

Effect of Electromagnetic Radiofrequency Radiation on the Rats' Brain, Liver and Kidney Cells Measured by Comet Assay

Ivančica Trošić, Ivan Pavičić, Sanja Milković-Kraus, Marin Mladinić and Davor Željezić

Institute for Medical Research and Occupational Health, Zagreb, Croatia

ABSTRACT

The goal of study was to evaluate DNA damage in rat's renal, liver and brain cells after *in vivo* exposure to radiofrequency/microwave (Rf/Mw) radiation of cellular phone frequencies range. To determine DNA damage, a single cell gel electrophoresis/comet assay was used. Wistar rats (male, 12 week old, approximate body weight 350 g) (N=9) were exposed to the carrier frequency of 915 MHz with Global System Mobile signal modulation (GSM), power density of 2.4 W/m², whole body average specific absorption rate SAR of 0.6 W/kg. The animals were irradiated for one hour/day, seven days/week during two weeks period. The exposure set-up was Gigahertz Transversal Electromagnetic Mode Cell (GTEM-cell). Sham irradiated controls (N=9) were a part of the study. The body temperature was measured before and after exposure. There were no differences in temperature in between control and treated animals. Comet assay parameters such as the tail length and tail intensity were evaluated. In comparison with tail length in controls (13.5±0.7 μm), the tail was slightly elongated in brain cells of irradiated animals (14.0±0.3 μm). The tail length obtained for liver (14.5±0.3 μm) and kidney (13.9±0.5 μm) homogenates notably differs in comparison with matched sham controls (13.6±0.3 μm) and (12.9±0.9 μm). Differences in tail intensity between control and exposed animals were not significant. The results of this study suggest that, under the experimental conditions applied, repeated 915 MHz irradiation could be a cause of DNA breaks in renal and liver cells, but not affect the cell genome at the higher extent compared to the basal damage.

Key words: microwave exposure, rat, brain, liver, kidney, DNA

Introduction

Delicate intracellular processes occurring at macromolecular level like microtubule arrangement which direct DNA assembly thereafter a proliferation of cells seems to be subtle targets for radiofrequency/microwave (Rf/Mw) radiation^{1–7}. The general goal of our investigation was to find out biomarkers of interaction of Rf/Mw and macromolecular structures within the cell, since the growing expands of mobile telephony cause a serious apprehension worldwide. Relationship between Rf/Mw radiation at low intensity exposures and biological markers of its undesirable effects on living matter are very near^{8–10}. Both, *in vivo* and *in vitro* investigation reveals that Rf/Mw radiation acts as biological stressor, since the effects are similar to stress response^{11,12}. Most studies that were published so far did not demonstrate convincingly DNA damage after acute or chronic exposure to Rf fields^{13,14}. Further, Malyapa's study which investigated DNA damage in rat brain cells after *in vivo* exposure to

Rf/Mw radiation no significant effect on direct DNA damage was found¹⁵. In contrary, Sakar et al., found evidence of an alteration in the length of a DNA microsatellite sequence in cells from brain and testis of mice exposed to 2.45 GHz fields yet in 1985¹⁶. Additional, Lai and Singh demonstrated that acute exposure to low-intensity Rf radiation increased DNA strand breaks in brain cells of rat¹⁷. Diem et al. reported that Rf exposure or cell phone signal 1800 MHz, SAR 1.2 or 2 W/kg induced DNA single- and double strand breaks in human fibroblasts and rat granulose cells as measured by comet assay¹⁸. Since radiation of cellular phone frequency is the primary concern for humans not for rodents, Luc Verschaeve reviewed cytogenetic biomonitoring studies of Rf-exposed persons. Majority of these studies show that Rf-exposed individuals have increased frequencies of genetic damage in their lymphocytes or exfoliated buccal cells. Author noticed that most of referred studies have a

number of shortcomings that actually prevents any firm conclusions. Thereafter, he suggested that large well-coordinated multidisciplinary investigations are needed in order to reach solid conclusions¹⁹. To contribute, our study was carried out to evaluate effects at macromolecular level, of carrier frequency 915 MHz with Global System Mobile (GSM) basic signal modulation on DNA in rat's brain, liver and kidney cells after in vivo irradiation.

Materials and Methods

Animals

Wistar rats (male, 12 week old, approximate body weight 350 g) were used for this experiment. All procedures have been performed in accordance with Croatian Animal Welfare Act (N.N. #19, 1999) and in compliance with the Guide for the Care and Use of Laboratory Animals DHHS Publ. (NIH #86-23, 1986). Before the exposure started, the animals had passed through a week accommodation period. Both sham-exposed control (N=9) and exposed animal group (N=9) were kept in steady-state microenvironment conditions ($22^{\circ}\text{C}\pm 1^{\circ}\text{C}$), and receiving standard laboratory food and water *ad libitum*, with alternating 12-hours light and dark cycles, except in one hour irradiation time daily when the Rf/Mw generator was switch on for experimental animals. Before and after exposure a body temperature was measured using a ThermoScan thermometer (Braun GmbH, Germany) to eliminate thermal effects on the observed variables. No significant changes in body temperature were observed in treated animals with respect to controls. At the end of the experiment, immediately after last exposure treatment, rats were sacrificed under the Narketan/Xylapan anesthesia (Narketan[®], 80 mg kg⁻¹ b.m. + Xylapan[®], 12 mg kg⁻¹ b.m., *i.p.* produced by Vétouquinol, Bern, Switzerland).

Ethical statement

Animal studies were carried out according to the guidelines force in Republic Croatia (Law on the Welfare of Animals, N.N. #19, 1999) and in compliance with the Guide for the Care and Use of Laboratory Animals, DHHS Publ. (NIH) #86-23 (1986).

Exposure equipment

Electromagnetic field was generated within the certified Gigahertz Transversal Electromagnetic Mode Cell (GTEM-cell), (Mod. 5402, ETS Lindgren, USA). A signal generator was used to produce electromagnetic field with frequency of 915 MHz (Antrisu 27211A, Japan). A signal amplifier (RF 3146 Power Amp Module RF Micro Devices, Greensboro, USA) and a signal modulator (RF 2722 Polaris chip, RF Micro Devices, Greensboro, USA) were a part of exposure set-up. The carrier frequency of 915 MHz with Global System Mobile (GSM) basic signal modulation was used in experiment. The temperature in the GTEM-cell was measured and preserved at 37°C throughout the experimental procedure.

Experimental design

Radiation exposure of animals lasted for one hour a day, seven days a week, each day at the same hour. The experiment was completed in 14 days. During irradiation procedure each animal was placed inside the individual Plexiglas cage (25 cm×7.5 cm×7.5 cm) which was put into the GTEM-cell. Free movements of animals throughout exposure were prevented by cage length, height and width. During the irradiation the incident electromagnetic field strength of 30 V/m was whole-body uniform. The power density of the field was 2.4 W/m² corresponding to the whole-body specific absorption rate (SAR) of 0.6 W/kg²⁰. Any thermal effect has been avoided by monitoring of whole-body temperature of animals before and after treatment as well as preservation of temperature inside the GTEM-cell at 37°C. The experimental design has been described in details elsewhere⁵.

Genotoxicity testing

Immediately after the animals were sacrificed, samples of liver, renal cortex, and frontal cortex were taken and immersed in chilled homogenization buffer (75mM NaCl and 24mM Na₂EDTA, pH 7.5) to obtain a 10% tissue solution. The tissues have been chosen on the basis of toxicological principles. Kidney and liver represents the main target tissues for evaluation of the undesirable effects of in vivo exposure to the physical or chemical toxicants. The brain is considered to be exceptional target tissue for non-ionizing radiation because of its electromagnetic activity^{21,17}. Samples were homogenized using a potter-type homogenizer. Tissue samples remained on ice during and after homogenization²². The comet assay was carried out under alkaline conditions, as described by Singh et al., Gamulin et al., and Željezić et al.^{23,24,25}. Two replicate slides per sample per method were prepared. Agarose gels were prepared on fully frosted slides coated with 1% and 0.6% normal melting point agarose. Cell suspension (5 μL) was mixed with 0.5% low melting point agarose, placed on the slides and covered with a layer of 0.5% low melting point agarose. The slides were immersed for 1 hr in freshly prepared ice-cold lyses solution (2.5 M NaCl, 100 mM Na₂EDTA, 10 mM Tris-HCl, 1% Na-sarcosinate, pH 10) with 1% Triton X-100 and 10% dimethyl sulfoxide (Kemika, Zagreb, Croatia). Alkaline denaturation and electrophoresis were carried out at 4°C under dim light in freshly prepared electrophoresis buffer (300 mM NaOH, 1 mM Na₂EDTA, pH 13.0). After 20 min. of denaturation, the slides were randomly placed side by side in the horizontal gel-electrophoresis tank, facing the anode. Electrophoresis at 1 V/cm lasted another 20 min. The slides were gently washed with a neutralization buffer (0.4 M Tris-HCl, pH 7.5) three times at 5 min. intervals and stained with ethidium bromide (20 μg/mL). Each slide was examined using a 250×magnification fluorescence microscope (Zeiss, Oberkochen, Germany) equipped with an excitation filter of 515–560 nm and a barrier filter of 590 nm. A total of 100 comets per sample were scored (50 from each of two replicate slides). Comets were randomly captured at

a constant depth of the gel, avoiding the edges of the gel, occasional dead cells and superimposed comets. As a measure of DNA damage, the following comet parameters were evaluated: tail length (μm) and tail intensity (%DNA in the tail). A computer-based image analysis system (Comet Assay II, Perceptive Instruments Ltd., Suffolk, UK) was used to perform the analysis. To avoid the variability, one well-trained scorer scored all comets.

Statistical analysis

Each experimental set contained duplicated slides. Prior to further analyses, all the parameters were log-transformed. Differences in the tail length and tail intensity for the standard comet assay for exposed and control samples were statistically evaluated using the non-parametric Mann-Whitney test. The level of statistical significance was set at $p < 0.05$.

Results

Table 1 represents descriptive statistics of tail length and tail intensity in kidney, liver and brain homogenates in animals exposed to the Rf radiation and matched controls.

In comparison with controls the performed comet assay established a significant increase in the tail length in

kidney and liver homogenates, but not in the brain cells. No significant differences were found in tail intensity for all examined organs. Results show that the mean value for kidney DNA migration was $13.9 \mu\text{m}$ for exposed and $12.9 \mu\text{m}$ for controls ($p < 0.05$). Mean value of liver DNA migration registered by tail length in irradiated animals was $14.5 \mu\text{m}$ and $13.6 \mu\text{m}$ in matched controls ($p < 0.01$). The standard comet assay performed at liver cells of irradiated animals revealed the greatest mean tail length when compared with the findings obtained for other tested organs. The mean tail length of brain DNA fragments was found to be $14.0 \mu\text{m}$, which is in comparison with the control brain samples slightly but not significantly elongated (significance level, $p = 0.05$) (Figure 1). Otherwise, differences in tail intensity (% DNA) between control and exposed animals were not significant (Figure 2). Comet assay microphotographs showing rat hepatocytes under Leitz Orthoplan epifluorescence microscope using 250 x magnifications are shown at Figure 3. Unexposed control cell is left assigned as A and Rf-exposed one is right marked as B. It is important to declare that there was no body temperature difference between irradiated and control animals before or immediately after exposure. The temperature difference between irradiated and control animal group was less than 1°C before or after exposure.

TABLE 1
DESCRIPTIVE STATISTICS OF TAIL LENGTH AND TAIL INTENSITY IN KIDNEY, LIVER AND BRAIN CELLS OF ANIMALS EXPOSED TO RADIOFREQUENCY (RF) RADIATION FOR ONE HOUR A DAY, SEVEN DAYS A WEEK DURING TWO WEEKS PERIOD (N=9) AND MATCHED CONTROLS (N=9)

Animal	Tail length / $\mu\text{m} \pm \text{S.D.}$			Tail intensity / % DNA in the tail, $\pm \text{S.D.}$		
	Kidney	Liver	Brain	Kidney	Liver	Brain
1	12.6 \pm 1.2	13.9 \pm 1.7	13.8 \pm 1.4	1.1 \pm 1.4	1.1 \pm 1.8	1.0 \pm 1.1
2	14.7 \pm 1.5	13.0 \pm 2.0	13.7 \pm 1.8	1.8 \pm 2.1	0.8 \pm 1.5	0.9 \pm 0.9
3	11.6 \pm 1.0	13.6 \pm 1.7	14.8 \pm 3.6	0.9 \pm 1.1	0.9 \pm 1.3	1.2 \pm 2.6
4	13.7 \pm 1.4	13.9 \pm 1.7	13.3 \pm 1.7	1.2 \pm 1.5	0.8 \pm 0.9	0.9 \pm 1.3
5	12.8 \pm 1.3	13.9 \pm 1.8	13.1 \pm 1.6	1.2 \pm 1.7	1.1 \pm 2.0	0.6 \pm 0.8
6	12.5 \pm 1.2	13.6 \pm 1.9	13.5 \pm 4.1	1.0 \pm 1.7	0.8 \pm 1.4	1.3 \pm 2.1
7	12.9 \pm 1.8	13.6 \pm 2.2	13.4 \pm 2.3	1.3 \pm 2.2	0.9 \pm 2.0	1.2 \pm 2.7
8	12.3 \pm 0.8	13.0 \pm 1.7	12.1 \pm 1.6	0.9 \pm 0.8	0.9 \pm 1.3	1.1 \pm 1.9
9	13.1 \pm 1.1	13.7 \pm 1.5	13.7 \pm 2.2	1.4 \pm 1.0	0.9 \pm 1.3	0.6 \pm 1.8
Mean	12.9 \pm 0.9	13.6 \pm 0.3	13.5 \pm 0.7	1.2 \pm 0.3	0.9 \pm 0.1	1.0 \pm 0.2
10	13.2 \pm 1.3	14.3 \pm 2.0	13.9 \pm 1.8	1.0 \pm 2.0	2.2 \pm 3.7	1.8 \pm 3.5
11	13.6 \pm 2.5	14.0 \pm 1.5	13.8 \pm 1.6	1.5 \pm 3.1	1.2 \pm 3.5	0.8 \pm 1.1
12	14.5 \pm 1.7	14.8 \pm 1.7	14.0 \pm 1.4	1.2 \pm 2.1	0.6 \pm 1.3	1.1 \pm 2.1
13	13.5 \pm 1.1	14.5 \pm 1.1	14.2 \pm 1.7	1.2 \pm 2.0	1.2 \pm 2.3	1.7 \pm 3.1
14	14.0 \pm 1.5	14.2 \pm 1.7	13.8 \pm 1.3	1.3 \pm 2.7	0.7 \pm 1.5	0.9 \pm 1.7
15	13.8 \pm 1.5	14.9 \pm 2.0	14.3 \pm 1.4	1.4 \pm 1.8	1.4 \pm 2.6	1.1 \pm 2.6
16	14.0 \pm 1.5	14.6 \pm 1.7	13.9 \pm 1.1	1.3 \pm 2.7	0.5 \pm 1.2	0.7 \pm 1.5
17	14.2 \pm 1.6	14.9 \pm 1.8	14.6 \pm 1.5	1.5 \pm 4.0	0.5 \pm 1.0	0.6 \pm 1.2
18	14.7 \pm 3.1	14.4 \pm 1.9	13.9 \pm 1.8	1.2 \pm 2.5	0.5 \pm 1.1	0.8 \pm 1.8
Mean	13.9 \pm 0.5*	14.5 \pm 0.3*	14.0 \pm 0.3	1.3 \pm 0.2	1.0 \pm 0.6	1.0 \pm 0.4

* $p < 0.05$, compared to control (Mann-Whitney test)

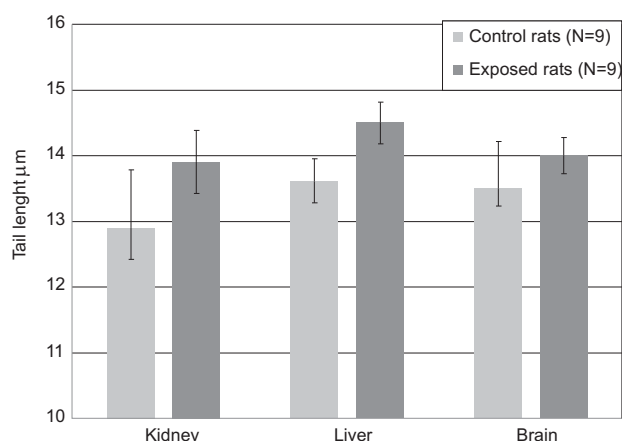


Fig. 1. Tail length in kidney, liver and brain cells of animals exposed to radiofrequency (Rf) radiation for one hour a day, seven days a week during two weeks period (N=9) and matched controls (N=9); comet assay.

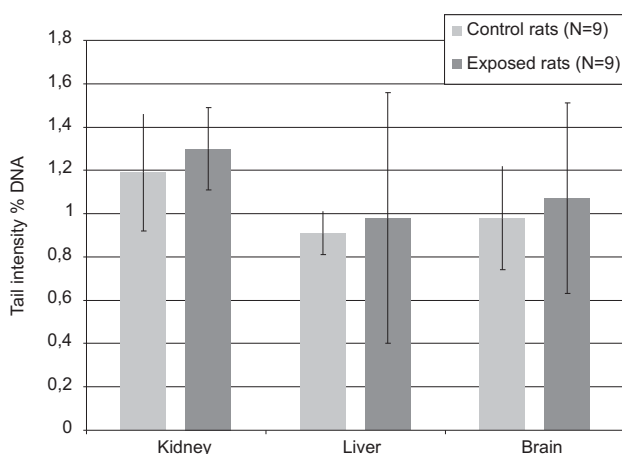


Fig. 2. Tail intensity in kidney, liver and brain cells of animals exposed to radiofrequency (Rf) radiation for one hour a day, seven days a week during two weeks period (N=9) and matched controls (N=9); comet assay.

Discussion

Besides the numerous reports of well conducted investigations, it would not be wisely to deny that Rf/Mw at low intensity might affect the macromolecular structures such as DNA. Until now, data available suggest a complex reaction of the intracellular protein system to Rf radiation. The response is not likely to be linear with respect to the intensity of the radiation. Other parameters of Rf exposure, such as frequency, duration, waveform, frequency- and amplitude-modulation are important determinants of biological responses and affect the shape of the dose (intensity)-response relationship²⁶. In 2009 Belyaev et al. pointed to the chromosomal DNA as a target for resonance interaction between living cells and microwave²⁷. It is important to appreciate when doing the comet assay on different tissues that normal DNA damage can be highly variable. Some of the factors that can

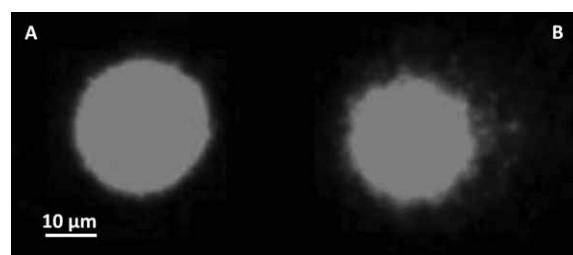


Fig. 3. Comet assay microphotographs showing rat hepatocytes under Leitz Orthoplan epifluorescence microscope using 250x magnifications (A – untreated control; B – treated cell).

influence DNA strand damage in a particular tissue include cell type heterogeneity, cell cycle, cell turnover frequency and culture or growth conditions. Different cell types may have very different background levels of DNA single-strand breaks due to variation in excision repair activity, metabolic activity, anti-oxidant concentrations or other factors²⁸. DNA damages in cells could have an important implication on health because they are cumulative. In general, DNA is capable of repairing itself efficiently. Stimuli which could influence equilibrium of cells maturation or proliferation activate a known feedback mechanism of homeostatic control mechanism²⁹. Moreover, through a homeostatic mechanism, cells maintain a delicate balance between spontaneous and induced DNA damage. DNA damage accumulates if such a balance is altered, which may in turn affect cell functions. When too much DNA damage is accumulated over time, the cell will die or may be the cause of slow onset diseases. Beside in 2005, the Diem's et al. report, Paulraj and Behari found that chronic exposure to this radiation causes significant increase in DNA strand breaks in brain cells of rats^{18,30}. The present study is in agreement with aforementioned ones showing a difference in DNA strand breaks in the brain cells of the exposed group (Table 1). The manuscript also demonstrates findings of single cell gel/comet assay application on rats' renal and liver homogenates after mobile phone frequency exposure *in vivo*. The significant DNA damage was found to be significant in kidney and liver cells of irradiated animals but only when tail length has been used as the parameter of DNA damage (see Figure 1). Since no significant effect on the content of DNA that moved from the head into the tail of the comet (tail intensity values) has been observed it may be suggested that selected radiation did not affect the cell genome at the higher extent compared to the basal damage. Thus, detected level of DNA damage does not justify the use of chromosome aberration or any other standard cytogenetic tests. At the reported extent, primary DNA damage is not likely to be transduced into morphological changes of chromosomes at the rate higher than basal. Detected level of primary DNA damage may be efficiently repaired within several hours following irradiation without any permanent impact on chromosome morphology. Similar observations have been reported by applying the comet assay in evaluation of DNA damage in athletes³¹. Thus, under the con-

ditions applied the observed extent of DNA damage does not pose a risk for inducing adverse health effects in irradiated animals. One has to notice that serious DNA strand breaks were common for highly metabolic tissues, for example kidney, liver or bone marrow³². Recently Ruediger exploited one hundred publications which have studied genotoxicity of radiofrequency electromagnetic fields *in vivo* and *in vitro*. Overview resulted in conclusion that cells are unusually sensitive to electromagnetic fields, because weak fields may accelerate electron transfer and thereby destabilize the H-bond of cellular macromolecules. Although the energy of weak fields such as Rf/Mw is not sufficient directly to break a chemical bond in DNA, he revealed that genotoxic effects are mediated by indirect mechanisms as micro thermal processes, gen-

eration of oxygen radicals or a disturbance of DNA-repair processes³³. It could be concluded that repeated whole-body exposure to carrier frequency of 915 MHz with Global System Mobile (GSM) basic signal modulation at power density of 2.4 W/m² and SAR 0.6 W/kg could be a cause of DNA strand breaks in renal and liver cells of rats, but not affect the cell genome at the higher extent compared to the basal damage.

Acknowledgements

This investigation was supported by the Croatian Ministry of Science, Education and Sports (Grants no. 0022-0222411-2406 and 022-0222148-2137).

REFERENCES

1. PAVICIC I, TROSIC I, Acta Biol Hung, 59(1) (2008) 67. — 2. PAVICIC I, TROSIC I, Biologia, 61(3) (2006) 321. — 3. TROSIC I, BUSLJETA I, KASUBA V, ROZGAJ R, Mutat Res, 521 (2002) 73. — 4. TROSIC I, BUSLJETA I, MODLIC B, Mutagenesis 19 (2004) 361. — 5. ZHAO TY, ZOU SP, KNAPP PE, Neurosci Lett, 412 (2007) 34. — 6. DEL VECCHIO G, GIULIANI A, FERNANDEZ M, MESIRCA P, BERSANI F, PINTO R, ARDOINO L, LOVISOLO GA, GIARDINO L, CALZA L, Neurosci Lett, 455 (2009) 173. — 7. PACINI S, RUGGIERO M, SARDI I, ATERINI S, GULISANO F, GULISANO M, Oncol Res, 13 (2002) 19. — 8. SCHWARZ C, KRATOCHVIL E, PILGER A, KUSTER N, ALDKOFER F, RÜDIGER W, Arch Occup Environ Health, 81 (2008) 755. — 9. TROSIC I, BUSLJETA I, Phys Scr, T118 (2005) 168. — 10. MAES A, VAN GORP U, VERSCHAEVE L, Mutagenesis, 21(2) (2006) 139. — 11. DE POMERAI D, DANIELLS C, DAVID H, ALLAN J, DUCE I, MUTWAKIL M, THOMAS D, SEWELL P, TATTERSALL J, JONES D, CANDIDO P, Nature, 440 (2006) 417. — 12. BLANK M, GOODMAN R, Pathophysiology, 16 (2009) 71. — 13. SAKUMA N, KOMATSUBARA Y, TAKEDA H, HIROSE H, SEKIJIMA M, NOJIMA T, MIYAKOSHI J, Bioelectromagnetics, 27 (2006) 51. — 14. VERSCHAEVE L, HEIKKINEN P, VERHEYEN G, VAN GORP U, BOONEN F, VANDER PLAETSE F, MAES A, KUMLIN T, MÄKI-PAKKANEN J, PURANEN L, JUUTILAINEN J, Radiat Res, 165(5) (2006) 598. — 15. MALYAPA RS, AHERN EW, BI C, STRAUBE WL, LAREGINA M, PICKARD WF, ROTI ROTI JL, Radiat Res, 149 (6) (1998) 637. — 16. SAKAR S, ALI S, BEHARI J, Mutat Res, 320 (1994) 141. — 17. LAI H, SINGH NP, Int J Radiat Biol, 69 (1996) 513. — 18. DIEM E, SCHWARZ C, ALDKOFER F, JAHN O, RUDIGER H, Mutat Res, 583 (2005) 178. — 19. VERSCHAEVE L, Mutat Res, 681 (2008) 259. — 20. DURNEY CH, MASSOUDI H, ISKANDER MF, Radiofrequency Radiation Handbook, (USAF School of Aerospace Medicine, Texas, 1986). — 21. HAYES AW, Principles and Methods of Toxicology, (Raven Press, New York, 1989). — 22. SASAKI YF, NISHIDATE E, IZUMIYAMA F, MATSUSAKA N, TSUDA S, Mutat Res, 391 (1997) 215. — 23. SINGH NP, MCCOY MT, TICE RR, SCHNEIDER LL, Exp Cell Res, 175 (1988) 184. — 24. GAMULIN M, GARAJ-VRHOVAC V, KOPJAR N, Coll Antropol, 31(3) (2007) 837. — 25. ZELJEZIC D, GARAJ-VRHOVAC V, Neoplasma, 51(3) (2004) 198. — 26. WORLD HEALTH ORGANIZATION, Environmental Health Criteria (EHC): 137 Electromagnetic fields (300 Hz – 300GHz), (World Health Organization, Genève, 1993). — 27. BELYAEV IY, MARKOVA E, HILLERT L, MALMGREN LO, PERSSON BR, Bioelectromagnetics, 30 (2009) 129. — 28. TICE RR, Applications of the single cell gel assay to environmental biomonitoring for genotoxic pollutants. In: BUTTERWORTH FM, CORKUM LD, GUZMAN-RINCON J (Eds) Biomonitoring and Biomarkers as Indicators of Environmental Change (Plenum Press, New York, 1995). — 29. TROSIC I, BUSLJETA I, Exp Toxicol Pathol, 57 (2006) 247. — 30. PAULRAJ R, BEHARI J, Mutat Res, 596 (2006) 76. — 31. WAGNER KH, REICHHOLD S, HÖLZL C, KNASMÜLLER S, NICS L, MEISEL M, NEUBAUER O, Toxicology, 278(2) (2010) 211. — 32. BUSLJETA I, TROSIC I, MILKOVIC-KRAUS S, Int J Hyg Environ Health, 207 (2004) 1. — 33. RÜDIGER HW, Pathophysiology, 16(2–3) (2009) 89.

I. Trošić

Institute for Medical research and Occupational Health, Ksaverska cesta 2, 10 000 Zagreb, Croatia
e-mail: itrosic@imi.hr

UTJECAJ ELEKTROMAGNETSKOG RADIOFREKVENCIJSKOG ZRAČENJA NA STANICE MOZGA, JETRE I BUBREGA U ŠTAKORA MJEREN KOMET TESTOM

S A Ž E T A K

Cilj studije bio je procijenjena oštećenja molekule DNK u bubregu, jetri i moždanim stanicama štakora nakon izlaganja radiofrekvencijsko/mikrovalnom (Rf/Mw) zračenju frekvencija mobilnih telefona. Za procjenu oštećenja DNA korišten je komet test. Wistar štakori (mužjaci, stari 12 tjedna, približne mase 350 g) (N=9) bili su izloženi zračenju moduliranog signala sustava mobilni telefonije (GSM), frekvencije od 915 MHz i gustoće snage od 2.4 W/m², i prosječnoj brzini apsorpcije energije (SAR) od 0.6 W/kg. Životinje su zračene po jedan sat dnevno tijekom dva tjedna. Sustav za ekspoziciju sastojao se od gigahercne transverzalne elektromagnetske komore (GTEM-stanica). Sham-kontrolna skupina životinja (N=9) bila je uključena u studiju. Temperatura tijela štakora mjerena je prije i nakon izlaganja. Nije bilo razlike

u temperaturi između kontrolnih i ozračenih životinja. Parametri komet testa su procijenjeni dužinom i intenzitetom repa. U usporedbi s kontrolom ($13,5 \pm 0,7 \mu\text{m}$), dužina repa u stanicama mozga ozračenih životinja je lagano izdužena ($14,0 \pm 0,3 \mu\text{m}$). Dužina repa u jetrenim stanicama ($14,5 \pm 0,3 \mu\text{m}$) i stanicama bubrega ($13,9 \pm 0,5 \mu\text{m}$) ozračenih životinja značajno se razlikovala od odgovarajućih kontrolnih uzoraka; ($13,6 \pm 0,3 \mu\text{m}$) i ($12,9 \pm 0,9 \mu\text{m}$). Intenzitet repa između kontrolnih i izloženih životinja bio je statistički neznačajan. Rezultati pokazuju da u eksperimentalnim uvjetima opetovano 915 MHz zračenje može uzrokovati lomove DNK u stanicama jetre i bubrega. Primijenjeno zračenje nema većeg utjecaja na genom stanice u odnosu na bazalna oštećenja.