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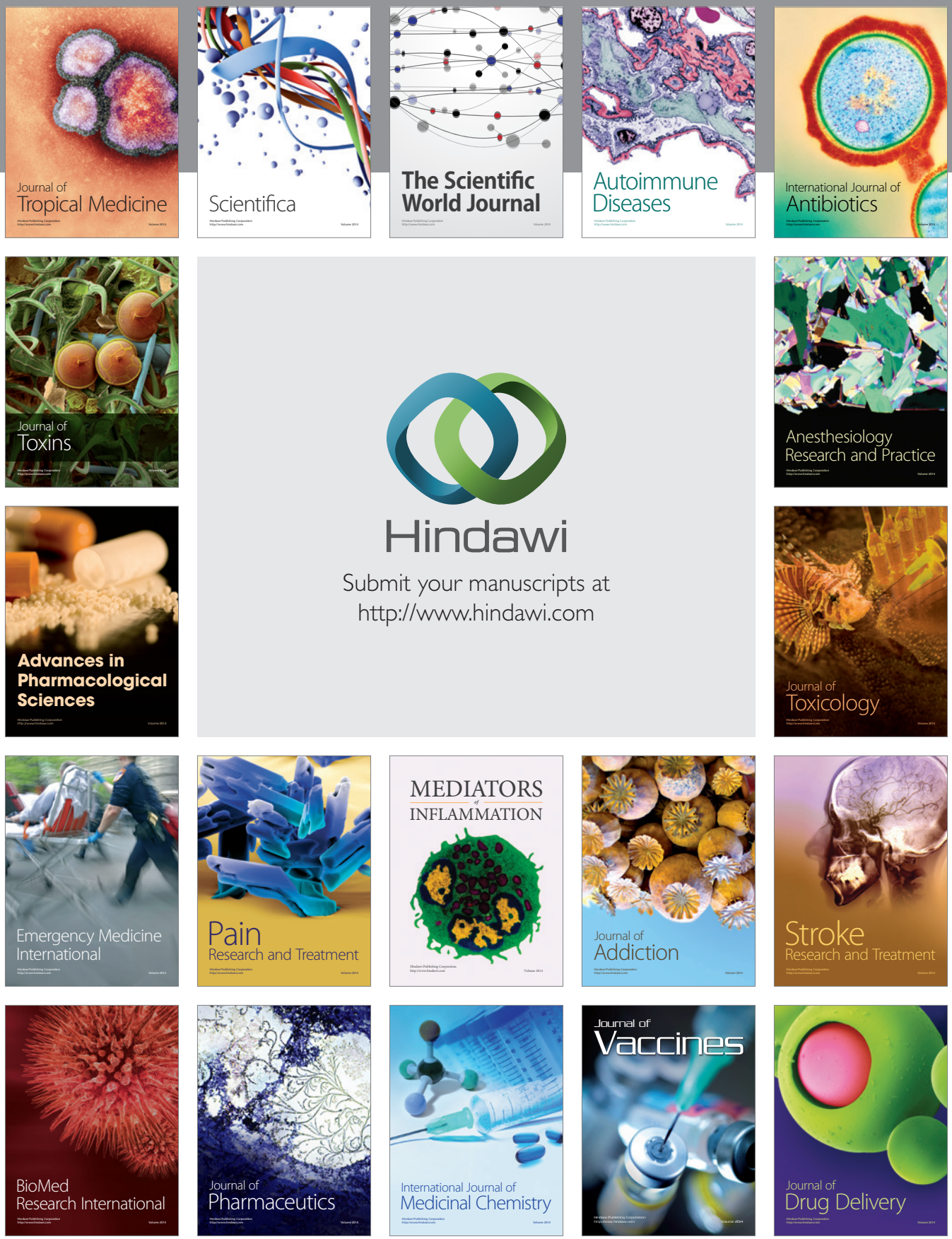
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# A Summary of Recent Literature (2007-2017) on Neurological Effects of Radiofrequency Radiation

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## Introduction

Neurological effects are caused by changes in the nervous system. Factors that act directly or indirectly on the nervous system causing morphological, chemical, or electrical changes in the nervous system can lead to neurological effects. The final manifestation of these effects can be seen as psychological/behavioral changes, e.g., memory, learning, and perception. The nervous system is an electrical organ. Thus, it should not be surprising that exposure to electromagnetic fields could lead to neurological changes. Morphological, chemical, electrical, and behavioral changes have been reported in animals and cells after exposure to nonionizing electromagnetic fields (EMF) across a range of frequencies. The consequences of physiological changes in the nervous system are very difficult to assess. We don't quite understand how the nervous system functions and reacts to external perturbations. The highly flexible nervous system could easily compensate for external disturbances. On the other hand, the consequence of neural perturbation is also situation-dependent. For example, an EMF-induced change in brain electrical activity could lead to different consequences depending on whether a person is watching TV or driving a car.

The following is a summary of the research literature on the neurological effects of exposure to radiofrequency radiation (RFR), a part of the EMF spectrum that is used in wireless communications, published between 2007- 2017. The database came from survey of the Medline and understandably does not include all the relevant papers published during the period.

### The Studies

There are many new studies on human subjects. Many of them are on changes in brain electrical activities after exposure to cell phone radiation. Bak et al (2010) (Global System for Mobile Communication (GSM) 935 MHz, 217 Hz pulses, 20 min, 0.0052 mW/cm$^2$) reported effects on event-related brain potentials. Maganioti et al. (2010) (900 MHz and 1800 MHz, 45 min) further reported that RFR affected the gender-specific components of event-related

potentials (see also Hountala et al., 2008). Croft et al (2008) (GSM 895 MHz, modulated at 217 Hz, 0.11 W/kg over 10 gm tissue, 30 min) reported changes of the alpha-wave power of electroencephalogram (EEG). They (Croft et al., 2010) further reported that effects differed between 2-G and 3-G cell phone transmission systems (2-G 894.6 MHz 217-Hz modulation, 0.7 W/kg over 10 gm tissue; 1900-MHz 3-G-modulated signal, 1.7 W/kg over 10 gm tissue; 55 min) on resting alpha activity in young adults. They observed effects after exposure to 2G but not 3G cell phone radiation, whereas Leung et al. (2011) (conditions similar to Croft et al. (2010)) found similar EEG effects (delayed ERD/ERS responses of the alpha power) with both 2G and 3G radiations. However, it is difficult to compare the 2-G and 3-G exposure conditions with different SAR and energy distributions. Ghosn et al. (2015) (GSM 900 MHz, peak specific absorption rate (SAR) 0.93 W/kg, 26 min) also reported GSM EMF affected alpha band of resting human EEG. Lustenberger et al. (2013) (900 MHz RFR pulsed with 500 msec bursts, spatial peak SAR 0.15 W/kg over 10 gm tissue) found increased slow-wave activity in humans during exposure to pulse-modulated RFR toward the end of the sleep period. Vecchio and associates reported that cell phone RFR affected EEG and the spread of neural synchronization conveyed by inter-hemispherical functional coupling of EEG rhythms (Vecchio et al., 2007) (GSM signal at 902.4 MHz, 8.33 and 217 Hz modulations, peak SAR 0.5 W/kg, 45 min) and modulated event-related desynchronization of alpha rhythms and enhanced human cortical neural efficiency (Vecchio et al., 2012a) (exposure conditions same as Vecchio et al., 2007). Naziroğlu and Gümral (2009) (2450 MHz pulsed at 217 Hz, 1.73 W/kg, 60 min/day for 28 days) reported a significant change in cortical EEG spikes in rats after chronic RFR exposure. RFR exposure modulated the spontaneous low frequency fluctuations in some brain regions (Lv et al., 2014a) (2573 MHz, spatial peak SAR 0.9 and 1.07 W/kg over 10 gm tissue, 30 min) and the synchronization patterns of EEG activation across the whole brain (Lv et al., 2014b) (exposure conditions similar to Lv et al., 2014a) in humans. An interesting finding is that RFR could interact with the activity of brain epileptic foci in epileptic patients (Tombini et al., 2013; Vecchio et al., 2012b). Roggeveen et al. (2015 a,b) (1929.1 to 1939.7 MHz, 0.69 W/kg, 15 min) reported significant changes in several bands of human EEG and detection of radiation peaks when exposed to the RFR from a 3G mobile phone. These effects were observed only when the phone was placed on the ear, and not on the heart. Yang et al. (2017) reported a reduction in spectral power in the alpha and beta bands in the frontal and temporal cortical regions of humans exposed to Long-Term Evolution (LTE) cell phone radiation. However, no significant effect on human EEG was reported by Perentos et al. (2007) (CW RFR 15 min, pulsed RFR 15 min) and Trunk et al. (2013) (1947-MHz 3G Universal Mobile Telecommunication System (UMTS), 1.75 W/kg 2 cm from surface of head model, 30 min), Trunk et al. (2014) (1947-MHz 3G UMTS signals, peak SAR 1.75 W/kg, 15 min)), and Kleinlogel et al. (2008 a, b) (1950 MHz UMTS (SAR 0.1 and 1 W/kg) and pulsed 900 MHz GSM (1 W/kg), ~30 min) also reported no significant effects on resting EEG and event-related potentials in humans after exposure to cell phone RFR. Furthermore, Krause et al. (2007) (902 MHz continuous-wave (CW) or pulsed at 217 Hz, pulse width 0.577 msec, averaged SAR 0.738 W/kg over 10 gm of tissue, peak 1.18W/kg) reported no significant effect of cell phone radiation on brain oscillatory activity, and Inomata-Terada et al. (2007) (800 MHz Time-Division Multiple Access (TDMA), 0.054 W/kg over 10 gm of tissue, 30 min) concluded that cell phone radiation does not affect the electrical activity of the motor cortex.

There are studies on the effects of cell phone radiation on EEG during sleep. Changes in sleep EEG have been reported by Hung et al. (2007) (GSM 900 MHz, SAR over 10 gm of tissue varied from < 0.001 – 0.133 W/kg depending the mode the cell phone was in, during sleep), Loughran et al. (2012) (894.6 MHz pulse-modulated at 217 Hz, peak spatial SAR 0.674 W/kg over 10 gm of tissue, 30 min prior to sleep), Lowden et al (2011) (GSM 884 MHz, spatial peak SAR 1.4W/kg, 3 hr prior to sleep), Regel et al. (2007) (pulse-modulated GSM 900 MHz signal, 0.2 or 5 W/kg, 30 min prior to sleep), and Schmid et al. (2012 a,b) (900 MHz modulated at 2 Hz, 2 W/kg). No significant effect was reported by Fritzer et al (2007) (GSM 900 with 2, 8, 217, 1733 Hz modulations, peak SAR within head 1 W/kg, during sleep), Mohler et al. (2010, 2012) (no details on exposure conditions), and Nakatani-Enomoto et al. (2013) (W-Code-Division Multiple Access (CDMA)-like signal, SAR over 10 gm tissue in the head and brain 1.52 and 0.13 W/kg, respectively, 3 hr). Loughran et al. (2012) provided an interesting conclusion in their paper: "These results confirm previous findings of mobile phone-like emissions affecting the EEG during non-rapid eye movement (REM) sleep. Importantly, this low-level effect was also shown to be sensitive to individual variability. Furthermore, this indicates that "previous negative results are not strong evidence for a lack of an effect…" More recently, Lustenberger et al. (2015) (900 MHz, 2 Hz pulse, peak spatial SAR 2 W/kg over 10 gm tissue, 30 min) reported pulsed-RFR-exposure-related increases in delta-theta EEG frequency range in several fronto-central brain areas in humans during non-REM sleep. Increase in REM sleep (Pelletier et al., 2013) (CW 900 MHz, 1 V/m, 0.0001 – 0.0003 W/kg, 5 weeks) and increases in duration and frequency of slow-wave sleep (Pelletier et al., 2014) (exposure conditions same as Pelletier et al., 2013) have been reported in developing rats after chronic RFR exposure. Mohammed et al. (2013) reported a disturbance in REM sleep EEG in the rat after long term exposure (1 hr/day for 1 month) to a 900-MHz modulated RFR.

Studies on the effects of RFR on the blood-brain barrier continued. Increase in blood-brain barrier permeability in animals after exposure to RFR was first reported in the 1970s. Such change could lead to entry of toxic substances into the brain. On the other hand, the possibility of using RFR to open up the blood-brain barrier to facilitate entry of therapeutic drugs into the brain has also be explored. In the last decade, the Salford group in Sweden continued to confirm their earlier findings on blood-brain barrier permeability and cell death in the brain (Eberhardt et al., 2008, Nittby et al., 2008a, 2009). Effects were observed after a single exposure (2 hr) to RFR at low SAR (0.00012-0.12 W/kg). In the meantime, there are several studies reporting effects of RFR on the blood-brain barrier. Sirav and Seyhan (2009, 2011) reported increased blood-brain barrier permeability in the rat after a 20-min exposure to continuous-wave 900 MHz and 1800 MHz RFR. The SARs in the 2011 study were 0.00426 W/kg for 900-MHz and 0.0014 W/kg for 1800 MHz. Interestingly, effect was observed only in male and not female rats. In a more recent study, Sirav and Seyhan (2016) studied the effects of pulse-modulated (217 Hz, 557 μs) 900-MHz and 1800-MHz RFR at 0.02 W/kg. They reported an increase in blood-brain barrier permeability in male rats after 20 min of exposure to either 900-MHz or 1800-MHz pulsed RFR, whereas an effect was found in female rats only after exposure

to the 900-MHz field. Tan et al. (2015) also reported an increase in blood-brain barrier permeability in rats after repeated exposure (14 or 28 days, 3 hr/day) to a 900 MHz field (brain SAR 2 W/kg). They suggested the involvement of the mkp-1/extracellular signal regulated kinase (ERK) for the effect. Wang LF et al. (2015), using an in vitro model, reported broadening of tight junctions in ECV304 cells and astrocytes. The authors implied the involvement of the vascular endothelial growth factor (VEGF)/Flk-1-ERK pathway in the effect. There is a related series of experiments on human subjects by Söderqvist et al. (2009 a,b,c). The authors reported a leakage of the blood-cerebrospinal fluid barrier and not the brain-brain barrier in subjects exposed to cell phone or cordless phone radiation. There are studies that reported no significant effect of RFR exposure on the blood-brain barrier. Kumlin et al. (2007) reported no neuronal cell death and significant change in the blood-brain barrier in juvenile rats after exposure to RFR (900 MHz, 0.3-3 W/kg, 2 hr/day, 5 days/week, 5 weeks). de Gannes et al. (2009) reported no significant effect on blood-brain barrier permeability and apoptosis of brain cells in rats after a 2hr-exposure to GSM 900 MHz at brain SAR of 0.14 and 20 W/kg. Finnie et al. (2009a,b) also reported no significant effects on the blood-brain barrier (based on expression of the water channel protein AQP-4 in the brain) in mice after exposure to RFR (900 MHz, 4 W/kg, 60 min or 60 min/day, 5 days/week for 104 weeks). More recently, Poulletier de Gannes et al. (2017) reported no significant changes in blood-brain barrier and neuronal degeneration in rats after a single (2 hr) or repeated (2 h/day, 5 days/week for 4 weeks) exposure to GSM-1800 and UMTS-1950 signals up to a brain-average SAR of 13 W/kg. However, an increase in albumin leakage was observed at 50 days after exposure in the brain of rats repeatedly exposed to both RF signals at 13 W/kg. Regarding “dark neurons” in the brain of rats exposed to RFR reported by Salford et al. (2003), which is apparently related to change in the blood-brain barrier. There are five reports showing an increase in dark neurons (Eberhardt et al., 2008; Kerimoğlu et al., 2016a; Köktürk et al., 2013; Jorge-Mora et al., 2013; Odaci et al., 2016), whereas de Gannes et al. (2009), Grafström et al. (2008), and Masuda et al. (2009) did not observe such an effect in the brain of RFR-exposed animals.

Related to the blood-brain barrier is a group of studies on astrocyte and microglia. These are cells in the blood-brain barrier that support the endothelial cells that form the barrier. Effects of RFR on these cells could conceivably affect the function of the blood-brain barrier. RFR-induced effects of astrocytes have been reported by Ammari et al. (2008a, 2010), Brillaud et al. (2007), Choi and Choi (2016), Liu et al. (2012), Lu et al. (2014), Maskey et al. (2010b, 2012), and Zhao et al. (2007), whereas no significant effect was reported by Bouji et al. (2012), Chen et al. (2014), Kumari et al. (2017) and Watilliaux et al. (2011). In studies on microglia, Hao et al. (2010), He et al. (2016), Lu et al. (2014) and Yang et al. (2010) reported effects of RFR exposure, whereas no significant effect was reported by Finnie et al. (2010), Hirose et al. (2010), and Watilliaux et al. (2011).

There are studies on the effects of cell phone radiation and the auditory system. Most research (Bhagat et al., 2016; Gupta et al., 2015; Kwon 2009, 2010a, b; Parazzini et al., 2009;

Stefanics et al., 2007, 2008) reported no effects, which seems to agree with the pre-2007 studies in this area. However, there are two reports by Kaprana et al. (2011) and Khullar et al (2013) showing effects on auditory brainstem response, two papers by Panda et al (2010, 2011) that concluded: "Long-term and intensive GSM and CDMA mobile phone use may cause damage to cochlea as well as the auditory cortex.", and a paper (Mandalà et al., 2014) reporting effect on auditory-evoked cochlear nerve response. Maskey and Kim (2014) reported a decrease in neurotrophins that are important in the regulation of neuron survival in the superior olivary complex, a neural component of the auditory system, in mice after chronic exposure to RFR. Velayutham et al. (2014) reported hearing loss in cell phone users and Sudan et al. (2013) observed weak associations between cell phone use and hearing loss in children at age 7. These effects may not be caused by the radiation. However, there is a study (Seckin et al., 2014) showing structural damage in the cochlea of the rat after prenatal exposure to RFR. And, Ozgur et al. (2015) reported neuronal degeneration in the cochlear nucleus of the auditory system in the rat after chronic exposure to RFR. Kwon et al. (2010a) reported that short-term exposure to cell phone radiation did not significantly affect the transmission of sensory stimuli from the cochlea to the midbrain along the auditory nerve and brainstem auditory pathways, and (Kwon et al., 2010b) no significant effect on auditory sensory memory in children. More recently, Çeliker et al. (2017) also reported no significant change in auditory brainstem responses, but increases in neuronal degeneration and apoptosis in the cochlear nucleus in rats exposed to a 2100-MHz field for 30 days.

There are several studies that showed neurological changes in humans after use of wireless devices, but those changes apparently were not caused by exposure to the radiation. Abramson et al. (2009) reported changes in cognitive functions in young adolescents. ("The accuracy of working memory was poorer, reaction time for a simple learning task shorter, associative learning response time shorter and accuracy poorer in children reporting more mobile phone voice calls"). Arns et al. (2007) observed more focused attention in frequent cell phone users, which was probably a "cognitive training effect". Yuan et al. (2011) reported morphological changes in the brain of adolescents with "internet addiction disorder".

There are several studies showing differential effects of different waveforms. This is an important consideration in understanding how EMF interacts with living organisms. Croft et al. (2010) reported that 2G, but not 3G, cell phone radiation affected resting EEG. Hung et al. (2007) showed that 2, 8, 217 Hz-modulated RFR differentially affected sleep. Lopez-Martin et al. (2009) reported that modulated and non-modulated RFR had different effects on gene expression in the brain. Nylund et al. (2010) found that different carrier-frequencies (900 MHz verses 1800 MHz) had different effects on protein expression. Schmid et al. (2012a) concluded that "modulation frequency components (of a RFR) within a physiological range may be sufficient to induce changes in sleep EEG". Mohammed et al. (2013) reported that EEG power spectrum during REM sleep is more susceptible to modulated RFR than the slow-wave sleep (SWS). Schneider and Stangassinger (2014) reported different effects of 900-MHz and 1.966-GHz EMFs on social memory functions in the rat. Zhang et al. (2008) reported that an intermittent exposure to RFR had a more potent effect on gene expression in the brain than continuous exposure. Apparently, extremely-low frequency (ELF)-modulation plays a role on

determining the biological effects of RFR. One can find many studies showing the same neurological effects of RFR described above in animals exposed to extremely-low frequency electromagnetic field (ELF EMF) e.g., Carrubba et al., 2007, 2010; Cook et al., 2009; Cui et al., 2012; Perentos et al., 2008. This is of considerable importance, since all cell phone signals are modulated by low frequency components. Furthermore, effects can also depend on the modulation frequency. Bawin et al. (1975) reported an increase in efflux of calcium ions from chick brain tissue after 20 min of exposure to a 147-MHz RFR (1 to 2 $mW/cm^2$). The effect occurred when the radiation was sinusoidally amplitude-modulated at 6, 9, 11, 16, or 20 Hz, but not at modulation frequencies of 0, 0.5, 3, 25, or 35 Hz. Blackman et al. (1979) also reported a "modulation-frequency window" in RFR-induced calcium ion efflux from brain tissue.

On the neurological effects of RFR, there are many papers published in the last decade indicating that oxidative stress played a role in the effects observed: Akbari et al., 2014; Bodera et al., 2015; Cetin et al., 2014; Dasdag et al., 2009, 2012; Del Vecchio et al., 2009a,b; Deshmukh et al., 2013a; Dragicevic et al., 2011; Eser et al., 2013; Gao et al., 2013; Ghazizadeh and Naziroglu, 2014; Hidisoglu et al., 2016; Hu S. et al., 2014; Hu et al., 2016; İkinci et al., 2016; Imge et al., 2010; Jing et al., 2012; Kerimoğlu et al., 2016a, b; Kesari et al., 2011; Kim JY et al., 2017; Liu et al., 2011; Maaroufi et al., 2014; Megha et al., 2012; Meral et al., 2007; Motawi et al., 2014; Narayanan et al., 2014; Nazıroğlu and Gümral, 2009; Nazıroğlu et al., 2012; Nirwane et al., 2016; Othman et al., 2017; Qin et al., 2014; Saikhedkar et al., 2014; Sharma et al., 2017; Shehu et al., 2016; Sokolovic et al., 2008; Varghese et al., 2017; Xu et al., 2010; Yang et al., 2010. (Dragicevic et al. (2011) reported a decrease in mitochondrial free radical production in the hippocampus and cerebral cortex of the mouse after RFR exposure.) There was one study (Poulletier de Gannes et al, 2011) that found no significant oxidative stress in brain cells after exposure to Enhanced Data rate for GSM Evolution (EDGE) signal. Kang et al (2014) reported that "neither combined RF radiation alone nor combined RF radiation with menadione or $H_2O_2$ influences the intracellular reactive oxygen species (ROS) level in neuronal cells." The mediating roles of cellular free radicals and oxidative status on the biological effects of EMF are worth looking into. Interestingly, there is a study (Cao et al., 2015) showing that RFR interacts with circadian rhythmicity on antioxidative processes in the rat.

An important issue that has been extensively debated in the media is whether children are more vulnerable to the effect of cell phone radiation than adults? The claim that children have thinner skulls and thus absorb more energy is not valid. And the claim that a child's head absorbs more energy from a cell phone is also debatable. It is quite possible that the pattern of energy distribution of cell phone energy absorption in the head is significantly different between a child and an adult (cf. Christ and Kuster, 2005; Christ et al. 2010; Gandhi et al. 2012). Scientific data on whether a child is biologically more vulnerable to cell phone radiation is sparse. There are several studies that indicate that animals (including humans) of different ages respond differently to cell phone radiation. Bouji et al. (2012) reported differences in neuro-immunity, stress, and behavioral responses to GSM signals between 'young adult' (6 weeks-old) and 'middle age' (12-month old) rats. Croft et al. (2010) showed that GSM signals affected certain electrical activities of the brain in young human adults (19-40 years old) but not in adolescents (13-15 years old) or elderly (55-70 years old) subjects. Leung et al. (2011)

reported that performance in a cognitive test was affected by GSM signal in adolescents but not in young or old human subjects. Noor et al. (2011) reported differences in neurochemical responses to 900-MHz RFR between adult and young rats. And, Vecchio et al. (2010) found differences in brain electric activities between young and elderly human subjects responding to GSM signals. It must be pointed out that although these studies reported an age-dependent effect of cell phone radiation, they do not necessarily imply that children are more vulnerable to cell phone radiation than adults. There are several papers showing effects of exposure to RFR during perinatal periods on the development and functions of the nervous system (Aldad et al., 2012; Bas et al., 2013; Cetin et al., 2014; Daniels et al. 2009; Divan et al., 2008, 2011, 2012; Erdem Koç et al., 2016; Gao et al., 2013; Haghani et al., 2013; İkinci et al., 2013; Jing et al., 2012; Kokturk et al., 2013; Lee and Yang, 2014; Odaci et al., 2008, 2013, 2016; Othman et al., 2017; Ragbetli e al., 2010; Razavinasab et al., 2016; Zareen et a., 2009; Zhang et al., 2015). These studies point to the vulnerability of the development nervous system to RFR. The cerebellum seems to be a structure especially vulnerable to the exposure (Eser et al. 2013; Haghani et al., 2013; Kokturk et al., 2013; Odaci et al., 2016; Ragbetli e al., 2010). Chen et al. (2014) reported that exposure to an1800-MHz RFR impaired neurite outgrowth of embryonic neural stem cells, which play a critical role in brain development. More recently, Xu et al. (2017) reported that effect of exposure to an 1800-MHz field on stem and progenitor cell proliferation in the hippocampus of mouse depended on the age of the animal. Stem cells play an important role in embryonic development. And, it turns out that they are very sensitive to electric current, particularly in their migration in the body during organogenesis. It has been suggested that electric current can be used as a guidance of migration of stem cells for the treatment of neurodegenerative diseases (Feng et al., 2017). On the other hand, disturbance of stem cells by induced electric currents of electromagnetic fields can cause defects in pre- and postnatal development. This can occur at low intensities of the field. Indeed, there are reports on effects of extremely-low frequency (ELF) magnetic and electric fields on stem cells (Bai et al., 2013; Choi et al., 2014; Cho et al., 2012; Kim et al., 2013; Takahashi et al., 2017). ELF EMF is more effective in generating induced electric currents.

With these physiological changes in the brain, what behavioral effects have been reported? Data are summarized in the tables below.

**Table 1. Behavioral Effects of Radiofrequency Radiation**

**Human studies that showed behavioral effects:**

| | **Behavior studied/Results** | **Experimental conditions** |
|---|---|---|
| Danker-Hopfe et al. (2015) | Sleep of individuals affected differently- showing both improvements and deteriorations. | GSM 900 MHz and Wideband Code Division Multiple Access (WCDMA)/UMTS, during sleep |
| de Tommaso et al. (2009) | Reduction in behavioral | GSM 900 MHz, 10 min |

| | | |
|---|---|---|
| | arousal | |
| Deniz et al. (2017) | Poorer attention in high exposure group | Low (<30 min/day) vs high (>90 min/day) cell phone radiation exposure |
| Hung et al. (2007) | Sleep latency | GSM 900 MHz with 2, 8, or 217-Hz modulations, 30 min |
| Leung et al. (2011) | Cognitive functions | 2G and 3G cell phone radiation, 10 min |
| Luria et al. (2009) | Spatial working memory (In a subsequent study (Hareuveny et al., 2011), the authors indicated that some of the effects observed may not be related to RFR exposure.) | GSM phone, 60 min |
| Lustenberger et al. (2013) | Sleep-dependent motor-task performance improvement | 0.25-0.8 Hz pulsed 900 MHz RFR, all-night |
| Mortazavi et al. (2012) | Decreased reaction time | Cell phone radiation, 10 min |
| Mortazavi et al. (2013) | Decreased reaction time; poorer short-term memory performance | Occupational exposure to military radar radiation |
| Movvahedi et al. (2014) | Better short-term memory in elementary school students | Cell phone radiation, 10 min |
| Redmayne et al. (2013) | Well-being | Use of cellphone and cordless phone |
| Regel et al. (2007) | Cognitive functions | pulse-modulated GSM 900 MHz signal, 0.2 or 5 W/kg, 30 min |
| Schoeni et al. (2015) | A change in memory performance | Based on cumulative duration of wireless phone use and RF-EMF dose over one year (GSM and UMTS) |
| Thomas et al. (2010) | Overall behavioral problems in adolescents | RFR measured by a personal dosimeter over 24 hr |
| Vecchio et al. (2012a) | Better performance in a | GSM signal at 902.4 MHz, 8.33 |

| | | |
|---|---|---|
| | cognitive- motor test | and 217 Hz modulations, peak SAR 0.5 W/kg, 45 min |
| Vecchio et al. (2012b) | Enhanced cognitive-motor processes in epileptic patients | GSM phone radiation, 45 min |
| Vecsei et al. (2013) | Decreased thermal pain perception | UMTS phone-like radiation, 1.75 W/kg, 30 min |
| Wiholm et al. (2009) | 'Virtual' spatial navigation task | 884 MHz, peak head SAR 1.4 W/kg, 150 min |
| Yogesh et al. (2014) | Sleep disturbance, latency and day dysfunction especially in females | > 2 hr/day of mobile phone use |
| Zheng et al. (2014) | Inattention in adolescents | Use of cell phone >60 min per day |

**Human studies that showed no significant behavioral effects:**

| | **Behavior studied** | **Experimental conditions** |
|---|---|---|
| Calvente et al. (2016) | No definite conclusion can be drawn on cognitive and behavioral functions of 10-year old boys | Environmental RFR 100 kHz to 6 GHz; root mean square 0.286 $mW/cm^2$; maximum power density 2.76 $mW/cm^2$ |
| Cinel et al. (2007) | Order threshold task | GSM or unmodulated carrier frequency wave to head, 40 min |
| Cinel et al. (2008) | Subjective symptoms | GSM or unmodulated carrier frequency wave to head, 40 min |
| Curcio et al. (2008) | Reaction time task, sequential figure tapping task | GSM (902.4 MHz, 217 Hz modulation, 0.5 W/kg), 3 x 15 min |
| Curcio et al. (2009) | objective and subjective vigilance | GSM (902.4 MHz, 8.33 Hz and 217-Hz modulation, 0.5 W/kg), 40 min |
| Curcio et al. (2012) | Somatosensory task | GSM (902.4 MHz, 8.33 Hz and 217-Hz modulation, 0.5 |

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| | | W/kg), 40 min |
| Danker-Hopfe et al. (2011) | Effect on sleep | GSM 900 or WCDMA/UMTS, during sleep |
| Eltiti et al. (2009) | Cognitive functions | GSM 900 or UMTS, 0.001 $mW/cm^2$, 50 min |
| Fritzer et al. (2007) | Sleep and cognitive functions | GSM900 with 2, 8, 217, 1733 Hz modulations, peak SAR within head 1 W/kg, during sleep |
| Haarala et al. (2007) | Cognitive functions | 902 MHz, continuous-wave or pulsed (27 Hz, 0.577 ms), head peak SAR 1.18 W/kg, 90 min |
| Irlenbusch et al. (2007) | Visual discrimination threshold | GSM 902.4 MHz 217 Hz pulses, 0.1 $mW/cm^2$, 30 min |
| Kleinlogel et al. (2008a) | Well being | 1950 MHz UMTS (0.1 and 1 W/kg) or 900 MHz GSM (1 W/kg), 30 min |
| Kleinlogel et al. (2008b) | Continuous performance test measuring reaction time and false reaction | 1950 MHz UMTS (0.1 and 1 W/kg) or 900 MHz GSM (1 W/kg), exposed during measurements |
| Krause et al. (2007) | Auditory memory task | 902 MHz CW or pulsed at 217 Hz, pulse width 0.577 msec, averaged SAR 0.738 W/kg over 10 gm tissue, peak 1.18 W/kg |
| Kwon et al. (2010b) | Auditory sensory memory in children | GSM 902 MHz pulsed at 217 Hz, temporal lobe peak SAR 1.21 W/kg, average 0.82 W/kg over 10 gm tissue |
| Loughran et al. (2013) | Cognitive effects and EEG in 11-13 years old adolescences | Modulated GSM900 (peak SAR 1.4 W/kg or 0.35 W/kg), 30-60 min |
| Malek et al. (2015) | Cognitive functions in | Pulse-modulated GSM (945 MHZ and 1840 MHz, 28 |

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| | sensitive humans | mW/cm$^2$) and UMTS (2140 MHz, 38 mW/cm$^2$), 1 V/m, whole body exposure, Short-term |
| Mohler et al. (2010, 2012) | Effect on sleep | Environmental far-field RFR and cell and cordless phone radiation |
| Nakatani-Enomoto et al. (2013) | Effect on sleep | W-CDMA, 3 hr |
| Redmayne et al. (2016) | Cognitive functions in 8-11 years old children | Use of cellular and cordless phone |
| Riddervold et al. (2008) | Trail making B test | 2140 MHz continuous-wave and 2140 MHz modulated as UMTS, 45 min |
| Roser et al. (2016) | No change behavioral problem and concentration capacity | Self-reported and operator-recorded wireless communication device use |
| Sauter et al. (2011) | Cognitive functions | GSM900 and WCDMA, 7 hr 15 min in two episodes |
| Sauter et al. (2015) | Cognitive functions and well-being | Terrestrial Trunked Radio (TETRA) (385 MHz) signals, 2.5 hr |
| Schmid et al. (2012a) | Cognitive functions | 900 MHz pulse modulated at 14 and 217 Hz, peak spatial SAR 2 W/kg, 30 min |
| Schmid et al. (2012b) | Cognitive functions | 900 MHz pulse modulated at 2 Hz, 2 W/kg, 30 min |
| Trunk et al. (2013) | Automatic deviance detection processes | 1947-MHz 3G UMTS, 1.75 W/kg 2 cm from surface of head model, 30 min |
| Trunk et al. (2014) | Reaction time to a stimulus | 1947-MHz 3G UMTS signals, peak SAR 1.75 W/kg, 15 min |
| Trunk et al. (2015) | Reaction time to a visual target detection task | 1947-MHz UMTS signals, peak SAR 1.75 W/kg, 15 min |

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| Unterlechner et al. (2008) | attention | UMTS signals, peak SAR 0.63 W/kg at cortex of temporal lobe, 90 min |
| Wallace et al. (2012) | Cognitive functions | 420 MHz TETRA, 0.001 $mW/cm^2$, 10- 50 min, whole body exposure |

**Animal studies that showed behavioral effects:**

| | **Behavior studied/results** | **Experimental conditions** |
|---|---|---|
| Aldad et al. (2012) | Hyperactive, impaired memory (mouse) | 800 and 1900 MHz cell phone radiation, gestation days 1-17 (24 hr/day), tested at 8, 12, and 16 weeks old |
| Arendash et al. (2010, 2012) | Improved cognitive behavior in mouse model of Alzheimer's disease | 918 MHz pulse modulated at 217 Hz, 0.25-1.05 W/kg, 2-6 months or 12 days, 2 hr/day |
| Banaceur et al. (2013) | Improved cognitive functions in mouse model of Alzheimer's disease | 2409 MHz, 1.6 W/kg, 2 hr/day for a month |
| Barthélémy et al. (2016) | Memory, emotionality, and locomotion in plus maze and open field (rat) | 900 MHz modulated at 217 MHz, 15 min (1.5 or 6 W/kg) or 45 min (6 W/kg) |
| Bouji et al. (2012) | Contextual emotional behavior deficit (rat) (age-depended effect observed) | 900 MHz, 6 W/kg, 15 min |
| Cammaerts et al. (2012) | Olfactory and/or visual memory deficit in ants | GSM900 MHz (GSMK modulated), 0.77 V/m, in several periods 1.5-6 days |
| Cammaerts et al. (2013) | Deterioration of food collection behavior in ants | GSM900 MHz (GSMK modulated), 0.77 V/m, 180 hr |
| Cammaerts et al. (2014) | Changes in locomotor and general behaviors in ants | 940 MHz pulse-modulated 577 μs width, 0.5-1.5 V/m, 10 min exposure before |

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| | | behavioral observation |
| Choi and Choi (2016) | Delayed hyperactivity-like behavior (mouse) | Smart phone, 10 min/day, 9-11 weeks |
| Daniels et al. (2009) | Decreased motor activity and increased grooming (rat) | 840 MHz, 6 x $10^{-6}$ mW/$cm^2$, pups exposed 3hr/day from postnatal day 2 to day14, tested at postnatal day 58 |
| Deshmukh et al. (2013a) | Impaired cognitive functions (plus maze and water maze) (rat) | 900 MHz, 8.47 x $10^{-5}$ W/kg, 2 hr/day, 30 days |
| Deshmukh et al. (2015) | Impaired cognitive functions (plus maze and water maze) (rat) | 900 MHz (5.953 x $10^{-4}$ W/kg), 1800 MHz (5.835 x $10^{-4}$ W/kg), 2450 MHz (6.672 x $10^{-4}$ W/kg), 2 hr/day, 180 days |
| Deshmukh et al. (2016) | Impaired cognitive functions (plus maze and water maze) (rat) | 900 MHz (5.953 x $10^{-4}$ W/kg), 1800 MHz (5.835 x $10^{-4}$ W/kg), 2450 MHz (6.672 x $10^{-4}$ W/kg), 2 hr/day, 90 days |
| Favre (2011) | Induced piping behavior in honeybee workers | Cell phone put close to bee hive |
| Fragopoulou et al. (2010) | Spatial memory deficit (mouse) | GSM 900 MHz, 0.41-0.98 W/kg, 2 hr/day, 4 days |
| Hao et al. (2013) | Learning and memory deficit (rat) | 916 MHz, 1 mW/$cm^2$, 6 hr/day, 5 days/week, 10 weeks |
| Hassanshahi et al. (2017) | Impaired object recognition (rat) | 2400 MHz, 12 hr/day, 30 days |
| Hu et al. (2014) | Spatial memory deficit (rat) | High power microwave, 30 mW/$cm^2$, average brain SAR 21 W/kg, 15 min/day, 14 days |
| İkinci et al. (2013) | Learning and memory deficit (rat) | 900 MHz, 13$^{th}$ to 21$^{st}$ day of pregnancy, 1 hr/day, offspring tested at 26 days old |

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| Júnior et al. (2014) | Observed stress behavioral patterns (rat) | GSM 180 MHz, 2 V/m, 25 sec every 2 min for 3 days |
| Kim JH et al. (2017a) | Hyperactivity-like behavior (mouse) | 835 MHz, 4 W/kg, 5 hr/day for 12 weeks |
| Kumar et al. (2009) | Hypoactivity, anxiety behavior (rat) | GSM 900 MHz and 1800 MHz, 50 missed call/day, 4 weeks |
| Kumari et al. (2017) | Spatial learning deficit and impairment of memory measured by passive avoidance test (mouse) | 7.5 KHz magnetic field, 12 or 120 μT, 5 weeks |
| Kumlin et al. (2007) | Improved spatial learning and memory (rat) | 90 MHz, 0.3 or 3 W/kg, 2 hr/day, 5 days/week, 5 weeks |
| Lee et al. (2015) | Locomotor activity after feeding (fish Poecilia reticulata and Danio rerio) | RFR from an 1800 MHz cell phone |
| Li et al. (2015) | Spatial learning and memory deficits (rat) | 2.856 MHz 5, 10, 20, or 30 $mW/cm^2$, 6 min 3 times a week up to 6 weeks |
| Li et al. (2012) | Spatial learning and memory deficits (rat) | GSM 900 phone, 2 hr/day for 1 month, 0.52-1.08 W/kg |
| Lu et al. (2012) | Spatial memory deficit (rat) | 2450 MHz pulsed, 1 $mW/cm^2$, 3 hr/day, 30 days |
| Maaroufi et al. (2014) | Spatial learning and memory deficit (rat) | 900 MHz, 0.05-0.18 $W/cm^2$, 1 hr/day, 21 days |
| Mathur (2008) | Analgesic effect (rat) | 73.5 MHz, amplitude-modulated at 16 Hz, 0.4 W/kg, 2 hr/day, 45 days |
| Megha et al. (2012) | Cognitive functions (plus maze and water maze) (rat) | 900 MHz ($5.953 \times 10^{-4}$ W/kg) or 1800 MHz ($5.845 \times 10^{-4}$ W/kg), 2 hr/day, 30 days |
| Mohammed et al. (2013) | Increased latency of REM sleep (rat) | 900 MHz continuous-wave, 900 MHz modulated at 8 and 16 Hz, spatial peak SAR 0.245 |

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| | | W/kg, 1 hr/day for 1 month |
| Narayanan et al. (2009) | Spatial learning and memory deficit (rat) | GSM 900/1800 MHz, 50 missed call/day, 4 weeks |
| Narayanan et al. (2010) | Passive avoidance deficit (rat) | GSM 900/1800 MHz, 50 missed call/day, 4 weeks |
| Narayanan et al. (2013) | Elevated plus maze-emotionality test deficit (rat) | GSM 900 MHz phone, peak power density 0.1466 $mW/cm^2$, 1 hr/day for 28 days |
| Narayanan et al. (2015) | Spatial memory deficit (rat) | GSH 90 MHz phone, peak power density 0.1466 $mW/cm^2$, 1.15 W/kg, 1 hr/day for 28 days |
| Nirwane et al. (2016) | Change in social behavior, anxiety behavior, learning impairment (zebrafish) | GSM 900 MHz phone, 1.34 W/kg, 1 hr/day for 14 days |
| Nittby et al. (2008b) | Reduced memory functions (rat) | GSM 900 MHz, 0.0006 and 0.06 W/kg, 2 hr/week, 55 weeks |
| Ntzouni et al. (2011) | Non-spatial memory deficit (mouse) | GSM 1800-MHz phone, 022 W/kg, 90 min/day, 17 days |
| Ntzouni et al. (2013) | Spatial and non-spatial memory deficit (mouse) | GSM 1800-MHz phone, 011 W/kg, 90 min/day, 66-148 days |
| Odacı et al. (2013) | Motor function (rat) | 900 MHz, 10 V/m, exposed 1 hr/day from day 13 to day 21 of pregnancy, offspring tested at 21 days of age |
| Othman et al. (2017) | Anxiety and deficits in neuromotor maturation mainly in male offspring (rat) | 2450 MHz, 2 hr/day from conception to parturition, offspring tested at 28, 30 and 31 days of age |
| Pelletier et al. (2013) | Food intake increase; changes in sleep parameters; increased food intake (rat) | 900 MHz, 1 V/m, 0.3-0.1 W/kg depending on age, 23.5 hr/day, 5 weeks |

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| Pelletier et al. (2014) | Preferred to sleep in a different temperature environment than controls; sleep parameters (rat) | 900 MHz, 1 V/m, 0.3-0.1 W/kg depending on age, 23.5 hr/day, 5 weeks |
| Qiao et al. (2014) | Spatial memory deficit (rat) | 2856 MHz, 30 mW/cm$^2$, 14 W/kg, 5 min |
| Qin et al. (2014) | Learning and memory deficits (mouse) | 1800 MHz, 0.208 mW/cm$^2$, 2 hr/day, 30 days |
| Razavinasab et al. (2016) | Passive avoidance and spatial learning and memory deficits (rat) | 900 MHz pulsed RFR, 0.3-0.9 W/kg, 6hr/day from conception to birth, tested at 30 days of age |
| Saikhedkar et al. (2014) | Learning and memory deficits (rat) | 900 MHz phone, 0.9 W/kg, 4 hr/day, 15 days |
| Sarapultseva et al. (2014) | Motor activity (protozoa Spirostomum ambiguum) | 1000 MHz 0r 10,000 MHz, 0.005-0.05 mW/cm$^2$, 0.05-10 hr |
| Schneider and Stangassinger (2014) | Social memory effect (rat) | GSM 900 MHz and UMTS 1966 MHz, 0.4 Wkg, up to 6 months |
| Sharma et al. (2014) | Spatial learning memory deficit (mouse) | 10,000 MHz, 0.25 mW/cm$^2$, 0.179 W/kg, 2 hr/day, 30 days |
| Sharma et al. (2017) | Spatial learning and memory deficit (mouse) | 10,000 MHz, 0.25 mW/cm$^2$, 0.179 W/kg, 2 hr/day, 15 days |
| Shehu et al. (2016) | Anxiety-like behavior (rat) | GSM 900/1800 phones, 10 min call per day for 4 weeks |
| Sokolovic et al. (2012) | Anxiety-related behavior (rat) | GSM900 phone, 9.88-13.356 V/m, 0.43-0.135 W/kg, 4 hr/day for 20, 40, 60 days |
| Tang et al. (2015) | Spatial long-term memory deficit (rat) | 900 MHz, 1 mW/cm$^2$, 0.016 W/kg, 3 hr/day for 14-28 days |

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| Vácha et al. (2009) | Magnetoreception disruption (cockroach) | Onset of disruption: 1.2 MHz 12-18 nT; 2.4 MHz 18-44 nT |
| Varghese et al. (2017) | Learning and memory deficits and expression of anxiety behavior (rat) | 2450 MHz, 4 hr/day for 45 days; at power density of 0.778 mW/cm$^2$, calculated power absorption in the body = 0.04728 W |
| Wang H. et al. (2013) | Spatial memory deficit (rat) | Pulsed 2856 MHz RFR, 5, 10, and 50 mW/cm$^2$, 6 min |
| Wang H. et al. (2015) | Spatial learning and memory deficits (rat) | Pulsed 2856 MHz RFR, 50 mW/cm$^2$, 6 min |
| Wang H. et al. (2017) | Spatial learning and memory deficits (rat) | 2856 MHz (1.75, 3.5, or 7 W/kg), 6 min/day, 5 days/week, 6 weeks |
| Wang K. et al. (2017) | Increased recognition memory (mouse) | 1800 MHz, >2.2 W/kg, 30 min |
| Wang LF et al. (2016) | Spatial memory impairment (rat) | GSM 1800 MHz, 30 mW/cm$^2$, 5 min/day, 5 days /week, 2 months |
| Zhang et al. (2015) | Increased anxiety-related behavior; spatial memory and learning deficits in male offspring (mouse) | 9417 MHz, 200 V/m, 2 W/kg, 12 hr/day on gestation days 3.5-18, offspring tested at 5 weeks of age |
| Zhang et al. (2017) | Increased anxiety-related behavior (mouse) | 1800 MHz, 6 hr/day for 28 days, whole body and brain SAR at 2.7 W/kg and 2.2 W/kg, respectively |

**Animal studies that showed no significant behavioral effects:**

| | **Behavior studied** | **Experimental conditions** |
|---|---|---|
| Ammari et al. (2008c) | spatial memory (rat) | GSM900, brain SRR 1.5 W/kg 45 min/day or 6 W/kg 15 |

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| | | min/day, 8 or 24 weeks |
| Fasseas et al. (2015) | Chemotaxis, short-term memory (Caenorhabditis elegans) | GSM 1800 MHz (15.4 V/m), WiFi router (9.7 V/m), Digital Enhanced Cordless Telecommunication (DECT) phone (11.3 V/m); various lengths of time (30 min to 24 hr) |
| Haghani et al. (2013) | Motor function (rat) | Pulsed 900MHz RFR, SAR 0.5-0.9 W/kg; 6 hr/day during gestation period |
| Klose et al. (2014) | Learning skills and motor behavior (rat) | GSM-modulated 900 MHz RFR, head only exposure 2 hr/day, 5 days/week from 14 days to 19 months old, 0.7, 2.5 or 10 W/kg |
| Shirai et al. (2014) | Spatial memory and motor function on $F_1$, $F_2$, and $F_3$ offspring (rat) | 2140 MHz WCDMA 20 hr/day from gestation Day 7 to weaning with dam, and offspring alone to 6 weeks old, 3-generations; 0.067-0.14 for a fetus, 0.12-0.36 W/kg for offspring before weaning, 0.12-0.24 W/kg for offspring after weaning |
| Salunke et al. (2015) | Anxiety, obsessive compulsive disorder (OCD) and depression-like behavior (mouse) | Bluetooth device, 2450 MHz, 60 min/day for 7, 30, 60, 90, or 120 days |
| Son et al. (2016) | Spatial and non-spatial memory functions (mouse) | 1950 MHz; 2 h/day, 5 days/ week, 3 months; 5 W/kg |

A majority of the animal studies reported effects, whereas more human studies reported no significant effects than effects. This may be caused by several possible factors: (a) Humans are less susceptible to RFR than are animals. (b) It may be more difficult to do human than animal experiments, since it is, in general, easier to control the variables and confounding factors in an animal experiment. (c) In the animal studies, the cumulative exposure duration was generally longer and studies were carried out after exposure, whereas in the human studies, the exposure was generally one time and testing was measurements were carried out

mostly during exposure. This raises the question of whether the effects of RFR are cumulative. This consideration could have very important implication on real life human exposure to EMF. However, it must be pointed out that neurophysiological and behavioral changes have been reported in both animals and humans after acute (one time) exposure to RFR, and most of the human EEG studies mentioned above are acute exposure experiments. (d) Most of the human studies are head exposure experiments whereas most of the animal studies involved whole body exposure. Could this have made a difference? Does it mean that effects of RFR on other parts of the body can also affect the nervous system? (e) The nervous system has the capability to adapt to perturbations. Physiological changes in the nervous system do not always manifest as behavioral effects, e.g., see Haghani et al. (2013) (changes in electrophysiology of cerebellar Purkinje cells after RFR exposure without behavioral effect in rats) and Schmid et al. (2012a) (RFR exposure induced EEG change but did not affect cognitive test performance in human subjects). May be the human brain has higher capability to tolerate and adapt to perturbations than other animals. (f) In the animal studies, the effects studies were mostly learning and memory functions. The hippocampus in the brain, particularly the cholinergic system, plays a major role in learning and memory functions. Various studies indicated that RFR affected electrical activities/morphology/chemistry of the hippocampus in animals (Aboul Ezz et al., 2013; Ammari et al., 2008 a, b; 2010; Barcal and Vozeh, 2007; Barthélémy et al., 2016; Baş et al., 2009, 2013; Carballo-Quintas et al., 2011; Choi and Choi, 2016; Erdem Koç et al., 2016;Fragopoulous et al., 2012; Gevrek, 2017; Gökçek-Saraç et al., 2017; Hao et al., 2013; Hassanshahi et al., 2017; Hu et al., 2014; İkinci et al., 2013; Kerimoğlu et al., 2016b; Kesari et al., 2011; Kim JH et al., 2017b; Kim JY et al., 2017; Kumari et al., 2017; Li et al., 2014; Lopez-Martin et al., 2009; Li et al., 2012; Lu et al., 2012; Maskey et al., 2010 a,b, 2012; Megha et al., 2015; Mugunthan et a., 2016; Narayanan et al., 2010, 2014, 2015; Ning et al., 2007; Nittby et al., 2008a; Odaci et al., 2008; Razavinasab et al., 2016; Şahin et al., 2015; Saikhedkar et al., 2014; Sharma et al., 2017; Tang et al., 2015; Tong et al., 2013; Wang H. et al., 2013, 2015, 2017; Wang K. et al., 2017; Wang LF et al., 2016; Xiong et al. 2015; Xu et al., 2017; Yang et al., 2012; Zhang et al., 2017). As early as 1987, we (Lai et al., 1987) have reported that RFR affected the cholinergic system in the hippocampus of the rat leading to spatial learning and memory deficits. Interestingly, the effect of RFR on the hippocampus apparently involves a sequence of neurological responses in the brain, including activation of endogenous opioids and release of the stress hormone corticotropin releasing factor (Lai, 1994). Thus, it is not surprising that 'learning and memory' functions are affected in the rodents by RFR since in most of the studies, the Morris water-maze was used to study learning and memory functions. The water-maze measures spatial memory, a function specifically involves the hippocampus. In the human studies listed above, the most common effect studied was cognitive functions. Since the exposure in most of these human studies was localized in the brain, particularly in the temporal cortical area, it is questionable whether the psychological tests used were appropriate.

**Discussion**

1. A major concern is that in some of the studies details of the exposure setup and dosimetry are not provided. This is important, since details of the independent variables

are very important in interpreting the validity of the experimental results, i.e., dependent variables. In many of these studies, a cell phone was used in the exposure of animals and humans. But information on how the cell phone was activated, in many instances, was not provided. Thus, the amount of energy deposited in the body was not known. Some studies used the phone in 'stand-by' mode. Mild et al. (2012) reported that when a stationary cell phone is on 'stand-by' mode, it actually infrequently emits a very small amount of energy. It is very surprising that in all papers on the effects of RFR on EEG mentioned at the beginning of this paper, only two provided significant information on the exposure parameters. This is alarming. It may indicate that the researchers did not understand the properties of the entity that they were studying. It is good that competent researchers from other disciplines are contributing to the advancement of bioelectromagnetics. But, I sincerely think that EMF researchers should get themselves acquainted with the physics of nonionizing electromagnetic fields.

2. Most of the studies were carried out with relatively high levels of RFR compared to environmental level. However, if you look through the narrative about, there are studied that reported effects at very low level, e.g., Bak et al, 2010. Indeed, biological/health effects of RFR at levels much lower than most international RFR-exposure guidelines, e.g., the International Commission on Non-ionizing Radiation Protection (ICNIRP), have been reported (see Table 1 in Levitt and Lai, 2010). This raises the question on whether the guidelines used in most countries nowadays are actually obsolete and new exposure guidelines have to be set.
3. Thus, there is ample evidence that RFR exposure affects the nervous system from both acute and long-term exposure experiments. Brain electric activities, nerve cell functions and chemistry and behavior can be affected. Some explanatory mechanisms for these effects have emerged. One consistent finding is that animals exposed RFR suffered from memory and learning deficits. These effects can be explained by the results of numerous reports that showed RFR affected the hippocampus, a brain region involved in memory and learning. However, the location and configuration of the human hippocampus are quite different from those of a rodent. There has not been much studies on the effect of RFR on the human hippocampus. Several studies did reported deficit in memory in human subjects exposed to RFR, particularly on short-term memory, a function specifically related to the hippocampus. One recent study (Deniz et al., 2017) showed that chronic cell phone use did not significantly affect the volume of the hippocampus in human subjects. But, the subjects showed poorer attention which is probably not related to the hippocampus. An interesting fact is that learning and memory deficits have also been reported in insects that do not have a hippocampus. Another related aspect is that several papers (Adrendash et al., 2010, 2012; Banaceur et al., 2013; Dragicevic et al., 2011) have indicated that RFR exposure could reverse some of the

defects in an animal model of Alzheimer's disease, a neurological disorder involved degeneration of cholinergic innervations in the hippocampus. Interestingly, similar claim has been reported (Hu et al., 2016) with exposure to extremely-low frequency magnetic field.

4. Another very consistent finding is that RFR affects free radical metabolism in the brain. This may explain some of the cellular and physiological effects of RFR on the nervous system. As a matter of fact, oxidative changes in cells and tissues after exposure to RFR is a very common phenomenon (cf. Yakymenko et al., 2016). This happened in many organs of the body and can provide explanation on many reported biological effects of RFR.
5. Many of the effects of RFR on the nervous system, e.g., on the hippocampus, oxidative effect, and behavioral effects, are also observed with exposure to extremely-low frequency electromagnetic field (cf. my section on the neurological effects of ELF EMF in the Bioinitiative Report, www.bioinitiative.info/bioInitiativeReport2012.pdf). There has been speculation whether biological effects observed with low-frequency modulated-RFR were actually caused by the modulation. There are two reports published in the last decade that seemed to refute this hypothesis. Perentos et al. (2013) reported in human EEG "…a suppression of the global alpha band activity was observed under the pulsed modulated RF exposure, and this did not differ from the continuous RF exposure. No effect was seen in the extremely low frequency condition." This means that pulsing is not essential for the effect observed. Schmid et al. (2012b) compared the effects of a 2-Hz modulated 900-MHz field with a 2-Hz magnetic field on human sleep EEG. Both fields affect sleep EEG but not identically. The authors concluded that "the study does not support the hypothesis that effects of radiofrequency exposure are based on demodulation of the signal only." However, in another study, Schmid et al (2012a) concluded in a study on sleep EEG that "…that modulation frequency components within a physiological range may be sufficient to induce these effects." In our earlier studies (e.g., Lai and Singh, 1995), we found that continuous-wave and pulsed RFR produced different effects. Indeed, different effects produced by continuous-wave and modulated RFR with the same frequency, exposure conditions, and SAR is a strong indication of the existence of "nonthermal" effects. Another question is whether one type of modulation is different from another in causing biological effects. Cell phone advances from one generation to another. Do the research data of 3G phone apply to 4G or 5G phone radiation? RFR is a complex entity. Its biological effects depend on many of its physical properties, e.g., frequency, direction of the incident waves relative to the object exposed, dielectric properties and size and shape of the exposed object, polarization of the waves, etc. Thus, it is unlikely that one can easily extrapolate the effects from one form of RFR to another. An assumption that 3G radiation is safe does

not necessary imply that 5G radiation is safe. Each one of them has to be investigated separately.

6. An important area of research is on how RFR in the environment affects humans and wildlife. Environmental RFR level has become higher and higher over the past decades due to the employment of RFR-wireless devices. Take the example of Bak et al. (2010) mentioned above, effect on human event-related brain potential was reported after 20 min of exposure to a GSM signal at a power density of 0.0052 $mW/cm^2$. This is very close to the levels found in some cities. The highest power density of ambient RFR measured near schools and Hospitals in Chandigarh, India was reported to be 0.001148 $mW/cm^2$ in 2012 (Dhami, 2012). The maximum total RFR power density emitted by FM and TV broadcasting stations and mobile phone base stations in centers of the major cities in the West Bank-Palestine was 0.00386 $mW/cm^2$ (Lahham and Hammash, 2012). One also has to take into consideration that exposure in the Bak et al. (2010) study was acute (20 min), whereas environmental exposure is chronic. Related to the neurological effect is the magnetic-sense possessed by many species of animals. It is essential for their survival. Interference by RFR of magnetic compass orientation in animals has been reported (e.g., Landler et al., 2015; Malkemper et al., 2015; Pakhomov et al., 2017; Schwarze et al., 2016; Vácha et al., 2009). Understanding the effects could help in preserving the ecosystem and ensure survival of the species on this earth.

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# Evidence of Neurological effects of Electromagnetic Radiation: Implications for degenerative disease and brain tumour from residential, occupational, cell site and cell phone exposures.

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**10th September 2002**

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# Evidence of Neurological effects of Electromagnetic Radiation: Implications for degenerative disease and brain tumour from residential, occupational, cell site and cell phone exposures.

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**Abstract**

**The brain it is a really sensitive bioelectromagnetic organ. Therefore it is scientifically plausible that brain will react to and be sensitive to external electromagnetic signals. It has been shown that has very strong evidence that the brain detects and responds to the Schumann Resonance signal of 0.1pW/cm$^2$. Since the first evidence that RF radiation damages chromosomes in 1959, many independent studies have identified broken DNA stands, chromosome aberrations and altered gene expression in animal cells, human cells and in living animals and humans from EMR exposure. Microwaves, including cell phone radiation, open the Blood Brain Barrier (BBB). Exposure to RF/MW is consistently associated with headaches, fatigue, loss of concentration and memory loss. These symptoms have been called "The Radiofrequency Sickness Syndrome" or "Microwave Syndrome". Because these are subjective symptoms they have been largely dismissed in the West. These symptoms are now shown with cell phone use in a significant dose-response manner. All of these effects are linked to electromagnetic radiation's ability to alter cellular calcium ions and GABA through cellular signal transduction processes not involving heat, to reduce melatonin and damage DNA, and enhance Apoptosis. A large and growing body of epidemiological research is revealing EMR associated neurological effects, degenerative disease and brain tumour. Cell phone radiation is involved in many of the biological effects and now shows significant increases in DNA damage and brain tumours. Residential exposures down to 0.4nW/cm$^2$, typically a thousand times stronger than the Schumann Resonance signal, and living within the vicinity of cell sites, are shown to have a causal relationship to the melatonin reduction related sleep disturbance. Therefore they will produce a host of other genotoxic and melatonin related health effects.**

Key Words: Electromagnetic radiation, calcium ion efflux, GABA, genotoxicity, melatonin reduction, neurological disease, suicide, brain cancer

## Introduction:

Our brains are exquisitely sensitive bioelectrochemical organs that are the seat of human creativity, memory, emotions and intelligence. We use electrical signals, including charged calcium ions, to think, remember and see, to regulate the beating of our heart, and for communication in our central nervous system and between and within our cells. Human brains were proven in the 1950's and 1960's, König (1974) and Wever (1974), to be very sensitive to and reactive to extremely small, naturally occurring Schumann Resonances. The Schumann Resonances are global low frequency signals that share the same part of the spectrum as the EEG. They are generated largely by tropical thunderstorms. They

propagate around the (at the speed of light), being ducted in the resonant cavity formed between the earth and the ionosphere. König observed highly significant alteration of human reaction times associated with the intensity of the Schumann Resonances, Figure 1. He then carried out laboratory experiments and could speed people up with 10 Hz signals and slow them down with 3 Hz signals. This work was independently confirmed by Hamer (1965, 1969). Wever (1974) carried out long-term isolation experiments and showed that isolation from sunlight resulted in significantly lengthened daily rhythms. Isolation also from all EMR extended the daily period significantly more. About 30 % of subjects were desychronized, producing very long and erratic daily rhythms. These could be corrected by the introduction of a very low intensity 10 Hz signal, similar to the primary Schumann Resonance peak.

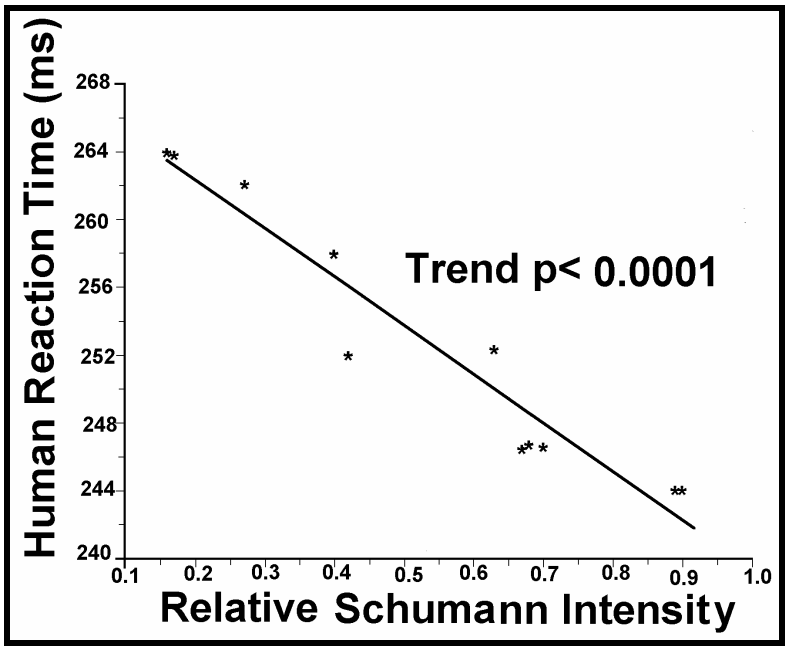


Figure 1: Human reaction times are causally correlated with natural variations in the Schumann Resonance Intensity, König (1974). The mean Schumann intensity (Relative Schumann Intensity =0.5) is 0.65mV/m or 0.1pW/cm$^2$. The range is 0.2 to 1.2 mV/m (0.01 to 0.4pW/cm$^2$).

The original König and Hamer experiments involved field strengths in the order of 1 V/m (0.27μW/cm$^2$) but results were still statistically significant when 1mV/m (0.27pW/cm$^2$) was used. The Schumann Resonances have a fundamental frequency of 7.8 Hz. The spectrum has other resonance peaks near 14.1, 20.3, 26.4 and 32.5 Hz. The three primary peaks between 7 and 21 Hz have a mean intensity of about 0.1pW/cm$^2$, Polk (1982). Thus the early German research concluded that there was significant proof that human beings react to electromagnetic radiation at extremely low intensities, including that naturally produced and called the Schumann Resonances. They speculated that humans had evolved to use the Schumann Resonances to timing synchronization, that is, they are a Zeitgeber.

Cherry (2002) shows that the Schumann Resonance (SR) signal modulation by Solar/Geomagnetic Activity (S/GMA) modulates human melatonin, Figure 2, and causes modulation of human health effect including, cancer, cardiac, reproductive and neurological diseases and mortality, with a mean intensity of 0.1pW/cm$^2$, with a magnetic field component about 1-3pT.

It is noted that cell sites produce signal intensities over 0.1μW/cm$^2$, out to 500m to 1000 m, depending on the power and height of the tower. This is 1 million times higher than the natural SR signals that it is proven that our brains detect and use. The possibility of interference with the natural signals and the processes that they alter is strongly evident.

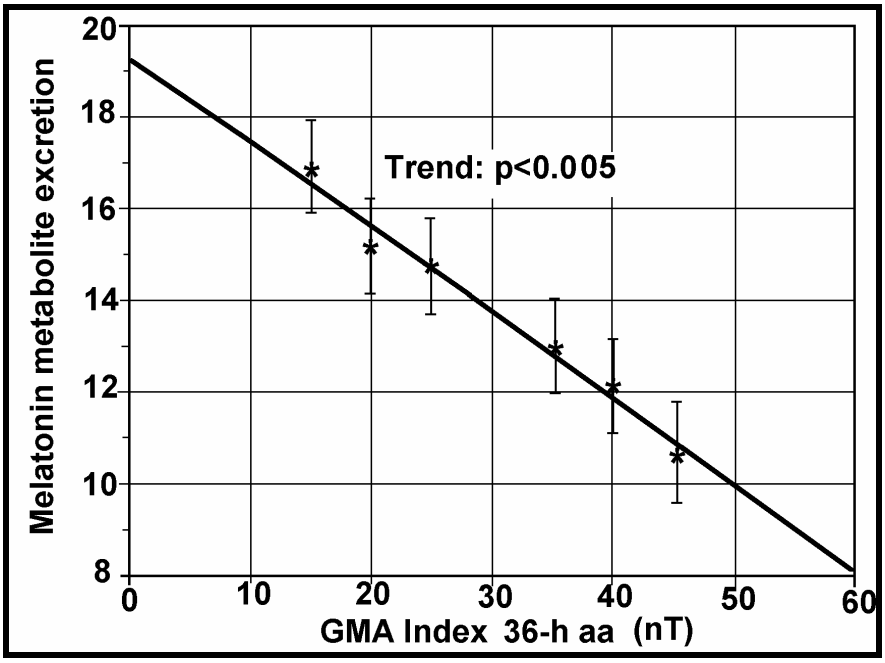


Figure 2: Reduction in the melatonin metabolite 6-OHMS in μg in urine from U.S. electric utility workers, as a function of the 36 hr global GMA aa-index, Burch et al. (1999b).

Figures 1and 2 show a classically causal relationship between the Schumann Resonance signal strength, which is extremely correlated with GMA indices, with altered human reaction times and modulation of human melatonin.

A core principle of Environmental Health is understanding and appreciating the natural system before assessing the impact of human activity on people, organs and cells. The failure of authorities to appreciate this and to apply this principle, has lead to massive trends in neurological illnesses from living in electric power produced ELF fields and RF/MW fields from radio and TV stations, computer screens, cordless phones, and occupational exposures of electrical workers, physiotherapists, airport staff, military, police and fire personnel, radio and TV personnel. Most recently this involves the introduction of a cellular phone network and wireless laptop systems.

Western public exposure guidelines and standards are typically in the range 0.1 to 10 billion times higher than the SR signal which is causally associated with modulation of human health effects. The evidence and conclusions of this report give a strong motivation for revising exposure standards and the development and application of safer technologies.

## Early Evidence of Neurological Symptoms from chronic radar exposure:

Evidence that radiofrequency/microwave (RF/MW) radiation also interacts with human brains at extremely low intensities comes from the U.S. Embassy in Moscow during the

1950s-1970s, Lilienfeld et al. (1978). Overall the mortality rate of Moscow personnel was 42% and in the comparative posts personnel it was 36% of the U.S. rate, showing the health employee effect and the healthier status of State Department staff through the selection of health staff.

The U.S. Embassy was deliberately irradiated by Soviet radar for over 20 years. It was aimed at the 5$^{th}$ floor of the west wall at one end of the Embassy Building. During most of this time the peak reading was 5$\mu$W/cm$^2$, over the working hours. The internal exposures are much smaller, Pollard (1979). Mean daily exposures are typically in the range 0.01 to 0.1$\mu$W/cm$^2$.

Several significant adverse health effects were identified. Several sickness symptoms were significantly increased with years of service in the Moscow Embassy, a dose-response relationship. They included Arthritis/Rheumatism (trend p=0.02), Back Pain (trend p=0.04), Ear problems (trend p=0.02), Skin/Lymphatic (trend p=0.02) and Vascular System (trend p=0.004)

The male staff who were chronically exposed to the radar signal showed a wide range of elevated neurological symptoms, some of which were significantly increased. These included depression (p=0.004), irritability (p=0.009), difficulty in concentration (p=0.001), memory loss (p=0.008). Dependents developed increased rates of cancer, including significant brain tumors, SMR = 20 (2.4-72.2). Children had increased mental and nervous conditions (RR = 5.0) and behavioural problems (RR= 2.06).

Baranski and Czerski (1976) give a description of a microwave exposure syndrome that was identified by Soviet researchers, e.g. Gordon (1966). Similar syndromes were reported in France by Deroche (1971) and in Israel by Moscovici et al. (1974). The Syndrome's symptoms included headaches, fatigue, irritability, nausea, vertigo, sleep disturbances and decreased libido. Johnson-Liakouris (1998) states that a literature review and the Lilienfeld study supports the Radiofrequency (RF) Sickness Syndrome as a medical entity. The headache symptoms were found with microwave exposure during "microwave hearing" experiments, Frey (1998) and in microwave exposure case studies, Forman et al. (1986).

The evidence was strong enough in 1982 for the Supreme Court of New York to award workers compensation for "Radiofrequency Sickness Syndrome" for chronic occupational microwave exposure to a technician servicing TV transmitters in the 87th floor of the Empire State Building, Yannon vs New York Telephone Co. The compensation also recognized that the chronic microwave exposure caused his death. Application for leave to appeal was declined. The primary expert witness in this case was Dr Milton Zaret.

**Biological Mechanisms for Neurological Effects:**

Biological mechanism for these effects have been well identified, especially, reduced melatonin. Pulsed and modulated RF/MW radiation is also shown to induce efflux of calcium ions and GABA from brain cells.

GABA related neurotransmitters are changed in a dose response manner by 915 MHz microwaves, Figure 3. Altered GAMA is shown to cause all of the neurological symptoms identified above. GABA (gamma-amino butyric acid) and glutamatergic synapses make up up to 60 % of the synapses in the CNS and 40 % in the brain, Kolomytkin et al. (1994). Hence induced alteration of GABA in the brain can have serious consequences. Figure 3

shows that a 5 minute exposure to pulsed microwaves have a dose-response effect on GABA related receptors.

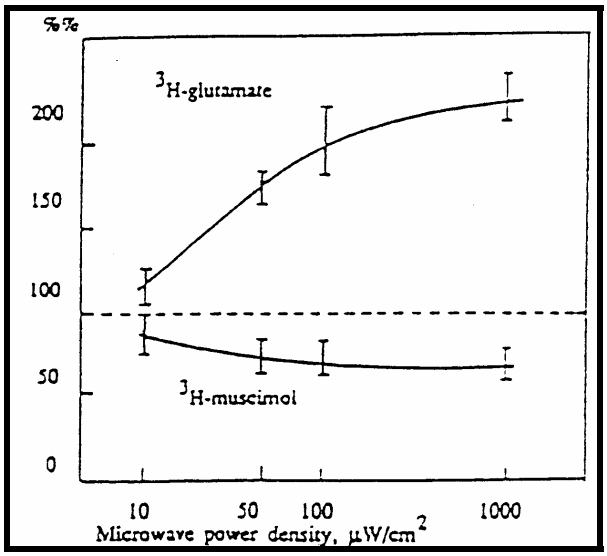


Figure 3: Exposure related alteration of GABA related molecules in rat brains exposed for 5 minutes to 915 MHz microwaves, pulsed at 16 pps. Differences from controls are still significant at 10µW/cm$^2$. Kolomytkin et al. (1994)

Frey (1995) concludes that EMR affects the dopamine systems of the brain through its effects on GABA. He also notes that the dopamine-opiate systems interact with the pineal melatonin/serotonin system.

**Altering Cellular calcium in homeostasis:**

The initial calcium ion research of Adey and Bawin was motivated by the observations that EMR altered reaction times in people, König (1974) and Hamer (1965, 1969), and monkeys, Gavalas-Medici and Day-Magdaleno (1976). Bawin, Gavalas-Medici and Adey (1973) showed alterations in cat EEGs and subsequently calcium ion efflux from cat brains under the same exposure conditions, Adey (1979). Shandala et al. (1979) show that microwaves significantly altered the EEG of animals.

Calcium ions are ubiquitous in cells throughout our bodies. The calcium ion ($Ca^{2+}$) is one of the most important substances in cells. $Ca^{2+}$ is a first, second and third signal transduction messenger, Alberts et al. (1994), Pahl (1999). Alberts et al. describes $Ca^{2+}$ as a prominent and ubiquitous intracellular messenger. This means that factors that induce changes of cellular $Ca^{2+}$ can cause significant changes of cells. As a signal transduction messenger $Ca^{2+}$ initiates and regulates many cellular processes, such as melatonin production. Given that EMR induces changes in cellular calcium ions it is reasonable to investigate whether EMR induces changes in melatonin. This biological plausibility of ELF detection by the brain is significantly strengthened by the observation that mammal brains contain and use phase-locked loop circuitry to detect and react to incoming ELF signals, Ahissar et al. (1997). Hence our brains contain a highly efficient, tuned FM receiver, Motluk (1997).

Chemical substances, such as TPA, are cancer promoters. They operate by altering the calcium ions. They cause calcium ion influx, which stops a damaged cell from going into apoptosis (programmed cell death) so that the cancer cell survives. Certain electromagnetic radiation combinations cause calcium ion influx, i.e. promoting cancer,

while other causes of calcium ion efflux, promoting apoptosis. Balcer-Kubiczek (1995) describes the ways in which TPA at low concentrations are able to switch the effect of calcium ion elevation from cell death to cell proliferation, probably by the activation of protein kinase C.

Dr Carl Blackman reviewed the extensive research literature on calcium ion efflux. He was well qualified to do this since he and his group at the U.S. E.P.A. had been responsible to replicating and extending all of the research shown in other laboratories. Blackman (1990) concludes:

**"Taken together, the evidence overwhelmingly indicates that electric and magnetic fields can alter normal calcium ion homeostasis and lead to changes in the response of biological systems to their environment".**

Blackman (1990) concludes that calcium ion efflux/influx is an established biological effect of EMR exposure and it changes the biological response of cells. Because modulation frequencies are critically involved, and low intensity exposures are observed under some circumstances to produce greater effects than some higher exposure conditions, resonant interactive processes are indicated and heating is definitely not involved except to establish a homeostatic range.

Blackman's group confirmed and significantly extended the "windows" concept of $Ca^{2+}$ efflux, as well as aspects of homeostasis, involving tissue temperature for example. $Ca^{2+}$ efflux is a function of modulation frequencies. Frequencies out to 510 Hz produce significant $Ca^{2+}$ efflux at some frequencies, but not at other frequencies on either side, Blackman et al. (1988). Power-Density windows are also identified, Bawin and Adey (1976), Blackman et al. (1989). The lowest intensity that has been published showing significant $Ca^{2+}$ efflux is 0.00015 W/kg, Schwartz et al. (1990). This involved a 16 Hz modulation carried on a 240 MHz carrier. This is equivalent to 0.08$\mu$W/cm$^2$.

Blackman et al. (1990a) showed the importance of the local static magnetic field. Blackman et al. (1991) showed that $Ca^{2+}$ efflux occurred for tissue temperatures of 36°C and 37 °C and not at 35°C and 38°C. $Ca^{2+}$ efflux is demonstrably not a thermal effect. It occurs at extremely low non-thermal levels and is a function of frequency, modulation frequency and it occurs in exposure windows. This appears to be a form of resonant interaction.

In some quarters the RF-Thermal View still dominates. This has been challenged many times since the Second World War. For example, Dr Adey gave the introductory paper to a 1974 conference, Adey (1975), on the effects of EMR on the nervous system. In this paper he states:

> **"Even a recent review body of the World Health Organization decided after discussion to dismiss from its concerns possible biological effects that might occur in the absence of significant heating. It has become clear, however, that interactions with the mammalian central nervous system can be reliably produced by oscillating electric and electromagnetic fields without significant heating of tissues."**

Blackman et al. (1991) comment that the windowing aspects of $Ca^{2+}$ efflux could be very good reasons why experimental outcomes have been difficult to confirm in some laboratories. Everything may appear to be the same but the local magnetic field is

different and completely changes the results. High exposure levels that raise the temperature outside the homeostatic range will produce no effects. Hence only non-thermal exposures produce these effects. Thus people are moving through constantly changing fields at home, at work and in the environment. They pass through windows of effect and no effect all the time. The cumulative effect of the 'effect' windows produces dose-response increases in health effects associated with extremely low mean exposures. The calcium ion effects are shown in brain tissue and heart muscle tissue, implicating neurological and cardiac effects. $Ca^{2+}$ efflux from pinealocytes (cell in the pineal gland) is likely to reduce melatonin production. This has implications for many kinds of sickness, cancer, miscarriage, neurological disease etc… because melatonin is a very potent free radical scavenger and

**EMR Reduces Melatonin in Animals and People**

DNA strand breaks, Chromosome Aberrations, impaired immune system competence and many other biological and health effects, are caused by reduced melatonin, Reiter and Robinson (1995). Light-at-night and electromagnetic radiation, are proven to reduce melatonin and hence pose significant adverse health effects.

Light-at-night and electromagnetic radiation, are proven to reduce melatonin and hence pose significant adverse health effects. The evidence for EMR is summarized here. Rosen, Barber and Lyle (1998) state that seven different laboratories have reported suppression of nighttime rise in pineal melatonin production in laboratory animals. They show that a 50 μT, 60 Hz field with a 0.06μT DC field, over 10 experiments, averages a 46% reduction in melatonin production from pinealocytes. Yaga et al. (1993) showed that rat pineal response to ELF pulsed magnetic fields varied significantly during the light-dark-cycle. They found that the rate-limiting enzyme in melatonin synthesis, N-acetyltransferase (NAT) activity showed that magnetic field exposure significantly suppressed NAT during the mid- to late dark phase.

Seventeen studies from show that ELF and RF/MW exposure reduces melatonin and enhances serotonin in people. Evidence that EMR reduced melatonin in human beings commenced with Wang (1989) who found that workers who were more highly exposed to RF/MW had a dose-response increase in serotonin, and hence indicates a dose-response reduction in melatonin. Sixteen studies have observed significant EMR associated melatonin reduction in humans. They involve a wide range of exposure situations. This includes 16.7 Hz fields, Pfluger et al. (1996); 50/60 Hz fields, Wilson et al. (1990), Graham et al. (1994), Wood et al. (1998), Karasek et al. (1998), Burch et al. (1997, 1998, 1999a, 2000), Juutilainen et al. (2000) and Graham et al. (2000); combination of 60 Hz fields and cell phone use, Burch et al. (1997,1999a); VDTs ELF/RF exposures, Arnetz et al. (1996), and a combination of occupational 60Hz exposure and increased geomagnetic activity around 30nT, Burch et al. (1999b). Two recent studies recorded significant melatonin reduction in women in EMF residential exposure situations, Davis et al. (2002) and Levallois et al. (2002).

The eighteenth human melatonin reduction study is from 6.1-21.8 MHz SW RF exposure as reported during the shutting down process of the Schwarzenburg shortwave radio tower, Professor Theo Abelin (seminar and pers.comm.). Urinary melatonin levels were monitored prior to and following the closing down of the Schwarzenburg short wave radio transmitter. This showed a significant rise in melatonin after the signal was turned off.

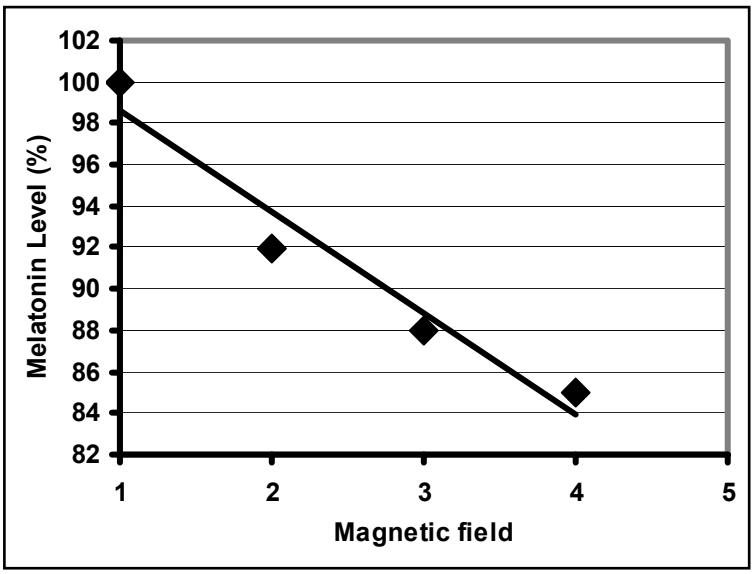


Figure 4: Human melatonin reduction from residential field exposures, the horizontal graph scale is in multiples from the lowest exposure (1), Davis (1997).

**Schwarzenburg Study:**

The Swiss research, Altpeter et al. (1995, 1997) - The Schwarzenburg Study) found a causal relationship between sleep disturbance and subsequent chronic fatigue, and short-wave radio exposures at extremely low mean levels. A wide range of symptoms were significantly elevated in a dose response manner, especially for those aged more than 45 years. In a multivariate analysis only the RF exposure was independently significantly associated with sleep disturbance, $p<0.01$.

The causal relationship between RF radiation exposure and deterioration in sleep quality is identified through a significant dose response relationship ($p<0.001$), Figures 5 and 6, improvements in sleep quality which changing the direction of the beams and turning the transmitter off, and reduced melatonin as the biological mechanism.

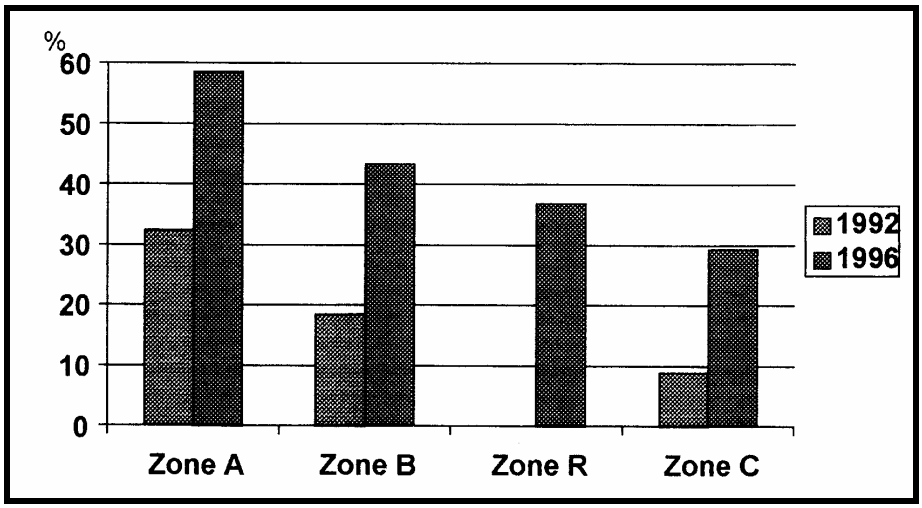


Figure 5: Adult Sleep Disturbance with RF exposure at Schwarzenburg, Switzerland, Altpeter et al. (1997).

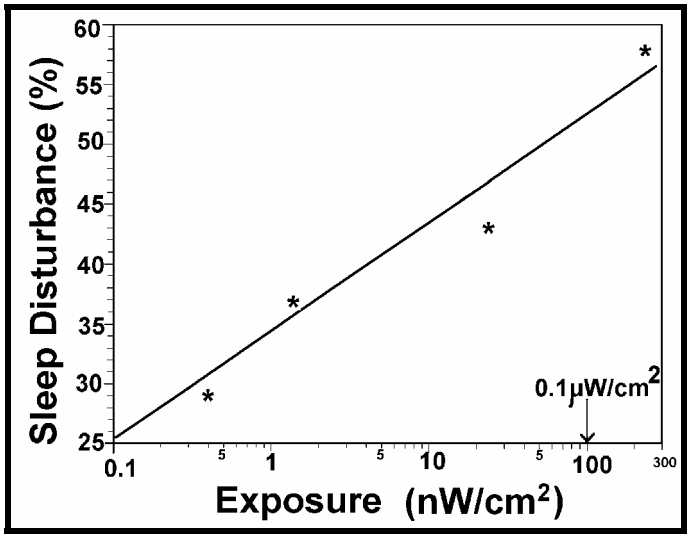


Figure 6: Dose-response relationship for Sleep Disturbance at Schwarzenburg with exposure in nW/cm$^2$. Note: 1nW/cm$^2$= 0.001μW/cm$^2$

Groups B, R and C are all exposed to a mean RF signal of less than 0.1μW/cm$^2$ and they experienced highly significant sleep disturbance and reduced melatonin. Since sleep disturbance and melatonin reduction has been observed with cell phone exposure, Mann and Roschkle (1995) and Burch et al. (1997), these observations also apply to cell sites. Assuming a normal sleep disturbance of 10 %, the approximate exposure level threshold for zero additional effect is near 1 pW/cm$^2$, near the natural level for the Schumann Resonances.

As an experiment, the transmitter was secretly turned off for three days. Sleep quality improved in all three groups being studied. Figure 7 shows Groups A and C.

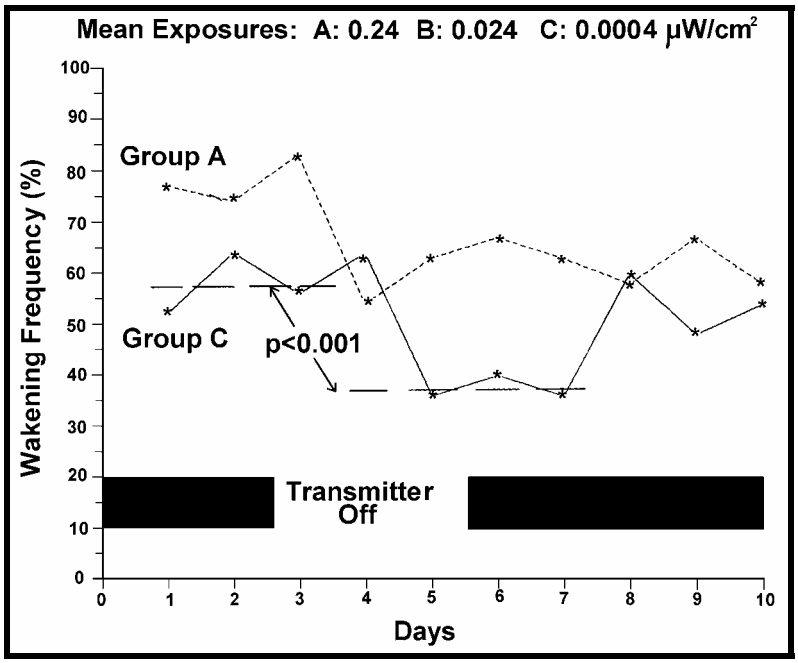


Figure 7: Sleep disturbance in people exposed to a short-wave radio stations which was turned off for three days, Altpeter et al. (1995), showing the highest exposed Group A, and lowest exposed Group C.

Both Groups show a delayed improvement in sleep of one to two days. The reduced wakening averaged over days 4 to 6 compared with days 1 to 3, for group A, and days 5

to 7 compared with days 1 to 4 for group C, are highly significantly reduced, $p<0.001$. Thus a significant ($p<0.001$) improvement in sleep quality is associated with a measured 24 hour mean and median exposure of 0.1 mA/m (0.4 nW/cm$^2$).

Human melatonin was sampled from urine in the morning. This is relatively ineffective because the important measure is the nocturnal peak. Altpeter et al. note that "Persons reporting sleep disorders, however, tend to have lower melatonin levels." When the decision was made to close down the transmitter permanently, melatonin readings were taken from a large group of residents before and after the closure. This showed a significant increase in melatonin following the closure, Professor Theo Abelin pers. Comm - seminar).

Two herds of 5 cows each, had salival melatonin sampled several times a day, including night-time. The "exposed" herd as at 500 m from the tower with a mean exposure of 0.095μW/cm$^2$. Their mean melatonin levels were 17.7 pg/ml compared with 19.0 pg/ml for the "unexposed" cows whose measured mean exposure as 0.00022μW/cm$^2$. Figure 8 shows the melatonin for these two herds during the experiment involving turning the tower off.

The small number of cows makes it difficult to show a significant difference. There is a persistent phase shift in the nocturnal melatonin peak with the exposed cows showing a delay. This reduced when the transmitter was off but returned when it was turned on. The exposed cows have lower mean melatonin prior to the off period. It rises progressively while the transmitter is off and is significantly higher on the third night. It then drops significantly when the transmitter is turned on again. This shows the "classic" effect of EMR reduction of melatonin. The "low" exposure cow's melatonin drops when the transmitter is turned on.

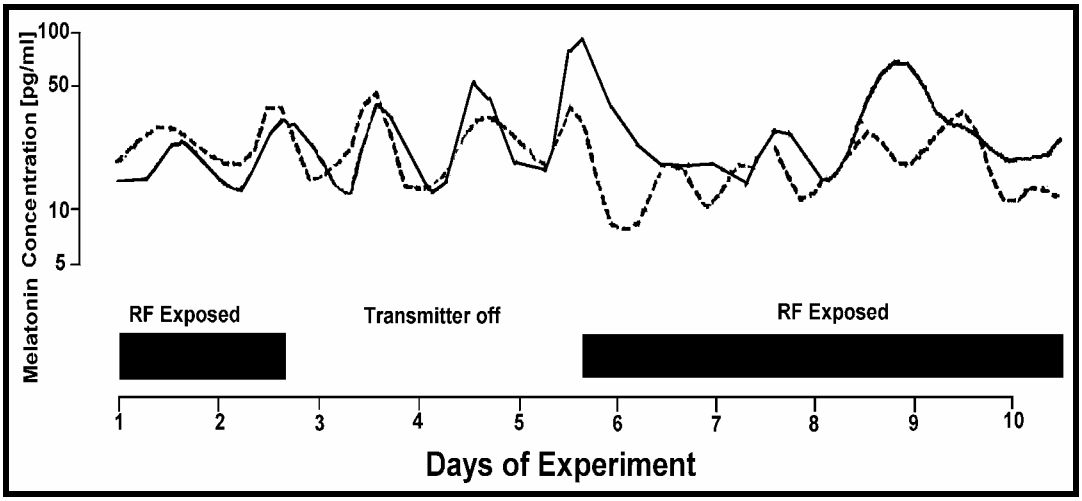


Figure 8: Salival melatonin from two herds of 5 cows, one exposed at 500 m, 0.095μW/cm$^2$, (solid line) and one "unexposed" at 4000 m, 0.00022μW/cm$^2$, (dashed line).

The causal relationship with human sleep disturbance is strong evidence of a significant neurological effect of RF radiation on people, associated with mean exposures down to less than 0.4 nW/cm$^2$. Hence, it is highly likely that cell phone users, with brain exposures many millions of times higher than the Schwarzenburg exposure levels, will experience significant neurological effects. The significant bovine behavioural effects of extremely low RF exposure is confirmed by Löscher and Käs (1998).

Table 1: Odd's ratios for an increase in 24-hour average exposure from 1 to 10 mA/m (0.04 to 3.8μW/cm$^2$) adjusted for age, sex, attribution and duration of time lived at the same place

| Symptoms | OR | 95% Confidence Intervals |
|---|---|---|
| Nervosity (Anxiety) | 2.77 | 1.62 - 4.74 |
| Diff. in falling asleep | 3.35 | 1.86 - 6.03 |
| Diff. in maintaining sleep | 3.19 | 1.84 - 5.52 |
| Joint pain | 2.46 | 1.37 - 4.43 |
| Limb pain | 2.51 | 1.15 - 5.50 |
| Cough and sputum | 2.80 | 1.18 - 6.64 |

All of the symptoms win Table 1 are consistent with reduced melatonin, Reiter and Robinson (1995). When the symptoms are ranked by exposure zone they form a dose-response relationship, Table 2. All of the symptoms in Table 1, except for cough and sputum, show very highly significant dose-response trends. Significant trends are also seen for Psychovegetative Index, Feeling Body excitement and Constipation. Non-significant trends are seen for Disturbed Concentration, Stomach pain and Neck and Shoulder pain. It is a common experience that lack of sleep, anxiety and depression lead to may other illnesses and symptoms.

Table 2: Complaints by Zones for all ages, showing the p-value for the trend.

| Symptoms | Zone A(%) | Zone B (%) | Zone C (%) | Trend p-value |
|---|---|---|---|---|
| Nervousness (Anxiety) | 25.0 | 18.0 | 7.0 | <0.001 |
| Difficulty in falling asleep | 22.9 | 17.6 | 6.7 | <0.001 |
| Difficulty in maintaining sleep | 32.4 | 18.5 | 8.9 | <0.001 |
| General weakness and tiredness | 22.0 | 13.0 | 6.0 | <0.001 |
| Limb pain | 14.3 | 6.7 | 3.3 | 0.003 |
| Joint pain | 22.9 | 10.1 | 10.0 | 0.004 |
| Psychovegetative Index | 12.5 | 5.2 | 3.4 | 0.010 |
| Feeling body excitement | 7.6 | 5.9 | 1.1 | 0.018 |
| Constipation | 7.6 | 6.7 | 1.7 | 0.034 |
| Disturbed concentration | 7.6 | 2.5 | 2.8 | 0.083 |
| Stomach pains | 9.5 | 5.9 | 3.9 | 0.152 |
| Neck and shoulder pain | 17.1 | 15.1 | 10.0 | 0.182 |

**Other Neurological Effects:**

Since it is now established that EMR alters calcium ions, GABA and melatonin, including overwhelming evidence for melatonin reduction in people, it is expected that EMR exposure will produce observable neurological effects, especially those that are known to related to calcium ions, GABA or melatonin reduction.

**Recent Research results from cell phone radiation exposure:**

In recent years research into the biological effects of cell phones have revealed many significant effects, especially neurological effects, Table 3.

**Table 3: Biological effects shown by mainly government and industry funded research that associates cell phone radiation with the following symptoms:**

- Alters brain activity including EEG, Von Klitzing (1995), Mann and Roschkle (1996)
- Disturbs sleep. Mann and Roschkle (1996), Bordely et al. (1999)
- Alters human reaction times, Preece et al. (1999), Induced potentials, Eulitz et al. (1998), slow brain potentials, Freude et al. (1998), Response and speed of switching attention (need for car driving) significantly worse, Hladky et al. (1999). Altered reaction times and working memory function (positive), Koivisto et al. (2000).
- Causes memory loss, concentration difficulties, fatigue, and headache, in a dose response manner, (Mild et al. (1998)). Headache, discomfort, nausea, Hocking (1998).

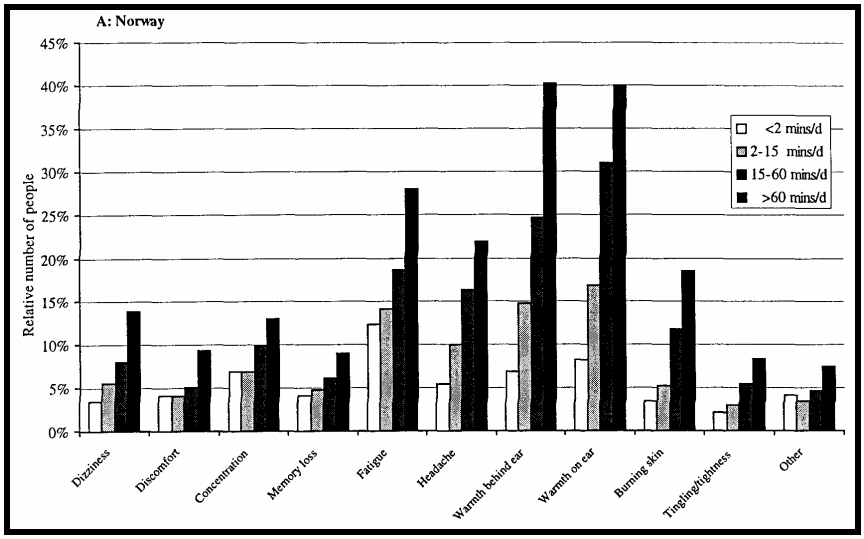


Figure 9: Prevalence of symptoms for Norwegian mobile phone users, mainly analogue, with various categories of length of calling time per day, Mild et al. (1998).

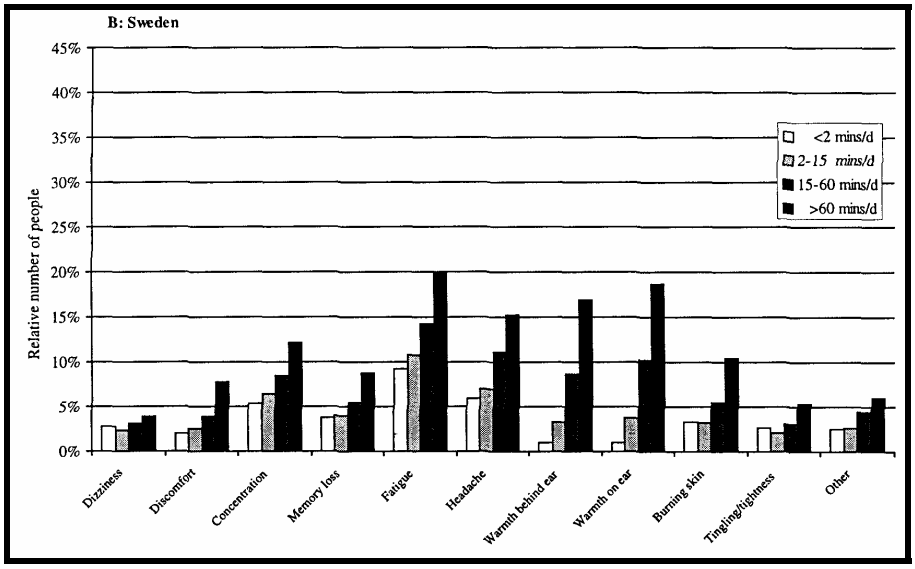


Figure 10: Prevalence of symptoms for Swedish mobile phone users, mainly digital, with various categories of length of calling time per day, Mild et al. (1998).