DO MOBILE PHONES AFFECT SLEEP?
Investigating Effects of Mobile Phone Exposure on Human Sleep EEG

Andrew Wood, Sarah Loughran, Rodney Croft, Con Stough
Australian Centre for Radiofrequency Bioeffects Research, Brain Sciences Institute
Swinburne University of Technology, Melbourne, Australia

Bruce Thompson
Allergy, Immunology and Respiratory Medicine, The Alfred Hospital, Melbourne, Australia

Keywords: Mobile phone radiation, sleep, EEG analysis.

Abstract: This paper will summarize the results of a human volunteer study on the effects on sleep parameters of exposure to RF emissions from a mobile phone handset for 30min prior to going to sleep. A cohort of 55 volunteers were tested over 4 nights in a double-blind design. The significant outcomes were: Rapid Eye Movement (REM) sleep latency reduced by 16%; EEG alpha power enhanced by 8% during 1st non-REM period. These results are compared for overall internal consistency and with studies from other laboratories. Part of the program of the Australian Centre for Radiofrequency Bioeffects Research extending these studies is described.

1 INTRODUCTION

The issue of whether or not mobile phone handset radiofrequency (RF) and other emissions are able to alter sleep patterns is controversial. The World Health Organisation has a RF research agenda which highlighted the need to extend and replicate earlier studies which demonstrated effects on sleep [WHO http://www.who.int/peh-emf/research/rf03/en/ index2.html]. A series of experiments have been carried out at Swinburne University in the period 1999 – 2007 involving human volunteers on a range of immediate psychological and physiological consequences of use of mobile phone handsets, including sleep. In terms of health risk assessment, alterations of sleep quality may not appear to be as severe as possible links with cancer, but in terms of society's expectations, if phone emissions are linked to any biological changes, these need to be thoroughly understood. Although the basic research question we have asked is 'do the emissions from mobile phone handsets lead to an immediate change in ability to get a good night's sleep?', we have specific hypotheses formulated on the basis of previous research. A review of literature conducted at the start of the period (Hamblin and Wood, 2002) identified EEG alpha band power increase (both in awake and sleep experiments) as being the most consistent observation. The present experiment was designed to specifically examine the 'increased alpha power' hypothesis.

2 MATERIALS AND METHODS

2.1 Exposure

A popular handset (Nokia 6110) has been used throughout the series of experiments. The manufacturer's software is used to set into GSM pulsed 'test' mode (0.25 W average) via a serial cable which is then disconnected once the setting is complete. Since the current drawn from the battery follows the GSM pulsing scheme (217 Hz, 1/8 duty cycle), there is a strong extremely low-frequency magnetic field associated with this, in addition to the RF at 914 MHz. The other house-keeping pulses (including the blank 26th frame) were absent. Since all exposures were carried out with neither the participants nor those involved in administering cognitive tests aware or the exposure status, it is necessary to have independent verification that the phone was in the correct mode at each testing session. The RF output was checked i) by holding

Wood A., Loughran S., Croft R., Stough C. and Thompson B. (2008).
DO MOBILE PHONES AFFECT SLEEP? - Investigating Effects of Mobile Phone Exposure on Human Sleep EEG.
In *Proceedings of the First International Conference on Bio-inspired Systems and Signal Processing*, pages 565-569
DOI: 10.5220/0001061505650569
Copyright © SciTePress

the handset near to a landline phone and checking for a 'buzz' ii) direct connection of the antenna feedpoint to a RF power meter iii) by measurements in SAR phantom. The first of these was performed on each occasion, the second at six-monthly intervals to check the constancy of RF output over a 3-hour period and the third was performed once to determine the appropriate Specific Absorption Rate (SAR) in the users' heads.

The peak SAR was 0.19 ± 0.03 W/kg (based on 1g average). At the relevant moment during the testing the phone was attached to a cradle in a normal position next to cheek, with the antenna approximately 2 cm from the skin. The phone was set in one of two modes: i) turned on and transmitting (active); ii) turned off (sham). In other experiments in our series we also used handsets in 'standby' mode (turned on, but not emitting RF, except in intermittent bursts every few minutes or so). In order to ensure blinding, the phone was checked for audible cues from the phone circuitry, an important requirement in a quiet sleep laboratory. This was done by asking participants to indicate whether or not they thought the phone was transmitting. In order to fully prevent participants picking up the faint 'buzz' even with the loudspeaker disabled, a plastic foam pad was placed around the phone in a pouch. This also minimised the sensation of warmth when the phone was in active mode.

The phone exposure consisted of the handset being placed next to the participants' cheek for 30 min just prior to having monitoring electrodes attached and getting into bed.

2.2 Subjects

60 subjects were recruited to the study, but five of these withdrew after provision for their participation had been made. Five more were excluded because of confirmed apnoeic event during at least one of their nights in the study. The final study sample thus comprised 50 healthy volunteers aged from 18 to 60 years (Mean =27.9 SD = 10.9 years). Subjects were recruited from advertisements in local and state newspapers, and posters located at several universities and organizations in Melbourne. In the final sample there were 27 males and 23 females, 45 of whom were right handed. No participant reported any psychological or neurological condition, serious head injury or extended periods of unconsciousness.

The study took place at a purpose-made sleep laboratory (Eastern Sleep Disorders Service, Mitcham Private Hospital, Vic), which consisted of three individual bedrooms, a central monitoring room, together with a kitchen and a bathroom..

2.3 Design

A double blind crossover design was used to collect the data i.e. both the subject and the tester were blind to the exposure condition. Participants attended the sleep laboratory on Saturday and Sunday nights on two consecutive weekends. The Saturday nights were adaptation nights, to enable participants to become accustomed to sleeping in a strange environment and with monitoring sensors attached. Full sleep monitoring data were obtained and stored for these nights. On Sunday nights participants were required to sit for 30 min prior to getting into bed with the phone in either the transmitting condition or switched off, with the opposite condition the following Sunday. During this time the participants were instructed to look at a blank wall. At the cessation of real/sham exposure electrodes and sensors were attached, a task which normally occupied 15 – 20 min.

2.4 Measures

Sleep was recorded and stages were visually scored for 30 s epochs according to standard criteria (Rechtschaffen and Kales, 1968) by an experienced independent sleep technician who was blind to the experimental conditions. During sleep, EEG (C3 and C4), ECG, EOG, EMG, SaO2 and nasal airflow were monitored along with thoracic, abdominal, and leg movements, using the Compumedics™ E-series polysomnography system. All EEG electrode impedances were below 5 kΩ initially. Data were sampled as shown in Table 1. Data was stored in records of 1 second in duration in European Data Format (EDF). This format stores data points as 2 byte binary representation and as such can be converted into continuous data records for each channel. This was then exported to Matlab™ in order to resample the data for subsequent analysis using Neuroscan data processing software.

Table 1: Sample rates for all recorded channels.

Channel	Number of samples in each data record
EEG	250*
EEG(second)	250*
EOG(L)	50
EOG(R)	50
EMG	250
ECG	50
Leg(L)	50
Leg(R)	50
SaO2	5
Airflow	25
Thoracic Respiration	25
Abdominal Respiration	25
Sound	25
CPAP	25
Oxygen	1
Total	1131

*In some records the EEG was sampled at 125 Hz because of monitoring constraints: the EDF header provided the recording-specific information.

2.5 Analysis

The sleep staging was carried out in accordance with routine procedures followed by the Eastern Sleep Disorders Service. Each 30 s epoch of sleep was assigned to a stage using the standard R & K (Rechtschaffen and Kales, 1968) classification. This analysis also provided timing markers for subsequent analysis of EEG records. Matlab was used to extract the first 6 channels of each participant's EDF file (EEG1, EEG2, EOGleft, EOGright, EMG, and ECG). The individual channel files were then converted to continuous files and opened using Matlab, where they were re-sampled (due to the original acquisition rates being different) so that all channels had the same number of points. The individual files were then recombined in Matlab as an EDF file for subsequent spectral analysis using Neuroscan software. Using the staging data, the first NREM period (the time from sleep onset, defined as the first occurrence of stage 2, until the onset of the first REM sleep period) was extracted and artefact removal was performed by visual inspection (with the experimenter blind to the exposure condition). Only artefact free epochs were used for further analysis. The first 30 minutes of each file was taken and the two EEG channels (C3, C4, referenced to linked mastoids) were extracted and spectral analysis was performed on the average of the two channels for each 20 second epoch (FFT routine, Hanning window, averages of five 4-second epochs). Data was then exported to SPSS statistical package Version 11.5 for further statistical analysis. Spectral data, with a resolution of 0.25 Hz, was thus obtained for each 20 second epoch for the first 30 minutes from first stage 2 occurrence. The spectrum for each participant (and for each night) was an average of the spectra for 3 x 30 = 90 epochs. For each individual, the averaged spectrum on the active exposure night is then divided by the spectrum for the sham night an the ratio converted to a percentage. These intra-subject ratios are then averaged over the number of subjects (n = 50) and the overall percentage (± SEM) calculated. This is shown in Fig. 1 below, along with the overall averaged spectra for active and sham exposure nights.

3 RESULTS

3.1 Sleep Parameters

Of the 10 sleep parameters measured all were non-significant, except for REM latency, which was reduced 16% by exposure ($p = 0.02$) (Loughran et al., 2005). This was contrary to previous work which found a suppression of REM sleep (Mann and Röschke, 1996) and when corrected for multiple comparisons, the level of significance is marginal.

3.2 Spectral Analysis of EEG

As outlined above, the prior hypothesis was that EEG alpha power would be increased. Spectral analysis of the sleep EEG in the first 30 minutes of the first NREM period revealed no significant effects of EMF exposure on EEG power density in the alpha frequency range (8-13Hz) as a whole. Two alpha sub-bands (11.5-12.25Hz, 13.5-14Hz) that have previously shown effects in the first NREM period of an overnight polysomnography following EMF exposure (Huber et al., 2003) were also analysed. EEG power density was found to be significantly enhanced by around 8% in the 11.5 – 12.25 Hz frequency range following EMF exposure, $F(1,48) = 5.56$, $p = 0.022$ (Figure 1). No significant enhancement was found to be present in the 13.5 – 14 Hz frequency range. Effect sizes (partial eta squared) were also calculated for the 0-25 Hz region and are shown in Figure 2. This shows a raised effect size for the 13.5 – 14 Hz sub-band which failed to reach significance.

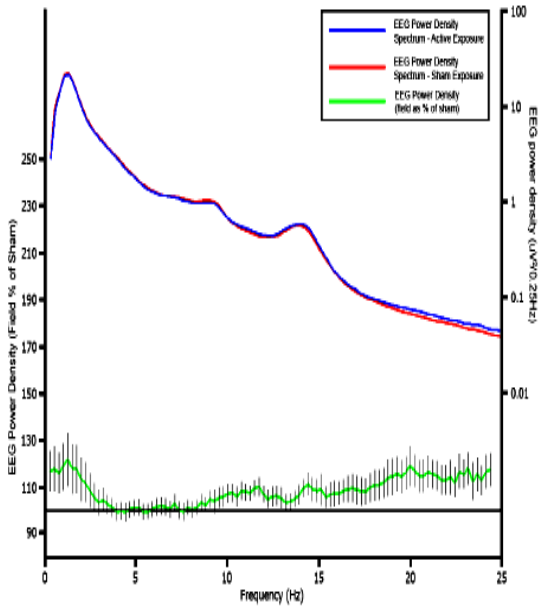

Figure 1: Upper: Averaged EEG spectra for active and sham exposure nights respectively (for 50 paricipants); Lower: Mean EEG Power Density Spectrum for real exposure as a % of sham. Bars represent Standard Error of Mean.

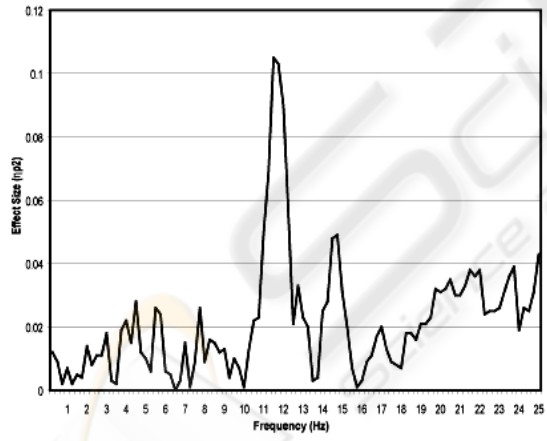

Figure 2: Effect sizes of EMF exposure on First 30 minutes of the first NREM period. Effect sizes for each 0.25 Hz bin (0 – 25 Hz) are illustrated and were calculated using the formula $\eta_p^2 = SS_{effect}/(SS_{effect} + SS_{error})$. (See Loughran et al. 2005).

It should be noted that in the region 0 – 3 Hz and 17 – 25 Hz there are enhancements of up to 20%, but as Figure 2 reveals, these are not statistically significant. Some of these data have previously been reported (Loughran et al., 2005). The averaged cross-participant spectra have a 1/f character (note log scale) with characteristic alpha and theta peaks shown. The differences in the spectra are only just distinguishable when plotted conventionally. Note that below 2 Hz the spectral estimates become unreliable.

4 DISCUSSION

Since our review paper which discusses papers published up to 2001 (Hamblin and Wood, 2002), we have continued to track the literature relating to reported EEG alpha band enhancements. Up to the end of 2006, we had noted that of the 18 papers reviewed, 9 showed data supporting alpha enhancement, 8 showed no effects, or a reduction and 1 showed both an enhancement and a reduction, based on gender. Although all reporting enhancement refer to the EEG band to be in the alpha region, further analysis shows that there is very little overlap between the actual sub-bands over which the significant changes were reported. In Figure 3 these bands are illustrated, in reverse chronological order of publication. Where multiple bands were shown to be significant in a single study these are shown as separate rows.

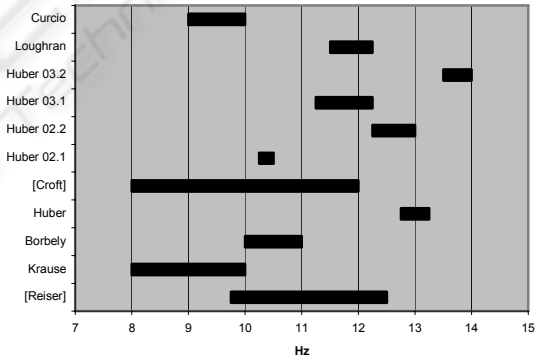

Figure 3: Frequency ranges over which increases in EEG power elicited by mobile phone radiation have been reported. In some cases, more than one sub-band was significantly enhanced. The studies are as follows: (Curcio et al., 2005, Huber et al., 2003*, Huber et al., 2002*, Croft et al., 2002, Huber et al., 2000*, Borbely et al., 1999*, Krause et al., 2000, Reiser et al., 1995. Those indicated thus (*) are during a non-REM period of sleep, the others were with awake subjects (Huber et al. 2002 showed increases with participants both awake and asleep.

5 FURTHER WORK

A repeat study is now underway in which 20 of the original cohort of 50 have repeated their participation. The aim is to discover if those who showed strong Alpha power changes in the first study show similar changes in the second. There has been some speculation that sensitivity to EMF may vary with the individual.

6 CONCLUSIONS

Alpha power findings are inconsistent across studies, but sleep studies may show slightly more consistency. The actual frequency range for significant increases varies between studies and even between studies from the same laboratory. Nevertheless, the preponderance is of reported increases in alpha power: this may relate to increased blood flow in superficial regions of the face or ear or increased tympanic membrane temperature. It is difficult however to envisage how these effects could persist several hours after exposure. Overall, the evidence is insufficiently strong to conclude that mobile phone emissions affect sleep.

ACKNOWLEDGEMENTS

Supported by the National Health and Medical Research Council of Australia, Grant No. 154905.

REFERENCES

Borbely, A. A., Huber, R., Graf, T., Fuchs, B., Gallmann, E. & Achermann, P. (1999) Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. *Neurosci Lett*, 275, 207-10.

Croft, R. J., Chandler, J. S., Burgess, A. P., Barry, R. J., Williams, J. D. & Clarke, A. R. (2002) Acute mobile phone operation affects neural function in humans. *Clin Neurophysiol*, 113, 1623-32.

Curcio, G., Ferrara, M., Moroni, F., D'inzeo, G., Bertini, M. & De Gennaro, L. (2005) Is the brain influenced by a phone call? An EEG study of resting wakefulness. *Neurosci Res*, 53, 265-70.

Hamblin, D. L., Anderson, V., Mcintosh, R. L., Mckenzie, R. J., Wood, A. W., Iskra, S. & Croft, R. J. (2007) EEG electrode caps can reduce SAR induced in the head by GSM900 mobile phones. *IEEE Trans Biomed Eng*, 54, 914-20.

Hamblin, D. L. & Wood, A. W. (2002) Effects of mobile phone emissions on human brain activity and sleep variables. *Int J Radiat Biol*, 78, 659-69.

Huber, R., Graf, T., Cote, K. A., Wittmann, L., Gallmann, E., Matter, D., Schuderer, J., Kuster, N., Borbely, A. A. & Achermann, P. (2000) Exposure to pulsed high-frequency electromagnetic field during waking affects human sleep EEG. *Neuroreport*, 11, 3321-5.

Huber, R., Schuderer, J., Graf, T., Jutz, K., Borbely, A. A., Kuster, N. & Achermann, P. (2003) Radio frequency electromagnetic field exposure in humans: Estimation of SAR distribution in the brain, effects on sleep and heart rate. *Bioelectromagnetics*, 24, 262-76.

Huber, R., Treyer, V., Borbely, A. A., Schuderer, J., Gottselig, J. M., Landolt, H. P., Werth, E., Berthold, T., Kuster, N., Buck, A. & Achermann, P. (2002) Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. *J Sleep Res*, 11, 289-95.

Krause, C. M., Sillanmaki, L., Koivisto, M., Haggqvist, A., Saarela, C., Revonsuo, A., Laine, M. & Hamalainen, H. (2000) Effects of electromagnetic field emitted by cellular phones on the EEG during a memory task. *Neuroreport*, 11, 761-4.

Loughran, S. P., Wood, A. W., Barton, J. M., Croft, R. J., Thompson, B. & Stough, C. (2005) The effect of electromagnetic fields emitted by mobile phones on human sleep. *Neuroreport*, 16, 1973-6.

Mann, K., Roschke, J. (1996) Effects of pulsed high-frequency electromagnetic fields on human sleep. *Neuropsychobiology*, 33, 41-47

Rechtschaffen, A. & Kales, A. (1968) *A manual of standardised terminology techniques and scoring system for sleep stages in human subjects*, Washington, DC, US Public Health Service.

Reiser, H., Dimpfel, W. & Schober, F. (1995) The influence of electromagnetic fields on human brain activity. *Eur J Med Res*, 1, 27-32.