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Effect of Microwave Radiation on Human EEG at Two Different Levels of Exposure

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This study is aimed at evaluating the effect of microwave radiation on human brain bioelectric activity at different levels of exposure. For this purpose, 450 MHz microwave exposure modulated at 40 Hz frequency was applied to a group of 15 healthy volunteers at two different specific absorption rate (SAR) levels: a higher level of 0.303 W/kg (field strength 24.5 V/m) and a lower level of 0.003 W/kg (field strength 2.45 V/m). Ten exposure cycles (1 min off and 1 min on) at fixed SAR values were applied. A resting eyes-closed electroencephalogram (EEG) was continuously recorded. Results showed a statistically significant increase in the EEG power in the EEG beta2 (157%), beta1 (61%) and alpha (68%) frequency bands at the higher SAR level, and in the beta2 (39%) frequency band at the lower SAR level. Statistically significant changes were detected for six individual subjects in the EEG alpha band and four subjects in the beta1 and beta2 bands at the higher SAR level; three subjects were affected in the alpha, beta1 and beta2 bands at the lower SAR level. The study showed that decreasing the SAR 100 times reduced the related changes in the EEG three to six times and the number of affected subjects, but did not exclude the effect. Bioelectromagnetics 34:264–274, 2013. © 2012 Wiley Periodicals, Inc.

Key words: microwave exposure; electroencephalogram; exposure level; dose-dependence; field strength

INTRODUCTION

The significant increase in wireless technology applications has caused public concern about possible microwave exposure effects on human health. Up to this time, the basic restrictions for limiting exposure are based on the International Commission on Non-Ionizing Radiation Protection (ICNIRP) guidelines [ICNIRP, 1998]. According to these guidelines, the recommended localized specific absorption rate (SAR) for the head and trunk is 2 W/kg for radiofrequency fields at a field strength of 61 V/m. However, experimental results reported by many authors have shown the effect of microwave radiation on human brain bioelectrical activity and cognitive behavior at exposure levels less than the recommended limits for SAR [Freude et al., 1998; Borbely et al., 1999; Huber et al., 2000, 2002; D'Costa et al., 2003; Maby et al., 2004; Curcio et al., 2005; Regel et al., 2007a; Hinrikus et al., 2008a, b; Croft et al., 2008, 2010; Vecchio et al., 2010]. Considering possible health effects, some countries have established more strict limitations on the exposure levels for the general public. The European Parliament in recently accepted resolutions recommends setting thresholds not exceeding 0.6 V/m for levels of long-term exposure to microwaves in all indoor areas, and reducing it to 0.2 V/m in the medium term [Council of Europe, 2011]. This is an alternative perspective that is not supported by any of the major expert bodies.

The electroencephalographic (EEG) signal has been frequently employed to assess the effect of microwave exposure on human brain bioelectrical activity because of its sensitivity to immediate changes in neural processes. Alterations related to exposure at 875 MHz frequency by Global System for Mobile Communications (GSM) technologies were demonstrated at an SAR value of 0.11 W/kg in the EEG alpha band (9–10 Hz) power, which was larger on the ipsilateral side in posterior regions compared to the contralateral side [Croft et al., 2008]. An increase

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in the resting EEG alpha (8–13 Hz), beta1 (15–20 Hz) and beta2 (22–38 Hz) power was detected with 450 MHz radiation modulated at 7, 14, 21, 40, 70, or 217 Hz frequencies at an SAR value of 0.3 W/kg [Hinrikus et al., 2008a, b]. An increase in the alpha band power was also observed in a study using a GSM 900 phone at SAR values of 0.5 W/kg during and after exposure [Curcio et al., 2005]. An increase in the alpha band (10.5–11 Hz) activity was reported after exposure to pulsed 900 MHz signals at an SAR of 1 W/kg in the eyes-closed condition; no effect was seen in the eyes-open condition [Regel et al., 2007a]. The EEG alpha band power enhancement was observed in resting EEG recorded at SAR values of 1 W/kg in the case of a 900 MHz pulse-modulated signal [Huber et al., 2000, 2002]. Compared to young subjects, elderly subjects showed a significant increase in the inter-hemispheric synchronization of frontal and temporal alpha bands following exposure to a GSM signal at 0.05 W/kg [Vecchio et al., 2010].

Several studies reported an increase in the EEG spectral power during sleep when exposed to pulse-modulated 900 MHz microwaves at SAR values of 1 W/kg [Borbely et al., 1999; Huber et al., 2002]. An increased EEG alpha range in the sleep EEG was also detected at an SAR value of 1.4 W/kg (884 MHz GSM) [Lowden et al., 2011]. Microwave exposure at a lower SAR value of 0.29 W/kg emitted by a digital mobile phone increased the EEG spectral power in the 11.5–12.25 Hz frequency range during the initial part of sleep following the exposure [Loughran et al., 2005, 2012]. A strong dependence on the modulation frequency and individual variability were reported at an SAR value of 2 W/kg [Schmid et al., 2012]. On the other hand, a GSM signal from a planar antenna at an SAR value of 0.6 W/kg, and from a horn antenna at an SAR value of 1.8 W/kg, had no effect on sleep architecture and the EEG [Wagner et al., 1998, 2000].

The effects of GSM signals on cognitive performance and auditory event-related potentials have been reported at SAR levels of 1.4 W/kg [Maby et al., 2004] and 0.61 W/kg [Hinrichs and Heinze, 2004]. The exposure to GSM signals of 902 MHz at an SAR of 0.65 W/kg also caused changes in EEG signals during a visual working memory task [Krause et al., 2000a]. However, the authors were unable to replicate their previous findings in later studies [Krause et al., 2000b, 2004]. They concluded that the effects of microwave radiation on brain oscillatory responses may be subtle, variable, and difficult to replicate for unknown reasons [Krause et al., 2007]. Another possibility, and the consensus view at present, is that there is no detectable effect of microwave radiation on cognitive performance and the reported findings may have been caused by chance [Regel and Achermann, 2011]. Therefore, it is not reasonable to select cognitive tasks as a measure for the evaluation of a dose-dependent relationship.

Only a few studies were aimed at comparing the effect of microwave radiation at two different levels of exposure. The effect of a GSM handset-like signal with 10 g averaged peak SARs of 0.2 and 5 W/kg was investigated on three different cognitive tasks and the sleep EEG [Regel et al., 2007b]. The study revealed a dose-response relationship between field intensity and its effects on brain physiology as demonstrated by changes in the sleep EEG [Regel et al., 2007b]. No dose-response relationship was found for accuracy in the cognitive tasks [Regel et al., 2007b]. The effects of two types of exposure, 1950 MHz Universal Mobile Telecommunications System (UMTS) signals at SAR values of 0.1 and 1 W/kg, and pulsed 900 MHz GSM signals at an SAR of 1 W/kg, were tested on well-being and the resting eyes-closed EEG [Kleinlogel et al., 2008]. However, the results of the study did not give any evidence for an effect at SAR values of both 0.1 and 1 W/kg [Kleinlogel et al., 2008].

Despite many investigations at different levels of exposure, the results do not provide sufficient systematic knowledge about how the reported effects depend on the level of exposure to microwave fields. The main problem is that data from different studies are not comparable due to different methods and experimental protocols as well as microwave and modulation frequencies. The alterations in brain electrical activity have been reported at SAR levels below 1 W/kg [Regel et al., 2007b; Croft et al., 2008; Hinrikus et al., 2008a, b; Vecchio et al., 2010]. On the other hand, no effects have been detected at SAR levels close to 1 W/kg or even higher [Wagner et al., 1998, 2000; Kleinlogel et al., 2008]. In a recent review, a conclusion was drawn confirming the lack of systematic data on the dependence of microwave effects on the level of exposure [Juutilainen et al., 2011].

The aim of this study was to evaluate the dependence of microwave effects on the level of exposure. For this purpose, alterations in the human EEG signal caused by modulated microwave exposure were compared at two different levels of exposure, both lower than the existing health protection limit. An SAR value of 0.303 W/kg (field strength 24.5 V/m) was selected and was equal to that in our previous studies where an effect was detected [Hinrikus et al., 2008a, b]. Another SAR value was selected 100 times lower, equal to 0.003 W/kg (field strength 2.45 V/m).

MATERIALS AND METHODS

Subjects

The experiment was carried out on a group of volunteers consisting of 15 healthy subjects (aged 23–32), nine male and six female. Their physical and mental condition was evaluated by a questionnaire and clinical interview before the experiment. All subjects selected reported themselves to be in good health and without medical or psychiatric disorders. Those who declared themselves tired or sleepy before the experiment were excluded. After the recordings, the subjects were asked to describe how they felt during the experiment. They reported no effect on their alertness and no experience of any strain during the EEG recordings. Each participant was aware of the purpose of the experiments and gave a written consent.

During the experiments, the subjects were asked to lie in a relaxed position, with eyes closed and ears plugged. The experiments were performed in a dark laboratory room. All subjects participated in two EEG recording sessions. For each recording session, the exposure condition was randomly assigned. The subjects were not informed of the exposure power level during the recording session, however, they were aware of the possibility of being exposed. Both recording sessions were performed on the same day. The interval between the two sessions was at least 15 min.

The study was conducted in accordance with the Declaration of Helsinki and was formally approved by the Tallinn Medical Research Ethics Committee (Tallinn, Estonia).

Microwave Exposure

The 450 MHz electromagnetic radiation was generated by a signal generator (SML02, Rhode & Schwartz, Munich, Germany). The radiofrequency signal was 100% pulse modulated using a pulse modulator (SML-B3, Rhode & Schwartz) at a frequency of 40 Hz and a duty cycle of 50%. The signal from the generator was amplified using a power amplifier (MSD-2597601, Dage, Stamford, CT). The generator and amplifier were carefully shielded and did not cause artifacts during recordings. The 1 or 0.01 W electromagnetic radiation output power was guided by a coaxial lead to a 13-cm quarter-wave antenna (NMT450 RA3206, Allgon Mobile Communication, Stockholm, Sweden). The antenna was located 10 cm from the skin on the left side of the head close to ear; the angle between axis of the head and antenna was 45°. The spatial distribution of the electric field was measured with a field strength meter (CA 43 Fieldmeter, Chauvin Arnoux, Paris, France). The measurements were performed by the Central Physical Laboratory of the Health Board (Tallinn, Estonia) under real experimental conditions. During the experiments, the stability of the electromagnetic radiation level was monitored with a field strength meter (Digi-Field C, IC Engineering, Thousand Oaks, CA).

The SAR was calculated using SEMCAD software (Schmid & Partners Engineering, Zurich, Switzerland). The finite-difference time-domain computing method with the specific anthropomorphic mannequin (SAM) specified in the Institute of Electrical and Electronics Engineers (IEEE) Standard 1528–2003 [IEEE, 2003] was applied. The results of the SAR distribution calculations are shown in Figure 1. Maximal exposure occurred in the left central region of the head; in this area, the calculated spatial peak SAR averaged over 1 g was 0.303 W/kg for the microwave output power of 1 W, and 0.003 W/kg for the microwave output power of 0.01 W.

Recording Protocol and Equipment

Each subject participated in two EEG recording sessions. The protocol of each recording session consisted of one series of reference followed by two series of microwave exposure; each series included five experimental cycles (Fig. 2). One experimental cycle consisted of a 60-s resting segment without microwave exposure and a 60-s exposure segment with microwave exposure. Each microwave series was conducted at a constant field power density of the

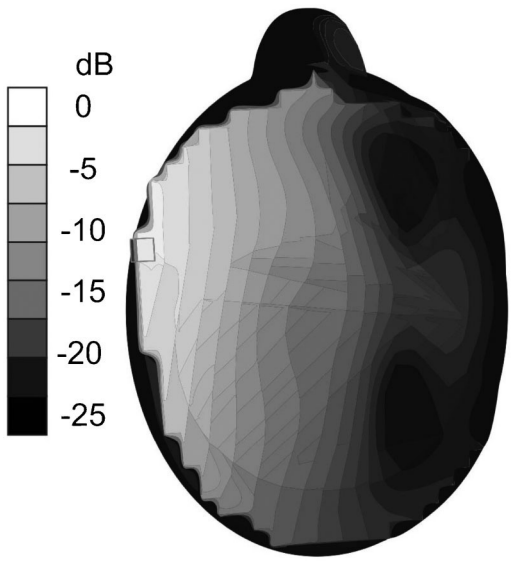

Fig. 1. Calculated SAR distribution in the SAM cross section. 0 dB corresponds to 0.003 W/kg averaged over 1 g for 0.01 W antenna input power and 0.303 W/kg for 1 W antenna input power.

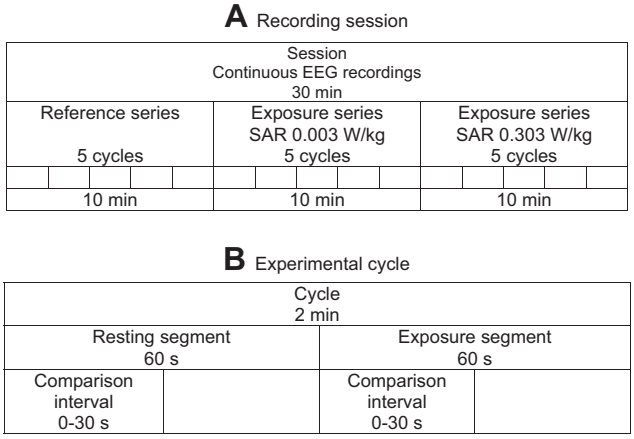

Fig. 2. Schedule of the recording protocol. **A**: Recording session; **B**: Experimental cycle.

modulated microwave radiation. The order of the two microwave exposures (SAR 0.303 and 0.003 W/kg) was randomized among subjects. However, the lower SAR level was selected in the first series and the higher SAR level in the second series in one session and vice versa in the other session for each subject. The reference series consisted of five cycles without exposure. One EEG recording lasted for 30 min, during which the EEG was continuously recorded while the subject was relaxed with eyes closed.

Cadwell Easy II EEG measurement equipment (Kennewick, WA) was used for the EEG recordings. The EEG was recorded using 19 electrodes, which were placed on the subject's scalp according to the international 10–20 system of electrode placement. EEG channels selected for analysis were identical to these in our previous studies [Hinrikus et al., 2008a, b]: frontal FP1, FP2; temporal T3, T4; parietal P3, P4; occipital O1, O2; and the reference electrode Cz. The raw data of the EEG recordings in the frequency band 0.5–48 Hz were stored on a computer at a 400 Hz sampling frequency. Signals for further analysis were selected in the frequency band 4–38 Hz. Such selection excluded the modulation frequency from analysis. Elliptical bandpass filters with an attenuation of 100 dB in the stopband were used. Preprocessing of the signals was performed in the LabVIEW (National Instruments, Austin, TX) programming and signal-processing environment using the analysis tool, Elliptic filter. An experienced neurologist examined the recorded EEG signals by visual inspection. Recordings made with drowsy subjects or recordings with apparent electrode artifacts were not used and the whole session was re-recorded on another day. To detect possible problems that could have occurred with electrodes, filtering, etc., the average group spectrum of recorded signals was calculated before further processing with the LabVIEW analysis tool.

Artifacts can be induced by parasitic demodulation of the radiofrequency electromagnetic components of the EEG electrodes and equipment. To detect possible parasitic interactions between the recording and radiofrequency equipment, the set-up was validated before the experiments. To conduct testing, an EEG cap was placed on a passive phantom of a human head [Hinrikus et al., 2008b]. The recordings of the phantom were conducted in accordance with the protocol of the present study. Multichannel recordings in the frequency band 0.5–48 Hz detected a spectral component of 40 Hz at the higher and lower SAR values. The component disappeared when the antenna was removed therefore it was not caused by the radiofrequency equipment but by the radiation from the antenna. No other spectral components were detected. The artifacts at the modulation frequencies were removed from the EEG signals by off-line filtering during the pre-processing of the signals.

EEG Analysis

The EEG analysis was similar to that applied in our previous studies [Hinrikus et al., 2008a, b]. Relative changes in the recorded EEG signals between the cycle segments with and without exposure were selected as a measure to detect the microwave effect on the EEG power.

The powers of the four basic EEG frequency bands, theta (4–7 Hz), alpha (8–13 Hz), beta1 (15–20 Hz), and beta2 (22–38 Hz), were extracted from the total EEG by filtering. Elliptical bandpass filters with an attenuation of 100 dB in the stopband were used.

The average powers of segments with and without exposure were compared. The comparison intervals were selected as 30 s from the beginning of the resting (unexposed) and microwave-exposed segments in one exposure cycle. The first half of the 60 s segment was selected considering possible physiological adaptations of the brain to exposure revealed in our previous study [Hinrikus et al., 2008a].

The average value of the EEG power s_i of an arbitrary comparison segment i was calculated as:

$$s_i = \frac{1}{N}\sum_{r=1}^{N}[x(r)]^2 \qquad (1)$$

where $x(r)$ is the amplitude of the recorded signal in a sample r, and N is the number of samples;

during 30 s, $N = 12000$. Parameter S_c, the relative change in the powers of the recording segments with and without exposure for one cycle, was calculated as:

$$S_c = \left(\frac{s_2}{s_1} - 1\right) \times 100\% \quad (2)$$

where s_1 and s_2 are the average powers inside the comparison segments without and with exposure, respectively.

The effect of microwave exposure on a subject was estimated by averaging the values of parameter S_c over 10 cycles of exposure (five cycles from each recording session) at a fixed SAR value. Parameter S_n, the average relative change in the EEG power of the recording segments with and without exposure at a fixed SAR value for a subject n, was calculated as:

$$S_n = \frac{1}{10}\sum_{c=1}^{10} S_c \quad (3)$$

where S_c is the value of the parameter calculated according to Equation (2) for one cycle.

Statistical Analysis

The hypothesis is that the exposure causes differences between data at the three exposure conditions. To reveal the effect, which is expected to be small, it is important to minimize the influence of other factors such as possible differences between EEG sites, frequency bands, subjects, etc. Therefore, the differences between exposure conditions were compared at all other identical conditions, in the same EEG channel (channel 1 was compared only to channel 1, channel 2 only to channel 2, etc.), frequency band (alpha was compared only to alpha, beta only to beta, etc.) and for the same subject (subject 1 was compared only to subject 1, subject 2 only to subject 2, etc.). To avoid additional differences caused by factors other than exposure conditions, the comparisons were not performed between combinations of different channels, frequency bands, and subjects. The statistical analysis was performed for three exposure conditions at each of eight EEG channels and 24 (3×8) comparisons at each of four EEG frequency bands. In the case of individual subjects, the statistical analysis was planned separately for each of the 15 subjects.

The software package Statistica 6.0 (StatSoft, Tulsa, OK) was used in the statistical analysis. For the group analysis, a mixed-design ANOVA was employed whereby the 10 reference, 10 SAR1 (0.003 W/kg) and 10 SAR2 (0.303 W/kg) measurements (calculated parameter S_c for 10 cycles, degrees of freedom (df) = 9) per participant were treated as independent (comprising three levels of the factor "Exposure Condition"), and the eight electrode sites were treated as levels of the within-subject factor "EEG Channel". The post hoc Bonferroni test was performed to evaluate the significance level of comparisons for the main effects of "Exposure Condition" (correction factor 3). Additionally, the planned Bonferroni correction was calculated for multiple comparisons in four EEG frequency bands (correction factor $3 \times 4 = 12$) and eight EEG channels (correction factor $3 \times 4 \times 8 = 96$). Finally, the initial P-values were multiplied by the correction factor 96 for all comparisons in the group analysis.

The averaging of relative changes in the EEG power over the whole group may mask larger effects for individual subjects. Therefore, individual changes in the EEG power for each subject were evaluated. The same analysis as for the whole group with the Bonferroni correction (correction factor 96) was applied for each individual subject, differing only in that the total number of independent measurements per test was 30 (as it only included one subject's data). An additional Bonferroni correction was applied for 15 subjects (correction factor 15). The total correction factor for each individual subject consisted of the correction for the group analyses (96) multiplied by the number of subjects ($96 \times 15 = 1440$).

RESULTS

The average group spectrum of recorded EEGs for the signal segments with and without exposure is shown in Figure 3. The spectra of the signal segments for further analysis selected by filtering in the 4–38 Hz band are similar. Small differences are caused by changes in the state of the brain at different times. Only a very small rise the in alpha band with exposure can be detected.

Figure 4 shows the relative changes in the EEG power caused by microwave exposure in the different EEG frequency bands. Microwave exposure at the higher SAR value of 0.303 W/kg caused an increase in the EEG average power changes compared to the reference series in the EEG alpha, beta1, and beta2 bands. No effect was detected in the theta band. At the lower SAR level of 0.003 W/kg, the obvious trend of increase was evident in the EEG beta1 and beta2 bands.

To demonstrate more clearly the dependence of the effect on exposure level, the values of changes in the exposed and reference series were compared. The graphs presented in Figure 5 show the relative difference of the changes in the exposed and reference

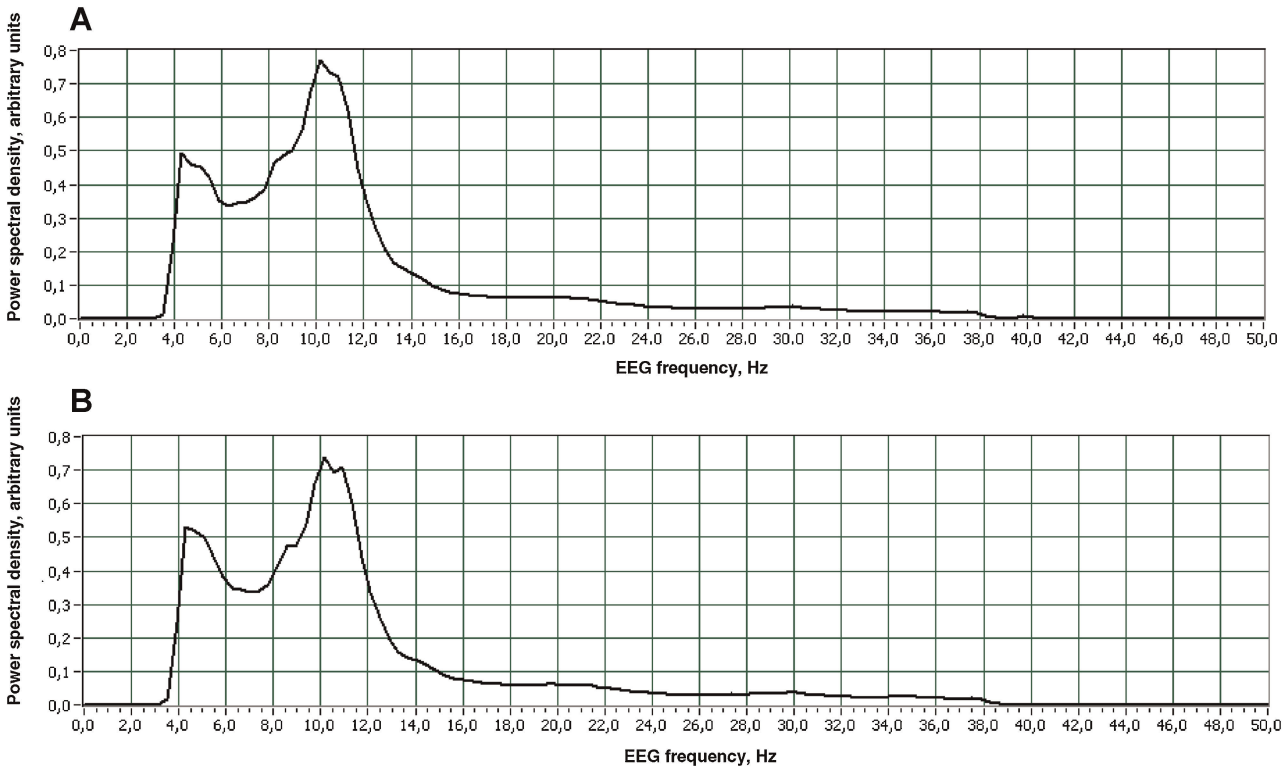

Fig. 3. Calculated EEG spectra selected by bandpass filtering in the frequency band 4–38 Hz averaged over the group of subject ($n = 15$) and all EEG channels. **A**: Spectrum of the segments of recorded signals with exposure; **B**: Spectrum of the segments of recorded signals without exposure.

series. Compared to EEG power changes in the reference series, the strongest increase in the changes at the SAR level of 0.303 W/kg occurred in the beta2 band (157%); the increases in the alpha (68%) and beta1 (61%) bands were somewhat smaller. A trend of increase in the EEG power changes at the SAR level of 0.003 W/kg was evident only in the beta2 (39%) and beta1 (23%) bands.

The results of the statistical evaluation for the whole group are presented in Table 1. These results revealed statistically significant differences between the reference and microwave-exposed series at the

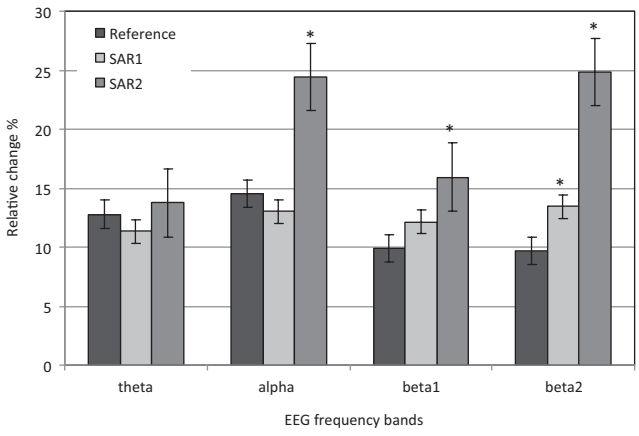

Fig. 4. Relative changes in the EEG power as parameter S_c values calculated for the theta, alpha, beta1 and beta2 EEG frequency bands averaged over 10 cycles and 15 subjects at three exposure conditions: reference without exposure; SAR1 = 0.003 W/kg; and SAR2 = 0.303 W/kg. Vertical bars denote standard error. *Significant difference between reference and exposed series indicated by Bonferroni corrected $P < 0.05$.

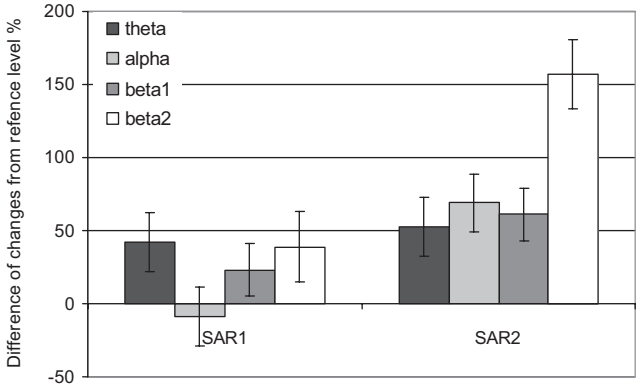

Fig. 5. Quantity of the microwave effect as the relative difference of change (parameter S_n) in the exposed (S_{ne}) and reference (S_{nr}) series for a subject n calculated as $S_{ne} - S_{nr}/S_{nr}$ for the theta, alpha, beta1 and beta2 EEG frequency bands averaged over 15 subjects at SAR1 = 0.003 W/kg and SAR2 = 0.303 W/kg. Vertical bars denote standard error.

TABLE 1. Results of the Post-Hoc Bonferroni Test for Comparison Between the Reference, Exposed at SAR1 (0.003 W/kg) and SAR2 (0.303 W/kg) Series for the Whole Group in the EEG Theta, Alpha, Beta1 And Beta2 Frequency Bands Calculated for 10 Cycles of Exposure Grouped for the eight EEG Channels

	P-values		
	Reference	SAR1	SAR2
Theta			
Ref	N/A	1.0000	1.0000
SAR1	1.0000	N/A	1.0000
SAR2	1.0000	1.0000	N/A
Alpha			
Ref	N/A	1.0000	**0.0002**
SAR1	1.0000	N/A	**0.0005**
SAR2	**0.0002**	**0.0005**	N/A
Beta1			
Ref	N/A	0.8573	**0.0030**
SAR1	0.8573	N/A	0.0732
SAR2	**0.0030**	0.0732	N/A
Beta2			
Ref	N/A	**0.0261**	**0.0008**
SAR1	**0.0261**	N/A	**0.0188**
SAR2	**0.0008**	**0.0188**	N/A

Significant values $P < 0.05$ in bold.

SAR level of 0.303 W/kg in the EEG alpha, beta1, and beta2 frequency bands. A significant difference was also revealed between the reference and exposed series at the SAR level of 0.003 W/kg in the EEG beta2 frequency band. Statistically significant differences were detected between exposure conditions at the higher and lower SAR levels in the EEG alpha and beta2 frequency bands.

The individual changes for each subject are shown in Figure 6. The graphs show relative alterations caused by microwave exposure in the EEG beta1 frequency band. The exposure caused a remarkable increase in changes for four subjects (2, 5, 12, and 15), whereas the increase occurred at both SAR levels for three of them (2, 12, and 15). Some trend of increase in changes compared with the reference series was evident at both SAR levels for an additional two subjects (11 and 14). A decrease in alterations related to exposure was mentioned for one subject (9) and the stability of alterations for two subjects (1 and 8). The values of bars lower than 10% (the level comparable with reference bars in Fig. 3) can be considered as natural variability in the EEG signal (six subjects: 3, 4, 6, 7, 10, and 13) and no trend of changes was revealed for these subjects.

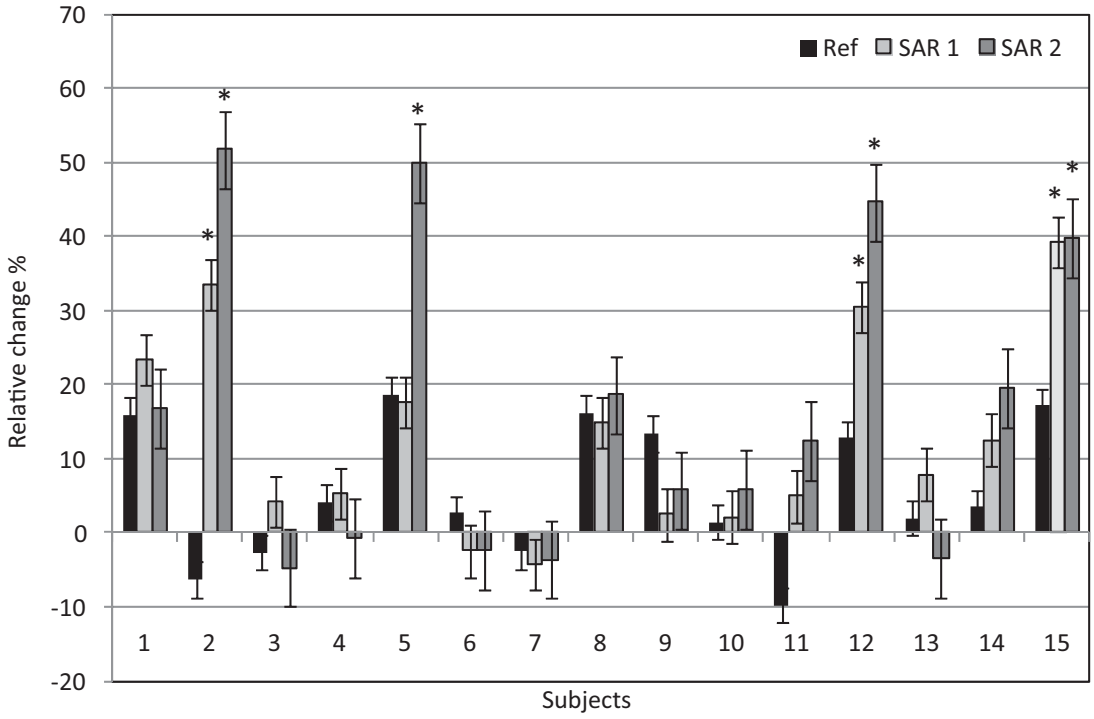

Fig. 6. Relative changes in the EEG power as parameter S_c values calculated for the beta1 frequency band averaged over 10 cycles for individual subjects ($n = 15$) at three exposure conditions: reference without exposure; SAR1 = 0.003 W/kg; and SAR2 = 0.303 W/kg. Vertical bars denote standard error. *Significant difference between reference and exposed series indicated by Bonferroni corrected $P < 0.05$.

TABLE 2. Bonferroni-Corrected *P*-Values for Comparison Between the Reference, Exposed at SAR1 and SAR2 Series for Individual Subjects in the EEG Alpha, Beta1 and Beta2 Frequency Bands Calculated for 10 Cycles of Exposure

	P-values					
	SAR1 = 0.003 W/kg			SAR2 = 0.303 W/Kg		
Subject	Alpha	Beta1	Beta2	Alpha	Beta1	Beta2
1	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
2	↑ **2×10^{-5}**	↑ **0.0025**	↑ **7×10^{-4}**	↑ **0.0455**	↑ **1×10^{-4}**	↑ **8×10^{-5}**
3	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
4	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
5	1.0000	1.0000	1.0000	↑ **2×10^{-4}**	↑ **0.0060**	0.1605
6	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
7	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
8	1.0000	1.0000	1.0000	↑ **2×10^{-5}**	1.0000	1.0000
9	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
10	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
11	↑ **0.0046**	0.8765	↑ **0.0321**	↑ **0.0465**	1.0000	↑ **1×10^{-5}**
12	↑ **0.0363**	↑ **0.0441**	0.8105	↑ **3×10^{-4}**	↑ **0.0063**	↑ **0.0321**
13	0.6462	1.0000	1.0000	1.0000	1.0000	1.0000
14	0.1052	0.6854	0.8064	0.1821	0.1915	0.5664
15	1.0000	↑ **0.0246**	↑ **0.0033**	↑ **0.0404**	↑ **0.0328**	↑ **0.0096**

Significant values $P < 0.05$ in bold; ↑ denotes an increase in the EEG power with exposure.

Table 2 specifies results of the statistical analysis for individual subjects in the EEG alpha, beta1, and beta2 frequency bands. No statistically significant changes were detected in the EEG theta band. The statistical analysis results show that the subjects affected by exposure had significant changes simultaneously in several EEG frequency bands. Microwave exposure introduced statistically significant changes in all or the majority (except one) of the EEG frequency bands for three subjects (2, 12, and 15) at both SAR levels. The changes were evident for the same subjects at both SAR levels. In the alpha band, six subjects were affected at the higher SAR level (2, 5, 8, 11, 12, and 15), and three at the lower SAR level (2, 11, and 12); in the beta1 band, four subjects were affected at the higher SAR level (2, 5, 12, and 15) and three at the lower SAR level (2, 12, and 15); in the beta2 band, four subjects were affected at the higher SAR level (2, 11, 12, and 15) and three at the lower SAR level (2, 11, and 15). The results show that the subjects affected at the lower SAR level were also affected at the higher SAR level in the same EEG frequency band.

For all subjects having a significant difference between the reference and microwave-exposed series, the average relative changes were positive; consequently, the level of power in segments with exposure was higher than in segments without exposure.

DISCUSSION

The experiments revealed the dose-dependent relationship of the modulated microwave effect: the average values of relative changes were higher at the higher level of exposure compared to those at the lower level. Microwave exposure caused an increase in the relative changes in the EEG power between exposed and resting segments compared to reference recordings for the whole group and for individual subjects at both SAR levels (Figs. 4–6).

The effect became evident in the EEG alpha, beta1, and beta2 frequency bands (Figs. 4–6). No effect was revealed in the EEG theta band. The increase in the EEG alpha band power caused by microwave exposure is in good accordance with findings reported by other research groups [Huber et al., 2002; Curcio et al., 2005; Croft et al., 2008, 2010]. The application of a more sensitive experimental method (repetitive cycles of exposure) made it possible to detect an increase in the EEG power not only in the alpha but also in the beta band. An increase in the EEG beta band power related to modulated microwave at similar exposure conditions was reported in our previous studies [Hinrikus et al., 2008a, b].

Several studies support the position that delayed effects may occur after 30–45 min of microwave exposure [Huber et al., 2002; Curcio et al., 2005]. The protocol of our experiment (comparison of 1 min

exposed and 1 min resting EEG during 10 cycles) was not sensitive for detecting the outlasting effect of exposure. The presence of the effect during both exposed and resting segments could cause a decrease in the difference between them and a diminishing of the detected effect. Moreover, the selection of the first half of the 60 s segment for analysis was the most unfavorable choice regarding an outlasting effect. Nevertheless, statistically significant changes were detected and the impact of the outlasting effect did not seem important. However, if the microwave effect on the EEG persisted after the exposure, the detected effect was smaller than its real value.

Relative changes averaged over the whole group showed a maximal increase in the beta2 band: 157% at the SAR level of 0.303 W/kg, and 39% at the SAR level of 0.003 W/kg. The increase in the changes in the alpha band was about two times lower: 68% at the SAR level of 0.303 W/kg and not evident at the lower SAR level. It does not seem random that the major effect was evident in the EEG beta band. According to the parametric model of excitation, modulated microwave radiation causes periodic changes in the brain electric susceptibility and leads to excitation of the EEG rhythms at parametric resonance frequencies [Hinrikus et al., 2011]. The linear model of parametric excitation determines a main resonance close to one-half of the external frequency (40 Hz modulation) [Butikov, 2004]. Therefore, the strongest effect is expected at the EEG beta band frequencies close to 20 Hz. Moreover, according to the nonlinear model of parametric excitation of the brain, the excitation of additional EEG rhythm components at one-quarter and three-quarters of the modulation frequency is expected [Hinrikus et al., 2011]. These components (close to 10 and 30 Hz) cause an increase in the EEG alpha and beta band power.

EEG alterations at reference conditions are caused by natural variability in the signal related to neural activity in the brain. The time course of the EEG spectrum power in resting conditions has been reported to have monotonic alterations toward the end of the recordings [Maltez et al., 2004]. The monotonic changes in the EEG power during the recordings lead to changes in the reference data. However, these processes do not cause significant differences between signal segments.

The number of individual subjects significantly affected by exposure was 40–60% higher (six subjects in the alpha band, four subjects in the beta1 and beta2 bands) at the higher SAR value of 0.303 W/kg compared with the lower SAR value of 0.003 W/kg (three subjects in the alpha, beta1, and beta2 bands). The same subjects were sensitive at the higher and lower level of exposure. The relative part of affected subjects was 40% for the alpha band and 27% for the beta bands at the higher SAR value, and 20% for the alpha and beta bands at the lower SAR value. These results are close to those in our previous study, where at the same exposure conditions (450 MHz microwave radiation, modulation frequency 40 Hz, SAR = 0.303 W/kg) and number of subjects ($n = 15$), the relative percentage of affected subjects in the beta1 band was 20% (three subjects) [Hinrikus et al., 2008b]. However, those results were based on the analysis of the EEG only in parietal channels P3–P4 [Hinrikus et al., 2008b].

As expected, alterations caused by exposure increase with increasing SARs (Figs. 4–6). This finding agrees with the results achieved in another study where exposure to 900 MHz GSM-like radiation had been applied [Regel et al., 2007b]. The reported data in that study provided evidence for a dose-response relationship: the spectral power of the sleep EEG in the fast spindle frequency range increased by 7.7% after radiofrequency exposure at an SAR of 0.2 W/kg, and 13.6% after exposure at 5 W/kg [Regel et al., 2007b].

The dependence of the effect on the exposure level is obviously not linear; the reduction in the effect is much slower than expected according to the decreasing SAR. In our study, the level of SAR decreases 100 times (20 dB), while the effect (relative changes of EEG power between resting and exposed segments) decreased only three to six times (5–8 dB; Figs. 4 and 5). In our study, both exposure levels were lower than the exposure limit for the general public. In the case of comparison of the effect at exposure levels higher (5 W/kg) and lower (0.2 W/kg) than the exposure limit for general public, the level of SAR decreases 25 times but the spectral power of the EEG decreases only about 1.8 times [Regel et al., 2007b]. One reason for the slow decrease in the effect is a possible parametric mechanism of excitation of the EEG rhythms by modulated microwave radiation [Hinrikus et al., 2011]. According to the theory of parametric excitation, even a weak external periodic force can cause remarkable excitation of the oscillatory system [Butikov, 2004]. Consequently, the modulated microwave radiation as a periodic factor can cause excitation of EEG rhythms even at a low level of exposure and the dose-dependent relationship can be less critical than expected.

It is difficult to predict how effective a further decreasing of the field strength at lower SAR values

is, and at which level the threshold of the effect is expected. A physical model in which cells are considered as possible detectors of very weak electric fields has been proposed and discussed [Weaver and Astumian, 1990]. The paradox is that according to experimental data, much smaller fields than the calculated thermal noise limit caused the effect and, according to the model, could be detected [Weaver and Astumian, 1990]. The parametric model of excitation can provide a threshold value lower than the thermal noise level of the system. However, there are still no data to estimate the threshold. Therefore, there is no certainty that reducing the preventive levels of exposure to microwaves down to 0.2 V/m, proposed by the Council of Europe, will fully eliminate the effect.

Further investigations, especially at lower levels of exposure, are needed to gain sufficient knowledge about the dependence of the microwave radiation effect on the level of exposure and the threshold of the effect.

CONCLUSION

The results of this experimental study confirm that exposure to 450 MHz microwave radiation modulated at 40 Hz has an effect on brain electrical activity depending on the level of exposure. A decrease in the exposure by 100 times reduces the effect, which results in a reduction in the relative changes of the EEG by three to six times and the number of subjects significantly affected by microwave exposure by 40–60%. However, a reduction in the effect is slower than expected according to the decrease in the level of exposure. This study showed that a decrease in the microwave field 100 times reduced but did not exclude microwave-related changes in the EEG.

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