

## Functional and Destructive Effects of Microwave Radiation on Rat Brain

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**W**HITE RATS were *in vivo* exposed to microwave (MW) radiation in the cellular phone communication frequency range to investigate whether such exposure produces functional or morphological damage in the brain.

Three groups were investigated: Mobile phone (MP) handset group, mobile stations tower (MT) group, and microwave source (MS) group. Subgroups of all exposed rats were taken for recovery studies. ECoG activities and histopathological changes were recorded before, during and after microwave exposure.

As compared to control animals, ECoG patterns in exposed animals were found to be distinctly altered in each case of exposure. Some fluctuations had been recorded in shape, frequency, and amplitude of ECoG pattern accompanied with structural changes in some brain tissue elements

The interaction of microwave radiation with biological systems has been extensively investigated<sup>(1,2,3)</sup>. The body organs affected by microwave exposure were reported to be susceptible, in terms of functional disturbance and/or structural alterations<sup>(4,5,6)</sup>. With the widespread use of the mobile telephones devices and the presence of mobile tower stations mounted on building roofs, there is concern regarding possible health hazards, particularly those related to brain tissues since the phone antenna lies alongside the head during usage<sup>(7)</sup>.

On repeated exposure to mobile phones<sup>(8-10)</sup>. Reported significant changes in the brain electrophysiology; evoked potentials, and EEG records. In addition, a pathological leakage of the blood-brain barrier was shown. Neural effects have also been reported including  $Ca^{++}$  changes (essential for cell communication and growth regulation), neurotransmitters (chemicals that conduct nerve signals and control such things as appetite, mood, behaviour, drug responses, sleep, learning and memory), behavioural changes and sleep disorder. Moreover, damage of brain tissues was noticed to be manifested by encephalo-proliferation of glial cells, neural degeneration and neuro-phagia<sup>(11)</sup>. However, the results of the studies are mostly inconsistent. Additional systematic studies are needed to

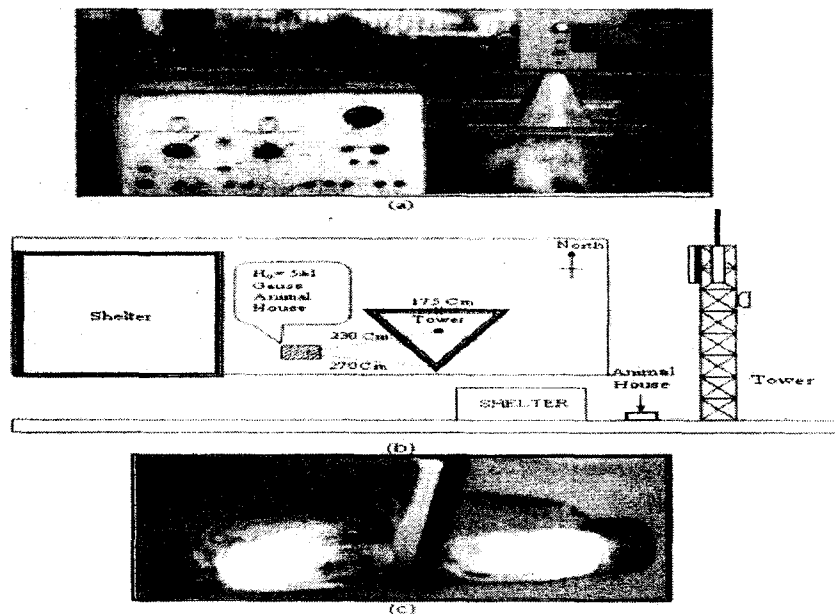
identify possible reproducible effects and the biologically active parameters controlling these effects. The present study was carried to investigate the effect of long term low intensity MW exposure could have on brain function and structure using continuous microwave source (MS), GSM mobile phone (MP) and mobile tower (MT) stations.

### Material and Methods

#### *Animal exposure*

80 female rats weighing 110-130 gram were used in this study. Ten of them were used for control studies. The rest of the animals were grouped into three groups:

- 1- Microwave source (MS) group; in which 30 rats were exposed, 1.5h daily, for 1, 2 or 3 months to a microwave source (Fig.1a) at a frequency of 9.86 GHz, average power density of  $0.248 \text{ mW/cm}^2$  (SAR=2mW/Kg).
- 2- Mobile tower (MT) group; in which 20 rats were continuously exposed to mobile station radiation, (Fig.1b), at a frequency of 950 MHz, intensity  $0.4 \text{ mW/cm}^2$  and a distance of 230cm from the tower center, for 1,2 or 3 months.
- 3- Mobile phone (MP) hand set group; (20 rats) where the rats were placed, 90 min daily, for 15, 30 & 40 days with their head in close contact with the phone set (Fig.1c) at a frequency 950 MHz and intensity  $0.4 \text{ mW/cm}^2$ .



**Fig. 1. a. A photograph for the arrangement of animal exposure to a microwave source, B; Schematic diagram of the tower, shelter of mobile station and the animal house and C; A photograph for the mobile phone exposure. The animal container is perfectly closed except for some holes in the side facing the mobile in order to force the animal to direct its head towards the mobile.**

Subgroups of the exposed rats were taken for recovery studies. Electrophysiological activities (ECoG) and histopathological changes of the brain were recorded before, during and after microwave exposure.

#### *Electrophysiological methods*

All the animal groups were housed in a good airing, feeding the same diet, and live nearly under the same conditions. After complete animal anesthesia, according to David and Arthur, (1997) the animal was fixed on a board, the skin of the head at the mid line, was cut and reflected towards the sides as far as possible, and the cranial bones were thoroughly cleaned. The ECoG activity was directly recorded from the cortical surface through a set of trephine openings drilled in the skull of rat brain. Two trephine openings, 2mm in diameter, were made in the skull over the frontal regions of the right hemisphere for ECoG recording using wick Ag-AgCl electrodes and another 1mm opening in the left hemisphere for a common reference earthed electrode. All the openings were made carefully by using a dental driller for removing only the skull bone without any injury of the brain duramatter cortex. The exposed regions of the brain were covered with warm Ringer's solution suitable for the brain.

#### *ECoG data analysis*

The ECoG is an extremely complex signal with a periodic waveform, making it very difficult to quantitate. On investigating the ECoG chart, it was noticeable that it contains relatively long period pulses (LP) of few parts of a mV separated by inactive duration regions and short time pulses of few microvolts (Fig. 2a). Each pulse consists of a number of peaks. The rate of appearance of these pulses may be regular (RP), or irregular according to the brain physiological conditions. The data from these records were represented as pulse amplitude  $\mathcal{A}$ , peak frequency  $\mathcal{F}$ , percentage of the appearance of long period pulses (LP)  $\mathcal{L}$  % and ECoG regularity  $\mathcal{R}$ , where

$$\mathcal{R} \% = \frac{\Sigma \text{ Regulation Time}}{\text{Total Time of the Chart}} (\%)$$

$$\mathcal{L} \% = \frac{\Sigma \text{ LP Time}}{\text{Total Time of the Chart}} (\%)$$

$$\mathcal{F} = \frac{\text{Peak's No.}}{\text{Pulse Time}}$$

#### *Histopathological Method*

Animals of all the above mentioned groups (control, MS, MT & MP groups) were used in this investigation. The brain of the anesthetized rats was carefully extracted from the skull. After saving in 10% formalin it was subjected to Haematoxyline-Eosin histological study. The brain samples of control and treated animals were processed and impeded in paraffin according to Drury and Walligton<sup>(12)</sup>. This technique is the most commonly used method since it gives

good and fast results. Semi thin sections (1-2  $\mu\text{m}$ ) were cut with the ultratome, mounted in glass slides, stained with Haematoxyline-Eosin, and examined by the light microscope.

### Results

#### *ECoG records*

Typical ECoG records from normal unexposed rat brain are shown in (Fig. 2b). The waveform of the ECoG, its characteristic parameters; amplitude  $A$ , frequency  $\mathcal{F}$ , regularity  $\mathcal{R}\%$  and the percentage long period pulse appearance  $\mathcal{L}\%$  depend on the general physiological conditions of the animal and on the position of the recording electrode. In this work, the electrode position in the brain opening was fixed during all the experiments because different brain regions do not display the same rhythms at the same time. Table 1 summarizes the calculated values of all the ECoG parameters in case of control animals. The results indicated that the F-values lie in the  $\delta$ -wave range (1-4Hz) of anesthetized and sleep EEG waveform.

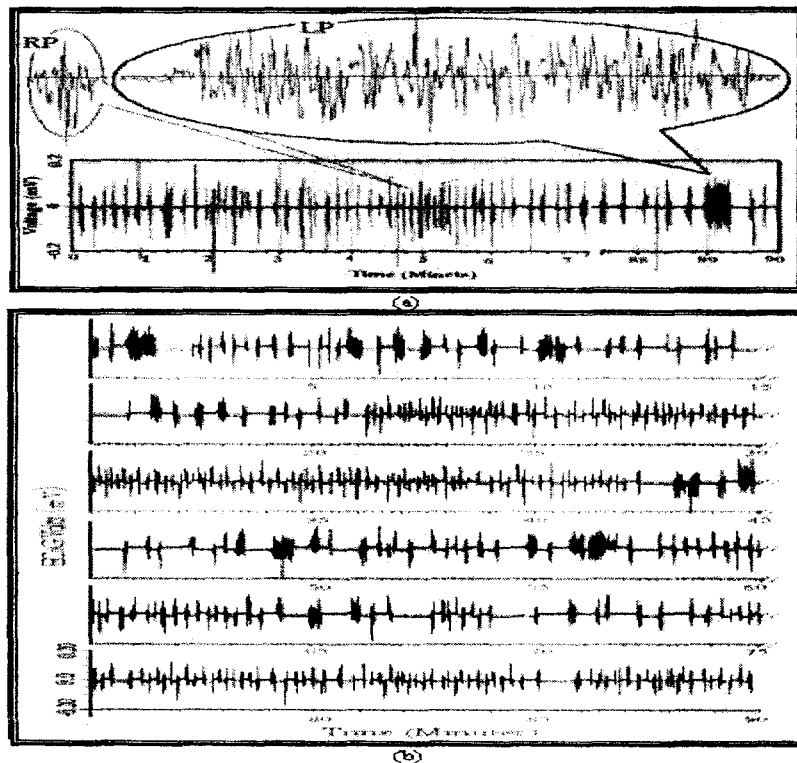


Fig. 2 a. RP and LP pulses in an ECoG chart sample and there and b; Typical ECoG pattern of a control experiment.

TABLE 1. The calculated ECoG parameters of control group ( $\pm$  SD) .

	RP		LP		$\mathcal{R}$ %	$\mathcal{L}$ %
	$\mathcal{A}$ (mV)	$\mathcal{F}$ (Hz)	$\mathcal{A}$ (mV)	$\mathcal{F}$ (Hz)		
Control	0.135 $\pm$ 0.064	1.657 $\pm$ 0.351	0.156 $\pm$ 0.084	1.587 $\pm$ 0.404	47.779 $\pm$ 8.733	8.678 $\pm$ 1.242

*1-Microwave source (MS) effects*

On exposing the animals to MS, for 1.5 h, the ECoG pattern showed significant deformations during and immediately after ending exposure. These deformations were reflected in the form of insanity in frequency, amplitude and  $\mathcal{L}$  %. An increase in  $\mathcal{F}$ , accompanied by a marked decrease in  $\mathcal{A}$ , was noticed during exposure and these variations became less pronounced after ending exposure. Also, a decrement in regularity  $\mathcal{R}$ % was obvious (Fig 3a&b) during and after exposure. In case of long term exposure of rats to MS for one, two or three months, ECoG records indicated more pronounced variations in the RP & LP (Fig. 4) and in all the calculated parameters. These variations appeared as a depression in regularity  $\mathcal{R}$ %, a rise in  $\mathcal{L}$ %,  $\mathcal{F}$ , and  $\mathcal{A}$  and they became more pronounced with the time of MS application.

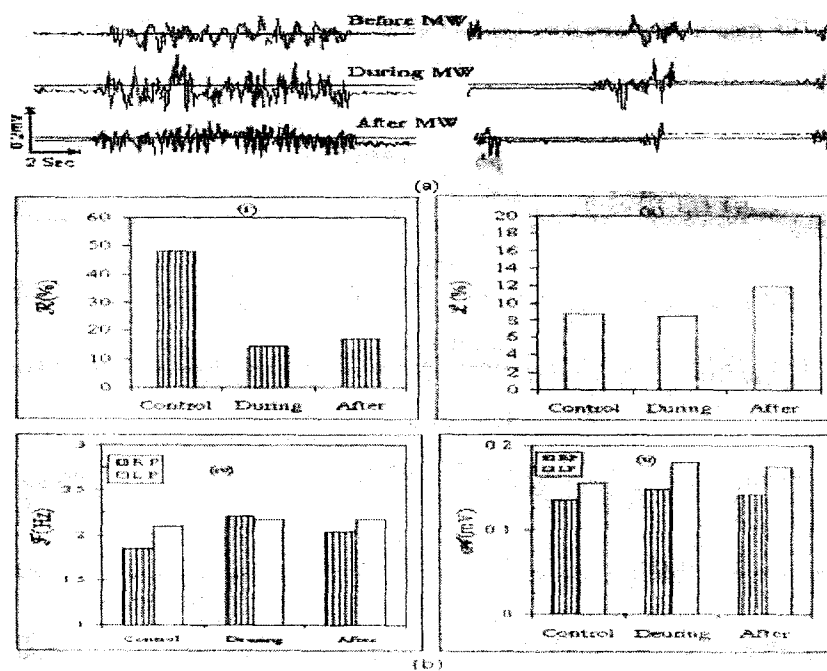


Fig.3. a. ECoG chart samples of R and L pulses for the same rat before, during and after exposing to MS and B. ECoG  $\mathcal{R}$  % (i),  $\mathcal{L}$ % (ii), frequency  $\mathcal{F}$  (iv) & amplitude  $\mathcal{A}$  (v) variations in RP and LP during, after exposure of immediate CMW comparing with control results.

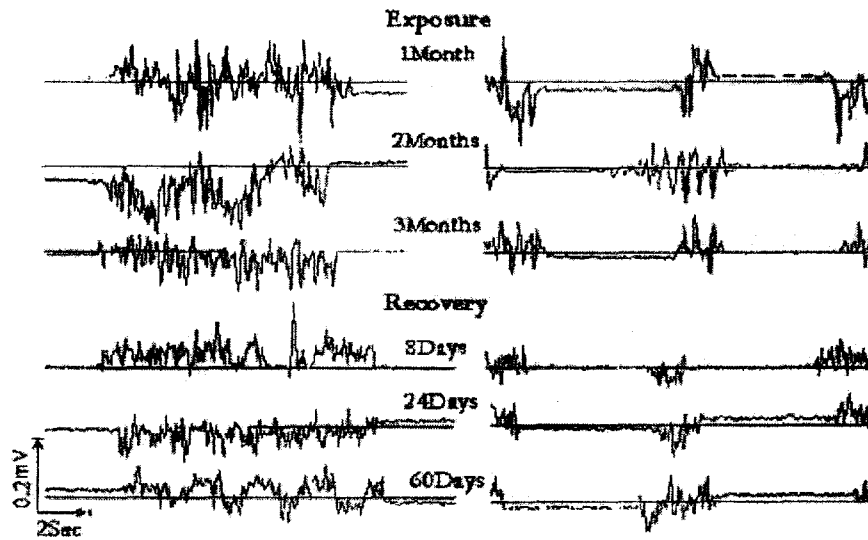


Fig. 4. Typical ECoG RP and LP sections after different periods of MS exposure and during recovery.

The measured parameters showed some sort of recovery after 8, 24, & 60 days of source removal but they did not return back to normal values (Fig. 5, a, b & c).

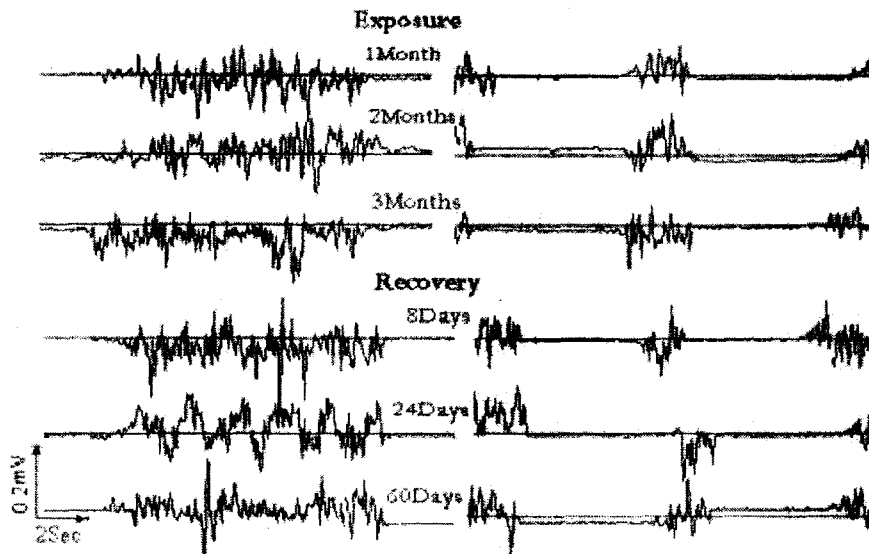


Fig. 5. Typical ECoG sections of RP and LP of rats after 1, 2 and 3 months of mobile tower (MT) exposure.

*Mobile tower (MT) effects*

Typical ECoG samples of LP and RP types, after exposure to MT for different periods; 1, 2, & 3 months and the variations in the calculated parameters are shown in (Fig. 6 & 7a, b, & c). A decrease in  $\mathcal{R}\%$ , increase in  $\mathcal{L}\%$  &  $\mathcal{F}$  were noticed and became more significant with exposure time. The amplitude  $\mathcal{A}$  showed an increase reached to about double its control value after 1 month exposure and then decreased with exposure time.

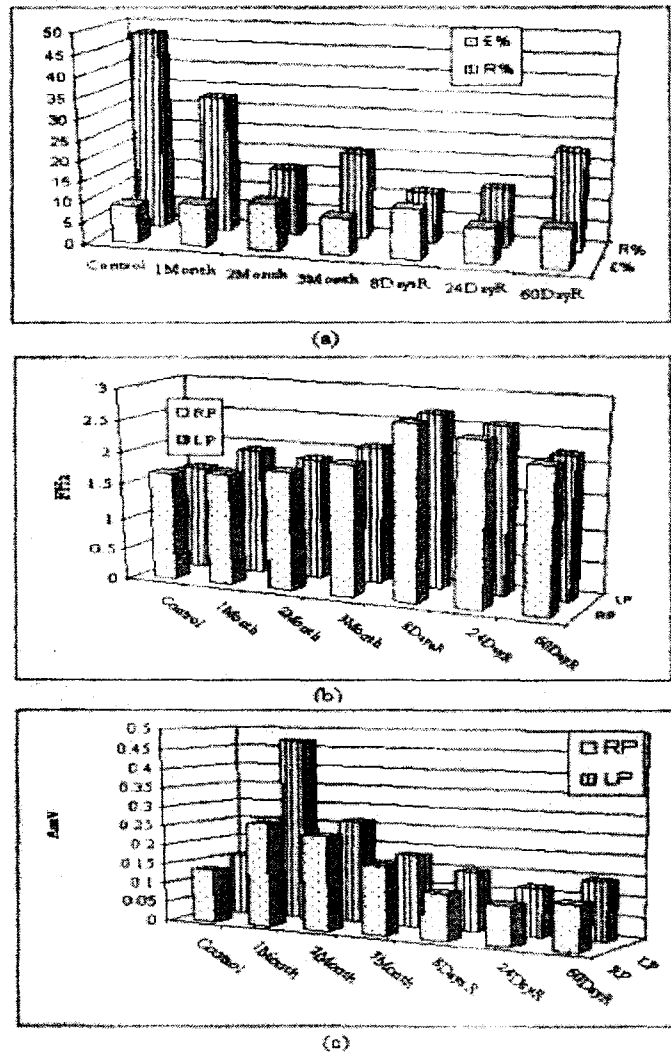


Fig. 6. variation of  $\mathcal{R}\%$  and  $\mathcal{L}\%$  values; (a), Frequency of RP and LP pulses; (b) &  $\mathcal{A}$  values of RP and LP; (c) during and after MS exposure and recovery periods.

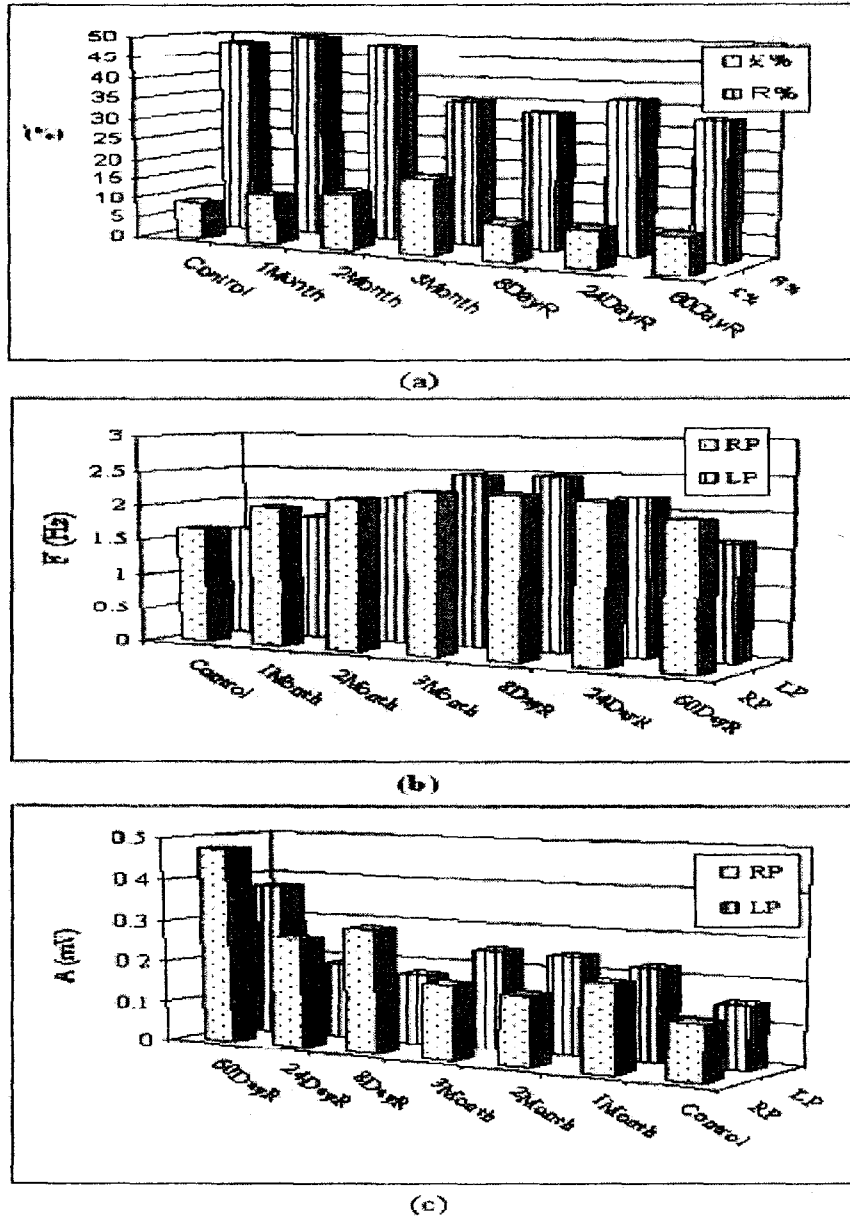


Fig. 7. a Variation of  $\mathcal{R}(\%)$  and  $\mathcal{L}(\%)$  values, (B); Frequency of RP & LP pulses, and (C) . Values of RP and LP during and after MT exposure and recovery periods.



The variations of these parameters were estimated after 8, 24 & 60 days of exposure cessation. Little uncompleted recovery was observed during the above mentioned periods. The ECoG pattern still irregular ( $\mathcal{R}$ =30% of control value) even after 60 days of exposure.

*Mobile phone (MP) effects*

ECoG records of LP and RP from animals exposed to MP source, for 1.5 hr, and after the removal of the handset mobile are illustrated in Fig. 8a. The variations in the calculated ECoG parameters are shown in Fig. 8b. The  $\mathcal{R}$ % value was noticeably decreased to about one third of the control value. The decrease is more pronounced after ending the call and removing the phone. On the other hand, the value of  $\mathcal{L}$ % showed a remarkable increase during the talking period and still larger than control value even after ending the call. Regarding the amplitude  $\mathcal{A}$  and the frequency  $\mathcal{F}$ , they both increased during exposure, decreased after ending the exposure but did not return back to control values even after 40 days.

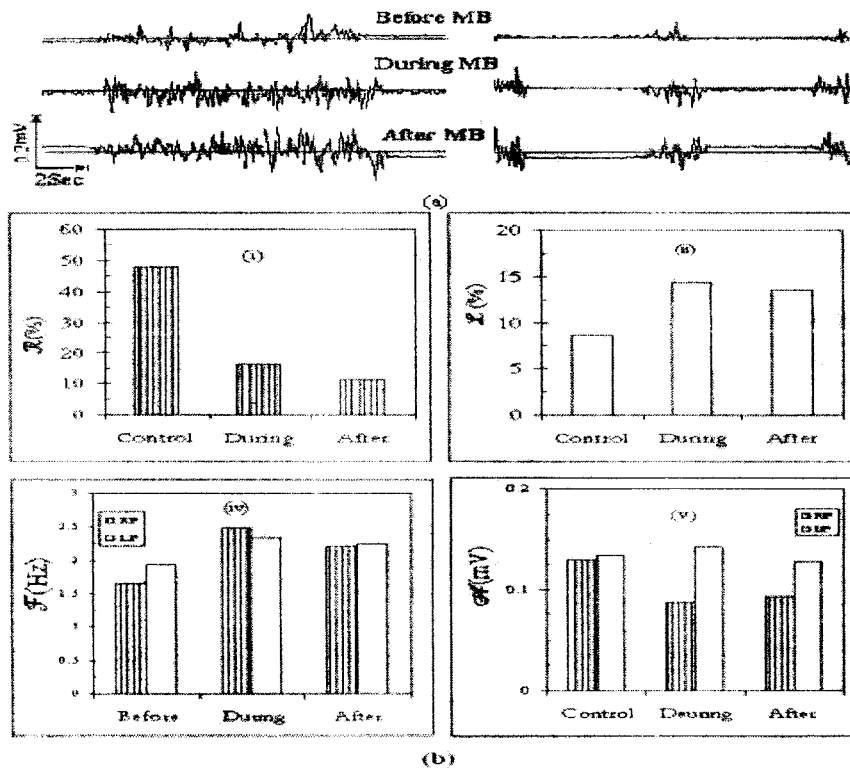


Fig. 8 a. ECoG chart samples of R and L pulses for the same rat before, during and after exposing to MS, and (b, i); ECoG  $\mathcal{R}$  % (ii); ECoG  $\mathcal{L}$ %, (iv); ECoG frequency  $\mathcal{F}$  & (v); ECoG amplitude  $\mathcal{A}$  variations in RP and LP during, after exposure of immediate MP comparing with control results .

The effects of long term exposure of animals to MP, 90 min daily, for 15, 30 & 40 days on the ECoG records were also studied (Fig. 9 & 10 (a, b, & c)). In these Figures the recovery results are also included. A significant decrease was noticed in both  $\mathcal{R}\%$  &  $\mathcal{L}\%$  whereas  $\mathcal{A}$  &  $\mathcal{F}$  were increased with exposure period. The recovery results indicated that  $\mathcal{R}\%$  &  $\mathcal{L}\%$  were increased with recovery time but their values were still smaller than the control ones. After 40 days of stopping exposure, the amplitude  $\mathcal{A}$  was approximately approached to control value whereas  $\mathcal{F}$  showed some recovery but still smaller than control.

#### *MS effects on rat brain histology*

Microscopic examination of rat brain tissue, after one & two months exposure to MS showed nearly similar changes. These changes appeared in the form of congestion of blood vessels with lymphocytic cellular infiltrations, neural degeneration and neurophagia of the brain tissues Fig.11 (b, c). After 3 month exposure, focal areas of encephomalacia represented by tissue vaculation with diffused gliosis were noticed. There was also severe congestion of blood vessels with perivascular and parenchymal hemorrhage in addition to perineural edema and demylin-ation of some neurons (Fig. 11d).

In recovery group, the examined brain samples still revealed congestion of blood vessels and multiple areas of hemorrhage with demylination of neurons after 8 days (Fig. 11e). After 24 days, similar recorded lesions were noted in addition to focal gliosis, neural degeneration, neurophagia and encephomalacia. After 60 days, only blood vessels congestion was seen in the examined sample.

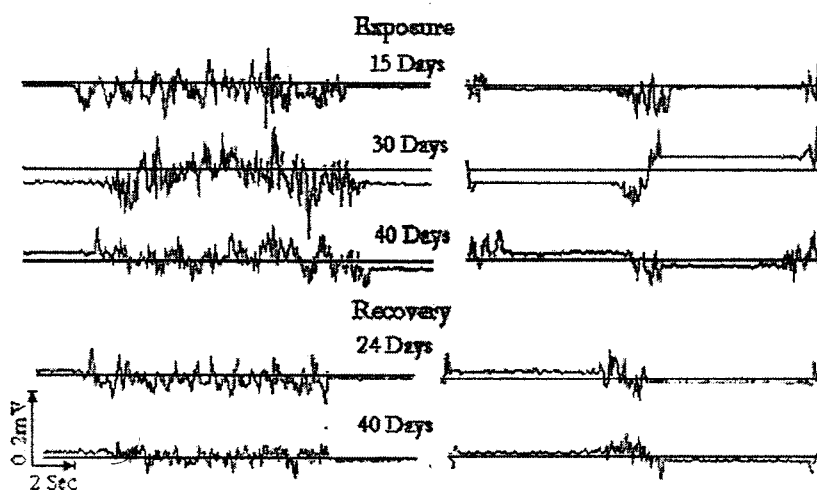


Fig. 9. ECoG charts samples of RP and LP pulses for exposing rat groups to mobile phone (MP).

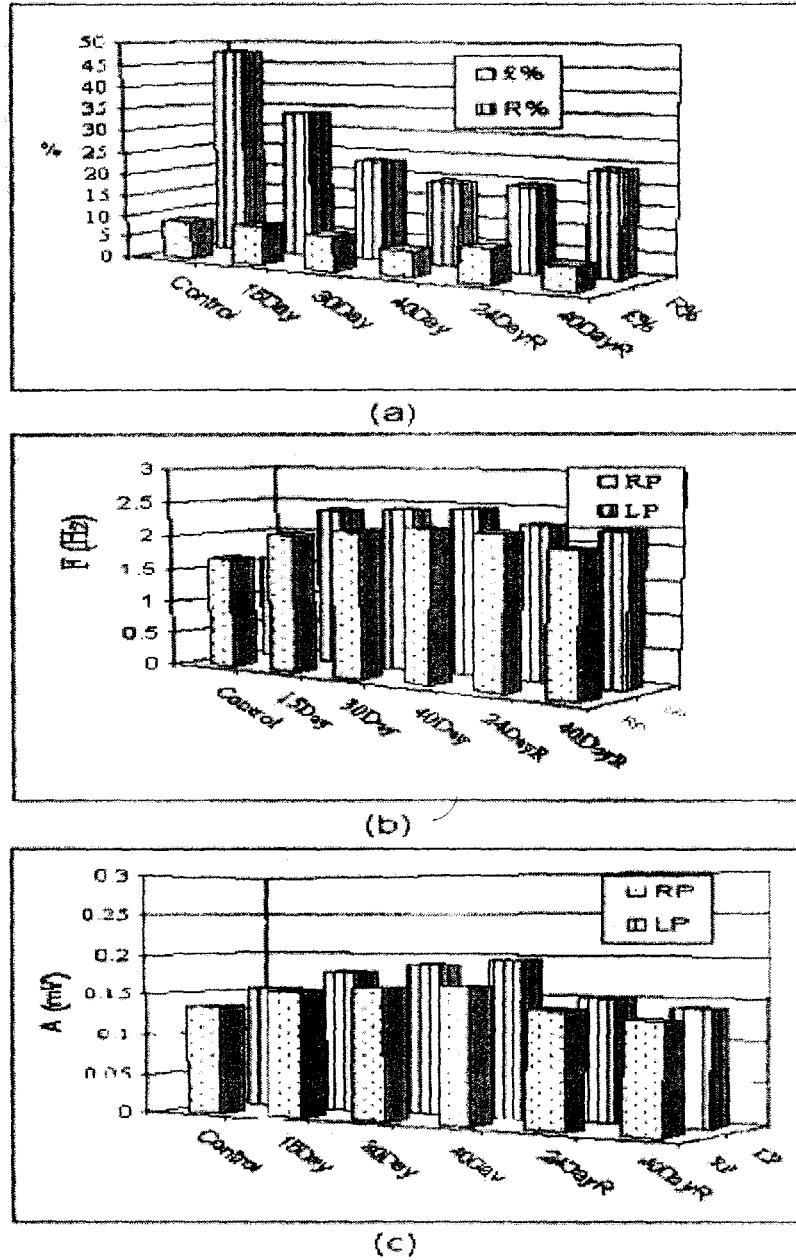


Fig. 10. A variation of  $\mathfrak{R}(\%)$  and  $\mathfrak{L}(\%)$  values, (B) ;Frequency of RP & LP pulses, and (C); A values of RP and LP during and after MT.

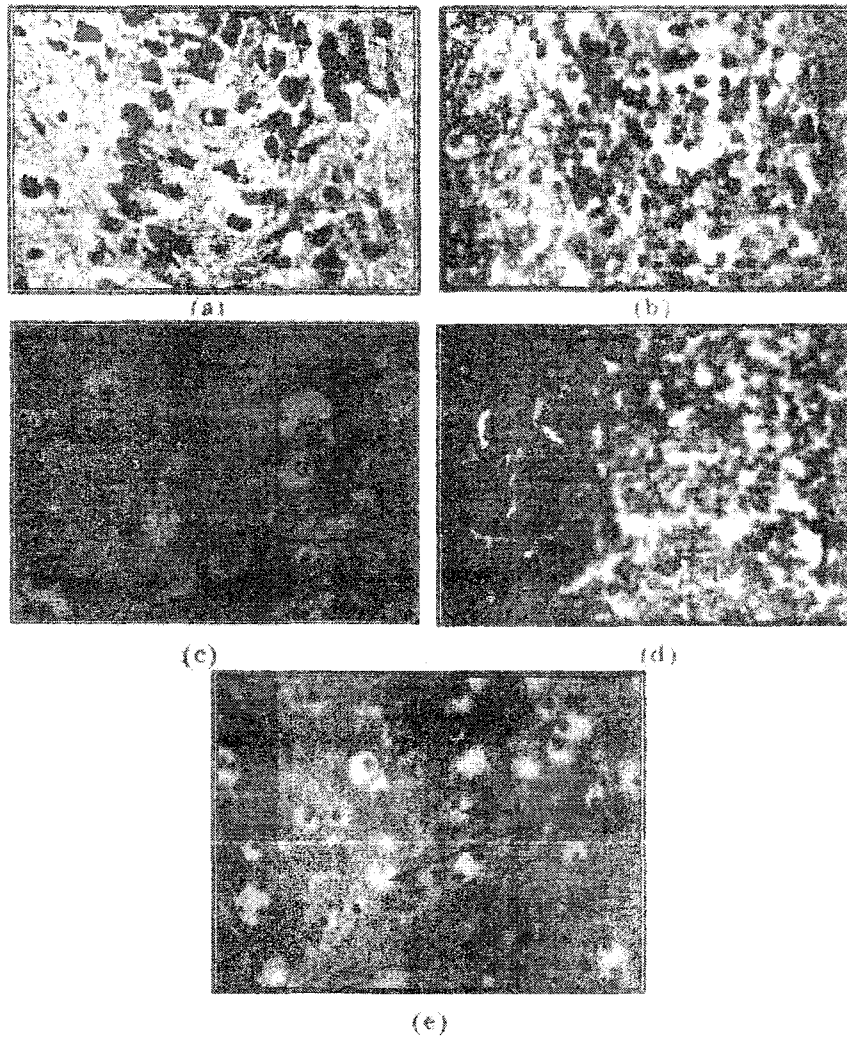


Fig. 11. Microphotographs of brain tissues: (Haematoxyline & Eosin stain  $\times 400$ );

- a: Brain of control rat, showing normal neurons with dendrites .
- b: Brain of rat exposed to Microwave source for 1 months showing focal lymphocytic cellular infiltration (LCI).
- c: Brain of rat exposed to Microwave source for 2 months showing degenerated neurons surrounded by glial cells, (Neurophagia).
- d: Brain of rat exposed to microwave source for 3months showing focal area of hemorrhage (PH).
- e: Brain of rat Recovery of 24 days Microwave source showing demyelination of some neurons (Dem).

*MT effects on rat brain histology*

Histopathological examination of one month exposed brain tissues to mobile tower field revealed blood vessels (BV) congestion, perivascular hemorrhage (PH), Fig.12a. Multiple areas of extravagation of erythrocytes in the cerebral cortex with degeneration of some neurons were also seen. After two months exposure, similar pathological changes were found, in addition to perivascular mononuclear cellular inflammation mostly lymphocytes. After 3 months exposure, the pathological brain lesions became more severe in blood vessels congestion, multiple large areas of hemorrhage, thrombosis of some blood vessels with neural degeneration. Proliferation of glial cells with neurophagia as evidenced by aggregation of some microglial cells around degenerated neurons to engulf them (Fig. 12 b) were prevalent. Moreover, demyelination of some neurons were also detected.

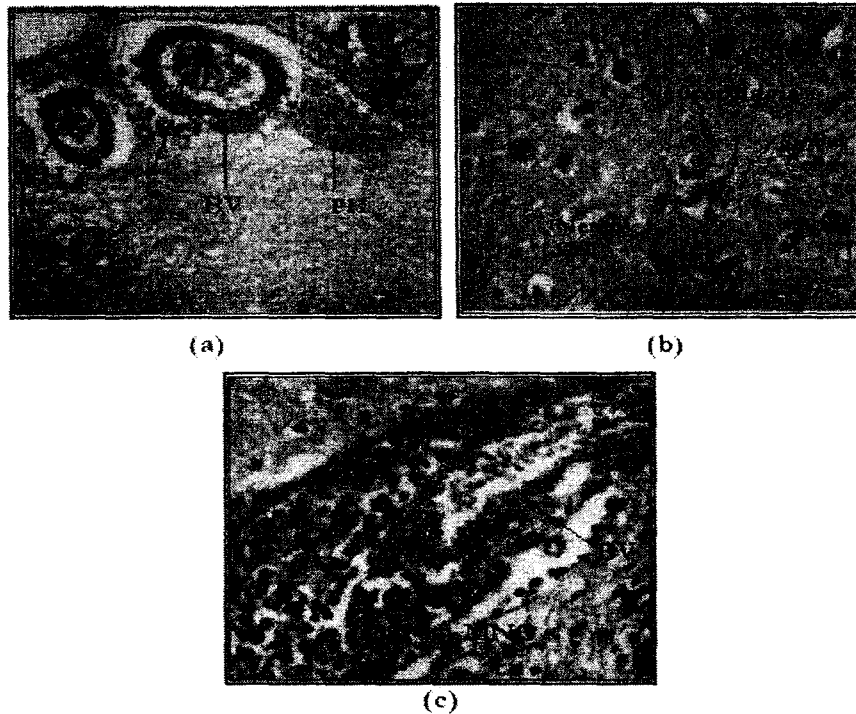


Fig. 12. microphotographs of different exposed recovered brain tissues stained with Haematoxyline & Eosin stain;

- a: Brain of rat exposed to Mobile Tower for 1month showing congestion (C) of blood vessels (B.V.) with perivascular hemorrhage, ( $\times 100$ ).  
 b: Brain of rat exposed to Mobile Tower for 3month showing Neurophagia, ( $\times 400$ ).  
 c: Brain of rat exposed to Mobile Tower after 24 days of recovery showing perivascular mononuclear inflammatory cellular aggregation (MNC), ( $\times 400$ ).

During recovery, the recorded lesions were less severe than that observed immediately after exposure. However, blood vessels congestion and perivascular mononuclear cellular aggregation mostly lymphocytes were noticed after 8 days (Fig. 12c). Later on, focal gliosis represented by aggregation of glial cells and focal area of cerebral hemorrhage was noticed after 30 days.

#### *MP effects on rat brain histology*

In case of 15 days mobile phone exposure the examined brain samples showed aggregation of few inflammatory cells and focal edema (Fig. 13). Microscopical examination of the brain samples of rats exposed to the phone for 30 & 40 days revealed nearly similar pathological changes. These changes were in the form of blood vessels congestion, perivascular hemorrhage and focal inflammatory cellular aggregation. Demyelination of some neurons and multiple areas of encephalomalacia were recognized. Moreover, degeneration of some neurons with loss in their dendrites Nissil's granules and appeared rounded in seated of satellite shape was observed.

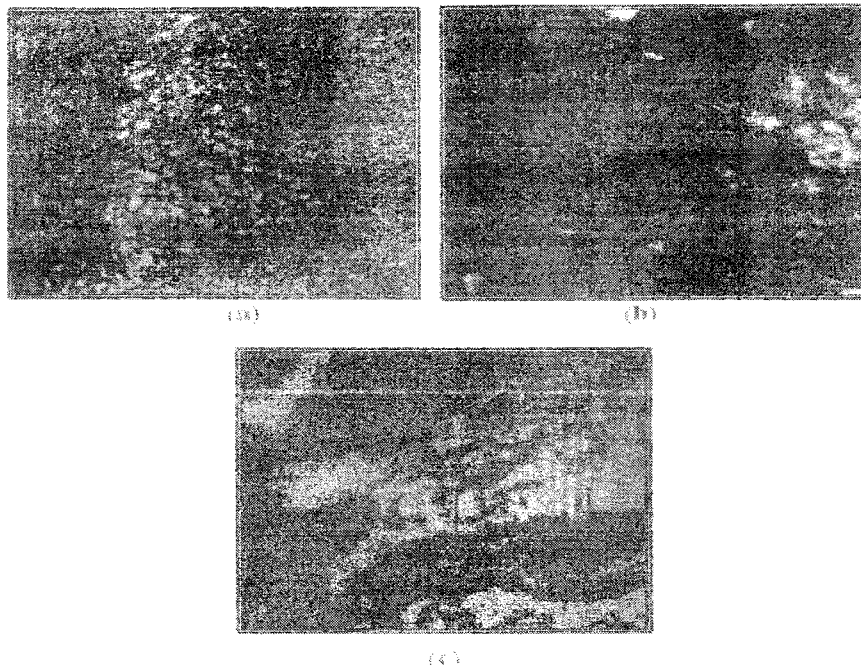


Fig. 13. Microphotographs of brain tissues; (Haematoxyline & Eosin stain);  
 a: Brain of rat exposed to Mobile Phone for 30 days showing degeneration of neurons (ND) which appeared rounded and loss their dendrites and Nissil's granules.( $\times 400$ ).  
 b: Brain of rat exposed to Mobile Phone for 1 month showing focal cerebral encephalomalacia (CE), ( $\times 100$ ).  
 c: Brain of rat after 40 days of recovery (Mobile Phone group) showing congestion of blood vessels (BVC) with perivascular inflammatory cellular infiltration (IC), ( $\times 400$ ).

During recovery, the samples showed nearly similar lesions to that noticed immediately after exposure. However, these lesions appeared severe and represented by blood vessels congestion, perivascular hemorrhage, edema and mononuclear cellular infiltration. Moreover, local area of encephalomalacia and demyelination were also observed.

### Discussion

Microwave radiation has been implicated in many diverse afflictions besetting humans and laboratory animals. The primary effect of microwave energy on living tissue was thought to be due to an increase in temperature<sup>(1)</sup> although non-thermal effects have now also been identified<sup>(13)</sup>. Stress reactions due to total body exposure<sup>(14)</sup>, physiological<sup>(15,10)</sup> biochemical changes<sup>(13)</sup> in rats after low level microwave exposure have been reported.

Transient functional changes referable to the central nervous system (CNS) have reported following "long term low level" ( $< 10 \text{ mW/cm}^2$ ) microwave exposure. Some reports describe non-thermal or "specific" MW effects at the molecular and cellular level<sup>(16)</sup>.

The results of the present investigation showed that long term-low level microwave fields alter distinct aspects of the brain's electrical activity as well as brain histology. This effect appears to be due to that MW field exposure may provide means to systematically alter the pattern ECoG activity and brain structure and function in consistent with<sup>(10,9)</sup>.

The obtained normal ECoG pattern results indicated  $\delta$ -waveforms in the frequency range of 1-4Hz similar to that known for anesthetized and sleep EEG waveforms<sup>(17)</sup>. On exposing the animal head to long term-low power microwave radiations from the MS, MT & MP, all of the recorded ECoG patterns showed a marked increase in  $\mathcal{F}$ ,  $\mathcal{A}$  &  $\mathcal{L}$  % values accompanied by a reduction in pulse regularity  $\mathcal{R}$ %. These variations in the ECoG parameters became more significant with increasing the time of microwave application, reaching their maximum variations in case of mobile phone (MP) exposure which needs a long time for recovery.

The recovery results indicated that  $\mathcal{R}$  % &  $\mathcal{L}$  % increased with recovery time but their values were still smaller than the control ones. After 40 days of stopping exposure, the amplitude  $\mathcal{A}$  was approximately approached to control value whereas  $\mathcal{F}$  showed some recovery but still smaller than control.

Histological examinations of brain tissue in all the exposed animals indicated that microwave of low intensity, low energy, and of long term exposure act as an irritant which stimulate inflammatory reaction in the tissue, represented by lymphocytic inflammation of the brain tissues, encephalo-alaein proliferation of glial cells, neural degeneration and neurophagia in addition to their effects on the

blood vessels walls leading to an increase in their permeability followed by hemorrhage. These findings are in agreement with those reported with<sup>(11,18)</sup> who showed that weak microwave radiation give rise to a significant leakage of albumin through the blood-brain barrier and this pathological leakage could be combined with damage to neurons in both the cortex, the hippocampus and the basal ganglia of exposed rats.

The observed changes of the ECoG parameters and brain histology after microwave exposure, can be attributed to some sort of non-specific stress created at low intensity microwave field and not as a result of the indirect thermal effect (as noticed by Sarkar *et al.*<sup>(19)</sup>). It is highly likely that at the power level used in these experiments, the microwave energy could not be converted into thermal energy and raise the body temperature of the treated animal significantly. The used microwave radiation could produce other effects and detectable changes can arise when the effect of the electric field, within the exposed biological system, is not masked by thermal noise that arise from the fact that all objects possess at temperature above absolute zero. So, all components of biological tissue-ions, molecules and cells- are in constant motion and the average value of their thermal energy will be  $= KT$  where  $K$  is Boltzman's constant  $= 86\mu\text{eV}/^\circ\text{C}$  and  $T$  is the absolute temperature, i.e. thermal energy  $= 26 \text{ meV}$  which is much larger than the energy produced by the field ( $\sim\mu\text{eV}$ ) and any effect of the field will be masked by the thermal noise. However, there was a special case in which the biological system was resonantly sensitive at a certain frequency and rather insensitive to others. The work of Pilla<sup>(20)</sup>, indicated the existence of a mechanism of interaction of weak electromagnetic fields with biological systems with no accompanying cell heating whereas<sup>(21)</sup> have reported that non-thermal levels of microwaves, showed resonance absorption in the frequency range of 1-12 GHz which is consistent with the range used in this work. Anderson and Joyner<sup>(22)</sup>, have measured maximum temperature rise of  $0.034^\circ\text{C}$ , for a local SAR of 0.83, in a phantom head exposed to microwave transmissions from a hand - held phone.

Lai<sup>(16)</sup> reported that low power microwave radiation induced non-thermal effects and that movement of calcium ions in brain tissue was changed. Since calcium ions control many brain and cell functions including the release and receptor function of neurotransmitters, any change in their functioning could significantly affect brain activity and health.

The correspondence between functional state of the organism and character of the ECoG recording may lead to interpretation of the functional consequences of changes in the character of spontaneous activity as a result of exposure to MW. Preece<sup>(23)</sup> and Koivisto *et al.*<sup>(24)</sup>, reported that MW at cell phone frequencies speeded the rate at which humans responded to tasks (reaction time). The higher the power from the cell phone signal, the faster the response time.



indicating the cell phone signal is not biologically neutral but can affect the brain activity. Moreover<sup>(25)</sup>, *et al.*, reported that microwave exposure from a GSM base station enhanced chemical mutagen and chromosome aberration in brain tissues.

Biological effects of the mobile phone microwave radiation were shown to depend mainly on many factors: the duration of the irradiation, individual characteristics of the CNS, and immune systems and others<sup>(9,10)</sup>. The cellular phone microwave irradiation can induce reversible unspecific adaptive responses if it is short and the organism is very radiosensitive. A long term exposure combined with the organism weakened immune system may produce a cumulative effect in the form of stress responses, various damages and in some cases, even cancer<sup>(26)</sup>. The ultimate result after microwave exposure depends on the balance between induced damage and the organism reparative ability.

Mobile phones emit a pulsed high frequency electromagnetic wave which may penetrate the scalp and the skull. In these communication systems operating within the frequency range 900-1100MHz, there is a concern that as a transceiver is brought close to the head there may be non-thermal insult produced by power deposition in tissue. This deposition can be quantified by the specific absorption rate, SAR. Guidelines have been issued<sup>(27)</sup>, expressing restrictions on exposure to high frequency electromagnetic fields of localized SAR which is defined as

$$SAR = \frac{\sigma}{\rho} |E|^2$$

Where  $\sigma$  is the electrical conductivity,  $\rho$  is the tissue density and  $E$  is the peak electric field.

Energy is preferentially absorbed in the high conductivity tissues such as the eye, the brain and muscle. The deposition from close-coupled antenna occurs mainly in the superficial structure of the head. Although bone has low conductivity resulting in low power deposition in the skull, there is some penetration into the brain through the skull<sup>(28)</sup>. Subcortical regions may contain the most sensitive structures to electromagnetic fields and this interpretation is supported by preliminary results of computer simulations of the distribution of the SAR of the electromagnetic fields in the brain<sup>(28)</sup>. An additional maximum in the SAR distribution was found in the subcortical regions indicating that exposure to microwave radiation in the mobile phone range has an effect on brain ECoG activity and so the brain physiology and histology. This effect would be commutative on repeated exposure to the radiation. Prolonged long term, low power microwave exposure leads to the conclusion about possible adverse effects on human health.

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## التأثير الوظيفي والهدمي لموجات الميكروويف على مخ السفال

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يهدف البحث إلى دراسة تأثير موجات الميكروويف على وظائف خلايا مخ الفأر. تم  
تعريض فئران تجارب بيضاء لأشعاع الميكروويف الصادر من سماعة التليفون المحمول  
وهوائي محطة إرسال التليفون المحمول وكذلك مولد موجات كهرومغناطيسية في مدى  
الميكروويف.

تم تسجيل نماذج رسم المخ من سطح القشرة المخية مباشرة بعد عمل فتحات في  
الجمجمة لمجموعة فئران ضابطة، وكذلك تم تسجيل رسم المخ لثلاث مجموعات أخرى  
من الفئران بعد تعريضها لموجات الميكروويف:

المجموعة الأولى تم عرضها على مدار الساعة أجهاز مولد موجات الميكروويف  
لمدة شهر، شهرين وثلاثة أشهر.

المجموعة الثانية كانت توضع الفئران فيها أسفل هوائي محطة إرسال المحمول فوق  
سطح المبنى على مدار الساعة لمدة شهر، شهرين وثلاثة أشهر في مكان ثابت بناء على  
توزيع الموجات وانتشارها حول البرج.

المجموعة الثالثة تم تعريض الفئران لسماعة التليفون المحمول مباشرة فترة ساعة  
ونصفا لمدة ١٥، ٣٠ و ٤٥ يوماً.

تم دراسة رسم المخ لكل من الحالات السابقة بعد انتهاء مدة التعرض وبعد انتهاء  
التعريض بفترات زمنية مختلفة لأراسة مدى العودة لنحالة الأصلية من عدمه بالإضافة  
إلى التأثير التدميري لموجات الميكروويف.

وتحليل نماذج رسم المخ تحت ظروف التعرض السابقة، تم الحصول على نتائج  
وعلاقات لتغيرات في شكل وزود وسمعة وفترة ظهور موجات رسم المخ مع تغير  
تدميري لبعض خلايا وأنسجة القشرة الصخية.