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

Dr Neil Cherry O.N.Z.M.
Associate Professor of Environmental Health

10th September 2002

Neil.Cherry@ecan.govt.nz

Human Sciences Department
P.O. Box 84
Lincoln University
Canterbury, New Zealand

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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.

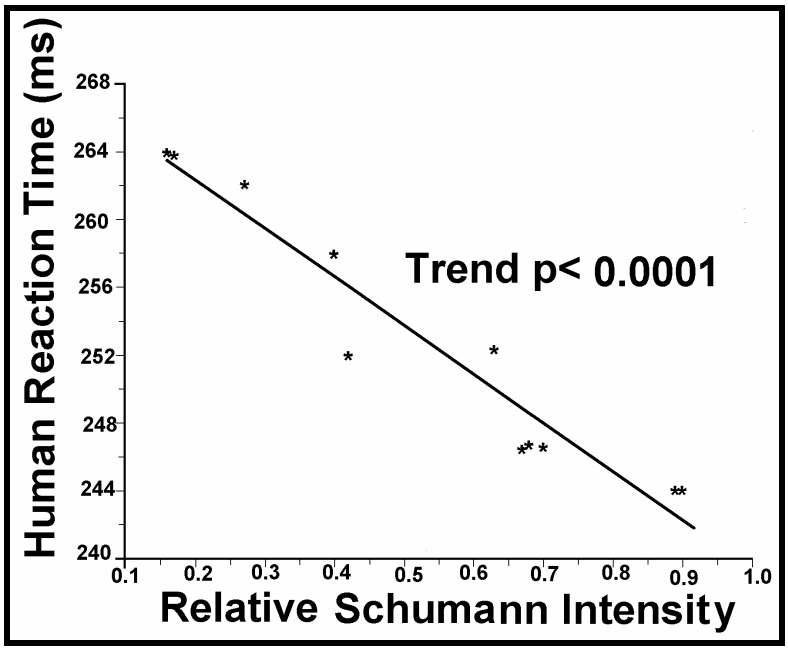

Figure 1: Human reaction times are causally correlated with natural variations in the Schumann Resonance Intensity, KÖnig (1974). The mean Schumann intensity (Relative Schumann Intensity =0.5) is 0.65mV/m or 0.1pW/cm^2. The range is 0.2 to 1.2 mV/m (0.01 to 0.4pW/cm^2).

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.

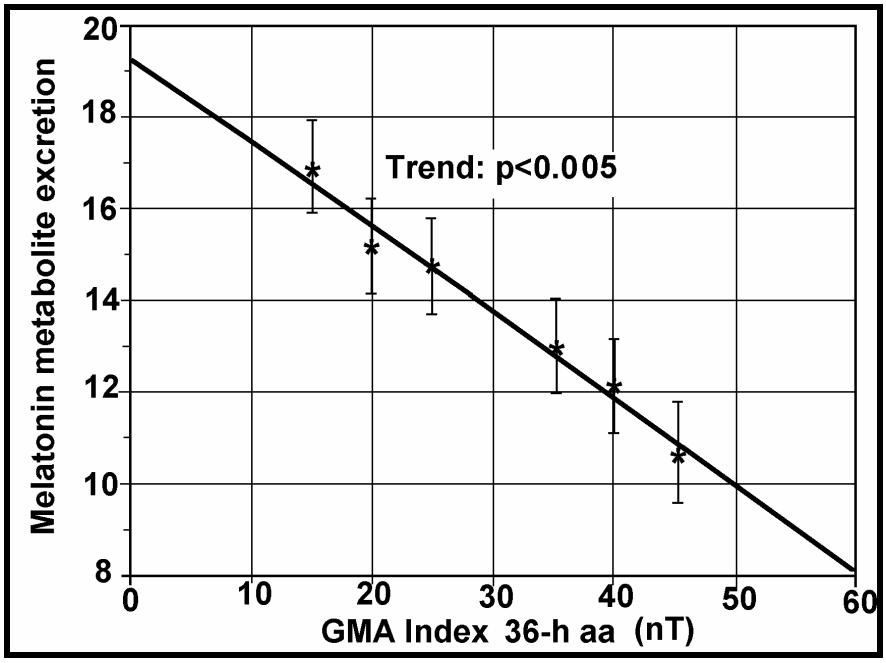

Figure 2: Reduction in the melatonin metabolite 6-OHMS in μg in urine from U.S. electric utility workers, as a function of the 36 hr global GMA aa-index, Burch et al. (1999b).

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 5th floor of the west wall at one end of the Embassy Building. During most of this time the peak reading was 5μW/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 0.1μW/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.

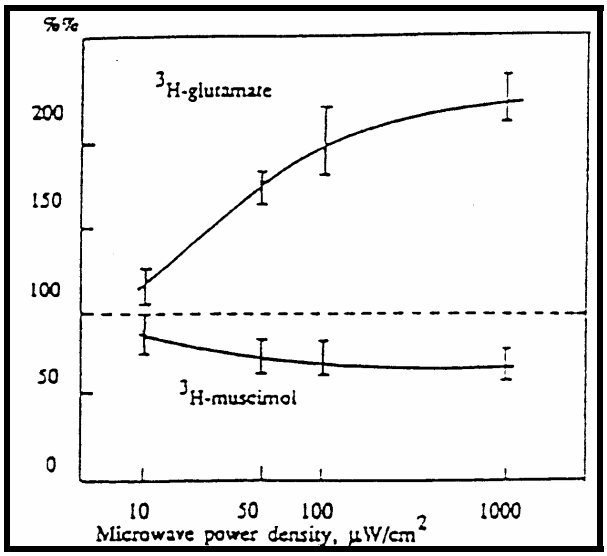

Figure 3: Exposure related alteration of GABA related molecules in rat brains exposed for 5 minutes to 915 MHz microwaves, pulsed at 16 pps. Differences from controls are still significant at 10μW/cm^2. Kolomytkin et al. (1994)

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 0.08μW/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.

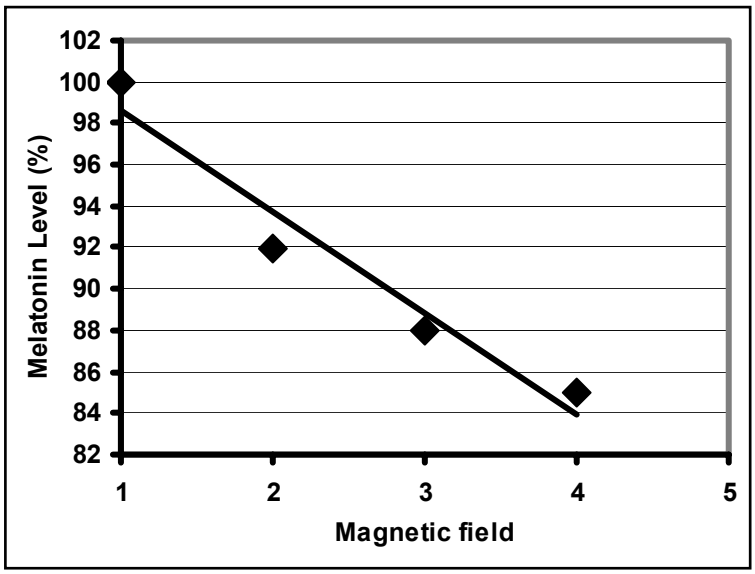

Figure 4: Human melatonin reduction from residential field exposures, the horizontal graph scale is in multiples from the lowest exposure (1), Davis (1997).

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.

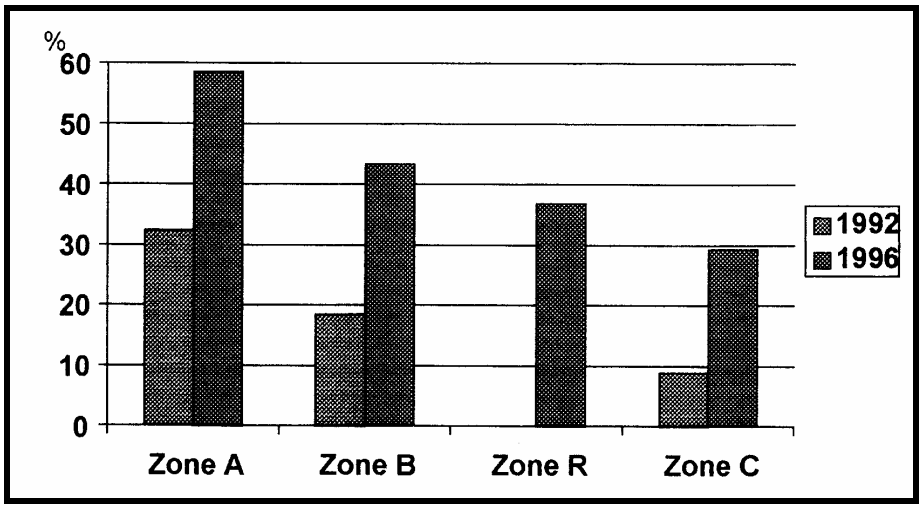

Figure 5: Adult Sleep Disturbance with RF exposure at Schwarzenburg, Switzerland, Altpeter et al. (1997).

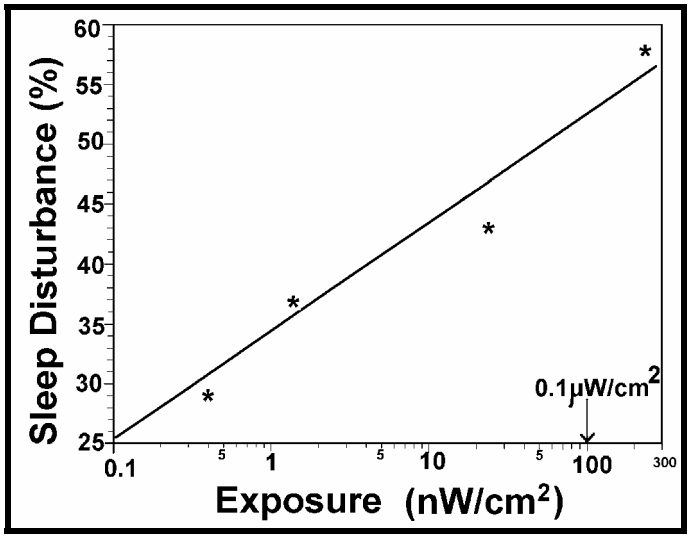

Figure 6: Dose-response relationship for Sleep Disturbance at Schwarzenburg with exposure in nW/cm^2. Note: 1nW/cm^2= 0.001μW/cm^2

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.

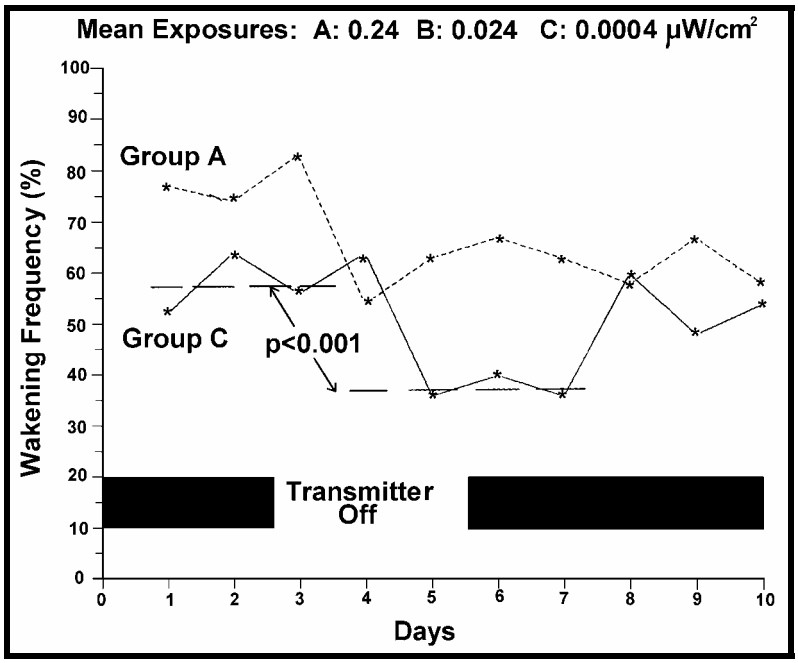

Figure 7: Sleep disturbance in people exposed to a short-wave radio stations which was turned off for three days, Altpeter et al. (1995), showing the highest exposed Group A, and lowest exposed Group C.

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.

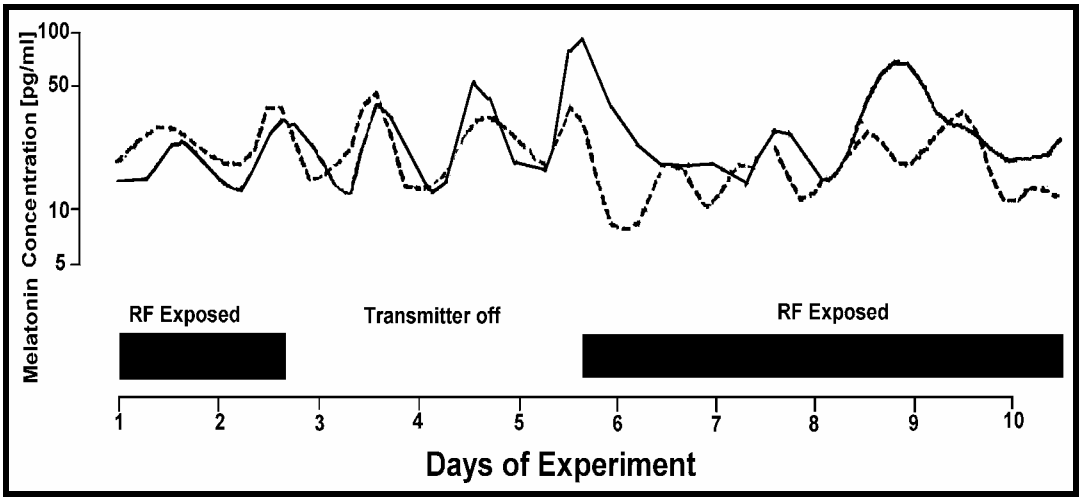

Figure 8: Salival melatonin from two herds of 5 cows, one exposed at 500 m, 0.095μW/cm^2, (solid line) and one "unexposed" at 4000 m, 0.00022μW/cm^2, (dashed line).

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 3.8$\mu W/cm^2$) 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).

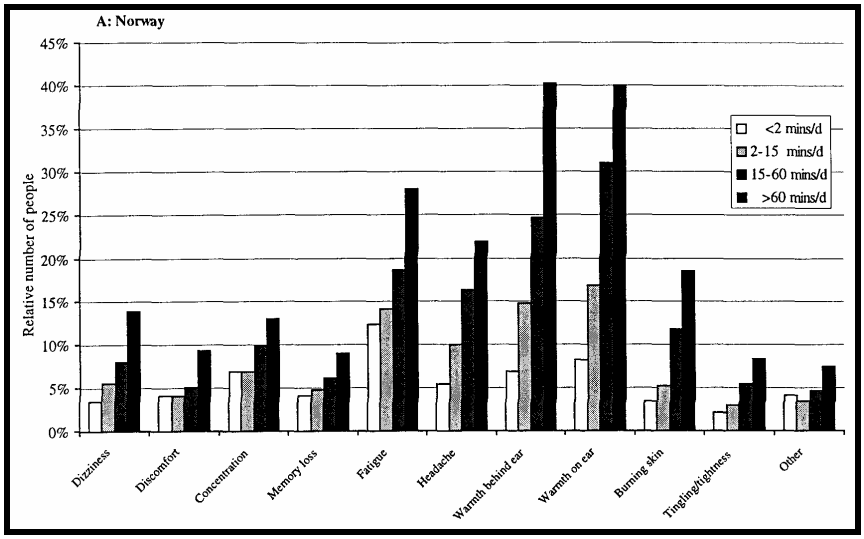

Figure 9: Prevalence of symptoms for Norwegian mobile phone users, mainly analogue, with various categories of length of calling time per day, Mild et al. (1998).

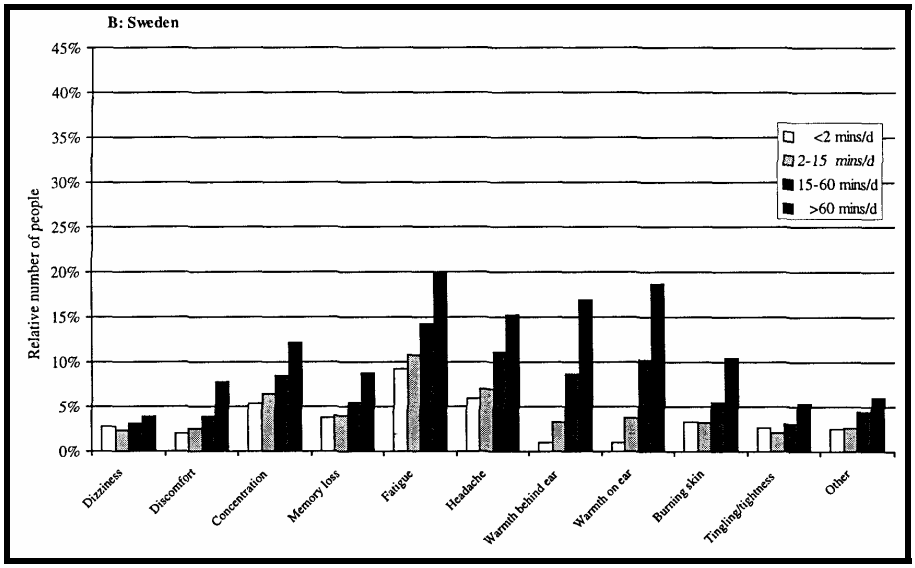

Figure 10: Prevalence of symptoms for Swedish mobile phone users, mainly digital, with various categories of length of calling time per day, Mild et al. (1998).

These are the same symptoms that have frequently been reported as "Microwave Sickness Syndrome" or "Radiofrequency Sickness Syndrome", Baranski and Czerski (1976) and Johnson-Liakouris (1998).

- A Fifteen minute exposure, increased auditory brainstem response and hearing deficiency in 2 kHz to 10 kHz range, Kellenyi et al. (1999).

- Highly significant Increased permeability of the blood brain barrier for 915 MHz radiation at SAR =0.016-0.1 (p=0.015) and SAR = 0.1-0.4 (p=0.002); Salford et al. (1994).

- Significant changes in local temperature, and in physiologic parameters of the CNS and cardiovascular system, Khdnisskii, Moshkarev and Fomenko (1999).

- Reduces the pituitary production of Thyrotropin (Thyroid Stimulating Hormone, TSH):

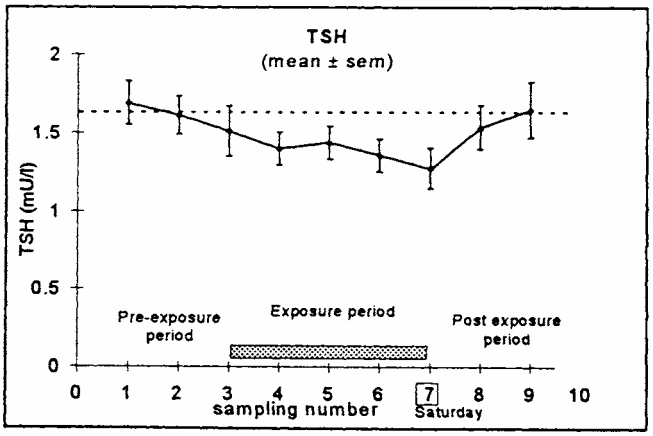

Figure 11: A significant reduction in Thyrotropin (Thyroid Stimulating Hormone) during cell phone use, de Seze et al. (1998).

- Decreases in sperm counts and smaller tube development in testes, Dasdag et al. (1999).

- Increases embryonic mortality of chickens, Youbicier-Simo, Lebecq and Bastide (1998).

- Increases blood pressure, Braune et al. (1998).

- Reduces melatonin, Burch et al. (1997).

- Breaks DNA strands (Verschaeve at al. (1994), Maes et al. (1997), Phillips et al. (1998)).

- Produces an up to three-fold increase in chromosome aberrations in a dose response manner from all cell phones tested, Tice, Hook and McRee, reported in Microwave News, April/May 1999.

- Doubles c-fos gene activity (a proto oncogene) for analogue phones and increases it by 41 % for digital phones, Goswami et al. (1999), altered c-jun gene, Ivaschuk et al. (1997), Increased hsp70 messenger RNA, Fritiz et al. (1997).

- Increase Tumour Necrosis Factor (TNK), Fesenko et al. (1999)

- DNA synthesis and cell proliferation increased after 4 days of 20 min for 3 times/day exposure. Calcium ions were significantly altered, French, Donnellan and Mc Kenzie (1997). Decreased cell proliferation, Kwee and Raskmark (1997), Velizarov, Raskmark and Kwee (1999)

- Doubles the cancer in mice, Repacholi et al. (1997).

- Increases human brain tumor rate by 2.5 times (Hardell et al. (1999)). Associated with an angiosarcoma (case study), Hardell (1999), significant increases in Brain Cancer, Hardell et al. (2000, 2002) and for Astrocytoma Hardell et al. (2002a).

An objective and independent scientific assessment would clearly state, cell phones are a strong risk factor for all of the adverse health effects identified for EMR. Hence, although a specific study is yet to be carried out, there is extremely strong evidence to conclude that cell phones are a risk factor for breast cancer. The biological mechanisms involving hormone change and altered cellular function and damage, and neurological effects, strongly supports the hypothesis that neurological effects found in association with EMR exposures are highly likely to be seen from chronic use of cell phones.

Alzheimer's disease:

Sobel et al. (1995) analysed three independent studies about AD and EMR exposure. All three studies had a consistent OR (2.9, 3.1, and 3.0). The combined results were very highly significant, OR = 3.0, 95%CI: 1.6-5.4, $p<0.001$, and for women OR = 3.8, 95%CI: 1.7-8.6, $p<0.001$. They concluded that the most obvious possibly etiological relevant exposure is that of electromagnetic fields.

Sobel et al. (1996) found that workers in industries with likely electromagnetic field exposure have a very significant ($p=0.006$) increase in incidence of Alzheimer's disease, OR = 3.93, 95% CI: 1.5-10.6. For males the adjusted odds ratio was 4.9, 95% CI: 1.3-7.9, $p=0.01$, and for females, OR = 3.40, 95% CI: 0.8-16.0, $p = 0.01$. They note that:

> **"These results are consistent with previous findings regarding the hypothesis that electromagnetic field exposure is etiologically associated with the occurrence of AD."**

Sobel and Davanipour (1996) outline the etiological process they hypothesize by which EMR produces Alzheimer's disease.

- The first step involves EMR exposure upsetting the cellular calcium ion homeostasis through calcium ion efflux from cells increasing the intracellular calcium ion concentrations. This cleaves the amyloid precursor protein to produce soluble amyloid beta (sAβ).

- sAβ is quickly secreted from cells after production, increasing the levels of sAβ in the blood stream. sAβ then binds to Apolipoprotein E and apolipoprotein J to be transported to and across the Blood Brain Barrier.

- Over time, when sufficient sAβ have been transported to the brain, a cascade of further events lead to the formation of insoluble neurotoxic beat pleated sheets of amyloid fibril, senile plaques, and eventually AD.

The biological mechanism for EMR to cause Alzheimer's disease is well advanced and entirely plausible, commencing with calcium ion efflux.

Multiple Sclerosis in Danish Electric Utility Workers:

A study of 26,124 men working in Danish utility companies were studied for their incidence of multiple sclerosis (MS) in relation to average work-related exposure to electromagnetic fields. A small group of 15 men were shown to have a dose-response incidence of MS as a function of EMF exposure, Figure 12.

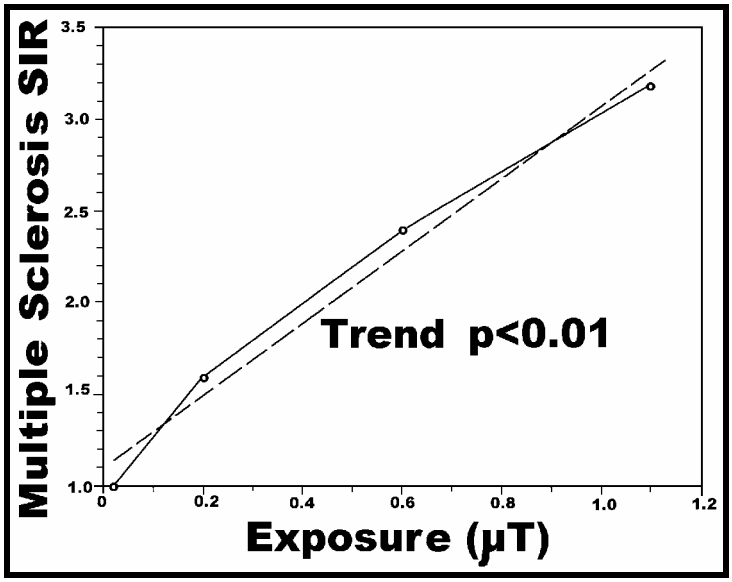

Figure 12: Dose response relationship of Multiple Sclerosis for a small group (N=15) of men occupationally exposed to typical peak magnetic fields in a Danish utility company, Johansen et al. (1999).

The authors conclude that they find no support for the hypothesis. In fact, despite the small sample size, their data shows very strong support for the hypothesis that EMR is associated with adverse neurological effects at extremely low mean exposure levels.

Suicide in U.S. Electric Utility Workers:

A very large study of men working in U.S. electric utility companies included monitoring time weighted average ELF exposures of 2842 people and the identification of 536 deaths from suicide and 5348 controls. For recent exposure and 1 to 5 years of recent exposure there were significant dose-response relationships with cumulative exposure to electromagnetic fields. The recent exposure result is shown in Figure 13.

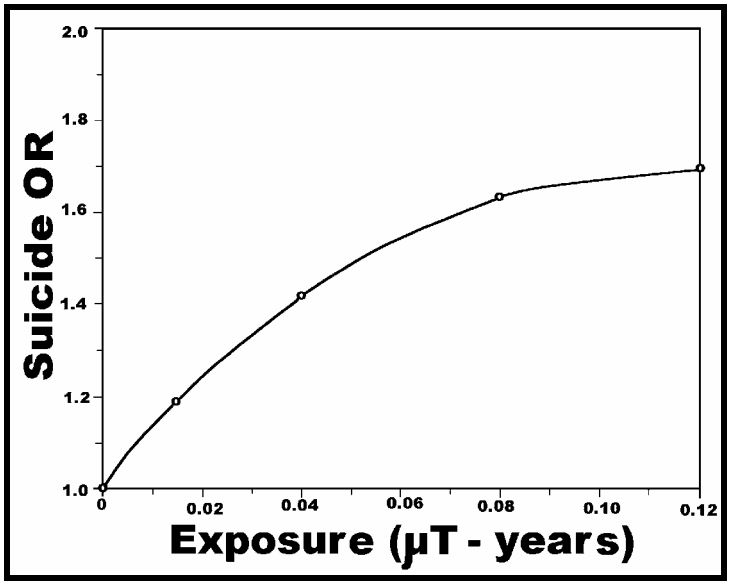

Figure 13: Dose response relationship of Suicide after recent monitored exposure to cumulative 50 Hz magnetic fields for men <50 years, adjusted for work, class, location and exposure to sunlight and solvents, Wijngaarden et al. (2000).

This confirms the results of Perry et al. (1981) who found a highly significant association between suicide and the exposure to magnetic fields from High Voltage Powerlines. Baris and Armstrong (1990) also found RF exposure shows a significant 53% increase in suicide or British Radio and Radar Mechanics, and 156 % increase for Telegraph radio operators. EMR is significantly associated with Clinical Depression, Verkasalo et al. (1997); Psychological symptoms, Beale et al. (1997); and ALS, Savitz et al. (1998a,b). Beale et al. found significant dose-response relationships for several symptoms including depression and anxiety

Non-linear response for neurological effects at extremely low exposure levels are evident in the three studies presented here for sleep disturbance, multiple sclerosis and suicide

MND/ALS, Parkinson's and Alzheimer's Disease:

Welders are occupationally exposed to a combination of lead and strong ELF/RF/MW fields. Welders have increased incidence of MND, OR = 5.3 and for electric plating OR = 8.0, 95%CI: 0.9-72, Strictland et al. (1996).

Electric utility workers are frequently exposed to elevated electric and magnetic fields and sometimes to electric shocks that send high currents through their bodies, including the Motor Neuron part of their central nervous system (CNS). Overall reported electromagnetic field exposures gave for MND/ALS, OR = 3.8, 95%CI: 1.4-13.0. For electric shocks producing unconsciousness, OR = 2.8, 95%CI: 1.2-9.9.

Parkinson's disease was also significantly elevated from ELF exposure, OR = 2.7, 95%CI: 1.1-7.6, Deapen and Henderson (1986). An independent study by Davanipour et al. (1997) compared MND/ALS rates between non-electrical and electrical occupations. They found that the higher the exposure the higher the rate of MND, Figure 14. Savitz, Loomis and Tse (1998) researched neurodegenerative disease and electrical occupations and found elevated Alzheimer's Disease (AD), Parkinson's Disease (PD) and Amyotropic

Lateral Sclerosis (ALS/MND). The highest rates were found in a very highly exposed group, the power plant operators:

For AD, Adj OR = 2.6, 95%CI: 1.3-5.1.

For PD Adj OR = 2.1, 95%CI: 0.9-4.7.

For MND Adj OR = 4.8, 95%CI: 1.9-12.4.

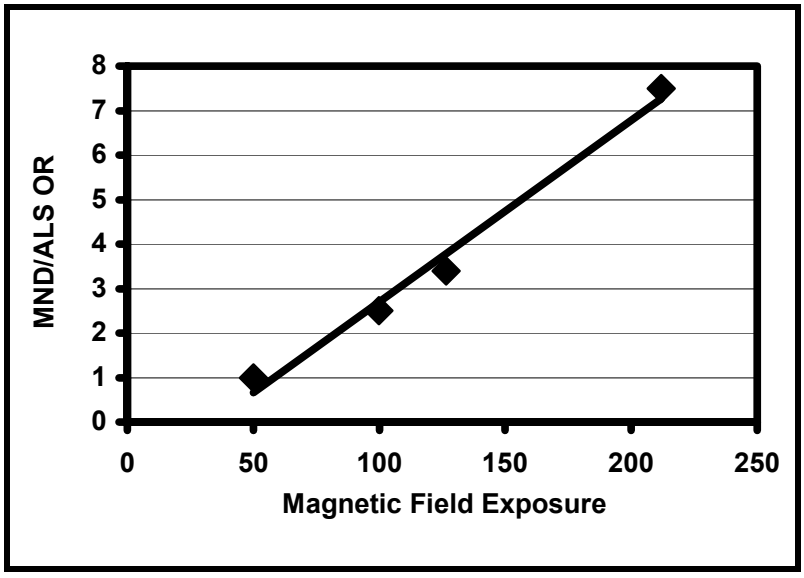

Figure 14: Dose-response increase in MND/ALS from chronic magnetic field exposures in electric utility workers, p<0.02, Davanipour et al. (1997)

A follow-up study, Savitz, Checkoway and Loomis (1998) also found a positive association with duration of electric occupational work and MND (ALS), RR = 2.0, 95%CI: 0.7-6.0. They also found that the longer you worked in these electromagnetic fields the higher the MND rate rose, Figure 15.

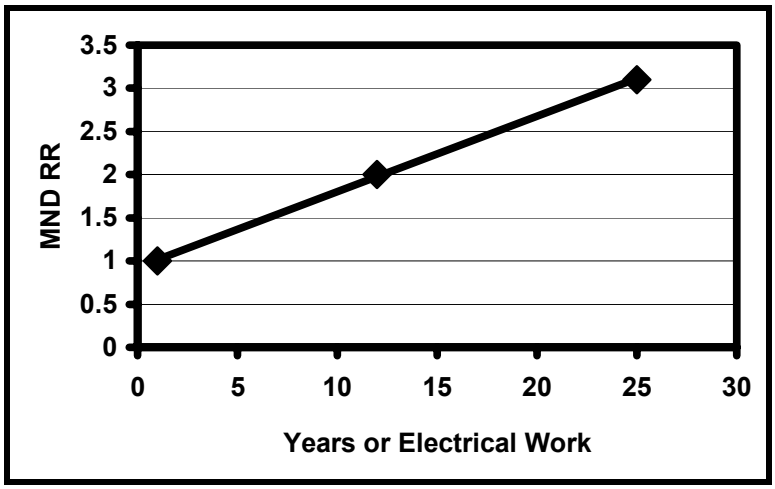

Figure 15: A significant dose-response relationship (p<0.001) between years of electrical work and MND (ALS), Savitz, Checkoway and Loomis (1998).

Electric utility workers in Denmark have the same risk factor for MND as U.S. utility workers, Figure 16. These three dose-response studies of EMF exposure show a causal link between chronic exposure to EMF and Motor Neuron Disease.

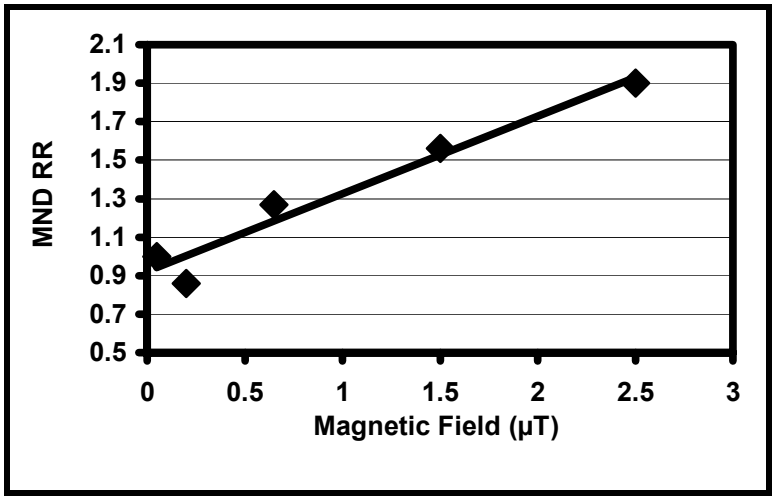

Figure 16: A significant dose-response ($p<0.0001$) increase in MND correlated with long-term mean ELF magnetic field exposures in electric utility occupations, Johansen (2000).

A recent review of Neurodegenerative Diseases in relation to EMF, Ahlbom (2001), identified 7 studies involving MND/ALS and electrical workers. When they were appropriately grouped, each group shows a significantly elevated MND rate, Table 4.

Table 4: Pooling across groups of studies on EMF and ALS, Ahlbom (2001).

Pooled studies	Number of studies	RR	95%C.I.
All	7	1.5	1.2-1.7
Clinically and ALS society based studies	3	3.3	1.7-6.7
Mortality registry and census based studies	2	1.3	1.1-1.6
Utility cohorts studies	2	2.7	1.4-5.0

This review confirms that EMF exposure is various situations significantly increases the incidence and mortality of MND/ALS.

Epidemiology of Brain Tumour:

Eminent academic epidemiologist Dr John Goldsmith reviewed the research of RF/MW health effects. He concludes, Goldsmith (1995):

"There are strong political and economic reasons for wanting there to be no health effect of RF/MW exposure, just as there are strong public health reasons for more accurately portraying the risks. Those of us who intend to speak for public health must be ready for opposition that is nominally but not truly, scientific. At present there seems to be little interest in or understanding of epidemiologic information among regulatory bodies that should provide protection."

Goldsmith (1997):

"Available data suggest that RF radiation be considered a carcinogenic risk, a position already taken in an internal U.S. E.P.A. document in 1990 when there was much less evidence of the potential harmfulness of RF radiation."

The evidence in this report confirms Dr Goldsmith's conclusions.

Early evidence of brain tumor associated with EMR exposure

Our strong attraction to the evident benefits of electronic technology easily masks the very strong evidence of the adverse health effects in our brains and bodies which radiation from this technology causes. This situation was described very clearly by Zaret (1977):

> **"As we are all well aware, many special societal benefits have been derived from the electronic revolution. What has not yet been established are the risks associated with exposure to stray radiation. In this context, my purpose in writing is to call attention to one largely over-looked possibility, that nonionizing radiation as an atmospheric pollutant may be carcinogenic."**
>
> **"Despite the paucity of published information a number of clusters, each by definition consisting of 2 or more cases, are known to exist."**
>
> **"Regarding the clusters of our immediate concern, one instance of brain tumors (2 cases with astrocytoma) appeared in a small group of workers (about 18) servicing microwave communication equipment."**
>
> **"Space limitations do not permit my developing a rationale nor citing the large number of supportive references already at hand, beginning with Heller and Teixeira-Pinto in 1959, which demonstrate that nonionizing radiation can induce mutagenesis."**

Zaret (1977) identifies 2 cases of astrocytoma (the most common primary brain tumour) in a group of 18 microwave exposed workers. Age standardized astrocytoma rates in the 1970's were about 6 per 100,000 p-yrs. Assuming a period of 25 years for these workers' exposures, this gives RR = 74.1, 95%CI: 15.0-367, $p<0.0001$, which is extremely significant. Heller and Teixeira-Pinto (1959) showed that pulsed RF radiation significantly increased chromosome damage.

Brain Tumour with VDT exposure:

Beall et al. (1997) found significant increases in brain tumour, especially glioma, among long-term workers using computers who are exposed to a mix of ELF and RF radiation from the VDTs. For long-term computer users, Engineering/technical users show a non-significant dose response, but computer programmers show a significant dose-response relationship, Figure 17.

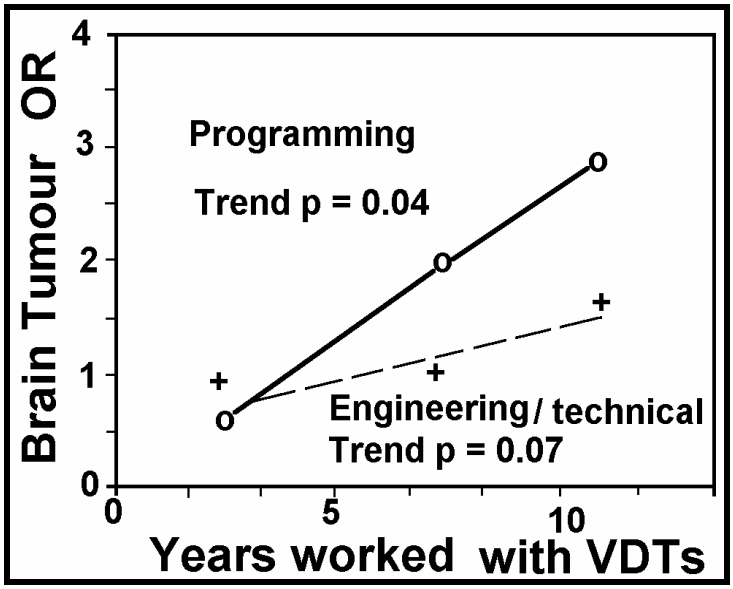

Figure 17 Dose-response increases in brain tumour from years of working with computers, Beall et al. (1997).

A search of the epidemiological literature, with the assistance of MEDLINE, and medical school libraries, reveals 96 studies covering over 420 separately exposed groups, that reveal that brain tumour incidence or mortality is increased with EMR exposure across the spectrum. Table 6 lists 28 studies showing dose response or significant dose response increases in brain tumour incidence with EMR exposure.

Table 5: Summary table of studies and groups showing elevated brain cancer from exposure to electromagnetic radiation across the EMR spectrum.

Total studies showing elevated brain cancer:	**96**
Total groups showing elevated brain cancer:	**423**
Total studies showing at least one significantly elevated brain cancer	**43**
Total groups showing significantly elevated brain cancer:	**153**
Total studies showing at least one dose response relationship	**28**
Total groups showing dose-response trends of brain cancer:	**78**
Total groups showing significant dose-response trends $p \leq 0.1$	**64**
Total groups showing significant dose-response trends $p \leq 0.05$	**48**

The studies showing dose-response relationships for EMF/EMR exposures are summarized in Table 6. Since the degrees of freedom (n-2) are very small for dose-response trends it is appropriate to use a threshold of $p \leq 0.1$. Despite this the number of trends have $p \leq 0.05$ and many have $p < 0.01$.

Table 6: Studies showing dose-response relationships for EMR exposure and brain tumor:

- Denver, United States, residential fields. Wertheimer and Leeper (1979)
  Childhood) Brain cancer is near 26% of all cancer.
  All cancer) Dose related for children living at same address, p<0.008.

- Electrical Occupations in Maryland, U.S. Lin et al. (1985)

	Glioma/Astrocytoma		Other Brain Tumors	
Definite Exposure	2.15 (1.10-4.06)*	n = 27	1.54 (0.68-3.38)	n = 15
Probable exposure	1.95 (0.94-3.91)	n = 21	1.30 (0.60-2.78)	n = 19
Possible exposure	1.44 (1.00-1.95)*	n =128	0.94 (0.68-1.31)	n = 87
No exposure	1.0	n =323	1.0	n =286
	Trend p<0.01		Trend p<0.05	

- Eastern U.S. Electronic Industries Thomas et al. (1987)
  Astrocytic brain tumours RR=4.9 (1.9-13.2)*

		Duration Employed (yr)		
	Unexposed	<5	5-19	≥ 20
RR	1.0	3.3	7.6	10.4
Solder fume adjusted RR	1.0	1.65	3.8	5.2
		(trend p<0.05)		

- United States, Meta Analysis Kheifets et al. (1995)
  Electrical Occupations

Pooled Dose-Response, Reference	RR = 1.0	
Low	RR = 1.23 (1.06-1.42)	
Middle	RR = 1.36 (1.11-1.68)	
High	RR = 1.61 (1.28-2.04)	Trend p=0.006

- East Texas, Males Glioma Speers, Dobbins and Miller (1988)
  n=202
  Electricity or electromagnetic fields OR = 3.94 (1.52-10.20)* Trend: p<0.01

- Los Angeles County, Occupational exposure Preston-Martin et al.(1989)
  High exposure to electric and magnetic fields n=272

Glioma 0 years	OR = 1.0	
Glioma 0-5 years	OR = 1.4 (0.7-3.1)	
Glioma >5 years	OR = 1.8 (0.8-4.3)	Trend p =0.05
Astrocytoma, >5 years empl.	OR = 4.3* (1.2-15.6)	Trend p = 0.008

- U.S., prenatal domestic appliances Savitz, John and Kleckner (1990)
  Electric Blanket usage:

Night duration use <8 hrs	OR = 1.5 (0.4-5.7)	n=3
Night Duration use = 8 hrs	OR = 3.1 (1.2-8.5)*	n=7
Night Duration use >8 hrs	OR = 4.5 (0.5-39.0)	n=1
(Assuming 4, 6 , 8, 10 hours)	Trend p = 0.019	

- United States, 16 States (Mortality) Loomis and Savitz (1990)
  adjusted for age & race

Dose-response relationship:

Exposure	Crude	Adjusted (age & race)
Possible only (Adj)	OR = 1.8* (1.4-2.3)	OR = 1.5* (1.1-1.9)
Unlikely (low) exposure (Adj)	OR = 1.3* (1.0-1.6)	OR = 1.2 (0.9-1.5)
Reference Control	OR = 1.0	OR = 1.0

- Los Angeles Country, electrical industry Mack et al. (1991)

Stratification from years worked in exposed situations:
Exposure Index 1, from Thomas et al. (1987)

Type of brain tumor	0	>0-5	>5-10	>10	Trend-p
All brain tumors	1.0	1.1 (0.6-2.0)	0.5 (0.1-2.0)	1.3 (0.3-3.0)	0.67
Glioma	1.0	1.1 (0.5-2.1)	0.4 (0.1-2.1)	1.7 (0.7-4.4)	0.21
Astrocytoma	1.0	1.1 (0.5-2.9)	0.4 (0.1-2.1)	10.3 (1.3-80.8)*	0.01**

Exposure Index 2, from Milham (1985)

Type of brain tumor	0	>0-5	>5-10	>10	Trend-p
All brain tumors	1.0	1.1 (0.6-2.2)	0.4 (0.1-1.7)	1.2 (0.5-2.8)	0.70
Glioma	1.0	1.2 (0.6-2.4)	0.4 (0.1-2.1)	1.4 (0.6-3.5)	0.32
Astrocytoma	1.0	1.3 (0.5-3.1)	0.4 (0.1-2.1)	4.6 (1.0-21.4)*	0.02*

- Sweden, Occupational exposure Floderus et al. (1993)

Time Above 0.2mT,	<16%	17-23%	24-28%	≥29%	≥39%	Trend p
All brain tumors	1.0	1.3 (0.9-2.0)	1.3 (0.9-1.9)	1.5 (1.0-2.2)*	1.9 (1.2-3.1)*	0.005**
Astrocytoma III-IV	1.0	1.6 (1.0-2.6)*	1.6 (1.0-2.5)*	1.7 (1.1-2.8)*	2.1 (1.2-3.8)*	0.011*

Median Exposure	<0.11μT	0.12-0.16μT	≥0.17μT	≥0.20μT	
Astrocytoma I-II<40	1.0	0.9 (0.3-2.9)	2.7 (1.1-6.8)*	5.7 (1.9-16.7)*	0.035*
Adjusted for Benzene	1.0	1.0 (0.7-1.5)	1.4 (1.0-2.0)*	1.6 (1.0-2.5)*	0.051
Possible solvents	1.0	0.9 (0.5-1.9)	1.4 (0.9-2.2)	1.9 (0.8-4.5)	0.066

Median Exposure	≤0.15μT	0.20-0.28μT	≥0.29μT	≥0.41μT	
Study participants	1.0	1.1 (0.8-1.5)	1.2 (0.9-1.7)	1.4 (0.9-3.3)	0.003**
Study and Nonrespondents	1.0	1.0 (0.8-1.4)	1.2 (0.9-1.6)	1.3 (0.9-2.0)	0.05*
Age Specific ≤40					
Study participants	1.0	0.8 (0.4-1.8)	1.4 (0.7-3.0)	2.7 (1.0-7.8)*	0.094
Study and Nonrespondents	1.0	0.9 (0.5-1.9)	1.6 (0.8-3.3)	2.9 (1.1-7.7)	0.065

- Canada, Provincial Residential Electric Consumption (REC) Kraut et al. (1994)
  Childhood brain cancer significantly increases with REC in a significant dose-response manner.

- Ontario, Quebec and France Theriault et al. (1994)

Dose-response for the median, 90th %ile, with a strongly skewed distribution, using exposure weights of 15, 25 and 92.

Ontario Hydro: Malignant Brain Cancer: Trend p-value

Median: OR = 1.85 (0.53-6.49) >90th OR = 5.45 (0.59-50.59) p=0.036*

All Companies: Malignant Brain Cancer:
0-20 yrs Median: OR = 1.05 (0.20-5.35) >90th OR = 5.90 (0.37-94.91) p=0.065
All Median: OR = 1.95 (0.98-3.86) >90th OR = 2.14 (0.80-5.72) p=0.44

All Companies: Astrocytoma:
0-20 yrs Median: OR = 3.99 (0.72-22.0) >90th OR = 11.1*(1.44-85.6) p=0.10
All Median: OR = 3.69 (0.61-22.2) >90th OR = 28.48*(1.76-461) p=0.02*

Cumulative exposure groups
Astrocytoma Trend OR = 9.41* (1.07-82.79)

- U.S. Electrical Workers, 1950-1988 Savitiz and Loomis (1995)
  Measured field assessment and confounders.

Cumulative exposures and duration windows:

Total Exposure (RR)

0-<0.6		0.6-<1.2		1.2-<2		2-<4.3		≥4.3		Trend p
1.0	1.61	0.99-2.63	1.47	0.84-2.56	1.65	0.92-2.95	2.29*	1.15-4.56	0.033*	

Past 2-10yrs

0		0-<0.2		0.2-<0.4		0.4-<0.7		≥0.7		
1.0	1.17	0.66-2.08	1.39	0.75-2.58	1.46	0.76-2.84	2.56*	1.35-4.86	0.009**	

Past 10-20 yrs

0		0-<0.3		0.3-<0.5		0.5-<0.9		≥0.9		
1.0	1.76*	1.07-2.91	1.26	0.69-2.29	1.47	0.76-2.84	1.63	0.92-2.90	0.44	

Past >20

0		0-<0.4		0.4-<1.1		1.1-<2.0		≥2.0		
1.0	0.76	0.45-1.27	0.89	0.51-1.56	1.12	0.59-2.14	1.26	0.64-2.48	0.074	

Mortality	Dose-response rates	RR per μT-yr	95%CI
	Total Exposure	1.07*	1.01-1.14
	Past 2-10 years	1.94*	1.34-2.81
	Past 10-20 years	1.35*	1.01-1.79
	Past>20 years	1.06	0.97-1.16

- U.S. electronics industry workers Beall et al. (1996)

Dose-response relationships:

Computer Programmers (>10 yrs)	OR = 2.8 (1.1-7.0)*	Trend p = 0.04
Engineering/Technical (>10 yrs)	OR = 1.7 (1.0-3.0)*	Trend p = 0.07
Glioma, All subjects, 5yr progrm.	OR = 3.9 (1.2-12.4)*	Trend p = 0.08

- United States, office workers Milham (1996)
  Transformer fields SIR = 389 (156-801)* N=410
  Exposure trend p=0.0034
  Employment period trend p<0.05

- Ontario Hydro male employees (Adjusted ORs) Miller at al. (1996)

Brain Tumour	Mod. Field	OR = 1.27 (0.32-5.41)	
	High Field	OR = 1.33 (0.52 -10.8)	Both show trends.
Benign Brain	Mod. Field	OR = 5.38 (0.42-69.3)	
Tumour	High Field	OR = 5.64 (0.3-105)	

- France, electrical utility workers, ELF exposures Guenel et al. (1996)

Crude and adjusted on Magnetic Fields (MF), Socioeconomic Status (SES) and solvent exposures (SOL), given with exposure percentiles:

Percentiles:	Cases	OR Adj SES	OR Adj MF+SES	OR Adj. SES+SOL
<50	29	1.0	1.0	1.0
50-<75	22	2.47 (0.99-6.16)	2.51 (1.00-6.34)*	2.29 (0.89-5.94)
>75-90	8	1.43 (0.46-4.45)	1.43 (0.45-4.48)	1.43 (0.45-4.57)
>90	10	3.08 (1.08-8.74)*	2.83 (0.97-8.28)	2.97 (1.00-8.80)*

Adjusting for a 5-year latency gives the highest Odds ratios:

Cumulative Exposure V/m-years	Cases	OR	95%CI
<202	22	1.00	
202-274	20	3.43*	1.25-9.40
275-342	9	2.40	0.73-7.90
≥343	9	3.69*	1.10-12.43

Adjusting for 10-year latency gives a linear dose-response:

Cumulative Exposure V/m-years	Cases	OR	95%CI
<166	22	1.00	
166-229	14	1.67	0.67-4.19
230-294	9	1.79	0.60-5.36
≥295	7	2.15	0.63-7.26
		Trend p = 0.013	

- Children in Los Angeles Preston-Martin et al. (1996b)

Dose-response relationship:

All years	Reference <1mG	OR = 1.0	
	>2mG	OR = 1.2 (0.5-2.8)	n=16
	>2.5mG	OR = 1.4 (0.5-3.8)	n=13
	>3mG	OR = 1.7 (0.6-5.0)	n=12
		Trend p = 0.036	

- Norway Tynes and Haldorsen (1997)

Children	<0.05μT	0.05-<0.14μT	>0.14μT	n=10
RR =	1.0	2.6 (0.5-12.0)	2.3 (0.8-6.6)	p=0.07

- Sweden, Electrical Occupations Rodvall et al. (1998)

Dose-response for groups with >10 cases:

Estimated daily Mean:	<0.2μT	0.2-0.4μT	>0.4μT	>0.4μT for ≥5yrs
Glioma (Mean)	1.0	1.1 (0.4-2.7)	1.9 (0.8-5.0)	1.8 (0.7-5.1)

Estimated daily Median:	<0.12μT	0.12-0.19μT	>0.19μT	>0.19μT for ≥5yrs
Glioma (Median)	1.0	1.1 (0.5-2.6	1.4 (0.5-3.6)	1.5 (0.6-4.1)

Dose-response

Estimated daily Mean:	<0.2μT	0.2-0.4μT	>0.4μT
Glioma (Mean)	1.0	1.3 (0.0.7-2.4)	1.3 (0.5-3.2)
Estimated daily Median:	<0.12μT	0.12-0.19μT	>0.19μT
Glioma (Median)	1.0	1.9 (1.0-3.5)	1.1 (0.5-2.6)

- United States Utility Workers — Kheifets et al. (1999)
  5 studies combined
  RR per 10 μT-years — RR = 1.12 (0.98-1.28)

- Swedish cohort study of ELF exposures — Floderus, Stenlund and Persson (1999)
  Occupational Exposure of Males:

1971-1977	Middle Exp 0.084-0.115μT	High Exp ≥0.116μT
Astrocytoma III-IV	RR = 1.1 (0.9-1.4)	RR = 1.2 (1.0-1.5)*
1971-1984	Middle Exp 0.084-0.115μT	High Exp ≥0.116μT
Astrocytoma III-IV	RR = 1.2 (1.1-1.4)*	RR = 1.3 (1.2-1.5)*

- United States Electric Utility Workers — Savitz et al. (2000)

Reference	OR = 1.0
Cumulative exposure	OR = 1.8 (0.7-4.7)
Average exposure	OR = 2.5 (1.0-6.3)*

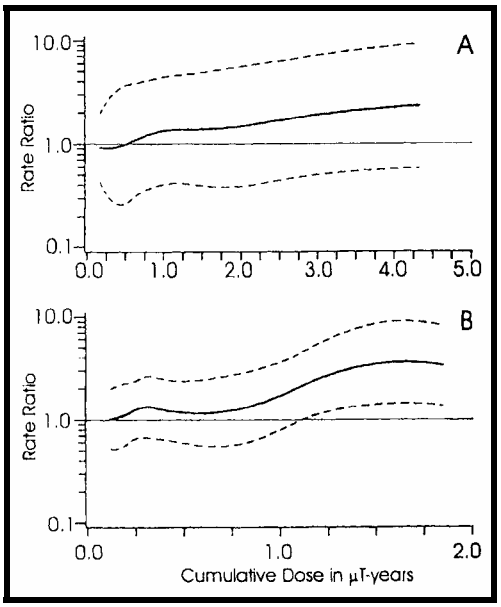

Figure 18: Brain cancer increases from refined case-cohort job-exposure matrix in the US Utility Worker Mortality Study 1950-1988, Savitz et al. (2000). Cumulative exposure over entire career with A: a 2-year lag; B: 2-10 years in the past.

- Cell phone users in Denmark — Johansen et al. (2001)

Duration of digital subscription		<1 yr	1-2yrs	≥3 yrs
Relative to reference group	SIR	0.7	0.9	1.2
Relative to <1 yr group	RR	1.0	1.29	1.71

- San Francisco, Sutra Tower (FM/TV) Cherry (2002)
  Children < 21 yrs Brain Cancer

Ring (km)	RR	95%CI	
0.1-1	15.5	3.14-76.8	
1-2	7.8	2.1-30.9	
2-3	3.3	0.84-13.4	
3-4	3.2	0.85-12.1	
4-5	3.07	0.81-11.6	Trend p-valuep=0.03
>5	1.00		Log/Lin Trendp<0.001

- Canadian Electrical Workers Villeneuve et al.(2002)

Highest average exposure	<0.3μT	≥0.3μT	≥0.6μT
All Brain Cancer OR(adj)	1.0	1.12 (0.83-1.51)	1.33 (0.75-2.36)
Glioblastoma multiforme	1.0	1.48 (0.89-2.47)	5.36 (1.16-24.78)
Other Brain Cancers	1.0	1.10 (0.58-2.09)	1.58 (0.56-4.50)
Average exposure	<0.3μT	≥0.3μT-<0.6μT	≥0.6μT
All Brain Cancer OR(adj)	1.0	1.13 (0.72-1.79)	1.50 (0.69-3.28)
Glioblastoma multiforme	1.0	1.99 (0.83-4.81)	12.59 (1.50-105.6)

- Cell phone users in Finland Auvinen et al. (2002)
  Significantly elevated brain cancer and salivary gland cancers from analogue phone usage.

  Trends of OR for years of usage are:

All Brain Cancers,	Analogue Phones	1.2* (1.0-1.3)
	Total Phones	1.1* (1.0-1.3)
Glioma	Analogue Phones	1.2* (1.1-1.5)
	Total Phones	1.2* (1.0-1.4)
Other Brain cancers	Analogue Phones	1.1 (0.8-1.4)
	Total Phones	1.1 (0.8-1.4)
Salivary gland cancers	Analogue Phones	1.3 (0.7-2.5)
	Digital Phones	1.5 (0.2-11.9)
	Total Phones	1.3 (0.7-2.6)

Summary of Brain Cancer studies:

The epidemiological studies on EMR associated brain cancer include 96 studies of over 400 separate groups with elevated brain cancer from EMR exposure from across the spectrum. Of the 423 groups, 153 show significant increases, 78 show dose response relationships and 48 of these are statistically significant. From acknowledged RF/MW exposure there are 35 groups, 19 with significant increases, 8 with dose responses, of which 6 are significant. This is strongly backed by evidence of genotoxicity. This evidence is more than sufficient, using the Bradford-Hill guidance, Hill (1965), to conclude that there is a causal relationship between EMR exposure across the spectrum and brain tumour incidence and mortality

Significant increases in brain cancer incidence or mortality are shown in welders, equipment repairers, microwave repairers, utility workers, telephone industry workers, amateur radio operators, military personnel exposed to radio and radar, commercial and

air force pilots and aircrew, computer users, children living near broadcast towers and high voltage powerlines and cell phone users.

Cell phones and Brain tumours:

Analogue cell phones use FM radio signals while digital cell phones use pulsed microwaves that are very similar to radar. The analogue phones expose the user's head to much higher intensities of microwaves than the more modern digital phones. The way the information in encoded in the pulsed differs but it is the pulsing action that is likely to enhance the biological effects. Since FM exposed populations show significant increases and significant dose-response increases in brain tumour at very low exposure levels, Cherry (2002a), and since cell phone produce far high mean exposures and the same neurological effects in a dose response manner, Mild et al. (1998), then analogue cell phones are highly likely to produced significant increases in brain cancer and degenerative neurological diseases.

Dr Andrew Davidson, an Oncologist in Western Australia, Davidson (1998), has noted a parallel increase in diagnosed brain tumours with the rising use of mobile phones. In a comment Dr Bruce Hocking refers to Rothman et al. (1996) who compared the mortality rates during 1994 of portable (hand-held) phone users and mobile (Bag) phone users. Dr Hocking quotes the results of RR = 0.86 to show that mobile phone users have a lower mortality rate. Actually Rothman et al. have deliberately reversed the ratio so that people will take the conclusion that Dr Hocking has drawn by reporting the rate of portable phone use over mobile phone use. When it is corrected, and mobile phone use is compared with portable phone use, for all users and for male users, mobile phone users have significantly higher mortality rates than portable phone users.

Table 7: Comparative mortality rates between mobile (Bag) phone users and portable (hand-held) phone users in the United States for 1994, Rothman et al. (1996).

Group	RR	95%CI	p-value
For Men	1.40	1.06 - 1.86	0.017
For Women	1.52	0.78 - 2.95	0.31
All People	1.38	1.07 - 1.79	0.013

Hardell et al. (1999) found no overall increase in brain cancer risk for mobile phone users compared with the general population. However they did find a marginally insignificant increase in a particular type of tumour at the position and side of the head that received the highest exposure from the mobile phone, based on 209 cases and 425 controls. Left side OR = 2.4, 95%CI: 0.52-10.9 and Right Side OR = 2.45, 95%CI: 0.78 - 7.76. They found a slightly higher rate for the digital GSM phones compared with the analogue NMT phones.

Dr George Carlo, former chairman of Wireless Technology Research (WTR), the industry funded research body in the U.S., reported that the studies they had funded had reported similar results to those in Sweden,

Digital phones are like radar and radar exposure has been shown to significantly increase brain tumour, Szmigielski (1996). Tice et al. (1999) reported that every cell phone tested in their U.S. laboratory caused chromosome aberrations in a dose response manner up to a 3-fold increase. Phillips et al. (1998), Maes and Verschaeve and Maes (1998) observed

significant DNA damage from a range of digital cell phone signals. Phillips et al. used exposures of 1.2 and 13μW/cm^2.

The largest and most careful case-control cell phone usage and brain cancer studies have been carried out in Sweden, Hardell et al. (1999, 2000, 2001, 2002a,b). Initially small case samples (n=270) showed elevated brain cancer from using an analogue mobile phone. When the results included more cases and adjusted the results for Xray and therapy exposures, the incidence of Brain Cancer on the side of the head that was exposed was significantly elevated, OR = 2.62 (1.02-6.71). The study group was significantly expanded to include 1429 cases. Using a cell phone for longer than a year raised the risk of Brain Cancer, OR = 1.26 (1.02-1.56). For longer latency periods, > 5 years gave OR = 1.35 (1.03-1.77) and > 10 years OR = 1.77 (1.09-2.86). For the side of the head the phone exposed OR = 2.50 (1.2-4.88).

For Acoustic Neuromas among analogue phone users OR =3.27 (1.67-6.43). The final study involved only patients with Astrocytomas (n=414). For analogue phone use OR = 1.29 (0.87-1.90) and digital phone use OR = 1.1 (0.81-1.53). When the side of the head where the phone was used was considered, for Analogue phone OR = 1.85 (1.12-3.39) for all Brain Cancers and OR = 1.95 (1.12-3.39) for Astrocytomas. For digital and cordless phones, the risk of side of head astrocytomas was OR= 1.59 (0.98-2.58) and OR= 1.70 (1.06-2.74) respectively, Hardell et al. (2002a). For astrocytomas in the temporal or occipital areas, OR=9.00 (1.14-71.0) based on 12 cases and 5 controls, Hardell et al. (2002b).

Summary and Conclusions:

Has a very sensitive electromagnetic organ. Since the published research shows that electromagnetic fields and radiation significantly enhance neurological responses by the brain acute exposure to mobile telephones. And there's a large body of evidence showing that people who live and work in electromagnetic fields have a significantly higher rate of neurological disorders, including Motor Neuron Disease, Alzheimer's disease, Parkinson's disease, and enhance epileptic fits. There is also robust evidence that there is a causal relationship between sleep disturbance, depression, suicide and brain cancer from chronic exposure to electromagnetic fields and radiation. The symptoms of radiofrequency or microwave syndrome must not be dismissed as simply subjective reactions. They are real and biologically sensible responses of an electromagnetic organ to electromagnetic interference.

All of these symptoms and disease rates are being enhanced by electromagnetic fields of homes, radiofrequency fields and radio and TV towers and the extensive installation of cell sites and the use of cellular telephones.

The causal association of neurological diseases and mortality from residential exposures to electromagnetic fields for about one million times higher than the Schumann Resonance signal, and cell phone exposures of one billion times higher than Schumann Resonance signal, there is highly scientifically sensible and well-established by multiple independent epidemiological studies.

Therefore public health protection standards should be set at 10nW/cm^2 to significantly reduce the risk rate in the public from the well identified and serious neurological symptoms, diseases and mortality rates.

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