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The Effect of Mobile Phone Electromagnetic Fields on the Alpha Rhythm of Human Electroencephalogram

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Mobile phones (MP) emit low-level electromagnetic fields that have been reported to affect neural function in humans; however, demonstrations of such effects have not been conclusive. The purpose of the present study was to test one of the strongest findings in the literature; that of increased ''alpha'' power in response to MP-type radiation. Healthy participants ($N = 120$) were tested using a double-blind counterbalanced crossover design, with each receiving a 30-min Active and a 30-min Sham Exposure 1 week apart, while electroencephalogram (EEG) data were recorded. Resting alpha power (8–12 Hz) was then derived as a function of time, for periods both during and following exposure. Non-parametric analyses were employed as data could not be normalized. Previous reports of an overall alpha power enhancement during the MP exposure were confirmed (relative to Sham), with this effect larger at ipsilateral than contralateral sites over posterior regions. No overall change to alpha power was observed following exposure cessation; however, there was less alpha power contralateral to the exposure source during this period (relative to ipsilateral). Employing a strong methodology, the current findings support previous research that has reported an effect of MP exposure on EEG alpha power. Bioelectromagnetics 29:1–10, 2008.

Key words: GSM900; acute exposure; EEG; alpha power

INTRODUCTION

Mobile phones (MP) operate using wireless technology, with communication typically occurring via a 400–3000 MHz signal that, depending on the system, may be pulsed at a lower frequency (e.g., 217 Hz). The signal results in low-level electromagnetic fields (EMF) that are in part absorbed by the user, and this feature has led to public concern about possible adverse health effects. At present there is no known mechanism to explain how MP-related EMF could affect biological organisms, prompting many to dismiss the possibility of MP-related health effects a priori. However, there is some evidence that MP-exposure may affect neural function in humans.

This study shall address one such line of research, that employing the electroencephalogram (EEG) to obtain indices of neural function in awake, resting humans, to determine whether MP-related EMF has an effect on neural function. The EEG measures coherent neural voltage fluctuations from electrodes placed about the scalp, and an important feature of the EEG is that it has millisecond resolution, which enables through spectral analysis of these fluctuations, the separation of functionally distinct neural processes.

The 8–12/13 Hz band of the spontaneous EEG spectrum of awake humans is often termed ''alpha.'' It is commonly hypothesized that EEG alpha power is generated by multiple processes in posterior regions of the brain [Andreassi, 2000]. In relation to the EEG, alpha is most easily recorded from occipital and parietal sites and can be driven simply by opening and closing the eyes, where opening the eyes attenuates the rhythm and closing the eyes enhances it. This very observation has traditionally led to increases in resting EEG alpha being interpreted as cortical idling, attenuated by

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mental effort [Niedermeyer and Lopes da Silva, 1987; Jacobs and Friedman, 2004]. However, recent research has demonstrated that this interpretation does not capture the complex functional significance of this rhythm [Schurmann and Başar, 2001; Cooper et al., 2003; Klimesch et al., 2003], and due to this, any change in alpha caused by MP phone exposure has the potential to be similarly complex.

Spontaneous resting alpha power has been the subject of much research into MP-related bioeffects. For example, Reiser et al. [1995], De Seze et al. [2001], and Croft et al. [2002] have all reported MP-related increases to alpha power. Further, Huber et al. [2002] reported an increase in the higher sub-band of this alpha range post exposure (PE) cessation while participants were preparing to sleep, and D'Costa et al. [2003], who employed a measure of alpha difference between the hemispheres, found a reduction of this difference metric. Such findings have prompted some to argue that alpha was emerging as a strong possibility in the search for a MP-related bioeffect [Hamblin and Wood, 2002].

There have also been null findings in relation to MP-related effects on alpha [Röschke and Mann, 1997; Hietanen et al., 2000], with this discrepancy in need of resolution. Croft et al. [2002] and Hamblin and Wood [2002] have argued that methodological issues can explain the discrepancy, with the null results more affected by methodological limitations. For example, Croft et al. [2002] argued that a crucial factor that differed between the positive and null findings was ''signal-to-noise ratio'' (SNR), with null results tending to come from studies with brief exposures (low signal) and/or large non-experimentally induced alpha variability (high noise). However, improving SNR is not trivial, as alpha variance will increase as a function of exposure duration [Maltez et al., 2004] due to the many factors to which alpha is related. For example, it has been shown that resting alpha relates to relaxation [Jacobs and Friedman, 2004] and fatigue [Cajochen et al., 1995], as well as performance on a number of tasks [Klimesch et al., 2003] and direction of attention [Worden et al., 2000; Cooper et al., 2003; Gladwin and de Jong, 2005]. Further, recent research [Cook et al., 2004, 2005] has suggested that MP-emissions may result in differing effects on alpha as a function of exposure duration, further emphasising the need to deal effectively with this variable.

It should be noted that Croft et al.'s [2002] report of increased alpha was with a regime that enhanced SNR by using a long exposure-duration and accounting for temporal changes in alpha statistically. However, there were important limitations that restricted its utility. For example, although many limitations were accounted for, it employed only a modest number of subjects ($N = 20$), a very low (and unverified) exposure level, used a non-standard ''resting'' EEG protocol, and was only single blind. Thus, an important possibility remains in that the results could have been due to modest sample size, inappropriate dosimetry or to experimenter bias, with this single-blind issue also a concern for Reiser et al. [1995], De Seze et al. [2001], and D'Costa et al. [2003]. Further, the alpha enhancement that Huber et al. [2002] reported immediately after MP-exposure was also limited in that although employing a double-blind design, it used a between-subject design with only 15 subjects, and only found a P-value of 0.04 that was not corrected for multiple comparisons (>20). Similarly, although De Seze et al. [2001] reported an alpha increase they did not provide statistics in support of the claim. The majority of studies thus suggest that MP exposure enhances rather than diminishes human alpha activity, both during exposure (DE) and after exposure cessation, but as the majority also suffer from serious limitations we cannot be confident with this conclusion.

However, a recent study by Curcio et al. [2005] overcame the above limitations, and again found a MP-related alpha enhancement DE. This substantially strengthens the claim that MP-exposure does increase alpha activity; however, with a modest sample size ($N = 20$), for this claim to be accepted the results need to be replicated with a larger sample. The current investigation was thus designed to provide a methodologically rigorous test of the thesis that MP-exposure increases alpha power, by employing similar methodologies to Curcio et al. [2005], but with a substantially larger sample. To this end, it tested a large sample's resting EEG during and after cessation of exposure, using a double blind, counterbalanced, crossover design with reliable dosimetry, where the MP handset was set to transmit at the maximum power that a standard MP is permitted to operate, and ''time on task'' was accounted for statistically. Consistent with Curcio et al. [2005], it was hypothesized that alpha power would increase during Active exposure relative to Sham exposure, and that this increase would be greater closer to the exposure source. As there have also been reports of MP-effects on alpha following exposure cessation, as well as interactions between MP-induced changes and both topography and exposure duration (both DE and after exposure cessation), these issues were also explored.

MATERIALS AND METHODS

Subjects

The sample originally comprised 120 healthy volunteers aged from 18 to 69 years (Mean = 31,

SD = 13 years). There were 46 males and 74 females, 108 of whom were right handed. Due to a combination of some subjects not completing all conditions and data loss due to technical reasons (see below), a reduced sample was employed for statistical analyses (ranging from 94 to 109, with numbers given in Section ''Results'' for each analysis). As this resulted in only partial counterbalancing, for any significant result, analyses were re-run to determine whether the results were related to order-effects. No participant reported any psychological or neurological condition, serious head injury or extended period of unconsciousness. All reported normal hearing, and normal (or corrected-to-normal) vision. Participants were informed in detail about the purpose and procedures of the study and written informed consent was obtained. The study design was approved by the Swinburne University Human Research Ethics Committee.

Procedure

A double blind, counterbalanced (Left vs. Right Hemisphere Exposure; between subject), crossover (Sham vs. Active) design was employed, where participants were required to attend two sessions 1 week apart. Both performance and event-related potential measures were collected in addition to the resting alpha described here, with performance and event-related potential results reported elsewhere [Hamblin et al., 2006].

At the beginning of each session, participants completed a demographics questionnaire, as well as a ''General Health Questionnaire'' (GHQ) to test for differences in subjects' general physical and psychological well-being between the Sham and Active sessions [Goldberg and Williams, 1998]. The GHQ is a self-report questionnaire comprised of four subscales which provide an indication of whether the respondent has experienced substantial impairment to physical or psychological well-being over the past week. The participant was then fitted with the EEG recording apparatus, seated in a comfortable chair 1.5 m in front of a computer monitor, and had a phone set to Sham attached over the temporal region. The baseline period then began where participants performed a battery of tests (described in Reference Hamblin et al. [2006]), followed by an electro-oculographic (EOG) calibration task (3 min). The Sham phone was then replaced with another phone (either Sham or Active), and participants were then instructed to ''sit quietly and relax with your eyes open,'' while 10-min of resting EEG data were collected (DE). Following this, the battery of tests was repeated, after which the phone was removed and another 10 min of Resting EEG was recorded (PE). The total exposure duration was 30 min.

Exposure Set-Up

A GSM MP (Nokia 6110) was set via laptop and manufacturer software to continuously transmit an 895 MHz digital signal at a mean power output of 250 mW (peak power of 2 W), thus avoiding discontinuous transmission (DTX) and adaptive power control (APC). This signal was an RF carrier pulse modulated at 217 Hz with a duty cycle of 12.5%, giving a pulse width of 576 μs. The spectrum of phone emissions (<250 Hz) was assessed using a pick-up coil positioned at the back of the handset in the z-axis orientation, where frequencies of 16 and 217 Hz were found. Although 16 Hz is not typically discussed in terms of MP-emissions, it has been suggested that such frequencies may occur due to battery operations [Andersen and Pedersen, 1997]. The 8 Hz component, associated with the 26th GSM frame, was not present in this test configuration.

Specific absorption rate (SAR) measurements were conducted inside a Specific Anthromorphic Mannequin (SAM) phantom using a precision robot RF Dosimetric Assessment System (DASY4). Without the EEG recording apparatus in place, a maximum SAR of 0.674 W/kg was measured high on the cheek bone, midway between the auricle and temple (10 g average), and the maximum in-line with the phone's antenna (approximately over the ''temporal lobe'') was 0.110 W/kg (10 g average). The effect of the EEG cap and leads on the measured SAR compared to that without the cap and leads in place was investigated by measurement and computational methods and is reported elsewhere [Hamblin et al., in press]. Both measurement and computation predict a decrease in SAR values to the head of up to 20%.

For both the Sham and Active exposure conditions, the phone was placed in a cradle over the subjects' EEG recording cap, either over the right ($n = 60$) or left ($n = 60$) temporal region, comparable to normal use (in ''touch'' position; FCC Guidelines, 2001). It should be noted that in a separate study [Wood et al., 2003], this type of cap did not display any EMF pickup within the physiologically important range relevant to this study (0.5–45 Hz).

The handset's audio circuitry was disabled so that participants would not be provided with acoustic cues to indicate the status of the phone. Additionally, padding was placed between the handset and its leather casing to effectively silence the just-perceptible ''buzz'' emanating from the circuit components during RF emission and to insulate against any heat generated by prolonged battery operation. In an independent test, two volunteers with normal hearing underwent a total of 20 1-min real/sham trials to test for acoustic cues or any interference with the ear-inserts that subjects were required

to use. Order of exposure was random and fully counterbalanced. In 18 of the 20 trials the volunteers reported to be unsure of the phone's status. When forced to make a choice between Active or Sham, only 11 out of the 20 choices were correct, which led us to believe that subjects would not be able to detect the phone in transmission mode. Further, we asked participants of the present study to guess whether they believed that the phone was on or not, with their responses reported in Section ''Results.''

To ensure that the power output of the test phone remained consistent over the entire duration of the study, output was measured over a series of tests via a power meter at the phone's antenna. These tests were conducted in an independent laboratory before testing commenced, after the 60th participant was tested, and upon completion of testing the last participants. This output was consistently observed to remain at the maximum value for approximately 90 min before declining slightly over the next hour and dropping off suddenly after a total of three and a half hours. For the purpose of the current study this meant that as the batteries were recharged after each testing session, the power output of the test phone remained at maximum for the critical 30 min of active exposure for each participant.

To ensure that participants received the appropriate exposure during the Sham and Active conditions, we checked to see whether the EEG from each session contained the appropriate machine signals (i.e., a well defined 217 Hz peak in the Active, and no 217 Hz peak in the Sham condition). This condition was not met for five participants and their data were thus not used in the statistical analyses.

Data Acquisition

EEG data were collected using Neuroscan Synamps amplifiers and 4.2 software, from tin electrodes at 58 scalp sites referenced to left mastoid, using an electrode cap arranged according to the Modified Combinatory Nomenclature [Fisch, 1999] and comparable to the 10/10 International System. EOG data were recorded using tin electrodes above and below the left eye, and on the outer canthus of each eye. All data were continuously sampled at 500 Hz with a 0.05–250 Hz bandpass filter. Electrode impedances were below 5 kΩ at the start of each session.

Data Analysis

For each of the DE and PE Resting EEG periods, data were EOG corrected [Croft and Barry, 2000], re-referenced to common average, divided into 2 s bins and baseline-corrected. Epochs were then removed from the analysis if they had EEG voltages greater than $\pm 200\,\mu V$, and were averaged in the frequency domain (Fast Fourier transform; cosine window; 10%) for each of the 1st and 2nd half of the Resting EEG period separately using Neuroscan Edit 4.2 software. The resultant alpha power (8–12 Hz) was then calculated for each site. Alpha power data were then grouped into front ipsilateral, front contralateral, posterior ipsilateral and posterior contralateral scalp regions. This resulted in two levels of each of the variables Anterior–Posterior axis (A–P axis) and Ipsilateral-Contralateral (Ipsi-Contra). For each of these grouped values, data were then converted to amplitude (square root function).

Statistical Analyses

A 2×2 Chi-square test was employed to determine whether participants were able to accurately report the status of the phone, where the independent variables were Decision (On vs. Off) and Exposure (Sham vs. Active). A 2 (Sham vs. Active) $\times$ 4 (GHQ subscales) repeated measures analysis of variance was conducted on GHQ data to check whether the general health of participants differed between the weeks preceding the Sham and Active conditions.

To reduce the effects of ''time on task'' on alpha activity, for each of the DE and PE periods, ''time on task'' was treated as a noise variable and scores within each of the 1st and 2nd half converted to z-scores separately, following Croft et al. [2002]. This has the effect of making the mean value for each of the two halves of the recording equal to zero, and avoids the possibility of treatment effects being masked by the alpha changes that normally occur as a function of testing duration. Due to a positive skew in the Active that was not present in the Sham condition, data could not be normalized and so non-parametric analyses were employed (Wilcoxon Signed Ranks Tests), and performed separately for each of the DE and PE periods. Non-parametric analyses have limited flexibility, and so the data were simplified by creating Difference Scores by subtracting the 1st from 2nd half of the variable ''time,'' the frontal from posterior scalp regions of the variable ''A–P axis,'' and the contralateral from ipsilateral regions of the variable ''Ipsi/Contra,'' for each of the Sham and Active, DE and PE periods separately. For example, the ''time'' manipulation produced an index of the change in alpha from the 1st to 2nd half of the recording period (and did so separately for each of the scalp regions, exposure conditions, DE, and PE periods separately), such that a positive value represented an increase in alpha from the 1st to 2nd half of the recording period, and a negative value represented a reduction in alpha. This allowed what we viewed to be the most important questions to be addressed without ''spending'' too many degrees of freedom (although

TABLE 1. Descriptive Statistics for the During Exposure (DE) Period

Exposure	Time	A–P axis	Ipsilateral	Contralateral
Sham	1st 5 min	Frontal	1.03 (0.20)	1.03 (0.20)
		Posterior	1.32 (0.34)	1.33 (0.33)
	2nd 5 min	Frontal	1.03 (0.22)	1.03 (0.22)
		Posterior	1.31 (0.33)	1.32 (0.33)
Active	1st 5 min	Frontal	1.09 (0.30)	1.09 (0.31)
		Posterior	1.35 (0.31)	1.34 (0.29)
	2nd 5 min	Frontal	1.10 (0.33)	1.10 (0.35)
		Posterior	1.33 (0.31)	1.33 (0.30)

Alpha means (and SDs) in units of Power (μV^2) for EEG recording made during Active/Sham exposure, for each exposure condition separately, as a function of time (1st vs. 2nd half), Anterior–Posterior (A–P) axis and laterality.

means and standard deviations of ''all'' levels are presented; see Tables 1 and 2). Hypothesis-driven and exploratory tests are described separately:

Hypothesis-Driven

For the DE period: (1) the main effect of Exposure was one-tailed, testing for a general increase in alpha based on the results of Curcio et al. [2005]; (2) the effect of exposure on the Ipsi/Contra Difference Score was one-tailed, testing for a greater alpha increase closer to the source of the exposure (ipsilateral) than further from the source (contralateral), based on the results of Curcio et al. [2005]. As each of these two analyses was employed to test specific findings generated from the literature, α was kept at 0.05 for each of the tests (as recommended by Tabachnick and Fidell [2001]).

Exploratory Analyses

To obtain further information about the data set, non-directional analyses were conducted to answer the following questions: For the PE period: (1) does exposure affect alpha overall? (2) Does exposure affect the Ipsi/Contra Difference score overall? For each of the DE and PE periods: (3–4) does exposure affect the Time Difference Score? (5–6) Does exposure affect the Ipsi/Contra Difference Score during the 1st half of the recording? (7–8) Does exposure affect the Ipsi/Contra Difference Score during the 2nd half of the recording? (9–10) Does exposure affect the A–P axis Difference Score? (11–12) Does exposure affect the Ipsi/Contra Difference Score at frontal sites? (13–14) Does exposure affect the Ipsi/Contra Difference Score at posterior sites?

The Dubey/Armitage & Parmar method was employed to maintain an overall α level for the exploratory analyses of 0.05 [Sankoh et al., 1997], by utilizing the mean correlation between all the dependent variables employed in the exploratory analyses (0.647) to adjust α to 0.020 for each of the 14 individual tests. This method is similar to the Bonferroni adjustment for multiple comparisons, but differs in that it takes into account dependencies between dependent variables when determining the adjustment factor that would otherwise inflate Type II error.

TABLE 2. Descriptive Statistics for the Post Exposure (PE) Period

Exposure	Time	A–P axis	Ipsilateral	Contralateral
Sham	1st 5 min	Frontal	1.04 (0.21)	1.04 (0.21)
		Posterior	1.33 (0.33)	1.34 (0.34)
	2nd 5 min	Frontal	1.06 (0.21)	1.05 (0.22)
		Posterior	1.32 (0.30)	1.33 (0.31)
Active	1st 5 min	Frontal	1.07 (0.30)	1.05 (0.26)
		Posterior	1.30 (0.30)	1.29 (0.29)
	2nd 5 min	Frontal	1.08 (0.26)	1.07 (0.25)
		Posterior	1.31 (0.32)	1.30 (0.31)

Alpha means (and SDs) in units of Power (μV^2) following exposure cessation are shown for Sham and Active conditions separately, as a function of time (1st vs. 2nd half of each 10-min recording period), Anterior–Posterior (A–P) and laterality.

RESULTS

The Chi-squared test showed that there was an overall bias in reporting whether the phone was transmitting or not ($\chi^2(1) = 195.08$, $P = 0.001$). This bias was towards reporting that the phone was turned ''off'' and did not differ between the exposure conditions, with 78% of participants responding that the phone was turned ''off'' during Active exposure ($\chi^2(1) = 48.13$, $P = 0.001$), and 84% responding that the phone was turned ''off'' during Sham exposure ($\chi^2(1) = 146.94$, $P = 0.001$). As described in Reference Hamblin et al. [2006], participants' GHQ scores did not differ between the Active and Sham weeks ($F[3357] = 0.38$; $P = 0.754$). Raw alpha means and standard deviations are shown for each of DE and PE, as a function of exposure, time, A–P axis, and laterality, in Tables 1 and 2, respectively. Power spectra for electrode locations ipsilateral and contralateral to the site of exposure are shown for DE and PE in Figures 1 and 3, respectively. Topographies of grand mean alpha power are shown for DE and PE in Figures 2 and 4, respectively.

Hypothesis-Driven

During the DE period: (1) as can be seen in Figure 1, there was more alpha in the Active than Sham condition, with this difference statistically significant ($Z[109] = 2.01$; $P = 0.022$; *one-tailed*). (2) The Ipsi/Contra Difference Score was larger (*trend-level*) during Active than Sham exposure overall ($Z[109] = 1.50$; $P = 0.066$; *one-tailed*), see Table 3.

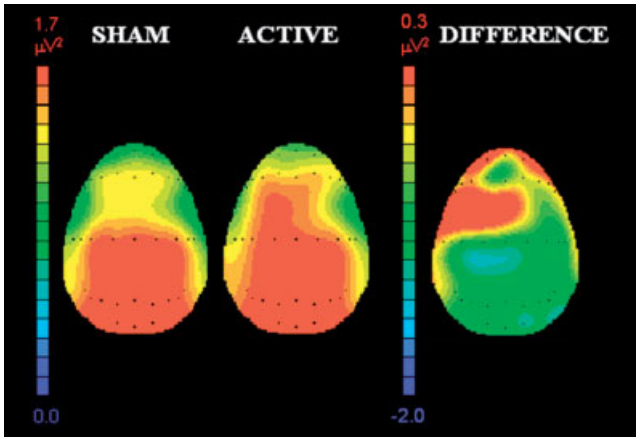

Fig. 1. Topographies of grand mean alpha power (8–12 Hz) are shown for EEG recordings made during Sham/Active exposure, as well as the difference between these exposure conditions. Ipsilateral (to the phone), contralateral, anterior, and posterior directions are represented on the maps as left, right, up, and down, respectively.

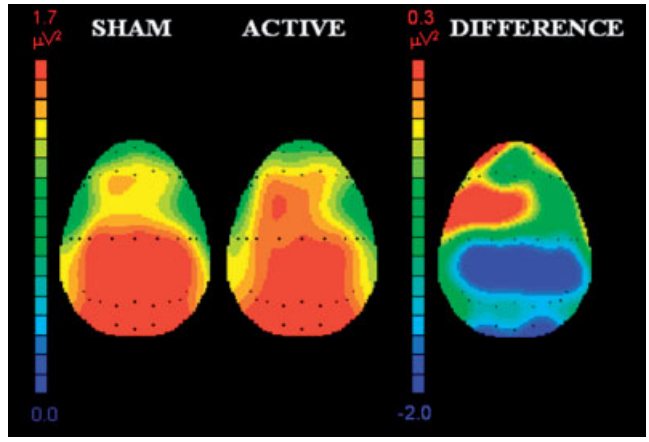

Fig. 3. Topographies of grand mean alpha power (8–12 Hz) are shown for EEG recordings made after Sham/Active exposure cessation, as well as the difference between these exposure conditions. Ipsilateral, contralateral, anterior, and posterior directions are represented on the maps as left, right, up, and down, respectively.

Exploratory Analyses

During the exposure period (DE): The Time Difference Score did not differ between the Active and Sham exposure ($Z[108] = 0.13$; $P = 0.899$). The Ipsi/Contra Difference Score did not differ between Active and Sham exposure during the 1st or 2nd half of the recording ($Z[109] = 1.66$; $P = 0.098$, and $Z[108] = 1.08$; $P = 0.281$, respectively). The A–P axis Difference

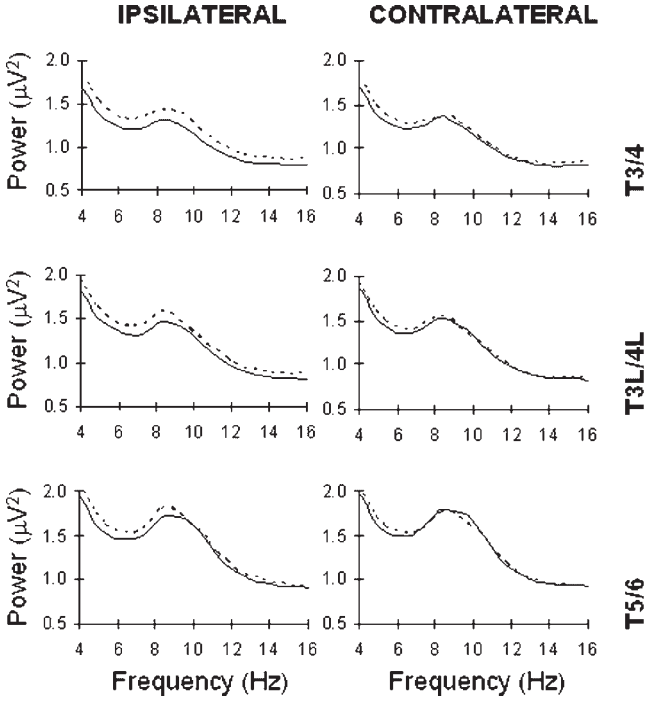

Fig. 2. EEG spectral power during exposure is shown: Grand mean power spectra are shown for sham (solid line) and active (dotted line) exposures, for ipsi and contralateral sites separately, at each of the T3/T4, T3L/T4L, and T5/T6 derivations. Note that due to counterbalancing, T3, T3L, and T5 sites were ipsilateral for some participants, and T4, T4L, and T6 were ipsilateral for the others.

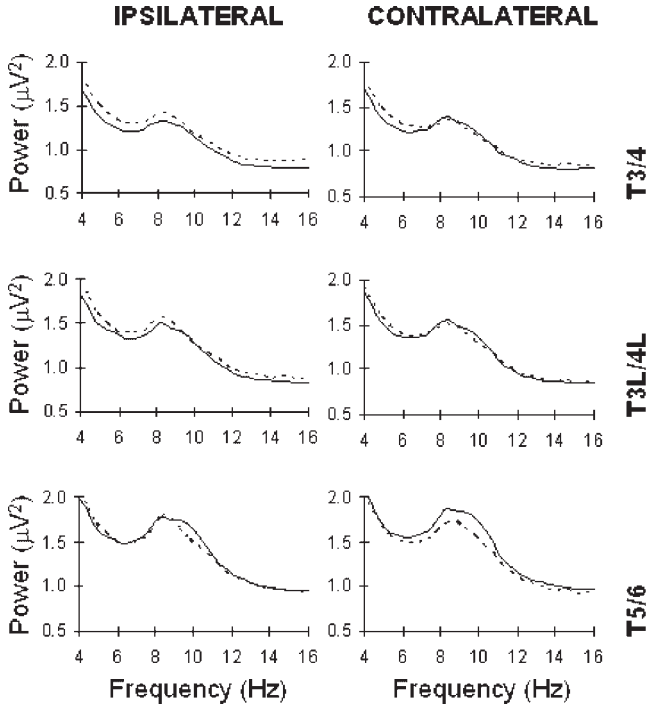

Fig. 4. EEG spectral power after exposure cessation is shown: Grand mean power spectra are shown for Sham (solid line) and Active (dotted line) exposures, for ipsi and contralateral sites separately, at each of the T3/T4, T3L/T4L, and T5/T6 derivations. Note that due to counterbalancing, T3, T3L, and T5 sites were ipsilateral for some participants, and T4, T4L, and T6 were ipsilateral for the others.

TABLE 3. Summary of Main Statistical Analyses

	Z	df	*P*
Hypothesis-driven (during exposure)			
A vs. S	2.01	109	0.022
$A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	1.50	109	0.066
Exploratory (during exposure)			
$A_{1st\ half\text{-}2nd\ half}$ vs. $S_{1st\ half\text{-}2nd\ half}$	0.13	108	0.899
1st Half of Recording: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	1.66	109	0.098
2nd Half of Recording: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	1.08	108	0.281
$A_{ant\text{-}post}$ vs. $S_{ant\text{-}post}$	0.04	109	0.965
Anterior: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	0.56	109	0.575
Posterior: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	2.90	109	0.004
Exploratory (post exposure)			
A vs. S	0.57	97	0.568
v$A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	2.75	97	0.014
$A_{1st\ half\text{-}2nd\ half}$ vs. $S_{1st\ half\text{-}2nd\ half}$	0.37	97	0.710
1st half of recording: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	2.68	97	0.007
2nd half of recording: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	2.15	94	0.031
$A_{ant\text{-}post}$ vs. $S_{ant\text{-}post}$	1.50	97	0.135
Anterior: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	0.92	97	0.358
Posterior: $A_{ipsi\text{-}contra}$ vs. $S_{ipsi\text{-}contra}$	2.46	97	0.014

Results for the main statistical analyses (Wilcoxon Signed Ranks tests) are shown. Hypothesis-driven tests were 1-tailed, with a critical $\alpha = 0.05$. Exploratory tests were 2-tailed, with the critical $\alpha = 0.02$. A, Active; S, Sham; ipsi, ipsilateral; contra, contralateral; 1st half, 1st half of the recording; 2nd half, 2nd half of the recording; ant, anterior scalp sites; post, posterior scalp sites.

Score did not differ between Active and Sham exposure ($Z[109] = 0.04$; $P = 0.965$). The Ipsi/Contra Difference Score did not differ between Active and Sham exposure frontally ($Z[109] = 0.56$; $P = 0.575$). The Ipsi/Contra Difference Score was larger during Active than Sham exposure at posterior sites ($Z[109] = 2.90$; $P = 0.004$).

During the PE period: Alpha did not differ between the Active and Sham conditions overall ($Z[97] = -0.57$; $P = 0.568$). The Ipsi/Contra Difference Score was larger during Active than Sham exposure overall ($Z[97] = 2.75$; $P = 0.014$). The Time Difference Score did not differ between Active and Sham exposure ($Z[94] = 0.37$; $P = 0.710$). The Ipsi/Contra Difference Score was larger during Active than Sham exposure during the 1st half of the recording ($Z[97] = 2.68$; $P = 0.007$). The Ipsi/Contra Difference Score was larger (*trend level*) during Active than Sham exposure during the 2nd half of the recording ($Z[94] = 2.15$; $P = 0.031$). The A–P axis Difference Score did not differ between Active and Sham exposure ($Z[97] = 1.50$; $P = 0.135$). The Ipsi/Contra Difference Score did not differ between Active and Sham exposure frontally ($Z[97] = 0.92$; $P = 0.358$). The Ipsi/Contra Difference Score was larger during Active than Sham exposure at posterior sites ($Z[97] = 2.46$; $P = 0.014$).

Further, to determine whether the positive findings were related to the lack of complete counterbalancing caused by the loss of participant's data (see Section ''Subjects''), Wilcoxon's Independent group analyses were performed using ''exposure order'' as the between subjects independent variable, and the difference between the two levels of the significant comparison as the dependent variable (i.e., DE: (1) Active [all sites combined]–Sham [all sites combined], (2) Active Ipsi/Contra Difference Score–Sham Ipsi/Contra Difference Score, (3) Active Ipsi/Contra Difference Score [posterior]–Sham Ipsi/Contra Difference Score [posterior]; PE: (1) Active Ipsi/Contra Difference Score–Sham Ipsi/Contra Difference Score, (2) Active Ipsi/Contra Difference Score [1st half]–Sham Ipsi/Contra Difference Score [1st half], (3) Active Ipsi/Contra Difference Score [posterior]–Sham Ipsi/Contra Difference Score [posterior]). The critical α levels that were employed for the initial analyses were also employed for this checking procedure.

DE: Neither the main effect of Exposure ($Z[109] = 0.26$; $P = 0.797$), the effect of proximity to the phone ($Z[109] = 1.57$; $P = 0.116$), nor the effect of proximity to the phone at posterior sites ($Z[109] = 1.60$; $P = 0.111$) were related to exposure order, indicating that exposure order did not account for the alpha changes during the active exposure. PE: The effect of proximity to the phone overall ($Z[97] = 1.90$; $P = 0.058$), the effect of proximity during the first half of recording ($Z[97] = 2.00$; $P = 0.046$) and at posterior sites ($Z[97] = 2.27$; $P = 0.023$), were related (*trend-level*) to exposure order. Specifically, those that received the Active exposure first exhibited more of a difference between the Sham and Active conditions than those that received it second.

To determine whether the significant effects of the Active exposure were related to age, gender or MP usage histories, difference scores (Effect = Active – Sham) were created to represent the effect of (1) Active exposure overall and (2) Active exposure on the Ipsi/Contra difference scores, and the following analyses performed: (1) non-parametric (Spearman's) correlations were performed between each of these difference scores, and each of ''age,'' ''daily MP use in last week (in minutes),'' ''daily MP use in last year (in minutes),'' ''years of use.'' (2) *t*-tests were performed with these difference scores as the dependent variables, and ''gender'' as the independent variable. No correlation was found between age or any MP usage variables, and either of the Active exposure effects ($r < 0.09$; $P > 0.34$). There were no differences between genders on either of the Active exposure

effects ($t < 1.20$, $P > 0.23$). Further, a Spearman's correlation failed to find any relation between alpha activity in the sham condition, and any of the MP usage history indices ($r < 0.10$; $P > 0.32$).

DISCUSSION

Results of the current investigation indicate that acute, low-level exposure to EMFs emitted by GSM MP, increases EEG activity in the alpha range of the awake resting EEG, and thus supports the view that there are MP-related bioeffects that occur as a result of the low-level EMF that MP emit. As well as being consistent with other reports of MP-related alpha enhancements that, as described in the Section ''Introduction,'' could not be confidently taken as evidence for a bioeffect due to methodological limitations, this finding is also consistent with a recent investigation that accounted for such methodological limitations and still found a MP-related alpha enhancement [Curcio et al., 2005]. As our current work also overcame concerns of earlier work in that it employed double-blinding techniques, dealt appropriately with multiple comparisons, employed adequate dosimetry and used a large sample size, this provides strong support for the view that MP-exposure does increase alpha activity in the human awake EEG.

The increased alpha was more pronounced at sites ipsilateral to exposure, particularly at posterior sites, which is consistent with Curcio et al. [2005], who found a *trend-level* enhancement over left (but not right) temporal regions with a left temporal scalp exposure. Importantly, in the present study this alpha enhancement was not due to contamination of the EEG by emissions from the MP, as there was not an 8 Hz signal emitted by the phone in the present exposure configuration (the 26th frame was turned on). These effects were also not related to age, gender, or self-reported MP-usage histories.

Although the ipsilateral effect was largest at posterior sites both during and after exposure cessation, the alpha patterns were very different. Specifically, as can be seen in Figures 1 and 2, whereas the significant posterior alpha laterality index was due to enhanced ipsilateral alpha DE, it was due to reduced contralateral alpha following exposure cessation (with almost identical ipsilateral alpha in the Active and Sham conditions). Thus, while there is apparent consistency in terms of the ipsilateral alpha increases more frontally (see Figs. 1 and 2) this effect was not statistically significant PE, and at posterior sites different processes were occurring during and after exposure. It is thus tempting to speculate that two different effects of the MP have been found in the present study; a direct effect on posterior alpha close to the phone that only lasts as long as the exposure, and a longer lasting alpha increase more frontally.

Four points are particularly noteworthy. First is that even though the effect closest to the phone (posterior ipsilateral effect described above) was the strongest statistically, the frontal increase in alpha was larger in magnitude, which suggests that the largest observable alpha effect of the phone may be that of secondary neural processes rather than the immediate ipsilateral effects (see Figs. 1–3). The second is that the magnitude of the MP-effect was very small, with only 3% (main effect) and 7–9% (ipsilateral vs. contralateral effects) of the variance in alpha power explained by the MP-exposure. This can be seen in Tables 1 and 2 (where the effect of MP exposure would likely be overlooked if not for the repeated-measures analysis technique employed) and goes some way to explaining why demonstrations have been equivocal in the past, as such small effect sizes are not easily detected with the smaller sample sizes that have previously been employed. Consistent with this small effect size it should be pointed out that not everyone exhibited an increase in alpha. For example, for the main effect of Exposure, during the Active period only 60% showed an increase in alpha, suggesting that the effect of the phone was not homogeneous, and that it may be related to individual differences between the participants. Third is that the ''increase in alpha as a function of exposure duration'' that was reported in Reference Croft et al. [2002], was not replicated in the present study.

Fourth, the present study made a distinction between *hypothesis-driven* and *exploratory* analyses, where increased Type I error due to multiple analyses of the data set was accounted for in the latter case only. This is the preferred method where the literature provides justification for making specific predictions (see Reference Tabachnick and Fidell [2001]), and may be interpreted as accounting for the low probability of obtaining the same ''chance finding'' repeatedly. For example, the probability of obtaining the same chance finding at $P = 0.05$ in two independent studies would be 0.0025 (which on average would only occur once in every 400 analyses).

The main effect of Exposure that we report is a significant MP-induced increase in alpha, during the exposure. As has been pointed out previously [Croft et al., 2002; Curcio et al., 2005], this alpha enhancement is not easily interpreted due to the range of neural functions to which alpha has been shown to relate. That is, although typically seen as representing idling or neural inactivity [Niedermeyer and Lopes da Silva, 1987; Başar et al., 2001; Jacobs and Friedman, 2004], recent research has demonstrated unequivocally that

under many circumstances enhanced alpha over the scalp corresponds to increased neural processing [Worden et al., 2000; Cooper et al., 2003; Klimesch et al., 2003; Gladwin and de Jong, 2005]. MP research that has looked at behavioral endpoints does not help resolve this quandary, as consistent behavioral effects of MP exposures are yet to be demonstrated. Thus, to determine which aspect of neural processing a particular alpha increase represents requires more-sophisticated protocols than those employed in the present study.

Having corroborated previous findings of MP-related increases in alpha, an important question arises; namely, ''how could an EMF bioeffect occur at such low emission levels?'' There are currently a number of theories regarding this, but as discussed in Section ''Introduction'' our view is that none of these have strong support at present. The proposed mechanisms include calcium efflux through RF stimulation of the outer layer of neurons [Bawin et al., 1975; Blackman et al., 1979; Adey, 1981], thermal increases of less than 1 °C that may affect the brain in subtle ways that we cannot predict (for review see Reference Adair and Black [2003]), increases in permeability of the blood–brain barrier (for review see Reference D'Andrea et al. [2003]), and a hypothesis put forward by Freude et al. [1998] has recently been given empirical support in that MP exposure has been reported to increase neural excitability [Ferreri et al., 2006].

As noted in the current WHO Research Agenda [2006] and in a recent review by Challis [2005], ''neural interference'' may also be a viable mechanism for MP-related changes such as those observed within the alpha frequency band. As the extremely low frequency (ELF) components (16 and 217 Hz; see Section ''Materials and Methods'') of the phone emissions are within the range that the brain naturally employs in its communication network (e.g., 16 Hz [Knyazeva et al., 2006]; 60–250 Hz [Edwards et al., 2005]; up to 1000 Hz [Mochizuki and Ugawa, 2005]), as suggested by Huber et al. [2002, 2005] this opens up the possibility that the exogenous (MP) ELF may interfere with the endogenous (neural) ELF. Thus, effects such as the alpha increase reported in the present study would be downstream consequences of the interference, representing a normal response to the event. This speculation is consistent with reports that RF has effects only when pulse-modulated, in terms of both alpha power [Huber et al., 2002] and PET-recorded dorsolateral prefrontal cortex activity [Huber et al., 2005]. It is also consistent with the similarity of the present MP-alpha findings and the increased alpha that has been reported as a result of pure ELF [Cook et al., 2004; Cook et al., 2005].

In conclusion, the present study has replicated previous reports of MP-related increases in resting alpha, and thus adds strength to the argument at which there are MP-related bioeffects at the low levels that MPs operate. Although the functional significance of this alpha change cannot be determined at present, it should be pointed out that alpha changes of the magnitude reported in this study have not previously, or in this study, been found to relate to health outcomes, either positive or negative.

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