to the increasing evidence that exposure to the RF EMF emitted by mobile phone handsets alters human brain activity during sleep [Mann and Roschke, 1996; Borbély et al., 1999; Huber et al., 2000, 2002; Loughran et al., 2005; Hung et al., 2007; Regel et al., 2007]. Furthermore, the observation that this effect is sensitive to individual variability provides a possible explanation for previous inconsistent results and suggests the possibility that, rather than effects being small or subtle, mobile phones may in fact have larger but differential effects on different people.

The EEG is known to show considerable variation between individuals, particularly in the range of sleep spindles [Werth et al., 1997], which is the frequency range where physiological effects of mobile phones are most commonly seen during sleep. Thus, the presence of individual differences may help to explain why previous studies have continued to find similar effects on the EEG, with results tending to differ only in terms of frequency range. This also challenges the technique of average group measures as used in previous research analyses because this does not account for the known individual variability in the EEG. This may explain why effects have slightly shifted frequency when using different samples, and could also lead to a masking of the effect if variability between participants was sufficiently high, particularly with the small sample sizes used in the majority of studies.

In relation to individual variability in EEG, both the sleep EEG and sleep architecture are known to show marked changes with age [Dijk et al., 1989; Ohayon et al., 2004]. A number of previous studies have included reasonably wide age ranges [Wagner et al., 1998, 2000; Loughran et al., 2005; Fritzer et al., 2007], which is problematic for studies with between-subjects designs [Fritzer et al., 2007]. The current study also included participants of a wide age range (20–51 years). However, the utilisation of a within-subjects design controls for individual differences in participants, meaning that unwanted variability is largely avoided and not a factor for the current results. It should also be noted that the possibility of carry-over effects from the exposure cannot be completely excluded in the current study, although again, the use of a within-subjects crossover design would largely overcome such issues.

At present, the possible mechanism behind this effect is unknown [Challis, 2005]. However, given that specific aspects of the signal, namely the pulse modulation employed by GSM mobile phone handsets, may be required to induce an effect on the sleep EEG [Huber et al., 2002] provides support for a non-thermal mechanism (as the temperature increase is the same for continuous and pulsed RF). Furthermore, it is also unclear where the site of interaction would likely be for such a non-thermal effect. However, as spindle oscillations are generated in the thalamus, subcortical regions such as the thalamus could be possible sites of interaction [Huber et al., 2003].

The significance of an enhancement of EEG spectral power in the spindle frequency range during the initial part of sleep remains unknown; however, there were no detrimental effects of mobile phone RF EMF on conventional sleep parameters or

subjective sleepiness observed in the current study or in previous studies. Therefore, it would be premature to draw any conclusions regarding health consequences related to this change in EEG spectral power, particularly as the overall quality of sleep derived from the sleep architecture variables was not changed. Given that previous studies have consistently shown alterations in the spindle frequency range following exposure, and that cognitive functioning such as memory consolidation and learning has been associated with sleep spindles and slow wave activity [Gais et al., 2002; Gais and Born, 2004; Stickgold, 2005; Walker and Stickgold, 2006], one consequence of a mobile phone-induced change in spindle frequency activity may be changes in cognitive functioning, either relating to the memory consolidation process itself or to subsequent cognition the next morning. However, the current study design does not address these functional consequences of enhanced EEG power and therefore subsequent studies are required to further explore the significance of the observed effect. In addition, these studies have only addressed short-term effects on sleep from a single exposure of RF EMF; hence, the long-term effects of repeated exposure on sleep remain unknown.

In conclusion, the present study provides further evidence that the RF EMF emitted by mobile phones affects the subsequent EEG spectral power during non-REM sleep. Importantly, this low-level effect was shown to be sensitive to individual variability, which consequently suggests that previous negative research on the EEG during sleep is not strong evidence for a lack of effects from mobile phones. Furthermore, the current results have important ramifications not only for mobile phone sleep research but all human RF bioeffects research, as it is predicated on the assumption that any effect will be relatively consistent across individuals. Given the far-reaching implications of any mobile phone-induced effects, it may be that we need to rethink the interpretation and conductance of research in this field and that we must conclude that we currently do not know if mobile phones affect health.

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