

**Actual or potential effects of ELF and  
RF/MW radiation on enhancing violence  
and homicide, and accelerating aging of  
human, animal or plant cells.**

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## **Abstract:**

The brain is a very sensitive Bioelectromagnetic organ and through classical resonance processes can be halted and damage of external electromagnetic fields and radiation. This review will explore the possibility that this could result in violence enhanced rates of homicide. The evidence that electromagnetic fields and radiation electromagnetic are genotoxic means that exposure to any electromagnetic fields and radiation will enhance cell death (Apoptosis). The natural ageing process involves oxygenated free radicals from the breathing process causing enhanced rates DNA damage, cancer and cell death. Exposure to electromagnetic fields and radiation also reduces melatonin which limits a body's ability to scavenge the free radicals and therefore contributes to enhanced Apoptosis and cancer rates. Melatonin is also necessary for a healthy immune system. Reduced melatonin is also associated depression and suicide and therefore is likely to be associated with violence of homicide. Since electromagnetic radiation damages the DNA and reduces melatonin it is scientifically logical that it also enhances many of the natural ageing process in people, animals and plants. These conclusions are strongly supported by robust evidence that natural weather related effects are caused by natural electromagnetic fields and radiation with extremely small intensities. Therefore it is logical and proven that humanly generated fields and radiation at intensities from a thousand to many billion times higher, also significantly enhance a wide range of adverse health effects, including cancer, heart disease, sleep disturbance, depression, suicide, anger, rage, violence, homicide, neurological disease and mortality.

## **1. Introduction:**

### **1.1 Brief:**

This report was commissioned by Bruce Ratcliff on behalf of Ratcliff Company Inc. in a letter dated 30 March 1998, with the following brief:

"We would like to know more about any possible relationship between microwave radiation as given off from relay towers and cell phones and premature, rapid ageing of humans, animals and/or plants. Please see page 22 in Firstenberg report on plants.

We recently had three news events which startled the public here in the U.S. They are listed as follows:

1. One where two apparently normal 11 and 13 year old boys shot and killed 4 people and wounded many others apparently with no known cause.

2. A mother in San Francisco smothered and killed her 3 babies. She was on medication which may have been a factor.
3. In another case in San Francisco, a student brought a gun to school and fired off a round.

What is the known and/or classified research relating microwave radiation to irritability, anger and violence?"

## 1.2 Context:

Levels of anger and violence in society are serious causes for concern. Well understood relationships between adult behaviour, parenting approaches, media, and movies and anger and violence, along with stress and frustration which can lead to rage such as road rage, and particularly the roles of alcohol and drugs, are associated with the development of lower self control, and more anger, rage and violence. This makes the social and environmental factors difficult to isolate.

This is not to say that environmental factors such as exposure to toxic substances in the environment should be ignored because of the difficulties involved. Rather, if there is evidence of potential or actual involvement of environmental toxins in enhancing the risk of anger, rage and violence, then it is vital that this be identified and addressed in order to develop strategies to reduce or minimize that particular risk factor. A toxic substance which is ubiquitous throughout a city, state or the whole country, would be of particular concern because of the vast number of people exposed to this enhanced risk.

The subject of this investigation is electromagnetic radiation in the radiofrequency and microwave (RF/MW) part of the electromagnetic radiation (EMR) and the ELF parts of the spectrum. This consideration is extremely important because humanly sourced EMR is ubiquitous and exposure levels are rising exponentially and at a rate which outstrips biological adaptation.

## 1.3 Biological Context:

For too long the RF/MW debate has been dominated by physics and physical concepts and arguments. These have been primarily revolved around exploration of the heating effects of RF/MW and determining "safe" levels of exposure which will avoid dangerous levels of tissue heating.

Many EMR researchers have noted the folly and error of this, e.g. Adey (1981), Frey (1994), and Goldsmith (1994). Human, animal and plant cells are bio-electro-chemical structures which interact in many ways including physically, chemically, biochemically and bioelectrically. Adey (1979) expresses this concern forcibly and scathingly:

**"Faced with the overwhelming complexity of the brain as a tissue and an organ of the mind, physical scientists and medical researchers alike have all too often retreated shamelessly into classicisms and the argots of their respective trades. Too many physicists and engineers cling desperately to thermal models as the alpha and omega of bioeffects from non-ionizing radiofrequency fields, shunning the exquisite beauty of long-range molecular**

interactions and resonant processes in biological macromolecules. In like fashion, medical physiologists, challenged by phenomena I have discussed here, have turned away and fixed their eyes in a glassy stare on the comparative crudity of ionic equilibria as the be-all and end-all of excitatory processes as described in the massive ionic exchanges of Hodgkin-Huxley models.

True science can never be a popularity contest. The time has surely come when we should place these scholasticisms of another age in a proper context, counting ourselves thrice blessed at the prospect that through the use of non-ionizing radiofrequency radiation as a research tool, the intrinsic organization of the brain tissue, the subtleties of neuroendocrine phenomena and the broad sweep of immunological interactions may at least be understood in terms of transductive coupling at the molecular level.”

#### **1.4 Summary of the Resolution from the European Parliament, 1992:**

The European Parliament, after a series of hearings on the matter of EMR health effects expressed grave concern (Resolution B3-0280/92):

**“Thus in the frequency range 100 kHz to 300 GHz, 50 years ago it was scarcely possible to measure 10 pW/cm<sup>2</sup> on the ground in our countries. Today, depending on the location, values one million to one thousand million times higher are recorded because of the explosion of telecommunications.”**

and the following clauses:

- A. having regard to the significant increase, in the environment, of power density of non-ionizing electromagnetic radiation in the various frequency ranges, associated with technological development over the last few decades,**
- B. having regard to the precautionary principle included in Article 130r of the Treaty establishing the European Community and the ALARA principle (a-slow-as-reasonably-achievable), according to which it is necessary, in this case to minimize exposure to electromagnetic radiation,**
- C. whereas such radiation interacts with matter by non-thermal mechanisms and whereas, as regards radiofrequencies and microwaves, these are therefore added to the purely thermal interaction mechanisms,**
- D. whereas, according to an increasing number of epidemiological and experimental studies, even slight exposure to non-ionizing electromagnetic fields increases the risks of cancer, can be accompanied by nervous disorders and disruption of the circadian rhythms and seems capable of affecting developing organisms,**
- E. whereas the results of many *in vivo* and *in vitro* studies show increasing clearly the interaction mechanisms underlying such disorders and illnesses, centred mainly in cell membrane, lead to disruption of melatonin secretions, ornithine**

**decarboxylase activity and T-lymphocyte efficacy, testifying to the probable role of non-ionizing radiation in promoting cancer,**

**F. whereas synergy phenomena must be expected between non-ionizing radiation and other physical agents, ...**

In clause D the European Parliament (E.P.) agree that electromagnetic fields affect the nervous system by nervous disorders and disrupts the circadian rhythms, such as sleep disruption leading to chronic fatigue syndrome, Altpeter et al. (1995). These also have a relation to behaviour, stress and advanced aging effects.

### **1.5 This Report's Approach:**

This review takes a number of innovative approaches that are scientifically sound. The biological processes caused by genotoxic, melatonin reducing, cellular calcium ion altering and gap junction altering substances are described and the evidence that EMF/EMR does all these things is presented. Melatonin related behaviour function, including irritability, anger, rage, violence, homicide and crime are correlated with weather and Solar/Geomagnetic Activity. The biophysical mechanism provides plausible scientific links, the natural environment correlations provide evidence of human reactions and links to extremely weak EMR signals to a wide range of human reactions. Occupational and residential studies are limited in availability and scope but they confirm a reasonable number of the symptoms to support and confirm the soundly based hypothesis which is centered on the extremely bioelectrical sensitivity of the brain, electrical interference of electric functions in the brain, heart and cells by external EMF/EMR signals, with genotoxic, melatonin reduction and calcium ion alteration.

Altered neurological function and behaviour related to the electrobiochemistry of the brain and evidence of changed behaviour. The links to the melatonin effects and the calcium ion mechanisms are through accelerated cell death and cancer in all body organs including the brain, and to the very serious problem of impaired sleep. Impaired sleep has a serious effect on immediate health and well-being, with lethargy, confusion, and chronic fatigue. Chronic sleep impairment, especially with loss of REM sleep efficiency, leads to memory and learning impairment, depression and risk of suicide. Depression is also linked to lung cancer because chronically depressed people are more prone to smoking. Evidence of induced sleep disruption is strongly indicative of a melatonin reduction mechanism in humans. Evidence is strong for EMR and EMF induced melatonin reduction in animals and observational evidence for this in humans is growing.

Some basic principles need to be understood and applied appropriately to study and interpret data in Environmental Health:

1) **The Straw Principle:** There is a classical saying about "The straw that breaks a camel's back". In assessing potentially cumulative environmental factors we need to identify whether a correlation of a symptom with an environmental factor is adding a "straw" or a "log". It is possible sometimes to identify a straw which is closely related to a log because in effect the log is a million straws tied together. On the other hand the straw or twig might be quite different from the log or a bunch of different twigs, and be working in a synergistic way with many different factors.

2) **The Ubiquitous Principle:** Some substances may only be found in special circumstances such as occupational situations. Other substances may expose the whole population in their homes and workplaces and can sometimes even expose them when they are in school, carrying out recreation and holiday travel. An invisible, silent, tasteless, odorless toxic substance that the vast majority of the population are unaware of, can be causing a high proportion of existing health effects.

3) **The Absence of a no-exposed reference group principle:** It is very important to when evaluating the effects of a ubiquitous substance that there is no non-exposed reference group. It is necessary for a case-control or cohort epidemiological study to compare the sickness or mortality rate of a non-exposed group with the rates of sickness or mortality in a group specifically exposed to a potential disease agent, not confused by any confounders. To possible ways of correcting for this factor at a) using a dose response relationship to identify the exposure rate, b) using a historically earlier general population disease rate for a period which is known not to have exposure to the currently studied ubiquitous substance.

In the first case the interpretation of the dose-response gradient must consider the effect of the ubiquitous agent on it because the gradient is dependent on the reference group disease rate. In the second case the careful consideration of any other historical or ubiquitous factors must be considered.

## 1.6 The links between ELF and EMR effects:

Induced electric fields alter the electric field and the charge characteristics at the cell membrane. Biological effects are primarily related to time varying induced fields since our biology has developed in the environment of the earth's static electric and magnetic fields. Induction of electric fields in tissue at the cellular level varies with the intensity and the nature of the environmental field. Typical endogenous EM fields, with ELF modulation, induce fields in the order of  $10^{-1}$  to  $10^{-7}$  V/cm in the pericellular fluid (fluid surrounding the cell). RF/MW fields penetrate the organ or body much more effectively than the ELF fields.

For example, when chick brains were exposed to an applied 56 V/m field ( $832 \mu\text{W}/\text{cm}^2$ ):

- An ELF field 1-32 Hz, induced a tissue gradient of  $10^{-7}$  V/cm.
- An RF field, 147 MHz, ELF modulated, produced a tissue gradient of  $10^{-1}$  V/cm.

Both of these signals significantly changed the calcium ion efflux from the chick brain tissue, Bawin and Adey (1976).

Thus the RF/MW field with a little over a million times higher frequency, produced a cellular tissue gradient 1 million times higher than the ELF field of the same external field strength. This shows the highly penetrative nature of RF/MW fields compared to ELF fields. Since the energy flux relates to the square of the electric field gradient strength, the energy and the electric field gradient imparted to the cell tissue by ELF modulated RF/MW radiation is many orders of magnitude higher than the same external strength of ELF field. This it is highly likely that effects which are found for exposures to ELF fields will be more likely to also be found in association with RF/MW exposure. For example, several studies have found depression and psychovegetative effects when living near high voltage powerlines. The Schwarzenburg Study, Altpeter et al. (1995), Altpeter (1998), found a causal

disturbance to sleep in association with extremely low mean exposures to a short-wave radio transmitter. Chronic sleep disturbance can lead to chronic fatigue and depression. The health study of the staff and families at the U.S. Embassy in Moscow, Lilienfeld (1978), associated depression, irritability, memory loss and difficulty in concentration with extremely low level exposures to a pulsed microwave, radar signal.

### **1.7 Setting the scene by a veteran researcher, Professor W Ross Adey:**

Adey, W.R., 1988a: "The cellular microenvironment and signaling through cell membranes". *Prog. Clin. Biol. Res.*, 257:81-106.

**"The structural and functional aspects of communication between cells have been reviewed, with emphasis on the cell membrane in detection and transductive coupling of oscillating electromagnetic fields in the peri-cellular environment. Imposed fields are powerful and highly specific tools in manipulation of the sequence of events in membrane transductive coupling. They have revealed nonlinear and non-equilibrium aspects of these interactions. In cerebral tissue, extra-cellular fields orders of magnitude weaker than the membrane potential can modulate cell firing patterns, entrain EEG rhythms, alter neurotransmitter release and modulate behavioral states.**

**These sensitivities have also been widely detected in non-neural tissues. It is therefore proposed that an intrinsic communication system between cells based on these weak electromagnetic influences may be a general biological property. A three-step model of transductive coupling is presented. First, a highly cooperative modification of calcium binding occurs in the plane of the membrane surface following a focal event at a receptor site. This "amplifying" stage releases substantially more energy than in the initial events.**

**Cerebral extracellular conductance changes accompanying physiological responses may arise in perineuronal fluid with a substantial macromolecular content and calcium ions may modulate perineuronal conductivity. In the second stage, coupling occurs along transmembrane helical proteins and may be mediated by solitons.**

**The third stage couples transmembrane signals to the cytoskeleton and to intracellular enzyme systems, including membrane-bound adenylate cyclase and the protein kinase system of intracellular messengers. Activation of these intracellular systems is calcium-dependent."**

Adey, W.R., 1988b: "Cell membranes: the electromagnetic environment and cancer promotion". *Neurochem. Res.*, 13(7):671-677.

**"Use of weak electromagnetic fields to study the sequence and energetics of events that couple humoral stimuli from surface receptor sites to the cell interior has identified cell membranes as a primary site of interaction with these low frequency fields. Field modulation of cell surface chemical events indicates a major amplification of initial weak triggers associated with binding of hormones, antibodies and neurotransmitters to their specific binding sites.**

**Calcium ions play a key role in this stimulus amplification, probably through highly cooperative alterations in binding to surface glycoproteins, with spreading waves of altered calcium binding across the membrane surface. Protein particles spanning the cell membrane form pathways for signaling and energy transfer.**

**Fields millions of times weaker than the membrane potential gradient of  $10^5$  V/cm modulate cell responses to surface stimulating molecules. The evidence supports nonlinear, non-equilibrium processes at critical steps in trans-membrane signal coupling. Powerful cancer-promoting phorbol esters act at cell membranes to stimulate ornithine decarboxylase which is essential for cell growth and DNA synthesis. This response is enhanced by weak microwave fields, also acting at cell membranes."**

Dr Adey summarizes the EMR/EMF interactional effects at the cell membrane, demonstrating the great deal of knowledge about this subject which scientific research has already revealed, this includes the cell membrane as the primary interactional site, calcium ions and ODC as key compounds, and very low level effects which are amplified to produce brain function, behavioural and health risk changes.

## **1.8 Outline of This Report:**

This report presents information about the natural weather-related electromagnetic fields and their biological and health effects, how the background artificial EMR/EMF levels have increased, especially over the 20<sup>th</sup> century, and is continuing. It will outline and document the genotoxic and melatonin mechanisms, and the cellular effects of calcium ions. It will give evidence that shows that EMR/EMF can alter these in people and animals, as well as effects in plants in relation to calcium ions and tree ring growth. It will then outline the evidence of effects in the neurological functions of the brain and in behavioural studies of animals. Then the epidemiological evidence of effects in humans will be summarized to lead to the conclusion that there is very strong evidence of serious adverse effects of EMR and EMF on the physical health and emotional and psychological wellbeing of people, as well as deleterious effects on plants and animals.

**An important evidence principle applied here is that a dose-response relationship is indicative of a causal link.**

## **2. The natural and humanly changed EMR/EMF environment:**

Extremely small natural electromagnetic signals are causally associated with plant, animal and human biological and health effects. Therefore Human Biometeorology strongly challenges that basis and appropriateness of human health protection standards for artificial EMF/EMR fields and radiation.

### **2.1 The electromagnetic environment changes with the weather:**

The primary natural electromagnetic signal that has biological links is the Schumann Resonance a signal. This is a global radio signal generated by lightning from tropical thunderstorms and radiating around the world in the resonance cavity between the lowest layer of the ionosphere of the earth. Local weather systems also have associated ELF modulated RF fields. Anticyclones and depression are characterized by very different



natural background of ELF modulated RF fields. Eichmeier and Buger (1969) measured the EMR/EMF characteristics of weather system. They summarize their measurements as:

Cyclone: 10-100 kHz, 30-100 Hz, > 100 mV/m, (RF Exposure  $\sim 0.0027 \mu\text{W}/\text{cm}^2$ )

Anticyclone: 10 kHz, 1-3 Hz, < 10 mV/m, (RF Exposure  $\sim 0.0000027 \mu\text{W}/\text{cm}^2$ )

They were investigating biological effects found with weather conditions, including altered liver function. Importantly Eichmeier and Buger found that under in controlled laboratory conditions the RF cyclonic-like conditions mouse liver respiration rates were 42 % higher than anticyclonic RF conditions, a highly statistically significant effect ( $p < 0.001$ ). Thus the stronger natural RF field strengths in cyclones is associated with significantly altered liver respiration.

The cyclone is generally much more electrically active because of the convective and thunderstorm activity and precipitation formation. Precipitation formation separates charge, rain and hail becoming negatively charged and cloud droplet positively charged. The positively charged ionosphere creates a static field formed between it and negatively charged earth, called the fair weather field. During rain events, negatively charged rain falling from the cloud to the ground, reduces the vertical gradient of the local electric field. During vigorous thunderstorms the strong negative charge at the base of the cloud induces attraction of positive ions on the surface features, reversing the ground level field.

The ELF modulation of cyclones (depressions) is in the 30-100Hz range, covering the upper frequency range of the daytime EEG rhythms. The anticyclone has modulation in the 1-3Hz range, the nocturnal Delta EEG rhythm. Cyclones are cloudy with less sunshine and higher RF intensities around  $3\text{nW}/\text{cm}^2$ . Both factors are associated with reduced melatonin/serotonin diurnal cycle amplitudes. The local anticyclonic RF field is about  $2.7\text{pW}/\text{cm}^2$ , which is nearly 30 times higher than the global Schumann Resonance signal  $0.1\text{pW}/\text{cm}^2$ .

The effects of the hot dry foehn winds were first identified in Europe. They are associated with depression and suicide in areas near European Alps. In Los Angeles the Santa Ana winds are associated with enhanced homicide, Miller (1968), summarized by Maunder (1970). Miller reports an indicative correlation between the days with Santa Ana winds and homicide in Los Angeles from a 2-year data-set. He observed that in the strongest Santa Ana conditions there was initially a fall in homicide rate (-0.8, -0.7) followed by a rise in homicide rate over 5 days reaching a significant peak on day 2 (+1.0, +3.6, +0.8, +0.2). Weather conditions significantly alter the fair weather fields, Reiter (1992), Figure 1.

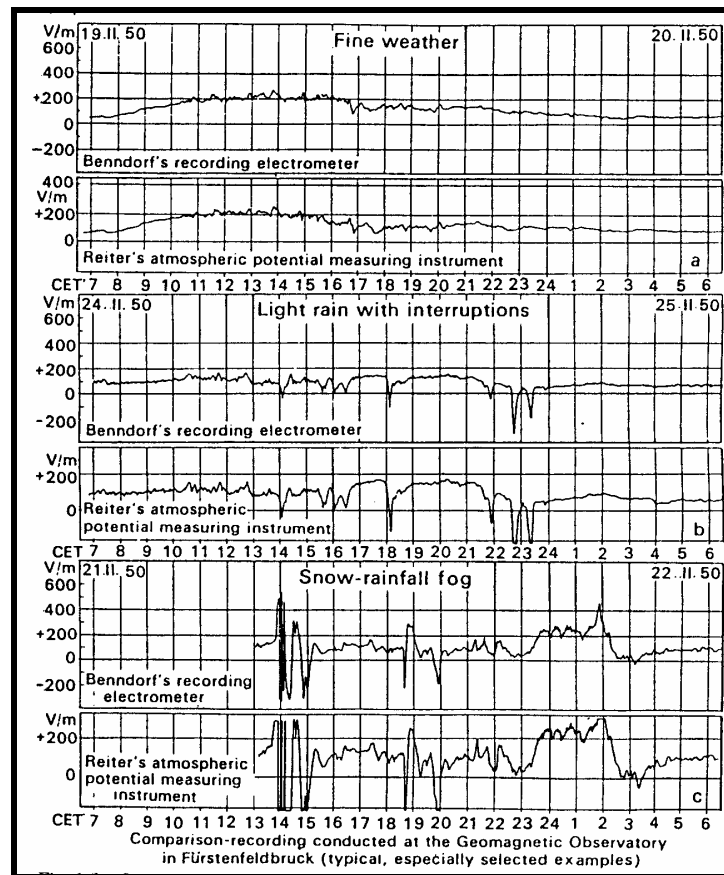


Figure 1: Electric field measurements for a range of weather conditions: (a) Fair weather field, (b), Light Rain with interruptions and (c) Snow/Rainfall and Fog, Reiter (1992).

In Canterbury, New Zealand our own research has shown some similar physical symptoms as those reported in Israel, Europe and America, associated with reduced melatonin. In our survey we found the same symptoms in people living inland and those living along the coast. The challenge was to identify the mechanism of how the people along the coast were affected by the hot dry winds that the people in the Inland were living in and experiencing, but along the coast the hot wind flowed over the sea breeze in the coastal zone and the people were in the cooler, high humidity marine air. It is well established that the hot, dry winds have much higher air ion content, enhancing the strength of the fair weather fields. The hot dry wind is very turbulent producing oscillating ELF electric fields which are associated with reduced melatonin. Therefore both the coastal and the inland living people with in the enhanced ELF electric fields and both experienced the symptoms associated with reduced melatonin.

Solar radiation from the high energy part of the solar spectrum, is absorbed high in the atmosphere, producing ionized atoms and molecules and free electrons. Because free electrons are more easily lost to space, this region, called the Ionosphere, has a net positive charge relative to the earth. This forms a static electric field which varies in strength from day to night, with season and with the presence of thunderstorms. The fair weather field is around 130V/m but can increase to 10kV/m beneath and within thunderstorms.

These ELF and RF signals are superimposed on the time varying static electric field some examples of which are given in Figure 1. The fair weather E field varies quite smoothly from about 20 V/m early in the morning to around 200 V/m during the afternoon. Light rain (b),

shows a nearly constant hourly mean value around 100 V/m, with reductions in E at times of rain, even becoming negative in heavier showers. During heavier precipitation the E-field is much more variable around a mean of about 100 V/m.

## 2.2 Pre-20th Century ambient exposures to ELF-UHF were extremely low:

Naturally occurring static electric and magnetic field strengths must not be confused with the strength of time varying EMR and EMF. Terrestrial biology has evolved in the long standing magnetic field of the earth and the static electric field produced between the ionosphere and the earth. The earth's static magnetic field varies with latitude, Figure 2, and year by year as the field rotates around the poles.

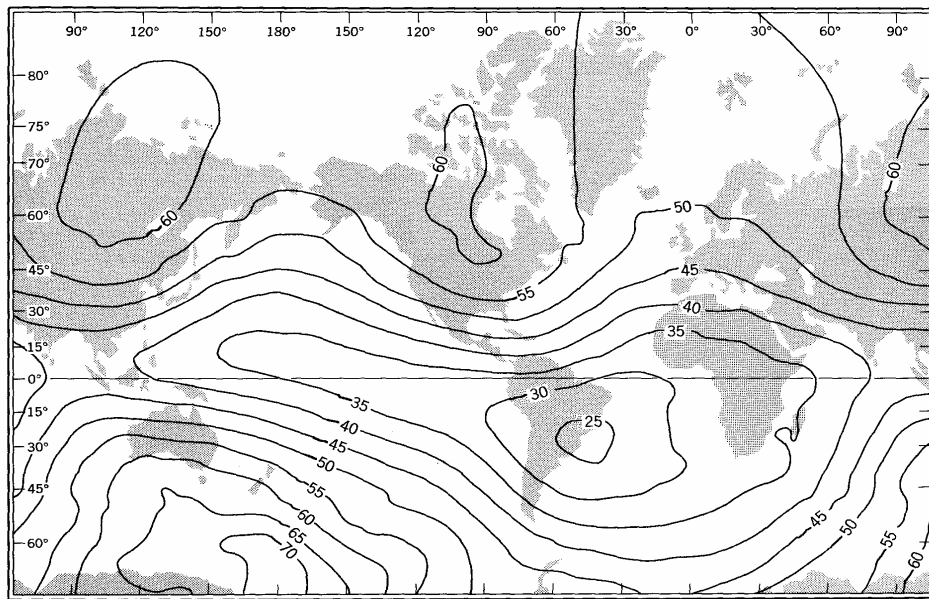


Figure 2: The earth's static magnetic field intensity in microTesla ( $\mu\text{T}$ ) for a given year, 1955.

The terrestrial magnetic field has a diurnal variation because of the distortion produced by the Solar Wind, Figure 3, and the effect of the sun.

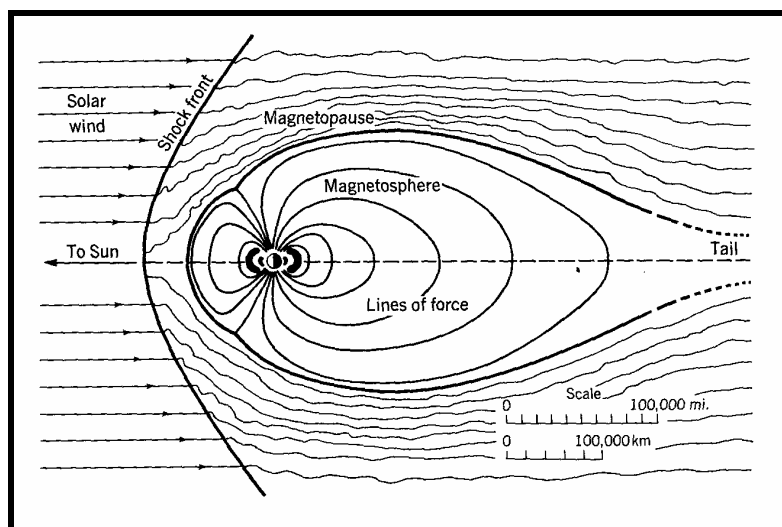


Figure 3: The earth's magnetosphere showing the effect of the solar wind which extends the lee-side magnetic field out into a "tail" region, Strahler (1963).

There are also annual solar and lunar cycles, 11-year sunspot cycles and other periodicities which create periodicities in the earth's magnetic and electric fields. An example of the diurnal magnetic field variation is seen in Figure 4.

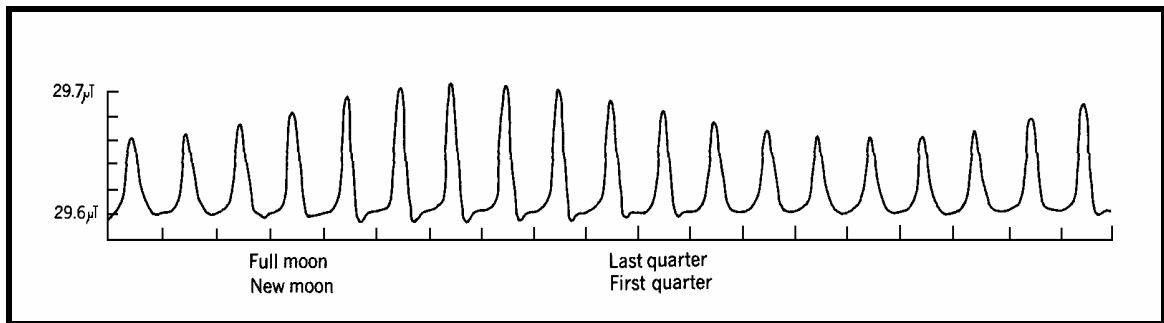


Figure 4: Daily magnetic intensity of the earth's field observed at Huancayo, Peru, throughout part of a month, showing also the moon's effect, Strahler (1963).

The natural sources of the natural radiation are galactic, solar and terrestrial. Detectable components of extraterrestrial sources at radio and microwave frequencies are extremely weak, around  $10^{-20}$  W/m<sup>2</sup>/Hz from a typical radio star. Even the sun cannot be considered a strong source of energy in the non-ionizing spectral region, making ancient ambient levels so low that the possibility of bioeffects is negligible, Adey (1981).

At 10 Hz the components of the solar flux do not exceed 0.001 V/m, with expected tissue gradients of around  $10^{-9}$  V/cm. A 10 Hz signal has a wavelength of 30,000 km making the head an extremely small fraction of the wavelength at EEG frequencies and incapable of significant radiation at these frequencies.

Terrestrial sources, primarily the Schumann Resonance Oscillations, have an amplitude around 0.0002 V/m/Hz. Schumann Resonance Oscillations originate from the radiation from lightning primarily in the tropics, the electric field radiation of which is ducted and resonates in the "electrical cavity" created between the ionosphere and the earth. The fundamental frequency is 7.8 Hz, with harmonics at 14.1, 20.3, 26.4 and 32.5 Hz. Assuming each resonant peak is associated with a 1 Hz bandwidth this results in a 0.001 V/m ELF field strength, or the same order as the solar 10 Hz flux. A combine signal of around 0.002 -0.003 V/m will produce tissue gradients in the order of  $2-3 \times 10^{-9}$  V/cm. The SR signal is in the range 0.1 to 0.25pW/cm<sup>2</sup>.

### **Human Detection of Schumann Resonances for Neurological and Cardiac Synchronization:**

Resonant absorption occurs when there is a frequency match. Figure 5 shows the typical human EEG spectrum and Figure 6 shows the typical daytime Schumann Resonance Spectrum.

Note the strong similarities between the natural E-field ELF variations and the ELF rhythms of the mammalian brain, Figure 7, not just the frequency range but also the diurnal pattern. Below evidence is given that these fields do in fact interact and alter human reaction times for example. Cherry (2002) shows that there is very strong and highly robust evidence that the SR signal is the biophysical mechanism linking Solar/ Geomagnetic Activity to human health effects related to reduced melatonin, including cancer, cardiac, reproductive and neurological health effects and mortality.

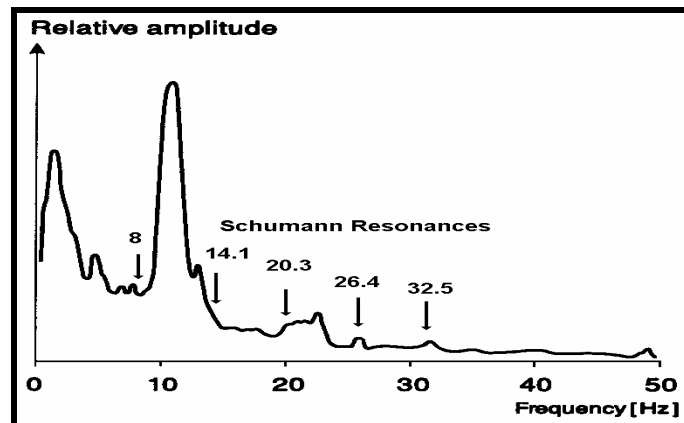


Figure 5: A typical EEG spectrum, with the Schumann Resonance peaks superimposed.

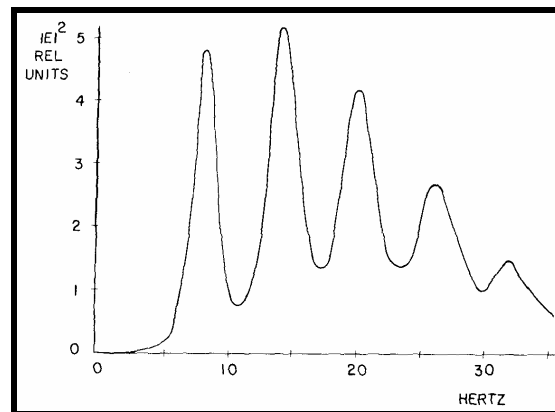


Figure 6: A Daytime Schumann Resonance Spectrum, Polk (1982).

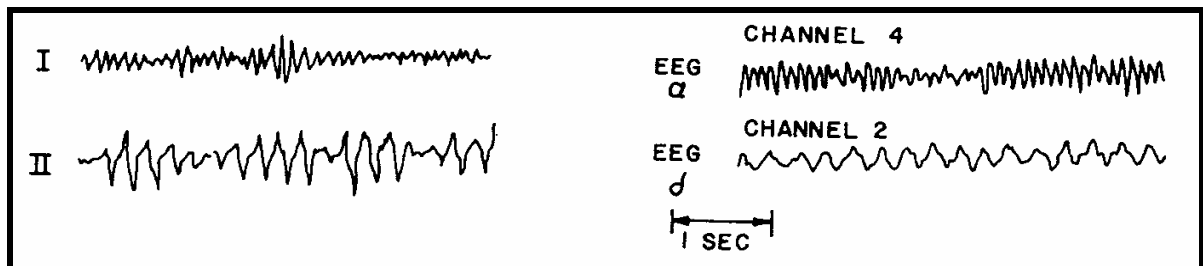


Figure 7: Electric fields from I, the Schumann-Resonance, II, Local fields of about 3 Hz and the  $\alpha$  (10 Hz) and  $\delta$  (3 Hz) human EEG channels, König (1974).

The frequency range of the Schumann Resonances and the human EEG overlap and some peaks coincide, showing the probability of resonant absorption and the likelihood of interaction. Both the high frequency peaks and the diurnal frequency shifts, high during the day and low at night, match between the two spectra. The 3Hz local field signal is very similar to the nocturnal SR signal.

It was proven, but not well known, that human brains detect, use and react to natural low frequency signals, the Schumann Resonances, König (1974). König (1974) reports on the results of an experiment carried out at the Munich Transport Exhibition of 1953, Figure 8. About 49,500 people were recorded in a visual reaction time experiment. Their reaction times were extremely highly correlated with the intensity of the Schumann Resonance signals.

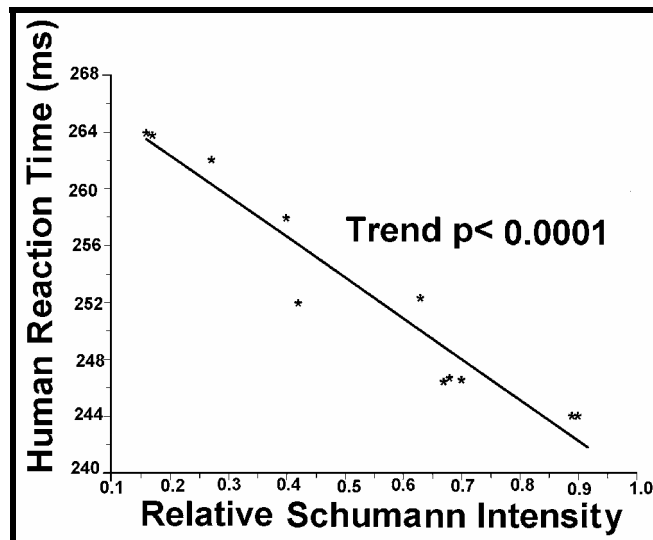


Figure 8: Human reaction times are causally correlated with natural variations in the Schumann Resonance Intensity, Konig (1974). The mean Schumann intensity (Relative Schumann Intensity =0.5) is 0.65mV/m or 0.1pW/cm<sup>2</sup>. The range is 0.2 to 1.2 mV/m (0.01 to 0.4pW/cm<sup>2</sup>).

The Schumann Resonances are global signals that radiate from tropical thunderstorms. They propagate around the world within the cavity created by the earth and the ionosphere. The intensity and spectrum of the Schumann Resonances vary markedly from day to night and with solar activity. At night both the EEG and the Schumann Resonances are dominated by very low frequencies (<5 Hz). With the coincidence of the frequency ranges, some of the high frequency peaks and the diurnal variation of the EEG and Schumann Resonances, it is biologically plausible that there is a resonant interaction between, and EEG reaction to the changing Schumann Resonance signals.

This biological plausibility 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).

This result was confirmed by laboratory experiments that showed that 10 Hz signals significantly and consistently increase the reaction speed and 3 Hz signals slowed them down, Konig (1974). These results were independently confirmed by Hamer (1966, 1969). Hamer observed that human reaction times were significantly altered at exposure levels down to 4mV/m, 4.2 pW/cm<sup>2</sup>. This is approaching the level of the Schumann Resonance signal, which averages about 0.08mV/m, 0.1pW/cm<sup>2</sup>.

These experiments give substantial proof that extremely small natural and artificial ELF signals interact significantly with human brains. The signal level of this interaction is 2,000,000,000 times below the ICNIRP ELF guideline. This guideline is based on avoiding acute shocks and not on avoiding proven neurological effects. The maintenance of the standard is obtained by ignoring or rejecting any and all evidence that contradicts it.

Independently Wever (1974), at the Max Planck Institute, showed that by shielding people from the Schumann Resonances the average day length in isolation experiments is significantly longer than simple sunlight isolation,  $p < 0.001$ . More significantly 30 % of

subjects in the Faraday Cage shielded room desynchronized,  $p < 0.001$ . This was corrected at will by the observers applying a small 10 HZ signal, Figure 9.

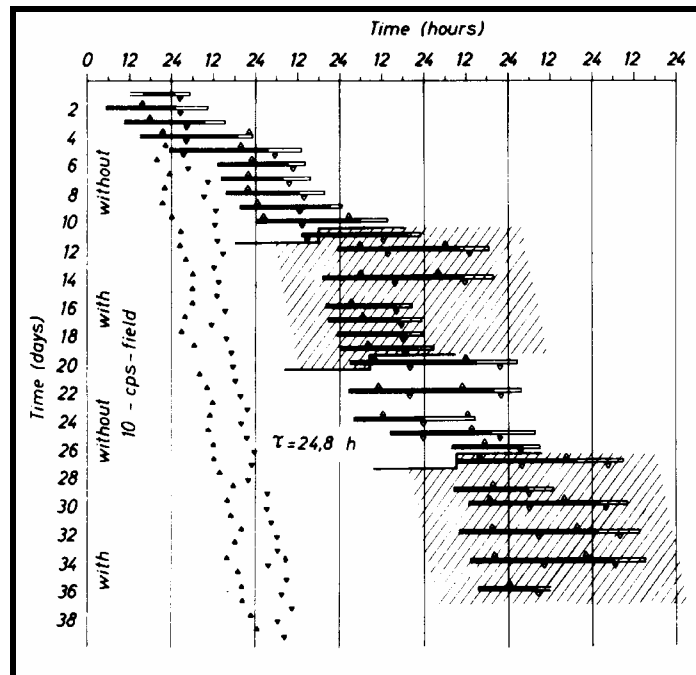


Figure 9: Free running circadian rhythm of a subject living under strict isolation from environmental time cues. The shaded period involve the secret application of 10 Hz pulsed signal with a peak-to-peak voltage of 2.5 V/m, corresponding to an rms voltage of 0.88V/m ( $S = 0.2\mu\text{W}/\text{cm}^2$ ).

**Wever concludes that these experiments “provide strong proof that electromagnetic fields in the ELF range influence human circadian rhythms, and therefore human beings”.**

Together Konig and Wever prove that human brains detect and react to the Schumann Resonance (SR) signal. This signal has a mean field strength of 0.08V/m,  $S = 0.1\text{pW}/\text{cm}^2$ . This is followed up and strongly confirmed by Cherry (2002), which also identified the melatonin mechanism and the human health effects.

This gives a very strong basis for this paradigm shift that recognizes the exquisite sensitivity of the human brain and its regulation and synchronization by these very weak naturally occurring, globally available, ELF signals. The paradigm shift is also based a classical public health approach setting standards based on epidemiology and strong evidence that electromagnetic fields and radiation is a genotoxic carcinogen.

Considering the SR signal, there is independent confirmation. In measuring the melatonin levels in electrical workers in the United States, Dr James Burch and his team at the Colorado State University, Fort Collins, found that ELF fields significantly reduce melatonin, as does cellphone use. Having removed the ELF and cellphone effects there was a residual variation in the data. This was dose-response related to Geomagnetic Activity, Figure 10.

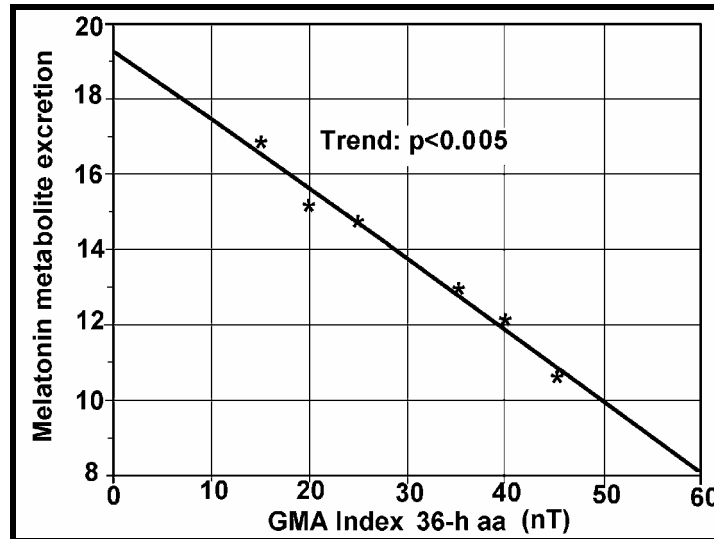


Figure 10: A dose-response reduction in human melatonin associated with increased geomagnetic activity, Burch et al. (1999).

An increase in GMA increases the nocturnal intensity of the Schumann Resonance signal that decreases the melatonin production in a causal manner (highly significant dose-response). Hence our brain's ability to detect and respond to the exquisitely minute signal of the Schumann Resonances results in changes in GMA causing physiological changes in human beings through the reduction in melatonin. A reduction in melatonin is associated with a very wide range of illnesses and diseases, including cancer, neurological, cardiac and reproductive health effects.

Cherry 2002 shows that the Schumann Resonance related health effects, operating through the melatonin mechanism, causes a homeostatic variation of cancer, cardiac, reproductive and neurological health effects in human populations. For example in relation to neurological effects, enhanced SR signal strength from enhanced S/GMA has been strongly correlated with:

Memory and attention deficit, aviation accidents, desynchronisation of the Alpha EEG rhythm, suppression of cortical activity, sensory deprivation (hearing and seeing), aggression, epileptic fits, convulsive seizures, dizziness, mental hospital admissions, suicide, migraine attacks, isolated sleep paralysis and multiple sclerosis incidence.

Lower levels of S/GMA are associated with increased levels of anxiety, vivid dreaming, crime, suicide, sports injuries, fatal work injuries, alcoholism and a psychiatric hospital admission rates.

Since the mechanism which causes these effects is a natural global radio electromagnetic signal with a mean field intensity of  $0.1 \text{ pW/cm}^2$  field strength of about  $1 \text{ pT}$ , it was predicted and confirmed in Cherry (2002) that similar effects would be found from electrical workers. The confirmation was given by multiple occupational epidemiological studies. Not all the identified effects have been found, but this is because not all effects have been investigated. However, the biological plausibility of altered melatonin related effects, produced by residential and occupational exposure to ELF and RF fields are producing all of the identified and many other health and biologically related effects.



### 2.3 The RF levels in U.S. cities in 1980:

The E.P.A. noted the extremely low levels of public RF exposure “50 years ago” was around  $10 \text{ pW/cm}^2$ , from a small number of radio stations. A survey of U.S. cities published in 1980, Tell and Mantiply (1980), found the mean urban public exposure was  $0.005 \text{ } \mu\text{W/cm}^2$ , or  $5 \text{ nW/cm}^2$ , with a median of  $6 \text{ nW/cm}^2$ , 600 times higher than 50 years earlier. At that time 0.59 % of the urban population of the U.S. was exposed to  $1 \text{ } \mu\text{W/cm}^2$  or more. This represents about a million people in 1980 exposed to more than 100,000 times more RF radiation than 50 years earlier. Figure 11 shows the frequency distribution of sampled exposures in U.S. cities in 1980.

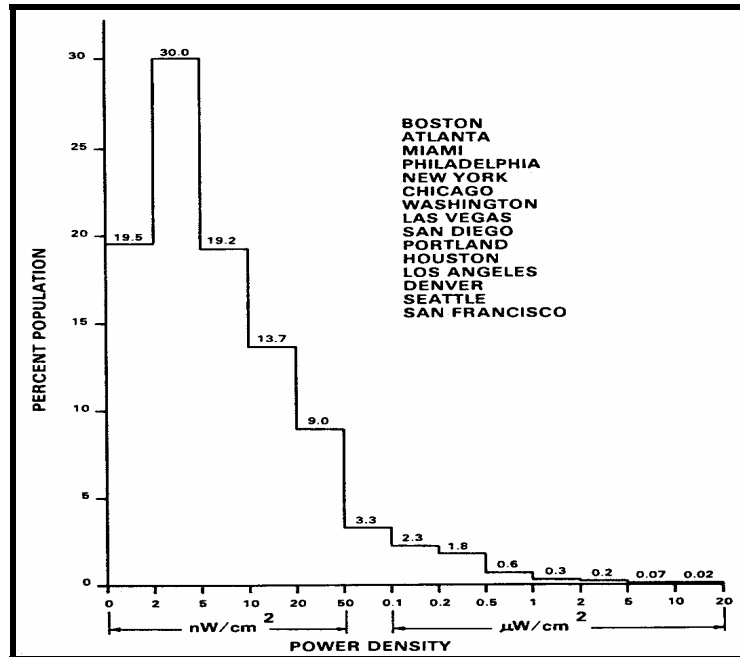


Figure 11: Frequency distribution of population exposures to RF radiation in 15 U.S. cities, Tell and Mantiply (1980).

With the explosion in the number of radio and TV stations, radio telephone networks, cordless phone and cell phones, the mean population exposures in the 21<sup>st</sup> Century will be 10-100 times higher than in 1980. Hence, compared with 50 years ago, the median public exposure levels to RF/MW radiation are generally 6,000 to 60,000 times higher now. For those living near transmission towers or the users of cell phones, the increase of the peak exposure is in the order of 1 million higher. Even the lowest of these, 6,000, is very concerning. If this was a visible or odorous substance, its presence and effects would be very evident and a grave cause for concern. Because RF/MW radiation is odorless, invisible, colorless and tasteless, its presence is virtually ignored and its effects are extremely hard for most people to conceive. Like many invisible toxic chemicals, RF/MW radiation is shown by many independent studies, including at isothermal and very low exposure levels, to be genotoxic. This puts the situation in a very different context if we ask the question,

**“Has a 6,000-fold or more increase in exposure level of people to genotoxic RF/MW had any potential effect or actual on individuals or populations of people ?”**

**Compare this with the public reaction to the question:**

**“Would you be concerned about, and urge the government to deal with your family being exposed to 6,000 times higher dioxin levels than people on Norfolk Island?”**

A genotoxic substance causes cellular DNA damage resulting in enhanced rates of cell death, mutation and cancer. If a substance is genotoxic, it causes cancer, cardiac, reproductive and neurological health effects and mortality. All of these health effects have shown rising rates in developed countries, over the past century, especially since the second World War.

### **3. The Melatonin Biological Mechanism:**

Melatonin reduction has many serious consequences including increased cancer risk for every organ in the body, increased risk of spontaneous miscarriage, impaired immunocompetence, impaired learning, memory, sleep, and enhanced incidence of anger, rage, depression and suicide. Hence when epidemiological studies identify statistically significant associations with any of these symptoms and EMR or EMF, then a melatonin mechanism is a plausible biological mechanism and that study adds to the weight of evidence that EMR and EMF alters melatonin in humans. Direct measurements of human melatonin in situations of EMR/EMF exposure are few in number but some have found reductions.

#### **3.1 The Pineal Gland:**

Melatonin is produced, primarily at night, by the pineal gland. The pineal gland is a cone shaped, pea sized body residing near the centre of the brain, Figure 12.

Light received by our eyes is the primary control source for melatonin. When it is light melatonin production is blocked. At night this blocking effect is removed. Then Tryptophan is converted to serotonin which is then converted to Melatonin at a rate controlled by an enzyme NAT (N-acetyltransferase) which has been activated or limited through protein synthesis from amino acids controlled by cyclic AMP, Figure 12.

Tryptophan, an amino acid from the blood, is converted to the hormone melatonin, which is quickly released into the capillaries of the gland. The enzymes which catalyze the conversion of serotonin to melatonin include N-acetyltransferase (NAT) and hydroxyindole-O-methyltransferase (HIOMT).

The pineal gland produces melatonin at night since the nerve endings which end in the pineal gland release the neurotransmitter norepinephrine (NE) which interacts with the b- and a-adrenergic receptors on the cell membrane; these interactions initiate the processes which control melatonin production. ATP, adenosine triphosphate; PVN paraventricular nuclei; SCN, suprachiasmatic nuclei; SCG, superior cervical ganglia. The melatonin easily passes through the cell wall into the blood stream to be dispersed throughout the body. The pineal gland is located near the centre of the brain. It is an endocrine organ which produces most of the melatonin which is found in the blood, Figure 13.

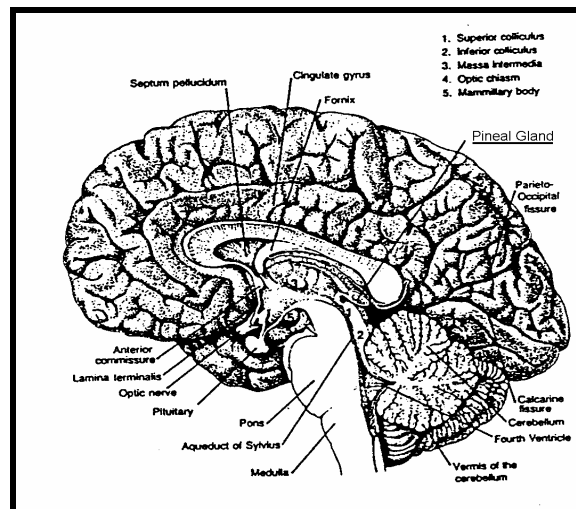


Figure 12: A mid-sagittal section of the human brain showing the location of the pineal gland, Reiter (1994).

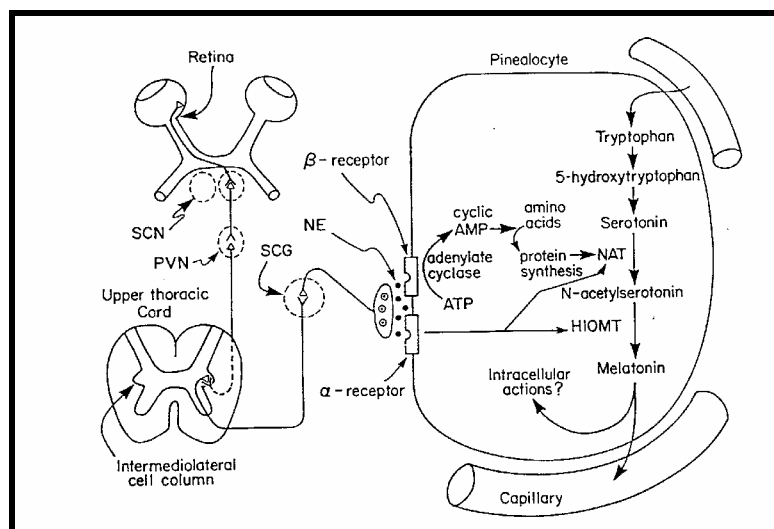


Figure 13: The connection of the eyes (retina) to the pineal gland, represented by a single pinealocyte, and the synthesis of melatonin within the gland.

Once melatonin is produced it is the molecule's high ability to pass through the cell membrane which allows it to escape from the pinealocyte to the blood. Once in the blood melatonin has access to every cell in the body. It passes through the cell membranes where many nuclei have receptors for it. Many body organs, needing to have a daily rhythm control have melatonin receptors. These may mediate the 24 h circadian rhythms of the endocrine, brain/CNS, cardiac, immune, metabolic and respiratory systems. In the nucleus melatonin plays a role in regulating the effects of the indole on gene expression. The ability of melatonin to enter all cells is also essential for one of the other important functions of melatonin, namely, its ability to scavenge the highly toxic hydroxyl radical ( $\bullet\text{OH}$ ).

### 3.2 Melatonin is a highly potent free radical scavenger:

The production of oxygen-based free radicals, such as  $\bullet\text{OH}$  is a consequence of the utilization of oxygen by organisms. About 1-2 % of inspired oxygen ends up as toxic free radicals. It is generally considered the  $\bullet\text{OH}$ , because of its high reactivity, is the most devastating to macromolecules such as DNA, proteins and lipids. The cellular damage produced by free radicals is generally referred to as oxidative stress, Reiter (1994).

Because of its action in removing free radicals, melatonin is probably the most efficient natural cell protection and oncostatic agent in our bodies. Every night, our pineal produces large quantities of melatonin which flood almost every cell in our body, cleaning out the free radicals and assisting cell division to take place with undamaged DNA. As we age our nocturnal peak melatonin production falls markedly, making elderly people much more prone to cancer. To test the cancer protecting properties of melatonin, Tan et al. (1993), injected rats with a chemical carcinogen, safrole. Safrole normally damages DNA because it induces the production of large numbers of free radicals. Rats injected with Safrole were found to have extensive DNA damage after 24 h. When melatonin was also injected, the DNA damage was reduced by 99 %. Since damaged DNA can undergo mutation it may result in the growth of a tumour. Thus melatonin is clearly a potent cellular protector against cancer initiation.

### 3.3 Effects of melatonin:

There is evidence of the change of melatonin and serotonin that is linked to depression, anger and rage. As melatonin is a very high free radical scavenger, and free radicals are known to damage DNA. Hence reduced melatonin can lead to cancer or damaged cells which are then programmed to die (apoptosis). Lai and Singh (1997) showed that microwaves increased the single and double-strand DNA breakage in living rats brains. They also showed that the breakage did not occur if either melatonin or a chemical free radicals scavenger (PBN) were infused. They noted that PBN has been shown to protect cells from free radical-induced apoptosis. Hence melatonin has the same oncostatic protection action and reduced melatonin reduces such protection leading to enhanced cancer risk and apoptosis. Lai and Singh (1997) conclude:

**“Since cumulated DNA strand breaks in brain cells can lead to neurodegenerative diseases and cancer and excess free radicals in cells has been suggested to be the cause of human diseases, data in this study could have important implications of the health effects.”**

Free radicals have an important role in aging processes (Reiter (1995)). Aging has been ascribed to accumulated oxidative damage to body tissues, Forster et al. (1996) and Sohal and Weindruch (1996), and involvement of free radicals in neurodegenerative diseases, such as Alzheimer's, Huntington's and Parkinson's, has also been suggested, Borlongan et al. (1996) and Owen et al. (1996). Hence there is evidence that RF/MW can cause serious health problems, such as cancer which can lead to earlier death, and neurodegenerative diseases which are associated with aging.

Once apoptosis begins, cell death occurs within hours through the inter-nucleosomal digestion of the genomic DNA. In most cell systems apoptosis is part of the cell protection system by killing damaged cells so they don't become cancer cells, for example. In brain cells, which have no regeneration, enhanced apoptosis leads to neurodegenerative diseases.

Pineal and serum melatonin concentration drops during the day and rises overnight, Figure 14. This shows the great differences in nocturnal melatonin levels between individuals. Similar variations can be seen for the same people on different nights, reflecting widely differing sleep quality.

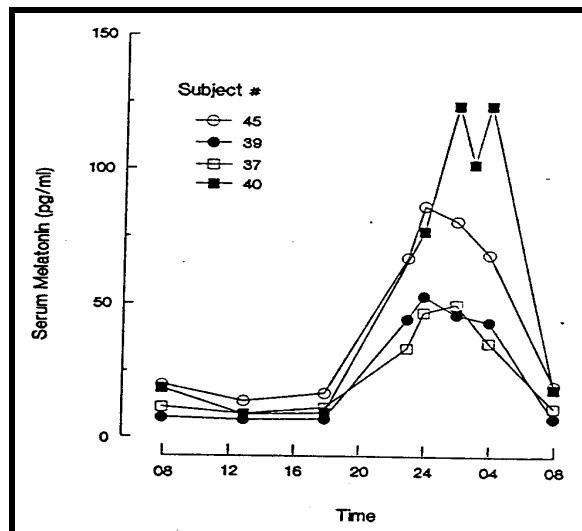


Figure 14: Blood melatonin levels for 4 healthy adult males over a 24 h period, Reiter (1994).

A natural part of aging is the reduced natural levels of melatonin which leads to increasing rates of cell damage, cancer and aging. Melatonin levels peak in childhood around 8 to 10 years and thence declines, Figure 15.

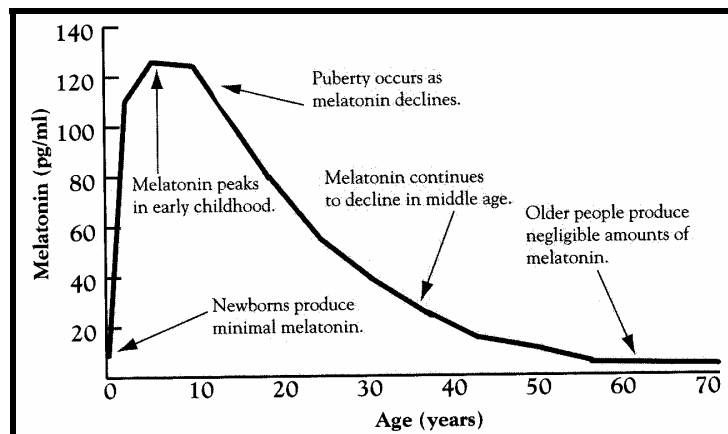


Figure 15: Peak night time melatonin varies throughout life span, Reiter and Robinson (1995).

### 3.4 Melatonin has a role in the immune system:

Melatonin is also linked to the immune system, with enhanced levels of melatonin protects and enhances the immune system. This happens through protecting lymphocytes from chromosome damage, Vijayalaxmi et al. (1995), and stimulating the T-cells, Maestroni et al. (1995), Figure 16.

### 3.5 Melatonin plays many roles:

High night-time melatonin is associated with healthy sleep, memory and learning. Thus the high nocturnal melatonin assists with physical health through good sleep and scavenging free radicals from cells throughout our bodies so that the cells divide and reproduce with healthier, less damaged DNA and chromosomes. Melatonin is critical to our DNA and cell repair systems and the next level of repair and protection system, our immune system.

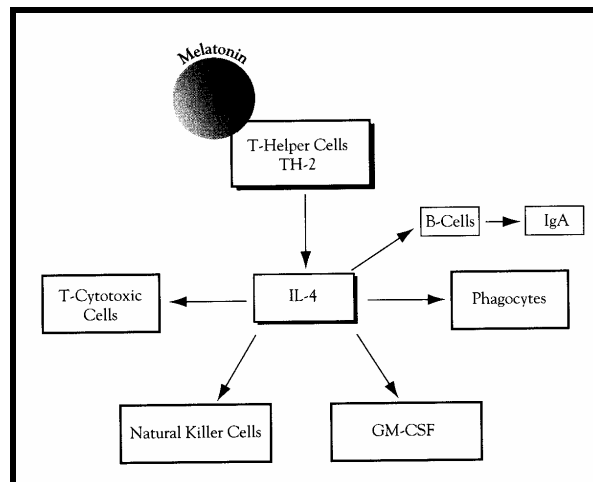


Figure 16: Melatonin stimulates T-helper cells, cited in Reiter and Robinson (1995).

Therefore any evidence that EMR can reduce melatonin, affects our sleep, memory or learning, impair our immune system and enhance apoptosis, has clear links to serious health and advanced aging problems. Reiter and Robinson (1995), identifying the following diseases that are associated with reduced melatonin “Arthritis, diabetes, heart disease, cancer Alzheimer’s Disease and Parkinson’s Disease”. In other sections they identify sleep disturbance that results in chronic fatigue, sudden infant death syndrome (SIDS), manic depression and suicide, Schizophrenia, chronic pain, seasonal affective disorder (SAD), alcoholism, and enhanced ageing. The key roles of melatonin in DNA and chromosome protection through free radical scavenging come together in the uterus where Sandyk (1992) notes that after genetic factors are taken into account, the next most important factors in the risk of spontaneous abortion are melatonin reduction and chromosome aberrations.

### 3.6 Melatonin production is inter-linked to intracellular calcium ions:

Melatonin production is interlinked with intracellular calcium ions through the fundamental role of calcium ions in all human cells, including the cells making up the pineal gland, the pinealocytes. The cyclic AMP and calcium ion dependent signal transduction processes are both involved in the regulation of melatonin production from serotonin within the pineal gland during dark periods, Figure 17.

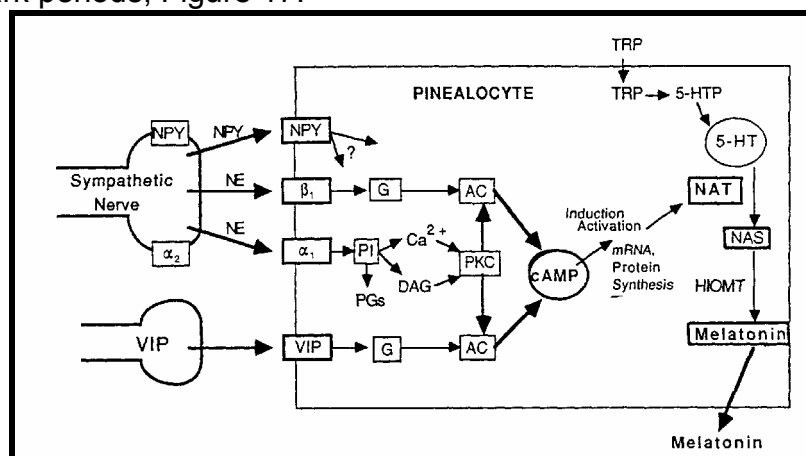


Figure 17: A schematic of the receptors and second messengers underlying the neural control of melatonin production, through which calcium ions mediate the cAMP through the protein kinase C (PKC) and thence the adenylyl cyclase (AC), Krause and Dubocoviich (1990).

### 3.7 EMR and EMF effects on the Melatonin Mechanism:

On the macro-scale, human and animal circadian rhythms are driven by the day/night cycle with a phase-lock synchronization provided by environmental ELF fields ( $E < 0.3 \text{ pW/cm}^2$ ). A fundamental physiological aspect of the circadian rhythm involves the pineal gland and the secretion of a neurohormone called melatonin. Light falling on the eye's retina produces signals which are biochemically amplified around a million times, to stimulate the pineal gland to reduce its melatonin output.

Four independent laboratories, Battelle PNL (Wilson), U.C. Riverside (Luben) and the U.S. EPA (Blackman), Lawrence Livermore Laboratory (Liburdy) have shown that 60 Hz modulated magnetic fields in the 1 to 12 mG range, almost completely negate the oncostatic effect of melatonin in human breast cancer cells, with a dose-response relationship. Wilson et al. (1986) showed significant reductions in pineal melatonin in living rats when they were chronically exposed to 60 Hz modulated electric field at 1.7-1.9 kV/m for 20 h per day, for 30 days, Figure 18.

Welker et al. (1983) studied a range of magnetic field configuration exposures on a group of Sprague-Dawley rats in relation to altered pineal serotonin-N-acetyltransferase (NAT) activity and melatonin. Experimental inversion of the horizontal component of the natural magnetic field, performed at night-time, led to a significant decrease of both parameters investigated. During day-time, this effect was less conspicuous.

During night-time, inversion of the horizontal component is followed by a reduced pineal secretory activity for about 2 h. After 24 h exposure to the inverted horizontal component, return to the natural condition was followed by a renewed clear depression of pineal NAT activity and melatonin content, indicating that the main stimulus is not the inverted magnetic field itself but rather its change. Changing the inclination of the local magnetic field from 63 degrees to 58 degrees, 68 degrees or 78 degrees, respectively also decreased the secretory activity of the rat pineal gland.

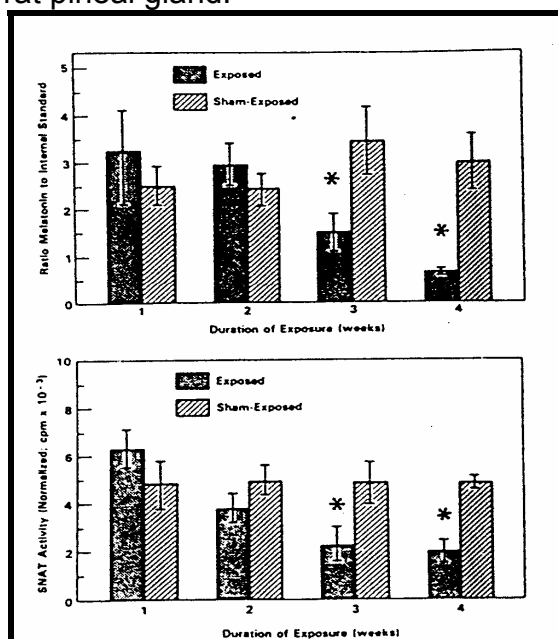


Figure 18: Pineal melatonin (top) and NAT activity (bottom) in groups of rats exposed to a modulated electric field for 1 to 4 weeks. The glands of the animals were collected at night.

In the sham-exposed animals the pineal melatonin and NAT levels were always high. However, after both 3 and 4 weeks of exposure to the electric field, both parameters were depressed ( $p < 0.001$ ).

Hence for rodents, whose cells in many ways behave as human cells, there is direct evidence that chronic electric field exposure over periods of weeks results in reduced pineal melatonin production, Figure 18, and increased serotonin production, Figure 19.

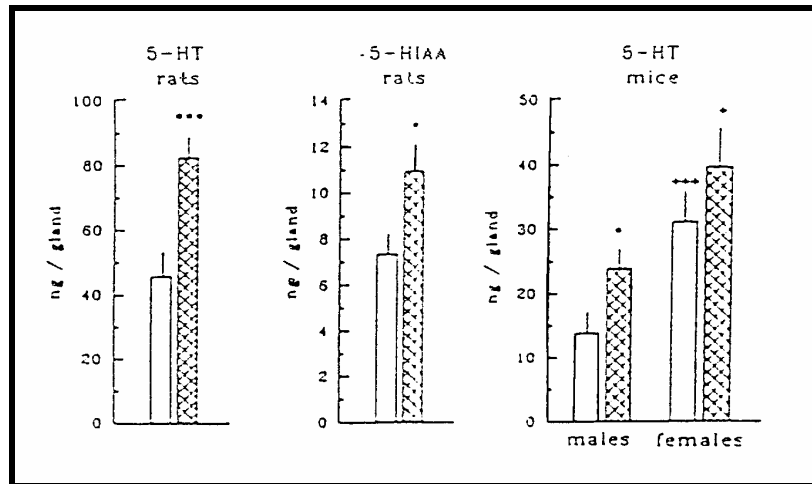


Figure 19: Pineal serotonin (5-HT) and 5-hydroxyindole acetic acid (5-HIAA) levels in rats and mice (cross-hatched bars) with and without (clear bars) exposure to pulsed static MF at night.

A highly statistically significant levels. Lerchl et al. (1988) samples for serotonin and its derivatives by periodically inverting the magnetic field at night.

In Figure 19, both 5-HT and 5-HIAA levels increased as a result of the exposure; these changes are consistent with a reduced melatonin production. \*  $p < 0.05$  and \*\*\*  $p < 0.001$  vs control; +++  $p < 0.05$  control male mice, from Lerchl et al. (1990). Thus it is shown that time varying electric and magnetic fields decrease the melatonin and increase the serotonin and NAT in rodents.

The review paper by Professor Russell Reiter (Reiter (1994)), was prompted by a number of epidemiological studies in which an increased incidence of cancer was reported in individuals living or working in an environment of higher than normal artificial electromagnetic fields. He concludes:

**“Reduction of melatonin at night, by any means, increases cell’s vulnerability to alteration by carcinogenic agents. Thus, if in fact artificial electromagnetic field exposure increases the incidence of cancer in humans, a plausible mechanism could involve a reduction in melatonin which is a consequence of such exposures.”**

He also notes:

**“Epidemiologists should look for other possible changes, including psychological depression, fatigue, sleep inefficiency, chronic feelings of jet lag, endocrine disturbances and other symptoms; all these may result from a chronically low melatonin rhythm.”**



Altpeter et al. (1995) [p 94] noted “a trend suggesting that an increased urine melatonin level is associated with the high probability of sleeping well during the night (OR = 0.903, 95% CI: 0.688; 1.455).”

A recent paper produced for the U.S. RAPID program, Burch et al (1997), reported statistically significant reductions of melatonin in a sample of 142 male electric utility workers. Those who reported occasional to frequent cell phone use had significantly lower melatonin secretion than those reporting no or infrequent cell phone use ( $p = 0.04$ ). After adjusting for age, light exposure and month of participation this association weakened slightly to  $p=0.05$ , whereas those with stable 60 Hz exposure and cell phone use the reduction was significant,  $p=0.03$ . They also concluded that “among cell phone users, there was a progressive decrease of mean 6-OHMS/cr (melatonin metabolite) concentrations in response to temporally stable magnetic field exposures. The results suggest that occupational cellular telephone use may be associated with reduced daytime melatonin production.”

Another study, Sait et al. (1997) concluded that in sensitive individuals there was a highly significant melatonin reduction,  $p= 0.005$ , with an average delay of 55 minutes ( $p<0.001$ ,  $n=11$ ), when exposed to an on/off 50 Hz magnetic field. Their nocturnal peak was reduced by 9 %, but this was not statistically significant. They found that a square wave form was more effective in reducing melatonin than a sinusoidal waveform and they concluded that for an effect to occur the magnetic field exposure had to precede the nocturnal rise in melatonin.

Hence there is direct evidence of melatonin reductions in people exposed to EMF and EMR, but that it varies considerably from person to person, that some people are much more responsive (sensitive) than others and that timing of the exposures is also a factor.

Thus in large exposed populations there are those whose melatonin will be reduced by combinations of EMF and EMR exposure and they are at increased risk of all the above identified health effects. The multiple independent studies showing a causal relationship are summarized below.

### **3.8 EMF/EMR Reduces Melatonin in Animals:**

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  $\mu$ T, 60 Hz field with a 0.06 $\mu$ T 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.

Stark et al. (1997) observed a significant increase in salivary melatonin in a group of 5 cows when the short-wave radio transmitter at Schwarzenberg, Switzerland, was turned off for three days, compared to 5 cows that had much lower RF exposure. Hence there are now at

least ten independent observations of melatonin reduction in animals from ELF and RF exposure.

### **3.9 EMF/EMR Reduces Melatonin in People**

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).

### **3.10 Health effect consequences of reduced melatonin:**

Melatonin reduction is a plausible and identified mechanism for epidemiological studies associating depression with living near power lines, Beale et al. (1997) [with a linear dose-response relationship] and Verkasalo et al. (1997) [RR= 4.7, 95% CI: 1.70-13.3, for severe depression in people living within 100 m of high voltage power lines]; for suicide in association with proximity to power lines, and suicide in electrical industries. The staff of the U.S. embassy in Moscow were studied in relation to their being chronically exposed to very low exposure to a radar which was aimed at a part of the embassy. They showed elevated cancer levels, Goldsmith (1995, 1998), and statistically significant elevation in the following symptoms: Depression ( $p=0.004$ ), Irritability ( $p=0.009$ ), Difficulty on Concentrating ( $p=0.001$ ) and memory loss ( $p=0.008$ ) [Table 6.31, Lilienfeld (1978)].

Zyss et al. (1997) noted that “Numerous reports suggest a relationship between the increased incidence of depressive and neurotic symptoms in humans and the exposure to extremely low frequency electromagnetic field (EMF) at the place of residence.” They studied 70 exposed persons (35 male, 35 female) and compared them to a control group of 37. the exposed group were residents of a suburb of Cracow, Poland, living in the vicinity of two 400 V powerlines. The stated: “Our investigation showed the increased psychopathological values in all clinical tests. The difference between the group exposed to EMF and the control population was statistically significant.”

Verkasalo et al. (1997) stated that electromagnetic fields have been suggested to contribute to the risk of depression by causing pineal dysfunction. Some epidemiologic studies have supported this possibility but have generally reported crude methods of exposure assessment and nonsystematic evaluation of depression. Using two available nationwide data sets, the authors identified from the Finnish Twin Cohort Study 12,063 persons who had answered the 21-item Beck Depression Inventory of self-rated depressive symptoms in 1990. The personal 20-year histories of exposure (i.e., distance and calculated annual average magnetic fields) before 1990 to overhead 110- to 400-kv power lines were obtained

from the Finnish Transmission Line Cohort Study. The adjusted mean Beck Depression Inventory scores did not differ by exposure, providing some assurance that proximity to high-voltage transmission lines is not associated with changes within the common range of depressive symptoms. However, the risk of severe depression was increased 4.7-fold (95% CI: 1.70-13.3) among subjects living within 100 m of a high-voltage power line. This finding was based on small numbers. The authors recommend that attempts be made to strive for a better understanding of the exposure characteristics in relation to the onset and course of depression.

Beale et al. (1997) studied 540 adults living near transmission lines completed neuropsychological tests in major domains of memory and attentional functioning, mental health rating scales and other questionnaires. Magnetic field measurements were taken in each room occupied for at least one hour per day to provide an estimate of total-time-integrated exposure. The data were subjected to joint multivariate multiple regression analysis to test for a linear relation between field exposure and dependent variables, while controlling for effects of possible confounders. Performance on most memory and attention measures was unrelated to exposure, but significant linear dose-response relationships were found between exposure and some psychological and mental health variables. In particular, higher time-integrated exposure was associated with poorer coding-test performance and more adverse psychiatric symptomatology. These associations were found to be independent of participants' beliefs about effects of electromagnetic fields.

Bonhomme-Faivre et al. (1998) studied a small group (13) of exposed workers matched to 13 controls and found that those exposed to 50 Hz in the mean range of 0.2  $\mu$ T-6.6  $\mu$ T had “significant increase in degree of certain neurovegetative disorders (i.e., physical fatigue, psychical asthenia, lipothymia, decreased libido, melancholy, depressive tendency, and irritability). In addition, the population experienced a significant fall in total lymphocytes and CD4, CD3, and CD2 lymphocytes, as well as a rise in NK cells. Leukopenia and neutropenia were also observed in two persons chronically exposed to doses of 1.2-6.6 microT. The disorders disappeared when exposure stopped, and they reappeared on re-exposure.”

#### **4. Intracellular Calcium ions:**

As with melatonin, calcium ions are ubiquitous throughout our cells and our bodies. Calcium has been identified as a very important factor of many cellular processes. Morphological changes, cytoskeletal damage, cell death and cytolysis followed the elevation of cytosolic free-calcium levels. Calcium ions are very important for cell homeostasis, in fact, they control many functions of a variety of cellular responses, including secretion, cell proliferation and apoptosis, Mattana et al. (1997).

##### **4.1 Roles of intracellular calcium ions:**

“Even a rather small change in intracellular calcium can exert profound changes in cellular activity. In synaptic terminals of neurons, for example, calcium induces release of neurotransmitter molecules.” Berridge (1985) (p148): Hence any externally induced changes in the concentrations of intracellular calcium ions can have many far reaching consequences for the health and well-being of cells, organs and people.

**Table 1: Calcium ions play many roles in processes within cells, including:**

- As a second messenger in cell regulation signal transduction.
- In a signal transduction process component inter-linked to the cyclic AMP process.
- In the development and activation of the immune system, proliferation of lymphocytes and a variety of other components of immune response.
- In the degranulation and superoxide generation of neutrophils in the immune system.
- In Cell proliferation promotion at key parts of the cell cycle.
- In timing the initiation of DNA synthesis in the cell cycle.
- In Apoptosis (programmed cell death).
- In setting a preference for either apoptosis or proliferation of a damaged cell.
- In Transcription of proto-oncogenes (Genes which produce cancer).
- As a regulator of the opening and closing of the gap junction.

#### 4.2 Calcium ions as cellular messengers:

Calcium ion ( $\text{Ca}^{2+}$ ) functions as a ubiquitous intracellular messenger, Alberts et al. (1994). The first evidence that  $\text{Ca}^{2+}$  functions as an intracellular mediator came from an experiment in 1947, showing that the intracellular injection of a small amount of  $\text{Ca}^{2+}$  causes a skeletal muscle cell to contract. In recent years it has become clear that  $\text{Ca}^{2+}$  also acts as an intracellular messenger in a wide variety of cellular responses, including secretion and cell proliferation. Two pathways of signaling have been well defined, one used mainly by electrically active (excitable) cells and the other by almost all eucaryotic cells.

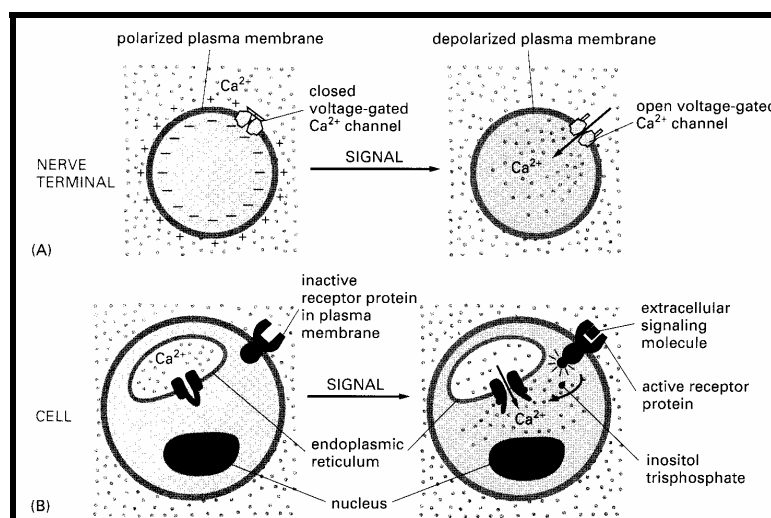


Figure 20: In (A)  $\text{Ca}^{2+}$  enters a nerve terminal from the extracellular fluid through voltage-gated  $\text{Ca}^{2+}$  channels when the nerve terminal is depolarized by an action potential. In (B) the binding of an extracellular signaling molecule to a cell-surface receptor generates inositol trisphosphate, which stimulates the release of calcium from the ER, Alberts et al. (1994).

The first of these pathways has been primarily described in nerve cells, in which depolarization of the plasma membrane causes an influx of  $\text{Ca}^{2+}$  into the nerve terminal, initiating the secretion of neurotransmitter; the  $\text{Ca}^{2+}$  enters through voltage-gated  $\text{Ca}^{2+}$  channels that open when the plasma membrane of the nerve terminal is depolarized by an invading action potential. In the second, ubiquitous pathway the binding of extracellular signaling molecules to the cell-surface receptors causes the release of  $\text{Ca}^{2+}$  from the endoplasmic reticulum (ER), Figure 20.

Calmodulin is a ubiquitous intracellular  $\text{Ca}^{2+}$  receptor. Calmodulin is a  $\text{Ca}^{2+}$  binding protein which is found in all eucaryotic cells that have been examined. A typical cell contains more than  $10^7$  molecules of calmodulin, which constitutes as much as 1 % of the total protein mass of the cell. Calmodulin functions as a multipurpose intracellular  $\text{Ca}^{2+}$  receptor, mediating many  $\text{Ca}^{2+}$  regulated processes. It is a highly conserved, single polypeptide chain of about 150 amino acids, with four high-affinity  $\text{Ca}^{2+}$ -binding sites, and it undergoes a conformational change when it binds to  $\text{Ca}^{2+}$ .

#### 4.3 Calcium ion and cAMP pathways are interlinked:

The cyclic AMP and  $\text{Ca}^{2+}$  intracellular signaling pathways interact at several levels in the hierarchy of cellular control. Firstly, cytosolic  $\text{Ca}^{2+}$  and cyclic AMP levels can influence each other. For example, some forms of enzymes that break down and make cyclic AMP are regulated by  $\text{Ca}^{2+}$ -calmodulin complexes. Also enzymes directly regulated by  $\text{Ca}^{2+}$  and cyclic AMP can influence each other, Alberts et al. (1994). In association with diacylglycerol release, calcium can activate protein Kinase-C dependent signal transduction pathways, thus modifying gene transcription, Clutton (1997). Thus induced alteration of intracellular calcium concentrations, disrupting the homeostasis of the cell, has serious consequences for the health and future development of the cell.

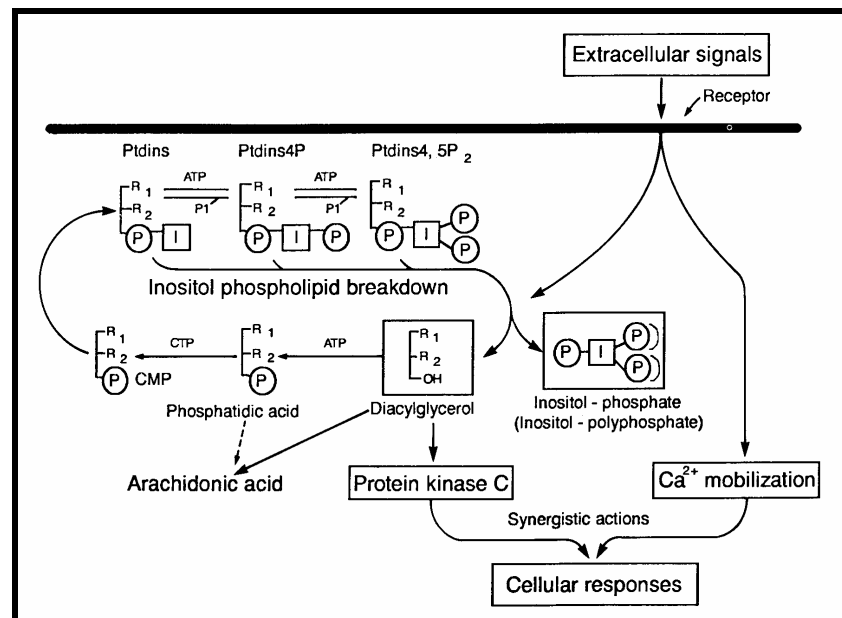


Figure 21: Illustrating the role of protein kinase C and calcium ions in signal transduction and cell responses to tumor promoters, Adey (1990).

Also, calcium ions, in partnership with cyclic AMP, control the proliferation of non-tumorigenic cells in vitro and in vivo. While it does not seem to be involved in the

proliferative activation of cells such as hepatocytes (in vivo) or small lymphocytes (in vitro), it does control two later stages of prereplicative (G1) development. It must be one of the very many regulatory and permissive factors affecting early prereplicative development, because severe calcium deprivation reversibly arrests some types of cell early in the G1 phase of their growth-division cycle in vitro, Whitfield et al. (1979).

However, calcium more specifically and much more often regulates a later (mid or late G1) stage of prereplicative development. Thus, regardless of its severity or the type of cell, calcium deprivation in vitro or in vivo reversibly stops proliferative development at that part of the G1 phase in which the cellular cyclic AMP content transiently rises and the synthesis of the four deoxyribonucleotides begins. The evidence points to calcium and the cyclic AMP surge being co-generators of the signal committing the cell to DNA synthesis.

The evidence is best explained so far by the cyclic AMP surge causing a surge of calcium ions which combine with molecules of the multi-purpose, calcium-dependent, regulator protein calmodulin (CDR) somewhere between the cell surface and the cytosol. The resulting Ca-calmodulin complexes then stimulate many different (and possibly membrane-associated) enzymes such as protein kinases, one of which produces the DNA-synthetic initiator, Whitfield et al. (1979).

#### 4.4 Calcium ions and gap junction communication:

Gap junctions are protein structures which link adjacent cells and provide a channel for the passing of small messenger molecules of 1000 daltons or less. The gap junction can open and close to control the flow. The opening and closing is regulated by the pH and calcium ion concentration. High  $\text{Ca}^{2+}$  shuts the gap junction while low  $\text{Ca}^{2+}$  open the gap junction, p 960 Alberts et al. (1994). Thus calcium ions play another key role in maintaining or interrupting the communication mechanisms for maintaining the health of cells because gap junctions are used to sense differences between cells and to initiate corrections in regulatory behaviour as necessary.

Both cAMP and calcium ions flow through gap junctions. A lowering of the intracellular calcium ion concentration has the effect of closing the gap junction and restricting this regulatory function.

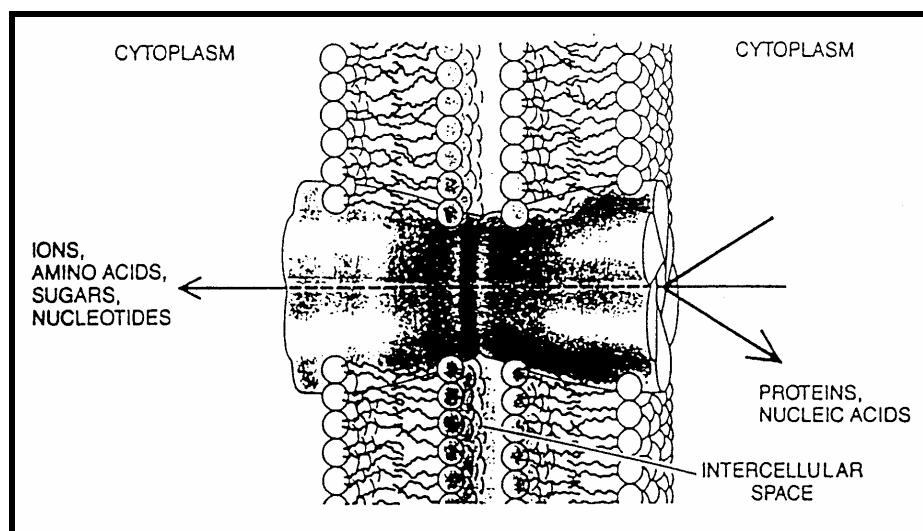


Figure 22: Schematic diagram of a gap junction between two cells.

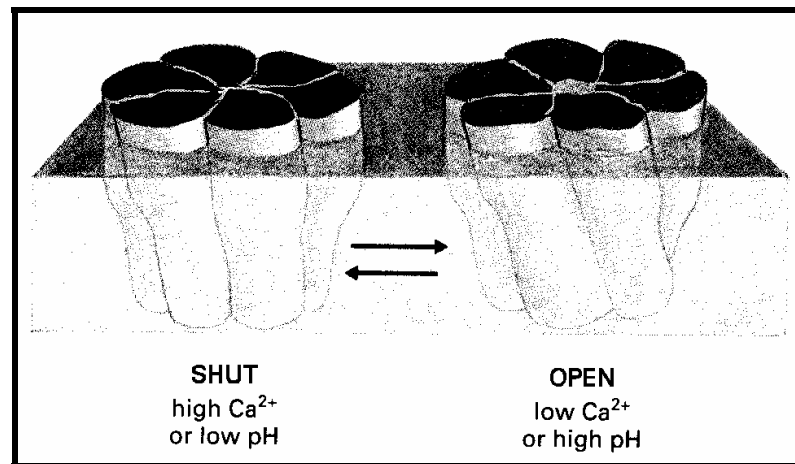


Figure 23: A small rotation triggered by high calcium ions or low pH causes the gap junction to close.

#### 4.5 Calcium ions and the immune system:

Grinstein and Klip (1989) state that:

**“It is now widely accepted that calcium plays a central role in the development of the immune system response. An elevation of  $\text{Ca}^{2+}$  (calcium ions) is a nearly universal feature associated with activation of cells of the immune system. Many responses normally elicited by physiological stimuli can be mimicked by simply elevating  $\text{Ca}^{2+}$  (calcium ions) by means of exogenous ionophores. These latter agents, which selectively increase the permeability of the membrane to divalent cations, such as  $\text{Ca}^{2+}$  (calcium ions), have been reported to induce degranulation and superoxide generation of neutrophils, proliferation of lymphocytes and a variety of other components of immune response.”**

#### 4.6 Calcium ions and cell proliferation:

Calcium ions ( $\text{Ca}^{2+}$ ) have been identified as mediators of proliferative and morphogenetic processes in many eukaryotic cells. Rodriguez-del Valle and Rodriguez-Medina (1993)

Durham and Walton (1982) outline the role of  $\text{Ca}^{2+}$  in cell proliferation.

**“Several lines of evidence suggest that  $\text{Ca}^{2+}$  ions control cell proliferation:  $\text{Ca}^{2+}$  entry into cytoplasm acts as a general mitogen; serum and serum-replacements induce  $\text{Ca}^{2+}$  influx; the  $\text{Ca}^{2+}$  concentrations in growth media required to support the proliferation of normal cells are much higher than those required for cancer cells; serum and growth factors reduce the  $\text{Ca}^{2+}$  requirements of normal cells; tumour promoters alter  $\text{Ca}^{2+}$  fluxes via a mechanism used principally by growth factors. Minor supporting evidence includes the effects of various drugs and viruses, and the behaviour of tumour cell mitochondria and intercellular junctions. It is still not possible to decide exactly where and when inside cells the critical effect of  $\text{Ca}^{2+}$  on proliferation occurs. Carried to its logical conclusion, present evidence suggests that an overridden or bypassed  $\text{Ca}^{2+}$  control process may be the key, common determinant of unrestrained proliferation in cancer cells.”**

Earlier work of Whitfield et al. (1979) revealed that altered intracellular calcium concentrations affected the cell cycle at particular points, producing different effects depending on the stage of the cell cycle.

**“Calcium, in partnership with cyclic AMP, controls the proliferation of non-tumorigenic cells in vitro and in vivo. Calcium must be one of the very many regulatory and permissive factors affecting early prereplicative development, because severe calcium deprivation reversibly arrests some types of cell early in the G1 phase of their growth-division cycle in vitro. However, calcium more specifically and much more often regulates a later (mid or late G1) stage of prereplicative development. Thus, regardless of its severity or the type of cell, calcium deprivation in vitro or in vivo reversibly stops proliferative development at that part of the G1 phase in which the cellular cyclic AMP content transiently rises and the synthesis of the four deoxyribonucleotides begins. The evidence points to calcium and the cyclic AMP surge being co-generators of the signal committing the cell to DNA synthesis.”**

This indicates that induced calcium ion efflux from a cell during the G1 phase of the cell cycle will reversibly stop the proliferation (stop or slow the cell cycle). Thus calcium ion efflux is expected to be associated with a slowing of the cell proliferation rate.  $\text{Ca}^{2+}$  also plays a key role in initiating the DNA synthesis.

#### **4.7 Calcium ions and apoptosis:**

A damaged cell can be programmed to die. This process is called “apoptosis”. Severe cell damage is caused by reactive oxygen species (ROS), which includes hydroxyl radical, hydrogen peroxide, superoxide etc.. After damage, if cellular balance is not restored, a number of pathological processes are elicited. Predominant processes resulting from oxidative stress include oxidative lipid degeneration, the loss of intracellular calcium homeostasis and alteration of metabolic pathways. All of these processes, including altered cellular calcium concentrations, have been recorded as apoptosis models. For example, calcium-dependent endonuclease activation is believed to initiate chromatin fragmentation frequently observed in apoptotic cells, Lemasters et al. (1987) and Orrenius et al. (1989).

#### **4.8 Intracellular calcium concentration alters the damaged cell's futures:**

Balcer-Kubiczek (1994) links intracellular calcium levels to the future of damaged cells between becoming neoplastic (cancer) or dying by apoptosis. This is demonstrated by the way that the cancer promoter TPA, in low concentrations, “has been shown to switch the effect of calcium elevation from cell death to cell proliferation, probably by the activation of protein kinase C.” and “TPA leads to the maintenance of malignant phenotype by blocking apoptosis, thus allowing potentially transformed cells to survive and to develop transformational damage.”

Cytolysis is the process of dissolution or destruction of cells. Morphological changes, cytoskeletal damage, cell death and cytolysis followed the elevation of cytosolic free-calcium levels. Calcium ions are very important for cell homeostasis, in fact, they control the functions of a variety of cellular responses, including secretion, cell proliferation and apoptosis, Mattana et al. (1997). Hence reductions of intracellular calcium ions has a very important effect. Lower than normal intracellular calcium concentrations fail to activate the



protein kinase C which is necessary for apoptosis to take place. Hence damaged cells survive and proliferate with their transformed genetic damaged or changed material.

## 5. The role of EMR and EMF on Calcium ions:

### 5.1 ELF fields and calcium ion effects in the immune system:

Electromagnetic fields, through their effect on calcium ions, play a vital role in the immune system, Walleczek (1992). Walleczek (1992) quotes research relating to the role of calcium, sodium and potassium ions, including research showing that EMF could alter the activity of the membrane incorporated  $\text{Ca}^{2+}$ -ATPase responsible for pumping  $\text{Ca}^{2+}$  out of the cell (calcium ion efflux).

In addition, data from two laboratories demonstrate that ELF fields alter the activity of another membrane ion pump,  $\text{Na}^+/\text{K}^+$ -ATPase with current densities as low as  $50\mu\text{A}/\text{cm}^2$  and estimated, by the authors, to also have an effect at  $1\mu\text{A}/\text{cm}^2$ . At  $50\mu\text{A}/\text{cm}^2$ ,  $J = 0.5 \text{ A}/\text{m}^2$ ;  $E=2.5 \text{ V}/\text{m}$ , assuming  $\sigma=0.2 \text{ S}/\text{m}$ . Hence  $S= 1.7 \mu\text{W}/\text{cm}^2$  and  $\text{SAR} = 0.00063 \text{ W}/\text{kg}$ . If the extrapolation to  $1\mu\text{A}/\text{cm}^2$  is confirmed then the EMR effects will be occurring at  $1/2500^{\text{th}}$  of the S and SAR levels estimated here.

This demonstrates the extremely low induced currents, SARs and energy densities which are associated with EMR induced changes in ion pumping and calcium, sodium and potassium efflux at the cellular level.

Walleczek and Budinger (1992) report that:

**“To date, at least 10 different laboratories, including our own, have reported ELF magnetic influences on lymphoid cells, and stimulatory as well as inhibitory effects on parameters related to calcium metabolism or RNA- and DNA-synthesis have been observed.”**

They also state that:

**“A plausible magnetic interaction mechanism based on radical pair recombination reactions which are linked to cellular signal transduction and application processes has been proposed (Grundler et al. (1992)). Magnetic field intensities similar to the intensities used in most experiments (e.g. 1-30 mT) are known from magnetochemistry to be able to influence *non-thermally* the kinetics and product yields from radical pair reactions *in vitro*, Steiner et al. (1989). The underlying reaction scheme is well known and is described by the radical pair mechanism.**

**For this mechanism to be applicable to the data reported here, a pathway by which magnetically-sensitive radical-dependent processes could influence mitogen-induced lymphocyte  $\text{Ca}^{2+}$  signaling must be postulated. There is new evidence that such pathways exist.**

**For example, Con A-induced  $\text{Ca}^{2+}$  uptake in rat thymic lymphocytes has been shown to depend on the generation of reactive oxygen radical species. There is also evidence from inhibition studies that cytochrome P-450 activity may be**

involved in  $\text{Ca}^{2+}$  uptake regulation in rat thymic lymphocytes, Alvarez et al. (1992), and it is known that P-450 function proceeds via radical pair recombination steps, Hollenberg (1992). Thus it is plausible to investigate if externally applied magnetic fields may interfere with radical pair reactions and as a consequence, may alter lymphocyte  $\text{Ca}^{2+}$  regulation.”

Calcium ion influx has been shown to play a role in the transcript levels of proto-oncogenes c-myc and c-fos which alters in the presence of electromagnetic fields, Karabakhtsian et al. (1994). (Proto-oncogenes: altered genes which become carcinogenic.) Lin et al. (1994) identified a specific part of the c-myc promoter which is responsive to electric and magnetic fields. Phillips (1993) exposed T-lymphoblastoid cells to 60 Hz magnetic field and found alterations to the transcription of genes encoding for c-fos, c-jun, c-myc and protein Kinase-C.

Modulated electric and magnetic fields alter key genetic characteristics and therefore are mutagenic. This is further confirmed with studies showing EMR/EMF exposure is associated with chromosome aberrations and DNA damage, e.g. Vijayalaxmi et al. (1997) and Lai and Singh (1997). Lindstrom et al. (1995) replicated and extended the research of Waliczek (1992), using the T-cell line (lymphocytes) for human leukaemia cells, and show that oscillating low-level magnetic fields produce the same calcium ion reaction as does an antibody. They show that weak magnetic fields initiate calcium ion oscillations with a threshold flux density of 40  $\mu\text{T}$ , a plateau at 150  $\mu\text{T}$  and a frequency range from 5 to 100 Hz, with a fairly broad peak at 50 Hz. Galvanovskis et al. (1996) report significant 30% reductions in the calcium ion oscillation amplitude in human leukaemia T-cells when exposed to 50 Hz magnetic fields. The key role of modulation frequency in the alteration of calcium ions was recognized early.

## 5.2 Calcium ion efflux and modulation frequency

The very early research on brain cells efflux and influx of calcium ions using external ELF frequency fields in the same range of EEG frequencies was carried out by Dr Ross Adey and his research team, e.g. Figure 24a.

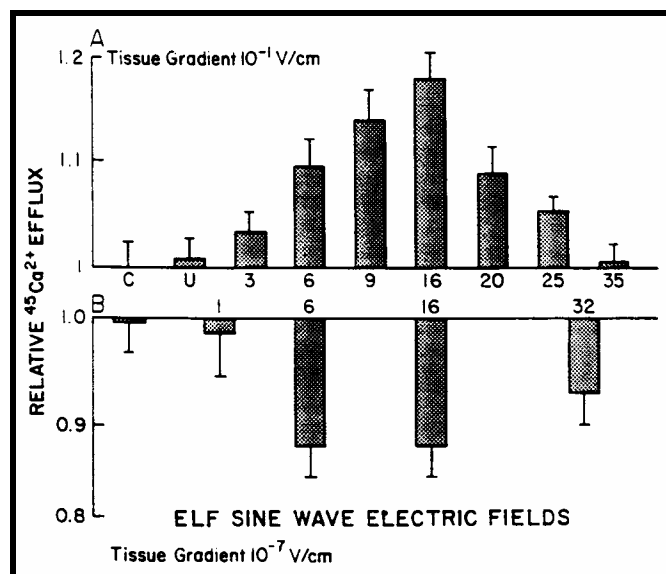


Figure 24a: ELF induced calcium ion efflux in chick brain cells from (A) an ELF modulated 147 MHz signal and (B) an ELF signal, Adey (1988).

Table 2 shows a concentration on using a modulation frequency of 16 Hz which was identified early to be an ELF frequency associated with strong calcium ion efflux compared with frequencies near it. A leading researcher in this area, Dr Carl Blackman of the U.S.E.P.A. has shown that research has identified modulation frequencies which significantly alter calcium ion efflux out to 510 Hz, Figure 24b.

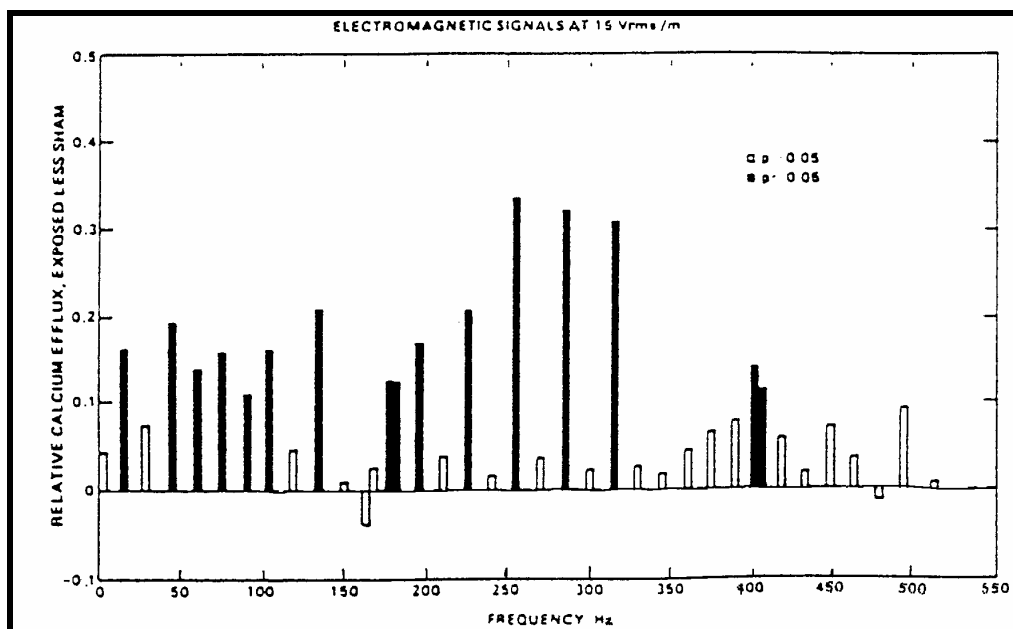


Figure 24b: The effect of 15 V/m electromagnetic fields on the efflux of calcium ions from chicken brain tissue as a function of modulation frequency. The relative efflux is the difference between exposed and unexposed samples. The data from 1 to 120 Hz are taken from Blackman et al. (1985). Blackman et al. (1988).

Their research further shows the involvement of polypeptide molecules, specifically poly-L-lysine, which the authors postulate may explain the intracellular calcium ion EMR effects on cell membrane surfaces, through the polylysine causing strong deformations on the cell surface which could trigger the release of stored calcium cations from intracellular pools, thus starting the oscillations.

Through replicating and extending the experiments of other laboratories, Dr Carl Blackman and his team at the U.S. Environmental Protection Agency have become the world leaders in calcium ion efflux research. That is why he was well qualified to review the research results and conclude, Blackman (1990) that

**"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".**

There is extremely strong evidence that both ELF and ELF modulated RF/MW radiation causes calcium ion efflux from cells which significantly alters the intracellular calcium concentrations, reducing the efficacy of lymphocytes in the immune system, participating in the alteration of transformation of pineal serotonin to melatonin and altering the damaged cells likelihood of becoming neoplastic or dying by apoptosis.

### 5.3 The melatonin - calcium ion efflux link:

The interdependence of cyclic AMP and calcium ions is outlined in section 3.3 above. The following outlines an hypothesis for modulated RF/MW effects on melatonin.

Since it has been shown:

- That ELF electric fields do reduce melatonin production in living rats brains; Wilson et al. (1986).
- That RF/MW signals produce tissue level electric fields about a million times higher than imposed ELF signals, Adey (1981).
- That RF/MW signals are resonantly absorbed at the cell membrane, Liu and Cleary (1995).
- That altering the electric and thermal fields on the surface of the cell membrane change the binding characteristics of  $H^+$  and  $Ca^{2+}$  ions on the outer surface of the membrane, Adey (1990).
- That modulated RF/MW has been shown to induce significant calcium ion efflux from cells, Table 2 above.
- That it known that the cyclic AMP signal transduction pathway and the Calcium ion signal transduction pathway interact, Alberts et al. (1994).
- That in the pinealocyte cell the cAMP pathway is involved in regulating the transformation of serotonin to melatonin.

The calcium ion mediated responses to neurotransmitters on the membrane of the pineal cells has been discussed by Wilson et al. (1989) in relation to ELF induced melatonin reduction. Thus it is highly probable that pinealocytes exposed to modulated RF/MW will experience an outflow of calcium ions, a reduction of the cAMP signal transduction activity and a reduction in the production of melatonin. This is a highly plausible mechanism to explain why RF/MW can reduce pineal melatonin production with consequent the adverse health effects.

**Table 2: Summary of Studies concerning Calcium ion efflux and ELF Modulation of RF.**

Effects	Species	RF (MHz)	Mod <sup>n</sup> (Hz)	Intensity (mW/cm <sup>2</sup> )	Time (min)	SAR (W/kg)	Reference
<u>Altered calcium-ion efflux in brain tissue in vitro:</u>							
Frequency specificity	Chicken	147	6-20	1-2	20	0.002*	Bawin et al.(1975)
influence of pH and lanthanum	Chicken	450	16	0.75	20	0.0035	Bawin et al.(1978)
frequency and intensity specificity	Chicken	147	16	0.83	20	0.0014	Blackman et al.(1979)
intensity specificity and sample spacing	Chicken	147	9,16	0.083	20	0.0014	Blackman et al.(1980a)
intensity specificity and sample spacing	Chicken	147	16	0.083	20	0.0014	Joines et al (1981)
intensity specificity	Chicken	450	16	0.1-1	20	0.005-0.005	Sheppard et al.(1979)
two intensity ranges	Chicken	50	16	1.5 3.6	20 20	0.0013 0.0035	Blackman et al.(1980b)
theoretical analysis of RF dependence	Chicken	50 147 450	16	-	20	~0.001	Joines and Blackman(1980) Athey (1981); Joines and Blackman (1981).
test of predictions of theoretical analyses	Chicken	147	16	0.37 0.49	20	0.0006 0.0008	Blackman et al.(1981)
no effect for pulse modulation	Rat	1000	16,32	0.5-15	20	0.15-4.35	Shelton and Merritt (1981)
no effect for pulse modulation	Rat	1000 2450	16 8,16,32	1,10 1	20	0.29-2.9 0.3	Merritt et al. (1982)
change in calcium efflux kinetics in synaptosomes	Rat	450	16	0.5	10	-	Lin-Liu and Adey (1982)
frequency and intensity specificity in cultured neuroblastoma cells	Human being	915	16	-	30	0.05	Dutta et al.(1984)
<u>Altered calcium ion efflux in brain tissue in vivo:</u>							
no effect for pulse mod.	Rat	2060	8,16,32	0.5-10	20	0.12-2.4	Merritt et al.(1982)
change in efflux kinetics	Cat	450	16	3	60	0.29	Adey et al.(1982)
Changes found in pancreatic slices	Rat	147	16	2	60-150	<0.075	Albert et al.(1980)
Suppressed T-lymphocyte Changes in Hearts	Mouse Frog	450 240	16-100 0.5,16	1.5 30	120 30	- 0.00015	Lyle et al.(1983) Schwartz et al (1990)

## **6. Neurological Effects of RF radiation:**

### **6.1 introduction to RF and Neurological functions of the brain:**

Dr Henry Lai summarizes this research in Lai (1994). His opening statement is:

#### **“INTRODUCTION**

Many reports in the literature have suggested the effect of exposure to radiofrequency electromagnetic radiation (RFR) (10 kHz-300,000 MHz) on the functions of the nervous system. Such effects are of great concern to researchers in bioelectromagnetics, since the nervous system co-ordinates and controls an organism's responses to the environment through autonomic and voluntary muscular movements and neurohumoral functions. As it was suggested in the early stages of bioelectromagnetics research, behavioral changes could be the most sensitive effects of RFR exposure. At the summary of session B of the proceedings of an international symposium held in Warsaw, Poland, in 1973, it was stated that "The reaction of the central nervous system to microwaves may serve as an early indicator of disturbances in regulatory functions of many systems" [Czerski et al., 1974].

Studies on the effects of RFR on the nervous system involve many aspects: morphology, electrophysiology, neurochemistry, neuropsychopharmacology, and psychology. An obvious effect of RFR on an organism is an increase in temperature in the tissue, which will trigger physiological and behavioral thermal regulatory responses. These responses involve neural activities both in the central and peripheral nervous systems. The effects of RFR on thermoregulation have been extensively studied and reviewed in the literature [Adair, 1983; Stern, 1980]. The topic of thermoregulation will not be reviewed in this chapter. Since this paper deals mainly with the effects of RFR on the central nervous system, the effect on neuroendocrine functions also will not be reviewed here. It is, however, an important area of research since disturbances in neuroendocrine functions are related to stress, alteration of immunological responses and tumor development [Cotman et al. (1987), Dunn (1989), Plotnikoff et al. (1991)]. “

Dr Lai's review reports several thermal effects, e.g. Baranski (1972) reported edema and heat lesions in the brain of guinea pigs exposed to a single 3 h session to 3000 MHz RFR at a power density of 25 mW/cm<sup>2</sup> (SAR 3.75 W/kg). After repeated exposure to a lower level, myelin degeneration and glia cell proliferation were reported for guinea pigs (3.5 mW/cm<sup>2</sup>, SAR 0.53 W/kg) and rabbits (5 mW/cm<sup>2</sup>, SAR 0.75 W/kg). Pulsed RFR produced more pronounced effects than continuous-wave radiation at the same power density. These second set of experiments are athermal and produce an athermal effect.

### **6.2 Blood Brain Barrier:**

Frey (1975) reports increases in the permeability of the microwave exposed blood-brain barrier (BBB). For example a 30 minute exposure to 1200 MHz RFR (2.4 mW/cm<sup>2</sup>, SAR 1.0 W/kg) as fluorescent dye was found to have crossed the BBB and was found mainly in the lateral and third ventricles of the brain. Other researchers found BBB changes only with

high exposures, e.g. Merrett et al. (1978). Dr Lai concludes: "It was apparent that in the majority of cases a high intensity RFR is required to alter the permeability of the BBB."

### 6.3 Evoked Potential:

Changes in the Evoked potential have been detected. For example, Taylor and Ashleman (1975) found that when the spinal cord was irradiated with a continuous wave 2450 MHz RFR at an incident power of 7.5 W, decreases in latency and amplitude of the reflex response were observed during exposure (3 min) and responses returned to normal immediately after exposure. They also reported that raising the temperature of the spinal cord produced electrophysiological effects similar to those of RFR.

### 6.4 Calcium ion balance in brain cells:

One of the most repeated effects of ELF modulated RF/MW is the calcium ion efflux from brain cells and muscle cells. Bawin et al. (1976) summarize some of the effects known up to that time. They are:

- Weak extracellular voltage gradients (1-5 mV/mm) have been shown to significantly affect the excitability or firing thresholds of the spinal neurons of cats.
- Nelson (1966) pointed out that the complex structural organization of brain tissues, as seen in the cerebrum, should be highly favorable for multiple electric field interactions, both in the intricate rate of overlapping dendritic trees and between macromolecules on the extracellular space and the glycoproteins of the out cell membrane.
- Weak pulsed electric currents (2-5 mV/mm, 200 pulses/sec) applied across the cat cortex were able to trigger the release of previously bound radioactive calcium ( $^{45}\text{Ca}^{2+}$ ).
- Intracranial injection of  $\text{Ca}^{2+}$  or  $\text{Mg}^{2+}$  in chronically implanted neonatal chicks resulted in an almost immediate synchronization of the hyperstriatal EEG, accompanied by behavioural depression, Bawin et al. (1984). During successive testing days, the animals appeared to recover behaviorally but never showed any sustained EEG arousal. By contrast animals treated with sodium chloride recovered completely within the first hour after injection.

Because of the high sensitivity of the chick forebrain to small perturbations of the extracellular divalent cations, this was chosen for investigating in vitro, possible interactions of weak voltage gradients induced by VHF radiation and ionic movements in cerebral tissue. The experiment showed that weak VHF fields (147 MHz, 1 mW/cm<sup>2</sup>), amplitude modulated at brain wave frequencies (6 Hz and 16 Hz) are able to increase the calcium efflux from the isolated brain of the neonatal chick. This result has been repeated by totally independent laboratories, and extended to a wide range of modulation frequencies up to 510 Hz, Blackman et al. (1988), and down to extremely low exposures. These include 10  $\mu\text{W}/\text{cm}^2$  (SAR=0.0075W/kg), Shandala et al.(1979) and an SAR of 0.00015 W/kg (S = 0.5  $\mu\text{W}/\text{cm}^2$ ), Schwartz et al. (1990).

Hence Calcium ion efflux is shown to alter mammal EEG and behaviour.

## 6.5 EEG alteration by EMR:

Professor Adey, and others, have been able to show that imposed oscillating electromagnetic fields can produce significant and repeatable changes in the behaviour of advanced mammals (cats and monkeys) in the laboratory, Adey et al. (1979). They used 450 MHz microwave signal at  $0.8 \text{ mW/cm}^2$ , modulated at 10 Hz, which produced an EEG level voltage gradient in the cat's brain of  $0.1 \text{ V/cm}$  and no detectable heating.

### 6.5.1 Altered Circadian Rhythm with extremely low exposure to RF and ELF:

Wever (1974) showed changes in human subjects isolated from environmental stimuli including ELF fields, which resulted in altered circadian rhythms which were corrected by applying a 10 Hz,  $2.5 \text{ V/m}$  field, which produces about  $10^{-7} \text{ V/cm}$  in tissue. The experiment was repeated using birds, with similar results, of lengthened circadian rhythms.

**“RF fields that are sinusoidally amplitude modulated at ELF frequencies produce a wide range of biological interactions. Induced electric gradients can be substantially higher than those produced by simple ELF electric fields, and at levels of 10-100 mV/cm, are the same range as intrinsic oscillations generated biologically, such as the electroencephalogram (EEG).”, Adey (1990)**

How does the brain cells sense these EMR fields ? The cell membrane outer surface is charged and the alpha-helix glycoprotein stands outside ends are highly charged. Calcium and hydrogen ions interact with the strands and its receptors, which is the first and most sensitive transductive coupling in brain tissue.

## 6.6 Neurotransmitters are altered by microwaves:

Many studies have shown significant efflux of calcium ions from cells exposed to ELF modulated RF and ELF fields. Since calcium ions ( $\text{Ca}^{2+}$ ) are known to stimulate specific glutamate binding to the synaptic membrane it is of value to determine whether modulated RF/MW alters glutamate binding.

An efflux has been recorded for the amino acid neurotransmitter gamma-aminobutyric acid (GABA), Kolomytkin et al. (1994), in association with microwaves modulated at 16 Hz. This is very significant since GABA and glutamateric synapses make up about 60 % of the CNS and calcium ions appear to hold the key to every aspect of cell-surface transduction, Adey (1979). Kolomytkin et al. (1994) showed that at 915 MHz microwave signal, modulated at 16 Hz, altered the binding of 3H-glutamate and 3H-muscimol in rats brains, at power densities below  $50 \text{ } \mu\text{W/cm}^2$ , which are statistically significantly different from controls to below  $10 \text{ } \mu\text{W/cm}^2$ , Figure 25.

Kolomytkin et al. (1994) link these changes to  $\text{Ca}^{2+}$  ions which have been shown to stimulate specific glutamate binding to synaptic membranes due to the activation of a calcium-dependent protease and resulting proteolysis (splitting into fragments) of cytoskeletal proteins. Since it is shown that modulated microwaves increase the glutamate uptake by synaptomes, Kolomytkin et al. pose the question as to whether microwaves directly affect the synaptosomes or does their sensitivity require some other brain system ? They determined that it was the synaptosomes which were sensitive to the microwaves. They then investigated whether it was a simple heating effect.



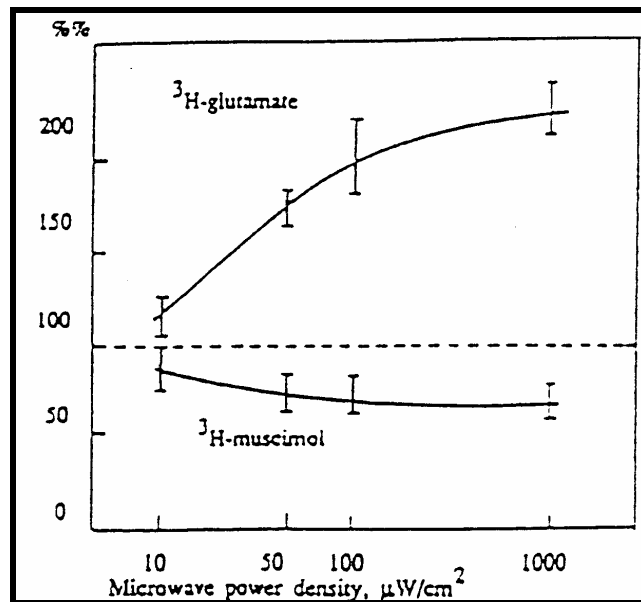


Figure 25: Altered the binding of  $^3\text{H}$ -glutamate and  $^3\text{H}$ -muscimol in rats brains versus microwave power density (915 MHz, modulated at 16 Hz), Kolomytkin et al. (1994).

Heating the samples to produce the same mean SAR did not produce the result. Hence they proposed the mechanism of localized microheating at the cell membrane. This membrane heating in the presence of microwaves has been demonstrated now by Liu and Cleary (1996). However, heating alone at the cell membrane level is unlikely to be the cause of the trends shown in Figure 20 since they follow a systematic change down to below  $10 \mu\text{W}/\text{cm}^2$ .

Kolomytkin et al. (1994) conclude that:

**“Our findings can be directly related to and complement the findings of Frey and Wesler (1990) and Kavakiers and Ossenkopp (1992). Frey found that dopamine and opiate systems of the brain were influenced by exposure to low intensity electromagnetic fields. Kavaliers has shown that electromagnetic fields can influence the functioning of multiple endogenous opoid systems and that the effects depend on the modulation of the field. Considering the great importance of GABA and glutamatergic systems in both normal and pathological brain processes, the finding of low intensity microwaves on these receptor systems is of significance.”**

This conclusion, which shows the findings of three independent studies, conforms to the requests of sceptics to show replication. Replication has been carried out and the effects are confirmed.

## 6.7 The Electroencephalograph (EEG) evidence from animals

The Electroencephalograph (EEG) provides a non-invasive means of monitoring the electrical activity of the brain.

Three studies show arousal in anesthetized rabbits with chronic athermal RFR exposure, Baranski and Edelwejn (1974) ( $7 \text{ mW}/\text{cm}^2$ , 200 h), Goldstein and Sisko (1974) (5 min,

9300 MHz, 0.7-2.8 mW/cm<sup>2</sup> ), and Servante et al. (1975) (rats, 10 days, 3000 MHz, 500-600 pps, 5 mW/cm<sup>2</sup> ).

Dumanskiy and Shandala (1974) and their colleagues reported altered conditioned reflex in rabbits and rats chronically exposed to extremely low levels of VHF and microwave fields. They used either 50 MHz or 2.5 GHz CW fields or 10 GHz 1  $\mu$ s pulses at 1,000 or 20 Hz, with 10-12h daily exposure with 50 MHz and 8 h with microwave fields. They found statistically significant effects with field intensities between 1.9 and 2.0  $\mu$ W/cm<sup>2</sup>.

In each experiment the animals were irradiated for 120 days, with a 60 day follow-up. For the first 10 days the animals were “somewhat excited” and reacted to the onset of exposure. Thereafter responses to conditioned stimuli has a longer latency, with weaker responses to positive stimuli and more numerous missed responses, leading to “pathologic stagnation and inertia”.

Shandala et al. (1979) found statistically significant changes in the EEG and brain biochemistry of rats and rabbits exposed to 2.375 GHz microwaves at 10, 50 and 500  $\mu$ W/cm<sup>2</sup>, for 7 hours/day over 30 days. The 10 $\mu$ W/cm<sup>2</sup> and 50 $\mu$ W/cm<sup>2</sup> initially stimulate brain activity, while 500 $\mu$ W/cm<sup>2</sup> causes suppression as seen from the increase in slow, high amplitude  $\Delta$ -waves. After 1 month of exposure to a power density of 10 $\mu$ W/cm<sup>2</sup> (for 7hr/day, i.e. averaging 2.9 $\mu$ W/cm<sup>2</sup>) a reliable ( $p < 0.05$ ) increase was observed in the alpha-rhythm in the sensory-motor and visual cortex due to a suppression of the slow EEG components.

Clifford et al. (1989), in an effort to duplicate research carried out in the Soviet Union. The U.S. group found significantly less Na<sup>+</sup>, K<sup>+</sup> and ATPase activity in microwave exposed animals compared to sham exposed animals. Both groups found incidences of statistically significant effects in the power spectrum analysis of EEG frequency, but not at the same frequency.

These interactions included entrainment of brain EEG rhythms at the same frequencies as the ELF components of the imposed fields, conditioned EEG responses to imposed fields, and modulation of brain and behavioural states, Bawin et al (1973); and in non-nervous tissues, strong effects on cell membrane functions, including modulation of intercellular communication through gap junctions mechanisms, Fletcher et al. (1986), reduction of cell mediated cytolytic immune responses, Lyle et al. (1983), and mediation of intracellular enzymes that are markers of signals arising at cell membranes and couple to the cell interior, Byus et al. (1984, 1988).

Vorobyov et al. (1997) studied short-term alterations of EEG in mice exposed to ELF fields carried on a 945 MHz microwave carrier with exposures in the range 100 to 200  $\mu$ W/cm<sup>2</sup>. They found an induced asymmetry in the EEG on each side of the brain of an ongoing EEG power decrease in the 1.5-3 Hz range in the left hemisphere and a power increase in the 10-14 Hz and 20-30 Hz ranges in the right hemisphere. Significant elevations of EEG asymmetry in the 10-14 Hz range were observed after the first 20 s after five onsets of the microwave field, when averaged spectra were obtained for every 10 s. In their conclusions they comment that :

**”One of the possible key links in this effect can be calcium ion exchange in brain tissue (Adey (1981)). Indeed it was found that the intracellular**

**calmodulin level was changed by modulated microwave fields, Katkov et al. (1992), This change, as is known, can cause the change in receptor sensitivity to mediators, because in neural tissues both the transmitter-receptor mechanism and the second messenger are  $\text{Ca}^{2+}$  dependent."**

Many studies show that RF/MW exposure at athermal levels alters the EEG in animals. This is, that brain function relating to thinking and mood, altering the various bands of the EEG spectrum. With so many effects found in the higher mammals, this suggests that human brain activity and function might also be altered by EMR exposure, especially in the microwave range and with the signal contain ELF modulation which might find some way to resonantly interact with the brains EEG signals and alter them.

Lai et al. (1989) studied the acute effect of  $1 \text{ mW/cm}^2$ ,  $\text{SAR} = 0.6 \text{ W/kg}$ , 2450 MHz radiation exposed rats for 20 to 45 min. in terms of spatial learning and memory function using a radial arm maze. The exposed rats made significantly more errors than sham exposed rats.

Adey (1991) goes a considerable way towards describing the mechanisms which underlie these changes in the brains of higher animals, including people. Dendritic cells in the brain, high levels of entrainment of ELF signals from RF/MW ELF modulated radiation, associated with changes in calcium ion concentrations and altered release and binding of neurohormones and neurotransmitters, such as GABA, serotonin and melatonin, have all been described and linked to EMR exposure.

Adey (1981) states that there is **"unequivocal experimental evidence that fields from ELF to UHF (10 Hz -450 MHz) interact directly with brain tissue"**. Some of the evidence for this is summarised in Table 3.

Table 3: Identified tissue level electric field gradients to which living species are responsive, Adey (1992).

Organism	Function	Tissue Gradient	Imposed Field
Sharks and rays	Navigation and predation	$10^{-8} \text{ V/cm}$	DC to 10 Hz
Birds	Navigation	$10^{-7} \text{ V/cm}$	0.3 G
Birds	Circadian rhythms	$10^{-7} \text{ V/cm}$	10 Hz, 2.5 V/m
Monkeys	Subjective time estimations	$10^{-7} \text{ V/cm}$	7 Hz, 10 V/m
Man	Circadian rhythms	$10^{-7} \text{ V/cm}$	10 Hz, 2.5 V/m
<u>Comparison with Intrinsic Cell and tissue Neurochemical Gradients</u>			
	Membrane Potential	$10^5 \text{ V/cm}$	
	Synaptic Potential	$10^3 \text{ V/cm}$	
	Electroencephalogram	$10^{-1} \text{ V/cm}$	

ELF fields in the order of  $10^{-7} \text{ V/cm}$  are used in orientation, navigation and prey attack in marine vertebrates, in bird navigation and in mammalian biorhythms. Similar ELF fields modify calcium binding in cat and chick cerebral tissue. At higher tissue gradients around

0.1 V/cm induced by RF fields with low-frequency modulation, there are alterations in “spontaneous” ELF fields potentials in EEG wave trains evoked as conditional responses, and in the release of calcium from cerebral tissue.

Dr Adey remarks that **“a striking feature of some of these observed interactions with weak radio-frequency (RF) fields is their relationship to the modulation frequencies in the ELF range and not to the radio carrier frequency.”**

This clearly demonstrates an athermal effect, an effect which is related to the modulation frequency and not to the intensity or frequency of the RF carrier. This data shows that biological systems involving the brain are sensitive to and reactive to induced field tissue gradients at far lower levels than assumed limitations imposed by the Membrane Potential and the Synaptic Potential, and well below field strengths associated with the EEG.

Just because effects are found in animals some claim that it does not necessarily apply to people. Cells and organs of mammals are highly similar to the cells of human beings, who are also mammals. Many toxic substances are tested on animals and the results are applied to people. In the case of EMR the reluctance to apply results shown in animal experiments to people as often related to a strong desire not to agree that there affects from commercially and militarily important technology. When it comes to the question of public health, these sectorial interests should not prevail. However, a strong reason why they should not prevail is also that the effects have also been found in people.

## **7. EMR induced EEG changes in humans:**

Are these effects found in humans? Two papers known to the author show EMR alteration of the human EEG. The first, Von Klitzing (1995) shows dominantly EEG delta to alpha rhythm change when exposed to GSM signal. The second shows sleep and EEG change with GSM phone exposure.

### **7.1 Human EEG delta to alpha when GSM exposed:**

Von Klitzing (1995) shows the same result, alpha enhancement and slow wave suppression in human subjects exposed to a GSM cell-phone like signal with an SAR of 0.001 W/kg ( $S = 0.7\mu\text{W}/\text{cm}^2$ ), (from Eq. 8 using  $\sigma=0.77 \text{ S/m}$ ) and a pulse frequency of 217 Hz. The power spectrum of one of the subjects is shown in Figure 26. Von Klitzing's paper presents an example of the 45 experiments from 17 students tested. Around 70 % of the students showed significant alteration in their EEG at these very low exposure levels.

The human subjects react much more quickly than the rat and rabbit subjects. Not all human being show this sensitivity. The author underwent the exposure and EEG test and showed no significant difference between the exposure and unexposed periods. He therefore joins the 30 % who show no effects.

### **7.2 Cell phone signal alters sleep EEG:**

Healthy people sleeping with a digital GSM cell phone on next to the bed, exposing their heads to about  $50\mu\text{W}/\text{cm}^2$  while their brain EEG was being monitored, Mann and Roschke (1996). This revealed a statistically significant disruption of alpha EEG frequency range and REM sleep. REM sleep decreased from 17.07 % to 13.91 %, which is significant at

$p < 0.05$ . In addition subjects went to sleep faster, a hypnotic effect also reported by Reite et al. (1994) who used a signal of 27.12 MHz modulated at 42.7 Hz.

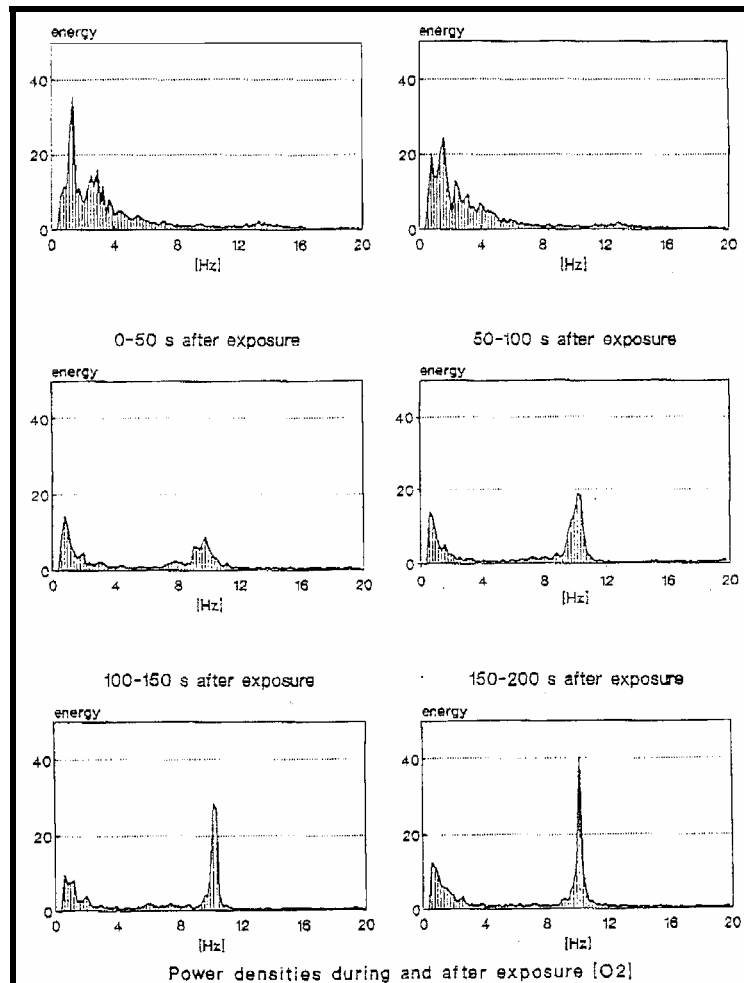


Figure 26: Human Alpha-EEG (O2-position) is altered by pulsed electromagnetic fields following first exposure, Von Klitzing (1995).

Mann and Roschke (1996) exposed 14 healthy, non-smoking, non-drinking, 21-34 year old male volunteers to 900 MHz, pulsed at 217 Hz with a pulse width of 580  $\mu$ s, digital GSM signal with a resultant average power density at the head of 50  $\mu$ W/cm<sup>2</sup>. They concluded that:

**“Besides a hypnotic effect with shortening of sleep onset latency, a REM suppressive effect with reduction of duration and percentage of REM sleep was found. Moreover, spectral analysis revealed quantitative alterations of the EEG signal during REM sleep with an increased spectral power density. Knowing the relevance of REM sleep for adequate information processing in the brain, especially concerning the mnemonic functions [Memory functions] and learning processes, the results emphasize the necessity to carry out further investigations on the action of this type of electromagnetic fields and the human organism.”**

The results are summarized in Figure 27.

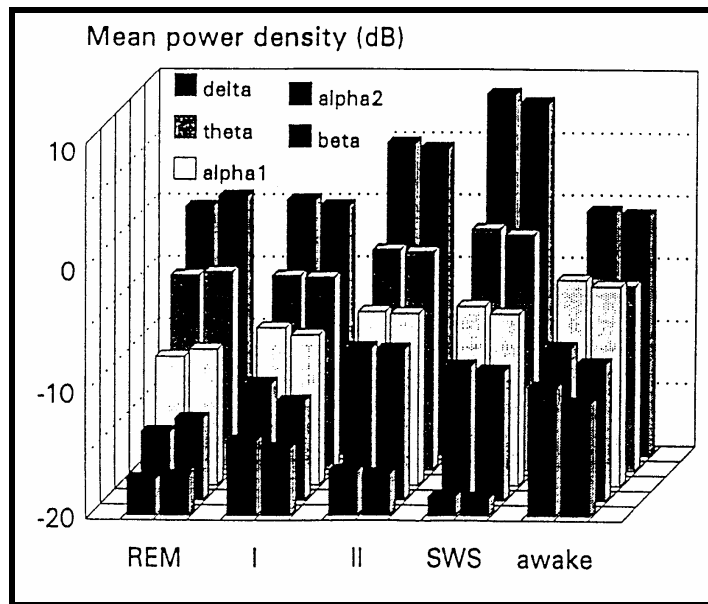


Figure 27: Mean power density (dB) of the averages of different sleep stages in 12 subjects averaged, left columns without field and right columns with GSM digital field of  $50 \mu\text{W}/\text{cm}^2$ .

Reite et al. (1994) also found an hypnotic effect when a 27.12 MHz signal, modulated at 42.7 Hz as applied over a 15 min. period. Exposed subjects reached a deeper state of sleep than sham exposed subjects.

The GSM exposed subjects also reported having fewer “bad dreams”. This is consistent with reduced melatonin. Post sleep subjective surveys found non-significant changes with GSM exposure such as reduced sleep quality, number of wakings. Post waking increased calmness and alertness, along side decreased concentration and increased anxiety. These latter two are frequently associated with increased daytime serotonin. The authors relate REM sleep impairment to memory and learning processes. Recently large numbers of cell phone users have been reporting headache, loss of concentration and memory impairment. This is consistent with these results.

Hence human studies, as with the animal studies above, shows that EMR from pulsed microwaves, including a cell phone signal, induces alterations in the human EEG in awake people, Von Klitzing (1995) and in sleeping people, Mann and Roschke (1996). Earlier German observations found that extremely low levels of EMR altered the reaction times and circadian rhythms of people. This suggests that there could be memory, and learning impairment in humans exposed to low level EMR.

## 8. Studies showing learning difficulties with EMR exposure:

Sound REM sleep is necessary for learning, memory and wellbeing. Any studies associating learning difficulties with EMR exposure would strengthen this association and the evidence of likely melatonin reduction and sleep disruption.

Three published papers or reports identify such effects:

- Chiang et al. (1989) found that visual reaction time, a measure of the function of the visual receptor and the central nervous system, varied with microwave exposure of children up to  $4 \mu\text{W}/\text{cm}^2$ . Children exposed to over  $10 \mu\text{W}/\text{cm}^2$  had lower scores in the

memory function test. They concluded “the data indicate that chronic exposure to EMFs are associated with significant changes in some physiological parameters.”

- Altpeter et al. (1995) showed a statistically significant delay in promotion from primary to secondary school in the more highly exposed school compared to a low exposure school, OR= 0.63, 95% CI: 0.43-0.85,  $p < 0.005$ . This involved short-wave radio exposure. The daily mean exposures in the highly exposed group were in the range 0.031 to  $9.1 \mu\text{W}/\text{cm}^2$ , median  $0.1 \mu\text{W}/\text{cm}^2$  and mean  $0.24 \mu\text{W}/\text{cm}^2$ .
- Kolodynski and Kolodynska (1996) investigated the effects of a RLS radar in Latvia, radiating at 154-162 MHz and pulsed at 24.4 Hz, on the performance of school children living several km in front of the radar compared to children living behind the radar. They concluded that “Motor function, memory and attention significantly differed between exposed and control groups. Children living in front of the RLS had less developed memory and attention, their reaction time was slower and their neuromuscular apparatus endurance was decreased.” Assuming that the closest child lived 2km in front of the radar, the maximum mean measured exposure is in the  $0.16 \mu\text{W}/\text{cm}^2$ .

Hence there is evidence from a wide range of RF/MW frequencies, at public exposure levels of around  $0.1 \mu\text{W}/\text{cm}^2$  and less, of learning, memory, sleep and physical performance of children; sleep disruption, aches, pains and chronic fatigue in adults. All of these symptoms are consistent with the hypothesis that RF/MW reduces nocturnal melatonin with consequent psychological and physical impairment.

## **9. Human Reactions to Atmospheric EMR/EMF changes:**

Thus the German work in the 1960's and 1970's established that naturally occurring EMR and EMF at extremely low levels influenced and altered sleep, circadian rhythm and reaction times. Later German work showed the cell phone and cellphone-like signals alter the human EEG and interfere with REM sleep. Impairment of REM sleep is associated with memory and learning difficulties. The Swiss research (Schwarzenburg Study) found a causal relationship between sleep disturbance and subsequent chronic fatigue, and short-wave radio exposures at extremely low mean levels.

### **9.1 Conclusions - EEG, Learning and EMR:**

These recent studies show unequivocal evidence that low level modulated and pulsed RF/MW signals, including GSM digital signals, alter the human EEG and affect the state of sleep in ways which interfere with information processing and learning. This confirms a neurological basis for the observed impairment of children's learning in Switzerland, Latvia and China.

Hence, far from being isolated examples, as this report brings together multiple studies showing adverse alterations of human EEG, learning, memory, reaction times, sleep efficiency in humans which has also been demonstrated in animals. In rabbits, Dumanskiy and Shandala (1974) and Shandala et al. (1979); and in cats, Bawin et al. (1973). Studies on altered reaction times and circadian rhythms in humans and animals are linked to EEG changes, Adey (1981).

These alterations are consistent with the calcium ion and melatonin effects, both of which also point to increased risks of cancer.

## 10. Cancer results from EMR/EMF

Evidence here shows that RF/MW and ELF are carcinogenic at the cellular level, increase tumours in animals with chronic exposure and increase the incidence of cancer in human populations. Leukaemia and brain tumor are commonly associated with EMR, but cancers of almost all organs have been associated.

### 10.1 The cell membrane is the primary site of many EMR interactions:

Adey (1990) reviewed the evidence for nonlinear cell membrane transductive coupling and concluded:

“We have reviewed event that couple chemical stimuli from the cell surface receptor sites to the interior. Weak imposed electromagnetic fields with low-frequency components (typically in the spectrum below 100 Hz) have proved unique tools in identifying the sequence and energetics in these events. Cell membranes are primary sites of interactions with these fields. recent observations have opened doors to new concepts of communication between cells as they whisper together across barriers of cell membranes. regulation of cell surface chemical events indicates a major amplification of initial weak triggers associated with binding of hormones, antibodies and neurotransmitters to their specific binding sites. Calcium ions play a key role in this stimulus amplification. The evidence supports nonlinear, nonequilibrium processes at critical steps in transmembrane coupling.”

“Communication between cells through gap junctions is also sensitive to low-frequency electromagnetic fields at athermal intensities. We hypothesize that cancer promotion may involve dysfunctions at cell membranes, disrupting inward and outward signal streams.”

“There is evidence that these fields may participate in the promotion phase of carcinogenesis by at least two mechanisms: through effects on immune responses and by direct effects on mechanisms regulating cell growth.”

### 10.2 Cancer and Cell Death are closely linked at the cellular level:

The cellular processes which have the potential to lead to cancer (neoplastic transformation) and to cell death (apoptosis) are very tightly related in terms of the factors which regulate cell growth and development. Apoptosis (programmed cell death) is a pathway of cell death characterized by internucleosomal digestion of the genomic DNA, Balcer-Kubiczek (1994). Neoplastic transformation involves the alteration of the genomic DNA, particularly involving certain genes which appear to suppress programmed cell death; these include the *bcl-2* oncogene, over-expression of *c-myc*, *c-ras*, *c-fos* and *c-jun*, ornithine decarboxylase (ODC) genes or reproduction of the wild-type *p53* tumor suppresser gene. DNA fragmentation can be induced in mammalian cells by a wide variety of stimuli, including cytotoxic, carcinogenic and anticarcinogenic agents. Among other factors, it can be overcome by an elevation of intracellular calcium. It can also be overcome by phorbol esters with tumor-promoting activity, such as TPA (12-O-tetradecanoyl-phorbol-13-acetate).

Low concentrations of TPA have been shown to switch the effect of calcium elevation from cell death to cell proliferation, Kizaki et al. (1989), probably by activation of protein kinase C, McConkey et al. (1988). Hence the calcium ion concentrations inside the cell are a central factor in influencing whether the damaged cell dies by Apoptosis or survives with altered



genetic structure which enhances cell proliferation and hence is neoplastic and potentially tumor forming.

One of the strongest and most repeated effects seen in brains, hearts and muscles of exposure to ELF and RF/MW with ELF modulations, is calcium ion efflux, e.g. Blackman et al. (1981), Adey et al. (1982), and Schwartz et al. (1990). Hence the many experiments which have found statistically significant efflux of calcium ions from cells in EMR exposed organs, which couples with the cellular understanding of the role of calcium in influencing the future of a damaged cell, whether it dies or survives to potentially produce a tumor, provides a clear mechanism for the role of low intensity EMR in promoting cell death and cancer. In most organs the desired outcome is enhancing cell death since damaged cell are removed this way. However, in the brain accelerated cell death is associated with neurodegenerative illnesses and premature senility.

Lai and Singh (1997) found increased DNA damage and enhanced cell death in the brains of living rats exposed to low level pulsed microwaves.

Balcer-Kubiczek (1994) states “In 1985 we published the first evidence of EMF carcinogenesis at the cellular level.”, referring to Balcer-Kubiczek and Harrison (1985). They used the standard cell line C3H/10T1/2 samples exposed to 2.45 GHz microwaves for 24 hours, then treated with 0.1 mg/ml of the cancer promoter TPA. One set of samples had no TPA, the remained had the TPA treatment and a range of microwave exposures for SARs of 0.1, 1.0 and 4.4 W/kg, Figure 28.

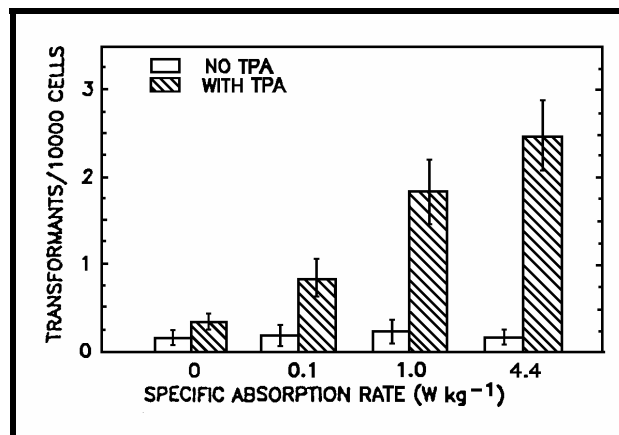


Figure 28: Dose-response relationship for the induction of neoplastic transformation in C3H/10T1/2 cells by a 24 hr exposure to 2.45 GHz microwaves modulated at 120 Hz, with and without post exposure treatment with TPA for 8 weeks, after Balcer-Kubiczek and Harrison (1985).

Balcer-Kubiczek (1994) states that “Our dose response data in terms of SAR for neoplastic effects provides evidence that the effect on tumor induction and development observed in a mouse skin model may operate at the cell level.”

The authors state that these results, and those of Szmigielski et al. (1982), are that **“2.45 GHz microwaves and 60-Hz magnetic fields, seem to act as an initiator or carcinogen, rather than as a promoter of malignant transformation.”**

Since genetic damage can lead to cancer, miscarriage, birth defects and health problem is future generations, evidence of DNA breakage and chromosome aberrations is crucial.

The first identified chromosome aberration study of non-thermal pulsed RF exposure was Heller and Teixeira-Pinto (1959). They concluded that the effects mimicked ionizing radiation and c-mitotic chemicals. More recently it has been shown that extremely significant DNA strand breakage was caused by cell phone radiation exposure to human cells,  $p < 0.0001$ , at a very low SAR level  $0.0024 \text{ W/kg}$ , Phillips et al. (1998). This followed pioneer work on direct DNA strand breakage observations using the Comet Assay by Lai and Singh (1995, 1996, 1997a,b,c).

Sagripanti and Swicord (1986) showed that non-thermal levels of microwave exposure can produce single and double-strand DNA breaks in *E. coli* in solution.

Garaj-Vrhovac et al. (1991) showed that cultured V79 Chinese Hamster fibroblast cell exposed to continuous wave (CW) 7.7 GHz microwaves at power density of  $0.5 \text{ mW/cm}^2$  for 15, 30 and 60 min., kept at a constant temperature, produced a significantly high frequency of specific chromosome aberrations such as dicentric and ring chromosomes in irradiated cells. The dose-response relationships were significant at  $p < 0.01$ . The also observed increased cell Apoptosis.

Garaj-Vrhovac et al. (1992) exposed whole human blood samples to the same exposure regime. With the addition of power densities of 10 and  $30 \text{ mW/cm}^2$ . The number of chromosome aberrations increased from 1.5 % in controls to 2.7 to 7.2 % at the rising power densities. There was a statistically significant dose response with  $p < 0.05$  for total aberrations,  $p < 0.001$  for Acentric and  $p < 0.0001$  for micronuclei.

Nordenson et al. (1994) reported that their recent studies have shown a significant increase in the frequency of chromosomal aberrations in human amniotic cells after exposure to a sinusoidal 50 Hz,  $30 \text{ } \mu\text{T}$  (rms) magnetic field. To evaluate further interactions between chromosomes and electromagnetic fields, they analyzed the effects of intermittent exposure. Amniotic cells were exposed for 72 h to a 50 Hz,  $30 \text{ } \mu\text{T}$  (rms) magnetic field in a 15 s on and 15 s off fashion.

Eight experiments with cells from different fetuses were performed. The results show a 4% mean frequency of aberrations among exposed cells compared to 2% in sham-exposed cells. The difference is statistically significant, with  $P < 0.05$  both excluding and including gaps. In another series of eight experiments, the cells were exposed in the same way but with the field on for 2 s and off for 20 s. Also in these experiments a similar increase in the frequency of chromosomal aberrations was seen, but only when the analysis included gaps. Continuous exposure for 72 h to  $300 \text{ } \mu\text{T}$ , 50 Hz, did not increase the frequency of chromosomal aberrations.

By 2002 I have identified over 20 studies showing that ELF exposures are genotoxic and over 50 studies showing that RF/MW are genotoxic, many at low exposure or isothermal levels showing that the mechanism is not thermal nor electric shocks with high induced currents as assumed originally by western scientists.

### **10.3 In vivo animal experiment results:**

Lai and Singh (1997a) used a highly sensitive microgel electrophoresis, COMET assay technique to identify single strand DNA breaks, Figure 27, and double strand DNA breaks,

Figure 28, from 2hr exposure to 0.1 mT and 0.25 mT 60 Hz magnetic fields in living rat brains.

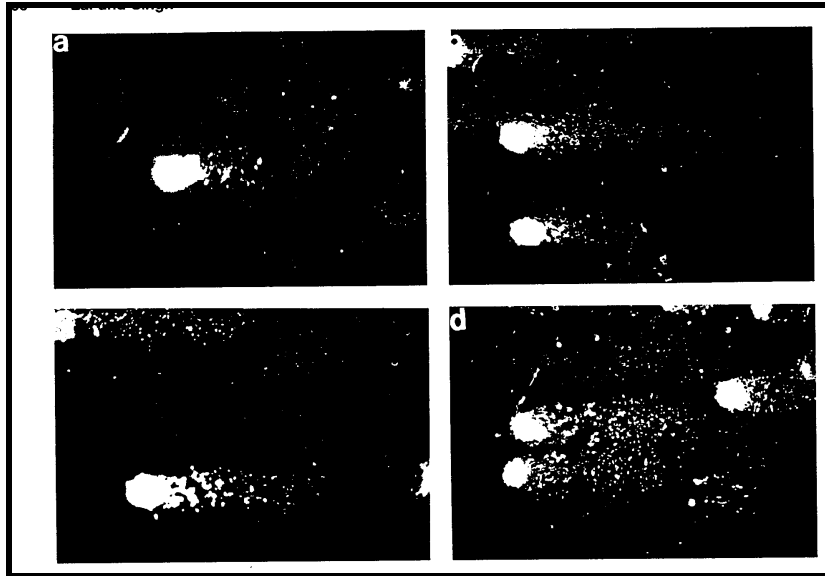


Figure 29: Photographs of single-strand DNA migration pattern of individual brain cells from rats exposed to (a) a bucking condition (0.1mT), magnetic fields of (b) 0.1 mT, (c) a 0.25 mT and (d) 0.5 mT. (x 400).

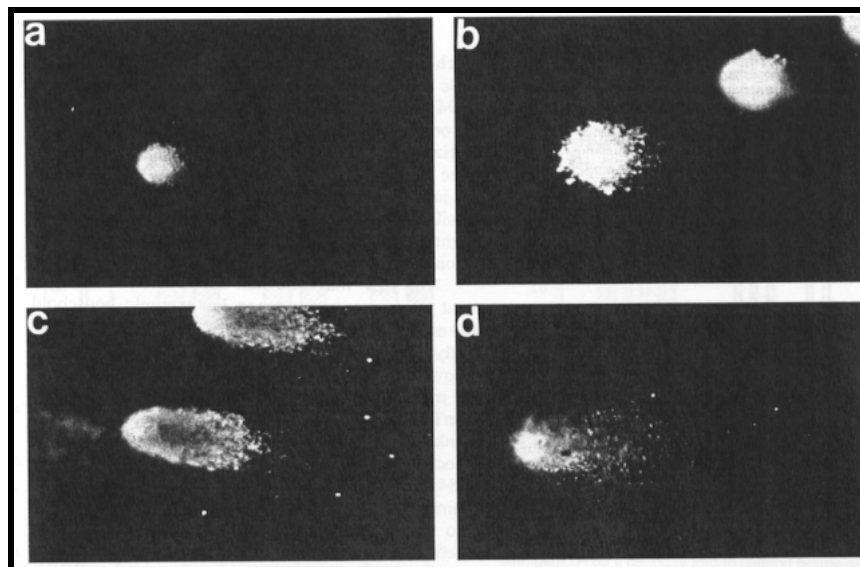


Figure 30: Photographs (expanded x 400) of double-strand DNA migration pattern of individual brain cells from rats exposed to (a) a bucking condition (0.1mT), magnetic fields of (b) 0.1 mT, (c) a 0.25 mT and (d) 0.5 mT.

Lai and Singh (1997a) conclude:

**“Because DNA strand breaks may affect cellular functions, lead to carcinogenicity and cell death, and be related to the onset of degenerative diseases, our data may have important implications for possible health effects of exposure to 60 Hz magnetic fields.”**

Lai and Singh (1997b) investigated the effect of melatonin and a spin trap compound (PBN) both of which scavenge free radicals. They found that rats injected with melatonin or PBN

before ELF field exposure and 2 hours after exposure. Both of these treatments blocked the magnetic field induced DNA single- and double-strand breaks.

Lai and Singh (1997b) conclude:

**“Since melatonin and PBN are efficient free radical scavengers, these data suggest that free radicals may play a role in magnetic field-induced DNA damage.”**

Lai and Singh further state that both melatonin and PBN can have other actions on cells in the brain that can prevent DNA damage therefore further support for their hypothesis can be obtained by studying whether other free radical scavenging compounds also block the effect of magnetic fields.

Timchenko and Ianchevskaia (1995) concluded that an electromagnetic field (EMF) at a frequency of 24 or 14 MHz and intensity of 400 or 200 V/m, increases numbers of epatocytes from rats with chromosomal aberrations 1.4-1.5-fold.

#### **10.4 Human studies of chromosome aberrations with EMR exposure:**

Ciccone et al. (1993) conducted a case control study of 50 acute myeloid leukemias (AML), 17 chronic myeloid leukemias (CML), 19 myelodysplastic syndromes (MDS), and 246 controls. The chromosome aberrations were recorded according to the International System for Human Cytogenetic Nomenclature. Chromosome aberrations were not associated with chemical exposures (OR = 1.0), but a non-statistically significant excess was noted in association with electromagnetic fields (OR = 2.1).

Valjus et al. (1993) sampled for chromosomal aberrations, sister chromatid exchanges (SCEs), replication indices and micronuclei in peripheral blood lymphocytes among 27 nonsmoking power linesmen with considerable long-term exposure to 50-Hz EM fields, and among 27 nonsmoking telephone linesmen serving as a reference group, pairwise matched with the exposed workers for age and geographical region. Blood samples from the two groups were collected, cultured and analyzed in parallel. No differences between the groups were observed on analysis of SCEs, replication indices or micronuclei. However, the mean rate of lymphocytes with chromatid-type breaks was higher among the power linesmen (0.96% gaps excluded, 1.41% gaps included) than among the reference group (0.44% and 0.70%, respectively). The excess of aberrant cells was concentrated among those power linesmen who had worked earlier in their life. Although the interpretation is somewhat complicated by the confounding effect of previous smoking, these results suggest that exposure to 50-Hz EM fields is associated with a slight increase in chromatid breaks.

Skyberg et al. (1993) studied 13 high-voltage laboratory employees and 20 referents participated in a cross-sectional, matched-pairs study of cytogenetic damage. During cable testing the workers were exposed to static, alternating, or pulsed electric and magnetic fields. The alternating magnetic field levels of 50 Hz were 5-10  $\mu$ T, occasionally much higher. Chromosome aberrations, sister chromatid exchanges, and aneuploidy were studied in peripheral blood lymphocytes. Among seven smoking laboratory employees the mean number of chromosome breaks/200 cells was 2.3, as compared with 0.7 for the job-matched referents. The comparable figures for inhibited cultures were 12.0 versus 6.0. No increase was detected in nonsmokers with either method. The results support, to some

extent, the hypothesis of an increased risk of genotoxic effects among high-voltage laboratory workers, particularly a synergistic effect with smoking.

Garson et al. (1991) studied 38 Australia Telecom radio-linesmen who had been exposed to RF EMR in their work and compared the chromosome damage in lymphocytes compared 38 non-exposed clerical staff. A very detailed assay of chromatid and chromosome gaps and breaks and other aberrations was carried out. Most categories showed a small but statistically insignificant increase in chromosome aberrations, with the sum of aberrations of 2.55% for linemen and 2.18% for controls (RR= 1.17, 95%CI: 0.9-1.6).

For Chromatid Gaps RR=1.2 (0.7-2.1); Chromosome Gaps: RR = 1.5 (0.6-3.5); and Chromosome Breaks (without outlier) 1.4 (0.8-2.3). Adjusting for confounding from recent X-rays and for smoking both produced a small increase in Rate Ratio. The absence of adjusting for coffee drinking is a limitation. Such an adjustment would be likely to favour reduction in the incidence among clerical workers, further increasing the Rate Ratio. The incidence of total chromosome aberrations among the controls does appear rather high.

Is chromosome damage in people evidence of an increased risk of cancer. Hagmar et al. (1994) trichotomize CA into the low (1-33%ile), medium (34-66%ile) and high (67-100%ile). The threshold for low CA is typically 1.0% but in the range 0.5 to 1.5 %, while medium is typically 1.0 to 2.0 %, and high >2 %, but may use a threshold between medium and high of 3 %. They concluded that there was a statistically significant linear trend (P=0.0009) in chromosome aberration strata with regards to subsequent cancer risk.

In addition to Hagmar et al. (1994), other studies finding increased subsequent cancer from earlier measured chromosome aberrations include Nordenson, et al. (1984), Nuzzo and Stefanini (1989), Sorsa et al. (1990), Brogger et al. (1990), Sorsa et al. (1992) and Bonassi et al. (1995).

Taking the typical classification the Australia Telecom study as both exposed and control groups in the high category. If the control group was in the “low” category  $\leq 1\%$ , then the Rate Ratio for the clerical staff would be 2.2 and for the linemen 2.6, both of which are significant ( $p < 0.01$ ) and, according to Hagmar et al. (1994), both have increased cancer risk.

There are multiple studies and hence strong evidence that RF/MW exposure increases chromosome aberrations in humans with a subsequent increase in cancer risk.

Garaj-Vrhovac and Fucic (1993) provide another important aspect to this data. Many factors cause chromosome aberration and repairs are being carried out all the time. The cancer risk occurs because not all chromosomes are repaired and mutated cells persist and tumors grow from them. The pulsed microwave exposure of a group of radar station personnel offered the opportunity to study the recovery rate of chromosome aberrations for 30 days after exposure was ceased. Hence this study gives more evidence that RF breaks chromosomes in humans, and shows the rate of recovery through repair. The most affected person is shown in Figure 31.

The recovery rate is very close to linear and the chromosome damage falls to 10% of the original damage is 28.6 weeks, dropping from 33% to 3.5% after 30 weeks, with a tendency for the repair rate to slow suggesting a more exponential recovery rate. For this person the typical daily recovery rate was 0.14% /day.

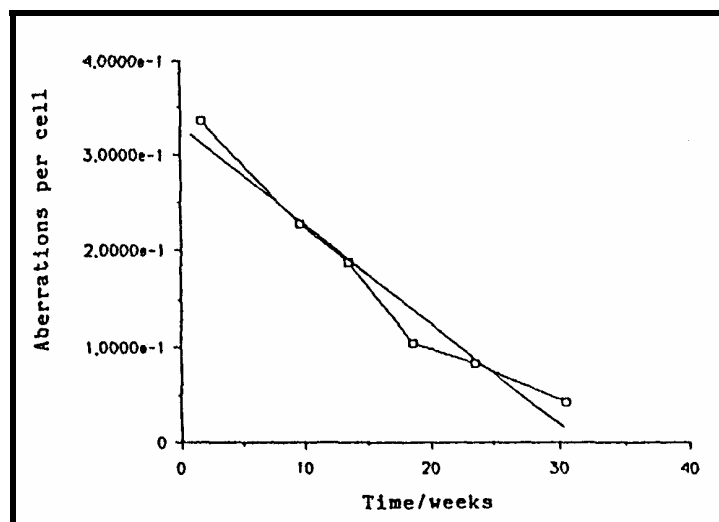


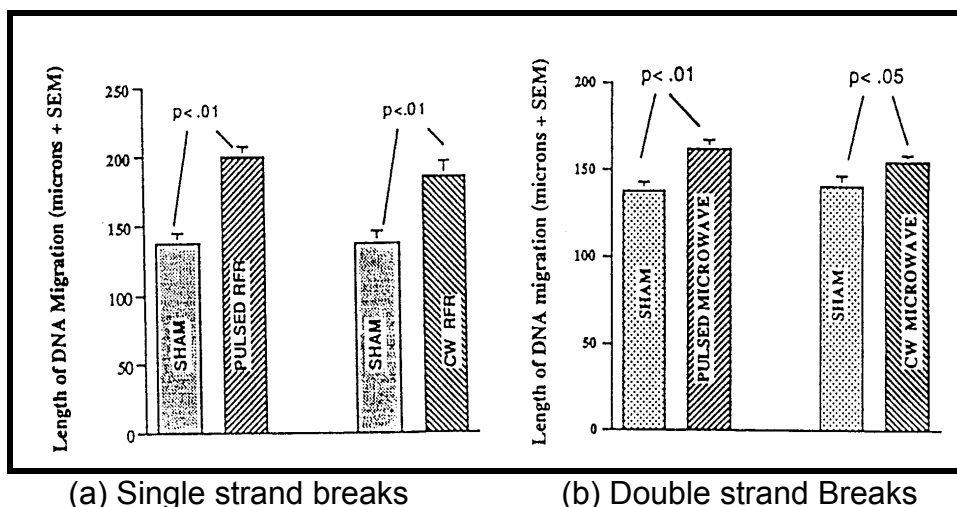
Figure 31: The time-dependent decrease in the number of chromosome aberrations for the subject with extremely high numbers of chromosome impairments,  $y = 0.318 - 0.010x$ ,  $R = 0.98$ .

For two others with lower damaged chromosome rates it was 0.08 and 0.03%/day. Hence daily repair rates are low, suggesting continuing exposure can easily lead to a build up of chromosome damage and an increased risk of cancer.

### 10.5 DNA breakage associated with RF/MW exposure:

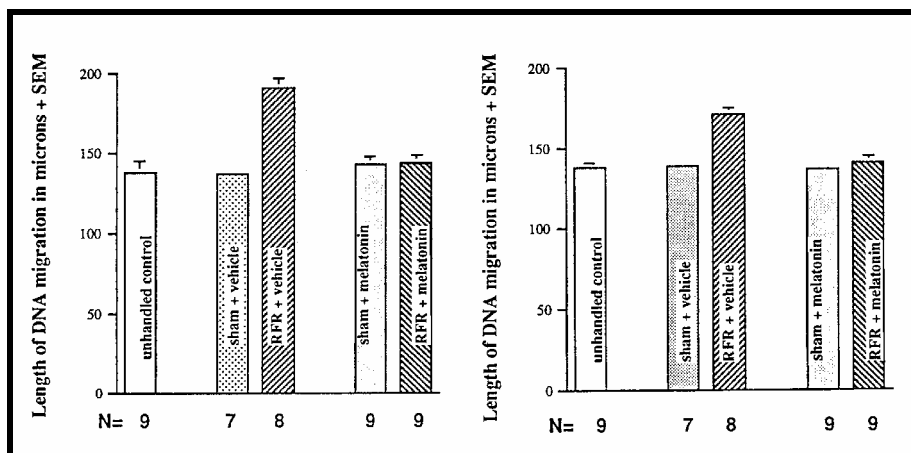
Sarkar et al. (1994) found significant modification of the DNA from mouse cells from brain and testes exposed to  $1 \text{ mW/cm}^2$  2.45 GHz microwaves for 2 hr/day for 120, 150 and 200 days.

Lai and Singh (1995) exposed living rats brains to a single 2 h exposure to microwaves at 2.45 GHz, pulsed at 500 pps, at SARs of 0, 0.6 and  $1.2 \text{ W/kg}$ . They found significant dose-response relationships for single strand DNA breaks in an assay carried out 4 hours after exposure for both the hippocampus and the rest of the brain

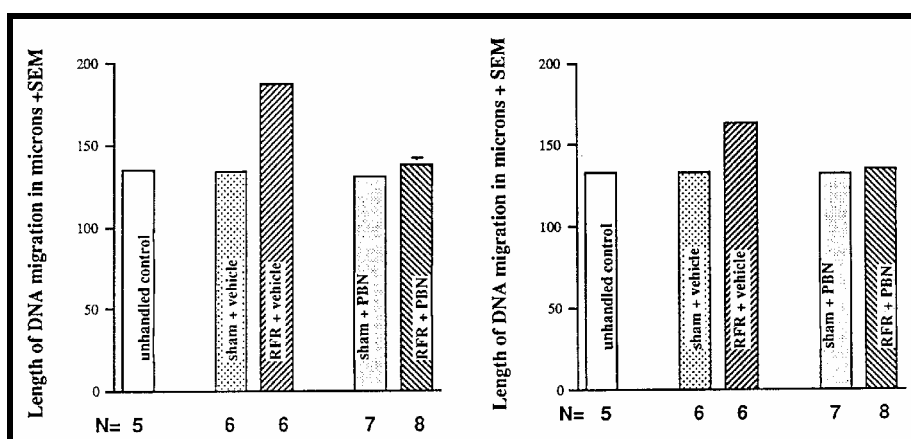


(a) Single strand breaks (b) Double strand Breaks  
Figure 32: DNA breakage in rat brains (SAR =  $1.2 \text{ W/kg}$ ), Lai and Singh (1996).

A second analysis involved assaying the whole brain and continuous wave microwaves at 2.45 GHz and 1.2 W/kg. This showed a statistically significant increase in single-strand DNA breaks between sham and exposed ( $p < 0.01$ ) but no significant difference between assays at 0 h and 4 h after exposure.



(a) Single strand DNA breakage. (b) Double-strand DNA breakage.  
Figure 33: Effect of melatonin on RF/MW induced DNA breakage, Lai and Singh (1997c).



(a) Single strand DNA breakage. (b) Double-strand DNA breakage.  
Figure 34: Effect of PBN (spin-trap compound) on RF/MW induced DNA breakage, Lai and Singh (1997c).

Lai and Singh (1996) repeated the experiment of Lai and Singh (1995) and extended the analysis to include and assay of double-strand DNA breaks and included both pulsed (500 pps) and continuous microwaves at 2.45 GHz. The exposed condition was 2mW/cm<sup>2</sup> (SAR = 1.2 W/kg). Statistically significant single-strand DNA breaks were found for both the CW and pulsed signals ( $p < 0.01$ ), and for double-strand DNA breaks (pulsed  $p < 0.01$  and CW  $p < 0.05$ ). This data was not available for the MacIntyre Case.

Their most recent work, Lai and Singh (1997c), shows that in the exposed rats brains there is enhancement of free radicals and the acceleration of cell death (apoptosis), which is eliminated by melatonin. It is not yet known whether this is caused by the MW radiation influencing the pineal gland or the retina of the eyes, to reduce melatonin production and hence enhance free radical numbers, or whether the MW radiation produces free radicals locally in the brain.

The implications of this study are very important. The authors, Lai and Singh (1997c),

conclude:

**“Data from the present experiment confirm our previous find in a [Lai and Singh, 1995, 1996] that acute RFR exposure causes an increase in DNA single- and double-strand breaks in brain cells of the rat. In addition, we have found that the effect can be blocked by treating the animals with melatonin or PBN. Since a common property of melatonin and spin-trap compounds is that they are efficient free radical scavengers [Carney and Floyd, 1991; Carney et al., 1991; Floyd, 1991; Lafon-Cazal et al., 1993 a,b; Lai et al., 1986; Oliver et al., 1990; Reiter et al., 1995; Sen et al., 1994; Zhao et al., 1994], these data suggest that free radicals may play a role in the RFR-induced DNA single- and double-strand breaks observed in brain cells of the rat. Consistent with this hypothesis is the fact that free radicals can cause damage to DNA and other macromolecules in cells. Particularly, oxygen free radicals have been shown to cause DNA strand breaks [McCord and Fridovich, 1978]. In addition, a study has implicated free radicals as the cause of some of the biological effects observed after exposure to RFR. Phelan et al. [1992] reported that RFR can interact with melanin containing cells and lead to changes in membrane fluidity consistent with a free radical effect.**

**If free radicals are involved in the RFR-induced DNA strand breaks in brain cells, results from the present study could have an important implication on the health effects of RFR exposure. Involvement of free radicals in human diseases, such as cancer and atherosclerosis, have been suggested. Free radicals also play an important role in aging processes [Reiter, 1995]. Aging has been ascribed to accumulated oxidative damage to body tissues [Forster et al., 1996; Sohal and Weindruch, 1996, and involvement of free radicals in neurodegenerative diseases, such as Alzheimer's, Huntington's, and Parkinson's, has also been suggested [Borlongan et al., 1996; Owen et al., 1996]. Furthermore, the effect of free radicals can depend on the nutritional status of an individual, e.g., availability of dietary antioxidants [Aruoma, 1994], consumption of ethanol [Kurose et al., 1996], and dietary restriction [Wachsman, 1996]. Various life conditions, such as psychological stress [Haque et al., 1994] and strenuous physical exercise [Clarkson, 1995], have been shown to increase oxidative stress and enhance the effect of free radicals in the body. Thus, one can speculate that some individuals may be more susceptible to the effects of RFR exposure.**

**However, it must be pointed out that both melatonin and PBN can have other actions on cells in the brain that can decrease DNA damage. Further support for our hypothesis can be obtained by studying whether other compounds with free radical scavenging properties can similarly block the effect of RFR, and by measurement of other free radical-related cellular effects, such as oxidative molecular damages in lipids, protein, and DNA.”**

This is also relevant to the study carried out by Adey et al. (1996) in which rats exposed to cellphone-like signals had 30 % fewer tumours than controls and the tumours were statistically significantly smaller. These results were reported to the 1996 BEMS conference in Victoria BC. Dr Singh raised the question with Dr Adey, of the possibility of cell death as an explanation for the result. Dr Adey agreed that this was possible, but stated that it



needed to be tested. Garaj-Vrhovac et al. (1991) and Lai and Singh (1997c) have found that result.

It has been shown that a sub-thermal dose of microwaves (0.6 W/kg and 1.2 W/kg) can enhance DNA breakage and accelerate the cell death (apoptosis) in living brains, through the increased production of free radicals. This is associated with a reduction in melatonin. With enhanced rate of cell death tumour cells can die at a faster rate than they grow, producing fewer and smaller tumours.

All of these above experiments were carried out without the use of cancer initiators nor co-carcinogens. They involve the direct application of RF/MW radiation to a sample or an animal and the observation of chromosome breakage, DNA breakage, tumours, free radicals and cell death. Hence they confirm the proposal of Reiter (1994) in section 4.1, that EMR would be both an initiator and promoter of cancer, in his case through melatonin reduction, in this case through direct observation of DNA damage which might involve melatonin reduction since free radicals are observed to be enhanced.

## **10.6 Conclusions on Mutagenic effects of EMR:**

DNA damage is shown by Chromosome Aberrations, Micronuclei Formation, DNA strand breakage, enhanced oncogene activity, neoplastic cell transformation. DNA damage has been found under non-thermal exposure to EMR in many independent studies. ELF and ELF modulated RF have been associated with chromosome aberration and micronuclei formation in cells and in exposed workers. Microwaves have been shown to produce DNA damage in living rats brains. Hence EMR is implicated in increasing cancer rates in exposed populations, Hagmar et al. (1994).

Increased cancer incidence can come about by the direct effect of a DNA damaging carcinogen or by the synergistic effect of co-carcinogens. The co-carcinogenic effect and cancer promotional effect of EMR has been widely suggested and demonstrated through a number of experiments, e.g. Adey (1992b), Byus et al. (1988). Direct effects (in the absence of a cancer initiator) include chromosome aberrations and DNA breakage which is most likely to be the result of the enhanced presence of free radicals in the RF/MW field. The role of melatonin is important here. Direct effects are likely to involve higher mean power densities than co-carcinogenic effects. In Lai and Singh (1995) the inter-animal variability is very small giving a small standard deviation for each exposure group. Even so a linear the dose-response relationship is statistically significant for the "rest of the brain" assayed 4 h after exposure ceased. This suggests that the smallest detectable increase in DNA breakage would be associated, with this small sample size, with an SAR of  $<0.2 \text{ W/kg}$ ,  $\sigma = 1.7$ ,  $S < 62 \mu\text{W/cm}^2$ .

Many early studies showing that microwaves caused chromosome aberrations assumed that the mechanism was the heat effects. We now know from many multiple independent published studies that DNA damage occurs at non-thermal and isothermal exposure levels. The lowest reported DNA damage from cellphone radiation ( $p < 0.0001$ ) was provided by Phillips et al. (1998). Two SAR exposures were in the range  $0.0024 \text{ W/kg}$  to  $0.026 \text{ W/kg}$ . These are equivalent to  $1.2$  to  $13 \mu\text{W/cm}^2$  for microwaves at  $813$  to  $837 \text{ MHz}$  and human brain tissues. This study also showed significant ( $p < 0.0001$ ) DNA repair rates in these human brain cells. We now know that induce DNA repairs is caused by DNA damage and brain cancer cells have very strong repair mechanisms because they cannot be

regenerated and it's a vital organ. The time exposure reaction of brain cells to cellphone radiation caused DNA damage is shown by the data in Figure 2 of Malyapa et al. (1997).

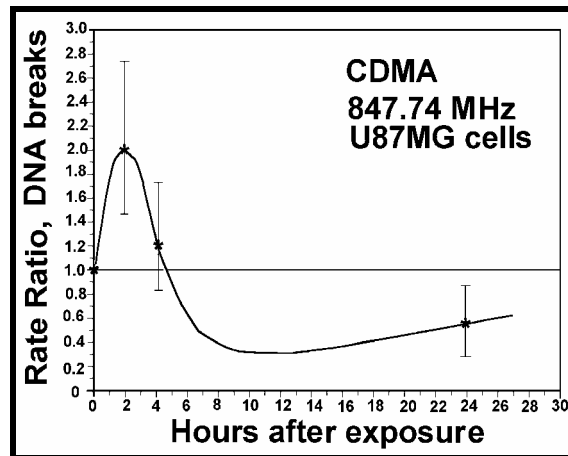


Figure 34a: DNA strand breakage Risk Ratio and 95% CI for the frequency distribution in Figure 2 Normalized Comet Moment of Malyapa et al. (1997).

Figure 34a clarifies the understating of the relationship of microwaves induced DNA damage with DNA repair.

## 11. Long-term Animal Studies:

A small number of long-term animal studies involving RF/MW exposure have been carried out, largely because of their extreme difficulty and very high cost. Balode (1996)

### 11.1 University of California, Berkeley:

Professor Charles Susskind and Dr Susan Prausnitz, Dept of Electrical Engineering, UC Berkeley carried out the first reported long term study for the US Air Force, Prausnitz and Susskind (1962). They exposed male Swiss albino mice to 9.27 GHz microwaves, pulsed with a 2  $\mu$ s pulse at 500 Hz, 4.5 minutes per day, 5 days per week for 59 weeks with an exposure level of 0.10W/cm<sup>2</sup>. This amounts to a mean weekly exposure of 223 $\mu$ W/cm<sup>2</sup>.

Detailed autopsies were carried out on 60 irradiated and 40 control mice who died during the experiment. Two adverse effects were more severe in the exposed compared to the control animals.

- (1) Testicular degeneration (sterility, atrophy with no sperm) occurred in 40 % (23/57) of the exposed animals and 8.1 % (3/37) of the control animals.
- (2) Cancer of the white cells or leukemia was seen in 35 % (21/60) of the exposed animals compared to 10 % (4/40) of the controls. This condition was described as monocytic or lymphatic organ tumours or myeloid leukaemia in the circulating blood.

At the 16-month interim kill, one month after exposure ceased, 30 % (6/20) if the exposed group had leukemia compared to 10 % (1/10) of the controls.

At the final kill at 19-months, 4 months after cessation of exposure testicular atrophy was seen in 21% (14/67) of the exposed group and 5 % (1/19) of the control group, and

testicular weights were lower for the exposed group. At this stage leukosis was the same in both groups at 18 % (12/67) for the exposed group and 21 % (4/19) for the control group.

This gives an overall rate for testicular degeneration of 29.8% (39/124) for the exposed group and 7.1% (4/56) for the control group, giving a Rate Ratio of  $RR=4.2$ . For leukosis the incidence was 26.5 % (39/147) for the exposed mice and 13.0% (9/69) for the control mice,  $RR = 2.04$ .

Cairnie et al. (1980) exposed mice to microwaves at power density of  $50 \text{ mW/cm}^2$ . They found that the absorbance in the abdomen area of the liver was 11 times greater than the testes, and while the abdomen temperature was increased the testicular temperature was not. This suggest that the  $100\text{mW/cm}^2$  exposure testicular degeneration could have been the genotoxic effect of microwaves rather than the temperature rise.

Leukosis (the initiation of leukaemia) requires damaged DNA and chromosome aberrations which are transferred from cell to cell through mutation. The same mechanism could cause testicular degeneration. An accumulated cellular level damage mechanism is not necessarily related to the intensity but can relate to total dose in relation to rates of repair. Hence the averaging of weekly exposure is a meaningful adverse effect related level. Actual public exposure levels of  $0.2\mu\text{W/cm}^2$  and less saw childhood leukaemia incidence and death rate rises at similar exposure levels (2.74 for mortality) in the North Sydney Study.

## 11.2 University of Washington Case Study:

Establishment of a potential adverse human health effect can be obtained from a suitably designed and executed animal experiment. Such an experiment was carried out at the University of Washington by Professor Arthur Guy and his associates, funded by the United State Air Force, Chou et al. (1992).

**Table 4: Crude incidence of neoplastic lesions (Tumours)**

Site/Type	Crude Tumor Incidence			
	Control		Exposed	
Adrenal Cortex	12/85	14.1 %	12/76	15.8 %
Adrenal medulla	1/73	1.4 %	7/67	10.4 %
Thyroid	9/85	10.6 %	12/76	15.8 %
Liver	1/85	1.2 %	3/76	3.9%
Pituitary	21/85	24.7 %	19/75	25.3 %
Testes	0/85	0 %	2/76	2.6 %
Epididymis	0/85	0 %	1/76	1.3 %
Pancreas	2/85	2.4 %	2/76	2.6 %
Urinary bladder	0/85	0 %	2/76	2.6 %
Stomach	4/85	4.7 %	4/76	5.3 %
Duodenum	0/85	0 %	1/76	1.3 %
Lymph node	0/85	0%	1/76	1.3 %
Soft Tissues, Thorax	0/85	0 %	2/76	2.6 %
Mesentery	0/85	0 %	2/76	2.6 %
Lymphosarcoma	3/85	3.5 %	4/76	5.3 %
Total	53/85	62.4 %	63/75	84.0%
(RR=1.35, 95%CI: 1.11-1.63,p=0.0022)				

The exposed a large group of rats to pulsed radar-like microwaves, 2,450 MHz, pulsed at 800 pps, 10  $\mu$ s pulse, at  $<0.4$  W/kg, the human exposure level allowable under the ANSI standard. These rats were compared to a similar group who were sham exposed. Guy found a total of 18 malignancies in the 100 exposed rats compared to 5 in the 100 sham exposed rats, a ratio of  $RR=3.6$  (1.34-9.70), in particular there were 9 endocrine tumours in the exposed group compared to 2 (ratio  $RR=4.5$  (1.0-20.8)) in the control group. On the other hand, the EPA review team worked with the original University of Washington research team, and undertook further detailed statistical analysis of their results and showed “a statistically significant elevation in the incidence of carcinomas at all sites combined.”

The experiment ran for 25 months with some mice being sacrificed and analyzed at 13 months. Their initial reports concluded no effects except a significant increase in the number of benign adrenal tumours. At 13 months the exposed group had a significantly larger number of B- and T-cells than do controls, but no difference was seen at the end of 25 months. This suggests the immune system was initially disrupted, but over a 2 year period it adapted to the exposure situation. Disturbance of the immune system is also consistent with the developing cancer and tumours growth.

These results were worrying to EPA researchers. Dr Robert McGaughy asked Dr Lawrence Kunz, the pathologist on the University of Washington study, for copies of the survival and histopathologic findings. Dr McGaughy was able to show that three statistical tests showed a statistically significant increase in carcinomas ( $P<0.05$ ) but no statistically significant increase in sarcomas. These results are listed in Table 13.

The EPA team argue that while most chemical carcinogens affect only one or a few tissues, the distribution of the EM field as a “toxic agent” is more uniform than a “typical” chemical agent, and therefore an “all sites” approach is justified..

McGaughy et al. (1990) point to the more ubiquitous action of melatonin as an example, since,

**“Nocturnal pineal melatonin activity is known to be inhibited by ELF electric fields (Wilson et al 1986) and that the pineal gland function is closely coupled to the function of other glands. Melatonin is known to inhibit tumour growth-enhancing hormones like prolactin and estrogen. The postulate has been made that when the blood melatonin concentration decreases because of the action of EM fields on the pineal gland, a tumour growth inhibitor has been reduce or effectively removed, thereby causing a stimulation of tumour growth.**

**Although only breast and prostate tumours have been discussed in this connection, the same regulation by melatonin might hold for other hormonally-regulated endocrine organs as well.”**

<p><b>The Guy et al. (1985) study, along with other supporting material, led to the recommendation that the US EPA classify RF/MW as a possible human carcinogen (Class C).</b></p>
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The data presented in this report indicate the progressively strengthening evidence of carcinogenicity and other adverse health effects from chronic non-thermal exposure to RF/MW radiation which raise the evidence to classify RF/MW radiation as a highly probable (Class B1) carcinogen.

Note: All you need in New Zealand Law is evidence of a potential irreversible adverse environmental effect to decline this application and to recommend the identification of a site in a less sensitive receiving environment, or a potential adverse effect to require mitigation or remediation.

### 11.3 Polish Study:

Szmigielski et al. (1982) measured the effects of 2.45 GHz microwave radiation at 5, 10 and/or 15 mW/cm<sup>2</sup>, 2h /day, 6 times/week exposure (average weekly exposure 360, 520 and 1,100  $\mu$ W/cm<sup>2</sup>), mice able to maintain core temperature under both exposures, specifically investigating lung cancer, breast cancer and skin cancer. Figure 32 shows the result of initiating skin tumours using 3,4 benzo-alpha-pyrene (BP) and assessing the cancer promoting effect of microwaves.

Cancer development started 2 months earlier for the MW exposed mice and reached the 50 % point for the population after 137 days compared to 305 days. Hence MW significantly accelerated the growth and proliferation of skin cancer tumours.

Figure 33 shows the results of planting lung cancer (sarcoma) cells and then exposing the mice to 5 and 15 mW/cm<sup>2</sup> MW radiation. The 5 mW/cm<sup>2</sup> exposure produced an enhancement of lung cancer modules at 2.5 times more than controls after 3 months, but at a similar level to the effect of an over-crowding stress factor. The 15 mW/cm<sup>2</sup> exposure produced about 5.5 times more lung cancer nodules.

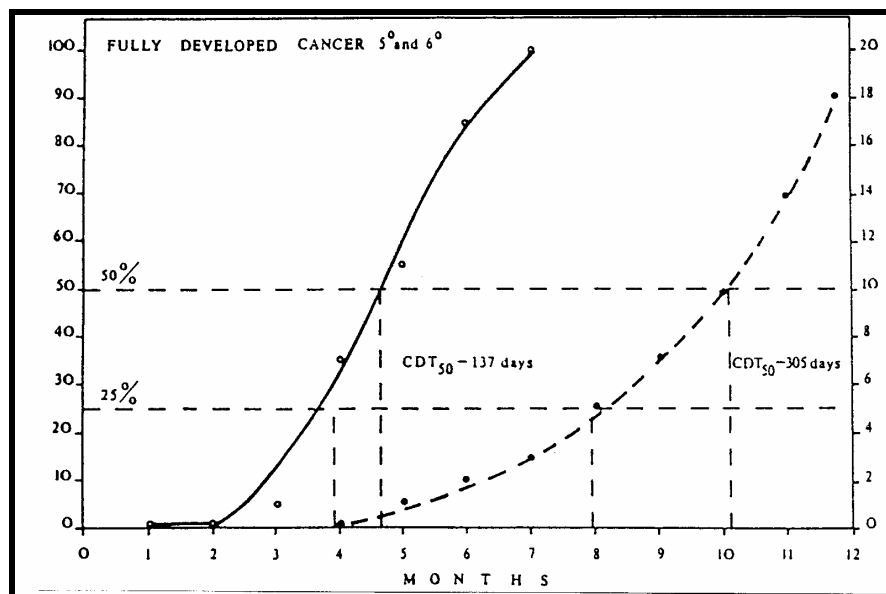


Figure 35: Growth curves of BP-induced skin tumour in mice exposed daily to 10 mW/cm<sup>2</sup> of 2.45 GHz microwave radiation for the whole period of tumour growth. CDT<sub>50</sub> refers to the cancer development time when 50 % of the animals have tumours.

A parallel experiment for breast cancer for control, overcrowding stress, 5 and 15 mW/cm<sup>2</sup> MW exposure, the 50 % development points were 322, 255, 261 and 219 days, respectively. These show a similar relationship to the results in Figure 26 for lung cancer, except that the stress and 5 mW/cm<sup>2</sup> effects are reversed.

These results show statistically significant increases in numbers and rates of development of chemically initiated skin, lung and breast tumours when exposed to low level microwaves, with a significant dose response relationship in each case.

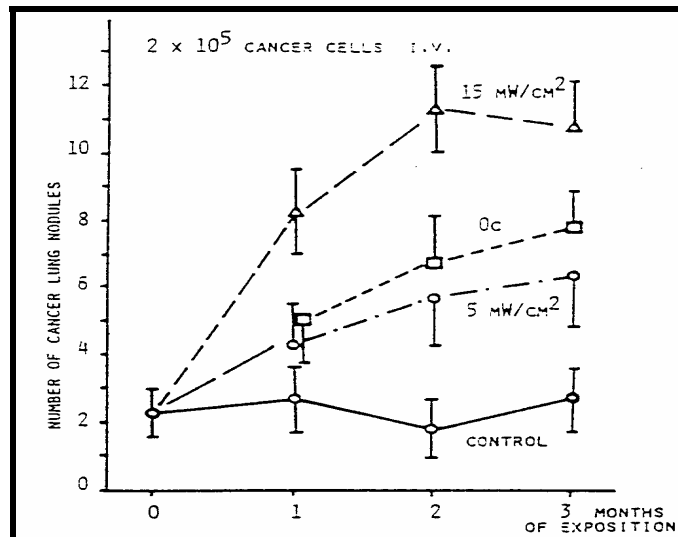


Figure 36: The number of lung tumours (following intravenous injection of  $2 \times 10^5$  viable sarcoma cells) in mice exposed during 1, 2 and 3 months to 2.45 GHz microwaves (2h daily) at 5 or 15 mW/cm<sup>2</sup>. Oc refers to mice treated with nonspecific stress of over crowding.

#### 11.4 Duke University Medical Center:

Eight week old female mice were exposed to 2.45 GHz microwaves at power densities of 5 to 15 mW/cm<sup>2</sup> for 30 min./day over periods between 1 and 17 days, Huang and Mold (1980). Daily mean exposures were about 100 to 300  $\mu$ W/cm<sup>2</sup>, and exposure conditions were essentially isothermal. The results showed, (a) A sustained activation of tissue macrophages resulting in suppression of lymphocyte responsiveness, and (b) a gradual but temporary stimulation directed to the lymphocytes.

Macrophage activation may have caused the early depression of lymphocyte responsiveness. The suppression is later overridden by the cumulative direct stimulation of lymphocytes by microwaves. Prolonged exposures is suggested to eventually result in depressed function in much the same as seen in rheumatoid arthritis which occurs from chronic immune stimulation.

They also conclude that 2.45 GHz microwaves affect the hematopoietic colony-forming abilities through altering the growth of both erythroid and myeloid cells. This is direct evidence of the ability of sub-thermal microwaves to cause chronic immuno-suppression.

### 11.5 Jawaharlal Nehru University Study:

Ray and Behari (1990) exposed young albino rats of both sexes to 7.5 GHz microwaves, pulsed at 1000 kHz and at a power of  $600 \mu\text{W}/\text{cm}^2$ , for 3 hr /day, averaging  $75 \mu\text{W}/\text{cm}^2$ .

Microwave exposed rats ate and drank less and thus showed smaller weight gain. Leukocyte count increased by 35 % in the exposed animals along with a 2-fold increase in eosinophils, and Spleen, Kidney, Brain and Ovaries were significantly smaller.

### 11.6 Royal Adelaide Hospital Project:

Repacholi et al. (1997) exposed genetically engineered mice to a cell phone signal for 1 hr/day. This was an Australian industry funded study to allay public fears of cell phone health effects was carried out by a team led by Dr Michael Repacholi at the Royal Adelaide Hospital. In an ABC Four Corners documentary Dr Repacholi describes this study:

**“We tried to get the most sensitive model of mouse that we could find that would get lymphoma and then see if we exposed them to radio frequency field, whether we could promote that cancer above its normal incidence.”**

Mice are often used to test toxins, chemicals and radiation effects because of the strong similarity of their cells to human cells. A search of Medline shows that since 1993 over 21100 cancer studies have used mice and 621 used tumorigenic mice.

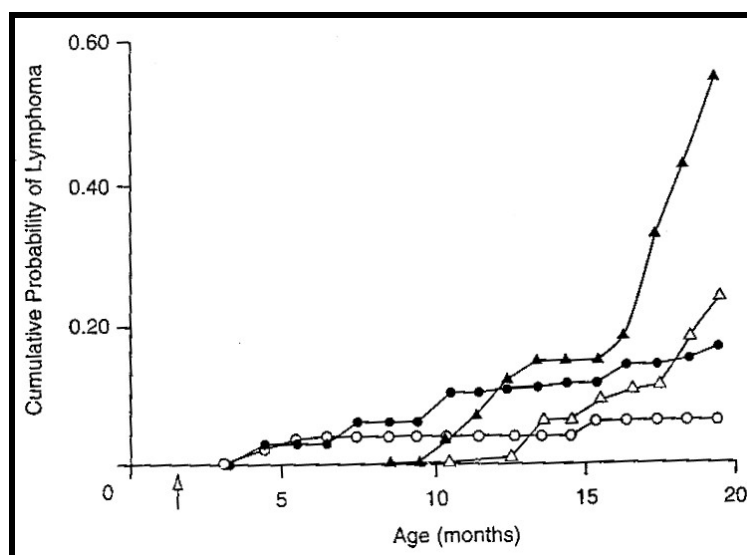


Figure 37: Rate of lymphomas increase in control and exposed groups of mice, Repacholi et al. (1997). The vertical arrow shows when the exposure began.

Their 200 genetically engineered mice normally had 22 % of them to get lymphomas in their immune system, including B-cells. About half of the mice were exposed to a moderate level of cell phone radiation for 1 hour per day for 18 months. The other half were treated the same way but not exposed. At the end of the study 43 % of the exposed mice had lymphomas. The overall Odds ratio was 2.4,  $p=0.006$ , 95% CI=1.3-4.5. This is a highly significant results in which the cell phone radiation more than doubled the cancer rate from a 1 hour per day exposure. Mean exposure range was measured as 0.13 to 1.4 W/kg.

Hence the mean daily exposure was 0.005 to 0.058 W/kg, averaging 0.03 W/kg. This is somewhat below the ICNIRP Guideline of 0.08 W/kg and clearly non-thermal.

### 11.7 Greek Mouse Fertility Study (1997)

Because of health concerns among residents living in the vicinity of an RF transmission tower in Greece, groups of mice were placed at various locations in relation to the tower, Magras and Xenos (1997). The mice fertility over several generations was monitored and related to the RF exposure. Figure 38 shows the fertility rate of the two exposed groups. Where group A the “Low” exposure group ( $0.168 \mu\text{W}/\text{cm}^2$ ) became infertile after 5 generations and B the “High” exposure group  $1.053 \mu\text{W}/\text{cm}^2$ , became infertile after only 3 generations. This is a highly significant result because so few multi-generation studies have been done and the effects of this study occur at extremely low levels and the effect is total infertility.

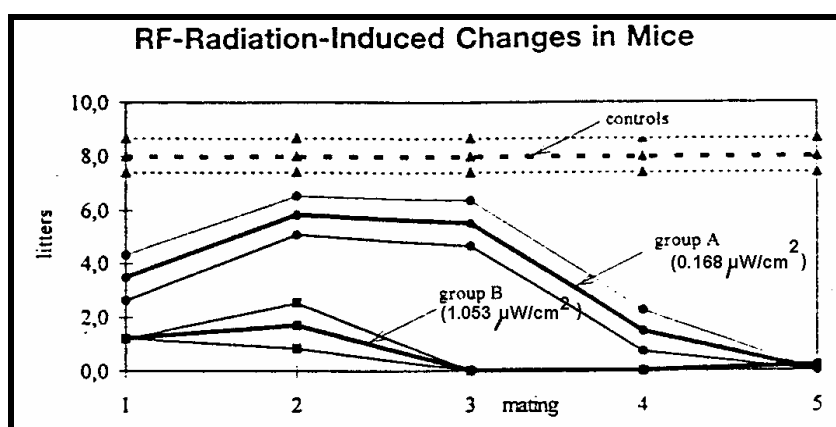


Figure 38: Reproductive rates in two groups of mice exposed to extremely low intensity radio signals, showing a dose response in the time taken to achieve full infertility of 3 matings for  $1.053 \mu\text{W}/\text{cm}^2$  and 5 matings for  $0.168 \mu\text{W}/\text{cm}^2$ .

### 11.8 University of Texas mice study

Vijayalaxmi et al. (1997) was designed to test whether microwaves were genotoxic by assaying the presence of micronuclei in peripheral blood and bone marrow of cancer-prone mice. Their threshold test was if there was a significant increase of micronuclei then microwaves were genotoxic. Their initial results had major errors and when they were corrected they did find significant increases of micronuclei in both blood ( $p < 0.02$ ) and bone marrow ( $p < 0.025$ ). In addition, 12 exposed mice had tumors compared to 8 mice in the control group, OR = 1.5. Therefore their results show that microwaves are genotoxic because they significantly enhance micronuclei formation and increase the tumour rate.

### 11.9 Summary and Conclusions about long-term animal experiments:

Animal experiments confirm that in mice pulsed RF/MW radiation is able to initiate statistically significantly more malignant tumours in many body organs at exposure levels assumed to be non-thermal and safe ( $0.4 \text{ W/kg}$ ), McGaughy (1990), and in the presence of a chemical cancer initiator to drastically increase the rate of development of lung, breast and skin cancer, Szmigielski et al. (1982), showing the strong co-promotional effects of microwave exposure. Prausnitz and Susskind (1962) found increased in testicular degeneration and increases in leukaemia at Rate Ratios from, chronic exposure to a short daily acute exposure to microwaves. Chou et al. (1992) found that radar microwaves more



than tripled the overall tumour rate in rats. Repacholi et al. (1997) shows that cellphone radiation enhances B-cell tumours in genetically engineered mice. Vijayalaxmi et al. (1997) show a similar result but also found significant increases in micronuclei formation in the microwave exposed mice. These are consistent with the research summarized above on the direct mutagenic affects of RF/MW radiation and the research showing alteration of signal transduction, cell communication which influence the cellular level growth regulation and can lead to cell proliferation and thence to tumour formation and cancer.

Magras and Xenos (1997) showed that mice became totally infertile with exposure to extremely low levels of radiofrequency radiation. This shows the strong value of multi-generational studies which are almost entirely lacking and almost impossible for epidemiology to carry out in relationship to human exposure to RF/MW. Human generations are over 2 decades long and hence a 5 generational study requires over 100 years of known exposure to RF/MW which is impossible at this time because the technology is too new.

With the very close parallel effects of mouse and rat studies and human studies, the results of chronic and multigenerational rodent studies must serve as a direct most probable indicator of effects on people. For example, Vijayalamxmi et al. (1997) show significant increases in chromosome breakage in the blood and bone marrow of cancer-prone mice chronically exposed to 2.45 GHz RF radiation, along with a 50 % increase in mammary tumors in the exposed vs sham exposed mice. Goldsmith (1995) reports enhanced chromosome damage and increased incidence of tumors in the Civil Service population of the U.S. Embassy in Moscow after they had been chronically exposed to a radar signal for varying but very low mean exposures less than  $0.1\mu\text{W}/\text{cm}^2$  (the mean range 1 to  $2.4\mu\text{W}/\text{cm}^2$  as measured on the outside of the walls).

Non-thermal microwaves also caused significant impairment of the immune system functioning. This was recently found in people in association with powerlines (Beale et al (1997)), and recall that powerlines emit RF radiation as well as ELF fields.

#### **11.10 Plant effects:**

The first published chromosome aberration study was for pulsed radiofrequency radiation and showed that in an isothermal situation the genotoxic effect of radiofrequency radiation mimicked ionizing radiation and C-mitotic chemicals. The subject of the exposure was garlic roots. Four of the Latvian studies published in 1996 refer to EMR effects on plants which were exposed to a radar RF pulsed signal. Balodis et al. (1996) measured a significant reduction ( $p<0.01$ ) in relative additional increment tree ring growth over 20 years of exposure. Figure 35 shows the incremental tree ring growth at 4 km from the radar. Figure 36 shows the measured exposures with distance giving a mean of about  $0.1\text{ V/m}$  which corresponds to  $0.003\mu\text{W}/\text{cm}^2$ .

Selga and Selga (1996) found significant cell structure changes in this exposure, including alterations of the Golgi apparatus switched to functions from synthesis of predecessors of cell walls to formation and export of resin predecessors an other cell stressors. These lead to accelerated resin production and promoted senescence of the pine trees.

Magone (1996) studied fast growing plants, *Spirodela polyrhiza* (L.) Schleiden. Generally, the vegetative reproduction rate was accelerated in the first 20 days after the end of exposure. Exposure of plants beginning formation lowered the vegetative growth rate.

Eighty-eight-hour exposure caused the appearance of some abnormal individuals after 30 days of growth. At 55 days growth, various morphological and developmental abnormalities appeared in 6-10 daughter plants from 10 exposed mother plants, compared to 0.1 plants per 10 in the control plants. Plants developed completely to daughter fronds under exposure from the electromagnetic field had a shorter life-span (67 days compared to 87 days in the control) and fewer daughters (8 compared to 10 in the controls).

Schmutz et al. (1996) studies young spruce and beech trees exposed to 2450 MHz for 3.5 years at a range of exposures from  $0.7 \mu\text{W}/\text{cm}^2$  to  $300 \text{ W}/\text{m}^2$ , depending on the location. A negative relationship existed for foliar concentrations of the calcium and sulfur in the beech and the exposure during the first 2 years.

Hence the Skrunda studies on plants show similar effects to those on human beings including stress symptoms, changes in calcium, growth reduction and reproductive changes consistent with chromosome damage. Adverse biological effects are found with exposures down to  $0.003 \mu\text{W}/\text{cm}^2$ .

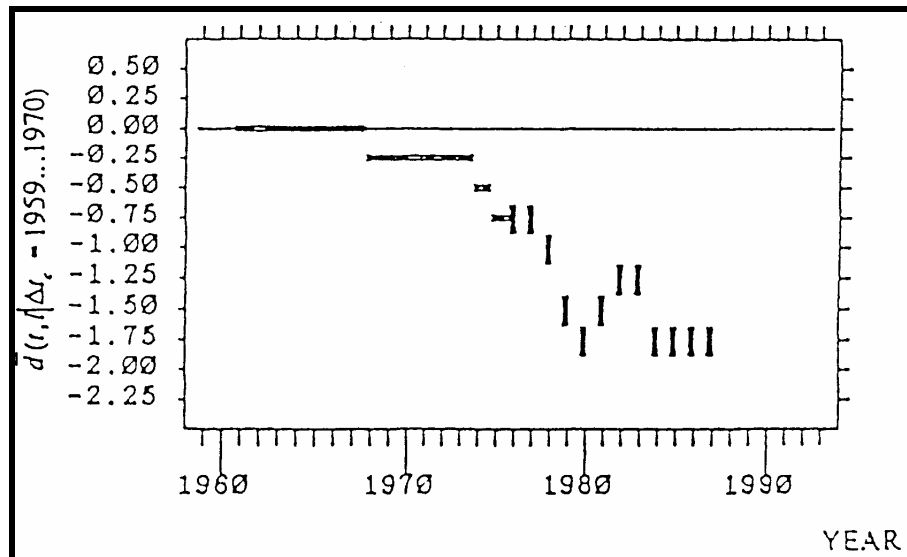


Figure 39: Reductions in annual incremental tree rings growth with  $0.003 \mu\text{W}/\text{cm}^2$  exposure to a pulsed radar signal. Balodis et al. (1996).

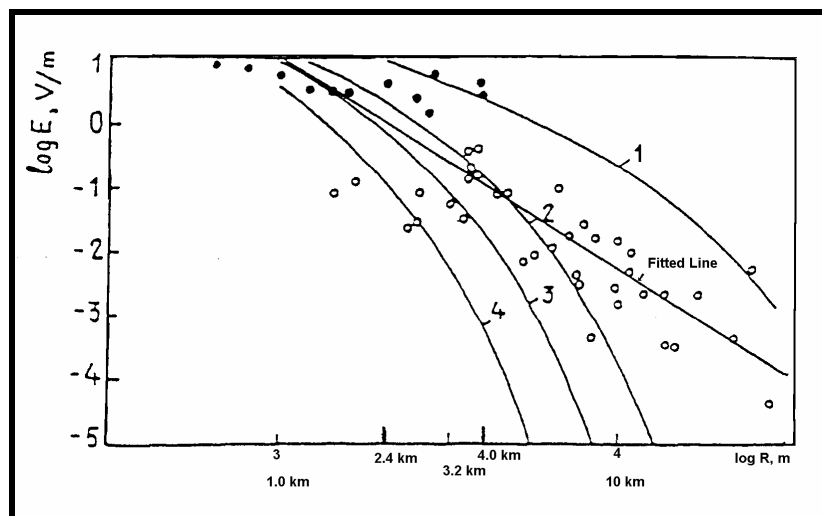


Figure 40: Measured exposure with distance from the Skrunda Radar.

## 12 Human health studies:

### 12.1 Reproductive

#### 12.1.1 Summary of Epidemiological Studies:

During the 1980's and early 90's there were three studies of the reproductive outcomes of physiotherapists (Physical Therapists) who were occupationally exposed to short-wave and infrequently to microwave, radiation during diathermy.

Kallen et al. (1982), in Sweden, used a survey of therapists recalling pregnancy outcomes and work place equipment use during pregnancy, involving 33 cases. None who reported pregnancy outcomes reported exposure to microwaves. They found increased congenital malformations, low birth weight, still birth and death within the first 7 days after birth in association with short-wave diathermy.

Taskinen et al. (1990) in Finland, with 204 cases, found increased spontaneous abortion with short-wave and microwave use (electric therapies  $\geq 5$ /week OR=2.0, CI: 1.0-3.9, n=17 ; shortwaves  $\geq 5$ h/week, OR= 1.6, CI: 0.9-2.7, n= 30; Microwaves, OR= 1.8, CI: 0.8-4.1, n=13), but stronger associations with ultrasound and heavy lifting (Ultrasound  $\geq 20$ /week, OR= 3.4, CI: 1.2-9.0, n=9 . Heavy lifting,  $> 10$  kg or patient transfers  $\geq 50$  times/week, OR=3.5, IC: 1.1-9.0, n=11). Odds ratios increased for pregnancies  $> 10$  weeks: electric therapies OR=2.2, shortwaves 2.5, Microwaves 2.4, ultrasound 3.4 and heavy lifting 6.7 .

They conclude "Physical exertion during early pregnancy seems to be a risk factor for spontaneous abortion. The findings raise suspicion of potential harmful effect of shortwaves and ultrasound on the pregnancy, but no firm conclusion can be drawn on the bases of these results alone." However, this study adds weight when grouped with other studies.

Larsen et al. (1991), studying pregnant physiotherapist's, identified 54 cases and 146 spontaneous abortion cases from Denmark, found a significant increase in malformations, still birth, low birth weight, cot death and prematurely when working with short-wave diathermy.

Taken together, the three Scandinavian studies show adverse changes in pregnancy outcomes associated with short-wave RF, including spontaneous abortion later in the pregnancy, fetal malformations, still birth, low birth weight, cot death and prematurely.

The only study to find effects with microwaves was Taskinen et al. (1990), and then an association with later pregnancy spontaneous abortion.

A further Finnish study on spontaneous abortion in association with the use of VDUs, Lindbohm et al. (1992), found a non-statistically significant elevated risk miscarriage of 10% with all VDU usage, but for those exposed to  $\geq 0.9 \mu\text{T}$ , the odds ratio was 3.4 (95% CI: 1.4-8.6) when compared to rates in workers exposed to  $< 0.4 \mu\text{T}$ . They investigated confounders such as ergonomic factors and mental workload but these change the odds ratio very little. An exposure assessment found a significant dose-response relationship.

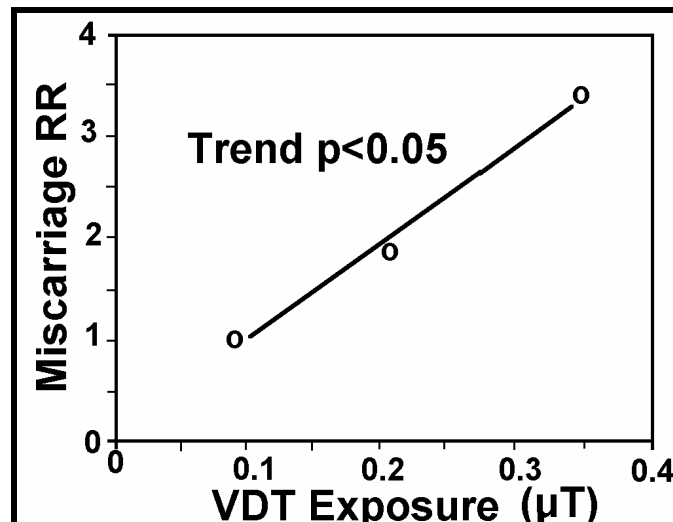


Figure 41: ELF/RF/MW exposure from VDT usage increases miscarriage in a dose-response manner, Lindbohm et al. (1992).

American studies include Vaughan et al. (1984) who found significantly increased risk of fetal death for last pregnancy for therapists,  $RR=2.0$ ,  $CI: 1.5-2.5$ ,  $n=169$ , and for electronic technicians,  $RR= 1.5$ ,  $CI:1.2-2.0$ ,  $n=202$ .

The most significant study was carried out by Ouellet-Hellstrom and Stewart (1993) who investigated early spontaneous miscarriage among U.S. Physical Therapists who had used short-wave (27 MHz) or microwave (915 MHz and 2.45 GHz) for diathermy. The sample involved 1753 case pregnancies (miscarriages) and 1753 matched controls. Exposed cases were women who had been exposed to EMR in the 6 months prior to the first trimester or during the first trimester of the pregnancy. They found statistically significantly increased early spontaneous abortions in the first trimester for those using microwave diathermy,  $OR= 1.28$ ,  $CI: 1.02-1.59$ , the odds ratio increasing with exposure (trend  $p < 0.005$ ) giving a statistically significant dose-response relationship, the odds ratio in the highest exposure group being  $OR= 1.59$ ,  $CI: 0.99-2.55$ .

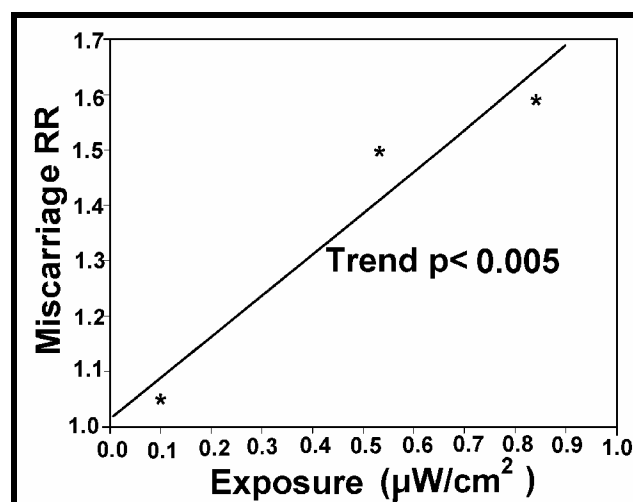


Figure 41a: Microwave exposure associated miscarriage for pregnant physiotherapists, Ouellet-Hellstrom and Stewart (1993).

For those for whom this was the first miscarriage, the association with microwave exposure gave  $OR= 1.26$ ,  $CI: 1.00- 1.59$ , the dose response trend had  $p<0.01$  and the highest

exposure group had  $OR = 1.55$ ,  $CI: 0.92-2.61$ . Short-wave exposure showed some positive odds ratios but none were statistically significant.

This study shows a very strong relationship between microwave exposure and early miscarriage, with a dose-response relationship, backed by several other studies, and evidence of melatonin reduction and chromosome aberrations, showing a causal relationship between RF/microwave exposures and miscarriage and congenital malformation.

Taken with the Scandinavian studies, the pattern emerges of late pregnancy miscarriage and fetal damage in association with short-wave exposure and early miscarriage in associate with microwave exposure.

A subsequently exchange of correspondence related to this study challenges those who dismiss an athermal mechanism, such as chromosome aberration or melatonin reduction which are known miscarriage risk factors, Sandyk et al. (1992). Hocking and Joyner (1995) show that microwaves produce very small SARs with the uterus, Figure 42.

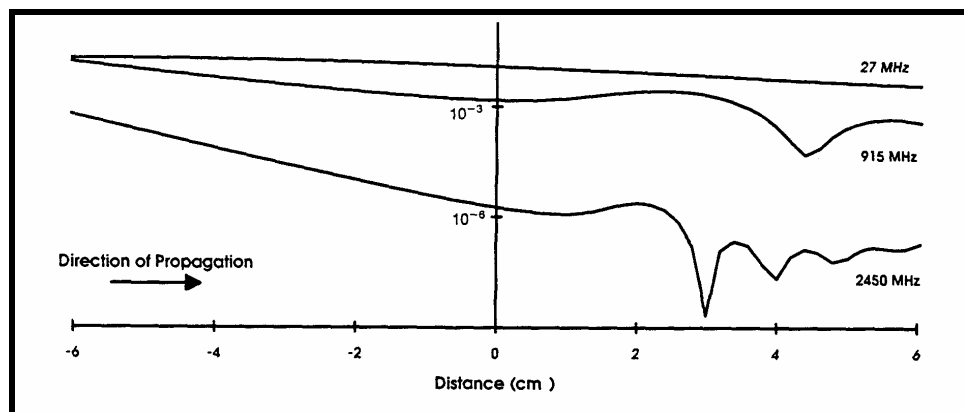


Figure 42: Specific absorption rate (SAR) profile across the uterus for a small woman exposed to  $1 \text{ mW/cm}^2$ , from Hocking and Joyner (1995).

Their table 2 shows maximum SARs in the uterus for the conditions in Figure 38 for short-wave (27.12 MHz) of  $0.209 \text{ W/kg}$ , for microwave (915MHz) of  $0.023 \text{ W/kg}$  and for microwave (2.45GHz) of  $0.000027 \text{ W/kg}$ . With a heating rate of  $0.0045 \times \text{SAR } ^\circ\text{C/min.}$ , Gandhi (1990) and a maximum exposure time per treatment of 5 minutes, the heating of the fetus will be  $0.005$ ,  $0.0005$  and  $0.0000006^\circ\text{C}$ , respectively. Not even the short-wave exposure can produce a detectable heating effect in the uterus environment. The purpose of providing this data was for Hocking and Joyner, employees at that time of Telstra, the Australian telecommunications company, to argue that microwaves cannot be associated with miscarriage because there is no heating. Ouellette-Hellstrom and Stewart correctly reply that the association with microwaves cannot be changed but the explanation (thermal mechanism) can be.

### 12.1.2 Biologically Plausible Mechanism:

Electromagnetically reduced melatonin could be related to spontaneous abortion. According to Sandyk et al. (1992):

**“The causes of spontaneous abortion can be divided into two main categories: those arising from chromosomal anomalies and those arising**

**from abnormalities in the intrauterine environment. In the following communication, we propose that deficient pineal melatonin functions in early pregnancy may be causally related to the development of spontaneous abortions in cases where chromosomal anomalies or structural abnormalities of the uterus have been excluded.”**

Microwaves are shown to be associated with DNA breakage in rats brains, Lai and Singh (1995, 1996) and to cause chromosome aberrations in living humans blood, Garaj-Vrhovac and Fucic (1993), and hence can produce the first cause of spontaneous abortion. Reduced melatonin allows greater concentrations of free radicals to exist. These damage the DNA and chromosomes, leading to a similar mechanism for miscarriage of the deformed fetus.

Therefore, thermal shock and cumulative buildup of thermal lesions is implausible and cumulative cell damage, including melatonin mediated free radical chromosome damage is a highly plausible mechanism.

### **12.1.3 The microwave dose associated with the risk:**

Given the SAR information and the dose-response relationship, it is appropriate to estimate the risk of spontaneous miscarriage in terms of monthly mean exposure.

The quoted mean exposure is 80 - 1200  $\mu\text{W}/\text{cm}^2$ . Assuming a conservatively long estimate of 2 minutes exposure per treatment, 1 treatment per month averages out to the range 0.004 to 0.056  $\mu\text{W}/\text{cm}^2$ , mean 0.03  $\mu\text{W}/\text{cm}^2$ . With 10 treatments per month from 0.04 to 0.56  $\mu\text{W}/\text{cm}^2$ , mean 0.3  $\mu\text{W}/\text{cm}^2$ ; and 20 treatments per month 0.08 to 1.11  $\mu\text{W}/\text{cm}^2$ , mean 0.6  $\mu\text{W}/\text{cm}^2$ . The lowest limit is very difficult to estimate with reliability but the mean level of the middle band is 0.3  $\mu\text{W}/\text{cm}^2$ . This suggests that a 20 to 50% increase in miscarriage occurred with a mean monthly microwave exposure of somewhat less than 0.3  $\mu\text{W}/\text{cm}^2$ . The dose-response relationships in Figures 41 and 41a points to a zero safe level. RF/Microwaves are genotoxic. A genotoxic substance has no safe threshold.

When applying the ubiquitous substance principle and the no non-exposure principle, along with two recently published that is showing that enhanced miscarriage rates are associated with residential power frequency fields, this confirms the conclusions above, Li et al. (2002) and Lee et al. (2002).

### **12.1.4 Relevance to mobile phone base stations:**

The fact that this level of microwave exposure is found near base stations and that there are currently no documented reports of increased incidence of miscarriage occurring near cell sites is not surprising nor a proof that the hypothesis advanced here is wrong. It simply results from the fact that miscarriage is not reported and no statistics are being collected for the general population. Each pregnant woman can only miscarriage once per child, with a several month wait until the next pregnancy. Each spontaneous miscarriage is isolated and does not form a pattern. Many causes are possible. Very few miscarriages are investigated, unless it becomes an issue from a cluster pattern and then a medical or environmental cause is sought. Initially few pregnant women lived near mobile phone base stations. However with the unrestricted sighting policy advocated by the companies and accepted by almost all states and councils, this is changing significantly month by month.

Increased incidence of miscarriage is potentially occurring right now and until it is scientifically assessed, we will not be able to rule out the scientifically indicated probability. The studies presented here give ample grounds for requiring the sighting of cell sites far enough away from residences to avoid an increase in risk of miscarriage.

## **12.2 Cancer**

### **12.2.1 WHO review team's results:**

The World Health Organization (WHO) has reviewed the EMR health research, including studies on cancer published up to about 1992 and published the review report "Environmental Health Criteria 137: Electromagnetic fields (300 Hz to 200 GHz)", Ed. Dr M.H. Repacholi (1993). The team selected 6 cancer related studies, 3 of which the authors conclude no effects, one "radar-exposed populations near air force bases" which has contradictory conclusions and two which do show increases in cancer risk, Figure 39. Given these conclusions on face value their chapter conclusion (8.2.5 in the box) of "no clear evidence of detrimental health effects" appears to be justified. However, two of the four "no effects" studies, Robinette et al. (1980) and Lilienfeld et al. (1978) have been closely studied and followed up by Professor John Goldsmith, Goldsmith (1995, 1998) who concludes that they both show increases in cancer risk. Given this analysis, of the six studies quoted, 5 actually do show increased risks of cancer. Below 24 studies are summarized showing EMR is associated with increased cancer

### **12.2.2 Brief Overview of Epidemiology and RF/MW association with cancer:**

There are many other studies which have found statistically significant increases in adverse health effects, including cancer. It is not that there is no evidence, nor even limited evidence of adverse effects. There is a large body of evidence, only part of which is reviewed here. There are sound scientific reasons for including studies involving ELF high voltage exposures (not reviewed here however), because of the similarity of cellular interactions and because high voltage are a localized source of RF radiation primarily in the 3 to 30 MHz range, which is why you hear a buzz on your car radio as you drive under a powerline.

The following give a brief summary of some of the published studies showing statistically significant increases of cancer from exposure to RF/MW on people.

A major study of radar and radio exposed U.S. Navy personnel, summarized as having no reported effects, includes data which shows statistically significant increases cancer between a group assessed as high exposure compared to a group assessed as low exposure, e.g. All death (RR=1.79 (1.52-2.12)), Accidental Death (RR=2.20 (1.72-2.82)), All Diseases (RR=1.55 (1.19-2.01)), Malignant tumours (RR=1.66 (1.06-2.60)), and Lymphatic and Hematopoietic cancer (RR=2.66 (1.02-4.81)). There was also statistically increased risks of a host of illness including, Musculoskeletal, Organs of Sense, Systematic conditions, Respiratory, Cardiovascular and digestive illness, Skin, Endocrine, Neurological and Mental conditions, Robinette et al. (1980).

EHC 137: Electromagnetic fields			Human responses		
Table 31. Morbidity and mortality studies			Table 31 (continued)		
Exposure conditions	Effect on exposed group	Reference	Exposure conditions	Effect on exposed group	Reference
Radar (pulsed), two groups: (i) <2 (ii) >2 up to 80 W/m <sup>2</sup> , for 1-10 years	No difference in health status between 841 adult males in groups (i) and (ii)	Czerski et al. (1974b); Siekierzynski et al. (1974a,b)	27 MHz shortwave diathermy (questionnaire to 3004 physiotherapists)	Association between heart disease and work with shortwave therapy (number of treatments/week)	Hamburger et al. (1983)
Radar (pulsed), <50 W/m <sup>2</sup> (<0.2 W/kg), for 5-10 years	No effects in clinical evaluations in comparisons between 322 radar workers and 220 non-radar workers; however, more neurasthenic symptoms in exposed group	Djordjevic et al. (1979)	Work at 27 MHz plastic sealers (70% of measurements at the head and hands >300 V/m)	Upper limb paraesthesia and eye irritation noted among 30 exposed workers compared with 11 partially exposed and 22 unexposed workers	Bini et al. (1986)
0.2-5 GHz (pulsed), approx. 10 W/m <sup>2</sup> , 0.05 W/kg (max). Occasional exposure to 1 kW/m <sup>2</sup>	No effect on mortality in male military personnel followed for over 20 years, exposed for 2 years on average (over 40 000 personnel)	Robinette & Silverman (1977); Robinette et al. (1980)	Military personnel exposed to RF/MW fields <2 W/m <sup>2</sup> with daily incidental (minutes) exposures of 2-10 W/m <sup>2</sup> (some times even 100-200 W/m <sup>2</sup> )	Increased risk of cancer morbidity in a retrospective cohort study of military personnel (study group size not given)	Szmigielski et al. (1988)
Males: 2.56-4.1 GHz (CW), 0.05 W/m <sup>2</sup> (max), 0.0002 W/kg (max); Females: 0.6-9.5 GHz (CW), 0.018 W/m <sup>2</sup> (max), 0.0007 W/kg (max), for 0.5-4 years average exposure	No effect on life span or cause of death of 1800 employees and 3000 dependents of US Embassy personnel	Lilienfeld et al. (1978)	51 male/62 female operators of plastic welding machines (27 MHz, 50% of welders exceeded 250 W/m <sup>2</sup> ) 23 female controls (sewing machine operators)	Increase rates of paraesthesia in hands, neurasthenia, and eye complaints; diminished 2-point discrimination ability	Kolmodin-Hedman et al. (1988)
Long-term microwave exposure of military personnel (interviews)	Higher frequency of microwave exposure in 14 polycythaemia cases than in 17 age-matched controls	Friedman (1981)	Amateur radio operators	Deaths from all causes less than expected from national rates; increased risk of leukaemia	Milham (1985)
Radar-exposed populations near air force bases	Increased cancer mortality compared with population-matched controls. No increase in cancer mortality compared with population-matched controls	Lester & Moore (1982); Lester (1985); Polson & Merritt (1985)	1.3-10 GHz, 0.1 to 10-μs pulses, RF exposure of radar mechanics often exceeded 10 W/m <sup>2</sup>	No differences in neurological symptoms and findings between 17 exposed and 12 controls; increased protein band in CSF in the exposed group	Nilsson et al. (1989)
Children exposed to various air pollutants and RF	Duration and severity of tonsillitis increased	Shandala & Zvinjatskovsky (1988)	<b>8.2.5 Conclusions</b>  In summary, the epidemiological and comparative clinical studies do not provide clear evidence of detrimental health effects in humans from exposure to RF fields. Some occupational groups, such as exposed physiotherapists and industrial workers, should be studied further. The question of whether RF might act as a carcinogen should be further evaluated in epidemiological studies.  Occupational exposure to RF will be at higher levels than that encountered by the general population, and, thus, there is less likelihood of health effects in the general population as a whole.		

Figure 43: Table 31 from WHO (1993) showing the review team's selection of studies on EMR and morbidity and mortality and their summary of the study's conclusions, and, in the box on the bottom right hand side, the review teams conclusions on this set of studies and the reproductive studies.

You will note that the WHO review, WHO (1993) refers to Robinette et al.(1980) as showing no effects. Similarly for Lilienfeld et al. (1978). The data contained in these papers and reports indicate otherwise, Goldsmith (1995, 1997). The following is a summary of the cancer based studies published over the past two decades.

- Cancer incidence in the vicinity of Wichita, Kansas was found to be higher on ridges which were exposed to radar transmissions than those residents who lived in the valleys, Lester and Moore (1982 a). Residents were potentially exposed to two radars, one radar and no radars with relative cancer incidences of 470, 429 and 303 per 100,000 (1.55: 1.42: 1.00), trend  $p=0.03$ . The association persisted through adjustments for age, sex, race and socio-economic factors.



- Lester and Moore (1982b) found significantly higher cancer rates in U.S. counties with Air Force bases compared to those without Air Force bases, which they related to prolonged environmental exposure to RF/MW from radar.
- Polson and Merritt (1985) criticized the analysis of Lester and Moore (1982b), pointing out weaknesses in their use of the data, such as a city could be in a country with no Air Force Base but be closer to a base in another country than a city in that county. Having made corrections for this, Lester and Moore (1985) found strengthened associations between cities and air force bases, with higher incidences of cancer related to radar transmissions.
- Lin et al. (1985) studied 951 cases of brain tumors among white male residents of Maryland during the period 1969-1982. Fifty cases of glioma and astrocytoma were observed among electrical workers exposed to EMR compared to an expected number of 18, i.e. an risk ratio of 2.8. While their exposure was mainly to ELF fields it shows the common link over a wide range of frequencies.
- Increased risk of leukaemia amongst amateur radio operators (Milham, 1985).
- In 1985 an unusual number of children with leukaemia were identified living in the vicinity of broadcasting facilities (OR = 3.4: CI=0.70 -16.41), Maskarinec et al. (1993).
- De Guire et al. (1987) report increased malignant melanoma of the skin in workers in a telecommunication industry, affecting only men, SIR = 2.7 CI : 1.31-5.02).
- Thomas et al. (1987) report a 10-fold increase in astrocytic brain tumor among electronics and repair workers employed for 20 years or more. Some risk was due to solvents, put at a factor of 2, placing RF/MW contribution at a factor of 5.
- Milham (1988) studied 67,829 amateur radio operators in Washington State and California. He concludes "The all-cause standardized mortality ratio (SMR) was 71 but a statistically significant increased mortality was seen for cancers of the other lymphatic tissues (SMR = 162), a rubric which includes multiple myeloma and non-Hodgkin's lymphomas. The all leukemia SMR was slightly but not significantly elevated (SMR = 124). However, mortality due to acute myeloid leukemia was significantly elevated (SMR = 176).
- Szmigielski et al. (1988) studied polish military personnel exposed to microwave radiation and reported that cancer morbidity was three times higher in the exposed group than the control group.
- Electrical workers in Los Angeles County have a 4.3-fold increased risk of certain brain tumors (Preston-Martin et al. 1989).
- An increased incidence of malignant brain tumors has been reported in children of fathers exposed to electromagnetic fields and electronic solvents (Johnson and Spitz, 1989).
- Hayes et al.(1990) report an Odds ratio for all testicular cancer of 3.1 (CI: 1.4-6.9) for a small sample of workers who were occupationally exposed to RF/MW radiation.

- U.S. Navy electrician's mates have an excess risk of leukaemia, RR=2.4 (1.0-5.0), Garland et al. (1990)
- Savitz and Chen (1990) show significant increased risk of childhood cancer (Neuroblastoma (OR=11.8\*), Brain Tumour (OR=2.7\*) and CNS tumors (OR=1.7)) associated with parents who work in electrical and electronic industries.
- Increased risk for all brain tumours (RR=2.9 (1.2-5.9)) and glioblastomas (RR=3.4 (1.1-8.0)) for assemblers, and repairmen in the radio and TV industry, Tornqvist et al. (1991)
- Goldsmith (1995) reports an up-dated analysis of the US embassy in Moscow which does show a significant elevated risk of a wide range of adult cancers, and including childhood leukaemia, after years of microwave irradiation, exposed to average levels of radar produced microwaves of long-term average indoor exposure of 0.2 to 0.5  $\mu\text{W}/\text{cm}^2$ , daily peaks between 5 and 18  $\mu\text{W}/\text{cm}^2$  on the outside walls.
- Increased risk of female breast cancer with exposure to radiofrequency EMF, RR=1.15 (1.1-1.2), Cantor et al. (1995).
- Hocking and Gordon (1996) found a 2.74-fold increase in childhood leukaemia death within 4 km of three TV and FM radio transmission masts in North Sydney between 1972 and 1990. Mean direct exposures were measured in the range 0.1 to 1  $\mu\text{W}/\text{cm}^2$ . Mean personal exposures are in the range 0.002 to 0.02  $\mu\text{W}/\text{cm}^2$ .
- Polish Military personnel (1971-85) exposed to above average radar and radio sourced RF/MW show large increases in leukaemia (Lymphoma: RR=5.8 (2.11-9.74); Chronic lymphocytic: RR=3.7 (1.45-5.18); Acute Lymphoblastic: RR=5.8 (1.22-18.16); Chronic myelocytic: RR=13.9 (6.72-22.12); Acute myeloblastic: RR=8.6 (3.54-13.67) and Total: RR=6.31 (3.12-14.32). Also show statistically significant associations for cancer of the esophagus and stomach, colorectal, skin (including melanomas), CNS and brain. (Szmigielski, 1996)
- U.S. Air Force personnel showed increased incidence of brain tumour with exposure to ELF (RR=1.28 (0.95-1.74)), and RF/MW (RR=1.39 (1.01-1.90)).
- Dolk et al. (1997 a, b) found small but significant increases in adult leukaemia, which decreases with distance from the transmitter, associated with 21 highly powerful Regional FM and TV transmission towers in the United Kingdom. The 20 site study shows adult leukaemia incidence following the typical exposure pattern as it varies with radial distance, i.e. low near the tower, peaking between 1.5 and 3 km and then decreasing with distance. This is a strong dose-response result.

The Sutton Coldfield result, Dolk et al. (1997a), differs from the 20 site result because of five people with leukaemia who live near the tower. These 5 people are a classic cancer cluster. Clustering of cancer has been used as a confounding factor to dismiss the association with a toxin. In this case the cluster is kept in the sample to weaken the result through inconsistency with the 20 site study. If the cluster, which has a highly probable chance of being random, is removed, then the Sutton Coldfield result is fully

consistent with the 20 site result when the exposure and population patterns are appropriately considered, Cherry (2001).

- Szmigielski (1998), a conference paper reported in microwave news, concludes that the data suggests that cancers “develop faster, with a shorter latency period” in servicemen with occupational RF/MW exposures. He also found a dose-response relationship with cancer rate against maximum microwave exposure.

Number of Men	Peak Exposure Range $\mu\text{W}/\text{cm}^2$	Cancer Rate Ratio
1900	100-200	1.69
1320	200-600	1.57
350	600-1000	4.62
280	1000-	4.93

The morbidity rate for hematological and lymphatic cancers was 5.33 times greater than unexposed servicemen, also a highly significant result. (MWN Jan/Feb, 1998 p10)

- Michelozzi, et al (2002) showed that chronic residential RF exposure of people living in the vicinity of the Vatican radio towers, outside Rome, show a significant dose-response increase in adult and childhood leukaemia.

### 12.2.3 Cancer studies conclusion:

A genotoxic substance causes cancer. There is extremely strong evidence that RF/microwave radiation is genotoxic. There is also overwhelming direct evidence that RF/MW radiation is carcinogenic, raising the incidence of cancer in exposed military, occupational and residential populations. Many dose-response relationships, even at residential exposure levels give substantial evidence of a causal link between of cancer and chronic RF/MW exposure. Cancer of many organs is found and is expected since RF/MW exposure is a whole body exposure. Most commonly found cancers are leukaemia and brain tumors.

Evidence that RF/MW causes cancer is also evidence of shortening life and accelerating aging, as cancer is a strong result of aging. In a toxic substances also enhance the apoptosis rate. This is also a basic mechanism for advancing the ageing range.

## 13. Conclusions:

Scientific studies at the cellular level, whole animal level and involving human populations, shows compelling and comprehensive evidence that RF/MW exposure down to very low residential exposure levels, levels which are a minute fraction of present “safety standards”, results in altered brain function, sleep disruption, depression, chronic fatigue, headache, impaired memory and learning, adverse reproductive outcomes including miscarriage, still birth, cot death, prematurely and birth deformities. Many other adverse health effects have been found, predominantly cancer of many organs, especially brain cancer, leukaemia, breast cancer and testicular cancer. Studies have also found that RF/MW exposed parents have more children with CNS cancers and other health defects. These effects are consistent with genetic damage caused by RF/MW. Many scientific studies have found chromosome aberrations and DNA damage with RF/MW exposure, the first being published in 1959. Three primary biological mechanisms are linked to these effects, genotoxicity, altered cellular calcium ions and melatonin reduction. With melatonin reduction there is

usually a rise in serotonin which is associated with awakesness, alertness, anxious, anger, rage and violence depending on the serotonin level, the person and the circumstances. Most of these and many other neurological and health effects are associated with geomagnetic activity, through the Schumann Resonance signal mechanism which alters the human melatonin levels. It strongly supports a causal connection between the extremely low intensity natural electronic signals and the thousands to billions of times higher humanly generated electromagnetic signals, and the listed health effects.

**Hence there is strong evidence that ELF and RF/MW is associated with accelerated aging (enhanced cell death and cancer) and moods, depression, suicide, anger, rage and violence, primarily through genotoxic damage, alteration of cellular calcium ions and the melatonin/serotonin balance.**

#### **References:**

- Adair, E.R., 1993: "Thermal physiology of radiofrequency radiation (RFR) interactions in animals and humans". pp 245-269, In "Radiofrequency Radiation Standards - Biological effects, Dosimetry, epidemiology and Public Health Policy", Ed Klauenberg, Grandolfo and Erwin, NATO ASI Series A: Life Sciences Vol 274, Plenum Press, New York.
- Adey, W.R., 1979: "Neurophysiologic effects of Radiofrequency and Microwave Radiation". Bull. N.Y. Acad. Med. 55(11): 1079-93.
- Adey, W.R., 1980: "Frequency and Power windowing in tissue interactions with weak electromagnetic fields". Proc. IEEE, 68:119-125.
- Adey, W.R., 1981: "Tissue interactions with non-ionizing electromagnetic fields". Physiological Reviews, 61: 435-514.
- Adey, W.R., Bawin, S.M., and Lawrence, A.F., 1982: "Effects of weak amplitude-modulated microwave fields on calcium efflux from awake cat cerebral cortex". Bioelectromagnetics, 3: 295-307.
- Adey, W.R., 1988a: "The cellular microenvironment and signaling through cell membranes". Prog. Clin. Biol. Res., 257:81-106.
- Adey, W.R., 1988b: "Cell membranes: the electromagnetic environment and cancer promotion". Neurochem. Res., 13(7):671-677.
- Adey, W.R., 1989: "The extracellular space and energetic hierarchies in electrochemical signaling between cells". pp 263-290. In "Charge and Field Effects in Biosystems - 2", eds Allen, M.J., Cleary, S.F. and Hawkrige, E.M., Plenum Press, New York.
- Adey, W.R., 1990: "Electromagnetic fields and the essence of living systems". In "Modern Radio Science", J Bach Anderson Ed., Oxford University Press, 1990, pp 1-37.
- Adey, W.R., 1991: "Signal functions of brain electrical rhythms and their modulation by external electromagnetic fields". pp323-351. In "Induced rhythms of the brain", Eds Basar, e., and Bullock, T.H., Birkhauser, Boston.

- Adey, W.R., 1992a: "Collective properties of cell membranes". pp47-77. In "interaction mechanisms of Low-level electromagnetic fields in living systems", Oxford University Press.
- Adey, W.R., 1992b: "ELF magnetic fields and promotion of cancer: experimental studies". In 'Interaction mechanisms of low-level electromagnetic fields in living systems', Bengt Norden and Claes Ramel (eds), Oxford Univ. Press.
- Adey, W.R., 1992c: "Collective properties of cell membranes". pp47-77 In 'Interaction mechanisms of low-level electromagnetic fields in living systems', Bengt Norden and Claes Ramel (eds), Oxford Univ. Press.
- Adey, W.R., 1993: "Biological Effects of electromagnetic fields". *Journal of Cellular Biochemistry*, 51: 410-416.
- Adey, W.R., Byus, C.V., Cain, C.D., Haggren, W., Higgins, R.J., Jones, R.A., Kean, C.J., Kuster, N., MacMurray, A., Phillips, J.L., Stagg, R.B., and Zimmerman, G., 1996: "Brain tumor incidence in rats chronically exposed to digital telephone fields in an initiation-promotion model". Bioelectromagnetic Society Annual Meeting, June 9-14, 1996, Victoria BC, Canada.
- Albert, E.C., Blackman, C.F., and Slaby, F., 1980: "Calcium dependent secretory protein release and calcium efflux during RF irradiation of rat pancreatic tissue slices". In *Ondes Electromagnetiques et Biologie*, A.J. Berteaud and B. Servantie, eds, Paris, France, pp 325-329.
- Alberts, B., Bray, D., Lewis, J., Raff, M., Roberts, K. and Watson, J.D., 1994: "Molecular Biology of the cell", 3rd edition, Publ. Garland Publishing, New York.
- Altpeter, E.S., Krebs, Th., Pfluger, D.H., von Kanel, J., Blattmann, R., et al., 1995: "Study of health effects of Shortwave Transmitter Station of Schwarzenburg, Berne, Switzerland". University of Berne, Institute for Social and Preventative Medicine, August 1995.
- Alvarez, J., Montero, M., and Garcia-Sancho, J., 1992; *FASEB J*, 6:786-792.
- Anderson, B.S., and Henderson, A.K., 1986: "Cancer incidence in census tracts with broadcasting towers in Honolulu, Hawaii". Honolulu City Council Report, Contact No. C17015, October 27, 1986.
- Athey, T.W., 1981: "Comparison if RF-induced calcium efflux from chick brain tissue at different frequencies: do scaled power density windows align ?" *Bioelectromagnetics*, 2: 407-409.
- Australian Standard: Radiofrequency radiation, Part 1: Maximum exposure levels - 100 kHz to 300 Ghz, Standards Australia AS 2772.1-1990.
- Barnett, S.B., 1994: "CSIRO report on the status of research on biological effects and safety of electromagnetic radiation: telecommunication frequencies. "Ultrasonics Laboratory, Radiophysics Division, CSIRO, 174 pp, June 1994.
- Balcer-Kubiczek, E.K. and Harrison, G.H., 1985: "Evidence for microwave carcinogenesis". *Carcinogenesis*, 6: 859-864.
- Balcer-Kubiczek, E.K., 1994: "Experimental studies of electromagnetic field induced carcinogenesis in cultured mammalian cells." Chapter 10 in "On the nature of the electromagnetic field interactions with biological systems". Ed. A.H. Frey, Publ. R.G. Landes Co.

- Balodis, V., Brumelis, G., Kalviskis, K., Nikodemus, O., Tjarve, D., and Znotina, V., 1996: "Does the Skrunda Radio Location Station diminish the radial growth of pine trees?". *The Science of the Total Environment*, Vol 180, pp 57-64.
- Balode, Z., 1996: "Assessment of radio-frequency electromagnetic radiation by the micronucleus test in Bovine peripheral erythrocytes". *The Science of the Total Environment*, 180: 81-86.
- Baranski, S., 1972: "Histological and histochemical effects of microwave irradiation on the central nervous system of rabbits and guinea pigs". *Am. J. Physiol. Med.*, 51: 182-190.
- Baranaski, S., and Edelwejn, Z, 1974: "Pharmacological analysis of microwave effects on the central nervous system in experimental animals". In "Biological effects and health hazards of microwave radiation: Proceedings of an International Symposium". P. Czerski eds, Polish Medical Publishers, Warsaw.
- Bassen, H., Herchenroeder, P., Cheung, A., and Neuder, S., 1977: "Evaluation of an implantable electric field probe within finite simulated tissues". *Radio Science*, 12, Suppl 6: 15-25.
- Bawin, S.M., Gavalas-Medici, R., and Adey, W.R., 1973: "Effects of modulated very high frequency fields on specific brain rhythms in cats." *Brain Research*, 58: 365-384.
- Bawin, S.M., Kaczmarek, L.K., and Adey, W.R., 1975: "Effects of modulated VHF fields on the central nervous system". *Ann. N.Y. Acad. Sci.* 247:74-81.
- Bawin, S.M. and Adey, W.R., 1976: "Sensitivity of calcium binding in cerebral tissue to weak electric fields oscillating at low frequency". *Proc. Natl. Acad. Sci. USA*, 73: 1999-2003.
- Bawin, S.M., Adey, W.R., and Sabbot, I.M., 1978: "Ionic factors in release of  $^{45}\text{Ca}^{2+}$  from chicken cerebral tissue by electromagnetic fields". *Proc. Natl Acad Sci. USA*, 75: 6314-6318.
- Beale, I.L., Pearce, N.E., Conroy, D.M., Henning, M.A. and Murrell, K.A., 1997: "Psychological effects of chronic exposure to 50 Hz magnetic fields in humans living near extra-high-voltage transmission lines." *Bioelectromagnetics*, 18(8):584-594 .
- Berman, E., Carter, H.B., and House D., 1982: "Reduce weight in mice offspring after in utero exposure to 2450 MHz (CW) microwaves". *Bioelectromagnetics*, 3(2): 285-291.
- Berridge, M., J., 1985: "The molecular basis of communication within the cell". *Scientific American*, 253 (4), (Oct) pp 142-152.
- Bini, M.G., Checcucci, A., Ignesti, A., Millanta, L., Olma, R., Rubina, N., and Vanni, R., 1986: "Exposure of workers to intense RF electric fields that leak from plastic sealers". *J. Microwave Power*, Vol 21, pp 33-40.
- Blackman, C.F., Elder J.A., Weil, C.M., Benane S.G., Eichinger, D.C., and House, D.E., 1979: "Induction of calcium-ion efflux from brain tissue by radiofrequency radiation: effects of modulation frequency and field strength". *Radio Science* 14(6S):93-98.
- Blackman, C.F., Benane, S.G., Elder, J.A., House, D.E., Lampe, J.A., and Faulk, J.M., 1980a: "Induction of Calcium-Ion Efflux from Brain Tissue by Radiofrequency Radiation: effect of sample number and modulation frequency on the power-density window". *Bioelectromagnetics*, 1: 35-43.

- Blackman, C.F., Benane, S.G., Joines, W.T., Hollis, M.A. and House, D.E., 1980b: "Calcium-Ion efflux from brain tissue: power density versus internal field-intensity dependencies at 50 MHz RF radiation.", *Bioelectromagnetics*, 1: 277-283.
- Blackman, C.F., Benane, S.G., House, D.E., and Joines, W.T., 1985: "Effects of ELF (1-120 Hz) and modulated (50Hz) RF fields on the efflux of calcium ions of brain tissue, in vitro". *Bioelectromagnetics*, 6:1-11.
- Blackman, C.F., Benane, S.G., Elliott, D.J., and Pollock, M.M., 1988: "Influence of Electromagnetic Fields on the Efflux of Calcium Ions from Brain Tissue in Vitro: A Three-Model Analysis Consistent with the Frequency Response up to 510 Hz". *Bioelectromagnetics*, 9:215-227.
- Blackman, C.F., Kinney, L.S., House, D.E., and Joines, W.T., 1989: "Multiple power-density windows and their possible origin". *Bioelectromagnetics*, 10: 115-128.
- Blackman, C.F., Benane, S.G., and House, D.E., 1991: "The influence of temperature during electric- and magnetic-field induced alteration of calcium-ion release from in vitro brain tissue". *Bioelectromagnetics*, 12: 173-182.
- Bonassi, S., Abbondandolo, A., Camurri, L., Dal Pra, L., De Ferrari, M., Degraffi, F., Forni, A., Lamberti, L., Lando, C., Padovani, P., et al, 1995: "Are chromosome aberrations in circulating lymphocytes predictive of future cancer onset in humans? Preliminary results of an Italian cohort study". *Cancer Genet. Cytogenet.*, 79(2):133-135.
- Bonhomme-Faivre, L., Marion, S., Bezie, Y., Auclair, H., Fredj, G. and Hommeau, C., 1998: "Study of human neurovegetative and hematologic effects of environmental low-frequency (50-Hz) electromagnetic fields produced by transformers." *Arch. Environ. Health*, Mar;53(2):87-92 .
- Borlongan, C.V., Kanning, K., Poulos, S.G., Freeman, T.B., Cahill, D.W., Sanberg, P.R. 1996: "Free radical damage and oxidative stress in Huntington's disease". *J Florida Med. Assoc.*, 83: 335-341.
- Brent, R.L., Beckman, D.A. and Landel, C.P., 1993: "Clinical teratology". *Current Opinion in Pediatrics*, 5(2): 201-211.
- Bretscher, M.S., 1985: "The molecules of the Cell Membrane". *Scientific American*, 253 (4), (Oct) pp 100-108.
- Brogger, A., Hagmar, L., Hansteen, I.L., Heim, S., Hogstedt, B., Knudsen, L., Lambert, B., Linnainmaa, K., Mitelman, F., Nordenson, I., et al, 1990: "An inter-Nordic prospective study on cytogenetic endpoints and cancer risk. Nordic Study Group on the Health Risk of Chromosome Damage". *Cancer Genet. Cytogenet.* 45(1):85-92.
- Brulfert, A., Miller, M.W., Robertson, D., Dooley, D.A., and Economou, P., 1985: "A cytohistological analysis of roots whose growth is affected by a 60 Hz electric field". *Bioelectromagnetics*, 6 (3): 283-291.
- Bullough, J., Rea, M.S., and Stevens, R.G., 1996: "Light and magnetic fields in a neonatal intensive care unit." *Bioelectromagnetics*, 17(5): 396-405.
- Burch, J.B., Reif, J.S., Pitrat, C.A., Keefe, T.J. and Yost, M.G., 1997: "Cellular telephone use and excretion of urinary melatonin metabolite". *Annual review of research in Biological Effects of electric and magnetic fields from generation delivery and use of electricity*, San Diego, CA, Nov 9-13, 1997.

- Burch, J.B., Reif, J.S., Yost, M.G., Keefe, T.J. and Pittratt, C.A., 1998: "Nocturnal excretion of urinary melatonin metabolite among utility workers". *Scand J Work Environ Health* 24(3): 183-189.
- Burch, J.B., Reif, J.S., Yost, M.G., Keefe, T.J. and Pittratt, C.A., 1999a: "Reduced excretion of a melatonin metabolite among workers exposed to 60 Hz magnetic fields" *Am J Epidemiology* 150(1): 27-36.
- Burch, J.B., Reif, J.S. and Yost, M.G., 1999b: "Geomagnetic disturbances are associated with reduced nocturnal excretion of melatonin metabolite in humans". *Neurosci Lett* 266(3):209-212.
- Burch, J.B., Reif, J.S., Noonan, C.W. and Yost, M.G., 2000: "Melatonin metabolite levels in workers exposed to 60-Hz magnetic fields: work in substations and with 3-phase conductors". *J of Occupational and Environmental Medicine*, 42(2): 136-142.
- Byus, C.V., Pieper, S., and Adey, W.R., 1987: "The effects of low-energy 60 Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase". *Carcinogenesis*, 8: 1385-
- Byus, C.V., Kartun, K., Pieper, S., and Adey, W.R., 1988: "Increased Ornithine Decarboxylase Activity in Cultured Cells Exposed to Low Energy Modulated Microwave Fields and Phorbol Ester Tumor Promoters", *Cancer Research*, 48, 4222-4226.
- Byus, C.V., 1994: "Alterations in Ornithine Decarboxylase Activity: a cellular response to Low-Energy Electromagnetic Field Exposure". Updated Sept 1994 from Summary and Results of the April 26-27, 1993 Radiofrequency Radiation Conference.
- Cairnie, A.B., Prud'homme-Lalonde, L.F., Harding, R.K., and Zuker, M., 1980: "The measurement of rectal and testes temperature in conscious mice, with observations on the effect of direct heating". *Phys. Med. Bio.*, 25(3): 317-322.
- Campbell, W.H., 1967: "Geomagnetic Pulsations", pp821-909, In "Physics of Geomagnetic Phenomena, Vol II", Matsushita, S. and Campbell W.H., eds, Academic Press.
- Cantor, K.P., Stewart, P.A., Brinton, L.A., and Dosemeci, M., 1995: "Occupational exposures and female breast cancer mortality in the United States". *Journal of Occupational Medicine*, 37(3): 336-348.
- Carney, J.M., and Floyd, R.A., 1991: "Protection against oxidative damage to CNS by ( $\alpha$ -phenyl-tert-butyl)nitron (PBN) and other spin-trapping agents: A novel series of nonlipid free radical scavengers". *J. Mol. Neurosci.*, 4:47-57.
- Carney, J.M., Starke-Reed, P.E., Oliver, C.N., Landum, R.W., Cheng, M.S., Wu, J.F. and Floyd, R.A., 1991: "Reversal of age-related increase in brain protein oxidation, decrease in enzyme activity, and loss in temporal and spatial memory by chronic administration of the spin-trapping compound N-tert-butyl- $\alpha$ -phenylnitron". *Proc. Nat. Acad. Sci. (USA)*, 88:3633-3636.
- Chazan, B., Janiak, M., Szmigielski, S., and Troszynski, M., 1983: "Development of murine embryos and fetuses after irradiation with 2450 MHz microwaves". *Problemy Medycyny Wieku Rozwojowego*, 12:164-173.



- Cherry, N, 2001: Re: "Cancer incidence near radio and television transmitters in Great Britain, II All high power transmitters", Dolk et al. 1997 a,b in *American J. of Epidemiology*, 145(1):1-9 and 10-17. Comment in *American J of Epidemiology* 153(2): 204-205.
- Cherry, N.J., 2002: "Schumann Resonances, a plausible biophysical mechanism for the human health effects of Solar/Geomagnetic Activity". *Natural Hazards* 56: 279-331.
- Chiang, H., Yap. G.D., Fang, Q.S., Wang, K.Q., Lu., D.Z., and Zhou, Y.K., 1989: "Health effects of environmental electromagnetic fields". *Journal of Bioelectricity*, 8:127-131.
- Chou, C-K., Guy, A.W., Kunz, L.L., Johnson, R.B., Crowley, J.J. and Krupp, JH., 1992: "Long-term, low-level microwave irradiation of rats". *Bioelectromagnetics* 13: 469-496.
- Cicchione, G., Mirabelli, D., Levis, A., Gavarotti, P., Rege-Cambrin, G., Davico, L., and Vineis, P., 1993: "Myeloid leukemias and myelodysplastic syndromes: chemical exposure, histologic subtype and cytogenetics in a case-control study". *Cancer Genetics & Cytogenetics* 1993 Jul 15;68(2):135-9.
- Clarkson, P.M., 1995: "Antioxidants and physical performance". *Crit. Rev. Food. Sci. Nutri.*, 35:131-141.
- Cleary, S.F., Liu, L.M., and Merchant, R.E., 1990a: "In vitro lymphocyte proliferation induced by radio-frequency electromagnetic radiation under isothermal conditions". *Bioelectromagnetics*, 11: 47-56.
- Cleary, S.F., Liu, L.M., and Merchant, R.E., 1990b: "Glioma proliferation modulated in vitro by isothermal radio-frequency radiation exposure". *Radiation Research*, 121: 38-45.
- Cleary, S.F., Liu, L.M., and Cao, G., 1992: "Effects of RF power absorption in mammalian cells". *Annals of the N.Y. Acad. Sci.*, 649: 166-175.
- Cleary, S.F., Cao, G., and Liu, L.M., 1996: "Effects of isothermal 2.45 GHz microwave radiation on the mammalian cell cycle: comparison with effects of isothermal 27 MHz radiofrequency radiation exposure". *Bioelectrochemistry and Bioenergetics*, 39: 167-173.
- Clemens, M.R., 1991: "Free radicals in chemical carcinogenesis". *Klinische Wochenschrift* , 69(21-23):1123-34.
- Clutton, S., 1995: "The importance of oxidative stress in apoptosis". *British Medical Bulletin*, 53 (3): 662-668.
- Conti, P., Gigante, G.E., Cifone, M.G., Alesse, E., Ianni, G., Reale, M., and Angeletti, P.U., 1983: "Reduced mitogenic stimulation of human lymphocytes by extremely low frequency electromagnetic fields". *FEBS* 0850, 162 (1): 156-160.
- Davis, S., 1997: "Weak residential Magnetic Fields affect Melatonin in Humans", *Microwave News*, Nov/Dec 1997.
- De Guire, L., Theriault, G., Iturra, H., Provencher, S., Cyr, D., and Case, B.W., 1988: "Increased incidence of malignant melanoma of the skin in workers in a telecommunications industry". *British Journal of Industrial Medicine*, Vol 45, pp 824-828.
- Demers, P.A., Thomas, D.B., Rosenblatt, K.A., Jimenez, L.M., Mc Tiernan, A, Stalsberg, H., Stemhagen, A., Thompson, W.D., McCrea, M.G., Satariano, W., Austin, D.F., Isacson, P., Greenberg, R.S., Key, C., Kolonel, L.N., and West, D.W., 1991: "Occupational exposure

- to electromagnetic fields and breast cancer in men", *Am. J. Epidemiology*, 134 (4): 340-347.
- Demers, P.A., Vaughan, T.L., Checkoway, H., Weiss, N.S., Heyer, N.J., and Rosenstock, L., 1992: Cancer Identification Using a Tumor Registry verses Death Certificates in Occupational Cohort Studies in the United States. *Am. Jour. of Epidem.*;136,10: 1232-1240
- Djordjevic, Z., Kolak, A., Stojkovic, M., Rankovic, N. and Ristic, P., 1979: "A study of the health status of radar workers". *Aviat. space environ. Med.*, Vol 50, pp 396-398.
- Dolk, H., Shaddick, G., Walls, P., Grundy, C., Thakrar, B., Kleinschmidt, I. and Elliott, P., 1997a: "Cancer incidence near radio and television transmitters in Great Britain, I - Sutton-Colfield transmitter". *American J. of Epidemiology*, 145(1):1-9.
- Dolk, H., Elliott, P., Shaddick, G., Walls, P., Grundy, C., and Thakrar, B., 1997b: "Cancer incidence near radio and television transmitters in Great Britain, II All high power transmitters". *American J. of Epidemiology*, 145(1):10-17.
- Dorland 28, 1994: "Dorland's illustrated medical dictionary, edition 28", Publ. W.B. Saunders and Co., Philadelphia, USA.
- Dumansky, J.D., and Shandala M.G., 1974: "The biologic action and hygiene significance of electromagnetic fields of super high and ultrahigh frequencies in densely populated areas". pp289-293, in "Biologic effects and Health Hazards of Microwave Radiation", Ed. P. Czerski, Warsaw Polish Medical Publication.
- Durham, A.C. and Walton, J.M., 1982: "Calcium ions and the control of proliferation in normal and cancer cells." *Biosci. Rep.*, Jan;2(1):15-30 .
- Dutta, S.K., Subramonian, A., Ghosh, B., and Parshad, R., 1984: "Microwave radiation-induced calcium ion efflux from human neuroblastoma cells in culture". *Bioelectromagnetics*, 5: 71-78.
- Enwonwu, C.O., and Meeks, V.I., 1995: "Bionutrition and oral cancer in humans." *Critical Reviews in Oral Biology & Medicine*. 6(1):5-17.
- Feychting, M., and Ahlbom, A., 1993: "Magnetic fields and cancer in children residing near Swedish High-voltage power lines". *Am J. Epidemiology*, 138 (7): 467-481.
- Feychting, M., Schulgen, G., Olsen, J.H., and Ahlbom, A., 1995: "Magnetic fields and childhood cancer- pooled analysis of two Scandinavian studies". *European J. of Cancer*, 31A (12): 2035-2039.
- Fletcher, W.H., Shui, W.W., Haviland, D.A., Ware, C.F., and Adey, W.R., 1986: " A modulated-microwave field and tumor promoters similarly enhance the action of alpha-lymphotoxin (aLT)". *Proce. Bioelectromagnetics Soc.*, 8th Annual Meeting, Madison, Wisconsin, p12, Bioelectromagnetics Society, Frederick, MD.
- Fletcher, W.H., Shiu, W.W., Ishida, T.A., Haviland, D.L., and Ware, C.F., 1987: "Resistance to the cytolytic action of lymphotoxin and tumor necrosis factor coincides with the presence of gap junctions uniting target cells". *J. Immunology*, 139: 956-
- Floyd, R.A., 1991: "Oxidative damage to behavior during aging". *Science*, 254:1597.

- Forster, M.J., Dubey, A., Dawson, K.M., Stutts, W.A., Lai, H., and Sotal, R.S., 1996: "Age-related losses in cognitive function and motor skills in mice are associated with oxidative protein damage in the brain." *Proc. Nat. Acad. Sci. (USA)*, 93: 4765-4769.
- Frey, A.H., 1971: "Biological function as influenced by low power modulated RF energy". *IEEE trans on Microwave Theory and Techniques*, MTT-19:153-164.
- Frey, A.H., Feld, S.R. and Frey, B., 1975: "Neural function and behaviour: defining the relationship". *Ann. N.Y. Acad. Sci.*, 247: 433-439.
- Frey, A.H., 1988: "Evolution and results of biological research with low intensity nonionizing radiation". pp 785-837, In "Modern Bioelectricity", Ed A. Marino, Publ. Marcel Dekker Inc, New York.
- Frey, A.H., 1994: "An integration of the data on mechanisms with particular reference to cancer", Chapter 2 in "On the Nature of electromagnetic Field Interactions with Biological Systems", Ed A.H. Frey, Publ. R.G. Landes Co. Medical Intelligence Unit, Austin, Texas.
- Friedman, H.L., 1981: "Are chronic exposure to microwaves and polycythemia associated [letter]". *New England J. Med.*, 304 (6), pp 357-358.
- Galvanovskis, J., Sandblom, J., Bergqvist, B., Galt, S., and Hamnerius, Y., 1996: "The influence of 50-Hz magnetic fields on cytoplasmic  $Ca^{2+}$  oscillations in human leukemia T-cells". *The Science of the Total Environment*, 180:19-33.
- Gandhi, O.P., 1980: "State of knowledge for electromagnetic absorbed dose in man and animals". *Proc. IEEE*, 68 (1), 24-32.
- Gandhi, O.P., 1990: "ANSI radiofrequency safety guide: Its rationale, some problems and suggested improvements". pp 28-46. In "Biological effects and medical applications of electromagnetic energy", Ed O.P. Gandhi, Publ. Prentice Hall.
- Garaj-Vrhovac, V., Fucic, A, and Horvat, D., 1990: "Comparison of chromosome aberration and micronucleus induction in human lymphocytes after occupational exposure to vinyl chloride monomer and microwave radiation"., *Periodicum Biologorum*, Vol 92, No.4, pp 411-416.
- Garaj-Vrhovac, V., Fucic, A, and Horvat, D., 1992: "The correlation between the frequency of micronuclei and specific aberrations in human lymphocytes exposed to microwave radiation in vitro". *Mutation Research*, 281: 181-186.
- Garaj-Vrhovac, V., and Fucic, A., 1993: "The rate of elimination of chromosomal aberrations after accidental exposure to microwave radiation". *Bioelectrochemistry and Bioenergetics*, 30:319-325.
- Garson, O.M., McRobert, T.L., Campbell, L.J., Hocking, B.A., and Gordon, I., 1991: "A chromosomal study of workers with long-term exposure to radio-frequency radiation". *The Medical Journal of Australia*, 155: 289-292.
- Giuliana, L., Vignati, M., Cifone, M.G., and Alesse, E., 1996: "Similarity of effects induced by ELF, amplitude modulated RF and ELF magnetic fields on PHB in vitro". *Radiation in Work*, Supplement PS 309, p 332.
- Goldhaber, M.K., Polen, M.R., and Hiatt, R.A., 1988: "The risk of miscarriage and birth defects among women who use visual display terminals during pregnancy". *Am. J. Industrial Medicine*, Vol 13, p695.

- Goldsmith, J.R., 1992: "Incorporation of epidemiological findings into radiation protection standards Public". *Health Rev* 1991/92; 19: 19-34.
- Goldsmith, J.R., 1995: "Epidemiological Evidence of Radiofrequency Radiation (Microwave) Effects on Health in Military, Broadcasting, and Occupational Studies". *International Journal of Occupational and Environmental Health*, 1, pp 47-57, 1995.
- Goldsmith, J.R., 1998: "Epidemiologic evidence relevant to radar (microwave) effects". *Environmental Health Perspectives*, 105 (Suppl 6), Dec 1997, pp 1579-1587.
- Goldstein, L. and Sisko, Z., 1974: "A quantitative electro-encephalographic study of the acute effect of X-band microwaves in rabbits". In "Biological effects and health hazards of microwave radiation: Proceedings of an International Symposium". P. Czerski eds, Polish Medical Publishers, Warsaw.
- Graham, C., Cook, M.R., Cohen, H.D. and Gerkovich, M.M., 1994: "A dose response study of human exposure to 60Hz electric and magnetic fields". *Bioelectromagnetics* 15: 447-463.
- Graham, C., Cook, M.R., Sastre, A., Riffle, D.W. and Gerkovich, M.M., 2000: "Multi-night exposure to 60 Hz magnetic fields: effects on melatonin and its enzymatic metabolite". *J Pineal Res* 28(1): 1-8.
- Grayson, J.K., 1996: "Radiation Exposure, Socioeconomic Status, and Brain Tumour Risk in the US Air Force: A nested Case-Control Study". *American J. of Epidemiology*, 143 (5), 480-486.
- Grinstein, S., and Klip, A., 1989: "Calcium homeostasis and the activation of calcium channels in cells of the immune system". *Bulletin of the New York Academy of Medicine*, 65 (1), 69-79.
- Grundler, W., and Keilmann, F., 1978: "nonthermal effects of millimeter microwaves on yeast growth". *Z. Naturforsch*, 33C:15-22.
- Grundler, W., and Kaiser, F., 1992: "Experimental evidence for coherent excitations correlated with cell growth". *Nanobiology* 1: 163-176.
- Grundler, W., Kaiser, F., Keilmann, F., and Walleczek, J., 1992: "Mechanisms of electromagnetic interaction with cellular systems". *Naturwissenschaften*.
- Guy, A.W., Chou, C.K., Kunz, L.L., Crowley, J, and Krupp, J., 1985: "Effects of long-term low-level radiofrequency radiation exposure on rats. Vol 9. Summary. University of Washington, USAFSAM-TR-85-64.
- Hagmar, L., Brogger, A., Hansteen, I.L., et al. (1994): "Cancer risk in humans predicted by increased levels of chromosomal aberrations in lymphocytes: Nordic Study Group on the health risk of chromosome damage". *Cancer Research*, 54: 2919-2922.
- Haider, T., Knasmueller, S., Kundi, M, and Haider, M., 1994: "Clastogenic effects of radiofrequency radiation on chromosomes of *Tradescantia*". *Mutation Research*, 324:65-68.
- Hamburger, S., Logue, J.N., and Sternthal, P.M., 1983: "Occupational exposure to non-ionizing radiation and an association with heart disease: an exploratory study". *J Chronic Diseases*, Vol 36, pp 791-802.

- Haque, M.F., Aghabeighi, B., Wasil, M., Hodges, S. and Harris, M. 1994: "Oxygen free radicals in idiopathic facial pain". Bangladesh Med. Res. Council Bul., 20:104-116.
- Harrison, G.H., McCulloch, D., and Balcer-Kubiczek, E.K., 1985: "Far-field 2.45 GHz irradiation system for cellular monolayers in vitro." J. Microwave Power, 20: 145-151.
- Hayes, R.B., Morris Brown, L., Pottern, L.M., Gomez, M., Kardaun, J.W.P.F., Hoover, R.N., O'Connell, K.J., Sutsman, R.E. and Nasser, J., 1990: Occupational and Risk for Testicular Cancer: A Case Control Study. International Journal of Epidemiology, 19, No.4, pp 825-831, 1990.
- Heller, J.H., and Teixeira-Pinto, A.A., 1959: "A new physical method of creating chromosome aberrations". Nature, Vol 183, No. 4665, March 28, 1959, pp 905-906.
- Hocking, B. and Joyner, K., 1995: "Re: Miscarriages among Female Physical Therapists who report using radio- and microwave- frequency electromagnetic radiation." - A letter to the Editor, American J. of Epidemiology, 141 (3): 273-274.
- Hocking, B., Gordon, I.R., Grain, H.L., and Hatfield, G.E., 1996: "Cancer incidence and mortality and proximity to TV towers". Medical Journal of Australia, Vol 165, 2/16 December, pp 601-605.
- Hollenberg, P.F., 1992; FASEB J, 6:686-694.
- Holly, E.A., Aston, D.A., Ahn, D.K., and Smith A.H., 1995: "Intraocular Melanoma Linked to Occupations and Chemical Exposure". Epidemiology, 7(1):55-61.
- Joines, W.T. and Blackman, C.F., 1980: "Power density, field intensity and carrier frequency determinants of RF-energy-induced calcium ion efflux from brain tissue". Bioelectromagnetics, 1: 271-275.
- Joines, W.T. and Blackman, C.F., 1981: "Equalizing the electric field intensity within chick brain immersed in buffer solution at different carrier frequencies". Bioelectromagnetics, 2: 411-413.
- Joines, W.T., Blackman, C.F., and Hollis, M.A., 1981: "Broadening of the RF power-density window for calcium-ion efflux from brain tissue". IEEE Trans on Biomedical engineering, BME-28 (8), pp 568-573.
- Johnson, C.C., and Spitz, C.C., 1989: "Childhood nervous system tumors: an assessment of risk associated with parental operations involving use, repair or manufacture of electrical and electronic equipment". International J. of Epidemiology, Vol 18, p 756.
- Juutilainen, J., Stevens, R.G., Anderson, L.E., Hansen, N.H., Kilpelainen, M., Laitinen, J.T., Sobel, E. and Wilson, B.W., 2000: "Nocturnal 6-hydroxymelatonin sulphate excretion in female workers exposed to magnetic fields". J Pineal Res 28(2): 97-104.
- Kallen, B., Malmquist, G., and Moritz, U., 1982: "Delivery Outcome among Physio-therapists in Sweden: is Non-ionising Radiation a Fetal Hazard ? Archives of Environmental Health, 37(2): 81-84.
- Kalnins, T., Krizbergs, R., and Romancuks, A., 1996: "Measurement of the intensity of electromagnetic radiation from the Skrunda radio location station, Latvia". The Science of the Total Environment, Vol 180, pp 51-56.

- Karabakhtsian, R., Broude, N., Shalts, N., Kochlatyi, S., Goodman, R., Henderson, A.S., 1994: "Calcium is necessary in the cell response to EM fields". *FEBS Letters*; 349(1):1-6. JUL 25 1994.
- Karasek, M., Woldanska-Okonska, M., Czernicki, J., Zylinska, K. and Swietoslawski, J., 1998: "Chronic exposure to 2.9 mT, 40 Hz magnetic field reduces melatonin concentrations in humans". *J Pineal Research* 25(4): 240-244.
- Kizaki, H., Tadakuma, T., Odaka, C. et al. (1989): "Activation of a suicide process of thymocytes through DNA fragmentation by calcium ionosphere and phorbol esters." *J. Immunology*, 143: 1790-1799.
- Kolmodin-Hedman, B., Mild, K.H., Jonsson, E., Andersson, M-C., and Eriksson, A., 1988: "Health problems among operators of plastic welding machines and exposure to radiofrequency electromagnetic fields". *Ind. Arch. Occup. Environ. Health*, 60(4): 243-247.
- Kolodynski, A.A. and Kolodynska, V.V., 1996: "Motor and psychological functions of school children living in the area of the Skrunda Radio Location Station in Latvia". *The Science of the Total Environment*, Vol 180, pp 87-93.
- Kolomytkin, O., Kuznetsov, V., Yurinska, M., Zharikova, A., and Zharikov, S., 1994: "Response of brain receptor systems to microwave energy exposure". pp 195-206 in "On the nature of electromagnetic field interactions with biological systems", Ed Frey, A.H., Publ. R.G. Landes Co.
- Kondo, T., Arai, M., Kuwabara, G., Yoshii, G., and Kano, E., 1985: "Damage in DNA irradiated with 1.2 MHz ultrasound and its effect on template activity of DNA for RNA synthesis". *Radiation Research*, 104: 284-292.
- König, H.L., 1974: "Behavioural changes in human subjects associated with ELF electric fields". In "ELF and VLF electromagnetic field effects", M.A. Persinger Ed, Publ. Plenum Press, New York.
- Krause, D.N. and Dubocovich, M.L., 1990: "Regulatory sites in the melatonin system of mammals". *TINS*, Vol 13 (11): 464-470.
- Kurose, I., Higuchi, H., Kato, S., Miura, S. and Ishii, H. 1996: "Ethanol-induced oxidative stress in the liver". *Alcohol Clin. Exp. Res.*, 20(1 Suppl): 77A-85A.
- Lafon-Cazal, M., Culcasi, M., Gaven, F., Pietri, S. and Bockaert, J., 1993a: "Nitric oxide, superoxide and peroxynitrite: putative mediators of NMDA-induced cell death in cerebellar granule cells". *Neurophysiol.*, 32:1259-1266.
- Lafon-Cazal, M., Pietri, S., Culcasi, M. and Bockaert, J. 1993b: "NMDA-dependent superoxide production and neurotoxicity". *Nature*, 354:535-537.
- Lai, E.K., Crossley, C., Sridhar, R., Misra, H.P., Janzen, E.G. and McCay, P.B. 1986: "In vivo spin trapping of free radicals generated in brain, spleen, and liver during  $\gamma$ -radiation of mice". *Arch. Biochem. Biophys.*, 244:156-160.
- Lai, H. and Singh, N.P., 1995: "Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells". *Bioelectromagnetics*, Vol 16, pp 207-210, 1995.
- Lai, H. and Singh, N.P., 1996: "Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation". *Int. J. Radiation Biology*, 69 (4): 513-521.

- Lai, H. and Singh, N.P., 1997a: "Acute exposure increases to a 60 Hz Magnetic Field increases DNA strand breaks in rat brain cells". *Bioelectromagnetics*, Vol 18: 156-165.
- Lai, H., and Singh, N.P., 1997b: "Melatonin and N-tert-butyl-a-phenylnitron Block 60 Hz magnetic field-induced DNA single- and double-strands Breaks in Rat Brain Cells." *Journal of Pineal Research*, 22:152-162.
- Lai, H., and Singh, N.P., 1997c: "Melatonin and Spin-Trap compound Block Radiofrequency Electromagnetic Radiation-induced DNA Strands Breaks in Rat Brain Cells." *Bioelectromagnetics*, 18:446-454.
- Larsen, A.I., Olsen, J., and Svane, O., 1991: "Gender specific reproductive outcome and exposure to high frequency electromagnetic radiation among physiotherapists". *Scand. J. Work Environ. Health*, Vol.17, pp 324-329.
- Lawrence, A.F., and Adey, W.R., 1982: "Nonlinear wave mechanisms in interactions between excitable tissue and electromagnetic fields". *Neurological Research*, 4: 115-153.
- Lednev, V.V. 1995: "Comments on 'Clarification and application of ion parametric resonance model for magnetic field interactions with biological systems', by Blanchard and Blackman. *Bioelectromagnetics*, 16: 268-269.
- Lee GM, Neutra RR, Hristova L, Yost M, and Hiatt RA., 2002: "A nested case-control study of residential and personal magnetic field measures and miscarriages". *Epidemiology* 13(1): 21-31.
- Lemasters, J.L., Di Fuisseppi, J., Nieminen, A.L., Blebbing H.B., 1987: "Free calcium and mitochondrial membrane potential preceding cell death in hepatocytes". *Nature* , 325: 78-81.
- Lerchl, A., Nonaka, K.O., Stokkan, K.A. and Reiter, R.J., 1990: "Marked rapid alterations in nocturnal pineal serotonin metabolism in mice and rats exposed to weak intermittent magnetic fields." *Biochem Biophys Research Communications*, 169: 102-108.
- Lerchl, A., Nonaka, K.O. and Reiter, R.J., 1991: "Pineal gland 'magnetosensitivity' to static magnetic fields is a consequence of induced electric currents (eddy currents)". *J. Pineal Research*, 10: 109-116.
- Lester, J.R., and Moore, D.F., 1982a: "Cancer incidence and electromagnetic radiation". *Journal of Bioelectricity*, 1(1):59-76.
- Lester, J.R., and Moore, D.F., 1982b: "Cancer mortality and air force bases". *Journal of Bioelectricity*, 1(1):77-82.
- Lester, J.R., and Moore, D.F., 1985: "Reply to: Cancer mortality and air force bases, a reevaluation". *Journal of Bioelectricity*, 4(1):129-131.
- Levallois, P., Dumont, M., Touitou, Y., Gingras, S., Masse, B., Gauvin, D., Kroger, E., Bourdages, M. and Douville, P., 2001: "Effects of electric and magnetic fields from high-power lines on female urinary excretion of 6-sulfatoxymelatonin". *Am J Epidemiology* 154(7): 601-609.
- Levin, M. and Ernst, S.G., 1995: "Applied AC and DC magnetic fields cause alterations in mitotic cycle of early sea urchin embryos". *Bioelectromagnetics*, 16 (4): 231-240.

- Li DK, Odouli R, Wi S, Janevic T, Golditch I, Bracken TD, Senior R, Rankin R, and Iriye R., 2002: "A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of miscarriage". *Epidemiology* 13(1): 9-20.
- Liburdy, R.P., Sloma, T.R., and Yaswen, P., 1993: "ELF magnetic fields, breast cancer and melatonin: 60 Hz fields block melatonin's oncostatic action on ER+ breast cancer cell proliferation". *Journal of Pineal Research*, 14 (2): 89-97.
- Liboff, A.R., Rozak, R.J., Sherman, M.L., McLeod, B.R., and Smith, S.D., 1987: "Calcium-45 cyclotron resonance in human lymphocytes.", *J. Bioelectromagnetics*, 6: 13-22.
- Lilienfeld, A.M., Tonascia, J., and Tonascia S., Libauer, C.A., and Cauthen, G.M., 1978: "Foreign Service health status study - evaluation of health status of foreign service and other employees from selected eastern European posts". Final Report (Contract number 6025-619073) to the U.S. Dept of State, July 31, 1978.
- Lin, H., Goodman, R., and Shirley-Henderson, A., 1994: "Specific region of the c-myc promoter is responsive to electric and magnetic fields". *J Cell Biochemistry*, Mar; 54(3): 281-288.
- Lin, R.S., Dischinger, P.C., Conde, J., and Farrel, K.P., 1985: "Report on the relationship between the incidence of brain tumors and occupational electromagnetic exposure". *Journal of Occupational Medicine*, 27: 413-419.
- Lin-Liu, S. and Adey, W.R., 1982: "Low frequency amplitude modulated microwave fields change calcium efflux rates from synaptomes". *Bioelectromagnetics*, 3: 309-322.
- Lindstrom, E., Lindstrom, P., Berlund, A., Lundgren, E., and Mild, K.H., 1995: "Intracellular calcium oscillations in a T-cell line after exposure to extremely-low-frequency magnetic fields with variable frequencies and flux densities". *Bioelectromagnetics*, 16: 41-47.
- Liu, L.M., and Cleary S.F., 1995: "Absorbed energy distribution from radiofrequency electromagnetic radiation in a mammalian cell model: effect of membrane-bound water". *Bioelectromagnetics*, 16 : 160-171.
- Lindholm, M.L., Hietanen, M., Kyronen, P., Sallmen, M., von Nandelstadh, P., Taskinen, H., Pekkarinen, M., Ylikoski, M., and Hemminki, K., 1992: "Magnetic fields of video display terminals and spontaneous abortion". *Am. J. Epidemiology*, 136(9): 1041-1051.
- Lissonin, P., Viviani, S., Bajetta, E., Buzzoni, R., Barreca, A., Mauri, R., Resentini, M., Morabito, F., Esposti, D., Esposti, G., et al., 1986: "A clinical study of the pineal gland activity in oncologic patients." *Cancer*, 57(4): 837-842.
- Lotmar, R., Ranscht-Froemsdorff, W.R. and Weise, H., 1969: "Dampfung der Gewebeatmung (CO<sub>2</sub>) von Mauseleber durch kunstliche impulsstrahlung", *Int. J. Biometeorology*, 13(3&4), 231-238.
- Luben, R., 1995: "Statement of Dr Richard A Luben on the Biology and Biochemistry of EMR, including RF/MW", Planning Tribunal Hearing, Decision A 15/96.
- Lyle, D.B., Schechter, P., Adey, W.R. and Lundak, R./L., 1983: "Suppression of T lymphocyte cytotoxicity following exposure to sinusoidally amplitude-modulated fields". *Bioelectromagnetics*, 4: 281-292.
- Maes, A., Collier, M., Slaets, D., and Verschaeve, L., 1996: "954 MHz Microwaves enhance the mutagenic properties of Mitomycin C". *Environmental and Molecular Mutagenesis*, 28: 26-30.



- Maestroni, G.J., 1995: "T-helper-2 lymphocytes as Peripheral target of melatonin signalling". J. Pineal Research, 18: 84-89.
- Magone, I., 1996: "The effect of electromagnetic radiation from the Skrunda Radio Location Station on *Spirodela polyrhiza* (L.) *Schleiden* cultures". The Science of the Total Environment, Vol 180, pp 75-80.
- Mann, K., and Roschke, J., 1995: "Effects of pulsed high-frequency electromagnetic fields on human sleep". Neuropsychobiology, 33: 41-47.
- Mar, P.K., Kumar, A.P., Kang, D.C., Zhao, B., Martinez, L.A., Montgomery, R.L., Anderson, L., and Butler, A.P., 1995: "Characterization of novel phorbol ester- and serum-responsive sequences of the rat ornithine decarboxylase gene promoter." Molecular Carcinogenesis. 14(4):240-50.
- Maskarinec, G., and Cooper, J., 1993: "Investigation of a childhood leukemia cluster near low-frequency radio towers in Hawaii". SER Meeting, Keystone, Colorado, June 16-18, 1993. Am. J. Epidemiology, 138:666, 1993.
- Mattana, A., Bennardini, F., Usai, S., Fiori, P.L., Franconi, F. and Cappuccinelli, P., 1997: "Acanthamoeba castellanii metabolites increase the intracellular calcium level and cause cytotoxicity in wish cells." Microb. Pathog., Aug 23 (2):85-93 .
- McConkey, D.J., Hartzell, P., Duddy, S.K. et al. , 1988: "2,3,7,8-Tetrachlorodibenzo-p-dioxin kills immature thymocytes by  $\text{Ca}^{2+}$ -mediated endonuclease activation." Science, 242: 256-257.
- McCord, J.M. and Fridovich, I., 1978: "The biology and pathology of oxygen radicals". Ann. Intern. Med., 89:122-127.
- McGauchy, R., 1990: "Evaluation of the potential carcinogenicity of electromagnetic fields". U.S. E.P.A. external review draft EPA/600/6-90/005B, October 1990.
- McLauchlan, K, 1992: "Are environmental magnetic fields dangerous?" Physics World. pp 41-45.
- McRee, D.I., 1970: "Soviet and Eastern Research on Biological effects of Microwave Radiation"., Proc. of the IEEE, Vol. 68 (1), 84-91.
- Maunder, W.J., 1970: "The value of the weather". Publ. Methuen & Co Ltd, London, 388pp.
- Merrett, J.H., Chamness, A.F. and Allan, S.J., 1978: "Studies on the blood-brain barrier permeability after microwave radiation". Radiation Environmental Biophysics, 15: 367-377.
- Merritt, J.H., Shelton, W.W., and Chamness, A.F., 1982: "Attempt to alter  $\text{Ca-}^{45}$  binding to brain tissue with pulse-modulated microwave energy". Bioelectromagnetics, 3: 457-478.
- Michelozzi, P., Capon, A., Kirchmayer, U., Forastiere, F., Biggeri, A., Baraca, A. and Perucci, C.A., 2002: "Adult and childhood Leukaemia near a high-power radio station in Rome, Italy". Am J. Epidemiology 155(12): 1096-1103.
- Milhan, S., 1985: "Silent Keys", Lancet 1, 815, 1985.
- Milham, S., 1988: "Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies". Am. J. Epidemiology, Vol 127, No.1, pp 50-54.
- Miller, W.H., 1968: "Santa Ana winds and crime". Prof Geog. 20: 23-27.

- Mitchel, L.M., McRee, D.I., Peterson, N.J., Tilson, H.A., Shandala, M.G., Rudnev, M.I., Varetiskii, V.V., and Navakatikyan, M.I., 1989: "Results of a United States and Soviet Union Joint Project on Nervous System Effects of Microwave Radiation." *Environmental Health Perspectives*, 81: 201-209.
- Murphy, J.C., Kaden, D.A., Warren, J., and Sivak, A., 1993: "International Commission for Protection Against Environmental Mutagens and Carcinogens. Power frequency electric and magnetic fields: a review of genetic toxicology". *Mutation Research*, 296(3):221-40.
- Murray, A., and Hunt, T., 1993: "The cell cycle: an introduction". Publ. Oxford University Press, Oxford.
- Mustelin, T., Poso, P., Lapinjoki, S.P., Gynther, J., and Andersson, L.C., 1987: "Growth signal transduction: rapid activation of covalently bound ornithine decarboxylase during phosphatidylinositol breakdown". *Cell*, 49: 171-176.
- Nelson, P.G., 1966: "Interaction between spinal motoneurons of the cat." *J. Neurophysiology*, 29: 275-287.
- Nilsson, R., Hamnerius, Y., Mild, K.H., Hansson, H-A., Hjelmqvist, E., Olanders, S., and Persson, L.I., 1989: "Microwave effects on the central nervous system - a study of radar mechanics". *Health Physics*, Vol 56 (5), pp 777-779.
- Nordenson, I., Beckman, L., Liden, S. and Stjernberg, N., 1984: "Chromosomal aberrations and cancer risk". *Hum. Hered.*, 34(2):76-81.
- Nordenson, I., Mild, K.H., Andersson, G., and Sandstrom, M., 1994: "Chromosomal aberrations in human amniotic cells after intermittent exposure to 50 Hz magnetic fields". *Bioelectromagnetics* 15(4):293-301.
- Nuzzo, F. and Stefanini, M., 1989: "Monitoring of genetic instability in subjects with increased risk of cancer". *Ann Ist Super Sanita*, 25(1):81-89.
- NZPT, 1996: "New Zealand Planning Tribunal Decision: J.M. McIntyre and others vs BellSouth New Zealand", Decision A 15/96.
- Oliver, C.N., Starke-Reed, P.E., Stadtman, E.R., Liu, G.J., Carney, J.M. and Floyd, R.A., 1990: "Oxidative damage to brain proteins, loss of glutamine synthetase activity, and production of free radicals during ischemia/reperfusion-induced injury to gerbil brain". *Proc. Nat. Acad. Sci. (USA)*, 87:5144-5147.
- Orrenius, S., McCabe, M.J. and Nicotera, P., 1992: "Ca(2+)-dependent mechanisms of cytotoxicity and programmed cell death". *Toxicol. Lett.*, Dec; 64-65.
- Ouellet-Hellstrom, R. and Stewart, W.F., 1993: "Miscarriages among Female Physical Therapists who report using radio- and microwave- frequency electromagnetic radiation." *American J. of Epidemiology*, 138 (10): 775-86.
- Ouellet-Hellstrom, R. and Stewart, W.F., 1995: "Re: Miscarriages among Female Physical Therapists who report using radio- and microwave- frequency electromagnetic radiation." (Reply), *American J. of Epidemiology*, 141(3), p274.
- Owen, A.D., Schapira, A.H., Jenner, P. and Marsden, C.D., 1996: "Oxidative stress and Parkinson's disease". *Ann. NY. Acad. Sci.*, 786:217-223.

- PCFE, 1996: "Public Authority Planning for Cellphone Transmission Facilities", Office of the Parliamentary Commissioner for the Environment, P.O. Box 10-241, Wellington New Zealand, 31 pp.
- Phelan, A.M., Lange, D.G., Kues, H.A., and Luty, G.A., 1992: "Modification of membrane fluidity in Melanin-containing cells by low-level microwave radiation". *Bioelectromagnetics*, 13 : 131-146.
- Phillips, J.L., 1993: "Effects of electromagnetic field exposure on gene transcription". *J Cell Biochemistry*, Apr; 51(4): 381-386.
- Pflugger, D.M. and Minder, C.E., 1996: "Effects of 16.7 Hz magnetic fields on urinary 6-hydroxymelatonin sulfate excretion of Swiss railway workers". *J Pineal Research* 21(2): 91-100.
- Prausnitz, S. and Susskind, C., 1962: "Effects of chronic microwave irradiation on mice". *IRE Trans on Biomed. Elecron.* 9: 104-108.
- Preston-Martin, S., Mack, W., and Henderson, B.E., 1989: "Risk factors for gliomas and meningiomas in males Los Angeles County". *Cancer Research*, Vol 49, p 6137.
- Reite, M., Higgs, L., Lebet, J.P., Barbault, A., Rossel, C., Kuster, N., Dafni, U., Amato, D., and Pasche, B.: "Sleep inducing effect of low energy emission therapy". *Bioelectromagnetics*, 15: 67-75.
- Reiter, R., 1992: "Phenomena in Atmospheric and environmental electricity". *Developments in Atmospheric Science*, 20, Publ Elsevier, Amsterdam.
- Reiter, R.J., 1994: "Melatonin suppression by static and extremely low frequency electromagnetic fields: relationship to the reported increased incidence of cancer". *Reviews on Environmental Health*. 10(3-4):171-86, 1994.
- Reiter, R., 1995: "Oxidative processes and antioxidative defense mechanisms in the aging brain". *FASEB J.*, 9:526-533.
- Reiter, R.J., Melchiorri, D., Sewerynek, E., Poeggeler, B., Barlow-Waiden, L., Chuang, J., Ortiz, G.G. and Acuna-Castroviejo, D., 1995: "A review of the evidence supporting melatonin's role as an antioxidant". *J.Pineal Res.*, 18:1 -11.
- Reiter, R.J., and Robinson, J., 1995: "Melatonin: your body's natural wonder drug". Bantam Books, New York.
- Repacholi, M.H., 1995: "Statement of Dr Michael Repacholi, New Zealand Planning Tribunal Hearing, Decision A 15/96, Christchurch, November 1995.
- Repacholi, M.H., Basten, A., Gebiski, V., Noonan, D., Finnie, JH., and Harris, A.W., 1997: "Lymphomas in E $\mu$ -*Pim1* Transgenic Mice Exposed to Pulsed 900 MHz Electromagnetic Fields". *Radiation Research*, 147:631-640.
- Rodriguez-del Valle, N., and Rodriguez-Medina, J.R., 1993: "Calcium stimulates molecular and cellular events during the yeast-to-mycelium transition in *Sporothrix schenckii*". *J. Med. Vet. Mycol*, 31(1): 43-53.
- Rosen, L.A., Barber, I. and Lyle D.B., 1998: "A 0.5 G, 60 HZ magnetic field suppresses melatonin production in pinealocytes". *Bioelectromagnetics* 19: 123-127.

- Roubinette, C.D., and Silverman, C., 1977: "Causes of death following occupational exposure to microwave radiation (radar) 1950-1974." Rockville, U.S. Dept of Health, Education and Welfare, pp 338-344 (US DHEW Publication (FDA) 77-8026).
- Robinette, C.D., Silverman, C. and Jablon, S., 1980: "Effects upon health of occupational exposure to microwave radiation (radar)". American Journal of Epidemiology, 112(1):39-53, 1980.
- Rowley, R., 1990: "Repair of radiation-induced chromatid aberrations: relationship to G2 arrest in CHO cells". International Journal of Radiation Biology, 58(3):489-98.
- Sagripani, J. and Swicord, M.L., 1976: DNA structural changes caused by microwave radiation. Int. J. of Rad. Bio., 1(1), pp 47-50, 1976.
- Sait, M.L., Wood, A.W., Armstrong, S.M., and Martin, M.J., 1997: "Are human plasma melatonin profiles delayed in some individuals in response to 50 Hz magnetic field exposure ?" A-44
- Sandyk, R., Anastasiadis, P.G., Anninos, P.A., and Tsagas, N., 1992: "The pineal gland and spontaneous abortions: implications for therapy with melatonin and magnetic field." International Journal of Neuroscience. 62(3-4):243-50, 1992.
- Sarkar, S., Sher, A., and Behari, J., 1994: "Effect of low power microwave on the mouse genome: A direct DNA analysis". Mutation Research, 320: 141-147.
- Savitz, D.A., and Chen, J., 1990: "Parental occupation and childhood cancer: Review of epidemiological studies". Environmental Health Perspectives, 88: 325-337.
- Schwan, H.P., 1969: "Interaction of microwave and radiofrequency radiation with biological systems". Proc. Symposium on Biological effects and health implications of microwave radiation, Richmond, VA.
- Schwan, H.P. and Foster, K.R., 1980: "RF-field interactions with biological systems: electrical properties and biophysical mechanisms". Proc. of the IEEE, 68(1): 104-113.
- Schwartz, J.L., House, D.E., and Mealing, A.R., 1990: "Exposure of frog hearts to CW or amplitude modulated VHF fields: selective efflux of calcium ions at 16 Hz." Bioelectromagnetics, 11: 349-358.
- Selga, T. and Selga, M., 1996: "Response of *Pinus sylvestris* (L.) needles to electromagnetic fields. Cytological and ultrastructural aspects". The Science of the Total Environment, Vol 180, pp 65-74.
- Sen, S., Goldman, H., Morenhead, M., Murphy, S. and Phillis, I.W., 1994: " $\alpha$ -phenyl-tert-butyl-nitrone inhibits free radical release in brain concussion". Free. Rad. Biol. Med., 16:685-691.
- Servantie, B., Servantie, A.M., and Etienne, J., 1975: "Synchronization of cortical neurons by a pulsed microwave field as evidenced by spectral analysis of electrocorticograms from the white rat". Ann. N.Y. Acad. Sci., 247: 82-86.
- Shandala, M.G., Dumanskii, U.D., Rudnev, M.I., Ershova, L.K., and Los I.P., 1979: "Study of Non-ionising Microwave Radiation Effects on the Central Nervous System and Behavior Reactions". Environmental Health Perspectives, 30:115-121.
- Shandala, M.G., and Zvinyatskonsky, Y.A., 1988: "Environment and health of the population", Kiev, Zdorovja, p150 (in Russian).

- Shelton, W.W., and Merritt, J.H., 1981: "In vitro study of microwave effects on calcium efflux in rat brain tissue". *Bioelectromagnetics*, 2: 161-167.
- Sheppard, A.R., Bawin, S.M., and Adey, W.R., 1979: "Models of long-range order in cerebral macromolecules: effect of sub-ELF and modulated VHF and UHF fields". *Radio Science*, 14 (6S): 141-145.
- Shore, M. (Ed), 1981: "Environmental Health Criteria 16: Radiofrequency and Microwaves", World Health Organization, Geneva, 1981.
- Sibbison, J.B., 1990: "USA: Danger from electromagnetic fields". *The Lancet*, July 14, 1990, p106.
- Silverman, C., 1979: "Epidemiologic approach to the study of microwave effects". *Bull. N.Y. Acad. Med.*, 55(11):1166-1181, December 1979.
- Skyberg, K., Hansteen, I.L., and Vistnes, A.I., 1993: "Chromosome aberrations in lymphocytes of high-voltage laboratory cable splicers exposed to electromagnetic fields". *Scandinavian Journal of Work, Environment & Health*.19(1):29-34.
- Snyder, S.H., 1985: "The molecular basis of communication between cells". *Scientific American*, 253 (4), (Oct) pp 132-144.
- Sohal, R.S. and Weindruch, R., 1996: "Oxidative stress, caloric restriction, and aging" *Science*, 273:59-63.
- Sorsa, M., Ojajarvi, A. and Salomaa, S., 1990: "Cytogenetic surveillance of workers exposed to genotoxic chemicals: preliminary experiences from a prospective cancer study in a cytogenetic cohort". *Teratog. Carcinog. Mutagen*, 10(3):215-221.
- Sorsa, M., Wilbourn, J. and Vainio, H., 1992: "Human cytogenetic damage as a predictor of cancer risk". *I.A.R.C. Sci. Publ.*, 116:543-554 .
- Speers, M.A., Dobbins, J.G., and Miller, V.S., 1988: "Occupational exposures and brain cancer mortality: a preliminary study of East Texas Residents". *American Journal of Industrial Medicine*, 13:629-638.
- Stark, K.D.C., Krebs, T., Altpeter, E., Manz, B., Griol, C. and Abelin, T., 1997: "Absence of chronic effect of exposure to short-wave radio broadcast signal on salivary melatonin concentrations in dairy cattle". *J Pineal Research* 22: 171-176.
- Stein, G.S., and Lian, J.B., 1992: "Regulation of cell cycle and growth control". *Bioelectromagnetics*, Suppl. 1: 247-265.
- Strahler, A.N., 1963: "The Earth Sciences", 2nd Edition, Publ. Harper and Row, New York.
- Stryer, L., 1986: "Cyclic AMP cascade of vision". *Annual review of Neurosciences*, 9: 87-119.
- Stuchly, M.A., and Stuchly, S.S., 1990: "Electrical properties of biological substances", pp75-112, In "Biological effects and medical applications of electromagnetic energy"., Ed. Om P. Gandhi, Prentice Hall, New Jersey.
- Sumiyoshi, H., Baer, A.R., and Wargovich, M.J., 1991: "Heterogenicity of ornithine decarboxylase during mouse colon carcinogenesis and in human colon tumors". *Cancer Research*, 51: 2069-2072.

- Suvorov, N.B., Boitsova, V.V., Medvedeva, M.V., Bogdanov, O.V., and Vasilevskii, N.N., 1994: "The biological action of physical factors in the critical periods of embryogenesis". *Zhurnal Evoliutsionnoi Biokhimii i Fiziologii*, 30(6):762-768.
- Szmigielski, S., Szudzinski, A., Pietraszek, a. et al., 1982: "Accelerated development of spontaneous and benzopyrene induced skin cancer in mice exposed to 2450 MHz microwave radiation." *Bioelectromagnetics*, 3: 179-191.
- Szmigielski, S., Bielec, M., Lipski, S., and Sokolska, G., 1988: "Immunological and cancer-related aspects of exposure to low level microwave and radiofrequency fields". In Marino (Ed), 'Modern Bioelectricity', Marcel Bekker, N.Y., pp 861-925.
- Szmigielski, S., 1996: "Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation". *Science of the Total Environment*, Vol 180, 1996, pp 9-17.
- Taskinen, H., Kyyronen, P., and Hemminki, K., 1990: "Effects of ultrasound, shortwaves and physical exertion on pregnancy outcome in physiotherapists". *J. of Epidemiology and Community Health*, 44:196-210.
- Taylor, E.M., and Ashleman, B.T., 1975: "Some effects of electromagnetic radiation on the brain and spinal cord of cats". *Ann. N.Y. Acad. Sci.*, 247: 65-73.
- Tell, R.A., and Mantiply, E.D., 1980: "Population exposure to VHF and UHF broadcast radiation in the United States". *Proc IEEE*, Vol.68, No.1, January 1980. pp 4-12.
- Thomas, T.L., Stolley, P.D., Stemhagen, A., Fontham, E.T.H., Bleecker, M.L., Stewart, P.A., and Hoover, R.N., 1987: "Brain tumor mortality risk among men with electrical and electronic jobs: A case-control study". *J. Nat. Canc. Inst.*, Vol 79, No.2, pp 233-238., August 1987.
- Timchenko, O.I., and Ianchevskaia, N.V., 1995: "The cytogenetic action of electromagnetic fields in the short-wave range". *Psychopharmacology Series*, Jul-Aug;(7-8):37-9.
- Tornqvist, S., Knave, B., Ahlbom, A., and Persson, T., 1991: "Incidence of leukaemia and brain tumours in some 'electrical occupations'". *British Journal of Industrial Medicine*, 48: 597-603.
- Ullrich, A., Coussens, L., Hayflick, J.S., Dull, T.J., Gray, A., Tam, A.W., Lee, J., Yarden, Y., Whittle, N., Waterfield, M.D. and Seeburg, P.H., 1985: "Human epidermal growth factor receptor cDNA sequence and aberrant expression of the amplified gene in A431 epidermoid carcinoma cells". *Nature*, 309:428-.
- Vagero, D., Ahlbom, a., Olin, R., and Sahlsten, S., 1984: "Cancer morbidity among workers in the telecommunications industry". *British Journal of Industrial Medicine*, Vol 42, pp 191-195.
- Valentino, A.R., 1972: "A small ELF electric field probe". *Proc. IEEE Int. Symp. Electromagnetic Compatibility*, p265.
- Valjus, J., Norppa, H., Jarventaus, H., Sorsa, M., Nykyri, E., Salomaa, S., Jarvinen, P., and Kajander, J., 1993: "Analysis of chromosomal aberrations, sister chromatid exchanges and micronuclei among power linesmen with long-term exposure to 50-Hz electromagnetic fields". *Radiation & Environmental Biophysics*, 32(4):325-36.
- Verkasalo, P.K., Kaprio, J., Varjonen, J., Romanov, K., Heikkila, K and Koskenvuo, M., 1997: "Magnetic fields of transmission lines and depression." *American J.Epidemiology*, Dec 15;146(12):1037-1045.

- Vijayalaxmi, B.Z., Reiter, R.J., Sewerynek, E., Meltz, M.L., and Poeggeler, B., 1995: "Melatonin protects human blood lymphocytes from radiation-induced damage". *Mutation Research*, 346(1): 23-31.
- Vijayalaxmi, B.Z., Frei, M.R., Dusch, S.J., Guel, V., Meltz, M.L., and Jauchem, J.R., 1997: "Frequency of Micronuclei in the peripheral blood and bone marrow of cancer-prone mice chronically exposed to 2450 Mhz radiofrequency radiation". *Radiation Research*, 147: 495-500.
- Von Klitzing, L., 1995: "Low frequency pulsed electromagnetic fields influence EEG of man". *Physica Medica XI* (2) April-June 1995, pp77-80.
- Vorobyov, V.V., Galchenko, A.A., Kukushkin, N.I., and Akoev, I.G., 1997: "Effects of weak microwave fields amplitude modulated at ELF on EEG of symmetric brain areas in rats". *Bioelectromagnetics*, 18:293-298.
- Vorst, A.V. and Duhamel, F., 1996: "1990-1995 Advances in investigating the interaction of microwave fields with the nervous system". *IEEE Trans. on Microwave Theory and Techniques*, 44(10),1898-1909.
- Wachsman, J.T., 1996: "The beneficial effects of dietary restriction: Reduced oxidative damage and enhanced apoptosis". *Mutation Research*, 350:25-34.
- Walleczek, J., 1992: "Electromagnetic field effects on cells of the immune system: the role of calcium signaling". *FASEB J.*, 6: 3176-3185.
- Walleczek, J. and Budinger, T.F., 1992: "Pulsed magnetic field effects on calcium signaling in lymphocytes: dependence on cell status and field intensity". *FEBS* 11896, 314 (3): 351-355.
- Wang, S.G. 1989: "5-HT contents change in peripheral blood of workers exposed to microwave and high frequency radiation". *Chung Hua Yu Fang I Hsueh Tsa Chih* 23(4): 207-210.
- Weinstein, I.B., 1988: "The origins of human cancer: molecular mechanisms of carcinogenesis and their implications for cancer prevention and treatment". *Cancer Research*, 48: 4135-4143.
- Weinstein, I.B., 1991: "Non-mutagenic Mechanisms in Carcinogenesis: Role of Protein Kinase C in Signal transduction and Growth Control"., *Environmental Health Perspectives*, 93: 175-179.
- Weaver, J.C., and Astumian, R.D., 1990: "The response of living cells to very weak electric fields: the thermal noise limit." *Science*, 247 (26 Jan 1990): 459-462.
- Wever, R., 1969: "Untersuchungen zur circadianen Periodik des Menschen mit besonderer Beruchsichtigung des Einflusses schwacher electrisscher Wechselfelder". *Bundesminst. f. wiss. Forschg., Forschungsbericht, W 69-21*, 212 pp.
- Wever, R., 1970: "The effects of electric fields on the circadian rhythmicity in men". *Life Sci. Space Res.*, 8: 177-187.
- Wever, R., 1974: "ELF-effects on Human Circadian Rhythms", pp 101-144 in "ELF and VLF Electromagnetic Field Effects", Ed. M.A. Persinger, Publ. Plenum Press, New York.

- Welker, H.A., Semm, P., Willig, R.P., Commentz, J.C., Wiltchko, W. and Vollrath, L., 1983: "Effects of an artificial magnetic field on serotonin N-acetyltransferase activity and melatonin content of the rat pineal gland." *Exp. Brain Res.*, 50(2-3):426-432 .
- Whitfield, J.F., Boynton, A.L., MacManus, J.P., Sikorska, M., and Tsang, B.K., 1979: "The regulation of cell proliferation by calcium and cyclic AMP". *Mol. Cell Biochem.*, Nov 1;27(3):155-179 .
- WHO, 1993: "Environmental Health Criteria 137: Electromagnetic fields (300 Hz to 300 GHz)". Ed. M. Repacholi, World Health Organization, Geneva, 1993.
- Wilson, B.W., Chess, E.K., and Anderson, L.E., 1986: "60 Hz electric field effects on pineal melatonin rhythms: time course and onset of recovery". *Bioelectromagnetics*, 7: 239-242.
- Wilson, B.W., Wright, C.W., Morris, J.E., Buschbom, R.L., Brown, D.P., Miller, D.L., Sommers-Flannigan, R. and Anderson, L.E., 1990: "Evidence of an effect of ELF electromagnetic fields on human pineal gland function". *J Pineal Research* 9(4): 259-269.
- Wood, A.W., Armstrong, S.M., Sait, M.L., Devine, L. and Martin, M.J., 1998: "Changes in human plasma melatonin profiles in response to 50 Hz magnetic field exposure". *J Pineal Research* 25(2): 116-127.
- Yoshida, M., Hayashi, H., Taira, M., and Isono, K., 1992: "Elevated expression of the ornithine decarboxylase gene in human esophageal cancer". *Cancer Research*, 52: 6671-6675.
- Zhao, Q., Pahlmark, K., Smith, M-L., and Siesjo, B.K., 1994: "Delayed treatment with the spin-trap  $\alpha$ -phenyl-N-tert-butyl nitron (PBN) reduces infarct size following transient middle cerebral artery occlusion in rats. *Acta. Physiol. Scand.*, 152:349-350.
- Zyss, T., Dobrowolski, J.W. and Krawczyk, K., 1997: "Neurotic disturbances, depression and anxiety disorders in the population living in the vicinity of overhead high-voltage transmission line 400 kV. Epidemiological pilot study". [Article in Polish]. *Med. Pr.*, 48(5):495-505.