HIGH-FREQUENCY ELECTROMAGNETIC RADIATION: NONTHERMAL EFFECTS AND SAR MEASUREMENT

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High-frequency electromagnetic field (HF EMF) becomes a common part of our environment because it is produced by many artificial sources as radars, transmitters and especially cellular (mobile) phones. The consideration of a possible harmful effect of it the term "electromagnetic smog" is often used for this situation. Among the possible sources frequently mentioned, first of all many mobile phones are in use and their number is rapidly rising (10 million devices were in use in the Czech Republic in 2005!). During a call the source of radiation is located close to the head. That is why there is a question of possible negative effects of HF EMF on the human body, especially on the brain. HF EMF can influence tissues by both thermal and nonthermal effects. While the thermal effect is relatively well known, the nonthermal effects are still discussed and remain unclear.

The basic effect of electromagnetic field is dielectric heating. In case of direct contact of antennal source (in switch-on mode) severe skin burns can be provoked. HF EMF with higher gradient and short wavelength heats up the tissue; the grade of heating depends on the dielectric loss according to its structure. The adipose tissue and elements have lower lossy values in comparison with body fluids; that is reason of lower increase of temperature. Generally, thermal gradients between lipophilic and hydrophilic structures in tissue exist. In fact, during exposure to radiation at a constant frequency, the wavelength is changed in the tissue and it depends on the dielectric constant. In the case of HF EMF, the "heating source" is directly a corresponding tissue. The energy distribution in different body structures is unequal and decreases in depth.

In our laboratory, the unique mouse strain with inborn neurodegeneration is studied. Lurcher mutant mice (Lurchers) represent a natural animal model of genetically determined olivocerebellar degeneration (53). Heterozygote individuals (+/Lc) are characterized by a complete postnatal loss of cerebellar Purkinje cells (excitotoxic apoptosis) and by decrease of number of granular cells and inferior olive neurons (secondary to the loss of Purkinje cells). The affected homozygotes (Lc/Lc) are not viable. Unaffected homozygotes, wild type mice (+/+) are completely healthy and serve as controls. Our previous results suggest that in Lurchers some cognitive functions are changed – in series of experiments studying the development of spatial learning in Lc/+ and +/+ during the first month of life using the standard Morris water maze (29, 46, 47, 53, 54). Some brain neurons in +/Lc

are more sensitive to neurotoxic substances (13) and the animals display a higher degree of CNS excitability compared with +/+ (14, 48). We obtained similar findings through the examination of brain cortical activity after previous electrical and drug stimulation (3, 39). Significant changes of hippocampal activity (LTP, 11) were also found in anesthetized Lc/+ in comparison with +/+ (4, 5).

Our working hypothesis was that some brain structures in Lurchers can be more sensitive (i.e. they can have a lower threshold) to different kinds of stimulation – including HF EMF – resulting in probable neuronal injury in comparison with wild-type mice.

Recent experimental results performed in our laboratory (900 MHz, 10 W, whole-body exposure, constant) were:

- The measurement of excitability after acute and long-term HF EMF exposure using audiogenic epilepsy method showed more intensive reaction in +/Lc than +/+ but those differences were only within the margin of statistical insignificance. An examination of CNS inhibition using pentobarbital sleep method suggested significantly lower mortality in HF EMF exposed animals and the difference was higher in +/+ than in +/Lc (15, 16).
- The spatial learning ability (tested in a Morris water maze) revealed only slight differences (some significant) between +/Lc and +/+. Strain and age related differences were also found (45).
- Histochemical examination showed higher NADPH-diaphorase positivity (NOS activity marker) in hippocampus of C57Bl/7 mice after HF EMF exposure in comparison with unaffected controls. Mice of C3H strain exhibited lower NADPH-d positivity generally and the differences between irradiated animals and controls were only slight (48).
- Immunocytochemical depiction of *c-fos* (as acute cellular stress marker) revealed differences between the irradiated and control animals; *c-fos* activity was present only in animals after HF EMF exposure (8).
- The evaluation of spontaneous brain cortical activity (ECoG) recorded simultaneously with HF EMF exposure) suggested changes in brain electrogeny. ECoG revealed a shift towards lower frequencies with significant differences between LMM and WTM. Spontaneous hippocampal activity showed opposite changes with an increase of theta oscilations during HF EMF exposure (6, 7, 54). Some mild somatic (body weight) and neurobehavioral (lower swimming speed and motor activity) changes were found in the HF EMF exposed newborn and young adult +/Lc and +/+ (49).

Nevertheless, one of crucial problem in above mentioned studies is the absence or only minimal information about real absorption/distribution of energy during HF EMF exposure. It seems to be generally accepted that the SAR quantification in studies are important with possible suggestions in the radiofrequency fields. Because it is impossible to measure SAR directly (42) this is the main reason to find possible methods for indirect quantification of Specific Absorption Rate in small laboratory animals.

METHODS

Exposure chamber

The exposure chamber operating at 900 MHz was designed with the following issues in mind: inducing uniform field, eliminating external radiation, determining accurate absorbed power, providing sufficient space for mice and maintaining costs within budget. The chamber consists of a circular waveguide terminated by matched loads. The waveguide is made of a copper sheet (Fig. 1). The electrical resistance of the matched load should grow linearly in a direction of the wave's propagation. Therefore, the shapes of matched loads are conical and they are made of plastics filled up with salted water. The loads serve as a prevention from possible unwanted resonances between the mouse and waveguide bottoms.



Fig. 1 A scheme of exposure chamber

The radius of the waveguide is 120 mm and the length 1650 mm. A circular polarized wave comprised by the dominant mode TE_{11} is excited in the waveguide. The wave is formed by two monopole antennas which have the mutually orthogonal orientation and the distance between them is equal to the wavelength divided by four. Circular polarized wave provides relatively constant coupling of the field to each mouse regardless of its position, posture or movement. In the waveguide there are two other monopoles serving for scattering parameters measurement with the aid of the vector analyser. The absorbed power in mouse is determined by the analysis of

scattering parameters. In the chamber there is a ventilation hole through which electric fan-forced ventilation air is introduced and let out through a second hole in order to reduce the stress of the mice and to maintain constant temperature inside the waveguide. The mice are placed in the circular plastic box.

Dosimetry

Dosimetry is an inherent task for the design and use of exposure systems for in vivo or in vitro experiments in the investigation of any possible biological effects of radiofrequency (RF) exposure. Dosimetry is a quantification of the magnitude and of distribution of absorbed electromagnetic energy within biological objects that are exposed to electromagnetic fields. At RF, the dosimetric quantity, which is called specific absorption rate (SAR), is defined as the rate at which energy is absorbed per unit mass. The SAR is determined not only by the incident electromagnetic waves but also by the electrical and geometric characteristics of the irradiated subject and nearby objects. It is related to the internal electric field strength as well as to the electric conductivity and the density of tissues. Therefore, it is a suitable dosimetric parameter even when a mechanism is determined to be "athermal". SAR distributions are usually determined from measurements in animal tissues. It is generally difficult to measure SAR directly in a living biological body, so dosimetry efforts are forced to rely on computer simulation.

We are able to compute the whole-body averaged SAR with the aid of an analysis of measured scattering parameters.

$$SAR_{WBAV} = \frac{P_i(IL - IL')}{m} \left[\frac{W}{kg}\right]$$

whereby P_i is incident power, IL is insertion loss of empty chamber (without the mouse) and IL' is insertion loss of the chamber with the mouse. Insertion losses can be computed with the aid of scattering parameters by the following formulas

$$IL = 1 - |S_{11}|^2 - |S_{21}|^2$$
, $IL' = 1 - |S'_{11}|^2 - |S'_{21}|^2$,

whereby S_{11} is the input port voltage reflection coefficient of the empty chamber, S_{21} is the forward voltage gain of the empty chamber, S'_{11} is the input port voltage reflection coefficient of the chamber with the mouse, S'_{21} is the forward voltage gain of the chamber with the mouse.

Simulation results



Fig. 2 A scheme of mouse position in the box

All mentioned simulations were dependent mainly on the mouse position (Fig. 2); the figure 3 shows the dependance of the power distribution on the mouse position according to Fig. 2 and feeding sources. It is obvious that for non-circular wave (usage source 1 or source 2 only) SAR strongly depends on the position of the mouse. The percentage difference of SAR is about 91% for perpendicular positions (between position 1 and position 2) of the mouse. For the circular polarization the percentage difference of SAR is about 7% (between position 1 and position 2) and 27% (between position 1, 2, and position 3).



Fig. 3 A comparison of dependance of SAR distribution on the mouse position

DISCUSSION

There is some experimental evidence about the nonthermal effect of HF EMF exposure, especially on functional-morphological characteristics of the brain. Repacholi (32) has recently concluded in the World Health Organization review of the literature that HF EMF fields, continuous or pulsed, can affect membrane channels, mainly at fairly high intensities, but even at levels that do not cause significant heating. There have been reports of decreased rates of channel formation, decreased frequency of channel openings, and increased rates of rapid, burst-like firing (43). Arber and Lin (1, 2) reported an increase in membrane conductance and a decrease in the spontaneous firing of impulses in neurons of the snail Helix aspersa when exposed for an hour to continuous and amplitude modulated 2.45 GHz radiation. The effects were abolished by the application of ethylenediamine tetraacetic acid (EDTA), which creates chelates with calcium. However, they occurred at a high RF intensity, hence a clear dependence on a rise of tissue temperature. McRee and Wachtel (24) described a decrease in the electrical amplitude of impulses and a reduction in the excitability of the frog sciatic nerve when exposed to 2.45 GHz radiation, but only at high levels. Wachtel et al. (50) and Seman and Wachtel (36) also described a decrease in spontaneous activity of neurons isolated from the marine gastropod Aplysia at relatively high intensities. In experiments performed by Salford et al. (34), 915 MHz electromagnetic field (pulse modulated) had a significant effect on the permeability of blood – brain barrier

and, more recently (35), a neuronal damage in the cortex, hippocampus and basal ganglia in exposed rats was described. In the other recent study (27), short-term exposure (15 min) to 900 MHz induced strong glial reaction in the brain.

Probably the most controversial findings originate from studies concerning effects of HF EMF on brain electrogeny. Bawin et al. (9, 10) exposed cats, which had been previously conditioned to produce selected EEG rhythms in response to a light flash, to low level HF EMF. Changes were reported in the performance of the conditioned EEG response task and in various other behavioural parameters. It was argued that the fields acted directly on the brain tissue, thereby causing a minute release of calcium, resulting in changes in membrane excitability, which could possibly affect EEG rhythms. Takashima et al. (40) reported changes in the EEG of rabbits following exposure to a modulated HF EMF of 1–10 MHz, a frequency range outside the main interest but Shandala et al. (37) and Thuroczy et al. (41) reported about subtle effects of HF EMF on EEG in rats and rabbits exposed to RF fields within the frequency range of interest. McRee et al. (25) described experiments performed by Rosensteig of the US Environmental Protection Agency, who exposed rats to HF EMF from late fetal life until adulthood. He saw no changes in either the spontaneous EEG or the electrical responses evoked by flashes of light (visual evoked responses).

Laboratory studies investigating the effects of mobile phone signals on the spontaneous EEG in awake subjects have produced ambiguous results. Reiser et al. (31) reported that exposure to GSM signals was associated with increases some 15 minutes later in the power of EEG frequencies of about 10 Hz and above Roschke and Mann (33), were unable to detect any differences in EEG spectra related to the exposure to GSM signals. Mann and Roschke (26) reported that exposure to GSM-like signals reduced latency to sleep onset, and altered spectral characteristics of REM sleep, although a subsequent study by the same group (51) failed to replicate these findings. In another study (12), exposure to a "pseudo-GSM signal" (15 minute on/off cycles, 900 MHz, duty cycle of 87.5% rather than the 12.5% used in phone signals, and an estimated whole-body SAR of 1 W/kg) was associated with a reduced wake state after sleep onset and changes in EEG power spectra during the first of the night's episodes of non-REM sleep. In three studies "event-related potentials" (ERPs) were investigated during exposure to GSM-like signals. In the first (44), visual sensory responses to checkerboard reversal were found to be unaffected during exposure. In two other studies (18, 19) positive effects were reported. A recent study from Huber et al. (20) suggests some changes in EEG (especially during sleep).

Effect on behavioral properties

Results of earlier studies on rodents have shown that the threshold at which acute RF exposure disrupts learned operant behaviour lies between 2.5 and 8 W/kg whole-body SARs, with an associated rectal temperature rise of about 1 °C. Deficits in the performance of a previously learned behaviour occur following long-term exposure to 2.45 GHz fields at SARs as low as 2.3 W/kg whole-body exposure. The initial acquisition of operant learned tasks by rats appears to be more sensitive to disruption by RF fields, the thresholds for long-term exposure to pulsed 2.8 GHz fields being between 0.7 and 1.7 W/kg whole-body exposure (43). The pulsed fields used in many of these studies involved brief, rather intense pulses such

as those produced by radar equipment, which may have elicited auditory sensations in the animals, a potentially confounding factor in the interpretation of the studies.

There is a distinct difference in the response between rodents and primates. Changes in operant performance responses in primates occur at higher threshold RF exposures. Such changes were detected after acute exposure of rhesus and squirrel monkeys to 1.3-5.8 GHz fields at whole-body SARs of 4-5 W/kg. Exposure of rhesus monkeys to RF field at which they absorb the maximum amount of energy (resonant frequency, 225 MHz) resulted in reduced task performance at a whole-body SAR of 2.5 W/kg. In rodents, these changes in performance were accompanied by a raised body temperature of about 1 °C (43). Since primates are much closer in size to people than rodents are, these data were used as the basis for standards limiting RF exposure. Under the most adverse environmental conditions, changes in behaviour may be observed with whole-body SARs as low as 1 W/kg. This interpretation is supported by the results of a study investigating working memory. Mickley et al. (28) found that acute exposure to 600 MHz fields at SAR of up to 10 W/kg for 20 minutes produced significant deficits in the memory of rats only when the exposure caused rises in rectal and brain temperatures of at least 1 °C. These changes were correlated with an increase in expression of the c-fos gene in the cortex. The authors concluded that the observed changes in the memory and behaviour were dependent on a rise in body temperature. In experiments done by Lai et al. (23) on spatial learning, the animals were exposed to low levels, pulsed 2.45 GHz fields (average whole-body SAR of 0.6 W/kg) for 20 minutes, immediately before daily training sessions in the maze. Learning was improved for the first two days, although the final performance and the overall accuracy were not affected. Wang and Lai (52) have also reported RF-induced changes in spatial memory as assessed in a circular water maze but an energy per pulse in this study can be calculated to be 2.4 mJ/kg (peak SAR of 2400 W/kg), which would have caused rapid, transient heating (38), using an experimental design very similar to that of (23) exposed mice to 900 MHz RF radiation pulsed at 217 Hz at a whole-body SAR of 0.05 W/kg. The behaviour of the animals was tested each day for 10 days in an eight-arm radial maze, either immediately after exposure for 45 minutes, or after delays of 15 and 30 minutes. There were no significant differences between exposed and control animals; however, the animals tested immediately after exposure took a longer time to complete the task and exhibited more erratic performances than the other animals. It is possible that these changes may have been induced by a mild stress associated with the auditory perception of the field. On the other hand (17), using a head-only exposure system emitting a 900-MHz GSM field reported no differences among exposed, sham and cage-control rats in the two spatial learning tasks. Studies of cognitive performance in human subjects: Preece et al. (30) and Koivisto et al. (21) suggest that exposure to mobile phone signals at power levels within existing exposure guidelines has biological effects that are of sufficient magnitude to influence behaviour. Both groups conjectured that their findings reflected the effect of small temperature increases on synaptic transmission in the region of the cerebral cortex directly under the headset antenna. An easily testable prediction of this account is that the tasks most sensitive to exposure to mobile phone signals should vary according to the position of the headset, and thus the cortical locus of the heating effect.

Taken together, the general result from functional-morphological, electrophysiological and behavioral studies suggests that exposure to HF EMF (corresponding with mobile phone signal) influences some of brain functions but common strong relevance of health risk remains unclear as well as any clear recommendation for the safe use of RF devices (22). The depicted exposure chamber for small laboratory animals will help us to measure an important parameter – SAR. After this first step, we can start other types of experiments which combine different procedures which may confirm or disprove the previous ambiguous results.

SUMMARY

Some interactions of high-frequency electromagnetic field (HF EMF) and neural functions are discussed. Using a unique animal model of neurodegeneration, the influence of HF EMF on its brain was confirmed. A special exposure chamber for small laboratory animals with a Specific Absorption Rate (SAR) measurement was designed.

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Vysokofrekvenční elektromagnetické pole: netermální účinky a měření SAR

SOUHRN

V práci jsou diskutovány některé interakce mezi vysokofrekvenčním elektromagnetickým polem (VF EMP) a funkcemi nervového systému. Užitím unikátního zvířecího modelu neurodegenerace byl potvrzen vliv VF EMP na funkce mozku. Byla navržena a zkonstruována speciální expoziční komora pro malá laboratorní zvířata s možností měření SAR (Specific absorption rate).

REFERENCES

1. Arber S. L., Lin J. C.: Microwave enhancement of membrane conductance: effects of EDTA, caffeine and tetracaine. Physiol. Chem. Phys. Med. NMR 16, 1984: 469–475. – 2. Arber S. L., Lin J. C.: Microwave induced changes in nerve cells: effects of modulation and temperature. Bioelectromagnetics 6, 1985: 257–270. – 3. Barcal J.,

Ježek K., Vožeh F., Žalud V.: Changes of excitability un the cerebellar degeneration model (Lurcher mutant mice). Physiol. Res. 49, 2000: P38. - 4. Barcal J., Vožeh F., Žalud V.: The differential cortical and hippocampal activity in the cerebellar degeneration model. Physiol. Res. 50 (5), 2001: P2. - 5. Barcal J., Vožeh F., Štenglová V. et al.: The effect of nitric oxide on hippocampal potentiation in the cerebellar degeneration model. Homeostasis 41 (1-2), 2001: 67-69. - 6. Barcal J., Cendelín J., Vožeh F. et al.: The spontaneous cortical and hippocampal activity of normal and neurodefective brain influenced by the high-frequency electromagnetic field. Homeostasis 42 (5), 2003: 229–233. – 7. Barcal J., Vožeh F.: Effect of whole-body exposure to high-frequency electromagnetic field on the brain cortical and hippocampal activity in mouse experimental model. NeuroQuantology 5 (3), 2007: 292-302. - 8. Barcal J., Jelínková D., Stopka P., Vožeh F., Vrba J.: Free radicals production and c-fos activity as markers of nonthermal effects of high-frequency electromagnetic exposure in animal model of neurodegeneration. SfN 42nd Annual meeting, New Orleans, USA 2012. - 9. Bawin S. M., Gavalas-Medici R. J., Adey W. R.: Effects of modulated very high frequency fields on specific brain rhythms in cats. Brain Res. 58, 1973: 365–384. – 10. Bawin S. M., Gavalas-Medici R. J., Adey W. R.: Reinforcement of transient brain rhythms by amplitude-modulated VHF fields. Llaurado J. G., Sances A., Battocletti H. eds. Biological and Clinical Effects of Low Frequency Magnetic and Electric Fields (p. 172) Springfield Charles C. Thomas. 1974. - 11. Bliss T. V. P., Collindridge G. L.: A synaptic model of memory: long-term potentiation in the hippocampus. Nature 361, 1993: 31-39. - 12. Borbely A. A., Huber R., Graf T. et al.: Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. Neurosci. Lett. 275, 1999: 207-210. - 13. Caddy K. W. T., Vožeh F.: The effect of 3-acetylpyridine on inferior olivary neuron degeneration in Lurcher mutant and wild type mice. Europ. J. Pharmacol. 330, 1997: 139-142. - 14. Cendelín J., Vožeh F.: Assessment of CNS excitability in natural model of cerebellar degeneration. Homeostasis 39, 1999: 115–116. – 15. Cendelín J., Žalud V., Jelínková D. et al.: The effect of high-frequency electromagnetic field on spatial learning in healthy and neurodefective mice. Phys. Res. 51, 2002: P2. - 16. Cendelín J., Schmidtmayerová B., Štenglová V. et al.: CNS excitability in normal and neurodefective C3H mice exposed to high-frequency electromagnetic field. Proceedings of "Biological Effects of EMFs 3rd Int. Workshop", Kos, Greece, 4-8 October 2004, Vol. II, 2004: 866-871. - 17. Dubreuil D., Jay T., Edeline J. M.: Does head-only exposure to GSM-900 electromagnetic fields affect the performance of rats in spatial learning tasks? Behav. Brain Res. 129, 2002: 203-210. – 18. Eulitz C., Ullsperger P., Frede G. et al.: Mobile phones modulate response patterns of human brain activity. NeuroReport 9, 1998: 3229-3232. - 19. Freude G., Ullsperger P., Ethert S. et al.: Effects of microwaves emitted by cellular phones on human slow brain potentials. Bioelectromagnetics 19, 1998: 384–387. – 20. Huber R., Schuderer J., Graf T. et al.: Radio frequency electromagnetic field exposure in humans: estimation of SAR distribution in the brain, effects on sleep and heart rate. Bioelectromagnetics 24, 2003: 262–276. – 21. Koivisto M., Revonsuo A., Krause C. M. et al.: Effects of 902 MHz electromagnetic field emitted by cellular phones on response times in humans. NeuroReport 11, 2000: 413-415. - 22. Kundi M., Mild K. H., Hardell L. et al.: Mobile telephones and cancer: a review of epidemiological evidence. J. Toxicol. Environ. Health 7, 2004: 351-384. - 23. Lai H., Horita A., Guy A. W.: Microwave irradiation affects radial-arm maze performance in the rat. Bioelectromagnetics 15, 1994: 95-104. - 24. McRee D. I., Wachtel H.: The effects of microwave radiation on the vitality of isolated frog sciatic nerves. Radiat. Res. 82, 1980: 536-541. - 25. McRee D. I., Doder J. A., Gage M. I. et al.: Effects of nonionizing radiation on the central nervous system, behavior and blood: a progress report. Environ. Health Perspect. 30, 1979: 123-126. - 26. Mann K., Roschke J.: Effects of pulsed high-frequency electromagnetic fields on human sleep. Neuropsychobiology 33, 1996: 41–47. – 27. Mausset-Bonnefont A. L., Hirbec H., Bonnefont X. et al.: Acute exposure to GSM 900 MHz electromagnetic fields induces glial reactivity and biochemical modifications in the rat brain. Neurobiol. Dis. 17, 2004: 445-454. - 28. Mickley G. A., Coby B. L., Mason P. A. et al.: Disruption of a putative working memory task and selective expression of brain c fos following microwave-induced hyperthermia. Physiol. Behav. 55, 1994: 1029–1031. – 29. Morris R. G. M.: Development of a water-maze procedure for studying spatial learning in the rat. J. Neurosci. Meth. 11, 1984: 47-64. - 30. Preece A. W., Iwi G., Davies-Smith A. et al.: Effect of a 915-MHz simulated mobile phone signal on cognitive function in man. Int. J. Radiat. Biol. 75, 1999: 447–456. – 31. Reiser H., Dimpfel W., Schober F.: The influence of electromagnetic fields on human brain activity. Eur. J. Med. Res. I, 1995: 27-32. - 32. Repacholi M. H.: Low level exposure to radiofrequency electromagnetic fields: health effects and research needs. Bioelectromagnetics 19, 1998: 1-19. - 33. Roschke J., Mann K.: No shortterm effects of digital mobile radio telephone on the awake human electroencephalogram. Bioelectromagnetics 18, 1997: 172-176. - 34. Salford L. G., Brun A. E., Eberhardt J. L. et al.: Permeability of the blood-brain barier induced

by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, and 200 Hz. Microsc. Res. Techn. 27, 1993: 535-542. - 35. Salford L. G., Brun A. E., Eberhardt J. L. et al.: Nerve cell damage in mammalian brain after exposition to microwaves from GSM mobile phones. Environ. Health Perspect. 111 (7), 2003: 881-883. -36. Seman R. L., Wachtel H.: Slow and rapid responses to CW and pulsed microwave radiation by individual Aplysia pacemakers. J. Microwave Power 13, 1978: 77-86. - 37. Shandala M. G., Dumanskii U. D., Rudnev M. I. et al.: Study of nonionizing microwave radiation effects upon the central nervous system and behavior reactions. Environ. Health Perspect. 30, 1979: 115–121. – 38. Sienkiewicz Z. J., Blackwell R. P., Haylock R. G. E. et al.: Low-level exposure to pulsed 900 MHz microwave radiation does not cause deficits in the performance of a spatial memory task in mice. Bioelectromagnetics 21, 2000: 151–158. – 39. Sobotka P., Barcal J., Žalud V. et al.: The effect of caffeine on the heart activity of mice with inborn cerebellar degeneration. Homeostasis 40, 2000: 128-129. -40. Takashima S., Onaral B., Schwan H. P.: Effects of modulated RF energy on the EEG of mammalian brains. Radiat. Environ. Biophys. 16, 1979: 15–27. – 41. Thuroczy G., Kubinyi G., Bodo M. et al.: Simultaneous response of brain electrical activity (EEG) and cerebral circulation (REG) to microwave exposure in rats. Rev. Environ. Health 10, 1994: 135-148. - 42. Trzaska H.: SAR? Proceedings of "Biological Effects of EMFs 3rd Int. Workshop", Kos, Greece, 4-8 October Vol. 1, 2004: 442-446. - 43. UNEP/WHO/IRPA Electromagnetic Fields (300 Hz-300 GHz). Environmental Health Criteria 137. Geneva, World Health Organization. 1993. - 44. Urban P., Lukas E., Roth Z.: Does acute exposure to the electromagnetic field emitted by a mobile phone influence visual evoked potentials? A pilot study. Centr. Eur. J. Public Health 6, 1998: 288-290. - 45. Voller J., Cendelín J., Vožeh F. et al.: The effect of long-term high-frequency electromagnetic field exposition on neural functions in normal and neurodefective mice of the C57Bl/7 strain. Homeostasis 42 (5), 2003: 225-229. - 46. Vožeh F., Cendelín J., Motáňová A.: The development of different types of learning in cerebellar degeneration model. Homeostasis 39, 1999: 248–250. – 47. Vožeh F., Cendelín J., Štenglová V., Barcal J., Záhlava J.: The development of spatial learning in a model of olivocerebellar degeneration. Homeostasis 41 (1-2), 2001: 64-66. - 48. Vožeh F., Barcal J., Cendelín J., Jelínková D., Křížková A., Štenglová V.: Functional expressions of activity dependent plasticity in a model of cerebellar degeneration. Acta Physiologica Hungarica 89 (1-3), 2002: 186. - 49. Vožeh F., Doněk A., Cendelín J. et al.: Study of high-frequency electromagnetic field effect on some somatic and neuro-behavioral characteristics in healthy and neurodefective mice. Environmentalist 27, 2007: 501-504. - 50. Wachtel H., Seman R., Joines W.: Effects of low-intensity microwaves on isolated neurons. Ann. N Y Acad. Sci. 247, 1975: 46-62. - 51. Wagner P., Roschke J., Mann K. et al.: Human sleep under the influence of pulsed radiofrequency electromagnetic fields: a polysomnographic study using standardized conditions. Bioelectromagnetics 19, 1998: 199-202. - 52. Wang, B., Lai, H.: Acute exposure to pulsed 2450-MHz microwaves affects water-maze performance of rats. Bioelectromagnetics 21, 2000: 52-56. - 53. Zuo J., Philips L., De Jager P. L. et al.: Neurodegeneration in Lurcher mice caused by mutation in d-2 glutamate receptor gene. Nature 388, 1997: 769-773. - 54. Žalud V., Barcal J., Cendelín J. et al.: EEG recording in mice during exposure to high-frequency electromagnetic field. Homeostasis 41 (5), 2001: 203-206.

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