

# CRITICISM OF THE PROPOSAL TO ADOPT THE ICNIRP GUIDELINES FOR CELLSITES IN NEW ZEALAND

## ICNIRP GUIDELINE CRITIQUE

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It is proposed to make the New Zealand Standard and Guideline for Cell Site public exposures of Radiofrequency and Microwave Radiation guidelines based on the International Commission on Non-Ionizing Radiation Protection (ICNIRP) guideline.

The ICNIRP guidelines and scientific assessment is published in Health Physics, Vol. 74 (4), p 494-522. This is the primary source document for this critique and will be referred to as ICNIRP 1998.

The ICNIRP assessment of effects, ICNIRP (1998) has been review and found to be seriously and fatally flawed, with a consistent pattern of bias, major mistakes, omissions and deliberate misrepresentations. Adopting it fails to protect public health from known potential and actual health effects and hence is unlawful according to the requirements of the Resource Management Act. Public health protection should be the objective of this process and this should be based on the identification of the Lowest Observed Adverse Effect Level, (LOAEL) and a reasonable safety factor to take into account the uncertainties and vulnerable members of the community.

Epidemiology currently identifies the LOAEL for RF/MW as  $0.06\mu\text{W}/\text{cm}^2$  for cancer and an reproductive effects, and  $0.0004\mu\text{W}/\text{cm}^2$  ( $0.4\text{nW}/\text{cm}^2$ ) for sleep disruption, learning impairment and immune systems suppression, for example. Hence the scientifically identified LOAEL is lower than the majority of the New Zealand population is currently exposed.

**Since background RF/MW levels in New Zealand cities are already in the range  $1\text{nW}/\text{cm}^2$  -  $3\text{nW}/\text{cm}^2$ , the only practical option to avoid these demonstrated effects is to set the initial public exposure limit at**

**$50\text{ nW}/\text{cm}^2$  ( $0.05\text{mW}/\text{cm}^2$ )**

**with the aim of reducing it to**

**$10\text{ nW}/\text{cm}^2$  ( $0.01\text{mW}/\text{cm}^2$ ) in 10 years.**

The ICNIRP guideline is based on the frequently stated claim that there are no adverse health effects unless a person is heated by more than  $1^\circ\text{C}$ , setting a level at which adverse effects can be avoided between 4 and 8 W/kg. This claim has been repeated in many statements and documents of ICNIRP, IRPA, WHO, NRL, ARL, and NRPB. It has also been stated publicly by the leaders of these bodies. A leading proponent of this position is Dr Michael Repacholi, WHO official and former chairperson of ICNIRP, IRPA

and the Australasian RF standards committee. Dr Michael Repacholi has expressed this view on TV, radio and in the press, in ICNIRP, IRPA and WHO reviews and in sworn evidence in a Planning Tribunal hearing in Christchurch in 1995. This is also the position taken by the staff of the National Radiation Laboratory (NRL) of the New Zealand Ministry of Health, the Australian Radiation Laboratory (ARL), the National Radiological Protection Board of the U.K. (NRPB).

This stands in strong contrast to the epidemiological and laboratory evidence given here and with the summary statement provided by one of the world's leading and most experienced, most scientifically published and respected EMR researchers, Dr William Ross Adey. The following is the abstract from his paper "Frequency and Power Windowing in Tissue Interactions with Weak Electromagnetic Fields": (Proc. IEEE 1980)

**"Abstract: Effects of non-ionizing electromagnetic (EM) fields that raise tissue temperature in general differ very little from effects of hyperthermia induced by other means. However, fields raising tissue temperature orders of magnitude less than 0.1C may result in major physiological changes not attributable to raised temperature *per se*. These weak fields have been observed to produce chemical, physiological, and behavioral changes only within windows in frequency and incident energy. For brain tissue, a maximum sensitivity occurs between 6 and 20 Hz. Two different intensity windows have been seen, one for ELF tissue gradients around  $10^{-7}$  V/m, and one for amplitude modulated RF and microwave gradients around  $10^{-1}$  V/m. The former is the level associated with navigation and prey detection in marine vertebrates and with the control of human biological rhythms; the latter is the level of the electroencephalogram (EEG) in the brain tissue. Coupling to living cells appears to require *amplifying* mechanisms that may be based on non-equilibrium processes, with long-range resonant molecular interactions. The *cooperative* processes are now recognized as important in immune and hormonal responses, as well as in nerve excitation. Polyanionic proteinaceous material forming a sheet on the cell membrane surfaces appears to be the site of detection of these weak molecular and neuroelectric stimuli."**

Professor Adey succinctly summarizes EMR research at that time. He does not claim, in the body of the paper, that there are only two intensity windows but that these are intensity windows that have repeatedly been shown to have significant effects. The paper contains evidence of other windows for ELF induced calcium ion efflux in chick and cat brains, e.g. 5, 10, 56 and 100 V/m, and other microwave intensity windows for calcium ion influx and efflux. This (Figure 4) shows significant biological effects at 0.1 and 1 mW/cm<sup>2</sup>.

Adey (1979) reviews a large body of research on the neurophysiologic effects of RF/MW radiation. This included the human biometeorological research on circadian rhythms in human subjects isolated from sunlight and EMR; their own work on altered monkey behaviour with a tissue gradient of  $10^{-7}$  V/m and other animal behaviour experiments. It also covered cellular evidence including calcium ion flux experiments on cats and chick brains. These show that ionic changes in amplitude modulated RF/MW fields are much

more related to modulation frequency than intensity of signal. Often higher effects are seen at lower exposure intensities than some higher intensities - in windows.

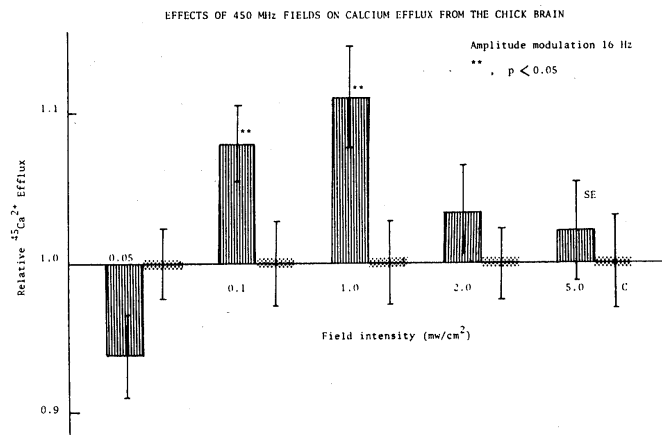


Fig. 4. Effects of changing intensity of 450-MHz field, amplitude modulated at 16 Hz, on efflux of <sup>45</sup>Ca<sup>2+</sup> from chick cerebral hemispheres. Cross-hatched bars show levels of efflux from exposed specimens in relation to control specimens (striped bars) tested simultaneously in the same experiments. Variance shown as SEM's. \*\*, p < 0.05 [27].

In great frustration at the intransigent position held by scientists who doggedly claim that there is only evidence of thermal effects, Professor Adey concludes:

**"Faced with the overwhelming complexity of the brain as a tissue and as the 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."**

**"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 interections may at last be understood in terms of transductive coupling at the molecular level."**

Dr Adey was basing his insights on a fascination with discovering how neurological tissue operated and altered in extremely low level RF/MW and ELF fields. Biochemists have now confirmed that RF/MW alters signal transduction, (e.g. Luben (1995), Byus (1994)), alters melatonin and damages the immune system, as will be shown below.

There is a wealth of laboratory evidence of cellular and animal changes at extremely low exposure levels to RF/MW radiation, accompanied by a massive body of epidemiological research which shows adverse health effects in human beings down to extremely low life-time mean exposure levels for chronic exposures. There is much more than Dr Adey had in 1979/80. It is simply not scientifically credible to claim that there are no established non-thermal effects and hence a public exposure standard that protects against warming by 1°C is adequate and should be adopted as a guideline in New Zealand.

The scientific evidence in relation to the requirements of the Resource Management Act 1991, makes it unlawful to adopt the ICNIRP guideline.

Professor John Goldsmith, as one of the world's leading epidemiologists was invited by the editor to provide a significant review paper to help to launch a new scientific journal, the International Journal of Occupational and Environmental Health. A couple of decades earlier Dr Goldsmith has been invited to be the opening key note speaker of the first conference of the newly formed International Society for Environmental Epidemiology. This illustrates the high standing with which he is held in the international epidemiological and public health community. The review, headed "Special Contributions" was carefully identified by Dr Goldsmith as an "opinion piece" which reviews and summarizes the "Epidemiologic Evidence of Radiofrequency Radiation (Microwave) Effects on Health in Military, Broadcasting and Occupational Studies".

This is a very relevant review for this assessment of what guideline level to choose. A member of the M.O.H./M.F.E. staff team in this process is a member of the National Radiation Laboratory, Mr Martin Gledhill. Mr Gledhill and Dr Andrew MacEwan were warmly praised and thanked for their large and dominant contributions to the recent Royal Society report "Radiation and the New Zealand Community - A scientific Overview".

The Royal Society report contains all of the omissions, biases and errors shown below in the preparation of the ICNIRP guideline and the WHO/UNEP/IRPA review. It takes the thermal view and at one key point makes the claim in relation to radiofrequency/microwave radiation, p67:

**"Some questions have been raised with respect to possible adverse effects of electric and magnetic fields, particularly those at low frequencies, in connection with high voltage lines, computer terminals, domestic appliances and wiring. However, no effects due to occupational exposure have been reported, nor are there any indications of adverse health effects on humans, other than from spark discharges and shock from direct contact."**

While this paragraph is mainly about ELF fields, it immediately follows the statement on RF/MW that only acknowledges a probable effect from a faulty microwave oven.

However, to claim in this alleged credible and high quality scientific report that no effects have been reported from occupational exposure and that there aren't any indications of adverse health effects on humans, is so grossly wrong, misleading and dishonest, that it puts this report's credibility and that of the Royal Society, seriously at risk.

There are hundreds of occupational studies showing significant adverse effects from ELF exposures, as well as scores of residential studies showing adverse effects on humans. In relation to RF/MW, a large number of such studies are reviewed by Dr Goldsmith's 1995 paper.

At the conclusion of the review, which covers statistically significant evidence of cancer and reproductive effects in exposed populations, as well as alterations in blood immune

factors and chromosome aberrations in RF/MW exposed people, Dr Goldsmith states in part:

**"There are strong political and economic reasons for wanting here to be no health effect from RF/MW exposure, as there are strong public health reasons for more accurately portraying the risks. Those of us who intend to speak for public health must be ready for opposition that is nominally but not truly, scientific."**

Dr Goldsmith's conclusion is exactly the same one I have come to in reviewing the ICNIRP assessment of effects. The position of the Ministry of Health as presented by the National Radiation Laboratory is scientifically flawed and shown to be biased and political, not based on public health protection. The Ministry for the Environment and the Ministry of Health should be above the influence of industry and its consultants, but in recommending the adoption of the ICNIRP guidelines, guidelines supported by industry around the world, will only favour putting more and more of the public at serious health risk.

It is easy to make strong and general dismissive and critical statements. The ICNIRP statement does this all the time. It is more difficult, and much more time consuming to carefully consider each claim and every paper cited in making those claims. I have done this in relation to the ICNIRP assessment of human reproduction and cancer evidence, and, to a lesser extent, to animal and laboratory evidence of RF/MW effects. This is set out below.

I show clearly and conclusively that there is a bias against finding and acknowledging adverse effects to the extent that most of the available scientific studies which show effects are ignored, the ones chosen are largely misrepresented, misinterpreted and mis-used.

A reductionist approach is taken rather than a comprehensive, integrative approach which is warranted by the nature and significance of the issues. It systematically dismisses individual papers:

- claiming papers don't show effects, when they do.
- claiming papers show no evidence of effects when they are not purporting to assess the effect under consideration.
- claiming papers don't show significant effects when they clearly do, and
- dismissing papers which show significant effects using incorrect, inappropriate and unjustified reasons.

A small number of studies are cited and reviewed, out of a large set of available material which shows potential, probable, taken together, actual adverse health effects. Whole bodies of research and the research results of complete disciplines, e.g. biometeorology, is totally ignored.

**This happens so consistently, systematically, demonstrably and blatantly that we can only conclude there is an unscientific motive behind the assessment and its conclusions.**

The guideline adopted in New Zealand must be based on an objective and independent assessment of the science, and epidemiological evidence, which is extremely strong and consistent, and not a simple adoption of a flawed and scientifically and legally challengeable approach and exposure level.

**ICNIRP Guideline seriously flawed and unlawful:**

The ICNIRP guideline should not be used as the New Zealand guideline or standard for three very important reasons. The use of the ICNIRP guideline is unlawful in New Zealand. It is grossly inappropriate for public health protection. It is scientifically challengeable because it is based on serious errors and omissions.

The ICNIRP guideline is unlawful since the ICNIRP assessment is based established and proven effects whereas the New Zealand law RMA (1991) is based on potential effects and cumulative effects, "regardless of scale, intensity, duration or frequency." Everybody in New Zealand is cumulatively exposed to electromagnetic radiation from power sources, appliances, cordless and cell phones, radio and TV stations and cell sites. Hence cell site radiation is a cumulate addition exposure in addition to all other exposures, and hence must be dealt with under Section 5(2)(c) of the RMA regardless of the level of exposure. It cannot be ignored claiming a "de minimus" level.

Public health protection, as outlined by Bradford-Hill (1965) and Goldsmith (1992), is should be based on epidemiological studies which show statistically significant results. Statistical significance is defined in terms of  $p=0.05$  and a 95% confidence interval. For a disease agent to which almost every person is exposed, a lower level of evidence is used as a threshold because of the importance and impact of the effect. Often in such cases an elevated Risk Ratio which lacks significance is sufficient for avoidance to be required. The ICNIRP guideline is not based on this approach and therefore fails to protect public health.

Goldsmith (1997) states:

**“To this day, the ICNIRP makes little use of epidemiological data, alleging that it is inconsistent and difficult to understand.”**

Professor Goldsmith, one of the world’s leading and most respected epidemiologists, then outlines detailed criticisms of the ICNIRP use of studies which are promoted to claim no effects are possible from RF/MW when the data in these studies actually does show significant adverse health effects.

The scientific assessment on which the ICNIRP guideline is based, contains major errors of scientific fact, research interpretation as well as taking the flawed approach to public health protection outlined above. Errors made in previous reviews, such as the UNEP/WHO/IRPA (1993) and NRPB (1991) reviews, are propagated through into the ICNIRP (1998) assessment through uncritical assessment. A small number of studies are

directly cited. In almost all cases the conclusions drawn are scientifically incorrect. This leads to the wrong conclusions and recommendations.

A major omission in the consideration of the effects of EMR on people is the results of the extensive research carried out by biometeorologists. Biometeorologists have identified many alterations in human conditions which are statistically significantly related to variations in naturally occurring electromagnetic fields. These results show conclusively that birds, mammals and people respond to extremely low and subtle changes in ELF and modulated RF field changes brought about by solar activity and the weather.

Public health protection is properly based on public health research from epidemiological studies. Many epidemiological studies show many statistically significant adverse health effects at levels of exposure to RF/MW which are hundreds to thousands of times lower than the proposed guideline of 200  $\mu\text{W}/\text{cm}^2$ . Under New Zealand law, the Resource Management Act, there is a legal requirement to “avoid, remedy or mitigate any adverse effects of an activity on the environment”. The environment includes the health and safety aspects of people and communities. An effect includes “any actual or potential effect”, any cumulative effect, “regardless of scale, intensity, duration or frequency”, and “includes any potential effect of high probability” and “any potential effect of low probability which has a high potential impact”.

### **Chemical Comparison:**

There are standard techniques for assessing the carcinogenicity of chemical substances, involving cell line studies, laboratory animal studies and human epidemiology. If EMR was treated in the same way it would have been declared a human carcinogen many years ago. EMR neoplastically transforms cell, causes cancer in mice, is found to increase cancer in exposed electrical workers and military personnel and in residential populations.

Chemical health risks are usually investigated around a single disease outcome, such as a particular kind of cancer. It may be a single form of leukaemia.

Once epidemiological studies find statistically significant increases in cancer from chemicals at a given mean concentration, safety factors of 1 to 10,000 are applied. The size of the safety factor depends on the nature of the critical effect and the size of the exposed population, Royal Commission on Environmental Pollution (U.K.), 21st Report, “Setting Environmental Standards”, cited at Houghton (1998).

### **Benzene as an Example: (from Houghton (1998))**

Benzene is classified as a genotoxic carcinogen which is primarily associated with non-lymphatic leukaemia. Benzene was shown to be a genotoxic carcinogen *In Vitro*, i.e. in cells in a test tube or a petri dish. It produced certain types of leukaemia in laboratory animals and was found to increase non-lymphatic leukaemia significantly in exposed workers, primarily in two cohort studies, which gave “evidence of an association between exposure to benzene and the likelihood of developing leukaemia”.

In these studies the risk of leukaemia in workers was not detectable when the average lifetime exposure was around 500 ppb (part per billion). To take into account the

difference between a working life (approximately 77,000 hours) and chronological life (about 660,000 hours), the figure of 500 ppb is divided by 10. A further factor of 10 was applied in order to extrapolate from the fit, young to middle-aged male working population to the general population that might reasonably contain individuals unusually sensitive to the effects of benzene. Because of uncertainties in the downward extrapolation of risk and to keep exposure as low as practicable, the U.K. Expert Panel on Air Quality Standards (EPAQS) recommended a target standard of 1 ppb as a running annual average exposure.

This gives a safety factor of 1000 below a level at which no effects could be seen in workers. It is important also to note that the EPAQS consists of five professional public health experts who are required to be totally independent of industry, the military and environmental lobby groups. These qualifications are not met by the ICNIRP council nor the Australasian Standards Association committee on RF/MW standards.

### **Grouping of Substances:**

Chemical substances are often grouped into classes of chemicals, such as the organochlorines or polycyclic aromatic hydrocarbons (PAHs). Within each tightly defined group some substances are classified as carcinogenic with particular disease outcomes and others are not.

### **EMR should be treated as multiple "Chemicals":**

At the Scientific Workshop on Biological Effects of Electromagnetic Radiation in Vienna, October 1998, Dr Carl Blackman, U.S. Environmental Protection Agency, presented the results of 30 years of research into cellular calcium ion efflux and influx which is induced by pulsed and modulated EMR. The work is well characterized as occurring within particular windows of intensity of signal ( $\mu\text{W}/\text{cm}^2$ ), modulation frequency, carrier frequency and temperature range. Statistically significant efflux or influx of calcium ions from exposed cells has been repeatedly observed for particular combinations of intensity, carrier frequency, modulation frequency and temperature, and not found at a nearby frequency intensity. These "windows" of effect have been found down to extremely low field intensities and are not found at some high but still athermal exposure levels.

Cellular calcium ion alteration in the presence of time varying electromagnetic fields is an established biological effect of EMR exposure. However, the "windowing" nature of this particular biological effect means, according to Dr Blackman, that **EMR must be considered as chemicals (plural) and not just a single chemical.**

Since alteration of cellular calcium ions concentration leads to many different health effects, and since many other biological changes have been identified, it is inappropriate to limit consideration of RF/MW exposure to single adverse health effects.

EMR exposes the whole human body and not a single target organ. Each organ has a different cellular structure which relies to a greater or lesser extent in electric and magnetic factors and forces for its growth and control. The brain, central nervous system and muscles, including the heart, make much stronger use of electrical signals than bones for example. However, every cell has an electric potential across its membrane and uses ions, such as calcium ions ( $\text{Ca}^{2+}$ ), sodium ions and potassium ions. Receptors on cells



are negatively charged and ions and neurotransmitters which initiate signal transduction are positively charged. DNA is negatively charged and the protein which is bound to it is positively charged.

Hence, every cell can interact with EMR and EMR can alter the growth regulation factors through alteration of the ionic concentration within the cells and in the intracellular fluid. Some higher functioning organs, especially the brain and CNS, are dependent on EMR for normal operation and have been shown to be altered by externally applied EMR, with consequent behaviour and neurological performance change, Bawin et al. (1976).

Because the whole body is exposed to RF/MW radiation, and since the brain and central nervous systems are electrically sensitive and active, it is not surprising that the most frequent adverse health effects identified in epidemiological studies are leukaemia and brain tumour. Leukaemia is a disease of the blood and bone marrow, whole body organs.

The ICNIRP approach, which at best can be seen as treating EMR as a single chemical, uses the observation that an effect shown in one laboratory or health study, but is not found in another when different frequencies, modulation frequencies, intensities and populations and effects are involved, as a reason to ignore the effects shown. By moving to the concept that EMR has different effects in different combinations of exposure parameters, much more accurate and appropriate interpretation of the scientific data is possible and more accurate.

### **Recommended Public Exposure Standard:**

At least 10 epidemiological studies have found increases in brain tumour in RF/MW exposed workers, including military personnel exposed to radio and radar. Eight of them reach statistical significance. A similar number of occupational studies have found a statistically significant increase in leukaemia. In addition there are many residential and occupational studies showing significantly increased adult and/or childhood leukaemia, some with significant dose response relationships. In addition there are several studies which report significant increases in "all cancer" from RF/MW exposure, some of these are also residential studies, and some have dose response relationships.

This body of studies alone, if applied to air pollution or toxic chemicals, would be sufficient to classify RF/MW as a human carcinogen, to identify an estimated lowest observed level adverse effect level (LOAEL) for residential exposure of about  $0.05\mu\text{W}/\text{cm}^2$  associated with childhood leukaemia. Applying a small safety factor of 50, which is conservative considering the diverse and sensitive members existing in the exposed population results in a public exposure standard of  $0.001\mu\text{W}/\text{cm}^2$  or  $1\text{nW}/\text{cm}^2$  ( $n = \text{nano} = 10^{-9}$ ).

At the turn of the century public exposures to RF/MW radiation were about  $10\text{pW}/\text{cm}^2$  ( $0.00001\mu\text{W}/\text{cm}^2$ ). Hence this initially proposed exposure standard allows for an increase of a factor of 100. However, since urban populations are already exposed to  $1$  to  $5\text{nW}/\text{cm}^2$ , a  $2\text{nW}/\text{cm}^2$  standard is impractical. Hence a  $10\text{nW}/\text{cm}^2$  ( $0.01\mu\text{W}/\text{cm}^2$ ) is proposed, allowing for a safety factor of 5 for leukaemia risk. As will be shown later, this allows for a safety factor of less than 1 for sleep, chronic fatigue, immune system impairment and learning impairment resulting from chronic low level RF/MW exposure.

An interim immediate target could be  $50\text{nW}/\text{cm}^2$  to allow industry time to adapt, but the recommended standard  $10\text{nW}/\text{cm}^2$  should be aimed to be achieved in 10 years.

This is despite the fact that the Swiss, Schwarzenburg Study, identified adverse effects on sleep, learning and a number of other serious health effects, down to mean levels of  $0.4\text{nW}/\text{cm}^2$ .

## **Biological Effects of RF/MW:**

### **Induced cellular calcium ion alteration:**

- of brain cells is associated with behavioural and reaction time changes and associated EEG alterations, Bawin et al. (1978);
- of the pineal gland reduces the nocturnal production of melatonin (which increases the cell damage throughout the body, reduces the integrity and competence of the immune system, and hence increases the incidence of cancer and immune system related disease and degenerative diseases of the brain, Reiter (1994) and Walleczek (1992);
- of lymphocytes reduced the competence of the immune system making the subject more vulnerable to allergens, toxins and viruses, and to leukaemia; and
- of damaged cells alters the ratio of surviving neoplastically transformed cells and those programmed to self destruct (apoptosis), Balcer-Kubiczek (1995).

Several studies show that RF/MW exposure and ELF exposure can reduce pineal melatonin production. Professor Russell Reiter, one of the worlds leading medical researchers into the effects of melatonin, summarizes melatonin's roles, Reiter and Robinson (1995), as being:

- Vital for healthy sleep, including lowering the body temperature, and assisting in maintaining health sleep states.
- Reduces cholesterol, with consequent reductions is risk of atherosclerosis and coronary heart disease.
- Reduces blood pressure and the tendency for blood clots, and hence reduces the risk of strokes.
- Scavenger of free radicals. This, along with the above factors, reduces the risk of heart attack, cancer, viral replication. Melatonin plays a vital free radical scavenging role in the brain where, because it is high in iron, has a high production rate of hydroxyl radicals ( $\text{OH}\bullet$ ). Free radical damage is now known to play a formative role in most brain disorders, including Alzheimer' disease, Lou Gehrig's disease, multiple sclerosis and Parkinson's disease. While the Blood Brain Barrier (BBB) denies access to most free radical scavengers, melatonin has free access.
- Enhances the effectiveness of the immune system. Specifically enhancing the T-cells, i.e. the T-helper cells and the T-killer cells. T-helper cells have a receptor for

melatonin. When melatonin is received a cascade of events is set in motion including stimulation of Interleukin-4 (IL-4) which then stimulates natural killer cells (NK), B-cells, IgA, phagocytes and T-Cytotoxic cells. The NK cells specialize in attacking cancer cells and virus infected cells.

### **Alzheimer's disease:**

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

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

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

- The first step involves EMR exposure upsetting the cellular calcium ion homeostasis through calcium ion efflux from cells increasing the intracellular calcium ion concentrations. This cleaves the amyloid precursor protein to produce soluble amyloid beta ( $sA\beta$ ).
- $sA\beta$  is quickly secreted from cells after production, increasing the levels of  $sA\beta$  in the blood stream.  $sA\beta$  then binds to Apolipoprotein E and apolipoprotein J to be transported to and across the Blood Brain Barrier.
- Over time, when sufficient  $sA\beta$  have been transported to the brain, a cascade of further events lead to the formation of insoluble neurotoxic beta pleated sheets of amyloid fibril, senile plaques, and eventually AD.

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

### **Breast Cancer**

Breast tissue is very sensitive to free radical damage and hence to melatonin reduction. While breast cancer has been associated with diet, stress levels and a number of chemical toxins, there is now compelling evidence that power frequency (50 Hz or 60 Hz) radiation can overcome the protective effect of melatonin in breast cancer cells. This research has now been carried out in 4 independent laboratories. This work shows a dose response relationship between 0.2 and 1.2 $\mu$ T (2 and 12 mG). At 1.2 $\mu$ T the protective effect of melatonin is completely negated. Several epidemiological studies have associated EMR and EMF exposure with breast cancer. With the progressively increase Mer exposure of the U.S. population, EMR cannot be ruled out as a contributory factor in the increase in rate of breast cancer in U.S. women under the age of 85 rising from 1-in-20 in 1940 to 1-in-8 by 1994.

Several epidemiological studies find statistically significant associations between EMF and EMR exposure and breast cancer, including Demers (1991), Tynes et al. (1996) and Hardell et al. (1995). Hardell et al. (1995) was an extensive independent review of the scientific literature published up to 1 July 1994 in relation to ELF exposures. One of their conclusions relates to “electrical occupations”. In such situations ELF and RF/MW signals are common. They conclude that there is “an increased risk of breast cancer, malignant melanoma of the skin, nervous system tumours, non-Hodgkin lymphoma, acute lymphatic leukaemia or acute myeloid leukaemia and certain occupations.”

Demers et al. (1991) found an elevated risk of male breast cancer in radio and communications workers, OR = 2.9, 95% CI: 0.8 - 10. Tynes et al. studied 2,619 Norwegian female radio and telegraph operators and their incidence of disease between 1920 and 1980. They compared the occupational incidence with the general population using a standardized incidence ratio (SIR). For all cancers SIR = 1.2, and for breast cancer SIR = 1.5 ( $p < 0.05$ ).

In Professor Reiter’s book, published in 1995, he describes the evidence that EMR/EMF does reduce melatonin as a “Smoking Gun” level of proof. That is, there is considerable scientific evidence but at that time it wasn’t sufficient for proof.

By considering more recent information, and the extensive results of biometeorological research, and linking the melatonin research to the calcium ion research, the level of proof can be seen as causal.

### **Biometeorological Research:**

This conclusion was drawn without reference to biometeorological work at the Max Planck Institute in Germany in the 1960s and 1970s involving isolating volunteers for many months from sunlight, and in some cases, from the earth’s fluctuation electromagnetic field by using a Faraday Cage, Wever (1974). The results included the fact that those in the Faraday Cage shielded room, identical to the other room in all other respects, had significantly longer circadian rhythms ( $p < 0.01$ ).

In addition, a significant proportion of the Faraday Cage group “desynchronized” while none of the other group did ( $p < 0.001$ ). This involved rapid lengthening of the circadian period from around 26-27 hours to 30 - 36 hours, Figure 1.

From the results of the experiments involving human subjects, their reaction times and altered circadian rhythm, the German researchers from the Max Planck Institute conclude:

**“Thus, it has been proven at a high statistical level that the artificial electric 10 cps field diminishes the tendency towards internal desynchronization, as does the natural field.”**

The desynchronization was removed through the application of a 10 Hz signal with a peak to peak field strength of 2.5 V/m. This is equivalent to  $0.83 \mu\text{W}/\text{cm}^2$ . The signal the Faraday cage had removed, which was replaced by this artificial signal, was the Schumann Oscillation which has a field intensity of about  $0.3 \text{ pW}/\text{cm}^2$ . Hence the

desynchronization was caused by the removal of a  $0.3\text{pW}/\text{cm}^2$  signal. Wever (1974) concludes that their research gives:

**“significant proof that electromagnetic fields in the ELF range influence the human circadian rhythms and therefore human beings.”**

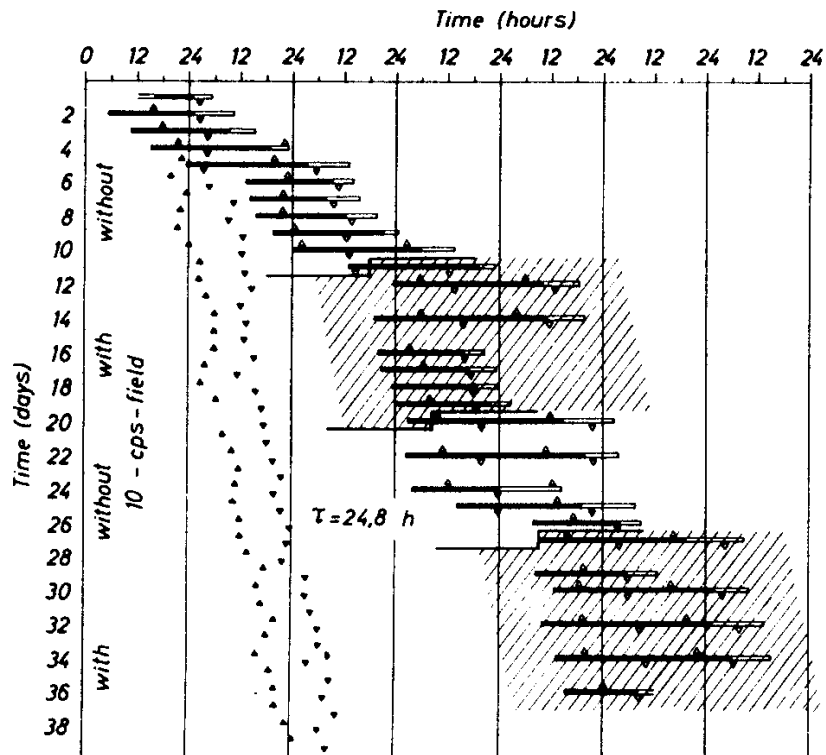


Figure 1: Free-running circadian rhythm of a subject living under strict isolation from environmental time cues. During the first and third section protected from natural and artificial electromagnetic fields, during the second and fourth sections (shaded area) under the influence of a continuously operating 10 Hz electric field of 2.5 V/m, Wever (1974).

A plausible biological mechanism was proposed by Konig (1974). He noted the strong similarity between the frequencies of the Schumann Oscillation and the alpha band of the human EEG, see the figure below. A resonant interaction is clearly feasible. Removing the Schumann Oscillation for some individuals, removes part of their circadian control.

The Type II signals on the left are naturally occurring, locally sourced ELF fields centred around 3 Hz, close to the delta EEG band. Konig (1974) showed that people's reaction time significantly slows in the presence of Type I signals and speed up when Type II signals were dominant, Figures a and b.

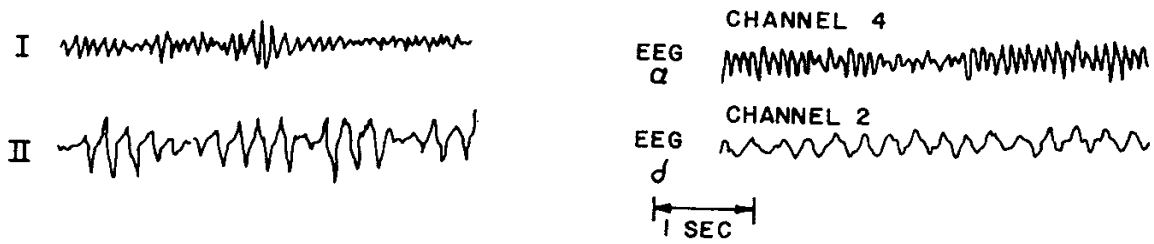


Figure 2: Electric fields from I, the Schumann-Resonance, II, Local fields of about 3 Hz and the  $\alpha$  (10 Hz) and  $\delta$  (3 Hz) human EEG channels, Konig (1974).

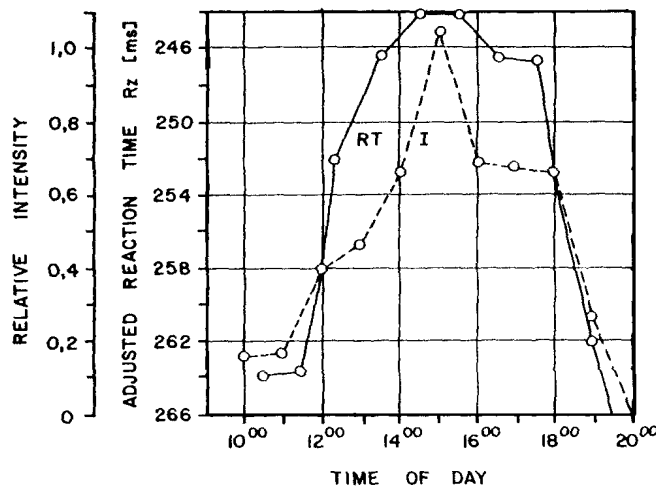


Figure 3: The solid line shows the reaction times of 4500 people per point, over the day in September 1953 in Munich, compared with (dashed line) the Type I (10 Hz) signals field intensity.

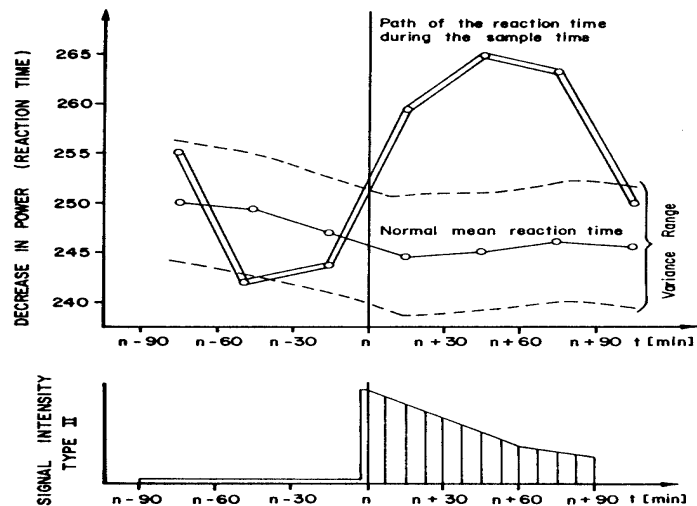


Figure 4: The speeding up of the reaction time of people in the 60 to 90 minutes following the onset of 3 Hz signals, from the Traffic Exhibition in Munich in 1953.

Signals of the Type II occurred during 10 occasions during the August-September period. Figure 4 shows the inter-relation for the change in reaction time relative to the onset of

Type II signals at time n hr. In the hour and a half after the onset of Type II signals the reaction times (involving between 2000 and 3000 people), are well above average.

At the same time that the Germans were publishing their biometeorological results showing that human being's reaction times vary with extremely low intensity naturally occurring and varying electromagnetic fields in the ELF part of the spectrum, Professor Ross Adey and Dr Susan Bawin were showing that altered human reaction times in ELF modulated microwave fields was associated with altered EEG and calcium ion efflux from the brain cells.

Hence the U.S. and German research jointly confirm both the effect and the mechanism.

### **Physiological Reactions to Atmospheric EMR/EMF changes:**

Very few people are aware that anticyclones and depressions are characterized by very different natural background of ELF modulated RF fields. Lomar et al. (1969) characterized these weather system EMR/EMF characteristics as:

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

Anticyclone: 10 kHz, 1-3 Hz, < 10 mV/m, (Exposure < 0.000027  $\mu\text{W}/\text{cm}^2$  )

Importantly Lomar et al. (1969) found that in the laboratory under simulated cyclonic conditions (using the above EMR fields) mouse liver respiration rates were 42 % higher than anticyclonic conditions, a highly statistically significant effect ( $p < 0.001$ ). It is well known and accepted that people generally feel fresher and more energetic in clear, sunny anticyclonic weather, compared to overcast, wet and windy depression weather. This is partly explained through a stronger serotonin/melatonin rhythm in sunny weather compared to cloudy weather. Sunlight drives daytime melatonin down and serotonin up producing sensations of clear headedness and alertness. The German research also shows that naturally occurring ELF modulated RF fields vary by a factor of about 100 in intensity, from 2.7 nW/cm<sup>2</sup> in depressions to 27 pW/cm<sup>2</sup> in anticyclones and that this is associated with a highly significant change in liver respiration.

Thus the German work in the 1960's and 1970's established that naturally occurring EMR and EMR at extremely low levels influenced and altered sleep, circadian rhythm and reaction times. In the 1990's German work showed the cell phones alter the human EEG and interfere with REM sleep, Von Klitzing (1995) and Mann and Roschkle (1996). Impairment of REM sleep is associated with memory and learning difficulties. The Swiss research (Altpeter et al. (1995) and Abelin (1998) - The Schwarzenburg Study) found a causal relationship between sleep disturbance and subsequent chronic fatigue, and short-wave radio exposures at extremely low mean levels.

In 1998 Mild et al. (1998) survey over 10,000 cell phone users in Norway and Sweden, Figure 5. They found significant dose response relationships for a number of crucial symptoms that had been clinically described and associated with cell phone use by Hocking (1998).

The symptoms include dizziness, a feeling of discomfort, difficulty with concentration, Memory Loss, Fatigue, Headache, Burning Skin and tinglingness and tightness of the skin near the phone. The symptoms were consistent across analogue and digital (GSM) phone users. A dominant physical symptom was a sensation of warmth on the ear and behind the ear. These is not a sensation which is experienced with a conventional telephone but are unique to the cell phone which exposes the user's head to moderate to high intensities of microwaves. It was significant that the neurological symptoms were highly correlated to the warm sensations. The symptoms are consistent with the Schwarzenburg symptoms. The headache symptoms were found with microwave exposure during "microwave hearing" experiments, Frey (1998).

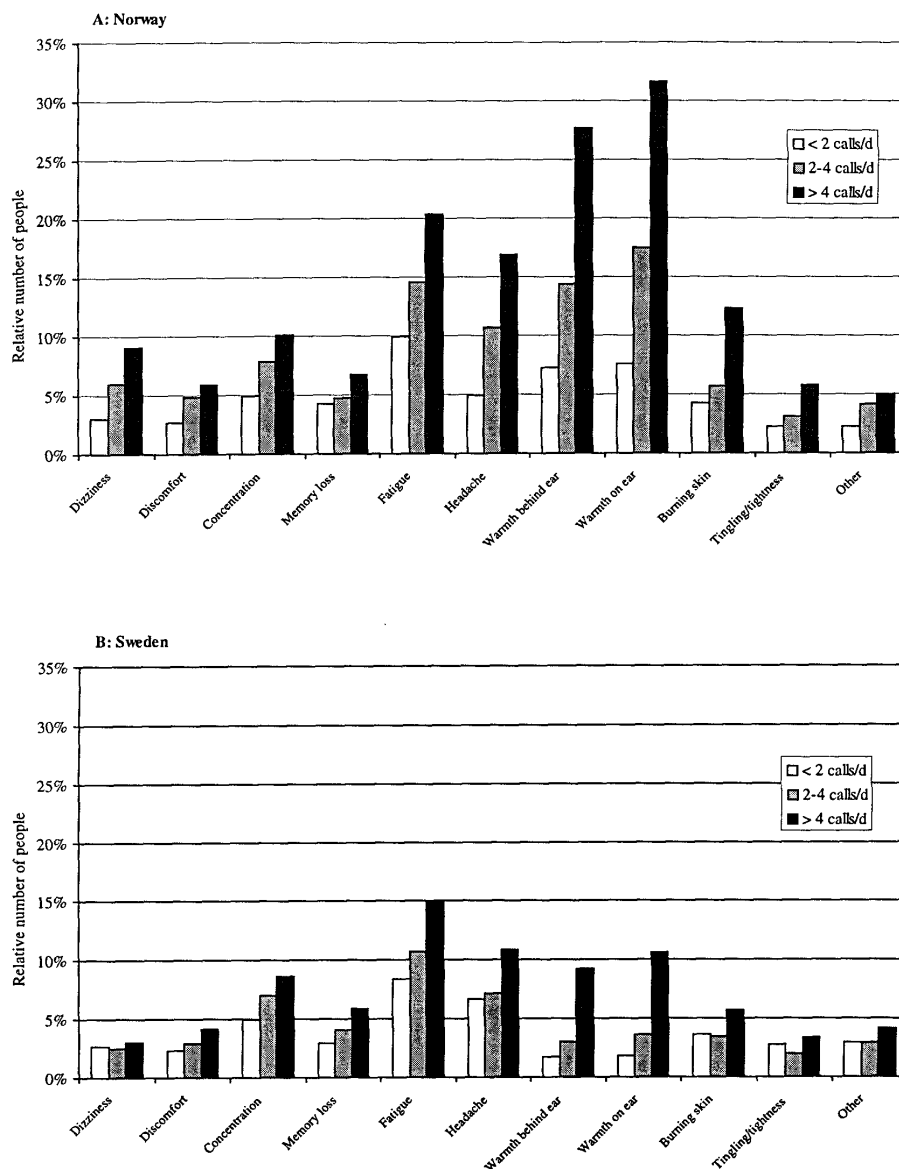


Figure 5: The prevalence of symptoms with various categories of calling times/day, A. Norway, B. Sweden, Mild et al. (1998).

The link with calcium ion efflux, altered EEG, behavioural change and EMR exposure is well established. The link with melatonin is stronger than the smoking gun proof accepted by Reiter (1995), with the circadian rhythm connection and the sleep disruption at Schwarzenburg. Salivary melatonin was measured in cows in the



Schwarzenburg study in 5 'exposed' cows and 5 'unexposed' cows. The exposed cows had lower mean melatonin levels but the difference was not statistically significant because the sample was too small. Human beings were sampled (using urine analysis). Samples were taken first thing in the morning when melatonin levels are naturally low, instead of at the correct time soon after midnight, when melatonin levels are high and reductions are easier to detect. However, the research team noted "Persons reporting sleep disorders, however, tend to have lower melatonin levels."

When the transmitter was off unexpectedly for three days, sleep quality improved markedly, and for those three nights the melatonin in the exposed cow herd reached their highest nocturnal peaks for that week. When the transmitter went on again, on that day the exposed cows' melatonin was statistically significantly lower than the unexposed cows.

In addition to these observations, two recent papers made direct human measurements of melatonin in association with power frequency exposure and one of them also associated cellphone usage, Armstrong and Martin (1997) and Burch et al. (1997). In both cases they found statistically significant reductions in melatonin.

**It is clearly a mistake to seek to classify the effects of EMR in terms of a single health outcome which should be expected to occur across the whole spectrum of carrier frequencies, modulation frequencies, intensities and ambient temperatures.**

### **Biological mechanisms:**

One of the primary reasons many skeptics about EMR health effects use to dismiss studies which show statistically significant effects and even dose-response relationships, is the apparent lack of a plausible biological mechanism for the EMR to alter the biological processes in an adverse way. While well documented biological mechanisms do exist, including calcium ion efflux and melatonin reduction. The EMR skeptics ignore these or claim that they must be invalid because of their pre-conceived notions that EMR must be benign because the EMR photons do not possess the energy to ionize atoms nor to break chemical bonds.

The EMR skeptics are wrong on two counts. There are plausible biological mechanisms, as stated above, and, the classifications of substances as carcinogens does not require the identification of detailed biological mechanisms if we are dealing with air pollutants or chemical carcinogens.

The absence of a detailed step by step biological mechanism is not a limitation on classifying chemicals, such as benzene, as carcinogens. A chemical which is observed to neoplastically transform cells, produces tumours in laboratory animals and is associated with increased incidence of cancer in exposed workers, is classified as a carcinogen.

Even two years ago Quinn (1997) noted that "although the role of ultraviolet radiation in human skin carcinogenesis has been supported by a wealth of epidemiological data, the mechanisms by which it leads to skin cancer are still poorly understood." This hasn't stopped the Cancer Society from running "slip, slap, slop" and cover-up campaigns for several years in order to reduce the risk of skin cancer. These programmes are targeted

at children for it is understood that UV damage in childhood leads to a higher incidence of skin cancer as adults.

### **Why is EMR treated differently from other toxic substances ?**

The history of EMR shows that it has always been treated differently from chemicals. This is largely a consequence of the controversies around the adverse health effects of “radiation” in contrast to the “national security benefits” of the use of “radiation”.

“Radiation” in this context is nuclear radiation and the alpha-, beta-, gamma- and X-rays which are released by nuclear explosions. The absence of reliable and repeatable acute effects was taken as evidence as the absence of effects. When the atomic bombs were dropped on Japan the only officially acknowledged effects were the explosive effects of blast and the shockwave.

The lingering health effects among the surviving populations of Hiroshima and Nagasaki were initially attributed to vitamin deficiency. Western scientists strongly denied that the sickness related to the after-effects of the bombs, largely because there was no known plausible mechanism. It took years for radiation sickness to be recognized and decades for radiation related cancers to be recognized. It took many more years to identify the mechanism through which the radioactive material released ionizing radiation which produced free radicals, which in turn caused single and double strand breakage of DNA, and cancer.

The observation that ionizing radiation can ionize atoms, produce free radicals and hence damage DNA, was incorrectly taken as assurance that non-ionizing radiation, which could not ionize atoms, must by this very fact, be benign.

It gave the EMR skeptics a sense of security and comfort to assume that ionizing radiation is harmful and all other parts of the electromagnetic spectrum is safe and benign.

Thus, it was assumed, the part of the solar spectrum which included ultraviolet (UV), visible and infrared (IR), were part of the benign spectrum, because the threshold for ionization lies above the UV region. Recently it has been established that UV radiation is carcinogenic, damaging the DNA of skin to produce melanoma and squamous cell carcinoma.

However, despite the clear evidence that UV radiation is carcinogenic without having the energy to ionize atoms and break chemical bonds, the EMR skeptics, which include most Health and Radiation Physicists have maintained their view that ionization and radiation induced chemical bond breakage means (to them) that EMR is benign apart from heating effects.

### **Ionization is not a prerequisite for cancer:**

Many generations of medical biologists and toxicologists do not assume that ionization is a necessary prerequisite for cancer producing agents since thousands of chemicals are cancer producing agents without the involvement of ionization. Chemicals change the biochemistry of cells and hence can cause neoplastic transformation.

**Free radicals occur naturally in our bodies:**

Free radical chemistry is quite straight forward. Atoms are held together to form molecules by sharing electrons. Two electrons shared between two atoms forms an ionic bond. Some atoms, especially oxygen, can easily gain only one of these bonding electrons, which means that it has an unpaired electron and hence is very reactive. This is a free radical, a molecule with unpaired electrons.

Free radicals are produced by many chemical reactions, including respiration. In breathing we all produce oxygen free radicals all of the time. Hence DNA and cellular damaging free radicals are a ubiquitous and ever present reality for all air breathing mammals. They are so reactive that they only last for a few nanoseconds but they are always present because they are always being generated.

**Damage and repair:**

The extent of the damage caused by free radicals and the amount and rate of repair which is necessary, is strongly dependent on the presence of free radical scavengers and a the health of the immune system. Our immune system has the job of identifying damaged cells and foreign agents and eliminating them. Our cells also have internal checking mechanisms.

When genetic damage is detected and a cell starts to behave abnormally, several systems seek to eliminated that rouge cell. The cell has an internal checking system and can start to digest the cellular protein in a damaged cell in a process called programmed cell death or apoptosis. If this doesn't happen and the damaged cell survives then the cell may be identified as "foreign" and the natural killer cells in the immune system can attack and eliminate them.

Thus in biological cellular based systems such as human and animal bodies, a healthy state is one in which the naturally occurring cellular damage is being detected, and eliminated or repaired. Ill health occurs when any situation or factor enhances the rate of damage or diminishes the effectiveness of the repair mechanisms.

Melatonin, a neurohormone produced from serotonin in the pineal gland, is the strongest known naturally occurring free radical scavenger. It also has the property that it can easily pass through the cell membrane so that it actively seeks to eliminate free radicals in the vicinity of the nucleus of the cell. It is the nucleus of the cell which houses the chromosomes and DNA. Hence melatonin plays a vital role in minimizing damage to chromosomes and DNA by free radicals. Melatonin levels are low during the daytime when respiration rates are high. Melatonin concentrations in the blood stream and cells is high at night when respiration rate, and hence free radical generation rates, are lowest. Hence a great deal of cellular repair is accomplished at night.

Melatonin also provides this protective effect for the immune system, assisting it to remain healthy and effective.

Any factor which reduces melatonin levels results in the greater risk of cell damage, faster cell death through apoptosis, and greater chance that a damaged neoplastic cell can survive to become cancerous. Factors which are known to reduce melatonin in mammals, including in people, includes older age, light at night, sleeplessness and electric and magnetic fields, of themselves or in combination with RF/MW fields.

In the same manner, EMR alters the electrobiochemistry of cells and hence can cause neoplastic transformation of cells. The way in which EMR does interact with cells is illustrated by considering a known cancer promoter, TPA. TPA is phorbol myristate acetate. It is very commonly used in laboratories as a cancer promoter. TPA acts by altering an already damaged (neoplastically transformed) cell by switching the effect of calcium elevation from cell death to cell proliferation. Thus TPA maintains the malignant phenotype by blocking apoptosis through altering the calcium ion status of the cell. It is already shown that calcium ion efflux and influx is induced by ELF modulated RF/MW. Hence RF/MW can enhance cancer in some situations and enhance apoptosis in others.

Calcium ion efflux has been documented in published papers down to an SAR of 0.00015W/kg, Schwartz et al. (1990), using 240 MHz microwaves modulated at 16 Hz, the rate of calcium ion efflux was 21 % higher than the control, with  $p < 0.05$ . The medium was isolated frog hearts. This is an exposure intensity of  $0.08 \mu\text{W}/\text{cm}^2$ ,  $0.3 \text{ V/m}$  and  $1.8\text{nT}$ . Dr Carl Blackman, pers. comm. informs me that his laboratory has found calcium ion efflux occurring in fT (femtoTesla =  $10^{-15} \text{ T}$ ) ELF fields. 10 fT is equivalent to  $2.4 \times 10^{-12} \mu\text{W}/\text{cm}^2$  or  $2.4 \text{ attoW}/\text{cm}^2$ . This might sound totally unrealistic until it is noticed that if the carrier was a 50 MHz signal,  $2.4 \text{ aW}/\text{cm}^2$  would still stand out against the blackbody background ( $1 \times 10^{-19} \text{ W}/\text{cm}^2$ ) by a factor of 24 .

For many people in the EMR area there is a “mind block” which stops them from accepting the possibility that EMR can have biological effects because of their assumptions about ionization, free radicals and radiation induced chemical bond breakage as prerequisites for biological action. A large body of scientific research contradicts this stance but this mind set persists and dominates the WHO, IRPA, ICNIRP, National Radiation Laboratory, Industrial and military personnel and their consultants, and the Standards setting bodies in Australasia and around the world.

To continue this mindset based on these challengeable assumptions continues to put millions of people at risk or severe health effects in New Zealand and billions of people around the world. To adopt the ICNIRP guideline will therefore be shown to be a disaster in New Zealand and thousands of people will suffer unnecessarily as a consequence.

### **Legal Guidance:**

The Environment Court (MacIntyre 1996) declared that the New Zealand Standard (and hence the ICNIRP guideline) is “not decisive” in New Zealand law but that the Sections 5 and 3 of the RMA are the appropriate legal basis for public exposure to electromagnetic radiation (EMR). In considering the evidence before it the court set a public exposure condition at that time and in that case of  $2 \mu\text{W}/\text{cm}^2$ , 1 % of the then allowed public exposure in NZS 6609, and of the proposed AS/NZS 2772.1 and ICNIRP guideline.

In the recent Shirley Primary School Case, Judge J. Jackson made an error in law and through his interpretation of the scientific evidence through his failure to properly apply sections 5 and 3 of the RMA.

The guideline discussion document legal section makes an error in referring to the MacIntyre case as having set a public limit of  $50 \mu\text{W}/\text{cm}^2$  when in fact it was  $2 \mu\text{W}/\text{cm}^2$ .

### **Scientific Critique of ICNIRP Assessment:**

#### **ICNIRP Discussion of 100kHz-300GHz effects:**

##### **Reproductive outcomes:**

There are several major errors and omissions in the ICNIRP (1998) assessment of reproductive effects, ICNIRP (1998), p 504.

This includes misrepresentation of two studies, inadequate interpretation of three studies and omission of several relevant epidemiological studies and failure to cite the relevant animal studies.

ICNIRP (1998) concludes that studies involving pregnancy outcome and microwave exposure suffer from poor assessment of exposure, small numbers of subjects and contrasting results. All of these claims and conclusions are wrong.

##### **The studies of Daels (1973 and 1976):**

The first claim is that there are two extensive studies on women treated with microwave diathermy to relieve the pain of uterine contractions during labour, with no evidence of adverse effects on the fetus, quoting Daels (1973 & 1976).

Daels (1973 (4 pages) & 1976 (2 pages)) are not an extensive studies on the effect on the fetus. They are small descriptive papers on an analgesic therapy for use in labour.

The subject of the study is the mother. A fully developed child is involved, immediately prior to birth, not the developing fetus which other studies are concerned about. The papers contain no assessments of the effects on the child. In Daels (1973) he simply states "No undesirable side effects of microwave heating of tissues are known." He references a single study, Leary (1959) to note that overheating can be a rare complication. Thus Daels (1973 & 1976) are neither extensive studies nor about fetal health.

These studies involve short term microwave heating of the uterine area for 30 to 40 minutes during labour. There was a maximum recorded neonate temperature of  $37.8^\circ\text{C}$  and amniotic fluid temperature of  $36.5^\circ\text{C}$ . These are well within the normal range. Heating was limited to levels where the mother felt skin heating as "agreeable". Since most of the microwaves are absorbed in the surface skin layers the fetal exposure will be extremely small, see Hocking and Joyner (1995) below. There is no reported follow-up on the children over subsequent years to determine any altered health status, which might have

resulted from chromosome aberrations which, could have occurred during the microwave exposure.

**It is therefore totally inappropriate and grossly misleading to cite these as “extensive studies” of the impact of microwaves on the fetus. They are not extensive, they do not relate to developing fetus and there is no actual assessment of the impact of the exposure on the children.**

### **Interpretation of Physiotherapy Studies:**

In assessing reproductive outcomes from physiotherapist studies it is important to distinguish short-wave exposure and microwave exposure, small study populations and larger study populations, and whole pregnancy including birth outcomes, in contrast to early pregnancy miscarriage alone. The effects of short-wave radiation are likely to be different from microwave effects. Small sample sizes may have elevated Risk Ratios but lack statistical significance solely by virtue of the small sample size.

### **Physiotherapist Studies Cited by ICNIRP (1998):**

In ICNIRP 1998 three physiotherapist studies are cited, Kallen et al. (1982), Larsen et al. (1991) and Ouellet-Hellstrom and Stewart (1993).

Kallen and Larsen involve small samples and short-wave exposure, and whole pregnancy outcomes, whereas Ouellet-Hellstrom and Stuart involves a large sample, studies only early pregnancy miscarriage and finds only microwaves to have an effect. Kallen et al. and Larsen et al. are cited in the review referred to as) with results which raise concerns about possible effects. The reviewers state however “The results suggest further study is necessary before conclusions can be drawn.”

Several other studies were available prior to 1993 but they were not used by UNEP/WHO/IRPA (1993).

In 1993 Ouellet-Hellstrom and Stewart was published with even more significant results.

When all the studies are taken together they form a comprehensive and compelling body of research to show that microwave exposure of mothers leads to a significant increase in early pregnancy miscarriage, with a significant dose response relationship, and that those using short-wave radio therapies and working in electrical industries, have more late pregnancy problems and malformed children.

The most likely mechanism is accumulated chromosome aberrations and damaged cells in the placenta and fetus because biophysics shows extremely small temperature increases can be expected from even very high RF/MW exposures.

### **Case by case assessment:**

ICNIRP states that there were “no statistically significant effects on rates of abortion or fetal malformation” in Kallen et al. (1982). This is wrong. even though Kallen et al. involves small sample numbers they conclude “The only positive finding was a higher

incidence of short-wave equipment use among the females with dead and deformed infant than among controls.” Very few therapists were involved with microwaves. Hence Kallen et al. associate fetal death and malformation with the use of short-wave diathermy equipment, with  $p=0.03$ . This is a statistically significant association, contrary to the ICNIRP claim.

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

Ouellet-Hellstrom and Stewart (1993) investigated early pregnancy miscarriage among U.S. physical therapists using short-wave (27 MHz) and microwave (915 MHz and 2.45 GHz) diathermy. The sample included 1753 case pregnancies (miscarriages) and 1753 control pregnancies. They found no significant increase in first trimester miscarriage amongst those using short-wave diathermy. They found a statistically significant increase in miscarriage in the first trimester with microwave exposure (OR= 1.28, 95%CI: 1.02-1.59) and a statistically significant dose response relationship ( $p<0.005$ ) using a dose measure of treatments per month. With more than 20 treatments per month OR = 1.59, 95%CI: 0.99-2.55 .

In addition to the three studies cited in ICNIRP (1998) there are several others with are relevant.

Vaughan et al. (1984), studying U.S. workers, 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.

Taskinen et al. (1990) in Finland, with 204 cases, found increased spontaneous abortion with short-wave and microwave use: Note that the statistical a significance is limited by the small sample sizes.

- Electric therapies >5/week OR= 2.0, CI: 1.0-3.9, n=17
- Shortwaves>=5h/week, OR= 1.6, CI: 0.9-2.7, n= 30
- Microwaves, OR= 1.8, CI: 0.8-4.1, n=13),

Stronger associations with ultrasound and heavy lifting:

- Ultrasound>=20/week, OR= 3.4, CI: 1.2-9.0, n=9
- Heavy lifting, > 10 kg or patient transfers >=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 OR=2.5
- Microwaves OR=2.4
- Ultrasound OR=3.4
- Heavy lifting OR=6.7 .

Taskinen et al. 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, in the context of all the other studies, is consistent and adds considerable weight to the conclusion that there are adverse health effects from RF/MW exposure. Taskinen et al. also found statistically significant increases in congenital malformations in the children of mothers using shortwave therapy. This confirms the results of Kallen et al, and Larsen et al.

Taskinen et al. (1990) was the only Scandinavian study to have a large enough sample to investigate the effects of miscarriage with microwaves. The sample was quite small (13), limiting the significance of the result. The Odds Ratio was (OR= 1.8, 95% CI 0.8-4.1). Exposure to ultrasound and short-wave showed significant increases in odds ratio for abortion after the 10th week of gestation, (OR = 3.4,  $p < 0.01$  and OR = 2.5,  $p < 0.03$ , respectively). Taskinen et al. concluded: “The effect of shortwaves and ultrasound on the ‘late’ spontaneous abortions was significant and increased in a dose response manner.”

Sanjose et al. (1991) investigated the incidence of low birthweight and preterm delivery in Scotland, 1981-84, in relation to parent’s occupation. They found statistically significant ( $p < 0.05$ ) increases in low birth weight (RR = 1.4) and preterm delivery (RR = 1.8) for mothers who work in the electrical industry. People who work in “electrical industries” are recognized as being exposed to a wide range of EMR giving them more than average EMR exposures.

Vaughan et al. (1984), Taskinen et al. (1990) and Sanjose et al. (1991) are consistent with Kallen et al. (1982) and Larsen et al. (1991) giving the conclusion that shortwave exposure takes longer to produce effects than do microwaves. Shortwave effects range from later pregnancy miscarriage, still birth, low birth weight, premature birth, cot death and congenital abnormalities.

Taskinen et al. (1990) and Ouellet-Hellstrom and Stewart (1993) confirm that microwave exposure is associated with early pregnancy miscarriage.

It is sobering to also note that breast cancer risk is over 4 times higher for women who miscarry in the first trimester, RR = 4.1, 95% CI: 1.5-11.3, Hadjimichael et al, (1986).

Genetic damage from RF/MW has been studied by a number of researchers. ICNIRP (1998) quotes Cohen et al. (1977) which found no association between radar exposure and Down’s syndrome in their off-spring. They failed to mention a previous paper from the same group, Sigler et al, (1965), which did find a significant risk from parental radar exposure.

Sigler et al. suggested that this result, along with research which found “tissue damage in humans and laboratory animals” and “a deleterious effect of rat testis” as evidence that microwaves might be ionizing radiation, since similar effects had been identified with exposure to ionizing radiation. We now know that chromosome aberrations do occur in microwave exposed subjects without the need for microwaves to be ionizing.



Flaherty (1994) presents “The effect of non ionizing electromagnetic radiation on RAAF personnel during World War II”. He found in a group of 302 surviving veterans, men had a ratio of single to twin births of 41:1, women 38:1 and overall the ratio was 40:1 . This contrasts with the ratio in the normal Australian population of 85:1. Hence radar exposed veterans had over twice the expected number of twins, a very significant result.

### **Animal Toxicology:**

ICNIRP (1998) fails to refer to the significant research involving animal experiments on reproductive effects when exposed to RF/MW.

Results range from testicular degeneration, resorption of the fetus and altered body weight at high but non-thermal levels of exposure to total infertility in multigenerational studies of mice exposed to  $0.168\mu\text{W}/\text{cm}^2$  and  $1.053\mu\text{W}/\text{cm}^2$ , Magras and Xenos (1997).

There are many animal studies showing that RF/MW is teratogenic, that is, it causes severe reproductive problems. Berman et al. (1982) introduce their paper by stating:

**“It has been repeatedly shown that microwaves have teratogenic potential. Rats and mice have been used almost exclusively in these studies.”**

Berman et al. (1982) were extending the studies to hamsters. They investigated the teratogenic potential of microwaves on Syrian hamsters, using 2.45 GHz at power densities of  $30\text{ mW}/\text{cm}^2$  for 100 minutes daily This caused a temperature rise of  $0.8\text{ }^\circ\text{C}$  and significant fetal resorptions or death ( $p = 0.0012$ ), decreased fetal body weight ( $p=0.0001$ ) and decreased skeletal maturity. Averaging this over a whole day the mean exposure is  $2.08\text{ mW}/\text{cm}^2$ . Maternal toxicity was not observed, only fetal damage and death. They conclude by comparing hamsters with mice.

**“In mice, SAR’s of 16 or 22 mW/g caused fetal changes. Comparing these two species, we see that 16 mW/g and above can cause decreased body weight and skeletal immaturity in mice, while only 9 mW/g in the hamster causes similar changes. Additionally, this lower SAR causes a significant increase in hamster fetal death (resorptions). Hamster fetus, appears to be more susceptible to microwave radiation than the mouse, exhibiting fetotoxic changes at lower SAR values.”**

Prausnitz and Susskind (1962) exposed male Swiss albino mice to 9.27 GHz microwaves, pulsed with a  $2\text{ }\mu\text{s}$  pulse at 500 Hz, 4.5 mins per day, 5 days per week for 59 weeks with an exposure level of  $100\text{ }\mu\text{W}/\text{cm}^2$ . This amounts to a mean weekly exposure of  $0.22\mu\text{W}/\text{cm}^2$ .

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 (atrophy with no sperm) occurred in 29.8% (39/124) of the exposed animals and 7.1 % (4/56) of the control animals, RR = 4.2.

- (2) Cancer of the white cells or leukosis was seen in 26.5% (39/147) of the exposed animals compared to 13.0% (9/69) of the controls, RR= 2.04. This condition was described as monocytic or lymphatic organ tumours or myeloid leukaemia in the circulating blood.

In these mice significant and severe (4.2-fold) testicular damage and a 2-fold increase in the initiation of leukaemia occurred in association with a mean exposure of  $0.22\mu\text{W}/\text{cm}^2$ .

Testicular damage has also been found in men who have radar exposures. Weyandt et al. (1996) studied U.S. service men who have radar exposures. **“The group of men with potential microwave exposures demonstrated lower sperm counts / mL (p = 0.009) and lower sperm/ejaculate (p= 0.027) than the comparison group.”**

Although as early as 1962 severe reproductive problems had been identified with an exposure regime averaging  $0.22\mu\text{W}/\text{cm}^2$  most of the research was carried out with the incorrect assumption that if an effect was real it would be demonstrated if the exposure was high enough. And if an effect was not detectable at extremely high levels of exposure, there was no way that an effect would occur at low levels of exposure.

Even so, high exposure experiments did show effects. Below shows the progression downwards until animal experiments have been carried out and found significant effects at the levels used in 1962 by Prausnitz and Suskind and are found in the vicinity of cell sites.

Chazan et al. (1983) investigated the development of murine embryos and fetuses after irradiation with 2450 MHz microwaves at  $40\text{ mW}/\text{cm}^2$ . They found indications of retardation of development in the early period of gestation in mice exposed to thermal MW fields. During the second half of pregnancy an increase in the number of resorptions, stillbirths and internal hemorrhages was noted. The living fetuses had lowered body mass compared to the offsprings of sham-irradiated mice.

Berman, Carter and House (1982) also found reduced weight in mice offspring after in utero exposure to 2450-MHz (CW) microwaves using an exposure level of  $28\text{ mW}/\text{cm}^2$ . They were exposed to for 100 minutes daily from the 6th through 17th day of gestation. This gives a mean exposure during that period of  $1.9\text{ mW}/\text{cm}^2$ . These data demonstrate that the decreased fetal weight seen in microwave-irradiated mice (-10 %) detected in utero and is retained at least 7 days after birth. Evidence from other published studies is presented to show that the retarded growth is persistent and might be interpreted as permanent stunting.

Suvorov et al. (1994) studied the biological action of physical factors in the critical periods of embryogenesis. The critical period in a chicken embryonic development (the 10-13 days of incubation) is revealed under total electromagnetic radiation. EMR is a physiologically active irritant which can influence functional state of the brain. The increased absorption of electromagnetic energy takes place in this incubation period. Its dynamics within 20 days of embryonic development has phasic, up and down character.

Electromagnetic exposure (4 hours a day) in the above mentioned period evokes a delay in embryo adaptive motor behavior (biofeedback learning). Morphological investigation shows significant pathological changes, specifically, destruction of share brain synapses. The delay in embryo hatching for a day is also detected. Radiation exposure within other periods of incubation (3-6th or 12-15th days) was not effective with respect to formation of normal motor pattern in biofeedback experiment. Unfortunately this paper is in Russian and no exposure levels are quoted in the English translation of the abstract.

The Australian ABC television investigative programme, Four Corners, claimed in a documentary on electromagnetic health effects, that in a factory which used radiofrequency heaters for sealing plastics, that of 17 women who worked at sealing machines, 14 had miscarried. Plastic sealers expose the operator to far higher levels than do physiotherapy diathermy devices. In association with the concern in Australia about the reproductive risks from plastic sealers, Brown-Woodman et al. (1989) exposed a set of rats to a repeated exposure to 27.12 MHz EM fields for 5 weeks. A reduction in fertility occurred as indicated by a reduced number of matings in exposed rats compared to sham-exposed rats, and a reduced number of conceptions after exposure. They conclude that:

**"The data suggests that female operators could experience reduced fertility, if they remain close to the console for prolonged periods. This has particular significance for the physiotherapy profession."**

Magras and Xenos (1997) responded to health concerns among residents living in the vicinity of an RF transmission tower in Greece, by placing groups of mice at various locations in relation to the tower. The mice fertility was monitored over several generations and related to the RF exposure.

The Figure below 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.

The Greek study confirms the Australian study, but shows that over several generations the infertility is complete at very low levels of mean RF/MW exposure, Figure 6.

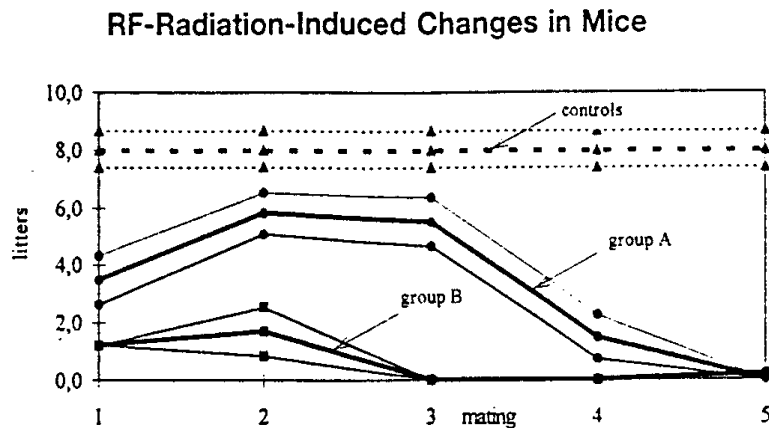


Fig.35. Comparison of the mean values  $\pm$  standard deviation of number of newborns per dam and mating from all experimental groups.

Figure 6: Multigenerational exposure of mice to low level RF leads to complete infertility.

### Summary and conclusions about teratological animal studies:

There is repeated evidence of RF/MW induced infertility in rodents strongly showing that RF/MW have genetically damaged the cells of the animals. This suggests that there could be reproductive and genetic damage in RF/MW exposed humans. The epidemiological studies below confirm that there is, and at very low mean levels of exposure comparable to the exposure of the mice in Greece.

Developing sperm, embryos and fetuses are very vulnerable to damage from toxins. At critical times in utero development damage to certain organs occurs. With sufficient fetal or placenta damage a spontaneous abortion is initiated. At other exposure levels and timing of damage a still birth can result. Thermal levels of microwave exposure has produced retardation of development if exposure is in early pregnancy, and resorptions, still births and hemorrhages with exposure in the second half of the pregnancy.

A much lower microwave dose was associated with significant reduction in birth weight and permanent stunting and slowing of bone hardening. Changes in chick embryo biofeedback learning is observed and testicular atrophy was observed with a mean exposure to a radar-like signal averaging  $0.22 \mu\text{W}/\text{cm}^2$  over a week. Total infertility occurred in mice after 5 weeks of exposure to  $0.17\mu\text{W}/\text{cm}^2$ .

**Thus in 1962 and 1997 it is been shown that chronic low level microwave exposure of animals leads to very significant adverse reproductive effects in males and females down. The effects were still significant at exposures of 0.22 and  $0.17\text{mW}/\text{cm}^2$ . These are close to the level of the lowest published results for calcium ion efflux,  $0.08\text{mW}/\text{cm}^2$  Schwartz et al. (1990).**

RF/MW radiation causes significant birth and reproductive damage in exposed animals down to very low short-term and extremely low average exposure levels.

### Reproductive Health Effects Conclusions:

The ICNIRP (1998) assessment of reproductive effects from RF/MW exposure is severely flawed. Animal studies show that chromosome aberrations and single and double strand DNA breakage occurs with EMR exposure, mice and rats have pregnancy, birth and fertility problems associated with EMR exposure which are also found in exposed human populations. There is consistency within human studies and between human studies and animal studies. Many human studies show statistically significant adverse reproductive outcomes. One large human study, Ouellet-Hellstrom and Stewart (1993), gave a statistically significant dose response relationship. This study allows an exposure assessment to be carried out, along with the multigeneration mice study, Magras and Xenos (1997).

### Exposure Assessment:

Ouellet-Hellstrom and Stewart (1993) report that the microwave exposure was primarily from leakage, which at waist level was measured in the range  $80 - 1200 \mu\text{W}/\text{cm}^2$ . At 15 cm from the source the highest reading was  $15 \text{ mW}/\text{cm}^2$ . The therapist needs to be leaning over the patient during the therapy to receive this dose. This is highly unlikely when the machine is turned on. Even so, this is not sufficient to cause a surface heating of the skin in the few minutes it is likely to involve.

Hocking and Joyner (1995) show that microwaves produce very small SARs with the uterus, in the following figure 7.

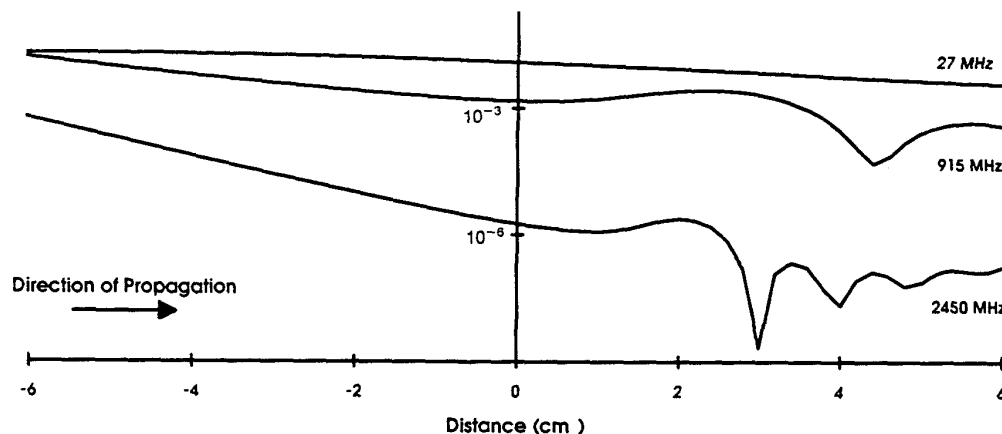


Figure 7: Specific absorption rate (SAR) profile across the uterus for a small woman exposed to  $1 \text{ mW}/\text{cm}^2$ , from Hocking and Joyner (1995).

In their table 2 Hocking and Joyner (1995) show maximum SARs in the uterus for the conditions in Figure 38 for short-wave (27.12 MHz) of  $0.209 \text{ W}/\text{kg}$ , for microwave (915 MHz) of  $0.023 \text{ W}/\text{kg}$  and for microwave (2.45 GHz) of  $0.000027 \text{ W}/\text{kg}$ .

Gandhi (1990) gives the relationship between SAR and temperature increase. The heating rate given is  $0.0045 \times \text{SAR } ^\circ\text{C}/\text{min}$ . With a maximum exposure time per treatment of 5 minutes, and an external field intensity of  $1,200 \mu\text{W}/\text{cm}^2$ , the heating of the fetus will be  $0.0055$ ,  $0.00062$  and  $0.00000073$   $^\circ\text{C}$ , respectively. Not even at  $15 \text{ mW}/\text{cm}^2$  does the short-wave exposure can produce a detectable heating effect in the uterus environment ( $0.071^\circ\text{C}$ ).

Since an acute thermal mechanism can be ruled out it is appropriate to calculate and use the cumulative average dose to determine the range of the exposure regime.

it is not the habit of therapists to stand close to the patient during the diathermy. In many cases the therapist leaves the room while the 15 to 30 minute diathermy is carried out. Hence a conservatively long exposure period of 2 minutes is chosen to be associated with the exposure range of  $80 - 1200 \mu\text{W}/\text{cm}^2$ . The dose-response relationship is expressed in terms of treatments per month. One treatment per month is associated with a mean monthly exposure in the range  $0.0038$  to  $0.056 \mu\text{W}/\text{cm}^2$ , and a mean exposure of  $0.03 \mu\text{W}/\text{cm}^2$ .

	No. of Exposures per Month		Odds Ratio	Exposure Regime ( $\mu\text{W}/\text{cm}^2$ )	
	Mean	Range		Mean	Range
All pregnancies	0		1.00	0.0	-
	<5	(2.5)	1.05	0.08	0.0095-0.14
	5-20	(12.5)	1.50	0.38	0.048 - 0.7
	>20	(25)	1.59	0.75	0.095 - 1.45

This table shows the results from Ouellet-Hellstrom and Stewart (1993) for microwave exposure for all pregnancies. The Number of exposures in brackets is the assumed mean number of treatments in the calculation of the Exposure regime.

There is a 5 % increase in miscarriage associated with a mean microwave exposure of  $0.08 \mu\text{W}/\text{cm}^2$ . This is totally consistent with the calcium ion efflux and animal toxicology experiments.

Hence for reproductive effects the Level of Lowest Observed Adverse Effect is  $0.08 \mu\text{W}/\text{cm}^2$ .

### Biologically Plausible Mechanism:

Calcium ion efflux lead to the survival of damaged cells which carry their chromosome aberrations into future generations of cells. A reduction in melatonin reduces the elimination of free radicals which enhances the chromosome damage. Calcium ion efflux and melatonin reduction also impairs the immune system with allows a greater population of damaged cells to survive. Cells with damaged chromosomes are a known cause of 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, 1997), Sarkar et al. (1994) and Phillips et al. (1998), and to cause chromosome aberrations, Heller and Teixeira-Pinto (1959), Garaj-Vrhovac et al. (1990, 1991, 1992, 1993), Haider et al. (1994) , and many others.

### **Cancer Assessment:**

#### **Laboratory Experiments:**

I have only alluded to some of the cell and animal laboratory studies to demonstrate the consistency of the flawed scientific approach taken by ICNIRP.

ICNIRP, p 506 Totally inappropriately down plays and misrepresents the calcium ion research. It is openly and importantly acknowledged that there calcium ion efflux and influx can occur, depending on the particular combination of intensity, temperature, modulation frequency and carrier frequency, and that there are windows of effect and no effect very close together. An attempt is made to dismiss the effects of alteration of cellular calcium ions by noting that there are “positive and negative” effects and by claiming the an attempted replication, Albert et al. (1987) was unsuccessful ignores dozens of other successful replications showing calcium ion efflux and influx. Albert et al. used chick brains, 147 MHz carrier frequency, and 16 Hz modulation, with an exposure level of 0.75 mW/cm<sup>2</sup>. At the same carrier and modulation frequency chick brains have been shown to have significant efflux at exposure intensities of 0.0014 W/kg three times, 0.006 and 0.008 and 0.002 W/kg. The fact that Albert et al. (1987) found no effect at a very high exposure level of 0.75 mW/cm<sup>2</sup>, equivalent to about 0.3 W/kg, simply means they are outside a window of intensity. Very few high intensity windows have been found.

This is an extremely poor and misleading assessment of calcium ion research and its health effect significance as set out above. The ICNIRP assessment totally misrepresents the nature and implications of laboratory experiments in their consistent efforts to dismiss evidence of effects.

The effect of microwaves neoplastically transforming a standard mice embryo cell line, a cell line which has been used several times in chemical carcinogen assessment are treated in the same inaccurately dismissive manner, p507, referring to the work of Balcer-Kubiczek and Harrison (1991). These researchers carried out a series of very careful and extensive laboratory assessments using a standard mouse cell line. One of their most significant results is presented below, Figure 8.

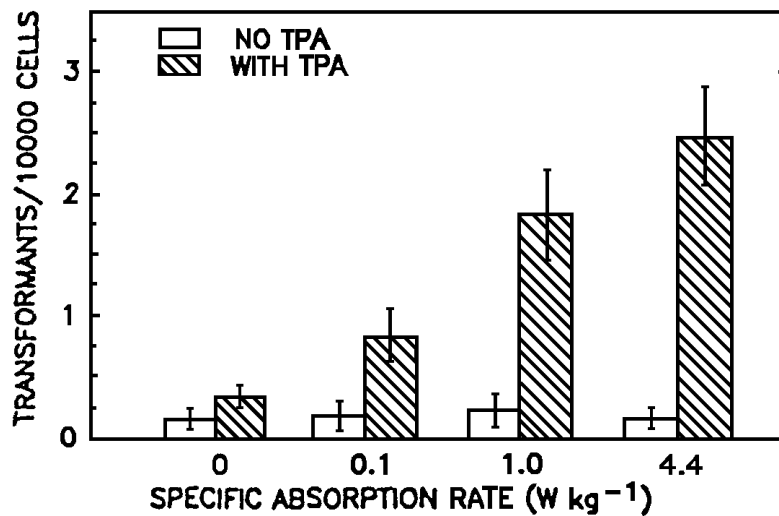


Figure 8: Dose response relationship for the induction of neoplastic transformation of C3H/10T1/2 cells by a 24 h exposure to 2.45 GHz microwaves at specific absorption rate indicated on the abscissa with or without TPA post-treatment for 8 weeks (Balcer-Kubiczek and Harrison (1991)).

This is a clear and simple result. TPA is a known and widely used cancer promotor. Together with TPA, microwaves significantly increase the number of neoplastically transformed cells in a significant dose response manner. states in a book chapter in 1995, Balcer-Kubiczek (1995):

**“In 1985 we published the first evidence indicative of EMF carcinogenesis at the cellular level.”**

Further on Dr Balcer-Kubiczek states:

**“The mouse data of Szmigielski et al. (1982) are also consistent with a general picture emerging from our in vitro data, in that 2.45 GHz microwaves, and possibly 60 Hz magnetic fields, seem to act as an initiator or carcinogen, rather than as a promotor of malignant transformation.”**

This is a very different and much stronger view than expressed by the ICNIRP review when it describes this work by saying: “This finding suggests that pulsed microwaves may exert co-carcinogenic effects in combination with a chemical agent that increases the rate of cell proliferation of transformed cells. To date, there have been no attempts to replicate this finding, and its implications are unclear.”

The use of the word “may” when the effect clearly does occur is wrong. The implication is clear if you want to see it, which the reviewer obviously does not. In context, animal skin, when treated with TPA or similar chemical cancer promoters, has the rate of cancer cell formation increased by microwaves. This experiment shows that it also does happen at the cellular level. That is, microwaves are carcinogenic at the tissue and cellular level. It is then not surprising that epidemiological studies also show that RF/MW increase cancer. But this review ignores and misrepresents that evidence too.

The extensive research into Melatonin and its implications are totally ignored.



## **Epidemiology of Cancer:**

ICNIRP (1998) p 504 concludes by referencing one review (UNEP/WHO/IRPA 1993) and 13 studies concludes: "Overall the results of the small number of epidemiological studies published provide only limited information on cancer risk."

The UNEP/WHO/IRPA 1993 contains errors, which are propagated through to the ICNIRP assessment.

Thirteen studies are cited directly:

1. Barron and Baraff (1958): The study group is too small (226) and the follow up period (4-13 years from first exposure) is too short to detect cancer. Cancer is not one of the paper's study's chosen outcomes. It is grossly dishonest and misleading to include this paper in a cancer assessment and to cite it as showing that there are no cancer risks from exposure to radar.
2. Robinette et al. (1980): Is widely claimed to show no effects when its data does show significant adverse human health effects.
3. Lilienfeld et al. (1978): Is widely claimed to show no effects when its data does show significant adverse human health effects.
4. Selvin et al. (1992): Is widely claimed to show no effects when it was aiming to develop an epidemiological method relating to spatial clustering. Its data does show significant adverse human health effects
5. Beall et al. (1996): Is quoted by ICNIRP as failing to show significant increases in nervous system tumours, when it does.
6. Grayson (1996) Is quoted by ICNIRP as failing to show significant increases in nervous system tumours, when it does.
7. Rothman et al. (1996a): ICNIRP acknowledges that it is still too early to observe an effect of cancer incidence and mortality from mobile telephone use as yet.
8. Rothman et al (1997b) ICNIRP acknowledges that it is still too early to observe an effect of cancer incidence and mortality from mobile telephone use as yet.
9. Szmigielski et al. (1988): finds significant increases in leukaemia incidence and mortality among Polish Military personnel exposed to radio and radar, which ICNIRP says is difficult to interpret because neither the size of the population nor the exposure levels are clearly stated. In fact the Polish Military microwave exposure regime is presented and the group is described by the authors as "large and well controlled".
10. Szmigielski (1996): ICNIRP acknowledges that Szmigielski found significant increases in leukaemia but criticizes the exposure assessment. Again, the exposure regime is

well described, but as in all large population studies, individual exposures are not monitored but group exposures can be well classified.

11. Hocking et al. (1996), (12.) Dolk et al. (1997a) and (13.) Dolk et al. (1997b) are acknowledged as “suggesting a local increase in leukaemia incidence” in population living in the vicinity of TV/FM transmission towers, but ICNIRP calls the results “Inconclusive”.

The conclusion that the results are “inconclusive” is mistakenly based on flawed previous assessments, UNEP/WHO/IRPA 1993, failure to review the data on effects (2, 3, and 4), incorrect claims of no significant effects when such effects are reported (5 and 6), inappropriate dismissal of significant studies (9 and 10) and inappropriate devaluing of residential studies (11, 12 and 13). A systematic and independent analysis of the data in these papers reveals a consistent and significant increase in cancer in these set of studies. There also exists many other studies which add considerable weight to this conclusion.

In order to scientifically justify the conclusion that RF/MW is a human carcinogen based on sound and extensive epidemiological research, backed by extensive animal toxicology and cellular research, a great deal of material must be presented, considered and taken into account. Such a presentation is required here and will be given.

First I will set out some principles and then present the data.

### **Significant Principles:**

- A significant problem of principle is involved here. It is easy to make a simple claim to dismiss as study of effects while it takes a substantial presentation to correct such a misleading claim.

Simple incorrect arguments are consistently used and internally reinforced in review after review. Claims are simply made and to correct them requires detailed and comprehensive scientific analysis and review.

- It is easier to present biased conclusions than to falsify data.
- Every scientist is a person with a degree of subjectivity and bias. Hence science uses principles and methods involving careful checking and peer review. Basic scientific training makes it very difficult (though not impossible) for a scientist to falsify data.
- Analysis of data is more subject to error and bias in its use and interpretation. Errors can be simple arithmetic errors or errors in programming and data entry. Checking procedures are usually in place to significantly reduce the chance of this occurring.
- Subjective bias is frequently involved in the choice and interpretation of statistics which makes the principles of the application of statistical methods and agreed systems of interpretation vital.

- Epidemiology is the basic science of preventive medicine and public health, and biostatistics is the quantitative foundation of epidemiology, Jekel et al. (1996).
- The test of statistical significance:
 

In epidemiology it is agreed that a statistically significant result is one which reaches the 1-in-20 or 5 % threshold for statistical probability. In calculating the value of the statistical probability or p-value (p), a single direction effect is tested against a one-tail distribution while a bi-directional effect is tested against a two-tailed distribution. This requires half the population to achieve statistical significance when searching for an adverse effect than when the hypothesis involves the possibility of a positive and a negative effect.
- Epidemiology deals with populations whereas the ICNIRP guideline is based on thermal effects on individuals, rather than evidence of disease in large populations. An important characteristic of epidemiology is its ecologic perspective. People are seen not only as individual organisms but also as members of communities in a social context.
- Classical epidemiologist studies the community origins of health problems. Classical epidemiologists are interested in discovering risk factors that might be altered in a population to prevent or delay disease or death.
- Death is only one of the outcomes of concern. In general many more people are made ill by a disease agent than those who die of it. Illness has a significant personal, social and economic cost which makes the prevention of illness a worthy goal.

#### **Detailed evaluation of cited papers and reports:**

##### **1. Barron and Baraff (1958): "Medical considerations of exposure to microwaves (radar)"**

The initial study contained 226 radar exposed workers, and 88 in the control group. In the radar group 37 had 5 - 13 years of exposure and 83 has 2 - 5 years. In the extended study 109 new workers were added placing them generally in the 2-5 year group. This is far too short a time for most cancers to appear, with latencies typically between 8 and 30 years. An article in the same volume of the J.A.M.A. records the initiation of a study on thousands of U.K. Radiologists, some of whom had started work in 1920. It is stated that in 1958 it is too early to see an increase in X-ray induced cancer and the sample is too small.

With the working age incidence of all cancers at about 100 per 100,000 per year, over the 4 years of this study the probable number of normally occurring cancers would be 0.9.

**To include this study in a cancer risk assessment is knowingly misleading and deceptive. This level of bias and error is unbecoming of an international assessment**

**of quality and merit. This, along with several other similar examples, must bring the scientific objectivity and professional credibility of the person or group who produce this assessment into serious question.**

This paper does report a high incidence of headache and nervousness, so called subjective or neurasthenic symptoms, consistent with stronger later findings, e.g. Djordevic et al. (1979), Lilienfeld et al. (1978), Hocking (1998), Mild et al. (1998) and Frey (1998) and significantly higher red blood cell counts and lower monocytes, and elevated white blood cell counts and reduced eosinophils and polymorphonuclear cells in the radar-exposed group compared with the control group. Altered blood cell counts were also found in radar exposed groups in the U.S. Embassy in Moscow, Tonascia and Tonascia (1976) and in radar technicians, Goldini (1990).

Barron and Barraf did not assay for chromosome aberrations and DNA breakage. Laboratory techniques were not as advanced in 1958 and they are now.

## **2. Robinette et al. (1980): "Effects upon health of occupational exposure to microwave radiation (radar)"**

- A report prepared for and funded by the U.S. Navy to determine whether service men who had been exposed to radio and radar signals on aircraft carriers during the Korean War, showed any adverse health effects at least two decades later.

An explanation of the background to the project and some of its results were reported to a symposium in 1979 and published in the Bulletin of the New York Academy of Medicine, Silverman (1979). The researchers had a difficulty in classifying the exposure of the subjects but went to considerable pains to reduce uncertainty.

All studies involving large populations have difficulty in separating exposed and unexposed members of that population. The ICNIRP assessors uncritically take this as a no effects study without reference to its exposure difficulties but consistently reject studies which do report effects, based on their alleged exposure uncertainties. This reveals a clear bias against finding effects.

The probability of exposure dilution was seriously addressed by Robinette, Silverman and Jablon. They sought occupational groups which naval assessors classified as unexposed and exposed primarily with unexposed groups being in "equipment operation" while exposed groups were involved with "equipment repair".

### **An error in exposure classification ?:**

In discussing the allocation of groups to exposure categories Robinette et al. state: "Radiomen and radar operators, whose duties keep them far from radar pulse generators and antennae, are exposed to levels well below 1 mW/cm<sup>2</sup>, whereas fire control technicians and electronic technicians are exposed to higher levels in the course of their duties." No mention is made as to why aircraft electrician's mates, men involved in repairs, were allocated to the low exposure group. This is a probable error.

Aircraft electrician's mates (AE) work with Aircraft electronics technicians in repairing equipment on planes and spend a great deal of time on the flight deck exposed to radar, and exposed to radar while it is tested under repair. The AE group should be in the high exposure group. Retaining AE in the low exposure group is a clear highly probable dilution factor.

It is observed that the AE group has rates of death and malignancies in line with the highly exposed FT and AT groups, (FT : Fire Control Technician), and hence including the AE group in the unexposed group provides a significant dilution of the effects. The two aviation technician groups AE and AT are linked by Robinette et al. through their common high incidence of aircraft accidents.

### **Hazard Number Assessment:**

Amongst those who were originally allocated to the exposed group, i.e. ET, FT and AT, around 5 % (1233 men) were randomly chosen to be assessed for individual exposure through a job matrix estimate of their Hazard Number. The results of this are in the following table:

Hazard Number	Electronics Technician (ET) %	Fire Control Technician (FT) %	Aviation Electronics Technician (AT) %
0	27.8	6.6	12.5
1 - 2000	28.3	23.4	16.9
2000-5000	20.0	31.1	17.6
5001+	10.6	25.8	48.6
Unknown	13.3	13.1	4.3
Mean HN	1770	3035	3782

There is a clear overlap between these groups with all groups having a large number in the 2000 + Hazard Number. There is a clear gradient in the proportion of each group with 5000+ Hazard Number.

At a preliminary presentation of the results at a seminar in Rockland, Maryland in 1977, Robinette and Silverman (1977) the follow up discussion records a former U.S. Coast electronic technician saying that when he was operating as a radioman he occasionally had his hands inside the equipment while it was operating. Hence the assumed low exposure group contains a group which is directly involved in repair and should be in the high exposure group, a significant number of men who are in the high exposure group were assessed as having low exposure (e.g. HN<2000), and low exposure operational staff are often highly exposed.

Two approaches can be used to reduce the uncertainties posed by these exposure uncertainties. The AE group, which has been inappropriately allocated to the low exposure group, could be removed altogether or moved to the high exposure group. The impact of both of these should be assessed. The second approach is to dichotomize the exposure group to provide greater certainty of exposure difference through having less chance of an

overlap. The effect of these approaches will be shown after a consideration of the original results.

### **Original Mortality Results:**

In presenting their original results Robinette et al. used a Mortality Ratio (MR) to standardize the data for age distributions within the groups. Their table 6 lists the MRs for All Diseases and for the Malignant Neoplasms. In every case, except "Other malignant neoplasms", the FT+AT group has the highest MR. Robinette et al. compare the lower Hazard Number group (ET) with the higher Hazard Number groups (FT+AT) and note that FT+AT is significantly higher than ET for "All Diseases",  $p < 0.01$ , and for "Other Diseases",  $p < 0.01$ .

Robinette et al.'s Table 9 lists the MRs for the group with assessed Hazard Number, ranked by Hazard Number.

The 5001+ group has the highest MR for all disease categories except "Digestive Organs" and "Other Disease". Within the High Exposure groups there is a significant dose response relationship for "All Diseases" with MR = 0.82, 0.91 and 1.23 for HZ = 0, 1-5000 and 5001+ respectively,  $p = 0.03$ .

Leukaemia is discussed in relation to Table 8. This reports 20 deaths in the low exposure group and 26 in the high exposure group. This shows a distinct dose response gradient from low, ET, FT+AT, with rates of 0.96, 1.16 and 1.57 respectively. The FT+AT/Low Risk Ratio is  $RR = 1.63$ , 95%CI: 0.78-3.40, which is not statistically significant. Comparing the very highly exposed AT group with the very low exposure RD+RM group gives rates of 3.055 and 1.033,  $RR = 2.96$ , 95%CI: 1.39-6.32, a highly significant result.

Hence Robinette et al. has imbedded in it several statistically significant results between rates of death in groups with well assessed radar exposure probability.

### **Original Morbidity Results:**

Naval Hospital records were used to assess the risk of increased disease from radar exposure using data from 1952-1954 and 1956-1959 for one analysis, Veteran's Hospital admission data from 1963-76 was used for a second analysis and a third for veteran's admissions up to December 1976.

The early Naval Hospital data set showed little difference between although the FT+AT group was had more total admissions than with the low exposure group and significantly more than the ET group.

A simple comparison between the low and high exposure groups is not very worthwhile because of the dilution problem. Robinette et al. make comparisons with the high exposure group by comparing ET with FT+AT. They note several statistically significant increases, Diseases of the ear, nose and throat, ( $p < 0.01$ ), acute respiratory disease ( $p < 0.01$ ), other respiratory diseases ( $p < 0.02$ ), diseases of the urinary and male genital organs ( $p < 0.05$ ) and accidents, poisonings and violence ( $p < 0.001$ ).

Robinette et al. devalue these results through comparison with the rates in the low exposure group. However, this is not appropriate because of the dilution effect of keeping the AE group in the low exposure group. The results are also diluted by combining FT with AT. In the morbidity statistics FT and AT are never presented separately so that we cannot compare AT with ET to more cleanly dichotomize the data.

The later data sets from Veteran's Administration Hospitals gives a longer time for chronic diseases such as cancer to occur and to accumulate a larger admissions data set. The results are set out in Table 11 of Robinette et al. (1980). The following table is derived directly from Table 11 with the Risk Ratio between the FT+AT group compared to the ET group shown with the 95% confidence interval.

All risk ratios are greater than 1.0 . Apart from Infective parasitic diseases and three marginally non-significant relationships for malignant neoplasms, other mental disorders and Skin (cellular) disorders, the remainder are significant or very significant

Number of hospitalizations and hospitalization rates per 10,000 per year, in VA hospitals, 1963 -1976, by diagnosis and exposure class: US enlisted Naval personnel exposed to microwave radiation during the Korean War period.

VA diagnostic class	Total		High exposures		RR	95% CI	
	No.	Rate	ET No.	ET Rate			FT + AT No.
Infective, parasitic	42	1.5	24	1.3	18	1.9	1.46 0.79-2.69
Neoplasms, malignant	34	1.2	17	1.0	17	1.8	1.80 0.92-3.53
Neoplasms, other	26	0.9	9	0.5	17	1.8	3.60 1.60-8.08
Allergic, endocrine system, metabolic and nutritional diseases	77	2.8	41	2.3	36	3.8	1.65 1.05-2.58
Blood, blood-forming organs	17	0.6	5	0.3	12	1.3	4.33 1.53-12.3
Alcoholism	105	3.8	45	2.5	60	6.3	2.52 1.71-3.71
Other mental disorders	276	10.1	166	9.3	110	11.6	1.25 0.98-1.58
Nervous system, sense organs	106	3.9	58	3.2	48	5.1	1.59 1.08-2.33
Circulatory	123	4.5	68	3.8	55	5.8	1.53 1.07-2.18
Respiratory	80	2.9	43	2.4	37	3.9	1.63 1.05-2.53
Digestive	255	9.3	132	7.4	123	13.0	1.76 1.38-2.25
Genitourinary	82	3.0	45	2.5	37	3.9	1.56 1.01-2.41
Skin, cellular	61	2.2	33	1.8	28	2.9	1.61 0.97-2.66
Bones, organs of movement	80	2.9	36	2.0	44	4.6	2.30 1.48-3.57
Trauma	108	3.9	53	3.0	55	5.8	1.93 1.32-2.81
Symptoms, ill-defined conditions, special exams and other	151	5.5	85	4.8	66	6.9	1.44 1.04-1.99
Person-years (1000)	27.39		17.89		9.50		

The next table shows the total cumulative data for men receiving VA compensation up to December 1976, from Robinette et al. Table 12. Again the vast majority of symptoms (apart from Nerves, and Genitourinary) are marginally significant to very significantly greater for the higher exposed FT+AT group compared to the lower exposed ET group. Note that the real differences between these groups will be considerably greater since the

FT+AT group contains many people with a history of low exposure (around 30%) and the ET group contains many with a high exposure (around 11%). The following table shows the effect of comparing FT+AT with ET for the mortality statistics.

**Table: Number of men receiving VA compensation and pension, December 1976 and rates per 1000 men per year by diagnosis and exposure class, and Risk Ratio (FT+AT)/ET.**

Diagnosis:	ET		FT+AT		Risk Ratio	
	No.	Rate	No.	Rate	RR	95% CI
Musculoskeletal	115	8.8	119	16.9	1.93	1.69-2.20
Organs of special sense	49	3.7	42	6.0	1.62	1.31-2.00
Systematic conditions	3	0.2	5	0.7	3.50	1.69-7.26
Respiratory	55	4.2	51	7.3	1.74	1.43-2.11
Cardiovascular	43	3.3	47	6.7	2.03	1.64-2.51
Digestive	74	5.7	55	7.8	1.37	1.15-1.64
Genitourinary	31	2.4	10	2.7	1.13	0.79-1.63
Skin	83	6.3	58	8.2	1.30	1.10-1.54
Endocrine	15	1.1	11	1.6	1.45	0.97-2.16
Neurological	21	1.6	16	2.3	1.44	1.03-2.01
Nerves	15	1.1	3	0.4	0.36	0.19-0.68
Mental Conditions	51	3.9	46	6.5	1.67	1.36-2.05

**Table: Mortality Incidence per 1000 and Risk Ratio (AT/ET) as an indication of the high exposure (AT) to low exposure (ET) difference.**

	Low	High	Risk Ratio	95 % CI
Causes of Death				
All Deaths	33.7	60.5	1.79	1.52 - 2.12
Accidental Death	13.5	29.6	2.20	1.72 - 2.82
Motor Vehicle Death	6.3	6.1	0.97	0.60 - 1.59
Suicide, Homicide, Trauma	4.4	6.1	1.38	0.83 - 2.29
Suicide	3.4	2.7	0.80	0.39 - 1.63
All Diseases	15.2	23.5	1.55	1.19 - 2.01
Malignant Neoplasms	5.0	8.2	1.66	1.06 - 2.60
Digestive and Peritoneum	1.1	1.2	1.07	0.35 - 3.21
Respiratory	1.2	2.1	1.75	0.72 - 4.25
Eye, Brain, CNS (FT/ET)	0.4	0.9	2.40	0.57 - 10.03
Skin	0.2	0.6	2.66	0.45 - 15.94
Lymphatic and Hematopoietic	1.4	3.1	2.22	1.02 - 4.81
Circulatory System Disease	7.6	9.5	1.24	0.83 - 1.85
Digestive System Disease	0.8	2.7	3.27	1.35 - 7.89
Other Diseases	1.6	2.7	1.71	0.78 - 3.74

This shows elevated Risk Ratios for all causes of death except motor vehicle and suicide. Significant increases in mortality were found for All Diseases, Malignant Neoplasms, and Lymphatic and Hematopoietic cancer. Very significant increases were found for All Causes of death, Accidental Death and Death from diseases of the Digestive System.



In that the original data shows a significant increase in morbidity and mortality incidence for the high exposure group, the stated conclusion in the abstract of the paper is **clearly wrong and misleading** when it states:

**“No adverse effects were detected in these indices that could be attributed to potential microwave radiation exposures during the period 1950-54.”**

The paper's incorrect conclusion is partly caused by misclassification of the Aircraft Electrician's Mate as an operational job when it is a repair job, thus significantly diluting the difference between the low and high exposure groups. In addition they carried out a job matrix assessment of three of the groups and then failed to use it to compare and contrast the groups which were identified to be low exposure and high exposure from this exposure assessment. They also failed to report that their findings which did show significant effects would in reality have stronger relationships if the data was much less dilute although exposure classification and data dilution was discussed as a limiting factor.

Dr Ruey Lin of the Maryland Department of Health, Lin (1985) reviewed this study and concluded that the exposed and control groups were in fact both exposed groups, leading to an under-estimate of the identified effects.

Robinette et al. (1980) stress that while considering the data about death, other disease would have been present which would not be reported:

**“Further, it is possible that effects involving cardiovascular, endocrine and central nervous system do exist, but are transient, disappearing with the termination of exposure or soon thereafter, or are not perceived to be sufficiently consequential to result in admission to hospital.”**

Robinette et al. do not compare rates for their sample with the general population even though they admit that every one in their sample has some exposure and hence the control group is called the “Low exposure” group. The standardized mortality for death from cancer for all causes of cancer in Males in New Zealand in the 25 to 49 age group is 2.21 per 1000. All of the Korean War veterans occupational groups studied have a far higher rate than this and all would have been exposed to more radar signals than the New Zealand population. Even the lowest rate for Radiomen at 4.21/1000 is 1.9 times higher than the New Zealand age adjusted male all cancer rate. The highest rate for Aviation Electronics Technicians (8.25/1000) is 3.73 times higher.

**Robinette et al. (1980) indicates, in a more than two decade study involving about 40,000 people, when a job matrix assessed group exposure classification was applied, and a low exposure group was compared with a high exposure group, that many statistically significant and very significant increases in sickness and death occur from exposure to radar and radio RF/MW radiation during the Korean War.**

**3. U.S. Embassy in Moscow: Lilienfeld, Tonascia, Tonascia, Libauer and Cauthen (1978). "Foreign Service Health Status Study - evaluation of health status of foreign service employees from selected eastern European posts"**

Lilienfeld et al. (1978) investigated the health effects of the staff and children who lived or worked in the U.S. Embassy in Moscow during a prolonged period when the embassy was

being irradiated by a Soviet radar. This study is frequently cited, along with the Korean War Study above, as indicating that there were no observed health effects from prolonged exposure to low intensity radar signals. It is then used as a reason to counter other studies which do show adverse health effects, as it is here in ICNIRP.

As with Robinette et al. (1980), the data presented in the Lilienfeld contract report is contrary to that stated in the report's conclusions.

The Lilienfeld data shows a significant increase in:

- neurological symptoms
- blood cell counts
- chromosome aberrations, and
- cancer in children and adults.

These symptoms are associated with chronic exposure to very low intensity pulsed microwaves in the range 1 to  $2\mu\text{W}/\text{cm}^2$ .

### **Study Structure:**

A two year study was carried out by the School of Hygiene and Public Health at Johns Hopkins University on behalf of the State Department, starting in 1976 following extensive publicity about the Russians irradiating the U.S. embassy in Moscow with radars. A 23 year study period was chosen, involving 1827 employees at the Moscow Embassy and 3000 of their dependants, and a comparison group of 2561 employees at eight other Eastern European Embassies and 5000 of their dependants.

For the purposes of the study, persons in the Moscow population were divided into three subgroups: the exposed, the unexposed and those with questionable exposure. Some comparisons were made internally and come with the comparison embassy populations.

### **Exposure Measurements:**

The radar was aimed at the Embassy from a small distance away. The direction and intensity of the microwave signal changed in 1975 but it was always directed at the upper floors of the Chancery, Silverman (1980). Measurements of maximum exposure were made at or near the windows of the upper central building. Exposures according to time period were determined for individual floors in the living and working areas. Apartment complexes in Moscow distant from the Chancery were monitored every few months and only background ( $1\mu\text{W}/\text{cm}^2$ ) were found, Silverman (1980). These are high background levels compared to the median for 15 U.S. cities surveyed in 1979/80 of  $0.005\mu\text{W}/\text{cm}^2$ , Tell and Mantiply (1980). The maximum exposure and exposed areas by time period were summarized by the State Department as follows:

Time Period	Exposed area of Chancery	Maximum Exposure
1953 to May 3, 1975	West Facade	Maximum of $5\mu\text{W}/\text{cm}^2$ , 9h/day
June 1975 to February 7, 1976	South and East	$15\mu\text{W}/\text{cm}^2$ , 18h/day

Since February 7, 1976	Facade South and East Facade	Fractions of $1\mu\text{W}/\text{cm}^2$ , 18h/day
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The daily mean maximum exposures (from the table above) were  $1.88\mu\text{W}/\text{cm}^2$  up to 1975,  $11.25\mu\text{W}/\text{cm}^2$ , June 1975 - February 7 1976, and less than  $0.7\mu\text{W}/\text{cm}^2$  since Feb 7th 1976. The higher exposure was for less than 10 months. The mean maximum exposure from 1953 - 1976 was  $2.17\mu\text{W}/\text{cm}^2$ .

Hence  $2.2\mu\text{W}/\text{cm}^2$  is the maximum possible long term exposure but ever persons mean exposure would be somewhat less than this because they worked away from the office which was maximally exposed, on other floors, lived even further away, had holidays etc.. A background of  $1\mu\text{W}/\text{cm}^2$  means that it is likely that in Moscow, nobody can be used as an unexposed group. Hence the comparisons with other Eastern Embassies and standardized morbidity and mortality ratios are preferred.

### Neurological Symptoms (Table 6.31):

A wide range of neurological symptoms in Lilienfeld et al. show high Risk Ratios but they don't reach statistical significance because of small sample sizes. Depression, Irritability, Difficulty with Concentrating and Memory Loss are highly significant.

Table: Standardized Morbidity Ratio for the rate of occurrence per 1000 person years, after the first tour of duty, for Moscow and comparison male employees, after Table 6.31

Symptom	SMBR			p-value
	Moscow	Comparison	RR	
Depression	1.3	0.73	1.78	0.004
Migraine	1.8	0.97	1.86	N.S.
Irritability	1.3	0.66	1.97	0.009
Nervous Disorders	1.5	0.64	2.34	N.S.
Difficulty in Concentrating	1.4	0.52	2.69	0.001
Memory Loss	1.6	0.50	3.2	0.008
Dizziness	1.2	0.85	1.41	N.S.
Finger Tremor	1.3	0.71	1.83	N.S.
Hallucinations	1.5	0.59	2.54	--
Insomnia	1.1	0.90	1.22	N.S.
Neurosis	1.4	0.62	2.26	--
Other Symptoms	1.3	0.76	1.71	N.S.

RF radiation is emitted by power lines and depression, anxiety and suicide has been associated with living near power lines, e.g. Perry et al. (1981), Beale et al. (1997), Zyss (1997) and Verkasalo et al. (1997) and in occupational exposures, e.g. Bobhomme-Faivre et al. (1998), who also found a significant fall in total lymphocytes and CD4, CD3 and CD2 lymphocytes, as well as a rise in natural killer (NK) cells. The use of cell phones is now causally related (dose response relationship) to Memory Loss, Headache and Concentrations problems, among others, Mild et al. (1998), Hocking (1998). There also is a growing number of occupational studies showing that workers exposed to RF/MW have

elevated incidence of psychological disorders, for example, Antoniazzi et al. (1983 and 1988) and Marraccini et al. (1990)

### Blood Sample Results:

The George Washington University report (August 4, 1969) entitled "Final report on contracts between the medical division, Department of State and the Reproductive Genetics Unit, of the George Washington University" covers analyses of blood from between 21/2/66-30/6/69. This covers the period when the external wall exposure was up to  $5\mu\text{W}/\text{cm}^2$  for 9 hours/day, averaging  $1.9\mu\text{W}/\text{cm}^2$ . They were analyzing for mutagenic effects by identifying chromosomal damage.

The report includes the comment: "The Contractor's opinion lies between these two extremes and the current risk is in a human adult population most likely exists solely in reproduction, however, some workers cite similarities in early malignancy."

**Table: Hematological Tests of chromosome and other damage in the blood of U.S. Foreign Service Workers from Moscow and other Eastern Embassies.**

Scale	Mutagenic Level	Clinical Significance	Patient X-Numbers
5	Extreme	Definite	None
4	Severe	Questionable	73,74,76,79,84,102
3.5	Intermediate		72,83,91,99,103
3	Moderate	Suspect	70,71,93,97,98,100,104
2.5	Intermediate		75,87,90,94,96
2	Mild	Questionable	69,81,85,92,95
1	Normal	None	77,78,80,82,86,101

A further report by James Tonascia and Susan Tonascia, 7 October 1976, entitled "Hematology Study", and included employees who arrived in Moscow before December 1975. The sample totaled 213 individuals from Moscow and they were compared with 981 other Foreign Service employees. The white blood cell counts "are strikingly higher in the Moscow Group. This means that the total as well as each of the four cell types are higher, ranging from 87% increase in eosinophil count to a 15 % increase in neutrophil count. The total white cell count was 25 % higher, lymphocytes were increased 41 %, and monocytes 31 %. They concluded :

**"There was a marked difference in white blood cell parameters. The total count as well as the counts for each individual cell type were substantially higher in Moscow than in the comparison group. This was especially true for the eosinophil (granular leukocyte) and lymphocyte counts."**

Leukocytes changes are related to Leukaemia and lymphocytes are involved in the immune systems. The microwave exposed Moscow group had significant alterations of blood cell counts. White blood cell counts were also found to alter in Lockheed employees frequently exposed to short-term, high intensity microwave exposures, but their WBC rose significantly.

### Cancer Increases:

Cancer rates in the Moscow Group were summarized in Tables 5.6, 7.12 and 7.16 of Lilienfeld (1978). Goldsmith (1995) reports:

Adult foreign service workers and their spouses showed marked increases in a number of cancers compared with the number expected for the same age-adjusted population.

After reviewing this data, an eminent epidemiologist, Professor John Goldsmith, referring to a “recent draft of criteria for health protection” which claims: “No effect on life span or cause of death of 1,800 employees and 3000 dependents of the U.S. Embassy personnel.”, states:

**“To ignore these findings on the basis of “No effect on life span or cause of death” in setting human exposure standards is wrong. In the first place the criteria are too narrow; mortality is not the only relevant end-point.”**

Table: Cancer Mortality rates for employees and dependents at the U.S. Embassy in Moscow:

Symptom	Moscow	Expected	SMR	p-value
All Cancer	33	24.83	1.33	N.S.
Adult Leukaemia	2	0.8	2.5	N.S.
Genital Cancer, Female	4	0.8	5.0	
Adult Brain Tumour	2	0.10	20.0	<0.05
Female Breast Cancer	2	0.50	4.0	<0.05
Childhood Leukaemia	4	1.33	3.0	<0.05

All of the above elevated rate ratios and significant increases in cancer death and neurological symptoms are associated with mean exposures somewhat less than  $2\mu\text{W}/\text{cm}^2$ . These results are backed up by measurements of significant ( $p < 0.001$ ) changes in blood counts and increases in chromosome aberrations.

This is an extremely significant study that has been replicated and confirmed in a number of long-term animal studies. Three are especially relevant, but one especially, shows broken chromosomes and increased cancers in RF/MW exposed mice.

However, just as the U.S. State Department tried to dismiss evidence of adverse effects in their staff in Moscow and other embassies by altering the conclusions of the Lilienfeld report and failing to carry out the follow-up studies he recommended, Goldsmith (1995), so the following two rodent experiments, funded by the U.S. Air Force, placed the authors under severe pressure not to show nor acknowledge significant effects.

**Chou et al. (1980)** report on exposing 100 rats to a radar signal and compared them with 100 sham exposed rats. The exposure regime exposed rats to 2.45 GHz microwaves pulsed at 800 pps. The power was measured at 0.144W which is equivalent to 0.4 W/kg for a 200g rat and 0.15 W/kg for 800g rats, for 21.5 h/day from the 8th week of age, for a period of 25 months. This corresponds to a mean lifetime SAR of 0.17W/kg, or about

425 $\mu$ W/cm<sup>2</sup>. The result was a significant increase in malignancies, 18 compared to 5, (RR = 3.6, 95% CI 1.34-9.70) and in benign endocrine tumours, 9 compared to 2 (RR = 4.5, 95%CI: 1.0-20.8). Thus non-thermal chronic exposure of rats to a radar signal produced a significant increase in malignant and benign tumours.

ICNIRP describes this as " In a large study of rats exposed to microwaves for up to 25 mo. An excess of primary malignancies was noted in exposed rats relative to controls." An conclude: "Taken as a whole, the results of this study cannot be interpreted as indicating a tumor-initiating effect of microwave fields."

What ICNIRP calls "an excess" the authors, Chou et al., describe as "a significant increase of primary malignancies in exposed rats vs. incidence in controls is a provocative finding" . It is scientifically misleading to term a "significant increase" as "an excess". The term "excess" is used when the increase is not significant.

The authors, of this U.S. Air Force funded research, then go on to say "but the biological significance of this effect in the absence of truncated longevity is conjectural."

Neither the sham exposed nor the microwave-exposed mice of these Sprague-Dawley rats had reached the end of their natural life after 25 months. The fact that the act of exposing a group to non-thermal microwaves for 25 months significantly increased malignancies and non-malignant tumours when taken as a whole, indicates that microwaves are mutagenic and carcinogenic. This is a significant result. Other rodent experiments confirm this conclusion, but in each case the authors make serious attempts to play down the significance of the results.

**Vijayalaxmi et al. (1997a. and b.)** report on the analysis of 62 exposed cancer-prone mice compared to 58 sham exposed mice. They found a statistically significant increase in chromosome damaged cells ( $p < 0.005$ ) and tumours ( $p < 0.05$ ) in the microwave exposed mice compared to the sham exposed mice. The radiation was 2.45 GHz microwaves, 20 h/day for 18 months, SAR = 1 W/kg. Vijayalaxmi et al. (1997 b.) is a correction of Vijayalaxmi et al. (1997a.).

This was also a U.S. Air Force funded project was published with the conclusion that RF/microwaves "are not genotoxic" because chronic exposure of cancer prone-mice did not show, in their estimation, a statically significant increase in chromosome aberration nor tumours. However, a serious error in calculation occurred and it was pointed out that it when it was corrected the data did show a significant increase in chromosome damage, and there was a significant increase in tumors. In writing the correction the authors continued to dismiss their own result with misleading statements and analysis. The grudgingly acknowledged that their was a significant increase in chromosome damage (micronuclei) but attempted to say it wasn't significant because it was only an increase of 1 cell in 200 sampled, i.e. 0.05 %. In fact a 0.05% increase is not significant. The actual increase in the exposed mice vs the sham exposed mice was from 8 to 9, an increase of 12.5 % which is the statistically significant result they were trying to hide. They also tried to hide the significant increase in tumours by using a two-tailed test for a one-direction effect. The appropriate significance test for a one-directional effect is a one-tailed test. Such as test shows the increase in tumours has  $p < 0.05$ .

This study, of a large sample of mice, according to the original criteria applied, shows that microwaves are genotoxic, increasing chromosome damage and cancer rates. These authors didn't try to dismiss their results because the mice didn't die.

One of the authors of this study, Dr Martin Meltz, appeared for Telecom as an expert witness in the Shirley Primary School case. His evidence was centred around the thesis that there were not reliable studies showing effects and all reliable studies show that there are no effects. He didn't willingly discuss the study of which he was the research leader, Vijayalaxmi et al. (1997a. and b.), and tried very hard to dismiss the significance of its results while avoiding suggesting that his own research was not reliable.

The third mouse study also used cancer-prone mice. Repacholi et al. (1997) also found a highly significant increase in cancer in EMR exposed cancer prone mice, OR = 2.4, 95% CI: 1.3-4.5 . The exposed group was irradiated for 2 periods of half and hour per day using a GSM cellphone signal, i.e. 900 MHz, pulsed at 217 Hz, with a mean expose in the range 0.13-1.4 W/kg during the exposure. This reduces to 0.0054 to 0.058 W/kg averaged over the whole day. This corresponds to 30 - 326 $\mu$ W/cm<sup>2</sup>. This study was primarily funded by Telstra and Motorola was a partner in the study, providing the transmitter and the exposure measurements.

A great deal of money was spent on a PR campaign to play down the significance of the result. The Australian Minister for Communications and Arts, Senator Richard Alston, when asked about the implications of this study through a question in the Senate, replied: "The study suggests that mice should not use cell phones." The researchers, including Dr Repacholi who played a leading role in designing and carrying out the study, used the cancer-proneness of the mice as a dismissive factor. The study was not funded by Telstra, supported by Motorola and the Medical and Health Research Council of Australia to see if cancer prone mice should or should not use cell phones. The whole purpose and design was to test the safety of cell phones for human use. The mice were not even exposed with the antenna next to their head. They were exposed to the far field whereas a cell phone user is exposed to the very intense near field. Even so, the cancer rate was doubled in the GSM exposed mice.

**Hence pulsed microwaves from radar and cell phones causes significant chromosome damage and cancer increase in rodents and in people, in the U.S. Embassy in Moscow, in mean exposures of 1 to 2mW/cm<sup>2</sup>.**

**4. Selvin et al. (1991): "Distance and risk measurements for the analysis of spatial data: a study of childhood cancer" - The Sutra Tower Study, San Francisco.**

Selvin et al. (1992) is widely quoted in national and international reviews as showing no evidence of health effects from a powerful telecommunications tower near a human population. The ICNIRP (1998) statement is typical when it says: "Selvin et al. (1992) reported no increase in cancer risk among children chronically exposed to microwaves radiation from a large microwave transmitter near their homes."

Selvin et al. are concerned with developing statistical data analysis techniques involved in comparing spatial clustering with risk approach to data analysis of potential effects from point sources of exposure. They apply their methods to the white, childhood cancer data for children <21 years living in the vicinity of the Sutra Tower to test the presence of

clustering. They conclude: "None of the three analytic approaches indicates the presence of clustering of childhood cancers associated with the Sutra Tower."

The absence of clustering bears no relevance as to whether the cancer rates relate to exposure from the tower, which is related to exposure levels. Exposure levels near a tower, such as the Sutra Tower, are very complicated in the few km around the tower because of the complex topography of the area, the presence of large institutions, parks and reserves which affect the population density.

The data in Selvin et al. makes a radial analysis of childhood cancer incidence in radial rings centred on the tower, with the mean exposure in each ring being well estimated with reference to a measurement and modelling RF survey which was carried by engineering consultants Hammett and Edison (1997).

### **Exposure regime:**

A computer model, using terrain data, produced a calculated ground level exposure pattern with 2 to 3 km of the Sutra Tower, Hammett and Edison (1997). The following diagram presents the computed data and adds the residence of children with leukaemia or brain tumour who live in the modelled area.

It is important to note that in the absence of terrain effects the exposure levels are higher to the east than to the west because the antennae horizontal patterns point the beams towards the greater populations living to the east, including cities such as Berkeley and Oakland on the eastern side of the Bay. For example, at the 2.5 km ring on Figure 9, to the NNE the calculated exposure is  $0.6\mu\text{W}/\text{cm}^2$ , but to the NNW it is  $0.4\mu\text{W}/\text{cm}^2$ , showing the eastern level is 50 % higher than the western exposure levels. All of the cancer rate patterns show higher rates to the east.

The model calculation shows a peak exposure at the base of the tower of  $24\mu\text{W}/\text{cm}^2$ , decreasing steadily down the slope of the hill on which the tower stands, to around  $2\mu\text{W}/\text{cm}^2$  at a radius of about 0.8 km. Within this ring there are three children with brain tumour (+). There are two more to the SW within a 1 km ring. This is a very high incidence for an area with a low density of homes as the area immediately around the tower is open grasslands, the N/MW slopes are occupied by the University of California Medical Centre and a reservoir lies to the SW. In this analysis a detailed street map of San Francisco was used to obtain a housing density factor (HDF) as a surrogate for population density.



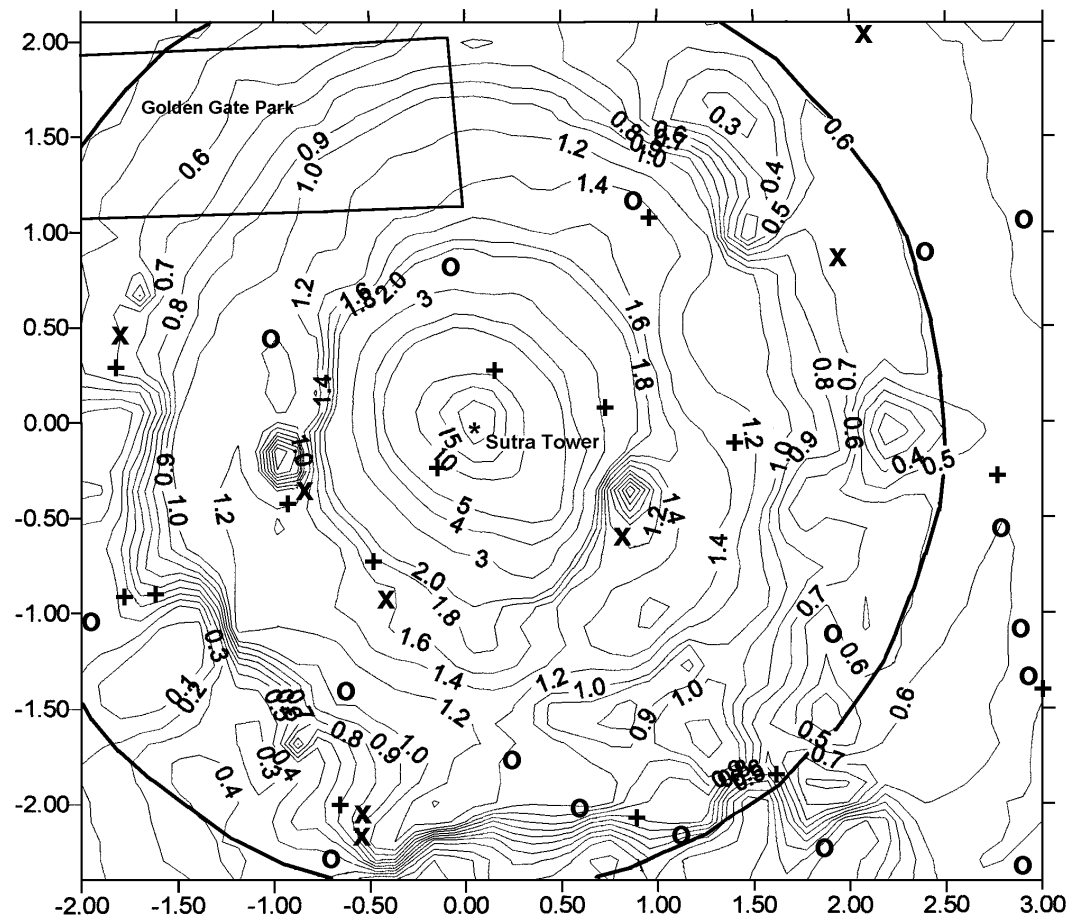


Figure 9: Calculated Radiation exposure pattern in  $\mu\text{W}/\text{cm}^2$ , from the Sutra Tower, San Francisco, Hammett and Edison (1997). Residences of white children <21 years, having cancer in the period 1973-1978, from Selvin et al. (1988). "o" for leukaemia, "+" for brain tumour and "x" for Lymphoma (Hodgkins and Non-Hodgkins). The circle indicates a radius of 2.5 km from the tower. The scales show east/west and north/south distances in km, with 0,0 being the site of the Tower.

For the 1 km ring the HDF is 0.65. The white childhood population density per residential square km is 557.3. Hence the residential area within 1 km is  $2.04\text{km}^2$ , containing an estimated white childhood population of 1138. This contains 5 cases of brain tumour in the period 1973-88, 16 years. Thus within 1 km of the Sutra Tower the rate of childhood brain tumour is 27.5 per 100,000 person years compared to the average for San Francisco of 4.31 / 100,000 p-yrs. This gives a rate ratio of  $\text{RR}=6.37$ , 95% CI: 2.08 - 19.47,  $p<0.001$ . A very highly significant result. Given the significance of this result it is probable that the Sutra Tower has increased the childhood brain tumour rate, especially with 4 to 5 km from the tower. The brain tumour rate for the population who live at least 4.5 km from the Sutra Tower is 2.15 /100,000 p-yrs. At this rate the Risk Ratio of brain tumour for those living within 1 km of the Tower is  $\text{RR} = 11.8$  95% CI: 3.8-36.1 ,  $p<0.001$ .

The Sutra Tower is a very high powered tower with 10 TV and 4 FM stations up to the mid-1990s. The total effective radiated power (ERP) of the TV and FM stations which existed in 1996 was 19,260 kW, with a central radiation height of about 240 m. The base of the

tower is at 253 m. In checking the model calculations, 10 spot readings were taken and compared with calculations. These are plotted in Figure 10.

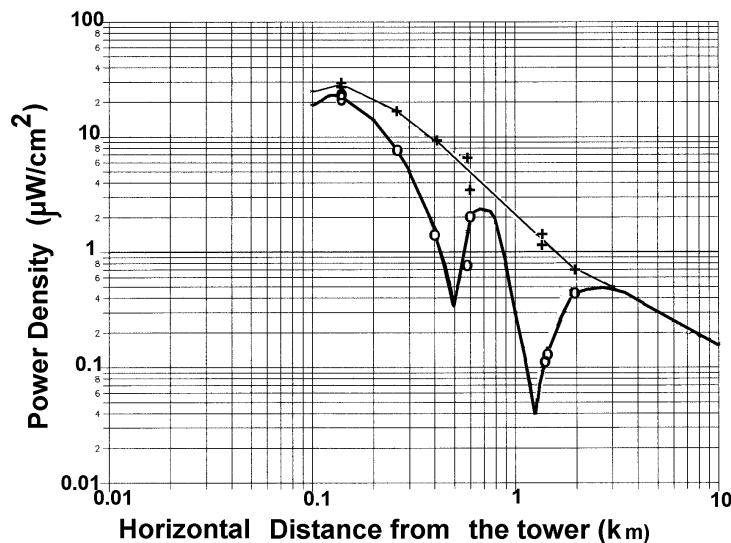


Figure 10: The measured power density (exposure) with distance from the Sutra Tower (bold solid line with circles showing measurement points - lower curve), and the calculated equivalent power density (in  $\mu\text{W}/\text{cm}^2$ ), from Hammett and Edison (1997).

All measurements were lower than calculations. Between 200m and 1.5 km the measurements are 5 to 10 times lower than the calculations suggesting that the mean vertical antenna radiation pattern and expressed by the Relative Field Factor is too high in this range. The figures come together around 2 km. The solid line on the figure, based on measurements, provides the best estimate of the mean exposure with radial distance. There will be local variation due to terrain effects as illustrated on the computer model above.

Very local measurements were taken along local streets in the vicinity of the Tower. Large reductions were measured along the street leading down the hill from the tower, Farview Crescent. The readings, in  $\mu\text{W}/\text{cm}^2$ , taken at 50 m intervals down the road, starting from the top were, 19, 29, 33, 12, 8, 6 and 3. This is over a radial distance from 125m to 360m. The readings follow the measured curve very closely. The measured curve has a minimum at a radial distance of 520 m. The streetscape measurements show at around 500m, reading below the sensitivity of the instrument (recorded as 0.000). The small number of readings given outside this distance are typically  $2\mu\text{W}/\text{cm}^2$ . This too is reflected by the measured curve.

The measurements show a second minimum around 1.25 km, with a third peak at around 2.5 km. Outside this the decline is close to and inverse square law.

These measurements show why a simple assumption of an inverse square law for the whole radial distance is inappropriate and why a cluster of cancers very close to this tower, and after a radial gap, a radial cluster around 1.5 to 4 km, both of which are correlated with exposure to microwave radiation from the Tower.

### **Estimation of personal mean exposure from measured outside power density:**

Once the outdoor mean radiation exposure (power density) is known the personal exposure of the exposed individuals needs to be assessed using a Personal Mean Exposure Factor which takes into account realistic estimates of the typical times spent at home and away, inside and outside.

The signal strength inside is assumed to 20% of the outside signal (thus could be as low as 10 % or less). The assumed ratio is 20% outside:80% inside. The home/away ratio is based on an average daily away time of 8 hours to allow for school, sports visits etc, and annual away ratio based on 6 weeks away annually. Hence the outside exposure is multiplied by  $(0.2 \times 0.2 + 0.8)(18/24)(46/52)$ . Hence the PMEF = 0.56 .

The measured outside signal at the five homes of the children with brain tumour is  $1.74 \mu\text{W}/\text{cm}^2$ . When the PMEF is applied this becomes  $1.0 \mu\text{W}/\text{cm}^2$ .

People who happen to live in a radial ring with very low local exposure, will have lower mean exposures than those who live on either side of the dip. However, since their local movements take them regularly through the higher exposure zones, their mean exposure will be a little higher than indicated by the estimates above. This won't be by much because of the dominance of the inside at home period.

### **Childhood cancer data:**

Childhood cancer rates and residential locations are given for the period 1973-1988 by Selvin et al. (1992). A total of 123 cases of cancer were identified among 50,686 white individuals at risk under the age of 21 years. These included 51 cases of leukaemia, 35 cases of brain tumour and 37 cases of lymphatic cancer. Selvin et al. estimate that these categories of cancer cover close to 50 % of all cancers. Each childhood cancer case is given a residential location on a spatial map, Figure 11.

Note the distinctly higher childhood leukaemia rates to the east of the tower, where beam mean intensities at a given radial distance are 50 % higher than to the west. This factor means that the simple radial ring analysis shown below, will underestimate the significance of the cancer/exposure relationships by mixing lower exposed populations to the west with higher exposed populations to the east.

This data has been digitized and radial distances from the tower has been calculated for each case. Selvin et al. recommend considering residents living inside 3.5 km as exposed compared to those living outside 3.5 km. The exposure data suggests that a 4.5 km cutoff is more appropriate. The following table shows the results of comparing the cancer rate within 4.5 km with the rates outside 4.5 km.

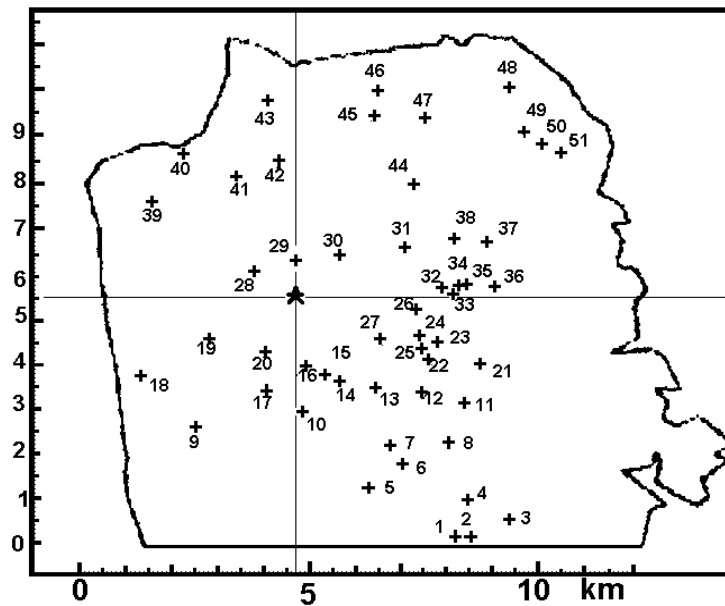


Figure 11: Spatial map of white childhood (<21 years) leukaemia for San Francisco, 1973-88, from Selvin et al. (1992).

This data shows that the cancer rates around the Sutra Tower are more than doubled within 4.5 km from the tower, a very highly significant result, since mean RF/MW exposures are somewhat higher inside the 4.5 km ring than outside it.

Table: Childhood (<21years) cancer rates for those residing within 4.5 km of the Sutra Tower compared to those living more than 4.5 km from the Tower, data from Selvin et al. (1992), 1973-1978, white children only. Total populations shown in brackets.

Symptom	Inside 4.5 km		Outside 4.5 km		Risk Ratio	95% Conf. Interval	p-value
	No. Cancers	Rate	No. Cancers	Rate.			
Brain Cancer	27	6.16	8	2.15	2.87	1.30-6.32	<0.01
Leukaemia	36	8.22	15	4.02	2.04	1.12-3.73	<0.05
Leuk. + Lymphoma	65	14.83	23	6.17	2.40	1.49-3.87	<0.001
All Cancer	92	20.99	31	8.32	2.52	1.68-3.79	<0.001

This is a strong indication that the data in Selvin et al. Shows significant adverse health effects of RF/ME on children, including brain cancer and leukaemia.

### Radial Ring Analysis:

The data also allows a radial ring analysis to be carried out to determine whether the cancer rates vary in a rational way related to the probable mean exposure to the RF/MW radiation from the tower. These data are summarized below using radial cutoff distances of 0.99, 1.49, 1.99, 2.49, 2.99, 3.49, 3.99, 4.49, 4.99, 5.99, and 8 km giving 10 radial rings. Both the Risk ratio and the cumulative risk ratio has been calculated in the manner of Dolk et al. (1997).

Table: Radial rings, with estimated population, Risk Ratios and Cumulative Risk Ratios, for white childhood brain tumour, Leukaemia, Leukaemia + Lymphoma, and All Cancer, in association with RF/MW exposure from the Sutra Tower, San Francisco.

Distance Intervals	<.99	1-1.99	2-2.49	2.5-2.99	3-3.49	3.5-3.99	4-4.49	4.5-4.99	5-5.99	6-8
Population	1138	4334	3558	4489	5146	5566	4939	5386	8141	7988
Symptom										
Brain Tumour	11.81	2.48	3.02	1.80	2.09	1.93	1.63	1.00	0.99	1.01
Cumulative	11.81	4.42	3.87	3.18	2.88	2.66	2.49	2.26	2.02	1.86
Leukaemia	1.26	1.32	2.02	1.92	1.67	1.80	2.03	1.33	0.53	1.26
Cumulative	1.26	1.31	1.59	1.70	1.69	1.72	1.77	1.70	1.48	1.44
Leuk + Lymphoma		2.47	1.08	2.63	2.08	2.54	1.85	2.27	1.56	0.57
	1.05									
Cumulative	2.47	1.37	1.86	1.94	2.10	2.05	2.08	2.00	1.73	1.62
"All Cancer"	4.88	1.44	2.73	2.01	2.43	1.87	2.35	2.11	0.68	1.04
Cumulative	4.88	2.16	2.38	2.26	2.31	2.43	2.21	2.19	1.80	1.68
Estimated personal mean dose in $\mu\text{W}/\text{cm}^2$ .	0.58	0.11	0.27	0.27	0.25	0.22	0.20	0.17	0.16	0.13

The cumulative RR deals to a small extent with the problem of small data sets and overcomes any small errors of bias in radial distance measurements. The reference cancer rate used is the rate in the data set for those living beyond 4.5 km. They are not an unexposed group but a low exposure group.

Despite the small numbers in the study, the "All Cancer" and Leukaemia + Lymphoma rate ratios and cumulative rate ratios follow the mean measured exposure curve very closely showing a strong dose-response curve. The low cancer rate is preserved in the 1-2 km ring which has a particularly low exposure. Inside 1 km where the exposures are above  $1\mu\text{W}/\text{cm}^2$  the total cancer rates are highest.

Figure 12 shows a significant dose response relationship ( $p < 0.005$ ). The trend line shows an estimated no observed adverse effect level (LOAEL) of  $0.063\mu\text{W}/\text{cm}^2$ . This is of the same order of size of the lowest published calcium ion efflux exposure level ( $0.08\mu\text{W}/\text{cm}^2$ ), Schwartz et al. (1990).

Contrary to the ICNIRP (1998) claim that this study shows no evidence of adverse effects, the authors simply stated that none of their three statistical methods for cluster analysis of childhood cancer was related to the Sutra Tower radiation.

The data presented however, does show highly statistically significant increases in Brain Tumor, Leukaemia, Leukaemia + Lymphoma and All Cancers when comparing the higher exposed group residing within 4.5 km of the Tower and when the radial rings RR rates are compared to the mean measured RF/MW exposures.

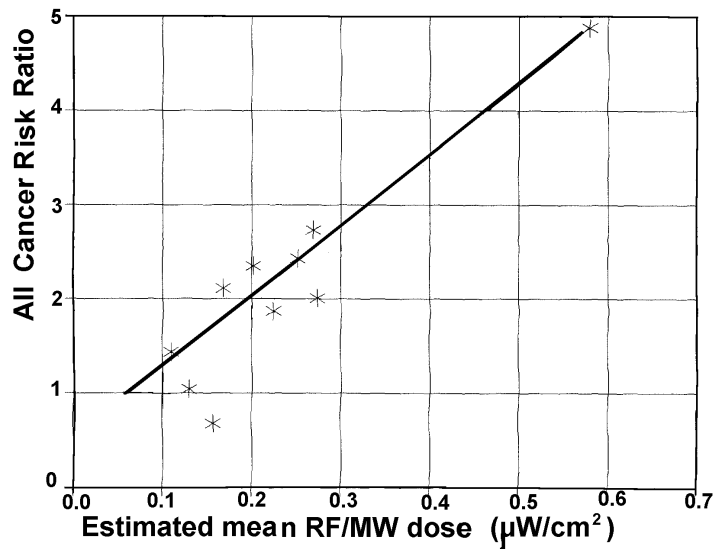


Figure 12: All Cancer Risk Ratio as a function of estimated mean group dose based on measurements of exposure at distances from the tower and a 58% estimate of the personal mean group exposure based on mean inside/outside, home/away times. The fitted line is a least squares fit ignoring the largest outlier.

**This results in the data in Selvin et al. (1992) show a highly significant dose response relationship which, when combined with other epidemiological studies, shows a causal relationship between RF/MW exposure and several childhood cancers, especially brain tumours and leukaemia, and all cancer.**

#### 4. Beall, Delzell, Cole and Brill (1996) “Brain Tumor among Electronics Industry Workers:

ICNIRP (1998) claims that this study showed no significant increases in nervous system tumours. This is factually wrong. The overall results of Beall et al as presented in their abstract is: There was elevated Ors “For 10 or more years of employment in engineering/technical jobs (OR = 1.7, 95% CI: 1.0-3.0) or in programming jobs (OR = 2.8, 95% CI: 1.1-7.0). The OR for glioma for all subjects who had accrued 5 years of programming work 10 tears before the case’s death was 3.9 (95% CI: 1.2-12.4).”

These are statistically significant relationships. The subjects were chosen and studied because of the possibility and concern that using VDTs (Visual Display Terminals, i.e. computers) a great deal in their occupations, which expose workers to a wide range of EMR for long periods, could be related to the increase in brain tumours. The researchers found differences between different occupations who use VDTs in different ways. For example, those in manufacturing VDT had OR = 0.8, while those who used them as going computers such as engineering and technical jobs, OR = 1.2 (95%CI: 0.8-1.9), programming, OR =1.5 (95%CI: 0.8-2.7) and systems engineering, OR = 2.2 (95%CI: 0.6-2.3). Odd ratios for brain tumours increased with the longer times in jobs using VDTs. After 10 years the engineering/technical jobs had an OR = 1.7 (95%CI: 1.0-3.0) and programming, OR = 2.8 (95% CI: 1.1-7.0). These show dose response relationships.

This study shows that the particular groups which use live computers regularly have elevated Odds Ratios (increased levels of risk of brain tumour), and significant increases after 10 years of service. The overall analysis, comparing gliomas and all brain tumours, men, women and total groups all show dose response relationships but the relationship is not assessed as statistically significant:

TABLE 1. Odds Ratios and 95% Confidence Intervals for Brain Tumor and for Glioma by Years of Employment

Tumor and Years of Employment	Men				Women				Total			
	Cases	Controls	OR	95% CI	Cases	Controls	OR	95% CI	Cases	Controls	OR	95% CI
All brain tumors												
<10*	8	47	1.0	Referent	8	31	1.0	Referent	16	78	1.0	Referent
10-19	32	130	2.0	0.7-5.7	7	23	1.2	0.4-4.0	39	153	1.5	0.7-3.1
≥20	90	339	2.3	0.8-6.5	4	21	0.7	0.2-2.8	94	360	1.6	0.7-3.3
Median	24	23	P = 0.20†		13	12	P = 0.68		23	22	P = 0.32	
Glioma												
<10*	7	39	1.0	Referent	5	22	1.0	Referent	12	61	1.0	Referent
10-19	24	103	1.6	0.5-4.9	6	16	2.0	0.4-9.8	30	119	1.5	0.6-3.7
≥20	67	247	2.2	0.7-6.3	2	14	0.6	0.1-3.8	69	261	1.7	0.7-4.1
Median	25	23	P = 0.08		13	12	P = 0.74		23	22	P = 0.14	

\* Reference category.

† P-value for linear trend.

Men show increasing risk of all brain tumours and gliomas with the increasing work time with VDTs but women only show an increase in the 10-19 year group. There is a good evident reason for this. Women's employment is not usually as long as men in these jobs. In the ≥20 year group there were only 6 women in the cases and 35 in the controls.

Exposures to EMR from VDTs has decreased over the decades with the introduction of "low radiation" monitors. Measured RF/MW exposures at the head level of a computer user, 0.5 m from the screen, have been measured at 0.1 to 5 μW/cm<sup>2</sup>. Using a mean lifetime exposure factor of 0.24, based on 0.3 for the time at/away from work and 0.8 for the time programmers are at/away from the computer of 0.8, gives an estimated average lifetime exposure in the range 0.024 to 1.2 μW/cm<sup>2</sup>. This is the same order of mean lifetime residential exposure for the children in San Francisco who had a very significant increase in brain tumour and other cancers.

**Beall et al. (1996) does show statistically significantly increases of brain tumours for those using VDTs in their work for more than a decade. Several relationships also showed dose response increases with brain tumours with longer periods of employment using VDTs, though the small sample sizes limit the statistical significance, these are indicative of probable relationship. The study is misrepresented by the ICNIRP reviewers as a no effect study.**

## 6. Grayson (1996) : "Radiation Exposure, Socioeconomic Status, and Brain Tumor Risk in the U.S. Air Force: A nested Case-Control Study".

Once more the ICNIRP (1998) paper claims that this paper "failed to show significant increases in nervous tumors". Grayson actually shows the opposite conclusion: "Although the present study has its limitations, particularly in exposure estimation, it does suggest that there is a small association between potential EMF exposures and brain tumor risk

among Air Force members, especially for personnel potentially exposed to Radiofrequency/microwave EMFs.” The Odds ratio was, OR = 1.39 (95%CI: 1.01-1.90), a statistical significant relationship.

There is an even more significant relationship of age and length of service as indicated by increase rank. Latencies for brain cancer can be several decades. Hence those who have early exposure and then remain but advance in rank could potentially show a greater incidence of brain tumors. The association with rank has OR = 2.11, (95% CI: 1.48-3.01), for age- race adjusted odds ratio.

Grayson acknowledges that EMF are generally considered to be able to promote cancer by interfering with intercellular communications but that Balcer-Kubiczek and Harrison have observed that microwaves may act alone as tumor initiators or as cocarcinogens. He also reviews several other epidemiological studies which support the association between RF/MW exposure and brain tumors. Ten such studies are known to the present author.

**Grayson (1996) is far from a “no effects” study. Thus far consistently the ICNIRP claims are scientifically wrong and misleading. The study does show statistically significant increases in brain tumours from RF/MW exposure.**

**7. Rothman, Cou, Morgan, Balzano, Guy, Funch, Preston-Martin, Mandel, Seffens and Carlo (1996): Assessment of Cellular Telephone and Other Radiofrequency Exposure for Epidemiologic Research, and**

**8 Rothman, Loughlin, Funch and Dreyer (1996): “Overall Mortality of Cellular Telephone Customers”**

These are the only papers with the ICNIRP reviewers properly assess. The first, however, is only a paper outlining a proposed epidemiological study and so it cannot be said to show no excess in total mortality as claimed by ICNIRP. The second is a study of the effects on cellphone users, but it is only a preliminary report. The authors state that the present preliminary findings have two major limitations. “First, they do not directly address the issue of the relationship between cellular telephone use and brain cancer, which comprises only a small proportion of deaths. Second, the time between exposure to radio frequency energy from portable cellular telephones and the death endpoints that we measured was comparatively short, and our study therefore addresses only short-term effects.”

Neither studies are able nor claim to be able to show mortality effects. Therefore, in the context of a cancer assessment, it is wrong for ICNIRP to claim, “Moreover, (in these papers) no excess mortality was apparent among users of mobile phones”. While the ICNIRP statement about being too early is correct, the way this statement about mortality is expressed is misleading. Neither of these papers are about studies which could show mortality effects and they clearly acknowledge that.

**Interim Conclusions (Papers 1 - 8.):**



All of the first 8 papers or reports cited by ICNIRP with the clear intention of dismissing the possibility of cancer being related to RF/MW exposure, are incorrectly and, in some cases, deliberately, misquoted and misused. In reality the reverse of what ICNIRP claims is true. The research and data set out in these papers alone is sufficient to provide strong evidence of a causal relationship between above RF/MW exposure and significant increases in cancer in military, occupational and residential groups, including children. The residential study shows that statistically significant increases in childhood cancer occur in a dose response manner down to a NOAEL of  $0.06\mu\text{W}/\text{cm}^2$ .

### **9. Szmigielski, Bielec, Lipski and Sokolska (1988) “Immunologic and Cancer-Related Aspects of Exposure to Low-Level Microwave and Radiofrequency Fields”**

ICNIRP describes the work of Szmigielski et al. (1988) in the phrase: “There has been a report of increased cancer among military personnel (Szmigielski et al. (1988)), but the results of the study are difficult to interpret because neither the size of the population nor the exposure levels are clearly stated.”

This is a very dismissive statement for a substantial and significant amount of research which shows highly significant results. In fact it is the largest and most carefully designed study up to that time. Its results are highly significant and confirm that RF/MW can cause cancer in every organ in the body, but especially the blood and lymph organs, e.g. leukaemia.

#### **Population:**

Szmigielski et al. state: “The total population of career servicemen (in the Polish armed forces) was analyzed, and a subgroup of personnel exposed occupationally to MW/RF radiation (on the basis of service records) was developed; the E (exposed) group counted about 3 % of the total population, the rest (97%) was considered as subjects without exposure to MW/RFs (the NE group).” The data set used was 1971-80. In the paper describing the analysis of the study using an extra 5 years of data, 1971-85, Szmigielski (1996), it is explained that over the 15 years there is a slight year to year variation in the population but it averages 128,000 person each year with 3700 MW/RF exposed. The data set is somewhat larger than that used by Robinette et al. (1980).

#### **Exposure Regime:**

As mentioned above, the exposed group was identified by individual service and medical records. The service records show occupations within the services which have RF/MW exposure. The medical records are very detailed since from 1968 it was required to report any occupational exposure to RF/MW in regular medical examinations. This gives a very detailed record of group and individual exposures. Individual exposure events were described in terms of time in zones based on an RF/MW Safety Hygiene Regime. Radar exposures dominated. A “safety zone” was one in which exposures for 4-8 hours were less than  $200\mu\text{W}/\text{cm}^2$ , with incidental (several minutes) daily in the range  $200\text{-}1000\mu\text{W}/\text{cm}^2$ . However, personnel mainly working with production and repair of MW devices, reported incidents of short-lasting higher power densities ( $10\text{-}20\text{ mW}/\text{cm}^2$ ). Szmigielski et al. state that “these exposures resulted from defying the safety rules and were more common in the

1960s when safety rules were not strictly enforced, but still occurred in the 1970s, despite awareness of the possible health hazards of MW/RF radiation.”

Thus Szmigielski et al. found that it was not possible to estimate individual exposures precisely for the whole E group so they divided the exposed subjects into large groups by 5 employment (exposure potential) classes; below 2 years, 2-5, 5-110, 10-15 and over 15 years, and by decadal age groups.

This is a far more detailed exposure classification than Robinette et al. Every exposed person was classified by their job and record of exposure. Even so, uncertainties required grouping into populations. There is no doubt that the E group is an exposed group which is subject to higher mean exposures than the comparison group (all other military personnel). However, the NE group is not truly unexposed. Living and working on military bases leads to everyone being exposed to higher intensities of RF/MW than the normal civilian population. Hence Risk Ratios will be underestimates of the significance of any effects identified.

### **Health Effects Assessment:**

Szmigielski et al. are acutely aware that evidence of immunological impairment with RF/MW exposure is evidence of increased cancer risk since the immune system is a vital part of the cellular repair mechanism of our bodies. Hence they first review evidence that RF/MW impairs the immune systems in cells and animals.

### **Cell line (*In Vitro*) studies:**

They found and present evidence of immunosuppression and immunostimulation associated with RF/MW exposure of cells to a wide range of frequencies, modulations and intensities. This is related to the hypothesis of Professor Ross Adey and his group about the modification of calcium ion binding at the cell membrane surface, and its flow on effects into the signal transduction regulation of the cells. We are now aware that both calcium ion efflux and influx occur at different combinations of RF/MW signal impacting on the cell membrane. This is consistent with immunosuppression and stimulation respectively.

### **Whole animal (*In Vivo*) Studies:**

Short-term exposures of experimental animals to low level RF/MW initially confused thermal effects with non-thermal effects. Careful control of exposure and better handling of animals found consistent transient and reversible increase lymphocyte proliferation and function. However, that time there was not convincing *in vivo* evidence of immune system impairment from short-term RF/MW exposure, and, at that time “There are no experiments *in vivo* involving exposure of animals to low-frequency modulated MW with examination of the immune functions. On the other hand, as discussed below, both the higher susceptibility of animals to chronically exposed bacterial and viral diseases, and the data on acceleration of development of neoplasms in mice exposed for months in non-thermal MW fields (the two phenomena that might result from suppression of immune functions in chronically exposed subjects) emphasize the problem of the response to long-term low-level irradiation in MW/RF fields, and they call for further investigation.”

However, Szmigielski et al. appear to be unaware of Shandala et al. (1983) which did find a highly significant (78%) and persistent suppression of the immune system rats when exposed to  $500 \mu\text{W}/\text{cm}^2$  for 3 months.

### **Integrated evaluation of immunity in MW/RF exposed animals:**

Szmigielski et al. proceed to describe their own experiments in this area. They conclude:

**“An overview of the available and of our own findings suggests the existence of a biphasic reaction of the immune system to MW/RF radiations - stimulation of the whole system (mainly humoral immunity) after a single or few days exposures, followed by gradual, but transient, suppression of the whole immunity with prolongation of the exposure period (up to several months) and/or increasing power density of the fields. Stimulation and suppression of immunity in MW/RF exposed animals both seem to be transient and inconsistent phenomena. At low power densities the system recovers soon after exposure.”**

This raises the question, what happens if exposure continues for years ?

### **Cancer related aspects of exposure to low-level microwave fields:**

Human populations contain a wide range of people, including those with already compromised immune systems. The evidence that chronic exposure of animals can suppress their immune system with some combinations of parameters of low-level microwave exposure promoted the study of the effects of MW exposure on cancer prone mice. This was a precursor for looking for cancer in MW exposed human populations.

Szmigielski et al. planted cancer cell in the lungs and on the skin of mice and chronically exposed them to non-thermal intensities of 2.45 GHz microwaves. The tumors grew faster and the mice died earlier in the exposed compared to the sham exposed mice. The MW exposed mice with induced skin cancer showed 50 % died after 137 days, compared to 305 days for the sham exposed mice. The lung tumors which all started at near  $2 \times 10^5$  viable cells. After 3 months, the control group stayed close to  $2 \times 10^5$ , while the exposed mice rose to 6 and 15 for 5 and 15  $\text{mW}/\text{cm}^2$  respectively.

They then showed that microwaves on their own and with a cancer promoter, significantly enhanced cyclic AMP activity in urine epidermis (scraped) samples in mice.

They concluded:

**“On the basis of Balcer-Kubiczek and Harrison’s reports, an the above investigations of his own group, Adey (personal communication) recently offered his own concept and initial model of the cancer-promotion process and its influence by MW/RF fields modulated at low frequencies. The promotion appears to relate to a distorted inward stream of signals from the cell membrane to the nucleus (where carcinogenesis was already initiated by other factors) and to intracellular organelles. MW/RF modulated at low frequencies may in certain cases (depending upon modulation and time**

**exposure)) act synergistically with the action of promoters, activating the same membrane receptors.”**

Hence, prior to presenting their human study of cancer in MW exposed military personnel, Szmigielski et al. outline a strong evidence trail indicating the probability of cancer being found based on cellular and animal experiments, based on immune system impairment, and synergistic activity of RF/MW with other cancer initiators and promoters.

### Polish Military Study:

Placing the study in context, the authors note several previously published studies showing increases in cancer (McLaughlin (1953)), in leukaemia with radar exposure (Lester and Moore (1982)), Milham (1982) and Wright (1982), and Vagero and Olin (1983).

They note that Robinette et al. (1980), the Korean War Study, reported no significant differences between high and low exposure groups, but point out: “However, when three sub-groups of the high-exposure group were developed to provide a gradient of potential exposure, a trend appeared for increased number of malignant neoplasms in the sub-group rated as highly exposed.” They also refer to the weakness of the Korean War study in terms of its size and subject selection.

Their own study, the Polish Military Study, has a very large study group, careful and well documented subject selection for membership in the exposed group, and used the entire military population as a reference group. The results are summarized by Figures 13 and 14.

The decadal age category results are presented in Figure 13.

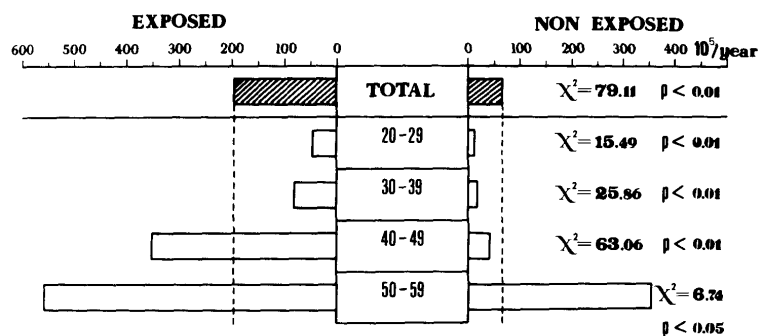


Figure 13: Cancer morbidity rates in RF/MW exposed and “non-exposed” personnel for all types of malignancies at various age groups.

Note the largest differences at the age group 40-49 years and statistical significance of differences for all age groups. For all cancer and for every age group  $p < 0.01$  except for the 50-59 age group, when the cancer rate in the general population rises rapidly, the difference is still significant ( $p < 0.05$ ) but less so than the early age groups for this obvious reason.