Dr. Henry Lai's Vienna Report on RFR Bioeffects
(October 25-28, 1998)

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"Since nerve cells do not divide and are not likely to become cancerous, more likely consequences of DNA damage in nerve cells are changes in functions and cell death, which could either lead to or accelerate the development of neurodegenerative diseases. Double strand breaks, if not properly repaired, are known to lead to cell death. Indeed, we have observed an increase in apoptosis (a form of cell death) in cells exposed to RFR (unpublished results). However, another type of brain cells, the glial cells, can become cancerous, resulting from DNA damage." - From this document (below)

"As regards exposure to cell mast radiation, chronic exposure becomes an important factor. Intensity and exposure duration do interact to produce an effect. We [Lai and Carino, In press] found with extremely low frequency magnetic fields that 'lower intensity, longer duration exposure' can produce the same effect as from a 'higher intensity, shorter duration exposure'." From the document. (below)

[Please note that this report is from 1998. As of 2010, there are twelve other peer-reviewed studies from around the world showing double strand DNA breaks and genome damage from exposure to low levels of RF.]

NEUROLOGICAL EFFECTS OF RADIOFREQUENCY ELECTROMAGNETIC RADIATION

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INTRODUCTION

Radiofrequency electromagnetic radiation (RFR), a form of energy between 10 KHz-300 GHz in the electromagnetic spectrum, is used in wireless communication and emitted from antennae of mobile telephones (handys) and from cellular masts. RFR can penetrate into organic tissues and be absorbed and converted into heat. One familiar application of this energy is the microwave ovens used in cooking.

The close proximity of a mobile telephone antenna to the user's head leads to the deposition of a relatively large amount of radiofrequency energy in the head. The relatively fixed position of the antenna to the head causes a repeated irradiation of a more or less fixed amount of body tissue. Exposure to RFR from mobile telephones is

of a short-term, repeated nature at a relatively high intensity, whereas exposure to RFR emitted from cell masts is of long duration but at a very low intensity. The biological and health consequences of these exposure conditions need further understanding.

Formal research on the biological effects of RFR began more than 30 years ago. In my opinion, the research has been of high quality, innovative, and intelligent. All of us who work in this field should be proud of it. However, knowledge of the possible health effects of RFR is still inadequate and inconclusive. I think the main barrier in understanding the biological effects of RFR is caused by the complex interaction of the different exposure parameters in causing an effect. An independent variable of such complexity is unprecedented in any other field of biological research.

In this paper, I have briefly summarized the results of experiments carried out in our laboratory on the effects of RFR exposure on the nervous system of the rat. But, before that, I will discuss and point out some of the general features and concerns in the study of the biological effects of RFR.

EXPOSURE CONDITIONS AND BIOLOGICAL RESPONSES

The intensity (or power intensity) of RFR in the environment is measured in units such as mW/cm2. However, the intensity provides little information on the biological consequence unless the amount of energy absorbed by the irradiated object is known. This is generally given as the specific absorption rate (SAR), which is the rate of energy absorbed by a unit mass (e.g., one kg of tissue) of the object, and usually expressed as W/kg. We may liken the intensity of RFR to a quantity of aspirin tablets. Lets say, there are 100 mg of aspirin per tablet (i.e., the intensity). This information tells us nothing about the efficacy of the tablets unless the amount taken is also known, e.g., take 2 tablets every 4 hrs (or 200 mg every 4 hrs) (analogous to the SAR). The amount of a drug absorbed into the body is the main determinant of its effect. Thus, in order to understand the effect of RFR, one should also know the SAR.

Unfortunately, RFR does not behave as simply as a drug. The rate of absorption and the distribution of RFR energy in an organism depend on many factors. These include: the dielectric composition (i.e., ability to conduct electricity) of the irradiated tissue, e.g., bones, with a lower water content, absorb less of the energy than muscles; the size of the object relative to the wavelength of the RFR (thus, the frequency); shape, geometry, and orientation of the object; and configuration of the radiation, e.g., how close is the object from the RFR source? These factors make the distribution of energy absorbed in an irradiated organism extremely complex and non-uniform, and also lead to the formation of so called 'hot spots' of concentrated energy in the tissue. For example, an experiment reported by Chou et al. [1985], measuring local energy absorption rates (SARs) in different areas of the brain in a rat exposed to RFR, has shown that two brain regions less than a millimeter apart can have more than a two-fold difference in SAR. The rat was stationary when it was exposed. The situation is more complicated if an animal is moving in an RF field. Depending on the amount of movement of the animal, the energy absorption pattern in its body could become either more complex and unpredictable or more uniform. In the latter situation, we are all familiar with the case that a microwave oven with a rotating carousel provides more uniform heating of the food than one without. However, the distribution of energy in the head of a user of a mobile telephone is more discrete because of the relatively stationary position of the phone. 'Hot spots' may form in certain areas of the head. As a reference, from theoretical calculations [e.g., Dimbylow 1993; Dimbylow and Mann 1994; Martens et al. 1995], peak (hot spot) SAR in head tissue of a user of mobile telephone can range from 2 to 8 W/kg per watt output of the device. The peak energy output of mobile telephones can range from 0.6-1 watt, although the average output could be much smaller.

Thus, in summary, the pattern of energy absorption inside an irradiated body is non-uniform, and biological responses are dependent on distribution of energy and the body part that is affected [Lai et al., 1984a, 1988]. Related to this is that we [Lai et al., 1989b] have found that different areas of the brain of the rat have different

sensitivities to RFR. This further indicates that the pattern of energy absorption could be an important determining factor of the nature of the response.

Two obviously important parameters are the frequency and intensity of RFR. Frequency is analogous to the color of a light bulb, and intensity is its wattage. There is a question of whether 'the effects of RFR of one frequency is different from those of another frequency.' The question of frequency is vital because it dictates whether existing research data on the biological effects of RFR can apply to the case of mobile telephones. Most previous research studied frequencies different from those used in wireless communication. Frequency is like the color of an object. In this case, one is basically asking the question "Are the effects of red light different from those of green light?" The answer to this is that it depends on the situation. They are different: if one is looking at a traffic light, 'red' means 'stop' and 'green' means 'go'. But, if one is going to send some information by Morse code using a light (on and off, etc.), it will not matter whether one uses a red or green light, as long as the receiver can see and decode it. We don't know which of these two cases applies to the biological effects of RFR.

It must be pointed out that data showing different frequencies producing different effects, or an effect was observed at one frequency and not at another, are sparse. An example is the study by Sanders et al [1984] who observed that RFR at frequencies of 200 and 591 MHz, but not at 2450 MHz, produced effects on energy metabolism in neural tissue. There are also several studies that showed different frequencies of RFR produced different effects [D'Andrea et al., 1979, 1980; de Lorge and Ezell, 1980; Thomas et al., 1975]. However, it is not certain whether these differences were actually due to differences in the distribution of energy absorption in the body of the exposed animal at the varous frequencies. In addition, some studies showed frequency-window effects, i.e., effect is only observed at a certain range of frequencies and not at higher or lower ranges [Bawin et al., 1975; Blackman et al., 1979, 1980a,b, 1989; Chang et al., 1982; Dutta et al., 1984, 1989, 1992; Lin-Liu and Adey, l982; Oscar and Hawkins, 1977; Sheppard et al., 1979]. These results may suggest that the frequency of an RFR can be a factor in determining the biological outcome of exposure.

On the other hand, there are more studies showing that different frequencies can produce the same effect. For example, changes in blood-brain barrier have been reported after exposure to RFRs of 915 MHz [Salford et al., 1944]; 1200 MHz [Frey et al., 1975], 1300 MHz [Oscar and Hawkin, 1977], 2450 and 2800 MHz [Albert, 1977], and effects on calcium have been reported at 50 MHz [Blackman et al., 1980b], 147 MHz [Bawin et al., 1975; Blackman et al., 1980a; Dutta et al., 1989], 450 MHz [Sheppard et al., 1979], and 915 MHz [Dutta et al., 1984]. If there is any difference in effects among different frequencies, it is a difference in quantity and not quality.

An important question regarding the biological effects of RFR is whether the effects are cumulative, i.e., after repeated exposure, will the nervous system adapt to the perturbation and, with continued exposure, when will homeostasis break down leading to irreparable damage? The question of whether an effect will cumulate over time with repeated exposure is particularly important in considering the possible health effects of mobile telephone usage, since it involves repeated exposure of short duration over a long period (years) of time. Existing results indicate changes in the response characteristics of the nervous system with repeated exposure, suggesting that the effects are not 'forgotten' after each episode of exposure. Depending on the responses studied in the experiments, several outcomes have been reported. (1) An effect was observed only after prolonged (or repeated) exposure, but not after one period of exposure [e.g., Baranski, 1972; Baranski and Edelwejn, 1974; Mitchell et al., 1977; Takashima et al., 1979]; (2) an effect disappeared after prolonged exposure suggesting habituation [e.g., Johnson et al., 1983; Lai et al., 1992a]; and (3) different effects were observed after different durations of exposure [e.g., Baranski, 1972; Dumanski and Shandala, 1974; Grin, 1974; Lai et al., 1989a; Servantie et al., 1974; Snyder, 1971]. As described in a later section, we found that a single episode of RFR exposure increases DNA damage in brain cells of the rat. Definitely, DNA damage in cells is cumulative. Related to this is that various lines of evidence suggest that responses of the central nervous system to RFR could be a stress response [Lai, 1992; Lai et al., 1987a]. Stress effects are well known to cumulate over time and involve first adaptation and then an eventual break down of homeostatic processes when the stress persists.

Another important conclusion of the research is that modulated or pulsed RFR seems to be more effective in producing an effect. They can also elicit a different effect when compared with continuous-wave radiation of the same frequency [Arber and Lin, 1985; Baranski, 1972; Frey and Feld, 1975; Frey et al., 1975; Lai et al., 1988; Oscar and Hawkins, 1977; Sanders et al., 1985]. This conclusion is important since mobile telephone radiation is modulated at low frequencies. This also raises the question of how much do low frequency electric and magnetic fields contribute to the biological effects of mobile telephone radiation. Biological effects of low frequency (< 100Hz) electric and magnetic fields are quite well established [see papers by Blackman, and Von Klitzing in this symposium].

Therefore, frequency, intensity, exposure duration, and the number of exposure episodes can affect the response to RFR, and these factors can interact with others and produce different effects. In addition, in order to understand the biological consequence of RFR exposure, one must know whether the effect is cumulative, whether compensatory responses result, and when homeostasis will break down.

EFFECTS OF VERY LOW INTENSITY RFR

For those who have questions on the possible health effects of exposure to radiation from cell masts, there are studies that show biological effects at very low intensities. The following are some examples: Kwee and Raskmark [1997] reported changes in cell proliferation (division) at SARs of 0.000021- 0.0021 W/kg; Magnras and Xenos [1997] reported a decrease in reproductive functions in mice exposed to RFR intensities of 160-1053 nW/square cm (the SAR was not calculated); Ray and Behari [1990] reported a decrease in eating and drinking behavior in rats exposed to 0.0317 W/kg; Dutta et al. [1989] reported changes in calcium metabolism in cells exposed to RFR at 0.05-0.005 W/kg; and Phillips et al. [1998] observed DNA damage at 0.024-0.0024 W/kg. Most of the above studies investigated the effect of a single episode of RFR exposure. As regards exposure to cell mast radiation, chronic exposure becomes an important factor. Intensity and exposure duration do interact to produce an effect. We [Lai and Carino, In press] found with extremely low frequency magnetic fields that 'lower intensity, longer duration exposure' can produce the same effect as from a 'higher intensity, shorter duration exposure'. A field of a certain intensity, that exerts no effect after 45 min of exposure, can elicit an effect when the exposure is prolonged to 90 min. Thus, as described earlier, the interaction of exposure parameters, the duration of exposure, whether the effect is cumulative, involvement of compensatory responses, and the time of break down of homeostasis after long-term exposure, play important roles in determining the possible health consequence of exposure to radiation emitted from cell masts.

THERMAL AND NONTHERMAL EFFECTS

When RFR is absorbed, it is converted into heat. A readily understandable mechanism of effect of RFR is tissue heating (thermal effect). Biological systems alter their functions as a result of change in temperature. However, there is also a question on whether 'nonthermal' effects can occur from RF exposure. There can be two meanings to the term 'nonthermal' effect. It could mean that an effect occurs under the condition of no apparent change in temperature in the exposed animal or tissue, suggesting that physiological or exogenous mechanisms maintain the exposed object at a constant temperature. The second meaning is that somehow RFR can cause biological effects without the involvement of heat energy (or temperature independent). This is sometime referred to as 'athermal effect'. For practical reasons, I think it is futile to make these distinctions simply because it is very difficult to rule out thermal effects in biological responses to RFR, because heat energy is inevitably released when RFR is absorbed.

In some experiments, thermal controls (i.e., samples subjected to direct heating) have been studied. Indeed, there are reports showing that 'heating controls' do not produce the same effect of RFR [D'Inzeo et al., 1988; Johnson and Guy, 1971; Seaman and Wachtel, 1978; Synder, 1971; Wachtel et al., 1975]. These were taken as an indication of non/a-thermal effects. However, as we discussed earlier, it is difficult to reproduce the same pattern of internal heating of RFR by external heating, as we know that a conventional oven cooks food

differently than a microwave oven. And pattern of energy distribution in the body is important in determining the effect of RFR [e.g., Frey et al., 1975; Lai et al., 1984a, 1988]. Thus, 'heating controls do not produce the same effect of RFR' does not really support the existence of nonthermal effects.

On the other hand, even though no apparent change in body temperature during RFR exposure occurs, it cannot really rule out a ' thermal effect'. In one of our experiments [Lai et al., 1984a], we have shown that animals exposed to a low SAR of 0.6 W/kg are actively dissipating the energy absorbed. This suggests that the brain system involved in body temperature regulation is activated. The physiology of body temperature regulation is complicated and can involve many organ systems. Thus, changes in thermoregulatory activity can indirectly affect biological responses to RFR.

Another difficulty in eliminating the contribution of thermal effects is that it can be 'micro-thermal'. An example of this is the auditory effect of pulsed RFR. We can hear RFR delivered in pulses. An explanation for this 'hearing' effect is that it is caused by thermoelastic expansion of the head of the 'listener.' In a classic paper by Chou et al. [1982], it was stated that "... one hears sound because a miniscule wave of pressure is set up within the head and is detected at the cochlea when the absorbed microwave pulse is converted to thermal energy." The threshold of hearing was determined to be approximately 10 microjoule/gm per pulse, which causes an increment of temperature in the head of one millionth of a degree centigrade! Lebovitz [1975] gives another example of a microthermal effect of RFR on the vestibulocochlear apparatus, an organ in the inner ear responsible for keeping body balance and sensing of movement. He proposed that an uneven distribution of RFR absorption in the head can set up a temperature gradient in the semicircular canals, which in turns affect the function of the vestibular system. The semicircular canals are very minute organs in our body.

What about in vitro experiments in which isolated organs or cells are exposed to RFR? Generally, these experiments are conducted with the temperature controlled by various regulatory mechanisms. However, it turns out that the energy distribution in culture disks, test tubes, and flasks used these studies are very uneven. Hotspots are formed. There is a question of whether the temperature within the exposed samples can be efficiently controlled.

In any case, my argument is not about whether a non/a-thermal effect can occur. The existence of intensity-windows, reports of modulated fields producing stronger or different effects than continuous-wave fields, and the presence of effects that occur at very low intensity described in the previous section could be indications of non/a-thermal effects. My argument is that it may not be practical to differentiate these effects experimentally due to the difficulty of eliminating thermal effects.

I propose the use of the term 'low-intensity' effects, which is based on the exposure guideline of your community. By multiplying the guideline level with the safety factor used to determine the guideline, one would get a level that supposedly causes an effect(s). Any experiment/exposure done below that level would be considered 'low-intensity'. For example, if the safety guideline is an SAR of 0.4 W/kg for whole body exposure, and a safety factor of 10 has been used to determine the guideline, then, the level at which effects should occur would be 4.0 W/kg. Any exposure below 4 W/kg would be considered a 'low-intensity' exposure. Any effect found at 'low-intensities' could conceivably contribute to the setting of future guidelines.

OUR RESEARCH ON NEUROLOGICAL EFFECTS OF RFR

When the nervous system or the brain is disturbed, e.g., by RFR, morphological, electrophysiological, and chemical changes can occur. A significant change in these functions will inevitably lead to a change in behavior. Indeed, neurological effects of RFR reported in the literature include changes in blood-brain-barrier, morphology, electrophysiology, neurotransmitter functions, cellular metabolism, calcium efflux, responses to drugs that affect the nervous system, and behavior [for a review of these effects, see Lai, 1994 and Lai et al., 1987a].

Our research on the effects of RFR exposure on the nervous system covers topics from DNA damage in brain cells to behavior. My research in this area began in 1980 when I investigated the effects of brief exposure to RFR on the actions of various drugs that act on the nervous system. We found that the actions of several drugs- amphetamine, apomorphine, morphine, barbituates, and ethyl alcohol- were affected in rats after 45 min of exposure to RFR [Lai et al., 1983; 1984 a,b]. One common feature of these responses was that they seemed to be related to the activity of a group of neurotransmitters in the brain known as the endogenous opioids [Lai et al., 1986b]. These are compounds that are generated by the brain and behave like morphine. We proposed that exposure to RFR activates endogenous opioids in the brain of the rat [Lai et al., 1984c]. One interesting finding was that RFR could inhibit morphine withdrawal in rats [1986a, which led me to speculate as to whether low-intensity RFR could be used to treat morphine withdrawal and addiction in humans. When I was in Leningrad, USSR in 1989, a scientist informed me that he had read my paper on 'RFR decreased morphine withdrawal in rats', and he had been using RFR to treat morphine withdrawal in humans. Also, unknown to us at that time was that the 'endogenous opioid hypothesis' could actually explain the increase of alcohol consumption in RFR-exposed rats that we reported in 1984 [Lai et al., 1984b]. In the summer of 1996, the United States Food and Drug Administration approved the use of the drug naloxone for the treatment of alcoholism. Naloxone is a drug that blocks the action of endogenous opioids. Increase in endogenous opioid activity in the brain can somehow cause alcohol-drinking behavior. In addition, our finding that RFR exposure alters the effect of alcohol on body temperature of the rat [Lai et al., 1984b] was replicated by Hjeresen et al. [1988, 1989] at an SAR half of what we used.

Interactions between RFR with drugs could have important implications on the health effects of RFR. They suggest that certain individuals in the population could be more susceptible to the effects of RFR. For example, an important discovery in this aspect is that ophthalmic drugs used in the treatment of glaucoma can greatly increase the damaging effects of RFR on the eye [Kues et al., 1992].

Subsequently, we carried out a series of experiments to investigate the effect of RFR exposure on neurotransmitters in the brain of the rat. The main neurotransmitter we investigated was acetylcholine, a ubiquitous chemical in the brain involved in numerous physiological and behavioral functions. We found that exposure to RFR for 45 min decreased the activity of acetylcholine in various regions of the brain of the rat, particularly in the frontal cortex and hippocampus. Further study showed that the response depends on the duration of exposure. Shorter exposure time (20 min) actually increased, rather than decreasing the activity. Different brain areas have different sensitivities to RFR with respect to cholinergic responses [Lai et al., 1987b, 1988b, 1989a,b]. In addition, repeated exposure can lead to some rather long lasting changes in the system: the number of acetylcholine receptors increase or decrease after repeated exposure to RFR to 45 min and 20 min sessions, respectively [Lai et al., 1989a]. Changes in acetycholine receptors are generally considered to be a compensatory response to repeated disturbance of acetylcholine activity in the brain. Such changes alter the response characteristic of the nervous system. Other studies have shown that endogenous opioids are also involved in the effect of RFR on acetylcholine [Lai et al., 1986b, 1991, 1992b, 1996].

At the same time, we speculated that biological responses to RFR are actually stress responses, i.e., RFR is a stressor (see Table I in Lai et al., 1987a). A series of experiments was carried out to compare the effects of RFR on brain acetylcholine with those of two known stressors: loud noise and body restraint [Lai, 1987, 1988; Lai and Carino, 1990a,b, 1992; Lai et al., 1986d, 1989c]. We found that the responses are very similar. Two other bits of information also support the notion that RFR is a stressor. We found that RFR activates the stress hormone, corticotropin releasing factor [Lai et al., 1990], and affect benzodiazepine receptors in the brain [Lai et al., 1992a]. Benzodiazepine receptors mediate the action of antianxiety drugs, such as Valium and Librium, and are known to change when an animal is stressed.

Another interesting finding is that some of the effects of RFR are classically conditionable [Lai et al., 1986b,c, 1987c]. Conditioning processes, which connect behavioral responses with events (stimuli) in the environment, are constantly modifying the behavior of an animal. In a situation known as classical conditioning, a 'neutral' stimulus that does not naturally elicit a certain response is repeatedly being presented in sequence with a

stimulus that does elicit that response. After repeated pairing, presentation of the neutral stimulus (now the conditioned stimulus) will elicit the response (now the conditioned response). You may have heard of the story of "Pavlov's dogî. A bell was rung when food was presented to a dog. After several pairing of the bell with food, ringing the bell alone could cause the dog to salivate.

We found that biological effects of RFR can be classically conditioned to cues in the exposure environment. In earlier experiments, we reported that exposure to RFR attenuated amphetamine-induced hyperthermia [Lai et al., 1983] and decreased cholinergic activity in the frontal cortex and hippocampus [Lai et al., 1987b] in the rat. In the conditioning experiments, rats were exposed to RFR in ten daily sessions (45 min per session). On day 11, animals were sham-exposed (i.e., subjected to the normal procedures of exposure but the RFR was not turned on) and either amphetamine-induced hyperthermia or cholinergic activity in the frontal cortex and hippocampus was studied immediately after exposure. In this paradigm, the RFR was the unconditioned stimulus and cues in the exposure environment were the neutral stimuli, which after repeated pairing with the unconditioned stimulus became the conditioned stimulus. Thus on the 11th day when the animals were sham-exposed, the conditioned stimulus (cues in the environment) alone would elicit a conditioned response in the animals. In the case of amphetamine-induced hyperthermia [Lai et al., 1986b], we observed a potentiation of the hyperthermia in the rats after the sham exposure. Thus, the conditioned response (potentiation) was opposite to the unconditioned response (attenuation) to RFR. This is known as 'paradoxical conditioning' and is seen in many instances of classical conditioning. We found in the same experiment that, similar to the unconditioned response, the conditioned response could be blocked by the drug naloxone, implying the involvement of endogenous opioids. In the case of RFR-induced changes in cholinergic activity in the brain, we [Lai et al., 1987c] found that conditioned effects also occurred in the brain of the rat. An increase in cholinergic activity in the hippocampus (paradoxical conditioning) and a decrease in the frontal cortex were observed after the session of sham exposure on day 11. In additon, we [Lai et al., 1984c] observed an increase in body temperature (approximately 1.0 oC) in the rat after exposure to RFR, and found that this RFR effect was also classically conditionable and involved endogenous opioids [Lai et al., 1986c].

Conditioned effects may be related to the compensatory response of an animal to the disturbance of RFR and whether it can habituate to repeated challenge of the radiation. For example, the conditioned effect on cholinergic activity in the hippocampus is opposite to that of its direct response to RFR (paradoxical conditioning), whereas that of the frontal cortex is similar to its direct response. We found that the effect of RFR on hippocampal cholinergic activity habituated after 10 sessions of exposure. On the other hand, the effect of RFR on frontal cortical cholinergic activity did not habituate after repeated exposure [Lai et al., 1987c].

Since acetylcholine in the frontal cortex and hippocampus is involved in learning and memory functions, we carried out experiments to study whether exposure to RFR affects these behavioral functions in the rat. Two types of memory functions: spatial 'working' and 'reference' memories were investigated. Acetylcholine in the brain, especially in the hippocampus, is known to play an important role in these behavioral functions.

In the first experiment, 'working' memory (short-term memory) was studied using the 'radial arm maze'. This test is very easy to understand. Just imagine you are shopping in a grocery store with a list of items to buy in your mind. After picking up the items, at the check out stand, you find that there is one chicken at the top and another one at the bottom of your shopping cart. You had forgotten that you had already picked up a chicken at the beginning of your shopping spree and picked up another one later. This is a failure in short-term memory and is actually very common in daily life and generally not considered as being pathological. A distraction or a lapse in attention can affect short-term memory. This analogy is similar to the task in the radial-arm maze experiment. The maze consists of a circular center hub with arms radiating out like the spokes of a wheel. Rats are allowed to pick up food pellets at the end of each arm of the maze. There are 12 arms in our maze, and each rat in each testing session is allowed to make 12 arm entries. Re-entering an arm is considered to be a memory deficit. The results of our experiment showed that after exposure to RFR, rats made significantly more arm re-entries than unexposed rats [Lai et al., 1994]. This is like finding two chickens, three boxes of table salt, and two bags of potatoes in your shopping cart.

In another experiment, we studied the effect of RFR exposure on 'reference' memory (long-term memory) [Wang and Lai, submitted for publication]. Performance in a water maze was investigated. In this test, a rat is required to locate a submerged platform in a circular water pool. It is released into the pool, and the time taken for it to land on the platform is recorded. Rats were trained in several sessions to learn the location of the platform. The learning rate of RFR-exposed rats was slower, but, after several learning trials, they finally caught up with the control (unexposed) rats (found the platform as fast). However, the story did not end here. After the rats had learned to locate the platform, in a last session, the platform was removed and rats were released one at a time into the pool. We observed that unexposed rats, after being released into the pool, would swim around circling the area where the platform was once located, whereas RFR-exposed rats showed more random swimming patterns. To understand this, let us consider another analogy. If I am going to sail from the west coast of the United States to Australia. I can learn to read a map and use instruments to locate my position, in latitude and longitude, etc. However, there is an apparently easier way: just keep sailing southwest. But, imagine, if I sailed and missed Australia. In the first case, if I had sailed using maps and instruments, I would keep on sailing in the area that I thought where Australia would be located hoping that I would see land. On the other hand, if I sailed by the strategy of keeping going southwest, and missed Australia, I would not know what to do. Very soon, I would find myself circumnavigating the globe. Thus, it seems that unexposed rats learned to locate the platform using cues in the environment (like using a map from memory), whereas RFR-exposed rats used a different strategy (perhaps, something called 'praxis learning', i.e., learning of a certain sequence of movements in the environment to reach a certain location. It is less flexible and does not involve cholinergic systems in the brain). Thus, RFR exposure can completely alter the behavioral strategy of an animal in finding its way in the environment.

In summary, RFR apparently can affect memory functions at least in the rat. The effects are most like reversible and transient. Does this have any relevance to health? The consequence of a behavioral deficit is situation dependent. What is significant is that the effects persist for sometime after RFR exposure. If I am reading a book and receive a call from a mobile phone, it probably will not matter if I cannot remember what I have just read. However, the consequence would be much more serious, if I am an airplane technician responsible for putting screws and nuts on airplane parts. A phone call in the middle of my work can make me forget and miss several screws. Another adverse scenario of short-term memory deficit is that a person may overdose himself on medication because he has forgotten that he has already taken the medicine.

Lastly, I like to briefly describe the experiments we carried out to investigate the effects of RFR on DNA in brain cells of the rat. We [Lai and Singh 1995, 1996; Lai et al., 1997] reported an increase in DNA single and double strand breaks, two forms of DNA damage, in brain cells of rats after exposure to RFR. DNA damages in cells could have an important implication on health because they are cumulative. Normally, DNA is capable of repairing itself efficiently. Through a homeostatic mechanism, cells maintain a delicate balance between spontaneous and induced DNA damage. DNA damage accumulates if such a balance is altered. Most cells have considerable ability to repair DNA strand breaks; for example, some cells can repair as many as 200,000 breaks in one hour. However, nerve cells have a low capability for DNA repair and DNA breaks could accumulate. Thus, the effect of RFR on DNA could conceivably be more significant on nerve cells than on other cell types of the body. Cumulative damages in DNA may in turn affect cell functions. DNA damage that accumulates in cells over a period of time may be the cause of slow onset diseases, such as cancer. One of the popular hypotheses for cancer development is that DNA damaging agents induce mutations in DNA leading to expression of certain genes and suppression of other genes resulting in uncontrolled cell growth. Thus, damage to cellular DNA or lack of its repair could be an initial event in developing a tumor. However, when too much DNA damage is accumulated over time, the cell will die. Cumulative damage in DNA in cells also has been shown during aging. Particularly, cumulative DNA damage in nerve cells of the brain has been associated with neurodegenerative diseases, such as Alzheimer's, Huntington's, and Parkinson's diseases.

Since nerve cells do not divide and are not likely to become cancerous, more likely consequences of DNA damage in nerve cells are changes in functions and cell death, which could either lead to or accelerate the

development of neurodegenerative diseases. Double strand breaks, if not properly repaired, are known to lead to cell death. Indeed, we have observed an increase in apoptosis (a form of cell death) in cells exposed to RFR (unpublished results). However, another type of brain cells, the glial cells, can become cancerous, resulting from DNA damage.

This type of response, i.e., genotoxicity at low and medium cumulative doses and cell death at higher doses, would lead to an inverted-U response function in cancer development and may explain recent reports of increase [Repacholi et al., 1997], decrease [Adey et al., 1996], and no significant effect [Adey et al., 1997] on cancer rate of animals exposed to RFR. Understandably, it is very difficult to define and judge what constitute low, medium, and high cumulative doses of RFR exposure, since the conditions of exposure are so variable and complex in real life situations.

Interestingly, RFR-induced increases in single and double strand DNA breaks in rat brain cells can be blocked by treating the rats with melatonin or the spin-trap compound N-t-butyl-a-phenylnitrone [Lai and Singh, 1997]. Since both compounds are potent free radical scavengers, this data suggest that free radicals may play a role in the genetic effect of RFR. If free radicals are involved in the RFR-induced DNA strand breaks in brain cells, results from his study could have an important implication on the health effects of RFR exposure. Involvement of free radicals in human diseases, such as cancer and atherosclerosis, has been suggested. As a consequence of increase in free radicals, various cellular and physiological processes can be affected including gene expression, release of calcium from intracellular storage sites, cell growth, and apoptosis. Effects of RFR exposure on free radicals in cells could affect these cellular functions.

Free radicals also play an important role in aging processes, which have been ascribed to be a consequence of accumulated oxidative damage to body tissues [Forster et al., 1996; Sohal and Weindruch, 1996], and involvement of free radicals in neurodegenerative diseases, such as Alzheimer's, Huntington's, and Parkinson's, has been suggested [Borlongan et al., 1996; Owen et al., 1996]. Furthermore, the effect of free radicals could depend on the nutritional status of an individual, e.g., availability of dietary antioxidants [Aruoma, 1994], consumption of alcohol [Kurose et al., 1996], and amount of food consumption [Wachsman, 1996]. Various life conditions, such as psychological stress [Haque et al., 1994] and strenuous physical exercise [Clarkson, 1995], have been shown to increase oxidative stress and enhance the effect of free radicals in the body. Thus, one can also speculate that some individuals may be more susceptible to the effects of RFR exposure.

CONCLUDING REMARKS

It is difficult to deny that RFR at low intensity can affect the nervous system. However, data available suggest a complex reaction of the nervous system to RFR. Exposure to RFR does produce various effects on the central nervous system. The response is not likely to be linear with respect to the intensity of the radiation. Other parameters of RFR exposure, such as frequency, duration, waveform, frequency- and amplitude-modulation, etc, are important determinants of biological responses and affect the shape of the dose (intensity)-response relationship. In order to understand the possible health effects of exposure to RFR from mobile telephones, one needs first to understand the effects of these different parameters and how they interact with each other.

Therefore, caution should be taken in applying the existing research results to evaluate the possible effect of exposure to RFR during mobile telephone use. It is apparent that not enough research data is available to conclude whether exposure to RFR during the normal use of mobile telephones could lead to any hazardous health effect. Research studying RFR of frequencies and waveforms similar to those emitted from cellular telephones and intermittent exposure schedule resembling the normal pattern of phone use is needed. At this point, since not much is known on the biological effects of mobile telephone use but there is indication that the radiation from the phones can cause biological effects which could lead to detrimental health effects, prudent usage should be taken as a logical guideline.

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