Potential and Actual Adverse Effects of Radiofrequency and Microwave Radiation at levels near and below 2 \(\mu\)W/cm\(^2\).

by

Dr Neil Cherry
Lincoln University
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1. Background:

1.1 Introduction:

There is wide community concern about the possible or actual health effects of electromagnetic radiation. Most recently the concern has focused on the placement of cell sites (mobile phone base stations) in communities, close to residences, schools, preschools, hospitals and work places, for example. Strong claims by industry representatives and their consultants that there is no scientific evidence to justify the public’s fears is scientifically demonstrably wrong.

This review cites a great deal of internationally published peer-reviewed scientific evidence from laboratories and universities around the world, sourced from reports from reputable institutions and in internationally published papers from peer-reviewed journals. This shows compelling evidence of athermal biological changes in cells and in animals which relate to brain function change, sleep disruption, chronic fatigue, reproductive problems and adverse health problems such as immune system impairment and cancers of many organs. Epidemiological studies have identified statistically significant increases of the incidence of most these symptoms and diseases associated with above average exposure to radio-frequency and microwave (RF/MW) exposure.

The Standards which are referred to and preferred by industry and military authorities are technical standards and not public health standards. The standards bodies in Australia and New Zealand are the Standards Associations. These are the bodies who are responsible for building and mechanical standards. When the Australian Government wished to set an RF/MW exposure standard they referred the matter to Standards Australia, who formed a committee of “stakeholders” who had an interest in the use of RF/MW technology. This included telecommunications and power industry representatives, government departments who deal with the radio spectrum, defence department personnel, along with the Australian Radiation Laboratory, C.S.I.R.O. and some scientists who had an interest in RF/MW, most of whom were consultants to the industry or the government in RF/MW matters. This is not a group which is totally independent of those who gain benefit from using the spectrum and is not a group working from the basis of public health research and protection.

Hence the standard, NZS 6609, is based on international standards and guidelines which are based on what is describes as “established health effect” which in the RF/MW range is only “tissue heating”. Thus these standards ignore the highly probable health effects shown by the combination of laboratory, animal and epidemiological studies and hence is not a public health protection standard and should not be promoted and used as such.
There is more than enough research and published studies to show that adverse health effects are established to a high degree of probability by epidemiological studies, backed up by plausible biological mechanisms from laboratory and animal experiments, associate serious adverse health effects with chronic low level exposures to RF/MW radiation in occupational and residential situations where the mean exposure level is near or below 0.1 \( \mu \text{W/cm}^2 \). This is 2000 times less than the guideline approved by the standards committee and over 100 times higher than the ambient exposure in rural New Zealand and Australia.

This is not the first review which shows that EMR at the occupational and residential levels of exposure with are on average significantly below the standards, which are based on heat avoidance, produce strongly associated adverse health effects. Two leading U.S. EMR researchers make similar conclusions and two highly eminent institutions also concur.


“First, I will detail some epidemiological and laboratory evidence indicating that em fields can promote cancer. Second, I will detail effects of electromagnetic fields have on neural and neuroendocrine systems. Then I will summarize the relevant information on how the neural and immune systems interact. With the foregoing as foundation, I will then integrate it all and spell out one means by which exogenous electromagnetic fields may promote cancer”

The biological mechanism presented involves the absorption of the em field at the cell membrane which perturbs the neurochemistry, with changes in dopamine, opiate and melatonin systems.

Cleary (1995), in the “Encyclopedia of energy Technology and the Environment” concludes in part:

“There is increasing evidence of possible health effects of environmental exposures to EMFs and EMR in the home and in the work place. Epidemiological evidence indicates possible associations of long-term exposure and cancer incidence, adverse reproductive outcomes, and behavioural and neurological changes.”

The U.S. Environmental Protection Agency staff produced a review in 1990 which recommended that ELF be classified as a probable carcinogen and RF/MW as a possible carcinogen, Sibbison (1990).

In 1992 the European Parliament passed resolution B3-0280/92 which included in part:

“E: whereas the results of many in vivo and in vitro studies show increasingly clearly that the interaction mechanisms underlying such disorders and diseases [cancer, nervous disorders and circadian rhythm changes], centred mainly in the cell membrane, lead to disruption of melatonin secretions, ornithine decarboxylase activity and T-lymphocyte efficacy, testify to the probable role of non-ionizing radiation in promoting cancer,”
Some military and occupational groups are exposed to moderate to high exposures to RF/MW on occasions during their work. The pervading international view is that the only effect of RF/MW is heating of tissue. It is also accepted that there is no scientific evidence that heating of tissue on a few degrees Celsius can cause cancer. Hence high military and occupational studies which find increased incidence of cancer cannot be dismissed on the grounds that their exposures are high. If it was true that high exposure groups were associated with high Risk Ratios for cancer, moderate exposure groups with moderate Risk Ratios and low exposure groups with low Risk Ratios then this would provide a form of gross dose-response relationship and would result in compelling evidence that RF/MW was probably or actually carcinogenic.

Using leukaemia as an example we might expect Polish Military personnel to be in a high exposure group, amateur radio operators to be moderately exposed group and residences of cities in the vicinity of TV and FM towers to be a low exposure group. The results of such studies are summarised in Table 1.

<table>
<thead>
<tr>
<th>Study</th>
<th>Reference</th>
<th>Exposure Category</th>
<th>Leukaemia Type</th>
<th>Risk Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polish Military (Mortality)</td>
<td>Szmigielski et al., 1996</td>
<td>High</td>
<td>ALL</td>
<td>5.75</td>
<td>1.22-18.16</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CML</td>
<td>13.90</td>
<td>6.72-22.12</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>CLL</td>
<td>3.68</td>
<td>1.45-5.18</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AML</td>
<td>8.62</td>
<td>3.54-13.67</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>All Leuk.</td>
<td>6.31</td>
<td>3.12-14.32</td>
</tr>
<tr>
<td>Amateur Radio (Mortality)</td>
<td>Milham (1988)</td>
<td>Moderate/High</td>
<td>AML</td>
<td>1.79</td>
<td>1.03-2.85</td>
</tr>
<tr>
<td>North Sydney TV/FM towers (Mortality)</td>
<td>Hocking et al.(1996)</td>
<td>Moderate</td>
<td>All Leuk.</td>
<td>1.17</td>
<td>0.96-1.43</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ALL+CLL</td>
<td>1.39</td>
<td>1.00-1.92</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>AML+CML</td>
<td>1.01</td>
<td>0.82-1.24</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other Leuk.1.57</td>
<td>1.01-2.46</td>
<td></td>
</tr>
<tr>
<td>UK TV/FM (Incidence)</td>
<td>Dolk et al. (1997b)</td>
<td>Low</td>
<td>Adult Leuk.</td>
<td>1.03</td>
<td>1.00-1.07</td>
</tr>
</tbody>
</table>

Note: ALL : Acute Lymphatic Leukemia; CLL: Chronic Lymphatic Leukaemia; AML Acute Myeloid Leukaemia; CML: Chronic Myeloid Leukaemia; and All Leuk.: All Adult Leukaemias.

By exposure ranking these studies a form of dose response relationship is found with increasing Risk Ratio with increasing exposure. This is referred to by Bradford Hill (1964) as a biological gradient which suggests a strong association between a disease agent (RF/MW) and disease (Leukaemia). Several other epidemiological studies have found statistically significant associations with RF/MW exposure and leukaemia, adding strength and consistency to this relationship. The Dolk et al. (1997b) study finds mean adult...
leukaemia incidence varying with radial distance in a manner which is close to the likely exposure curve. This is a strong individual study’s dose-response relationship.

This evidence more than is sufficient under the resource Management Act to establish a plausible biological mechanism and a potential adverse effect on the environment. Without the evidence presented above the Planning Tribunal in the Maclntyre/Bell South Case stated that the New Zealand Standard NZS 6609 as “not decisive” in RMA considerations where there is evidence of potential adverse health effects.

The Tribunal held that:

“we cannot avoid our duty to decide the resource consent application on the evidence by simply accepting the New Zealand Standard as decisive of the issue. The law does not give the standard that status. It is the Tribunal's duty to consider all the evidence and find whether or not there would be actual or potential effects on the environment of allowing the activity. we hold that compliance with the New Zealand Standard is not decisive of that question; and any challenge to the adequacy of the levels set in the standard is collateral to it.”

It is important to note the context of this appeal. The applicant, BellSouth, was appealing the CCC imposition of a $50\mu W/cm^2$ exposure limit, seeking to have it changed to $200\mu W/cm^2$, the level in NZS 6609. The Tribunal clearly rejected this based on the evidence given, including evidence of potential adverse effects below $3\mu W/cm^2$ and the applicants willingness to reduce emissions to produce a public exposure of $1.6\mu W/cm^2$, and set the public exposure condition at the nearest dwellinghouse of $2\mu W/cm^2$.

Since this is the only legally and scientifically contested case about the health effects of cell sites in New Zealand it has the strongest precedent weight. Hence for the applicant and the CCC officers to recommend conditions based compliance with NZS 6609 is legally challengable and inappropriate. It is a clear example of the way in which the officers' recommendations are ignoring legal and scientific evidence showing that under the RMA legal framework the standard has no status nor standing and the environment court has moved to a public exposure level of 1 % of the standard. The finding of the Maclntyre case also state that the level should be revised when there is new evidence. There is a host of new evidence which is summarised here.

1.2 Summary of Evidence:

In this report will give evidence which will show the following:

- That the New Zealand Standard is based on avoidance of heating effects and does not protect public health from adverse health effects linked to chronic extremely low level exposure to RF/MW radiation as indicated by biochemistry, animal studies and epidemiology.

- That the committee which sets the standard is comprised largely but not exclusively by pecuniary interested stakeholders who derive financial benefit from the production and usage of RF/MW radiation.
• That athermal biological effects have been identified which relate to adverse health effects.

• That biological effects identified for ELF exposures also apply to RF/MW which is pulsed or modulated at ELF frequencies with the difference that RF/MW has a far higher penetration into tissues than does ELF signals.

• That experiments on animals, which are normally used in chemical, cosmetic and drug tests of human safety, when used to test the safety of electromagnetic radiation show adverse health effects.

• That epidemiological studies of populations who are known to experience above average exposures to RF/MW radiation, form a strong and coherent set of evidence, which is consistent with the cellular changes and animal experiments, which shows that RF/MW radiation is a very highly probable or even an actual adverse health causal agent.

• That, given the above evidence which also shows that effects occur in residential populations at very low exposure levels, consistent with those experienced with hundreds of metres of cell phone base stations, that cell phone base stations should be located a considerable distance from residences, worksites, schools, preschools and hospitals.

• That given the health effect are real risks, the strong and supportable anxiety that such scientific information provides, the costs to the community and the health system cannot be offset against any additional costs which might be entailed in locating cell sites base stations away from homes and rural towns. These costs should be borne by the users and producers of the cell phone technology by requiring them to locate their base stations well away from public assess places where people spend more than 4 hours per day.

• Because of the results of epidemiological studies of public exposure then:

  The national standard for public exposure should be set at 0.1 \( \mu \text{W/cm}^2 \).

1.3 Professional Scientific background:

My name is Neil James Cherry, I have the degrees of B.Sc. (Hons) and Ph.D. in Physics from the University of Canterbury. I am a member of a number of learned societies, including Fellow of the Royal Meteorological Society (F.R.Met.S.), member of in the Royal Society of N.Z. (M.R.S.N.Z.) and member of the International Society for Environmental Epidemiology (M.I.S.E.E.). I was Senior Lecturer in Agricultural Meteorology in the Department of Natural Resources Engineering at Lincoln University for 23 years and hence I am a professional biophysicist. I have 28 years of post graduate research experience, about 25 years of research into air pollution, including air pollution epidemiology and health effects. I teaching experience in undergraduate and graduate courses in environmental engineering, environmental physics, air pollution meteorology, modeling and health effects, atmospheric chemistry, environmental epidemiology, biometry and human biometry.
I have over 15 years research experience in human biometeorology and environmental epidemiology, and in studying the effects of the natural environment on human physiology, psychology and brain function. Repacholi (1993) (The WHO/IRPA/UNEP sponsored review) notes: “The biological effects of electromagnetic fields is multidisciplinary; it draws from physics, engineering, mathematics, biology, chemistry, medicine and environmental health”. My academic qualifications, teaching and research experience, which include professional training, teaching, research in physics, engineering, mathematics, biology, chemistry and environmental health, provides a broad and sound foundation for researching the adverse health effects of EMR from an integrative, multi-disciplinary approach.

I have used my wide multi-disciplinary experience over about the last 4 years to review the published research of the effects of artificial electromagnetic radiation on human physiology, brain function and health. This included a recent international study tour to major universities and laboratories who are engaged in researching EMR effects. I have authored several widely internationally circulated reviews of the scientific literature on EMR health effects.

1.2 Planning and RMA background:

I am an elected member of Canterbury Regional Council and Chairperson of the Resource Planning Committee, with considerable experience in the use, interpretation and implementation of the Resource Management Act 1991, through academic study and lecturing, Resource Consent hearings, Environment Court hearings, and policy and plan development.

1.3 Independence of Evidence:

I bring a totally independent scientific and professional approach to this evidence, with no pecuniary interest in the subject. My professional base is Lincoln University. I have strong community support, shown by the large number of New Zealand, Australian, U.S., U.K. and Irish individuals, community groups and councils who have sought my reviews and my evidence at hearings, review panels, Inquiries, pension claims and insurance claims.

My interpretation of the research results is frequently at variance with scientific and expert evidence submitted by consultants engaged by industry and applicants for resource consents. Because of these differences I have taken great and careful pains to check and cross-check my information and interpretation with the authors of the papers and reports where possible and practical, and with other independent scientists, doctors and researchers in laboratories and universities and in private practice.

A totally independent public authority, the New Zealand Parliamentary Commissioner for the Environment, in her 1996 report “Public Authority Planning for cellphone transmission facilities”, states:

“Scientific and technical information on the subject should be ‘neutral’. Much of the technical and scientific expertise, however, resides in the companies and may not be perceived as neutral by the community. Local authorities need an independent source of advice in order to fulfill their responsibilities under the RMA. Where a public agency is seen to support the industry, local
authorities and the public may perceive that an agency is not truly independent and may distrust the information provided.”

I strive to provide totally independent, high quality, scientific advice with the primary emphasis on assessing the public health impacts and taking a public health protection approach as a matter of priority.

A valued colleague, an eminent epidemiologist and public health professional, Professor John Goldsmith, who was a member of our team of expert witnesses for the MacIntyre/BellSouth Planning Tribunal appeal, stated in the conclusions to one of his published reviews, Goldsmith (1995):

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

At present there seems to be little interest in or understanding of epidemiologic information among regulatory bodies that should provide protection. While we conduct epidemiologic studies as well as we possibly can, we who are concerned with health protection and careful identification of risks must also keep pressure in regulatory agencies to include epidemiologic thinking in their work.”

There is strong, and growing, epidemiologic evidence, with reinforcing and related animal and cell biological research, to show that chronic low-level exposure to RF/MW produces increased risk of adverse health effects in occupational groups and residential populations.

As the evidence for adverse health effects from EMR continues to mount year by year, experts on behalf of industrial clients still claim that there is no substantial evidence of effects below the thermal threshold, no evidence of a causal adverse effect and/or no proof of adverse effects. They also say that experiments need to be replicated and that more and more research is required. Enough evidence existed many years ago for the following two reviews to come to strong conclusions. The RMA requires only the establishment of a potential adverse effect in order to invoke section 5(2)(c) and Section 3(f), to avoid, remedy or mitigate any potential adverse effect.

1.4 New Zealand’s Law, The Resource Management Act 1991:

The Resource Management Act 1991 (RMA) regulates all construction and industrial and trade discharges of contaminants into the environment. A cell site requires a building resource consent from a territorial authority. If RF/MW radiation is recognized as an issue in a Regional Air Plan then the discharges into the environment can be controlled through rules in that Plan. The Canterbury Regional Council has legal advice that RF/MW is a contaminant under the RMA and therefore can be regulated through a regional air plan. Irrespective of this in deciding on any resource consent or district or regional rule, an assessment of the effects on the environment must be carried out according to the Fourth Schedule. Over-riding this is a general duty, Section 17, for every person to avoid,
remedy or mitigate any adverse effects of an activity on the environment, enforceable through an Enforcement Officer or the Environment Court. “Effects” include any potential effects of low potential probability which has a high potential impact, Section 3 (f).

The RMA threshold test is that it is more likely than not that there is a potential effect of low probability which has a high potential impact.

1.4.1 Definition of contaminant:

In relation to cell sites and the discharge of RF/MW into the environment, Section 2 of the RMA defines a “contaminant as “includes any substance (including gases, liquids, solids, and micro-organisms) or energy (excluding noise) or heat, that either by itself or in combination with the same, similar, or other substances, energy, or heat (b) When into air, changes or is likely to change the physical, chemical, or biological condition of the air into which it is discharged. RF/MW radiation is a form of energy which heats and changes the biological conditions of cells irradiated by it in the environment having passed into the air.

1.4.2 Purpose of the RMA, Sustainable Management, Adverse Effects:

The purpose of the RMA is to promote the sustainable management of natural and physical resources. This means managing the use, development, and protection of natural and physical resources in a way, or at a rate, which enables people and communities to provide for their social, economic, and cultural well-being and for their health and safety while -

(a) Sustaining the potential of natural and physical resources (excluding minerals) to meet the reasonably foreseeable needs of future generations; and

(b) Safeguarding the life-supporting capacity of air, water, soil, and ecosystems; and

(c) Avoiding, remedying, or mitigating any adverse effects of activities on the environment.

where (Section 3) the definition of “Effect” includes -

(a) Any positive or adverse effect; and

(b) Any temporary or permanent effect; and

(c) Any past, present, or future effect; and

(d) Any cumulative effect which arises over time or in combination with other effects - regardless of the scale, intensity, duration, or frequency of the effect, and also includes -

(e) Any potential effect of high probability; and

(f) Any potential effect of low probability which has a high potential impact.
The definition of “Environment” in this Act, includes “Ecosystems and their constituent parts, including people and communities;...”.

1.4.2.1 Cumulative potential effects:

The Resource Management Act directly recognizes cumulative effects and potential effects. The cumulative risk of cell damage from chronic low level exposure to the RF/MW radiation form a cell site, clearly conforms to the definition of a cumulative effect, “regardless of scale, intensity, duration and frequency” and a “potential effect of low probability which has a high potential impact”, e.g. cancer, miscarriage, learning difficulties, sleep disruption or chronic fatigue syndrome.

1.4.2.2 Alternative sites - alternative receiving environments:

In giving guidance about the rules in plans about discharges into the environment the RMA states (Section 70 (2)) that, “Before a regional council includes in a regional plan a rule requiring the adoption of the best practicable option to prevent or minimise any actual or likely adverse effect on the environment of any discharge of a contaminant, the regional council shall be satisfied that, having regard to -

(a) The nature of the discharge and the receiving environment; and
(b) Other alternatives, including a rule requiring the observance of minimum standards of quality of the environment, -

the inclusion of that rule in the plan is the most efficient and effective means of preventing or minimising those adverse effects on the environment.”

The Fourth Schedule lists the Matters that should be included in an assessment of effects on the environment, which includes: (f) Where the activity includes the discharge of any contaminant, a description of-

(i) The nature of the discharge and the sensitivity of the proposed receiving environment to adverse effects; and
(ii) Any possible alternative methods of discharge, including discharge into any other receiving environment:

(g) A description of the mitigation measures (safeguards and contingency plans where relevant) to be undertaken to help prevent or reduce the actual or potential effect:

1.4.3 Avoiding siting cell sites near sensitive receiving environments:

Cell sites radiate low levels of RF/MW radiation into the environment surrounding the site. Hence when considering the siting of cell sites the nature and sensitivity of the receiving environment is critical. Applicants for Resource Consents are required to investigate other receiving environments, i.e. alternative sites with less sensitivity to the effects of the discharge. The effect of the discharge is required to be prevented or minimised, including the consideration of less sensitive sites. Because of the epidemiological evidence of health effect risks, environments which include long term exposure of people, especially the young, the elderly, pregnant women and the sick, i.e. schools, preschools, hospitals, retirement homes and residences. In addition some work places involve vulnerable
people for 9 hours a day (8am to 5 pm) and hence could be considered as sensitive receiving environments.

2. New Zealand Court precedents which guide the scientific approach:

Three Planning Tribunal/Environment Court (The Court) cases are considered relevant to the consideration of the approach to scientific evidence and expert witnesses:

- **Rodney** District Council/Transpower (A85/94), a high voltage powerline appeal which relates to adverse health effects, plausible mechanisms, the balance of probabilities, and conflict of evidence as well as the role, approach and status of expert witnesses.

- **MacIntyre**/BellSouth (A15/96), a cellsite appeal based on adverse health effects, and focuses on Section 3(f) of the RMA 1991 and establishing a condition for public exposure under which there was no evidence of adverse effects, “even a potential effect of low probability and high potential impact”. It also recognizes the need for a review in the light of new evidence. Scientifically contestable evidence was presented and tested under cross-examination.

- **Beckenham**/Telcom (W165/96) a cellsite appeal based on visual effects, but includes scientifically uncontested and legally unargued discussion of health effects where the Court considers that “the onus of proof is Telecom to persuade us on the balance of probabilities”.


This case was about potential adverse health effects from a high tension powerline. The key finding relates to decisions made under situations of scientific uncertainty. It addresses issue relating to “plausible mechanisms”, “balance of probabilities” and “scientific consensus” under conditions of a conflict of evidence. The decision (A85/94) states (p21):

“It is our duty to make findings about actual or potential effects of the proposed activity on the environment. To make our decision on a question on which there is a conflict of evidence, we have to be satisfied on the balance of probabilities, having regard to the gravity of the matter in question.”

“We accept the validity of statements of Dr Repacholi that it is not possible for scientists to prove that exposure to electrical magnetic fields from high voltage transmission lines does not have adverse effects on health; and that an appropriate approach is that with open minds we carefully consider the evidence from studies that suggests that there is or is not an effect. Yet although we can accept that scientific knowledge about the potential adverse effects of the fields may be incomplete, it is our duty to make a decision now, on the present state of knowledge. It would be an abdication of that duty if we were to allow opponents of proposals to prevent them from proceeding on the basis that science might discover effects that had not yet been established. That is not to reject the precautionary approach, but there needs to be some plausible basis, not mere suspicion or innuendo, for adopting the approach.”
This gives guidance on the application of the precautionary approach when applying the provisions of the RMA relating to potential adverse health effects that: “there needs to be some plausible basis, not mere suspicion or innuendo, for adopting the approach.”

2.1.1 Weight of expert evidence:

In order to establish a plausible mechanism scientific evidence must be presented. A plausible mechanism may be proposed by one side of a case and be attacked as implausible by the other. When there is conflict between experts about that evidence the status and plausibility of the experts becomes relevant.

2.1.2 Status of experts:

The Court notes that there is a “conflict of evidence” and set about resolving that conflict through ranking the quality of the experts and their evidence.

Three expert witnesses presented evidence. Dr Michael Repacholi and Dr Andrew McEwan presented evidence on behalf of the applicant and Dr Ivan Beale gave independent evidence. The decision is very instructive on the status of experts and the use and interpretation of expert scientific evidence in the Environmental Court.

The decision stated: “As a judicial body it would not be appropriate for us to weight suspicion, even when expressed by one who is qualified as an expert witness, against the opinions of even better qualified experts which are consistent with the consensus of the international scientific community.”

At that time none of these experts had published any fundamental research that they had carried out on the adverse health effects of powerlines, although Dr Beale was carrying out such a project at that time. Dr Beale’s project was given no weight by the Tribunal, assisted by comment from Dr McEwan, because it was not written up, not peer-reviewed and not published. On the other hand, while Drs Repacholi and McEwan had no relevant publications of their own. Their evidence was given weight because it was consistent with the claimed “consensus of the international scientific community” and that the exposures of the residents would be a small fraction of the allowed international guidelines.

A further reason for according the evidence of Drs Repacholi and McEwan a higher status was because Dr Beale “did not produce the reports of the studies on which he relied, and did not refute his colleagues claims that they (the reports) were inconclusive because of methodological weaknesses”.

Dr Repacholi’s status is clear as the chairman of two highly significant international commissions but he has never published any basic scientific research on health effects of powerlines which was relevant in this case. The nature of this claimed “consensus of the international scientific community” needs to be viewed critically since so much emphasis is placed on it by the Court and the experts for the applicant.

2.1.3 International Scientific Consensus:
The applicant’s case was that there was no evidence of adverse effects, neither through a biological mechanism, nor through epidemiological studies. Dr McEwan deposed that “there is no established interactive mechanism which explains how biological cells might be affected by external weak fields; that epidemiological studies show a lack of consistency and had not established any causal association between health effects and exposure to ELF magnetic fields.” He further stated that the NRL recommends the use of the interim 1990 ICNIRP guidelines which were reviewed and confirmed by the commission in 1993. “Dr McEwan deposed that the guidelines had been based on careful examination of the research data on effects of exposure to ELF electric and magnetic fields, and include a margin of safety.”

Expert “confirmation” of Dr McEwan’s evidence is given by Dr Repacholi. Dr Repacholi “deposed that laboratory evidence does not support there being a link between exposure to 50/60 Hz fields and carcinogenesis or any form of cancer” and he says that epidemiological studies has not established an association between cancer and 50/60 Hz fields neither for workers nor for residents.

This appears to establish a very strong four-tier case for no adverse effects:

- No plausible biological mechanism.
- No epidemiological association of risk of an adverse health effect.
- International consensus that there are no biological mechanisms nor public health risks.
- Conformation that public exposure falls within international guidelines.

In critically assessing this approach it is vital to note that very different approaches to assessment of evidence and levels of proof are being used. One seeks “Proof of an Effect” and another seeks “Evidence of Risk of an Effect”.

2.1.4 Proof of an Effect Approach (Strong Proof):

This approach uses the terms of scientific proof and seeks a very high level of confidence that evidence of an effect is available. It requires that the research results referred to have to be highly reliable and repeated before identification of an adverse effect is made. The requirement is for strong links to be made and for proof of causation to be established before an adverse health effect is accepted. Thus only reliably proven adverse effects can be used to set guidelines and standards. Hence only thermal effects are used are used to set the international and national standards because they are the only effects accepted by the selected reviewers as scientifically proven.

2.1.5 Proof level used by applicant experts and international guidelines:

The “proof stance” an expert witness or a group of experts takes is revealed by the phrases and language used in their evidence.

The terms such as “not supporting there being a link” and “established” or “established any causal association”, show that the high level of proof is being applied. Drs McEwan and Repacholi both use this strong proof language.
In reviewing the documents of the international bodies which set the EMR guidelines we find the same strong proof language. By applying such tests epidemiological evidence is almost entirely excluded from standards setting because by its nature it does not provide proof of causation.

Jammet (1990) outlines the criteria used by UNEP/WHO/IRPA/ and ICNIRP for limiting exposure to 50/60Hz fields:

“The limits recommended in these guidelines were developed primarily on established or predicted immediate health effects produced by currents induced in the body by external electric and magnetic fields.”

Epidemiological research was found to:

“provide some support for the findings of a previous study on childhood cancer and exposure to weak electric fields”

but

“To date, chronic low-level exposure to 50/60 Hz fields has not been established to increase risk of cancer.” (Strong proof language).

Given this rejection of the epidemiologic evidence the IRPA set public exposure limits at an induced current of 2 mA/m² which is equivalent to an electric field of E=5 kV/m or B=0.1 mT, which will avoid shocks and perception of the presence of the field. This is far above the levels in which children live who have been shown to have increased risk of leukaemia, for example, which is around 0.2 to 0.5 μT, or down to 500 times lower than the recommended standard.

Protecting workers from gross, immediate effects of induced currents is very different from protecting residents from chronic low level exposure when residing near high voltage power lines. The expert witnesses for the applicant, Dr Repacholi and Dr McEwan seek status and strength of argument by referring to the international consensus that there are no established effects and that international guidelines give levels of exposures under which there will be no proven adverse effects.

2.1.6 Level of Scientific proof used by the Court:

The Court appears to be unaware of the strong proof approach of these experts and in the UNEP/WHO/IRPA/ and ICNIRP guidelines. Dr Repacholi is the chair of the major international commissions and was chair of the Australasian EMR standards sub-committee, indicating the likelihood of consistency of the strong proof approach being applied locally and internationally.

In contrast, the legal guidance given by the Court in its interpretation of the Resource Management Act requires the adoption of the “evidence of risk” approach. The Court states that there must be “some plausible basis” for adopting a precautionary approach. Sections 5(2)(c) and 3(f) of the RMA 1991 apply and the court states that “we have to be satisfied on the balance of probabilities, having regard to the gravity of the matter in question. The possibility of adverse effects on the health of the people who may be exposed to electric and magnetic fields from high voltage power lines has sufficient gravity to deserve a higher standard of proof.”
This statement points to the “evidence of risk” approach because of the “gravity of the situation with respect to the health of the people”.

However, the Court was steered away from this to a strong proof approach advocated by the expert witnesses on behalf of the applicant and in the relative absence of presentation of evidence to the contrary. It is very difficult for the Court when International and National experts do not present the available evidence when it would be contrary to their client’s interests and no equivalent standing independent expert is sought and available to provide the total sweep of evidence available.

2.1.7 The Nature of the International Group setting the International Consensus and Guidelines:

The WHO 1993 review team was derived primarily from the International Radiation Protection Association (IRPA), which in 1992 formed an independent commission called the International Commission on Non-Ionizing Radiation Protection (ICNIRP). Evidently three apparently separate and independent bodies, the IRPA, the ICNIRP and the WHO, contain primarily the same group of people, Table 2.

It is seen that there is a strong overlap in membership of international commissions which all involve national representatives and draw from a small pool of eminent scientists. The first eight members are on both commissions, giving a total scientific membership of the two commissions of 19 people, Table 2.

<table>
<thead>
<tr>
<th>Table 2: Membership of three of the major international commissions which produce the “international consensus” on EMR health effects.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WHO 1993 Review Team</strong></td>
</tr>
<tr>
<td>Dr M. Repacholi (Chairman) (Aust)</td>
</tr>
<tr>
<td>Prof. M. Grandolfo (Italy)</td>
</tr>
<tr>
<td>Prof. J. Bernhardt (Germany)</td>
</tr>
<tr>
<td>Dr L.A. Court (France)</td>
</tr>
<tr>
<td>Dr A. McKinlay (Observer) (UK)</td>
</tr>
<tr>
<td>Dr M. Swicord (USA)</td>
</tr>
<tr>
<td>Dr A.J. Stolwijk (USA)</td>
</tr>
<tr>
<td>Dr L.D. Szabo (Hungary)</td>
</tr>
<tr>
<td>Dr C.F. Blackman (USA)</td>
</tr>
<tr>
<td>Dr R.D. Saunders (UK)</td>
</tr>
<tr>
<td>Prof. M.G. Shandala (USSR)</td>
</tr>
<tr>
<td>Dr M.A. Stuchly (Canada)</td>
</tr>
<tr>
<td>Dr S. Szmigielski (Poland)</td>
</tr>
<tr>
<td>Mme A. Duchene (Secretary) (France)</td>
</tr>
<tr>
<td>From the International Non-Ionizing Radiation Committee of the IRPA.</td>
</tr>
</tbody>
</table>

Hence, the “international consensus” claimed is not as substantial as might initially be assumed. A broader group of scientists were involved in the U.S.E.P.A. 1990 report, 7 with the writing, 22 as reviewers and a further 9 who participated in a Peer Review
Workshop, totally 38 of the United States’ top EMR researchers and environmental health experts.

2.1.8 Available Evidence was not presented to the Court:

Many available published papers were not presented to the court. For example, Perry et al. (1981) studied the incidence of suicide near powerlines in the West Midlands of England and found “a significant correlation between suicide locations and the measured power-frequency magnetic field strength. Significantly more suicides occurred at locations of high magnetic field strength.” This result is consistent with depression which is associated with melatonin disruption in Seasonally Affective Disorder and studies presented here showing probable melatonin disruption with EMR exposure.

The Court can only deal with evidence which is placed before it. The eight of the most comprehensive research reviews of EMR health effects published to date are:

- 1990: The U.S.E.P.A. review “Evaluation of the potential Carcinogenicity of Electromagnetic Fields”
- 1997: The U.S. National Research Council report “Possible Health effects of Exposures to Residential Electric and Magnetic fields”.

The second, third and fifth are relevant to ELF and were available for the Rodney hearing in September 1994 but only thr third was referred to. The eighth is also about ELF effects but was not available for the Rodney case. The 1990 E.P.A. review includes the statement:

“in view of these laboratory studies (about EMR biological effects), there is reason to believe that the findings of carcinogenicity in humans is biologically plausible”.

It concludes its epidemiological review with:
“In conclusion, after examination of the available epidemiologic data over the past 15 years, there is evidence of a positive association of exposure to magnetic fields with certain forms of site-specific cancer, namely leukemia, cancer of the CNS, and, to a lesser extent, lymphomas.”

This report originally recommended that ELF be classified as a probable carcinogen and RF/MW as a possible carcinogen, Sibbinson (1990). This strongly conflicts with the evidence presented at the Rodney hearing which relied to some extent on the ORAU review.

The court recorded Dr McEwan’s evidence as concluding:

“that there was no convincing evidence to support the contention that exposures to extremely low frequency electric and magnetic fields generated by sources such as power lines are demonstrable health hazards; that epidemiological findings of an association between electric and magnetic fields and childhood leukemia or other childhood or adult cancers are inconsistent and inconclusive; that no plausible biological mechanism had been presented that would explain causality; and that lack of epidemiological support was consistent with calculations of quantities based on Fundamental laws of physics for describing electric and magnetic fields which showed that the fields induced in the human body from external extremely low frequency sources are generally much weaker than intrinsic fields created by the normal activity of the body. They concluded that any assessment of a health risk associated with fields emitted by those sources would be speculative and seemingly unjustified. Having made subsequent reviews of more recent studies, the authors of those reports considered that the later results did not give sufficient ground to change their conclusions.

In summary, it was Dr McEwan’s opinion that there is no established interactive mechanism which explains how biological cells might be affected by external weak fields; that epidemiological studies show a lack of consistency and had not established any causal association between health effects and exposure to extremely low frequency magnetic fields; and that while the evidence does not demonstrate a risk to health, if the risk does exist it would be very small.”

Plausible Biological Mechanism:

Dr McEwan ignores the evidence about melatonin reduction and calcium ion efflux and concludes: “that no plausible biological mechanism had been presented that would explain causality.” and “there is no established interactive mechanism”.

“Causality” and “established” are language from the “strong proof approach”. This is the approach adopted by the ORAU team and Dr McEwan. This approach is inappropriate in the RMA legal environment. Dr McEwan generally accurately reports the reviewers conclusions but in many cases the conclusions can be challenged. In chapter 2 of the review calcium ion efflux is dismissed as a biological mechanism because the fields associated with the observed effects are below the thermal threshold and the observed
thermal field voltage gradients of about 0.02 V/m at the cell level and an electric field gradient of 280 V/m across the cell membrane and the lack of a known physical coupling model.

Calcium ion efflux has been repeatedly observed in fields modulated at ELF frequencies at cell membrane exposure levels far below the thermal threshold, with non-linear responses being identified.

The icing on the cake for those taking a strong proof approach is their inability to accept an athermal biological effect when their physics calculations apparently show induced fields at cellular level which are less than the intrinsic fields of the cell membrane for example and hence are assumed not to be able to cause change. With ELF biological research it has been shown repeatably that the modulation is critical and the magnitude of the modulation can be very much smaller than a static field and still be detected and reacted to. For example Liburdy et al. (1993) found that melatonin reduces the growth rate of human breast cancer cells (MCF-7) in culture, but that 12 mG 60 Hz magnetic field completely blocks the protective effect of melatonin. They suggest a threshold between 2mG and 12 mG.

Changes in rodents’ pineal melatonin are dismissed for similar physics-based reasons in Chapter 2, by Dr Bennett of the Department of Applied Physics, Yale University. However, the chapter on neurobehavioural effects, co-authored by a medical researcher from the University of Texas, Dr Russell Reiter, author of many papers on melatonin, is much more positive about EMF reductions in pineal melatonin in rodents. It includes the conclusion: “Thus, at this stage, although pineal effects seem to have been documented in nonhuman mammals as a result of EMF exposure, in humans there in no evidence suggesting adverse health effects related to disturbance of pineal physiology.” “However the positive data relating to EMF effects on pineal serotonin metabolism should not be ignored.”

The ORAU review reported several nonhuman mammals which showed melatonin reduction with electric, magnetic and combined electric and magnetic fields. Yellon (1991) who studied the effect of 60 Hz magnetic fields on Djungarian Hampsters, and found daytime levels of Pineal and Serum Melatonin were the same but exposed animals has a lower and later nocturnal pineal peak and a much lower serum melatonin concentration. In relation to this study, pVII-27, “this finding makes a very strong argument for magnetic field effects on the circadian melatonin rhythm. Furthermore, the findings show that daytime exposures to such fields may have very significant consequences in terms of the subsequent night’s melatonin rise.”

The review's final conclusions include the statement: “When considered collectively, however, the findings suggest either a direct or indirect interaction of fields with at least one aspect of pineal metabolism, namely the nocturnal conversion of serotonin to the primary pineal hormone, melatonin.”

This and other statements within the ORAU report can be used to construct a “plausible biological mechanism” such as is required by the Court for the identification of a potential effect, but in a strong proof approach the possibility of melatonin reduction is as yet unproven in human beings and therefore is not part of a causal effect.

**Epidemiological evidence:**
Dr McEwan quoted the results of a review carried out by Dr Dockerty in Dunedin. Dr Dockerty reviewed 15 studies, only two of which reached the 95% confidence level of statistical significance. Dr McEwan’s primary source is the ORAU review carried out on behalf of the Department of Labour by an eleven member team from the Oak Ridge Associated Universities. They claim to have reviewed about 1000 journal articles which had been published in the past 15 years (to 1992). The total number cited which relate to biological effects is about 420 and to epidemiological effects is about 80.

A detailed reading of the ORAU report reveals considerable epidemiological and biological evidence of a potential effect but the conclusion the court recorded in relation to Dr McEwan’s use of this review was expressed in “strong proof” language.

Of the 35 epidemiological studies cited on leukaemia, for example, 23 have rate ratios greater than 1.0 and eleven of these reach the 95% confidence level. Of the 12 which have RR<=1.0, 8 have 40 or fewer exposed people with leukaemia.

Table 3 summarizes the integrated results of the occupational studies reviewed and clearly shows statistically significant Risk Ratios for proportional incidence and mortality and for Cohort studies. The case control studies were marginally non-significant. Notice that the review only covers papers published up to 1991.

<table>
<thead>
<tr>
<th>Leukemia Type</th>
<th>Design</th>
<th>Summary RR (95% CI)</th>
<th>References*</th>
</tr>
</thead>
<tbody>
<tr>
<td>All leukemia</td>
<td>Proportional mortality (n=618)</td>
<td>1.2 (1.1-1.2)</td>
<td>1,2,3,4,5,6,7</td>
</tr>
<tr>
<td></td>
<td>Proportional incidence (n=148)</td>
<td>1.2 (1.0-1.4)</td>
<td>8,9</td>
</tr>
<tr>
<td></td>
<td>Case-control</td>
<td>1.1 (0.9-1.3)</td>
<td>10,11,12,13,14,15,16</td>
</tr>
<tr>
<td></td>
<td>Cohort (n=599)</td>
<td>1.1 (1.0-1.2)</td>
<td>17,18,19,20,21,22,23,24,25,26,27,28</td>
</tr>
<tr>
<td>Acute myeloid</td>
<td>Proportional mortality (n=93)</td>
<td>1.2 (1.0-1.5)</td>
<td>2, 4, 7</td>
</tr>
<tr>
<td></td>
<td>Proportional incidence (n=55)</td>
<td>1.5 (1.1-1.9)</td>
<td>8,9</td>
</tr>
<tr>
<td></td>
<td>Case control</td>
<td>1.6 (1.2-2.0)</td>
<td>11,13,14,15,16,29,30,31</td>
</tr>
<tr>
<td></td>
<td>Cohort (n = 128)</td>
<td>1.2 (1.0-1.5)</td>
<td>21,22,23,26</td>
</tr>
</tbody>
</table>


If the Court had been shown this table then their conclusions could well have been different. In fact the data presented in the ORAU Table V-14 is consistent with the conclusions of the EPA 1990 review and Hardell (1995) with their greater number of reviewed papers.
In contrast, the Hardell review considered nearly 100 epidemiological studies, dozens of which achieved statistical significance, and hence the conclusions were drawn that cancers, particularly leukaemia, breast cancer, melanoma and nervous system tumours, are associated with ELF exposure. Similar conclusions were also found by the U.S. E.P.A. 1990 review.

Hardell et al. (1995) reviewed the scientific literature published up to July 1994 and concluded that there was:

- An increased risk of leukaemia in children and the existence of, or distance to, power lines in the vicinity of their residence.
- An increased risk of chronic lymphatic leukaemia and occupational exposure to low frequency electromagnetic fields.
- An increased risk of breast cancer, malignant melanoma of the skin, nervous system tumors, non-Hodgkin lymphoma, acute lymphatic leukaemia and acute myeloid leukaemia and certain occupations, involving design, manufacture, installation or maintenance of electronic or electric equipment.

The conclusions of the Swedish and U.S.E.P.A. reviews are very different from those quoted at the Court hearing by the experts for the applicant, even though there is a great deal of published review material held in common. It is the difference in approach to the interpretation of the data which leads to the difference in conclusion. A “strong proof approach” finds many reasons to dismiss evidence of highly probable health effects because it does not constitute causal proof. The language used is very important. For example the use of the word “risk”. Hardell et al. (1994) conclude from statistically significant epidemiological research that there is “increased risk of” many adverse health effects. On the other hand Dr McEwan used the absence of causal proof as evidence of no “demonstrated risk to health”. For public health epidemiologists, final causal proof of an effect is not necessary for there to be an avoidable risk of an adverse health effect.

2.1.9 Plausible Mechanism:

The Court decision on the application of the RMA provides some guidance on a “plausible mechanism” which is required to establish a potential effect.

The fact that an available plausible biological mechanism was not presented to the Court by the expert witnesses shows the role of evidence in RMA decision making and the problem when the expert witnesses appearing are unaware of the most recent research or take a selective approach to it on behalf of their client.

Reiter (1994) published a review of the research on melatonin suppression by ELF electromagnetic fields and the relationship to the reported increased incidence of cancer. From papers and reports published up to 1993, hence they were available for the Rodney Hearing, Reiter concludes that animal experiments show that ELF suppresses nocturnal melatonin, melatonin is a potent free radical scavenger and so is a “potent oncostatic agent and it prevents both initiation and promotion of cancer.” Reiter (1994) therefore states:
“Thus, if in fact artificial electromagnetic radiation increases the incidence of cancer in humans, a plausible mechanism could involve the reduction of melatonin which is the consequence of such exposures.”

Hence a plausible mechanism, melatonin reduction, was available, and a much larger set of epidemiological studies than cited by Dockerty, with statistically significant associations of ELF exposure with cancer incidence, was available, but was not presented by any of the expert witnesses. Hence the finding of no adverse health effects was made in the absence of the substantial body of evidence which was available but was not presented at that time.

2.1.10 Commentary:

Dr Beale has now completed and published his research in a highly reputable, international, peer-reviewed journal, and it does show statistically significant psychological and adverse health effects, with linear dose-response relationships for several health variables. Significant dose-response relationships, coupled to plausible mechanisms, are very close to being accepted as causal. Swedish studies, Feychting and Ahlbom (1993, 1995), identify linear dose-response (p=0.005) relationships for childhood leukaemia and magnetic field strength in relation to power line proximity.

Other internationally credible bodies had reviewed the international literature and concluded that there was strong evidence of adverse health effects associated with powerlines, contrary to the claimed “scientific consensus” deposed by Drs Repacholi and McEwan. These include a review team of U.S. EPA scientists, Sibbison (1990), the European Parliament (1992), Cleary (1994), and the Swedish reviews, Hardell et al. (1995), for example. Even the 1997 U.S. National Research Council (NRC) report “Possible health effects of exposure to residential electric and magnetic fields”, which was widely reported as a “no effects report”, had epidemiological findings which included the statement:

“We found the highest wire-code category is associated with a rate of childhood leukemia (a rare disease) that is about 1.5 times the expected rate.”

According to Dr Richard Luben, a member of the review panel, this conclusion survived the very high replicated study standard imposed by the review committee.

A scientifically comprehensive and objective presentation of studies available to the court in late 1994 would have more than satisfied the requirement to show a plausible biological mechanism and a potential adverse health effect from living near powerlines. The court was not presented with this data and made its findings accordingly. In giving its findings clear guidance is given about the nature and quality of the scientific evidence required. Subsequent publication of research results strengthens the conclusions which could have been drawn at the time of the hearing, that there are adverse health risks of living near powerlines.

The expert witnesses appearing for the applicant take a high proof approach, which in the RMA legal environment, effectively misled the Court into concluding that there were no plausible mechanisms and no risk of adverse health effects, when a more comprehensive approach based on seeking evidence of potential effects would have easily identified
reduced melatonin as a plausible mechanism for increased cancer, and there exists in epidemiological research evidence of a significant risk of leukaemia, especially acute myeloid leukaemia.

This case illustrates the way in which expert witnesses assist the applicant to construct a set of evidence which supports the granting of a Resource Consent. It is seen that this can involve careful selection of approaches and scientific studies which support the case. The Court appears to be unaware of the use of a strong proof approach being taken by witnesses for the applicant when the law under which the hearing is being held is a potential effects approach.

2.2 MacIntyre/BellSouth Case:

In New Zealand the Environment Court (as the Planning Tribunal) in 1995 heard the case of residents against BellSouth, and the decision is recorded as Decision A96/15 (MacIntyre Case), NZPT (1996). The decision of the Chief Planning Judge, His Honour Judge Sheppard, was based on the part if the New Zealand Standard NZS 6609 which advocates that exposure should be “as low as reasonably achievable” (ALARA), and on the application of the Precautionary Principle, in conjunction with Sections 5 and 3 of the Resource Management Act 1991 (RMA). The RMA requires that people “avoid, remedy or mitigate any adverse effects of an activity on the environment” (Section 5(2)(c)), including “any cumulative effect which arises over time or in combination with other effects - regardless of scale, intensity, duration, or frequency of the effect, (section 3(d)), and also includes” ... “any potential effect of low probability which has a high potential impact” (Section 3(f)).

The court finding summarises the case as:

“In this case, the applicant has asserted that there would not be an actual or potential effect on the environment from the radiation emitted from the cell phone transmitter; and it has adduced evidence tending to establish that. The appellants have challenged the applicant's assertion, and have adduced evidence which they claim will satisfy us that there is a potential of an effect of low probability but high impact on the environment from the health effects of radiation from the transmitter.”

The Court further defined the word “potential” as an adjective of: "capable of coming into being or action", “possible but not yet actual; capable of being or-becoming”

2.2.1 Expert evidence:

Dr Michael Repacholi, EMR Consultant to the World Health Organization, and Mr Martin Gledhill, Senior Scientist at the National Radiation Laboratory, presented scientific evidence on behalf of the applicant, BellSouth, while Dr Neil Cherry, Biophysicist at Lincoln University, Professor John Goldsmith, Head of Epidemiology, Ben Gurion University of the Negev, Israel, and Associate Professor Richard Luben, Medical Biochemist and Endocrinologist from University of California, Riverside, presented evidence for the residents in support of the appeal.

The evidence structure of the applicant was similar to that in the Rodney case. Mr Gledhill and Dr Repacholi both gave evidence that the cell site conformed to the national standard, NZS 6609, and to international guidelines.
Dr Repacholi gave detailed scientific evidence denying the existence of proven biological mechanisms and claiming that epidemiological evidence had not established adverse health effects. We can see the strong proof approach to scientific evidence by Dr Repacholi in this case. For example, in his evidence to the Planning Tribunal he stated:

Section 54.2: “To produce any adverse effect, RF exposure above a threshold must occur. This threshold level is the RF exposure needed to increase tissue temperature by at least 1°C. The low RF power levels from base transceiver stations cannot possibly cause this temperature rise.”

Section 55: “The International (IRPA 1988) RF standard is based on reviews of scientific literature which indicate (WHO 1993 - Repacholi (1993)) that a threshold exposure of 4 W/kg is needed before any adverse health effect occurs. For the cellular telephone frequencies 800-955 MHz the current International standard recommends an exposure limit of 400-470 µW/cm² (0.08 W/kg) for the general public.”

Section 54.4: “Exposure to RF fields has not been established to cause cancer.”

Section 54.9: “SAR (W/kg) of 0.00016 has no effect, and this is the level from cellular telephone base stations.”

Section 85: “On page 14 Dr Cherry suggests that a 2-3 fold increase in miscarriage occurred with a monthly mean microwave exposure of less than 1 µW/cm². This is patently absurd. Dr Cherry emits 0.3µW/cm², does he suggest if 4 people are in a room they will cause miscarriages?”

Apart from the final item, these statements summarize the official position of most national government bodies and standards setting bodies. Apart from the last sentence of Section 54.2 and Section 55, all of these claims are able to be shown to be scientifically wrong. They stand in strong contrast the European Parliament resolution, which was passed after extensive hearings of expert testimony, and will be shown conclusively here to be wrong. Taking the last point first, for example.

### 2.2.1 Repacholi Claim of 0.3µW/cm² of microwaves from Black body emissions:

Dr Repacholi’s claim that people emit 0.3µW/cm² of microwave radiation from Black body emissions so that 4 people would emit 1.2µW/cm² of microwave radiation is in itself clearly wrong and patently absurd. Dr Repacholi knows well the background to this because he is referring to his edited review report, Repacholi (1993), which in turn refers to his own published paper, Repacholi (1983). These both note that the 0.3 µW/cm² is emitted by black bodies “when integrated up to 300 GHz”. Over 0.22 µW/cm² of which is from above 100 GHz, which above the microwave radiation and is referred to as Extremely High Frequency (EHF) radiation and part of the Far Infrared portion of the spectrum. In the whole band from 100 to 1000 MHz the black body irradiance is only about 0.0092 pW/cm². (0.0000092 µW/cm²).

Dr Repacholi’s statement is also wrong because the number of people in a room makes very little difference to the total black body irradiance since the intensity varies as the...
fourth power of the absolute temperature and the surfaces of the room will be at comparable absolute temperatures to people’s skin and clothing surfaces.

Only under persistent cross-examination from John Fogarty Q.C. Dr Repacholi eventually reluctantly admitted that this claim was made "with tongue in cheek".

2.2.2 Dr Repacholi claimed the support of “Science”:

Dr Repacholi claimed that “science” supported his position. When asked what he meant by “science” he referred to the WHO 1993 review. He acknowledged that he had chaired the review team and was the technical editor of the review. He claimed that the report represented official WHO policy. He was then asked to read the statement in the opening pages of the review which states:

“This report contains the collective views of an international group of experts and does not necessarily represent the decisions or the stated policy of the United Nations Environment Programme, the International radiation Protection Association, or the World Health Organization.”

2.2.3 Characteristics of the Applicant’s Case:

Thus the applicants case in MacIntyre was similar to the evidence presented in the Rodney case, an international scientific consensus was claimed for the position that there were no plausible mechanisms and no epidemiology proved (casually) that there were adverse health effects.

2.2.4 Residents Case:

The residents’ case was based on establishing that there was at least one plausible biological mechanism that, according to Rodney, could be used to establish a potential adverse health effect. That there also existed cellular and animal experiments to give strong substance to the plausible biological mechanism and epidemiological studies which showed statistically significant associations with RF/MW radiation and adverse health effects. This would establish a potential adverse health effect from low level RF/MW radiation at levels well below the national guideline but exceeding levels of evidence required by RMA Section 3 (d), (e) and (f).

2.2.4.1 Overview of Scientific Evidence:

On behalf of the residents Dr Cherry gave the overview of the biological mechanisms and epidemiological studies showing statistically significant associations between cancer, brain changes and reproductive effects such as miscarriage from exposure to RF/MW radiation. He also summarised exposure data and showed that many experiments involved only a few hours per day and often only 5 days per week. Hence, if there was a cellular biological mechanism, the chances of which could lead to tumorogenic outcomes, a cumulative exposure characterised by the long term mean exposure was an appropriate measure to relate to a public exposure standard.

2.2.4.2 Biological Mechanisms for EMR effects:
Professor Luben gave detailed evidence on the biochemical changes observed in cellular systems with EMR exposure, including calcium ion efflux, signal transduction alteration, ornithine decarboxylase changes and the relevance of these to cancer. He concluded:

“Clearly, any environmental influence (e.g. electromagnetic fields) that modifies signal transduction pathways in normal cells could also influence the potentially tumorigenic pathways in susceptible cells, either by enhancing the likelihood of transformation by other tumorigenic stimuli or by acting in a directly tumorigenic manner. Thus, it is not necessary to hypothesize, as some have done, that EMF must cause genetic damage to cells in order to cause cancer or developmental abnormalities. Nor is it necessary to hypothesize that EMF must alter the expression of genes in cell directly (indeed, recent studies make this hypothesis seem rather unlikely). By influencing signal transduction pathways, which in turn regulate cell proliferation, cell differentiation, and even transformation to a cancer phenotype, EMF can potentially be involved in a host of disease processes without ever penetrating the cell membrane in any significant manner.”

Dr Luben referred to Byus (1994) that there was scientifically published evidence showing that athermal levels of RF/MW modulated at ELF frequencies had increased ODC in a significant and long-lasting fashion in cultured Reuber H35 rat hepatoma cells, following several other laboratories showing that ELF exposure had altered ODC concentrations in a wide range of biological media.

Hence plausible biological mechanisms have been identified, the primary one promoted by Dr Luben being altered signal transduction of cell regulation pathways which are potentially tumorogenic.

2.2.4.3 Epidemiological evidence of EMR effects:

Dr John Goldsmith referred to evidence of radar exposes being associated with testicular cancer, two previous studies which had been reported as “no effect” studies which he deposed both showed increases in cancer with radar exposure, namely, the U.S. Embassy in Moscow and the Korean War Study, a number of the epidemiological studies involving cancer and the U.S. Physiotherapist study associating early term spontaneous abortion with microwave exposure during diathermy. In addition he referred to measurements of changes to blood in microwave exposed people, including chromosome aberrations and somatic mutations. He related the blood changes to “a potential effect of high probability” and the data on cancer near broadcasting facilities and at the Moscow Embassy as “a potential effect of high probability which has a high potential impact.”

When asked in cross-examination whether epidemiological evidence on the adverse health effects of RF/MW could be described as a “weak link”, Professor Goldsmith replied (evidence transcript: p137, line 36):

“I disagree. I think when children die of cancer between 5 and 18 µW/cm² over a period of time - exposure is not weak. It is significant.”

Dr Goldsmith is referring to a 4 to 5-fold increase in childhood leukaemia in the U.S. Embassy in Moscow, the top floors of which were irradiated by a radar for several hours a
day, days a week and over several years. Dr Cherry pointed out to the Court that the mean exposure was in the range 1 to 2.4 \( \mu \text{W/cm}^2 \).

2.2.4 Treatment of the scientific evidence:

With high level scientific evidence and legal argument on both sides of the case, the Court gives detailed discussion of the legal and scientific evidence.

2.2.4.1 Status of the New Zealand Standard, NZS 6609:

The Court determined that compliance with the Standard is not decisive. They said that in the absence of challenge by another party resource consent proceedings are entitled to rely on compliance with the relevant New Zealand Standard. However, parties to a Resource Consent are not bound to accept that compliance “because New Zealand standards are not given particular status in law, parties must be free to assert that significant adverse effects on the environment would occur despite compliance with the standard. In this case the appellants assertion of environmental harm notwithstanding compliance with NZS 6609, “was supported by expert witnesses.”

2.2.4.2 Status of scientific publications:

Referring to a U.S. Supreme Court Case, Daubert v Merrill Dow Pharmaceuticals Inc., the decision, in which a lower court had rejected evidence because it hadn’t yet been published in peer-review journals. The Supreme Court ruled:

“402. All relevant evidence is admissible, except as otherwise provided. ... Evidence which is not relevant is not admissible.

702. If scientific, technical or other specialized knowledge will assist the trier of fact to understand the evidence or to determine the fact of an issue, a witness qualified as an expert by knowledge, skill, experience, training or education, may testify thereto in the form of an opinion or otherwise.”

In the RMA legal environment, when potential effects are considered on the basis of probative evidence:

“The Court [Supreme Court] noted that some propositions are too particular, too new, or of too limited interest to be published, but held that submission to the scrutiny of the scientific community is a component of ‘good science’, in part because it increases the likelihood that substantive flaws in methodology will be detected. So the fact of publication (or lack of it) in a peer-reviewed journal, while not decisive, is a relevant consideration on assessing the scientific validity of a particular technique or methodology on which an opinion is premised.”

On the treatment of evidence and discretion in decision making, Macintyre says:

“On the second issue, the Planning Tribunal is free to receive anything in evidence that it considers appropriate, and is not bound by the rules of law about evidence that apply to judicial proceedings. Even so, the basic
principles of evidence developed by the general courts provide a valuable guide for fact-finding by the Tribunal. It is our understanding that there are three requirements for us to make a finding on a question of fact. There needs to be material of probative value, i.e., tending logically to show the existence of facts consistent with the finding. Also, the evidence must satisfy us of the fact (i.e., that there will or will not be such an effect) on the balance of probabilities and having regard to the gravity of the question; but we are not to put either party to having to prove its assertion of fact beyond reasonable doubt. Further, the heart of a finding of fact is that we ourselves need to feel persuaded that it is correct.

We do not accept that the existence of a serious scientific hypothesis, or even one that is regarded as deserving priority for testing, is necessarily sufficient by itself to establish a potential effect, even a potential effect of low probability which has a high potential impact. Nor do we accept that the Tribunal should impose a threshold based on current scientific knowledge before taking notice of a scientific hypothesis. We hold that like any other evidence tending to establish a contested fact, the grounds for the hypothesis have to be exposed to testing (as discussed in Daubert's case) and scrutinised to determine whether they meet a basic threshold of reliability (as discussed in Mohan's case) to assist the Tribunal to weigh the evidence and make a finding one way or the other."

Application of the Precautionary Principle in the RMA:

“On the general precautionary principle, we note that a consent authority is entitled to have regard to any other matter not listed in section 104(1) which it considers relevant and reasonably necessary to determine the application; and that the definition in section 2(1) of the term "environment" extends to include people. The purpose of the Act is to promote the sustainable management of natural and physical resources. The term "sustainable management" is described by reference among other things to enabling people to provide for their health and safety.

There may be resource consent applications in which a consent authority may consider it relevant and reasonably necessary to have regard to the precautionary principle. In the context of the Resource Management Act the principle can apply to people and their health as well as for the rest of the natural and physical environment. So a consent authority may allow its discretionary judgment to grant or refuse consent to be influenced by the precautionary principle to the extent consistent with the statutory purpose of promoting the sustainable management of natural and physical resources and with judicial exercise of that discretion.”

2.2.4.2 Nature of the appeal relating to exposure limits:

This appeal was base on a public exposure limit applied by the Christchurch City Council of 50 $\mu$W/cm$^2$ being appealed by BellSouth who sought a limit of 200 $\mu$W/cm$^2$ as provided for by NZS 6609. The Court’s decision to set the public exposure condition at 2 $\mu$W/cm$^2$ based on the evidence before it, shows a clear and strong departure from the New Zealand Standard and the Christchurch City Council’s draft plan limit.
2.2.4.3 Court errors of fact and approach:

On page 37 of the findings the Court incorrectly implied that two studies which did show effects were no effects studies by claiming that Dr Silverman resiled from the Polish Military study which did show effects, when she resiled from the conclusions of the Korean War study which had incorrectly concluded that there were no effects.

The Court records the study of Dr M.C. Shandala and colleagues reporting stimulated changes to brains at microwave exposures of 10, 50 and 500 $\mu$W/cm$^2$. Even though it was pointed out (by Dr Cherry) that 10$\mu$W/cm$^2$ for 7 hours per day averaged 2.9$\mu$W/cm$^2$, the court concluded that “there was no evidence of health effects from exposures to radiation at or below 10 $\mu$W/cm$^2$.” The Court also incorrectly identified the Moscow Embassy exposures as estimates as not being below 10$\mu$W/cm$^2$. Sliverman (1980) details the measurements. The peak ranges from 5$\mu$W/cm$^2$ from 1953 to May 1975, 15$\mu$W/cm$^2$ from June 1975 to Feb 1976 and no more than 1 from Feb 1976 onwards. These values were measured on the outside walls of the embassy. The radar signals were not continuous but varied with time of day and time of year.

From these readings the all-time estimate of their mean value is in the range 1 to 2.4$\mu$W/cm$^2$ which is associated with a 5-fold increase of leukaemia in children. The Court received evidence of this mean range of exposure and the increase in childhood cancer. Hence there was evidence presented of adverse health effects below 3$\mu$W/cm$^2$. This makes sense of the Court’s decision to set the condition at 2$\mu$W/cm$^2$ when the applicant had agreed that operating conditions would not generally exceed 1.2$\mu$W/cm$^2$.

Hence it is incorrect and misleading to claim that the MacIntyre decision found that “there is no evidence of adverse health effects”. There was evidence of adverse health effects below 3$\mu$W/cm$^2$, from Shandala et al., the U.S. Physiotherapist study, Von Klitzing’s study and from the U.S. Embassy in Moscow, which enabled the Court to set the public exposure level at 2$\mu$W/cm$^2$ and say that with this condition there would be no known adverse health effects. The court is factually in error when it records that “there is no evidence of health effects from exposures at or below 10 $\mu$W/cm$^2$.”

An epidemiological methodological error found in the written decision relates to isolating studies rather than assessing the sweep of evidence. The Court decided to select only those studies it considered to be in the relevant narrow frequency and modulation range to be applicable to cell phone frequencies and modulations, rather than considering the wider question as to whether RF/MW could be considered to be carcinogenic, backed up by animal experiments and plausible biological mechanisms and a consistent pattern of epidemiological studies.

For example, the claim that the only radiation which is proven to be carcinogenic is ionizing radiation and that non-ionizing radiation cannot produce cancer, is disproven by noting that Ultraviolet radiation, which is non-ionizing, is shown to produce skin cancer.

The claim that photons of RF/MW radiation cannot initiate nor promote cancer because their energy is insufficient to break chemical bonds and hence cannot damage DNA and thus cannot produce a tumour, is at odds with the strong evidence of childhood leukaemia in association with proximity to high voltage powerlines and other cancers associated with occupational EMR exposures. Many researchers have asserted that there are many other
ways of initiating and promoting cancer than simply through damage to DNA. Research showing single and double strand DNA breakage in RF/MW fields implicates free radicals as the source of the damaged DNA, Lai and Singh (1997). Free radicals are also involved with DNA damage in the presence of ionizing radiation. Hence the biological mechanisms are very similar but for ionizing radiation the enhanced free radical concentration is a direct consequence of energy imparted by the quanta of ionizing radiation, whereas for non-ionizing radiation the reduction in the free radical scavenger melatonin results in enhanced concentrations of free radicals which have been produced by other processes, such as oxidative stress.

Evidence that studies had shown DNA breakage and chromosome aberrations in RF/MW exposure were presented to the Court. The melatonin mechanism was also presented but not with the compelling evidence of Reiter (1994) and Lai and Singh (1997).

It is inappropriate in public health effects to focus on only four studies and attempting to dismiss each one based on methodological weakness. Each study is part of a suite of studies which, taken together, form a strong set of evidence.

For example, the American physiotherapist epidemiological study followed two earlier studies from Sweden and Denmark which showed adverse pregnancy outcomes with those working with short-wave diathermy (Kallen et al. (1982) and Larsen et al. (1991)) and two rodent studies showing reproductive problems with low level microwave exposure, Il’chevich and Gorodetskaya (1976) and Chazan et al. (1983). Chromosome aberrations and melatonin involvement is recognised as causes of spontaneous miscarriage, Sandyk et al. (1992).

The Korean War Study can be compared with the Polish Military Study, Szmigielski et al. (1988 and 1996) and the U.S. Air Force Study, Garyson (1997), both of which found increases of cancer with RF/MW exposure. this gives at lease three studies showing cancer increase with exposure to military radio and radar.

Von Klitzing’s observations that human EEG’s change with exposure to modulated microwaves is consistent with Adey (1981), Dumanskiy and Shandala (1974), Shandala et al. (1979) and Gvopzdikcova et al. in McRee (1970).

Residential cancer increases as found in the U.S. Embassy in Moscow were also found in North Sydney by Hocking et al. (1996), in Hawaii by Maskarinec and Cooper (1993) and in Wichita, Kansas by Lester and Moore (1982a), as well as in the U.K. by Dolk et al. (1997 a,b).

One of the principles of epidemiology, Hill (1965), is to seek consistency to determine if the effect has been “repeatably observed by different persons, in different places, circumstances and times”. Another principle is to look for a “biological gradient” or dose-response relationship. Such a relationship is found in Dolk et al. (1997 a and b) which finds the adult leukaemia incidence peaks at some distance from TV and FM radio regional transmission towers, and then it decreases with distance in line with the decrease in exposure with distance.

Some of these papers were cited to the court in MacIntyre but the later published papers are included to demonstrate the approach and the strengthening of evidence over time.

2.2.5 The Decisions:
The Tribunal (A96/15) decided that a precautionary approach to the adverse health effects issue was warranted, p 49. In the next paragraph it states:

“However, this case focused on the possibility of adverse health effects from radiofrequency radiation, and after careful consideration of the evidence we have found that the transmissions would not have any actual or potential adverse effects on the public, not even a potential effect of low probability which has a high potential impact. This can be assured by the amended condition that we would substitute [2 \( \mu \text{W/cm}^2 \)], and by the provisions of the Act that could be invoked if it should turn out, contrary to the evidence before us, that the transmissions have an adverse effect, including the ability to review the condition.”

The converse of this is that without a condition of 2\( \mu \text{W/cm}^2 \), the finding of no actual nor potential adverse effect will not be assured. To underscore and strengthen this position the Tribunal stated the consent should be reviewed in the light of new evidence of adverse health effects near or below 2\( \mu \text{W/cm}^2 \). The Resource Consent was granted with the following condition:

“This 3. That the incident power flux density of radiofrequency radiation emitted by the facility, measured at any dwellinghouse, is not to exceed 2 microwatts per square centimetre.”

Hence, while setting the Public Exposure Limit at 2\( \mu \text{W/cm}^2 \), the decision requires the review of this condition if there is any new evidence. “New Evidence” includes re-evaluation and interpretation of the research presented to the original hearing if sound principles warrant that, as well as the results of studies not presented to the original hearing because of lack of awareness of them or access to them, or they were not published at that time. A great deal of new evidence is now available and will be presented in detail later.

**2.3 Evidence of Risk of an effect approach:**

Seeks sound scientific evidence of risk of an effect as a basis for risk reduction or risk avoidance through exposure reduction or minimization.

This approach seeks to identify evidence of risk of an effect in a sound scientific manner, not just reacting to suspicion of an effect. It looks for biochemical changes in cells which might indicate a possible effect, it views animal exposure experiments as providing indications that the cellular or molecular changes could have a human health effect since the effects are found in living mammal cells or organs. It looks at epidemiological studies which show statistically significant increases in incidence of disease in groups which are more highly exposed to a suspected disease agent, in which all identified confounding factors have been adequately dealt with, as evidence of risk of an effect.

The court decision in the MacIntyre case was seen as a win-win-win decision. The applicant was granted the resource consent. The residents obtained the strictest exposure condition in the western world and the scientists gained extensive guidance on the presentation and treatment of scientific evidence in an RMA legal environment.
2.4 The Beckenham Case:

Telecom and CCC Officers favor the Beckenham Decision (W165/96) of November 1996 for it reverts to the 200 μW/cm² exposure level of the standard NZS 6609, rather than the Maclntyre case which sets a public exposure limit of 2 μW/cm², based on probative scientific and legal evidence of potential adverse health effects as required by the RMA 1991.

Thus there was no contestable scientific evidence nor legal argument, and the Beckenham case has no standing in the issue of adverse health effects compared to Maclntyre which does.

The Beckenham was based on an appeal by Telecom NZ against the CCC decision to decline the consent based on visual impact. As it was explained in the Maclntyre case that if there was no challenge of the other party then a consent authority may treat the standard as setting an appropriate level of emissions. This is just what the court did. However the Court also has the discretion to set as lower public exposure level based on probative evidence, such as choosing to adopt the findings of the Maclntyre case. The Court chose not to do this. The findings include the statement:

“We agree with Mr Cooke-Willis that it is wrong in principle for the Court to set arbitrary limits on RF emissions from cell sites which are below those set by the relevant New Zealand Standard 6609: 1990, unless there is compelling evidence given in individual cases that the public interest requires such a course. There is no such evidence in this case.”

This implies that the Court accepted the Telecom evidence from Mr Cook-Willis and Dr Black, that the Maclntyre limits are arbitrary. This is demonstrably not the case. The limit of 2 μW/cm² lies below four cases of scientific evidence presented showing effects below 3 μW/cm² and the applicant’s agreement to operate at a maximum of about 1.2 μW/cm².

In the Beckenham case in an agreed memorandum defining the issues counsel for the appellant and the respondent recorded the following matters as being agreed:

“Any actual or potential health or environmental effects of alleged or perceived effects of radiofrequency emissions from the appellant’s proposed cellular base station are not issues which either party intend to raise or give evidence on, in this proceeding. The respondent does not raise these issues in opposition to the appeal.”

Despite this agreement between Telecom and the CCC, the minister of the Colombo Street Baptist Church, Mr D.W. Haliday, appearing for the church and the Archer Home, “did not share the view taken by the respondent (CCC) on the matter of the possible health risk to adjoining residents which may result from the building of the facility. To the contrary he took the view that it is the responsibility of the appellant (Telecom) to prove to an acceptable standard that there is no health risk.” This was occasioned by Telecom seeking the confirmation of the public exposure guideline of NZS 6609 against the decision of the Court in the Maclntyre case of 2 μW/cm².

Although Mr Haliday cited scientific evidence for possible health risk from that presented to the original resource consent hearing by Dr Neil Cherry and in articles and letters from
other professionals, he presented no professional legal nor scientific evidence by experts available for cross-examination. Hence the only expert witness evidence was provided by the appellant (Telecom).

The Court notes that, “It is only when the Court has decided that the evidence is sufficiently relevant and probative to be admitted to consideration that it is then enjoined to have regard to the matters raised in the sections of the Resource Management Act...”

The Court required the appellant (Telecom) to provide legal and scientific evidence based on the situation that “the onus of proof is [on] Telecom to persuade us on the balance of probabilities.”

It appears that in the absence of professional adversarial evidence the Court relies on the appellant to show at the level of balance of probabilities, that there is no adverse effect, but where there is contestable legal argument and scientific evidence, as in MacIntyre, the Court is able to apply Section 3 (f) of the RMA.

In the Beckenham findings, referring to Dr Black’s in summary of his evidence and expressing his view on the appropriate conditions, the findings record:

“Significantly, however, he concludes that it would be quite wrong in his view to set what he describes as pseudo standards which he is fearful is the approach being adopted by some councils in the light of the MacIntyre case. ”

Dr Black’s statement is highly challengable. It misrepresents the court’s decision in the MacIntyre case, but it reinforces Telecom's case which is arguing for the adoption of NZS 6609 in order to weaken the precedent effect of MacIntyre. Telecom was fearful of the court endorsing the findings of the MacIntyre case, which rejected the use of the public exposure guideline given in NZS 6609 and imposed and RMA probative evidence based condition at 1 % of the guideline based on RMA Section 3 (f).

In accepting Telcom’s legal and scientific evidence in the Beckenham case the Court could have exercised a discretion to adopt the condition set in the MacIntyre case, a public exposure limit of 2 μW/cm² at the nearest dwelling house. They chose not to, and decided in favour of Telecom’s appeal to apply NZS 6609’s 200 μW/cm².

It therefore appears that the Court in the Beckenham case has accepted Telecom’s incorrect and challengable interpretation of the MacIntyre case which led them to make their own highly challengable statement:

“We question the value of inviting this court to continue to entertain and make findings in respect of detailed technical and medical evidence in order to decide in each case whether on not a health hazard exists in relation to these facilities. We would have thought that by now the findings in cases such as MacIntyre and others v the Christchurch City Council Decision 15/96 dated 5/3/96 would have been sufficient, in the absence of any fresh evidence to ally concerns of residents about possible health hazards emanating from cell sites such as those proposed in this case.”
In making this statement the Court reveals its misinterpretation of the MacIntyre decision. If the Court and Telecom had accepted the public exposure levels in the MacIntyre decision then health evidence would not have been necessary in the Beckenham case. New evidence in relation to the MacIntyre case was presented to the Resource Consent hearing panel on behalf of the residents by Dr Neil Cherry, but was not available to the Court because of Dr Cherry’s absence overseas. The Court in exercising its discretion and by ruling in Telecom’s favour and not choosing to confirm the MacIntyre decision, has exacerbated the problem of protecting public health from exposures from cell sites.

Telecom and CCC officers by the way in which they misrepresent the Beckenham and MacIntyre cases in relation to adverse health effects are misleading the hearing.

Only witnesses on behalf of Telecom presented evidence on health effects. Dr Black mounted an argument around the premise that there are no proven effects. For example the findings state:

“Dr David Black notes that the only known, and scientifically proven effect of RF emissions on the human body is that heating occurs at certain levels of exposure.”

Dr Black does acknowledge that:

“there is a significant body of research which is looking at the possibility that there may be biological effects other than those caused by heating” but he goes on to say “to date however no athermal effects have been confirmed to the standard of proof required in scientific debate. The research is unconfirmed and anyway does not infer adverse effect or hazard to health.”

With the assistance of legal advice from Telecom and CCC, and scientific advice from Telecom’s witness, especially Dr Black who made some scientifically and legally challangable statements concerning epidemiological evidence, biochemistry and the MacIntyre case, instead of accepting the evidence and findings of the MacIntyre case, the Court itself made a highly challenging statement.

The evidence presented here challenges this statement. Some of the research findings in this report were presented in the MacIntyre, but none of it was presented to the Court in the Beckenham Case.

It is assumed by the Court that expert witnesses give objective evidence which is full and comprehensive to enable the court to come to an informed judgement.

I assert that in the Beckenham case, Dr Black’s evidence is carefully crafted to meet the desires of the applicant. He does this by using high proof language and a selective quoting of scientific research. The following demonstrates this through an analysis of Dr Black’s statements as quoted in Beckenham.

(a) Almost all of Dr Black’s statements about health effects are cast as a personal opinion only or in high proof terms.

For example:
1. Dr Black: “To date however no athermal effects have been confirmed to the standard of scientific proof required for scientific debate. The research is unconfirmed and anyway does not infer any adverse effect of hazard to health.”

2. Dr Black: “I am of the opinion that cancer from any level of RF is extremely unlikely. I could not even construct a hypothesis as to how cancer could conceivably be caused from these low levels of RF.”

3. Dr Black: “There is no evidence to actually link rises in ODC (which is the enzyme in question) with cancer promotion”.

4. Dr Black: “There is no coherent evidence that electromagnetic fields do actually affect cell membranes. Even if they might there is no reason to believe that this would pose a hazard to health.”

Personal opinion: 2. “I am of the opinion”, and “I could not even”, 4. “there is no reason”.

High Proof Language: 1. “have been confirmed to the standard of proof required in scientific debate”. “The research is unconfirmed”. 3. “Actually link”. 4. “no coherent evidence .. do actually affect” and “would pose a hazard”.

Comment 1: Calcium ion efflux is a well confirmed athermal effect of RF/MW exposure. The melatonin mechanism is a highly probable and therefore plausible mechanism for cancer, immune system impairment, spontaneous miscarriage, sleep, learning and memory disruption. Since there is statistically significant, multiple epidemiological evidence for all of these adverse health effects, and since there is a plausible mechanism and animal laboratory evidence, it is highly probable that RF/MW exposure increases the risk of, and incidence of these health effects. Dr Black has used a standard high proof approach to ignore or dismiss a mountain of evidence by implying that for there to be an adverse health effect there needs to be an established and proven athermal mechanism. Then he claims that there is no proven athermal effect therefore, he argues, there is no health hazard.

By moving away from the high proof environment to the potential effects environment we ask different questions and treat evidence in a different way. If we asked the question, is there evidence of an adverse health effect such as cancer from occupational or residential exposure to RF/MW radiation, based on internationally published, peer-reviewed research of statistically significant associations, sound exclusion of likely confounders and in some cases, dose-response relationships, the answer is YES !!!

That is, there is strong scientific evidence of a highly probable risk of a health hazard.

If we further ask if there have been controlled animal laboratory experiments which have shown health effects consistent with the epidemiological studies, the answer is YES !!!

If we further ask, have there been laboratory in vitro studies showing that cell lines change their behaviour in ways which are consistent with the above evidence, the answer to this is also, YES !!!

Comment 2: To avoid the many epidemiological studies which find statistically significant increased incidence of cancer or mortality of cancer for those exposed to RF/MW Dr Black refers only to his own opinion not the large published scientific studies. The fact
that Dr Black cannot conceivably construct a hypothesis of how cancer can be caused he ignores many published papers which do provide such hypothesis including Reiter (1994), Adey (1989, 1990, 1993a, 1993b), Luben (1995), Szmigielski et al. (1988), McLauchlin (1992), and Cleary (1994).

Comment 3: There is a great deal of evidence making a highly probable link, reported by Luben in MacIntyre, but the term used, which is high proof, “Actually link” is pedantically correct. This stands in strong contract to Professor Richard Luben’s evidence in MacIntyre, and Dr Luben is a well recognised EMR researcher and past president of the Bioelectromagnetics Society. He stated:

“The activity of ODC is believed to represent a measure of the rapidity of cell division; higher ODC levels are associated with more rapidly proliferating cells. This rapid proliferation of cells is a characteristic of cancer. Agents which stimulate ODC activity in some cells are known as tumor promoting agents. Byus and colleagues (Byus et al. , 1987) reported that three different cell lines, human lymphoma cells (CEM), mouse myeloma cells (P3), and rat heptoma (Reuber H35) cells exhibited increases of 50 to 300% in ODC activity when exposed to sinusoidal 60 Hz electric fields at 10 mV/cm (1 V/m). Increases in ODC were detected as low as 0.1 V/m in Reuber H35 cells. The investigators interpreted these results as indicative of and EMF effect on the cell membrane, resulting in a signal transduction effect on ODC activation by mechanisms not directly investigated in these studies.”

Comment: Dr Luben’s evidence, based on extensive published research, identifies the cell membrane as a primary point of interaction between ELF and RF and the cell. Professor Luben is backed up by a large array of scientific research. The well know calcium ion efflux observations relate to cell membrane induced effects by ELF and ELF modulated RF/MW. Adey (1990) reviews and summarizes Cell membrane transductive coupling and concludes the “Both low-frequency fields and radiofrequency fields with low frequency modulation can act separately and synergistically with chemical cancer promoters at cell membranes.” This confirms the cancer promotion and co-promotion effects of EMR and the site of activity as the cell membrane.

Comment 4: Calcium ion efflux occurs through the cell membrane. There is a high number of replicated studies of the calcium ion efflux from cells. This is a well confirmed athermal effect of modulated RF/MW radiation, related to modulation frequency not the exposure level, even down to SAR level of 0.00015 W/kg, Schwartz et al. 1990. Repacholi (1993) stated: “This field-induced effect is of interest because it occurs at SARs too low to implicate heating, and because calcium ions play a prominent role in the transductive coupling of many cell membrane-mediated responses.” Hence Dr Black is factually wrong in claiming that there are no confirmed athermal biological effects except heating. There are several others but this serves to illustrate the problem.

The calcium ion concentrations in cells is related to the fidelity of DNA replication, the cyclic AMP signal transduction pathway which regulates cell energy supplies, the protein kinase C activity which is fundamental for cell regulation, Adey (1990). Walleczek (1992) states in his review “Based on these findings it is proposed that membrane mediated Ca^{2+} signalling processes are involved in the mediation of field effects on the immune system.” The immune system produces killer cells which attack cancer cells (target cells) in an
attempt to eliminate them. These killer cells are T-lymphocytes and the act of killing the target cells is called cytotoxicity. Inhibition of cytotoxicity of T-lymphocytes was reduced by up to 20% by a sinusoidally modulated 450 MHz field (1.5 mW/cm$^2$), Lyle et al. (1983). Quan et al. (1992) showed that microwave heating of human breast milk eliminated a large proportion of the immunoglobulin factors for fighting E-coli bacteria, by up to a factor of 18 compared to conventional heating to 95°C.

Dr Black's carefully worded statements can all be justified in the context of a high proof approach, but in the RMA potential effects legal environment they are incorrect and mislead the court.

It is my experience as a Regional Councillor that in none of the Resource Consent Hearings I have sat on over the past 5 years has a lawyer nor an expert witness for the applicant knowingly given evidence which was likely to lead to the consent being declined. The applicant prepares a case to the best of their ability and resources with the purpose of gaining the resource consent. None of our Regional councillors and none of our Investigating officer staff can recall a case to counter this.

Hence our experience and the compelling logic of the situation suggests that it is self evident that legal council, planner and expert witness evidence on behalf of an applicant will be aimed at obtaining the resource consent, whether it is at the initial hearing stage or in the Environment Court.

This makes good sense of the Court’s evidence requirements as set out above in the three cases reviewed. The courts have exercised discretion and judgement based on their interpretation of the evidence presented and the legal advice given.

What has become clear also in every case the expert evidence on behalf of the applicant was based on a strong proof approach. In writing up the decisions and findings only in the MacIntyre case is the potential effects environment of RMA decision making properly recognised. However, in every case no comment was made and no indication was given that the court recognised and rejected the strong proof approach.

2.5 Comparative use of evidence for levels of legal or scientific proof:

In presenting scientific evidence in the RMA legal framework, it is important to note where the Section 3 (f) potential effect level lies compared to other levels of evidence and scientific proof, Table 4.

<table>
<thead>
<tr>
<th>Percentage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 %</td>
<td>Scientific Proof, Causation established. (NZS 6609)</td>
</tr>
<tr>
<td>99%</td>
<td>Multiple epidemiologic studies with dose response relationships, plausible biological mechanisms and animal experiments.</td>
</tr>
<tr>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>95%</td>
<td>Multiple epidemiologic studies, one with dose response relationship, plausible biological mechanisms and animal experiments.</td>
</tr>
<tr>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>90%</td>
<td>Highly Probable (Beyond Reasonable Doubt)</td>
</tr>
</tbody>
</table>
Dose response relationship with significant risk ratio and plausible mechanism, with animal experiments.

Animal experiment(s).

Multiple epidemiologic studies with statistically significant risk ratios and a plausible biological mechanism.

<table>
<thead>
<tr>
<th>51 %</th>
<th>Probable (Balance of probabilities, more likely than not)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 %</td>
<td></td>
</tr>
<tr>
<td>5 %</td>
<td></td>
</tr>
<tr>
<td>1 %</td>
<td>Potential effect of low probability and high potential impact.</td>
</tr>
<tr>
<td>0.000001 %</td>
<td>Potential effect, regardless of scale. Plausible mechanism</td>
</tr>
</tbody>
</table>

The MacIntyre Case is the only EMR case with competitive scientific evidence and legal argument and hence is based on Section 3 (f) and demonstrates the fundamental difference between the “strong proof” approach in the Rodney case, the lack of competitive probative evidence in the Beckenham case and the “evidence of risk” approach taken in the MacIntyre case.

3. Significant National and International Reviews:

3.1 European Parliament Resolution:

After extensive hearings, involving dozens of expert witnesses from around the world, the European Parliament on 19 March 1992 adopted resolution B3-0280/92, which included the following statement:

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

and the following clauses:

A. having regard to the significant increase, in the environment, of power density of non-ionizing electromagnetic radiation in the various frequency ranges, associated with technological development over the last few decades,

B. having regard to the precautionary principle included in Article 130r of the Treaty establishing the European Community and the ALARA principle (a-slow-as-reasonably-achievable), according to which it is necessary, in this case to minimize exposure to electromagnetic radiation,
C. whereas such radiation interacts with matter by non-thermal mechanisms and whereas, as regards radiofrequencies and microwaves, these are therefore added to the purely thermal interaction mechanisms,

D. whereas, according to an increasing number of epidemiological and experimental studies, even slight exposure to non-ionizing electromagnetic fields increases the risks of cancer, can be accompanied by nervous disorders and disruption of the circadian rhythms and seems capable of affecting developing organisms,

E. whereas the results of many in vivo and in vitro studies show increasing clearly the interaction mechanisms underlying such disorders and illnesses, centred mainly in cell membrane, lead to disruption of melatonin secretions, ornithine decarboxylase activity and T-lymphocyte efficacy, testifying to the probable role of non-ionizing radiation in promoting cancer,

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

The conclusions of the European Parliament strongly accept the adverse effects of EMR and more than fulfill the legal requirements of Section 3 of the RMA for the definition of an adverse health effect. In presenting new evidence, evidence not present to the European Parliament, conclusions beyond those of the New Zealand Environment Court and the European Parliament can be drawn that adverse health effects occur below $2 \mu W/cm^2$, and over 1000 times below the Australian/New Zealand guideline of $200 \mu W/cm^2$.

3.2 U.S. E.P.A. Reviews and draft recommendations:

The United States Environmental Protection Agency (U.S.E.P.A.) Health Effects Laboratory has a responsibility to study a wide range of toxic health studies including ELF and radiofrequency radiation health effects. A major review of RF/MW biological effects was published in 1984, Elder and Cahill (1984). In 1990 a scientific team of U.S. Environmental Protection Agency research staff produced a review of the then available literature. This was peer reviewed internally and externally. It recommended that:

a) ELF radiation be classified as a “probable human carcinogen”, and

b) RF/MW be classified as a “possible human carcinogen”.

The publication of this result was strongly opposed by the White House and the U.S. Air Force on the grounds of public alarm and national security, Sibbison (1990). In 1996, after years and years of delays, the review was left unpublished because of EPA budget cuts, much to the disappointment of the review team leader, Dr Robert McGaughy and the lead epidemiologist, Dr Doreen Hill, who both remarked that the evidence is now much stronger, Microwave News Jan/Feb 1996.

The recommendations were based in the E.P.A.’s public health protection mandate but the classifications were not made for political reasons. If the revised, stronger recommendations would have been published last year and history would have been made. The U.S. E.P.A. would have classified both ELF and RF/MW as highly probable human carcinogens. Even the U.S. Air Force now allows papers to be published linking
exposure to RF/MW and ELF to increased brain tumours in U.S. Air Force staff, Grayson (1996).

Hence the U.S. E.P.A. recommendations, consistent with the European Parliament recommendations, add significant weight to the evidence of an adverse health effect from public exposure to RF/MW radiation.

3.3 World Health Organization Reviews:

3.3.1 WHO 1981 Review:

In 1981 the World Health Organisation (WHO) issued a report "Environmental Health Criteria 16: Radiofrequency and Microwaves", Shore (1981). The human effects in the 1981 report rely heavily on the USSR and Eastern Europe studies with the exception of the Korean War Study, Robinette and Silverman (1977). The USSR and Polish studies showed that microwaves exposure under 10 mW/cm$^2$ produced “marked disturbance in cardiac rhythm, expressed by variability or pronounced bradycardia” and “a higher incidence of changes in the nervous and cardiovascular systems in the exposed group”. The initial findings of the Korean War Study in 1977 were “no significant differences were found between the 2 groups”.

The 1981 WHO report in part concludes:

"Effects have been reported at power densities too low to produce biologically significant heating."

and

"The general population includes persons of different ages (infants, small children, young adults and senior citizens) and different states of health, including pregnant women. The possible greater susceptibility of the developing fetus to microwave/RF exposure may deserve special consideration. Exposure of the general population should be kept as low as possible and limits should generally be lower than for occupational exposure. In view of the fact that data are still required to clarify interaction mechanisms and determine threshold levels for effects, it is recommended that microwave and RF exposure of occupationally exposed workers and the general population should be kept as low as readily achievable."

The 1981 WHO report, if coupled with the Precautionary Principle would have led the central and local government to adopt policies for the siting of radio and TV transmitters and cell sites away from places where children and women of child-bearing age would reside and/or work.

Between 1980 and 1992 there was a large volume of research published substantiating the concerns expressed in the 1981 report. Many published papers reported repeated studies on non-thermal effects such as calcium ion efflux, cell cycle changes, DNA breakage and chromosome aberrations. Several long-term rodent studies observed increased cancer and reproductive effects, and many epidemiological studies associated RF/MW exposure with increases in cancer, birth defects and miscarriage, for example. Some of these studies are reviewed in the 1993 WHO report, Repacholi (1993).
3.3.2 WHO 1993 Review:

The evidence of athermal effects has progressed from “Effects have been reported” in the WHO 1981 review to “A substantial body of data exists describing in vitro biological responses to amplitude modulated RF at SARs too low to involve any response to heating.” in WHO 1993 review (p 20 & 154), Repacholi (1993). Thus clearly recognizes the increased evidence of athermal effects which might well be biological mechanisms explaining and strengthening the epidemiological studies results. Several western epidemiological studies in the 1993 review report increased cancers with RF exposure and the report states: “However there are studies indicating an increase in cancer in RF field-exposed populations.” (p 167).

Despite the recognition of stronger evidence, the 1993 WHO review’s recommendations are weaker in terms of public health protection and moving more towards a high proof approach. This the 1993 WHO report emphasizes uncertainty, recommends carrying out more research and delays recommending precautionary risk avoidance, concluding:

“There is increasing concern about the possibility that RF exposure may play a role in the causation or promotion of cancer, specifically of the blood forming organs or in the CNS. Similar uncertainties surround possible effects on reproduction, such as increased rates of spontaneous abortion and of congenital malformations.

Effects of RF exposure on CNS function, with resulting changes in cognitive function are also surrounded by uncertainties. In view of the potential importance of these interactions and the disruptive effects of the uncertainty on society, a high priority should be placed on research in this area. It is important that research efforts be coordinated to clarify rather than increase the level of uncertainty. Research on possible mechanisms, such as weak field interactions, should be closely coordinated with appropriately designed animal toxicology studies and with human epidemiology.”

It should also be noted that several of the studies used in the 1993 review which are reported to show there are no cancer effects, actually do show increased risks of cancer with exposure to RF fields.

- Early results of the health effect of staff and children in United States Embassy in Moscow which is irradiated by a radar for many years showed no significant incidence of cancer, Lilienfield et al. (1978) but mutations in blood and increased cancer is reported in Goldsmith (1995).

- The cancer study in Wichita Kansas which showed a dose-response relation of cancer for populations exposed to no radar, one radar and two radars by Lester and Moore (1982a) was ignored even though it was in the same journal as Lester and Moore (1982b) which showed that county cancer rates were higher in counties which has air force bases which the authors related to the existence of radars. The 1993 WHO review team uses Polson and Merritt (1985) to claim contradictory findings, whereas in fact Lester (1985) accepts the criticism from Polson and Merritt that some cities in another county might be closer to a radar in a neighbouring county, re-evaluates the study by apply this correction.
This concludes: “Thus the 91 counties that contained cities nearest each of the 91 Air Force bases were found to have higher incidences of cancer than the control counties. This strengthens the possibility of an association between some factor associated with AFBs - our original hypothesis was microwave radiation - and cancer incidence because we now explicitly recognize the use of county containing the city nearest the base...”.

Since all of these papers were available and quoted by the review team, the failure to quote the conclusions of Lester (1985) raises the question of the possible bias approach of the review team towards the “no effect” stance.

- Analysis of the Korean War Study data, used in the 1993 WHO review as a “no effect” study, reveals that the identified high exposure group compared to the identified low exposure group has malignant neoplasms of 8.2 and 5.0 per 1000, with a risk ratio of RR = 1.66 (95% CI: 1.06-2.60).

The review team also ignores several other studies which do show increased cancer with exposure to RF/MW including Lin et al. (1985), Maskarinac et al. (1983), De Guire et al. (1987), Thomas et al. (1987), Preston-Martin et al. (1989), Johnson and Spitz (1989), Savitz and Chen (1990), Garland et al. (1990) and Tornqvist et al. (1991), for example.

In considering mutagenic effects the review claims that “In general, no changes in mutation rate has been observed, except in cases where substantial temperatures may also have occurred.” The WHO review reports only one exception, Yee (1982). This ignores Heller and Teixeira-Pinto (1959) [which was cited in the 1981 review], Sagripanti and Swicord (1986) and Garaj-Vrhovac et al. (1991, 1992), all of which report athermal condition increased incidence of chromosome aberrations with exposure to RF/MW radiation. Even though it was denied for many years, blood samples were taken from the staff of the U.S. Embassy in Moscow and these were shown to have increased mutations, Goldsmith (1995). This was clearly an athermal effect with peak external wall exposures of 15 μW/cm², mean external wall, upper floor exposes less than 2.4μW/cm² and mean indoor, all floors expose of less than 0.1μW/cm².

At a recent seminar involving international experts I asked if there was any scientific evidence showing that high temperatures and excess heat can cause cancer. The unanimous answer was “no”. There are no known scientific studies shown that high temperatures, which won’t kill an organism, has caused cancer, broken DNA or mutations.

Hence the WHO review team incorrectly interpreted the studies they used which they claimed to show no effects when the data published in the studies actually showed increased cancer incidence. They claimed contradictory findings when none actually existed and they ignored many studies which showed human cancer increased effects and chromosome aberration increases with RF/MW exposure, and where studies showed mutations they claimed that these were caused by heating. This adds considerable weight to the impression that there was a “no effect” and “strong proof” bias in the review team approach.

The strong proof (No effect) approach was revealed by the chairman of the review team, Dr Michael Repacholi in his evidence on behalf of BellSouth in the Planning Tribunal hearing in 1995 when he stated: “To produce any adverse effect, RF exposure above a threshold must occur. This threshold level is the RF exposure needed to increase
tissue temperature by at least 1°C.” This clearly ignores the epidemiologic evidence of cancer associated risk with RF exposure with life-time mean RF/MW exposures around and much less than 1 to 5 μW/cm².

There is extremely strong official government, military and industry pressure not to acknowledge the possibility or probability of adverse health effects from RF/MW radiation because of the clear and expensive implications of meaningful public health risk avoidance and legal liability. This pressure is reported by Sibbinson (1990).

The continuing WHO EMR program under Dr Repacholi’s leadership is under continual criticism for


Professor Cleary summarizes the scientific literature on EMR and health effects. He includes a discussion of interaction mechanisms, including hormonal alterations:

“Hormonal alterations have been reported to occur in laboratory animals exposed to ELF fields. Specifically, exposure affected biorhythms resulting in the suppression of the normal nocturnal increase in melatonin [Wilson et al. (1983,1986)]. This finding is potentially significant due to the interaction of melatonin with other hormones. Reduction in plasma melatonin concentrations causes increased levels of circulating steroid hormones such as estrogen and testosterone as well as increased protactin release by the pituitary gland [Reiter et al. (1990)]. Such hormonal alterations increase the rate of proliferation of breast tissue and suppress the immunological system, effects consistent with increased breast cancer risk [Stephens (1987)].”

After reviewing epidemiological, animal and in vitro studies he came to the following conclusions:

Epidemiological:

“Epidemiological studies, although quite limited in number, provide evidence of an association of long term exposure to RF/microwave and lower frequency EMFs and cancer incidence. Although a number of different cancers have been reported to result from such exposure, leukemia and brain cancer appear to be the most prevalent.”

Animal:

“Animal studies have also provided evidence that low-intensity EMR alters brain neurochemistry under conditions not involving tissue heating. Effects of microwave exposure on: (1) actions of various psychoactive drugs, (2) the activity of the cholinergic systems of the brain, and (3) on neural mechanisms in the rat, were investigated by Lai (1992). Neurological alterations were induced in specific parts of the rat brain by 45 minute exposures to 2450 MHz EMR at SARs of 0.6 W/kg. It was concluded that alterations of levels of endogenous opiates were responsible for the
observed EMR effects and that the effects depend upon the exposure parameters, Lai (1992). The results of these studies are of interest since they provide evidence that low-intensity EMR can alter brain function which is consistent with numerous reports of behavioral, neurological and neuroendocrine alterations in humans due to EMR exposure.”

In Vitro:

“In summary, in vitro studies of the effects of low-intensity RF and microwave radiation indicate dose rate dependent increases in neoplastic transformation frequency and proliferation. In view of limitations on the extrapolation of in vitro results to in vivo responses, these results cannot be related directly to cancer incidence in human populations exposed to such radiation. However, these results are not inconsistent with the hypothesis that human exposure to RF or microwave radiation, under presently not well-defined conditions, may affect cancer incidence.”

CONCLUSIONS

“There is increasing evidence of possible health effects of environmental exposures to EMFs and EMR in the home and in the work place. Epidemiological evidence indicates possible associations of long-term exposure and cancer incidence, adverse reproductive outcomes, and behavioral and neurological changes. Inherent limitations on exposure assessment, common to epidemiological studies, provide imprecise knowledge regarding time- or exposure intensity thresholds for these effects, thus making risk assessment difficult at this time. Whereas the results of animal experimentation and cellular studies of ELF EMFs and EMR effects are generally consistent with results of epidemiological studies, they provide insufficient data for meaningful risk assessment.

The greatest impediment to understanding the effects of EMFs and EMR on living systems is the limited knowledge of interaction mechanisms. One consequence is that research in this area has been treated with scepticism that has, together with other factors, resulted in serious limitations on research support. In view of the diverse nature of the physical properties of electromagnetic fields reviewed here, as well as the great variety of reported effects in living systems, the large gaps in our understanding are perhaps not surprising. The potential magnitude of exposure-related health effects in industrial societies indicates that these uncertainties must be resolved.”

Dr Cleary's review gives conclusions which support the U.S.E.P.A. review team's classification of ELF and RF/MW as probable and possible carcinogens and the probable causes of other adverse health effects.

3.5 Conclusions about reviews:

These national and international reviews provide more than sufficient evidence of a potential adverse health effect from RF/MW exposure to satisfy the section 3(f) level of proof. The material below provides an even more comprehensive review to strengthen this conclusion.
4. Levels of proof:

4.1 The Strong Proof Approach:

International and National EMR standards are based on established effects, the only one of which the bodies which set the standards will recognise is heating of tissue. This is a “strong proof” approach.

The “strong proof” approach is characterized above by Goldsmith (1995) by its rejection of epidemiologic information. The academic and professional roots of radiation standards committee members is Biophysics and Health Physics relating to ionizing radiation health studies. The history of investigations concerning cancer effects from Ionizing radiation reveal an initial reluctance among the scientists involved to accept that radioactive material which produces ionizing radiation was actually carcinogenic. Later it was recognized that the products of radioactive decay can ionize atoms, producing ions and free radicals which can damage DNA, cause chromosome aberrations and hence is mutagenic and carcinogenic. Scientists with this background and associated training have been a strong proponents of the strong proof approach in relation to the potential health effect of EMR. This largely relates to a conviction that unless radiation and ionize molecules it cannot cause cancer.

The nub of the basic difference of opinion between the two polarized sides of experts in this debate related to the existence of athermal effects which would provide a plausible mechanism to support epidemiological evidence of public health effects, in the absence of ionization. Those on national and international standards setting commissions and committees, and those presenting evidence on behalf of telecommunications companies and power companies take a strong proof approach and claim that the only proven adverse effects are heating and that athermal effects and public health effects are unproven and therefore cannot be used in standards setting nor Resource Consent decisions.

The strong proof approach is dominant in Government Radiation Laboratories, such as the New Zealand National Radiation Laboratory (NRL), Australian Radiation Laboratory (ARL) and the U.K.’s National Radiation Protection Board (NRPB). From these national bodies the representative take the strong proof approach into the IRPA, WHO and ICNIRP.

4.2 Evidence of athermal effects and health risks:

There is a well established “Public Health” approach, Hill (1965) and Goldsmith (1996), which is not generally applied in relation to standards setting for electromagnetic radiation, which is more dominated by a “strong proof” approach.

4.2.1 The Bradford-Hill ‘viewpoints’:

The ‘classic’ public health approach is outlined by Sir Austin Bradford-Hill in his presidential address to the then newly formed Royal Society section on Occupational Medicine on 14th January 1965. He was addressing the question of how much and what kind of evidence of epidemiological association was necessary to point to causation, as well as what level or type of association was of itself sufficient for prudent avoidance to be taken to reduce or eliminate the potential or probable risk. In his introduction he states:
"But with the aims of occupational, and almost synonymously preventative, medicine in mind the decisive question is whether the frequency of an undesirable event B will be influenced by a change in an environmental feature A. How such a change exerts an influence may call for a great deal of research. However, before deducing ‘causation’ and taking action we shall not invariably have to sit around awaiting the results of that research. The whole chain may have to be unravelled or a few links may suffice. It will depend on the circumstances."

Sir Austin outlines his factors or criteria as:

1. **Strength of Association:**

He gives the example of an enormous increase of scrotal cancer amongst chimney sweeps on one hand and the less than doubling of coronary thrombosis amongst smokers compared with non-smokers. He addresses the problem of confounders with the smoking group, refers a single occupational group, doctors, which showed increases the rates of lung cancer with increased cigarette usage concluding that the increasing ratios of incidence are “far more informative” that absolute differences in death rates.

Sir Austin finally refers to John Snow’s analysis of the 1854 cholera epidemic when the death rate from a sewage contaminated water source was 71 in 10,000 compared to a nearby water source which was sewage free, which was 5 per 10,000.

He concludes this section saying” In thus putting emphasis on strength of an association we must, nevertheless, look to the obverse of the coin. We must not be too ready to dismiss a cause-and-effect hypothesis merely on the grounds that the observed association appears to be slight. There are many occasions in medicine when this is in truth so.”

2. **Consistency of the observed association:**

This raises the question has the association been repeatedly observed by different persons, in different places, circumstances and times? This is to sort out those chance associations from real rare hazards. He says “Whether chance is the explanation or whether a true hazard has been revealed may sometimes be answered only by repetition of the circumstances and observations.” He points to a paradox that “The different results of a different enquiry certainly cannot be held to refute the original evidence; yet the same results from precisely the same form of enquiry will not invariably strengthen the original evidence. I would myself put a good deal of weight upon similar results reached in quite different ways, e.g. prospectively and retrospectively.

Once again looking at the obverse of the coin, there will be occasions when repetition is absent or impossible and yet we should not hesitate to draw conclusions.”

Sir Austin illustrated the obverse by referring to an example involving Sir Richard Doll’s study of nasal cancers among certain workers and pensioners from a particular chemical industry, and later only those working with a particular chemical process. The nasal cancer rate was 10 times expected but no causal agent was known for several decades.

Sir Austin states:
“No causal agent of these neoplasms has been identified. Until recently no animal experimentation had given any clue or any support to this wholly statistical evidence. Yet I wonder if any of us would hesitate to accept it as proof of a grave industrial hazard.”

(3) Specificity:

If the association is limited to specific workers and to specific sites and types of disease and there is no association between the work and other modes of dying, then clearly that is a strong argument in favour of causation.

He immediately warns, however, not to over-emphasize the importance of the characteristic, as even in the example he uses, there is a cause and effect relationship with two sites of cancer and how milk can be the source of a wide range of sicknesses. The actual example used is smoking and lung cancer which is raised by a factor of 9 to 10 in smokers compared to non-smokers. Sir Austin notes that there is specificity and a specificity in the magnitude of the association.

As with all other factors, he points to the obverse, that diseases may have more than one cause, indeed single causes he decides are not frequent. He then concludes:

“In short, if specificity exists we may be able to draw conclusions without hesitation; if it is not apparent, we are not thereby necessarily left sitting irresolutely on the fence.”

(4) Temporality:

The exposure to a disease causing agent must occur before the disease occurs, otherwise the agent cannot be associated with the disease. It can be more subtle, such as does a particular diet cause a disease or does the onset of the disease lead to a change in diet?

(5) Biological gradient:

“If the association is one which can reveal a biological gradient, or dose-response curve, then we should look most carefully at such evidence.”

Sir Austin again refers to smokers concluding:

“The clear dose-response curve admits of a simple explanation and obviously puts the case in a clear light.” “Often the difficulty is to secure some satisfactory quantitative measure of the environment which would permit us to explore this dose-response. But we should invariably seek it.”

(6) Plausibility:

“It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon biological knowledge of the day.” “In short, the
association we observe may be a one new to science or medicine and we
must not dismiss it too light-heartedly as just too odd.”

(7) Coherence:

“On the other hand the cause-and-effect interpretation of our data should not
seriously conflict with the generally known facts of the natural history and
biology of the disease.”

The examples used refer to smoking and cholera. Laboratory evidence of cancer of the
skin of laboratory animals in cigarette smoke and the lack of a biological agent in the case
of cholera from sewage contaminated water. Sir Austin is here giving the obverse that
the lack of a plausible agent to give coherence to the result did not weaken the
epidemiological case for cholera hazard.

(8) Experiment:

Sir Austin notes that occasionally it is possible and desirable to experiment when a health
effects is suspected from exposure to a possible disease agent because of an
epidemiological association. By removing the suspected disease agent, if the association
disappears than the cause-and-effect relationship is greatly strengthened but if the
disease association continues the association is significantly weakened.

(9) Analogy:

In some circumstances parallel effects can be compared by analogy.

Bradford Hill’s closing comments:

“Here are nine different viewpoints from all of which we should study
association before we cry causation. What I do not believe - and this has
been suggested - is that we can usefully lay down some hard and fast rules
of evidence that must be obeyed before we accept cause and effect. None
of my nine viewpoints can bring indisputable evidence for or against the
cause-and-effect hypothesis and not can be required as a sine qua non.
What they can do, with greater or less strength, is to help us to make up our
minds on the fundamental question - is there any other answer equally, or
more, likely than cause and effect?”

Sir Austin does not require that all of the nine viewpoints to be satisfied, as some have
suggested, rather he argues against this. He goes so far as to say that none of them are
essential conditions (sine qua non). A judgement is required in each case and the
viewpoints are valuable tools to assist in an assessment of the significance or otherwise
of a set of studies. Bradford-Hill’s approach is strongly at odds with the reductionist
approach used by the Court in the MacIntyre case. It is consistent with the approach
advocated by Professor Goldsmith and most other public health professionals.

4.2.2 Goldsmith on Epidemiology and EMR:
Eminent international epidemiologist, Professor John Goldsmith has rightly criticized IRPA EMR standards because of their failure to take into account the epidemiological findings of adverse public health effects, Goldsmith (1992): His abstract states:

“In standard setting there is a tendency to use data from experimental studies in preference to findings from epidemiological studies. Yet the epidemiological studies are usually the first and at times the only source of data on such critical effects as cancer, reproductive failure, and chronic cardiac and cardiovascular disease in exposed humans.

A critique of the protection offered by current and proposed standards for ionizing and non-ionizing radiation illustrates some of the problems. Similar problems occur with water and air pollutants and with occupational exposures of many types. The following sorts of problems were noted:

(a) Consideration of both thermal and non-thermal effects especially of non-ionizing radiation.

(b) Interpretation of non-significant results as equivalent to no effect.

(c) Accepting author’s interpretation of a study, rather than examining its data independently for evidence of hazard.

(d) Discounting data on unanticipated effects because of poor fit to preconceptions.

(e) Dependence on threshold assumptions and demonstration of dose response relationships.

(f) Choice of insensitive epidemiological indicators and procedures.

(g) Consideration of each study separately, rather than giving weight to the conjunction of evidence from all available studies.

These problems may be minimized by greater involvement of epidemiologists and their professional organizations in decisions about public health protection.”

Dr Goldsmith concludes:

“Existing standards (IRPA, and Tables 2 and 3), lacking appropriate use of epidemiological data, are not sufficient to provide a reasonable level of protection against long-term cancer hazards or against symptomatic, or physiological effects of R.F. radiation. This is in part because of the failure to evaluate currently available epidemiological information. Omission of critical studies occurs, as well as acceptance of a superficial conclusion concerning the meaning of the studies which were cited. The standards do provide a reasonable level of protection against thermal effects.
These remarks are also applicable to the recently published Israeli guidelines which propose to reduce the standards shown in Table 3 by 2/3 for new exposures. They are, in our opinion, a step in the right direction. However, due to the present lack of inclusion of current data on athermal (behavioral and chronic disease) effects, these guidelines cannot be considered an adequate basis for protection of the health of populations in the 1990s.

In conclusion, in setting health protection guidelines it is reasonable to expect that examples be given in which data on symptoms, carcinogenic hazards, and biological indices have been incorporated. When such data are available and are not used, the level of health protection may be insufficient.

Hence it is clear, from the standards documents themselves, from members of the standards setting bodies and from criticisms by epidemiologists, that RF/MW standards do not incorporate epidemiological findings and thus do not protect public health from adverse health effects.

The alternative, epidemiologically-based view holds that there is sufficient evidence of athermal effects to establish highly plausible biological mechanisms for cancer, brain and sleep disturbance, immune system impairment and reproductive effects, and sufficient animal and epidemiological evidence to require the setting of public and occupational exposures at much lower levels across the whole EMR spectrum.

5. Standards Setting:

5.1 History of Approaches:

Since RF/MW radiation is non-ionizing it is the widely held presumption in the Health Physics profession and in the Government Radiation Laboratories, for example, that RF/MW radiation is not mutagenic and carcinogenic because quanta of RF/MW lack the energy to ionize atom nor to break chemical bonds. This conviction lay behind the Tri-Service program in the United States, Steneck and Cook (1980) and is widely held in official government, military and industrial sectors today. With this assumption being widely adopted, the Tri-Service Program undertook extensive in-depth research in an attempt to determine the limits of the thermal effects which could be born by service personnel without thermal injury.

Michaelson (1971) summarizes the Tri-Service Program and the pioneering work of Professor Herman. Michaelson shows the extensive mathematical lengths which were taken to calculate the thermal effects of RF/MW exposure, especially by Schwan and his group at the University of Pennsylvania.

Dr Alan Frey, Frey (1988), in his outline of the historical development of research on low intensity non-ionizing radiation, points out that most of the research done during the 1960’s and 1970’s was irrelevant to his topic since the Department of Defense sponsors who determined what would be done were interested only in high power levels relevant to thermoregulation.

This was based to a significant extent on the notions about the nervous system function which Professor Schwan had developed. Schwan set up a mathematical model of the nerve’s axon membrane, and assumed that this was a reasonable representation of the
nervous system, Schwan (1969). His calculations with the model indicated that at field strengths that are “not thermally significant”, the induced potentials across the nerve membrane are many orders of magnitude smaller than the nerve resting potential. He stated that such induced fields applied to the resting potential of the axon cannot excite the nerves, and essentially, on the basis of this, he concluded that the nervous system could not be influenced by low intensity RF radiation.

For example, Schwan and Foster (1980) conclude:

“The considerations above do not suggest any weak nonthermal mechanism by which biological systems could react to low-intensity microwave fields.”

Dr Frey points out that he had identified two primary faults in Prof Schwan’s model. One was that the model was unrealistic. Nerves function, and the resting potential, is only one extreme of the continuum of potentials of the axon. Schwan ignored most of the nerve cell, including the most important part, when he considered only the axon in his model. Further, nerves interact with each other (see the dendritic cell in Figure 16). The points of interaction on the synapses are the most sensitive to disturbance, not the axon. Thus Schwan’s model, based on the resting potential of the axon, did not conform to reality.

Secondly, Schwan assumed that we had a good understanding of the nervous system at the time he developed his model. What we have learnt since then shows how complex it is. Cellular biochemistry has identified and quantified many processes in cells, including nerve cells, many of which are altered by very small and subtle EMR induced signals, through such processes as gap junction changes, ionic balance and signal transduction. Frey (1971) showed that by changing one of Schwan’s model parameters to a more realistic value, and then re-doing his calculations, leads to the conclusion that the nervous system would be affected by low level RF radiation.

Frey’s work in 1971 has been vindicated by many studies but those studies and Frey (1971) have been totally ignored by those favouring a thermal view. More sophisticated models are now in use with considerable experimental verification. These are outlined by Adey (1981, 1991) for example. Prof Adey concludes:

“To claim that there are no nonthermal mechanisms for the interaction of weak RF/MW signals with human and animal organs and cells is simply not scientifically credible.”

5.2 Setting the U.S. Standard C 95.1: Avoiding thermal hazard.

It is totally clear that the U.S. and subsequent standards are based on thermal effects. The U.S. safety standard C95.1 was set at 10 mW/cm² after a large number of experiments involving quite intense short-term exposure at this or somewhat higher levels, e.g. 50 to 200 mW/cm², with no apparent irreversible effects, Steneck and Cook (1980). They also report that a letter from the Raytheon Company to Senator Warren G. Magnuson, dated 31 August 1967, contended that:

“The Tri-Service program had led to ‘three basic conclusions’: the biological effects involved were (i) thermal, (ii) non-cumulative, and (iii) of little concern
since ‘man has a built-in alarm system coupled to his threshold of pain that protects him from thermal injury.”

Steneck and Cook (1980) note:

“In the push to set the standard, there can be no doubt that possible evidence against its safety was ignored and that research which might have clarified certain details was not undertaken.”

Steneck and Cook also note that: “few animal studies involving long-term exposure to low-level radiation were undertaken.”

In setting up one of the rare long-term animal exposure studies, Guy et al. (1980) note that:

“Although there are more than 5000 published articles in the literature pertaining to the biological effects of electromagnetic radiation, the question of whether long-term low-level radio-frequency radiation exposure is hazardous to health remains highly controversial.”

5.3 IRPA Standards - protection from thermal hazards:

The exposure standard of 10 mW/cm² is associated with a Specific Absorption Rate (SAR) of 4 W/kg which is referred to in Dr Repacholi’s evidence, Section 1.6.2. Repacholi (1990) states:

“The international (IRPA, 1988) and most Western Standards are accepted, on the basis of reviews of all appropriate scientific literature, a threshold RF exposure of 4 W/kg as necessary under normal environmental conditions to produce behavioural changes in animals. ..... Standards have generally required that limits of RF absorption be set that do not allow more than one tenth of the threshold SAR value (that is 0.4 W/kg) to be exceeded.”

Gandhi (1990) states that the observed highest rates of temperature rise in human subjects is given by 0.0045 x SAR (W/kg) in °C/min. An SAR of 4 W/kg will raise the temperature of a human body by about 1°C in 1 hour. For workers to avoid significant heating a safety factor of 10 is used giving an occupational exposure limit of 0.4 W/kg. Localized heating can occur in human bodies exposed to RF/MW radiation, especially in places such as the head, ankles and testes. Jammet (1988) says:

“The 'hot spot' range, extending from about 400 MHz up to 2000 MHz or even 3000 MHz, where significant localized energy absorption can be expected at incident power densities of 100 W/m² (10,000 μW/cm²). The size of hot spots ranges from several centimeters at 915 MHz to about 1 centimeter at 3000 MHz. Hot spots are caused by resonance or quasioptical focusing of incident fields. The former mechanism prevails at lower frequencies, the latter at higher ones (Foster et al. (1978) and Schwan (1982)). For the human head, the hot spot range extends from 300 MHz to 2000 MHz (Foster et al. 1978).”
It is also recognized that a person’s ability to dissipate absorbed RF/MW energy will also depend on their physical activity, the ambient temperature, exposure to bright sunlight and health status. Hence a further safety factor of 5, i.e. 0.08 W/kg is used to avoid RF ‘hot spots’ (Repacholi (1993) p 185 and p 189) and avoid heating of the chronically ill, frail people on very hot, sunny humid days, (p 177) Repacholi (1993).

Jammet (1984), reporting the IRPA exposure guidelines remarks

"The basic limit above 10 MHz (0.4 W/kg for occupational exposure or 0.08 W/kg for the general public) protects against potential thermal hazards."

Hence it is made clear by both the WHO technical group (Repacholi (1993)) and the IRPA that the 0.08 W/kg standard is a thermal protection standard and makes no attempt to protect from epidemiologically identified adverse health effects from chronic exposure to non-thermal levels of exposure to RF/MW radiation.

Michaelson (1971) also summarizes the animal experiments which were used to establish and validate the 10 mW/cm$^2$ “safe” exposure standard, which is openly and clearly related to the avoidance of heating effects.

A further indication of the thermal basis of the standard is the inverse “U” shape of the allowable exposure as it changes with frequency, following the inverse of the whole body resonant absorption curve.

5.4 Australian/ New Zealand Standard:

The EMR standards committee is a committee of Standards Australia and it is a joint committee with New Zealand. Over the post war years EMR standards in the West have been the interest of the military and industry. The Australian and New Zealand RF/MW standards sub-committee reflects this. Its membership is described as “stakeholders”. This is not universally accepted as the right way to go. People with a financial “stake” should not be involved in setting public health standards.

The U.K. air quality standards committee consists of 5 independent scientific experts with high standing in the community. They are not allowed to have any direct nor indirect financial involvement with industry. They are required to consult widely with industry, the community and with experts, and then to make recommendations based on epidemiology and toxicology, on public exposure levels which will not produce a detectable rise in public health risk.

In the U.K. the Government and the Public can have confidence that public health is being protected by these standards. They are reviewed regularly and have progressed downwards, with lower and lower allowable exposure levels to pollutants as epidemiological studies show adverse health effects at lower and lower levels of exposure. Contrast this with the membership and brief of the committee which originally set our radiofrequency public exposure guideline.

AS 2772 was set in 1990 by Committee TE/7 of the Australian Standards Association, comprising:

- Australian Electronics Industry Association
- Australian Radiation Protection Association
- Civil Aviation Authority
The membership illustrates the technical nature of the committee. One of the more benign looking members, Royal Adelaide Hospital, is represented by Dr Michael Repacholi, the then Chairman of the Committee, and a frequent consultant paid by industry. The committee has been expanded to include New Zealand representatives, including Telecom NZ (Mr Simon Cooke-Willis), NorthHealth (Dr Black), BCL Ltd, National Radiation Laboratory (Dr Andrew McEwan), Ministry of Commerce, Local Body Association and a Public Representative (Prof Ivan Beale). Both Dr Black and Dr McEwan have appeared as paid consultants to industry.

The International Radiation Protection Association (IRPA) and the Australia/ New Zealand Standard are acknowledged to be thermally based, thermally derived and thermally expressed. They are based on the Specific Absorption Rate (SAR) of radiation by human bodies and the body's ability to dissipate heat and thus retain homeostasis (a constant Core Temperature of about 37°C).

At the Waituna II workshop in Auckland, February 1998, and at a seminar sponsored by Telecom NZ on Monday 29th January 1998, Dr Miala Hietenan, Senior Lecturer in Non-Ionizing Radiation from Finland, and a member of the International Commission on Non-Ionizing Radiation Protection (ICNIRP) confirmed that the ICNIRP standard was based on “established” health effects and that the only established health effect of RF/MW was tissue heating. When asked if the standard was based in physics she said “yes”, and when asked why it did not relate to physiology, biochemistry or epidemiology she stated that there was only weak evidence which did not establish health effects.

Hence, it is clearly shown and admitted by those involved that the international and national standards are based on established effects only, and the only such effect of RF/MW is heating. As such they have no standing in New Zealand where actual and potential effects must be taken into account.

5.5 Mind changing case study:

*Once established, this thermally-based mind-set is very hard to change.*

The strength of the thermally-based position is very strongly held and very hard to change. Dr Michael Repacholi provides a good case study.

In November 1995 Dr Repacholi gave evidence in the MacIntyre Case in Christchurch to the effect that there are only thermal adverse effects and epidemiological studies, such as the Korean War Study showed no effects from radio and radar exposure of exposed service men.
A primary method used to dismiss adverse effects, the strong proof approach, is to require increasingly higher levels of proof, dismiss projects one at a time as insignificant, and to continue to recommend that more and more research is necessary to clarify uncertainties. In the light of the recommendations in the WHO review team’s report which he edited (and was chair of the task group) that “research efforts be coordinated to clarify rather than increase the level of uncertainty” and the careful design and execution of the project, Dr Repacholi and his research team were at pains to play down the implications of the results that cancer prone mice exposed to cell phone signals for 1 hour a day were observed to double their cancer rate.

Mice were chosen because evidence from mice is indicative of probable effects in people. To claim that the effects can be ignored because the happen in mice is not consistent nor credible.

This is definitely an athermal, carcinogenic result of RF/MW exposure.

Several other mouse or rat studies carried out in the U.S. have found increases of cancer incidence in mice or rats exposed to radar signals. It was previously claimed that these other projects could not be applied to cell phones and so until an experiment was done with an actual cell phone signal we shouldn’t worry about potential health effects. Now that this experiment has been carried out, and it was found that mobile phones double the lymphomas in B-cells of the immune system of these mice, there is little reason to assume that it couldn’t happen in the B-cells of the human immune system. However, Dr Repacholi still claims there is no evidence to link cell phones to cancer in humans. This ignores seven epidemiological studies which show increases in brain tumours associated with RF/MW exposure. Dr Repacholi stated in a TV link from Geneva to Australia:

“This is the first scientific study to show such an effect and as such in science it is necessary before you can really use this result for any health risk assessment, to repeat the study and extend it. By extending it I mean to look at other mouse models. Also to expose the mouse model to different levels of radiofrequency field so that we find out whether, first of all, this result was only a characteristic of these genetically engineered mice or whether it has wider implications for health. So we have a lot of research to do before the result can be used in the debate as to whether electromagnetic fields have an influence on cancer.”

Hence, while the study was specifically and carefully designed to answer a particular question as to whether there were any health risks from cell phones, according to Dr Repacholi, this study has not answered the question nor has it decreased uncertainty at all.

The timing of the release of the results poses some serious questions about expert advice in court when the expert involved has a commercial contract requiring silence until 3 months after the contractor is told of the results and the results are published.

The Adelaide experiment is a very important result, as Dr Repacholi now acknowledges, showing an athermal effect. “I believe that this is the first study showing a true nonthermal effect”, Dr Repacholi told Microwave News, May/June 1997.
The paper was received by Radiation Research on 8 July 1996. The paper was submitted to at least two other journals who declined to publish it, a process which could have taken at least six months. The writing up of the paper after the completion of the experiments would also take at least two or three months. Hence it is highly likely that final results were known by Dr Repacholi when he testified under oath in the MacIntyre case, and at least the 15 month results would have been known.

On 21-22 November 1996 the WHO held a workshop on athermal effects in Munich (organized by Dr Repacholi). Dr Repacholi’s mouse experiment results could not be presented here either. In fact Dr Repacholi circulated pre-prepared conclusions that evidence for nonthermal effects was weak at best. Papers presented identified the following athermal cellular responses to EMR exposure.

- release of calcium ions from chick brains, frog hearts, cat brains, rat brains and human cells.
- altered brain activity
- inhibition of T-lymphocyte cyclotoxicity
- decrease in non-cyclic AMP dependent kinase activity in lymphocytes
- transient increases in ornithine decarboxylase in various cell lines
- cell cycle alteration
- cell proliferation
- neoplastic formation
- biomolecular/biochemical changes
- membrane ion transport binding changes and altered single channel kinetics

Animal experiments involving athermal exposure reported showing effects including:

- Altered behaviour in cats, 147 MHz, 0.001 W/kg, AM at 1-25 Hz, Bawin and Adey.
- Structural rearrangement of DNA in mice exposed to 1 W/kg for 120-200 days, Sarker (1994).
- Single-strand DNA breakage, 0.6 and 1.2 W/kg for 2 hr, Lai and Singh (1994).
- Double-strand DNA breakage, 1.2 W/kg for 2 hr, Lai and Singh (1996).
- Increase in lung metastases in rats exposed to 2.45 GHz, Szmigielski (1982).
- Altered EEG rhythm in rabbits exposed to 1-10 MHz, 0.001 W/kg modulated at 14-16 Hz. Takashima.
• Ocular damage in primates exposed to continuous and pulsed radiation 2.66 - 7.8 W/kg, Kues.

As reported in the E.P.A. review, the Cleary review and the WHO 1981 and 1993 reviews, this workshop confirms that there is a growing body of scientific research which shows that very low, non-thermal levels of RF/MW radiation alters the basic biochemistry of cells which have a potential to cause altered brain function, carcinogenesis and impaired immune system functioning. Animal experiments show that these effects occur in living animals, including DNA breakage, cancer at many body sites, behavioural alteration, EEG change and ocular damage. Many epidemiological studies show increased risks of cancer and many other health effects with increase RF/MW exposure.

6. Biophysics and Geophysics factors:

In order to understand the way in which EMR can influence people it is helpful to be aware of the physical absorption factors, biochemical changes at the cell level and how these have been shown to change with EMR exposure.

6.1 Physical Variables, Units and Formulas Used:

At any point in the EM wave the electric field (E in V/m) and the magnetic field (H in A/m) are proportional to each other, such that \( E/H = 120\pi = 377\,\Omega \), the free space impedance.

The flux of E-M energy which is radiated through space is called the Energy Flux (S), and when it impacts onto an object it is called the Exposure. Exposure relates to the electric field through:

\[
S = \frac{E^2}{377} \quad [\text{W/m}^2] \quad (1)
\]

or to the magnetic field:

\[
S = 377\,H^2 \quad [\text{W/m}^2] \quad (2)
\]

where H is the magnetic field strength in Ampere/m (= A/m) which is related to the Magnetic Flux Density (B) through:

\[
B = \mu \, H \quad [\text{Tesla}] \quad (3)
\]

where \( \mu \) is the magnetic permeability, \( \mu \approx \mu_0 = 1.257 \times 10^{-6} \, \text{H/m} \) (H=Henry).

Exposure is frequently expressed in \( \mu \text{W/cm}^2 \).

\[
1 \, \text{W/m}^2 = 100 \, \mu \text{W/cm}^2 \quad (4)
\]

As the EM field propagates away from its radiating source (in the far field) the Energy Flux decreases as the square of the radius (r) and:

\[
S = \frac{P}{4\pi \, r^2} \quad (5)
\]

where \( P \) is the total radiated power, and \( r \) is the distance from the antenna.

Hence the Exposure of people and objects gets rapidly smaller as distance increases from a transmission facility, such as a radar, a cell site or a TV tower. For each doubling
of the distance the Exposure is reduced by one quarter. However, since $S$ varies as the square of electric field and magnetic field, $E$ and $H$ reduce linearly with distance from the antenna in the far field condition.

For an isotropic antenna, radiating equally in all directions, the exposure at any distance from the antenna can be calculated from Eq (5). Almost all commercial antennae have considerable directional focus to send their radiation more intensely in chosen directions so that TV, radio and cell site stations direct their signals towards potential receivers and not out to space. Hence for a given antenna power ($P$), the exposure at a given radius will be greater than that given by Eq. (5) by factor related to the Antenna Gain ($G$). The radiation pattern is further complicated by the existence of side-lobes in addition to the primary beam. Hence close to an antenna the exposure pattern is complicated. In the more distant field, outside the influence of the side lobes, the intensity decreases as the inverse square law of Eq.(5), but with a higher initial power determined by the antenna gain characteristics modified by the loss characteristic of the feeds etc. An overall gain factor of 6 to 7 is common for omni-directional antennae on Telecom cell sites.

The Energy Flux which impacts on a object is reflected, scattered and absorbed by the object. The proportion of the energy absorbed is a function of the wavelength of the wave compared to the linear dimension of the object. The most efficient energy absorption occurs when the wavelength of the EM wave is close to twice the size of the object. Larger objects have more efficient energy absorption at longer wavelengths, i.e. at lower frequencies, while smaller objects have higher absorption efficiencies at shorter wavelengths and higher frequencies. A 1.8 m man has a peak absorption rate at about 70 MHz, a monkey at about 300 MHz, an adult head at about 915 MHz and a mouse at about 2,450 MHz.

On a gross scale the energy absorption is expressed according to the incident absorbed energy’s heating ability and it is expressed as a Specific Absorption Rate (SAR), which in terms of the incident electric field is (Gandhi (1990))

$$\text{SAR} = \sigma \frac{E^2}{2\rho} \quad [\text{W/kg}]$$ (6)

where $\sigma$ is the electrical conductivity of the tissue, in Siemens/m$^2$ or S/m$^2$, and $\rho$ is the density of the tissue, in kg/m$^3$.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>100 kHz</th>
<th>1 MHz</th>
<th>10 MHz</th>
<th>100 MHz</th>
<th>1 GHz</th>
<th>10 GHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skeletal Muscle</td>
<td>0.50</td>
<td>0.72</td>
<td>0.83</td>
<td>0.90</td>
<td>1.42</td>
<td>11.5</td>
</tr>
<tr>
<td>Liver</td>
<td>0.16</td>
<td>0.28</td>
<td>0.45</td>
<td>0.66</td>
<td>0.98</td>
<td>8.9</td>
</tr>
<tr>
<td>Spleen</td>
<td>0.62</td>
<td>0.63</td>
<td>0.67</td>
<td>0.89</td>
<td>1.2</td>
<td>10.1</td>
</tr>
<tr>
<td>Kidney</td>
<td>0.25</td>
<td>0.37</td>
<td>0.59</td>
<td>0.86</td>
<td>0.98</td>
<td>9.7</td>
</tr>
<tr>
<td>Brain</td>
<td>0.15</td>
<td>0.18</td>
<td>0.42</td>
<td>0.72</td>
<td>1.00</td>
<td>9.1</td>
</tr>
<tr>
<td>Bone</td>
<td>0.014</td>
<td>0.017</td>
<td>0.024</td>
<td>0.057</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

At ELF frequencies electrical conductivities are 0.1-0.35 S/m for cardiac muscle, 0.1-0.3 S/m for nerve tissue, 0.2 S/m for cerebral tissue and 0.25 S/m for myocardial tissue, Repacholi (1993). Typical values for the electrical conductivity of tissue for MW are 0.05
S/m for bone, 0.95 S/m for muscle and 0.77 S/m for visceral organs such as heart, liver, brain etc.

Density is close to 1000 kg/m$^3$ for most tissue because of the presence of water. However lung tissue is about 100 kg/m$^3$ since it contains pockets of air.

While in terms of the tissue heating rate it is:

$$\text{SAR} = C \frac{\Delta T}{dt} \quad (7)$$

where $C$ is the specific heat of the tissue, in J/kg-$^\circ$C, and

$\frac{\Delta T}{dt}$ is the rate of increase in tissue temperature ($^\circ$C/s)

Combining Eq. (1) and Eq. (6),

$$S = \left(\frac{2\rho}{3.77\sigma}\right) \text{SAR} \quad [\mu\text{W/cm}^2] \quad (8)$$

and using $\rho = 1000$ kg/m$^3$

$$S = 530.5/\sigma \quad \text{SAR} \quad [\mu\text{W/cm}^2] \quad (9)$$

Using the data in Table 1 a relationship between $\sigma$ and frequency has been derived, for example,

for brain tissue: $\sigma = 0.27 + 0.0973 \ln (f) \quad (10)$

and for muscle tissue: $\sigma = 0.672 + 0.0877 \ln (f) \quad (11)$

where $f$ is the frequency in MHz.

Hence, for example, an SAR of 0.00015 W/kg for isolated frog hearts at 240 MHz ($\sigma = 1.15$ S/m) corresponds to exposure of 0.1$\mu$W/cm$^2$ and for Von Klitzing’s human brain EEG at 150 MHz ($\sigma = 0.76$ S/m) and 0.001 W/kg to 0.7$\mu$W/cm$^2$.

The estimate of the SAR for a whole body or body part is a complex calculation because of different tissue densities and electrical conductivities of each tissue type, and the variable size of components of the body which influences the efficiency of absorption of the EMR, all of which varies with wavelength and frequency of the EMR.

Gandhi (1980) gives empirical formulae for the whole body averaged SAR for a 1 mW/cm$^2$ exposure as a function of the signal frequency, for when the electric field vector is parallel to the length dimension. Expressing the coefficient in Eq.10 as $R (=2\rho/(3.77\sigma))$ and using the units for $R$ of W/kg per $\mu$W/cm$^2$, Gandhi’s model is:

Resonant Frequency $f_r = 114/L$ MHz

$$S_{res} = 15.2 \sqrt{(L^3/m)}$$

For the sub-resonant range: $0.5 f_r < f < f_r$

$$R = 5.2 \times 10^{-3} L^2/m \quad (f/f_r)^{2.75} \quad (12)$$
For the supra-resonant range: \( f_r < f < 1.6 S_{res} f_r \)

\[
R = 0.595 \frac{L}{(m \cdot f)}
\]

where \( f \) is the frequency of the incident signal in MHz.
\( L \) is the long dimension in m, and
\( m \) is the mass of the person in kg.

For example, for an incident signal of 300 MHz, an adult with \( L = 1.8 \) m and \( m = 80 \) kg, \( f_r = 63 \) MHz, it is in the supraresonant range and \( R = 4.44 \times 10^{-5} \). For a child with \( L = 0.9 \) m and \( m = 25 \) kg, \( f_r = 127 \) MHz and \( R = 7.14 \times 10^{-6} \). Hence an incident RF signal at 300 MHz and power density of 20 \( \mu W/cm^2 \) would produce an SAR for the adult of 0.0009 W/kg and the child of 0.00179 W/kg, two times higher for the child than the adult. This ratio remains the same for all frequencies since it is determined by the \( L/m \) ratio.

Figure 1 shows the calculated SAR levels produced by a content energy flux of 1 mW/cm\(^2\) as a function of frequency, for various body sizes.

When an electric field is induced in a person by the incident RF signal, an electric current flows through the person to earth. Gandhi (1990) has shown that the electric current which is flowing through the feet of a grounded man (\( I_h \) in mA) as the result of an incident electric field \( E \) (V/m) is given by:

\[
I_h = 0.108 \cdot E \cdot h_m^2 \cdot f \tag{13}
\]

Figure 1: The average SAR for 3 species exposed to 1 mW/cm\(^2\) with the E vector parallel to the long axis of the body, Durney et al. (1978).
For a 1.75m person at 40 MHz, at the limit field exposure for the ANSI C95.1 safety standard, 63.2 V/m (1 mW/cm$^2$) this gives a current of 836 mA.

The localized SAR is a function of $I_h$, which flows down two legs and since $\text{SAR} = J^2/\sigma \rho$,

$$\text{SAR} = \frac{(I_h/2)^2}{(A_e^2 \sigma_c \rho)}$$

where $J$ is the current density (A/m$^2$), $A_e$ is the effective cross sectional area of the legs, at the ankles it is about 9.5 cm$^2$ even though the physical cross section is about 40 cm$^2$. Hence, assuming a density of 1000 kg/m$^3$ and $\sigma_c=0.77$ S/m, this person has an SAR at their ankles of 251 W/kg. The ANSI standard is based on thermal protection and states that SAR should not exceed 8 W/kg for any 1 g of tissue in occupational exposures and 1.6 W/kg for public exposures. Since the exposure $S$ is proportional to SAR, to protect a well grounded adult person from heat exceeding 1.6 W/kg in the ankles the limit exposure near the resonant frequency would need to be reduced to 6.4 $\mu$W/cm$^2$. Insulating people with rubber soled shoes for example, reduces the induced current by 60 to 80 %. However, very young children frequently play with bare feet and so can often be in a well grounded state.
6.2 Thermal limits for young children:

An exposure of 1 mW/cm$^2$ (whole body mean SAR=0.4 W/kg), for a 10 yr old child (h = 1.38m, f = 50.7 MHz, $A_e=6.1$ cm$^2$) SAR = 371 W/kg; for a 5 yr old child (h = 1.12 m, f = 62.5 MHz, $A_e=4.2$ cm$^2$) SAR = 534 W/kg (Gandhi (1990) and for a 2.5 yr old child (h= 0.9 m, f = 74 MHz, $A_e= 3.0$ cm$^2$) SAR = 603 W/kg. Hence, using simple ratios since S is directly proportional to SAR, for children the allowable exposure to limit the heating of any 1 g of tissue to 1.6 W/kg is $4.2 \mu W/cm^2$ for a 10 yr old, $3.0 \mu W/cm^2$ for a 5 year old and $2.65 \mu W/cm^2$ for a 2.5 yr old.

Professor Gandhi notes that an empirical fit to heating rate as a function of SAR yields $0.0045 \times$ SAR (W/kg) °C/min. If a 2.5 yr old toddler is exposed to 1 mW/cm$^2$, then the heating rate will be 2.73 °C/min. Prof Gandhi concludes that for the ankle section “substantial rates of surface temperature elevation are anticipated.”

6.3 Standards give inadequate protection even for thermal effects in children:

The Australian and New Zealand joint standard does not protect from these localized heating effects through its use of electric field intensity and related exposure limits based on whole body average SARs only, allowing 0.4 W/kg for occupational exposure and 0.08W/kg for public exposure.

Even at our lower public exposure level of 0.08 W/kg maximum heating rates in toddler’s ankles is about 0.55°C/min. This has serious implications when children are exposed to FM signals in the frequency band 20 to 150 MHz especially. Since the public exposure standard must be adequate to protect even the most vulnerable, if they are set to only deal with thermal effects, then in the sub-150 MHz range they should be adequate to protect a baby from adverse heat levels. This would require the allowed maximum level to be set at 2 $\mu W/cm^2$ or less, simply to meet the 1.6 W/kg limit.

These limits will be somewhat higher away from the optimal frequency for maximum induced current flow, but these figures illustrate the vulnerability of body parts to high levels of localized heating and to the greater vulnerability of young children.

While short-term exposures can give dangerous heating effects down to exposure levels far below the current “safety standard” when near the absorption frequency maximum, legitimate concerns exist about changes in our brains and at the cellular level the short-term exposures might cause. There is also clear evidence about chronic exposures at far lower levels of exposure which have the potential to alter the ambient electromagnetic environment in ways which are potentially harmful through effects on reproductive processes, brain function and metabolism, sleep disruption, immune system suppression and EMR probably causes an increase in the risk of cancer.

6.4 Natural Electromagnetic Environment:

The earth’s static magnetic field is about 30-50 $\mu T$ and static electric field (in fair weather conditions) is about 150 V/m. Our bodies are well adapted to these static fields and to the radiation from the sun. We are now seeing how small changes to a minute part of the solar spectrum, UVA and UVB, are producing significant increases of skin cancer in non-black skinned people. Legitimate concern can be raised about the significant increases in
population exposures to other parts of the EM spectrum from ELFs to millimeter microwaves.

Figure 2: The signal of a typical cell site near the mast ($2 \mu W/\text{cm}^2$) against the thermal radiation background in the frequency range around that used by cell sites.

Our background thermal environment emits around 400 W/m$^2$ (40,000 $\mu W/\text{cm}^2$) of "thermal" radiation, the vast majority of which is in the infrared. The part of this which forms the natural sources of oscillating EMR fields summed over the range up to 300 GHz is 0.3 $\mu W/\text{cm}^2$, Repacholi (1983). In the radiofrequency range it is less than 10 pW/cm$^2$ because over 99.996% of the 0.3$\mu W/\text{cm}^2$ comes from above 10 GHz, whereas most of our spectrum use for telecommunications, (radio, TV, RT and cell phones, satellite communication, radar) are in specific frequency bands below this frequency. Hence from the ELF to RF/MW parts of the EM spectrum we have massively increased our ambient exposure, especially following developments since the Second World War.

Figure 2 shows how a cell site local exposure level of 2 $\mu W/\text{cm}^2$ stands out against the thermal background, by a factor or about $2 \times 10^8$.

The ionosphere has a net positive charge which creates a static electric field and the dynamics of thermal convection in the earth’s core produce the earth’s magnetic field. Thunderstorms create short-term localized field variations as they strongly alter the local electric field and lightning produces emissions broad spectrum emissions of RF/MW in bursts called sferics. These can be heard on radios as a burst of static.

Long-term natural ELF signals arise from thunderstorm sourced electromagnetic energy being ducted around the world in the cavity between the earth and the ionosphere. These are called the Schumann Oscillations. Their fundamental frequency is 7.8 Hz, with
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harmonics at 14.1, 20.3, 26.4 and 32.5 Hz. Their amplitude is about 0.2 mV/m for a 1 Hz bandwidth, Campbell (1967). Assuming each resonant frequency is associated with a 1 Hz bandwidth, the total field strength will be 1 mV/m or 0.1 V/cm. From Eq. 3 this is equivalent to an energy flux of 0.27 pW/cm$^2$.

6.5 Modification of the natural environment:

While life on earth is well adapted to the static magnetic and electric fields and to minute intensities of naturally occurring ELF and RF/MW fields primarily from thunderstorms, biological tissues and organisms have, in this century, become exposed to ELF, RF/MW (VHF and UHF) fields at intensities which are thousands of times higher than they were at the turn of the century (<10 pW/cm$^2$). The median public RF exposure measured by the U.S.E.P.A. in U.S. cities in 1979 was 0.005 $\mu$W/cm$^2$ or 5,000 pW/cm$^2$, Tell and Mantiply (1980). About 1% of the population lived in more than 1 $\mu$W/cm$^2$ or 1 million pW/cm$^2$.

6.6 Healthy vs vulnerable people:

People who live or work close to transmission facilities have mean daily exposures of about 1 to 20 $\mu$W/cm$^2$, but when averaged over a year their mean is of the order of 0.2 to 5 $\mu$W/cm$^2$. In the work and military environment a “healthy worker” effect exists. Employment selection screens out the sick, young and elderly. Hence while occupational and military exposures can be somewhat greater than average in certain cases, studies on these workers tend to significantly underestimate the effect similar exposures would have on the vulnerable in society. It is the vulnerable groups in which overall sickness rates are higher. So when reviewing national morbidity and mortality statistics the vulnerable groups are over represented and there are large groups of “healthier than average” people who are under-represented in the health statistics.

The IRPA RF exposure standard is an acknowledged thermal standard based on an occupational exposure limit of 0.4 W/kg. Greater protection is offered to the general public because this includes the vulnerable. Hence the Public Exposure Limit of 0.08 W/kg, which will avoid burns, and slight heating effects in infants and the frail elderly on hot sunny days when they exercise, Repacholi (1993). However such standards do not deal well with hot spots, nor with potential nor actual health effects from chronic exposure.

7. Absorption Mechanisms:

Many epidemiological studies show statistically significant associations between EMR exposure and adverse health effects such as cancer, genetic and reproductive problems; animal experiments show increases in cancer, immune system impairment and reproductive problems; and in vivo cell experiments show many biological and biochemical changes with exposure to ELF and RF/MW at nonthermal levels of exposure. While these are sufficient to show highly probable effects, a large body of opinion which dominates the international and national standards setting bodies, holds that the above set of research is fundamentally suspect because there is no physically plausible mechanism through which ELF fields or RF/MW radiation can be absorbed in and alter the physical electromagnetic environment of molecules and cells.

While this physics dominated view is no real reason to ignore the implications and findings of biochemical, toxicological and epidemiological research. For example, the highly repeated result that ELF and ELF modulated RF/MW radiation causes and efflux
of calcium ions through the cell membrane. Many studies show broken DNA and damaged chromosomes with EMR exposure, providing evidence of genetic damage and cancer potential. We cannot ignore these simply because we cannot at this time identify and describe every step in the process. However, it would be scientifically significant and very satisfying to those which wish to complete the picture of the ways in which EMR interacts with biological systems, if satisfactory and identifiable physical mechanisms could be found.

7.1 Whole Body Absorption:

Just as a radio or television can detect and decode modulated RF/MW signals at RMS intensities at a minute fraction of the static electric and magnetic fields, using frequency banded tuned oscillators, so can biological organisms absorb and resonate in modulated RF/MW fields. Human and animals bodies act as antennae for whole body energy absorption and tissue cells act as resonant absorbers. The whole body absorption determines the energy absorbed, the fields induced and the currents which flow through the body. Many fundamental cellular processes involve electric fields and the flow of ions. Hence induced changes from imposed EM fields can and do alter the cellular processes and their ionic balance.

Molecular and cell level electric charges, and electric and magnetic fields play a fundamental role in these processes and in the organization of the complex cellular and macrocellular structures. With these understandings has come new insights. Gone from the outset are notions of isotropy. We see schemes of biochemical and biophysical organization of unparalleled complexity. Concepts of linear systems disappear and are replaced by non-linear, non-equilibrium thermodynamics. Observations and models are consistent with quantum processes involving long-range interactions between electrical charges on cell surface macromolecules, Adey (1992c).

It is at the level of the cell that non-thermal effects become very evident. It has been asserted by some that thermal noise, expressed in the term kT as a function of the Boltzmann constant and the absolute temperature, must remain a monolithic threshold below which no biological threshold can exist. Some biophysicists and others still hold this view despite the wealth of physiological evidence that sensory thresholds descend substantially below the floor of thermal noise, as happens for example in the auditory system of the ear, Adey (1993). Also, attention is now directed to newly defined roles for free radicals, that may also participate in highly cooperative detection of weak electromagnetic fields, “even at levels below the thermal (kT) noise, McLaughlan (1992), and Grundler et al. (1992).

7.2 Radical Pair Mechanism

Many researchers have suggested that thermodynamical considerations do not necessarily impose a final limit with respect to the primary EMR interaction step, Walleczek (1994). Nonlinear biological processes provide a number of possibilities, one of which is the Radical Pair Mechanism (RPM). During such processes the magnetic field does not change the nature of biochemical reaction, only the product yield.

Free radicals are chemical species possessing one or more unpaired electron, which makes them generally very reactive. They are produced continuously in cells either as
the accidental by-products of metabolism or deliberately during, for example, phagocytosis. The most important reactants in free radical biochemistry in aerobic cells are oxygen and its radical derivatives, superoxide and hydroxyl radical, hydrogen peroxide and transition metals.

The reactivity of free radical molecules is determined by the overall spin state of their outer shell electrons. For this reason, their chemical reactivity is spin-selective. Spinning electrons are associated with a magnetic component. The key to understanding the RPM is the fact that applied static or time varying magnetic fields can modify the electron spin states during free radical formation steps, and consequently, alter radical-dependent biochemical reaction rates. Numerous radical-dependent reactions are known to occur in cells, including reactions which may affect T-cell signal transduction steps.

7.3 Magnetic Resonance Mechanisms:

The search for mechanisms by which ELF signals could create biological resonances was first directed towards possible joint actions of static fields and oscillating ELF magnetic fields by Liboff et al. (1985) and Blackman et al. (1985). Blackman proposed a nuclear magnetic resonance model based on the oscillating field being perpendicular to the static field. Liboff proposed the cyclotron resonance model for situations where the fields are parallel. Lednev (1991), using a different theoretical approach, characterized field exposure parameters with sufficient precision to test possible cellular interations with specific combinations of oscillating and static magnetic fields. The Lebnev ion parametric resonance model describes protein-bound ions as spatial oscillators with a series of vibrational frequencies that depend on the bond energy, and the charge and mass of the ligand-bound ion. With the formation and breaking of coordination bonds between protein and a chain of ions, the ions oscillate around a mean energy level, due to random thermal motion. The energy level of the bound ions split into two sub-levels in the presence of a static magnetic field, and the splitting of the two levels occurs at a frequency equal to the cyclotron frequency (proposed by Liboff et al.)

Evidence for sensitivity of biological systems under cyclotron conditions has been extensive but so far inconclusive, with inconsistencies that suggest action of uncontrolled intercurrent factors, Liburdy (1995).
7.4 Cell-based resonant absorption of RF/MW:

Water is known to strongly absorb microwaves. This characteristic is used in microwave cooking. Living biological tissue is water-rich. It is reasonable then to expect that water in tissue will absorb microwave energy.

The surprise is the response to absorbed RF radiation. During the MacIntyre Case, the Court was not told about the research of Liu and Cleary (1995) in which they showed, using classical scattering theory, that both radiofrequency (RF) and microwave (MW) radiation is resonantly absorbed in the bound water layers on the cell membrane. This creates an electric field across the cell membrane in the case of RF and thermal fields in the case of MW, Figure 3.

This research provides vital link between the whole body absorption of RF/MW radiation by human beings and animals and the altered cellular biochemistry demonstrated in isolated cell lines in laboratory experiments. It is another direct rebuttal of the thermal view because the effect varies strongly with frequency between electric field and heating.

Liu and Cleary (1995) have shown, in a biophysical model of cells, that the bound water layer on each side of the cell membrane, resonantly absorbs radiofrequency and microwave radiation at the surface of the cell membrane.

![Figure 3: Spatial distribution of the maximum induced E-field component (E_x) and SAR (SAR_x) on the x-axis of a five component mammalian cell model (extracellular medium, bound-water layer on exterior cell membrane surface, cell membrane, bound-water layer on inside surface of cell membrane surface, cytoplasm) exposed to 27 or 2450 MHz continuous plane-wave electromagnetic radiation. Incident field strength, 1 V/m. (0.3μW/cm²)](image)

There is a vast difference between the effect of an RF (27 MHz) signal and a microwave (2.45 GHz) signal. For the 27 MHz signal the cell has a massive change in the electric
field in the membrane and very little heat absorption. For the 2.45 GHz microwave signal there is a very small change in the electric field from the outside to the inside of the cell, but there is a massive, resonant absorption of heat energy on the inside and outside surface of the cell due to the presence of the bound water layers. I have asked Professors Liu and Cleary to re-run the model for 915 MHz, a common mobile phone frequency. The biophysics suggests that this would show a lower heating response compared to 2450 MHz and a lower electric field difference than the 27 MHz signal.

This very significant paper shows that classical physics predicts resonance absorption of RF/MW radiation at the cell level. Hence there are energy, thermal and electric gradients which can alter the biochemical and chemical reactions at the cellular level. Professor Cleary pointed out to me that this paper also shows a difference in the resonant absorption on the various axes of the cell, depending on the polarization of the incident EM radiation. In a spherical model these differences are as great as a factor of 13 above average. That means, he explained, a mean SAR of 0.007 W/kg could well have localized cellular based SARs or 0.10 W/kg. He was worried about the effects of cell phones on brain cells in the head near the antenna, in the light of this and his other work on cell-cycle timing changes and DNA changes in Chinese Hamster Ovary (CHO) cells, for example.

The absorption of the RF/MW energy at the cell membrane gives a basis to move on to the biochemistry of altered cellular processes, including the effect on signal transduction, cell cycle timing, on ODC in tumour development, on calcium ions and the immune system, and on melatonin in relation to free radical control and elimination. These in turn relate to adverse health effects such as cancer and immuno-competence, sleep disturbance, memory dysfunction and concentration disruption.

8. The Melatonin Mechanism:

On the macro-scale, human and animal circadian rhythms are driven by the day/night cycle with a phase-lock synchronization provided by environmental ELF fields (E<0.3 pW/cm²). A fundamental physiological aspect of the circadian rhythm involves the pineal gland and the secretion of a neurohormone called melatonin. Light falling on the eye’s retina produces signals which are biochemically amplified around a million times, to stimulate the pineal gland to reduce its melatonin output.

8.1 Pineal Melatonin - A plausible mechanism for EMR effects

Pineal and serum melatonin concentration drops during the day and rises overnight, Figure 4.

Melatonin production is very well understood. A schematic of the way in which the light induced signal passes from the retina of the eye, being amplified to release many molecules of the neurotransmitter, norepinephrine (NE), which is received by receptors on the surface of the pineal gland cell (Pinealocyte).
Figure 4: Blood melatonin levels for 4 adult males over a 24 h period, Reiter (1994).

Tryptophan is converted to serotonin which is then converted to Melatonin at a rate controlled by an enzyme NAT (N-acetyltransferase) which has been activated or limited through protein synthesis from amino acids controlled by cyclic AMP, Figure 5.

Figure 5: The connection of the eyes (retina) to the pineal gland, represented by a single pinealocyte, and the synthesis of melatonin within the gland.
Tryptophan, an amino acid from the blood, is converted to the hormone melatonin, which is quickly released into the capillaries of the gland. The enzymes which catalyze the conversion of serotonin to melatonin include N-acetyltransferase (NAT) and hydroxyindole-O-methyltransferase (HIOMT). The pineal gland produces melatonin at night since the nerve endings which end in the pineal gland release the neurotransmitter norepinephrine (NE) which interacts with the b- and a-adrenergic receptors on the cell membrane; these interactions initiate the processes which control melatonin production. ATP, adenosine triphosphate; PVN paraventricular nuclei; SCN, suprachiasmatic nuclei; SCG, superior cervical ganglia. The melatonin easily passes through the cell wall into the blood stream to be dispersed throughout the body. The pineal gland is located near the centre of the brain. It is an endocrine organ which produces most of the melatonin which is found in the blood, figure 6. 

Once melatonin is produced it is the molecule’s high ability to pass through the cell membrane which allows it to escape from the pinealocyte to the blood. Once in the blood melatonin has access to every cell in the body. It passes through the cell membranes where every nucleus has receptors for it. A few cell membranes have receptors. These may mediate the 24 h circadian rhythms of the endocrine system. In the nucleus melatonin plays a role in regulating the effects of the indole on gene expression. The ability of melatonin to enter all cells is also essential for one of the other important functions of melatonin, namely, its ability to scavenge the highly toxic hydroxyl radical (•OH).

The production of oxygen-based free radicals, such as •OH is a consequence of the utilization of oxygen by organisms. About 1-2 % of inspired oxygen ends up as toxic free radicals. It is generally considered the •OH, because of its high reactivity, is the most devastating to macromolecules such as DNA, proteins and lipids. The cellular damage produced by free radicals is generally referred to as oxidative stress, Reiter (1994).
Because of its action in removing free radicals, melatonin is probably the most efficient natural cell protection and oncostatic agent in our bodies. Every night, our pineal produces large quantities of melatonin which flood almost every cell in our body, cleaning out the free radicals and assisting cell division to take place with undamaged DNA. As we age our nocturnal peak melatonin production falls markedly, making elderly people much more prone to cancer. To test the cancer protecting properties of melatonin, Tan et al. (1993), injected rats with a chemical carcinogen, safrole. Safrole normally damages DNA because it induces the production of large numbers of free radicals. Rats injected with Safrole were found to have extensive DNA damage after 24 h. When melatonin was also injected, the DNA damage was reduced by 99%. Since damaged DNA can undergo mutation it may result in the growth of a tumour. Thus melatonin is clearly a potent cellular protector against cancer initiation.

Three independent laboratories, Battelle PNL (Wilson), U.C. Riverside (Luben) and the U.S. EPA (Blackman), have shown that 60 Hz modulated magnetic fields in the 1 to 12 mG range, almost completely negate the oncostatic effect of melatonin in human breast cancer cells, with a dose-response relationship. Wilson et al. (1986) showed significant reductions in pineal melatonin in living rats when they were chronically exposed to 60 Hz modulated electric field at 1.7-1.9 kV/m for 20 h per day, for 30 days, Figure 7.

![Graph](image)

Figure 7: Pineal melatonin (top) and NAT activity (bottom) in groups of rats exposed to a modulated electric field for 1 to 4 weeks. The glands of the animals were collected at night. In the sham-exposed animals the pineal melatonin and NAT levels were always high. However, after both 3 and 4 weeks of exposure to the electric field, both parameters were depressed (p<0.001).
The review paper by Professor Russell Reiter (Reiter (1994), was prompted by a number of epidemiological studies in which an increased incidence of cancer was reported in individuals living or working in an environment of higher than normal artificial electromagnetic fields. Because of the key role of melatonin is decreasing the likelihood of cancer because of its effect of removing free radicals, Prof. Reiter has been researching the effects of EM fields on melatonin production. He concludes:

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

He also notes:

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

Lerchl et al. (1988) samples for serotonin and its derivatives by periodically inverting the magnetic field at night. Figure 8.

Figure 8: Pineal serotonin (5-HT) and 5-hydroxyindole acetic acid (5-HIAA) levels in rats and mice (cross-hatched bars) with and without (clear bars) exposure to pulsed static MF at night. Both 5-HT and 5-HIAA levels increased as a result of the exposure; these changes are consistent with a reduced melatonin production. * p<0.05 and *** p<0.001 vs control; +++ p<0.05 control male mice, from Lerchl et al. (1988).
Hence there exists a plausible mechanism for cancer and a host of disorders, most of which will be identified below as discovered in epidemiological studies. Often the papers or reports which identify statistically significant increases in cancer or other complaints associated with above average exposure to EM fields, have rather weak conclusion, citing the lack of a plausible mechanism. In fact their conclusions can be much stronger because of the existence of the melatonin mechanism and several others which will be described.

Dr Reiter’s review paper, quoted above, demonstrates the fundamental role of cells and the vast amount of cellular biochemistry which is known. It also documents biological mechanisms which are chemical and biochemical and are definitely not thermal.

8.2 Hypothesis for modulated RF/MW effects on melatonin:

Since it has been shown:

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

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

8.3 Direct and Indirect evidence of EMR effects on melatonin:
Melatonin is associated with sleep quality, and sleep quality can be assessed by EEG measurements and poor sleep quality can be reflected in loss of energy, chronic fatigue symptoms, poor concentration and impaired learning and memory. Hence research association these symptoms with EMR exposure are relevant to identifying the potential or actual effects of EMR in reducing melatonin. Reduced nocturnal melatonin leads to reduced nocturnal serotonin because of the large nighttime rise in melatonin output from the pineal gland through conversion of serotonin. However the daytime levels of melatonin and serotonin are not so strongly related.

8.3.1 Biological Sensors of Environmental Fields:

Biological systems are sensitive to external EM fields for many functions. There is unequivocal experimental evidence that fields from ELF to UHF (10 Hz to 450 MHz) interact directly with brain tissue, Adey (1981). Dr Adey cites bird navigation, bird circadian rhythms, monkeys’ subjective time estimations and human circadian rhythms which are all related to tissue level gradients of about $10^{-7}$ V/cm. Weaver and Astumian (1990) use a mathematical model to calculate that membrane macromolecules can directly respond to $10^{-3}$ V/cm while much smaller fields, about $10^{-7}$ V/cm, can be detected if signal averaging occurs through field-induced variation in the catalytic activity of membrane associated enzymes.

The fact that intrinsic cell neuroelectric gradients are far higher than these observed tissue gradients, e.g. Membrane Potential $10^5$ V/cm, Synaptic Potential $10^3$ V/cm and Electro-encephalogram 0.02 to 0.05 V/cm, attests to the vital role of modulation of EMR and the existence of amplification processes at the cellular level, Adey (1989).

8.3.2 Far greater tissue penetration of ELF modulated RF/MW than ELF alone:

Induction of electric fields in tissue at the cellular level varies with the intensity and the nature of the environmental field. Typical endogenous EM fields, with ELF modulation, induce fields in the order of $10^{-1}$ to $10^{-7}$ V/cm in the pericellular fluid (fluid surrounding the cell). RF/MW fields penetrate the organ or body much more effectively than the ELF fields.

For example, when chick brains were exposed to an applied 56 V/m field:

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

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

Thus the RF/MW field produced a cellular tissue gradient 1 million times higher than the ELF field of the same external field strength. This shows the highly penetrative nature of RF/MW fields compared to ELF fields. Since the energy flux relates to the square of the electric field gradient strength, Eq. 1, the energy imparted to the cell tissue by RF/MW modulated radiation is many orders of magnitude higher than the same external strength of ELF field.
8.3.3 The Max Planck Institute Identifies EMR Circadian Rhythm Effects:

Human biometeorology has a great deal to teach us about the effects of natural electromagnetic fields and biological reactions. There is a strong evidence, Wever (1974), that natural ELF signals such as the Schumann Oscillations, coupled with the earth’s magnetic field, help to phase lock the 24 h circadian rhythm in people and animals.

Isolation experiments show that the dark/light cycle is insufficient to fully regulate the circadian rhythm but other environmental stimuli, called “Zeitgebers” by the German researchers, are also required to synchronize the rhythm. These must be globally available, naturally occurring signals since almost all terrestrial life is tuned to the 24 hr cycle The day/night light cycle is the primary driver of circadian rhythm for when people are deprived of this light cycle their daylength drifts, generally becoming longer. An extensive research program has been carried out by the Max Planck Institute over several years and involving over 200 subjects.

Two isolation rooms were used, one of which was also shielded from environmental electromagnetic fields (Room 2). In a simple “free running” experiment, it was found that the mean day period was 24.87 h for Room 1 and 25.26 h for Room 2. The difference between the light isolated (Room 1) and the light and electromagnetic field isolated subjects (Room 2) was significant at the p<0.01 level, Figure 8. Those isolated from the extremely small environmental electromagnetic fields had mean daylengths that were significantly longer and more variable. The standard deviation of their variation in daylength was also significantly larger Room 2 and the number of internal desynchonizations was greater in Room 2 with p<0.001.

Depriving people of access to the natural electromagnetic fields made a very significant difference in their daily rhythm. Other experiments with a very low level artificial ELF signal were carried out. This 2.5 V/m (peak-to-peak) 10 Hz signal (rms-amplitude of 1.77 V/m giving S=0.83μW/cm$^2$) reduced the desynchronization significantly (p<0.001). In many experiments, no case of internal desynchronization occurred as long as the 10 Hz field was in operation, Weber (1974), Figure 9. No effects were found with static electric fields.

Weber (1974) concludes that their research gives:

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

People who were deprived of the light/dark cycle and natural electromagnetic fields with intensities of the order of 0.3 pW/cm$^2$ showed significant shifts in circadian rhythm while an artificial ELF field of 0.8μW/cm$^2$ significantly reduced the desynchronization, mean period and variance of the circadian rhythm.
Figure 9: 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 under the influence of a continuously operating 10 Hz electric field of 2.5 V/m, Wever (1974).

The biological mechanism involved in brain detection of extremely low intensity ELF signals is not discussed by Wever. This substantial project, carried out by a prestigious laboratory, establishes that human beings have the ability to sense and react to extremely small electromagnetic signals. The involvement of the circadian rhythm points to a pineal gland involvement.

8.3.4 Human Research relating to EMR and melatonin reduction:

Clearly mammals can sense and react to extremely low levels of EMR but can and do human beings? The Max Planck Institute research shows a strong and repeatable effect on human circadian rhythm involving ELF signals. This implies a pineal gland reaction and is likely to involve melatonin. As set out in sections 8.3.1 and 8.3.2 above, greater tissue penetration of ELF signals carried by RF/MW radiation, strongly implies that effects on sleep, brain function, immune system, reproduction and cancer should be found in people since the pineal gland and melatonin is involved in each of these. for example:

- Lissoni et al. (1988) report: “It has been known for many years that the pineal gland is involved in regulating tumor growth”. Through the “functional activity of the pineal gland in neoplastic diseases.”

- Bullough et al. (1996) state: “Effects of light and electromagnetic fields (EMFs) on pineal function could have implications for long term risk of breast cancer, reproductive irregularities, or depression.”
Sandyk et al. (1992) write “In the following communication, we propose that the 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.”

Reiter (1994) concludes: “Epidemiologists should look for other possible changes including psychological depression, fatigue, sleep inefficiency, chronic feelings of jet lag, endocrine disturbances and other symptoms; all these may result from chronically low melatonin rhythm.”

Few projects have attempted to directly measure the reduction of melatonin in human beings due to EMR exposure. Some which have, have only assessed daytime alterations. These are prone to great variability and little statistical significance because of individuals’ large variations in melatonin from day to day and the low daytime levels of melatonin in all people. The Schwartenburg Study, Altpeter et al. (1995) for example, sample melatonin in people after they had woken up. The people exhibited a statistically significant dose-response relationship for sleep difficulty, and indication of melatonin reduction, but their early morning melatonin levels showed no statistically significant reductions. This is not surprising because the melatonin levels which related to sleep are those of the evening and nighttime. The exposure involved a shortwave radio transmitter in Switzerland. The primary description of a combination of the symptoms reported was called “Chronic Fatigue Syndrome”. Sleep disruption improved when the transmitter went off unexpectedly and unknowingly, showing a strong connection.

8.3.4 The Schwarzenburg Study

8.3.4.1 Introduction:

Following many years of reported health complains from the vicinity of a shortwave transmission mast, a study by Altpeter et al. (1995) was carried out and revealed statistically significant health and well-being effects which varied systematically with exposure zone. Follow up studies gave strong evidence of the involvement of melatonin as a biological mechanism in relation to sleep disturbance and the reported group of symptoms referred to a “Chronic Fatigue Syndrome”.

8.3.4.2 Exposure levels associated with the radio tower:

Daily average exposures in the frequency range 3 to 30 MHz for each zone are given in Table 14.

The background exposure level in the frequency range 3 to 30 MHz, was measured during a 3-day period when the transmitter was turned off. Levels averaged 0.083 mA/m (0.00183 μW/cm²), with a nocturnal peak of 0.169 mA/m (0.0076 μW/cm²) and an afternoon minimum at about 1pm to 2pm of 0.041 mA/m (0.00045 μW/cm²). This diurnal variation is likely to be related to changes in atmospheric conditions such as those which give better shortwave reception at night than during the day.

Globally this relates to the height of the reflecting electron concentration in the ionosphere, which is higher at night and lower during the day. Locally it relates to atmospheric stratification and ducting of high frequency RF signals, such as radios and radars, as has been observed in Canterbury during nor’westers. This suggests the
probability of nocturnal ducting of the radio signals from the tower, increasing the nocturnal strength in Zones A, B, and C, and hence making them all prone to sleep disturbance effects (if such is the result of RF exposure as is strongly suggested by Table 18).

The exposure range, mean and median exposures measured in each zone during the transmitter’s operation are set out in Table 9.

<table>
<thead>
<tr>
<th>Exposure Zone</th>
<th>Exposure Range</th>
<th>Median</th>
<th>Mean  ((\mu)W/cm(^2))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zone A : High</td>
<td>0.031 - 9.1</td>
<td>0.10</td>
<td>0.24</td>
</tr>
<tr>
<td>Zone B: Medium</td>
<td>0.0034 - 0.074</td>
<td>0.024</td>
<td>0.024</td>
</tr>
<tr>
<td>Zone C: Low</td>
<td>0.00046 - 0.0074</td>
<td>0.0004</td>
<td>0.0004</td>
</tr>
</tbody>
</table>

The median exposure gives the most likely exposure for the population in each zone. For the high exposure (Zone A) this is 0.1 \(\mu\)W/cm\(^2\). The maximum exposure is about 1% of the allowable public exposure level for SW transmissions, and the Zone A median is about 0.01% of the current standard.

Zone C has readings in the range of frequencies produced by the tower from above and below the mean background level. The higher than background levels are likely to be more prevalent at nighttime due to ducting phenomena except during Föhn conditions.

8.3.4.3 Effects associated with the RF Exposure:

The statistically elevated symptoms in the high and medium exposure groups, compared to the low exposure group, include Nervosity and restlessness, Disturbances in falling asleep and difficulty in maintaining sleep, Joint pains, Psychovegetative Index changes, Disturbances of Concentration, General Weakness and Tiredness, Constipation, Diarrhea and Lower back pain, all significant at \(p<0.02\) except the first for which \(p=0.034\) which is less than the usual significance level of \(p<0.05\).

An increased exposure from 1 mA/m to 10 mA/m (0.038\(\mu\)W/cm\(^2\) to 3.8\(\mu\)W/cm\(^2\)) had on Odds Ratio for insomnia of 1.13 (CI: 1.04-1.23) and from 0.1 mA/m to 1 mA/m (0.00038\(\mu\)W/cm\(^2\) to 0.038\(\mu\)W/cm\(^2\)), OR=2.1 (CI: 0.95-4.57). Table 10 presents the adjusted Odds Ratios for the primary effects found, which show significant dose response relationships and a highly statistically significant increase with mean exposure increase.
Table 10: Odds Ratios for an increase in 24-hour average exposure from 1 mA/m (0.04 μW/cm^2) to 10 mA/m (3.8 μW/cm^2) adjusted for age, sex, attribution, and duration of time lived at the same place.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>OR</th>
<th>95% Conf. Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nervosity, Restlessness</td>
<td>2.77</td>
<td>1.62-4.74</td>
</tr>
<tr>
<td>Diff. in falling asleep</td>
<td>3.35</td>
<td>1.86-6.03</td>
</tr>
<tr>
<td>Diff. in maintaining sleep</td>
<td>3.19</td>
<td>1.84-5.52</td>
</tr>
<tr>
<td>Joint Pain</td>
<td>2.46</td>
<td>1.37-4.43</td>
</tr>
<tr>
<td>Limb Pain</td>
<td>2.51</td>
<td>1.15-5.50</td>
</tr>
<tr>
<td>Cough and Sputum</td>
<td>2.80</td>
<td>1.18-6.64</td>
</tr>
</tbody>
</table>

Figure 10: Frequency of different psychovegetative disorders by Zone A, B, and C. The light bars concern persons aged over 45 years, the dark ones aged 45 or less, Altpeter et al. (1995).

It is important to note that the Odds Ratio in the range from 2.46 to 3.35 is much more significant in this study than the incidence of cancers since the occurrence of symptoms in the low group in this study is around 10% compared to about 0.007% in total leukaemia incidence in the Polish Military Study.

Figure 10 and Table 11 show that several complaints, especially for the over 45 age-group, have statistically significant dose-response curves which Bradford-Hill viewpoint (5) Biological Gradient ascribes a strong weight of evidence. When linked to the melatonin mechanism (6) Biological Plausibility this is strong evidence of a probable cause-and-effect. Symptoms which reach this standard (p<0.05) include Nervocity/restlesness and genral weakness and tiredness (all ages), and difficulties in falling asleep and maintaining sleep, joint pain, psychovegetative index, constipation and diarrhea (>45).
Table 11: Complaints by zone and age-groups. P-value calculated for the dose-response curve using a $X^2$ within each stratum.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Zone A (%)</th>
<th>Zone B (%)</th>
<th>Zone C(%)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nervosity, restlessness (&lt;45)</td>
<td>12/55 (21.6)</td>
<td>15/61 (24.6)</td>
<td>8/85 (9.4)</td>
<td>0.034</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>14/50 (28.0)</td>
<td>7/58 (12.1)</td>
<td>4/94 (4.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Difficulties in maintaining sleep (&lt;45)</td>
<td>7/55 (12.7)</td>
<td>4/61 (6.6)</td>
<td>4/84 (4.7)</td>
<td>0.194</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>14/50 (28.0)</td>
<td>5/58 (8.5)</td>
<td>5/94 (5.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Limb pain (&lt;45)</td>
<td>1/55 (1.8)</td>
<td>3/61 (4.9)</td>
<td>1/85 (1.2)</td>
<td>0.329</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>14/50 (28.0)</td>
<td>5/58 (8.5)</td>
<td>5/94 (5.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Difficulties in falling asleep (&lt;45)</td>
<td>8/55 (15.4)</td>
<td>6/58 (10.3)</td>
<td>3/86 (3.5)</td>
<td>0.062</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>16/50 (32.0)</td>
<td>15/58 (25.9)</td>
<td>9/94 (9.6)</td>
<td>0.002</td>
</tr>
<tr>
<td>Joint pain (&lt;45)</td>
<td>5/55 (9.1)</td>
<td>2/61 (3.3)</td>
<td>4/86 (6.7)</td>
<td>0.353</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>19/50 (38.0)</td>
<td>10/58 (17.2)</td>
<td>14/94 (14.9)</td>
<td>0.003</td>
</tr>
<tr>
<td>Psychovegetative index (&lt;45)</td>
<td>1/54 (1.9)</td>
<td>1/60 (1.7)</td>
<td>1/84 (1.2)</td>
<td>0.947</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>12/50 (24.0)</td>
<td>5/55 (9.1)</td>
<td>5/90 (5.6)</td>
<td>0.004</td>
</tr>
<tr>
<td>Disturbances of concentration (&lt;45)</td>
<td>1/55 (1.8)</td>
<td>2/61 (3.3)</td>
<td>2/86 (2.3)</td>
<td>0.874</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>7/50 (14.0)</td>
<td>1/57 (1.8)</td>
<td>3/94 (3.2)</td>
<td>0.009</td>
</tr>
<tr>
<td>General weakness, tiredness (&lt;45)</td>
<td>10/54 (18.5)</td>
<td>9/61 (14.8)</td>
<td>3/86 (3.5)</td>
<td>0.011</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>13/50 (26.0)</td>
<td>7/58 (12.1)</td>
<td>8/94 (8.5)</td>
<td>0.014</td>
</tr>
<tr>
<td>Constipation (&lt;45)</td>
<td>2/55 (3.6)</td>
<td>5/61 (8.2)</td>
<td>2/86 (2.3)</td>
<td>0.222</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>6/50 (12.0)</td>
<td>3/58 (5.2)</td>
<td>1/94 (1.1)</td>
<td>0.016</td>
</tr>
<tr>
<td>Diarrhoo (&lt;45)</td>
<td>1/55 (1.8)</td>
<td>1/61 (1.6)</td>
<td>2/86 (2.3)</td>
<td>0.952</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>6/50 (12.0)</td>
<td>3/58 (5.2)</td>
<td>1/94 (1.1)</td>
<td>0.016</td>
</tr>
<tr>
<td>Stomach trouble (&lt;45)</td>
<td>3/55 (5.5)</td>
<td>2/61 (3.3)</td>
<td>4/86 (4.7)</td>
<td>0.845</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>7/50 (14.0)</td>
<td>5/58 (8.6)</td>
<td>3/94 (3.2)</td>
<td>0.57</td>
</tr>
<tr>
<td>Lower back pain (&lt;45)</td>
<td>5/55 (9.1)</td>
<td>9/61 (14.8)</td>
<td>11/86 (12.8)</td>
<td>0.644</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>15/50 (30.0)</td>
<td>12/58 (20.7)</td>
<td>14/94 (14.9)</td>
<td>0.100</td>
</tr>
<tr>
<td>Feelings of excitement on body (&lt;45)</td>
<td>3/55 (5.5)</td>
<td>3/61 (4.9)</td>
<td>0/85 (0.0)</td>
<td>0.034</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>5/50 (10.0)</td>
<td>4/58 (6.9)</td>
<td>2/94 (2.1)</td>
<td>0.102</td>
</tr>
<tr>
<td>Irregular heartbeat (&lt;45)</td>
<td>2/22 (3.7)</td>
<td>0/61 (0.0)</td>
<td>0/86 (0.0)</td>
<td>0.067</td>
</tr>
<tr>
<td>... (&gt;45)</td>
<td>0/50 (0.0)</td>
<td>0/58 (0.0)</td>
<td>0/93 (0.0)</td>
<td>0.120</td>
</tr>
</tbody>
</table>

The insomnia is related to a disturbance of nocturnal melatonin and leads to a general debilitation and lack of mental alertness. This is therefore a very important symptom which warranted extra study. Follow up studies of people’s melatonin detected no significant change. However readings were taken after people awoke in the morning and so they did not and would peak which is reached about 2 hours after falling asleep. This problem, and several others, is acknowledged by the authors.

8.3.4.4 Unexpected and unaware transmitter breakdown:

A fortuitous event is very revealing. For three days during the study, the transmitter broke down and emissions ceased, and the people did not know about it. During these three days reported sleep quality was markedly improved from the first night in the high exposure group and from the second night in the middle and low exposure group. Averaging over 3 day intervals for each zone, for before, during and after, starting on day 2 gives the following percentage awakenings, Table 12.

“Before” shows the level of awakenings increasing with exposure, “After” shows a rate of recovery which is slow and smallest in the high exposure group and is quick and highest in the Low exposure group.
Table 12: Sleep disturbance rates (%) for 3-day periods before, during and after the transmitter went off, the brackets show the % difference from the previous 3-day period.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Before</th>
<th>During</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zone A (High)</td>
<td>70</td>
<td>64 (-6)</td>
<td>61 (-3)</td>
</tr>
<tr>
<td>Zone B (Middle)</td>
<td>68</td>
<td>52 (-16)</td>
<td>63 (+11)</td>
</tr>
<tr>
<td>Zone C (Low)</td>
<td>61</td>
<td>37 (-24)</td>
<td>54 (+17)</td>
</tr>
</tbody>
</table>

“During” shows a significant reduction in awakenings in Zones B and C, which is greatest and most significant in the Low exposure group, and they show the fastest recovery. It appears that the High exposure group takes many days to react to the shut down. This is fully consistent with the study on ELF exposed rats by Wilson et al. (1986) whose melatonin dropped slowing over a period of weeks.

Hence the High exposure group, which is shown to be most strongly affected by sleep difficulties, is very slow in their reaction time, which might limit the level of relief shown by a three day shut down. The Low exposure group gets the greatest relief and the fastest reactions. This strongly suggests that even in Zone C the effects of the transmissions on sleep quality is significant. Hence the Odds Ratios of “exposed” to “unexposed” are significantly under estimated since the group which is assumed to be unexposed clearly is reacting to the exposure they are getting, even though it is in the range 0.00046-0.0074 μW/cm².

8.3.4.5 Nocturnal Melatonin measured in cows:

Mean measurements of bovine salival melatonin showed that exposed cows had lower mean melatonin, 17.7 pg/ml compared to 19.0 pg/ml, but this was not statistically significant. It must again be pointed out that it is not the mean melatonin levels which relates to sleep but it is the nocturnal melatonin peak.

The Bovine melatonin study shows that when the transmission was off the cows in the exposed group had strong nocturnal peaks not seen as that pronounced and high at any time when the transmission was on. Since it is the nocturnal peak and not the daily mean nor daytime levels of melatonin which are implicated in sleep and other probable health effects, this data is important ion showing that the nocturnal peak in the exposed group is constantly higher than the unexposed group, while the transmitter is off but it is frequently, and on average, lower when the transmitter is on.

Stark et al. (1997) report further analysis of this bovine data stating that while there was a statistically significant 21-fold higher exposure on the exposed farm compared to the control farm, the lack of statistical significant of the reduce levels of salivary melatonin between the groups suggested that a chronic melatonin effect was unlikely. Figure 11 illustrates one of the fundamentals of melatonin production and that is great variability. The short observational period is also significant in the result. If the reduction between the exposed group compared to the unexposed group had been evident in observations continued for another 10 days then the result is likely to have reached statistical significance.
The authors actual did find a significant observation: “However, on the first night of re-exposure after the transmitter had been off for 3 days, the difference in salivary melatonin concentration between the two farms (3.89 pg/ml, CI: 2.04-7.41) was statistically significant, indicating a two- to seven-fold increase in melatonin concentration. This a delayed acute effect effect of EMF on melatonin cannot be completely excluded.”

Figure 11: Melatonin concentration measured in the saliva of EMF-exposed (continuous line) and unexposed (dashed line) cows: acute effect of EMF exposure. The lines at 100 pg/ml indicate the dark phases when melatonin concentration was measured, black box is the period of no EMF exposure of both groups. (Altpeter et al. (1995))

The rate of response and the possibility of a degree of adaption should be considered when interpreting this data. The human sleep alteration data indicate this, with the lowest and lowest response in the highly exposed group and the greatest and quickest response from the lowest exposure group, Table 12.

These combined observations point strongly to the effect of the RF EMR in decreasing the nocturnal peak of melatonin with an accompanying significant degradation in the quality and maintenance of sleep.

There appears to be little reason to assume that human nocturnal peak melatonin levels were not affected in the same way that the bovine melatonin levels were. Sleep disturbance was a highly reported effect, nocturnal peak melatonin relates to sleep quality, bovine exposed melatonin levels were reduced, bovine nocturnal peak melatonin levels improved when the transmitter was off, sleep improved when the transmitter was off. Hence human nocturnal melatonin reduction this is a reasonable and likely
mechanism to explain the reported sleep disturbance and the Chronic Fatigue Syndrome condition, as projected by Reiter (1994), Section 4.1.

Reduced nocturnal melatonin also has significant health implications relating to cancers, such as breast cancer, which has been related to melatonin reduction in laboratory studies, Liburdy et al. (1993) and related to ELF exposure in epidemiological studies, Demers et al (1991) and Section 3.1. This study implicates RF exposure.

8.3.4.6 Heart and Blood Pressure:

Concerns about heart problems were investigated with a blood pressure and hypertension survey. Normal blood pressure was reported in 55 % in Zone A, 56 % in Zone B and 74 % in Zone C. Arterial hypertension was reported in 14 % in Zone A, 8.4 % in Zone B and 7.9 % in Zone C. An extensive review of reporting and measurements revealed that there was an Odds ratio of 1.4 (CI: 0.75-2.52) for blood pressure difference in Zone A compared with Zones B and C combined. Hence blood pressure was slightly elevated with exposure to EMR, but not significantly so.

8.3.4.7 Effect of school children:

Sleep and concentration disruption also relates to the performance of school children. Children from a school in Zone A were compared with children from a school which was unexposed, in terms of rate of promotion from primary to secondary school. They found a significantly slower promotion rate in the exposed school. They conclude that even though the association is weakened by a small sample size, “an adverse effect (from the transmitter) cannot be excluded.” (p130)

The children in Zone A are in the range of exposures (0.031-9.06μW/cm^2) which Von Klitzing (1995) shows that significant changes in human EEG occur in 70 % of subjects (0.7μW/cm^2). Reduced nocturnal melatonin and sleep disturbance would also be contributory factors. Mann and Roschke (1996) found that sleeping near to a cell phone caused an EEG monitored adverse change in REM sleep, accompanied by learning and memory problems. Hence a slowing of promotion was found in the Swiss study and scientifically identified mechanisms also exist to reinforce this observations link to EMR. Learning difficulties associated with RF/MW exposure was also found by Latvian researchers, Kolodynski and Kolodynska (1996), and Chinese researchers, Chiang et al. (1989).

Hence very strong evidence exists, with identified mechanisms involving nocturnal melatonin reduction, for RF/MW radiation significantly reducing the intellectual and memory functions of children and adults, at exposure levels which correspond to those found within many hundreds of metres of cell sites.

9. Alterations in brain chemistry, CNS and EEG:

The brain and its accompanying organs such as the pineal body, the pituitary gland and the hypothalamus play fundamental roles in human mood, sleep, immune system, health and well-being. Considerable data exists to suggest that EMR acts in a similar way to sunlight in altering the hormone relationships between the pineal and the pituitary. Visible light exposure modulates the pituitary and pineal gland which leads to neuroendocrine changes, Roberts (1995). Melatonin, norepinephrine and acetylcholine decrease with
light activation while cortisol, serotonin, GABA and dopamine increase. All of these neuroendocrine changes can lead to alterations in mood, circadian rhythm and immune modulation, Roberts (1995).

9.1 Calcium ion balance in brain cells:

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

- Weak extracellular voltage gradients (1-5 mV/mm) have been shown to significantly affect the excitability or firing thresholds of the spinal neurons of cats.

- Nelson (1966) pointed out that the complex structural organization of brain tissues, as seen in the cerebrum, should be highly favourable for multiple electric field interactions, both in the intricate rate of overlapping dendritic trees and between macromolecules on the extracellular space and the glycoproteins of the out cell membrane.

- Weak pulsed electric currents (2-5 mV/mm, 200 pulses/sec) applied across the cat cortex were able to trigger the release of previously bound radioactive calcium ($^{45}$Ca$^{2+}$).

- Intercranial injection of Ca2+ or Mg2+ in chronically implanted neonatal chicks resulted in an almost immediate synchronization of the hyperstriatal EEG, accompanied by behavioural depression, Bawin et al. (1984). During successive testing days, the animals appeared to recover behaviourally but never showed any sustained EEG arousal. By contrast animals treated with sodium chloride recovered completely within the first hour after injection.

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

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

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

Wever (1974), section 8 above, showed changes in human subjects isolated from environmental stimuli including ELF fields, which resulted in altered circadian rhythms.
which were corrected by applying a 10 Hz, 2.5 V/m field, which produces about \(10^{-7}\) V/cm in tissue. The experiment was repeated using birds, with similar results, of lengthened circadian rhythms.

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

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

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

An efflux has been recorded for the amino acid neurotransmitter gamma-aminobutyric acid (GABA), Kolomytkin et al. (1994), in association with microwaves modulated at 16 Hz. This is very significant since GABA and glutamateric synapses make up about 60% of the CNS and calcium ions appear to hold the key to every aspect of cell-surface transduction, Adey (1979). Kolomytkin et al. (1994) showed that at 915 MHz microwave signal, modulated at 16 Hz, altered the binding of 3H-glutamate and 3H-muscimol in rats brains, at power densities below 50 \(\mu\text{W/cm}^2\), which are statistically significantly different from controls to below 10 \(\mu\text{W/cm}^2\), Figure 12. Kolomytkin et al. (1994) link these changes to \(\text{Ca}^{2+}\) ions which have been shown to stimulate specific glutamate binding to synaptic membranes due to the activation of a calcium-dependent protease and resulting proteolysis (splitting into fragments) of cytoskeletal proteins.

Since it is shown that modulated microwaves increase the glutamate uptake by synaptomes, Kolomytkin et al. pose the question as to whether microwaves directly affect the synaptosomes or does their sensitivity require some other brain system? They determined that it was the synaptosomes which were sensitive to the microwaves. They then investigated whether it was a simple heating effect. Heating the samples to produce the same mean SAR did not produce the result. Hence they proposed the mechanism of localized microheating at the cell membrane. This membrane heating in the presence of microwaves has been demonstrated now by Liu and Cleary (1996).
Figure 12: Altered the binding of 3H-glutamate and 3H-muscimol in rats brains versus microwave power density (915 MHz, modulated at 16 Hz), Kolomytkin et al. (1994).

Kolomytkin et al. (1994) conclude that:

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

Dumanskiy and Shandala (1974) and their colleagues reported altered conditioned reflex in rabbits and rats chronically exposed to extremely low levels of VHF and microwave fields. They used either 50 MHz or 2.5 GHz CW fields or 10 GHz 1 μs pulses at 1,000 or 20 Hz, with 10-12h daily exposure with 50 MHz and 8 h with microwave fields. They found statistically significant effects with field intensities between 1.9 and 2.0 μW/cm².

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

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

Shandala et al. (1979) found statistically significant changes in the EEG and brain biochemistry of rats and rabbits exposed to 2.375 GHz microwaves at 10, 50 and 500 μW/cm², for 7 hours/day over 30 days. The 10 μW/cm² and 50 μW/cm² initially stimulate brain activity, while 500 μW/cm² causes suppression as seen from the increase in slow, high amplitude Δ-waves. After 1 month of exposure to a power density of 10 μW/cm² (for 7 hr/day, i.e. averaging 2.9 μW/cm²) a reliable (p<0.05) increase was observed in the alpha-rhythm in the sensory-motor and visual cortex due to a suppression of the slow EEG components.

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

Vorobyov et al. (1997) studied short-term alterations of EEG in mice exposed to ELF fields carried on a 945 Mhz microwave carrier with exposures in the range 100 to 200 μW/cm². They found an induced asymmetry in the EEG on each side of the brain of an ongoing EEG power decrease in the 1.5-3 Hz range in the left hemisphere and a power increase in the 10-14 Hz and 20-30 Hz ranges in the right hemisphere. Significant elevations of EEG asymmetry in the 10-14 Hz range were observed after the first 20 s after five onsets of the microwave field, when averaged spectra were obtained for every 10 s. In their conclusions they comment that:

"One of the possible key links in this effect can be calcium ion exchange in brain tissue (Adey (1981)). Indeed it was found that the intracellular calmodulin level was changed by modualted microwave fields, Katkov et al. (1992). This change, as is known, can cause the change in receptor sensitivity to mediators, because in neural tissues both the transmitter-receptor mechanism and the second messenger are Ca²⁺ dependent."

9.2 EMR induced EEG changes in humans:

Are these effects found in humans?

Two papers known to the author show EMR alteration of the human EEG. The first, von Klitzing (1995) shows dominantly EEG delta to alpha rhythm change when exposed to GSM signal. The second shows sleep and EEG change with GSM phone exposure.
9.2.1 Human EEG delta to alpha when GSM exposed:

Von Klitzing (1995) shows the same result, alpha enhancement and slow wave suppression in human subjects exposed to a GSM cell-phone like signal with an SAR of 0.001 W/kg (S = 0.7 μW/cm²), (from Eq. 8 using σ=0.77 S/m) and a pulse frequency of 217 Hz. The power spectrum of one of the subjects is shown in Figure 13. Von Klitzing's paper presents an example of the 45 experiments from 17 students tested. Around 70% of the students showed significant alteration in their EEG at these very low exposure levels.

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

9.2.2 Cell phone signal alters sleep EEG:

Healthy people sleeping with a digital GSM cell phone on next to the bed, exposing their heads to about 50 μW/cm² while their brain EEG was being monitored, Mann and Roschke (1996). This revealed a statistically significant disruption of alpha EEG frequency range and REM sleep. REM sleep decreased from 17.07 % to 13.91 %, which is significant at p<0.05. In addition subjects went to sleep faster, a hypnotic effect also reported by Reite et al. (1994) who used a signal of 27.12 MHz modulated at 42.7 Hz.

Mann and Roschke (1996) exposed 14 healthy, non-smoking, non-drinking, 21-34 year old male volunteers to 900 MHz, pulsed at 217 Hz with a pulse width of 580 μs, digital GSM signal with a resultant average power density at the head of 50 μW/cm². They concluded that:

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

The results are summarized in Figure 14.
Figure 13: Human Alpha-EEG (O2-position) is altered by pulsed electromagnetic fields following first exposure, von Klitzing (1995).
Reite et al. (1994) also found an hypnotic effect when a 27.12 MHz signal, modulated at 42.7 Hz as applied over a 15 min period. Exposed subjects reached a deeper state of sleep than sham exposed subjects.

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

9.3 Studies showing learning difficulties with EMR exposure:

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

Three published papers or reports identify such effects:
Chiang et al. (1989) found that visual reaction time, a measure of the function of the visual receptor and the central nervous system, varied with microwave exposure of children up to 4 $\mu$W/cm$^2$. Children exposed to over 10 $\mu$W/cm$^2$ had lower scores in the memory function test. They concluded “the data indicate that chronic exposure to EMFs are associated with significant changes in some physiological parameters.”

Altpeter et al. (1995) showed a statistically significant delay in promotion from primary to secondary school in the more highly exposed school compared to a low exposure school, OR= 0.63, 95% CI: 0.43-0.85, p<0.005. This involved shortwave radio exposure. The daily mean exposures in the highly exposed group were in the range 0.031 to 9.1 $\mu$W/cm$^2$, median 0.1 $\mu$W/cm$^2$ and mean 0.24 $\mu$W/cm$^2$.

Kolodynski and Kolodynska (1996) investigated the effects of a RLS radar in Latvia, radiating at 154-162 MHz and pulsed at 24.4 Hz, on the performance of school children living several km in front of the radar compared to children living behind the radar. They concluded that “Motor function, memory and attention significantly differed between exposed and control groups. children living in front of the RLS had less developed memory and attention, their reaction time was slower and their neuromuscular apparatus endurance was decreased.” Assuming that the closest child lived 2 km in front of the radar, the maximum mean measured exposure is in the 0.16 $\mu$W/cm$^2$.

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

### 9.4 The relationship between EEG brain states and moods:

Typical electroencephalographs (EEG) are given below, from Dorland 28, p535. As noted above, cells have electric charges on their surfaces. By placing sensor electrodes on the surface of the skull electrical signals are detected. Voltages are detected which have been produces by currents emanating from nerve cells in the brain. The dominant frequency of these signals in about 8 to 10 Hz with an amplitude of 10 to 100 $\mu$V.

EEGs are characterized by frequency bands which are associated with various brain states, Dorland 28:

**Alpha Rhythm:** 8 - 13 Hz

Are typical of the normal person awake and in a quite resting state, and principally in the occipital region. Alpha amplitude increases with joy and anger and decreases with fear and sorrow.

**Beta Rhythm:** 18 - 30 Hz.

Are typical during periods of intense activity of the nervous system, occurring principally in the parietal and frontal regions.

**Delta Rhythm:** < 3.5 Hz.
Typically occurs in deep sleep, in infancy and in serious brain disorders.

**Theta Rhythm: 4 - 7 Hz.**

Occurs mainly in children but also in adults during periods of emotional stress.

![Figure 15: EEG recordings made while the subject was excited, relaxed and in various stages of sleep. During excitement the brain waves are rapid and small amplitude, whereas in sleep they are much slower and of greater amplitude.](image)

Adey (1979) notes that the electric process between dendrites (in the brain) is one of slow waves, not pulses. The integral of the slow wave activity of the dendrites constitutes the electroencephalogram (EEG). When the brain is awake the electro-encephalogram is fast, as are the waves outside the cell.

In classical axodendritic synapse, Figure 16, (a) synaptic vesicles in the axon of one neuron release neurotransmitter toward the receptors on the dendrite of a target neuron. It is also possible for a dendrite to pass a message to another dendrite by way of dendrodendritic synapses. In a reciprocal dendrodendritic synapses (b) each dendrite passes messages to the other by way of a separate synapse. In some synapses, called axoaxonic synapses (c), the axon of one neuron passes a message through the axon of another neuron to the dendrite of a third neuron. In synaptic glomerulus (d) the axon of one neuron passes messages to dendrites of two others; the dendrites may pass messages to each other as well. Snyder (1985) [p138]

The electroencephalogram is produced by the leakage of these big waves from inside the dendrites into the fluid around the cell. The electroencephalogram recorded over the dimensions of the cell is a few microvolts. The neuronal wave inside the cell is of the order of 5 to 15 mV. Thus the difference in amplitudes is about 200 to 1.
Figure 16: Communication between neurons takes place across gaps called synapses.

It is very evident that brain activity changes a great deal with rest and activity, with health and illness, and with stress and emotion. During these wide ranging changes, significant changes in neurotransmitters such as serotonin and adrenaline have also been monitored. These biochemical changes send neurohormones throughout the body to change heart beat, vasodilation etc. They also change the brain cell behaviour in such a way that electrical signals in regions of the brain show altered, coordinated and repeatable changes in oscillating voltages which are indicative of coordinated electrical communication between large groups of brain cells.

A key scientific question is: is the EEG simply a product of the changing electrical environment within the brain, or does it provide the opportunity for external oscillating electrical fields to superimpose changes in the electrical behaviour of the brain which would then produce imposed changes in neurotransmitters and neurohormone production? Is the brain, or parts of the brain, sensors which can pick up external
electrical signals which can change the psychological and/or physiological state of the brain and body?

### 9.5 Conclusions - EEG and EMR:

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

Hence, far from being an isolated example, as this data was considered by the Planning Tribunal, the von Klitzing results for human beings is consistent with research on people and rabbits, Dumanskiy and Shandala (1974) and Shandala et al. (1979); and in cats, Bawin et al. (1973). Studies on altered reaction times and circadian rhythms in humans and animals are linked to EEG changes, Adey (1981).

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

Hence Dr von Klitzing’s results are consistent with animal experiments, have a basis in Neurophysiology and therefore stand as a serious concern about the impact of very low intensity modulated RF/MW signals on the fundamental processing of information by our brains. The intensities of exposure which show effects are all non-thermal and reach levels well below the current public exposure standard (200 $\mu$W/cm$^2$), at 50, about 1 and 0.7 $\mu$W/cm$^2$. With isolation from natural environment’s 0.3 pW/cm$^2$ Schumann Oscillations also having measurable effects of circadian rhythms.

### 10. Cellular Biology:

#### 10.1 Introduction:

The fundamental basis of biologic activity is the cell. Cell biology and biochemistry has advanced our understanding of cell behaviour and cellular processes to highly advanced levels. The structure of cells is well described, the processes which regulate cell growth and development, the genetic basis of cell reproduction and the amino acid typing of complex molecules, including RNA and DNA is being advanced daily. This shows the importance of understanding and appreciating cellular characteristics and processes in order to understand the interactions of EMR with living tissue and potential health hazards from EMR exposure.

In cellular aggregates that form tissues of higher animals, cells are separated by narrow fluid channels that take on a special importance in signaling from cell to cell. These channels act as windows on the electrochemical world surrounding each cell. Hormones, antibodies, neurotransmitters and chemical cancer promoters, for example, move along then to reach binding cells on the cell membrane receptors.
10.2 Biochemistry and cell biology:

During the MacIntyre Case, Associate Professor Richard Luben presented evidence of biochemical mechanisms which are observed to change under exposure to modulate RF fields, Luben (1995). This included changes in calcium ion efflux and Ornithine Decarboxylase (ODC), both of which are involved in the signal transduction aspects of control of the growth and development of cells in the human body and other animals.

Two of Dr Luben’s key statements are worth recalling. He stated that laboratory studies had shown the similarities and parallels in the biological effects of ELF and RF modulated by ELFs. For example, calcium ion efflux and ODC are observed to vary in similar ways in ELF fields and in RF fields modulated at ELF frequencies, Byus (1994), Giuliana, et al. (1996).

Dr Luben also stated that the electromagnetic radiation did not need to enter the cell in order to change its behaviour, it just needs to be absorbed by the cell surface, and then the altered signal transduction process changes the cell behaviour.

A more detailed and updated review of biophysics and biochemistry reveals clear means of EMR altering the biochemical behaviour at the cellular level with a great deal of detailed existing understanding about these processes. These form a set of plausible mechanisms to explain the way in which EMR can change cell behaviour and hence can cause adverse health effects. Where these are matched by appropriate epidemiology an extremely strong association is established. Where there is a pattern of epidemiology which is consistent with animal experiments and for which there are detailed biophysical and biochemical mechanisms, the evidence approaches the level required to establish cause and effect.

The research here reviews our current understanding of cell structure and processes, including resonant absorption of radiofrequency and microwave radiation at the cell membrane, gap junction communication between cells, signal transduction processes from regulating cell growth and behaviour, the vital role of calcium ions, the implications for changes in ODC, the formation and effects of free radicals, the dendritic structure of the brain which relates to neurotransmitters and EEG, and the role of neurotransmitters such as serotonin and adrenaline, and neurohormones such as melatonin.

Evidence will then be presented showing cellular and molecular changes which occur with exposure to electromagnetic radiation which are directly or indirectly related to known biophysical biochemical characteristics of cells. The implications of this for public health will be discussed. The identified biophysical and biochemical mechanisms will then be related to epidemiological research and public health implications will be discussed and exposure standards will be recommended where supportable by sound research.

10.3 Cell Biochemistry and Neurophysiology

The last 10 to 15 years has seen an exciting and challenging revelation of the complex biochemistry at the cellular and molecular level in living systems. As laboratory techniques have advanced we have progressed from the study of organs, to tissues, to cells and to molecules. The behaviour and organisation of cells and molecules in living tissue relies on sequences of reactions which use and transform energy in the continual creation and re-creation of molecular complexes including material to form cell walls (membrane), cell nuclei, inside to outside cell communications, cell to cell
communications, enzymes to stimulate and slow cell division and cell growth, and the RNA and DNA molecules which code the genetic structure of the host of different types of cell, and cell and tissue structures which make up highly organized living systems.

10.4 The Cell:

The cell is an identifiable entity of all living organisms and is recognized as the fundamental unit of biologic activity. Cells consist of a nucleus which is surrounded by cytoplasm, which contains various organelles, and is enclosed in a cell or plasma membrane, Figure 17.

The nucleus contains the hereditary material of DNA and chromosomes, along with the proteins and enzymes which are necessary for the sustenance of the nucleus and the processes of chromosome separation during mitosis.

The cytoplasm is the protoplasm of a cell exclusive of that nucleus. It consists of a continuous aqueous solution (cytosol) and the organelles and inclusions suspended in it. It is the site of most biochemical activities of the cell.

The cell membrane is a bimolecular layer of lipids which encloses the cytoplasm and nucleus. It has permeable to some substances and contains protein structures which pass through the membrane, providing for processes such as signal transduction, see Figure 13 below.

Cells form many shapes and have a host of different functions. Some cells are bound together to form tissue such as in skin and muscles, some are near spherical and float in fluid, such as T-cells, and others are dendritic, with long dendrite structures extending to several times the diameter of the central cell body, such as many brain and central nervous system cells.

10.5 The Cell cycle:

A cell is a cooperative of molecules which is capable of reproducing itself. Cells are discrete entities that grow and divide. Most cells must complete four tasks during the cell cycle. They must grow, replicate their DNA, segregate their chromosomes into two identical sets and divide. To do this a cell needs between 2000 and 5000 different enzymes and structural proteins.
The cell cycle is divided into four discrete phases. Rapidly dividing human cells have a cell cycle which lasts about 24 hours.

Some of the molecules, like ribosomal proteins and RNAs are present in the millions per cell, while DNA are present as only one or two. Cells contain many different types of proteins, each specialized for a particular role in the life of the cell. Important classes include enzymes that produce the building blocks for the synthesis of DNA, RNA and proteins, and the enzymes which build these blocks to replicate DNA, transcribe DNA into RNA, and translate mRNA into protein. The form and function of cells depend on the structural proteins that form the cytoskeleton and on the motor proteins that move objects along elements of the cytoskeleton, such as chromosomes. Mammals are estimated to have as many as 200 different cell types.

The cell cycle is divided into two fundamental parts: interphase, which occupies the majority of the cell cycle, and mitosis, which lasts about 30 minutes, ending with the division of the cell. During interphase DNA is diffusely distributed throughout the nucleus, and individual chromosomes cannot be distinguished. Little activity can be seen in the microscope but two important classes of process are occurring, continuous processes (referred to collectively as ‘growth’) and stepwise processes which occur once per cycle.

For example, chromosome replication is restricted to a specific part of interphase called S phase (for DNA synthesis). S phase occurs in the middle of interphase, preceded by a gap called G1 and followed by a gap called G2, Figures 11a and 11b. After each chromosome has been replicated, the two daughter chromosomes remain attached to each other at multiple points along their length and are referred to as sister chromatids.
In a typical animal cell cycle, G1 lasts 12 hours, S phase 6 hours, G2 6 hours and mitosis (M) about 30 minutes.

![Cell growth and DNA content during the cell cycle.](image1)

### Figure 18: (a) Cell growth and DNA content during the cell cycle. Mass increases continuously throughout the cell cycle while DNA content is constant for most of the cycle, increasing during the S phase as DNA replicates, then falling dramatically during chromosome segregation.

(b) Stages of the cell cycle, which in adult vertebrates rapid cell cycles take about 24 hours. Mitosis (M) represents about 5% of the cycle, and there is a substantial gap (G1) between mitosis and DNA synthesis (S), as well as a gap (G2) between replication and mitosis.


The interaction mechanisms of radiation and some chemicals which have strong adverse health effects occur through alteration or interference with the cell cycle. X-ray radiation is known to damage cells and their DNA, while caffeine is known to accelerate mitosis. Rowley (1990) reported on studies into the repair of radiation-induced chromatid aberrations: relationship to G2 arrest in CHO cells. The literature suggests that the function of radiation-induced G2 arrest is to allow repair of potentially lethal damage before cell-entry into, and damage expression in, mitosis. The nature of the damage repaired is not known, but chromosome aberrations have been considered.

To examine this possibility in G2 cells, Rowley (1990) compared the rate of repair of chromatid aberrations in CHO cells progressing to or arrested in G2, with the rate of repair of the damage which gives rise to G2 arrest. To measure aberration repair rates, exponentially growing CHO cells arrested in G2 with 1.5, 2.5 or 3.5 Gy of X-rays were released into mitosis by treatment with 5 mM caffeine immediately or 1, 2 or 3 h after irradiation. Aberration frequencies in these cells were then related to the caffeine-free (repair) interval. To measure the rate of repair of arrest-causing damage a split-dose procedure was used. The half-times for aberration repair were approximately 1 h for
achromatic gaps and 1.5 h for breaks, intrachanges and interchanges. The half-time for arrest damage repair varied with radiation dose. This result suggests that chromatid aberrations are not a primary cause of radiation-induced G2 arrest.

While Rowley (1990) has shown arrest during G2 of the cell cycle under X-ray irradiation, DeFrank et al (1996) show that UV radiation arrests the cell cycle in G1, slowing the transit of cells into the S-phase, which is reduced by the application of caffeine. DeFrank et al. investigate the role of the p53 tumour suppressor protein whose function is inactivated in malignant cells. They find that p53-null cells are more sensitive to UV light, only in the presence of caffeine, implicating caffeine in processes which reduce cell repair and enhance cell damage under UV exposure.

The role of caffeine in accelerating mitosis before DNA repair can take place implicates caffeine in enhancing chromosome aberrations. This shows the quite complex interactions of diet, environmental exposure and other factors such as familial genetics, in the susceptibility of people to adverse health effects.

10.6 EMR alteration of the cell cycle time:

Brulfert et al. (1985) studied the growth of plant roots in 2 day exposure to a strong (430 V/m) ELF (60 Hz) electric field in vivo. They found that exposed roots were shorter because cell elongation was reduced in exposed roots compared to controls. Heller and Teixeira-Pinto (1959) showed that a strong pulsed RF (27 MHz) field caused chromosome breaks which probably occurs in the replication of DNA in the S-phase. These pose the question as to whether animal cells are similarly affected.

Levin and Ernst (1995) report that 60 Hz fields (3.4-8.8 mT) and magnetic fields over the range DC-600 kHz (2.5-6.5 mT) can alter the early embryonic sea urchin embryos by inducing alterations in the timing of the cell cycle. Their results, as for the cellular studies above, were dose-dependent and biphasic as a function of frequency, duration and timing of the exposure. Low frequencies advanced mitosis and higher frequencies delayed mitosis. Stein and Lian (1992) point out the importance of cell cycle perturbations since the loss of growth control in transformed and tumour cells is accompanied by an abrogation of developmental regulatory mechanisms that are functionally coupled to proliferation.

Do the differences of these sea urchin cells and human cells mean that people will not experience alteration of their cell cycle in exposure to EMR?

Conti et al. (1983) investigated the effects of extremely low frequency EMR on immature human peripheral blood lymphocytes which were also exposed to substances which participate in the mitosis of the cells. They found that a frequency window (3-50 Hz) significantly inhibited the conA-induced blastogenesis, while the pokeweed mitogen (PWM) was significantly affected only at 3 Hz. Conti et al. explored the mechanisms which EMR might have and excluded a direct effect on thymidine incorporation. They focus on the flux of calcium ions. A reduction of calcium ions upon exposure to EMR (through the outward flow through the cell membrane - calcium ion efflux). The effect on lymphocytes of calcium loss represents a decrease in the rate of DNA synthesis in all cells and/or a reduction in the number of cells undergoing DNA replication.

Hence they conclude: “Ca$^{2+}$ ions are involved in the control of lymphocyte proliferation. In fact, mitogenic lectins produce a rapid, initial calcium influx and calcium is required for
DNA synthesis some 18-72 h after the mitogenic stimulus.” Considering the theoretical (and observed) effects of EMR on cellular efflux of calcium ions “we think that an alteration of calcium fluxes by EMF may be the most realistic hypothesis to explain the observed inhibitory effect on human lymphocyte blastogenesis.”

Human lymphocytes are the primary agents in the immune system, in the form of T-cells, B-cells and NK-cells (natural killer). The calcium ion efflux is now well documented to increase with ELF modulated RF signals and hence these signals, at levels down to SAR of 0.00015 W/kg, Schwartz et al. (1988) have the effect of reducing the protection to infection offered by the immune system cells (white blood cells), Walleczek (1992). This corresponds to an energy flux of about 0.04 $\mu$W/cm$^2$ for isolated frog hearts or about 0.4 $\mu$W/cm$^2$ for a human body.

Professor Stephen Cleary's group has been studying the effects of cell cycle changes when exposed to RF (27 MHz) and MW (2.45 GHz) radiation. Clearly et al. (1990a) exposed human blood to these EMR frequencies under isothermal conditions (37 $\pm$ 0.2°C) for 2 h and observed a statistically significant biphasic, dose-response dependent effects of the radiation on human lymphocyte proliferation, both with and without a mitogenic stimulation.

Cleary et al. (1990b) carried out a similar exposure experiment on human glioma cells. They found alterations of the cell proliferation which were not caused by RF-induced cell heating. The dose-response for both frequencies was biphasic. Lower exposures enhanced proliferation while exposures over 50 W/kg suppressed DNA and RNA synthesis. Statistically significant time-dependent alterations were detected up to 5 days postexposure, suggesting a kinetic cellular response to RF radiation and the possibility of cumulative effects of cell proliferation. Cleary et al. (1992) concluded that there is direct, nonthermal cellular effects of RF radiation which included effects on the mitotic cell cycle but no mechanisms had been identified.

Cleary et al. (1996) exposed Chinese hamster ovary (CHO) cells to within 37+/0.1°C, to 5 W/kg and 25 W/kg signals of 27 MHz and 2.45 GHz radiation. They studied the effects at each phase of the cell cycle, including DNA distributions. They found that a 2hr exposure induced significant time-dependent cell cycle alterations for up to about 4 days. These effects were generally reversible over 96 hours and were twice as great for the 2.45 GHz microwave signal as they were for the 27 MHz radiofrequency signal. They considered this to be a real effect of relatively low magnitude and in "agreement with predictions of a theoretical analysis", referring to Liu and Cleary 1995.

10.7 Cell Structure and EMR Alterations Conclusion:

RF and MW electromagnetic radiation has frequency and intensity effects on the alteration of cell cycles which are not heat induced effects. Highly probable mechanism involves the change in cell cycle with effects on DNA efficacy and altered membrane permeability to key agents. For example, the efflux of calcium ions which alters the synthesis of DNA and other aspects of the cell cycle in plant, animal and human cells, including cells of the central nervous system, immune system and cardiac system.

11. Cellular Control Factors

11.1 Gap-Junction Communication:
Cell-to-cell communication takes place through signals transmitted through the intercellular fluid, and through direct cell-to-cell contact through two apposed epithelial cells made of two hexagonal studs embedded in the membrane layer, called a Gap Junction, Figure 19.

Through this structure, ions, amino acids, sugars, nucleotides and other molecules which are smaller than $20 \, \text{Å}$ in diameter pass, but proteins, nucleic acids and larger molecules cannot, from Bretscher (1985).

![Diagram of Gap Junction](image)

Figure 19: Gap Junction between adjacent cells (Schematic) from Bretscher (1985).

### 11.2 Gap Junction Alteration by EMR:

A 2 mT 50 Hz field induced a 160 % flow of cAMP through gap junctions in a monolayer of mouse fibroblast cells, measured immediately after a 5 minute exposure, Schimmelpfeng et al. (1995). Cyclic AMP is a primary “second messenger” of the cell developmental biochemistry along side calcium ions. Cooper (1995) calculated that millisecond applications of electric fields on the range 0.01 to 0.1 V/cm can result in significant hyperpolarizations and depolarizations across the gap junction. Cell-to-cell is a vital biological function. Disruption of the gap junction communication is associated with unregulated cell growth, Adey (1989).

Fletcher et al. (1987) noted that the blockage of the entry of natural cytolytic substances, alpha-lymphotoxin (LT) and recombinant tumour necrosis factor (TNF), into Chinese hamster ovary cells depends on their ability to form gap junctions, a function which varies between different strains of these cells. Fletcher found that the phorbol ester cancer promoter (TPA) opens gap-junctions to permit the entry of LT, leading to cell death (lysis) in a dose-dependent fashion.

Weak RF fields (450 MHz, 1-1.5 mW/cm$^2$ incident energy) with 16 Hz sinusoidal modulation, enhanced this ability of TPA to impair gap-junction communication. The effect did not occur without modulation.
Oncogenes may also interrupt gap-junction communication. Hence, EMR modifies gap-junction communication in ways which are potentially adverse to the health of tissue, either through cell death or through disrupted growth control which leads to cancer cells.

11.3 Extra-cellular environment:

In cellular aggregates that form tissues of higher animals, cells are separated by narrow fluid channels that take on a special importance in signaling from cell to cell. These channels act as windows on the electrochemical world surrounding each cell. Hormones, antibodies, neurotransmitters and chemical cancer promoters, for example, move along them to reach binding sites on cell membrane receptors, Adey (1992a). These narrow fluid “gutters”, typically not more than 150 Å wide, are also preferred pathways for intrinsic and environmental electromagnetic (EM) fields since they offer a much lower electrical impedance than cell membranes. Although this intercellular space (ICS) forms only 10 % of the conducting cross section of typical tissue, it carries at least 90 % of any imposed or intrinsic current, directing it along cell membrane surfaces.

11.4 Signal Transduction:

The division of labour among the cells of a multicellular organism requires that each cell population be able to call on the services of some cell populations and respond to the requirements of others. Much of this is accomplished with chemical and electrical signals. Yet most of the arriving signals never invade the privacy of the cell. They are picked up on the surface of the cell by molecular antennae called receptors. This initiates the communication into the cell in a process termed “signal transduction”.

“Signal Transduction refers to reactions by which the cell receives and acts upon regulatory information from outside the cell. Information-containing signals may include neural messages, hormones, growth regulatory factors, chemical substances, physical forces, and electromagnetic variables such as heat, light, and internal currents from bones and muscles. Signal Transduction is very specific and sensitive. Only particular cells respond to signals and some signal transduction systems can amplify the incoming signal by many orders of magnitude, for example a single photon of light in the eye can induce the synthesis of millions of molecules of neurotransmitters in the nerves leading from the eye to the brain (Stryer, 1986).”

11.5 Signal Transduction structures:

Numerous stranded protein molecules protrude from within the cell into this narrow ICS. Their glycoprotein tips form the glycocalyx which senses the chemical and electrical signals in the surrounding fluid. Their highly negatively charges tips form receptor sites for hormones, antibodies, neurotransmitters and for many metabolic agents, including cancer promoters.

These charged terminals form an anatomical substrate for the first detection of weak electrochemical oscillations in the pericellular fluid, including the field potentials arising
from activity of adjacent cells or as tissue components of environmental EM fields, Adey (1993).

A schematic of the cell plasma membrane is given in Figure 20, from Bretscher (1985).

These stranded protein molecules are the structures providing signal transduction of biochemical messages into the cell to alter cell metabolism or behaviour as a response to external (cell to cell, or environmentally sourced) stimuli.

![Cell membrane schematic](image)

Figure 20: Cell membrane (schematic) showing the bimolecular layer in which cholesterol and other protein molecules are imbedded, with the stranded (alpha-helix) protein which has a coiled hydrophobic section within the membrane and “Y” shaped receptor sites on the extracellular strands, Bretscher (1985).

The amino acid sequence of these stranded proteins reveals a hygrophobic segment of 23 amino acids in the portion which passes through the cell membrane, and the response of these strands to epidermal growth factor (EGF) results in the proposition that this short segment produces vibration modes in the helical proteins which act as a nonlinear amplifier of the signal, Ullrich et al. (1985), Lawrence and Adey (1982).

11.6 Signal transduction messengers:

An external signal (first messenger) is provided by a messenger binding to a receptor on the stranded protein. The alpha helix transfers the message by changing shape successively down its length. At some point the signal is transferred to ions or chemicals in the cytoplasm through the action of an "amplifier" enzyme. A typical amplifier process involves adenylyl cyclase which converts adenosine triphosphate (ATP) to cyclic adenosine monophosphate (AMP) (cAMP) by removing two of the three phosphate groups. ATP serves the cell by donating energy to chemical reactions. The intracellular signals are carried by "second messengers" such as cAMP.
The number of second messengers is surprisingly small, in other words, the intracellular signal pathways are remarkably universal. Yet the known messengers are capable of regulating a vast variety of physiological and biochemical processes.

Three of the major signal pathways are:

1. The Adenylate cyclase pathway, converting ATP to cAMP, both enhanced by stimulation and reduced by inhibition. This also modifies the calcium pathway.

2. The calcium ion, IP₃ and DG, pathway. This plays a central role in the regulation of cell growth and is not known to be inhibited.

3. The polyamine pathway, which involves the enzyme ornithine decarboxylase (ODC).

Figure 21 shows a schematic of the first two of these signal transduction pathways.

The third signal transduction pathway involves polyamine biosynthesis. The polyamines are found ubiquitously in nature and have been closely linked to the processes of cell proliferation, hypertrophy and differentiation in eukaryotic cells, Byus (1994). (Eukaryotic cells are cells of higher plant and animals, having a true nucleus). ODC decarboxylates, or removes, the carboxyl group from ornithine to yield putrecine or diainobutane, and by a further series of reaction yields spermidine and spermine, Byus (1994). Enhancement of Ornithine decarboxylase (ODC), the key regulatory enzyme in mammalian polyamine biosynthesis, is rapidly induced by mitogens and tumor promoters, Mar et al. (1995).

Elevated levels of ODC have been found in a number of animal and human tumours, for example stomach, colon and esophagus, Yoshida et al. (1992). A detailed analysis of ODC in Human Colon Cancer suggests that ODC activity is influenced by kinase activity, with protein kinase C being the most likely candidate, Sumiyoshi et al. (1991). Mustelin et al. (1987) have shown that ODC is also linked to T-cells membrane so that activation of ODC can be linked to neoplastic changes in cells and to alteration of immune system cells.

Due to the high sensitivity of this enzyme (ODC) to a large variety of stimuli and the involvement of changes in ODC activity and polyamines in a variety of pathologies, including cancer, ODC appeared to be a logical choice to investigate as a potential marker of exposure of cells or tissues to low-energy electromagnetic fields, Byus (1994).

Given the fundamental role of the signal transduction processes in the regulation and control of cell processes, including proliferation which occurs in cancer cells, and in the development of all cells in human bodies including brain, CNS and immune system, any evidence of changes in these processes because of exposure to environmental electromagnetic radiation is of grave concern.

The research reported below documents many induced changes at the cellular level due to EMR exposure. This follows with epidemiology showing increased health risks associated with increased EMR exposure.
11.7 Signal Transduction Alteration by EMR:

Many laboratories have now observed increases in the enzyme ODC in cultured cells following a variety of electromagnetic fields, from pulsed static and ELF fields to modulated microwave fields. At least six separate laboratories have observed changes in ODC activity comparable to what is reported here when monolayer cultured cells were exposed to a number of ELF exposure paradigms, including pulsed electromagnetic fields, 50 Hz amplitude modulated 450 MHz fields, and 50-65 Hz electromagnetic fields, Byus (1994).

Luben (1995) summarizes the concept of signal transduction in cells and the effect of EMR:

“Clearly, any environmental influence (e.g. electromagnetic fields) that modifies signal transduction pathways in normal cells could also influence the potentially tumorigenic pathways in susceptible cells, either by enhancing the likelihood of transformation by other tumorigenic stimuli of by acting in a direct tumorigenic manner. Thus, it is not necessary to hypothesize, as some have done, that EMF must cause genetic damage to cells in order to cause cancer or developmental abnormalities. Nor is it necessary to hypothesize that EMF must alter the expression of genes in
cells directly (indeed, recent studies make this hypothesis seem rather unlikely).

By influencing signal transduction pathways, which in turn can generate cell proliferation, cell differentiation, and even transformation to a cancer phenotype, EMF can potentially be involved in a host of disease processes without ever penetrating the cell membrane in any significant manner."

There is clear biological evidence at the cellular and molecular level which shows that RF/MW radiation can be absorbed at very low levels and produce significant changes in cell behaviour and structure through signal transduction, including carcinogenic activity. Galvanovskis et al. (1996) show that modulated EMR fields change the concentrations of calcium ions and their oscillations in human leukaemia T-cells. T lymphocytes (T-cells) recognize intracellular antigens, presented at the surface of the cells. Thus there are biological mechanisms which could be related to the production of childhood leukaemia as identified through epidemiology.

11.8 Alterations in Ornithine Decarboxylase activity:

Being a frequency-related effect, the degree of coherence of the ELF signal or the modulation frequency can be relevant. Litovitz et al. (1993) investigated this matter using L929 mouse fibroblast cells exposed to 915 MHz microwaves modulated at 55, 60 and 65 Hz, with an SAR of 2.5 W/kg. They found that, as for ELF signals, a period of coherence of about 10 s was required to gain the full ODC enhancement. Litovitz et al. (1994), using a 60 Hz signal, imposed noise containing frequencies from 30 to 90 Hz.

They determined that full ODC enhancement was obtained when the rms value of the noise was less than one tenth of that of the coherent signal. These results could well have been influenced by the thermal noise of the rather intense microwave signal used. Referring back to table 2, no effect was found for calcium-ion efflux at 0.2 W/kg and higher but very significant effects were found between 0.00015 and 0.075 W/kg.

Byus et al. (1987) investigated the ODC activity in a number of established cell lines under the influence of low-energy 60 Hz EM fields. They used a 1 hr exposure to a 10 mV/cm 60 Hz field which produced a 5-fold increase in ODC activity in human lymphoma CEM cells and a 2- to 3-fold increase in mouse myeloma cells (P3) relative to unexposed cultures. Depending on the cell type, the ODC activity remain elevated for several hours after the 1 hr exposure had ceased. Reuber H35 hepatoma cells grown in monolayer culture had a 30 % increase in ODC activity with a 0.1 mV/cm field applied for 1 hr, but no effect from a 10 mV/m field applied for 2 or 3 hrs. This is another example where high intensities find no change but lower intensities do cause biological changes. Hence, while results vary with exposure interval and field strength, this shows that EMR alters ODC activity in such a way that 60 Hz fields are shown to have the potential ability as a tumour promoting stimulus in the same way that ELF modulated RF/MW also does.

Byus et al. (1988) showed a 50 % increase in ODC activity in Reuber H35 hepatoma cells with 450 MHz microwaves modulated at 16 Hz for 1 h. This was an athermal exposure,
giving less than 0.1°C temperature rise, with a 1 mW/cm² peak-envelope-power and an SAR of 0.08 W/kg. With σ=1.2 S/m, Eq. 11 estimates the exposure as S=35 µW/cm². The effect persisted for several hours following exposure. Modulation frequencies of 60 Hz and 100 Hz had no effect. A phorbol ester tumour promoter (TPA) enhanced the ODC activity in combination with the EMR. Similar ODC activity changes were observed when Chinese Hamster ovary cells and 294T melanoma cells were exposed to the radiofrequency EMR regime.

While the mechanism by which EM fields increase ODC activity is still unknown, from the observation that brief exposure of cells to EM fields altered the cell’s responsiveness to TPA, and the fact that TPA has a specific receptor in the membranes of all cells, this suggests that this, and other data, are consistent with the concept that protein kinase C in the membrane may be a target for low energy EM fields.

The observation, Balcer-Kubiczek and Harrison (1985), that prior exposure to microwaves (2.45 GHz, 130 pps) led to the enhanced effect of benzpyrene- or X-ray-induced transformation frequencies, provided the cells were treated with TPA, is also consistent with the hypothesis that primary cellular effects of low level microwave fields and of TPA, is at the level of the cell membrane.

Balcer-Kubiczek and Harrison (1985) conclude that this is further evidence that microwaves are cancer promoters using mechanisms which are athermal and act at the cell membrane level. Direct application of this to animal and human cancer is found in Sumiyoshi et al. (1991). They state:

“ODC is a rate-limiting enzyme in the biosynthesis of polyamines linked with normal and neoplastic cell proliferation. Induction of ODC has been suggested to play an important role in tumor including skin, urinary bladder, stomach and colon carcinogenesis in rodent models. ... Studies have shown that human colonic mucosal levels of ODC activity are lowest in colonic mucosa from healthy controls but are increased in normal-appearing mucosa from subjects with colonic polyps and from colon cancer patients.”

Yoshida et al. (1992) investigated levels of ODC gene in human cancers, found that the ratios of ODC mRNA in tumours compared to normal tissue was 14.6 ± 3.7 for all
esophageal cancers, 2.9 ± 0.9 for stomach cancer, 2.1 ± 0.9 for colon cancers, and 0.9 ± 0.2 for liver tumours.

11.9 ODC Summary and Conclusions:

Research shows that ODC, a growth regulating enzyme in the polyamine signal transduction pathway, is enhanced in a number of cell lines, including human cells, in the presence of ELF or ELF modulated RF/MW radiation. The mechanism is at yet unknown but could well involve protein kinase C in a receptor on the surface of the cell membrane.

This is relevant to the effects of signal transduction pathways on the formation and promotion of cancer for it is found that ODC levels are highly elevated in neoplastic tissue in many human cancers. The relationships between signal transduction processes, cell growth, differentiation and neoplastic transformation of cells is very complex.

What is relevant here is that many genes known to be oncogenes are clearly analogous to membrane receptors or to molecules involved in the signal transduction pathways activated by membrane receptors. Intracellular regulatory pathways such as the cell division cycle and the promotion of differentiation and gene expression are very likely to be modulated by a multitude of signal transduction pathways in both normal cells and in neoplastically transformed cells, Luben (1995).

Clearly, any environmental influence, such as electromagnetic fields, that modifies signal transduction pathways in normal cells could also influence the potentially tumorigenic pathways in susceptible cells, either by enhancing the likelihood of transformation by other tumorigenic stimuli or by acting in a direct tumorigenic manner.

Thus it is not necessary to hypothesize, as some have done, that EMF must cause genetic damage directly to cells in order to cause cancer or developmental abnormalities. Nor is it necessary to hypothesize that EMF must alter the expression of genes in cells directly. By influencing the signal transduction pathways, which in turn can regulate cell proliferation, cell differentiation and even transformation to a cancer phenotype, EMF can potentially be involved in a host of disease processes without ever penetrating the cell membrane in any significant manner, Luben (1995).

12. Calcium Ions:

12.1 Calcium Ion Processes:

12.1.1 Ionic calcium is ubiquitous in mammalian cells.

Calcium is nearly ubiquitous in human cells. Calcium ions (Ca$^{2+}$) play vital roles in many biological processes of living tissues, including signal transduction processes at the cell level, which includes processes which control the binding and release of molecules to the surfaces of cells which influence primary cellular behaviour. The intracellular fluid (fluid inside the cell membrane surrounding the cellular nucleus), is rich in calcium ions. When calcium ions flow outwards through the cell membrane, it is called “calcium-ion efflux”.

A molecular analysis of the cAMP pathway shows that cAMP often activates the calcium ion pathway and modulates its activity. The heart provides a now classic example. There epinephrine acts through the cyclic AMP pathway to modulate the level of
intracellular calcium. This the force of each heart beat which is governed by a brief calcium pulse.

In certain cells, such as neurons, the source of calcium ions is well known: it is the extracellular fluid. Nerve signals arriving at the synaptic terminals of a neuron decrease the voltage across the neuronal cell membrane; the resulting “depolarization” opens voltage-sensitive calcium channels through the cell membrane. Before depolarization, the Ca$^{2+}$ concentration in the cytoplasm is about $6 \times 10^{14}$ ions/cc. The Ca$^{2+}$ concentration outside the neuron is about 10,000 times higher. Hence the depolarization enables calcium ions to flood into the neuron and trigger a cell response. Even a rather small change in intracellular calcium can exert profound changes in cellular activity, Bretscher (1985). In the synaptic terminals of neurons, for example, calcium induces the release of neurotransmitter molecules.

The extracellular fluid cannot be the sole source of calcium ions. For one thing the absence of extracellular calcium does not prevent external messenger acetylcholine from stimulating the pancreas to release the digestive enzyme amylase. Thus it has become apparent that calcium employed by a cell for internal signaling not only enters the cell from outside but is also released from internal reservoirs. There turn out to be many examples of hormones or neurotransmitters employing internal calcium to control physiological processes, Bretscher (1985).

Hence external stimuli which can cause influx or efflux of calcium ions from the cell have clear and important consequences for cell growth regulation, cell death, neurotransmitter and hormone balance.

12.2 Calcium ions and Electromagnetic Interactions:

A perspective on the EM properties involved can be seen by noting that the characteristic membrane potential of most cells is about 0.1 V in a resting state. Since this exists across the very thin (40 Å) plasma membrane, it creates an enormous barrier of the order of $10^5$ V/cm. However imposed ELF and amplitude modulated RF fields produce tissue gradients in the range $10^{-7}$ to $10^{-1}$ V/cm, which are gradients involved in essential physiological functions in marine vertebrates, birds and mammals, Adey (1981). In vitro studies have reported similar sensitivities for cerebral Ca$^{2+}$ efflux, and in a wide range of calcium-dependent processes that involve cell membrane functions, including bone growth, modulation of intercellular communication mechanisms that regulate cell growth, reduction of cell-mediated cytolytic immune responses, and modulation of intracellular enzymes in signal transduction. These processes have been confirmed for many human cell types, including lymphocytes, ovary cells, bone cells, fibroblasts, cartilage cells and nerve cells, Adey (1992a).

Since the electric field strength varies as the square root of the exposure (Eq. 3), for a 147 MHz modulated RF field with an environmental exposure of 1μW/cm$^2$ or 0.1μW/cm$^2$ the Tissue Gradients are estimated at $3.5 \times 10^{-3}$ V/cm and $1.1 \times 10^{-3}$ V/cm, respectively. These are still at least 10,000 times higher than the lower limit of $10^{-7}$ V/cm.

Calcium ion efflux from within cells clearly alters the intracellular calcium ion concentration, which alters the Calcium ion signal transduction process which is vital to balanced regulation of cell growth and, in neuron tissue, neurotransmitter and
neurohormone production and reception. In other tissue it alters the reaction to the stimulation of antibodies because the role of calcium ion homeostasis in activation of channels of cells in the immune system.

Luben (1995) summarizes research through which RF radiation which is modulated at ELF frequencies changes the calcium ion efflux in Table 5.

It is now widely accepted that calcium plays a central role in the development of the immune response, Grinstein and Klip (1989). Changes in the cytoplasmic free calcium concentration (Ca^{2+}) are thought to be essential for responses as varied as bacterial killing by neutrophils and the synthesis and secretion of antibodies by lymphoid cells.

It is pertinent to note that the “no effects” studies of Merritt et al. (1982) are consistent with the power intensity windows identified by Blackman et al. (1980a, 1988). It is well established that calcium ion efflux changes are not linearly related to intensity, but rather to particular combinations of intensity, modulation frequency and temperature range. It is also pertinent to note that although a great deal of calcium ion efflux research has focused on ELF exposures, the table 5 below is for modulated RF/MW exposure, with effects being found for carriers in the range 50 MHz to 915 MHz and modulation frequencies in the range 0.5 to 32 Hz.

Calcium ion signaling is a function of the central nervous system (CNS). Walleczek (1992) proposes that research findings show that membrane-mediated calcium ion signaling processes are involved in the mediation of ELF effects on the immune system. ELF modulated microwaves have similar effects.

Shandala et al. (1979) found that calcium ion efflux varies in living animal cells at 10\(\mu\)W/cm\(^2\) using microwaves (about 0.0075 W/kg), consistent with Kolomytkin et al. (1994).

The understanding of the role of intercellular calcium ions has been growing and evolving rapidly over recent years. The fact that ELF radiation, and RF/MW radiation which is modulated at ELF frequencies, significantly alters the calcium ion concentrations and efflux in intracellular fluid is well proven and documented down to SARs of 0.00015 W/kg. Electromagnetic fields, through their effect on calcium ions, play a vital role in the immune system, Walleczek (1992). Walleczek (1992) quotes research relating to the role of calcium, sodium and potassium ions, including research showing that EMF could alter the activity of the membrane incorporated Ca\(^{2+}\)-ATPase responsible for pumping Ca\(^{2+}\) out of the cell (calcium ion efflux).

In addition, data from two laboratories demonstrate that ELF fields alter the activity of another membrane ion pump, Na\(^+/K^+\)-ATPase with current densities as low as 50\(\mu\)A/cm\(^2\) and estimated, by the authors, to also have an effect at 1\(\mu\)A/cm\(^2\). At 50\(\mu\)A/cm\(^2\), \(J = 0.5\)

<table>
<thead>
<tr>
<th>Effects</th>
<th>Species</th>
<th>RF Mod (MHz)</th>
<th>Intensity (mW/cm(^2))</th>
<th>Time (min)</th>
<th>SAR (W/kg)</th>
<th>Reference</th>
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<tbody>
<tr>
<td>Altered calcium-ion efflux in brain tissue in vitro:</td>
<td>Chicken</td>
<td>147</td>
<td>6-20</td>
<td>1-2</td>
<td>20</td>
<td>0.002* Bawin et al.(1975)</td>
</tr>
<tr>
<td>Factor</td>
<td>Species</td>
<td>Frequency (Hz)</td>
<td>Intensity</td>
<td>Sample Spacing</td>
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<tr>
<td>Influence of pH and lanthanum</td>
<td>Chicken</td>
<td>450</td>
<td>0.75</td>
<td>20</td>
<td>0.0035</td>
<td></td>
</tr>
<tr>
<td>Frequency and intensity</td>
<td>Chicken</td>
<td>147</td>
<td>0.83</td>
<td>20</td>
<td>0.0014</td>
<td></td>
</tr>
<tr>
<td>Specificity</td>
<td>Chicken</td>
<td>147</td>
<td>0.083</td>
<td>20</td>
<td>0.0014</td>
<td></td>
</tr>
<tr>
<td>Intensity specificity and sample spacing</td>
<td>Chicken</td>
<td>147</td>
<td>0.083</td>
<td>20</td>
<td>0.0014</td>
<td></td>
</tr>
<tr>
<td>Intensity specificity and sample spacing</td>
<td>Chicken</td>
<td>147</td>
<td>0.1-1</td>
<td>20</td>
<td>0.005-0.005</td>
<td></td>
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<tr>
<td>Two intensity ranges</td>
<td>Chicken</td>
<td>50</td>
<td>1.5</td>
<td>20</td>
<td>0.0013</td>
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<tr>
<td>Theoretical analysis of RF dependence</td>
<td>Chicken</td>
<td>50</td>
<td>-</td>
<td>20</td>
<td>~0.001</td>
<td></td>
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<tr>
<td>Test of predictions of theoretical analyses</td>
<td>Chicken</td>
<td>147</td>
<td>0.37</td>
<td>20</td>
<td>0.0006</td>
<td></td>
</tr>
<tr>
<td>No effect for pulse modulation</td>
<td>Rat</td>
<td>1000</td>
<td>0.5-15</td>
<td>20</td>
<td>0.15-4.35</td>
<td></td>
</tr>
<tr>
<td>No effect for pulse modulation</td>
<td>Rat</td>
<td>1000</td>
<td>0.5-10</td>
<td>20</td>
<td>0.29-2.9</td>
<td></td>
</tr>
<tr>
<td>Change in calcium efflux kinetics in synaptosomes</td>
<td>Rat</td>
<td>450</td>
<td>0.5</td>
<td>10</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Frequency and intensity specificity in cultured neuroblastoma cells</td>
<td>Human being</td>
<td>915</td>
<td>-</td>
<td>30</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Altered calcium ion efflux in brain tissue in vivo</td>
<td>Rat</td>
<td>2060</td>
<td>8,16,32</td>
<td>0.5-10</td>
<td>0.12-2.4</td>
<td></td>
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<tr>
<td>Change in efflux kinetics</td>
<td>Cat</td>
<td>450</td>
<td>3</td>
<td>60</td>
<td>0.29</td>
<td></td>
</tr>
<tr>
<td>Changes found in pancreatic slices</td>
<td>Rat</td>
<td>147</td>
<td>2</td>
<td>60-150</td>
<td>&lt;0.075</td>
<td></td>
</tr>
<tr>
<td>Suppressed T-lymphocyte</td>
<td>Mouse</td>
<td>450</td>
<td>16-100</td>
<td>1.5</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td>Changes in Hearts</td>
<td>Frog</td>
<td>240</td>
<td>0.5,16</td>
<td>30</td>
<td>0.00015-</td>
<td></td>
</tr>
</tbody>
</table>
A/m²; E=2.5 V/m, assuming σ=0.2 S/m. Hence from Eq.(1) S = 1.7 μW/cm² and SAR = 0.00063 W/kg from Eq.6. If the extrapolation to 1μA/cm² is confirmed then the EMR effects will be occurring at 1/2500th of the S and SAR levels estimated here.

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

Walleczek and Budinger (1992) report that:

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

They also state that:

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

For this mechanism to be applicable to the data reported here, a pathway by which magnetically-sensitive radical-dependent processes could influence mitogen-induced lymphocyte Ca²⁺ signaling must be postulated. There is new evidence that such pathways exist.

For example, Con A-induced Ca²⁺ uptake in rat thymic lymphocytes has been shown to depend on the generation of reactive oxygen radical species. There is also evidence from inhibition studies that cytochrome P-450 activity may be involved in Ca²⁺ uptake regulation in rat thymic lymphocytes, Alvarez et al. (1992), and it is known that P-450 function proceeds via radical pair recombination steps, Hollenberg (1992). Thus it is plausible to investigate if externally applied magnetic fields may interfere with radical pair reactions and as a consequence, may alter lymphocyte Ca²⁺ regulation.”

Calcium ion influx has been shown to play a role in the transcript levels of proto-oncogenes c-myc and c-fos which alters in the presence of electromagnetic fields, Karabakhtsian et al (1994). (Proto-oncogenes: altered genes which become carcinogenic.)

Lindstrom et al. (1995) replicate and extend the research of Walleczek (1992), using the T-cell line (lymphocytes) for human leukaemia cells, and show that oscillating low-level magnetic fields produce the same calcium ion reaction as does an antibody. They show that weak magnetic fields initiate calcium ion oscillations with a threshold flux density of 40 μT, a plateau at 150 μT and a frequency range from 5 to 100 Hz, with a fairly broad peak at 50 Hz.
Galvanovskis et al. (1996) report significant 30% reductions in the calcium ion oscillation amplitude in human leukaemia T-cells when exposed to 50 Hz magnetic fields.

Figure 18: The effect of 15 V/m electromagnetic fields on the efflux of calcium ions from chicken brain tissue as a function of modulation frequency. The relative efflux is the difference between exposed and unexposed samples. The data from 1 to 120 Hz are taken from Blackman et al. (1985). Blackman et al. (1988).

The key role of modulation frequency in the alteration of calcium ions was recognized early. A leading researcher in this area, Dr Carl Blackman of the U.S.E.P.A. has shown that research has identified modulation frequencies which significantly alter calcium ion efflux out to 510 Hz, Figure 18.

This takes us away from the long term concentration on the 16 Hz calcium ion oscillation which first attracted attention. We can only speculate on what the results would have been in the above quoted experiments if the modulation of ELF frequency had been extended out to 500 Hz.

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

"These results allow us to suggest that 50 Hz; 100 μT magnetic fields might influence some step in the chain of biochemical events leading to the sustained calcium ion oscillation."

They further note:
“That more than 20 enzymes are thought to incorporate radical chemistry in the conversion of substrates to products. It is possible that some enzymes or intermediates containing radicals are involved in the complex system responsible for intracellular calcium ion regulation. It has been shown that such biochemical reactions may be sensitive to the magnetic field.”

12.3 Intensity and Frequency Windows:

Calcium ion efflux is shown to be enhanced in intensity and modulation frequency windows, suggesting a pseudo-quantum effect. The early work of Dr Susan Bawin and Professor Ross Adey, Bawin and Adey (1976) and Adey (1980) for example, identified differences with exposure intensity and modulation frequency suggesting intensity and frequency “windows” in relation to calcium ion efflux from chick brains. Much of this work was replicated by Dr Carl Blackman at the U.S.E.P.A., e.g. Blackman et al. (1989). In Blackman’s paper he comments that they could not replicate the Bawin and Adey results until his team had carefully examined the power-density dependence of the field and discovering that only certain power densities and certain modulation frequencies were capable of eliciting the response. Blackman et al. (1989) found that at 16 Hz modulation of a 50 MHz carrier with a highest SAR of 0.005 W/kg was far to low to cause heating. They report that statistically significant effects were found at power densities of 1.44-1.67, 1.75, 3.85, 5.57, 6.82, 7.65, 7.77, and 8.82 mW/cm², but not at 0.37, 0.72, 0.75, 2.17, 2.30, 4.32, 4.50, 5.85, 7.08, 8.19, 8.66, 10.6, and 14.7 mW/cm².

Blackman et al. (1989) propose a fractal process with a non-integer dimension of 1.4 to explain a series of highly peaked responses which correspond to cell membrane level amplificational processes. Using the probability of the statistical significance (p) which is p<0.001 for the strong peaks, they note that there is no decrease at the lower power densities making it impossible to extrapolate to a lowest threshold. Dr Adey has shown nonlinear dynamical responses at the cellular level for some time, but Dr Blackman and his group claim to be the first to apply fractal geometry to the problem and in doing so, open the possibility for functional alterations to the CNS due to very weak stimuli.

The lowest reported SARs with statistically significant increases in calcium ion efflux at 0.00015 and 0.0003 W/kg from Schwartz et al. (1990), using 16 Hz modulation and a 240 MHz carrier.

Blackman et al. (1991) define a temperature window for the calcium ion efflux from avian brain tissue. Effects are seen for 36 and 37°C but not for 35 and 38°C. The effects are evident within the normal core temperature range but not outside it.

These results, for power density and temperature windows, explain why no effects were seen in early experiments which used high power densities and raised the temperature of the sample. It is not a simple matter of higher exposure gives greater effects, i.e. there is not a simple dose-response relationship. The effects are highly quantized by particular sets of conditions which trigger cell membrane reactions which involve enzyme amplifiers in the signal transduction process which are thought to be poised at a phase or cooperative transition.

12.4 Calcium-ion Signaling Summary:
ELF and RF/MW modulated at ELF frequencies, change the oscillation frequency and amplitude and they change the influx and efflux of calcium ions in and around the cell membrane.

The changing oscillation frequency and amplitude is related to the immune response of the cell and shows that the oscillating applied field produces an antibody-like reaction as though the cell has been attacked.

The influx and efflux changes relate to the signal transduction pathway in which calcium ions participate. This is one of the biochemical pathways which regulate cell behaviour. This is altered by the applied oscillating electromagnetic field. Since signal transduction controls the cell division, cell differentiation and cell proliferation, this EMR induced alteration to signal transduction has the strong potential to participate in tumour formation or promotion. Alteration of T-lymphocytes and other immune system factors suggests that EMR exposure causes immuno-suppression, partly through induced calcium ion efflux.

The following section on DNA damage and chromosome aberrations is consistent with this. While research shows that DNA is damaged and chromosome aberrations are found in EMR exposed cells, animals and people, the evidence does not point to direct DNA breakage but to the involvement of free radicals or some other cellular level mechanism such as altered signal transduction pathways.

12.5 Calcium ion Conclusion:

Courtesy of Professor Ross Adey, Adey (1993):

“Life on earth has evolved in a sea of natural electromagnetic (EM) fields. Over the past century, this natural environment has sharply changed with the introduction of a vast and growing spectrum of man-made EM fields. From models based on equilibrium thermodynamics and thermal effects, these fields were initially considered too weak to interact with biomolecular systems, thus incapable of influencing physiological functions. Laboratory studies have tested a spectrum of EM fields for bioeffects at cell and molecular levels, focusing on exposures at athermal levels. A clear emergent conclusion is that many observed interactions are not based on tissue heating. Modulation of cell surface chemical events by weak EM fields indicates a major amplification of initial weak triggers associated with the binding of hormones, antibodies, and neurotransmitters to their specific binding sites. Calcium ions play a key role in this amplification. These studies support new concepts of communication between cells across barriers of cell membranes; and point with increasing certainty to an essential physical organization in living matter, at a far finer level than the structural and functional image defined by the chemistry of molecules. New collaborations between physical and biological scientists define common goals, seeking solutions to the physical nature of matter through a strong focus on biological matter. The evidence indicates mediation by highly nonlinear, nonequilibrium processes at critical steps in signal coupling across cell membranes. There is increasing evidence that these events relate to quantum states and resonant responses in biomolecular
systems, and not to equilibrium thermodynamics associated with thermal energy exchanges and tissue heating.”

13. Free Radicals

13.1 Introduction:

A free radical is an extremely reactive molecule which carries an unpaired electron and which has a very short half-life of $10^{-5}$ s or less. Although superoxide anions ($\text{O}_2^-$) are the primary oxygen radicals produced in biological systems, they can also give rise to a cascade of other radicals such as hydroxyl, carbonate and lipoperoxy radicals.

Medical literature documents the role of free radicals in carcinogenesis, Guyton and Kensler (1993):

“Cancer in humans and animals is a multistep disease process. In this process, a single cell can develop from an otherwise normal tissue into a malignancy that can eventually destroy the organism. The complex series of cellular and molecular changes that occur through the development of cancers can be mediated by a diversity of endogenous and environmental stimuli. Active oxygen species and other free radicals have been known to be mutagenic; further these agents have more recently emerged as mediators of other phenotypic and genotypic changes that lead from mutation to neoplasia. Free radical production is ubiquitous in all respiring organisms, and is enhanced in many disease states. Free radicals may therefore contribute widely to cancer development in humans.”

13.2 Cumulative effects:

Commonly used chemicals and drugs produce damaging levels of free radicals, which produce chromosome and DNA damage and suppress the immune system. Enwonwu and Meeks (1995) review the free radical chemistry of tobacco and alcohol in relation to oral cancer. The abstract is included here to illustrate the central role of free radicals in cancer and immune system suppression, whether they are produced by chemicals, ionizing radiation or non-ionizing electromagnetic radiation. They also address the role of free radical scavengers, such as anti-oxidants.

“Abstract:

As shown in this report, abuse of alcohol and tobacco has serious nutritional implications for the host, and generates increased production of reactive free radicals as well as eliciting immunosuppression. Maintenance of optimal competence of the immune system is critical for cancer surveillance. Active oxygen species and other reactive free radicals mediate phenotypic and genotypic alterations that lead from mutation to neoplasia. Consequently, the most widely used chemopreventive agents against oral cancer (e.g., vitamins A, E, C, and beta-carotene) are anti-oxidants/free radical scavengers. These anti-oxidants, both natural and synthetic, neutralize metabolic products (including reactive oxygen species), interfere with activation of procarcinogens, prevent binding of carcinogens to DNA, inhibit chromosome aberrations, restrain replication of the transformed cell,
suppress actions of cancer promoters, and may even induce regression of pre-cancerous oral lesions such as leukoplakia and erythroplakia.

13.3 EMR effects on free radicals:

Modulated EMR has been shown to reduce melatonin levels and to lead to an increase in free radicals and increased cell death.

Barnett (1994) reviews possible mechanisms relating microwave exposure to the action of free radicals:

“There is increasing support for the theory that free radicals play an important role in discrete, important sub-cellular events during exposure to microwaves. The field of magneto-chemistry is beginning to have an impact on the understanding of subtle effects in molecular biology of cell systems. Chemical bonds consist of paired electrons with opposite spins. Free radicals are highly charged and can only form bonds between radicals of opposite spins. Electron spins may be altered by EM fields and radicals prevented from uniting. Recent information on the small unstable molecule, nitric oxide (NO), as a physiological mediator has shown the importance of oxygen free radicals in biological systems. NO is understood to modulate neurotransmission and regulate cerebral arterial blood flow and has been implicated in the pathogenicity of Alzheimer’s Disease.

Microwave-induced lowering of phase transition temperature and increasing membrane permeability is inhibited by the presence of antioxidants, thereby implicating free radical involvement. A number of laboratories have reported enhanced permeability to sodium cation in erythrocytes during exposure to microwave fields.”

Adey (1993) discusses McLauchlan (1992) which proposes a model for the production of free radicals by ELF fields. McLauchlan concludes from his model that “the effect begins at the lowest applied field strength, even at levels below thermal noise (kT). The all-important interaction has an energy very much less than the thermal energy and is effective exclusively through its influence on the dynamics; this is counter intuitive to most scientists.” Adey (1993) goes on to consider the work of Grundler and Keilmann (1978) and Grundler and Kaiser (1992) in which around 42 GHz they found highly tunes resonances in yeast cells, with clear responses down to 5 picowatt/cm².

Grundler et al. (1992) present a synthesis of interaction of nonthermal EM fields with cellular systems. They present a model of EM field transductive coupling, based on magnetic field-dependent chemical reactions, including cytochrome-catalyzed reactions that involve free radicals, such as reactive oxygen or nitric oxide, leading to a further highly cooperative amplification step. They conclude that in such a system “imposed fields can be active even at intensities near zero.”
Lai and Singh, pers. comm. have exposed rat brains to sub-thermal pulsed microwaves (2.45 GHz) and found enhanced single- and double-strand DNA breakage in the presence of enhanced free radicals and accelerated cell death.

The models are now being confirmed by experiments in living tissue as laboratory techniques allow detection of cellular level effects. It is highly likely that the many examples of observed chromosome damage in the presence of RF/MW fields is due to the involvement of free radical mechanisms and/or disruption to intra- and inter-cellular communication. The consequence of this is impairment of the immune system and increased risk of cancer and birth defects, for example.

14. Carcinogenesis processes:

14.1 Introduction:

It has been estimated that 75-80% of all human cancers are environmentally induced, Clemens (1991), 30-40 % of them by diet. The remaining cancers, 35-50%, are primarily from environmental toxins, among which epidemiological research strongly implicates electromagnetic radiation.

Two distinct types of process which lead to neoplasm of cells which can lead to malignant cancer can involve electromagnetic radiation, signal transduction alteration and genetic damage. The first involves the change in signal transduction process in the cells which controls the cells development, and involves calcium ions and/or ODC for example, Byus (1994), Luben (1995), and Weinstein (1991). The second involves DNA and chromosome damage through the action of such agents as free radicals.

A multi-stage process for developing cancer is often described. This starts with initiation, then promotion and finally progression, Weinstein (1988). Adey (1992b) adds "synergism" to include the effects of co-carcinogens, Figure 21.

Initiation involves a single exposure to a carcinogen which damages the nuclear DNA. A single agent (a complete carcinogen) or two or more agents may be necessary, working together in the proper sequence. Promotion involves multiple exposures at certain intervals to agents which do not damage DNA directly. Promotion leads to conversion from benign to malignant tumours. Progression involves the increasing degree of malignancy.

The latency period for most cancers (the time between initiation and appearance of the disease) is often 20 years or more. Initiation is generally thought to change the cell's genetic stores of DNA, but the change is not expressed and a tumour does not result unless one or more promoting agents act repeatedly at a later time. Initiated cells may remain quiescent if they are not stimulated by a promoter, and cancer may never develop if sufficient exposures to promoters do not occur.
Figure 21: Model of multistage carcinogenesis from studies in mouse skin. Initiation results from only a single exposure to a carcinogen that appears to damage nuclear DNA. Promotion involves multiple exposures at certain intervals to agents that do not damage DNA directly. Promotion leads to conversion of benign to malignant tumours, with progression increasing the degree of malignancy. (After Weinstein, 1988).

In a specific context, tobacco proteins are both initiators and promoters. Because of cigarette smoking’s promotional attributes, risks of lung cancer decline after a smoker rejects the habit.

Promotion and progression agents have very weak or no carcinogenic activity when tested alone, but they markedly enhance tumour yield when applied repeatedly following a low or sub-optimal dose of a carcinogen. Promoting agents are not mutagenic and thus are not cancer initiators by an action on the DNA in the nucleus.

Many papers give evidence of EMR as a cancer promoter, e.g. Adey (1992b). Agents which disrupt the gap-junction communication or alter the signal transduction in order to increase proliferation, can be cancer promoters. EMR does change these cellular processes in the same way that known cancer promoters do. In cell cultures the ability of T-lymphocytes (T-cells) to destroy tumour cells is shown, pointing to the importance of the immune system in reducing and eliminating cancer cells. Both 60 Hz ELF and modulated RF fields (450 MHz) fields, Lyle et al. (1983), reduce the lymphocyte killing ability.

Synergism is another form of interaction which occurs when two or more substances potentiate each other’s actions, producing more cancers than can be accounted for by the separate effects of each. The phorbol ester TPA is known to activate the membrane bound enzyme protein kinase C (PKC). Studies of these interactions show that PKC plays a critical role in signal transduction in normal cells and it is irreversibly activated by phorbol esters, Adey (1992b). PKC belongs to a group of cAMP-dependent protein kinases identified as sensitive to weak RF fields amplitude-modulated at ELF frequencies, Byus et al. (1984). Many experiments in cell-lines and in animals have shown synergistic effects of EMR and chemical cancer promoters, benzpyrene or TPA for example.

However, evidence is growing that ELF modulated RF/MW radiation not only alters the cellular level growth regulation processes in a cancer promoting way, but also is involved under some circumstances in the breakage of nuclear DNA. Hence EMR appears to be both a cancer initiator and a cancer promoter, which also enhances progression. In this
way the similarity with cigarettes is quite strong, as are the similarities to the effects of
ionizing radiation, but at a lower, but not insignificant level of impact, particularly because
of the near universal exposure of people to RF/MW radiation.

14.2 DNA breakage and Chromosome aberrations (CA) by EMR:

Carcinogenesis can be initiated through breakage of DNA which leads to the aberration of
chromosomes. This can happen by the direct action of free radicals or by the inactivation
of tumour suppresser genes. Thus it is generally accepted that chromosomal mutations
are causal event in the development of neoplasia, Hagmar et al. (1994). Hence, at the
population level, an increased frequency of CA has thus been generally considered
indicative of increased cancer risk for those exposed to the damage-inducing agent. Thus
it is important to review research which shows CA under EMR exposure.

Two of the important agents identified in these processes are melatonin and free radicals,
Liburdy et al. (1993), Reiter (1994) and possibly also calcium ions. CA may be enhanced
directly by physiological responses to EMR which reduce the production of melatonin or
indirectly by substances such as cysteamine which enhance free radicals. This effect was
found in by Kondo et al. (1985) when investigating DNA damage observed after exposure
to 1.2 MHz infrasound. Alternatively they may impair the DNA repair mechanism, by
altering the cell cycle for example. In either case the result is damaged nuclear DNA.

14.3 Early Biomedical Result: Pulsed RF breaks chromosomes.

Nature, in March 28th, 1959 contains a paper in the Genetics section entitled “A New
Physical Method of creating Chromosome Aberrations”. The authors, Drs Heller and
Teixeira-Pinto at the New England Institute for Medical Research, report a method they
use to prepare medical samples which contain high levels of chromosome aberrations.
They use a radiofrequency source of 27 MHz, a pulse length of about 50 μs and between
80 and 180 pulses per second (pps).

They report asymmetrical particles aligning themselves along the field lines. They
observe that motile bacteria or protozoa migrate along field lines when the RF is on, but
resume random movement when the field is turned off. This can be repeated as often as
desired. They note that the thermal component is so low as not to affect the viability of
these organisms or of mammalian cells. No increase of the temperature of the water was
noted. In the larger organisms, they were able to observe intracellular orientation of the
subcellular particles. They say that this led them to believe that this force might be used
as a powerful and controlled mutagenic agent.

Growing garlic roots were exposed to the field and the water they were in was monitored
and no temperature rise was seen. The tips were exposed to the RF field for 5 mins and
examined 24 h later. They observe, Heller and Teixeira-Pinto (1959):

“Among those aberrations seen were linear shortening of chromosomes,
pseudochiasmata, amitotic division, bridging, irregularities in the
chromosomal envelope. The effects noted mimic those produced by ionizing
radiation and c-mitotic substances.”

Of the papers and reviews I have, it is only cited in Shore (1981), the WHO review
“Environmental Health Criteria 16: Radiofrequency and Microwaves” This short paper is
remarkable for its significance and the fact that it is been almost totally ignored by subsequent reviews and reports. The conclusion about the similarity of effects to those of ionizing radiation and other cell damaging agents is telling. It also related to the role of free radicals, which are known to be produced and cause DNA damage under exposure to ionizing radiation and have been observed in vivo under microwave exposure, Lai and Singh pers. comm. It is also noted in a mouse reproductive study by Dimberg (1995), who used 20 kHz magnetic field with a peak-to-peak amplitude of 15 μT (sawtooth wave). He concludes: “Most of the effects of MF (magnetic field) treatment during the embryonic period were similar to those induced by ionizing radiation but much weaker”.

14.4 ELF studies involving chromosome aberrations, DNA breakage and cancer:

Several ELF exposure studies have been carried out on workers which are of relevance because to the strong similarity between effects of ELF EMR and ELF modulated RF/MW EMR. As noted above the effect of RF/MW modulated by ELF should be even stronger than ELF alone because of RF/MW penetrative effects into mammal bodies.

Murphy et al. (1993) note that since epidemiologic studies have reported a modestly increased risk of childhood leukemia associated with certain electric power wire configurations and since cancer it likely to involve DNA damage, this review discusses the evidence of direct and indirect genetic toxicity effects for both electric and magnetic fields at 50- and 60-Hz and miscellaneous pulsed exposures. Exposure conditions vary greatly among different end points measured, making comparisons and conclusions among experiments difficult. Also in 1993 Liburdy et al. (1993) provided “The first evidence that ELF frequency magnetic fields can act at the cellular level to enhance breast cancer cell proliferation by blocking melatonin’s natural oncostatic action. In addition there appears to be a dose threshold between 2 and 12 mG.”

Lai and Singh (1997a) used a highly sensitive microgel electrophoresis, COMET assay technique to identify single strand DNA breaks, Figure 22, and double strand DNA breaks, Figure 23, from 2hr exposure to 0.1 mT and 0.25 mT 60 Hz magnetic fields in living rat brains.

Lai and Singh (1997a) conclude:

“Because DNA strand breaks may affect cellular functions, lead to carcinogenicity and cell death, and be related to the onset of degenerative diseases, our data may have important implications for possible health effects of exposure to 60 Hz magnetic fields.”
Figure 22: Photographs of single-strand DNA migration pattern of individual brain cells from rats exposed to (a) a bucking condition (0.1 mT), magnetic fields of (b) 0.1 mT, (c) a 0.25 mT and (d) 0.5 mT. (x 400)

Figure 23: Photographs of double-strand DNA migration pattern of individual brain cells from rats exposed to (a) a bucking condition (0.1 mT), magnetic fields of (b) 0.1 mT, (c) a 0.25 mT and (d) 0.5 mT. (x 400)
Lai and Singh (1997b) investigated the effect of melatonin and a spin trap compound (PBN) both of which scavenge free radicals. They found that rats injected with melatonin or PBN before ELF field exposure and 2 hours after exposure. Both of these treatments blocked the magnetic field induced DNA single- and double-strand breaks.

Lai and Singh (1997b) conclude:

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

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

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

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

Skyberg et al. (1993) studied 13 high-voltage laboratory employees and 20 referents participated in a cross-sectional, matched-pairs study of cytogenetic damage. During cable testing the workers were exposed to static, alternating, or pulsed electric and magnetic fields. The alternating magnetic field levels of 50 Hz were 5-10 μT, occasionally much higher. Chromosome aberrations, sister chromatid exchanges, and aneuploidy were studied in peripheral blood lymphocytes. Among seven smoking laboratory employees the mean number of chromosome breaks/200 cells was 2.3, as compared with 0.7 for the job-matched referents. The comparable figures for inhibited cultures were 12.0 versus 6.0. No increase was detected in nonsmokers with either method. The results support, to some extent, the hypothesis of an increased risk of genotoxic effects among high-voltage laboratory workers, particularly a synergistic effect with smoking.
Nordenson et al. (1994) reported that their recent studies have shown a significant increase in the frequency of chromosomal aberrations in human amniotic cells after exposure to a sinusoidal 50 Hz, 30 $\mu$T (rms) magnetic field. To evaluate further interactions between chromosomes and electromagnetic fields, they analyzed the effects of intermittent exposure. Amniotic cells were exposed for 72 h to a 50 Hz, 30 $\mu$T (rms) magnetic field in a 15 s on and 15 s off fashion.

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

14.5 RF/MW studies:

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

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

Hagmar et al. (1994) trichotomize CA into the low (1-33%ile), medium (34-66%ile) and high (67-100%ile). The threshold for low CA is typically 1.0% but in the range 0.5 to 1.5 %, while medium is typically 1.0 to 2.0 %, and high >2 %, but may use a threshold between medium and high of 3 %. Taking the typical classification the Australia Telecom study gas both exposed and control groups in the high category. If the control group was in the “low” category <= 1%, then the Rate Ratio for the clerical staff would be 2.2 and for the linesmen 2.6, both of which are significant (p<0.01).

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

14.6 DNA breakage associated with RF/MW exposure:
Sagripanti and Swicord (1986) showed that non-thermal levels of microwave exposure can produce single and double-strand DNA breaks in E. coli in solution.

Garaj-Vrhovac et al. (1991) showed that cultured V79 Chinese Hamster fibroblast cell exposed to continuous wave (CW) 7.7 GHz microwaves at power density of 0.5 mW/cm² for 15, 30 and 60 min produced a significantly high frequency of specific chromosome aberrations such as dicentric and ring chromosomes in irradiated cells. The dose-response relationships were significant at $p<0.01$.

Garaj-Vrhovac et al. (1992) exposed whole human blood samples to the same exposure regime. With the addition of power densities of 10 and 30 mW/cm². The number of chromosome aberrations increased from 1.5 % in controls to 2.7 to 7.2 % at the rising power densities. There was a statistically significant dose response with $p<0.05$ for total aberrations, $p<0.001$ for Accentric and $p<0.0001$ for micronuclei.

Sarkar et al. (1994) found significant modification of the DNA from mouse cells from brain and testes exposed to 1 mW/cm² 2.45 GHz microwaves for 2 hr/day for 120, 150 and 200 days.

Lai and Singh (1995) exposed living rats brains to a single 2 h exposure to microwaves at 2.45 GHz, pulsed at 500 pps, at SARs of 0, 0.6 and 1.2 W/kg. They found significant dose-response relationships for single strand DNA breaks in an assay carried out 4 hours after exposure for both the hippocampus and the rest of the brain. A second analysis involved assaying the whole brain and continuous wave microwaves at 2.45 GHz and 1.2 W/kg. This showed a statistically significant increase in single-strand DNA breaks between sham and exposed ($p<0.01$) but no significant difference between assays at 0 h and 4 h after exposure.

![Figure 24: DNA breakage in rat brains (SAR = 1.2 W/kg), Lai and Singh (1996).](image-url)
Lai and Singh (1996) repeated the experiment of Lai and Singh (1995) and extended the analysis to include and assay of double-strand DNA breaks and included both pulsed (500 pps) and continuous microwaves at 2.45 GHz. The exposed condition was 2mW/cm$^2$ (SAR = 1.2 W/kg). Statistically significant single-strand DNA breaks were found for both the CW and pulsed signals ($p<0.01$), and for double-strand DNA breaks (pulsed $p<0.01$ and CW $p<0.05$). This data was not available for the MacIntyre Case.

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

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

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

If free radicals are involved in the RFR-induced DNA strand breaks in brain cells, results from the present study could have an important implication on the health effects of RFR exposure. Involvement of free radicals in human diseases, such as cancer and atherosclerosis, have been suggested. Free radicals also play an important role in aging processes [Reiter, 19951. Aging has been ascribed to accumulated oxidative damage to body tissues [Forster et al., 1996; Sohal and Weindruch, 19961, and involvement of free radicals in neurodegenerative diseases, such as Alzheimer’s, Huntington’s, and Parkinson’s, has also been suggested [Borlongan et al., 1996; Owen et al., 19961. Furthermore, the effect of free radicals can depend on the nutritional status of an individual, e.g., availability of dietary antioxidants [Aruoma, 19941, consumption of ethanol [Kurose et al., 1996], and dietary restriction [Wachsman, 19961. Various life conditions, such as psychological stress [Haque et al., 1994 and strenuous physical exercise [Clarkson, 1995], have been shown to increase oxidative stress and enhance the effect of free radicals in the body. Thus, one can speculate that some individuals may be more susceptible to the effects of RFR exposure.

However, it must be pointed out that both melatonin and PBN can have other actions on cells in the brain that can decrease DNA damage. Further support for our hypothesis can be obtained by studying whether other compounds with free radical scavenging properties can similarly block the effect of RFR, and by measurement of other free radical-related cellular effects, such as oxidative molecular damages in lipids, protein, and DNA.”
This is also relevant to the study carried out by Adey et al. (1996) in which rats exposed to cellphone-like signals had 30% fewer tumours than controls and the tumours were statistically significantly smaller. These results were reported to the 1996 BEMS conference in Victoria BC. Dr Singh raised the question with Dr Adey, of the possibility of cell death as an explanation for the result. Dr Adey agreed that this was possible, but stated that it needed to be tested. Lai and Singh (1997c) have found that result.

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

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

14.7 Cellular Base Station radiation's synergistic mutagenic effect with MMC:

A Belgian research team has found that “very-close-range” exposure to microwaves from a cellular telephone base station increases the effect of a chemical mutagen on human blood cells, Maes et al. (1996). Whole blood samples were exposed to 954 MHz microwaves from an actual GSM base station and then to the DNA damaging agent mitomycin C (MMC). Other samples were exposed to either microwaves or MMC alone.

The exposure was at 5 cm from a GSM digital 15 W antenna, giving an SAR of 1.5 W/kg, for a period of 2 h and S = 514 μW/cm². This is a high, but significantly non-thermal exposure.

In this experiment, base station levels of microwaves alone showed no significant mutagenic effects. However, blood samples exposed to microwaves and then to MMC showed a considerably higher, statistically significant number of chromosomal abnormalities than those exposed to MMC alone. Microwave exposure increased the subsequent effect of MMC by about 20 to 50%, the higher levels being produced by higher concentrations of MMC.

It is important to determine what the dose-response relationship of this exposure is. Clearly a non-thermal mechanism is operating, as will many other chromosome aberration observations reported here. These results show that GSM digital microwave radiation is co-carcinogenic with other natural or environmental carcinogens.

Thus people who are exposed to GSM bases station microwaves might have a higher risk of cancer and reproductive effects by making chemical carcinogens more potent in damaging chromosomes. A potentizing effect with skin cancer and UV is a possibility.

On the other hand, the research of Lai and Singh shows that the microwave exposure levels produced by cellular telephones in users heads, free radical production is enhanced, breaking DNA and enhancing the rate of cell death in the brain. The Belgian
research also suggests that the head’s exposure to the cellular telephone antenna could enhance the risk of chemical damage of chromosomes.

14.8 Conclusions on Mutagenic effects of EMR:

Chromosome Aberrations and DNA damage has been found under non-thermal exposure to EMR. ELF and ELF modulated RF have been associated with chromosome aberration in cells and in exposed workers. Microwaves have been shown to produce DNA damage in living rats brains. Microwaves have also been shown to potentize cancer initiators (MMC) and to enhance the chromosome aberrations with exposure to a GSM digital base-station near field signal. Hence EMR is implicated in increasing cancer rates in exposed populations, Hagmar et al. (1994).

Increased cancer incidence can come about by the direct effect of a DNA damaging carcinogen or by the synergistic effect of co-carcinogens. The co-carcinogenistic effect and cancer promotional effect of EMR has been widely suggested and demonstrated through a number of experiments, e.g. Adey (1992b), Byus et al. (1988). Direct effects (in the absence of a cancer initiator) include chromosome aberrations and DNA breakage which is most likely to be the result of the enhanced presence of free radicals in the RF/MW field. The role of melatonin is important here. Direct effects are likely to involve higher mean power densities than co-carcinogenic effects. In Lai and Singh (1995) the inter-animal variability is very small giving a small standard deviation for each exposure group. Even so a linear the dose-response relationship is statistically significant for the “rest of the brain” assayed 4 h after exposure ceased. This suggests that the smallest detectable increase in DNA breakage would be associated, with this small sample size, with an SAR of \(< 0.2 \text{ W/kg} , \sigma = 1.7, S < 62\mu\text{W/cm}^2\). No clear lower limit is able to be estimated.

15. Long-term Animal Studies:

Very few long-term animal studies involving RF/MW exposure have been carried out, largely because of their extreme difficulty and very high cost. The significant studies, also reviewed by the U.S. E.P.A., are reported here.

15.1 University of California, Berkeley:

Professor Charles Susskind and Dr Susan Prausnitz, Dept of Electrical Engineering, UC Berkeley carried out the first reported long term study for the US Air Force, Prausnitz and Susskind (1962). They exposed male Swiss albino mice to 9.27 GHz microwaves, pulsed with a 2 \(\mu\text{s} \) pulse at 500 Hz, 4.5 mins per day, 5 days per week for 59 weeks with an exposure level of 100 \(\mu\text{W/cm}^2\). This amounts to a mean weekly exposure of 0.22\(\mu\text{W/cm}^2\).

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 40 % (23/57) of the exposed animals and 8.1 % (3/37) of the control animals.
Cancer of the white cells or leukosis was seen in 35% (21/60) of the exposed animals compared to 10% (4/40) of the controls. This condition was described as monocytic or lymphatic organ tumours or myeloid leukaemia in the circulating blood.

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

At the final kill at 19-months, 4 months after cessation of exposure testicular atrophy was seen in 21% (14/67) of the exposed group and 5% (1/19) of the control group, and testicular weights were lower for the exposed group. At this stage leukosis was the same in both groups at 18% (12/67) for the exposed group and 21% (4/19) for the control group.

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

These combinations of symptoms pose some challenging interpretations. Testicular degeneration is not associated with the brief heating effect of the daily exposure (4.5 mins at 100 μW/cm²), because this is usually taken to be a non-thermal exposure. The current A/NZ standard for public exposure for microwaves is 200 μW/cm² and there is a proposal to relax it to 1000 μW/cm², which is also claimed by those who believe that only thermal effects exists, to be harmless and non-thermal. In addition, Cairnie et al. (1980) exposed mice to microwaves at power density of 50 mW/cm². They found that the absorbance in the abdomen area of the liver was 11 times greater than the testes, and while the abdomen temperature was increased the testicular temperature was not.

Leukosis (the initiation of leukaemia) requires damaged DNA and chromosome aberrations which are transferred from cell to cell through mutation. The same mechanism could cause testicular degeneration. An accumulated cellular level damage mechanism is not necessarily related to the intensity but can relate to total dose in relation to rates of repair. Hence the averaging of weekly exposure is a meaningful adverse effect related level. Actual public exposure levels of 0.2 μW/cm² and less saw childhood leukaemia incidence and death rate rises at similar exposure levels (2.74 for mortality) in the North Sydney Study.

15.2 University of Washington Case Study:

Establishment of a potential adverse human health effect can be obtained from a suitably designed and executed animal experiment. Such an experiment was carried out at the University of Washington by Professor Arthur Guy and his associates, funded by the United State Air Force. The exposed a large group of rats to pulsed radar-like microwaves, 2,450 MHz, pulsed at 800 pps, 10 μs pulse, at <0.4 W/kg, the human exposure level allowable under the ANSI standard. These rats were compared to a similar group who were sham exposed. Guy found a total of 18 malignancies in the 100 exposed rats compared to 5 in the 100 sham exposed rats, a ratio of RR=3.6 (1.34-9.70), in particular there were 9 endocrine tumours in the exposed group compared to 2 (ratio RR=4.5 (1.0-20.8)) in the control group.
On the other hand, the EPA review team worked with the original University of Washington research team, and undertook further detailed statistical analysis of their results and showed “a statistically significant elevation in the incidence of carcinomas at all sites combined.”

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

### Table 13: Crude incidence of neoplastic lesions (Tumours)

<table>
<thead>
<tr>
<th>Site/Type</th>
<th>Crude Tumor Incidence</th>
<th>Control</th>
<th>Exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal Cortex</td>
<td>12/85 14.1 %</td>
<td>12/76 15.8 %</td>
<td></td>
</tr>
<tr>
<td>Adrenal medulla</td>
<td>1/73  1.4 %</td>
<td>7/67 10.4 %</td>
<td></td>
</tr>
<tr>
<td>Thyroid</td>
<td>9/85 10.6 %</td>
<td>12/76 15.8 %</td>
<td></td>
</tr>
<tr>
<td>Liver</td>
<td>1/85  1.2 %</td>
<td>3/76  3.9 %</td>
<td></td>
</tr>
<tr>
<td>Pituitary</td>
<td>21/85 24.7 %</td>
<td>19/75 25.3 %</td>
<td></td>
</tr>
<tr>
<td>Testes</td>
<td>0/85  0 %</td>
<td>2/76  2.6 %</td>
<td></td>
</tr>
<tr>
<td>Epididymis</td>
<td>0/85  0 %</td>
<td>1/76  1.3 %</td>
<td></td>
</tr>
<tr>
<td>Pancreas</td>
<td>2/85  2.4 %</td>
<td>2/76  2.6 %</td>
<td></td>
</tr>
<tr>
<td>Urinary bladder</td>
<td>0/85  0 %</td>
<td>2/76  2.6 %</td>
<td></td>
</tr>
<tr>
<td>Stomach</td>
<td>4/85  4.7 %</td>
<td>4/76  5.3 %</td>
<td></td>
</tr>
<tr>
<td>Duodenum</td>
<td>0/85  0 %</td>
<td>1/76  1.3 %</td>
<td></td>
</tr>
<tr>
<td>Lymph node</td>
<td>0/85  0 %</td>
<td>1/76  1.3 %</td>
<td></td>
</tr>
<tr>
<td>Soft Tissues, Thorax</td>
<td>0/85  0 %</td>
<td>2/76  2.6 %</td>
<td></td>
</tr>
<tr>
<td>Mesentery</td>
<td>0/85  0 %</td>
<td>2/76  2.6 %</td>
<td></td>
</tr>
<tr>
<td>Lymphosarcoma</td>
<td>3/85  3.5 %</td>
<td>4/76  5.3 %</td>
<td></td>
</tr>
</tbody>
</table>

Total 53/85 62.4 % 63/75 84.0% (RR=1.35, p<0.05)

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

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

McGaughy et al. (1990) point to the more ubiquitous action of melatonin as an example, since,
"Nocturnal pineal melatonin activity is known to be inhibited by ELF electric fields (Wilson et al 1986) and that the pineal gland function is closely coupled to the function of other glands. Melatonin is known to inhibit tumour growth-enhancing hormones like prolactin and estrogen. The postulate has been made that when the blood melatonin concentration decreases because of the action of EM fields on the pineal gland, a tumour growth inhibitor has been reduce or effectively removed, thereby causing a stimulation of tumour growth.

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

The Guy et al. (1985) study, along with other supporting material, led to the recommendation that the US EPA classify RF/MW as a possible human carcinogen (Class C).

The data presented in this report indicate the progressively strengthening evidence of carcinogenicity and other adverse health effects from chronic non-thermal exposure to RF/MW radiation which raise the evidence to classify RF/MW radiation as a highly probable (Class B1) carcinogen.

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

15.3 Polish Study:

Szmigielski et al. (1982) measured the effects of 2.45 GHz microwave radiation at 5, 10 and/or 15 mW/cm², 2h /day, 6 times/week exposure (average weekly exposure 360, 520 and 1,100 µW/cm²), mice able to maintain core temperature under both exposures, specifically investigating lung cancer, breast cancer and skin cancer. Figure 26 shows the result of initiating skin tumours using 3,4 benzo-alpha-pyrene (BP) and assessing the cancer promoting effect of microwaves.
Figure 26: Growth curves of BP-induced skin tumour in mice exposed daily to 10 mW/cm$^2$ of 2.45 GHz microwave radiation for the whole period of tumour growth. \( \text{CDT}_{50} \) refers to the cancer development time when 50 % of the animals have tumours.

Figure 27: The number of lung tumours (following intravenous injection of $2 \times 10^5$ viable sarcoma cells) in mice exposed during 1, 2 and 3 months to 2.45 GHz microwaves (2h daily) at 5 or 15 mW/cm$^2$. \( \text{Oc} \) refers to mice treated with nonspecific stress of over crowding. Cancer development started 2 months earlier for the MW exposed mice and reached the 50 % point for the population after 137 days compared to 305 days. Hence MW significantly accelerated the growth and proliferation of skin cancer tumours.
Figure 27 shows the results of planting lung cancer (sarcoma) cells and then exposing the mice to 5 and 15 mW/cm² MW radiation. The 5 mW/cm² exposure produced an enhancement of lung cancer modules at 2.5 times more than controls after 3 months, but at a similar level to the effect of an over-crowding stress factor. The 15 mW/cm² exposure produced about 5.5 times more lung cancer nodules.

A parallel experiment for breast cancer for control, overcrowding stress, 5 and 15 mW/cm² MW exposure, the 50 % development points were 322, 255, 261 and 219 days, respectively. These show a similar relationship to the results in Figure 26 for lung cancer, except that the stress and 5 mW/cm² effects are reversed.

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

15.4 Duke University Medical Center:

Eight week old female mice were exposed to 2.45 GHz microwaves at power densities of 5 to 15 mW/cm² for 30 min/day over periods between 1 and 17 days, Huang and Mold (1980). Daily mean exposures were about 100 to 300 µW/cm², and exposure conditions were essentially isothermal. The results showed, (a) A sustained activation of tissue macrophages resulting in suppression of lymphocyte responsiveness, and (b) a gradual but temporary stimulation directed to the lymphocytes. Macrophage activation may have caused the early depression of lymphocyte responsiveness. The suppression is later overridden by the cumulative direct stimulation of lymphocytes by microwaves. Prolonged exposures is suggested to eventually result in depressed function in much the same as seen in rheumatoid arthritis which occurs from chronic immune stimulation.

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

15.5 Jawaharlal Nehru University Study:

Ray and Behari (1990) exposed young albino rats of both sexes to 7.5 GHz microwaves, pulsed at 1000 kHz and at a power of 600 µW/cm², for 3 hr /day, averaging 75µW/cm².

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

15.6 Royal Adelaide Hospital Project:

Repacholi et al. (1997) exposed genetically engineered mice to a cell phone signal for 1 hr/day. This was an Australian industry funded study to allay public fears of cell phone health effects was carried out by a team led by Dr Michael Repacholi at the Royal Adelaide Hospital. In an ABC Four Corners documentary Dr Repacholi describes this study:
"We tried to get the most sensitive model of mouse that we could find that would get lymphoma and then see if we exposed them to radio frequency field, whether we could promote that cancer above its normal incidence."

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

![Figure 28: Rate of lymphomas increase in control and exposed groups of mice, Repacholi et al. (1997).](image)

Their 200 genetically engineered mice normally had 22% of them to get lymphomas in their immune system, including B-cells. About half of the mice were exposed to a moderate level of cell phone radiation for 1 hour per day for 18 months. The other half were treated the same way but not exposed. At the end of the study 43% of the exposed mice had lymphomas. The overall Odds ratio was 2.4, p=0.006, 95% CI=1.3-4.5. This is a highly significant results in which the cell phone radiation more than doubled the cancer rate from a 1 hour per day exposure. Mean exposure range was measured as 0.13 to 1.4 W/kg. Hence the mean daily exposure was 0.005 to 0.058 W/kg, averaging 0.03 W/kg, somewhat below the New Zealand Standard of 0.08 W/kg.

15.7 Summary and Conclusions about long-term animal experiments:

Animal experiments confirm that in mice pulsed RF/MW radiation is able to initiate statistically significantly more malignant tumours in many body organs at exposure levels assumed to be non-thermal and safe (0.4 W/kg), McGaughy (1990), and in the presence of a chemical cancer initiator to drastically increase the rate of development of lung, breast and skin cancer, Szmigielski et al. (1982), showing the strong co-promotional effects of microwave exposure. Prausnitz and Susskind (1962) found increased in testicular degeneration and increases in leukaemia at Rate Ratios and mean weekly
exposure levels which are compatible with the North Sydney Study. Cell phone radiation, Repacholi et al. (1997) enhances B-cell tumours in genetically engineered mice. These are consistent with the research summarized above on the direct mutagenic affects of RF/MW radiation and the research showing alteration of signal transduction, cell communication which influence the cellular level growth regulation and can lead to cell proliferation and thence to tumour formation and cancer.

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

16. Reproductive effects and Teratology:

16.1 Introduction:

The cellular level changes discussed and documented above are pertinent also to the consideration of potential or actual effects of RF/MW radiation on the development of human embryos, miscarriage and adverse birth outcomes.

Altered signal transduction and gap-junction communication, or DNA breakage and Chromosome Aberration in the developing human embryo is potentially damaging or fatal. Brent et al. (1993) record that 20 to 25 % of human birth defects are caused by genetic factors. Laboratory studies using mice and other animals have typically employed exposure levels in the range 10 to 100 mW/cm², or even higher, in the belief that the higher the dose the more likely the change of detecting a result.

The cellular processes discussed above show the fallacy of this, as does the calcium ion windowing effects, which have been monitored to change in association with environmental exposure levels of less than 10 μW/cm². When moderate to high exposure levels are used great care must be taken to discern between thermal effects and non-thermal effects. Real non-thermal effects can be masked by large thermal effects. The following are a sample of laboratory experiments involving mice and chickens exposed to microwaves.

16.2 Animal Studies:

Chazan et al. (1983) investigated the development of murine embryos and fetuses after irradiation with 2450 MHz microwaves. They found indications of retardation of development in the early period of gestation in mice exposed to thermal MW fields. In mice exposed to microwaves at 40 mW/cm² during the second half of pregnancy increased 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) found reduced weight in mice offspring after in utero exposure to 2450-MHz (CW) microwaves. They were exposed to 28 mW/cm² for 100 minutes daily from the 6th through 17th day of gestation. The offspring were examined either as fetuses after hysterotomy on the 18th day of gestation or as naturally born neonates on the 1st and 7th day of age. Fetuses of half of the dams were examined on the 18th day of gestation. The incidence of pregnancy and the numbers of live, dead, resorbed, and total fetuses were similar in both groups.
The mean weight was significantly lower (10%) in live microwave-irradiated fetuses, and ossification of sternal centers was significantly delayed. In the offspring that were born naturally, the mean weight of microwave-irradiated 7-day-old suckling mice was significantly lower (10%) than that of the sham-irradiated group. Survival rates of neonates in these two groups were not different. These data demonstrate that the decreased fetal weight seen in microwave-irradiated mice 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.

Prausnitz and Susskind (1962) were not studying reproductive effects, but atrophy of the testes would have severe effects on any sperm which survived. Such sperm are unlikely to have undamaged DNA. Their exposure regime was 100 \( \mu \text{W/cm}^2 \) for 4.5 mins/day, averaging 0.22 \( \mu \text{W/cm}^2 \) /week.

16.3 Summary and conclusions about teratological animal studies:

The in utero developing embryo is very vulnerable to damage from toxins. At critical times damage to certain organs occurs. With sufficient foetal or placenta damage a spontaneous abortion is initiated. At other 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 \( \mu \text{W/cm}^2 \) over a week.

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

17. Epidemiological Studies:

Now that we have established that there are non-thermal or athermal biological changes and observed changes in exposed animals which are consistent with possible or probable
adverse health effects, we review the epidemiological studies which find statistically significant adverse health effects in human populations, both occupational and residential.

The use and mis-use of epidemiological studies in radiation standards setting is discussed in Goldsmith (1991). He first notes that there is a tendency to use experimental studies in preference to findings from epidemiological studies. “Yet the epidemiological studies are usually the first and at time the only source of data on such critical effects such as cancer, reproductive failure and chronic cardiac and cardiovascular disease in exposed humans.” A public health protection to the use of epidemiological studies is well covered by Bradford-Hill, section XX above.

Evidence published since 1990 and 1993 can extend the reviews of the U.S.E.P.A. and the W.H.O. review team. Several very recent public health studies include the North Sydney TV tower study of Hocking et al. (1996), the set of papers on the Skrunda Radar in Latvia, the analysis of the effects of the short-wave tower in Schwarzenburg, Switzerland, Altpeter et al. (1995), the U.K. study of Dolk et al. (1997a, 1997b), the brain tumour study of Grayson (1996), the mouse study of Repacholi et al (1997), and the Chinese Study of Chiang et al. (1989). These studies all have exposure measurements or calculations associated with them, which increases their power. The study by Dolk et al. (1997a, 1997b) adds confirmation to the Hocking et al. (1996) study. All of these studies show increased risk of adverse effects, on health, well-being and the environment, at mean exposure levels at a fraction of the Australian/New Zealand Standard AS 2772 (NZS 6609) and well below 2μW/cm².

17.1 Brief Overview of Epidemiology and RF/MW association with health effects:

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

The following give a brief summary of some of the published studies showing adverse effects from RF/MW effects on people.

- More neurasthenic symptoms (chronic mental and physical weakness and fatigue) in group exposed to radar (Djordevic et al., 1979).

- A major study of radar and radio exposed U.S. Navy personnel, summarized as having no reported effects, includes data which shows statistically significant increases cancer between a group assessed as high exposure compared to a group assessed as low exposure, e.g. All death (RR=1.79 (1.52-2.12)), Accidental Death (RR=2.20 (1.72-2.82)), All Diseases (RR=1.55 (1.19-2.01)), Malignant tumours (RR=1.66 (1.06-2.60)), and Lymphatic and Hematopoietic cancer (RR=2.66 (1.02-4.81)). There was also statistically increased risks of a host of illness including, Musculoskeletal, Organs of Sense, Systematic conditions, Respiratory, Cardiovascular and digestive illness, Skin, Endocrine, Neurological and Mental conditions, Robinette et al. (1980).
- Higher frequency of polycythemia (increase in red blood cells) with microwave exposure (Friedman, 1981).

- Swedish physiotherapists who gave birth to a deformed child or who had perinatal death had higher recorder RF/MW diathermy exposures, Kallen et al. (1982).

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

- Lester and Moore (1982b) found significantly higher cancer rates in U.S. counties with Air Force bases compared to those without Air Force bases, which they related to prolonged environmental exposure to RF/MW from radar.

- Association between heart disease and work with shortwave therapies, increasing with the number of treatments/week (Physiotherapists using 27 MHz diathermy) (Hamburger et al., 1983).

- Polson and Merritt (1985) criticized the analysis of Lester and Moore (1982b), pointing out weaknesses in their use of the data, such as a city could be in a country with no Air Force Base but be closer to a base in another country than a city in that country. Having made corrections for this, Lester and Moore (1985) found strengthened associations between cities and air force bases, with higher incidences of cancer related to radar transmissions.

- Lin et al. (1985) studied 951 cases of brain tumors among white male residents of Maryland during the period 1969-1982. Fifty cases of glioma and astrocytoma were observed among electrical workers exposed to EMR compared to an expected number of 18, i.e. an risk ratio of 2.8. While their exposure was mainly to ELF fields it shows the common link over a wide range of frequencies.

- Increased risk of leukaemia amongst amateur radio operators (Milham, 1985).

- In 1985 an unusual number of children with leukaemia were identified living in the vicinity of broadcasting facilities (OR = 3.4: CI=0.70 -16.41), Maskarinec et al. (1993).

- Upper limb paraesthesia and eye irritation among 30 exposed workers using 27 MHz plastic sealers (Bini et al., 1986);

- De Guire et al. (1987) report increased malignant melanoma of the skin in workers in a telecommunication industry, affecting only men, SIR = 2.7 CI : 1.31-5.02).

- Thomas et al. (1987) report a 10-fold increase in astrocytic brain tumor among electronics and repair workers employed for 20 years or more. Some risk was due to solvents, put at a factor of 2, placing RF/MW contribution at a factor of 5.

- Increased rates of paraesthesia in hands, neurasthenia and eye complaints, using 27 MHz plastic welders and sewing machines (Kolmodin-Hedman et al., 1988).
Milham (1988) studied 67,829 amateur radio operators in Washington State and California. He concludes "The all-cause standardized mortality ratio (SMR) was 71 but a statistically significant increased mortality was seen for cancers of the other lymphatic tissues (SMR = 162), a rubric which includes multiple myeloma and non-Hodgkin's lymphomas. The all leukemia SMR was slightly but not significantly elevated (SMR = 124). However, mortality due to acute myeloid leukemia was significantly elevated (SMR = 176).

A doubling of miscarriage rates has been reported in women working at computer terminals for more than 20 hours/week in the first three months of pregnancy (Goldhaber et al. 1988). Note that VDU's emit a wide range of RF radiation.

Szmigielski et al. (1988) studied polish military personnel exposed to microwave radiation and reported that cancer morbidity was three times higher in the exposed group than the control group.

Duration and severity of tonsillitis increased with combined air pollution and RF exposure (Shandala and Zvinjatskovsky, 1988).

Electrical workers in Los Angeles county have a 4.3-fold increased risk of certain brain tumors (Preston-Martin et al. 1989).

An increased incidence of malignant brain tumors has been reported in children of fathers exposed to electromagnetic fields and electronic solvents (Johnson and Spitz, 1989).

Increased protein band in CSF in exposed group or radar mechanics (Nilsson et al., 1989).

Hayes et al.(1990) report an Odds ratio for all testicular cancer of 3.1 (CI: 1.4-6.9) for a small sample of workers who were occupationally exposed to RF/MW radiation.

U.S. Navy electrician's mates have an excess risk of leukaemia, RR=2.4 (1.0-5.0), Garland et al. (1990)

W Shao-Guang, et al. (1990) reports a Chinese study which found a significant increase in neurasthenic syndrome backed up by blood biochemical changes.

Savitz and Chen (1990) show significant increased risk of childhood cancer (Neuroblastoma (OR=11.8*), Brain Tumour (OR=2.7*) and CNS tumors (OR=1.7)) associated with parents who work in electrical and electronic industries.

Danish physiotherapists working with shortwave diathermy, who were "highly exposed" to RF, only 17% of newborn infants were boys, and exposure was associated with stillbirth/death within a year, prematurity and low birth weight, Larsen et al. (1991).

Increased risk for all brain tumours (RR=2.9 (1.2-5.9)) and glioblastomas (RR=3.4 (1.1-8.0)) for assemblers, and repairmen in the radio and TV industry, Tornqvist et al. (1991)
Microwave heating reduces immune system factors in human breast milk, compared to conventional heating. Microwave heating significantly reduces the IgA for E coli bacteria, producing five times more E coli for 25°C heating and 18 times more after 3 hours for 98°C heating, Quan et al. (1992).

United States physical therapists (Ouellet-Hellstrom and Stewart (1993)) show a 1.59-fold increase in miscarriage in the first 7 weeks of gestation when using microwave diathermy, and a dose-response relationship with increased treatments/month.

Goldsmith (1995) reports an up-dated analysis of the US embassy in Moscow which does show a significant elevated risk of a wide range of adult cancers, and including childhood leukaemia, after years of microwave irradiation, exposed to average levels of radar produced microwaves of long-term average indoor exposure of 0.2 to 0.5 µW/cm², daily peaks between 5 and 18 µW/cm² on the outside walls.

Increased risk of female breast cancer with exposure to radiofrequency EMF, RR=1.15 (1.1-1.2), Cantor et al. (1995).

Altpeter et al (1995) studied populations living near and further away from a shortwave transmitter in Schwarzenburg, Switzerland. The statistically elevated symptoms in the high and medium exposure groups, compared to the low exposure group, include Nervosity and restlessness, Disturbances in falling asleep and difficulty in maintaining sleep, Joint pains, Psychovisual Index changes, Disturbances of Concentration, General Weakness and Tiredness, Constipation, Diarrhea and Lower back pain, all significant at p<0.02 except the first for which p=0.034 which is less than the usual significance level of p<0.05. Children’s advancement from primary school to secondary school was significantly slower in the exposed group. They conclude that even though the association is weakened by a small sample size, an adverse effect from the transmitter “cannot be excluded”.

An increased exposure from 1 mA/m to 10 mA/m (0.038µW/cm² to 3.8µW/cm²) had an Odds Ratio for insomnia of 1.13 (CI: 1.04-1.23) and from 0.1 mA/m to 1 mA/m (0.0038µW/cm² to 0.038µW/cm²), OR=2.1 (CI: 0.95-4.57). Table 7 presents the adjusted Odds Ratios for the primary effects found, which show significant dose response relationships and a highly statistically significant increase with mean exposure increase.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>OR</th>
<th>95% Conf. Interval</th>
</tr>
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<tbody>
<tr>
<td>Nervosity</td>
<td>2.77</td>
<td>1.62-4.74</td>
</tr>
<tr>
<td>Diff. in falling asleep</td>
<td>3.35</td>
<td>1.86-6.03</td>
</tr>
<tr>
<td>Diff. in maintaining sleep</td>
<td>3.19</td>
<td>1.84-5.52</td>
</tr>
<tr>
<td>Joint Pain</td>
<td>2.46</td>
<td>1.37-4.43</td>
</tr>
<tr>
<td>Limb Pain</td>
<td>2.51</td>
<td>1.15-5.50</td>
</tr>
<tr>
<td>Cough and Sputum</td>
<td>2.80</td>
<td>1.18-6.64</td>
</tr>
</tbody>
</table>
Hocking and Gordon (1996) found a 2.74-fold increase in childhood leukaemia death within 4 km of TV and FM radio transmission masts in North Sydney between 1972 and 1990. Mean exposures were measured in the range 0.04 to 0.4 $\mu$W/cm$^2$.

Polish Military personnel (1971-85) exposed to above average radar and radio sourced RF/MW show large increases in leukaemia (Lymphoma: RR=5.8 (2.11-9.74); Chronic lymphocytic: RR=3.7 (1.45-5.18); Acute Lymphoblastic: RR=5.8 (1.22-18.16); Chronic myelocytic: RR=13.9 (6.72-22.12); Acute myeloblastic: RR=8.6 (3.54-13.67) and Total: RR=6.31 (3.12-14.32). Also show statistically significant associations for cancer of the esophagus and stomach, colorectal, skin (including melanomas), CNS and brain. (Szmigielski, 1996)

U.S. Air Force personnel showed increased incidence of brain tumour with exposure to ELF (RR=1.28 (0.95-1.74)), and RF/MW (RR=1.39 (1.01-1.90)).

The Skrunda Radar provides a living laboratory for the chronic low level effects of exposure to RF/MW radiation. To date investigations have revealed a number of statistically significant changes associated with exposure to the radar signal. These include:

- Impaired scholastic performance of children in the open field exposure range of 0.0008-0.41 $\mu$W/cm$^2$, mean measured level in the range 0.0028-0.039$\mu$W/cm$^2$.

- A 6-fold increase in broken chromosomes in the peripheral erythrocytes of the exposed cows (p<0.01). for a measured exposure would be in the range 0.042 to 6.6$\mu$W/cm$^2$, mean exposures in the range 0.157 to 0.63$\mu$W/cm$^2$.

- A statistically significant (P<0.01) negative correlation between the relative additional increment in tree growth and the intensity of the electric field. The Pine trees at 4 km were exposed to a range of 0.011 to 0.41$\mu$W/cm$^2$, a mean open field exposure of 0.039$\mu$W/cm$^2$ and measured distance exposure of 0.0027$\mu$W/cm$^2$ (for the radar signal). A probable biological mechanism was identified through observed changes in physiological conditions.

- Chromosome and reproductive damage in plants exposed RF/MW in the range 0.042 to 6.6$\mu$W/cm$^2$.

- Chronic exposure to pulsed RF radar signals is associated with chromosome damage in plants and animals, with associated reproductive aberration in plants, and growth reduction in pine trees linked to observed physiological changes, and scholastic impairment of school children occurs in relation to exposure levels which fall well below 2$\mu$W/cm$^2$, below 0.1$\mu$W/cm$^2$. and even below 0.01$\mu$W/cm$^2$.

Dolk et al. (1997 a, b) found small but significant increases in adult leukaemia, which decreases with distance from the transmitter, associated with 21 FM and TV transmission towers in the United Kingdom. This is a strong dose-response result.

Study after study shows cancer and other health problems associated with RF/MW exposure. This shows why independent scientists continually find it disturbing and unprofessional when government and international reviews, such as that carried out for
the WHO, Repacholi (1993) publish conclusions which are weak or even misleading, by stating that there is "**no clear evidence of detrimental effect**". This is strongly at odds with the data presented.

When asked whether epidemiological evidence on the adverse health effects of RF/MW could be only described as a "weak link", Professor Goldsmith replied (p137, line 36)

"I disagree. I think when children die of cancer between 5 and 18 $\mu$W/cm$^2$ over a period of time - exposure is not weak. It is significant."

Here Professor Goldsmith is referring to the children of the staff of the U.S. Embassy in Moscow and other Eastern European embassies and the range of peak exposure levels on the outside walls of the United States Embassy in Moscow. The daily mean external wall exposure was in the range 1.0 to 2.4$\mu$W/cm$^2$, internal exposure being less than 0.2 to 0.5$\mu$W/cm$^2$.

17.2 Occupational Studies:

17.2.1 U.S. Physiotherapists Spontaneous Miscarriage Study:

17.2.1.1 The Study:

Ouellet-Hellstrom and Stewart (1993) carried out the largest study of physiotherapists in relation to early (first trimester) spontaneous miscarriage associated with exposure to shortwave and microwave leakage fields from diathermy equipment. In a sample of 11,598 pregnant physiotherapists in the U.S., 6684 reported having ever used microwave or shortwave diathermy, 1791 of whom had experienced early spontaneous miscarriage. From these a case group (miscarriage) was selected for a microwave exposed group and a shortwave exposed group.

<table>
<thead>
<tr>
<th>Table 15: Unconditional odds ratios for the association between risk of recognised miscarriage and reported exposure to microwave diathermy during the 6 months prior to and the first trimester of pregnancy: Physical Therapists Study, 1989-1990.</th>
</tr>
</thead>
<tbody>
<tr>
<td>**No. of exposures</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>All pregnancies</td>
</tr>
<tr>
<td>&lt;5</td>
</tr>
<tr>
<td>5-20</td>
</tr>
<tr>
<td>&gt;20</td>
</tr>
<tr>
<td>p&gt;0.005</td>
</tr>
<tr>
<td>Total no.</td>
</tr>
<tr>
<td>No prior fetal loss</td>
</tr>
<tr>
<td>&lt;5</td>
</tr>
<tr>
<td>5-20</td>
</tr>
</tbody>
</table>
17.2.1.2 Types of Biological Mechanisms:

Two possible types of biological mechanisms have been proposed to explain this associated effect of microwaves and early spontaneous miscarriage. The first, favoured by the National Radiation Laboratory (NZ) and the Australian Radiation Laboratory staff, is a thermal mechanism, such as the production of heat lesions in the placenta and/or foetus, causing damage and subsequent miscarriage. This requires a significant temperature rise in the affected tissue. The second involves cumulative mutational DNA and aberrated chromosomes, probably involving enhanced free radicals. This mechanism is suggested by observations of aberrated chromosomes in blood analysis of people exposed to microwaves from radar, Garaj-Vrhovac and Fucic (1993).

17.2.1.3 The Heating Issue:
The key to resolving this is the ability of the known exposure regime to produce significant foetal heating or not. The highest exposure at 15 cm from the pads is 15 mW/cm², while the top of the usual range is 1,200 µW/cm². Hocking and Joyner (1995) in their criticism of the paper, suggest that the result is implausible because shortwaves penetrate the foetus much more easily than do microwaves. Hocking and Joyner use a model developed by Telecom Australia to show that for a frontal exposure of 1000 µW/cm², the maximum SAR in the uterus is 0.209 W/kg for 27.12 MHz, 0.023 W/kg for 915 MHz and 0.000027 W/kg for 2,450 MHz. For the maximum conceivable exposure (15 mW/cm²), the 915 MHz and 2,450 MHz SARs would be 0.345 W/kg and 0.00041 W/kg, respectively.

Ouellet-Hellstrom and Stewart (1995) in reply to Hocking and Joyner's comments state:

“**We disagree with the general scientific view of Hocking and Joyner as expressed in their closing statement. In general, one should examine the extent to which competing explanations are supported by the data, not whether the data is supported by the explanations. The data are fixed, but the explanations are not.”**

For normal blood flow the temperature rise rate at 4 W/kg is about 0.02 °C/min at 20 °C and 50 % RH, Adair (1993). This gives a 1 °C rise after 50 minutes. At 0.345 W/kg the heating rate will be about 0.0017°C/min. Hence a 2 to 5 minute exposure at the maximum conceivable rate would result in a foetal temperature rise of 0.004 to 0.009 °C. This is far too low to cause thermal lesions and therefore rules this out as a possible mechanism.

**17.2.1.4 Biologically Plausible Mechanism:**

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

“**The causes of spontaneous abortion can be divided into two main categories: those arising from chromosomal anomalies and those arising from abnormalities in the intrauterine environment. In the following communication, we propose that deficient pineal melatonin functions in early pregnancy may be causally related to the development of spontaneous abortions in cases where chromosomal anomalies or structural abnormalities of the uterus have been excluded.”**

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

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

Thus, it is appropriate to estimate the risk of spontaneous miscarriage in terms of monthly mean exposure since the dose-response relationship is expressed in terms of treatments
per month. A significant occupational exposure will only occur then, if many treatments are given and the operator stands very close to the equipment for prolonged periods. Assuming a conservatively long estimate of 2 minutes exposure per treatment, the dosage per treatment is 0.01 to 0.144 J/cm².

17.2.1.5 Microwave dose associated with the risk:

One treatment per month is in the range 0.004 to 0.056 μW/cm², mean 0.03 μW/cm²; 10 per month 0.04 to 0.56 μW/cm², mean 0.3 μW/cm²; and 20 treatments per month 0.08 to 1.11 μW/cm², mean 0.6 μW/cm². The lowest limit is very difficult to estimate with reliability but the mean level of the middle band is 0.3 μW/cm². This suggests that a 20 to 50% increase in miscarriage occurred with a mean monthly microwave exposure of somewhat less than 0.5 μW/cm².

17.2.1.6 Relevance to mobile phone base stations:

The fact that this level of microwave exposure is found near base stations and that there are currently no documented reports of increased incidence of miscarriage occurring near cell sites is not surprising nor a proof that the hypothesis advanced here is wrong. It simply results from the fact that nobody reports or records miscarriage. Several other factors exists. Miscarriage is not reported and no statistics are being collected. Each pregnant woman can only miscarry once per child, with a several month wait until the next pregnancy. Each spontaneous miscarriage is isolated and does not form a pattern. Many causes are possible. Very few miscarriages are investigated, unless it becomes an issue from a cluster pattern and then a medical or environmental cause is sought. Few pregnant people live near mobile phone base stations. However with the unrestricted siting policy advocated by the companies and accepted by almost all councils, this is changing significantly month by month.

Increased incidence of miscarriage is potentially occurring right now and until it is scientifically assessed, we will not be able to rule out the scientifically indicated probability. It remains a potential adverse effect under the definitions of the Resource Management Act 1991. The studies presented here give ample grounds for requiring the siting of cell sites far enough away from residences to avoid an increase in risk through sections 5 and 3 of the Act.

A statistically significant 50% increase in miscarriage risk was identified with 5 to 20 treatments per month. Taking the typical number in this range as 10 treatments per month the mean exposure is in the range 0.04 to 0.56 μW/cm². A public exposure limit of 0.1 μW/cm² should be adopted until the effect was conclusively show to occur at or below this level, or not at all.

17.2.2 Korean War U.S. Navy Study:

Robinette et al. (1980) was quoted by Dr Michael Repacholi in evidence for BellSouth, following the conclusions stated in the original paper, as showing no effects from exposure to radio and RF/MW radiation. This comes from taking the authors’ conclusions and not looking at the data itself. Doubt was thrown on these conclusions at the hearing by Professor of Epidemiology, Dr John Goldsmith on the basis of published comments from Dr Charlotte Silverman, a leading epidemiologist and co-author of the original paper when she said in relation to the results of this study, Silverman (1979), “while some
significant differences among the occupational groups classified by level of potential exposure have been found with respect to all end points studied, the differences could not be interpreted as a direct result of microwave exposure.” Professor Goldsmith was clearly stating that Dr Silverman had concluded that significant differences has been found in all studied end points, and that this was at variance with the conclusions of the 1980 paper.

Robinette et al. (1980) acknowledge the strong possibility of mis-classification of exposure hazard. They carried out a survey of the assumed high exposure groups and a hazard number was allocated to each. Electronics Technicians (ET) were 1620, Fire Control Technicians (FT) were 2870 and Aviation Electronics Technicians were 3700. Hence a low versus high exposure comparison can be made taking the incidence ratio (Risk Ratio) between ET and AT, Table 16.

This analysis is much more consistent with Dr Silverman’s conclusions reported in the 1979 conference paper, Silverman (1979). It is also consistent with the substance of the material in the original paper.

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

In that the original data shows a significant increase in mortality risk 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.”

Adverse effects are even more clear with a consideration of the morbidity data derived from men receiving VA compensation for treatment, Table 12 from Robinette et al. (1980).

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.

Morbidity effects were investigated using VA compensation claims. The average exposure of the FT+AT group is 3286. Table 10 is extracted from the data in Table 12 in Robinette et al. (1980). Table 17 shows that sickness was considerably higher amongst the highly exposed group compared with the low exposure (ET) group. The following have Risk Ratios elevated by 30% or more: Musculoskeletal (RR = 1.93), Organs of special sense (RR = 1.62), Systematic conditions (RR = 3.5), Respiratory (RR = 1.74), Cardiovascular (RR = 2.03), Digestive (RR = 1.37), Skin (RR = 1.30), Endocrine (RR = 1.45), Neurological (RR = 1.44), and Mental Conditions (RR = 1.67).

### Table 17: 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.

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

Note that the Risk Ratios in Table 6 are smaller than they would be if AT was compared with ET, and the “high exposure” group was not diluted by including FT.

A part of the Respiratory disease and Cardiovascular Disease increase could be attributable to increased incidence of smoking. However these do not account for all of the increase in these diseases, nor of the wide range of disease increase detected.

Note also that the reference group, ET, have an elevated Hazard Number compared to other servicemen and considerably elevated compared to the general public. Hence the Risk Ratios are quite large underestimates of the effect of increased chronic exposure to microwaves emitted by radar.

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.
Since all of the subjects are acknowledged to have some radar exposure on a regular basis, such as when they are on deck, it is relevant to compare the incidence of mortality of the servicemen with a large group of unexposed men. Cancer mortality statistics are available for New Zealand men. Since rates of cancer death rise with age a well defined age cohort is necessary.

As of 1952, 88.7% of the studied service men were 25 or younger. Mortality analysis covered the period to 1974 making around 89% being 47 years old or less. 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 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.

17.2.3 Polish Military Study:

The Polish Military Study, Szmigielski et al. (1988) reported significant health effects from chronic RF/MW exposure but did not have exposure estimates associated with it. Analysis of the exposure regime described allows an annual mean career range of exposure to be estimated with a degree of reliability because of the hygiene regime which was in place and measurements which were made. Another 5 years of data has now been added and reported, Szmigielski (1996).

17.2.3.1 Background:

Szmigielski et al. (1988) carried out an extensive retrospective study of Polish Military personnel with radar exposure, covering a longer period to allow for the latency of cancers. This was updated to extend the period involving cancer morbidity to between 1971 and 1985, Szmigielski (1996). The mean annual population was 128,000 with around 3700 (3%) being considered to occupationally exposed to RF/MW. In this group statistically significant increases in many forms of cancer were detected, consistent with the Moscow study below. No analysis of the exposure regime was presented to the Planning Tribunal. While it was impossible to assign an exposure to each individual, the exposure regime was extensively studied and it is possible to make an estimate of the likely career mean range of exposure for those with very high exposures, setting the likely maximum exposure range for the effects identified.

17.2.3.2 Exposure Assessment:

Szmigielski (1996) states that the exposure regime was considerably more uniform than most exposures because of exposure hygiene controls and reporting of high exposures. Szmigielski (1988) describes the daily exposures as 4-8 hours below 200\(\mu\)W/cm\(^2\) with several minutes in the range 200-1000\(\mu\)W/cm\(^2\). Incidents of short-lasting exposures estimated up to 10-20 mW/cm\(^2\) were reported but were more frequent before 1960 when the hygiene controls were introduced.

Exposures were extensively measured by military safety groups, with the finding that 80%-85% of the posts were in the 10-200\(\mu\)W/cm\(^2\) range, and 15 % in the 200-600\(\mu\)W/cm\(^2\) range, and where EM fields mostly pulse-modulated RF/MWs at 150 to 3500 MHz. Safety rules limiting exposure were established in 1961 and are outlined in Table 18.
A highly exposed person would spend most of the working day in Zone 2, a small number of hours in Zone 3 and a few minutes in Zone 4 at most, since a person in a highly exposed occupation was generally required to follow hygiene principles set out in the Standard. About 16 hour/working day would be spent away from the occupational exposure zone. Hence the maximum exposure regime is suggested in Table 18.

Because the exposure distribution is skewed, the mean and median exposures are closer to the lower bound of the range. From Tell and Mantiply (1980), the distribution of population exposures in major US cities, the mean is 0.01μW/cm² and the 15 percentile is 0.069μW/cm². Hence the ratio of the upper 15 percentile to the mean is 0.153. Applying this factor to the upper limit of each exposure class gives an approximate estimate of the mean exposure.

### Table 18: Polish Occupational exposure standards (Czerski (1985)) for RF/MW exposure, 300 MHz - 300 GHz. Exposure in μW/cm²

<table>
<thead>
<tr>
<th>Zone</th>
<th>Stationary Antennae</th>
<th>Rotating</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Safe, Human occupancy unrestricted</td>
<td>&lt; 10</td>
<td>&lt; 100</td>
</tr>
<tr>
<td>2) Intermediate, access limited to authorized personnel, occupational exposure permissible during work shift.</td>
<td>10 - 200</td>
<td>100 - 1000</td>
</tr>
<tr>
<td>3) Hazardous, access limited to authorized personnel, duration of exposure (t in hrs) defined by the formula in parentheses.</td>
<td>200 - 10,000</td>
<td>1,000 -</td>
</tr>
<tr>
<td>10,000 (p in W/m²: 1 W/m² = 100μW/cm²)</td>
<td>(t=32/p²)</td>
<td>(t=800/p²)</td>
</tr>
<tr>
<td>4) Danger Zone, human occupancy prohibited.</td>
<td>&gt; 10,000</td>
<td>&gt; 10,000</td>
</tr>
</tbody>
</table>

### Table 19: Estimated maximum average daily career exposure scenario for a very highly exposed serviceman.

<table>
<thead>
<tr>
<th>Zone classification and safety limits</th>
<th>Daily Time</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Danger Zone (&gt;10,000μW/cm², average 15,000μW/cm²)</td>
<td>1 minutes</td>
<td>37.4</td>
</tr>
<tr>
<td>Hazard Zone (200-10,000 μW/cm², average 2750 μW/cm²)</td>
<td>5 minutes</td>
<td>34.4</td>
</tr>
<tr>
<td>Intermediate Zone (10-200 μW/cm², average 30.6 μW/cm²)</td>
<td>6 hours</td>
<td>27.5</td>
</tr>
<tr>
<td>Safety Zone (&lt;10 μW/cm², average 1.5 μW/cm²)</td>
<td>1.9 hours</td>
<td>0.4</td>
</tr>
<tr>
<td>Residential (&lt;1 μW/cm², average 0.153 μW/cm²)</td>
<td>16 hours</td>
<td>0.4</td>
</tr>
</tbody>
</table>

The regime in Table 19 gives a high exposure workday mean of 27.8μW/cm², and a working week average of 19.9 μW/cm², and an annual average (assuming 46 working
weeks) of 17.6 $\mu$W/cm$^2$. This is 1760 times higher than the mean U.S. urban exposure of 0.01 $\mu$W/cm$^2$. The residential exposure in Table 12 (0.153 $\mu$W/cm$^2$) is over 15 times higher than the U.S. urban mean. This is appropriate because most servicemen live on or near the military base and hence are exposed to radar signals. Lester and Moore (1985) found increased cancer rates in cities adjacent to air force bases. It is unlikely that a serviceman would be in this highly exposed regime for all of their career and so long-term mean maximum exposures are likely to be less than this, say 40-80%. Using the extremes of each of these gives the range 7-14 $\mu$W/cm$^2$ for the career maximum average exposure for Polish servicemen.

17.2.3.3 Summary of Health effects:

Prof. Szmigielski has published the health effects data in two parts, Szmigielski (1988) and Szmigielski (1996), the first covering the period 1971 to 1980 and the second 1971-1985. Szmigielski (1988) reached the following conclusions:

"In summary, from a retrospective study that covered a large, well controlled population with a known population of subjects, and that has a relatively long period of observation (1971-1980) the following conclusions can be drawn:

- The risk of developing clinically detectable neoplastic disease was about 3 times higher for the personnel exposed occupationally to MW/RF radiation. The higher risk appeared for malignancies originating from hemato-lymphatic systems (morbidity about 7 times higher). Other more frequent neoplasms were located in the alimentary tract and in skin (including melanomas).

- The highest risk factor of cancer morbidity related to occupational exposure to MW/RFs appeared for subjects at the age of 40-49 who had a 5-15 year period of exposure.

- Morbidity rates of neoplasms in personnel exposed occupationally to MW/RFs showed a strong correlation with the period of exposure.

- Neoplasms (cancer tumors) of the same localization and/or type developed earlier (by about 10 years) in personnel exposed occupationally to MW/RFs than in those not working in the MW/RF environment.

The extension to 15 years of data was reported in Szmigielski (1996). The results are in Tables 20,21 and 22.

<table>
<thead>
<tr>
<th>Type of malignancy</th>
<th>Incidence</th>
<th>Incidence</th>
<th>RR</th>
<th>95% Confid.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Significance</td>
<td>Non-exposed</td>
<td>Exposed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hodgkin's disease</td>
<td>1.73</td>
<td>5.12</td>
<td>2.96</td>
<td>1.32 - 4.37</td>
</tr>
<tr>
<td>Lymphoma (non-Hodgkin and lymphosarcoma)</td>
<td>1.82</td>
<td>10.65</td>
<td>5.82</td>
<td>2.11 - 9.74</td>
</tr>
<tr>
<td>Chronic lymphacytic leukaemia</td>
<td>1.37</td>
<td>5.04</td>
<td>3.68</td>
<td>1.45 - 5.18</td>
</tr>
<tr>
<td>Acute lymphoblastic leukaemia</td>
<td>0.32</td>
<td>1.84</td>
<td>5.75</td>
<td>1.22 - 18.16</td>
</tr>
<tr>
<td>Chronic myelocytic leukaemia</td>
<td>0.88</td>
<td>12.23</td>
<td>13.90</td>
<td>6.72 - 22.12</td>
</tr>
<tr>
<td>Acute myeloblastic leukaemia</td>
<td>0.71</td>
<td>6.12</td>
<td>8.62</td>
<td>3.54 - 13.67</td>
</tr>
<tr>
<td>Total</td>
<td>6.83</td>
<td>43.12</td>
<td>6.31</td>
<td>3.12 - 14.32</td>
</tr>
</tbody>
</table>

Table 21: Incidence of neoplasms (tumors) (per 100,000 subjects annually) in age groups of military personnel exposed and non-exposed (control) to radiofrequency and microwave radiation, Szmigielski (1996).

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Incidence Non-exposed</th>
<th>Incidence Exposed</th>
<th>OR</th>
<th>95 % Confidence limits</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>11.62</td>
<td>21.11</td>
<td>2.33</td>
<td>1.23 - 3.12</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>30-39</td>
<td>18.37</td>
<td>42.28</td>
<td>2.30</td>
<td>1.04 - 3.06</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>40-49</td>
<td>84.29</td>
<td>161.62</td>
<td>1.92</td>
<td>0.98 - 2.84</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>50-59</td>
<td>186.71</td>
<td>274.13</td>
<td>1.47</td>
<td>0.92 - 2.21</td>
<td>N.S.</td>
</tr>
<tr>
<td>All Ages</td>
<td>57.60</td>
<td>119.12</td>
<td>2.07</td>
<td>1.12 - 3.58</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Haemopoietic/lymphatic malignancies

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Incidence Non-exposed</th>
<th>Incidence Exposed</th>
<th>OR</th>
<th>95 % Confidence limits</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>2.12</td>
<td>17.30</td>
<td>8.16</td>
<td>3.11 - 22.64</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>30-39</td>
<td>3.08</td>
<td>26.43</td>
<td>8.58</td>
<td>3.46 - 19.58</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>40-49</td>
<td>8.32</td>
<td>73.25</td>
<td>8.80</td>
<td>4.13 - 15.27</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>50-59</td>
<td>24.13</td>
<td>108.62</td>
<td>4.47</td>
<td>2.56 - 6.81</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>All ages</td>
<td>6.83</td>
<td>43.12</td>
<td>6.31</td>
<td>3.12 - 14.13</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 22: Incidence of neoplasms (per 100,000 subjects annually) in military personnel exposed and non-exposed (control) to radiofrequency and microwave radiation, Szmigielski (1996).

<table>
<thead>
<tr>
<th>Localization of malignancies</th>
<th>Incidence (Expected)</th>
<th>Incidence (Exposed)</th>
<th>Risk Ratio</th>
<th>95% CI limits</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharynx</td>
<td>1.96</td>
<td>2.12</td>
<td>1.08</td>
<td>0.82-1.24</td>
<td>N.S.</td>
</tr>
<tr>
<td>Esophageal and stomach</td>
<td>4.83</td>
<td>15.64</td>
<td>3.24</td>
<td>1.85-5.06</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Colorectal</td>
<td>3.96</td>
<td>12.65</td>
<td>3.19</td>
<td>1.54-6.18</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Liver, pancreas</td>
<td>2.43</td>
<td>3.58</td>
<td>1.47</td>
<td>0.76-3.02</td>
<td>N.S.</td>
</tr>
<tr>
<td>Laryngeal, lung</td>
<td>21.89</td>
<td>23.26</td>
<td>1.06</td>
<td>0.72-1.56</td>
<td>N.S.</td>
</tr>
<tr>
<td>Skin, including melanomas</td>
<td>3.28</td>
<td>5.46</td>
<td>1.67</td>
<td>0.92-4.13</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Nervous system including brain tumour</td>
<td>2.28</td>
<td>4.36</td>
<td>1.91</td>
<td>1.08-3.47</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Thyroid</td>
<td>1.38</td>
<td>2.12</td>
<td>1.54</td>
<td>0.82-2.59</td>
<td>N.S.</td>
</tr>
<tr>
<td>Haematopoietic system and lymphatic organs</td>
<td>6.83</td>
<td>43.12</td>
<td>6.31</td>
<td>3.12-14.32</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>All malignancies</td>
<td>57.60</td>
<td>119.12</td>
<td>2.07</td>
<td>1.12-3.58</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>
Hence the Polish Military study gives conclusive and strong associations of RF/MW exposure and increases in a host of cancers and a large increase in cancer death. Table 20 shows the incidence ratios (Odds Ratio: OR) for the ratio of exposed to non-exposed personnel, after Szmigielski (1996).

The analysis here identifies the likely highly exposed regime as being between 7 and 14μW/cm². Professor Szmigielski forms the following conclusions concerning cancer risk from this study, Szmigielski (1996):

“The main results obtained in the present study were a doubled incidence of all neoplasms, with a three fold increase of cancers of the alimentary tract and a six-fold increase of malignancies of the haemopietic system and lymphatic organs in 20-59 year old career military servicemen exposed to pulse-modulated 150 - 3500 MHz RF/MW radiation.”

### 17.2.3.4 Conclusions:

Dr Szmigielski states that this does not prove a causal link but the high incidence of certain forms of neoplasms in personnel exposed to pulse-modulated RF/MW radiation clearly shows a need for urgent identification of causal factors present in the occupational environment. However, in the context of the studies presented in this review, there are plausible mechanisms to relate the observed increases in cancer to altered cellular behaviour, these results are consistent with animal experiments, and with many other epidemiological studies.

### 17.3 Residential Studies:

#### 17.3.1 North Sydney Study:

**17.3.1.1 Introduction:**

Hocking et al. (1996) undertook a population based study of people in three municipalities which surround three TV and FM radio towers in North Sydney. The health status for leukaemia and brain tumour in the three inner municipalities was compared to the health status in a ring of six outer municipalities, Figure 30.

**17.3.1.2 Effects Associated:**

Among children, the rate ratio for total leukaemia incidence was 1.58 (CI: 1.07-2.34) and for total leukaemia mortality it was 2.32 (CI: 1.35-4.01). For childhood lymphatic leukaemia, the most common type, the rate ratio was 1.55 (CI: 1.00-2.41) for incidence and 2.74 (CI: 1.42-5.27) for mortality.

The exposed population compared to the New South Wales population has a non-statistically significant increase in childhood brain tumour incidence of 30% (SIR/SMR= 1.3; CI:0.7-2.3), while the “outer” group has a 20% increase (SIR/SMR= 1.2; CI:0.9-1.6). Total leukaemia incidence for all ages was 1.24 (95%CI: 1.09-1.40), Table 16.
Figure 30: Municipalities in northern Sydney and the TV towers (numbered 1, 2 and 3). The circle has a 4 km radius and is for reference only. Willoughby, Lane Cove and North Sydney are the inner “exposed” municipalities, Hocking et al. (1996).

Table 23: Rate Ratios (RR) and 95% confidence intervals (CI) for cancer incidence and mortality in the population of the inner area compared to the outer area, adjusted for age, sex and calendar period.

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>RR (95% CI)</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain Tumour</td>
<td>0.89 (0.71-1.11)</td>
<td>740</td>
</tr>
<tr>
<td>Total Leukaemia</td>
<td>1.24 (1.09-1.40)</td>
<td>1206</td>
</tr>
<tr>
<td>Lymphatic Leukaemia</td>
<td>1.32 (1.09-1.59)</td>
<td>536</td>
</tr>
<tr>
<td>Myeloid Leukaemia</td>
<td>1.09 (0.91-1.32)</td>
<td>563</td>
</tr>
<tr>
<td>Other Leukaemia</td>
<td>1.67 (1.12-2.49)</td>
<td>107</td>
</tr>
<tr>
<td><strong>Mortality</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain Tumour</td>
<td>0.82 (0.63-1.07)</td>
<td>606</td>
</tr>
<tr>
<td>Total Leukaemia</td>
<td>1.17 (0.96-1.43)</td>
<td>847</td>
</tr>
<tr>
<td>Lymphatic Leukaemia</td>
<td>1.39 (1.00-1.92)</td>
<td>267</td>
</tr>
<tr>
<td>Myeloid Leukaemia</td>
<td>1.01 (0.82-1.24)</td>
<td>493</td>
</tr>
<tr>
<td>Other Leukaemia</td>
<td>1.57 (1.01-2.46)</td>
<td>87</td>
</tr>
</tbody>
</table>

These data clearly show the greater susceptibility of children to leukaemia in association with RF exposure than adults, Table 24.
### Table 24: Rate Ratios (RR) and 95% confidence intervals (CI) for cancer incidence and mortality in childhood (0-14 years) in the population of the inner area compared to the outer area, adjusted for age, sex and calendar period.

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>RR (95% CI)</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain Tumour</td>
<td>1.01 (0.59-2.06)</td>
<td>64</td>
</tr>
<tr>
<td>Total Leukaemia</td>
<td>1.58 (1.07-2.34)</td>
<td>134</td>
</tr>
<tr>
<td>Lymphatic Leukaemia</td>
<td>1.55 (1.00-2.41)</td>
<td>107</td>
</tr>
<tr>
<td>Myeloid Leukaemia</td>
<td>1.73 (0.62-14.81)</td>
<td>9</td>
</tr>
<tr>
<td>Other Leukaemia</td>
<td>1.65 (0.33-8.19)</td>
<td>8</td>
</tr>
<tr>
<td><strong>Mortality</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain Tumour</td>
<td>0.73 (0.26-2.10)</td>
<td>30</td>
</tr>
<tr>
<td>Total Leukaemia</td>
<td>2.32 (1.35-4.01)</td>
<td>59</td>
</tr>
<tr>
<td>Lymphatic Leukaemia</td>
<td>2.74 (1.42-5.27)</td>
<td>39</td>
</tr>
<tr>
<td>Myeloid Leukaemia</td>
<td>1.77 (0.47-6.69)</td>
<td>11</td>
</tr>
<tr>
<td>Other Leukaemia</td>
<td>1.45 (0.30-6.99)</td>
<td>9</td>
</tr>
</tbody>
</table>

### 17.3.1.3 Exposures:

Exposure levels were calculated for the 4 individual TV stations. They were combined and plotted against the geographic centre of the three TV towers, Figure 31. The frequencies involved are in the range 63 - 219 MHz and 626-633 MHz.

Within a radius of about 1 km or so the area is inside the circle of the towers themselves. The high readings between 4 to 8 $\mu$W/cm$^2$ at about 1 km are the areas immediately adjacent to each of the towers where few, if any, people reside. At the geographic centre, between the towers, the calculated exposures are in the range 1 to 2 $\mu$W/cm$^2$.

Outside the circle of the towers themselves their combined calculated level of exposure falls from about 1$\mu$W/cm$^2$ at 2 km from the centre, to 0.2$\mu$W/cm$^2$ at 4 km and 0.08$\mu$W/cm$^2$ at 8km. Thus the exposed population resides in calculated outdoor levels between 0.2 and 2$\mu$W/cm$^2$. Measurements found that in the region of Tower 1 the actual levels, among the rolling terrain, trees and buildings, were 5 times lower than those calculated. Indoor exposures would be 4 to 8 times lower again. Hence mean residential exposure for the inner group is in the range 0.01 to 0.2$\mu$W/cm$^2$, or less.

People who reside on ridgetops will receive greater exposure than those who live in valleys. Dr Hocking is following this study up through more detailed interviews and locations of those identified with cancer.
17.3.1.4 Dealing with confounders:

Hocking et. al (1996) searched diligently to find confounders to explain these results. They investigated and found no bias due to socio-economic class, proximity to industry, density of traffic (and hence benzene levels), air pollution, ionising radiation, high voltage power lines, population movement, nor the location of hospitals.

Recent suggestions that the increase in incidence and mortality of leukaemia was caused by a high incidence in one municipality (Lane Cove) was checked with the primary author, Dr Bruce Hocking. Dr Hocking says that this is not true. The team checked this out and stated in the paper:

"To see whether results within each municipality were similar, we performed tests of homogeneity for childhood leukaemia incidence and mortality. No significant heterogeneity was found (P=0.10 for incidence and P=0.13 for mortality)."
17.3.1.5 Conclusions:

It came as a complete surprise to the research team that 39 children had died of lymphatic leukaemia when only 14.2 were expected in the exposed population, especially when the mean measured RF exposure levels are in the range of the MW exposures found within a few hundred metres of a cell site.

This is a highly significant study, carried out very carefully, with no pre-determined view of a positive association being expected. The study was originally sponsored by Telstra (formerly Telecom Australia) as a “toe in the water study” to ally fears about health effects of cell sites according to Dr Bruce Hocking on National Radio with Kim Hill. The results prove the opposite conclusions, that there is a probable but not proven effect from RF exposure from cell sites of increased risk of childhood leukaemia and lower, but still statistically significant increased risk of adult leukaemia.
Consistency with other, less detailed and less comprehensive studies, which have also found positive associations with cancer, including leukaemia and brain tumour in association with elevated RF/MW radiation exposure, provides compelling evidence of increased risk of cancer, especially leukaemia in children, from chronic low level RF exposure at the level of a probable to highly probable human carcinogen.

The Planning Tribunal in setting the 2 $\mu$W/cm$^2$ condition in the McIntyre case was not aware of this study as it had not been published at that time. It was also not told of the desire of a U.S.E.P.A. review team to have RF/MW radiation classified as a possible human carcinogen in 1990. With the Hocking and other more recent studies E.P.A. officials agree that the evidence in now even stronger.

17.3.2 The Skrunda Radar Study:

17.3.2.1 Introduction:

A radar location station in Latvia, near the town of Skrunda, has been operating for over 20 years. People live on the land in front of the radar, with villages, farms and forestry being the predominant features.

17.3.2.2 Associated Human Effects:

Although the local population have recorded many health complaints, the health effects have not been reported yet. A study has been carried out on children’s performance, comparing the children in a village up to 20 km from Skrunda but in front of the radar, with children who live behind the radar.

Kolodynski and Kolodynski (1996) studied a group of 966 children (425 males and 541 females) aged 9-18 years. A total of 609 children were examined from the Kuldiga and Saldus regions within a 20 km radius of the Skrunda RLS. Of these, 224 pupils live in directly exposed areas to the west of the radar. The control group were 357 pupils from the Preili region, behind the radar.

For the populations living in front of the radar and behind it, and for the control group, groups of similar age and sex were selected. They examined similar social groups of farming communities, and 95% of subjects lived on small farms.

They conclude that "the weak correlations between the distance from the children’s homes to the RLS, and the children’s responses, are certainly consistent with the idea of an electromagnetic field effect."

Statistically significant differences were observed between the performance of exposed and control groups of children which leads to the conclusion:

“The children living in front of the Skrunda RLS have less developed memory and attention, slower reaction times and decreased endurance of neuromuscular apparatus. On the basis of the data obtained, one could propose the working hypothesis that the decreased endurance of muscular apparatus, slower reaction time and less developed memory and attention are the results of chronic electromagnetic radiation effects. Evidence for a
factor other than electromagnetic field having caused the observed results was not found, but its existence cannot be ruled out, for example, differences in the past experiences of the children, local small pollution effects, differences in family behaviour, etc."

While confounding effects cannot be ruled out, the evidence for the changes in children’s performance is most plausibly related to the very low emissions of pulsed RF radiation from the Skrunda radar. Whether it is the RF energy or the pulse rate or size is not known. The pulse rate is unlikely to have had the operative effect on plants, people and animals. The “working hypothesis” proposed constitutes evidence of a potential adverse environmental effect.

17.3.2.3 Human Exposures:

Exposures were measured with frequency sensitive equipment, Kalnins et al. (1996), and so the measured readings relate to the output of the radar as against the broad band ambient radiofrequency exposure from all emissions which is likely to be around 0.001\(\mu\)W/cm\(^2\). Hence measurements or estimates of radar signals below this level will be irrelevant, unless the effects are frequency specific. Any levels above 0.001\(\mu\)W/cm\(^2\) are localized exposures above the ambient whose effects can potentially be associated with the radar irrespective of whether or not the effects are frequency sensitive.

Children living in the exposed zone have been tested for a range of performance parameters. The children lived and went to school in a zone about 4 to 15 km from the radar, with open exposure measurements in the range 0.0008-0.41 \(\mu\)W/cm\(^2\), mean in the 0.0028- 0.039\(\mu\)W/cm\(^2\). The measurements range is far wider than the mean range because of short-term variations in the output of the radar and weather conditions, etc. The local measurements (for the radar signal) among the trees and buildings, are in the range 1.7x 10\(^{-6}\) - 0.0027 \(\mu\)W/cm\(^2\). The frequency was in the range 156-162 MHz and the radar is pulsed at a rate of 24.4 Hz and pulse width of 8 ms.
17.3.2.4 Bovine Effects:
A herd of female Latvian Brown Cows were studied, Balode (1996), using cytogenetical evaluate of chromosome breakage in blood, using a micronuclei method. Comparing a herd of the same type of cows from in front of the radar with a herd housed behind the radar, they found a small incidence of broken chromosomes in the peripheral erythrocytes of the exposed cows of 0.6 per 1000. However this was six times that of the unexposed cows, which is significant at the p<0.01 level.

17.3.2.5 Bovine exposures:

Assuming the herd was within 1 to 2 km of the front of the radar, in open grassy pastures, the measured exposure would be in the range $0.042$ to $6.6\mu W/cm^2$, mean exposures in the range $0.157$ to $0.63\mu W/cm^2$.

17.3.2.6 Pine tree growth increments:

Many stands of pine trees were studied and reduction in growth ring increment was found. A particular set of stands lie at a radius of about 4 km from the radar, Balodis et al. (1996). Figure 34 shows the growth response of a set of stands of pine trees at a 4 km distance from the radar, where measured exposure levels are in the order of $0.0027\mu W/cm^2$.

![Figure 34: The mean relative additional increment of pine trees for a plot 4 km distance in from of the Skrunda RLS radar. The solid bars depict significant deviations from normal growth.](image)

They conclude,
“There is a statistically significant (P<0.01) negative correlation between the relative additional increment in tree growth and the intensity of the electric field. The radial growth of pine trees is diminished in all plots that received electromagnetic radiation. This decrease in growth began after 1970, which coincided with the start of the operation of the Skrunda RLS, and was subsequently observed throughout the period of the study.”

The study team investigated other environmental and anthropogenic factors but found no significant effects to relate to tree growth.

Selga and Selga (1996) investigated the effect of RF exposure on the needles of *Pinus sylvestris* L. They found physiological changes with exposed trees which they state would explain the difference detected in tree ring width. They conclude:

“Evidently, EMF induces modification of the Golgi apparatus and switches its functions from synthesis of predecessors of cell walls (lignins) to formation and export of resin predecessors. The stress due to the RF EMF generated by the Skrunda RLS causes an unspecific response - accelerated resin production and promoted senescence (aging) of pine trees.” ...

“Phenol-induced senescence of pine trees can explain the decrease in tree ring increment width and viability of pine forests caused by direct pulsed RF EMF irradiation.”

Hence there is strong evidence from plant physiology that gives a biological mechanism to explain the observed decrease in growth rate of pine tree which are exposed to pulsed RF radiation.

**17.3.2.7 Plant reproductive damage:**

Magone (1996) investigated the vegetative growth and morphology of the duckweed *Spirodela polyrhiza (L.) Schleiden*. These plants have high vegetative reproduction rates, and genetically uniform clones can be used for experimentation. The results of exposure to the Skrunda RLS irradiation was dramatic.

“At 55 days, various morphological and developmental abnormalities appeared in 60 to 100 % of the exposed plants and 1 % of the control plants. Plants developed completely from daughter fronds under exposure from the electromagnetic field had a shorter life-span (67 days compared to 87 days in the controls) and fewer subsequent daughters (8 compared to 10 in the control group).”

It is also noted that the decrease in reproduction rates and the occurrence of deformities in future generations after 30 days of exposure to pulse-type RF irradiation comes from small cellular changes that become evident only after replication in cell division. This was also supported by the fact that in the experiment to determine life-span, where only the daughters directly produced from the mother were observed, the number of deformities was almost two times lower than when all descendants were observed.

They conclude:
“Our work suggests that studies of non-thermal radiofrequency electromagnetic fields on organisms must be comparable to the life-span of the organism. If short-term observations are made, only the organism response to electromagnetic radiation as a stress factor can be seen. Long-term studies can yield different conclusions due to more effects becoming evident only at later times.”

These results are consistent with observed chromosome damage in plants exposed to RF radiation, Haider et al. (1994), who used 10 to 27 MHz broadcast antennae with very high, but sub-thermal exposures of at least 424$\mu$W/cm$^2$ (40 V/m).

17.3.2.8 Plant exposure levels:

The Pine trees at 4 km were exposed to a range of 0.011 to 0.41 $\mu$W/cm$^2$, a mean open field exposure of 0.039$\mu$W/cm$^2$ and measured distance exposure of 0.0027$\mu$W/cm$^2$ (for the radar signal). Indications are that the duckweed study was done closer to the radar and so the bovine exposure levels would be likely to apply, i.e. range 0.042 to 6.6$\mu$W/cm$^2$, mean exposures in the range 0.157 to 0.63$\mu$W/cm$^2$.

17.3.2.9 Summary and conclusions:

The Skrunda Radar provides a living laboratory for the chronic low level effects of exposure to RF/MW radiation. To date investigations have revealed a number of statistically significant changes associated with exposure to the radar signal. These include:

- Impaired scholastic performance of children in the open field exposure range of 0.0008-0.41 $\mu$W/cm$^2$, mean measured level in the range 0.0028- 0.039$\mu$W/cm$^2$.

- A 6-fold increase in broken chromosomes in the peripheral erythrocytes of the exposed cows (p<0.01). for a measured exposure would be in the range 0.042 to 6.6$\mu$W/cm$^2$, mean exposures in the range 0.157 to 0.63$\mu$W/cm$^2$.

- A statistically significant (P<0.01) negative correlation between the relative additional increment in tree growth and the intensity of the electric field. The Pine trees at 4 km were exposed to a range of 0.011 to 0.41$\mu$W/cm$^2$, a mean open field exposure of 0.039$\mu$W/cm$^2$ and measured distance exposure of 0.0027$\mu$W/cm$^2$ (for the radar signal). A probable biological mechanism was identified through observed changes in physiological conditions.

- Chromosome and reproductive damage in plants exposed RF/MW in the range 0.042 to 6.6$\mu$W/cm$^2$.

Chronic exposure to pulsed RF radar signals is associated with chromosome damage in plants and animals, with associated reproductive aberration in plants, and growth reduction in pine trees linked to observed physiological changes, and scholastic impairment of school children occurs in relation to exposure levels which fall well below 2$\mu$W/cm$^2$, below 0.1$\mu$W/cm$^2$. and even below 0.01$\mu$W/cm$^2$. 
17.3.4 Chinese Study:

Chiang et al. (1989) studied subjects living and working near radio antennae and radar installations. Most of the studied exposures were above 10\(\mu\)W/cm\(^2\), and they included microwaves and AM EMF. The tests carried out included visual reaction time, standardized written tests and white blood cell (WBC) phagocytosis. Visual reaction time increased significantly for Male Soldiers exposed to microwaves in the range 10-15\(\mu\)W/cm\(^2\) (\(p<0.01\)) and Male College students exposed to 13-42\(\mu\)W/cm\(^2\) of microwaves (\(p>0.05\)). Memory Function scores were also significantly reduced for both of these high exposed groups, \(p<0.01\) for both groups. Secondary school boys and girls showed non-significant changes in visual reaction time (0-4\(\mu\)W/cm\(^2\)) but a significant improvement in memory Function Scores (\(p<0.05\) for boys and \(p<0.01\) for girls).

Blood tests associated with microwave exposure showed significantly elevated white blood cell counts for high school students (\(p<0.05\)) for exposure 0-4\(\mu\)W/cm\(^2\). Male Soldiers in the middle range showed no effect and Male College Students in the high range showed a significant reduction in WBC.

AM exposure was associated with a 15 % increase in WBC in kindergarten children exposed to 3-4 V/m (2.4 - 4.2\(\mu\)W/cm\(^2\)) and a significant (\(p<0.05\)) 31.2 % increase at 4-11 V/m (4.2-32 \(\mu\)W/cm\(^2\)). Non significant reductions in WBC were found in 3rd year high school students at moderate exposures 10-18 V/m, but WBC were significantly reduced to 45.7 % for boys (\(p<0.01\)) and to 81.2 % for girls (\(p<0.05\)) in the range 22-23 V/m (128-140\(\mu\)W/cm\(^2\)).

The authors conclude “The data indicate that chronic exposures to EMFs are associated with significant changes in some physiological parameters.”

Increased WBC at low exposures have been observed for example in the staff of the U.S. embassy in Moscow, exposed to mean exposures of somewhat less than 1/5th of the outdoor, upper floor measure mean exposures of 1 to 2.4\(\mu\)W/cm\(^2\), i.e. less than 0.2 to 0.5\(\mu\)W/cm\(^2\), produced a total WBC 25 % higher than average, with a 41 % increase in lymphocytes and 31 % increase in monocytes, Lilienfeld et al. (1978).

17.3.5 U.K. TV/FM studies:

The Small Area Statistics Unit, Department of Epidemiology and Public Health at Imperial College, London, Dolk et al. (1997a), was contracted to study the possible health effects in the population living around the regional TV/FM transmitter in Sutton Coldfield, just north of Birmingham. Peak exposures for the TV signal were measured at 2.5 m AGL at 1.3 \(\mu\)W/cm\(^2\), and for the FM signal at 5.7 \(\mu\)W/cm\(^2\). These don’t occur at the same radius and so the total exposure peaks between 6 and 6.5 \(\mu\)W/cm\(^2\). Mean exposures amongst built-up areas, because of the scattering and absorbing effect of tree and buildings, is around 1/5th of the measured well exposed value. That is, residential mean exposures will peak at around 1.2 to 1.4 \(\mu\)W/cm\(^2\), between about 1 and 3 km from the tower, and decrease with distance at higher distances. At 10 km it is expected to be around 0.05\(\mu\)W/cm\(^2\).

17.3.5.1 Study 1: The Sutton Coldfield Tower.

Primary findings of the study are:
“the results of this study confirm that there was an excess of adult leukaemia within the vicinity of the Sutton Coldfield TV/FM transmitter in the period 1974-1986, accompanied by a decline in risk with distance.”

The risk of adult leukaemia within 2 km was 1.83 (CI: 1.22-2.74) and the decline with distance was significant at p<0.001.

Table 25 shows the results for cancer.

Table 25: All cancers, all leukaemias, and non-Hodgkin’s lymphomas near the Sutton Coldfield transmitter, West Midlands, England: observed and expected numbers of cases, observed/expected (O/E) ratios, and cumulative O/E ratios, by distance of residence from the transmitter, in person aged >= 15 years, 1974-1986. Dolk et al. (1997a).

<table>
<thead>
<tr>
<th>Distance from transmitter (km)</th>
<th>All cancers*</th>
<th>Cumulative O/E ratio</th>
<th>All leukaemias</th>
<th>Cumulative O/E ratio</th>
<th>Non-Hodgkin's lymphomas</th>
<th>Cumulative O/E ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obs.</td>
<td>Expd</td>
<td>O/E</td>
<td>Obs.</td>
<td>Expd</td>
<td>O/E</td>
</tr>
<tr>
<td>0-0.5</td>
<td>2</td>
<td>5.61</td>
<td>0.36</td>
<td>1</td>
<td>0.11</td>
<td>0.36</td>
</tr>
<tr>
<td>0-1.0</td>
<td>96</td>
<td>137.19</td>
<td>0.70</td>
<td>5</td>
<td>2.72</td>
<td>1.84</td>
</tr>
<tr>
<td>1-2.0</td>
<td>605</td>
<td>504.59</td>
<td>1.20</td>
<td>17</td>
<td>9.76</td>
<td>1.74</td>
</tr>
<tr>
<td>2-3.0</td>
<td>282</td>
<td>279.01</td>
<td>1.01</td>
<td>9</td>
<td>5.56</td>
<td>1.62</td>
</tr>
<tr>
<td>3-4.9</td>
<td>1,002</td>
<td>1,050.86</td>
<td>0.95</td>
<td>25</td>
<td>20.22</td>
<td>1.24</td>
</tr>
<tr>
<td>4.9-6.3</td>
<td>2,414</td>
<td>2,301.25</td>
<td>1.05</td>
<td>54</td>
<td>41.96</td>
<td>1.29</td>
</tr>
<tr>
<td>6.3-7.4</td>
<td>2,734</td>
<td>2,650.62</td>
<td>1.03</td>
<td>48</td>
<td>46.54</td>
<td>1.03</td>
</tr>
<tr>
<td>7.4-8.3</td>
<td>2,827</td>
<td>2,798.65</td>
<td>1.01</td>
<td>51</td>
<td>49.22</td>
<td>1.04</td>
</tr>
<tr>
<td>8.3-9.2</td>
<td>3,363</td>
<td>3,213.75</td>
<td>1.05</td>
<td>40</td>
<td>57.35</td>
<td>0.70</td>
</tr>
<tr>
<td>9.2-10</td>
<td>4,084</td>
<td>3,919.59</td>
<td>1.04</td>
<td>54</td>
<td>68.90</td>
<td>0.78</td>
</tr>
</tbody>
</table>

* All cancers excluding non-melanoma skin cancer.

Secondary findings of the study were: “declines in skin melanoma and bladder cancer with distance from the transmitter site.”

An excess risk ratio (O/E) close to the tower, with a decline in risk (incidence) with distance, is an extremely significant result. It is a dose-response relationship as it follows the ground level exposure of low exposure close to the tower, rising to a peak some distance from the tower and thence decreasing with the inverse square law with additional distance from the tower. The study found these highly significant results for adult leukaemia, skin melanoma and bladder cancer.

These results prompted a follow-up study of 20 other regional TV/FM transmitters throughout the U.K.

Table 26: Skin Melanoma and bladder cancers in the vicinity of the Sutton Coldfield transmitter, West Midlands, England: observed and expected numbers of cases, observed/expected (O/E) ratios, and cumulative O/E ratios, by distance of residence from the transmitter, in person aged >= 15 years, 1974-1986. Dolk et al. (1997a).

<table>
<thead>
<tr>
<th>Distance from transmitter (km)</th>
<th>Skin melanoma</th>
<th>Bladder cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-4.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.9-6.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.3-7.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.4-8.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.3-9.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.2-10</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
17.3.5.2 Study 2: The 20 tower study:

Dolk et al. (1997b) carried out a similar analysis to Study 1, for 20 other regional TV/FM transmission towers throughout the U.K.. These towers have a range of output powers and arrangements of TV and FM signals. The results are presented for all towers combined and for groups of towers according to power groupings, Table 27.

Study 2 produced the following conclusions:

“A decline in risk of adult leukaemia, with distance, was found for all transmitters combined (p=0.05), two of the transmitter groups, and three single transmitters; for all transmitters combined, observed excess risk was no more than 15 % at any distance up to 10 km.”.

The highest O/E ratio for the group of leukaemias was 1.20 for Chronic Lymphatic Leukaemia, for skin melanoma was 1.11 (within the 2km radius) and no relationship was found for bladder cancer. For the group of high powered transmitters (Group 1) the peak ratio for all leukaemias was 1.29 at 2-3 km; Moderate power group TV (Group 2), 1.17 at 2-3 km; Moderate Power Group FM (Group 3), 1.28 at 3-4.9 km and Low Power group (Group 4), 1.28 at 3-4.9 km. For all combined and Groups 1 and 2 the cumulative O/E ratio is still positive at 10 km. In comparing the 20 site results with Sutton Coldfield the following was stated:

“In conclusion, while there is evidence of a decline in leukaemia risk with distance, the pattern and magnitude of the risk associated with residence near the Sutton Coldfield transmitter do not appear to be replicated around other transmitters.”

### TABLE 27: Cancer incidence near 20 high power radio and TV transmitters in Great Britain—all leukemias: observed (0) and expected (E) numbers of cases, 0/E ratios, and cumulative O/E ratios, for all transmitters combined, for transmitter groups, and selected individual transmitters, by distance of residence from transmitter, in persons aged ≥15 years, 1974-1986.

<table>
<thead>
<tr>
<th>Distance (km)</th>
<th>Observed</th>
<th>Expected</th>
<th>O/E ratio</th>
<th>Cumulative Observed</th>
<th>Expected</th>
<th>O/E ratio</th>
<th>Cumulative O/E ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-0.5</td>
<td>0</td>
<td>0.09</td>
<td>0.00</td>
<td>0</td>
<td>0.24</td>
<td>0.00</td>
<td>0</td>
</tr>
<tr>
<td>0.5-1.0</td>
<td>2</td>
<td>2.02</td>
<td>0.99</td>
<td>4</td>
<td>5.96</td>
<td>0.67</td>
<td>0.65</td>
</tr>
<tr>
<td>1.0-2.0</td>
<td>11</td>
<td>6.99</td>
<td>1.57</td>
<td>39</td>
<td>22.17</td>
<td>1.76</td>
<td>1.52</td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>12</td>
<td>5.03</td>
<td>2.39</td>
<td>11</td>
<td>11.94</td>
<td>0.92</td>
<td>1.34</td>
</tr>
<tr>
<td>3.0-4.9</td>
<td>16</td>
<td>16.16</td>
<td>0.99</td>
<td>43</td>
<td>45.27</td>
<td>0.95</td>
<td>1.13</td>
</tr>
<tr>
<td>4.9-6.3</td>
<td>26</td>
<td>28.77</td>
<td>0.90</td>
<td>119</td>
<td>100.31</td>
<td>1.19</td>
<td>1.16</td>
</tr>
<tr>
<td>6.3-7.4</td>
<td>28</td>
<td>27.93</td>
<td>1.00</td>
<td>131</td>
<td>114.85</td>
<td>1.14</td>
<td>1.15</td>
</tr>
<tr>
<td>7.4-8.3</td>
<td>32</td>
<td>30.90</td>
<td>1.04</td>
<td>117</td>
<td>120.64</td>
<td>0.97</td>
<td>1.10</td>
</tr>
<tr>
<td>8.3-9.2</td>
<td>28</td>
<td>35.66</td>
<td>0.79</td>
<td>169</td>
<td>140.13</td>
<td>1.21</td>
<td>1.13</td>
</tr>
<tr>
<td>9.2-10</td>
<td>34</td>
<td>43.08</td>
<td>0.79</td>
<td>155</td>
<td>167.45</td>
<td>0.93</td>
<td>1.08</td>
</tr>
<tr>
<td>Group 1</td>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-0.5</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5-1.0</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1.0-2.0</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.0-4.9</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.9-6.3</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.3-7.4</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.4-8.3</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.3-9.2</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.2-10</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 3</th>
<th>Group 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-0.5</td>
<td>0.00</td>
</tr>
<tr>
<td>0.5-1.0</td>
<td>0.00</td>
</tr>
<tr>
<td>1.0-2.0</td>
<td>0.00</td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>0.00</td>
</tr>
<tr>
<td>3.0-4.9</td>
<td>0.00</td>
</tr>
<tr>
<td>4.9-6.3</td>
<td>0.00</td>
</tr>
<tr>
<td>6.3-7.4</td>
<td>0.00</td>
</tr>
<tr>
<td>7.4-8.3</td>
<td>0.00</td>
</tr>
<tr>
<td>8.3-9.2</td>
<td>0.00</td>
</tr>
<tr>
<td>9.2-10</td>
<td>0.00</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Crystal Palace</th>
<th>Wenvoe</th>
<th>Rowridge</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-0.5</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>0.5-1.0</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>1.0-2.0</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>3.0-4.9</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>4.9-6.3</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>6.3-7.4</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>7.4-8.3</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>8.3-9.2</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>9.2-10</td>
<td>0.00</td>
<td>0.00</td>
</tr>
</tbody>
</table>

* Group 1: Highest power TV transmitters of 870-1,000 kW erp; Group 2, all TV transmitters of 500-1,000 kW erp; Group 3, all FM transmitters of 250 kW erp; Group 4, all transmitters with a combination of TV (≥500 kW erp) and FM (250 kW erp) transmission.

17.3.5.3 Result Conflict Resolution:

It might be tempting to discount the results of both of the projects on the grounds of the inconsistencies in the results. This would be scientifically incorrect. Natural variability of the transmission powers, frequency, and antennae patterns makes the exposures quite different from site to site. Within this variability there is a great deal of consistency between the results and the differences could well be explained in terms of these physical factors.

For example, a cellsite transmitter at 20m above ground level, produces a 2m AGL side-lobe peak at about 20-60m from the tower, and the main-beam ground level peak is at 120 to 210m from the tower base. In proportion, the ground level exposure peaks from a 240m tower are likely to be in the range 240-720 m for the side lobes, and 1400 to 2500m for the main beam peak. The populations exposed to the main-beam peak is generally far greater because it covers a much larger area. With a range of 1400 to 2500 m the
area within this range inside 2km (6.4 km$^2$) is close to the area outside 2 km (7.0 km$^2$). Hence, depending on the physical arrangement of the antennae, the peak exposed population is almost equally likely to live inside or outside the 2 km ring.

In addressing the 2km/3km inconsistency it should be noted that in relation to leukaemia, the All Transmitters, Group 1, Group 2, Rowridge and Crystal Palace peak O/E ratios occur in the 2-3 km zone, whereas at Wenvoe, Group 4 and Sutton Coldfield have O/E peaks in the 1-2 km zone. The highest peaks are clustered around 2 km (1-2 km and 2-3 km). The significantly different antenna powers, mixtures of TV and FM stations and physical configurations are likely to be more than sufficient to account for these differences.

Consistencies include finding an excess of adult leukaemia at some distance from the towers, which then decreases with distance. Skin melanoma was higher in the first 2 km in both cases. Bladder cancer incidence was elevated but did not decrease with distance nor attain statistical significance in the 20-site study.

The primary inconsistency is with bladder cancer which is significant in the Sutton Coldfield study and erratically related to distance in the 20 site study.

The very high rate ratios associated with <2 km for the Sutton Coldfield transmitter involves 11 people. The cumulative rate ratio does not become statistically significant at p=0.05 until the 4.9 km radius (RR=1.49, 95%CI: 0.99-2.42). Hence the difference between adult leukaemia incidence at the Sutton Coldfield site and the “All transmitters” data, is not statistically significant.

These papers do show excess incidence of adult leukaemia in association with living in proximity to TV/FM towers, with a decrease in incidence with distance from individual towers and from all transmitters combined. This is consistent with the excess of adult leukaemias found by Hocking et al. (1996), Milham (1985, 1988) and Szmigielski (1996). The increase in skin melanoma is consistent with De Guire et al. (1987) and Szmigieliski (1996).

It is hopeful that Dr Hocking can repeat this level of radial analysis in a follow-up study in North Sydney. If this is accomplished then the results will be highly significant in the light of the U.K. and other residential leukaemia studies.

17.3.6 The Moscow (U.S.) Foreign Service Workers Study

For many years in the late 1960’s through to the 1970’s the Soviet government aimed radar signals at the US embassies in Moscow and other European cities. State Department staff worried about the increasing incidence of cancers in the staff, spouses and children of diplomatic staff working in Moscow and other Eastern Block countries. This led to an extensive epidemiological study. The first stage was controversial but was ended with the death of the team leader, Professor Lilienfeld. Lilienfeld (1978) himself recommended follow-up studies because of the cancer latency periods. The Moscow Group appeared to be most highly affected. Blood tests revealed statistically significant increases (p<0.001) in hematocrit and decreases in neutropil, for example, Goldsmith (1995). White blood cell counts were strikingly higher in the Moscow group.
Professor Goldsmith was not directly involved at that time, but after Prof. Lilienfeld’s death he was asked to become more involved with individual staff as rates of cancer appeared to increase, Goldsmith (1995). Professor Goldsmith’s most recent analysis shows significant increases in cancers in adults and children, some of which were not evident in the earlier study because of the case reference approach taken and because the latency period for some cancers being 8 to 20 years, Goldsmith (1995,1996).

The State Department arrange for exposure reading to be taken.

Table 28: Maximum exposure and exposed areas by time period as estimated by the State Department, Lilienfeld et al.(1978).

<table>
<thead>
<tr>
<th>Time Period</th>
<th>Exposed Area of Chancery</th>
<th>Maximum Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1953 to May 3, 1975</td>
<td>West Facade</td>
<td>5 - 14 $\mu$W/cm$^2$, 9h/day</td>
</tr>
<tr>
<td>June 1975 to Feb 7 1976</td>
<td>South and East Facade</td>
<td>15 $\mu$W/cm$^2$, 18 h/day</td>
</tr>
<tr>
<td>Since Feb 7, 1976</td>
<td>South and East Facade</td>
<td>&lt; 1 $\mu$W/cm$^2$, 18 h/day</td>
</tr>
</tbody>
</table>

The peak values were 5 to 15 $\mu$W/cm$^2$.

Assuming the latter period ended in December 1978, and assuming the maximum exposures are the means, there was 281 months at 1.88$\mu$W/cm$^2$, 20 months at 11.25$\mu$W/cm$^2$ and 23 months at about 0.5$\mu$W/cm$^2$. This gives a highest possible mean exposure over the whole period of 2.4$\mu$W/cm$^2$ and a likely mean exposure of less than about 1$\mu$W/cm$^2$. These exposure readings are for the outside walls of the embassy. The beams were always directed at the upper floors of the chancery. Staff and children would be inside most of the time, not often on the upper floors, and therefore would be exposed to a fraction of this, probably less than 10 %, or 0.1 to 0.24$\mu$W/cm$^2$. Whether it is this low or slightly higher, it is within the range of that produced at ground level near this cell site.

I have received copies of reports on the analysis of the blood of the Moscow Embassy staff. These reports were classified and have only been released and stamped “declassified” a few years ago, even though they were written in 1969 and 1976. Both reports found major mutagenic changes in blood samples. 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”. It covered analyses of blood from between 21/2/66-30/6/69. This covers the period when the external wall exposure was 5-14$\mu$W/cm$^2$ for 9 hours/day, averaging then 1.9-5.3$\mu$W/cm$^2$. They were analysing for mutagenetic effects by identifying chromosomal damage. The results were expressed as:

Table 29: Hematological Tests of chromosome and other damage in the blood of U.S. Foreign Service Workers from Moscow and other Eastern Embassies.
<table>
<thead>
<tr>
<th>Scale</th>
<th>Mutagenic Level</th>
<th>Clinical Significance</th>
<th>Patient X-Numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Extreme</td>
<td>Definite</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>Severe</td>
<td>Questionable</td>
<td>73,74,76,79,84,102</td>
</tr>
<tr>
<td>3.5</td>
<td>Intermediate</td>
<td>Suspect</td>
<td>72,83,91,99,103</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>Suspect</td>
<td>70,71,93,97,98,100,104</td>
</tr>
<tr>
<td>2.5</td>
<td>Intermediate</td>
<td>Questionable</td>
<td>75,87,90,94,96</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>Questionable</td>
<td>69,81,85,92,95</td>
</tr>
<tr>
<td>1</td>
<td>Normal</td>
<td>None</td>
<td>77,78,80,82,86,101</td>
</tr>
</tbody>
</table>

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.”

The later, 7 October 1976 report by James Tonascia and Susan Tonascia, titled “Hematology Study”, and included all employees who arrived in Moscow before December 1975. This totaled 213 individuals from Moscow and they were compared with 981 other Foreign Service employees. They found:

“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.

What was the resulting health effects. 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:

- Two leukaemia deaths in Moscow when 0.8 are expected (RR = 2.5).
- Three leukaemia deaths in other eastern block embassies when 1.7 are expected (RR=1.8).
- Four deaths due to female genital cancer in Moscow compared with 0.8 expected (RR=5.0).
- Three deaths due to female genital cancer in other embassies compared with 1.3 expected (RR=2.3).
- Four dependent children died of cancer among Moscow families compared to 1.5 expected (RR=2.7).

Table 30 gives a break down of the cancer deaths of the children involved.

**Table 30: Cancer Mortality of the Children Exposed to Microwave Radiation in the Moscow and Western Embassies, Goldsmith (1995).**
Overall these reports include 16 cancer deaths when 6.1 were expected, an overall risk ratio of 2.62. Hence the hematological samples showing increased mutations in exposed foreign service workers is reflected in significant increases in the incidence of cancer in the exposed population. **This is very compelling evidence.**

These are very significant increases in the incidence of cancer mortality which are hard to dismiss or ignore, and the exposures to the pulsed microwave radiation are extremely low, between 0.1 and 0.24 μW/cm², based on 10% of the external, one exposed wall only measurements of less than 1 to 2.4 μW/cm² on average and between 5 and 15 μW/cm² for peaks. Remember that the radar was directed at the top floor only.

These very low mean exposures are associated with a 4- to 5-fold increase in childhood leukaemia, a 3.8 fold increase in all cancers in Moscow and an overall increase in cancer of 4.0 for living in and 1.36 for living outside the compound in Moscow. This is a low level doess-response relationship with risk increasing with increasing mean probable exposure. Note that the readings were taken on the outside walls of the embassy chancellery, not inside where the people were. Hence the increased health risk is associated with somewhat lower mean exposure levels than are reported in Table 28.
17.3.6 Parental Occupation and Risk of Birth Defects in Offspring:

Schnitzer et al. (1995) state that several epidemiological studies indicate some parental occupations are associated with an increased risk of birth defects. They review a large number of papers and extend the analysis using data collected as part of the Metropolitan Atlanta Congenital Defects Programme between 1968 and 1980. Of particular interest here are the group known as Electricians and Electrical workers. “Electronic equipment operators is another category in this exploratory analysis that has elevated odds ratios for several birth defects.

**Table 31: Birth defects associated with parental occupations, Schnitzer et al. (1995).**

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Anencephalus</th>
<th>OR</th>
<th>CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electricians, electrical workers (N=229)</td>
<td>7</td>
<td>1.3</td>
<td>0.6-2.8</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>10</td>
<td>1.2</td>
<td>0.6-2.5</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>3</td>
<td>0.6</td>
<td>0.2-1.9</td>
</tr>
<tr>
<td>Coactation of aorta</td>
<td>6</td>
<td>3.0</td>
<td>1.2-7.5</td>
</tr>
<tr>
<td>Rectum, anus atresia/stenosis</td>
<td>4</td>
<td>1.7</td>
<td>0.6-5.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Anencephalus</th>
<th>OR</th>
<th>CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electronic equipment operators (N=123)</td>
<td>4</td>
<td>1.6</td>
<td>0.5-4.8</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>6</td>
<td>1.9</td>
<td>0.7-4.7</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>4</td>
<td>2.6</td>
<td>0.9-7.9</td>
</tr>
<tr>
<td>Cleft Palate</td>
<td>3</td>
<td>2.1</td>
<td>0.6-7.3</td>
</tr>
<tr>
<td>Cleft lip and palate</td>
<td>4</td>
<td>1.7</td>
<td>0.6-5.4</td>
</tr>
<tr>
<td>Pyloric Stenosis</td>
<td>7</td>
<td>1.7</td>
<td>0.7-3.9</td>
</tr>
<tr>
<td>Reduction defects upper limb</td>
<td>4</td>
<td>4.2</td>
<td>1.3-13.7</td>
</tr>
</tbody>
</table>

This group includes announcers, air traffic controllers; and broadcast equipment computer and telephone operators. Some or all of these workers are potentially exposed to electromagnetic fields, including radiofrequency radiation. The electricians and electrical workers category has similar potential exposures.

The authors acknowledge the limitation of parental occupation as an indicator of exposure and the existence of many potential confounders. What they have done is to identify potential risk factors which are significant for some industrial exposure situations.

Savitz and Chen (1990) summarize studies of parental occupations and childhood nervous system cancers. The following table covers electrical and electronic workers:

**Table 32: Results of studies of parental occupation and childhood nervous system cancers, Savitz and Chen (1990).**

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Cancer site</th>
<th>No.Cases</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electronics Workers</td>
<td>Neuroblastoma</td>
<td>6</td>
<td>11.8*</td>
</tr>
</tbody>
</table>
The results mean that we can't ignore the possibility of birth defects in offspring for parents exposed to electromagnetic radiation. That potential clearly exists.

### 17.3.7 Summary of Epidemiological Evidence:

These studies together show statistically significantly increased risk and incidence in many important health and well-being factors in human beings exposed to a range of frequencies and intensities of residential expose and school exposure at levels well below \(2 \mu\text{W/cm}^2\), being in the range of about 0.04 to 0.2\(\mu\text{W/cm}^2\) in the North Sydney cancer study; 0.0034\(\mu\text{W/cm}^2\) (the bottom on Zone B) in the Schwarzenburg Study (though Zone C also shows sleep disturbance effects); in Latvia at similar exposure levels, school children’s performance is impaired and pine trees show decreased growth rates in mean measured exposure of 0.0027\(\mu\text{W/cm}^2\) at 4 km from the radar, in the Skrunda Study, and in the U.K., with increased adult leukaemia out to 10 km, about 0.05 \(\mu\text{W/cm}^2\).

The U.S. embassy in Moscow provides additional consistency evidence for childhood leukaemia, in addition to the Hawaii, and North Sydney studies. It also adds biological plausibility to the many other studies showing chromosome aberrations and DNA breakage. Adult cancer increase in Moscow is not unusual in the light of the scores of other studies associating adult cancers to RF/MW exposure.

There is also growing evidence of childhood defects being associated by parents’ EMR exposure.

### 18. Classification of Carcinogens:

The task now is to place the evidence in a more objective assessment context. A classification scheme for carcinogens has been assembled to assist with this.

#### 18.1 Background:

Many substances, originally assumed to be benign, are now classified as carcinogens. The classification as several levels or ranking from a possible human carcinogen to a proven human carcinogen. The vast majority of listed substances are potential carcinogens in various stages of investigation and weights of evidence of possible or probable carcinogenicity. Associate Professor Neil Pearce, epidemiologist at the Wellington Clinical school, says that about 19 of the 20 major human carcinogens were first identified by epidemiology, pers comm. Classification of human carcinogens must involve human epidemiological studies to receive a high classification. Animal studies give toxicological and biochemical evidence of processes which can give reinforcement to human epidemiological studies.

An epidemiological study which finds a statistically significant increase in cancer incidence in association with an identified risk factor may well be equivocal because of unknown confounding factors. The search to resolve this issue can involve further
epidemiological studies and/or animal experiments when test animals are challenged with known levels of the possible carcinogen. If the results of a replicated animal experiment point strongly to the risk factor present and not discounted in the epidemiological studies then the risk factor would be classed as a Class C, possible human carcinogen.

Stronger animal data accompanied by stronger epidemiological evidence, moves the classification further up the scale.

18.2 A classification scheme:

A recent comprehensive review by a team of Swedish researchers, Hardell et al (1995), applied criteria for evaluating groups of epidemiological studies to identify (a) no association, (b) probably no association, (c) possible association and (d) association. The US EPA use a class C to A classification. The following table illustrates the US EPA approach, which combines animal and epidemiological input:

Possible Carcinogen: Class C.: The human evidence is inadequate but there is some "limited" animal experiments indicate increased cancer with increased exposure.

Probable Carcinogen: Class B2: Sufficient animal experiments with some, but not adequate human evidence.

Probable Carcinogen: Class B1: Sufficient animal experiments and limited human epidemiological evidence.

| Table 33: Potential Carcinogens EPA “Weight-of-Evidence” classification and Hazard Ranking |
|-----------------------------------------------|-------------------------------------------------|-----------------------------|
| Class A: Human Carcinogen                     | Class B: Probable Human Carcinogen               | Class C: Possible Human Carcinogen |
| B1: Limited Epidemiologic Evidence            | B2: Sufficient Animal Evidence                   |                                    |
| Arsenic (H)                                   | Acrylonitrile (M)                                | Beryllium (M)                 |
| Asbestos (H)                                  | Cadmium (M)                                      | Methyl Chloride (L)           |
| Benzine (M)                                   | Creosote (H)                                     | DDT (M)                       |
| Diethylstilbestrol (H)                         | Ethylene Oxide (M)                               | PCBs (M)                      |
| Vinyl chloride (H)                             | Formaldehyde (M)                                 | Carbon tetrachloride (M)      |

Hazard Ranking: (H)=High; (M)=Medium; (L)=Low. Source Office of Health and Environmental Assessment, EPA June 1988

Human Carcinogen: Class A.: Coherent, strong evidence of human epidemiology, backed up by strong animal evidence.

In relation to the RMA, where a “potential effect of low probability but high potential impact” gives a threshold for level of evidence based on epidemiological analysis. Through decisions of the Planning Tribunal the concept of “potential effect” incorporates a “plausible mechanism”. This conforms to the EPA approach thought the plausible mechanism does not necessarily use an animal model, but could equally be a biophysical/biochemical model. For ease of applying these criteria, they have been
slightly modified by combining the epidemiological criteria from Hardell (1995) with the EPA approach.

1. **No association** based on: three or more studies showing no association with measured, estimated or evaluated EMF exposure or exposure hygienic classification. Confounding can be ruled out with reasonable confidence. Absence of animal or cell tissue experiments showing biological effects which could be potentially associated with cancer.

2. **Possible (Potential) carcinogen** (Class C) based on a “plausible mechanism and not mere innuendo”, i.e. animal and cell tissue experiments reveal one or more plausible mechanisms by which the increased EMF exposure can reasonably lead to cancer initiating or cancer promoting cellular behaviour, or an replicated animal experiment showing responses consistent with cancer causing factors, such as impaired immune system, chromosome breakage, DNA damage, gene mutations, etc. A well performed animal experiment exists showing increased carcinomas but beyond this the animal evidence is “limited”.

3. **Probable carcinogen** (Class B2) based on: three or more studies showing a pattern of an association with measured, estimated or evaluated EMR exposure or hygienic classification, backed up by plausible mechanisms through replicated animal and/or tissue experiments, as for Class C above.

4. **Highly Probable carcinogen** (Class B1) based on: consistent results in two or more well-performed studies showing excess risks and a dose-response in terms of measured, estimated or evaluated EMR exposure, or a consistent pattern of an association with excess risks in exposure hygienic classification. Confounding can be ruled out with reasonable confidence; backed up by plausible mechanisms through replicated animal or tissue experiments.

5. **Human carcinogen** (Class A) based on: consistent results in four or more well-performed studies showing excess risks and a dose-response in terms of measured, estimated or evaluated EMR exposure, or a consistent pattern of an association with excess risks in exposure hygienic classification. Confounding can be ruled out with strong confidence. Reliably demonstrated consistent causal mechanisms are available from more than one laboratory for each mechanism, through in vitro and/or in vivo experiments.

18.3 A current classification assessment:

Several causal mechanisms are set out and well described above, including melatonin suppression, alteration of the signal transduction process at cellular level, alteration of the cell cycle at critical times such as the s-phase, co-carcinogenic effects with other carcinogens, co-promotion to enhance cancer incidence and chromosome aberrations from the action of liberated free radicals.

Long term animal experiments show statistically significantly increased benign and malignant tumours without chemical initiation, and very much enhanced incidence of lung, skin and breast tumours with chemical initiation, showing cancer initiation and promotional attributes for RF/MW radiation, consistent with the cellular changes observed in vitro.
Szmigielski’s results are consistent with the re-evaluation of the U.S. Navy Korean War study, with the increase in Lymphatic and haematopoietic cancers, brain, eye and CNS, cancers, skin cancer and cancer of the respiratory and digestive organs.

Brain cancer associations were also found by Thomas et al (1987), Speers et al. (1988), Tornqvist et al. (1991) and Grayson (1996); skin cancer by Vagero et al. (1985), De Guire et al. (1987), Szmigielski (1996); eye cancer by Holly et al. (1995) and adult leukaemia by Milham (1988), Goldsmith (1995), Szmigielski (1996), Dolk et al. (1997a, 1997b) and Hocking et al. (1996).

Breast cancer in women exposed to radar was significantly raised in Moscow, Goldsmith (1995). It is consistent with the melatonin mechanism and has been found in electrical industries, Cantor et al. (1995) and in men, Demers et al. (1991).

The Moscow Embassy staff and dependents, chronically exposed to low intensity radar signals, experienced statistically significant increases in childhood leukaemia incidence and death, Goldsmith (1995). This is consistent with Hocking et al. (1996), Maskarinec and Cooper (1993), and Anderson and Henderson (1986).

The Moscow Embassy study also involved blood tests which showed significantly elevated hematocrit and monocyte count, and lower neutrophil concentrations. White blood cells were strikingly higher, Goldsmith (1995). He also reports that the occurrence of multiple-site cancers was unusually high, 1.33 sites/person compared to 1.02 expected from the Third National Cancer Survey.

Residential studies finding statistically significant increases in cancer with exposure to RF/MW radiation, some with dose-response relationships (*), include: Lester and Moore (1982)*, Lester and Moore (1985), Maskarinec and Cooper (1993), and Anderson and Henderson (1986), Hocking et al. (1996), Dolk et al. (1997a*, 1997b*)

Together these results, reinforced by animal experiments, make the strong case of classifying RF/MW radiation exposure a highly probable human carcinogen, Class B1, or even Class A, according to U.S. EPA classification.

Exposure levels at which Risk Ratios are significantly raised average less than 10 \( \mu \text{W/cm}^2 \) for military and occupational studies and less than 0.1\( \mu \text{W/cm}^2 \) for residential studies. For example Hocking et al.(1996) where calculated exposures of the “exposed” group residences are in the range 0.2 to 2\( \mu \text{W/cm}^2 \), but measured outdoor levels were around 1/5th of this, i.e. 0.04 to 0.4\( \mu \text{W/cm}^2 \), but mean exposures, including indoor time, will be even lower. This is consistent with the results of Lester and Moore (1982, 1985).

18.4 Mortality statistics significantly under-estimate morbidity:

The absence of specifically directed studies involving a comprehensive assessment of potential health effects probable leads to a major underestimate of the possible adverse effects. This arises because mortality statistics are more robust and more readily available than is the incidence of non-fatal tumors and lesions for example. Demers et al. (1992) demonstrate this using a Tumor Registry verses Death Certificates in an Occupational Cohort Studies in the United States. Their abstract records the following conclusion:
“As expected, an increased ability to study relatively common cancers with low fatality rates was demonstrated by the incidence data. The most dramatic example was seen for bladder cancer. Twenty-four bladder cancers were diagnosed among the study cohort (consisting of 4,528 Tacoma fire fighters and police officers) between 1974 and 1989, whereas only two deaths were attributed to this malignancy.”

Hence most studies which are related to mortality statistics grossly underestimate the adverse health effects. Thus studies of the full potential impact of any particular environmental stressor are difficult and rare because of the limitations in available data and the complexity of human subjects. This is more likely to lead to an under-estimate of the impact of a particular stressor than an over-estimate.

Thus it is vital to remember that most epidemiological studies used to assess carcinogenicity use mortality statistics. There is between 10 and 20 times more tumours produced which do not result in death but are a major cost on the health system, extremely worrying to the person and causes loss of earnings.

19. Conclusions

19.1 Standards and standard setting:

The Australia/ New Zealand standards committee for EMR is a technical committee of “stakeholders” which includes a majority of those who derive direct or indirect benefit from the production and use of EMR. It is not independent of industry as a public health protection standards committee should be. The U.K. provides a good alternative model.

The standard derives from western approaches to standards setting which has been dominated by the United States, the Tri Services Program (Army, Navy and Air Force), ANSI and IEEE and relate closely to thermal effects protection. They are not based on epidemiological results for they are, in the main, assumed to be faulty under the mistaken assumption that there are only thermal effects. Recent moves to relax the Australia/ New Zealand standard were based on conforming to these U.S. standards, on the basis that they are “more scientific”. This is only true to the extent that they are well based in the science of heating.

19.2 Athermal biological mechanisms:

Observations, reinforced by mathematical models, show that time varying signals interact at the cellular and tissue level producing voltage gradients and cell development changes at extremely low exposure levels corresponding to tissue gradients of the order of $10^{-7}$ V/cm. Changes in brain function are particularly evident, with altered circadian rhythm associated with the removal of the extremely weak Schumann Oscillations (0.3 pW/cm$^2$) and altered human EEG at 0.7 $\mu$W/cm$^2$. Most aspects of cell cycle activity have been shown to be altered by imposed RF/MW signals, including cell cycle time, signal transduction controls of cell development, differential and proliferation, cell ion balance, DNA synthesis, cell membrane permeability, and melatonin reduction and free radical damage, associated with chromosome aberrations.
Calcium ion efflux and melatonin/free radical processes are implicated in impaired immune system performance. This relates to carcinogenesis, spontaneous miscarriage, birth deformity, and a host of other diseases.

Melatonin reduction is a central and primary mechanism, which is also involved with sleep disruption, chronic fatigue syndrome, learning and memory impairment.

This can be well summarized by referring to the following very recent papers. Resonant absorption at the cell membrane was demonstrated by Liu and Cleary (1995). A review of research on effects of microwaves on the nervous system 1990-1995, published in an IEEE journal in October 1996, states:

"The use of weak electromagnetic fields to study the sequence and energetics of events that couple humoral stimuli from surface receptor sites to the cell interior has identified the cell membrane as a primary site of interaction with these low frequency fields in the pericellular fluid. Field modulation of the cell surface chemical events indicates a major amplification of initial weak triggers associated with the binding of hormones, antibodies and neurotransmitters to their specific binding sites. Calcium plays a key role in this stimulus amplification, probably through highly cooperative alterations in binding to surface glycoproteins, with spreading waves of altered calcium binding across the membrane surface. Protein particles spanning the cell membrane form pathways for signaling and energy transfer. Fields millions of times weaker than the membrane potential of $10^7$ V/m modulate cell responses to surface stimulating molecules.

The evidence supports non-linear, non-equilibrium processes at critical steps in transmembrane coupling. Cancer promoting phobol esters act at cell membranes to stimulate ornithine decarboxylase which is essential for cell growth and DNA synthesis. This response is enhanced by weak microwave fields also acting at the cell membrane. There is strong evidence that cell membranes are powerful amplifiers of weak electrochemical events in their vicinity", Vorst and Duhamel (1996).

Microwaves, 915 MHz, whether pulsed or continuous, open the Blood-Brain Barrier, making the brain more open to toxic polar molecules and weakening the BBB changes the system which controls the stability of the fluid movement of the brain's intracellular compartment. The specific results are, Vorst and Duhamel (1996):

1) Exposed animals are at risk for opening the BBB (Odds Ratio =3.8, p=0.0004).

2) The response is independent of pulse repetition rate, and the response is the same for CW as compared to pulsed modulation.

3) The response is independent of SAR in the interval 0.016< SAR < 2.5 W/kg (Odds Ratio = 3.3), but rises for SAR > 2.5 W/kg.

19.3 Animal Studies:
Long term animal studies have shown increases in benign and malignant tumours (carcinomas) at a multitude of sites, consistent with the whole body coverage of EM radiation; with skin cancer, breast cancer, lung cancer, cancer of the white cells, lymphatic tumours and myeloid leukaemia, atrophy of the testes, lower birth weight, still birth, resorption, hemorrhage and stunted growth, immune system impairment and altered brain activity (EEG), reaction times and learning retention.

Short term exposure experiments have found DNA breakage in living rats brains, associated with free radicals and melatonin reduction.

19.4 Public Health and Epidemiological Studies:

Several human studies show reduction in melatonin directly through blood or urine samples, and indirectly through monitoring sleep quality and scholastic performance. At least three studies show impairment of children’s learning and memory functions at residential levels of exposure. The Schwarzenburg study shows sleep difficulties which have a strong dose-response relationship and were confirmed when the transmitter went off unknowingly. The effects were seen as far as 5 km away. Chronic fatigue syndrome was very evident. The effects are seen across the RF/MW spectrum. Sleeping with a mobile phone next to your bed changes your EEG and interferes with REM sleep, causing learning and memory problems, in the same manner as radar in Latvia and SW radio in Switzerland.

Scores of occupational studies have linked RF/MW exposure to increased incidence of cancer and cancer related death. The strongest rate ratios in occupational studies and the strongest results from residential studies involves Leukaemia. Residential studies associate increased incidence of leukaemia in adults and children at measured mean exposure levels down to about 0.04 $\mu W/cm^2$ (0.2 $\mu W/cm^2$ / 5: North Sydney).

With cell changes, animal experiments and scores of epidemiological studies, RF/MW can be classified as a Class B1 (highly probable) human carcinogen or even a Class A human carcinogen. Avoidance or significant risk reduction will only be achieved for chronic residential exposure where mean exposure levels are somewhat less than 0.1 $\mu W/cm^2$.

Some results are grouped in Table 34, with higher exposures corresponding to higher risk ratios, giving a grouped dose-response. These are more than sufficient to establish a potential adverse health effect “of low potential probability and high potential impact”.

<table>
<thead>
<tr>
<th>Study</th>
<th>Exposure Range $\mu W/cm^2$</th>
<th>Risk Ratio Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polish Military</td>
<td>&lt; 7 - 14</td>
<td>3.0 - 13.9</td>
</tr>
<tr>
<td>Moscow Embassy</td>
<td>&lt; 0.1 - 2.4</td>
<td>1.7 - 5.0</td>
</tr>
<tr>
<td>Korean War</td>
<td></td>
<td>1.9 - 3.3</td>
</tr>
<tr>
<td>U.K. 21 sites</td>
<td>&lt; 0.05 - 1.6</td>
<td>1.01 - 3.57</td>
</tr>
<tr>
<td>North Sydney</td>
<td>&lt; 0.04 - 1.6</td>
<td>1.61 - 2.74</td>
</tr>
</tbody>
</table>
19.5 Reproduction Studies:

Studies on the adverse effects of RF and MW exposure on pregnancy involving physiotherapists, show MW to be a risk factor for early spontaneous miscarriage and RF to be a risk factor for perinatal death and congenital deformity. Mean exposure levels in the range 0.04 to 0.56 $\mu$W/cm$^2$, corresponding to 10 treatments per month, is associated with an Odds Ratio of 1.50 (CI:1.04-2.17). This approach to averaging is appropriate because of the very high plausibility of a non-thermal mechanism, such as chromosome aberrations from the release of free radicals. A higher rate of damage from a given MW exposure compared with RF rate of damage, could well explain the early pregnancy effect of MW and the late pregnancy effects of RF.

19.6 Biological Studies:

Human beings are not the only part of the environment which have shown adverse biological impacts of exposure to RF/MW radiation at very low mean ambient levels.

Adverse biological effects on plants and animals have been identified in the Schwarzenburg and Skrunda Studies. Pine tree growth ring annual increments were significantly reduced at 4 km from the Skrunda radar, in mean measured exposure levels of 0.0027$\mu$W/cm$^2$, a six-fold chromosome damage level in cattle blood was found in the absolute range 0.04 to 6.6$\mu$W/cm$^2$, and mean measured exposure range 0.16 - 0.63$\mu$W/cm$^2$, a similar range for the plants which demonstrated massive disruption of their reproductive system.

19.7 Children’s performance:

Adverse effects are found at very low mean environmental levels of exposure to RF/MW which relate to performance rather than health. Children’s intellectual and physical performance levels were significantly impaired in both the Swiss and Latvian studies, in mean residential exposure levels in the range 0.03 to 9.06$\mu$W/cm$^2$, median 0.1$\mu$W/cm$^2$ and mean 0.24$\mu$W/cm$^2$ in Switzerland and 0.003 to 0.04$\mu$W/cm$^2$ in Latvia.

These results are consistent with the very significant human EEG changes observed by Von Klitzing (1995), at exposure levels of 0.7$\mu$W/cm$^2$.

The Chinese study, Chiang (1988) also showed significant changes in children mental and physical performance, but at slightly higher levels of exposure, 0-4$\mu$W/cm$^2$.

19.8 Sleep disruption, fatigue, aches and blood pressure:

These Swiss exposures were also associated with significant increases in reported disorders, especially in those over 45 years, involving sleep disruption and chronic fatigue syndrome, related to melatonin reduction, as well as aches, pains, lung problems and heart problems. These were associated with mean exposure levels (Zones A and B) in the range 0.024 - 0.24$\mu$W/cm$^2$.

20. Recommendations:
There is extensive and compelling scientific research which links RF/MW exposure, down to very low mean exposure levels, to severe health problems and mortality risks.

**This suggests setting the Public exposure limit at:**

0.1μW/cm² if cancer risk is to be reduced, and

0.01μW/cm² if miscarriage risk, sleep disruption, children’s performance impairment and chronic fatigue symptoms are to be reduced.

This requires that cell sites, wherever possible, be located away from residential areas. In Australia, with so much open space. There is no practical impediment to placing base stations in open rural settings, outside rural and suburban settlements and towns. The small costs of extra cables is trivial compared to the health costs associated with chronic exposure of residents.

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Yee, 1992:

