EFFECT OF ELECTROMAGNETIC RADIATION FROM MOBILE PHONE ON THE LEVELS OF CORTICAL AMINO ACID NEUROTRANSMITTERS IN ADULT AND YOUNG RATS

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Abstract. The present study aims to investigate the effect of electromagnetic radiation (EMR) generated by mobile phones on the levels of amino acid neurotransmitters; glutamate, aspartate, GABA, glycine and taurine in the cortex of adult and young rats. Several studies showed that EMR could influence normal brain physiology, probably by changing cortical excitability. In the present study, adult and young rats were exposed to EMR for one hour/day. Amino acids were measured after 1 hour, 1, 2 and 4 months of daily EMR exposure and after 1 month of stopping exposure that extended daily for 4 months. The present data showed that in adult rats EMR induced significant changes in the cortical levels of some studied amino acids throughout the exposure periods. However, in young rats EMR induced significant changes after 4 months of daily exposure and after stopping exposure. It could be suggested that the changes in amino acid neurotransmitters may underlie the EMR-induced changes in cortical excitability.

Key words: Electromagnetic radiation, amino acid neurotransmitters, cortex, rats.

INTRODUCTION

The increasing number of telecommunication devices available and length of time spent using mobile telephones has aroused interest of possible interactions between human and radiofrequency radiations [14]. The study of Schörnborn *et al*. showed that the adult human head absorbs 80% of the radiation emitted by a cellular telephone [32].

Recent studies have reported contradictory results regarding potential effects of electromagnetic fields (EMF) of digital mobile phone on the nervous system. Several studies have indicated that EMF emitted from mobile phone could affect brain activity; including sleep [11], attention [8], learning and memory [20, 21] and cognitive performance [29]. However, several authors did not find any effect for

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EMF on memory and cognitive function [10, 22] and histopathological parameters [9] when replicating their previous findings.

It has been suggested that the brain harm from mobile phone might derive from the proven ability of electromagnetic fields such as those from mobile phone to modify electrophysiological activity in human brain [13] and to alter neurotransmission [1, 16, 23, 25]. Moreover, evidence of oxidative damage was found in the brain tissues when rats were exposed to (RF/MW) radiation [15]. It has been concluded that EMF like the ones emitted from mobile phones influence normal brain physiology, probably by means of changing cortical excitability [7].

The aim of the present study is to investigate the effect of EMR at a frequency of 900 MHz, power density 0.02 mW/cm^2 and an average specific absorption rate (SAR) 1.165 W/kg on amino acid neurotransmitters in the cortex of adult and young male albino rats.

MATERIALS AND METHODS

EXPERIMENTAL ANIMALS

The experimental animal used in this study is the male albino rat *Rattus norvegicus*. Both young (one month old) and adult (four months old) animals were used and provided with food and water *ad libitum*. All experiments were carried out in accordance with research protocols established by the animal care committee of the National Research Center, Egypt.

ELECTROMAGNETIC EXPOSURE SETUP

The electromagnetic field was generated by a radiofrequency (RF) source which simulated the actual sources encountered in mobile communications such as stations, handsets, etc. This source consisted of: (1) an RF signal generator (Aeroflex Company, Model : 2025,UK) with an RF output level up to 13 dBm and a frequency range of 0 – 2.5 GHz, (2) two monopole antennas set in parallel with the help of a T-junction, each being designed so that the reflection coefficient at its input is not more than –12 dBm and fed by a coaxial line through a BNC connector, (3) a power amplifier (Stealth Microwave, Model: SM 0520-36, SSB Technologies, Inc.) to supply a power intensity of 0.02 mW/cm^2 at the rat location, which is equal to the average power radiated from an actual mobile communication source and (4) a field survey meter (Narda, EMR200, frequency from 0 to 4 GHz, Germany) to measure the power intensity of electromagnetic fields at different distances from the generator.

ELECTROMAGNETIC FIELD EXPOSURE

At the beginning of the experiment, the RF signal generator output was connected to the power amplifier whose output was in turn connected to the T-junction, to feed concurrently the two monopole antennas. Each monopole antenna was placed at the center of a circular equally radiated container, made of an opaque acrylic substance called presidex, pointing downwards. The length of the monopole was set so that the antenna is resonant at the operating frequency. The antenna had an omni directional pattern in the azimuth plane through which the rats were to be uniformly distributed. Before placing the rats in the container, an intensity field meter was used to measure the electromagnetic field distribution at the different locations of the circular container especially near the expected locations of the rat heads. The antenna was removed and the rats (16) were placed in the sectors (16) of the container with their heads towards the antenna. The container was designed in such a way that the animals were not restrained. The animals were left for a period to adapt and direct themselves spontaneously towards the antenna as the air openings are concentrated in the cover of the container around the antenna. The antenna was then placed again at the center of the container and the rats were exposed to the RF fields at a frequency of 900 MHz and a power density of 0.02 mW/cm^2 for 1 hour. The average SAR was 1.165 W/kg as calculated by the Finite-Difference-Time-Domain (FDTD). A geometric/electric model was constructed for the animal's head from the stereotaxic atlas of Paxinos and Watson [30]. An ellipsoid model with the internal anatomic layers was used. The electric properties were assigned to each layer. The animal head model was then subjected to EMR in the form of a plane wave with the power density as that measured by the field survey meter through the experimental exposure process. The FDTD algorithm was then applied to calculate the electric field distribution everywhere inside the head model. The SAR was then calculated at the desired points as $(E)^2/2\sigma$ where E is the electric field value at the point and σ is the conductivity of the tissue at this point.

EXPERIMENTAL DESIGN

The groups of adult and young rats were exposed to EMR (frequency 900 MHz, power density 0.02 mW/cm^2 and average SAR 1.165 W/kg) simultaneously for 1 hour daily. A group of both adult and young animals was placed at the same time in a similar container for 1 hour away from the RF source and served as control animals. A group from each treated and control animals was sacrificed after 1 hour, 1 month, 2 months and 4 months of daily exposure. One subgroup from the treated animals (adult and young) was left for 1 month without exposure (after 4 months of daily exposure) to study the withdrawal effect of the radiation and were then sacrificed with a group of the control animals.

The animals were killed by sudden decapitation and the cortex was dissected out, weighed and kept frozen until analyzed. The quantitative determination of the amino acids (glutamate, aspartate, glutamine, GABA, glycine and taurine) was carried out by using the high performance liquid chromatography (HPLC) method employed by Márquez *et al*. [24].

STATISTICAL ANALYSIS

All data are expressed as mean±S.E.M. (*n*), where *n* refers to the number of animals. Statistical comparisons between the means of animals exposed to EMR and those of control animals were carried out by the independent t-test using SPSS (Statistical Package for Social Sciences) version 14 for each of the adult and young rats in each time interval separately. Significance was determined at $p < 0.05$.

RESULTS

Data showing the effects of EMR on the concentrations of cortical amino acid neurotransmitters of adult rats are presented in Table 1.

The exposure of adult rats to EMR for 1 h induced a significant increase in glutamate, aspartate, GABA and glycine, but a significant decrease in glutamine. After one month of daily EMR exposure, a significant decrease was obtained in glutamate and aspartate. However, a significant increase in glutamate and aspartate accompanied by a significant increase in glycine and taurine were recorded after two months of daily EMR exposure. After four months of daily exposure, a significant increase in aspartate, GABA, glycine and taurine was obtained, however, glutamine decreased significantly. After stopping EMR exposure for one month, non significant changes in the concentrations of the studied cortical amino acids occurred.

The data on the effects of EMR on the concentrations of amino acid neurotransmitters in the cortex of young rats are shown in Table 2.

The acute effect of EMR recorded after a single exposure for 1h showed a significant decrease in taurine. This was followed by non significant changes in the concentrations of the amino acid neurotransmitters after one and two months of daily EMR exposure, except for a significant increase in glutamine after one month and taurine after two months. The exposure of young rats to EMR daily for four months induced a significant decrease in glutamine; while a significant increase in GABA and taurine was recorded. After one month of stopping exposure to EMR, a significant decrease in glutamine accompanied by a significant increase in GABA and glycine was observed.

Table 1

Effect of electromagnetic radiation on the concentrations of amino acid neurotransmitters (μmol/g fresh tissue) in the cortex of adult rats

	Time of Exposure	Control	Treated	%*d*
Glutamate	1 hour	5.57 ± 0.17 (7)	6.59 ± 0.22 (7)	18.31*
	1 month	4.43 ± 0.15 (6)	3.84 ± 0.12 (6)	–13.32*
	2 months	5.67 ± 0.24 (6)	6.52 ± 0.19 (6)	14.99*
	4 months	6.71 ± 0.11 (6)	6.86 ± 0.04 (7)	2.24
	4 months + 1 month recovery	6.08 ± 0.07 (6)	6.07 ± 0.11 (7)	–0.16
Aspartate	1 hour	1.17 ± 0.05 (6)	1.56 ± 0.07 (7)	33.33*
	1 month	3.12 ± 0.25 (6)	1.99 ± 0.15 (5)	–36.22*
	2 months	1.62 ± 0.06 (6)	2.11 ± 0.05 (6)	30.25*
	4 months	1.34 ± 0.03 (5)	1.48 ± 0.04 (6)	10.45*
	4 months + 1 month recovery	1.33 ± 0.08 (6)	1.20 ± 0.05 (7)	–9.77
Glutamine	1 hour	3.32 ± 0.09 (7)	2.97 ± 0.12 (6)	–10.54*
	1 month	2.72 ± 0.10 (7)	2.61 ± 0.10 (7)	–4.04
	2 months	3.82 ± 0.15 (6)	3.65 ± 0.09 (7)	–4.45
	4 months	4.15 ± 0.17 (6)	3.65 ± 0.09 (6)	–12.05*
	4 months + 1 month recovery	3.65 ± 0.12 (6)	3.46 ± 0.11 (8)	–5.21
GABA	1 hour	1.85 ± 0.03 (7)	2.18 ± 0.05 (8)	17.84*
	1 month	1.53 ± 0.08 (7)	1.47 ± 0.06 (7)	–3.92
	2 months	2.23 ± 0.07 (7)	2.39 ± 0.05 (7)	7.17
	4 months	1.81 ± 0.04 (6)	2.01 ± 0.07 (6)	11.05*
	4 months + 1 month recovery	2.21 ± 0.04 (6)	2.19 ± 0.03 (8)	–0.90
Glycine	1 hour	1.17 ± 0.05 (6)	1.43 ± 0.02 (7)	21.37*
	1 month	0.91 ± 0.08 (7)	0.87 ± 0.09 (7)	–4.39
	2 months	1.23 ± 0.05 (6)	1.54 ± 0.03 (7)	25.20*
	4 months	1.34 ± 0.05 (6)	1.53 ± 0.03 (6)	14.18*
	4 months + 1 month recovery	1.15 ± 0.02 (6)	1.16 ± 0.03 (8)	0.87
Taurine	1 hour	5.16 ± 0.13 (7)	5.21 ± 0.19 (8)	0.97
	1 month	2.47 ± 0.08 (7)	2.30 ± 0.05 (7)	–6.88
	2 months	5.03 ± 0.14 (6)	5.79 ± 0.15 (6)	15.11*
	4 months	7.27 ± 0.16 (5)	7.96 ± 0.11 (6)	9.49*
	4 months + 1 month recovery	4.85 ± 0.17 (6)	4.80 ± 0.15 (8)	–1.03

Mean ± SEM is represented with number of animals between brackets; *$p < 0.05$ significant; %*d*: percentage difference from control.

Table 2

Effect of electromagnetic radiation on the concentrations of amino acid neurotransmitters (μmol/g fresh tissue) in the cortex of young rats

	Time of exposure	Control	Treated	%*d*
Glutamate	1 hour	6.63 ± 0.07 (6)	6.06 ± 0.26 (5)	–8.59
	1 month	6.01 ± 0.24 (6)	6.08 ± 0.08 (7)	1.16
	2 months	4.11 ± 0.08 (7)	4.12 ± 0.15 (7)	0.24
	4 months	6.69 ± 0.19 (6)	6.38 ± 0.10 (7)	–4.63
	4 months + 1 month recovery	6.27 ± 0.16 (7)	6.65 ± 0.26 (6)	6.06
Aspartate	1 hour	1.53 ± 0.07 (6)	1.56 ± 0.09 (6)	1.96
	1 month	1.43 ±0.08 (7)	1.40 ± 0.08 (7)	–2.10
	2 months	0.86 ± 0.02 (7)	0.84 ± 0.03 (7)	–2.33
	4 months	1.71 ± 0.10 (5)	1.47 ± 0.03 (6)	–14.04
	4 months + 1 month recovery	1.31 ± 0.06 (7)	1.47 ± 0.12 (6)	12.21
Glutamine	1 hour	4.02 ± 0.07 (7)	3.89 ± 0.15 (7)	–3.23
	1 month	2.97 ± 0.08 (6)	3.29 ±0.11 (6)	10.77*
	2 months	2.35 ± 0.09 (7)	2.36 ± 0.07 (7)	0.43
	4 months	4.19 ± 0.17 (6)	3.69 ± 0.07 (7)	–11.93*
	4 months + 1 month recovery	3.43 ± 0.07 (6)	3.09 ± 0.05 (6)	–9.91*
GABA	1 hour	2.07 ± 0.07 (7)	2.00 ± 0.07 (7)	–3.38
	1 month	1.74 ± 0.03 (7)	1.76 ± 0.03 (7)	1.15
	2 months	1.56 ± 0.03 (7)	1.53 ± 0.02 (7)	–1.92
	4 months	1.96 ± 0.05 (5)	2.19 ± 0.08 (6)	11.73*
	4 months + 1 month recovery	1.77 ± 0.02 (6)	1.99 ± 0.04 (6)	12.43*
Glycine	1 hour	1.31 ± 0.14 (7)	1.36 ± 0.09 (7)	3.82
	1 month	1.07 ± 0.06 (7)	1.13 ± 0.06 (7)	5.61
	2 months	0.99 ± 0.16 (7)	0.96 ± 0.05 (7)	–3.03
	4 months	1.29 ± 0.04 (6)	1.41 ± 0.04 (6)	9.30
	4 months + 1 month recovery	1.03 ± 0.05 (6)	1.30 ± 0.02 (6)	26.21*
Taurine	1 hour	5.56 ± 0.18 (6)	4.56 ± 0.24 (6)	–17.99*
	1 month	3.80 ± 0.11 (6)	3.86 ± 0.08 (7)	1.58
	2 months	2.62 ± 0.13 (6)	2.99 ± 0.05 (6)	14.12*
	4 months	4.82 ± 0.14 (5)	5.27 ± 0.10 (6)	9.34*
	4 months + 1 month recovery	3.87 ± 0.16 (7)	4.03 ± 0.14 (7)	4.13

Mean ± SEM is represented with number of animals between brackets; *p< 0.05 significant; %*d*: percentage difference from control.

DISCUSSION

Huber *et al*. [13] reported an increase in cerebral metabolism immediately after electromagnetic irradiation over some exposed regions of the scalp and this effect was interpreted as a consequence of altered activity induced by EMF exposure. This increase in cerebral metabolism may result in an increase in the rate of cerebral glucose utilization that represents the main substrate for the brain [33]. Glucose is metabolized in the neurons and glia into amino acid neurotransmitters [6]. Thus, the rapid increase in cortical glutamate, aspartate, GABA and glycine after one hour of exposure in the present study may be due to the increase in cerebral metabolic rate induced by EMR.

After one month of daily exposure of adult animals to EMR, a significant decrease in the excitatory amino acid neurotransmitters, glutamate and aspartate was observed in the present study that was accompanied by a non significant decrease in the inhibitory amino acid neurotransmitter.

In the brain, a low extracellular concentration of neurotransmitters is normally maintained between times of exocytotic transmission in order to prevent continued activation or desensitization of receptors and for excitatory transmitter glutamate to avoid excitotoxicity [5].

It has been reported that an acute exposure to high frequency power GSM (900 MHz) microwaves is able to induce a significant reduction in the presence of NMDA receptors at the postsynaptic membrane level in the cortex and striatum and biochemical modifications of the neurotransmitter receptor properties [25].

Accordingly, it may be assumed that the significant decrease in cortical glutamate and aspartate observed in the present study may be attributed to their release under the effect of EMR exposure. This in turn may result in the reported significant decrease in NMDA receptors in the cortex.

Glutamate has many other fates in the brain, including oxidation for energy, incorporation into proteins and formation of glutamine, GABA and glutathione [26]. Moreover, it has been demonstrated that mobile phone can cause oxidative damage biochemically by increasing the level of nitric oxide, malondialdehyde as well as xanthine oxidase and adenosine deaminase activities in the brain tissue [15, 27]. In addition, the brain tissues and blood glutathione concentrations were significantly lowered in the EMF-exposed animals than in the control [27, 36]. In the brain there is a transport system that mediates the exchange of glutamate for cystine [3, 17]. Under physiological conditions, this transporter provides a source of cystine to cells that need to produce glutathione [2].

Thus, it may be suggested that the significant decrease in the cortical glutamate level after one month, in the present study, may explain the reported decrease in glutathione level in the brain tissues.

In the present study, after two months of daily exposure of adult rats to EMR, a significant increase in the concentration of glutamate, aspartate, glycine and taurine was observed.

Taurine has been found to act as a neuroprotector against glutamate-induced excitotoxicity and cell death [35]. It has been observed that taurine suppressed the synaptic release of D-aspartate and glutamate [28].

Therefore, it can be assumed that the significant increase in the cortical glutamate and aspartate induced by EMR exposure for two months, in the present study, may be due to the inhibition of their release under the neuroprotective effect of taurine and consequently their accumulation in the nerve terminals.

It has been suggested that glycine is a major causative molecule released from microglia that potentiates NMDA-induced current [12]. Glycine acts as a coagonist at NMDA receptors which could enhance the effectiveness of normal NMDA receptors-mediated glutamatergic neurotransmission [34]. Moreover, it has been reported that NMDA receptors are regulated not only by glutamate but also by the amino acids glycine and D-serine which bind to glycine binding sites of NMDA receptor complex [18] where glycine has been found to work as an excitatory agent via activating NMDA receptors [19].

Therefore, the significant increase in cortical glycine reported in the present study after two months of EMR exposure may be a compensatory mechanism for the attenuated glutamate release induced by taurine.

After four months of daily EMR exposure, aspartate, GABA, glycine and taurine increased significantly; however, glutamine decreased significantly. It is clear from the data of the present study that there is a parallel significant increase in aspartate, taurine and glycine levels after two and four months; however, the increase in taurine was reduced from 15.11% after two months to 9.49% after four months of daily EMR exposure. This in turn may attenuate the neuroprotective role of taurine on aspartate and subsequently permit its release. This may explain the parallel reduction of aspartate increase from 30.25% after 2 months to 10.45% after four months. Supporting this notion is the reduction in glycine increase from 25.20% after two months to 14.18% after four months as glycine has excitatory effects at NMDA receptors. Thus, it may be suggested that the increase in aspartate release together with the elevated level of glycine may induce a state of hyperexcitability. However, the increase in cortical GABA level in the present study may be a mechanism by which the brain ameliorates the state of hyperexcitability. Furthermore, this increase in GABA could be at the expense of the decreased glutamine level. It has been suggested that 50% of the endogenous GABA released is derived from glutamine [4].

From the present results, it may be speculated that the changes in the cortical amino acid neurotransmitters induced by EMR exposure in adult rats may underlie the changes in cortical excitability reported after EMR exposure.

In the cortex of young rats, non significant changes in most of the studied amino acid neurotransmitters have been recorded after one hour, one month and two months of daily exposure except for a significant decrease in taurine after one hour of acute exposure to EMR.

It has been demonstrated that both NMDA and non-NMDA subtypes of glutamate receptors are involved in glutamate-mediated toxicity but their relative contribution is highly dependent on the age of the animals [31]. Indeed, in young individuals cellular defenses against free radical-induced protein oxidation by antioxidant enzymes are likely in prime shape and the proteases and protein synthesis machinery are fully functional [15].

Thus, the non significant changes in the cortical amino acid neurotransmitters of young rats mostly observed in the first studied three time intervals may be attributed to the ability of young rats to resist the cell stress induced by EMR.

After four months of daily exposure to EMR, the cortex of young rats showed a significant decrease in glutamine that was accompanied by a significant increase in GABA and taurine. It is clear that as young rats reached adulthood, the results obtained after four months revealed a state of inhibition as evident from the increase in GABA, at the expense of the decreased level of glutamine. However, unlike the adult stage, these changes continued after stopping EMR for 1 month.

The present study suggests that EMR exposure especially in young animals induced a delayed effect on the cortical amino acid neurotransmitters.

It may be suggested that the recorded changes in amino acid levels in the cortex of adult and young rats, in the present study, may underlie and explain the changes in cortical excitability reported after exposure to electromagnetic radiation emitted from mobile phone. However, further studies are needed to verify these effects and investigate their significance to brain functions.

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REFERENCES

1. AHMED, A.N., A. ASSAD, H.S. ABOUL EZZ, N.M. RADWAN, Effect of Exposure to Electromagnetic Radiation from the Mobile Phone on Acetylcholinestrase Activity in the Hippocampus and Striatum of Young and Adult Male Rats , *Med. J. Cairo Univ.*, 2006, **74**(2) (Suppl.), 129–135.
2. BAKER, D.A., Z.X. Xi, H. SHEN, C.J. SWANSON, P.W. KALIVAS, The origin and neuronal function of in vivo glutamate, *J. Neurosci.*, 2002, **22**, 9134–9141.
3. BANNAI, S., Exchange of cystine and glutamate across plasma membrane of human fibroblasts, *J. Biol. Chem.*, 1986, **261**, 2256–2263.
4. BRADFORD, H.F., H.R. WARD, A.J. THOMAS, Glutamine-a major substrate for nerve endings, *J. Neurochem.*, 1978, **30**, 1453–1459.
5. CAVELIER, P., M. HAMANN, D.J. ROSSI, P. MOBBS, D. ATTWELL, Tonic excitation and inhibition of neurons, *Prog. Biophys. Mol. Biol.*, 2005, **87**, 3–16

6. CHOWDHURY, G.M., M. GUPTA, K.M. GIBSON, A.B. PATEL , K.L. BEHAR, Altered cerebral glucose and acetate metabolism in succinic semialdehyde dehydrogenase-deficient mice: evidence for glial dysfunction and reduced glutamate/glutamine cycling, *J. Neurochem.*, 2007,**103**, 2077–2091.
7. CURCIO, G., M. FERRARA, F. MORONI, G. D'INZEO, M BERTINI, L.DE GENNARO, Is the brain influenced by a phone call? An EEG study of resting wakefulness, *Neurosci. Res.*, 2005, **53**, 265–270.
8. EDELSTYN, N., A. OLDERSHAW, The acute effects of exposure to the electromagnetic field emitted by mobile phones on human attention. *Neuroreport*, 2002, **13**, 119–121.
9. GRAFSTRÖM, G., H. NITTBY, A. BRUN, L. MALMGREN, B.P. PERSSON, L.G. SALFORD, J. EBERHARDT, Histopathological examinations of rat brains after long-term exposure to GSM-900 mobile phone radiation, *Brain Res Bull.*, 2008, Sep 6 [Epub ahead of print].
10. HAARALA, C.F., F. TAKIO, T. RINTEE, M. LAINE, M. KOIVISTO, A. REVONSUO, H. HÄMÄLÄINEN, Pulsed and continuous wave mobile phone exposure over left versus right hemisphere: effects on human cognitive function, *Bioelectromagnetics*, 2007, **28**(4), 289–295.
11. HAMBLIN, D.L., A.W. WOOD, Effects of mobile phone emissions on human brain activity and sleep variables, *Int. J. Radiat. Biol.*, 2002, **78**, 659–669.
12. HAYASHI, Y., H. ISHIBASHI, K. HASHIMOTO, H. NAKANISHI, Potentiation of the NMDA receptor-mediated responses through the activation of the glycine site by microglia secreting soluble factors, *Glia*, 2006, **53**(6), 660–668.
13. HUBER, R., V. TREYER, A.A. BORBÉLY, J. SCHUDERER, J.M. GOTTSELIG, H.P. LANDOLT, E. WERTH, T. BERTHOLD, N. KUSTER, A. BUCK, P. ACHERMANN, Electromagnetic fields such as those from mobile phones alter regional cerebral blood flow and sleep and waking EEG, *J. Sleep Res.*, 2002, **11**, 289–265.
14. HYLAND, G.J., Physics and biology of mobile telephony, *Lancet*, 2000, **356**, 1833–1836.
15. ILHAN, A., A. GUREL, F. ARMUTCU, S. KAMISLI, M. IRAZ, O. AKYOL, S. OZEN, *Ginko biloba* prevents mobile phone-induced oxidative stress in rat brain, *Clin. Chim. Acta*, 2004, **340**, 153–162.
16. INABA, R., K. SHISHIDO, A. OKADA, T. MOROJI, Effect of whole body microwave exposure on the rat brain contents of biogenic amines, *Eur. J. Appl. Physiol. Occup. Physiol.*, 1992, **65**, 124–128.
17. ISHII, T., H. SATO, K. MIURA, J. SAGARA, S. BANNAI, Induction of cystine transport activity by stress, *Ann. N.Y. Acad. Sci.*, 1992, **663**, 497–498.
18. JOHNSON, J.W., P. ASHER, Glycine potentiates the NMDA responses in cultured mouse brain neurons, *Nature*, 1987, **325**, 529–531.
19. KEMP, J.A., P.D. LEESON, The glycine site of the NMDA receptor: Five years on, *Trends Pharmacol. Sci.*, 1993, **14**, 20–25.
20. KOIVISTO, M., C.M. KRAUSE, A. REVONSUO, M. LAINE, H. HÄMÄLÄINEN, The effects of electromagnetic field emitted by GSM phones on working memory, *Neuroreport*, 2000, **11**, 1641–1643.
21. KOIVISTO, M, A. REVONSUO, C.M. KRAUSE, C. HAARALA, L. SILLANMÄKI, M. LAINE, H. HÄMÄLÄINEN, Effects of 902 MHz electromagnetic field emitted by cellular telephones on response times in humans, *Neuroreport*, 2000, **11**, 1–3.
22. KWON, M.S., M. KOIVISTO, M. LAINE, H. HÄMÄLÄINEN, Perception of the electromagnetic field emitted by a mobile phone, *Bioelectromagnetics*, 2008, **29**(2),154–159.
23. LAI, H, M.A. CARINO, A. HORITA, A.W. GUY, Corticotropin-releasing factor antagonist blocks microwave-induced decrease in high-affinity choline uptake in the rat brain, *Brain Res Bull.*, 1990, **25**, 609–612.
24. MÁRQUEZ, F.J., A.R. QUESADA, F. SÁNCHEZ-JIMÉNEZ, I. MÚÑEZ DE CASTRO, Determination of 27 dansyl amino acid derivatives in biological fluids by reversed-phase high performance liquid chromatography, *J Chromatogr.*, 1986, **380**, 275–283.

25. MAUSSET-BONNEFONT, A.L., H. HIRBEC, X. BONNEFONT, A. PRIVAT, J. VIGNON, R. DE SEZE, Acute exposure to GSM 900-MHz electromagnetic fields induces glial reactivity and biochemical modifications in the rat brain, *Neurobiol. Dis.*, 2004, **17**(3), 445–454.
26. MCKENNA, M.C., The glutamate-glutamine cycle is not stoichiometric: fates of glutamate in brain, *J. Neurosci Res.*, 2007, **85**(15), 3347–3358.
27. MERAL, I., H. MERT, N. MERT, Y. DEGER, I. YORUK, A. YETKIN, S. KESKIN, Effects of 900-MHz electromagnetic fields emitted from cellular phone on brain oxidative stress and some vitamin levels of guinea pigs, *Brain Res.*, 2007, **1169**(12), 120–124.
28. MOLCHANOVA, S.M., S.S. OJA, P. SARANSAARIA, Taurine attenuates D-[3H]aspartate release evoked by depolarization in ischemic corticostriatal slices, *Brain Res.*, 2006, **1099**, 64–72.
29. NITTBY, H., G. GRAFSTRÖM, D.P. TIAN, L. MALMGREN, A. BRUN, B.R. PERSSON, L.G. SALFORD, J. EBERHARDT, Cognitive impairment in rats after long-term exposure to GSM-900 mobile phone radiation, *Bioelectromagnetics*, 2008, **29** (3),219–232.
30. PAXINOS, G., C. WATSON, *The Rat brain in Stereotaxic Coordinates*, Academic Press, San Diego, 1986.
31. SANGANAHALLI, B.G., P.G. JOSHI, N.B. JOSHI, NMDA and non-NMDA receptors stimulation causes differential oxidative stress in rat cortical slices, *Neurochem. Int.*, 2006, **49**(5), 475–480.
32. SCHÖRNBORN, F., M. BUCKHARDT, N. KUSTER, Differences in energy absorption heads of adults and children in the near of field sources, *Health Phys.*, 1998, **74**, 160–168.
33. SCHOUSBOE, A., L.K. BAK, H.M. SICKMANN, U. SONNEWALD, H.S. WAAGEPETERSEN, Energy substrates to support glutamatergic and GABAergic synaptic function: role of glycogen, glucose and lactate, *Neurotox. Res.*, 2007, **12**, 263–268.
34. SUR, C., G.G. KINNEY, Glycine transporter 1 inhibitors and modulation of NMDA receptor-mediated excitatory neurotransmission, *Curr. Drug Targ.*, 2007, **8**(5), 643–649.
35. WU, H., Y. JIN, J. WEI, H. JIN, D. SHA, J-Y. WU, Mode of action of taurine as a neuroprotector, *Brain Res.*, 2005, **1038**, 123–131.
36. YUREKLI, A.I., M. OZKAN, T. KALKAN, H. SAYBASILI, H. TUNCEL, P. ATUKEREN, K. GUMUSTAS, S. SEKER, GSM base station electromagnetic radiation and oxidative stress in rats, *Electromag. Biol. Med.*, 2006, **25**, 177–188.