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Effect of 7, 14 and 21 Hz modulated 450 MHz microwave radiation on human electroencephalographic rhythms

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Abstract

Purpose: The aim of this study was to evaluate the effect of microwaves modulated at different frequencies on human electroencephalographic (EEG) rhythms.

Materials and methods: Thirteen healthy volunteers were exposed to microwaves (450 MHz) pulse-modulated at frequencies of 7, 14 and 21 Hz. The field power density at the scalp was 0.16 mW/cm^2. Our experimental protocol consisted of two five-cycle (1 min on and 1 min off) series of exposures at fixed modulation frequencies. A relative change in the EEG power with and without exposure was used as a quantitative measure. EEG frequencies recorded in the theta (4 – 6.8 Hz), alpha (8 – 13 Hz), beta1 (15 – 20 Hz), and beta2 (22 – 38 Hz) bands were analyzed.

Results: Modulated microwaves caused an increase in the average EEG alpha (17%) and beta (7%) power but the theta rhythm remained unaffected. Increases in the EEG alpha and beta power were statistically significant during the first half-period of the exposure interval (30 s) at the modulation frequencies of 14 and 21 Hz. Differences were found in individual sensitivity to exposure. Increases in the EEG beta power appeared statistically significant in the case of four subjects.

Conclusions: Our findings suggest that the effect of the 450 MHz microwave radiation modulated at 7, 14 and 21 Hz varies depending on the modulation frequency. The microwave exposure modulated at 14 and 21 Hz enhanced the EEG power in the alpha and beta frequency bands, whereas no enhancement occurred during exposure to the modulation frequency of 7 Hz.

Keywords: *EEG rhythms, brain bioelectrical activity, radio-frequency radiation, low-frequency modulation, electromagnetic field effect, modulated microwaves*

Introduction

Increased use of telecommunication equipment has raised the problem of possible effects of the radio-frequency electromagnetic fields (EMF) on human brain physiology (D'Andrea et al. 2003). During recent decades, discussions have focussed on the effects of low-level EMF on human electroencephalographic (EEG) signals and on cognitive effects (Cook et al. 2006). Several investigators have reported that exposure to low-level microwaves produces alterations in the resting or sleep EEG signal and/or brain behaviour (Reiser et al. 1995, Mann & Roschke 1996, Borbely et al. 1999, Huber et al. 2000, Krause et al. 2000a, Lass et al. 2002, Hinrikus et al. 2004, Curcio et al. 2005). Reports on possible non-thermal EMF effects are often contradictory. Difficulties experienced in independent replication of experimental findings have caused doubts concerning these effects; moreover, mechanisms behind the effects are still unclear.

Earlier results related to the effects of the EMF on the EEG have been derived from animal studies. Investigators have reported that exposure to an electromagnetic field of 0.147 GHz, amplitude modulated at brain rhythm frequencies, influenced the EEG patterns in cats (Bawin et al. 1973). Interestingly, these amplitude-modulated fields induce changes only when the modulation frequency approaches that of the rhythms of physiological bioelectric function. No effects were found at modulation frequencies either below 8 Hz or above

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16 Hz. An increase in the number of slow rhythms and spindle-shaped firings in the EEG signal as well as a change in the discharge frequency of neurons have been observed in the visual cortex of rabbits when exposed to 2.4 GHz (Chizhenkova 1988). In another experimental series, an averaged EEG frequency spectrum was studied in rats exposed to an intermittent (1 min on, 1 min off) weak (0.1–0.2 mW/cm^2) microwave (945 MHz) field, amplitude-modulated at 4 Hz (Vorobyov et al. 1997). A significant elevation of the EEG hemispheric asymmetry was observed in the frequency range of 10–14 Hz during the first 20 seconds of the stimulation period (Vorobyov et al. 1997).

The majority of recent studies have been conducted on human volunteers. In these experiments microwave exposure has been reported to correspond to that of an average cellular GSM (Global System for mobile communication) phone (Borbely et al. 1999, Huber et al. 2000, 2005, Curcio et al. 2005, Krause et al. 2000b, 2004, Loughran et al. 2005). Such a pulse modulated radio frequency exposure consists of 900 MHz carrier radio frequency EMF and includes low frequency modulation components of 2, 8, 217, 1736 Hz and the corresponding harmonics (Huber et al. 2002).

Several studies have covered the influence of pulsed high-frequency electromagnetic fields of digital mobile phones on human sleep and waking EEG. An increase in the spectral power of the EEG during sleep when exposed to the EMF before and during sleep has been reported (Borbely et al. 1999, Huber et al. 2000). According to these results, a maximum rise occurs in the frequency bands of 9.75–11.25 Hz and 12.5–13.25 Hz rhythms during the initial part of sleep. The authors also report that unilateral exposure induces no hemispheric asymmetry of the EEG power and changes of brain function induced by the EMF last longer than the exposure period. The effect of the EMF beyond the exposure period as well as an increase in the alpha and beta power in the EEG was also reported in an earlier study (Reiser et al. 1995). The EMF emitted by a digital mobile phone increased the electroencephalogram spectral power in the 11.5–12.25 Hz frequency range during the initial part of sleep following the exposure (Loughran et al. 2005).

The exposure to pulse-modulated 900 MHz EMF has been reported to enhance the sleep and waking EEG power in the alpha frequency range (Huber et al. 2002, Curcio et al. 2005). Exposure to the same EMF without pulse modulation has been reported not to enhance the waking or sleep EEG power (Huber et al. 2002). The authors have demonstrated that exposure to a pulse-modulated microwave will alter not only the EEG but also regional cerebral blood flow (Huber et al. 2002, Huber et al. 2005, Curcio et al. 2005). Exposure to the EMF modulates the responses of the EEG oscillatory activity in the 6–8 and 8–10 Hz frequency bands, specifically during the cognitive processes (Krause et al. 2000a). However, these authors have not been able to confirm their early findings in further studies (Krause et al. 2004). Other researchers have reported that exposure to a microwave does not alter the resting EEG (Hietanen et al. 2000, Krause et al. 2000b). A recent study suggests that microwaves emitted by mobile phones have effects on brain oscillatory responses during cognitive processing in children (Krause et al. 2006). However, the observed effects of the pulse-modulated microwave were subtle and the underlying mechanisms still remain unknown.

On the other hand, recent achievements in the electric and transcranial magnetic stimulation (TMS) in brain research and neurotherapy have renewed interest in the possible effects of EMF at different frequencies (Lemon 2002, Rohan et al. 2004). Changes in the behavioural state of the brain are typically accompanied by changes in the frequency and spatial coordination of rhythmic bioelectrical activity in the neocortex. Cortical excitability by magnetic fields has been shown to vary at different frequencies: low-frequency TMS caused inhibition whereas stimulation at higher frequencies produced activation (Lemon 2002). Therefore, we can assume that any effects caused by the electromagnetic radiation on the bioelectrical activity of the human brain may also depend on the stimulation frequency. Whereas microwaves cannot cause any regular changes in the movement of ions due to their small cross-section of absorption (the wavelength of the microwave is much larger than the dimensions of a cell) as well as their inertial properties and viscosity of the liquid medium (Adair, 2002), the effect may depend on the modulation frequency rather on the radio frequency. Experimental findings have also demonstrated that low-frequency pulse modulation of radio frequency EMF is necessary to induce changes in the waking and sleep EEG, and that pulse modulation is crucial for radio frequency EMF-induced alterations in brain physiology (Huber et al. 2002, Huber et al. 2005). However, the effects of microwaves modulated at different frequencies cannot be differentiated in experiments conducted with microwave exposures that correspond to the radiation of a cellular phone.

The aim of this study was to evaluate the effect of microwaves modulated at different frequencies within the physiological spectrum of the EEG of human EEG rhythms. To accomplish this purpose, three modulation frequencies: 7, 14 and 21 Hz were selected. Changes in the power of the EEG theta, alpha and beta rhythms, caused by microwaves

modulated at these frequencies, were analyzed. The hypothesis of our study is that changes in the EEG power, caused by modulated low-level microwave exposure, differ under different modulation conditions.

Methods and equipment

Subjects

Our experiments were carried out on a group of healthy volunteers, consisting of 13 young persons (aged 21 – 30), 4 male and 9 female. Their physical and mental condition (tiredness, sleepiness) was evaluated by a questionnaire and a clinical interview before the experiment. All the subjects selected had neither medical nor psychiatric disorders and tired or sleepy persons were excluded. After the recordings, they were asked to describe how they felt during the experiment. The subjects reported no effect on their alertness and no experience of any strain during the recordings. Each participant was aware of the purpose of the experiments and gave a written consent.

During the experiments, the experimenter and the subject stayed in the same laboratory room. The room was dark and the subjects were lying in a relaxed position, with eyes closed and ears blocked during the experiments.

All the subjects passed the recording sessions (Figure 1) with exposure and sham twice. The subjects were not informed of their exposure during the session, however, they were aware of the possibility of being exposed. During each double-blind recording session, the exposed and sham-exposed subjects were randomly assigned. The succession of the modulation frequencies was randomly assigned by using a computer program. Subjects were subjected to only one experimental EEG recording session daily between the times of 9 a.m. to noon.

The study was conducted in accordance with the Declaration of Helsinki and was formally approved by the local Medical Research Ethics Committee.

Microwave exposure

Microwave exposure at the non-thermal level of field power density was selected to be identical to that in our previous studies (Lass et al. 2002, Hinrikus et al. 2004). The only change was that three different modulation frequencies were applied. Exposure conditions were the same for all subjects.

Electromagnetic radiation of 450 MHz was generated by a signal generator (model SML02, Rhode & Swartz Munich, Germany). The radio frequency signal was 100% pulse-modulated using a modulator (SML-B3, Rhode & Swartz, Munich, Germany) at

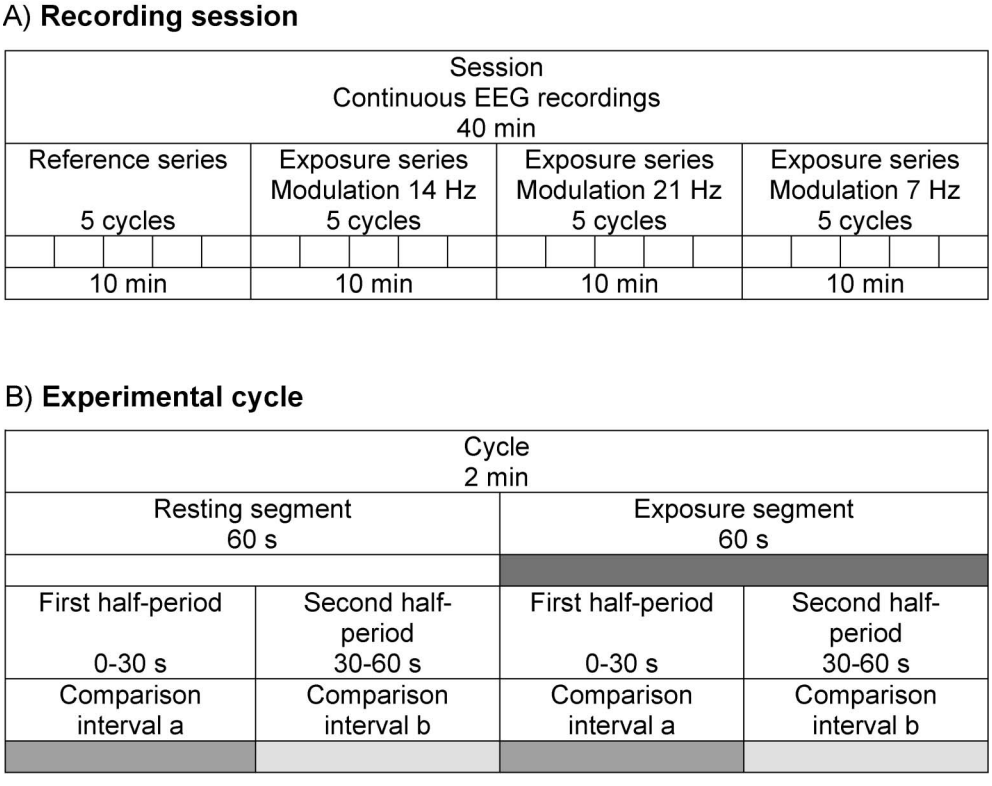

Figure 1. Schedule of the recording protocol: (a) recording session; (b) experimental cycle.

frequencies of 7, 14 or 21 Hz (duty cycle 50%). The signal from the generator was amplified by a power amplifier (model MSD-2597601, Dage Corporation, Stamford, CT, USA). The generator and the amplifier were carefully shielded. The output power of 1 W electromagnetic radiation was guided by a coaxial lead to the 13 cm quarter-wave antenna, (NMT450 RA3206, Allgon Mobile Communication AB, Stockholm, Sweden), located 10 cm from the skin on the left side of head.

The spatial distribution of the power density of electromagnetic radiation was measured by a field strength meter (Fieldmeter C.A 43, Chauvin Arnoux, Paris, France). The measurements were done by the Central Physical Laboratory of the Estonian Health Protection Inspection. The calibration curves of the dependence of field power density on the distance from the radiating antenna were obtained from these measurements, performed under real experimental conditions. During the experiments a Digi Field C field strength meter (IC Engineering, Thousand Oaks, CA, USA) was used to monitor the stability of the electromagnetic radiation level. The average field power density of the modulated microwave at the skin on the left side of head was 0.16 mW/cm^2 as estimated from measured calibration curves, The spatial distribution of the field density around head and upper torso was non-uniform: the Digi Field C field strength meter indicated a maximal level of 0.16 ± 0.02 (mean plus or minus the standard error of the mean, SEM)mW/cm^2 at the left side of the head and a minimal level of 0.025 ± 0.01 mW/cm^2 at the right side of the head and 0.08 ± 0.01 mW/cm^2 above head. The measured field power density was 0.8 ± 0.2 mW/cm^2 near the neck and shoulder on the left side and 0.03 ± 0.01 mW/cm^2 near the neck and shoulder on the right side. The estimated value of absorption rate was 0.35 W/kg (Hinrikus et al. 2004).

Recording protocols and equipment

Our experimental study was conducted according to the recording protocol that was identical for all subjects.

The protocol of a recording session consisted of one series of reference recordings and three series of microwave exposure recordings, with five cycles of exposure in each series (Figure 1A). Each series was conducted at a constant modulation frequency of 7, 14 or 21 Hz. The frequencies were randomly assigned between the series. A recording session lasted for 40 minutes, during which the subject was resting with eyes closed. EEG was continuously recorded. Each subject went through two recording sessions.

One experimental cycle (Figure 1B) consisted of a 60-second exposure segment with microwave radiation and a 60-second resting segment without microwave radiation. For the EEG analysis, the exposure and resting segments were divided into a first (0 – 30 s) and a second (30 – 60 s) half-period which were selected as intervals for comparison. Average values of the EEG power (a) in the first half-period of the exposure and resting segments, and (b) in the second half-period of the exposure and resting segments, respectively, were compared.

Sham recordings were performed according to the same protocol except the microwave power was switched off. In the case of reference and sham recordings, the exposure and resting segments were considered as odd and even minutes of the recordings. The sham protocol was also applied twice to each subject.

The EEG recordings were made using a Cadwell Easy II EEG measuring equipment (Kennewick, WA, USA). To record EEG, nine electrodes were placed on the subject's head according to the international classification system of 10 – 20-electrode position. Channels were selected to cover the entire head: frontal – FP1, FP2; temporal – T3, T4; parietal – P3, P4; occipital – O1, O2; and the reference electrode Cz. The EEG recordings were stored on a computer at the sampling frequency of 400 Hz. 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 parasitic interaction between the recording and radio-frequency equipment, the instrumentation was validated before the experiments. Artifacts can be induced by parasitic demodulation of the radio-frequency electromagnetic components and interference between low-frequency components of the EEG electrodes and equipment. To conduct testing, an EEG cap was placed on a passive phantom of a human head and recordings were conducted according to the procedures to be used in the experimental study. Multichannel recordings detected spectral components at the modulation frequencies (7, 14 or 21 Hz) and their third harmonics in some of the channels. Spectral components of the third harmonics of the modulation frequencies were depressed over 30 dB. No other spectral components were detected. Artifacts at the modulation frequencies of 7, 14 and 21 Hz were removed from the EEG signals by off-line filtering during the pre-processing of the signals in the programming and signal-processing environment (LabVIEW, National Instruments, Austin, TX, USA).

Data analysis

Separate EEG analyses were performed for each EEG frequency band (theta, alpha, beta1 and beta2) and the first half and second half of the resting and exposure segments of the experimental cycle.

Initially, the power of four basic EEG frequency bands: theta (4 – 6.8 Hz), alpha (8 – 13 Hz), beta1 (15 – 20 Hz) and beta2 (22 – 38 Hz) were extracted from the total EEG (0.5 – 48 Hz) by filtering. Elliptic bandstop filters with an attenuation of 50 dB in the stopband were used. Such a selection of the EEG frequency bands excluded the frequencies of 7, 14 and 21 Hz and possible related artifacts from the analysis.

The method of our analysis in each EEG frequency band and half-period of the segments of the experimental cycle consisted of two main steps.

First, the average value of the EEG power within a comparison interval in a time-window T was calculated as

$$e_i = \frac{1}{N}\sum_{r=1}^{N}[V(r)]^2, \tag{1}$$

where $V(r)$ is the amplitude of the recorded signal in a sample r and N is the number of samples in the time-window T.

The width of the time-window T is a free (adjustable) parameter. The value of T must be large enough to minimize the influence of natural EEG fluctuations but not too large. It must not exceed the time constant of brain adaptation. In this case, the selected value of T was 30 s. During the 30 seconds, the number of samples was $N = 12000$. Finally, the 30-second half-periods of the segments of the experimental cycle in the presence and absence of microwaves were selected as signal intervals for comparison.

Second, change of the EEG power in a cycle as a relative change of the average EEG power between the exposure and resting segments of the experimental cycle was calculated as follows:

$$S_c = \frac{e_{2c} - e_{1c}}{e_{1c}} \times 100\%, \tag{2}$$

where e_{1c} and e_{2c} are the average values of the EEG power at comparison intervals in the half-period segments of the experimental cycle without and with microwaves, respectively. The values of the parameter S_c were calculated for two sessions ($5 + 5$ cycles or 10 values), each exposure condition (4) and EEG channel (8). The total number of S_c values for each subject amounted to 320 in each EEG frequency band.

The value of S_c, which describes the relative change in the EEG power between the recording segments with and without microwave exposure during an experimental cycle, was selected as the parameter for further statistical analysis.

The effect of the condition of exposure was estimated by averaging the S_c values over ten cycles of exposure at the fixed modulation frequency. The parameter S_n, the average relative change in the EEG power at a fixed modulation frequency for a subject n, was calculated as follows:

$$S_n = \frac{1}{10}\sum_{c=1}^{10} S_c, \tag{3}$$

where S_c is the value of parameter calculated according to (2) for a single cycle. Values of S_n were calculated for each subject in each EEG channel. The total number of S_n values for a subject was 8 (EEG channels) in each EEG frequency band (4 bands) and half-period segments of the experimental cycle. The values of S_n, the average relative change in the EEG power at a fixed modulation frequency for a subject n were calculated for each member of the group ($n = 13$).

For the reference EEG, the same parameters were calculated at 30-second comparison intervals using the first or second half-periods of the odd and even minutes of the recordings. The same procedure was followed in sham recordings.

Signal processing and parameter calculations were performed in the LabVIEW (National Instruments, Austin, TX, USA) programming and signal processing environment.

In the statistical analysis each EEG frequency band (theta, alpha, beta1 and beta2) and the 30-second half-periods of the segments of the experimental cycle were considered separately. In each database calculated parameters S_c for ten cycles were indexed by three characters: EEG channels (8), exposure conditions (4) and individual subjects (13).

Software package Statistica 6.0 (StatSoft. Inc., Tulsa, OK, USA) was used in the statistical analysis. For statistical comparisons, analysis of variance (ANOVA) was performed. Dependent variable data (calculated parameter S_c for ten cycles) were entered into these analyses with two types of categorical factor: EEG channel (8 channels) and exposure condition (reference, 7, 14 or 21 Hz). The post-hoc Bonferroni correction (software package Statistica 6.0, StatSoft. Inc., Tulsa, OK, USA) for multiple comparisons was performed to evaluate the significance level of comparisons between individuals under different exposure conditions.

ANOVA was performed separately for each EEG frequency band and the half-period of the cycle. Therefore an additional Bonferroni correction for

multiple-comparisons was applied: *p*-values (evaluated using Statistica 6.0 package) were multiplied by the number of separate analysis (4 EEG bands and 2 half-periods). The confidence level of 0.05 was considered statistically significant to the Bonferroni corrected *p*-values.

Results

Figure 2 illustrates the effect of the microwave exposure modulated at the frequencies of 7, 14 and 21 Hz and the reference, in different EEG frequency bands for the whole group ($n = 13$). It shows the calculated values of the relative percentage change of the EEG power between the exposure and resting segments of the experimental cycle S_c averaged over 10 cycles of exposure, 8 electrodes and 13 subjects. The results obtained are different for the first and the second half-period of the segments of the experimental cycle: changes are most evident during the first half-period, except for the theta rhythm. Microwave exposure at the higher modulation frequencies of 14 and 21 Hz caused an increase in the EEG average power (except for the theta rhythm) during the first half-period of the segments of the experimental cycle. The increase is less noticeable at the modulation frequency of 7 Hz. The values of the relative change are about twice as high in the EEG alpha band (16–17%) as in the EEG beta band (6–7%) at modulation frequencies of 14 and 21 Hz. Changes in the EEG average power during reference recordings were lower (less than 3% for alpha and 1.5% for beta frequencies) and values of the reference bars are close during the first and the second half-period of the segments (except the theta rhythm). In the case of sham recordings, values shown by the bars are 5% or less for alpha and 2% or less for beta frequencies and values shown by the bars are close during different series.

The average values of changes in the EEG power for the whole group and all the channels may have had masked larger changes in effects for individuals and different channels. Therefore, individual changes in the EEG power in different channels for each subject were calculated. The changes caused by microwaves in the EEG P-channel powers of each of the 13 subjects are presented in Figure 3. The greatest effects, up to 150%, were witnessed in the EEG alpha frequency range for two subjects. The EEG power inside the beta rhythm increased with exposure at a maximum of up to 50% for two subjects and up to 25% for five subjects. No obvious outliers were found in the theta frequency range.

Results of ANOVA for the whole group are presented in Table I. Our analysis detected significant differences between the EEG channels only in the case of the beta2 rhythm. Therefore, spatial distribution of the effect in different EEG channels was excluded in the post-hoc analysis for the group. The ANOVA revealed a significant difference between the exposure conditions during the first half-period of the segments of the experimental cycle for the alpha, beta1 and beta2 frequency bands. The differences were not statistically significant during the second half-period of the segments except for the alpha band. No significant differences were detected between the exposure conditions in the case of the EEG theta band. Neither were any statistically significant differences detected between sham recordings.

Table II shows the Bonferroni test results for the post-hoc comparisons of the reference and the exposed series of recordings for the whole group. The results demonstrate a significant difference between the reference and the exposed series at different modulation frequencies only during the first half-period of the segments of the experimental cycle. Significant differences appear between the reference and the exposed series at the modulation frequency of 14 Hz for the EEG alpha and beta1 rhythms and at the modulation frequency of 21 Hz for the EEG alpha, beta1 and beta2 rhythms. No significant differences between the reference and the exposed series were detected at the lower modulation frequency of 7 Hz. In the case of the EEG theta rhythm, no significant differences were found between the reference and the exposed series at any modulation frequency or half-period of the segments of the experimental cycle.

Discussion

The results demonstrate that clear differences exist in the EEG power between recordings with and without microwave exposure. Changes in the EEG power became evident during the first half-period of the segments of the experimental cycle at the EEG band frequencies lower than the modulation frequency or close to that (see Figures 2 and 3). The findings show that the EEG power level increases at the modulated microwave exposure in all EEG band frequencies except the theta band. Changes caused by the exposure were higher than natural fluctuations in the reference EEG power level and equivalent changes in sham recordings and the effect were statistically significant. No effect was observed at the theta frequencies.

Based on stable changes in the EEG power during the first and the second half-period of the segments on reference recordings (Figure 2) it can be concluded that our results provide an adequate picture of the cortical activity: there is no difference between the first and the second half-period of the segments of the experimental cycle without exposure.

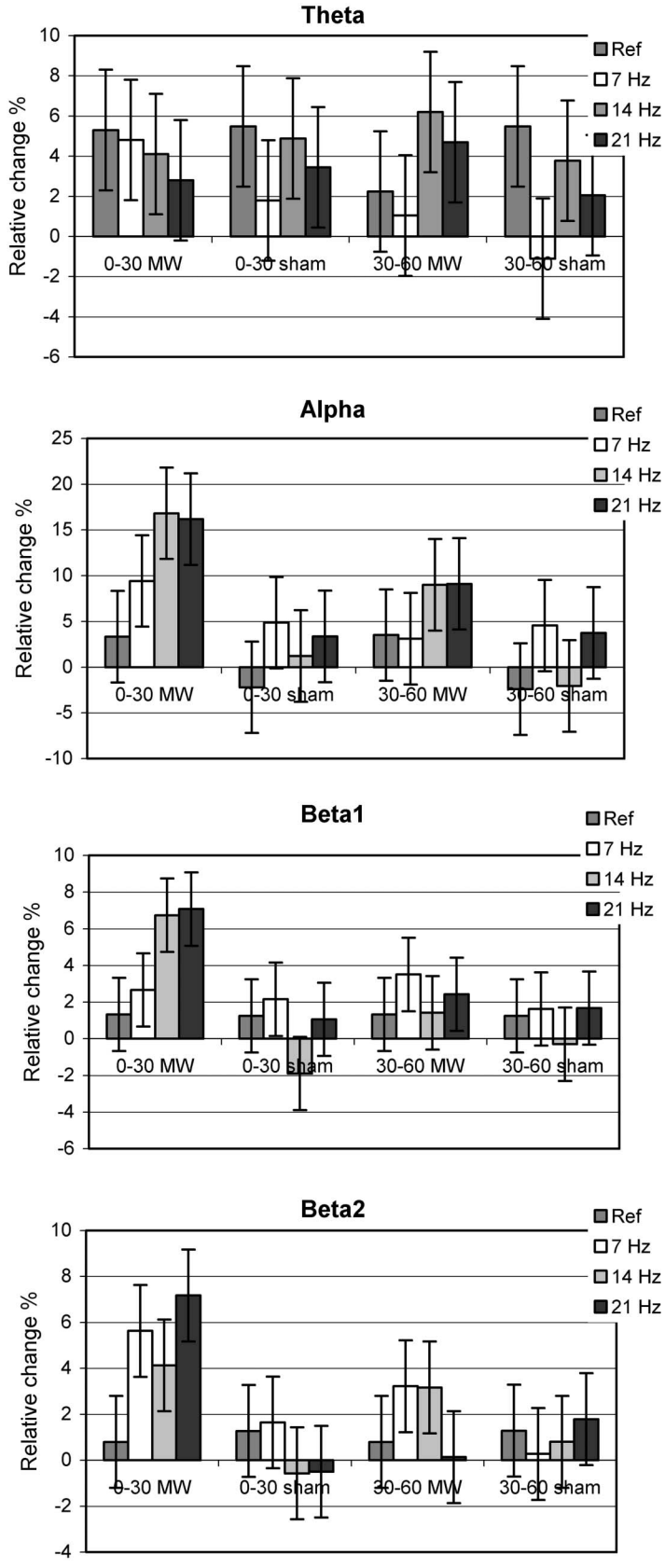

Figure 2. Mean values of relative changes in the EEG power (the average relative change in the EEG power at a fixed modulation frequency for a subject n parameter S_n averaged over all subjects $n = 13$ and 8 EEG channels) for the four different EEG frequency bands (theta, alpha, beta1 and beta2) and exposure conditions (Reference, 7, 14 and 21 Hz) in exposed (MW) and sham recordings during the first (0 – 30 s) and the second (30 – 60 s) half-periods of the segments of the experimental cycle. Error bars indicate standard deviation for m = 1040 measurements.

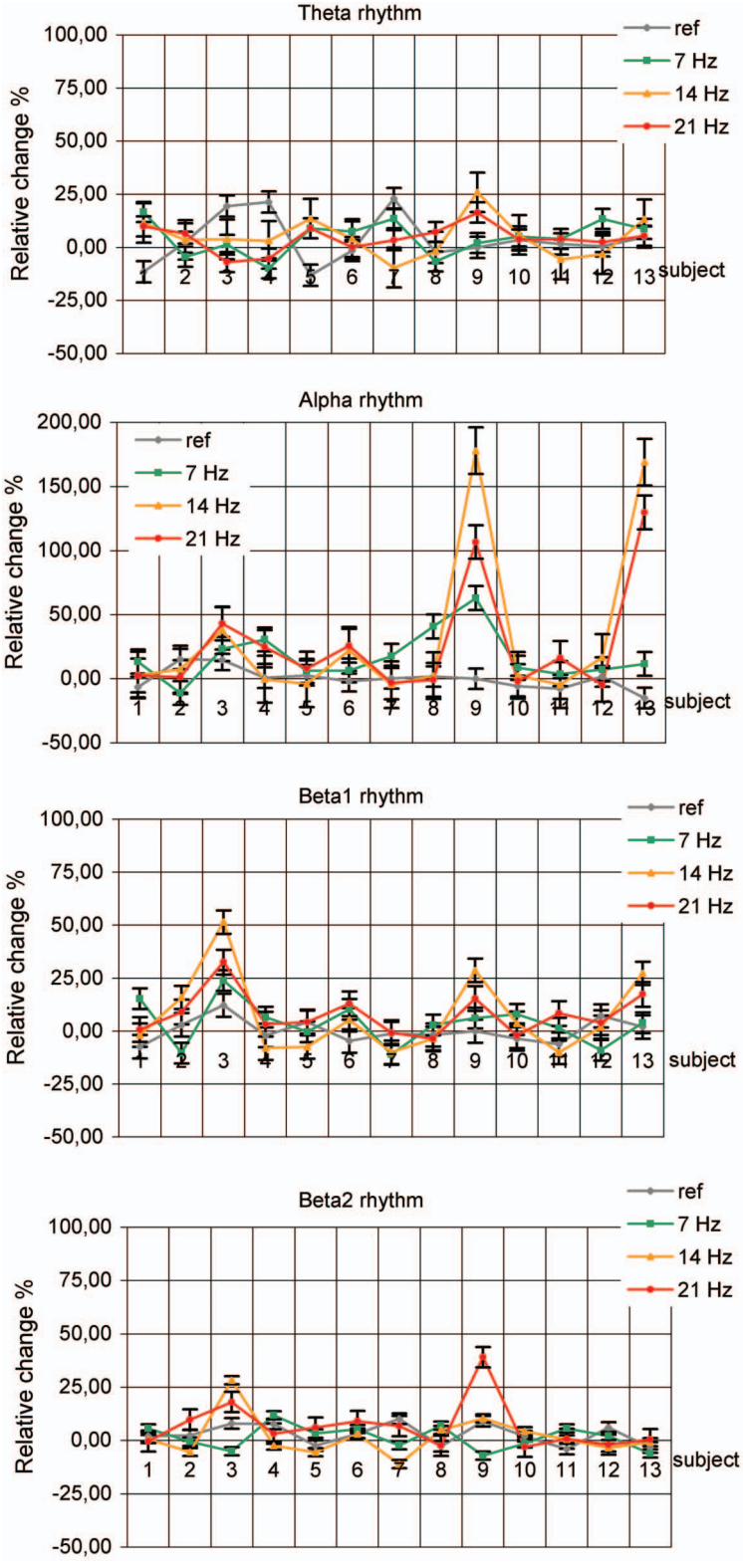

Figure 3. Percentages of relative change in the EEG power at different modulation frequencies (the average relative change in the EEG power at a fixed modulation frequency for a subject n parameter S_n) during the first (0 – 30 s) half-periods of the segments of the experimental cycle in P3 – P4 channels for individual subjects. Error bars indicate standard deviation for m = 20 measurements.

The changes that appear in the EEG power promptly after the microwave exposure onset (during the first half-period of the exposure segment) demonstrate that the brain reacts to the external stimulus within some seconds. The decrease of the changes within a certain time (smaller value or

Table I. Results of 3-D ANOVA for relative changes in the EEG power (parameter S_c) for eight different EEG channels and four microwave exposure conditions (reference, 7 Hz, 14 Hz and 21 Hz) during the first (0–30 s) and the second (30–60 s) half-period of the segments of the experimental cycle for the whole group of subjects ($n = 13$) (significant values are marked in bold).

	Differences between EEG channels				Differences between exposure condition			
	0–30 s		30–60 s		0–30 s		30–60 s	
	F(7.384)	*p*	F(7.384)	*p*	F(3.384)	*p*	F(3.384)	*p*
Microwave exposed recordings								
Theta	0.3036	0.752	1.1308	0.342	1.3029	0.273	1.4839	0.218
Alpha	1.0924	0.367	0.7042	0.668	3.2719	**0.021**	3.5989	**0.013**
Beta1	0.0885	0.998	0.1702	0.990	5.0796	**0.001**	2.1919	0.064
Beta2	4.9812	**<0.001**	3.1770	**0.002**	4.0472	**0.007**	1.2673	0.285
Sham recordings								
Theta	0.2780	0.788	0.2426	0.809	0.0180	0.982	1.1764	0.861
Alpha	0.3585	0.724	0.2628	0.793	0.1449	0.885	0.1963	0.905
Beta1	0.1697	0.865	0.1495	0.881	0.0722	0.944	0.1700	0.865
Beta2	1.1326	0.257	1.0838	0.279	0.8646	0.387	0.7950	0.424

Table II. Results of post-hoc Bonferroni test for comparison of reference and exposed series at different modulation frequencies and during the first (0–30 s) and the second (30–60 s) half-periods of the segments of the experimental cycle (significant values are marked in bold) for the whole group of subjects ($n = 13$).

	p-values					
	7 Hz		14 Hz		21 Hz	
EEG Bands	0–30 s	30–60 s	0–30 s	30–60 s	0–30 s	30–60 s
Theta	1.000000	1.000000	1.000000	0.077599	0.338937	0.731238
Alpha	1.000000	1.000000	**0.041827**	0.158027	**0.040963**	0.135282
Beta1	1.000000	0.759753	**0.018230**	1.000000	**0.009988**	1.000000
Beta2	0.071871	1.000000	0.495683	1.000000	**0.005771**	1.000000

absence of changes during the second half-period of the exposure segment) can be explained by the physiological adaptation of the brain to the external stressor.

The microwave radiation modulated at 14 and 21 Hz as an external stressor caused an increase in the EEG alpha and beta1 power (14 and 21 Hz modulation) and beta2 power (only 21 Hz modulation). At the lower modulation frequency of 7 Hz, a minor effect occurred and it was not statistically significant. Thus it can be suggested that the effect of an external stimulus on brain oscillations is stronger if the frequency of the stimulus is higher or close to the physiological frequency of brain rhythms. Therefore, the 7 Hz modulated radiation could affect only the EEG theta power, but the theta band appears to be insensitive to the exposure. Neither were any statistically significant changes detected in our previous study with a 450 MHz microwave modulated at 7 Hz (Hinrikus et al. 2004).

A major increase in the EEG average power of 16–17 % was observed in the EEG alpha frequency range. For some subjects, the increase reached up to 150%. This result is in agreement with the findings reported recently, where the GSM mobile phone radiation caused an increase in the EEG power mainly in the alpha frequency band (Huber et al. 2002, Curcio et al. 2005). On the other hand, that trend in the alpha band is similar to the phenomena caused by TMS, where lower frequencies cause inhibition and higher frequencies cause cortical activation (Lemon 2002).

Statistically significant increases of 6–7% for the whole group were observed in the EEG power in the beta frequency range (for some subjects up to 50%). A similar trend was observed during TMS, where stimulation at the higher modulation frequency of 21 Hz produced an increase of the bioelectrical activity of the brain in the EEG beta band (Lemon 2002). It is interesting to mention that the effect caused by a chemical stressor results in similar changes in the resting EEG: an increased beta absolute power has also been observed in alcohol-dependent subjects (Rangaswamy et al. 2002).

The exposures applied in this study had no effect on the EEG power in the theta frequency band: the relative changes of 5–6% remained the same for the first and the second half-period of the segments of the experimental cycle and were statistically insignificant, obviously caused by the natural variability of the EEG.

The experimental data showed that changes in the EEG rhythms caused by pulse-modulated low-level microwave exposure are different at different modulation frequencies. Our results support the idea that the influence of the microwave exposure on the cortical activation depends on the frequency. This conclusion is in good agreement with the results reported by other authors who have demonstrated that the exposure to a pulse-modulated microwave alters brain physiology, as reflected in the regional changes of cerebral blood flow. Furthermore, the modulation paradigm was shown to be critical for microwave-induced increases of cerebral blood flow (Huber et al. 2005).

The mechanisms of the observed effects of a pulse-modulated microwave are unknown. However, we can assume that the high-frequency field causes fluctuations and damped vibrations of the charged particles and membranes. The fluctuations initiated by the microwave in the movement of ions and membranes affect the gating variables and nerve cell properties. This phenomenon is similar to the effect caused by Brownian motion initiated by temperature, and results in the same effects – the mobility of the ions and fluctuations in membrane motion will increase. Such a quasi-thermal field-tissues mechanism is supported by the findings that low-level electromagnetic radiation exposure induces changes in heat shock proteins (Leszczynski et al. 2002). Therefore, the quasi-thermal effect produced by a microwave can lead to altered neuronal-cortical activity. An increase in cerebral metabolism regarded as a consequence of an altered cortical activity induced by exposure to a modulated microwave has been described in other studies (Huber et al. 2002, 2005).

Experimental effects that depend on the low-frequency modulation of electromagnetic radiation can also be related to more complicated nonlinear responses in biological tissues and living cells (Balzano & Sheppard, 2003).

Conclusion

The results of the experimental study with a low-level 450 MHz microwave pulse-modulated at 7, 14 and 21 Hz support our hypothesis that the effect of the modulated microwave on the human EEG differs at different modulation frequencies. The microwave exposure modulated at 14 and 21 Hz enhanced the EEG power in the alpha and beta frequencies whereas no enhancement of the EEG power was found during exposure at the modulation frequency of 7 Hz. Thus, the EEG power increased in the frequency bands that were close to or lower than the modulation frequency of the microwave. The changes were more obvious at the beginning of the exposure segments. No changes in the EEG theta power were detected for any modulation frequencies.

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