BEFORE THE
PENNSYLVANIA PUBLIC UTILITY
COMMISSION

Michael T. Jennings	:	
	:	
v.	:	**Docket No. C-2018-3006031**
	:	
West Penn Power Company	:	

TESTIMONY OF DR. MICHAEL SEMELKA, DO ON BEHALF OF COMPLAINANT MICHAEL T. JENNINGS

September 9, 2019

1. Q. Please state your name and business address.

A. Michael Semelka

508 S. Church Street Mt. Pleasant, PA 15666

2. Q. *What is your occupation?*

A. Family Physician

3. Q. Where are you employed and what is your position?

A. Excela Health. I am the family medicine residency director.

4. Q. Please describe your educational background.

A. Bachelors of Science in Biology at the Pennsylvania State University, Doctor of Osteopathic Medicine at Philadelphia College of Osteopathic Medicine, Family Medicine Residency at the Medical Center of Beaver, PA, Fellowship in Faculty Development at Duke University.

5. Q. Please briefly describe your professional experience.

A. I have practiced as a board certified family physician since 2003.

6. Q. Are you McKenzie Jennings' Primary Care Physician?

A. Yes.

7. Q. Are you Susan Jennings' Primary Care Physician?

A. Yes.

adverse consequences in persons who are more susceptible thereto?

A. Yes.

17. Q. Relative to an infrequent exposure to a pollutant, would the maximizing of exposure duration to 24-7-365-forever likely produce more adverse effect?

A. Yes.

18. Q. In your medical opinion, does McKenzie qualify for Americans with Disabilities Act (ADA) accommodations?

A. Yes.

19. Q. Is an individual required to prove he has already been adversely affected before an accommodation through ADA is granted?

A. I do not believe so.

20. Q. Mobile carriers classify non-ionizing radiation, also called radiofrequency (RF) radiation, as a pollutant. T-Mobile's Insurance Warranty Guide defines pollution as, "...artificially produced ionizing or non-ionizing radiation and or waste."

Source: **https://www.phoneclaim.com/t-mobile/pdf/New-Ts-and- Cs.pdf**

Given this information, would you recommend that McKenzie be, or not be, exposed to T-Mobile's RF radiation at home?

A. I would recommend that he not be exposed to RF radiation.

31. Q. Do you consider radio frequency radiation a pollutant?

A. Possibly.

32. Q. Can a pollutant or toxin affect the central nervous system?

A. Yes.

33. Q. Does this conclude your testimony?

A. Yes, although I reserve the right to supplement my direct testimony.

References:

Exhibit A

What is the impact of electromagnetic waves on epileptic seizures? med./bio. By: Cinar N, Sahin S, Erdinc OO Published in: Med Sci Monit Basic Res 2013; 19: 141-145

Exhibit B

Effect of 450 MHz Microwave Modulated with 217 Hz on Human EEG in Rest med./bio. By: Bachmann M, Säkki M, Kalda J, Lass J, Tuulik V, Hinrikus H Published in: Environmentalist 2005; 25 (2-4): 165-171

Exhibit C

Integration of differences in EEG Analysis Reveals Changes in Human EEG Caused by Microwave. med./bio. By: Bachmann M, Lass J, Kalda J, Sakki M, Tomson R, Tuulik V, Hinrikus H Published in: IEEE 28th Annual International Conference of the Engineering in Medicine and Biology Society (EMBC), 2006. IEEE, 2006, ISBN 9781424400324: 1597-1600

Exhibit D

Alterations of human electroencephalographic activity caused by multiple extremely low frequency magnetic field exposures. med./bio. By: Cvetkovic D, Cosic I Published in: Med Biol Eng Comput 2009; 47 (10): 1063-1073

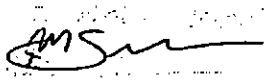
Exhibit E

Long-Term Evolution electromagnetic fields exposure modulates the resting state EEG on alpha and beta bands. med./bio. By: Yang L, Chen Q, Lv B, Wu T Published in: Clin EEG Neurosci

2017; 48 (3): 168-175

Exhibit F

Radiofrequency signal affects alpha band in resting electroencephalogram. med./bio. By: Ghosn R, Yahia-Cherif L, Hugueville L, Ducorps A, Lemarechal JD, Thuroczy G, de Seze R, Selmaoui B Published in: J Neurophysiol 2015; 113 (7): 2753-2759



Michael William Semelka, DO, FAAFP

(electronically signed)

EXHIBIT A

Dr. Michael Semelka, DO

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EXHIBIT A

Med Sci Monit Basic Res. 2013; 19: 141–145.
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What is the impact of electromagnetic waves on epileptic seizures?

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[B]Data Collection
[C]Statistical Analysis
[D]Data Interpretation
[E]Manuscript Preparation
[F]Literature Search
[G]Funds Collection

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Abstract

Background

The effects of electromagnetic waves (EMWs) on humans and their relationship with various disorders have been investigated. We aimed to investigate the effects of exposure to different frequencies of EMWs in various durations in a mouse epilepsy model induced by pentylenetetrazole (PTZ).

Material/Methods

A total of 180 4-week-old male mice weighing 25–30 g were used in this study. Each experimental group consisted of 10 mice. They were exposed to 900, 700, 500, 300, and 100 MHz EMWs for 20 hours, 12 hours and 2 hours. Following electromagnetic radiation exposure, 60 mg/kg of PTZ was injected intraperitoneally to all mice. Each control was also injected with PTZ without any exposure to EMW. The latency of initial seizure and most severe seizure onset were compared with controls.

Results

The shortest initial seizure latency was noted in the 12-hour group, followed by the 700 MHz. The mean initial seizure latencies in the 2-hour EMW exposed group was significantly shorter compared to that in the 12- and 20-hour groups. There was no significant difference between 12- and 20-hour EMW

exposed groups. There was a significant difference between control and 2- and 10-hour EMW exposed groups. No statistically significant differences were noted in mean latencies of the most severe seizure latency, following 20-, 12-, and 2- hour EMW exposed groups and control groups.

Conclusions

Our findings suggest that acute exposure to EMW may facilitate epileptic seizures, which may be independent of EMW exposure time. This information might be important for patients with epilepsy. Further studies are needed.

Keywords: epilepsy, pentylenetetrazole, mice, electromagnetic waves

Background

The effects of electromagnetic waves (EMWs) on humans and the relationship of EMSs with various disorders have been investigated [1]. As a result of advances in technology, people are constantly exposed to EMW. Alternating electric currents, computer screens, radio, television, cell phones, and radar devices are examples of sources of electromagnetic fields. All of these EMW sources operate at different frequencies. Several studies have suggested serious negative effects of EMWs on health [2–4].

We aimed to investigate the effects of different frequencies of EMWs at various durations on time of seizure onset (seizure latency) and the most severe seizure latency in a mouse model of epilepsy induced by pentylenetetrazole (PTZ) and to compare these results with controls.

Material and Methods

Study groups

The study was approved by our local Animal Ethics Committee. It was conducted on 180 4-week-old male albino mice weighing 25–30 g. Experiments were started after 10 A.M. Animals were kept at 24±1°C in a 12–12 hour light-dark cycle and were provided with water and food before and during the study.

Six experimental groups were established, including controls. Each group consisted of 10 mice. They were exposed to 900, 700, 500, 300 and 100 MHz EMW for 20, 12, and 2 hours. Following the exposure, 60 mg/kg of PTZ was injected intraperitoneally (IP) into all mice. PTZ was injected into the controls after the same periods without exposure to EMWs. All mice were then monitored and seizures were scored. Time to the first myoclonic jerk was recorded as initial seizure latency. Seizure severity was scored on a scale from 0 to 6.

Generation of EMWs

An antenna was used to generate EMWs. The antenna intercepts the EMWs and converts them into electrical currents. A 0.5-mm diameter transmitter dipole antenna with electromagnetic field frequencies of 900, 700, 500, 300, 100 MHz was fabricated for use in this experimental study [5]. Electromagnetic wave frequency was measured by a spectrum analyzer and frequency meter before the dipole antenna generated electromagnetic waves.

Administration of pentylenetetrazole (PTZ)

PTZ is a convulsant drug used in experimental epileptic seizure models, which causes convulsions similar to absence and myoclonic type seizures in humans when administered subcutaneously, intravenously, or intraperitoneally in mice and rats [6]. PTZ acts by binding to the GABA-A/benzodiazepine receptor complex and blocks the GABA-gated chloride channels. Drugs effective against PTZ model exert their anticonvulsant effects through their effects on T-type Ca^{+2} currents and GABA-A [7]. In this study, PTZ was administered at a dose of 60 mg/kg IP in mice.

Evaluation of seizures

After 20-hours, 12-hours and 2-hours groups of mice were exposed to 900, 700, 500, 300, and 100 MHz electromagnetic wave fields respectively, 60 mg/kg of PTZ was injected intraperitoneally. The control groups were also injected with 60 mg/kg of PTZ. All mice were monitored for 20 minutes. The seizure score was recorded according to the following scale; 0: No response, 1: Ear and facial twitching, 2. Mild myoclonic jerks of the limbs, 3: Severe myoclonic jerks of the limbs and rearing, 4: Forelimb convulsions, 5: Increase in general muscle tone in combination with rearing and falls, and 6: Status epilepticus and death [8].

Time to onset of initial myoclonic jerk was defined as first seizure onset latency. Mice were kept in a bell glass for 20 minutes following PTZ injection and the highest score (most severe seizure onset latency) was recorded [9].

Statistical analysis

SPSS 13.0 for Windows software was used for analysis. Groups were compared using one-way ANOVA, Kruskal Wallis test and chi-square tests.

Results

The shortest initial seizure latency was noted at 500 MHz ($p<0.05$) and the longest at 300 MHz ($p<0.05$) in the 2-hour group. The shortest initial seizure latency was noted at 700 MHz ($p<0.05$) and the longest in the control group, followed by 100 MHz ($p<0.05$) in the 12-hour group. The shortest initial seizure latency was noted at 300 MHz ($p<0.05$) and the longest in the 500 MHz ($p<0.05$) in the 20-hour group.

The shortest initial seizure latency was noted in the 12-hour group, followed by 700 MHz and the longest in the 20-hour group, followed by the 500 MHz. Mean initial seizure latencies of EMW-exposed groups were; 2 hour-group: 18.5 s, 12-hour group: 22.3 s, and 20-hour group: 27.2 s. The mean initial seizure latencies of the 2-hour EMW exposed group was significantly shorter compared to that in the 12- and 20-hour groups ($p<0.05$). There was no significant difference between 12- and 20-hour EMW exposed groups ($p>0.05$). There was a significant difference between the control group and the 2- and 10-hour EMW exposed groups, and no significant difference between controls and the 12-hour EMW exposed group (Table 1). No statistically significant difference was noted in the mean latencies of the most severe seizure latency following 20-, 12-, 2-hour groups and the control groups (Table 2).

EXHIBIT A

Table 1

Comparison of the initial seizure latencies (sec) without EMW exposure and with 900, 700, 500, 300, 100 MHz EMW exposure in the 2, 12 and 20-hours experimental groups and controls.

Open in a separate window

*The shortest and the longest latencies.

Table 2

Comparison of the latency of the most severe seizure (sec) following 900, 700, 500, 300, 100 MHz EMW exposure in the 2, 12, 20 hours experimental groups and controls.

Open in a separate window

Discussion

There are several published studies on the effect of EMWs on tumors [10,11], but there has not yet been a detailed study on EMWs and epilepsy. We investigated different frequencies of EMW on seizures in this comparative experimental epilepsy model. Five different frequencies (100, 300, 500, 700, 900 MHz) of EMWs were applied for 3 different time periods (2, 12, and 20 hours).

The shortest initial latency was observed in the 12-hour group, followed by the 700 MHz in our study. Tattersall et al. found excitability changes in rat hippocampal tissue exposed to 700 MHz radiofrequency without heating effect [12]. Our finding is compatible with results of that study.

EMWs are known to have a heating effect, which is more prominent at higher frequencies [13]. Previous studies have reported that radiofrequency leads to an increase in body core temperature [14]. It has been shown that energy absorption occurs via radiofrequency waves in target organs, the hypothalamic thermoregulatory center, and peripheral regions. Adair et al. used 450 MHz and 2450 MHz magnetic waves applied in humans, and found an increase in skin temperature [15]. Chou et al., exposed rats to 2450 MHz microwaves, and found that tail regions of the rats absorbed more energy than the head or other body regions, and that specific absorption rates (SAR) were higher in the anterior hypothalamus compared to other brain regions [16]. The role of the tail and anterior hypothalamus in the temperature regulation system should be considered during interpretation of these results. In our study, no specific body region was chosen as the target region. Whole body application of EMWs can eliminate the misinterpretation caused by regional applications. Carratala and Moya used microwaves as an indicator of febrile seizures in neonatal mice and concluded that they had no harmful effects [17]. Body temperature was not measured in the current study. Different effects of EMW exposure on seizure latency might be attributed to blood-brain barrier (BBB) damage caused by heating effect of EMWs, but this effect was reported in studies using gigahertz-levels of EMWs [18,19]. We did not perform any histological examination of BBB damage.

There have been some electroencephalography (EEG) studies of EMW exposure [20,21]. For example, awake and healthy individuals were exposed to 900 MHz EMW and no significant EEG changes were noted [21]. Hietanen et al. used 5 different models of cell phones and did not find any significant changes in resting EEG, but noted some EEG changes during memory tests [22]. Vorobyov et al. compared the effect of scopolamine in the electroencephalogram of rats and found that repeated low-level exposure to extremely low frequency microwaves can alter activity of the cholinergic system in the brain [23]. In our study we did not perform EEG recording.

Some studies have investigated the effects of EMWs on nervous system tissue. Carballo-Quintás et al. found c-fos and glial markers were increased by the combined stress of non-thermal irradiation and the toxic effect of picrotoxin on cerebral tissues exposed to 900 MHz [24]. In study of López-Martín et al., 900 MHz GSM radiation stimulated c-fos expression in different areas of the limbic system and triggered a marked increase in neuronal excitability in seizure-prone rats [4]. Ammari et al. showed that sub-chronic exposures to a 900 MHz EMF signal for 2 months could adversely affect rat brains (indicating potential gliosis) [25]. Also, adverse effects of free radicals on myocardium have been shown previously [26]. Tissue investigation was not performed in our study.

Servantie et al. investigated the effects of 5±1 mW/cm^2 EMW on PTZ-induced seizure latency in mice. Chronic EMW exposure of different durations were tested, and the most significant shortening in latency was noted at day 27 [27]. Although the shortest latencies were recorded in the 2-hour group in our study, obvious short latencies were also recorded in the 12- and 20-hour groups. These results suggest that the duration of EMW exposure is not the only factor affecting the occurrence of epileptic seizures.

Previous studies have shown a trigger effect of EMWs on seizure activity [4,7]. Our results also showed a trigger effect of EMWs on seizures by shortening initial seizure latency. However, there was no significant effect on most severe seizure latencies. This suggests that EMWs affect only seizure threshold, without any effect on seizure severity.

Canseven et al. did not find any effect of 50 Hz EMWs on PTZ-induced epileptic seizures [28]. Among our study groups, the shortest initial seizure latency was 9.5±12.9 seconds in the 700 MHz group at 12 hours exposure. Higher frequencies of EMW were used in our study and we found to have significant effects on initial latency of epileptic seizures. This result may indicate a relationship between the seizure threshold and higher frequencies of EMWs.

Conclusions

Our findings suggest that acute exposure to EMWs may facilitate occurrence of epileptic seizures and that this could be independent of EMW exposure time. This information might be important for patients with epilepsy. Further studies are needed to evaluate the acute effects of EMW exposure generated by cell phones and other electromagnetic devices used in everyday modern daily life.

Footnotes

Statement

There is no conflict of interest in this study.

Source of support: Self financing

EXHIBIT A

References

1. Hossmann KA, Hermann DM. Effects of electromagnetic radiation of mobile phones on the central nervous system. Bioelectromagnetics. 2003;24(1):49–62. [PubMed] [Google Scholar]

2. Zhao TY, Zou SP, Knapp PE. Exposure to cell phone radiation up-regulates apoptosis genes in primary cultures of neurons and astrocytes. Neurosci Lett. 2007;412:34–38. [PMC free article] [PubMed] [Google Scholar]

3. Ragbetli MC, Aydinlioğlu A, Koyun N, et al. Effect of prenatal exposure to mobile phone on pyramidal cell numbers in the mouse hippocampus: a stereological study. Int J Neurosci. 2009;119(7):1031–41. [PubMed] [Google Scholar]

4. López-Martín E, Relova-Quinteiro JL, Gallego-Gómez R, et al. GSM radiation triggers seizures and increases cerebral c-Fos positivity in rats pretreated with subconvulsive doses of picrotoxin. Neurosci Lett. 2006;398:139–44. [PubMed] [Google Scholar]

5. Balanis CA. Antenna Theory, Analysis, and Design. 2nd ed. John Wiley & Sons; New York: 1997. pp. 249–338. [Google Scholar]

6. Kral R, LJK, Penry BG, et al. Antiepileptic drug development. II Anticonvulsant drug screening. Epilepsia. 1978;19:409–28. [PubMed] [Google Scholar]

7. Bashkatova V, Vitskova G, Narkevich V. Nitric oxide content measured by ESR-spectroscopy in the rat brain is increased during pentylenetetrazole induced seizures. J Mol Neurosci. 2000;14:83–190. [PubMed] [Google Scholar]

8. Ziylan Z, Ateş N. Age releated changes in regional pattern of blood brain breakdown during epileptiform seizures induced by pentilenetetrazol. Neuroscience Lett. 1989;96:179–84. [PubMed] [Google Scholar]

9. White HS, Johnson M, Wolf HH, et al. The early identification of anticonvulsant activity: role of the maximal electroshock and subcutaneous pentylenotetrazol seizure models. Ital J Neurol Sci. 1995;16:73–77. [PubMed] [Google Scholar]

10. Fedorowski A, Steciwko A, Rabczynski J. Influence of low-frequency electromagnetic field, on growth of endogenous Morris hepatoma and its metastatic ability. Med Sci Monit. 1998;4(5):BR765–73. [Google Scholar]

11. Beniashvili D, Avinoach'm I, Baasov D, Zusman I. The role of household electromagnetic fields in the development of mammary tumors in women: clinical case-record observations. Med Sci Monit. 2005;11(1):CR10–13. [PubMed] [Google Scholar]

12. Tattersall JE, Scott IR, Wood SJ, et al. Effects of low intensity radiofrequency electromagnetic fields on electrical activity in rat hippocampal slices. Brain Res. 2001;15;904(1):43–53. [PubMed] [Google Scholar]

13. Bortkiewicz A, Gadzicka E, Szymczak W, Zmyślony M. Changes in tympanic temperature during the exposure to electromagnetic fields emitted by mobile phone. Int J Occup Med Environ Health. 2012;25(2):145–50. [PubMed] [Google Scholar]

14. Swicord M, Morrissey J, Zakharia D, et al. Dosimetry in mice exposed to 1.6 GHz microwaves in a carrousel irradiator. Bioelectromagnetics. 1999;20:42–47. [PubMed] [Google Scholar]

EXHIBIT A

15. Adair ER, Cobb BL, Mylacraine KS, et al. Human exposure at two radio frequencies (450 and 2450 MHz): Similarities and differences in physiological response. Bioelectromagnetics. 1999;20:12–20. [PubMed] [Google Scholar]

16. Chou CK, Guy AW, McDougall JA, et al. Specific absorbtion rate in rats exposed to 2, 450-MHz microwaves under seven exposure conditions. Bioelectromagnetics. 1985;6:73–88. [PubMed] [Google Scholar]

17. Carratala F, Moya M. Febrile convulsions induced by microwaves and the alteration in behavior of albino mouse of OF1. Biol Neonate. 1991;60:62–68. [PubMed] [Google Scholar]

18. Neilly JP, Lin JC. Interaction of ethanol and microwaves on the blood-brain barrier of rats. Bioelectromagnetics. 1986;7:405–14. [PubMed] [Google Scholar]

19. Salford LG, Brun A, Sturesson K, et al. Permeability of the blood brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, 200 Hz. Microscopy Tech. 1994;27:535–42. [PubMed] [Google Scholar]

20. Regel SJ, Tinguely G, Schuderer J, et al. Pulsed radio-frequency electromagnetic fields: dose-dependent effects on sleep, the sleep EEG and cognitive performance. J Sleep Res. 2007;16:253–58. [PubMed] [Google Scholar]

21. Roschke J, Mann K. No short term effects of digital mobile radio telephone on the awake human electroencephalogram. Bioelectromagnetics. 1997;18:172–76. [PubMed] [Google Scholar]

22. Hiatenen M, Kovala T, Hamalainen AM. Human brain activity during exposure to radiofrequency fields emitted by cellular phones. Scand J Work Environ Health. 2000;2:87–92. [PubMed] [Google Scholar]

23. Vorobyov V, Pesić V, Janać B, Prolić Z. Repeated exposure to low-level extremely low frequency-modulated microwaves affects baseline and scopolamine-modified electroencephalograms in freely moving rats. Int J Radiat Biol. 2004;80:691–819. [PubMed] [Google Scholar]

24. Carballo-Quintás M, Martínez-Silva I, Cadarso-Suárez C, et al. A study of neurotoxic biomarkers, c-fos and GFAP after acute exposure to GSM radiation at 900 MHz in the picrotoxin model of rat brains. Neurotoxicology. 2011;32:478–94. [PubMed] [Google Scholar]

25. Ammari M, Gamez C, Lecomte A, et al. GFAP expression in the rat brain following sub-chronic exposure to a 900 MHz electromagnetic field signal. Int J Radiat Biol. 2010;86(5):367–75. [PubMed] [Google Scholar]

26. Tayefi H, Kiray A, Kiray M, et al. The effects of prenatal and neonatal exposure to electromagnetic fields on infant rat myocardium. Arch Med Sci. 2010;6(6):837–42. [PMC free article] [PubMed] [Google Scholar]

27. Servantie B, Bertharion G, Joly R. Effect of a very high frequency electromagnetic radiation on sensitivity to pentetrazol in white mice. C R Seances Soc Biol Fil. 1971;165:1952–56. [PubMed] [Google Scholar]

28. Canseven AG, Keskil ZA, Keskil S, Seyhan N. Pentylenetetrazol-induced seizures are not altered by pre- or post-drug exposure to a 50 Hz magnetic field. Int J Radiat Biol. 2007;83(4):231–35. [PubMed] [Google Scholar]

EXHIBIT A

EXHIBIT A

EXHIBIT B

Dr. Michael Semelka, DO

Effect of 450 MHz Microwave Modulated with 217 Hz on Human EEG in Rest

Article *in* The Environmentalist · December 2005

DOI: 10.1007/s10669-005-4279-5

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EXHIBIT B

The Environmentalist, 25, 165–171, 2005

Effect of 450 MHz Microwave Modulated with 217 Hz on Human EEG in Rest

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Summary. This study focuses on discrimination of changes, produced by low-level microwave exposure in intensity and time variability of the human EEG at rest. The power spectral density (PSD) method and nonlinear scaling analysis of the length distribution of low variability periods (LDLVP) were selected for analysis of the EEG signal. During the study, 19 healthy volunteers were exposed to a microwave (450 MHz) of 217 Hz frequency on-off modulation. The field power density at the scalp was 0.16 mW/cm^2. The experimental protocol consisted of ten cycles of repetitive microwave exposure. Signals from frontal, temporal, parietal and occipital EEG channels on EEG theta, alpha and beta rhythms were analysed. Exposure to microwave causes average increase of EEG activity. LDLVP analysis discriminated significant effect in time variability for 2 subjects (11%). PSD method detected significant changes in intensity for 4 subjects (21%). The effect of low-level microwave exposure is stronger on EEG beta rhythm in temporal and parietal regions of the human brain.

Keywords: EMF effects, nonionising radiation, microwave radiation, time variability, scaling analysis, spectral analysis, EEG rhythms

1. Introduction

Starting with the new era of portable telecommunication solutions, artificial electromagnetic fields present stronger radiation than the fields created by natural sources. For most of the time, people may not be aware of such radiation, so they solely rely on safety standards.

Modulated microwave radiation at non-thermal level of field power density can affect human central nervous system in a sensible way (D'Andrea *et al.*, 2003; Salford *et al.*, 2003). Except in unhealthy artificial conditions, the effect of electromagnetic field is weak and difficult-to-detect. With carefully planned measurement technique and recording protocol, the measurement of the bioelectrical activity of the brain has been proven to be one of the most successful ones and selected as our primary data source. The measurement and data analysis must take into account the normal fluctuations of EEG signal and presence of other complicate detectable factors. Thus, quantitative measures should be carried out to estimate the overall effect.

During recent years, non-thermal effect of low-level electromagnetic field on human nervous system has become a subject of discussions. The reports of

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possible non-thermal effects are often contradictory. Several investigators have reported that low-level exposure produces alterations in the EEG signal and brain behavior (Bawin *et al.*, 1973; Vorobyov *et al.*, 1997; Mann *et al.*, 1996; Wagner *et al.*, 1998; Huber *et al.*, 2000; Lass *et al.*, 2002; Hinrikus *et al.*, 2004). The conclusion reported by the other researchers is that the exposure to electromagnetic field does not alter the resting EEG (Hietanen *et al.*, 2000; Krause *et al.*, 2000; Krause *et al.*, 2000). Mechanisms behind the effects are still unclear and the question about the existence of any feasible effect of a low-level radiation on brain bioelectric activity has been left open.

In our previous studies the relative changes in the EEG rhythms energy, mainly in theta and alpha bands, were investigated and effect, produced by microwave exposure, reported (Hinrikus *et al.*, 2004). Modulation of microwave at 7 Hz frequency, which belongs to the band of physiological frequencies of the brain, was applied. However, those results did not present statistically important changes. Likewise, the power spectrum analysis could not differentiate sham recordings from recordings under the influence of microwave stimulation at 7 Hz on-off modulation. However, nonlinear scaling analysis of the length distribution of low variability periods (LDLVP) detected significant effect of exposure to the EEG signal for about 25% of subjects (Bachmann *et al.*, 2005). Increase in EEG variability, caused by microwave exposure, was reported.

The analyzed frequencies are lower, than modulation and pulse frequencies in technical systems. Therefore, here we study the physiological effect of the modulation frequency 217 Hz. To this end, we compare the EEG signals recorded at the presence of a modulated low-level microwave field, with sham signals. 217 Hz is the GSM signal's pulse frequency and the population is most widely exposed to microwave modulated at that frequency. The mechanisms of low-level microwave interaction with biological tissues are not clear. Therefore, it is not possible to predict the character of changes in brain bioelectrical activity, caused by microwave exposure. The effect could be related to stimulation or depression of the brain activity, which leads to changes in intensity of the EEG signal. The effect could be related to changes on neurons spiking frequency or processes in synapses, which leads to changes in time variability of the EEG signal. Experimental effects that depend on low frequency modulation of microwave radiation can also be related to more complicated nonlinear responses in biological tissue and living cells (Balzano *et al.*, 2003). Therefore, two different methods for analysis of the EEG signals were used in this study: the first for discrimination of changes in intensity, and the second for discrimination of changes in time variability of the EEG signals.

The intensity of the EEG signal is most completely described by power spectrum of the EEG signal. The power spectral density method, a widely used method in quantitative EEG, was selected for intensity analysis of the EEG signals. The powers of EEG theta, alpha and beta rhythms bands were analyzed.

The LDLVP analysis provides a simple route to detecting the multifractal characteristics of a time-series and yields somewhat better temporal resolution than the traditional multifractal analysis. Thus, it can be expected that this method is sensitive with respect to small "hidden" changes in such a complicated physiological signal as EEG. The LDLVP method was selected for time variability analysis of the EEG signals.

The hypothesis, evaluated in this study, is that modulated at 217 Hz microwave exposure increases variability of the EEG signal and causes changes in the power spectrum of the human EEG.

2. Method and equipment

2.1. *Subjects*

An experimental study was carried out on a group of volunteers. The group consisted of 19 healthy, young people (aged 21–24): 8 male and 11 female. Their physical and mental condition (tiredness, sleepiness) before the experiment was evaluated by a questionnaire and a clinical interview. After the recordings, they described how they felt during the experiment. The subjects reported neither alertness nor any strain experienced during the recordings.

The experiments were conducted with the understanding and written consent of each subject. The study was conducted in accordance with the Declaration of Helsinki and has formally approved by the local Medical Research Ethics Committee.

The measurements were performed in a dark laboratory, but no other special conditions were provided. The subjects lay in a relaxed position, with eyes closed and ears blocked during the experiments.

All the subjects were exposed and sham exposed. Only one experimental EEG recording was performed for a subject during a day. The measurements were

EXHIBIT B

double blinded. During each test session, the exposed and sham-exposed subjects were randomly assigned. The subjects were not informed of their exposure; however, they were aware of the possibility of being exposed. Subjective factors were also excluded from the computer-performed data analysis: the same algorithms were applied for all the recordings (both for exposed and sham-exposed subjects).

2.2. *Microwave exposure*

The modulated microwave radiation at non-thermal level of field power density, identical to our previous studies, except modulation frequency (LASS *et al.*, 2002), was used. Microwave exposure conditions were the same for all subjects. The 450 MHz microwave radiation was 100% amplitude modulated at 217 Hz frequency (duty cycle 50%). The 1 W EMF output power was guided by a coaxial lead to the 13 cm quarter-rhythm antenna, located 10 cm from the subject's skin on the left side of the head.

Estimated field power density at the skin was 0.16 mW/cm^2. The level of power density was so low that thermal effects were extremely unlikely.

2.3. *Recording protocols and equipment*

The study consisted of two experimental protocols, identical for all subjects. The first protocol was recorded as described below.

First, the reference EEG was recorded over 60 s. Secondly, modulated microwave radiation was applied. The duration of the exposure was 60 s, and the compensatory pause after the exposure was 60 s. Continuous EEG recordings were made during and 60 s after exposure. The procedure of the cycle was repeated ten times. The microwave exposure was switched on every first 60 s of the cycle. During ten cycles of microwave exposure, the modulation frequency always remained at 217 Hz.

The recording protocol for one subject lasted for 21 min, during which the EEG was continuously recorded.

The second protocol for the sham-exposure included the same steps, except that the microwave generator was switched off.

The Cadwell Easy II EEG measurement equipment was used for the EEG recordings. The EEG was recorded by means of 19 electrodes, placed on the subject's head according to the international 10–20-electrode position classification system, with Cz as reference. The recorded EEG signals were examined by an experienced neurologist. Artifacts were detected by visual inspection. The recordings containing multiple artifacts were removed, and the whole recording was repeated.

For the analysis, EEG spectrum 0.5–40 Hz was selected, as the results of the preceding validation of the set-up confirmed the absence of modulation components, caused by parasitic interference between EEG and radio frequency equipment.

2.4. *Selection of signals*

Recordings from the following channels were selected for further power analysis: frontal: FP1, FP2; temporal: T3, T4; parietal: P3, P4; occipital: O1, O2. For scaling analysis, only channels FP1 and FP2 were used, as formerly shown, the results from different EEG channels did not differ (Bachmann *et al.*, 2005).

Initially, all the EEG recordings were divided into two sub-signals. The recordings performed with the first recording protocol were divided as follows: the first subsignal contained all 1 min periods without microwave exposure (all the odd minutes from the initial EEG recording); the second subsignal contained all minutes with microwave exposure (all even minutes of the initial EEG recording).

The recordings performed with the second recording protocol (sham) were divided similarly: the first sham subsignal contained all the odd minutes; the second sham subsignal contained all the even minutes of the initial recording.

2.5. *Scaling analysis of the EEG signal based on the LDLVP method*

The LDLVP method has been used and described in details in our previous studies (Kalda *et al.*, 2001; Säkki *et al.*, 2004; Bachmann *et al.*, 2005). The analysis consists of several steps.

First, we define the local variability as the deviation of the current value of the signal from the local average. The time-window width T, for the local average, is a free (adjustable) parameter. For EEG signals, a reasonable value is provided by $T = 60$ ms, cf (Bachmann *et al.*, 2005).

Secondly, low-variability periods are defined as continuous intervals where local variability is smaller than δ_0. The value of δ_0 was adjusted for each record-

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ing individually, reaching a minimum value so that, for both subsignals, the length of the longest low-variability period was at least 3750 ms.

Finally, the number of low-variability periods N exceeding length T_0 is plotted against length T_0.

The weighted area of the function $T_0 = T_0(N)$ was selected as the non-linear quantitative measure.

2.6. *Power spectral density analysis*

The power spectral density (PSD) was estimated by means of Welch's averaged periodogram method. The subsignals were divided into overlapping sections (50%), with a length of 2048 points, and windowed by a Hanning window.

Afterwards, the power W_{mnf} was computed for each subject (indexed by $n \in [1, 19]$), subsignal (indexed by $m = 1, 2$) and frequency band ($f = \theta$ for theta band [4–8 Hz], $f = \alpha$ for alpha band [8–13 Hz] and $f = \beta$ for beta band [13–40 Hz]), as the area under the spectrum for the corresponding frequency band (integral of the band).

To locate the possible influences of microwave exposure, difference of two sub-signals was selected as the PSD measure for further analysis.

The same procedure was repeated with sham subsignals, resulting in spectral powers $\bar{W}_{mnf}$.

2.7. *Statistical analysis*

For sham recordings, subsignals were completely equivalent. According to the "zero hypothesis", the EEG recordings of subjects under microwave exposure cannot be distinguished from sham signals. Consequently, if the zero hypothesis is true, the ratio of the computed power difference to the standard deviation of the differences is an f-distributed random quantity and it can be used as a quantitative measure, showing how well the zero hypothesis is satisfied; respective p-values are obtained by means of the cumulative f-distribution.

The same technique has been applied to the non-linear quantitative measure (derived from LDLVP), resulting in another series of p-values.

3. Results

The results of LDLVP analysis for a subject are presented in Fig. 1. The number of low-variability periods N exceeding the length T_0 is plotted versus the length T_0 for the first and second sub-signal for exposed recording. As can be seen, microwave exposure lowers the curve at the right-hand part of the graph (large values of T_0). Such a change in curve indicates that microwave exposure increases variability of the EEG signal: owing to higher variability there are fewer long low variability periods.

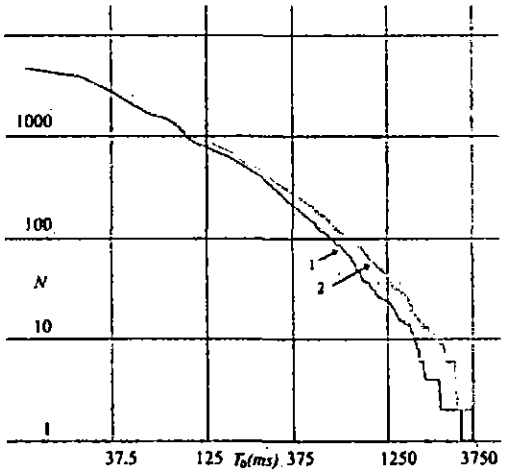

Figure 1. The number of low-variability periods N exceeding the length T_0 for a significant subject: Line 1—second sub-signal of exposed recording (intervals with microwave); line 2—first sub-signal of exposed recording (intervals without microwave).

Figure 2 presents the average values of calculated relative changes in PSD measures for different frequency bands, for exposed and sham recordings. While in sham recordings the power in theta frequency band increases, the power decreases for alpha and beta frequencies. Average values of the measure for microwave-exposed recordings are always positive, therefore, the power of all frequency bands is increasing during microwave stimulation.

Statistical analysis of the PSD and LDLVP quantitative measures for microwave-exposed and sham recordings, were calculated for each subject. The ratio of the computed power difference to the standard deviation of differences of more than three, and p values not larger than 0.001 were considered as significant deviations from the zero hypothesis.

The analysis based on the PSD measures resulted in the ratio of the computed power difference to the

EXHIBIT B

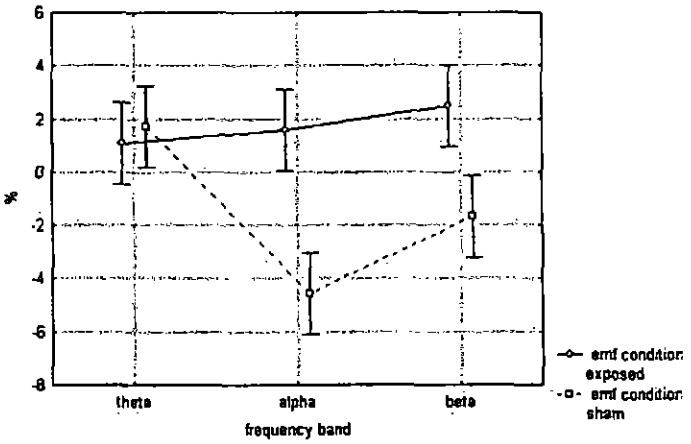

Figure 2. Calculated relative changes in intensity between exposed and non-exposed segments of the EEG signals on main EEG rhythms. Vertical bars denote 0.95 confidence intervals.

standard deviation of differences (calculated on the basis of sham signals) being higher than three for 12 cases in the case of microwave exposure. For sham recordings, 2 cases were significant. The analysis based on the LDLVP measures for exposed recordings resulted in the ratio of the computed power difference to the standard deviation of differences exceeding the value of three for 2 subjects and for no subjects in case of the sham recordings.

All of the results, except for two cases in frontal region (PSD measures), remain significant even after application of the modified Bonferroni correction. The number of subjects having significant results after Bonferroni correction for microwave exposed and sham recordings are presented in *Table 1*. As for PSD measures, there was no significant result for theta and alpha frequency band, only beta band is presented.

Table 1. Number of subjects with significant results after Bonferroni correction in the case of microwave exposed and sham recordings

Method	LDLVP	PSD							
Frequency band	*full EEG*	*beta*							
Channel	FP1/FP2	FP1	FP2	T3	T4	P3	P4	O1	O2
Exposed	2	0	0	3	4	2	1	0	0
Sham	0	0	0	1	1	0	0	0	0

4. Discussion

LDLVP measures resulted in significant results for two subjects in the case of microwave exposure and for none of the subjects in the case of sham recordings (Table 1). Accordingly, significant effect of exposure to the EEG signal was detected for about 11% of subjects. However, for one subject under the exposure, the computed LDLVP weighted area decreased and for other, it increased. For both subjects, the departure from the sham behavior is statistically reliable. This is somewhat different from what has been observed for the modulation frequency 7 Hz, when the sign of the departure was always negative (corresponding to increased *variability*) (Bachmann *et al.*, 2005). This observation gives us a hint that the physiological effect of the microwave stimulation depends on the modulation frequency (at least there is a difference between the 7 Hz and 217 Hz frequencies).

The PSD measures exceeded the limit of significant deviation from zero hypothesis only in beta frequency band (Table 1). In temporal region, the PSD measures provided the most results: 3–4 cases out of 19, ~16–21%. The influence was somewhat smaller in parietal region: 1–2 cases out of 19, ~5–11%. The frontal region did not present significant changes after Bonferroni correction, while occipital region did not present any significant change.

As for sham recordings, the PSD measure resulted in significant results for one subject in channel $T3$ and

in *T*4. However, those were very close to the limit of significance −0.05—and therefore, can be arguably explained with the statistical variability.

Looking at the average values calculated for sham recordings (Fig. 2), one can see that the results are in a good agreement with the findings of Maltez *et al.*, 2004. They showed that alpha and beta power decreased towards the end of recording session during resting conditions, while delta and theta power showed a systematic increase. Except for delta power, which was not under investigation, our results showed the same trend.

However, average values for microwave-exposed recordings reveal an increase of power for all frequency bands. For theta frequency band the level is almost the same as for sham recordings, referring probably to the normal time course and variability of power. At the same time, the average values for alpha and beta band are opposite from sham, implying to the influence of microwave stimulation by increase of power.

The analysis by the LDLVP and PSD methods detected the effect of exposure for about 11 and 21% of subjects respectively. For instance, the rate of multiple chemical sensitivity (MCS) occurrence is estimated to be between 2 and 10% in the general population (Cullen, 1987). MCS is characterized by recurrent symptoms involving multiple organ systems and occurring in response to demonstrable exposures to multiple chemically unrelated compounds at doses far below those established to cause harmful effects. Taking this into consideration, low-level microwave exposure influences even higher part of population than multiple chemically unrelated compounds.

Conclusion

1. Modulated at 217 Hz low-level 450 MHz microwave exposure produced statistically significant changes in time variability and intensity of the EEG signal for 10–20% of healthy subjects.
2. The effect of low-level 450 MHz microwave exposure is stronger on EEG beta rhythm in temporal and parietal regions of the human brain.
3. Exposure to modulated at 217 Hz low-level 450 MHz microwave causes average increase in EEG activity.

The mechanism of these changes is not clear and the effect needs further investigation.

Acknowledgments

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References

Bachmann, M., Kalda, J., Lass, J., Tuulik, V., Säkki, M. and Hinrikus, H.: 2005, 'Non-Linear Analysis of the Electroencephalogram for Detecting Effects of Low-Level Electromagnetic Fields,' *Med Biol Eng Comput* **43**, 142–149.

Balzano, Q. and Sheppard, A.: 2003, 'RF Nonlinear Interaction in Living Cells-I: Nonequilibrium Thermodynamic Theory,' *Bioelectromagnetics* **24**, 473–482.

Bawin, S.M., Gavalas-Medici, R.J., Adey, WR.: 1973, 'Effects of Modulated Very High Frequency Fields on Specific Brain Rhythms in Cats,' *Brain Res* **58**, 365–84.

Cullen, M.R.: 1987, 'Workers with Multiple Chemical Sensitivities Occupational Medicine: State of the Art Reviews', *Philadelphia: Hanley & Belfus, Inc.* **2**, 655–661.

D'Andrea, J.A., Chou, C.K., Johnston, S.A. and Adair, E.R.: 2003, 'Microwave Effects on Nervous System,' *Bioelectromagnetics S6: 107–147.*

Hietanen, M., Kovala, T. and Hamalainen, A.M.: 2000, 'Human Brain Activity During Exposure to Radiofrequency Fields Emitted by Cellular Phones,' *Scand J Work Environ Health* **26**, 87–92.

Hinrikus, H., Parts, M., Lass, L. and Tuulik, V.: 2004, 'Changes in Human EEG Caused by Low-Level Modulated Microwave Exposure,' *Bioelectromagnetics* **25**(6), 431–440.

Huber, R., Graf, T., Cote, K.A., Wittmann, L., Gallmann, E., Matter, D., Schuderer, J., Kuster, N., Borbely, A.A. and Achermann, P.: 2000, 'Exposure to Pulsed High-Frequency Electromagnetic Field During Waking Affects Human Sleep EEG,' *Neuroreport* **11**, 3321–3325.

Kalda, J., Säkki, M., Vainu, M. and Laan, M.: 2001, 'Zipf's Law in Human Heartbeat Dynamics', Available: http://arxiv.org/abs/physics/0110075.

Krause, C.M., Sillanmäki, L., Koivisto, M., Häggqvist, A., Saarela, C., Revonsuo, A., Laine, M. and Hämäläinen, H.: 2000, 'Effects of Electromagnetic Field Emitted by Cellular Phones on the EEG During a Memory Task,' *Neuroreport* **11**, 761–764.

Krause, C.M., Sillanmäki, L., Koivisto, M., Häggqvist, A., Saarela, C., Revonsuo, A., Laine, M. and Hämäläinen, H.: 2000, 'Effects of Electromagnetic Fields Emitted by Cellular Phones on the Electroencephalogram During a Visual Working Memory Task,' *Int J Radiat Biol* **76**, 1659–1667.

Lass, J.V., Tuulik, R., Ferenets, R., Riisalo, and Hinrikus, H.: 2002, 'Effects of 7 Hz-Modulated 450 MHz Electromagnetic Radiation on Human Performance in Visual Memory Tasks,' *Int. J. Radiat. Biol* **78**, 937–944.

Maltez, J., Hyllienmark, L., Nikulin, V.V. and Brismar, T.: 2004, 'Time Course and Variability of Power in Different Frequency Bands of EEG During Resting Conditions', *J. Clin. Neurophysiol.* **34**, 195–202.

Mann, K. and Roschke, J.: 1996, 'Effects of Pulsed High-Frequency Electromagnetic Fields on Human Sleep,' *Neuropsychobiology* **33**, 41–47.

EXHIBIT B

Salford, L.G., Brun, A.E., Eberhardt, J.L., Malmgren, L. and Persson, B.R.: 2003, 'Nerve Cell Damage in Mammalian Brain After Exposure to Microwaves from GSM Mobile Phones,' *Environ Health Perspect* **111**(7), 881–883.

Säkki, M., Kalda, J., Vainu, M. and Laan, M.: 2004, 'The Distribution of Low-Variability Periods in Human Heartbeat Dynamics,' *Physica A* **338**, 255–260.

Vorobyov, V.V., Galchenko, A.A., Kukushkin, N.I. and Akoev, I.G.: 1997, 'Effects of Weak Microwave Fields Amplitude Modulated at ELF on EEG of Symmetric Brain Areas in Rats,' *Bioelectromagnetics* **18**, 293–298.

Wagner, P., Roschke, J., Mann, K., Hiller, W. and Frank, C.: 1998, 'Human Sleep Under the Influence of Pulsed Radiofrequency Electromagnetic Fields: A Polysomnographic Study Using Standardized Conditions,' *Bioelectromagnetics* **19**, 199–202.

EXHIBIT C

Dr. Michael Semelka, DO

Bioelectromagnetics. 2004 Sep;25(6):431-40.

Changes in human EEG caused by low level modulated microwave stimulation.

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Abstract

This study focuses on the effect of low level microwave radiation on human EEG alpha and theta rhythms. During the experiment, 20 healthy volunteers were exposed to a 450 MHz microwaves with 7 Hz on-off modulation. The field power density at the scalp was 0.16 mW/cm2. Signals from the following EEG channels were used: FP1, FP2, P3, P4, T3, T4, O1, and O2. The experimental protocol consisted of one cycle of short term photic and ten cycles of the repetitive microwave stimulation. The changes caused by photic as well as microwave stimulation were more regular on the alpha rhythm. In the majority of cases, photic stimulation caused changes in the EEG energy level in the occipital and microwave stimulation in the frontal region. Our experimental results demonstrated that microwave stimulation effects became apparent, starting from the third stimulation cycle. Changes varied strongly from subject to subject. Therefore, photic and microwave exposure did not cause statistically significant changes in the EEG activity level for the whole group. For some subjects, clear tendencies of changes in microwave on-off cycles were noticeable.

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[Indexed for MEDLINE]

EXHIBIT C

Publication types, MeSH terms +

LinkOut - more resources +

EXHIBIT D

Dr. Michael Semelka, DO

Alterations in Human EEG Activity Caused by Extremely Low Frequency Electromagnetic Fields

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EXHIBIT D

Alterations in Human EEG Activity Caused by Extremely Low Frequency Electromagnetic Fields

D. Cvetkovic, E. Jovanov, *Senior Member*, and I. Cosic, *Senior Member*

Abstract—**This study has investigated whether extremely low frequency (ELF) electromagnetic fields (EMFs) can alter human brain activity. Linearly polarised magnetic flux density of 20μT (rms) was generated using a standard double Helmholtz coils and applied to the human head over a sequence of 1 minute stimulations followed by one minute without stimulation in the following order of frequencies 50, 16.66, 13, 10, 8.33 and 4Hz. We collected recordings on 33 human volunteers under double-blind counter-balanced conditions. Each stimulation lasted for two minutes followed by one minute post-stimulation EEG recording. The same procedure was repeated for the EMF control sessions, where the order of control and exposure sessions was determined randomly according to the subject's ID number. The rest period between two conditions (exposure and control) was 30 minutes. The results indicate that there was a significant increase in Alpha1, Alpha2, and Beta1 at the frontal brain region, and a significant decrease in Alpha2 band in parietal and occipital region due to EMF exposure.**

I. Introduction

Several studies have been conducted to asses whether electromagnetic field (EMF) exposures at characteristic frequencies of brain electrical activity could influence alterations in the EEG and other physiological parameters. Studies on 16.66 Hz and 50 Hz have reported adverse effects on humans and animals [1]-[4]. A single-blind study on 61 volunteers exposed to alternating 3Hz magnetic field of 0.1mT for 20 minutes caused relative spectral power increase at theta and alpha EEG bands and decrease in beta EEG band at the occipital head regions [5]. It was previously reported that applications of electromagnetic fields (EMFs) in the range 0-60Hz and intensity 20 – 100 μT, altered EEG activity in animals and human subjects during 2-second exposure epochs [6]. It was concluded that a weak EMF applied continuously to human subjects for 10 minutes resulted in a reduction in brain electrical activity at the frequency of the EMF during the 1-minute interval following termination of the field. A similar study reported on the effects of 1.5 and 10 Hz EMFs, 20- 40μT, and the results indicated altered brain EEG activity [7]. A recent double blind study on 20 subjects suggested that exposure to ELF magnetic fields altered human EEG activity, specifically within the alpha frequency band (8-13Hz) [8]. The findings indicated that alpha activity was significantly higher over the occipital electrodes and marginally higher over the parietal electrodes at post-exposure.

The purpose of this study was to investigate whether combined extremely low frequencies EMFs (50, 16.66, 13, 10, 8.33 and 4Hz, in that order) which have previously been investigated in literature only as individual frequencies, could cause changes in the EEG activity under double-blind and counter-balanced conditions.

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II. Materials and Methods

A. Design of Helmholtz Coil Magnetic Field Exposure Apparatus

The preliminary [9],[10] and final studies investigated whether multiple sinusoidal extremely low frequency (ELF) (50, 16.66, 13, 10, 8.33 and 4Hz) linearly polarised magnetic flux density of 20±0.57μT (rms) applied to the human head over a non-continuous period of 12 minute, could causes alterations in the EEG rhythms on 33 human volunteers [11]. Standard circular Helmholtz pair of coils have been designed to pass the current of approximately 140mA. The total coil impedance was 71 Ω, designed with average radii of 65cm, copper wire of 0.8mm in diameter and 250 turns each. A signal generator effective in producing high quality sine waveforms of high stability/accuracy ELF signals was designed and developed using EXAR XR-2206 monolithic IC. Also, an audio amplifier was designed and constructed with the approximate gain of 10 to deliver sufficient current to the coils. The magnetic flux density was verified by direct measurement using "Wandel and Goltermann" EFA-200 EMF Analyser. The linearly polarized filed was perpendicular to the Earth's North-South magnetic field.

B. Subjects and EEG Montages and Procedures

The final experiments were conducted on 33 healthy subjects, 24 male and 9 female, with mean age of 30 years, SD 11 years, range 20-59 years. The RMIT ethics committee approved the study and all subjects gave written informed

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consent prior to the experiment. During the EEG recording sessions, subjects were asked to lie down between the coils in saggital plane direction perpendicular to the coil axis and in the supine position. The entire experiment was performed in a darkened and sound proof RF anechoic chamber to prevent erroneous recordings due to the standing waves and power line interference.

C. EEG Recording and Experimental Protocol

The EEG equipment used throughout testing was the Mindset MS-1000 recording system. Neuroscan 19 Channel Caps electrodes were used with referential montage of 16 channels. The left brain hemisphere electrodes: Fp1, F7, F3, T7, C3, P7, P3 and O1 were all referenced to M1 (left mastoid), while the right brain hemisphere electrodes: Fp2, F8, F4, T8, C4, P8, P4 and O2 were referenced to right mastoid M2. The baseline EEG was recorded prior to any stimulation for one minute. Each stimulation (50, 16.66, 13, 10, 8.33 and 4Hz) lasted for two minutes followed by one minute post-stimulation EEG recording. Therefore, total length of an experiment was 19 minutes. The same procedure was repeated for the EMF control sessions. The order of control and exposure sessions was determined randomly according to the subject's ID number. Subjects with odd ID numbers were first tested with control condition (no EMF exposure) followed by EMF stimulation after 30 minute break. Double-blind counterbalanced condition was exercised. The two EMF sessions were highly considered in the analysis as a factor that might reveal that if the 1st session was EMF exposure, the EEG activity results during the 2nd EMF control session could still be influenced or dependent on the results of the 1st EMF exposure session.

III. Signal Processing and Statistical Method

All the collected EEG data was processed using Matlab tool. The main Matlab script was written to process all 16 channel EEG data of all subjects and generate valuable parameters that would be used in the further statistical analysis, such as Total spectral power of each stimulation EEG data (i.e. before, 50Hz, 16.66Hz, 13Hz, 10Hz, 8.33Hz and 4Hz); Spectral power in the stimulated band, before/after; Central band frequency before/after and Relative difference "ratio" between the individual band and total spectral power before/after. Spectral function was written to compute the windowed discrete-time Fourier transform of a signal using a sliding window. The EEG band intervals were as Theta (3-5Hz), Alpha1 (7.5-9.5Hz), Alpha2 (9-11Hz), Beta1 (12-14Hz), Beta2 (15.5-17.5Hz) and Gamma (49-51Hz). Delta and Gamma band data was excluded from this particular analysis. We compared the EEG activity "before" and "after" stimulation for each frequency stimulation and band. Throughout this method, "before" stimulation EEG data was regarded for every next recording of the "after". For example, if 1st recording was before any stimulation, 2nd was 50Hz stimulation (gamma band), 3rd was 16.66Hz stimulation (beta2 band). The script used for this signal processing computed all the parameters mentioned above as 1 second epochs, maximum of 60 epochs per recording. Throughout this investigation, only the relative difference (ratio) parameter between the individual bands and total spectral power (before and after) was used for the statistical analysis.

IV. Results

Multiple paired samples 2-tailed t-tests and ANOVA's 3-way mixed design for within and between-subject measures were employed. The factors considered were the "before and after", "exposure and control" and "first and second session." The first test conducted was for the first session of EMF exposure and there were 16 subjects used for this session. The second test was the second session EMF control ($df=15$), the third test was the first session EMF control ($df=16$) and the fourth test was the second session EMF exposure ($df=16$).

A. EMF Exposure followed by EMF Control Results

In Alpha1 band and 8.33Hz stimulation under EMF control (2nd session), *t*-test results revealed a significant relative difference increase from before to after at T7 ($t(15)= -2.397$, $p<0.030$). ANOVA test revealed a significant difference for the interaction between exposure/control and sessions factors (T7) ($F(1,31)= 5.992$, $p<0.020$). In Alpha2 band after 10Hz stimulation, 2nd control session, the relative difference has decreased, highlighted by a high difference observed in parietal and occipital regions, P3, that the relative difference at before (M=0.1789, SE=0.0201) was significantly higher than after (M=0.1573, SE=0.0140), $t(15)= 3.081$, $p<0.008$. At P4, the relative difference before (M=0.1861, SE=0.0223) was significantly higher than after (M=0.1510, SE=0.0134), $t(15)= 2.812$, $p<0.013$. The occipital regions, O1 before (M=0.1399, SE=0.0156) and after (M=0.1243, SE=0.0111), $t(15)= 2.256$, $p<0.039$; and O2 before (M=0.1383, SE=0.0137) and after (M=0.1203, SE=0.0104), $t(15)= 3.283$, $p<0.005$, as shown in Figure 1. There was a largest decrease in relative difference from before to after by 12% (P3), 18.4% (P4), 11.2% (O1), and 13% (O2) than at any other electrode and stimulation. The 3-way ANOVA revealed a significant difference at the interaction between exposure/control and sessions (P3) $F(1,31)= 11.918$, $p<0.002$ and the main factor before/ after $F(1,31)= 5.230$, $p<0.029$. At P4 electrode, a significant difference between exposure/control and sessions was $F(1,31)= 14.827$, $p<0.001$ and before/after $F(1,31)= 4.406$, $p<0.044$; O1 revealed $F(1,31)=9.346$, $p<0.005$ (exposure/control and sessions); and O2 $F(1,31)= 13.071$, $p<0.001$. The t-test results for 13Hz stimulation in Beta1 band revealed no significant differences at any electrode, as shown in Figure 2.

EXHIBIT D

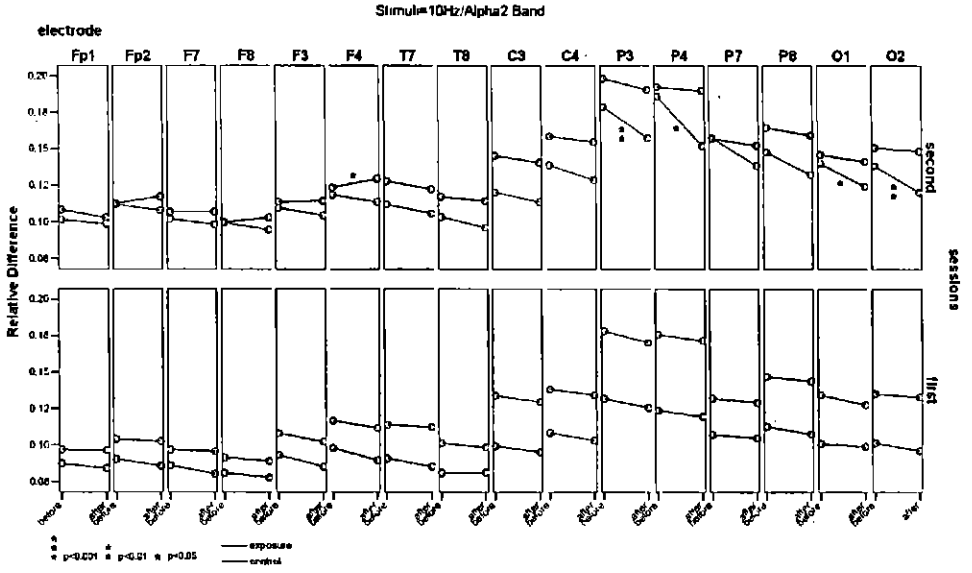

Figure 1. The Relative Differences Versus "Before" and "After" Results Represented at 10Hz Stimulation in a Alpha2 band for EMF Exposure/Control and First/Second Session Conditions.

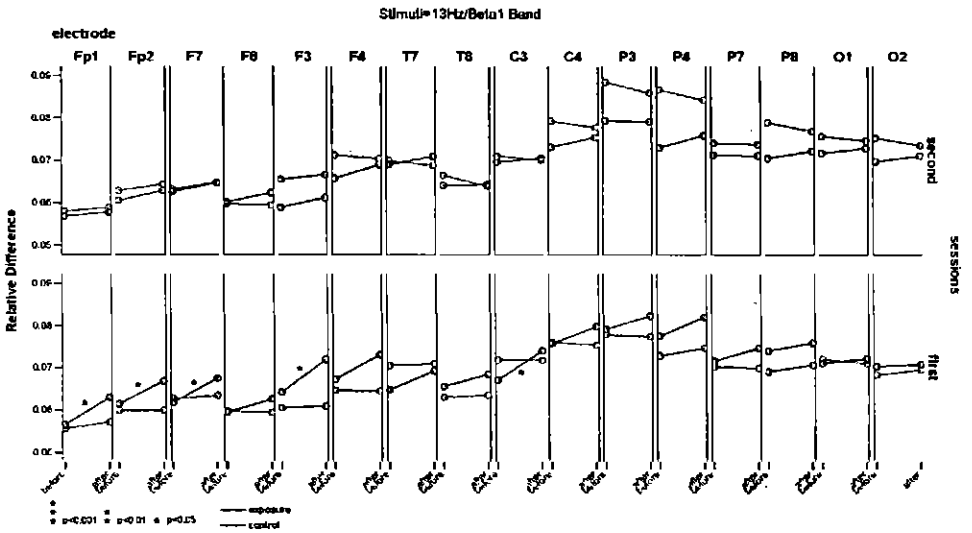

Figure 2. The Relative Differences Versus "Before" and "After" Results Represented at 13Hz Stimulation in a Beta1 band for EMF Exposure/Control and First/Second Session Conditions.

For the 1st EMF exposure session, the t-test results revealed a significant increase at Fp1, Fp2, F7, F3 and C3 for 13Hz stimulation in Beta1 band. At F7 before, $t(1,15)= -2.798$, $p<0.014$; F3 before $t(1,15)= -2.659$, $p<0.018$; and C3 before $t(1,15)= -2.391$, $p<0.030$. There was an increase in relative difference from before to after by 10.1% (Fp1), 8% (Fp2), 8.4% (F7), 10.8% (F3) and 9.3% (C3). The ANOVA results revealed a significant differences between before and after main factors at Fp1 $F(1,31)= 12.852$, $p<0.001$; Fp2 $F(1,31)= 7.058$, $p<0.012$; F7 $F(1,31)= 15.730$, $p<0.0001$; and C3 (NS). In 1st EMF exposure Beta1 band (13Hz), ANOVA's significant results for before and after main factor, were very similar with the t-test's results.

B. EMF Control followed by EMF Exposure Results

For the 2nd EMF exposure session, the t-tests were conducted for 8.33Hz stimulation in Alpha1 band, that relative difference at electrodes Fp1, F7, F3, F4 and C4 was significantly higher before than after stimulation. The results of t-tests were: F7 before $t(1,16)= 2.120$, $p<0.050$; F3 before $t(1,16)= 2.862$, $p<0.011$; F4 before $t(1,16)= 2.682$, $p<0.016$; and C4 before $t(1,16)= 2.872$, $p<0.011$. There was a decrease

in relative difference from before to after by 11.1% (Fp1), 11.3% (F7), 10% (F3), 9.8% (F4) and 8.8% (C4). The ANOVA results indicated a significant difference at: F7 $F(1,31)= 6.485$, $p<0.016$ (exposure/control and sessions) and $F(1,31)= 4.485$, $p<0.042$ (before/after and sessions); F3 $F(1,31)= 4.524$, $p<0.041$ (exposure/control and sessions) and $F(1,31)= 4.297$, $p<0.047$ (before/after and sessions); F4 $F(1,31)= 11.554$, $p<0.002$ (exposure/control and sessions); and C4 $F(1,31)= 5.121$, $p<0.031$ (exposure/control and sessions) and $F(1,31)=6.035$, $p<0.020$ (before/after and sessions). Under the 2nd EMF exposure session, the t-test revealed a significant difference between before and after stimulation of 10Hz in Alpha2 band at F4, where a relative difference was higher before than after the 10Hz stimulation $t(16)=-2.130$, $p<0.049$, as shown in Figure 1. ANOVA revealed a significant difference for the interaction between exposure/control and session's factor, $F(1,31)= 11.043$, $p<0.002$. For 13Hz stimulation, there was no significant difference.

V. Discussion

The statistical EMF exposure/control tests have been conducted and the summary of its entire hypothesis tested have been illustrated in Figure 3.

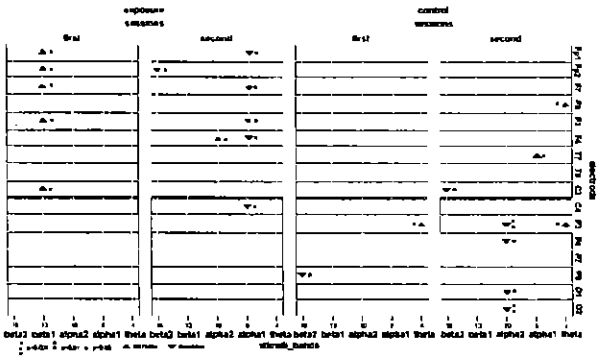

Figure 3. Summary of All the T-test Significant Relative Increase/Decrease for the Standard EMF Exposure/Control and First/Second Session Conditions at Individual Bands/Stimuli and Electrodes.

The alternative hypothesis test for EMF Exposure 1st and Control 2nd session results signify a possibility that the EEG activity could remain altered for at least 50 minutes after the exposure (30 minutes break between the exposure and control conditions with additional 20 minutes for EMF control EEG recordings and stimulations). For the corrected alpha rate value of multiple tests, Bonferroni test was used with the new modified alpha rate of $p<0.0025$. No significant differences were observed as a result of this correction. However, the final analysis results suggest that EEG activity in Alpha1, Alpha2 and Beta1 band could be altered due to EMF exposures, which are mainly associated with stimulation frequencies of 8.33, 10 and 13Hz.

VI. Conclusion

The results from the EEG study on 33 subjects have indicated that under the first EMF exposure there was a shift from a significant increase in Beta1 band at frontal region of the brain to a significant decrease in Alpha2 band at the back region (parietal and occipital) under the post-EMF exposure. However, when the subjects were exposed to EMF after 60 minutes of rest, they exhibited a decrease in Alpha1 band.

Acknowledgment

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References

[1] H. Brendel, M. Niehaus, A. Lerchl, "Direct supressive effects of weak magnetic fields (50Hz and 16.66Hz) on melatonin synthesis in the pineal gland of Djungarian hamsters (Phodopus sungorus)", *Journal of Pineal Research*, vol. 29, pp. 228-233, 2000.

[2] M.J. Azanza, A.C. Calvo, A.D. Moral, "Evidence of synchronisation of neural activity of molluscan brain ganglia induced by alternating 50Hz applied magnetic field", in *Electromagnetic Biology and Medicine, Electromagnetic Biology and Medicine*, 2002.

[3] T. Akerstedt, B. Arnetz, G. Ficca, L.E. Paulsson, A. Kallner, "A 50Hz Electromagnetic field impairs sleep", *Journal of Sleep Research*, vol. 8, pp. 77-81, 1999.

[4] Y. Kurokawa, H. Nitta, H. Imai, M. Kabuto, "Acute Exposure to 50Hz Magnetic Fields with Harmonics and Transient Components: lack of effects on nighttime hormonal secretion in men", *Bioelectromagnetics*, vol. 24, pp. 12-20, 2003.

[5] K. Hausser, D. Tellschaft, F. Thoss, "Influence of an Alternating 3Hz Magnetic Field with an Induction of 0.1 mT on Chosen Parameters of the Human Occipital EEG", *Neuroscience Letters*, vol. 239, pp. 57-60, 1997.

[6] G.B. Bell, A.A. Marino, A. Chesson, "Frequency-Specific Blocking in the Human Brain Caused by Electromagnetic Fields", *Neuro Report*, vol. 5, pp. 510-512, 1994.

[7] G.B. Bell, A.A. Marino, A. Chesson, "Frequency-Specific Responses in the Human Brain Caused by Electromagnetic Fields", *Journal of the Neurological Sciences*, vol. 123, pp. 26-32, 1994.

[8] C.M. Cook, A.W. Thomas, F.S. Prato, "Resting EEG is affected by exposure to a pulsed ELF magnetic field", *Bioelectromagnetics*, vol. 25: pp. 196-203, 2004.

[9] D. Cvetkovic, I. Cosic, "The induced rhythmic oscillations of neural activity in the human brain" in *Proc. IASTED (BIOMED) Conf.*, ACTA Press, 2004.

[10] D. Cvetkovic, I. Cosic, "Automated ELF Magnetic Field Stimulation on the Human EEG Activity", *Integrated Computer-Aided Engineering, IOS Press*, 2006 (accepted).

[11] D. Cvetkovic, "Electromagnetic and Audio-Visual Stimulation of the Human Brain at Extremely Low Frequencies" Ph.D. dissertation, School of Electrical and Computer Engineering, RMIT Univ., Melbourne, Australia, 2005.

EXHIBIT E

Dr. Michael Semelka, DO

Format: Abstract

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Long-Term Evolution Electromagnetic Fields Exposure Modulates the Resting State EEG on Alpha and Beta Bands.

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Abstract

Long-term evolution (LTE) wireless telecommunication systems are widely used globally, which has raised a concern that exposure to electromagnetic fields (EMF) emitted from LTE devices can change human neural function. To date, few studies have been conducted on the effect of exposure to LTE EMF. Here, we evaluated the changes in electroencephalogram (EEG) due to LTE EMF exposure. An LTE EMF exposure system with a stable power emission, which was equivalent to the maximum emission from an LTE mobile phone, was used to radiate the subjects. Numerical simulations were conducted to ensure that the specific absorption rate in the subject's head was below the safety limits. Exposure to LTE EMF reduced the spectral power and the interhemispheric coherence in the alpha and beta bands of the frontal and temporal brain regions. No significant change was observed in the spectral power and the inter-hemispheric coherence in different timeslots during and after the exposure. These findings also corroborated those of our previous study using functional

magnetic resonant imaging.

EXHIBIT E

KEYWORDS: EEG; electromagnetic field exposure; functional magnetic resonance imaging; long-term evolution; resting state; specific absorption rate

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EXHIBIT F

Dr. Michael Semelka, DO

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EXHIBIT F

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Radiofrequency signal affects alpha band in resting electroencephalogram

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Abstract

The aim of the present work was to investigate the effects of the radiofrequency (RF) electromagnetic fields (EMFs) on human resting EEG with a control of some parameters that are known to affect alpha band, such as electrode impedance, salivary cortisol, and caffeine. Eyes-open and eyes-closed resting EEG data were recorded in 26 healthy young subjects under two conditions: sham exposure and real exposure in double-blind, counterbalanced, crossover design. Spectral power of EEG rhythms was calculated for the alpha band (8–12 Hz). Saliva samples were collected before and after the study. Salivary cortisol and caffeine were assessed by ELISA and HPLC, respectively. The electrode impedance was recorded at the beginning of each run. Compared with the sham session, the exposure session showed a statistically significant ($P < 0.0001$) decrease of the alpha band spectral power during closed-eyes condition. This effect persisted in the postexposure session ($P < 0.0001$). No significant changes were detected in electrode impedance, salivary cortisol, and caffeine in the sham session compared with the exposure one. These results suggest that GSM-EMFs of a mobile phone affect the alpha band within spectral power of resting human EEG.

Keywords: electroencephalogram, radio frequency, mobile phone, alpha band

EMERGING TECHNOLOGIES IN MOBILE telecommunications, such as radio frequency fields (RF) and microwave radiation, are widely used in our modern society. Prominent examples are the wireless Internet network and mobile phone communications, which are particularly widespread. The extensive use of mobile phones (MP) increases the exposure of human beings to radiofrequency electromagnetic fields. During a phone call, given the close proximity of the MP to the user's head, a part of the electromagnetic field (EMF) can be absorbed by the head and the brain (Schönborn et al. 1998). This exposure to EMF has raised questions about possible effects of the EMF of mobile phones on brain activity.

Some earlier studies have investigated the effects of EMFs on resting cerebral activity with somewhat mixed results, but more recently, there has been consistent data, indicating the existence of exposure effects on the alpha bands of the resting EEG.

Indeed, data reported by some authors showed an increase in EEG power in the alpha frequency band (Cook et al. 2004; Croft et al. 2002, 2008, 2010; Curcio et al. 2005; Hinrikus et al. 2008; Huber et al. 2002; Kramarenko and Tan 2003; Regel et al. 2007; Reiser et al. 1995), whereas other studies reported a decrease in EEG power or coherence in the alpha band (Maby et al. 2006; Perentos et al. 2013; Vecchio et al. 2007, 2010, 2012). Finally, other studies failed to show an effect on EEG power in the alpha bands (D'Costa 2003; Hietanen et al. 2000; Röschke and Mann, 1997; Perentos et al. 2007).

As the literature cited demonstrates, the most consistent effect observed is a change in alpha band power. However, these changes sometimes correspond to an increase in alpha power and sometimes to a decrease. The reason why alpha band power reacts differently to RF exposure remains unclear. The main problem lies in the use of different methods, different experimental protocols, and/or different intensities or frequencies (van Rongen et al. 2009), thus making the comparison of data more difficult. As also reported by Loughran et al. (2012), individual variability is also one of the important factors that may explain the discrepancies between the results.

Moreover, several other parameters could impact the EEG results as confounding factors. Among these parameters are electrode impedance changes. The battery and electronics of the phone causes it to heat up, which, in turn, causes heating of the skin and underlying tissue (Anderson and Rowley 2007; Ghosn et al. 2012, Straume et al. 2005). As exposure to heat causes the dilation of blood vessels, this phenomenon may result in a change in the skin impedance (Luck 2005), which, in turn, could explain some observed changes in the recorded EEG power.

In addition, changes in the alpha band power are related to changes in parameters, such as cortisol or caffeine, which, to our knowledge, have never been concretely measured in relation to EMF exposure. Changes in cortisol and ECG could result from stress linked to the experimental environment and protocol, and therefore, these parameters need to be controlled.

The aim of the present study was to examine the potential impact of GSM (global system for mobile) RF (radiofrequency) exposure to the alpha band of the resting EEG under controlled parameters and to, thus, bring additional information to fill certain gaps in our current knowledge of the effects of GSM RF exposure. This study was conducted on awake volunteers in two different conditions: open eyes and closed eyes. In addition, some parameters that are known to affect alpha band, such as electrode impedance, cortisol levels and caffeine concentrations were also investigated to ensure that if any effect was observed,

that it was not attributable to one of the aforementioned parameters. Hence, electrode impedance was checked after each block of EEG recordings, and caffeine and cortisol were concurrently evaluated in the saliva.

EXHIBIT F

METHODS

Participants. Twenty-six healthy volunteers participated in the experiment (13 females and 13 males; mean age = 23.5 ± 3.1 yr). All women reported having regular menstrual cycles (25–32 days) during the year preceding the study, no vasomotor complaints (i.e., hot flashes, night sweats). These women were studied in the laboratory during the follicular phase of their menstrual cycle to avoid any interference with EEG rhythms and hemispheric activity. All participants provided informed written consent and were compensated for their participation. All procedures were approved by the local ethics committee (ID no. = RCB: 2011, A01455-36). The volunteers were selected following a routine clinical examination. The mean body mass index of the subjects was 22.3 ± 1.8. Systolic and diastolic blood pressures were 113.3 ± 9.2 and 74 ± 7.7 mmHg (mean ± SD), respectively. Inclusion criteria included regular sleep habits, no medication, no chronic disease or disability, no recent acute illness, no smoking, and no neurological or psychiatric illness. All participants were right-handed and had normal or corrected-to-normal vision. Those selected were instructed to abstain from consuming alcohol and coffee for 24 h before and during each experimental session. They were instructed to abstain from using a mobile phone on the day of the experiment. Participants declared that they did not use the mobile phone at all on the day of the experiment. Moreover, we are quite sure that they did not use their phones 2 to 3 h before the start of the experiment, since they were admitted into the facility of the hospital to fill some documents related to the experiment 2 to 3 h prior to the exposure.

Experimental design. Participants attended two EEG recording sessions in a crossover, randomized, double-blind, and counterbalanced design experiment. During each session, the subject was exposed to 26 min and 15 s of sham or real GSM RF exposure (Fig. 1). In the case of sham exposure, the mobile phone was switched "on" but without RF radiation, while for real exposure, the mobile phone was switched "on" with RF radiation. For the same subject, the two sessions were at a 1-wk interval. Both the subjects and experimenters were unaware of the exposure condition. The experiment was conducted in a dimly lit, electrically shielded room. Subjects were seated in a comfortable chair, and a screen was placed 1 m in front of the volunteer to keep their eyes in a well-defined direction. In addition to the EEG recordings, ECG and galvanic skin responses (GSR) [also called electrodermal response (EDR)] were simultaneously recorded (EDR data will not be reported in the present paper). During the recordings, volunteers were asked to fix their eyes on a center point on the screen represented by a white square in the center of a black background. Each recording session was composed of seven experimental blocks distributed across the 3 experimental conditions: preexposure, exposure, and postexposure. Each block consisted of three recordings: EDR assay (2 min and 45 s), resting EEG with open eyes (3 min), and resting EEG with closed eyes (3 min) (Fig. 1). Vocal instructions were previously recorded by the experimenter. Loudspeakers placed on either side of the screen in front of the volunteer connected to a computer in the acquisition room allowed instructions to be sent to the volunteers. Auditory instructions to inform the volunteers when recording starts, when to open or to close their eyes, and the fixation point were given with Omnistim (stimulus presentation software developed at the MEG-EEG Center). Transistor-transistor logic (TTL) pulses were used to synchronize stimulus presentation and the EEG/BIOPAC systems. Instructions were at the beginning of the recording block, the open eyes and the closed-eye periods, and at the end of the block.

The timeline of the two experimental sessions is presented in Fig. 1. The preexposure period consisted of two blocks of recordings (*run 1* and *run 2*) with no mobile phone (baseline). Three blocks (*run 3*, *run 4*, and *run 5*) were recorded during the exposure period in which the actual mobile phone (genuine) was

positioned and activated for the exposure session, and the sham phone was used in the sham session. The mobile phone was then removed, and two blocks were recorded in the postexposure period (*runs 6* and *7*).

Exposure system and dosimetry. Subjects were exposed to RF EMF by a commercial dual-band GSM mobile phone (Nokia 6650). The mobile phone was positioned against the left ear. To set the standard exposure parameters, the phone was connected to a personal computer to control the required frequency and RF power by service software (Phoenix, Nokia, Finland). The sham or genuine exposure was carried out using a "load" or a "dummy load", respectively. For this purpose an external power load was connected to the external antenna connector of the phone. A 50-Ω resistive load and an open-circuit dummy load were developed for sham and exposed conditions with the same shape and structure to allow for the double-blind protocol of the study. This implied that, when the telephone was on, the internal circuitry was regularly active, but no radiofrequency power was delivered in space by the antenna. The participants received GSM-modulated exposure with the full power of the mobile phone (2 W peak, 250 mW average, pulse modulated with 1/8 duty cycle) at 900 MHz for 26 min. The maximum specific absorption rates (SARs) were averaged on 10 g tissue, 1 g tissue, and the peak value was measured at 0.49 W/kg, 0.70 W/kg, and 0.93 W/kg, respectively. The SAR of the sham phone was below the detection level of the system (0.001 W/kg) at any position of the phantom, and no electric field was detected on the surface of the sham phone (for more details, see Ghosn et al. 2012).

EEG recording and data acquisition. Electroencephalography data were recorded using BrainCap (EASYCAP Products, Herrsching, Germany) with 29 passive electrodes (Fp1, Fp2, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, PO3, PO4, O1, and O2) placed according to the international extended 10/10 system. The reference electrode was the AFz, and the ground electrodes were placed on the right shoulder of each participant. Repeated EEG blocks were recorded with respect to the AFz reference at a sampling rate of 1,000 Hz. The signal was amplified and band-pass filtered online between 0.016 Hz and 250 Hz. We used three bipolar derivations to monitor eye movements: one electrode was placed below the right eye for vertical eye movements, and two electrodes were placed at the outer canthi of the eyes for horizontal movements. Data acquisition was performed using BRAINAMP MR plus Amplifiers (Brain Products, Munich, Germany).

EEG interference with RF-EMF. To test possible interference between radio frequencies emitted by the mobile phone and EEG signals recorded during exposure, a polystyrene phantom head was constructed to simulate a complete EEG chain. Time-frequency analysis was performed on the three recordings blocks (without a phone, with the sham phone, and the real phone) to detect any interference signals. Results showed no disturbance in the recording in the absence or presence of the two phones for the frequencies between 1 and 20 Hz. The two sham and real phones used in our experiment seem not to have disturbed the EEG recordings assessed during exposure.

Measurement of electrode impedance. Electrode impedance was checked to be below 5 kΩ and was recorded throughout the experiment at the beginning of each run.

Heart rate data acquisition. Heart rate was recorded by BIOPAC MP150 (GSR100C and ECG100C modules) at a sampling rate of 1,000 Hz by using two electrodes. One was placed at the base of the neck (above the right clavicle), while the other was placed on the left forearm.

ELISA for salivary cortisol. Saliva was collected using a Salivette device (Sarstedt) and then centrifuged and immediately frozen. Each volunteer provided two saliva samples, the first before starting the experiment (T0) and the second after the recordings were complete (Tf). The cortisol was quantified in two samples collected at T0 and Tf using commercialized sandwich ELISA kits (human cortisol), according to the manufacturer's instructions.

EXHIBIT F

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Samples were centrifuged (1,000 g/20 min/4°C), and the supernatant was collected. Raw data were presented for sham and exposed groups.

EXHIBIT F

Salivary caffeine concentration using HPLC. Salivary caffeine concentration was assessed in T0 samples. A rapid HPLC method was used for the salivary caffeine analysis. The HPLC system consisted of a Spectra SYSTEM Pump and a Spectra SYSTEM UV detector (Ultimate 3000 Photodiode Array detector). An Envirodur C18 (3 μm) column (250 × 4.6 mm; Macherey Nagel) was used for the separation. The mobile phase was made of 85% of a 0.012 M KH_2PO_4 and 15% acetonitrile. The flow rate was set at 1 ml/min, and the injection volume was set at 20 μl. The detection wave length was set at 280 nm. The caffeine solution concentrations used for the standard curves were 1, 1.5, 2, 5, 8, 15, 25, 50, and 100 μg/ml. Standard curves were constructed by plotting concentration vs. area under the curve. Caffeine retention time was 5.2 min.

EEG data analysis. Resting EEG data were analyzed for the periods "open eyes" and "closed eyes," which lasted 3 min each for each run. In total, 7 runs were performed: the first two runs (*runs 1* and *2*) consisted of the preexposure period, the three following runs (*runs 3*, *4*, and *5*) constituted the exposure period, and the last two runs (*runs 6* and *7*) represented the postexposure period. Markers were placed in the data at 4-s intervals, and then we performed the time-frequency wavelet transform on individual EEG epochs comprising data from −2.5 to 2.5 ms around each marker. We used a family of complex Morlet wavelets, with an *m* parameter of 10 and a Blackman window of 100 ms, resulting in an estimate of signal power at each time sample and at each frequency between 1 and 20 Hz, with a frequency step of 1 Hz. The time-frequency transformed data were then averaged across epochs for each experimental eye condition, each run and for each subject, separately for the baseline trials and the exposure and postexposure trials to obtain spectral power, which were then subsequently averaged in the alpha (8–12 Hz) bands. The alpha band was divided into two subbands: the upper (10–12 Hz) and the lower (8–10 Hz) and were then analyzed. The log-transformation of the data was used to approach a normal distribution. Finally, the data were averaged over the three conditions of interest: preexposure (baseline), during exposure, and postexposure period, for each subject and for the grand mean of the 26 volunteers.

Statistical analysis. A four-way repeated-measures ANOVA was run to determine the effect of exposure (sham or exposed), frequency bands (delta, theta, or alpha), period (before, during, or after), and eye conditions (closed or open) across subjects. Then, we restricted the analysis to alpha band (8–12 Hz) in closed-eye condition as follows: for each period (before, during, and after), we performed a paired *t*-test for each electrode across subjects in the two conditions (real exposure vs. sham exposure). Then, we averaged frequency power values for each portion of the alpha band (8–12 Hz, 8–10 Hz, and 10–12 Hz) on each electrode across subjects and performed a paired *t*-test in the two conditions (sham or real exposure). The family-wise error rate was controlled via permutations tests as showed by Groppe et al. (2011), which is, at most, as conservative as Bonferroni.

Heart rate, impedance, and cortisol data analyses were performed using two-way repeated-measures ANOVA. Statistical significance was set for $P < 0.05$.

RESULTS

EEG interference with RF-EMF. No disturbance was seen in any recording in the absence or presence of the phones (actual or genuine) for the analyzed frequencies between 1 and 20 Hz. The two sham and real phones used in our experiment did not disturb EEG recordings assessed during exposure (data not shown).

Alpha spectral power. There were significant differences between frequency bands and eye conditions for all of the electrode measurements. Period levels (before, during, and after) were statistically significantly different on all electrodes except in the frontal region (Fp1, Fp2, F7, F3, Fz, F4, FC1, FC2).

In the closed-eyes condition, a significant difference between sham and real exposure was found in alpha band power (8–12 Hz) for all electrodes during the exposure (except FP2, FC5, and P8) and postexposure period (except Cz, CP2, P7). Indeed, a paired permutation *t*-test analysis detected a significant and important decrease in alpha band power (8–12 Hz) ($P < 0.0001$) during the exposure and postexposure period ($P < 0.001$) (Table 1).

Furthermore, the alpha band (8–12 Hz) was divided into two subbands—the upper (10–12 Hz) and lower (8–10 Hz) alpha bands—which were analyzed separately. Results showed that in the 8–10 Hz frequency band, alpha spectral power significantly decreased during the exposure and postexposure period ($P < 0.001$ and $P < 0.0001$, respectively). Likewise, data within the upper alpha band (10–12 Hz) showed a decrease in the spectral power during and also after exposure ($P = 0.0001$ and $P < 0.0001$, respectively) (Table 1).

Electrode impedance. Figure 2 represents electrode impedance recorded at the beginning of each run. No significant differences have been detected when comparing sham and real exposure between runs. Repeated-measures two-way ANOVA and Bonferroni post hoc tests were applied. *P* and *F* values are given in Table 2. Impedance was not affected by the factor session (sham and real exposure) recorded 1 wk apart in all runs. Moreover, no significant differences were found in all electrode impedances when comparing the seven runs separately in the sham sessions and in the exposure sessions.

Heart rate. There were no significant variations in heart rate (Fig. 3), whether it be between the two sessions (sham and real exposure), eye condition (open eyes and closed eyes) within and between sessions [two-way ANOVA: exposure ($F = 0.1$, $P = 0.75$), and eye condition ($F = 0.58$, $P = 0.71$)].

Salivary cortisol. Figure 4 shows the salivary cortisol concentration in sham and exposed sessions separately for participants recorded in the morning or in the afternoon. ANOVA analyses showed no significant differences in cortisol concentrations when comparing sham to exposed sessions, no differences between volunteers, and no significant interaction between exposure × subjects in the morning, respectively, in T0 and Tf ($F = 2.72$, $P = 0.12$; $F = 0.08$, $P = 2.27$; interaction: $F = 0.42$, $P = 0.87$). In the afternoon, no significant difference was observed between T0 and Tf when comparing sham to exposed sessions ($F = 0.67$, $P = 0.78$), but a significant difference was noted between subjects ($F = 2.08$, $P = 0.04$), and no exposure × subjects interaction ($F = 0.55$, $P = 0.89$).

Salivary caffeine. Results showed that caffeine concentrations in all samples were negligible and below the detection limit of 2 μg/ml.

EXHIBIT F

DISCUSSION

The present study evaluated the effect of GSM (global system for mobile) signals of a mobile phone on the electrical activity of the human brain, especially in the alpha band of the resting EEG in young adults. In this study, healthy adults underwent two sessions of EEG recordings 1 wk apart as a washout period. Results showed that alpha spectral power decreased during exposure period to GSM signals. These results concur with previous findings on the effects of GSM signals on alpha power of resting EEG in humans (Croft et al. 2002; Curcio et al. 2005; Kramarenko and Tan 2003). When analyzing lower (8–10 Hz) and upper (10–12 Hz) alpha bands separately, results showed a similar significant decrease. This effect persisted in the postexposure period (Table 1), suggesting that the effect is sustained with lasting physiological changes and not solely during immediate interaction between exposure and the target tissue. This is in line with the results obtained in other studies that have exposed participants prior to the EEG

recording (Curcio et al. 2005; Huber et al. 2002; Reiser et al. 1995), and where an effect of RF-EMF has been observed on brain activity. The persisted effect of RF-EMF on brain activity was also observed on the EEG during sleep, during which time some authors have reported a modification following the active period of exposure (Loughran et al. 2005, 2012; Regel et al. 2007).

EXHIBIT F

As we know, interpreting alpha wave activity from the amplitude/power measurement is dependent on several factors, mainly the experimental conditions under which the amplitude is measured, such as open or closed eyes (Bazanova and Vernon 2013). Indeed, it was reported that an increase in the amplitude seen with closed eyes indicates less activation, whereas when eyes are open, there is a decrease in amplitude, indicating an increase in activation (Barry et al. 2007). It was assumed that neuronal activity generating the alpha rhythm is associated with areas of the cortex that are not processing information at rest. This is the usual explanation of why the rhythm may disappear when the eyes are open, while processing the visual information. Similarly, when a subject concentrates on a particular modality, the EEG activity in the alpha band specifically decreases in the corresponding brain region. Also, reduction in the power of alpha rhythms has been related to the speed of information processing, the subject's global attention, and cognitive performance (Klimesch 1997, 1998, 1999, 2003; Krause et al. 2000a, b; Neubauer and Freudenthaler 1995; Vogt et al. 1998).

The possible reasons why an effect was found only for eyes closed but not for eyes open may reside in the fact that amplitudes of alpha waves diminish when subjects open their eyes and, thereby, the effect of radiofrequency could not be significantly detected. However, when the subject is awake and relaxed with eyes closed, the alpha rhythm is prominent, thereby facilitating the observation of any effect.

According to these data, it seems that the effects observed in our study mimic, to some extent, the global reductions in alpha-band power observed in eyes-opened vs. eyes-closed conditions. One would suggest that the power decrease in alpha-band frequency resulting from the GSM signal exposure could be beneficial for memory process, global attention, and cognitive performance. The potential clinical significance of this effect, in this area, could be assessed in further studies.

The mechanisms behind these exposure-induced changes still remain unclear. However, on the basis of earlier reported data, it has been shown that intracortical excitability of the motor cortex was modified by acute exposure to GSM 900 (Ferreri et al. 2006). Intracortical inhibition/facilitation (ICI/ICF) curves were investigated, and results showed that ICI is reduced and ICF is enhanced after exposure to GSM signal (Ferreri et al. 2006). It has been suggested that ICI is mediated by GABA-A receptors (Hanajima et al. 1998), ICF is mediated by glutamatergic *N*-methyl-D-aspartate (NMDA) (Ziemann et al. 1998), and an imbalance between ICI and ICF may lead to changes in the intracortical excitability (Sanger et al. 2001). It has also been suggested that oxidative stress may play a role in this phenomenon, since it reduces the release of GABA and the activity of GABA-A receptors at presynaptic and postsynaptic sites (Sah et al. 1999, 2002), which correlates with the observed decrease in EEG amplitude.

The data reported in the present study were obtained while controlling certain parameters considered as confounding factors. Indeed, alpha rhythm is known to be sensitive to several factors, including caffeine and cortisol. To our knowledge, previous studies on RF's effect on EEG did not concretely and concurrently measure such factors that may modify alpha power. Therefore, our study was designed to include and assess salivary cortisol and caffeine.

As alpha rhythm has long been known to be sensitive to overall attentional states (i.e., intensity aspects such as arousal) (Adrian and Matthews 1934) and is also involved in the biasing of selective attention (Foxe et al. 1998; Kelly et al. 2006), we instructed subjects to refrain from any caffeinated drinks (e.g., coffee, tea, caffeinated soft drinks) 24 h before the study. It has been reported that caffeine increases

alertness and speeds reaction time, dominant factors in relation to alpha power (Fredholm et al. 1999; Smith 2002). In addition, previous studies reported a drop in absolute alpha power during rest with eyes open when caffeine was ingested at high doses (Deslandes et al. 2005; Siepmann and Kirch 2002). In our study, caffeine assessed in the saliva did not show detectable values (above the device's quantification limit = 2 μg/ml), suggesting that caffeinated drinks did not bias the observed results.

Moreover, salivary cortisol was assessed because it has been shown that concentrations of cortisol within the blood or saliva can vary spontaneously with EEG power across a range of 6.5–14.0 Hz, which includes the alpha rhythm (Sannita et al. 1999). Our results showed no significant variations in salivary cortisol between sham and real exposure.

In regard to electrical impedance, no differences were detected in all runs when comparing sham to real exposure sessions. Thus, the reported effects could not be related to differences in electrode impedance throughout the experiment, caffeine consumption before the experiment, or cortisol differences between groups.

Conclusions. Exposure to GSM-EMFs of a mobile phone can influence human dominant alpha rhythms in a resting state. Our results showed a power decrease of alpha band during and after exposure to GSM-EMFs compared with sham exposure in an eyes-closed condition. These findings were not correlated with impedance electrodes, cortisol, or caffeine, factors that can influence alpha power. However, extended postexposure duration should be tested since the observed effect persisted until the end of the postexposure period. Furthermore, it is also important to stress the potential clinical significance of this effect.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

Author contributions: R.G., L.H., A.D., and J.-D.L. performed experiments; R.G., L.Y.-C., L.H., and A.D. analyzed data; R.G., L.Y.-C., L.H., A.D., G.T., R.d.S., and B.S. interpreted results of experiments; R.G. prepared figures; R.G. and B.S. drafted manuscript; R.G., J.-D.L., G.T., R.d.S., and B.S. edited and revised manuscript; R.G., L.Y.-C., L.H., A.D., J.-D.L., G.T., R.d.S., and B.S. approved final version of manuscript; G.T., R.d.S., and B.S. conception and design of research.

ACKNOWLEDGMENTS

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REFERENCES

Adrian ED, Matthews BHC. The Berger rhythm: Potential changes in the occipital lobes in man. Brain 57: 355–385, 1934.

EXHIBIT F

Anderson V, Rowley J. Measurements of skin surface temperature during mobile phone use. Bioelectromagnetics 28: 159–162, 2007. [PubMed: 17080453]

Barry RJ, Clarke AR, Johnstone SJ, Brown CR. EEG differences between eyes-closed and eyes-open resting conditions. Clin Neurophysiol 118: 2765–2773, 2007. [PubMed: 17911042]

Bazanova OM, Vernon D. Interpreting EEG alpha activity. Neurosci Biobehav Rev 44: 94–110, 2014. [PubMed: 23701947]

Cook CM, Thomas AW, Prato FS. Resting EEG is affected by exposure to a pulsed ELF magnetic field. Bioelectromagnetics 25: 196–203, 2004. [PubMed: 15042628]

Croft RJ, Chandler JS, Burgess AP, Barry RJ, Williams JD, Clarke AR. Acute mobile phone operation affects neural function in humans. Clin Neurophysiol 113: 1623–1632, 2002. [PubMed: 12350439]

Croft RJ, Hamblin DL, Spong J, Wood AW, McKenzie RJ, Stough C. The effect of mobile phone electromagnetic fields on the alpha rhythm of human electroencephalogram. Bioelectromagnetics 29: 1–10, 2008. [PubMed: 17786925]

Croft RJ, Leung S, McKenzie RJ, Loughran SP, Iskra S, Hamblin DL, Cooper NR. Effects of 2G and 3G mobile phones on human alpha rhythms: resting EEG in adolescents, young adults, and the elderly. Bioelectromagnetics 31: 434–444, 2010. [PubMed: 20564174]

Curcio G, Ferrara M, Moroni F, D'Inzeo G, Bertini M, De Gennaro L. Is the brain influenced by a phone call? An EEG study of resting wakefulness. Neurosci Res 53: 265–270, 2005. [PubMed: 16102863]

D'Costa H, Trueman G, Tang L, Abdel-rahman U, Abdel-rahman W, Ong K, Cosic I. Human brain wave activity during exposure to radiofrequency field emissions from mobile phones. Australas Phys Eng Sci Med 26: 162–167, 2003. [PubMed: 14995060]

Deslandes AC, Veiga H, Cagy M, Piedade R, Pompeu F, Ribeiro P. Effects of caffeine on the electrophysiological, cognitive and motor responses of the central nervous system. Braz J Med Biol Res 38: 1077–1086, 2005. [PubMed: 16007279]

Ferreri F, Curcio G, Pasqualetti P, De Gennaro L, Fini R, Rossini PM. Mobile phone emissions and human brain excitability. Ann Neurol 60: 188–196, 2006. [PubMed: 16802289]

Foxe JJ, Simpson GV, Ahlfors SP. Parieto-occipital ~10-Hz activity reflects anticipatory state of visual attention mechanisms. Neuroreport 9: 3929–3933, 1998. [PubMed: 9875731]

Fredholm BB, Bättig K, Holmén J, Nehlig A, Zvartau EE. Actions of caffeine in the brain with special reference to factors that contribute to its widespread use. Pharmacol Rev 51: 83–133, 1999. [PubMed: 10049999]

Ghosn R, Thuróczy G, Loos N, Brenet-Dufour V, Liabeuf S, de Seze R, Selmaoui B. Effects of GSM 900 MHz on middle cerebral artery blood flow assessed by transcranial Doppler sonography. Radiat Res 178: 543–550, 2012. [PubMed: 23106209]

Groppe DM, Urbach TP, and Kutas M. Mass univariate analysis of event-related brain potentials/fields I: A critical tutorial review. Psychophysiology 48: 1711–1725, 2011. [PMCID: PMC4060794] [PubMed: 21895683]

Hanajima R, Ugawa Y, Terao Y, Sakai K, Furubayashi T, Machii K, Kanazawa I. Paired-pulse magnetic stimulation of the human motor cortex: differences among I waves. J Physiol 509: 607–618, 1998. [PMCID: PMC2230978] [PubMed: 9575308]

Hietanen M, Kovala T, Hämäläinen AM. Human brain activity during exposure to radiofrequency fields emitted by cellular phones. Scand J Work Environ Health 26: 87–92, 2000. [PubMed: 10817372]

Hinrikus H, Bachmann M, Lass J, Tomson R, Tuulik V. Effect of 7, 14 and 21 Hz modulated 450 MHz microwave radiation on human electroencephalographic rhythms. Int J Radiat Biol 84: 69–79, 2008. [PubMed: 18058332]

EXHIBIT F

Huber R, Treyer V, Borbély AA, Schuderer J, Gottselig JM, Landolt HP, Werth E, Berthold T, Kuster N, Buck A, Achermann P. Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. J Sleep Res 11: 289–295, 2002. [PubMed: 12464096]

Kelly SP, Lalor EC, Reilly RB, Foxe JJ. Increases in alpha oscillatory power reflect an active retinotopic mechanism for distracter suppression during sustained visuospatial attention. J Neurophysiol 95: 3844–3851, 2006. [PubMed: 16571739]

Klimesch W, Russegger H, Doppelmayr M, Pachinger T. A method for the calculation of induced band power: implications for the significance of brain oscillations. Electroencephalogr Clin Neurophysiol 108: 123–130, 1998. [PubMed: 9566625]

Klimesch W, Sauseng P, Gerloff C. Enhancing cognitive performance with repetitive transcranial magnetic stimulation at human individual alpha frequency. Eur J Neurosci 17: 1129–1133, 2003. [PubMed: 12653991]

Klimesch W. EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. Brain Res Brain Res Rev 29: 169–195, 1999. [PubMed: 10209231]

Klimesch W. EEG-alpha rhythms and memory processes. Int J Psychophysiol 26: 319–340, 1997. [PubMed: 9203012]

Kramarenko AV, Tan U. Effects of high-frequency electromagnetic fields on human EEG: A brain mapping study. Int J Neurosci 113: 1007–1019, 2003. [PubMed: 12881192]

Krause CM, Sillanmäki L, Koivisto M, Häggqvist A, Saarela C, Revonsuo A, Laine M, Hämäläinen H. Effects of electromagnetic field emitted by cellular phones on the EEG during a memory task. Neuroreport 11: 761–764, 2000a. [PubMed: 10757515]

Krause CM, Sillanmäki L, Koivisto M, Häggqvist A, Saarela C, Revonsuo A, Laine M, Hämäläinen H. Effects of electromagnetic fields emitted by cellular phones on the electroencephalogram during a visual working memory task. Int J Radiat Biol 76: 1659–1667, 2000b. [PubMed: 11133048]

Loughran SP, McKenzie RJ, Jackson ML, Howard ME, Croft RJ. Individual differences in the effects of mobile phone exposure on human sleep: rethinking the problem. Bioelectromagnetics 33: 86–93, 2012. [PubMed: 21812009]

Loughran SP, Wood AW, Barton JM, Croft RJ, Thompson B, Stough C. The effect of electromagnetic fields emitted by mobile phones on human sleep. Neuroreport 16: 1973–1976, 2005. [PubMed: 16272890]

Luck SJ. An introduction to the event-related potential technique. In: Averaging, Artifact Rejection and Artifact Correction. Cambridge, MA: MIT Press, 2005, chapt. 4, p. 166.

Maby E, Le Bouquin Jeannes R, Faucon G, Le Bouquin Jeannes R, Faucon G. Short-term effects of GSM mobiles phones on spectral components of the human electroencephalogram. Conf Proc IEEE Eng Med Biol Soc 1: 3751–3754, 2006. [PubMed: 17946579]

Neubauer AC, Freudenthaler HH. Ultradian rhythms in cognitive performance: no evidence for a 15-h rhythm. Biol Psychol 40: 281–298, 1995. [PubMed: 7669837]

Perentos N, Croft RJ, McKenzie RJ, Cvetkovic D, Cosic I. Comparison of the effects of continuous and pulsed mobile phone like RF exposure on the human EEG. Australas Phys Eng Sci Med 30: 274–280, 2007. [PubMed: 18274067]

Perentos N, Croft RJ, McKenzie RJ, Cosic I. The alpha band of the resting electroencephalogram under pulsed and continuous radio frequency exposures. IEEE Trans Biomed Eng 60: 1702–1710, 2013. [PubMed: 23358937]

Regel SJ, Gottselig JM, Schuderer J, Tinguely G, Rétey JV, Kuster N, Landolt HP, Achermann P. Pulsed radio frequency radiation affects cognitive performance and the waking electroencephalogram. Neuroreport 18: 803–807, 2007. [PubMed: 17471070]

EXHIBIT F

Reiser H, Dimpfel W, Schober F. The influence of electromagnetic fields on human brain activity. Eur J Med Res 1: 27–32, 1995. [PubMed: 9392690]

Röschke J, Mann K. No short-term effects of digital mobile radio telephone on the awake human electroencephalogram. Bioelectromagnetics 18: 172–176, 1997. [PubMed: 9084868]

Sah R, Galeffi F, Ahrens R, Jordan G, Schwartz-Bloom RD. Modulation of the $GABA_A$-gated chloride channel by reactive oxygen species. J Neurochem 80: 383–391, 2002. [PubMed: 11905987]

Sah R, Schwartz-Bloom RD. Optical imaging reveals elevated intracellular chloride in hippocampal pyramidal neurons after oxidative stress. J Neurosci 19: 9209–9217, 1999. [PMCID: PMC6782903] [PubMed: 10531424]

Sanger TD, Garg RR, Chen R. Interactions between two different inhibitory systems in the human motor cortex. J Physiol 530: 307–317, 2001. [PMCID: PMC2278414] [PubMed: 11208978]

Sannita WG, Loizzo A, Garbarino S, Gesino D, Massimilla S, Ogliastro C. Adrenocorticotropin-related modulation of the human EEG and individual variability. Neurosci Lett 262: 147–150, 1999. [PubMed: 10218877]

Schönborn F, Burkhardt M, Kuster N. Differences in energy absorption between heads of adults and children in the near field of sources. Health Phys 74: 160–168, 1998. [PubMed: 9450585]

Siepmann M, Kirch W. Effects of caffeine on topographic quantitative EEG. Neuropsychobiology 45: 161–166, 2002. [PubMed: 11979068]

Smith A. Effects of caffeine on human behavior. Food Chem Toxicol 40: 1243–1255, 2002. [PubMed: 12204388]

Straume A, Oftedal G, Johnsson A. Skin temperature increase caused by a mobile phone: a methodological infrared camera study. *Bioelectromagnetics 26: 510–519, 2005. [PubMed: 15931679]*

Van Rongen E, Croft R, Juutilainen J, Lagroye I, Miyakoshi J, Saunders R, de Seze R, Tenforde T, Verschaeve L, Veyret B, Xu Z. Effects of radiofrequency electromagnetic fields on the human nervous system. J Toxicol Environ Health B Crit Rev 12: 572–597, 2009. [PubMed: 20183535]

Vecchio F, Babiloni C, Ferreri F, Curcio G, Fini R, Del Percio C, Rossini PM. Mobile phone emission modulates interhemispheric functional coupling of EEG alpha rhythms. Eur J Neurosci 25: 1908–1913, 2007. [PubMed: 17432975]

Vecchio F, Babiloni C, Ferreri F, Buffo P, Cibelli G, Curcio G, van Dijkman S, Melgari JM, Giambattistelli F, Rossini PM. Mobile phone emission modulates inter-hemispheric functional coupling of EEG alpha rhythms in elderly compared to young subjects. Clin Neurophysiol 121: 163–171, 2010. [PubMed: 20005167]

Vecchio F, Buffo P, Sergio S, Iacoviello D, Rossini PM, Babiloni C. Mobile phone emission modulates event-related desynchronization of alpha rhythms and cognitive-motor performance in healthy humans. Clin Neurophysiol 123: 121–128, 2012. [PubMed: 21873111]

Vogt F, Klimesch W, Doppelmayr M. High-frequency components in the alpha band and memory performance. J Clin Neurophysiol 15: 167–172, 1998. [PubMed: 9563585]

Ziemann U, Hallett M, Cohen LG. Mechanisms of deafferentation-induced plasticity in human motor cortex. J Neurosci 18: 7000–7007, 1998. [PMCID: PMC6792971] [PubMed: 9712668]

Figures and Tables

EXHIBIT F

Fig. 1.

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The experimental protocol included three periods: preexposure, exposure, and postexposure. Each volunteer participated in two recording sessions (sham and active exposure) in a crossover randomized double-blind design. Electrodermal response (EDR), open eyes (OE), and closed eyes (CE) were performed during resting EEG recordings.

Table 1.

Statistical analyses of alpha band spectral power

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For each period (before, during, and after), we performed a paired *t*-test for each electrode across all subjects. Then, the frequency power values were averaged across all subjects, and a paired *t*-test was performed on the averaged electrode values for the two conditions (sham/exposure). Comparisons were made between sham vs. real exposure (i.e., $t > 0$ corresponds to a decrease, and $t < 0$ corresponds to a power increase in the truly exposed condition).

Fig. 2.

Open in a separate window

Changes in the electrical impedance of EEG electrodes during sham (○) and exposed (■) sessions. The impedances were maintained below 5 kΩ. No significant differences were detected comparing sham and real exposure in all runs.

Table 2.

Statistical findings: electrode impedance with two factors: session (sham and exposed), electrodes (29 electrodes), and interaction between the two factors

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Fig. 3.

Open in a separate window

Heart rate during open eyes (OE) and closed eyes (CE) periods in sham and exposed sessions. Results are expressed as means ± SE

EXHIBIT F

Fig. 4.

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Salivary cortisol concentration (ng/ml) before starting the study protocol (T0) and after the end of the protocol (Tf) in sham and exposed sessions for the volunteers who attended the experiment in the morning or in the afternoon.

EXHIBIT F

EXHIBIT G

Dr. Michael Semelka, DO

Exhibit G

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Doctor of Osteopathic Medicine
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Excela Health, August 2005 – present

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Program Director, AOA-accredited Family Medicine Residency at Latrobe Hospital, November 2006 – June 2020

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Responsibilities include residency program administration and oversight, education of residents, direct patient care in hospital as well as in Excela Square Frick Family Medicine, and oversight of three residency family medicine offices.

Duke University / Southern Regional Area Health Education Center Family Medicine Residency Program, July 2004 – July 2005

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AWARDS

2011 Fellow of the American Academy of Family Physicians (FAAFP)
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2005 Southern Regional AHEC Family Medicine Residency Faculty Teacher of the Year Award

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2003 Family Practice Center/Beaver Medical Center Staff Award
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2013 Clinical Assistant Professor of Family Medicine, Philadelphia College of Osteopathic Medicine

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PUBLICATIONS

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PRESENTATIONS

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