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SAFE EXPOSURE LEVELS

INTRODUCTION:

1.1 Background to this critique

There is a strong push from the WHO and the ICNIRP of harmonize national RF/MW exposure standards by individual states adopting the ICNIRP Guideline. This would be a good thing if the ICNIRP Guideline was set at an exposure level that provided sound protection of public health. The evidence presented here shows that the ICNIRP Guideline exposure level is set many orders of magnitude too high to accomplish this. It is based on the preconceived and long held view of Western Government Authorities that the only possible and only established biological effect of RF/MW exposure is tissue heating. This is referred to here as the RF-Thermal View. This view has been intransigently maintained in the face of compelling laboratory and epidemiological evidence of adverse health effects that would have had a chemical declared carcinogenic, neuropathogenic, cardiogenic and teratogenic for humans many years ago.

This critique was originally written when the New Zealand Ministries of Health and Environment proposed to adopt the ICNIRP Guideline as the Public Health Standard for Cell Site exposures. At the same time the New Zealand RF Standards Committee was proposing to use the ICNIRP Guideline as the New Zealand RF/MW Standard. ICNIRP is the International Commission on Non-Ionizing Radiation Protection. The ICNIRP RF/MW guideline and scientific assessment was published in Health Physics, Vol. 74 (4): 494-522, 1988. This is the primary source document for this critique and will be referred to as ICNIRP (1998).

The ICNIRP (1998) assessment of effects has been reviewed against the research literature cited and other published research. It is found that both the basic approach of ICNIRP and its treatment of the scientific research have serious flaws. The ICNIRP assessment is determined to maintain the RF-Thermal View and it rejects or omits all evidence that conflicts with this view. This may be termed "Constructive Dismissal" for a preconceived concept is used to inappropriately dismiss all evidence that challenges it.

ICNIRP is particularly dismissive of epidemiological evidence because all existing studies involve nonthermal exposures. Hence accepting the validity of these studies would directly challenge the RF-Thermal View. In this way the approach to dealing with health effects from non-ionizing radiation was developed to follow a completely different method than for toxic chemicals, drugs or air pollution. Both the approach of ICNIRP and the assumptions made are severely scientifically challenged in this report.

Overview of this report:

Public health protection standards for toxic substances, chemicals, drugs, air pollution, ionizing radiation are set by WHO, IARC, E.U., U.S. EPA and the U.K. Royal Commission on Environmental Pollution primarily using epidemiological evidence and secondarily using animal evidence. WHO and ICNIRP base non-ionizing radiation protection standards on a single biological mechanism, Tissue Heating. They systematically reject or ignore all epidemiological and animal evidence of non-thermal effects, for which there is a large body.

The history and basis of the RF-Thermal View which dominates ICNIRP, WHO, and national authority approaches, is documented and summarized. It will be shown that throughout the post-War period scientific research and leading biological and medical scientists have challenged the RF-thermal assumptions. They present very strong evidence, amounting to proof, that biological systems intrinsically use EMR for body, organ, hormone and cellular functions and regulation, and that extrinsic EMR interferes with these at extremely low exposure levels. These biological effects do not involve heat but do involve non-linear, non-equilibrium resonant interactions between ELF oscillating signals.

The well documented and established nonthermal biological effects of EMR include significant alteration of cellular calcium ion homeostasis, reduction of melatonin and the detection of Schumann Resonances by human and avian brains, DNA strand breakage and enhanced chromosome aberrations.

The human health implications of these biological effects are discussed and documented. This shows that calcium ion efflux/influx and melatonin reduction are separately and jointly linked to DNA strand breaks, chromosome aberrations, enhanced proto oncogene activity, impaired immune system competence and impaired neurological and cardiac functioning. Many projects, from independent labotories, have observed and reported that all of these effects are significantly related to EMR exposure.

Human Biometeorology is a whole body of research that is ignored by ICNIRP. This has provided the proof over 30 years ago that human brains detect and use the Schumann Resonances for synchronization of biological rhythms, i.e. as a Zeitgeber. This observation on its own is an absolute challenge to the validity of the ICNIRP assumptions that there are no established non-thermal biological effects.

Epidemiological reviews by Dr John Goldsmith show that adverse health effects, such as neurological, reproductive and cancer effects have been observed in EMR exposed populations. Based on this, and the traditional public health protection approach, Dr Goldsmith challenges the validity of the ICNIRP guideline and approach.

To summarize the scientific evidence an initial set of eight bioelectromagnetic principles are proposed and a brief summary of the scientific research that supports them is given. They are:

EMR is intrinsic to our bodies.

Our brains are the most electrically sensitive organs in our bodies.

Our hearts are electrically sensitive.

Cells are sensitive to EMR

Our whole body acts as an aerial

The brain is linked to organs and cells through EMR-sensitive hormones.

The EMR Spectrum Principle.

The Intrinsic Free Radical Principle

These principles provide a sound and scientifically reliable approach to assessing EMR impacts on people and animals. They soundly challenge the ICNIRP assumptions and approach. The ICNIRP assessment of biological mechanisms is reviewed and found to be selective, limited and flawed. Their assessment of RF/MW effects on reproductive outcomes is shown to be limited, misleading and flawed.

The cancer assessment is shown to be selective, misleading, inappropriate and flawed. An incorrect epidemiological approach is consistently applied.

From the data in the studies cited (and misused) by the ICNIRP and WHO reviews, and supported by a great deal of other available research evidence, a public health protection standard is recommended based on residential dose-response relationships for cancer, neurological effects and reproductive effects.

2. Public Health Protection Standards are based on Epidemiology:

The background to identifying environmental factors that produce cancer will be given, along with an example using the chemical Benzene. Then the principles of epidemiology relating to assessment of cause and effect will be outlined and the particular principles in the epidemiology of EMR will be discussed.

2.1 Cancer Assessments are based on environmental epidemiology:

Public health Protection Standards are based on Epidemiological Evidence. A primary textbook on Cancer, De Vita, Hellman and Rosenburg (1993), states:

"In contrast to laboratory studies, epidemiology directly evaluates the experience of human populations and their response to various environmental exposures and host factors (the risk of disease)".

Del Regato, Spjut and Cox (1985) introduce their medical textbook on cancer by discussing the use of Incidence Rates in human populations as the means of detecting human cancers. Fraumeni et al. (1993) outline the historical role that epidemiology has played in identifying carcinogenic agents and the range of methods which are classically used.

Setting public health standards for environmental carcinogens is the role of the United States Environmental Protection Agency (USEPA). Their website includes the Integrated Risk Information System (IRIS), _ HYPERLINK "http://www.epa.gov/ngispgm3/iris/rfd.htm" __http://www.epa.gov/ngispgm3/iris/rfd.htm_, that details the procedures for carrying out assessments and the results for a wide range of carcinogens. This is primarily based on epidemiological assessments. Under the heading "Hazard Identification" the following statement relates to the use of epidemiological studies:

"Human data are often useful in quantitatively establishing the presence of an adverse effect in exposed human populations. When there is information on the exposure level associated with an appropriate endpoint, epidemiologic studies can also provide the basis for a quantitative dose-response assessment. The presence of such data obviates the necessity of extrapolating from animals to humans; therefore, human studies, when available, are given first priority, with animal studies serving to complement them."

An environmental epidemiologist of considerable standing, alongside Sir Austin Bradford Hill, was Professor Abraham Lilienfeld, of Johns Hopkins University. He was the epidemiologist responsible for the survey of health effects at the U.S. Embassy in Moscow. In his paper "Practical limitations of Epidemiologic methods" (Lilienfeld, 1983), Professor Lilienfeld discussed some of the difficulties of demographic studies, including the issue of the "ecological fallacy". In relation to his study on the staff and dependents at the U.S. Moscow Embassy, he states:

"The problems associated with these studies are illustrated by reviewing some of the details of the study of effects of microwave radiation on embassy employees in Moscow. The study population had to be reconstructed, individuals had to be located and information on exposure status has to be obtained by

questionnaire. The relatively small size of the exposed group permitted the detection of only fairly large relative risks. Despite these limitations, epidemiologic studies have been remarkably productive in elucidating etiological factors. They are necessary since 'the proper study of man is man' ".

Dr Lilienfeld describes a classical epidemiological approach and problems. Epidemiology is complex and difficult, but it is the best and most appropriate science for the study of the effects of environmental exposures on human populations.

2.2 A Chemical Example - Benzene:

An example is the carcinogenic assessment for Benzene. Benzene is classified by the U.S.E.P.A. as a known human carcinogen (Category A) based on "convincing human evidence as well as supporting evidence from animal studies". At the end of the section on "Human Carcinogenicity Data", having outlined the epidemiological evidence, the conclusion is:

"All of the epidemiological studies referred to above have some methodological problems, i.e. confounding exposures, lack of sufficient power, and other limitations, but the consistent excess risk of leukaemia across all of these studies argues that such problems could not be entirely responsible for the elevated risks of cancer. Most of these epidemiologic studies have been reviewed in peer-reviewed publications. They provide clear evidence of a causal association between exposure to Benzene and ANLL. The evidence is suggestive with respect to CNLL and CLL."

ANLL: Acute Nonlymphocytic Leukaemia.
CNLL: Chronic Nonlymphocytic Leukaemia.
CLL: Chronic Lymphocytic Leukaemia.

The Benzene Assessment is based on a total of 15 epidemiological papers covering 6 separate studies, one showing a significant dose-response relationship. Several papers found insignificantly elevated leukaemia rates. Some of these reached significance when follow-up studies involved more cases. In summary the dose-response data gives:

Table 1: Air concentrations at specific risk levels:

Risk Level	Concentration of Benzene
1 in 10,000	13.0 to 45.0 (g/m3
1 in 100,000	1.3 to 4.5 (g/m3
1 in 1,000,000	0.13 to 0.45 (g/m3

Figure 1: An example of standard setting using Benzene from the Royal Commission on Environmental Pollution, Houghton (1998).

The United Kingdom, Royal Commission on Environmental Pollution, 21st report "Setting Environmental Standards", Houghton (1998), also shows the reliance on epidemiology in setting such standards and outlines the procedures followed. The are very similar to the USEPA and IARC. The Royal Commission also uses Benzene as an example, Figure 1.

There is no discussion at all in the EPA Benzene assessment, nor the Royal Commission summary, about biological mechanisms. It is wholly sufficient that consistent human studies, two cohort studies and one dose-response relationship shows increases in leukaemia. A MEDLINE search reveals a large number of cytogenetic studies showing that Benzene enhances chromosome damage in animals, worker

and human blood. None of these are cited by the EPA Assessment. The epidemiological studies give the necessary and sufficient evidence for the carcinogenicity assessment.

It is stated in Figure 1 that human studies were more useful than animal studies. Most human studies involved high occupational exposure that were probably under-estimated, making their results and over-estimate of the risk of effects. They refer to the Expert Panel on Air Quality Standards (EPAQS) who considered that the risk of leukaemia in workers was undetectable when average exposure over a working lifetime is around 500 ppb. Taking into account working lifetime (77,000 hours) compared with chronological lifetime (660,000 hours) the figure is reduced by a factor of 10. A further factor of 10 is applied to extrapolate from fit, young to middle-aged workers to the general population giving 5 ppb. Allowing for uncertainties in the ambient exposure, and following the principle of keeping exposure as low as practicable, a target standard of 1 ppb was adopted as a running annual average. This applies an overall safety factor of 500 below the NOAEL for moderate to highly exposed workers.

The UK report refers to the number and importance of international conventions relating to the environment. This includes the Maastricht Treaty that sets out the basis for the European Union's environmental policy, which includes protecting human health. The basic procedure of human health risk characterization is to compare the estimated human dose (EHD) of a given substance with either the no observed adverse effect level (NOAEL) or the lowest observed adverse effect level (LOAEL). The NOAEL is the greatest concentration of a substance that produces no observed adverse effect. The LOAEL is the lowest concentration of a substance, found by experiment or observation, that causes any adverse alteration of morphology, functional capacity, growth, development, or life-span, which is distinguishable from control organisms of the same species and strain.

For the epidemiology of human populations the NOAEL approach involves the search for the study with the highest exposure that shows no adverse effect, with no studies that do show elevated risks below it. Then a safety factor is applied to take into account the uncertainties, the susceptibilities, and size of the exposed populations. The LOAEL approach uses dose-response relationships to determine the lowest threshold for the observation of an adverse effect. In using the epidemiological studies, careful consideration of bias and confounding is undertaken and then the Bradford Hill viewpoints are used to guide consideration of the likelihood of cause and effect, Figure 2.

In Figure 2, Houghton (1998), uses the term "criteria" and in the final quote the term "feature". The word "viewpoint" was very carefully chosen by Sir Austin Bradford Hill. They are points from which to view the evidence and not criteria that must the achieved. This is the importance of the note at the bottom of Figure 2. These are not criteria, they are viewpoints with either greater or lesser strength from which we can decide "is there any other way of explaining the set of facts before us, is there any other answer more likely than cause and effect. Epidemiology does not provide "scientific truth". It provides a weight of evidence that must be considered in an informed fashion, and decisions made with incomplete facts.

Figure 2: A summary of the Bradford-Hill viewpoints for deciding on cause and effect from epidemiological evidence, Houghton (1998).

2.3 The Bradford Hill Guidance:

It is also an absolute prerequisite that the exposure takes place prior to the effect occurring (temporality). This is the only viewpoint that could be termed a "criteria".

2.3.1 Viewpoints NOT criteria:

Expert witnesses who appear in court and at hearings for companies assess the epidemiological evidence using what they call the "Bradford Hill Criteria", Black (1998), Elwood (1999) or the "Hill Criteria", Moulder (2000). Dr Moulder states that "The Hill criteria should be viewed as a whole; no individual criterion is either necessary or sufficient for concluding that there is a causal relationship between exposure to an agent and a disease." This is an approach of people who are determined to dismiss the epidemiological evidence. It is totally inappropriate and leads to a significant failure to protect occupational and public health protection.

This is not the approach taken by IARC, US EPA, the U.K. Royal Commission on Environmental Pollution, nor Sir Austin Bradford Hill himself. In fact Sir Austin strongly opposed this approach. He states, Hill (1965):

"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 viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non. What they can do, with greater or less strength, is to help us make up our minds on the fundamental question - is there any other way of explaining the set of facts before us, is there any other answer equally or more, likely than cause and effect."

Sir Austin also rejects strict statistical thresholds such as the 95% confidence interval. He cites a group of cotton mill workers who persistently showed a higher incidence of respiratory disease than similar group of unexposed workers, but the difference was never statistically significant. He says that the evidence was so clear-cut that no formal test could contribute anything of value to the argument:

"No formal tests of significance can answer those questions. Such tests can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis."

Hill (1965) gives the example of respiratory sickness in workers in the cardroom of a spinning mill where they were exposed to dust. The 30 to 60 year olds suffered three times as much illness as other workers not exposed to the dust. The data never reached statistical significance but Dr Bradford Hill concluded that it was a causal effect because the evidence was so clear cut.

With EMR the usual experience is that a small occupational group, people living near powerlines and an electric blanket study show non-significant but elevated rates of disease or mortality. Larger occupational groups and military personnel who experience radar and radio exposures show significantly elevated rates of disease and death. Attempts are then made to identify a gradient of exposure to determine whether a dose response relationship exists. Dose response relationships and significant dose response relationships are then identified using sub-groups who have a graduated risk of exposure by virtue of their jobs and tasks. This builds the picture that across the EMR spectrum similar health effects and biological effects are observed, supporting an integrated approach to the assessment of the data, and indicating a level of evidence that rises progressively towards the point where we can decide that there is a causal relationship. Many years previously we surpassed the threshold for a potential effect for a wide range of EMR related health effects at very low level of exposure with the initial residential and occupational studies showing elevated risks.

With the sensitivity of the brain to electromagnetic signals it is expected that neurological effects will be measurable from EMR exposures. There are many EMR studies of exposed workers that show elevated by non-significant increases or neurological effects. It is a common conclusion of the authors that the result does not support the hypothesis that EMR is associated with neurological disease. This is incorrect, especially if small samples are involved since tests of statistical significant are strongly dependent on

sample size. An instructive example of this is Johansen et al. (1999). They investigated the incidence of Multiple Sclerosis among utility workers. With 32 cases of MS diagnosed when 23.7 were expected, they found a Standardized Incidence Ratio, SIR = 1.35, 95%CI: 0.92-1.91. They then conclude: "We found no support for the hypothesis of an association between occupational exposure to electromagnetic fields and the risk of MS."

What would Sir Austin conclude if he looked closely at their data? Based on only 14 cases, for low, medium and high exposures they observed SIRs of 0.8, 1.4 and 1.6. This is a remarkable result with a biological gradient. This "admits of a simple explanation and puts the case in a clearer light". Actually this study provides very strong support for the hypothesis. Along with several other studies it supports the cause and effect hypothesis.

2.3.2 Specificity:

Specificity is valuable in situations where a specific disease agent is observed to produce a specific disease in specific workers exposed to a specific situation. Sir Austin says that when this occurs it is a strong argument in favour of cause and effect. He immediately cautions that we must not over-emphasize this characteristic because many agents are known to produce more than one cancer or a range of illnesses. He also observed that many diseases are produced by multiple agents. The epidemiological evidence for EMR shows that it enhances a wide range of cancer and sickness in many body organs, under a wide range of exposure conditions across the spectral range. Sir Austin summarizes this with:

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

An application of specificity to EMR arises when we consider which body elements are particularly bioelectromagnetically sensitive and reactive. Our brains and hearts are immediately identified as sensitive organs. However all cells are sensitive, especially in the immune system and the endocrine system, through the actions of calcium ions and melatonin.

2.3.3 Experimentation:

Experimentation is not always possible but where it is, it is very powerful. For example, in the Schwarzenburg Study, involving a shortwave radio tower, a significant dose-response relationship for sleep disturbance was observed. Confirmation of cause and effect came from turning the transmissions off for 3 days without notifying the residents. Sleep quality improved significantly ($p<0.001$), with a delay of about one day, even in the group with the lowest exposure (Group C). This shows that even though they experienced the lowest exposure, the RF signal was still interfering with their brains and their sleep. When the transmission was turned off permanently, measured human melatonin levels rose significantly (Prof. Theo Abelin Pers. Comm.). This is a biological mechanism but it was identified after the assessment of cause and effect was concluded.

2.3.4 First Priority - Dose-Response Relationship:

In relation to dose-response Sir Austin states:

"If the association is one which can reveal a biological gradient, or dose-response curve, then we should look most carefully for such evidence. For instance, the fact that the death rate from cancer of the lung rises linearly with the number of cigarettes smoked daily, adds a great deal to the simpler evidence that cigarette smokers have a higher death rate than non-smokers." … "The clear dose-response curve admits of a simple explanation and obviously puts the case in a clearer light."

Hence a dose-response relationship is highly indicative of a cause and effect.

2.3.5 Second Priority - Strength of Association:

For Strength of Association Sir Austin cites the example of John Snow's classic analysis of the cholera epidemic in 1854. He found 71 deaths per 10,000 in the group whose water came from the Southwark and Vauxhall Company and 5 deaths per 10,000 from those using the Lambeth Company, a factor of 14. No known biological mechanism was available at that time but this is sufficient to decide cause and effect, especially when the Lambeth Company water was sewage-free and the other Company's water wasn't.

Sir Austin warns, however, not to place too much emphasis on strength of association, for some important effects might wrongly be dismissed. He also dismissed the requirements to achieve statistical significance as an absolute requirement. For Dr Bradford Hill, elevated risks are important evidence to be considered in context. A group of mill workers were consistently observed to have elevated respiratory disease, but this never reached statistical significance. Nevertheless it was concluded to be a causal effect.

2.3.6 Third Priority - Consistency

Consistency is a feature to be specially considered. Has the effect been consistently observed to be associated in different persons in different places, circumstances and times? But consistency is not absolute. He states:

"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."

For example, we cannot repeat the Korean War and wait another 20 years to see if cancer and sickness is greater in high exposure groups compared with low exposure groups. A repetition is impossible but we can look for consistency between the Polish Military Study, studies of RF/MW exposed electrical workers, and large population studies of residents around high powered broadcast towers.

2.3.7 Lowest Priority - Biological Mechanism:

Dr Bradford Hill's comments on "biological plausibility" or "biological mechanism" place them at the lowest priority. He states:

"It will be helpful if the causation we suspect is biologically plausible. But it is a feature I am convinced we cannot demand. What is biologically plausible depends on the biological knowledge of the day".

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

Just two years ago Quinn (1997) noted that:

“Although the role of ultraviolet radiation in human skin carcinogenesis has been supported by a wealth of epidemiological data, the mechanisms by which it leads to skin cancer are still poorly understood.”

It is accepted from the epidemiological evidence that UV radiation is carcinogenic, causing melanoma and other skin cancers. It also reduces immune system competence. This is a strong reason for being

very concerned about the depletion of the Ozone Layer and the formation of the "Ozone Holes" over the Arctic and Antarctic.

2.4 ICNIRP's inappropriate reliance on a Biological Mechanism

In setting public health protection standards, epidemiological evidence is the strong guiding evidence. It does not need a biological mechanism for it to be interpreted as a probable or even causal effect. For ICNIRP to concentrate on and rely on a single biological mechanism, Tissue Heating, is inappropriate and wrong. Large portions of official documents are devoted to extensive discourses on SARs and determination of the thermal threshold. This whole methodology is flawed. Health effects assessments start with epidemiological evidence and the existence of a plausible biological mechanism is irrelevant.

WHO, ICNIRP and their international and national counterparts have developed a highly sophisticated system of approaches to dismiss all epidemiological evidence and animal and cellular evidence which conflicts with their RF-Thermal view of the world. As the epidemiological and laboratory evidence has grown stronger and stronger, the dismissive methodology has lost all sophistication and, as demonstrated by ICNIRP (1998), it is blatantly selective, reductionist, biased and scientifically dishonest.

It is selective through carefully selecting positive and negative evidence to maintain their 'no effects' stance through a 'balance of evidence approach'.

It is reductionist by seeking reasons, valid or not, for dismissing each study on its own so that none are left at the end. A set of experimental and epidemiological "criteria" have been adopted in an attempt to make the process look like good science.

It is biased towards accepting evidence of no effects and dismissing all evidence of effects as weak, questionable and unreliable.

It is scientifically dishonest because it cites papers that clearly report significant increases in cancer as showing no evidence of cancer. It deliberately chooses to accept conclusions that claim no association between radar and health effects when the data in the report or paper proves that this is incorrect. ICNIRP also includes studies in its assessment that are incapable of showing effects, as though they provide evidence that there are no effects.

This is a process of "Constructive Dismissal" in which a pre-conceived position is defended at all costs by inappropriately dismissing all evidence that challenges it.

The epidemiological evidence, when appropriately assessed following the Bradford Hill Viewpoint approach, an approach endorsed by Drs Lilienfeld and Goldsmith, provides sufficient weight to establish cause and effect relationships between EMR and many health effects, and to set public health standards. It is wrong in science, and in public health protection policy terms to retain the RF-Thermal view. ICNIRP's 1998 assessment must be rejected and urgently revised in the light of these revelations.

2.5 Cancer Epidemiology:

The science of epidemiology has developed to deal with complex human situations, as are found with almost every potential disease agent, whether it is chemicals, drugs, smoking, air pollution or ionizing radiation. Large groups of the population are identified whose occupation, location or activity involves exposure to the disease agent of concern. On some occasions the level of potential or probable exposure can be reasonably well stratified to allow a dose-response comparison to be made. In all cases, the exposure varies from day to day, week to week, year to year, and person to person. Hence there is a frequency distribution of hourly or daily exposures for each person and for each group. As a consequence

almost all retrospective studies deal only with potential or probable exposures. The frequency distribution of exposures accumulates towards a mean exposure. By judicious choice of occupational groups or residential situations the exposed groups can be dichotomized to compare groups with higher mean exposures compared with other similar groups who have lower or no exposure to the agent. In some circumstances it is possible to identify a gradient of exposure which might allow a dose-response curve to be investigated. Elevated rates of disease and death are then considered and assessed in the light of uncertainties and the important of the evidence for public health.

A commonly accepted model of cancer development involves initiation, promotion and progression, Figure 3. For cancer studies a significant time delay between initiation and the development of malignant cancer can be many years or even several decades. When cancer does develop it occurs in a small number of people when we are younger than 50 years.

For example in New Zealand for male brain and CNS cancer, in the 30-34 age group the incidence is near 4.4 and the mortality near 2.3 per 100,000 person years. In a city of half a million people there are about 25,000 men in the 30-34 yr age group. This gives an annual brain tumor incidence rate of 1.1/yr. If four 30-34 yr old men develop brain tumours they won't usually be noticed but the rate is 3.64 times average. In order to detect the influence of a particular carcinogen, very large samples, very long periods and highly elevated rates are necessary.

The age standardized leukaemia incidence rate for all people is near 8/100,000 p-yr. With a population density of 800 /km2 and population of about 2500 could live within 1 km of a tower. Over a 13 year period this is 32,500 p-yrs giving an expected leukaemia rate of 2.6. This is very similar to the Sutton Coldfield situation, Dolk et al. (1997a). Six leukaemias were observed when 2.83 were expected.

Figure 3: Model of multistage carcinogenesis. Initiation involves a single exposure to a carcinogen that damages the nuclear DNA. Promotion involves multiple exposures at certain intervals to agents that do not damage DNA directly. Many chemical promoters alter cell regulation through signal transduction or gap junction alteration. Promotion leads from benign to malignant tumours. Progression increases the degree of malignancy, Adey (1990).

Hence studies, undertaken a few years after exposure are very unlikely to detect any increase in cancer, even in large populations. In small populations it is impossible because only a small proportion of people who are exposed show increased cancer at younger ages than about 50 to 60 years. As the total cancer rate increases with age in normal populations, especially after 50-60 years, it becomes harder to detect the influence of a specific carcinogen in older decadal age groups.

2.6 Exposure Dilution:

One of the fallacious reasons used to criticize and dismiss EMR epidemiological studies is that there is an ill defined or unknown exposure regime between the occupational or military exposure at an early age and the health survey data decades later. The time delay is essential to allow time for cancer to develop. During the time between the initiating and promoting exposures, and the collection of the health and mortality survey data, a complex exposure regime will be experienced by every person. Rather than creating uncertainty, it is certain what the effect of this is in large groups. The stochastic and randomized nature of this will dilute the differences between the groups and reduce any initial stratification or dichotomization based on the original exposure regime. Hence any observed adverse health effects will be significantly under-estimated. Thus, rejecting the study because of intra-exposure uncertainty is wrong and unjustified. In fact the effects seen can reliably be assumed to be even more elevated with higher significance than the analysis indicates.

The exposure complexity over decades significantly reduces the progressive exposure gradient that might have produced a dose-response curve. Thus any observed dose-response curve for cancer will be a very significant indication of cause and effect, even if it technically fails to achieve p(0.05.

EMR is particularly problematic because it is ubiquitous. Every member of society is exposed to some extent. Epidemiological method aims to minimize the inclusion of confounding factors. Hence exposed populations are compared with controls who are as similar in an as many respects as possible except for the exposure. Hence similarly trained and aged military groups are used as controls for radar exposed groups. In the Korean War Study, radar repairers we chosen as exposed groups and radio and radar operators as the comparison control group. Exposure surveys show that radio and radar operators are in a moderate exposure situation that is far higher than the general public. Hence if EMR exposure increases cancer, then the observed difference between the operators and repairs will not be as great as the real difference between them, and between both of them and the general male public of the same age. In military and industrial situations the lack of a 'no exposure' group is another significant source of exposure dilution.

A job exposure matrix can significantly reduce the uncertainties between groups classified by job. Such a survey was carried out in the Korean War Study. Despite this advanced exposure analysis, the authors try to claim that the observed adverse outcomes cannot be related to radar exposure since it was only based on potential exposure.

The technological advancements in society have exacerbated this further. Exposures occur from radio and TV broadcast towers, powerlines and home appliances. Computers, portable phones, mobile phones and cell sites have significantly raised individual EMR exposures in recent decades. Hence there is no true "no-exposure" population. These and other similar effects are strong sources of exposure dilution.

Residential studies are subject to dilution by 'migration'. Residential samples are diluted by exposed people leaving the area and unexposed people arriving into the study area. This is a significant effect because of the long periods necessary to allow for cancer to develop.

Thus all EMR studies have an extremely high probability of significantly under-estimating the Relative Risks.

The Korean War Study, Robinette et al. (1980), gives a good example. They surveyed exposures in a 5% sample of the "high exposure" repairers groups. They found frequency distributions within the three occupational groups being studied. This resulted in distinctly different distribution and mean Hazard Number for each occupational group that enabled a dose-response exposure gradient to be identified. The health and mortality survey data collected about 20 years later revealed a significant dose-response gradient in the mortality for each of the sailors surveyed when grouped in Hazard Number ranges. Despite 20 years of exposure dilution, the initial exposure dichotomy using occupational group produces elevated and significantly elevated mortality and morbidity differences 20 years later. Many of the elevated Relative Risks don't quite reach the p(0.05 threshold. The very large exposure dilution effect in this case is highly likely to raise them so that they do. This is accentuated by realizing that the comparison or control group was also regularly exposed and so this too produces its own exposure dilution effect, artificially reducing the observed RR and its significance.

An elevation in the rates of a wide range of sicknesses, neurological and cardiac disease and death and cancer incidence and mortality were observed in the Korean War Study, Robinette et al. (1980) and the Moscow Embassy study, Lilienfeld et al. (1978). Neither the authors of these studies, nor the WHO and ICNIRP assessors appreciate the effects of dilution for even though they found significant effects, they sought to dismiss all evidence of adverse effects, even when the data and appropriate interpretations strongly clash with this.

2.7 Dr Goldsmith is critical of ICNIRP standards approach

Eminent, internationally recognized, environmental epidemiologist, the late Dr John Goldsmith, Goldsmith (1997c), states:

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

Dr Goldsmith's own conclusions, Goldsmith (1997b), after reviewing some of the epidemiological data on RF health effects include:

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

Dr Goldsmith sets out guidance on how to use epidemiological research in setting standards, Goldsmith (1992). Dr Goldsmith was critical of approaches taken to date and identified problems including:

Failure to consider both thermal and non-thermal effects especially of non-ionizing radiation.
Interpretation of non-significant results as equivalent to no effect.
Accepting the author's interpretation of a study, rather than examining its data independently for evidence of hazard.
Discounting data on unanticipated effects because of poor fit to preconceptions.
Dependence on threshold assumptions and demonstration of dose response relationships.
Choice of insensitive epidemiological indicators and procedures.
Consideration of each study separately, rather than giving weight to the conjunction of evidence from all available studies.

2.8 Dr Goldsmith reviews EMR epidemiological evidence:

Professor John Goldsmith was one of the world's most eminent environmental epidemiologists. A couple of decades ago when the International Society for Environmental Epidemiology was formed, Dr Goldsmith was invited to give the opening key note address to the first session of the first conference. This illustrates the high standing that he was accorded by the international epidemiological and public health community.

Because of his standing, the editor a new scientific journal, the International Journal of Occupational and Environmental Health, invited Professor Goldsmith to help to launch the first issue of the new journal by providing a significant review paper. The review, headed "Special Contributions" was carefully identified by Dr Goldsmith as an "opinion piece" which reviews and summarizes the "Epidemiologic Evidence of Radiofrequency Radiation (Microwave) Effects on Health in Military, Broadcasting and Occupational Studies".

Goldsmith (1995, 1996 and 1997b) reviews many epidemiological studies of radiofrequency and microwave exposures. Many of these studies show increases of cancer and some show increases of miscarriage and neurological effects. In all of these studies exposures involving heating are extremely rare and mean long-term exposures are a very small fraction of the heating threshold. Goldsmith (1995) concludes:

"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 on the regulatory agencies to include epidemiologic thinking in their work."

This report continues Dr Goldsmith's work and extends his reviews and conclusions. I strongly contend that the approach and conclusions of ICNIRP and the WHO position is methodologically and factually scientifically flawed. They place public health severely and demonstrably at risk.

2.9 The Special case of Broadcast Tower Epidemiological Studies:

For residential studies around broadcast Radio and TV towers the cause and effect relationships can be much more decisive because to the complex nature of the radiation patterns. For example, broadcast antennae usually focus the signal more in one direction than another, towards the major population centres. A cancer rate that is higher on the high emission side than the low emission side is a first indication of a dose-response.

For example, the San Francisco City Department of Public Health, San Francisco (1988), analysed the childhood and adult cancer rates in the vicinity of the Sutra Tower. For childhood All Cancer, leukaemia and brain tumour the whole San Francisco Standard Incidence Ratios were SIR = 0.98, SIR = 1.16 and 0.90, respectively. For a community on the western side of the tower, Sunset East, these rates were SIR = 1.47, SIR = 1.88 and SIR =1.00. For the Noe/Eureka Valley to the easy of the tower they were SIR = 1.78, SIR = 1.94 and SIR = 2.27. Antenna patterns and measurements show higher exposures to the east of the tower (Noe/Eureka Valley) than to the west (Sunset East). Hence there is a dose response relationship.

The radial public exposure pattern around broadcast towers is complex and undulating. If health effect rates follow these complex patterns then they form significant dose response relationships that cannot be explained by any other factor. Hence they show a causal effect.

Radial ground level exposure levels vary with the antenna pattern and the frequency of the carrier. The higher the frequency the better the signal is focussed towards the horizon. The antenna elevation tilt is crucial in determining the position and strength of the main beam when it eventually strikes the ground several km from the base of the tower. Closer to the tower than this the exposure pattern varies with distance as the side-lobes intercept with the ground and the interference between the direct and reflected beams go into and out of phase. An example of an antenna pattern with side-lobes is given in Figure 4, for a VHF antenna.

Figure 4: A typical vertical antenna pattern for a 4-element dipole array at about 98 MHz.(VHF), Units in dB.

The side-lobes have elevation angles of 72, 57, 40, 15 and 8 degrees. For an antenna at 500 m above ground level, the ground level side-lobe peaks occur at 160, 390, 600, 1870 and 3560 m from the base of the tower, with significant troughs of low exposure between them. The amplitudes of the peaks and troughs are very large because of the logarithmic nature of the dB units. It is common to tilt the antenna pattern slightly downwards so that the main beam is directed towards major population centres in the listening and viewing area rather than towards the far horizon.

Figure 5a is street level measurements around the Empire State Building for a 44 MHz VHF transmitter, taken in 1933. These VHF signals have peaks inside 1 mile from the tower which are repeated near 2 4, 8, and 16 miles. Beyond 10 miles the signal generally declines as the inverse square law. Figure 5b shows the way in which VHF radial signal patterns, and hence ground level exposure intensity peaks and troughs, vary with carrier frequency. Figure 6 shows horizontal radiation patterns from UHF and VHF broadcast stations.

Cancer or other health effects which follow these complex radial patterns cannot be caused by any other confounder and hence firmly establish cause and effect. In the North Sydney Study of Hocking et al. (1996) there is a higher than average leukaemia rate in Lane Cove that is in the position of Christchurch in Figure 6b, and lower than average in Willoughby, which is north of the towers. In Sydney the major populations live to the SW of the site. Hence it is likely that a similar antenna pattern to Figure 6b has been used.

Epidemiologists and statisticians who are unaware of these patterns, such as Dolk et al. (1997a,b) and Selvin et al. (1992) have made serious errors in the interpretation of their data. In the first case they assumed a simple inverse square law and a linear relationship in the second. In both cases their cancer data follows a complex radial pattern which closely approximates the radial exposure pattern.

Figure 5: Ground level radiation pattern for (a) the 44 MHz (VHF) signal from the Empire State Building in New York City, from Jones (1933) by merging his figures 6 and 8, and (b) a theoretical set of antennae Antenna height 1000', receiver height 30 ', power 1 kW, Reference data for Engineers, Jordon (1985).

Figure 6: Horizontal antenna radiation patterns showing the relative filed strength for, (a) UHF Digital TV (linear scale) from the Sutra Tower, and (b) 99 MHz VHF for 8 dipole array (logarithmic scale), Ouruhia transmission site, Christchurch, New Zealand.

This indicates a significant dose-response relationship in relation to mean exposure. The authors' weak and dismissive conclusions are favoured by ICNIRP but their data give conclusive evidence of cause and effect between RF radiation and cancer, especially for leukaemia, brain tumor and all cancer. These conclusions are supported by many other studies showing significant increases in these and other cancers in higher than average RF/MW exposures, and by established biological mechanisms.

2.10 Residential Exposure Factor:

There is also a significant difference between direct exposure intensity at a residential site from the tower and personal mean exposure. People spend time inside and out, at home and away. Hence observed health effects need to be related to a residential exposure estimate that takes these factors into account.

When considering cancer, the appropriate exposure metric is the cumulative personal exposure over many years as this relates to cumulative cell damage. The long-term cumulative exposure is the product of time and mean personal exposure. The mean personal exposure is a combination of indoor/outdoor and home/away times. Based on local measurements, the indoor exposure is assumed to be 1/15th of the outdoor exposure. The away exposure is assumed to be 1/30th of the home exposure. Taking the indoor/outdoor ratio as 20:4, the weekly home/away ratio as 108:60 and annual ratio of 44:8. This produces a personal exposure factor or 0.136, which is rounded up to 0.15. Thus the residential exposure factor (REF) is taken to be 15 % of the direct exposure.

McKenzie, Yin and Morrell (1998) took readings in Sydney, Australia, of direct roof level, street level and inside RF exposures from the North Sydney TV Towers. At a particular house these were 3.0, 0.066 and 0.017(W/cm2, respectively. These give reduction factors of 45 and 176 for street level and inside, showing how that the REF of 0.15 is likely to be too high.

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

Cancer latencies and exposure dilution will also reduce the size of the observed RR and its significance, under-estimating the magnitude of any actual adverse health effects in residential studies.

Very Close Proximity:

In residential exposure studies around broadcast facilities the identification of associated health effects in the immediate vicinity of the tower, inside 1 km for example, is very limited. Limitations are produced by a very small area that is unlikely to be able to contain a sufficiently large population to detect elevated or significantly elevated effects. The area is smaller than 3.14 km2 because a portion of the area is taken up with the facility itself, which often requires a very tall mast with guy wire bracing. This means that the exposed population is quite small. In rural areas it is very small. A further factor is the radiation pattern must involve high powered emissions and the mix of VHF and UHF transmissions since VHF antenna have side lobes that produce high exposures inside 1 km and UHF antennas don't.

Hence the only circumstances that are likely to show high cancer rates near a broadcast tower is one that combines a high population density, a VHF signal and an RF sensitive cancer. The only place in the UK study of Dolk et al. (1997a,b) that had a high cancer rate near the tower, was Sutton Coldfield. It is the only high power tower in a highly populated area that had VHF stations on it.

2.12 A more appropriate approach:

These more fundamental considerations of method and approach, with more realistic radiation exposure patterns and placing epidemiology well above biological effects, strongly supports a fresh approach and significantly altered conclusions. The WHO and ICNIRP assessments suffer from all of the problems identified by Dr Goldsmith and Sir Austin Bradford Hill. This critique attempts to correct this and to incorporate epidemiological evidence into processes for setting public health protection standards.

A scientifically objective and open-minded approach should start with an open question:

What is the epidemiological evidence of adverse health effects, and what does this evidence suggest in terms of potential, probable or actual adverse health effects?

3. History of the RF-Thermal View:

A long-held Western mind-set:

ICNIRP follows a long-held Western position that arose after the Second World War when no epidemiological studies of radio and radar exposed populations were available. In this situation it is appropriate to deal with the known effect of tissue heating and to determine the levels of acute exposure which would avoid burns and shocks. In the 1970's and 1980's time had elapsed between initial exposure and the potential development of chronic health problems. Studies were carried out, significant effects

were observed, but the thermal mind-set was so well established, reinforced by the Cold War politics between the U.S.A. and the U.S.S.R. so that these results were dismissed or hidden by government officials changing the conclusions of epidemiological studies. Through this period the West became focussed on the single "proven" biological effect of RF/MW, Tissue Heating.

It is demonstrable that acute high level exposure causes Tissue Heating. Exposed people and animals had their temperature measured and it rose, reliably and repeatably. It makes sense. Absorbed energy raises the temperature as a function of the 'aerial' properties of the object (person), relative to the wavelength of the electromagnetic wave.

Given the central and dominating role of the RF-Thermal View it is important to trace its history. In the period immediately following the Second World War, when radio and radar had come into widespread use for the first time, there was no epidemiology to challenge the developing view that Tissue Heating was the only possible effect. Early on there were anecdotal, case-by-case reports of leukaemia, ocular defects, reproductive problems, heart problems and neurological symptoms of tiredness and headache. For example, McLaughlin (1953), Cleary and Pasternack (1966), Rosenthal and Beering (1968), Forman et al. (1982), and Archimbaud et al. (1989). Some of these involved quite high acute exposures. Most were relatively isolated and they were claimed not to be confirmed to be RF/MW related.

The U.S. Tri-Service Program:

The conviction that the only possible effect of RF/MW exposure is tissue heating is sourced largely from the Tri-Service Program. One of the primary aims was to determine the thermal threshold so that exposed personnel could be protected from dangerous over heating. This is documented through Steneck et al. (1980) and published conference proceedings from the United States. Steneck et al. document the detailed history of the development of the U.S. standard C95.1. They note that Dr John T. McLaughlin, a medial consultant of the Hughes Aircraft Corporation assessed the research into the ill effects of radar exposure. He wrote a report and sent it the military. It listed purpura hemorrhagica (internal bleeding), leukaemia, cataracts, headaches, brain tumors, heart conditions and jaundice as possible effects.

No weight was given to this report and calculations proceeded to determine the heating exposure that people could tolerate, based on their ability to deal with solar radiation. After some basic arithmetic errors were corrected a figure of 10mW/cm2 was arrived at in about 1960. This became the basis of standard C95.1 ten years later. It was supported by a large body of research that was coordinated through the Tri-Service Program. Steneck et al. summarized this research, pointing to the high acute exposures that were involved.

3.3 The U.S./U.S.S.R. double standard:

In 1970 Dr Leo Inglis presented a paper to an IEEE forum on EMR entitled "Why the double standard - a critical review of Russian work on the hazards of microwave radiation", Inglis (1970). He notes that a major difference between the U.S. and Soviet work was:

"In the U.S., the thermal effects are generally believed to be the only ones of significance; other contentions are usually dismissed as lacking a provable basis. In the U.S.S.R., non-thermal effects are considered the most significant and are overwhelmingly the ones most studied."

The non-thermal effects identified by the Soviet researchers were often referred to as the "Radiofrequency Sickness Syndrome" or the "Microwave Syndrome". It has symptoms of functional changes in nervous and cardiovascular systems, such as headaches, fatigue, irritability, sleep disturbance, weakness, decreased libido, chest pains and bradychardia. Hypotonia (muscle weakness) and related symptoms had been reported in the USSR (Gordon, 1966), France (Deroche, 1971) and

Israel (Moscovici et al., 1974). Western scientists rejected these symptoms as "subjective". A set of them have now been identified with cell phone usage, including a significant dose-response relationship, Mild et al. (1998).

3.4 Determining and challenging the thermal threshold:

The RF-Thermal View dominance is confirmed in the proceedings of the 1974 conference on "Biological effects of Non-Ionizing radiation", held at the New York Academy of Sciences, 12-15 February 1974, and published in Annals of the NY Academy of Sciences, February 28, 1975. The conference chairman was Dr Paul E. Tyler of the EMR project office in the U.S. Dept of the Navy. His opening remarks include comments about the Tri-Service Program and the very high levels of exposures generally used, Tyler (1975). He states:

"After I had read and analyzed of the publications for this program (the Tri-Service Program), I was left with the feeling that the research was conducted with the preconceived idea that all of the effects were thermal in nature. It appears that the protocols were designed only to determine gross thermal effects."

"Although the Tri-Service research addressed essentially only the problem of thermal hazard, the idea that the sole hazard was thermal became dominant, and in the early 1960's, an air of complacency settled over this country. At the end of the Tri-Service Program in 1960, United States research in this area decreases to a very low level and remained there for the next decade."

The general acceptance or complacency about the RF-thermal view was scientifically challenged time and time again throughout this period. For example, Dr Adey gave the introductory paper to this 1974 conference, Adey (1975), on the effects of EMR on the nervous system. In this paper he states:

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

It is a very strong statement to say that interactions can be reliably produced in the CNS without the heating of tissues. Dr Adey refers to the work of König and Wever in Germany and to work from his own laboratory, on behavioural effects, such as changed reaction times and altered circadian rhythms in ELF exposures. These effects were associated with induced electric field gradients in monkey phantoms in the range 0.1 to 0.01(V/cm. The effects were also linked to changes in EEG and significant effluxes of calcium ions and GABA, Kaczmarek and Adey (1973). During the 1970's was as shown that calcium ion efflux occurred at non-thermal exposure levels and was primarily related to modulation frequency, i.e. a non-thermal, possible resonant, biological mechanism.

Two large epidemiological studies were carried out in the 1970's, Lilienfeld et al. (1978) and Robinette et al. (1980) in the middle of the Cold War. These found small but significant increases in cancer, cardiac problems and neurological symptoms. However, the authors were under strong pressure, for a range of reasons, to not relate these results to the radar exposure. In one case, Lilienfeld et al., the U.S. State Department case officer, Dr Herbert Pollock, actually changed the conclusions, Goldsmith (1996).

Tell and Harlen (1979) outline the Thermogenic properties of RF/MW. From a number of studies that recorded rectal temperatures under various exposure conditions. This was to give guidance in setting RF/MW exposure standards. The 10 mW/cm2 standard was confirmed as protecting from temperature rise of less than 1(C.

An official attempt to declare EMR carcinogenic (1990):

In 1990 an internal review team of the U.S. E.P.A. recommended that ELF be classified as a probable human carcinogen and RF/MW as a possible human carcinogen. Under pressure from the Bush White House, EPA administrators changed the conclusions of the review and the classification never became official EPA policy, Sibbison (1990). The rationale was based on the preferred public policy stance "We don't want to scare the public". Public health protection was not considered as important.

3.6 U.K.'s NRPB retains the RF thermal view (1991):

In May 1991 the United Kingdom's NRPB issued a series of reports on EMR, which included a report on the Biological Effects of ELF, Sienkiewicz, Saunders and Kowalczuk (1991) and the Biological Effects of RF/MW, Saunders, Kowalczuk and Sienkiewicz (1991). This second report reviews many cell and animal studies that used thermal exposures and produced some observable effects. These thermal and behavioural effects were not seen when SAR's dropped below 4 W/kg. They don't find anything reliably significant in the long-term mouse study of Guy et al. (1985) in which found a significant increase in primary malignant tumors at an SAR of 0.4 W/kg. The U.S. E.P.A. internal review team found this study much more relevant and used it as an important support for their recommendation to classify RF/MW as a possible human carcinogen, McGaughy et al. (1990). Epidemiology played no role at all in the NRPB review, which was solely concerned with biological mechanisms. However it did play a major role in the EPA review.

U.S. IEEE/ANSI review retains RF Thermal View (1993):

In 1993 the U.S. based IEEE published their revision of the IEEE/ANSI RF/MW standard C95.1-1991, IEEE(1991). This report is solely about thermal biophysical interactions that create heat and the SAR levels that will avoid dangerous heating, burns and shocks. The assessment criteria all related to thermal absorption mechanisms. The primary revision is a relaxation of power density limits for all body parts except eyes and testes. This relates to a revised calculation of the 6-min dose that produces an SAR of 0.4 W/kg.

Conclusion:

Of all the major western authorities who are responsible for setting RF/MW exposure standards, the only body which is departed from solely considering thermal effects, was an internal review team of the U.S. E.P.A.. They also considered epidemiologic and animal evidence at non-thermal levels that did involve increases in cancer. However, they were not allowed to retain their recommended carcinogenic classification because EPA administrators bowed to political pressure.

4. The ICNIRP and WHO Approach in the 1990's:

Introduction:

The world Authorities, WHO and ICNIRP, in the early and late 1990's, also retain the RF-Thermal View and recommend guidelines based on avoiding tissue heating. They have undertaken more comprehensive reviews that considered epidemiological and long-term animal evidence. Their reviews of this evidence did not sway them from the RF-Thermal View. A detailed analysis of their reviews and the research papers cited reveals evidence of predetermination to reject any evidence that contradicted this view. The long history of holding the RF-Thermal View has brought extensive comfort and complacency. This is partly through the great degree of precision, repeatability and reliability of SAR calculations and heat protection. This is such a long-held view that it has become a mind-set. This way of thinking makes it

extremely difficult to move review teams from the RF-Thermal View to the Public Health Protection approach. It requires a complete change of thought and approach to move from a comfortable and well understood mechanism to the much more complicated consideration of epidemiological data derived from complex human situations. But, as Dr Lilienfeld reminded us:

"The proper study of man is man".

The Constructive Dismissal Approach:

In order to maintain the RF-Thermal View against the extremely strong evidence from epidemiology, animal experiments and of non-thermal mechanisms, the WHO and ICNIRP assessors and their colleagues have developed as set of dismissive methodologies. These include:

Maintaining that the RF-Thermal view as the "consensus of science". This allows the biological mechanism to dominate and epidemiology and animal evidence is dismissed.

Maintaining a contrast between Ionizing radiation and Non-ionizing radiation.

Moving the level of evidence goalpost where for a study to become "evidence" it must first be replicated, whereas in the past each study was evidence and to "establish" a biological effect replication was required.

Promoting strict sets of scientific criteria which are proposed as being necessary for reliable use of the results, e.g. the Bradford Hill "criteria", instead of "viewpoints", and Dr Martin Meltz's 13 experimental criteria for testing genotoxicity, Meltz (1995). In this way all non-thermal evidence is rejected.

Citing studies which are too small and have small follow-up periods so there is little or no opportunity for cancer to develop, as evidence that radar exposure does not cause cancer.

Citing studies which do show significant increases in cancer as showing no evidence of increases in cancer.

Preferring to simply quote the conclusions of papers and reports that state that there were no adverse effects found, while failing to recognize that the data and analysis within the documents do show significant associations, including significant dose-response relationships.

Dismissing epidemiological studies on the grounds that populations and exposures are not well defined. Lilienfeld explains that this is a difficulty but results are still relevant and important.

Dismissing research results one by one and failing to assemble and interpret the whole pattern of research results - the divide to conquer approach.

All of these are demonstrated methods used by WHO and ICNIRP which amounts to a systematic approach to wrongly dismiss evidence of effects, i.e. Constructive Dismissal.

The evidence of a leading WHO/ICNIRP member:

In the 1990's a major WHO review was published, WHO (1993). The latest ICNIRP Guideline assessment has been published in 1998, ICNIRP (1998). Both of these maintain the RF-Thermal View. A leading scientist, Dr Michael Repacholi, was involved as the technical editor of the WHO review and in chairing both the WHO review team and ICNIRP until April 1996. He is now is Chairman emeritus of ICNIRP.

Insights into his mind-set, which is reflected by WHO and ICNIRP, is seen in his evidence in a New Zealand cell site case in November 1995, the MacIntyre Case. In this case the local residents of the suburb of Ilam, in Christchurch, New Zealand, appealed a City Council decision to allow a cell site to be installed on the roof of an old suburban movie theatre in the middle of their community. The site would irradiate a number of local residences and the local kindergarten that was about 70 m from the site.

Dr Repacholi appeared in this case as an expert witness on behalf of BellSouth Ltd,. In sworn testimony contained in his evidence-in-chief he states: (Note that the emphasis on 'any' is Dr Repacholi's)

"To produce any adverse effect, RF exposure above a threshold level must occur. This threshold level is the RF exposure needed to increase tissue temperature by at least 1(C."

"Multiple exposures to sub-threshold levels of RF have not been found to have any adverse health impact."

"Exposure to RF fields has not been established to cause cancer."

"No accumulation of damage occurs to tissues from low level (sub-threshold) RF exposures".

"The science has also not found any evidence for adverse health effects from repeated exposures at levels below the threshold."

Dr Repacholi's evidence is fully consistent with the ICNIRP conclusions outlined above and were referenced by Dr Repacholi to the WHO/IRPA/UNEP review, WHO(1993). To back up Dr Repacholi's claim that the RF-Thermal position was the "consensus of science", Dr Repacholi referenced WHO (1993), for which he had a major responsibility.

4.4 Evidence vs Established:

Around the time of this court case Dr Repacholi was supervising a research project in Australia in which genetically modified mice were exposed to a sub-thermal dose of a GSM cell phone signal, for two half-hour periods per day. This gave SARs averaging 0.13 to 1.4 W/kg during the exposures, giving a daily average range of 0.005 to 0.058W/kg. They concluded that "Lymphoma risk was found to be significantly higher in the exposed mice than in the controls (OR = 2.4, p=0.006, 95%CI: 1.3-4.5)."

Hence Dr Repacholi's own research results, which were published after the NZ court case was concluded, contradict his claims in court. In an industry-sponsored press conference in Vienna at the time of the Vienna EMR Workshop in October 1998, Dr Repacholi stated that there was no evidence of adverse effects from GSM cell phones. When questioned in the Workshop about his own research results, he took the position that a scientific experiment can only be considered as "evidence" once it has been independently replicated. This is not the definition of "evidence" which most people and most courts accept. A research result is "evidence". Replication is required in order to establish a biological effect. Both the original and the replicate experiments contribute evidence with amounts to the establishment of a biological effect.

Two other long-term rodent studies have observed increases of cancer in exposures involving RF/MW. Chou et al. (1992) chronically exposed rats to a non-thermal radar-like signal, observed a significant increase in benign tumors and highly significant increase in primary malignant tumors, RR=3.6, 95%CI: 1.34-9.7, p=0.0036. Vijayalaxmi et al. (1997, 1998) exposed cancer-prone mice to a 2.45 GHz continuous wave signal and observed a 41 % increase in tumors and highly significant ($p<0.01$) 12.5% increase in chromosome damage in bone marrow and blood. Hence the evidence consists of three studies in which RF/MW radiation significantly increases cancer in rodents, including one which also associates this with

chromosome damage. The chromosome damage is evidence of genotoxicity, the ability to damage DNA and cause mutations and cancer.

These projects serve to illustrate one of the fundamental problems with EMR research. While three independent laboratories have observed increases in cancer in rodents with non-thermal RF/MW exposures, all rodent species were different, all exposure regimes were different. One was a GSM carrier of 900 MHz pulsed at 217 Hz for 2 periods of half an hour per day with mean daily SAR in the range 0.005 to 0.058 W/kg. Two used 2.45 GHz carriers but the first was pulsed at 800pps, modulated at 8 Hz, and involved 21.5 hr of daily exposure with a daily mean SAR in the range 0.13 to 0.36 W/kg. The second used a continuous wave exposure for 20 hr/day with a daily average SAR of 0.83W/kg.

For those, like ICNIRP, who maintain the RF-thermal view, these projects do not provide "evidence" that RF/MW produces cancer in rodents because every experiment has differences in animals and exposure regimes and none have been precisely replicated.

Alternatively, taking the more traditional scientific and legal approach, there are three studies, from independent laboratories, which show significant increases in cancer in rodents at non-thermal levels of exposure to RF/MW radiation. Hence there is animal evidence to support the epidemiological evidence that RF/MW exposed populations develop significantly higher rates of cancer incidence and mortality. Both the animal evidence and the human evidence covers a wide range of RF/MW exposure conditions. Across the same frequency range multiple independent laboratories have observed significant DNA-strand breakage and enhanced chromosome aberrations. Hence there is strong evidence that RF/MW is genotoxic, mutagenic, carcinogenic and teratogenic in animals and people at non-thermal levels of RF/MW exposure.

4.5 Ionizing Radiation vs Non-Ionizing Radiation?

The history of EMR shows that it has always been treated differently from chemicals. One reason for this is an argument related to radiation. The argument runs as follows:

"Ionizing radiation has the photon energy to break chemical bonds and produces free radicals in cells. These enhance the damage to DNA and other macromolecules which increases the risk of cancer. Non-Ionizing radiation does not have the photon energy to break bonds and produce free radicals. Therefore non-ionizing radiation cannot damage DNA and cannot cause cancer."

Many chemicals cause cancer, such as benzene, without involving ionization. We can immediately note that UV-B radiation is non-ionizing but it is known to cause cancer, particularly skin cancer. Among a number of identified mechanisms is UV's ability to cause mutation in the tumor suppressor gene p53, Leffell (2000). This proves that non-ionizing radiation does cause cancer and acts through altering the p53 gene activity. This illustrates the point that cancer is caused by both enhancing cell damage and by inhibiting cell damage repair rates and efficiencies. UV radiation also enhances free radical formation, Collins, Poehler and Bryden (1995) and Jurkiewicz and Buettner (1994, 1996).

There is evidence that non-ionizing radiation dose enhance free radical activity. Phelan et al. (1992) investigated membrane fluidity in Melanin-containing cells that were exposed to low level microwave radiation, 1 hr at 0.2 W/kg. They conclude:

"The data indicate significant, specific alteration of cell-membranes was due, at least in part, to the generation of oxygen radicals".

Lai and Singh (1997) showed that significant microwave induced DNA-strand breakage could be eliminated through the application either of melatonin, a natural free radical scavenger, or of PBN, a spin-trap compound. This indicates that melatonin reduction and enhanced free radicals is a plausible mechanism.

Hence UV-B and RF/MW non-ionizing radiation are both associated with enhanced free radical activity in cells, either by enhancing the free radicals or by reducing the free radical scavenger, melatonin. Thus the effect on ionizing and nonionizing radiation can be very similar, but may involve different mechanisms. Either way, the effect is the same. They both produce genetic damage and are carcinogenic.

4.6 Ionization is not a prerequisite for cancer:

Many generations of medical biologists and toxicologists do not assume that ionization is a necessary prerequisite for cancer producing agents, since thousands of chemicals are cancer producing agents without the involvement of ionization. Chemicals are carcinogens, Baxter (1995), when they:

Alter DNA, initiating cancer.
Corrupt cellular growth control, thus acting as cancer promoters.
Act with other carcinogenic agents, working as Co-promoters of cancer.

There is evidence that EMR acts in all of these ways.

4.7 Examples of extreme lengths gone to retain the RF-Thermal view:

ICNIRP and individual national authorities are so wedded to the RF-Thermal view that they not only attempt to reject studies by claiming weakness and inconsistencies, they also descend to use demonstrably incorrect scientific statements.

4.7.1 ICNIRP misquotes results:

In the ICNIRP (1998) cancer assessment the following statement appears:

"More recent studies have failed to show significant increases in nervous tissue tumors among workers and military personnel exposed microwave fields (Beall et al. 1996 and Grayson 1996)."

Statistical significance is defined as p(0.05 and/or a 95% confidence interval where the lowest side of the range is close to 1.0 or higher. Beall et al. studied the increase in brain tumor with exposure to computer monitors (VDTs). Beall et al.'s abstract states:

"Other results included and elevated OR for 10 or more years of employment in engineering/technical jobs [OR = 1.7; 95% confidence interval (CI) = 1.0-3.0] or in programming jobs (OR = 2.8, 95%CI= 1.1-7.0). The OR for glioma for all subjects who had accrued 5 years of programming work 10 years before each case's death was 3.9 (95%CI = 1.2-12.4)."

The abstract reports significant increases in brain tumors. The data in the paper show that for engineering/technical jobs there is a dose-response for brain tumor death and years of work, p=0.07, and for computer programming, p=0.04. Thus the paper does show significant increases in brain tumor death from EMR exposure with dose-response increases and one significant dose-response relationship.

Grayson (1996) investigated a large sample (880,000 with 11.17 million p-yrs) of U.S. Air Force personnel, some of whom were occupationally exposed to EMR and ionizing radiation, with exposure assessed through a job exposure survey. From this very large sample only 275 were exposed to RF/MW,

94 of whom developed brain tumors. This yielded OR = 1.39, 95%CI: 1.01-1.90. This is a statistically significant result.

ICNIRP's statement about Beall et al. (1996) and Grayson (1996) is demonstrably scientifically wrong and misleading. It reveals a strong predetermination to dismiss evidence of effects.

4.7.2 Recently in New Zealand a similar situation occurred:

Late in 1998 the Royal Society of New Zealand released a review report on radiation health effects. Being the Royal Society it was assumed that it would be a high quality, up to date and authoritative publication. The report was entitled "Radiation and the New Zealand Community - A scientific Overview". The major contribution of two staff members of the National Radiation Laboratory is acknowledged. The report contains statements about the health effects of EMR being totally wrong and misleading, and reveal a determination to ignore evidence of adverse health effects.

The N.Z. Royal Society report takes the thermal view and at one key point makes the claim in relation to ELF EMR, p67:

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

It is glaringly untrue to state that "no effects due to occupational exposure have been reported". Many hundreds of studies have reported ELF biological and human health effects. Three have even been carried out in New Zealand. Preston-Martin et al. (1993) found for all brain cancer elevated risks were found for electrical engineers (OR= 8.2, 95%CI: 2.0-34.7) and electricians (OR = 4.6, 95%CI: 1.7-12.2). Beale et al. (1997) investigated health effects near high voltage powerlines in Auckland and found significant linear dose-response relationships for some health and psychological variables and magnetic field exposure. Dockerty et al. (1998) studied childhood cancers in relation to EMF exposure. Electric blankets produced elevated adjusted rates of leukaemia (OR= 2.2, 95%CI: 0.7-6.4), CNS cancer (OR = 1.6, 95%CI: 0.4-7.1) and other solid cancers (OR = 2.4, 95%CI: 1.0-6.1). Leukaemia risk was highest when bedroom magnetic field was (0.2(T compared with (0.1 (T, (OR= 15.5, 95%CI:1.1-224).

A totally independent team of Swedish medical scientists, reviewed over 100 epidemiological papers, an over 300 studies in total, published up to July 1994, Hardell et al. (1995). They concluded:

"Epidemiological and experimental studies concerning extremely low frequency electromagnetic field exposure and malignant diseases published up to 1 July 1994 were evaluated to assess the possible carcinogenicity of electromagnetic fields and the scientific basis for environmental and occupational standard setting. We concluded that there are possible associations between

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 and,

an increased risk of breast cancer, malignant melanoma of the skin, nervous system tumours, non-Hodgkin lymphoma, acute lymphatic leukaemia or acute myeloid leukaemia and certain occupations.

There is no scientific basis for occupational or environmental standard setting for low frequency electric or magnetic fields."

The final statement about standards setting is based on the lack of good exposure measurement in most occupational studies and the lack of dose response relationships in order to determine an ELF field level which will avoid the observed association to risk factors. The fact that the mean daily exposure of even the highly exposed workers is a small fraction of the current standards demonstrates the gross inadequacies of the standards and guidelines. The ICNIRP (1998) Guideline recommends a 24-hr occupational limit of 500(T and residential limit of 100(T.

Many more ELF health studies have been published since July 1994. Four laboratories have shown that ELF below 1.2 (T reduces the oncostatic protection of melatonin in human breast cancer cells, with a threshold of around 0.1 to 0.2(T. Also 4 laboratories have shown the ELF radiation is associated with significant increases in DNA strand breaks. One replication is usually necessary to confirm a biological effect. Four independent studies definitely establish a biological effect. These biological effects are biological mechanisms which confirm the plausibility of the epidemiological associations found in Hardell et al. (1995), giving the classification to the level of probable or actual human carcinogen with the addition of the post-1994 studies.

Residential powerline studies on childhood leukaemia, such as Feychting and Ahlbom (1993), found for a cut-off point of 0.2(T a Relative Risk of RR=2.7 (95%CI: 1.0-6.3) and a trend with p=0.02. For a cut-off point of 0.3(T, RR= 3.8 (95%CI: 1.4-9.3, for the trend p= 0.005 . By pooling data from Norwegian and Swedish studies, Feychting et al. (1995) found a relative risk of RR=2.0 (95%CI: 1.0-4.1) for a 0.2(T cut-off and RR=5.1 (95%CI: 2.1-12.6) for 0.5(T of, a significant dose response relationship, p=0.03.

Hence it is now possible to determine that a current threshold level for no observed effect for childhood leukaemia and breast cancer is near 0.1(T. This is 1000 times below the current guideline and has yet to have a safety factor incorporated.

Thus it is grossly wrong for the report of Royal Society of NZ to claim that "no effects have been reported from occupational exposure" and "nor are there any indications of adverse health effects on humans, other than from spark discharges and shock from direct contact". This is so grossly misleading and dishonest, that it puts this report's credibility, and that of the Royal Society of New Zealand, seriously at risk. In coming to its conclusions the Royal Society of NZ relied heavily on the Director of the National Radiation Laboratory, Dr Andrew McEwan.

Scientists and the public expect much more scientific accuracy and integrity from Government employees who advise the Minister of Health, and of the Royal Society.

4.7.3 Canada does it a little better:

In contrast, the Royal Society of Canada in their March 1999 report "Potential health risks of Radiofrequency fields from wireless telecommunication devices", carried out a detailed review of biological mechanisms. They involved current researchers in the review team who concluded that most RF exposures used in experiments exceed the limits set in the Canadian Safety Code 6 (SAR = 0.08 W/kg). They also state:

"However, effects on cell proliferation, Ca2+ efflux, blood brain barrier (BBB) permeability, behaviour and ornithine decarboxylase (ODC) activity have all been repeated in independent laboratories. Because these effects occur at exposures not thought to elicit thermal effects, it is likely that these effects, even if they also occur at higher exposure levels, are non-thermal biological effects."

This critique will show that some key non-thermal biological mechanisms are well established by replication in many independent laboratories. These established biological mechanisms are totally supportive of and consistent with a large body of epidemiological evidence, which includes many statistically significant associations and dose-response relationships. In doing so this critique will show that the ICNIRP assessment takes a predetermined dismissive approach that is highly selective and unscientific. It even involves deliberate and repeated misquoting and misrepresentation of study results. It becomes clear that the thermally based guideline is being defended at all costs, even at the cost of putting public health severely at risk all around the world.

5. ICNIRP's 1998 assessment of the RF/MW Guideline:

ICNIRP Review Conclusions:

The failure to use epidemiological evidence as the primary source and animal evidence secondarily, and the predetermination to retain of the RF-Thermal View is seen in the conclusions of ICNIRP (1998), p507:

“Data on human responses to high-frequency EMF that produce detectable heating have been obtained from controlled exposure of volunteers and from epidemiological studies on workers exposed to sources such as radar, medical diathermy equipment and heat sealers. They are supportive of the conclusions drawn from laboratory work, that adverse biological effects can be caused by temperature rises in tissue that exceed 1(C. Epidemiological studies on exposed workers and the general public have shown no major health effects associated with typical exposure environments. Although there are deficiencies in epidemiological work, such as poor exposure assessment, the studies have yielded no convincing evidence that typical exposure levels lead to adverse reproductive outcomes or an increased cancer risk in exposed individuals. This is consistent with the results of laboratory research on cellular and animal models which have demonstrated neither teratogenic nor carcinogenic effects of exposure to athermal levels of high frequency EMF.”

ICNIRP conclusions are scientifically wrong:

The Constructive Dismissal approach is evident. The thermally-based guideline is retained. Apart from the statement about there being adverse effects of tissue warming, every other statement made is scientifically challengable and misleading.

For example: "epidemiological studies on exposed workers and the general public have shown no major health effects associated with typical exposure environments".

Epidemiological studies of exposed workers and the general public have shown significant increases in major health effects, including dose-response relationships which are indicative of a casual effect. This includes multiple studies on miscarriage and significant dose-response relationship between microwave exposure and first trimester miscarriage, Ouellet-Hellstrom and Stewart (1993). Many laboratory studies on cells and animals have demonstrated athermal carcinogenic and teratogenic effects, Chou et al. (1992), Repacholi et al. (1997), Vijayalaxmi et al. (1997) and Magras and Xenos (1997). These statements are demonstrably incorrect and misleading. It is conclusions such as these that continue to put thousands of lives at risk in New Zealand alone, and millions at risk around the world. Many occupational studies have found significant increases in cancer, e.g. Lilienfeld et al. (1978), Robinette et al. (1980), Milham (1985 a,b, 1988), Thomas et al. (1987), Demers et al. (1991), Cantor et al. (1995), Szmigielski (1996), Grayson (1996), Beall et. al. (1996). Residential studies showing significant increases in cancer from RF/MW exposure, some of which show significant dose-response relationships include: Hocking et al. (1996), Selvin et al. (1992), Dolk et al. (1997a,b), and Michelozzi et al. (1998).

In the middle of the frequency spectrum, where the ICNIRP Guideline exposure level is at its lowest, 200 (W/cm2, there are residential epidemiological studies that give dose-response relationships for adult and childhood leukaemia with a threshold near 0.025 (W/cm2. This is 8,000 times lower than the ICNIRP Guideline. In Switzerland, significant sleep disturbance was observed at an RF exposure level of 0.0004(W/cm2, Altpeter et al. (1995). For this RF frequency (6.1-21.8 MHz) for which the ICNIRP Guideline is about 200-1000(W/cm2. The adverse effect occurs at a factor of 0.5 to 2.5 million times lower than the Guideline.

5.3 The ICNIRP 1998 Guideline:

By ignoring the epidemiological evidence ICNIRP settles on a thermally-based guideline by accepting a thermal threshold of 4 W/kg, a workers safety factor of 10 (0.4 W/kg) and a further factor of 5 for the general public (0.08 W/kg). This is plotted in Figure 7 in terms of electric field strength and exposure intensity, as a function of carrier frequency.

Figure 7: The ICNIRP (1998) Guideline for public and occupational exposures as a function of carrier frequency. On the left the units are electric field strength (V/m) and on the right the exposure intensity ((W/cm2). The three plateau regions above 1 kHz are 87, 28 and 61 V/m, corresponding to 2000, 200 and 1000 (W/cm2, respectively.

The ICNIRP methodology and use of scientific research is blatantly incorrect. A long-held mind-set dominates the EMR radiation authorities. It needs to be exposed and changed.

5.4 A sports analogy of the different approaches:

5.4.1 The situation of conflict:

The primary issue in this report is the ICNIRP retention of a thermally-based guideline in the face of the dominant international approach which requires the use of epidemiological evidence when setting public health protection standards. A phrase comes to mind when reading the ICNIRP Guideline report, "They appear to be playing their own game and making up the rules as they go along". This analogy appears to be helpful. These two approaches are like two different games.

5.4.2 The ICNIRP Game:

ICNIRP is playing its own game and setting its own rules. It is the game that is played by national authorities which, as a team, they feel very comfortable with it. The name of the team is "The Consensus of Science". However, it involves quite a small and very select team that includes national experts who come from national authorities who subscribe to the rules of the ICNIRP game. In the ICNIRP game the first rule is that there is only a tissue heating effect from RF/MW exposure. You must agree with this rule to play the ICNIRP game. As a consequence of this rule, in the ICNIRP game, all other biological effects are not real and any epidemiological study that shows an effect with non-thermal exposure, must be faulty and will be rejected. In other words, if you break this rule you are out of the game. In this game it is fine to change the rules about acceptable significant, what is evidence, and criteria for how a biological effect is established. In this game a study does not provide evidence until it has been exactly replicated. You set up 13 criteria which must be achieved for an experiment to be reliable, for example Meltz (1995). If even one criteria is breached then you can reject the findings. Similarly the ICNIRP team uses the Bradford-Hill Criteria to criticize and reject all epidemiological studies. One criticism, valid or not, is sufficient to reject a whole study.

For a long time Dr Repacholi has been the captain of the ICNIRP team, he has helped to make and change the rules. His own study shows a significant non-thermal effect. He was able stay in the team by changing the rules of evidence.

The Public Health Protection Game:

In the Public Health Protection Game the first rule is that public health protection is paramount. Standards are based on public health studies, i.e. epidemiology. Epidemiological evidence is sufficient to set standards where there are dose-response relationships or when studies have shown significant adverse effects a sound study which involves exposures below those which have found effects, determines that there is no evidence, not even an elevated risk, at that exposure level. Then exposure associated with the lowest reported adverse effect has a safety factor is applied to deal with the uncertainty. Avoidance action and experiment are a vital part of this game.

Avoidance action is taken long before scientific proof of cause and effect is reached. This is because it is recognized that many disease agents cause sickness and death years or even decades after the initiating exposure. Unnecessary delay is avoided and action is taken to protect public health, once the evidence is judged sufficient under the circumstances. A reversible adverse effect can be treated differently than permanent damage that hastens disease and death, such as miscarriage, congenital malformation, brain damage or cancer. The Bradford-Hill viewpoints inform this decision-making, Hill (1965).

6. Non-Thermal Biological Mechanisms

Veteran EMR biological researchers Dr Ross Adey, Dr Carl Blackman, and Dr Alan Frey and eminent epidemiologist, the late Dr John Goldsmith, cite sound evidence which totally refutes the claim that there are no established non-thermal biological mechanisms.

6.1 Dr Ross Adey directly challenges the thermal view:

Dr W. Ross Adey is one of the world's most respected veteran EMR researchers. His pioneering work on neuroscience gives deep insights into biological functions and processes. The following is the abstract from his paper "Frequency and Power Windowing in Tissue Interactions with Weak Electromagnetic Fields", Adey (1980):

"Effects of non-ionizing electromagnetic (EM) fields that raise tissue temperature in general differ very little from effects of hyperthermia induced by other means. However, fields raising tissue temperature orders of magnitude less than 0.1(C may result in major physiological changes not attributable to raised temperature per se.

These weak fields have been observed to produce chemical, physiological, and behavioral changes only within windows in frequency and incident energy. For brain tissue, a maximum sensitivity occurs between 6 and 20 Hz. Two different intensity windows have been seen, one for ELF tissue gradients around 10-7 V/m, and one for amplitude modulated RF and microwave gradients around 10-1 V/m. The former is the level associated with navigation and prey detection in marine vertebrates and with the control of human biological rhythms; the latter is the level of the electroencephalogram (EEG) in the brain tissue.

Coupling to living cells appears to require amplifying mechanisms that may be based on non-equilibrium processes, with long-range resonant molecular interactions. The cooperative processes are now recognized as important in immune and hormonal responses, as well as in nerve excitation. Polyanionic proteinaceous material forming a sheet on the cell membrane surfaces appears to be the site of detection of these weak molecular and neuroelectric stimuli."

Professor Adey succinctly summarizes EMR research at that time. He does not claim, in the body of the paper, that the two observed intensity windows are the only intensity windows, but that these are intensity windows that have repeatedly been shown to have significant effects.

In his conclusion Dr Adey directly challenges the thermal view.

"Too many physicists and engineers cling desperately to the thermal models as the alpha and omega of bioeffects from nonionizing radiofrequency fields, shunning the exquisite beauty of long-range molecular interactions and resonant processes in biological macromolecules."

In Adey (1993) intercellular communications are described as "whispering between cells". Dr Adey notes new work that involves free radicals:

"that may also participate in highly cooperative detection of weak magnetic fields, 'even at levels below thermal (kT) noise'."

The key role of resonance and tuning is addressed.

"In recent studies (Grundler and Kaiser (1992)), they noted that the sharpness of the tuning increases as the intensity of the imposed field decreases; but the tuning peak occurs at the same frequency when the field intensity is progressively reduced. Moreover, clear responses occur with incident fields as weak as 5 picowatts/cm2."

A 5 pW/cm2 signal is billions of times below the ICNIRP guideline for GHz signals. The studies cited by Dr Adey show the fundamental biological role of frequencies, tuning and resonance. Kaczmarek and Adey (1973) showed that weak oscillating electric gradients, no larger than the EEG (50-100 mV/cm), increase efflux of calcium ions and GABA from cat cerebral cortex by almost 20%. Cellular calcium ions play many vital roles in cell growth and development regulation. Hence the ability of EMR to induce changes in cellular calcium ions is fundamentally important in assessing the biological mechanisms which contribute to adverse health effects.

This evidence that biological systems use tune frequencies which involve electromagnetic signals on the surface of cells and between cells, even though these signals are orders of magnitude below the thermal noise level, is quite reasonable. Close to an AM tower where the 1 MHz signal had a strength of 100 (W/cm2 it was still possible to tune into a remote AM and remote FM signals on an ordinary radio even through their field intensity was less than 0.01pW/cm2. Resonant circuits within the radio were tuned into the carrier of the extremely weak but easily detectable radio signal.

Calcium ion (Ca2+) efflux:

The calcium ion (Ca2+) is one of the most important substances in cells. Ca2+ is a first, second and third signal transduction messenger, Alberts et al. (1994), Pahl (1999). Alberts et al. describes Ca2+ as a prominent and ubiquitous intracellular messenger. This means that factors that induce changes of cellular Ca2+ can cause significant changes of cells.

Adey (1979) contains evidence of other windows for ELF induced Ca2+ efflux in chick and cat brains, e.g. 5, 10, 56 and 100 V/m (Figure 8), and other microwave intensity windows for Ca2+ influx and efflux. The field intensity and modulation frequency were shown to be important parameters in EMR causing Ca2+ efflux. Since extremely low SAR's produce significant effects and the modulation frequency is critical, this is not a thermal effect but it is a biological effect acting at the cellular level affecting cellular functions. ELF and RF/MW signals cause Ca2+ efflux and ELF signals don't involve tissue heating. enceH

Figure 8: The effects of extremely low frequency fields on 45Ca2+ efflux from chick forebrain, for ELF fields of 5, 10, 56 and 100 V/m. * : p<0.05; **:p<0.01, Bawin and Adey (1976).

Figure 9 shows significant Ca2+ efflux with exposure intensity at 0.05, 0.1 and 1 mW/cm2, but not at 2 and 5 mW/cm2 with a 450 MHz carrier. Particular higher exposures do not have the same significant effects as lower specific exposures, indicating that this is a non-thermal mechanism.

Figure 9: Effects of changing intensity of 450 MHz field amplitude modulated 16 Hz as efflux of 45Ca2+ from chick cerebral hemispheres. Cross-hatched bars show levels of efflux exposed specimens in relation to control specimens (stripped bars) tested simultaneously in the same experiments. Variance shown as SEMs **, p<0.05., Bawin, Sheppard and Adey (1978)

Figure 10: Relative Ca2+ efflux (positive and negative) from isolated chick cerebral hemisphere exposed to (A) weak RF field (147 MHz, 0.8 mW/cm2, 56 V/m in air), amplitude modulated at low frequencies (abscissa) (Bawin et al. (1975) and (B) ELF electric field (56 V/m in air) over the same modulation frequency range (Bawin and Adey (1976). The tissue gradients differ by 106 between A and B.

It was established very early on that an ELF signal carried on a RF carrier produced altered cellular Ca2+ fluxes, as the ELF signal on its own, but with a very much higher induced tissue electric field gradient, Bawin and Adey (1976), Figure 10.

Adey (1979) reviews a large body of research on the neurophysiological effects of RF/MW radiation. This included the human biometeorological research on circadian rhythms in human subjects isolated from sunlight and EMR; their own work on altered monkey behaviour with a tissue gradient of 10-7 V/m and other animal behaviour experiments. It also covered cellular evidence including Ca2+ flux experiments on cats and chick brains. These show that ionic changes in amplitude modulated RF/MW fields are much more related to modulation frequency than intensity of signal. Often higher effects are seen at lower exposure intensities than some higher intensities - in windows.

Significant effects occur in fields that are too low to produce any detectable thermal effects. In great frustration at the intransigence of the position held by scientists who doggedly claim that there is only evidence of thermal effects. Professor Adey concludes:

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

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

Dr Adey was basing his insights on a fascination with discovering how neurological tissue operated and how it was altered in extremely low level RF/MW and ELF fields.

The current world leader in Ca2+ efflux research is Dr Carl Blackman of the U.S.E.P.A.. Dr Blackman has replicated and significantly extended the studies carried out by Dr Adey's group and other groups. He and

his team have produced over 2 dozen peer-reviewed publications in this area, including several major reviews. Blackman et al. (1989) identified multiple power density windows for Ca2+ efflux, using a 50 MHz carrier modulated at 16 Hz. Their results, using units of mW/cm2, are summarized as follows:

Enhanced efflux		1.75	3.85	5.57	6.82	7.65	7.77	8.82	
No change	0.75	2.30	4.50	5.85	7.08	8.19	8.66	10.6	14.7

The intensity window data was considered as an example of non-linear dynamics because there appears to be no progressive decline in the magnitude of the effects at low exposure intensities. This data is consistent with a fractal process with a non-integer dimension that is approximately 1.4, Blackman et al. (1989).

The lowest published RF intensity that has been documented to produce significant Ca2+ efflux is 0.00015 W/kg from Schwartz et al. (1990). They used frog hearts, exposed for 30 mins, to a 16Hz modulated 240 MHz RF signal. This has an exposure intensity of about 0.08 (W/cm2.

Figure 11: Effect of 15 Vrms/m electromagnetic fields on the efflux of Ca2+ from chicken brain tissue as a function of modulation frequency, Blackman et al. (1988). The solid bars show significant alteration, $p<0.05$.

Blackman's group confirmed and significantly extended the "windows" concept of Ca2+ efflux, as well as aspects of homeostasis, involving tissue temperature for example. Figure 11 shows how modulation frequencies out to 510 Hz produce significant Ca2+ efflux at some frequencies, but not at other frequencies on either side.

Blackman et al. (1990) showed the importance of the local static magnetic field. Blackman et al. (1991) showed that Ca2+ efflux occurred for tissue temperatures of 36(C and 37 (C and not at 35(C and 38(C. They comment that these could be very good reasons why experimental outcomes have been difficult to confirm in some laboratories. After reviewing the many studies in the published literature on EMR induced Ca2+ efflux. Blackman (1990) concludes:

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

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

At the Scientific Workshop on Biological Effects of Electromagnetic Radiation in Vienna, October 1998, Dr Carl Blackman, U.S. Environmental Protection Agency, presented the results of 30 years of research into cellular calcium ion efflux and influx which is induced by pulsed and modulated EMR. Having established that EMR acts in quite different and consistent biological manner in a complex set of windows, Dr Carl Blackman concluded that it makes a great deal of sense to move away from the concept that EMR should act like a single chemical. The variable nature of the response, as indicated by complex exposure 'windows', indicates that EMR acts like chemicals (plural) rather than acting like a single chemical, Blackman (1998). This addresses the concepts around 'consistency' and 'specificity'.

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

EMR exposes the whole human body and not a single target organ. Each organ has a different cellular structure that relies to a greater or lesser extent in electric and magnetic factors and forces for its growth and control. The brain, central nervous system and muscles, including the heart, make much stronger use of electrical signals than bones for example. However, every cell has an electric potential across its membrane and uses ions, such as calcium ions (Ca2+), sodium ions and potassium ions. Receptors on cells are negatively charged and ions and neurotransmitters which initiate signal transduction are positively charged. DNA is negatively charged and the protein which is bound to it is positively charged.

Hence, every cell can interact with EMR and EMR can alter the growth regulation factors through alteration of the ionic concentration within the cells and in the intracellular fluid. Some higher functioning organs, especially the brain and CNS, are dependent on EMR for normal operation and have been shown to be altered by externally applied EMR, with consequent behaviour and neurological performance change, Bawin et al. (1973).

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

Health implications of induced alterations in calcium ion homeostasis:

Induced alteration of cellular calcium ions:

of brain cells is associated with behavioural and reaction time changes and associated EEG alterations, Bawin et al. (1978);

of the pineal gland reduces the nocturnal production of melatonin (which increases the cell damage throughout the body, reduces the integrity and competence of the immune system, and hence increases the incidence of cancer and immune system related disease and degenerative diseases of the brain, Reiter (1994) and Walleczek (1992);

of lymphocytes reduced the competence of the immune system making the subject more vulnerable to allergens, toxins and viruses, and to leukaemia; and

of damaged cells alters the ratio of surviving neoplastically transformed cells and those programmed to self destruct (apoptosis), Balcer-Kubiczek (1995).

Cells have voltage-gated Ca2+ channels in the cell membrane to allow the influx and efflux of calcium ions in order to regulate cellular processes, Adey (1993).

The neurological role of Ca2+ is well described and documented by Dr Adey. A university text on the molecular biology of the cell, Alberts et al. (1994), documents many cellular processes which depend on Ca2+, including cell-cell adhesion, gap junction gating, intracellular mediation, cyclic AMP and ATPase processes, and signal transduction as a second messenger. Ca2+ mediate process in the hypocampus involved with learning. They also mediate apoptosis. Chemical carcinogens, such as the tumor promoting phorbol esters, for example TPA, act by elevating intracellular calcium, Balcer-Kubiczek (1995).

Ca2+ mediate gene expression processes, development and plasticity of the nervous system, activity-dependent cell survival, modulation of synaptic strength and calcium mediated cell death, Ghosh and Greenburg (1992). They are involved in the Ca2+-cAMP signal transduction process that mediates several cellular functions, including melatonin production in pinealocyte, Zurawska and Nowak (1992). Li et al. (1999) showed that 50 Hz fields in TPA treated cells produce a significant dose-response decrease in gap junction communication with magnetic fields of 0.2, 0.4 and 0.8 mT.

Neurological effects intimately involve Ca2+ as shown originally by Dr Adey's group. This includes mediation of sodium ion activity in the brain, Charpentier and Kado (1999). Walleczek (1992) reviewed the roles of Ca2+ in the immune system including regulation of leukocytes, lymphocytes and Natural Killer Cells, mainly through signal transduction processes. Through their synergistic activity with cAMP, Ca2+ mediate some key hormones including luteinizing hormone, testosterone, prolactin and Growth Hormone, Veldhuis et al. (1984), Kotwicka and Warchol (1998), Rillema (1980), Vacher et al. (1994), Ilondo et al. (1994), Ray and Wallis (1982), and Davis et al. (1987).

Cardiac regulation occurs using calcium ion signaling, Reuter (1987) and Ugarte et al. (1998). Takahashi et al. (1992) found that altered expression of Ca2+-dependent genes are involved in end-stage heart failure.

Calcium ion influx is critical to mitogen action, Hadden (1987). In this process Ca2+ acts directly and indirectly through its action on calmodulin and protein kinase C to the activation of a number of enzymatic processes. The tumor suppresser gene p53 is regulated by Ca2+, Metcalf et al. (1999). Ca2+ also regulates the transcription of the c-fos proto oncogene, Montminy et al. (1990), Thompson et al. (1995) and Werlen et al. (1993). One of the key roles on Ca2+ in the carcinogenic process is outlined by Fanelli et al. (1999) who showed a dose response relationship for Ca2+ influx over a static magnetic field range from 60 to 600(T. This Ca2+ influx was observed to inhibit apoptosis. Fanelli et al. stated that magnetic fields thus might interfere with human health by altering/restoring the equilibrium between cell death and proliferation. Indeed, they conclude, the rescue of damaged cells may be the mechanism explaining why magnetic fields that are not mutagenic per se are often able to increase mutation and tumor frequencies.

Hence EMR's proven ability to induce calcium ion fluxes and to significantly alter cellular calcium in homeostasis is a direct biological mechanism for all of the biological effects associated with EMR exposure. Taken together, this provides an established biological mechanism for genetic damage, reproductive problems, cardiac disease, cancer and increased risk of viral and bacterial infection. The key cancer mechanisms involve reduced melatonin, DNA strand breaks, chromosome aberrations, altered proto oncogene expression and impairment of the immune system. These have all been linked to EMR exposure across the EMR spectrum from ELF to RF/MW.

Ca2+ have been implicated in essentially every step of the transductive coupling of neurotransmitter substances in effects of every step of the immunological reactions and every step of the coupling of hormonal binding at the membrane surfaces to cellular mechanisms, Adey (1979). Ca2+ efflux is the initial biological mechanism for almost all of the observed significant adverse health effects of EMR exposure, including neurological, cardiac, reproductive and cancer effects.

Biochemists have now confirmed that RF/MW alters signal transduction, (e.g. Luben (1995), Byus (1994)), alters melatonin and damages the immune system, as will be shown below.

Dr Alan Frey directly challenges the RF Thermal view.

Dr Frey, an eminent U.S. biologist, has been carry out EMR research for several decades. He was the discoverer of "Microwave Hearing". In the introductory chapter of a book that he edited, Dr Frey describes the historical tendency to use the toxicological model that treats EMR as an external agent,

Frey (1995). He then refers to Burke and others who have made it clear that "our frame of reference determines what we look at and how we look. And as a consequence, this determines what we find." This is demonstrably true for the ICNIRP assessors. Dr Frey than states "Theory and data show that this is the wrong model. Electromagnetic fields are not a foreign substance to living beings, like lead or cyanide."

"To model how em fields affect living beings, one might compare them to the radio we use to listen to music. The em signal the radio picks up and transduces into the sound of music is almost unmeasurably weak. At the same time there are, in toto, strong em fields impinging on the radio. We don't notice the stronger em signals because they are not the appropriate frequency or modulation. Thus they don't disturb the music we hear. However, if you impose on the radio an appropriately tuned em field or harmonic, even if it is very weak, it will interfere with the music. Similarly, if we impose a very weak em signal on a living being, it has the possibility of interfering with normal function if it is properly tuned. This is the model that much biological data and theory tell us to use, not a toxicology model."

Wever (1974) and König (1974) proved that human brains are tuned to detect and use the Schumann Resonances that have an intensity of the vertical electric field component of about 0.1pW/cm2. This is 2.65x1014 times lower than the ICNIRP guideline for low frequency signals, Figure 7. Ahissar et al. (1997) demonstrated that mammal's brains contain biochemical phase-locked loop circuits to detect the phase difference between incoming ELF signals in the same manner as FM radio receivers.

EMR Reduces Melatonin in Animals and People

The calcium ion efflux research demonstrates one of the fundamental principles of EMR research. Under given specific conditions the calcium ion efflux (positive or negative) does occur at some combination of exposure conditions, but not at a nearby slightly different set of conditions. This is because of the "window" non-linear nature of the effect with respect to modulation frequency and intensity in particular. Also, one set of conditions that produce a significant effect in one laboratory does not produce any observed effect in another laboratory because it has a different geomagnetic field. On the other hand, in real world situations workers or residents are continually passing through effective and non-effective windows of exposure.

This means that despite the great difficulties of detecting melatonin reduction in people because of the large intra-personal differences from day to day, and the very large inter-personal differences, on average there is a dominance of exposure conditions that do cause calcium ion efflux and reduced melatonin, so that it is observed to differ in most monitored populations in the real world.

Since calcium ions in the pinealocytes are associated with the regulation of cAMP, which in turn mediates the transformation of serotonin to melatonin, Reiter (1994), it is postulated that EMR's ability to alter calcium ion homeostasis will produce reductions in melatonin over a wide range of exposure window conditions across the EMR spectrum from ELF to RF/MW.

Light-at-night and electromagnetic radiation, are proven to reduce melatonin and hence pose significant adverse health effects. The evidence for EMR is summarized here. Rosen, Barber and Lyle (1998) state that seven different laboratories have reported suppression of nighttime rise in pineal melatonin production in laboratory animals. They show that a 50 (T, 60 Hz field with a 0.06(T DC field, over 10 experiments, averages a 46% reduction in melatonin production from pinealocytes. Stark et al. (1997) observed a significant increase in salival melatonin in a group of 5 cows when the short-wave radio transmitter at Schwarzenberg, Switzerland, was turned off for three days, compared to 5 cows that had much lower RF exposure. Hence there are now nine independent observations of melatonin reduction in animals from ELF and RF exposure.

Fifteen studies from show that ELF and RF/MW exposure reduces melatonin in people and a serotonin enhancement. Evidence that EMR reduced melatonin in human beings commenced with Wang (1989) who found that workers who were more highly exposed to RF/MW had a dose-response increase in serotonin, and hence indicates a reduction in melatonin. Fourteen studies have observed significant EMR associated melatonin reduction in humans.

They involve a wide range of exposure situations, including 50/60 Hz fields, Wilson et al. (1990), Graham et al. (1994), Wood et al. (1998), Karasek et al. (1998), and Burch et al. (1997, 1998, 1999a, 2000), Jutilainen et al. (2000) and Graham et al. (2000); 16.7 Hz fields, Pfluger et al. (1996), VDTs Arnetz et al. (1996), a combination of 60 Hz fields and cell phone use, Burch et al. (1997), and a combination of occupational 60Hz exposure and increased geomagnetic activity around 30nT, Burch et al. (1999b).

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

Hence it is established from multiple, independent studies, that EMR from ELF to RF/MW reduces melatonin in animals and human beings.

The health implications of reduced melatonin:

Melatonin has many biological effects. The melatonin receptor regulates several second messengers: cAMP, cGMP, diacylglycerol, inositol trisphosphate, arachidonic acid, and intracellular Ca 2+ concentration ([Ca2+]j). In many cases, its effect is inhibitory and requires previous activation of the cell by a stimulatory agent. Melatonin inhibits cAMP accumulation in most of the cells examined, but the indole effects on other messengers have been often observed only in one type of the cells or tissue, until now. Melatonin also regulates the transcription factors, namely, phosphorylation of cAMP-responsive element binding protein and expression of c-Fos. Molecular mechanisms of the melatonin effects are not clear but may involve at least two parallel transduction pathways, one inhibiting adenylyl cyclase and the other regulating phosphohpide metabolism and [Ca 2+]j, Vaneeck (1998).

Professor Russell Reiter, one of the world's leading medical researchers into the effects of melatonin, summarizes melatonin’s roles, Reiter and Robinson (1995), as being:

Vital for healthy sleep, including lowering the body temperature, and assisting in maintaining health sleep states.

Reduces cholesterol, with consequent reductions is risk of atherosclerosis and coronary heart disease.

Reduces blood pressure and the tendency for blood clots, and hence reduces the risk of strokes.

Scavenger of free radicals. This, along with the above factors, reduces the risk of heart attack, cancer, viral replication. Melatonin plays a vital free radical scavenging role in the brain where, because it is high in iron, has a high production rate of hydroxyl radicals (OH(). Free radical damage is now known to play a formative role in most brain disorders, including Alzheimer’ disease, Lou Gehrig’s disease, multiple sclerosis and Parkinson’s disease. While the Blood Brain Barrier (BBB) denies access to most free radical scavengers, melatonin has free access.

Enhances the effectiveness of the immune system. Specifically enhancing the T-cells, i.e. the T-helper cells and the T-killer cells. T-helper cells have a receptor for melatonin. When melatonin is received a

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

In Professor Reiter's book, published in 1995, he describes the evidence that EMR/EMF does reduce melatonin as a "Smoking Gun" level of proof. That is, there is considerable scientific evidence but at that time it wasn't sufficient for scientific proof. By considering more recent information, and the extensive results of biometeorological research, and linking the melatonin research to the calcium ion research, the level of proof can be seen as causal. The multiple observations of melatonin reduction in EMR exposed populations means that EMR exposure increases the incidence of all of the conditions identified by Reiter and Robinson above, including immune system, cancer, neurological and cardiac effects. Epidemiological evidence of exposed workers and residential populations confirms that these adverse health effects do occur.

Human Biometeorology:

Dr Ross Adey refers to the work of Wever and König in Germany in the 1960's and 70's. The work was carried out at the Munich Technical University and the Max Planck Institute, Adey (1981). Wever and his colleagues constructed two isolation rooms to remove all daily time signals. One, Room 2, as also surrounded by a Faraday Cage to exclude electromagnetic signals, Wever (1974). The results included the fact that those in the Faraday Cage shielded room, identical to the other room in all other respects, had significantly longer circadian rhythms ($p<0.01$).

In addition, a significant proportion (30%) of the Faraday Cage group "desynchronized" while none of the other group did ($p<0.001$). This involved rapid lengthening of the circadian period from around 26-27 hours to 30 - 36 hours, Figure 12.

Long-term isolation experiments at the Max Planck Institute proved that removing sunshine led to significantly longer mean circadian periods. Also shielding subjects from natural and artificial EMR further significantly extends mean circadian periods. Around 30 % of subjects desynchronized. When a weak 10 Hz signal was secretly introduced the desynchronization was removed, Figure 12. This proved the role of the Schumann Resonances that act with sunshine as a Zeitgeber.

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

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

The desynchronization was removed through the application of a 10 Hz signal with a peak to peak field strength of 2.5 V/m. This is equivalent to 0.83(W/cm2. The signal the Faraday cage had removed, which was replaced by this artificial signal, was the Schumann Resonance which has a field intensity of about 0.12 pW/cm2. Hence the desynchronization was caused by the removal of a 0.1pW/cm2 signal. Wever (1974) concludes that their research gives:

"Significant proof that electromagnetic fields in the ELF range influence the human circadian rhythms and therefore human beings."

Figure 12: Free-running circadian rhythm of a subject living under strict isolation from environmental time cues. During the first and third section protected from natural and artificial electromagnetic fields, during

the second and fourth sections (shaded area) under the influence of a continuously operating 10 Hz electric field of 2.5 V/m, Wever (1974).

The plausible biological mechanism involving local Schumann Resonances (Type I) and sferics (Type II) signals, was proposed by König (1974). He noted the strong similarity between the frequencies of the Schumann Oscillation and the alpha band of the human EEG, see the Figure 13 below.

Figure 13:Electric fields from, (, the Schumann-Resonance, ((, Local fields of about 3 Hz and the ((10 Hz) and ((3 Hz) human EEG channels, König (1974).

A resonant interaction is entirely feasible. Human brains use the same frequency bands as the weather produces. The EEG signals are electromagnetic and so are the weather signals. Removing the Schumann Oscillation from some individuals, removes part of their circadian control. This confirms the interaction between human brains and the Schumann Resonance. Konig provides independent confirmation of this interaction though a completely different set of experiments involving human reaction times.

The Type II signals on the left of Figure 13 are naturally occurring, locally sourced ELF fields centred around 3 Hz. They are very similar to the EEG delta-band. König (1974) showed that people's reaction time significantly sped up in the presence of Type I (Schumann Resonance) signals and slowed down when Type II (Local Sferic) signals were dominant, Figures 14 and 15.

Figure 14:The solid line shows the reaction times of 4500 people per point, over the day in September 1953 in Munich, compared with (dashed line) the Type ((10 Hz) signals field intensity, König (1974).

Converting the data in Figure 14 to a dose response relationship produces Figure 15.

Figure 15: The dose-response relationship for human reaction time as a function of the intensity of the Schumann Resonance signal, from König (1974).

Type ((Signals occurred on 10 occasions during the August-September period. Figure 16 shows the inter-relation for the change in reaction time relative to the onset of Type ((signals at time n hr. In the hour and a half after the onset of Type ((signals the reaction times (involving between 2000 and 3000 people), are well above average.

To confirm the indicated results from these public measurements Konig carried out controlled experiments with volunteers. He was able to produce slowed reaction times with 3 Hz signals and faster reaction times with 10 Hz signals, at will. This proves that human brains detect and react to external electromagnetic signals of extremely low intensity, including naturally occurring Schumann Resonances and local sferics.

Polk (1982) summarizes many observations of the Schumann Resonances. This reveals that the vertical electric field gradient is in the range 0.06 to 0.3 mV/m/Hz-1/2 for the 8 Hz to 21 Hz frequency range. This becomes 0.22 to 1.12 mV/m, averaging 0.67 mV/m. The field intensity (S) as a function of electric field (E) is S = E2/3.77 (W/cm2. Hence the mean Schumann Resonance field intensity is 0.12 pW/cm2.

At the same time that the Germans were publishing their biometeorological results showing that human being's reaction times vary with extremely low intensity naturally occurring and varying electromagnetic fields in the ELF part of the spectrum, Professor Ross Adey and Dr Susan Bawin were showing that

altered human reaction times in ELF modulated microwave fields was associated with altered EEG and calcium ion efflux from the brain cells.

Figure 16: The speeding up of the reaction time of people in the 60 to 90 minutes following the onset of 3 Hz signals, from the Traffic Exhibition in Munich in 1953, König (1974).

Hence the U.S. and German research jointly confirm both the effect and the mechanism. Human brains detect oscillating EMR signals at very low intensities through resonant absorption interactions. This causes the altered reaction times and circadian rhythms through induced changes in brain synchronization, cellular calcium ions and reduced melatonin. The later two biological effects are shown to occur over a wide range of exposure conditions from ELF to RF/MW and at very low exposure intensities.

7. Bioelectromagnetic Principles:

A more appropriate scientific approach than that taken by ICNIRP is one that recognizes some fundamental principles concerning the nature of biological systems and their use and interaction with EMR. Eight Bioelectromagnetic Principles are set out below with some of the scientific studies that support or confirm the principles.

Bioelectromagnetic Principle 1:

EMR is intrinsic to our bodies.

Intrinsic EMR signals are used for timing regulation at all levels, from seasonal and circadian rhythms, heart beat, cell ion oscillations, cell cycle timing, and synchronizing the EEG bands, Adey (1980), Becker and Seldon (1985), Frey (1993), König (1974) and Wever (1974).

Bioelectromagnetic Principle 2:

Our brains are the most electrically active organs in our bodies.

Interference with timing leads to arrhythmia of brain, neurological effects and diseases and brain tumours.

Supporting Evidence:

König (1974) and Wever (1974) proved that human brains detect local lightning signals and the globally radiated Schumann Resonance signals by showing that these signals alter human reaction times and regulate human circadian rhythms, at intensities around 0.1pW/cm2, Polk (1982). This work is indicative of a resonant absorption interaction between the Schumann Resonance Spectrum and human brain waves (EEG-rhythms).

Shandala et al. (1979) show than microwaves significantly altered the EEG of animals, Von Klitzing (1995) shows that a GSM signal alters the EEG of human volunteers and Mann and Roschke (1996) show sleep disturbance and EEG change from sleeping next to a cell phone. Mild et al. show that cell phone users exhibit a significant dose response increase in neurological symptoms, including dizziness, memory loss, loss of concentration and headaches.

Over sixty studies identify increases in brain tumours with EMR exposures across the spectrum; over 30 are statistically significant and 13 have dose-response relationships, at least half of which are significant, Section 16.3.

Several epidemiological studies show significant increases in neurological effects and diseases in residential and occupational EMR exposures, Amyotropic Lateral Sclerosis (ALS) and Parkinsonism, Deapen and Henderson (1986); Suicide, Baris and Armstrong (1990), Perry et al. (1991); Alzheimer's Disease, Sobel et al. (1995, 1996); Clinical Depression, Verkasalo et al. (1997); Psychological symptoms, Beale et al. (1997); and ALS, Savitz et al. (1998a,b). Beale et al. found significant dose-response relationships for several symptoms including depression and anxiety and Johansen et al. (1999) for Multiple Sclerosis and Savitz et al. (1998a) for ALS. Van Wijngaarden et al. (2000) identified significant dose response increases in suicide in electrical utility workers in relation to recent cumulative magnetic field (60Hz) exposures. The strongest associations were for younger people (<50 years).

7.2.2 Alzheimer's disease:

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

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

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

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

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

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

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

7.2.3 Sleep disturbance:

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

The causal relationship between RF radiation exposure and deterioration in sleep quality is identified through a significant dose response relationship (p<0.001), Figures 17 and 18, improvements in sleep quality which changing the direction of the beams and turning the transmitter off, and reduced melatonin as the biological mechanism.

Figure 17: Adult Sleep Disturbance with RF exposure at Schwarzenburg, Switzerland, Abelin (1999).

Figure 18: Dose-response relationship for Sleep Disturbance at Schwarzenburg with exposure in nW/cm2. Note: 1nW/cm2= 0.001(W/cm2

Groups B, R and C are all exposed to a mean RF signal of less than 0.1(W/cm2 and they experienced highly significant sleep disturbance and reduced melatonin. Since sleep disturbance and melatonin reduction has been observed with cell phone exposure, Mann and Roschkle (1995) and Burch et al. (1997), these observations also apply to cell sites. Assuming a normal sleep disturbance of 10 %, the approximate exposure level threshold for zero additional effect is near 1 pW/cm2, near the natural level for the Schumann Resonances.

As an experiment, the transmitter was secretly turned off for three days. Sleep quality improved in all three groups being studied. Figure 19 shows Groups A and C.

Figure 19: Sleep disturbance in people exposed to a short-wave radio stations which was turned off for three days, Altpeter et al. (1995), showing the highest exposed Group A, and lowest exposed Group C.

Both Groups show a delayed improvement in sleep of one to two days. The reduced wakening averaged over days 4 to 6 compared with days 1 to 3, for group A, and days 5 to 7 compared with days 1 to 4 for group C, are highly significantly reduced, $p<0.001$. Thus a significant ($p<0.001$) improvement in sleep quality is associated with a measured 24 hour mean and median exposure of 0.1 mA/m (0.4 nW/cm2).

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

Two herds of 5 cows each, had salival melatonin sampled several times a day, including night-time. The "exposed" herd as at 500 m from the tower with a mean exposure of 0.095(W/cm2. Their mean melatonin levels were 17.7 pg/ml compared with 19.0 pg/ml for the "unexposed" cows whose measured mean exposure as 0.00022(W/cm2. Figure 20 shows the melatonin for these two herds during the experiment involving turning the tower off.

Figure 20: Salival melatonin from two herds of 5 cows, one exposed at 500 m, 0.095(W/cm2, (solid line) and one "unexposed" at 4000 m, 0.00022(W/cm2, (dashed line).

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

The causal relationship with human sleep disturbance is strong evidence of a significant neurological effect of RF radiation on people, associated with mean exposures down to less than 0.4 nW/cm2.

Hence, it is highly likely that cell phone users, with brain exposures many millions of times higher than the Schwarzenburg exposure levels, will experience significant neurological effects. The significant bovine behavioural effects of extremely low RF exposure is confirmed by Löscher and Käs (1998).

7.2.4 Neurological effects of cell phone usage:

In 1998 Mild et al. (1998) survey around 11,000 cell phone users in Norway and Sweden, Figure 19. They found significant dose response relationships for a number of crucial symptoms that had been clinically described and associated with cell phone use by Hocking (1998).

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

Bioelectromagnetic Principle 3:

Our hearts are electrically sensitive.

Supporting Evidence:

Hearts use EMR signals that are detectable by the ECG. An electric pulse produces a cascade of calcium ions that causes the heart muscle to contract and produces a heart beat.

7.3.2 Heart Disease:

Satre, Cook and Graham (1998) observed significantly reduced heart rate variability (HRV) in volunteers sleeping in 60Hz fields. Extrinsic EMR signals interfere with hearts and cause heart disease and death. Bortkiewicz et al. (1995, 1996, 1997) and Szmigielski et al. (1998) found that RF exposure altered heart rate variability and blood pressure. Braune et al. (1998) showed that cell phone significantly increased blood pressure. Savitz et al. (1999) found a highly significant dose response relationship for mortality from Arrhythmia related heart disease and heart attack (Acute Myocardial Infarction) for exposed electrical occupations and for individual occupations of electrician, lineman and power plant operator.

This is a powerful set of epidemiological evidence showing that EMR across the spectrum increases the incidence and mortality from arrhythmia related heart disease and from heart attack.

Figure 21: The prevalence of symptoms with various categories of calling times/day, A. Norway, B. Sweden, Mild et al. (1998).

Geomagnetic Activity adverse effects:

Solar activity alters the earth's geomagnetic field, electron concentrations in the ionosphere, the Q-value of the earth/ionosphere resonant cavity and intensity and frequency of the Schumann Resonance Spectrum. Since human brains an hearts are sensitive to subtle changes in environmental electromagnetic

fields, evidence of correlations in cardiac functions and geomagnetic activity (GMA) would be indications of human sensitivity to very small EMR signals.

Watanabe et al. (1994) report that a 35-yr old cardiologist with a family history of heart disease, monitored himself with a blood pressure monitor at 15-min intervals for 3 years. Systolic and diastolic blood pressure and heart rate were significantly correlated with the 27.7 day solar cycle and with the geomagnetic disturbances. Pikin et al. (1998) observed that blood coagulation and platelet aggregation increased with increasing GMA. Gurfinkel et al. (1995) observed significant alterations in capillary blood flow in heart patients, correlated with GMA. These are biological effects that are risk factors for heart disease and heart attack. Many studies have found significant correlations between geomagnetic activity (GMA) and Ischemic Heart Disease and Heart Attack, for example Sitar (1990), Villoresi et al. (1998), Stoupel et al. (1996, 1999), and Oraevskii et al. (1998).

Hence blood pressure changes and reduction of heart rate variability is observed with changes in GMA, working in RF/MW environments and using a cell phone. Significant cardiac disease and death are highly correlated with subtle changes in GMA, work in electrical industries (in a dose-response manner).

Bioelectromagnetic Principle 4:

7.4.1 Cells are sensitive to EMR.

7.4.2 Supporting Evidence

Cells have a voltage across their membrane, voltage-gated ion channels through their membrane and use ions (e.g. Ca2+) for many cell regulatory processes including signal transduction and gap junction gate regulation. Altering the electric field on the surface of cells alters the receptor efficiency and interferes with the voltage-gated ion channels.

Induced alteration of calcium ion homeostasis has profound and serious effects for every cell. Calcium ion efflux/influx is an established biological effect of modulated EMR exposure, Blackman (1990). Cellular calcium ions have many profound effects on cells. These include the regulation of the neurotransmitter GABA and the neurohormone melatonin, as well as being associated with DNA synthesis, chromosome aberrations, gene transcription and protein expression, gap junction regulation, reaction times, immune system competence, heart beat regulation, apoptosis, cancer, cardiac, reproductive and neurological effects.

Bioelectromagnetic Principle 5:

7.5.1 Our whole body acts as an aerial.

Unlike many chemicals, no particular body organ is the target of an RF signal. The whole body acts as an aerial and electric current flows down through our bodies to earth. Hence RF/MW radiation impacts on every organ in our bodies.

Whole body organs such as our circulatory system and bone marrow are sensitive to the altered electric fields and the currents flowing through them, impairing our immune system and producing leukaemia, and cancer and illness throughout human bodies.

Supporting Evidence:

Large epidemiological studies, Robinette et al. (1980), Milham (1985 a,b, 1988), Szmigielski (1996) and Dolk et al. (1997 a,b) show that diseases and cancer across many body organs is produced by RF/MW

and electrical occupational EMR exposures. In all of these studies, and in many other epidemiological studies, ELF and RF/MW exposures produce significant increases in leukaemia, including residential studies with significant dose-response relationships.

7.6 Bioelectromagnetic Principle 6:

7.6.1 The brain is linked to organs and cells through EMR sensitive hormones.

Normal brain functions are communicated to the body through neurotransmitters (such as serotonin) and neurohormones (such as melatonin).

Melatonin reduction (and serotonin enhancement) by EMR has highly significant impacts on all organs and cells in our bodies, including brains, hearts and immune systems, Reiter and Robinson (1995).

Supporting Evidence

Natural EMR, the Schumann Resonances, are used for circadian synchronization, using phase-locked loop biochemical circuits, Ahissar et al. (1997). Artificial EMR interferes with these processes leading to desynchronization of circadian and cellular rhythms, and alteration of the timing and magnitude of the melatonin/serotonin cycle. De Seze et al. (1999) showed that cell phone use significantly reduces the pituitary output of Thyrotropin (thyroid stimulating hormone (TSH)). TSH is a primary regulator of metabolic function.

Many animal studies and fourteen human studies show that EMR significantly reduces melatonin, Section 6.5 above. This is a plausible mechanism for cancer in all organs but especially breast cancer, immune system impairment, SIDS, heart disease and reproductive effects such as congenital malformation and miscarriage.

Breast Cancer:

Epidemiological studies have shown significant increases and male and female breast cancer from exposure to EMR from ELF to RF/MW, as with leukaemia and brain tumor. Table 2 summarizes the studies for Female Breast Cancer.

There is a tendency for higher rates in pre-menopausal women and those with estrogen-receptor-positive breast cancer, and for black women. Elevated incidence and significantly elevated incidence of breast cancer and breast cancer mortality has been found with electric blanket use, residence near powerlines, electrical industry employment, radio telegraph operators, and RF/MW exposure.

7.6.4 Epidemiological Studies of congenital malformation and miscarriage:

Epidemiological studies of physiotherapists and electrical occupations identify significant increases on congenital malformation and miscarriage, Kallen et al. (1982), Vaughan et al. (1984), Taskinen et al. (1990), Larsen et al. (1991), Sanjose et al. (1991), including a significant dose-response associating first trimester miscarriage to MW exposure, Ouellet-Hellstrom and Stewart (1993).

Hence metabolic functions, cancer and reproductive effects are produced and alterations occur in many other hormone regulatory functions with EMR exposure.

Table 2: Epidemiological studies of Female Breast cancer associated with EMR exposure

Group	SIR/RR/OR	95%CI/(p-value)	Reference
Radio-telegraph operators	SIR=1.5		Tynes et al. (1996)
Electrical Engineers	OR = 1.73	0.92-3.29	Loomis, Savitz and Ananth (1994)
Electrical technicians	OR = 1.28	0.79-2.07	"
Telephone installers repairers, line work	OR = 2.12	1.17-4.02	"
Electrical Workers	OR = 1.38	1.04-1.89	"
Radiofrequency EMR			
Low Exp. White	OR = 1.15	p<0.05	Cantor et al. (1995)
High Exp. White	OR = 1.14	p<0.05	"
Low Exp. Black	OR = 1.23	p<0.05	"
High Exp. Black	OR = 1.34	p<0.05	"
High Exposure ELF	OR = 1.43	0.99-2.09	Coogan et al. (1996)
Pre-menopausal	OR = 1.98	1.04-3.78	"
Post-menopausal	OR = 1.38	0.82-2.17	"
Computer equipment operators, high Exp.	OR =1.80	1.04-3.12	[Trend p = 0.06]
Electric Blankets, heavy use, pre-menopausal	RR = 1.43	0.94-2.17	Vena et al. (1994)
All women	OR = 1.45	1.08-1.94	"
> 2 years of use	OR = 1.60	1.15-2.22	"
> 5 years of use	OR = 1.56	1.09-2.25	"
Positive Estrogen receptor aged 45 - 55 years.	RR = 1.12	0.78-1.43	Gammon et al. (1998)
Powerline, Sweden			
> 0.2 (T, men	RR = 2.1	0.3-14.1	Feychting et al. (1998)
>0.2 (T, women < 50 yr	RR = 1.8	0.7-4.4	"
>0.01 (T, women with + estrogen receptor	RR = 1.6	0.6-4.1	"
>0.01 (T, women with + estrogen receptor, aged < 60 years	RR = 7.4	1.0-178.1	"

Bioelectromagnetic Principle 7:

7.7.1 The EMR Spectrum Principle.

The EMR spectrum should be treated as an integrated whole, with biological impacts generally increasing with increasing carrier frequency.

Supporting evidence:

Biological and epidemiological studies show that biological effects, including calcium ion efflux, melatonin reduction, DNA damage, and chromosome aberrations, and human health effects, including neurological, cardiac and cancer disease and death, all have been shown to occur from ELF exposure, exposures in electrical and military occupations, and with RF/MW exposure.

Biophysics shows that the Dielectric Constant varies progressively, decreasing with increasing carrier frequency, Schwan and Foster (1980). This implies, as has been observed, Bawin and Adey (1976), and calculated, Vignati and Giuliani (1997), that for a unit field exposure induced tissue electric field gradients and induced tissue currents, increase with increasing frequency.

This strongly indicates that if a biological or epidemiological effect is observed for ELF exposures, then that effect will be more likely to occur from RF and MW exposures. It also indicates that epidemiological assessments can be carried out by integrating ELF and RF/MW exposure studies. The uncertainties of mixed occupational exposures are significantly reduced. Vignati and Giuliani suggest, in support of this principle, that the biological effects and adverse effects observed from powerlines could well be the result of the greater biological impact of the lower field strength but measurable RF signals emitted by the powerlines.

Bawin and Adey (1976) observed significant calcium ion efflux/influx from both an ELF modulated 147 MHz signal and a pure ELF signal. Both had an ambient electric field strength of 56 V/m but the RF signal produced a tissue gradient of 10-1 V/cm and the ELF signal 10-7 V/cm. This implies that the RF field could have been a million times smaller, i.e. 5.6x10-5 to produce a tissue gradient of 10-7 V/cm that would also cause altered cellular calcium ions. This smaller RF field has an exposure intensity of 0.83pW/cm2. This is of the same order as the intensity of the Schumann Resonance Spectrum during solar storms, which has been shown to have adverse health effects, for example S.I.D.S., O'Connor and Persinger (1997), Heart Attack, Oraevskii et al. (1998) and Epileptic Seizures, Ilipaev (1978).

7.8 Bioelectromagnetic Principle 8:

The Intrinsic Free Radical Principle.

Supporting Evidence: Free radicals

Oxygen free radicals, and other radical species, occur naturally in human bodies, Guyton and Kensler (1993). Free radicals are highly reactive and damage macromolecules such as DNA. Hence they provide a direct mechanism for causing cancer. Melatonin, as a potent free radical scavenger, and our immune system which detects and attempts to eliminate foreign cells, such as neoplastically transformed cells, are vital parts of a well developed cell repair system which is fundamental to health. Any factors or agents which reduce melatonin or impair the health of the immune system is thus carcinogenic and teratogenic.

It is the assumption of ICNIRP and those with the RF-thermal view that non-ionizing radiation cannot directly break chemical bonds and form free radicals, and therefore NIR cannot be genotoxic and cannot cause cancer and reproductive effects unless the exposure levels are high enough to cause significant tissue heating. Microwaves were observed to enhance free radicals in cell membranes, Phelan et al. (1992).

Several independent laboratories have observed significant genetic damage from nonthermal intensities of EMR, such as DNA strand breaks, chromosome aberrations enhanced oncogene activity. Lai and Singh (1997 a,b) have shown that ELF and microwave exposures involve free radical damage of DNA-strands.

7.8.3 DNA strand breakage

The first report that microwaves at non-thermal levels could produce single- and double-strand DNA breaks in E. Coli in solution, was Sagripanti and Swicord (1986). A much more advanced method, the "Comet Assay", was used on brain cells extracted from rats that had been exposed while alive by Lai and Singh (1995,1996,1997b). They observed single and double strand breaks in a dose-response manner, and identified the involvement of free radicals and the protective effect of melatonin.

Two other laboratories have recorded RF/MW produced significant DNA stands breaks. Verschave et al. (1994), who used a GSM cell phone signal to expose human and rat peripheral blood lymphocytes, found significantly increased strand breaks at high, but non-thermal exposure levels. Phillps et al. (1998) exposed Molt-4 T-lymphoblastoid cells to a number of cell phone technologies in the exposure range SAR = 0.0024W/kg to 0.026W/kg. At both of these exposure levels they observed significantly increased DNA damage ($p<0.0001$) for one cell phone and decreased damage for three cell phone signals. Induced DNA repair is also a sign of DNA damage, Meltz (1995). Hence RF/MW radiation has been confirmed to enhance DNA damage under RF/MW exposure from radar-like and cell phone exposures, including an exposure level which is 3% of the ICNIRP guideline. Using the ICNIRP relationship of 0.08 W/kg and 450(W/cm2 for 1 GHz, the exposure range is 13.5 to 146.3(W/cm2. Using the more basic equations [SAR = (E2/2(, (=1 S/m, (=800 kg/m3, and S = E2/3.77 (W/cm2] results with 1.0 to 11.0(W/cm2.

Four independent laboratories have also published data on ELF induced DNA strand breaks confirming that ELF EMR damages DNA strands; Lai and Singh (1997a), Svedenstal et al. (1998) Phillips et al. (1998), and Ahuja et al. (1997). Lai and Singh (1997a) also demonstrate the involvement of free radicals and the protective effect of melatonin. With the evidence above that EMR reduces melatonin this confirms that reduced melatonin causes higher concentrations of free radicals which produce more DNA strand breaks from EMR exposure from ELF to RF/MW frequencies. Increased DNA strand breaks will result in increased chromosome aberrations.

Multiple evidence from independent laboratories established that EMR from ELF to RF/MW causes DNA strand breaks at very low, non-thermal exposure levels.

Answers to the Criticisms of the Lai-Singh Results:

Attempts have been made to counter the Lai/Singh DNA results. Motorola funded Dr Joseph Roti Roti's group at Washington University in St Louis to 'replicate' the Lai/Singh work. Their research, Malyapa et al. (1998), did not confirm the DNA strand breakage but it did not replicate the Lai/Singh research. A very different assay method was used that differed in several key aspects such that it should be much less sensitive.

Dr Narendra Singh provided the following explanation:

"Here are the major differences between the technique we are using and what Dr Joseph Roti-Roti is using to detect DNA damages in cells after RF exposure in our technique, DNA is precipitated in agarose so as even tiny pieces can become confined to a dense spot to be visible. If a small piece of DNA is stretched out over long distance, we cannot see it under the microscope but once it precipitates, it becomes many folds shorter but denser and can be seen. The Motorola funded work is not doing that, in

fact only a few labs around the world are doing this. This is important for those involved in detection of low levels of DNA damage.

We treat our samples with proteinase K to digest proteins bound to DNA so DNA is made free to move in electric current towards the positive pole as DNA is a negatively charged molecule. Most proteins on the other hand are negatively charged and thus if not removed will drag the DNA in the opposite direction, I call it a "protein drag". The Motorola study did not use proteinase K.

We used an in vivo system where cells have very low levels of damage to start with, animals were exposed to radiofrequency at 2450 MHz, while Motorola used in vitro system, where cells have high background DNA damage as the culture conditions are not as good as in whole animals. In their study, cells were grown in culture which were exposed to cellular phone frequency of 837 MHz.

Anyway, it is very difficult for me to understand why when they said they wanted to replicate our data but didn't use exactly the same methodology of our experiment. Instead, they chose to use a much less sensitive assay to do their study."

Criticism has been raised about the delay of 4 hours in observing the DNA breakage. Williams (1996) states that "The presence of Single Strand DNA Breaks (SSDB) immediately after microwave exposure is consistent with findings from other types of radiation. However, the finding of no decrease in migration after 4 hr must be assumed to reflect no repair of putative breaks, which again does not conform to knowledge of the processing of DNA damage resulting from other forms of radiation."

Lai and Singh (1996b) respond by stating that there is a known difference when short term (seconds) of exposure are used compared with longer period (2 hr) exposures, and that this is observed in the use of drugs, to produce different dynamics. Dealing with the 4 hr questions they state: "However, it is not true that maximal DNA strand breaks are always observed at the end of exposure to radiation or chemical agents. For example, in a study by Arlett et al. (1993), a maximal level of DNA strand breaks was observed in lymphocytes at 1 hr after exposure to UV radiation, and a recent study by us [Singh et al. (1995)] showed that ethanol-induced SSDB in brain cells of the rat at 4 hrs after an oral administration." Several other explanations and examples are given to show that the dynamics of DNA strand breakage and repair is more complex than often assumed and there is considerable support for the feasibility of their finding of significant SSDB 4 hrs after the 2 hr exposure to microwaves. The replication in two independent laboratories is significant.

Malyapa et al. (1998) and Moulder et al. (1999) claim that the DNA damage observed by Lai and Singh is caused by the killing method: "It appears likely that the effects observed by Lai and Singh were confounded by the euthanasia procedure or by some unknown aspect of the animal handling of of the comet assay they used."

This is answered by Dr Lai. He states:

"Why do we use carbon dioxide (CO_2) in our study? First, we did not euthanaze rats with CO_2, we anaesthetised them. CO_2 is a well known anaesthetic agent, because it causes constriction of blood capillaries in the brain. We use it because it decreases blood flow, thus, less haemoglobin from red blood cells will get to brain tissue. Haemoglobin is known to cause oxidative DNA damage. Thus, use of CO_2 can actually decrease the artefact of haemoglobin-induced DNA damage.

What we did was to place a rat in a box containing dry-ice (CO_2) for 65 seconds to anaesthetise it (the rat was not dead yet), then we decapitated it and dissected the brain out for DNA comet assay. Dr Singh actually insisted that I picked up the animal after CO_2 by the neck (instead of by the tail, so that the rat is up-side-down and blood can flow to the brain), so that less blood would flow to the brain. Malyapa/Roti-

Roti misread our paper and euthanazed their rats with CO2. They basically asphyxiated their rats by putting them in a box containing dry-ice for several minutes (instead of 65 sec). Yes, asphyxiation is known to cause DNA damage. They also found that the longer you waited after asphyxiation, the more DNA damage would occurred. I also believe this is true.

But, what is unbelievable is that they claimed that our results were caused by the 'artefact' that we 'killed' our rats with CO2. It is so illogical. First, one has to wait longer to do assay on the 'exposed' rats (than the controls) to produce the artefact. We didn't do that. We killed our animals one at a time. So, the time between CO2 anaesthesia and start of comet assay was the same for each animal. Second, the order of doing 'control' and 'exposed' was at random. We didn't wait longer for the 'exposed' animals to produce the artefact."

This discussion reveals a great deal about the carrying out of laboratory experiments and the lengths some go to dismiss other's results. It is important to hear the story from the researcher and I thank Drs Lai and Singh for telling their side of the story to clarify the issues.

7.8.4 Chromosome aberrations

In 1959 Heller and Teixeira-Pinto (1959) showed that non-thermal pulsed RF signals could cause complex chromosomes breaks which mimicked the effect of ionizing radiation and c-mitotic chemicals. Hence significant chromosome aberrations (CA) produced by RF/MW have been reported by eight independent groups: the staff at the U.S. Embassy in Moscow, Goldsmith (1997); Garaj-Vrhovac et al. (1990a,b, 1991, 1992, 1993); Timchenko and Ianchevskaia (1995), Balode (1996), Haider et al. (1994) and Vijayalaxmi et al. (1997). In the Mar/Apr 1999 edition of Microwave News it is reported that Drs Tice, Hook and McRee showed chromosome damage from all cell phones tested, all being statistically significant and all but one highly significant with dose-response relationships up to a factor of three increase in chromosome aberrations.

El Nahas and Oraby (1989) observed significant dose-response dependent micronuclei increase in 50 Hz exposed mice somatic cells. Elevated CA have been recorded in a number of workers in electrical occupations. In Sweden Nordenson et al. (1988) found significant CA in 400 kV-substation workers and with 50 Hz exposures to peripheral human lymphocytes, Nordenson et al. (1984) and human amniotic cells, Nordenson et al. (1994). Significant CA in human lymphocytes exposed to 50 Hz fields are also reported by Khalil and Qassem (1991), Garcia-Sagredo and Monteagudo (1991), Valjus et al. (1993) and Skyberg et al. (1993). Skyberg et al. collected their samples from high-voltage laboratory cable splicers and Valjus et al. from power linesmen.

Hence chromosome damage has been recorded from exposes across the EMR spectrum from ELF to RF/MW exposures, in plants, mammal and human cells, animals and human beings, and from many independent laboratories. This confirms that EMR does damage chromosomes and establishes EMR induced chromosome aberrations as a biological effect. For a neoplastic cell to survive it must have an altered genetic structure to store the damage and to hide this from the immune system so that NK cells do not kill the neoplasm transformed cells.

7.8.5 Gene transcripts and activity

It is shown above that EMR induces alterations in cellular calcium ion fluxes and that calcium ion fluxes mediate gene transcription and expression. Calcium ion fluxes occur in “windows” of exposure parameter combinations. Two studies associate EMR exposure alteration of gene transcription with exposure windows. Litovitz et al. (1990) identified amplitude (intensity) windows, and Wei et al. (1990) frequency windows in the range 15 to 150 Hz. They observed a peak effect in c-myc gene transcription at 45 Hz. Liburdy et al. (1993) show that c-myc induction occurs in a direct sequence from calcium ion influx.

Increased c-myc gene transcripts by 50/60 Hz fields has also been observed, Goodman et al. (1989, 1992) and Lin et al. (1994). Phillips et al. (1992, 1993) observed time-dependent changes in the transcription of c-fos, c-jun, c-myc and protein kinase C, from 60 Hz exposure and a linear reduction in ras p21 expression by a 72 Hz signal. 50/60 Hz signals altered c-jun and c-fos gene expression as observed by and Lagroye and Poncy (1998) and c-fos expression by Rao and Henderson (1996) and Campbell-Beachler et al. (1998). The ppSom gene is very important in human neurological disorders, and is regulated by calcium ions Capone, Choi and Vertifuille (1998).

Cell phone radiation (836.55 MHz) significantly altered c-jun transcript levels, Ivaschuk et al. (1997). Cell phone radiation significantly enhances the proto oncogene c-fos activity in C3H 10T 1/2 cells, from a 40 % (p=0.04) increase from a digital cell phone and a 2-fold increase (p=0.001) from an analogue cell phone, Goswami et al. (1999).

Hence proto oncogene activity is altered and enhanced in multiple independent experiments from ELF and RF/MW exposure, including cell phone radiation.

7.8.6 Immune system impairment by EMR

Impairment of the immune system is related to calcium ion efflux, Walleczek (1992) and to reduced melatonin, Reiter and Robinson (1995). Cossarizza et al. (1993) showed that ELF fields increased both the spontaneous and PHA and TPA- induced production of interleukin-1 and IL-6 in human peripheral blood. Rats exposed to microwaves showed a significant reduction in splenic activity of natural killer (NK) cells, Nakamura et al. (1997). Dmoch and Moszczynski (1998) found that microwave exposed workers had decreased NK cells and a lower value of the T-helper/T-suppressor ratio was found. Moszczynski et al. (1999) observed increased IgG and IgA and decreased lymphocytes and T8 cells in TV signal exposed workers. Quan et al. (1992) showed that microwave heating of human breast milk highly significantly suppressed the specific immune system factors for E.Coli bacteria compared with conventional heating. Chronic, 25 year, exposure to an extremely low intensity (<0.1(W/cm2) 156-162 MHz, 24.4 Hz pulse frequency, radar signal in Latvia produced significant alterations in the immune system factors of exposed villagers, Bruvere et al. (1998).

Since calcium ion efflux and melatonin reduction are established biological effects of EMR exposure from ELF to RF/MW, impair immune systems should be observed in EMR exposures. Multiple independent evidence is available for RF exposures, down to extremely low chronic mean levels. This evidence establishes that EMR is genotoxic. This occurs through enhancing free radical damage by reducing melatonin, by altering the signal transduction within cells in such a manner that proto oncogene activity is increased, and by reducing the competence of the immune system through both reducing melatonin and altering calcium ion homeostasis.

Gap Junction Communication:

Cells in tissues are regulated by a large number of processes that involve cell-to-cell communication, including intercellular communication through gap junctions. When the natural opening of gap junctions is impaired then cell damage and cell transformation can take place more frequently. Gap junction opening is regulated by calcium ions and pH, Alberts et al. (1994). Li et al. (1999) observed that when a 50 Hz magnetic field was combined with the application of the cancer promoter TPA then the gap junction flow was impaired in a significant dose-response manner as a function of the magnetic field exposure, Figure 22.

Figure 22: Gap junction flow as a function of 50 Hz magnetic field strength, Li et al. (1999).

The EMR Spectrum Principle predicts that these effects are probable with low intensity RF/MW exposure.

7.10 Conclusions:

These Bioelectromagnetic Principles scientifically sound. They are supported by a large body of reliable internationally published peer-reviewed scientific research. They provide an integrative link between biology, EMR interactions, biological mechanisms and epidemiology. When considered with the supporting scientific evidence they provide a very substantial scientific challenge to the validity of the ICNIRP guideline.

8. The ICNIRP Guideline is seriously flawed and unlawful in New Zealand:

Environment Court Support:

The Environment Court (MacIntyre 1996) declared that the New Zealand Standard (and hence the ICNIRP guideline) is “not decisive” in New Zealand law but that the Sections 5 and 3 of the RMA are the appropriate legal basis for public exposure to electromagnetic radiation (EMR). This requires evidence to be considered of actual and potential adverse effects. The applicant, BellSouth, appealed the condition that restricted their emissions to 50(W/cm2, seeking to have the then New Zealand Standard which was 200 (W/cm2. In considering the evidence before it, including evidence of actual or potential adverse effects which occurred about 3 (W/cm2, the court set a public exposure condition for a cell site at that time and in that case of 2 (W/cm2. This is 1 % of the then allowed public exposure in NZS 6609, and 0.4% of the recently adopted NZS 2772.1:1999 and ICNIRP guideline.

The sections of the law that this is based on are Section 5, which requires that we "Avoid, remedy or mitigate any adverse effect of an activity on the environment." The definition of the 'environment' in Section 2 includes 'people and communities'. The definition of 'effect', Section 3, includes 3(d) "any cumulative effect of itself or in combination with any other effect, regardless of scale, intensity, duration or frequency, including (3f) "any potential effect of low probability which has a high potential impact."

Thus the Chief Environment Court Judge, Judge Sheppard, has accepted evidence that renders the ICNIRP guideline unlawful when evidence is given that there are potential adverse effects below the standard.

Additional strong reasons for rejecting the adoption of the ICNIRP guideline in New Zealand is the position that ICNIRP is based on 'established' effects whereas the legal evidence threshold in New Zealand is 'potential' effects, which have already been accepted by the Environment Court. It is grossly inappropriate for any country to adopt the ICNIRP guidelines for public health protection because it is scientifically challengable as it is based on serious errors and omissions.

In an earlier case for which no epidemiological evidence was presented on adverse human health effects from power lines, Transpower vs Rodney District Council, Judge Sheppard defined the basis of a 'potential effect' as being based on a plausible biological mechanism not mere innuendo.

The MacIntyre case was presented with evidence of plausible biological mechanism by Dr Richard Luben and epidemiological evidence of actual or potential human health effects by Dr John Goldsmith, with the exposure conditions associated with these effects being given by Dr Neil Cherry. Based on this expert evidence the public exposure condition of 2 (W/cm2 was imposed.
Environment Court Judge's error:

In the most recent case, the Shirley Primary School vs Telecom, a cell site case, it is submitted here that the Judge, Judge Jackson, made errors in law and evidence by ignoring the guidance given by Judge Sheppard, received evidence of potential or actual effects below 2 (W/cm2. This included the North Sydney study, Hocking et al. (1997), presented by Dr Hocking, who recommended an exposure level of 0.2 (W/cm2 based on his study. It also included the sleep disturbance study from Schwarzenburg, Switzerland, Altpeter et al. (1995) and the U.S. physical therapist miscarriage study of Ouellet-Hellstrom and Stewart (1993).

In the decision the Judge set out they way the court considered the evidence. "The first general level is epidemiological studies. The second level is a study of biological mechanisms. The levels are generally hierarchical (biological mechanisms above epidemiology) in that they are perceived as having increasing power in terms of establishing cause and effect." This is the reverse of the assessment that is usually followed, as shown by the MacIntyre Case and Sir Austin Bradford Hill, Hill (1965), IARC, USEPA and many other assessments. This is simply stated by Dr Abraham Lilienfeld: "The proper study of man is man".

The epidemiological evidence given in this case led the decision to conclude that the main factors they had to balance including the first three that were:

The very low risk, subjectively but reasonably assessed, of adverse learning effects and/or sleeplessness from exposure of pupils at the school to RF radiation;

A very low risk to pregnant women of miscarriage;

The extremely low risk of exposure to RFR causing cancer, e.g. leukaemia in humans;

Having stated this, based largely on the court's incorrect rejection of the established existence of a biological mechanism, the judgement allows the ICNIRP Guideline of 450(W/cm2 in this case. This there was stronger evidence than given on the MacIntyre Case but the accepted exposure level was 225 times higher.

The court was not shown the dose response relationships contained in this report for each of these effects. Therefore they did not know that with the probable exposure level of 2(W/cm2 the Risk Ratio (RR) for childhood leukaemia would be about RR = 22 and for miscarriage about RR = 3.5. For learning disturbance effects almost all of the children exposed to measured levels much less than 0.32(W/cm2, showed significant adverse learning effects and this almost all of the children at the Shirley Primary School could be adversely effected which is not a "very low risk". Even so, Section 3(d) and 3(f) of the New Zealand Resource Management Act requires effects of any potential low probability irreversible effects that have a high potential impact (i.e. miscarriage, leukaemia or learning impairment) must the avoided.

Hence this is clearly challengable as a misapplication of the provisions of the Resource Management Act, especially in the light of earlier, more senior guidance from Judge Sheppard.

9. The ICNIRP treatment of Biological mechanisms:

9.1 Inappropriate reliance on a plausible biological mechanism:

One of the primary reasons many skeptics about EMR health effects, such as ICNIRP, use to dismiss studies that show statistically significant effects and even dose-response relationships, is their claim of the lack of a plausible biological mechanism. When a study reveals a significant biological effect at nonthermal levels then groups such as the ICNIRP state that it must be independently replicated before it

can be accepted as an established biological mechanism. Based on this criteria calcium ion efflux/influx, GABA fluxes, melatonin reduction, DNA damage, chromosome aberrations and altered proto oncogenes are established biological mechanisms. All have been reported from two or more independent laboratories, most in 4 or more laboratories.

ICNIRP RF/MW assessment of Calcium Ion Efflux:

ICNIRP cites only three calcium ion efflux papers of the over 30 which have been published. Two are cited as showing significant effects, Bawin et al. (1975) and Blackman et al. (1979). One is cited as showing no effect, Albert et al. (1987).

The overall conclusion, which applies to all biological mechanisms, including calcium ion efflux, states:

"Overall, the literature on athermal effects of AM electromagnetic fields is so complex, the validity of reported effects is so poorly established, and the relevance to human health is so uncertain, that it is impossible to use this body of information as a basis for setting limits on human exposure to these fields".

This is a carefully and deliberately constructed dismissal of athermal (nonthermal) effects so that epidemiological effects can also be dismissed with the lack of a biological mechanism to justify dismissal. In challenge and contrast to this Dr Carl Blackman, Blackman (1990) concludes that calcium ion efflux is an established biological effect having considered about 20 papers, section 6.2.

ICNIRP ignores most evidence of genotoxicity:

The ICNIRP assessment totally ignores the vast literature on DNA strand breakage, chromosome aberrations, oncogene activity enhancement, melatonin reduction and Schumann Resonance interactions, summarized in Sections 6 and 7.

10. Reproductive outcomes: 100kHz-300GHz

The ICNRIP Statement:

There are several major errors and omissions in the ICNIRP (1998) assessment of reproductive effects, ICNIRP (1998), Figure 19.

This includes misrepresentation of two studies, inadequate interpretation of three studies and omission of several relevant epidemiological studies and failure to cite the relevant animal studies. ICNIRP (1998) concludes that studies involving pregnancy outcome and microwave exposure suffer from poor assessment of exposure, small numbers of subjects and contrasting results. All of these claims and conclusions are wrong.

10.2 The studies of Daels (1973 and 1976):

The first claim is that there are two extensive studies on women treated with microwave diathermy to relieve the pain of uterine contractions during labour, with no evidence of adverse effects on the fetus, quoting Daels (1973 & 1976). Daels (1973 (4 pages) & 1976 (2 pages)). They are very small papers on an analgesic therapy to ease the mother's pain in delivery. They report on the subjective Apgar Score of the new-born child. The score is the sum of indexes related to heart rate, respiratory effort, muscle tone, reflex irritability and colour. Ten is a perfect score. The test is carried out within 30 minutes of the exposure, 1 minute after birth. This is a small fraction of the cell cycle time and therefore cannot detect cellular damage.

Figure 23: The ICNIRP (1998) epidemiological assessment of reproductive effects, p504.

These studies involve short-term microwave heating of the uterine area for 30 to 40 minutes during labour. They recorded a maximum neonate temperature of 37.8(C and amniotic fluid temperature of 36.5(C. These are well within the normal range. Heating was limited to levels where the mother felt skin heating as “agreeable”. Since most of the microwaves are absorbed in the surface skin layers the fetal exposure will be extremely small, see Hocking and Joyner (1995) below.

The Apgar Score showed that the "microwave group" had a slightly higher mean score of 9.1 compared to 8.8 for the "control group". A very low Apgar Score (0-3) indicates gross problems and have been correlated with long-term problems, such as significantly lower Bayley mental scores, Serunian and Broman (1975). Lan et al. (1991) found that low (4-6) and very low (0-3) Apgar Scores were significantly associated with low birth weight. In Daels the lowest Apgar Score was 7, within the normal range. The Daels papers show that the slight, imperceptible heating of the mother during delivery by microwave diathermy, results in a slight improvement in the Apgar Score, attributed to the more relaxed mother because of the warming.

A fully developed child is involved, exposed at extremely low levels for minutes immediately prior to birth, and assessed immediately after birth. There is no assessment of the developed pre-schooler to determine if there was any brain damage or developmental problems that could have resulted from a small risk of chromosome damage.

In Daels (1973) he simply states “No undesirable side effects of microwave heating of tissues are known.” He references a single study, Leary (1959) to note that overheating can be a rare complication. Thus Daels (1973 & 1976) are neither extensive studies nor about subsequent new-born health in the months or years following the birth and the exposure.

It is therefore totally inappropriate and grossly misleading to cite these as “extensive studies” of the impact of microwaves on the fetus. The exposure of the fetus is extremely low and very short. The studies are not extensive, they do not relate to developing fetus and there is no actual assessment of the long-term impact of the exposure on the children.

10.3 Interpretation of Physiotherapy Studies:

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

10.4 Physiotherapist Studies Cited by ICNIRP (1998):

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

Kallen and Larsen involve small samples and short-wave exposure, and whole pregnancy post-natal outcomes. Kallen et al. report significant increases in malformed children and perinatal deaths for physiotherapists using RF diathermy. Larsen et al. observed very few boys, and many more perinatal deaths, premature births and low birthweight children for therapists using shortwave diathermy. Given

these confirming results the reviewers state however “The results suggest further study is necessary before conclusions can be drawn.”

A further study was carried out. Ouellet-Hellstrom and Stuart involves a very large sample, studies only early pregnancy (first trimester) miscarriage and finds only microwaves to have an effect. They observe a significant dose-response increase in first trimester miscarriage for female therapists using microwave diathermy. Following the Bradford Hill guidance, this is indicative of a cause and effect relationship. In addition to Larsen et al. and Kallen et al. this additional study confirms that RF/MW exposure of pregnant women is associated with adverse reproductive outcomes. Despite this ICNIRP found reasons why this data is difficult to interpret.

Several other studies were available prior to 1993 but they were not cited by UNEP/WHO/IRPA (1993). The total available published research on EMR associated reproductive effects was not cited by WHO (1993) nor by ICNIRP (1998).

10.5 Case by case assessment:

10.5.1 ICNIRP misrepresentation:

ICNIRP states that there were “no statistically significant effects on rates of abortion or fetal malformation” in Kallen et al. (1982). This is wrong. Even though Kallen et al. involves small sample numbers they conclude “The only positive finding was a higher incidence of short-wave equipment use among the females with dead and deformed infant than among controls.” Very few therapists were involved with microwaves. Hence Kallen et al. associate fetal death and malformation with the use of short-wave diathermy equipment, with $p=0.03$. This is a statistically significant association, contrary to the ICNIRP claim.

Papers cited by ICNIRP:

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

Ouellet-Hellstrom and Stewart (1993) investigated early pregnancy miscarriage among U.S. physical therapists using short-wave (27 MHz) and microwave (915 MHz and 2.45 GHz) diathermy. The sample included 1753 case pregnancies (miscarriages) and 1753 control pregnancies. They found no significant increase in first trimester miscarriage amongst those using short-wave diathermy. They found a statistically significant increase in miscarriage in the first trimester with microwave exposure (OR= 1.28, 95%CI: 1.02-1.59) and a statistically significant dose response relationship ($p<0.005$) using a dose measure of treatments per month. With more than 20 treatments per month OR = 1.59, 95%CI: 0.99-2.55. In addition to the three studies cited in ICNIRP (1998) there are several others with are relevant.

10.5.3 Additional Studies not cited by ICNIRP (1998):

Male sexual functions are significantly reduced in ELF and RF/MW occupational exposure situations.

Lancranjan et al. (1975) studied 31 young men, average age 33 yr, with long-term (mean 8 yr) exposure to microwaves. "This investigation showed a high frequency of libido decrease and sexual dynamic disturbances in the framework of asthenic syndrome (70% of subjects) as well as various alterations of spermatogenesis ($p<0.001$) in 74% of the subjects. Exposures were to frequencies in the range of tens to hundreds of (W/cm2, and hence were non-thermal.

Nordstrom, Birke and Gustavsson (1983) observed a significant decrease in "normal" pregnancies in high voltage substations in Sweden, almost exclusively as a result of congenital malformations when the father was a high voltage switchyard worker. Nordenson et al. (1988) measured a significant increase in chromosome aberrations in similar workers.

A small group (n=30) of U.S. Military personnel exposed to radar had significantly lower (p=0.009) lower sperm counts, Weyandt et al. (1996). This confirms Lancranjan et al.

Women exposed to ELF to RF/MW radiation experience significant changes in reproductive functions.

Vaughan et al. (1984), studying U.S. workers, found significantly increased risk of fetal death for last pregnancy for therapists, RR=2.0, CI: 1.5-2.5, n=169, and for electronic technicians, RR= 1.5, CI:1.2-2.0, n=202.

Wertheimer and Leeper (1986) found a seasonal pattern of developmental delay and spontaneous abortion which significantly correlated with the use of times when electrically heated beds were used. They were not able to correlate the reproductive outcomes directly with electric field exposure. Subsequent studies have found this, confirming this result could well be EMR related.

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

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

Stronger associations with ultrasound and heavy lifting:

Ultrasound>=20/week, OR= 3.4, CI: 1.2-9.0, n=9
Heavy lifting, > 10 kg or patient transfers >=50 times/week,
OR=3.5, IC: 1.1-9.0, n=11

Odds ratios increased for pregnancies > 10 weeks:

Electric therapies OR=2.2
Shortwaves OR=2.5
Microwaves OR=2.4
Ultrasound OR=3.4
Heavy lifting OR=6.7 .

Taskinen et al. conclude “Physical exertion during early pregnancy seems to be a risk factor for spontaneous abortion. The findings raise suspicion of potential harmful effect of shortwaves and ultrasound on the pregnancy, but no firm conclusion can be drawn on the bases of these results alone.”

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

Taskinen et al. (1990) was the only Scandanavian study to have a large enough sample to investigate the effects of miscarriage with microwaves. The sample was quite small (13), limiting the significance of the result. The Odds Ratio was (OR= 1.8, 95% CI 0.8-4.1). Exposure to ultrasound and short-wave showed

significant increases in odds ratio for abortion after the 10th week of gestation, (OR = 3.4, p<0.01 and OR = 2.5, p<0.03, respectively). Taskinen et al. concluded: “The effect of shortwaves and ultrasound on the ‘late’ spontaneous abortions was significant and increased in a dose response manner.”

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

Larsen (1991) found a non-significant elevation in congenital malformations in a small (n=54) group of RF exposed Danish physiotherapists, OR = 1.7, 95%CI: 0.6-4.3.

Lindbohm et al. (1992) observed a dose-response relationship between the level of exposure to VDTs and miscarriage. VDTs also emit RF radiation.

Exposure	RR	95%CI
<0.13(T	1.0	Reference
0.13-0.3 (T	1.9	0.9-3.9
>0.3 (T	3.4	1.4-8.6

Evans et al. (1993) compared reproductive outcomes between Magnetic Resonance Workers and other groups. MRI workers had elevated outcomes compared with other workers but compared with homemakers they were highly elevated:

Outcome	RR	95%CI
Miscarriage	3.22	1.74-5.97 (p=0.0001)
Early Delivery	1.71	0.87-3.38
Low Birth Weight	1.52	0.52-4.41

Juutilainen et al. (1993) observed a significant early pregnancy loss associated with "high" residential 50 Hz exposures ((0,63 (T at the front door), OR = 5.1 (1.0-25).

Savitz et al. (1996) investigated the association between maternal occupation and pregnancy outcomes. For women using electrical equipment the following results were found.

	Any time in Pregnancy			Fifth Month of Pregnancy		
		Adjusted			Adjusted	
Symptom	N	OR	95%CI	N	OR	95%CI
Preterm delivery	57	1.8	0.9-3.3	46	1.8	0.9-3.5
Very Low Birth Wt	36	1.5	0.9-2.3	24	1.2	0.7-2.1
Mod. Low Birth Wt	33	1.4	0.9-2.3	30	1.5	0.9-2.5
Stillbirth	54	1.2	0.8-1.7	50	1.2	0.8-1.9
Infant death	70	1.0	0.7-1.3	54	0.9	0.6-1.4
Small for Gestational Age	33	1.4	0.9-2.3	30	1.5	0.9-2.5

The electrical occupation was the most consistently the highest OR for these reproductive effects. They are marginally non-significant because of the very small sample size.

Belanger et al. (1998) conducted a prospective study (N= 2967) to evaluate the relation between spontaneous abortion and the use of electrically heated beds. Electric blanket use was associated with increased spontaneous abortion, OR = 1.84, 95%CI: 1,08-3.13 for unadjusted data, and OR = 1.74, 95%CI: 0.96-3.15 for data adjusted for other risk factors such as alcohol, smoking, age and caffeine intake.

Summary and conclusions:

ICNIRP ignores several male studies showing significant reduction in sexual function. The ICNIRP reproductive assessment also fails to take into account 11 relevant studies that reconfirm the conclusions of Kallen et al., Larsen et al. and Ouellet-Hellstrom and Stewart. This shows how limited and therefore unprofessional the ICNIRP assessment is.

The studies involving low frequency EMR exposure reinforce and support the RF/MW exposure studies through the EMR Spectrum Principle.

Vaughan et al. (1984), Taskinen et al. (1990), Sanjose et al. (1991), Lindbohm et al. (1992) and Larsen (1991) are consistent with Kallen et al. (1982) and Larsen et al. (1991) giving the conclusion that shortwave exposure takes longer to produce effects than do microwaves. Shortwave effects range from later pregnancy miscarriage, still birth, low birth weight, premature birth, cot death and congenital abnormalities.

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

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

When all the studies are taken together they form a comprehensive and compelling body of research to show that microwave exposure of mothers leads to a significant increase in early pregnancy miscarriage. There are two significant dose-response relationships. They occur with those using short-wave radio and microwave therapies and working in electrical industries, who have more late pregnancy problems and malformed children. This amounts to a causal relationship between EMR exposure and adverse reproductive outcomes.

10.5.6 Plausible Mechanism:

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

Calcium ion efflux lead to the survival of damaged cells that carry their chromosome aberrations into future generations of cells. A reduction in melatonin reduces the elimination of free radicals which enhances the chromosome damage. Calcium ion efflux and melatonin reduction also impairs the immune system with allows a greater population of damaged cells to survive. Cells with damaged chromosomes are a known cause of spontaneous abortion.

According to Sandyk et al. (1992):

"The causes of spontaneous abortion can be divided into two main categories: those arising from chromosomal anomalies and those arising from abnormalities in the intrauterine environment. In the following communication, we propose that deficient pineal melatonin functions in early pregnancy may be

causally related to the development of spontaneous abortions in cases where chromosomal anomalies or structural abnormalities of the uterus have been excluded."

Microwaves are shown to be associated with DNA breakage in rats brains, Lai and Singh (1995, 1996, 1997b), Sarkar et al. (1994) and Phillips et al. (1998), and to cause chromosome aberrations, Heller and Teixeira-Pinto (1959), Garaj-Vrhovac et al. (1990, 1991, 1992, 1993), Haider et al. (1994) and others. Lai and Singh (1997b) show the links to melatonin reduction and free radicals.

ICNIRP (1998) quotes Cohen et al. (1977) which found no association between radar exposure and Down's syndrome in their off-spring. They failed to mention a previous paper from the same group, Sigler et al, (1965), which did find a significant risk from parental radar exposure.

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

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

10.6 Animal Toxicology:

ICNIRP (1998) fails to refer to the significant research involving animal experiments on reproductive effects when exposed to RF/MW. Results range from testicular degeneration, resorption of the fetus and altered body weight at high but non-thermal levels of exposure to total infertility in multigenerational studies of mice exposed to 0.168(W/cm2 and 1.053(W/cm2, Magras and Xenos (1997). There are many animal studies showing that RF/MW is teratogenic, that is, it causes severe reproductive problems. Berman et al. (1982) introduce their paper by stating:

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

Berman et al. (1982) extended the studies to hamsters. They investigated the teratogenic potential of microwaves on Syrian hamsters, using 2.45 GHz at power densities of 30 mW/cm2 for 100 minutes daily This caused a temperature rise of 0.8 (C and significant fetal resporptions or death ($p = 0.0012$), decreased fetal body weight ($p=0.0001$) and decreased skeletal maturity. Averaging this over a whole day the mean exposure is 2.08 mW/cm2. Maternal toxicity was not observed, only fetal damage and death. They conclude by comparing hamsters with mice.

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

Prausnitz and Susskind (1962) exposed male Swiss albino mice to 9.27 GHz microwaves, pulsed with a 2 (s pulse at 500 Hz, 4.5 mins per day, 5 days per week for 59 weeks with an exposure level of 100

mW/cm2. This is a thermal exposure load which would result in a temperature rise of about 2(C. This amounts to a mean weekly exposure of 22(W/cm2.

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

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

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

In these mice significant and severe (4.2-fold) testicular damage and a 2-fold increase in the initiation of leukaemia occurred is association with a mean exposure of 22(W/cm2.

Testicular damage has also been found in men who have radar exposures. Lancranjan et al. (1975) and Weyandt et al. (1996).

Although as early as 1962 severe reproductive problems had been identified with and exposure regime averaging 22 (W/cm2 most of the research was carried out with the incorrect assumption that if an effect was real it would be demonstrated if the exposure was high enough. And if an effect was not detectable at extremely high levels of exposure, there was no way that an effect would occur at low levels of exposure.

Chazan et al. (1983) investigated the development of murine embryos and fetuses after irradiation with 2450 MHz microwaves at 40 mW/cm2. They found indications of retardation of development in the early period of gestation in mice exposed to thermal MW fields. During the second half of pregnancy an increase in the number of resorptions, stillbirths and internal hemorrhages was noted. The living fetuses had lowered body mass compared to the offsprings of sham-irradiated mice.

Berman, Carter and House (1982) also found reduced weight in mice offspring after in utero exposure to 2450-MHz (CW) microwaves using an exposure level of 28 mW/cm2. They were exposed to for 100 minutes daily from the 6th through 17th day of gestation. This gives a mean exposure during that period of 1.9 mW/cm2. These data demonstrate that the decreased fetal weight seen in microwave-irradiated mice (-10 %) detected in utero and is retained at least 7 days after birth. Evidence from other published studies is presented to show that the retarded growth is persistent and might be interpreted as permanent stunting.

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

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

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

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

Magras and Xenos (1997) responded to health concerns among residents living in the vicinity of an RF transmission tower in Greece, by placing groups of mice at various locations in relation to the tower. The mice fertility was monitored over several generations and related to the RF exposure. Figure 10 shows the fertility rate of the two exposed groups. Where group A the “Low” exposure group (0.168 (W/cm2) became infertile after 5 generations and B the “High” exposure group (1.053 (W/cm2), became infertile after only 3 generations. This is a highly significant result because so few multi-generation studies have been done and the effects of this study occur at extremely low levels and the effect is total infertility.

The Greek study confirms the Australian study, but shows that over several generations the infertility is complete at very low levels of mean RF/MW exposure, Figure 24.

Figure 24: Multigenerational exposure of mice to low level RF leads to complete infertility.

10.7 Summary and conclusions about teratological animal studies:

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

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

A much lower microwave dose was associated with significant reduction in birth weight and permanent stunting and slowing of bone hardening. Changes in chick embryo biofeedback learning is observed and testicular atrophy was observed with a mean exposure to a radar-like signal averaging 22 (W/cm2 over a week. Total infertility occurred in mice after 5 weeks of exposure to 0.17(W/cm2.

Thus in 1962 and 1997 it is been shown that chronic mean low level microwave exposure of animals leads to very significant adverse reproductive effects in males and females down. The effects were still significant at exposures of 22 and 0.17(W/cm2. These are close to the level of the lowest published results for calcium ion efflux, 0.00015 W/kg (0.08(W/cm2) Schwartz et al. (1990).

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

10.8 Reproductive Health Effects Conclusions:

The ICNIRP (1998) assessment of reproductive effects from RF/MW exposure is severely flawed. Animal studies show that chromosome aberrations and single and double strand DNA breakage occurs with EMR exposure, mice and rats have pregnancy, birth and fertility problems associated with EMR exposure which are also found in exposed human populations. There is consistency within human studies and between human studies and animal studies. Many human studies show statistically significant adverse reproductive outcomes. Two human studies, Lindbohm et al. (1992) and Ouellet-Hellstrom and Stewart (1993), gave a statistically significant dose response relationship. This study allows an exposure assessment to be carried out, along with the multigeneration mice study, Magras and Xenos (1997).

This evidence supports a causal relationship between EMR exposure and serious adverse reproductive outcomes such as miscarriage, prematurely, still birth, low birth weight, SIDS and congenital malformations.

10.9 Exposure Assessment:

Ouellet-Hellstrom and Stewart (1993) report that the microwave exposure was primarily from leakage, which at waist level was measured in the range 80 - 1200 (W/cm2. At 15 cm from the source the highest reading was 15 mW/cm2. The therapist needs to be leaning over the patient during the therapy to receive this dose. This is highly unlikely when the machine is turned on. Even so, this is not sufficient to cause a surface heating of the skin in the few minutes it is likely to involve. Hocking and Joyner (1995) show that microwaves produce very small SARs with the uterus, in the following Figure 25.

In their table 2 Hocking and Joyner (1995) show maximum SARs in the uterus for the conditions in Figure 11 for short-wave (27.12 MHz) of 0.209 W/kg, for microwave (915 MHz) of 0.023 W/kg and for microwave (2.45 GHz) of 0.000027 W/kg.

Gandhi (1990) gives the relationship between SAR and temperature increase. The heating rate given is 0.0045 x SAR (C/min. With a maximum exposure time per treatment of 5 minutes, and an external field intensity of 1,200 (W/cm2, the heating of the fetus will be 0.0055, 0.00062 and 0.00000073 (C, respectively. Not even at 15 mW/cm2 does the short-wave exposure can produce a detectable heating effect in the uterus environment (0.071(C).

Figure 25: Specific absorption rate (SAR) profile across the uterus for a small woman exposed to 1 mW/cm2, from Hocking and Joyner (1995).

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

Dose-Response Relationship for MW exposure:

It is not the habit of therapists to stand close to the patient during the diathermy. In many cases the therapist leaves the room while the 15 to 30 minute diathermy is carried out. Hence a conservatively long mean exposure period of 3 minutes is chosen to be associated with the exposure range of 80 - 1200 (W/cm2, average 600(W/cm2. The dose-response relationship is expressed in terms of treatments per month.

One treatment per month is associated with a mean monthly exposure of 0.042(W/cm2.

The results are plotted in Figure 26, showing a significant dose response relationship with a threshold near zero exposure, The trend line is fitted using a least squares fit.

Figure 26: Microwave exposure associated miscarriage for pregnant physiotherapists, Ouellet-Hellstrom and Stewart (1993).

Table 3: Estimated mean exposure ranges, from Ouellet-Hellstrom and Stewart (1993).

	No. of Exposures per Month		Odds Ratio	Exposure Regime ((W/cm2)
		Mean		
All pregnancies		0	1.00	0.0
	<5	(2.5)	1.05	0.105
	5-20	(12.5)	1.50	0.53
	>20	(21)	1.59	0.88

11. Cancer Assessment:

11.1 Laboratory Experiments:

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

The effect of microwaves neoplastically transforming a standard mice embryo cell line, a cell line which has been used several times in chemical carcinogen assessment are treated in the same inaccurately dismissive manner, p507, referring to the work of Balcer-Kubiczek and Harrison (1991). These researchers carried out a series of very careful and extensive laboratory assessments using a standard mouse cell line. One of their most significant results is presented below, Figure 27.

Figure 27: Dose response relationship for the induction of neoplastic transformation of C3H/10T1/2 cells by a 24 h exposure to 2.45 GHz microwaves at specific absorption rate indicated on the abscissa with or without TPA post-treatment for 8 weeks, Balcer-Kubiczek and Harrison (1991).

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

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

Further on Dr Balcer-Kubiczek states:

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

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

The use of the word “may” when the effect clearly does occur is wrong. The implication is clear if you want to see it, which the reviewer obviously does not. In context, animal skin, when treated with TPA or similar chemical cancer promoters, has the rate of cancer cell formation increased by microwaves. This experiment shows that it also does happen at the cellular level. That is, microwaves are carcinogenic at the tissue and cellular level. It is then not surprising that epidemiological studies also show that RF/MW increase cancer. But ICNIRP (1998) ignores and misrepresents that evidence too.

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

12. Epidemiology of Cancer:

12.1 Summary of ICNIRP's assessment:

The cancer assessment, ICNIRP (1998) p 504, Figure 24, references one review (UNEP/WHO/IRPA 1993), WHO (1993), and 13 papers covering 11 studies. The WHO (1993) review, is limited by citing only 6 epidemiological studies and, by not reviewing the actual results, contains errors, which are propagated through to the ICNIRP assessment.

In ICNIRP (1998), only 13 papers are cited directly:

Barron and Baraff (1958): The study group is too small (226) and the follow up period (4-13 years from first exposure) is too short to detect cancer. Cancer is not one of the paper’s studies chosen outcomes. It is grossly dishonest and misleading to include this paper in a cancer assessment and to cite it as showing that there are no cancer risks from exposure to radar.

Robinette et al. (1980): Is widely claimed to show no effects when its data does show significant adverse human health effects, including a significant dose-response relationship for respiratory cancer.

Lilienfeld et al. (1978): Is widely claimed to show no effects when its data does show significant adverse human health effects, including neurological, cardiac and cancer effects and includes a significant dose-response relationship for rates of sickness as a function of years in Moscow.

Figure 28: The Epidemiological assessment of cancer effects in ICNIRP (1998).

Selvin et al. (1992): Is widely claimed to show no effects when it was aiming to develop an epidemiological method relating to spatial clustering. Its data does show significant adverse human health effects, including significant dose-response relationships when radial cancer rates are related to radial exposure measurements.

Beall et al. (1996): Is quoted by ICNIRP as failing to show significant increases in nervous system tumours. Actually it does show many significant increases of brain tumors, and it includes a significant dose-response relationship between years of exposure and rates of brain tumor for computer programmers.

Grayson (1996) Is quoted by ICNIRP as failing to show significant increases in nervous system tumours, when it does show a significant increase in brain tumor for RF/MW exposed personnel.

Rothman et al. (1996a): ICNIRP acknowledges that it is still too early to observe an effect of cancer incidence and mortality from mobile telephone use as yet.

Rothman et al (1997b) ICNIRP acknowledges that it is still too early to observe an effect of cancer incidence and mortality from mobile telephone use as yet.

Szmigielski et al. (1988): finds significant increases in cancer across the body, especially leukaemia incidence and mortality among Polish Military personnel exposed to radio and radar. ICNIRP says is difficult to interpret because neither the size of the population nor the exposure levels are clearly stated. In fact the Polish Military microwave exposure regime is presented and the group is described by the authors as “large and well controlled”.

Szmigielski (1996): ICNIRP acknowledges that Szmigielski found significant increases in leukaemia but criticizes the exposure assessment and the description of the population. Again, the overall group exposure regime is well described, but as in all large population studies, individual exposures are not monitored but group exposures can be well classified.

Hocking et al. (1996), (12.) Dolk et al. (1997a) and (13.) Dolk et al. (1997b) are acknowledged as “suggesting a local increase in leukaemia incidence” in populations living in the vicinity of TV/FM transmission towers, but ICNIRP calls the results “Inconclusive”. When the radial cancer rates are compared with realistic broadcast transmission patterns, they form highly significant dose response relationships and very strong evidence of a causal relationship.

ICNIRP's overall cancer assessment conclusion that: "Overall, the results of the small number of epidemiological studies published provide only limited information on cancer risk."

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

Much more evidence of RF/MW induced cancer is available:

Zaret (1977), Lester and Moore (1982 a,b) and Lester (1985), Milham (1985, 1988), Thomas et al. (1987), De Guire et al. (1987), Archimbaud et al. (1989), Hayes et al. (1990), Tornqvist et al. (1991), Maskarinec and Cooper (1993), Band et al. (1996), etc. In addition, the reviews of Goldsmith (1995, 1996, 1997a,b) are ignored. Manyl other papers are relevant. Occupational cancer studies identify a wide range of exposure agents, including RF/MW in occupational groups. For example, for "electrical, electronic manufacture and communications", such as Kaplan et al. (1997), who found an elevated risk of brain tumour (OR=2.2 (0.5-9.3)). Cantor et al. (1995) found significant increases in breast cancer for RF/MW exposed women in the United States. Thus there is at least three times as many papers available than those cited by ICNIRP.

It is a difficult and challenging task for an individual scientist to take on and criticize the largest and most prestigious bodies in the world, such as the WHO and the ICNIRP. However, science not only allows this to occur, but supports and even demands a comprehensive review of the data contained in the published material, an inter-comparison between studies and an accurate quotation of the results and analyses given. It also supports correction of analyses where errors are identified, and new analyses where data suggests that more can be shown by standard scientific methods.

The summary above gives a strong indication of the ways in which ICNIRP have selectively used and consistently misquote the studies they have chosen to assess. Hence the conclusions which should be

drawn are quite different than those ICNIRP arrived at. In order to substantiate the brief claims made above this review will outline and list the detailed data contained in the studies cited.

12.3 Data analysis and presentation principles:

Some analysis principles are set out and then the detailed data is presented.

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

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

It is easier to present biased conclusions than to falsify data. In many studies the data shows significant health effects which are ignored in the abstracts and conclusions.

Every scientist is a person with a degree of subjectivity and bias. Hence science uses principles and methods involving careful checking and peer review. Basic scientific training makes it very difficult (though not impossible) for a scientist to falsify data.

Analysis of data is more subject to error and bias in its use and interpretation. Errors can be simple arithmetic errors or errors in programming and data entry. Checking procedures are usually in place to significantly reduce the chance of this occurring.

Subjective bias is frequently involved in the choice and interpretation of statistics which makes the principles of the application of statistical methods and agreed systems of interpretation vital.

Epidemiology is the basic science of preventive medicine and public health, and biostatistics is the quantitative foundation of epidemiology, Jekel et al. (1996).

The test of statistical significance:

In epidemiology it is agreed that a statistically significant result is one which reaches the 1-in-20 or 5 % threshold for statistical probability. In calculating the value of the statistical probability or p-value (p), a single direction effect is tested against a one-tail distribution while a bi-directional effect is tested against a two-tailed distribution. This requires half the population to achieve statistical significance when searching for an adverse effect than when the hypothesis involves the possibility of a positive and a negative effect.

Epidemiology deals with chronic exposure of populations whereas the ICNIRP guideline is based on acute thermal effects on individuals. An important characteristic of epidemiology is its ecologic perspective. People are seen not only as individual organisms but also as members of communities in a social context.

Classical epidemiologist studies the community origins of health problems. Classical epidemiologists are interested in discovering risk factors that might be altered in a population to prevent or delay disease or death.

Death is only one of the outcomes of concern. In general many more people are made ill by a disease agent than those who die of it. Illness has a significant personal, social and economic cost which makes the prevention of illness a worthy goal.

Dose response relationships are indicative of causal relationships and need to be taken very seriously, Hill (1965).

Chronic mean exposures are much lower than acute peak exposures. Health risks such as cancer are related to cumulative cellular damage from inaccuracy on DNA repair processes and failure of the body to eliminate genetically damaged cells. Hence the chronic mean exposure is the appropriate metric for assessing health effects.

There are a large number of studies that involve RF/MW exposures and show elevated and significantly elevated cancer incidence and mortality. These are dismissed by WHO and ICNIRP assessors because of the lack of a well defined exposure measurement. Dr Szmigielski confirms, pers.comm., that even the highest acute military exposures in the Polish Military study are nonthermal, and are associated with daily average exposures around 1-5% of the daily peak exposure, and lifetime mean exposures about 20-30% of the daily mean work day exposure. These chronic mean exposures are at a small fraction of the ICNIRP guideline.

Significant increases in cancer are also found in residential RF/MW exposures around TV/FM towers with annual mean exposures around 15% of the direct exposure at the primary residence. Hence there is a large body of epidemiological evidence showing significant increases in cancer in RF/MW exposed populations whose chronic mean direct exposures of less than 0.1 to 0.2(W/cm2, and hence involve chronic mean exposures in the range 0.015 to 0.03(W/cm2. This firm knowledge of the actual mean exposure is not necessary in order to revise the exposure standard when significant health effects occur at exposure levels of less more than 1000 times below the present standard.

13. Detailed evaluation of ICNIRP cited papers and reports:

13.1 Barron and Baraff (1958): "Medical considerations of exposure to microwaves (radar)"

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

With the working age incidence of all cancers at about 100 per 100,000 per year, over the 4 years of this study the probable number of normally occurring cancers would be 0.9. This paper cannot and does not assess cancer risk from radar exposure.

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

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

To include this study in a cancer risk assessment is knowingly misleading and deceptive. This level of bias and error is unbecoming of an international assessment of quality and merit. This, along with several other similar examples, must bring the scientific objectivity and professional credibility of the person or group who produce this assessment into serious question.

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

13.2.1 Introduction

This is one of two epidemiological studies which ICNIRP states "found no evidence of increased morbidity or mortality from any cause". Both WHO (1993) and ICNIRP (1998) treat this is a large and reliable study which shows that there are no effects from radar exposures. Actually it is a large reliable study which does show significant health effects.

Epidemiological studies regarding cancer are affected by the complexities and long time scales involved in the initiation, promotion and progression of cancer. This process can take decades from the initial cell damage and genetic transformation of cells to the development of tumors and malignant cancers. To some extent the individual complexities and the complex nature of post war exposures to carcinogens over 20 years are smoothed by considering large populations. This study involves around 40,900 sailors with advanced technical training who served on ships during the Korean War and were exposed to radio and radar signals. Their mortality statistics and health status about 20 years later was obtained and analyzed for evidence of differences which could be related to the RF/MW exposure. If the EMR exposure had caused a great amount of initiation and/or promotion of cancer then this study has the ability to reveal it.

An early challenge was to identify exposure groups so that the health status of a large group with lower mean exposures could be compared with a group that had received higher mean exposures. Comparing technical sailors with similar age structures reduced confounding. The naval advisors recommended that operators of radio and radar would have lower mean exposures compared with those sailors who repaired and maintained the radio and radar equipment. Hence the low exposure group included Radioman (RM) and Radarman (RD). The technical people, including Electronics Technician (ET), Fire Control Technician (FT) and Aviation Electronics Technician (AT) were placed in the high exposure group.

A fourth technical group, Aviation Electrician's Mate (AE), a group which is clearly involved with repairs and maintenance, was placed in the operators group, the low exposure group. The AE group has a moderately high mortality rate and plays the role of diluting the difference between the groups.

The problem of high exposures for the radar and radio operators on ships was pointed out when the preliminary results were presented to a conference, Robinette and Silverman (1977).

13.2.2 Hazard Number Assessment:

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

Table 4: Distribution of assessed Hazard Number for the three assumed high exposure groups, Robinette et al. (1980).

Hazard	Electronics	Fire Control	Aviation Electronics

Number		Technician (ET)		Technician (FT)	Technician (AT)
	%	%	%		
0	27.8	6.6	12.5		
1 - 2000		28.3	23.4	16.9	
2000-5000		20.0	31.1	17.6	
5001+		10.6	25.8	48.6	
Unknown		13.3	13.1	4.3	
Mean HN		1610	2870	3700	

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

Health survey results:

From the exposure survey there was a group of individual for whom each person was assigned a Hazard Number that was proportional to his exposure risk. Of those who had died, they identified 63 sailors with Hazard Number of 0, 160 with 1-5000 and 86 with 5001+. The mortality results are presented in Table 5.

Table 5: Number of deaths from disease and mortality ratios by Hazard Number: US enlisted Naval personnel exposed to microwave radiation during the Korean War period, from Table 9, Robinette et al. (1980). The Rate Ratio is calculated as the ratio of the Mortality ratio for Hazard Number 5001+ exposure and 0 Hazard Number exposure.

Cause of Death	Hazard Number			Trend					
	No.	0	1-5000	5001+	p-value	RR	95%CI		
All diseases	309	0.82	0.91	1.23	0.03	1.50	1.08-2.08		
Malignant Neoplasms	96	0.99	0.90	1.44	N.S.	1.45	0.83-2.52		
Digestive Organs	20	1.49	1.14	0.78	N.S.	0.52	0.13-2.08		
Respiratory Tract	24	0.82	0.86	2.20	<0.05	2.68	0.84-8.55		
Lymphatic and hematopoietic System	26	1.09	1.04	1.64	N.S.	1.50	0.52-4.32		
Other Malignant neoplasms	26	0.78	0.70	1.17	N.S.	1.50	0.52-4.32		
Disease of Circulatory System	150	0.94	0.83	1.17	N.S.	1.24	0.79-1.94		
Other Disease	63	0.30	1.13	1.08	N.S.	3.60	1.14-9.20		

Given the exposure dilution factors, all but digestive organs would probably have RR>2 and be significantly be increased. This small sample analysis shows a significant dose response trend for mortality from all diseases (p=0.03) and for Respiratory Cancer (p<0.05). This is remarkable given the exposure dilution. The analysis also shows that for every disease cause but one there is an elevated risk of mortality due to a range of cancers, Circulatory Disease and Other Disease.

The mean Hazard Number for each group is calculated using a mean hazard number of 0, 1000, 3500 and 6000 for the defined ranges. The mean exposure estimate also shows a gradient and suggests that the best dichotomy will be achieved by comparing AT as a high exposure group to ET as a low exposure group. This was not done by Robinette et al. who preferred to compare ET with the FT and AT groups combined (FT+AT). This maintains larger numbers in the high exposure group by reduces the exposure separation.

The mortality dose-response gradient persists when the total mortality rate is calculated for the ET, FT and AT groups: MR (ET) = 1.0; MR(FT) = 1.29; and MR(AT) = 1.79.

Having identified that the FT and AT groups had higher hazard numbers than the ET group, Robinette et al. combined FT + AT and compared their mortality rates with ET, Table 6. Table 6 shows elevated mortality rates compared with the ET group, for all causes of death listed. The text records that they are significantly elevated for All Disease ($p<0.01$) and Other Diseases ($p<0.01$).

Table 6: Mortality rates, Risk Ratios and Confidence Intervals between the ET group and AT group of US enlisted personnel exposed to microwave radiation during the Korean War.

Cause of death	No.(FT+AT)		ET	FT+AT	RR	95%CI
All diseases	140	0.83	1.19	1.43	1.14-1.79	
Malignant Neoplasms	40	0.95	1.18	1.24	0.83-1.86	
Digestive Organs	8	1.10	1.19	1.08	0.44-2.65	
Respiratory Tract	9	1.13	1.15	1.02	0.45-2.33	
Lymphatic and Hematopoietic System	11	1.06	1.40	1.32	0.61-2.87	
Other malignant neoplasms	12	0.68	1.06	1.56	0.72-3.37	
Diseases of the Circulatory System	64	0.85	1.08	1.27	0.92-1.75	
Other disease	36	0.61	1.46	2.39	1.45-3.94	

Robinette et al.'s Table 5 sets out the mortality data by cause of death for each occupational group, giving the opportunity to compare AT rates with ET rates of mortality. The results are shown in Table 6.

Table 7: Mortality Incidence per 1000 and Risk Ratio (AT/ET) as an indication of the high exposure (AT) to low exposure (ET) difference.

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

In Table 5 where exposures are more dichotomized, mortality due to Malignant Neoplasms and Lymphatic/Hematopoietic cancers are both significantly elevated but when FT and AT are combined these results are no longer significantly different. It is interesting too that in the dose-response analysis using the individual's hazard number, respiratory cancer shows a significant trend, but in these occupational group comparisons this cancer is elevated but not significantly elevated. The comparisons between Tables 6 and 7 clearly show the effect of dilution through combining the FT and AT groups. Table 7 shows elevated Risk Ratios for all causes of death except motor vehicle and suicide. Significant

increases in mortality were found for All Diseases, Malignant Neoplasms, and Lymphatic and Hematopoietic cancer. Very significant increases were found for All Causes of death, Accidental Death and Death from diseases of the Digestive System.

Figure 29: Naval occupations grouped by exposure category, showing dose response increases in mortality for all mortality, all disease, cancer and Lymphatic/Leukaemia. Low exposure (RM+RD), Intermediate exposure ET+FT), High exposure (AT).

Figure 29 shows that the overall mortality rates for all death, disease, cancer and leukaemia/lymphoma, for the large occupation groups increase significantly and in a dose response manner with radar exposure. This is similar to the 5% exposure survey sample. The Aviation Electrician's Mate (AE) group has been omitted because it is a repairer group included in the operator's group by the original authors.

Morbidity Data:

Robinette et al. obtained two morbidity data sets. The first was from the periods 1952-54 and 1956-59 for admissions to naval hospitals. This is very close to the period of exposure and allows little time for cancers to develop. For the immediate post-war data set the following significant increases in sickness were identified by Robinette et al.:

Diseases of the ears, nose and throat ($p<0.01$),
Acute respiratory disease ($p<0.01$),
Other respiratory disease ($p<0.02$),
Diseases of the urinary and male genital organs ($p<0.05$), and
Accidents, poisonings and violence ($p<0.001$).

The second data set was from Veterans' Administration Hospitals for the period 1963-76.

Table 8 gives a more detailed description of the results of the later morbidity data set. It is not inconsistent with the significant results cited by Robinette et al. but it does show a wider range of significant adverse health effects.

In the later, to 1976, VA compensation data Robinette et al. found significantly increase in sickness for Musculoskeletal system and other organs, including:

Loss of part extremities, osteomyelitis and neoplasms of bone or muscle ($p<0.001$);
Organs of special sense which includes eye cataracts ($p<0.05$);
Respiratory system, excluding pulmonary tuberculosis ($p<0.01$);
Cardiovascular system ($p<0.001$); and
Mental disorders, including psychoses, psychoneurotic disorders and so-called "psychophysiologic disorders" ($p<0.05$).

Table 8: Number of hospitalizations and hospitalization rates per 10,000 per year, in VA hospitals, 1963 - 1976, by diagnosis and exposure class: US enlisted Naval personnel exposed to microwave radiation during the Korean War period. The significance p-value is calculate from the Mantel-Haenszel Chi-squared estimate.

			High exposures						
VA diagnostic class	Total		ET		FT + AT				
	No.	Rate	No.	Rate	No.	Rate	RR	95% CI	p-value
Infective, parasitic	42	1.5	24	1.3	18	1.9	1.46	0.79-2.69	0.26
Neoplasms, malignant	34	1.2	17	1.0	17	1.8	1.80	0.92-3.53	0.04

Neoplasms, other	26	0.9	9	0.5	17	1.8	3.60	1.60-8.08		<0.001
Allergic, endocrine system, metabolic and nutritional dis.			77	2.8	41	2.3	36	3.8	1.65	1.05-2.58	0.01
Blood, blood-forming organs	17	0.6	5	0.3	12	1.3	4.33	1.53-12.3			0.001
Alcoholism	105	3.8	45	2.5	60	6.3	2.52	1.71-3.71		<0.001	
Other mental disorders			276	10.1	166	9.3	110	11.6	1.25	0.98-1.58	0.02
Nervous system, sense org.	106	3.9	58	3.2	48	5.1	1.59	1.08-2.33			0.009
Circulatory	123	4.5	68	3.8	55	5.8	1.53	1.07-2.18		0.007	
Respiratory	80	2.9	43	2.4	37	3.9	1.63	1.05-2.53		0.014	
Digestive	255	9.3	132	7.4	123	13.0	1.76	1.38-2.25		<0.001	
Genitourinary	82	3.0	45	2.5	37	3.9	1.56	1.01-2.41		0.02	
Skin, cellular	61	2.2	33	1.8	28	2.9	1.61	0.97-2.66		0.04	
Bones, organs of movement	80	2.9	36	2.0	44	4.6	2.30	1.48-3.57			<0.001
Trauma	108	3.9	53	3.0	55	5.8	1.93	1.32-2.81		<0.001	
Symptoms, ill-defined cond., special exams and other			151	5.5	85	4.8	66	6.9	1.44	1.04-1.99	0.007
Person-years (1000)	27.39		17.89		9.50						

The Table 9 shows all of the diagnosis groups detailed in Robinette et al. Table 12, for VA compensation claims up to December 1976. Again the vast majority of symptoms (apart from Nerves, and Genitourinary) are marginally significant to very significantly greater for the higher exposed FT+AT group compared to the lower exposed ET group. Except for "Nerves" all symptoms are elevated and some, as also identified by Robinette et al., are significantly and highly significantly elevated.

Table 9: 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, Robinette et al. Table 12.

	ET		FT+AT		Risk Ratio		Significance	
	No.	Rate	No.	Rate	RR	95% CI		p-value
Diagnosis:								
Musculoskeletal	115	8.8	119	16.9	1.93	1.49-2.49		<0.001
Organs of special sense	49	3.7	42	6.0	1.62	1.07-2.45		0.010
Systematic conditions	3	0.2	5	0.7	3.50	0.84-14.65		0.080
Respiratory	55	4.2	51	7.3	1.74	1.19-2.55		0.001
Cardiovascular	43	3.3	47	6.7	2.03	1.34-3.07		<0.001
Digestive	74	5.7	55	7.8	1.37	0.97-1.94		0.02
Genitourinary	31	2.4	10	2.7	1.13	0.55-2.30		0.32
Skin	83	6.3	58	8.2	1.30	0.93-1.82		0.052
Endocrine	15	1.1	11	1.6	1.45	0.67-3.16		0.86
Neurological	21	1.6	16	2.3	1.44	0.75-2.76		0.29
Nerves	15	1.1	3	0.4	0.36	0.10-1.24		0.14
Mental Conditions	51	3.9	46	6.5	1.67	1.12-2.49		0.003

Discussion of Results:

This project was conducted with the objective of determining whether radar exposure to service personnel during the Korean War produced health hazards. It appears evident that the authors of the study were under pressure not to identify any adverse health effects. They identified significant dose response increases of mortality and respiratory cancer with exposure and many elevated and significantly elevated cancer and health mortality and morbidity from the separated radar exposed groups. Even so, they decided to conclude that they couldn't associate these effects to radar exposure. Their abstract includes the conclusion:

"No adverse effects were detected in these indices that could be attributed to potential microwave radiation exposures during the period 1950-1954."

This is clearly not true. Their published conclusion is expressed differently by Dr Silverman in a 1979 conference paper, Silverman (1979). She states:

"While some significant differences among occupational groups classified by 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."

Here Dr Silverman uses the term "direct radar exposure". She points out that no measures of actual as opposed to potential exposure were available. Hence occupational groups consisted of mixed exposure experiences. Dr Silverman notes the dilution effects that this produces because the high exposure AT group contained nearly 30 % of people with Hazard Number <2000, while the low exposure ET group had nearly 24 % who were in the >5000 category.

Dr Silverman is arguing that the results relate to potential and not actual exposure, which conflicts with the published paper that refuses to relate the effects even to potential exposure. What both Dr Silverman on her own and Robinette, Silverman and Jablon together fail to recognize and appreciate are that all dilutionary influences, by their very nature, weaken the dichotomization and reduce the contrasts between exposed and control groups. These data sets are strongly influenced by several dilutionary factors.

A high exposure 'repairer group' (AE), was placed in the 'operator', low exposure control group.

All participants are more highly exposed to radar than the average male population of the same age, Lin et al. (1985).

Combining the FT and AT groups reduces the exposure separation.

All of the three 'high exposure' groups contain a mixture of low, middle and high exposure individuals.

Dilution weakens and destroys dichotomization. Hence it is remarkable and highly significant that elevated, significant, highly significant and dose-response differences still persisted through to the health statistics 20 years after the war.

After discussing this actual vs potential exposure problem, 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.”

ICNIRP and the authors are wrong to conclude that this study shows no increases in cancer from radar exposure.

This study shows that exposure to radar (pulsed microwaves) results, several years later, in large, severe and highly significant health problems and death across all surveyed organs, including neurological, respiratory, endocrine, circulatory and cardiac, and cancer morbidity and mortality. Dose response relationships show strong relationships between radar exposure, mortality and morbidity.

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

13.3.1 The context:

The Soviets irradiated the U.S. Embassy in Moscow for over 20 years between 1953 and 1976 using radars. Measurements taken on the outside walls on the upper floors at which the radar was aimed showed peak exposure values of 5(W/cm2 between 1953 and May 1975, 15(W/cm2 between June 1975 and Feb 1976. After this it was a fraction of 1(W/cm2. Exposure lasted for 9hr/day in the first period and 18 hr/day subsequently. Hence for over 20 years the daily average outside exposure was 1.9(W/cm2. Inside the exposure was in the range of 10 to 50 times lower, i.e. 0.038 to 0.19(W/cm2.

Pollack (1979) confirms that the employees and dependents were exposed to extremely low intensity radar signals:

"The maximum intensity up to May 28, 1975, was 5 (W/cm2. To achieve a 5 microwatt exposure the individual has to stand in from of an open window fully undressed for a full part of the working day. The characteristics of microwaves are such that once away from the open window, inside the room, a variety of field intensities fluctuate depending upon the wall, the furniture, the presence of steel cabinets, and so forth. A few feet from the window, the intensity was down to fractions of a microwatt most of the time with occasional points there where one could measure one or two microwatts."

The employees and dependents were studied for possible health effects from the radar exposure by a team from the John Hopkins University under the direction of highly respected epidemiologist, Professor Abraham Lilienfeld. Dr Lilienfeld noted that the group was quite small and the follow-up time too short to generally identify significant health effects such as cancer. He thus recommended that continued health status surveillance should be carried out. This was not done. The incidence of sickness and death were compare with the average US rates for similar age groups for both the Moscow Embassy and other Eastern European Embassies.

There was great pressure for the group not to identify adverse health effects. Dr Herbert Pollack, the U.S. State Department Contract Officer is recorded to have changed the conclusions of the report, Goldsmith (1995a, 1997). In a published conference paper Dr Pollack criticized the media and a particular journalist, Paul Brodeur for their claims that there were blood tests showing effects, Pollack (1979a). Pollack states: "He (Brodeur) goes on to day that types of chromosome aberrations observed in this study are the same as those induced by ionizing radiation in other organisms, including humans. Obviously, no such data were available." This final claim is untrue. The chromosome aberrations were recorded and reported by Tonascia and Tonascia (1976) and their results are published in Goldsmith (1997), Table 13 below.

13.3.2 The key results included:

The all cause mortality rate for Moscow males as 0.42 (0.3-0.6) and for females 1.1 (0.5-1.9). Hence males, primarily State Department employees, were much healthier and females were as healthy as the average U.S. residents. This is a good example of the "healthy worker" effect. State Department selection procedures rule out a range of unhealthy people and favour healthy people.

Table 10: Sickness rates increased in Moscow with years of service: (Table 6.18)

	Under 2 yrs	2-3 years		4 + years	p-value for trend
Number of people		316	455	45	
Person-years	3709	5570	568		
Male Conditions (%)					
Present Health Summary		5.4	9.7	16.2	0.05
Arthritis/rheumatism	4.3	6.5	8.8	0.02	
Back Pain	4.0	7.7	11.8	0.04	
Ear problems	3.8	5.6	14.7	0.02	
Vascular system		0.8	2.7	11.8	0.004
Skin & Lymphatic		9.4	12.2	28.0	0.02
Female Conditions (%)					
Vaginal discharge		4.2	13.8	17.5	0.04

The sickness rates increased independent of the age of arrival and faster than the influence of aging.

Table 11: Neurological Symptoms per 1000 p-y, Male employees: (Table 6.31)

	Moscow	Comparison		RR	p-value
Depression	1.3	0.73	1.78	0.004	
Migraine	1.8	0.97	1.86		
Lassitude	1.2	0.78	1.54		
Irritability	1.3	0.66	1.97	0.009	
Nervous Disorders		1.5	0.64	2.34	
Difficulty in Concentrating			1.4	0.52	2.96	0.001
Memory Loss	1.6	0.50	3.20	0.008	
Dizziness	1.2	0.85	1.41		
Finger Tremor	1.3	0.71	1.83		
Insomnia	1.1	0.90	1.22		
Neurosis	1.3	0.76	1.71		

These symptoms are consistent with the "Microwave Syndrome" of the "Radiofrequency Radiation Sickness", Johnson-Liakouris (1998). Mild et al. (1998) identified significant dose-response relationships for the following symptoms from the use of mobile phones: Memory Loss, Difficulty in Concentrating, Headache, Fatigue. Hence it is now shown and known that RF/MW exposure from extremely low but chronic exposure over many years, occupational exposure and cell phone use all produces significant and consistent neurological symptoms. The Risk Ratios were quite large but they were not quite significant because of the very small sample numbers.

Table 12: Congenital Malformations of children after the first tour:

Conditions	Moscow	Comparison	RR	Number of children
	SMBR	SMBR		

Leukaemia and cancer	1.2	0.84	1.43	1
Blood Disorders	1.7	0.42	4.05	7
Mental, Nervous Condn.	1.8	0.36	5.0	8
Behavioural Problems	1.4	0.68	2.06	7
Chronic Disease	1.1	0.88	1.25	7

Table 13: Blood samples showed a high proportion of the staff had significantly altered red and white blood cell counts and well above average chromosome aberrations (CA). The CA data is set out in Goldsmith (1997), i.e.

Mutagenic Level		Designator	Subjects, No.
5	Extreme	0	
4	Severe	6	
3.5	Intermediate	5	
3	Moderate	7	
2.5	Intermediate	5	
2	Questionable	5	
1	Normal	6	

Patients with mutagenic level of 3 and above were advised not to reproduce until 6 months after somatic levels had returned to 2 or 1. This warning applied to 68 % of the patients in this sample. Staff with elevated chromosome aberration rates were advised not to have children for until six months after they had returned to near normal.

A survey of cancer mortality rates is summarized in Table 14. This shows that despite the extremely small sample size and the very significant exposure dilution in the years between residence in Moscow and the survey results, there are highly elevated and significantly elevated rates of mortality from cancer Lilienfeld et al. shows significantly increases chromosome aberration and cancer. This was recently also found in mice, Vijayalaxmi et al. (1997).

The dominant cancers are brain tumor and leukaemia and reproductive organ cancer. But this study, like the Korean War Study, confirms that extremely low level chronic microwave exposure is associated which very significant increases in illness and mortality in organs across the whole body, consistent with widespread cellular chromosome damage. Significantly elevated chromosome aberrations were measured in this case, Table 13, as well as significant alterations in white and red blood cell counts, Jacobson (1969). This would also be the expected result from reduced melatonin.

Table 14: Cancer Mortality Rates:

Male employees (Table 6.37)	Moscow SMBR	Comparison SMBR	RR
Skin Cancer	1.5	0.69	2.17
Benign Neoplasms	1.4	0.75	1.87

Female employees (Table 6.38)

Malignant Neoplasm (Excl. skin)	1.7	0.63	2.86 (p=0.06)

Adult Dependents: (Tables 7.12, 7.13)

	Obs.	Exp	SMR	(95%CI)
Live-in				

All malignant Neoplasms				5	1.5	3.3	(1.1-7.7)
Digestive Organs Cancer				1	0.26	3.8	(0.1-21.2)
Pancreas Cancer	1	0.03	33.3	(0.8-185)		
Breast Cancer 1	0.4	2.5	(0.1-13.9)			
Ovarian Cancer			3.0			
Multiple Myeloma			1.5			
Arteriolosclerotic Heart Disease	2	0.59	3.4	(0.4-12.3)		
Live-out						
All malignant Neoplasms				7	3	2.3	(0.9-4.7)
Brain tumor	2	0.1	20.0	(2.4-72.2)		
Lung cancer	1	0.44	2.3	(0.4-93)		
All Accidents	4	1	4.0	(1.1-10.2)		

Table 14: cont'd:

	Obs.	Exp	SMR	(95%CI)
Suicide	1	0.36	2.8	0.1-15.6)
Children Living In (Table 7.16)				
All Malignant Neoplasms			2	0.5	3.8	(0.5-13.7)
Leukaemia	1	0.2	5.3	(0.1-29.5)		
Suicide	1	0.29	3.4	(0.0-1.6)		
Children Living out						
All Malignant Neoplasms			2	0.83	2.4	(0.3-8.7)
Leukaemia	1	0.3	3.4	(0.1-18.9)		
Suicide	1	0.3	3.3	(0.1-18.4)		

13.3.3 Report conclusions challenged:

It is stated by both Bradford Hill (1965) and Goldsmith (1992) that elevated Odds and Risk Ratios are also relevant to the public health protection basis in epidemiology,

Professor Goldsmith was closely associated with the staff affected by the chronic radar exposure of the U.S. Embassy in Moscow and obtained information through the Official Information Act. This included the blood test results and minutes of meetings which record the fact that the State Department case officer, Dr Herbert Pollack, changed the conclusions of the final report compared with the draft report, to state that no effects could be associated with the radar exposure, Goldsmith (1997). The data and Dr Goldsmith show that this is not true. After reviewing this data, an eminent epidemiologist, Professor John Goldsmith, Goldsmith (1995), referring to a "recent draft of criteria for health protection" which claims: "No effect on life span or cause of death of 1,800 employees and 3000 dependents of the U.S. Embassy personnel", states:

"To ignore these findings on the basis of "No effect on life span or cause of death" in setting human exposure standards is wrong. In the first place the criteria are two narrow; mortality is not the only

relevant end-point. The positive or 'findings for concern' are ignored. Increased cancer incidence among dependents is a nontrivial endpoint."

A highly remarkable result is the dose-response relationship for a range of sicknesses, Table 10. The results must be very highly significant to survive the exposure dilution effect with the disease gradient intact and statistically significant. As with Robinette et al. (1980), the data presented in the Lilienfeld contract report is contrary to that stated in the report's stated (an altered) conclusions. Despite the small numbers, the lack of long latency period and dilutionary factors, the Lilienfeld data shows a significant increases in:

Cardiac symptoms
Neurological and psychological symptoms
Altered blood cell counts
Increased chromosome aberrations, and
Elevated cancer in children and adults
Sickness increasing in a dose-response manner with years of residence.

These symptoms are associated with chronic exposure to very low intensity pulsed microwaves in the range < 0.04 to 0.2(W/cm2.

In a sense too, the fact that the State Department case officer, Dr Herbert Pollack, altered the conclusions, attests to the significance of this study, the results of which would be embarrassing to the U.S. Government, both in terms of compensation and in terms of the validity of the U.S. exposure standard.

13.4 Selvin et al. (1992): "Distance and risk measurements for the analysis of spatial data: a study of childhood cancer" - The Sutra Tower Study, San Francisco.

Background:

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

Broadcast tower residential exposure patterns:

Selvin et al. (1992) made a major error by assuming that the public exposure varies linearly with distance from the tower. Their conclusions were firmly based on this assumption and therefore are wrong. Radio engineers know a great deal about broadcast antennae radiation patterns. Some typical VHF examples are given in section 1.9 and Figures 4 to 6.

Figure 30: Ground level exposure for a typical UHF TV broadcast signal, from an antenna pattern from Hammett and Edison (1997), for a 2.4 MW ERP transmitter at 400m AGL, for a flat surface.

The ground level radial pattern shows a complex of undulating patterns whose peaks and troughs vary with the wavelength of the signals and the height of the antennae. The transmissions from the Sutra Tower have weak VHF and powerful UHF signals. Figure 30 shows a typical UHF signal taken from an antenna vertical pattern in Hammett and Edison (1997). Figure 30 shows that the strongest peaks for UHF signals occur between 2 and 4 km, and the main beam peaks outside 10 km, around 11 to 15 km from the base

of the tower. Measurements taken around the Sutra Tower are presented in Figure 31, showing the mix of VHF and UHF broadcast stations.

The high peak close to the tower and the peak near 1 km are from the VHF (FM radio) transmissions, as shown in Figure 5. The peaks outside 2 km are primarily UHF signals, as shown in Figure 30.

Residential Exposure Factor:

The direct exposure measurements or calculations need to be adjusted for epidemiological purposes because people largely live inside and move around a great deal. The mean Personal exposure Factor has (PEF) has been estimated as 0.15, Section 1.10. For example, the measured outside signal at the five homes of the children who live within 1 km of the tower and who have brain tumour, averages 1.74(W/cm2. When the PEF of 0.15 is applied this becomes 0.26(W/cm2.

Figure 31: The measured and estimated power density (exposure in (W/cm2) with distance from the Sutra Tower. Circles show measurements. The line follows measurement points and the radial pattern of Figure 30 beyond 3 km. From Hammett and Edison (1997) and readings taken by the author in 1999.

The objective of Selvin et al.:

Selvin et al. were concerned with developing statistical data analysis techniques involved in comparing spatial clustering with risk approach to data analysis of potential effects from point sources of exposure. They apply their methods to the white, childhood cancer data for children <21 years living in the vicinity of the Sutra Tower to test the presence of clustering. An example, of the spatial distribution for childhood leukaemia, is given in Figure 30.

13.4.5 The results and errors in Selvin et al.:

Selvin et al. were totally unaware of the reality of radiation patterns and simply assumed that exposure varied linearly with radial distance. This was used to test three method of statistical clustering in an attempt to define the exposed vs unexposed populations. These approaches showed that peak cancer rates occurred at a radius of 1.75 km from the tower. From their methods this defines the exposed group to be within twice this distance, i.e. within 3.5 km. Because they assumed a linear decline in exposure with distance from the tower they conclude: “None of the three analytic approaches indicates the presence of clustering of childhood cancers associated with the Sutra Tower.” If they knew the actual radial radiation pattern then their conclusion would have been very different.

Figure 32: Spatial map of white childhood (<21 years) leukaemia for San Francisco, 1973-88, from Selvin et al. (1992).

Figure 33: All cancer for children (<21 years) from 1973-88, from Selvin et al. (1992), involving 123 cases; brain tumor (35), leukaemia (51) and Lymphoma (37). The non-residential areas of Golden Gate Park, the Central Business District (CDB) and Lake Merced are shown, along with the 1 km ring around the Sutra Tower.

Detailed spatial cancer incidence and exposure analysis:

Childhood cancer rates and residential locations are given for the period 1973-1988 by Selvin et al. (1992). A total of 123 cases of childhood cancer were identified among 50,686 white individuals at risk

under the age of 21 years. These included 51 cases of leukaemia, 35 cases of brain tumour and 37 cases of lymphatic cancer. Selvin et al. estimate that these categories of cancer cover close to 50 % of all cancers. Each childhood leukaemia case is given a residential location on a spatial map, Figure 30, the four cancers reported are plotted in Figure 31.

It is immediately evident in Figure 32 that there are higher childhood leukaemia rates in the eastern sector compared to the western, northern and southern sectors. Antenna radiation patterns and model calculations for all the antennae on the Sutra Tower, are given by Hammett and Edison (1997). These show that readings and model calculations give highest radiation intensities in the eastern sector. The broadcasters aim their signals at the greatest population in the city and across the Bay in Oakland and Berkeley. This is the first indication of a dose-response relationship.

Figures 32 and 33 also reveal the lack of cancer and residence in Golden Gate Park, the broad low density housing area of the Army Base, the Presidio to the NW, a large park area and hills to shade suburbs to the SW, the Central business district to the ENE the Lake Merced recreational area, and the port and industrial area along the eastern coastline. These were all taken into account when the residential population density was calculated below. In figure 32 the cluster 48-51 are residences on a western facing hill slope. They have higher exposure levels from the Tower than the radial distance implies. They contribute to the higher cancer rate in the 6-8 km ring compared with the 5-6 km ring and explains some of the scatter about the dose response line.

Table 15: Radial rings, with estimated population, Risk Ratios and Cumulative Risk Ratios, for white childhood brain tumour, Leukaemia, Leukaemia + Lymphoma, and All Cancer, in association with RF/MW exposure from the Sutra Tower, San Francisco.

Distance (km)	<0.99	1-1.99	2-2.49	2.5-2.99	3-3.49	3.5-3.99	4-4.49	4.5-4.99	5-5.99	6-8
Est. Population		1138	4334	3558	4489	5146	5566	4939	5386	8141	7988
Estimated personal mean dose in (W/cm2.	0.50	0.09	0.15	0.09	0.12	0.11	0.06	0.045	0.03	0.014	
Symptom											
Brain Tumour	11.81	2.48	3.02	1.80	2.09	1.93	1.63	1.00	0.99	1.01	
Cumulative	11.81	4.42	3.87	3.18	2.88	2.66	2.49	2.26	2.02	1.86	
Leukaemia	1.26	1.32	2.02	1.92	1.67	1.80	2.03	1.33	0.53	1.26	
Cumulative	1.26	1.31	1.59	1.70	1.69	1.72	1.77	1.70	1.48	1.44	
Leuk + Lymph	2.47	1.08	2.63	2.08	2.54	1.85	2.27	1.56	0.57	1.05	
Cumulative	2.47	1.37	1.86	1.94	2.10	2.05	2.08	2.00	1.73	1.62	
“All Cancer”	4.88	1.44	2.73	2.01	2.43	1.87	2.35	2.11	0.68	1.04	
Cumulative	4.88	2.16	2.38	2.26	2.31	2.43	2.21	2.19	1.80	1.68	

In order to calculate radial ring cancer rates a detailed map of San Francisco was used to remove areas of ocean, park, hill shading, lakes, port and central business district as outlined above, to make an estimate of the mean residential population density in each ring. These factors are recorded in Table 15, which also shows the Risk Ratios for each childhood cancer group.

The mean radial exposure regime, for this analysis, was assumed to be isotropic and given by Figure 31. Direct exposures were reduced by a factor of 0.15 to allow for mean residential exposure. These

estimates are given in Table 15. Thus the radial childhood cancer rates can be compared with a much more realistic radial radiation exposure pattern. The resulting estimates are summarized in Table 15.

Figures 34 and 35 show extremely significant dose-response relationships ($p<0.0001$) for Childhood brain tumor and All Cancer. The trend line passed through zero within the uncertainty of the data.

The dose-response trend analysis uses a least squares fit. The Mantel-Haenszel estimate of t with a two-tailed t-test was used for the significance test. For All Cancer $t = 14.05$ and for Brain Tumour $t = 13.70$. For leukaemia ($t = 3.31$, $p<0.01$), Leukaemia and Lymphoma combined ($t = 3.81$, $p<0.005$), Non-Hodgkin's Lymphoma ($t = 1.94$, $p<0.05$) and Hodgkin's Lymphoma ($t = 7.26$, $p<0.001$).

Contrary to the conclusion of Selvin et al. and ICNIRP (1998), who claim that this study shows no evidence of adverse effects, the spatial data when related to actual radial radiation exposure patterns forms significant linear dose-response relationships, with All Cancer and Brain Tumour having extremely significant dose-response relationships.

Figure 34: Brain Tumour Risk Ratio as a function of estimated radial group mean personal exposure to RF/MW radiation from the Sutra Tower, San Francisco, using the spatial childhood cancer data presented in Selvin et al. (1992). The linear dose-response relationship is extremely significant ($p<0.0001$).

Figure 35: All Cancer Risk Ratio as a function of estimated radial group mean personal exposure to RF/MW radiation from the Sutra Tower, San Francisco, using the spatial childhood cancer data presented in Selvin et al. (1992). The dose-response relationship is extremely significant ($p<0.0001$).

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

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

ICNIRP (1998) claims that this study showed no significant increases in nervous system tumours. This is factually wrong. The overall results of Beall et al. (1996), as presented in their abstract is: There was elevated ORs:

“For 10 or more years of employment in engineering/ technical jobs (OR = 1.7, 95% CI: 1.0-3.0) or in programming jobs (OR = 2.8, 95% CI: 1.1-7.0). The OR for glioma for all subjects who had accrued 5 years of programming work 10 years before the case’s death was 3.9 (95% CI: 1.2-12.4).”

These are statistically significant relationships. The subjects were chosen and studied because of the possibility and concern that using VDTs (Visual Display Terminals, i.e. computers) a great deal in their occupations. VDTs expose workers to a wide range of EMR for long periods, could be related to the increase in brain tumours. The researchers found differences between different occupations who use VDTs in different ways.

For example, those in manufacturing of VDTs they found OR = 0.8, while those in manufacturing VDTs who also used them for programming, OR =1.5 (95%CI: 0.8-2.7) and those in manufacturing VDTs who used them for information, OR = 1.3 (95%CI: 0.4-4.1).

Odd ratios for brain tumours increased with the longer times in jobs using VDTs. After 10 years the engineering/technical jobs had an OR = 1.7 (95%CI: 1.0-3.0) and programming, OR = 2.8 (95% CI: 1.1-7.0). These show dose response relationships, Figure 36.

Figure 36: Dose-response relationships for brain tumor mortality from Table 3 in Beall et al. (1996). These show linear dose-response relationships with years of using computers with the complex EMR exposure from the VDT.

This study shows that the particular groups which use live computers regularly have elevated Odds Ratios (increased levels of risk of brain tumour), and significant increases after 10 years of service. For computer programmers it is significant, p=0.04. The overall analysis, Figure 37, comparing gliomas and all brain tumours, men, women and total groups all show dose-response relationships but the relationship is not assessed as statistically significant:

Figure 37: Dose-response relationships for all brain tumors and Glioma for years of using VDTs.

The sample of men is somewhat larger than that for women. Men show increasing risk of all brain tumours and gliomas with the increasing work time with VDTs but women only show an increase in the 10-19 year group. Only 4 women are in the (20yr-group. There is a good evident reason for this. Women's employment is not usually as long as men in these jobs.

Exposures to EMR from VDTs has decreased over the decades with the introduction of low radiation" monitors. Measured RF/MW exposures at the head level of a computer user, 0.5 m from the screen, have been measured at 0.1 to 5(W/cm2. Using a mean lifetime exposure factor of 0.25 , based on 0.3 for the time at/away from work and 0.8 for the time programmers are at/away from the computer of 0.8, gives an estimated average lifetime exposure in the range 0.025 to 1.25 (W/cm2. The range is of the same order of mean lifetime residential exposure for the children in San Francisco who had a very significant increase in brain tumour and other cancers with a dose-response relationship.

Beall et al. (1996) does show statistically significantly increases of brain tumours for those using VDTs in their work for more than a 2 decades. Several relationships also showed dose response increases with brain tumours with longer periods of employment using VDTs, though the small sample sizes limit the statistical significance, these are indicative of probable relationship. The study is misrepresented by the ICNIRP reviewers as a study that shows no effects.

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

The ICNIRP claim:

The ICNIRP (1998) paper claims that this paper "failed to show significant increases in nervous tumors". Grayson actually shows the opposite conclusion.

13.6.2 The Context of this Study:

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

have been identified by the present author. Grayson cites Thomas et al. (1987) who found a significant dose-response relationship for Astrocytoma, the most common form of brain tumour, and years of service in the electronics industry, with a co-carcinogenic relationship with lead from solder fumes. The RF/MW exposure had the greater effect.

A study published earlier in 1996, Grayson and Lyons (1996) investigated the incidence of cancer in United States Air Force Aircrew. Aircrew are moderately exposed to ELF and RF/MW during their flight times and on bases. Grayson and Lyons found that Aircrew had significantly higher cancer rates than other USAF officers, RR = 1.31 (95%CI: 1.11-1.54, n=342). For brain tumor the incidence was elevated, RR = 1.20 (95%CI: 0.52-2.78, n=13), but not significantly so, largely because of the small case sample size (n). For other cancers, cancer of the testes and urinary bladder were significantly elevated.

Grayson notes that EMF (ELF) studies generally found negative results or suggest a small excess risk. Sahl, Kelsh and Greenland (1993), Tynes, Jynge and Vistnes (1994) found no increase in brain tumor in electric utility or railway workers. Theriault et al. (1994) found an elevated risk (OR = 1.95, 95%CI: 0.76-5.00) and Floderous et al. (1993) a significantly increased risk (OR= 1.5, 95%CI: 1.0-2.2) in electrical workers.

Mack, Preston-Martin And Peters (1991), Speers et al. (1988) and Loomis and Savitz (1990) found highly significant increases; and Lin et al. (1985), Thomas et al. (1987), Preston Martin, Mack and Henderson (1989) found significant dose-response relationships for increased brain tumors in EMR exposed populations. Using the EMR Spectrum Principle, this amounts to very strong evidence which is indicative of a causal relationship.

13.6.3 Grayson's results:

Grayson carried out a job title-time-exposure matrix utilising potential intensity scores for both ELF and RF/MW EMR exposures. Data on ionizing radiation exposure was also available.

“Although the present study has its limitations, particularly in exposure estimation, it does suggest that there is a small association between potential EMF exposures and brain tumor risk among Air Force members, especially for personnel potentially exposed to Radiofrequency/microwave EMFs.”

The results for the three types of radiation exposure, after adjustment for: Age-race-senior military rank, were:

Ionizing Radiation OR = 0.58 95%CI: 0.22-1.52
ELF Radiation OR = 1.28 95%CI: 0.95-1.74
RF/MW radiation OR = 1.39 95%CI: 1.01-1.90

The relationship for age-race adjusted Odds Ratios for rank were:

Rank OR = 2.11 95%CI: 1.48-3.01
Senior Rank OR = 3.30 95%CI: 1.99-5.45

The ELF and RF/MW exposure results are consistent with the second part of the EMR Spectrum Principle, that there are likely to be stronger effects at higher frequencies. These results are consistent with and confirming of the studies cited above, section 13.6.2.

The rank-related results are independent of the exposure-related results. They raise the question of the influence of socio-economic status, which is accurately represented by military rank. Preston Martin

(1989) and Preston-Martin et al. (1993) also find that brain tumour risk increases with socio-economic status.

Grayson (1996) is far from a “no effects” study. Thus far consistently the ICNIRP claims are scientifically wrong and misleading. This study does show a small but statistically significant increase in brain tumour from RF/MW exposure.

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

The Context:

This and the next paper, Rothman et al. (1996 a and b), are used to imply that there is "no excess total mortality was apparent among uses of mobile phones". This is the ICNIRP context of citing papers, as the six above are quoted, claiming that they show no increases or no significant increases in brain tumour or cancer. This paper, Rothman et al. (1996a), is the description of an epidemiological study in progress about the potential association of mobile phone use and brain tumour.

An important feature of radiation is the contrast between ionizing and non-ionizing radiation. The threshold for the ability of a photon to strip and electron off an atom, forming an ion, is a photon energy or 12.4 eV or a wavelength of 100 nm, Hitchcock and Patterson (1995). By this criteria, ultraviolet radiation, with a wavelength range of 200 to 400 nm is not ionizing radiation. However, the spectrum in Rothman et al. (1996a) shows UV radiation as part of the ionizing radiation segment. This may be because UV radiation is shown to produce skin cancer and cause immunosuppression in people, Gochfeld et al. (1995). With the assumption of many people that cancer can only be caused by ionizing radiation and not by non-ionizing radiation, then rather than accepting that non-ionizing UV radiation does cause cancer, they prefer to change the definition.

The Conclusion:

ICNIRP's follow up statement is actually true: "but it is still too early to observe an effect on cancer incidence or mortality". The latencies of brain tumours is decades. This is not an appropriate paper to cite in a cancer assessment because it has no evidence value for or against the incidence of cancer in RF/MW exposure.

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

This study was prompted by case reports of brain cancer among cellular telephone users. To evaluate the possible effect of using cellular telephones on the risk of death they compared the rates of mortality between mobile phone users and portable phone users. For the cancer latency reasons above it is a very preliminary report. The authors state:

“First, they do not directly address the issue of the relationship between cellular telephone use and brain cancer, which comprises only a small proportion of deaths. Second, the time between exposure to radio frequency energy from portable cellular telephones and the death endpoints that we measured was comparatively short, and our study therefore addresses only short-term effects.”

Both mobile and portable phone users have lower mortality rates than the general population. However, there are differences between these two groups. This paper is a prime example of how to obscure significant results. They conclude that for users with phone accounts at least 3 years old, portable phones produce a lower mortality risk than mobile phones, MR = 0.86 (90% CI: 0.47-1.53). This is deliberately avoiding the alternative true statement that mobile phone users have a higher mortality rate than portable

phone users. They also use a 90% confidence interval rather than the normal 95%. Their data, Table 1, shows for all ages, significantly higher death rates among cell phone users. The following is the result of a 2x2 analysis for all men, all women and all people:

	RR	95%CI	p-value
For men:	1.40	1.06-1.86	0.017
For women:	1.52	0.78-2.95	0.31
All people:	1.38	1.07-1.79	0.013

Since the portable phone users are exposed to RF/MW radiation they are not an unexposed group. Hence the difference is likely to be larger. A crude analysis of mortality with years of phone usage showed RR = 1.35 for 2 yrs and RR = 1.56 for 3 yrs. When adjusted for confounding, these reduced to 1.075 and 1.163 respectively. This shows that the adjusted comparative mobile phone mortality elevation more than doubles from 7.5% to 16.3 % with the extra year of usage.

Therefore, in the context of a cancer assessment, this paper does not identify the cause of death and therefore is not a cancer assessment. The authors hide the results by reversing the comparison. Overall the study does show that for a large (256,284) 1 year sample there is a significantly higher mortality rate among mobile phone users compared with portable phone users, especially among older people, and increases with years of usage.

13.9 Interim Conclusions (Papers 1 - 8.):

All of the first 8 papers or reports cited by ICNIRP with the clear intention of dismissing the possibility of cancer being related to RF/MW exposure. All are inappropriately or incorrectly cited. In some cases they are deliberately, misquoted and misused. In reality the reverse of what ICNIRP claims is true. Often the data challenges the conclusions.

Two of the papers, Barron and Baraff (1958) and Rothman et al. (1996a) are inappropriately included when they are not giving epidemiological results of cancer resulting from RF/MW exposure.

The remaining six reports or papers all show statistically significant increases in mortality from RF/MW exposure, including cancer of various body organs, especially brain tumour and leukaemia. Three of them also show significant dose-response relationships for a range of cancer, including respiratory cancer, Robinette et al.; Brain Tumour, Leukaemia, Hodgkins and Non-Hodgkin's Lymphoma and All Cancer, Selvin et al., and Brain Tumour, Beall et al.. Lilienfeld et al. (1978) shows significant dose response increases in a range of illnesses. Rothman et al. (1996b) shows significantly increased mortality from mobile phone usage, increasing in a dose response manner with years of usage.

Two of these, Lilienfeld et al. and Selvin et al., show results with extremely low mean residential RF/MW exposures, one moderate to low exposures from computer screens and two moderate to high exposures from military radio and radar radiation. The residential study shows that statistically significant increases in childhood cancer occur in a dose-response manner. For All Cancer, Selvin et al.(1992), the threshold is near to zero.

Studies acknowledged by ICNRIP to show increases in cancer from RF/MW exposure:

In this section there are five papers covering three studies. Szmigielski et al. (1988) and Szmigielski (1996) cover the Polish Military Study, Dolk et al. (1997a and b) cover the U.K. Regional TV Tower Study and Hocking et al. cover the North Sydney Broadcast Tower Study.

14.1 The Polish Military Study:

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

Szmigielski (1996): “Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation”.

ICNIRP Dismissal:

ICNIRP's complete, comprehensive and in-depth assessment of this project is fully quoted in the following two sentences:

“There has been a report of increased cancer among military personnel (Szmigielski et al. (1988)), but the results of the study are difficult to interpret because neither the size of the population nor the exposure levels are clearly stated. In a later study, Szmigielski (1996) found increased rates of leukaemia and lymphoma among military personnel exposed to EMF fields, but the assessment of EMR exposure was not well defined.”

This is a woefully inadequate and thoroughly unprofessional treatment of this large and significant epidemiological study. The ICNIRP response represents a total misunderstanding or misrepresentation of epidemiology and the results of this study.

Cancer Epidemiology:

Most cancer studies use data from cancer registers that cover several decades to allow for cancer latencies These registers involve the large populations are necessary for statistical quality and significance. Retrospective studies, such as cohort and case control studies rarely, have records of the hourly or daily mean exposure of every participant for the period of the register. Hence occupational activities involving exposure to a potential disease agent is the most common surrogate for exposure. Sometimes a job exposure matrix assessment is undertaken of a typical sample of tasks involved in a job. This improves the exposure assessment but it remains an estimate of potential or probable exposure. Because of these practical limitations epidemiology is based on careful selection of occupational groups in order to compare morbidity and mortality rates between exposed and non-exposed or low exposure control groups.

Hence many studies involve "electrical occupations", "power station workers", "electric train drivers", "electric utility workers", "computer programmers", "sewing machine operators", "TV repairmen", etc… Their cancer and illness rates are then compared with a set of controls who are selected because they have the same age-race-income-geographic- … characteristics to make them as similar as possible to the exposed people with the one exception of the exposure.

Polish Military Exposure Assessment:

Years of service become a reasonable estimate of cumulative exposure and thus a source of a dose-response gradient. For some occupations the EMR exposures are intermittent and associated with a particular activity. For example, physiotherapists using RF/MW heating for diathermy prior to muscular manipulation. Their exposures are generally for only a minute or two after the machine has been turned on. The monthly number of treatments is a good measure of the cumulative monthly dose or the monthly mean exposure.

Thus, most EMR epidemiological studies rely solely on occupational descriptions as a surrogate for exposure. Refinements include job exposure matrix surveys or reported exposure incidents. In this later case any exposure radiation from an active radio antenna or radar antenna is reported and recorded,

along with the estimated level of exposure and time of exposure. This is because strict daily limits are maintained based on cumulative dose. This is the EMR Hygiene reporting regime. It has been used in the Polish Military since about 1968. Hence the Polish Military Study uses one of the more advanced exposure assessment regimes of any study published. Szmigielski (1996) states:

"Data on exposure of personnel to RF/MW were collected from EM military safety groups operating as health hygienic services. These groups are responsible for measurements of RF/MW field intensities at and around service posts where EM emitting equipment is used, repaired or serviced, and keep health records of personnel working on these posts. The number of personnel considered to have been exposed occupationally to RF/MW was easily established, but the evaluation of the exposure rate appeared to be quite difficult."

Thus to be considered as a member of the exposed group, exposure episodes were required to be recorded. All other personnel are used as controls. This is one of the most advanced exposed group selection criteria ever used.

Population Description:

The data set used by Szmigielski et al. was 1971-80, and Szmigielski (1996) updated this to use 1971-85. Szmigielski et al. state:

“The total population of career servicemen (in the Polish armed forces) was analyzed, and a subgroup of personnel exposed occupationally to MW/RF radiation (on the basis of service records) was developed; the E (exposed) group counted about 3 % of the total population, the rest (97%) was considered as subjects without exposure to MW/RFs (the NE group).”

Szmigielski (1996) explained that over the 15 years there is a slight year to year variation in the population but it averages 128,000 person each year with 3700 being RF/MW exposed. The data set is somewhat larger than that used by Robinette et al. (1980) and exposure dichotomization is significantly better. Over the 15-year data set there are 1.92 million p-yrs in the control group and 55,500 p-yrs in the RF/MW exposed group.

Thus the Polish Military study is a very large study with a well defined population with a high quality criteria for identifying the exposed vs control groups. ICNIRP is wrong in its criticism and wrong to dismiss this highly significant study.

14.1.5 The Results of Szmigielski et al. (1988)

14.1.5.1 Health Effects Assessment:

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

14.1.5.2 Cell line (In Vitro) studies:

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

aware that both calcium ion efflux and influx occur at different combinations of RF/MW signal impacting on the cell membrane. This is consistent with immunosuppression and stimulation respectively.

14.1.5.3 Whole animal (In Vivo) Studies:

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

However, Szmigielski et al. appear to be unaware of Shandala et al. (1983) which did find a highly significant (78%) and persistent suppression of the immune system rats when exposed to 500 (W/cm2 for 3 months.

14.1.5.4 Integrated evaluation of immunity in MW/RF exposed animals:

Szmigielski et al. outline their own experiments in this area. They conclude:

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

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

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

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

Szmigielski et al. planted cancer cell in the lungs and on the skin of mice and chronically exposed them to non-thermal intensities of 2.45 GHz microwaves. The tumors grew faster and the mice died earlier in the exposed compared to the sham exposed mice. The MW exposed mice with induced skin cancer showed 50 % died after 137 days, compared to 305 days for the sham exposed mice, Figure 38.

Figure 38: Growth curves of 3,4-benzo-alpha-pyrene (BP) induced skin tumour in mice exposed daily (2 hours) to 2450 MHz radiation at 10 mW/cm2 (SAR 4 W/kg) for a whole period of tumour growth. CDT50, cancer development time for 50% of the animals.

The lung tumors which all started at near 2 x 105 viable cells. After 3 months, the control group stayed close to 2 (x 105), while the exposed mice rose to 6 and 15 for 5 and 15 mW/cm2 respectively, Figure 39.

Figure 39: Number of lung tumours (following intravenous injection of 2 x 105, viable sarcoma cells) in mice exposed during 1, 2, 3 months to 2450 MHz microwaves (2 hours daily), at 5 or 15 mW/cm2. Oc mice were treated with nonspecific stress of overcrowding (confinement in cages with out exposure to microwaves, as positive controls. Control: sham exposed mice).

Figure 39 shows a dose response relationship for the growth of cancer nodules as a function of microwave exposure level.

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

They concluded:

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

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

14.2 Polish Military Study (1971-80):

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

They note that Robinette et al. (1980), the Korean War Study, reported no significant differences between high and low exposure groups, but point out:

“However, when three sub-groups of the high-exposure group were developed to provide a gradient of potential exposure, a trend appeared for increased number of malignant neoplasms in the sub-group rated as highly exposed.”

They also refer to weakness of the Korean War study in terms of its size and subject selection. They didn't investigate the Korean War Study data in depth as is done here. The preliminary results of the Polish Military Study, using the 1971-80 data set, is presented in Figure 40 and 41.

Figure 40: Cumulative yearly morbidity rate of neoplasms during 1971-80 (expressed as the number of new cases per 100,000 person years) for all ages (20-59 years) in MW/RF exposed subjects and non-exposed subjects.

The top histogram shows organ localization of malignancies for oral cavity; pharynx and larynx; esophagus and stomach; colo-rectal, liver and pancreas; lungs; bones; skin including melanoma; kidneys-urogential tract- prostate; eyes and central nervous system; thyroid gland and other endocrine glands; hematopoietic and lymphatic organs.

The high incidence of cancer of the hemato-lymphatic organs allows the break down given in the lower half of the diagram. LGR: malignant lymphogranulamatosis; LS, LM, lymphosarcomas and lymphomas; CLL, chronic lymphatic leukemia; ALL, acute lymphoblastic leukemia; CML, chronic myelocytic leukemia; AML, acute myeloblastic leukemia and PL, plasmocytoma (plasma cell leukemia).

This data shows that the microwave exposed group, compared to the low exposure group, had increased malignancies in every category of organ, significantly increased in esophagus and stomach, colo-rectum, skin cancer including melanoma and thyroid, and highly significant in blood and lymph organs. Individual leukemia's which were significant were Acute Myeloblastic leukaemia and highly significant were chronic myelocytic leukaemia and lymphosarcomas and lymphomas.

The decadal age category results are presented in Figure 41.

Figure 41: Cancer morbidity rates in RF/MW exposed and “non-exposed” personnel for all types of malignancies at various age groups.

Note the largest differences at the age group 40-49 years and statistical significance of differences for all age groups. The Chi Squared values indicate that the differences are much more significant than $p<0.01$, they are actually $p<0.001$. This analysis shows that RF/MW exposure initiates earlier cancer and accelerates it in the young and middle decades, 20-49. The difference is reduced a little in the oldest age group as the reference cancer rate accelerates rapidly in the 50+ age group.

14.3 Polish Military Study (1971-85):

Szmigielski (1996) is a follow-up study from the previous study, adding a further 5 years of morbidity data. With the larger data set the significance of the observed increases in cancer are increased. The data is summarised in three tables, in parallel with the summary diagrams of Szmigielski et al. (1988) above, showing morbidity of body organs, haemopoietic malignancies and age-grouped relationships.

As in the 1988 analysis, this data shows that RF/MW exposure increases cancer across the body with elevated Risk Ratios, and several organs show significantly and very significantly higher cancer rates, including Esophageal and stomach cancer, Colorectal cancer, Skin cancer, Brain and CNS cancer and all malignancies, Table 16.

Table 16: Incidence of neoplasms (per 100,000 subjects annually) in military personnel exposed and non-exposed (control) to radiofrequency and microwave radiation, Szmigielski (1996).

Localization of malignancies	Incidence (Expected)	Incidence (Exposed)	Risk Ratio	95% CI	p-value
Pharynx	1.96	2.12	1.08	0.82-1.24	N.S.
Esophageal and stomach	4.83	15.64	3.24	1.85-5.06	<0.01
Colorectal	3.96	12.65	3.19	1.54-6.18	<0.01
Liver, pancreas	2.43	3.58	1.47	0.76-3.02	N.S.
Laryngeal, lung	21.89	23.26	1.06	0.72-1.56	N.S.
Skin, including melanomas	3.28	5.46	1.67	0.92-4.13	<0.05

Nervous system including brain tumour			2.28	4.36	1.91	1.08-3.47	<0.05
Thyroid	1.38	2.12	1.54	0.82-2.59		N.S.	
Haematopoietic system and lymphatic organs			6.83	43.12	6.31	3.12-14.32	<0.001
All malignancies		57.60	119.12	2.07	1.12-3.58		<0.05

The Haematopoietic and Lymphatic cancers are very highly significantly elevated and so are separated further in Table 17.

Table 17: Incidence of haemopoietic and lymphatic malignancies (per 100,000 subjects annually) in military personnel exposed and non-exposed (control) to radiofrequency and microwave radiation.

Localization of malignancies	Incidence (Expected)	Incidence (Exposed)	Risk Ratio	95% CI	p-value
Hodgkin's disease	1.73	5.12	2.96	1.32 - 4.37	<0.05
Lymphoma (non-Hodgkin and lymphosarcoma	1.82	10.65	5.82	2.11 - 9.74	<0.001
Chronic lymphacytic leukaemia	1.37	5.04	3.68	1.45 - 5.18	<0.01
Acute lymphoblastic leukaemia	0.32	1.84	5.75	1.22 - 18.16	<0.05
Chronic myelocytic leukaemia	0.88	12.23	13.90	6.72 - 22.12	<0.001
Acute myeloblastic leukaemia	0.71	6.12	8.62	3.54 - 13.67	<0.001
Total	6.83	43.12	6.31	3.12 - 14.32	<0.001

In the 1988 data analysis, three sub-categories of leukaemia and lymphoma were significantly increased with RF/MW exposure. In this larger data set all are significantly increased and 4 are very highly significantly increased, Lymphoma, Chronic Myelocytic Leukaemia, Acute Myeloblastic Leukaemia and Total leukaemia/lymphoma.

The age-group relationships show the same initiation and advancement of the cancer rate in the exposed group. Table 18 shows that the Risk Ratio decreases with increasing age as the unexposed cancer rate increases due to normal aging processes. The Haemopoietic/lymphatic cancers are all highly significantly increased in every age group.

Table 18: 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).

All sites:

Age Group	Incidence (Expected)	Incidence (Exposed)	Risk Ratio	95% Conf.	p-value
20-29	11.62	21.11	2.33	1.23 - 3.12	<0.05
30-39	18.37	42.28	2.30	1.04 - 3.06	<0.05
40-49	84.29	161.62	1.92	0.98 - 2.84	<0.05
50-59	186.71	274.13	1.47	0.92 - 2.21	N.S.
All Ages	57.6	119.12	2.07	1.12 - 3.58	<0.05

Haemopoietic/lymphatic malignancies

20-29	2.12	17.30	8.16	3.11 - 22.64	<0.01
30-39	3.08	26.43	8.58	3.46 - 19.58	<0.01
40-49	8.32	73.25	8.80	4.13 - 15.27	<0.01

50-59	24.13	108.62	4.47	2.56 - 6.81	<0.01
All ages		6.83	43.12	6.31	3.12 - 14.13	<0.001

14.4 Polish Military Preliminary Prospective Study Results:

Szmigielski (1998) is a prospective study on exposed Polish Military personnel between 1986 and 1990. He concludes that the data suggests that cancers “develop faster, with a shorter latency period” in servicemen with occupational RF/MW exposures. He also found a dose-response relationship with cancer rate against maximum microwave exposure. Individual exposure monitoring places 92.8% of the exposed men in situations where peak exposures were less than 1000(W/cm2, and 83.7 % below 600(W/cm2. This data also includes the all cancer risk ratio for these groups of people.

Table 19: Cancer rates as a function of typical peak exposures in a prospective study extending the Polish Military study, Szmigielski (1998).

Number of Men		Peak Exposure Range (W/cm2	Cancer Rate Ratio
1900	(49.4%)	100-200	1.69
1320	(34.3%)	200-600	1.57
350	(9.1%)	600-1000	4.62
280	(7.2%)	>1000	4.93

14.5 Simplified peak and mean exposure regime:

Based on the Polish Military study measurements the following simplified exposure regime has been proposed. The simplified regime could consist of life-time means being half of annual means while working, annual mean is 20% of the weekly working mean, the weekly peak is 10 times the weekly mean and the monthly peak is 10 times the weekly peak. For example:

Table 20: Estimated life-time, annual, weekly mean and weekly and monthly peak exposure relationships.

Exposure Category	Exposure ((W/cm2) Life-time Mean			Annual Mean	Weekly Mean	Weekly Peak	Monthly Peak
High	10	20	100	1,000	10,000		
Medium		5	10	50	500	5,000	
Low	2	4	20	200	2,000		

14.6 Conclusions:

The three published papers in the Polish Military cancer morbidity study shows that RF/MW is associated with increased cancer in many major organs of the body, with the highest risks occurring for Leukaemia and Lymphoma. A dose-response relationship has been found for the latest study which is a prospective study following a large number of exposed servicemen and monitoring their peak exposures associated with their military work.

The significance of these studies cannot be dismissed because of exposure uncertainties. There is a very good separation of exposed and low exposure populations through one of the world's most advanced personnel exposure monitoring systems. These are also one of the largest of any study thus far. Hence the ICNIRP criticism of these studies is completely unfounded.

These studies, when taken together with the other studies presented here, show a causal relationship between exposure to RF/MW and sickness and death due to cancer increases and very low, mean life-mean exposure levels. All peak exposures are non-thermal (Szmigielski pers. comm.).

15. Residential Studies:

15.1 Introduction

We have already seen that the residential study in San Francisco showed-dose response related childhood cancer death, brain tumor and leukaemia for residential exposure to low intensity microwaves from a TV/FM tower on Mt Sutra. Szmigielski shows that the highest effect of RF/MW exposure of military personnel is Leukaemia and Lymphoma.

ICNIRP cites two residential studies contained in three papers, Hocking et al. (1996) and Dolk et al. (1997a. and b.). They are described by ICNIRP as “have suggested a local increase in leukaemia incidence”, “but the results are inconclusive.”

ICNIRP consistently uses very simple statements to dismiss any adverse effects. Every time a careful consideration of principles, methods, application of epidemiological approaches and consideration of the actual data and exposure regimes, produces a significantly different conclusion. And when sets of studies are considered together, very strong conclusions are drawn. These studies are no exception.

15.2 Hocking, Gordon, Grain and Hatfield (1996): “Cancer incidence and mortality and proximity to TV towers.”

The Study Context:

This study was carried out to allay public fears about siting cell sites in residential properties in Australia, Hocking (pers. Comm.). The authors correctly recognized that mobile phone base stations (cell sites) have not been exposing people long enough to produce cancer because of the cancer latency periods are long. Because of the then dominance of analogue cell phones using FM radiation they decided to study the residents exposed to FM signals from FM radio and TV stations around three tall towers in North Sydney. When the study was commenced Dr Hocking was the Medical Director of the Telstra Research Laboratory. At the time of publication Dr Hocking had become an independent public health consultant and the paper was published with the support of his professional colleagues.

15.2.2 The population Sample:

The cancer data covered 9 municipalities in the north side of Sydney Harbour for the period 1972 to 1990, Figure 42.

Figure 42: 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).

The exposed population was chosen to the three municipalities that surrounded three large TV towers, Lane Cove, Willoughby and North Sydney. This gives an "exposed" population of 135,000. The control group came from six surrounding municipalities, Ryde, Ku-ring-gai, Warringah, Manly, Mosman and Hunters Hill, population 450,000, Figure 40.

The cancer incidence (mortality) cases were adjusted for sex, age and calendar period and resulted in 1206 (847) leukaemia cases for the total population and 134 (59) for children 0-14 years. For brain tumour the sample was 740 (606) for the total population and 64 (30) for children.

15.2.3 Exposure situation:

There are three towers. The frequencies involved are in the range 63 - 219 MHz and 626-633 MHz. Tower 1 has the highest TV/FM output power, 500 kW, while Tower 2 has 180 kW and Tower 3, 110 kW. The exposure of each municipality is also a function of the horizontal radiation patterns. See Figures 6 for example. One of the horizontal transmission patterns from North Sydney is given in Figure 43.

Figure 43: A horizontal radiation pattern for one North Sydney transmitter.

Figure 43 shows a 6-peak pattern with the peak rotated towards the SW. This is appropriate to reach the largest population in the viewing area, as for the patterns in Figure 6. The SW peak is over Lane Cove. Most of the Lane Cove population lives with 2.5 km of the towers, Figure 40. The centre of North Sydney is on the side of the SE peak and the centre of Willoughby is at the low point between the two northern peak which cover the sides of Willoughby. If the exposure of the centroid was assessed this would rank Lane Cove, North Sydney and Willoughby from high to low. This would then produce a dose response in the adult leukaemia rates that are 16.7 (9.7-26.8), 7.1 (2.8-14.6) and 6.1 (3.0-10.8) respectively.

This suggests that the Lane Cove population could experience the highest mean exposure and North Sydney the second highest. Towers 1 and 2 were increased to these high powers in 1980 with the addition of 340 kW to Tower 1 and 70 kW to Tower 2. In terms of cancer, this is likely to influence the childhood leukaemia and brain tumor rates more than the adult rates because of longer adult cancer latencies and their age structure.

Two exposure estimates were cited by Hocking et al. The first estimate is expressed as a function of radius from the centre point of the three towers. At the center between the towers this gave 1(W/cm2. The highest calculated exposures were between 4 to 8 (W/cm2 in a narrow ring at about 1 km. This would be a result of the VHF transmissions. These are the areas immediately adjacent to each of the towers where few people reside. Outside this the calculated exposure declines as an inverse square to be 0.2(W/cm2 at 4 km, the limit of the "exposed" population, Figure 44.

Figure 44: Logarithm of the calculated power densities (in (W/cm2) for TV signals from the three TV towers against distance from the centre of the towers.

The second data set was a number of actual readings taken by the Commonwealth Dept of Communications. These were generally about 1/5th of the calculated values at any point. This is largely explained by sheltering effects of the line-of-site signals, by hills and buildings. It must also be remembered that the population spends a proportion of its time inside, typically at least 10-12 hours, during which the RF exposure will be significantly reduced. A factor of 2 is conservative and is still likely to over estimate the mean population exposures. The results of the calculated and measured exposures are given in Figure 44.

The lower limit at the 4 km circle, enclosing the "exposed" population, the estimated mean residential exposure (50% of measured) is 0.025(W/cm2 or 25 nW/cm2. With the population of greater Sydney being to the SW of the towers it is probable that their horizontal antenna pattern is similar to Figure 6b, giving much lower exposures to Willoughby and lower exposures to North Sydney than to Lane Cove at the same radial distance.

15.2.4 North Sydney Study Results:

Hocking et al. shows statistically significantly increased incidence and mortality for total leukaemia, Lymphatic Leukaemia and Other Leukaemia for the whole population, with Risk Ratios in the range 1.09 to 1.67 for leukaemia incidence and 1.01 to 1.57 for leukaemia mortality. The highest relationship is for Lymphatic Leukaemia mortality, RR = 1.39 (95% CI: 1.00-1.92), Table 21.

For childhood leukaemia the relationships are generally stronger even though the sample size is smaller. Significant relationships exist for Total Leukaemia and Lymphatic leukaemia incidence and mortality.

Table 21: 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.

Cancer Type	RR (95% CI)	Cases
Incidence		
Brain Tumour	0.89 (0.71-1.11)	740
Total Leukaemia	1.24 (1.09-1.40)	1206
Lymphatic Leukaemia	1.32 (1.09-1.59)	536
Myeloid Leukaemia	1.09 (0.91-1.32)	563
Other Leukaemia	1.67 (1.12-2.49)	107
Mortality		
Brain Tumour	0.82 (0.63-1.07)	606
Total Leukaemia	1.17 (0.96-1.43)	847
Lymphatic Leukaemia	1.39 (1.00-1.92)	267
Myeloid Leukaemia	1.01 (0.82-1.24)	493
Other Leukaemia	1.57 (1.01-2.46)	87

The strongest relationship is for childhood lymphatic leukaemia death, RR=2.74 (95%CI: 1.42-5.27). The study found that 59 children had died from having leukaemia when the expected number was 25.43, an excess of 33.6 deaths. For childhood lymphatic leukaemia 39 children died when 14.2 were expected, an excess of nearly 25 children, Table 22.

The authors searched diligently for confounding factors, including social economic factors, air pollution (benzene), ionizing radiation, migration, hospitals, high voltage power lines and local industries. None affected the relationships found. They investigated the possibility of clustering and found that no significant heterogeneity was found (p=0.10 for incidence and p=0.13 for mortality).

Table 22: 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.

Cancer Type	RR (95% CI)	Cases
Incidence		
Brain Tumour	1.01 (0.59-2.06)	64
Total Leukaemia	1.58 (1.07-2.34)	134
Lymphatic Leukaemia	1.55 (1.00-2.41)	107
Myeloid Leukaemia	1.73 (0.62-14.81)	9
Other Leukaemia	1.65 (0.33-8.19)	8

Mortality			
Brain Tumour	0.73	(0.26-2.10)	30
Total Leukaemia	2.32	(1.35-4.01)	59
Lymphatic Leukaemia	2.74	(1.42-5.27)	39
Myeloid Leukaemia	1.77	(0.47-6.69)	11
Other Leukaemia	1.45	(0.30-6.99)	9

15.2.5 North Sydney Study Critique:

McKenzie, Yin and Morrell (1998) produced a very useful critique of Hocking et al. (1996). They carried out an analysis cancer rates of more of the municipalities in the Sydney metropolitan area. They also showed that socio-economic status is a risk factor for acute lymphoblastic leukaemia (ALL) in N.S.W. The concentrated of ALL because this was found by Hocking et al. to be the most elevated childhood cancer in the vicinity of the North Sydney TV towers. McKenzie et al. also undertook a number of exposure calculations in an attempt to characterize the mean exposure for each of municipalities.

Their calculations used a simple inverse square formula that does not produce side lobes, which elevated VHF and UHF signals do produce. These side-lobes influence ground level exposures out to beyond 4 km, Figure 5. They are also unaware of horizontal radiation pattern differences. They highlighted the role of shadowing as a source of lower measured values compared with calculated values. They showed with measurements at a particular location how the exposure varies from direct exposure on the roof (3(W/cm2), in the garden on the street (0.066(W/cm2) and inside the home (0.017(W/cm2). This verifies the factors used for the mean exposure estimates made in section 2.10.

Using these 'representative' calculated exposures for each municipality, McKenzie et al. plotted the total childhood ALL incidence as a function of their calculated exposure, Figure 45. The second diagram shows the improvement if Willoughby is reduced to its more probable mean exposure level.

Figure 45: A dose-response relationship for total childhood cancer in Sydney, Australia, from McKenzie, Yin and Morrell (1998), with the 95% confidence intervals added. RFR exposure is the calculated exposure at the geographic centroid of each municipality. The right hand diagram shows the effect of moving Willoughby to 0.3(W/cm2 to adjust for the horizontal radiation pattern.

Hocking et al. (Hocking, Gordon and Hatfield (1999)) reject the substance of the criticisms of McKenzie, Yin and Morrell, concluding:

"In summary, we consider that the second look at our study had important deficiencies regarding post hoc analysis of data. Their conclusion that their analysis 'casts doubt on the apparent association between childhood incidence of acute lymphoblastic leukaemia and television RFR' is not justified. If anything, their analysis confirms our own finding of a modest association, which warrants further study."

In this report the contention is that the conclusion could be even stronger than seen by Hocking et al. The calculated exposure used by McKenzie et al. does not take into account the very much higher power being radiated from Tower 1. When this is noted, Lane Cove has the highest calculated mean exposure, North Sydney is next, closely followed by Willoughby. These ignore the horizontal patterns in Figure 6b, Figure 43. If in Figure 45, Willoughby was moved to 0.3(W/cm2 then the results form an even stronger dose-response relationship than is shown by their original assumptions. The error of not taking into account the radiative power of each tower is significant.

The North Sydney Study shows significant increases in adult and childhood leukaemia incidence and death. When realistic estimates of the mean exposure of each municipality is used, a dose-response

relationship results. Measurements confirm that the estimates of the mean population exposure at the 4 km ring, the outer edge of the 'exposed population', is about to 25 nW/cm2.

15.3 United Kingdom Regional TV Tower Study:

Dolk, Shaddick, Walls, Grundy, Thakrar, Kleinschmidt and Elliott (1997): “Cancer Incidence near Radio and Television Transmitters in Great Britain: 1. Sutton Coldfield Transmitter”.

Dolk, Elliott, Shaddick, Walls, and Thakrar (1997): “Cancer Incidence near Radio and Television Transmitters in Great Britain: 2. All High Power Transmitters.”

15.3.1 The Study Context:

Dr Helen Dolk and her colleagues responded to concerns about a cluster of seven cases of leukaemia and lymphoma who were patients of a Birmingham GP, Dr Mark Payne, and who lived near the Sutton Coldfield Transmitter. They obtained data from the cancer registry and found a high incidence of adult leukaemia near the tower, which declined with distance. They assumed that this was a dose-response relationship that was following an inverse square law for exposure decline with distance from the transmitter. Before they published this result they decided to extend the study to 20 other regional TV towers throughout the United Kingdom.

At these individual sites, and for all the 20 sites combined, the adult leukaemia rate was found to be low near the tower, rose to form a broad variable peak between about 1 km and 5 km, and then declined with distance. Over all distance it didn't follow an inverse square law and therefore it failed to confirm the result found at Sutton Coldfield, Figure 46. Thus Dolk et al. (1997b) concludes that the follow-up study "at most gives very weak support to the Sutton Coldfield findings." ICNIRP accepts this conclusion and states that the results of these U.K. studies "are inconclusive".

Figure 46: Cumulative radial adult leukaemia patterns for the 21 site UK study, Dolk et al.

There are two types of radial transmission signals and two types of radial cancer patterns:

Type A : UHF signals that are low near the tower, rise to a broad peak between 2 and 6 km and then decline with distance, Figure 30.

Type B: VHF signals have a peak within 1 km and decline with distance in an undulating fashion, Figure 5.

For a high cancer rate to be detectable near a tower three factors are necessary:

There must be a large population. This requires a high population density because there is only a small area within 1 km radius of the tower and a high proportion of this is likely to be the open field in which the tower itself is sited.

There needs to be a high radiation exposure for the radiation to be able to elevate the cancer rate. This occurs for the lower frequency, VHF, FM signals, Figure 5, Figure 31.

The cancer type needs to be RF-radiation sensitive to assist in raising the cancer incidence above the background level. Leukaemia and Lymphoma are very RF-sensitive cancers, Szmigielski (1996), Milham (1985, 1988), Hocking et al. (1996).

These factors completely explain these results. Sutton Coldfield is the only tower that has these three factors. All other towers lack at least one factor and therefore cannot show a high cancer rate near the tower. In fact they all follow a Type A pattern which is a dose response relationship of cancer rate as a function of mean exposure. This for all radial cancers outlined in the Tables they follow a dose response relationship appropriate to their radiation patterns.

15.3.2 Sutton Coldfield Cancer Study Results:

For adult cancers the results are presented in the following two tables. These tables show two radial cancer patterns:

Type A: For All Cancer, Non-Hodgkin's Lymphoma, Skin Melanoma and Bladder cancer the cancer rates are low near the tower, rise to a complex broad peak between 1 km and 10 km. The skin melanoma drops faster than all the others. This is typical of UHF radiation patterns, Figure 28.

Type B: Adult leukaemia has 6 people within 1 km. This gives a high O/E ratio close to the tower. In all other respects it is similar to the first group. This is typical of mixed VHF/UHF radiation patterns, Figure 31.

In following the radiation patterns appropriate for each type of site these data follow dose response relationships with radial distance and also between high powered TV, moderate powered TV and low powered FM radio sites. This is very strong evidence that there is a causal relationship between low intensity RF exposure and adult leukaemia.

Figure 47: Radial cancer rates around the Sutton Coldfield TV Transmitter.

Figure 48: Radial adult cancer rates around the Sutton Coldfield TV transmission tower.

Figure 49: Radial cancer incidence around the Sutton Coldfield TV/FM tower near Birmingham, with both VHF and UHF transmissions, Dolk et al. (1997a).

The radial cancer incidences around the Sutton Coldfield tower shows that they are generally high in the 1 to 3 km range, low in the 4-5 km range, slightly higher in the 6-8 km range and diverging up and down outside 8 km. All Cancer follows this pattern. Being a high powered tower with VHF FM transmissions the radial exposure pattern is very likely to be similar to the Sutra Tower, Figure 29.

15.3.3 Childhood Cancer results:

This study involves a far smaller sample than the San Francisco study, less than half a million compared to several million total population, but this study considers a wide range of cancer types for adults as well as for children. However, the population of children involved is very small, especially in the “exposed” group, and therefore reaching statistical significance is unlikely. For example, at Sutton Coldfield there are 97 children with cancer with 10 km of the tower. Within 2 km of the tower there were two childhood leukaemia cases when 1.1 was expected. This gives RR = 1.82 which is elevated but non-significant. Hence the Sutton Coldfield study cannot reliably address the childhood cancer issue. This problem, of small numbers, also limits the reliability of relationships with individual cancer types, especially close to the towers where population numbers are necessarily small.

15.3.4 The 20-Site Study:

Figure 48 (Table 1) shows in the course radial analysis that Skin Melanoma is higher close to the towers whereas Bladder Cancer is more broadly elevated, consistent with Sutton Coldfield. All of these sites show the Type A pattern from above, with leukaemia rates being low near the tower, rising to a broad, complex peak and then declining with distance. The rate of decline is slower with the more high-powered transmitters, Group 1, Group 2, Crystal Palace and Wenvoe.

Figure 50: Cancer data for the 20 site U.K. study from Dolk et al. (1997b).

The elevated cancers identified by Dolk et al. include Leukaemia, Non-Hodgkin's Lymphoma, Skin Melanoma and Brain, Bladder, Male and Female Breast, Colorectal, Stomach and Prostate Cancer. Some are elevated only in the 0-2 km ring and some in the 0-10 km zone, Figure 50.

Figure 51: Adult leukaemia as a function of radial distance from regional TV transmission towers, for all 20 sites and for a number of individual sites throughout the United Kingdom, Dolk et al. (1997b).

Figure 52: Other cancer sites in the broad radial analysis from Dolk et al. (1997b).

15.3.5 21-Site Childhood Cancers:

The ability of these studies to detect childhood cancers was severely limited by small number of children who live within 10 km of these TV Transmission Towers. While in the 21 sites there were 3609 adults ((15 years) with leukaemia, there were only 317 children ((14 years). Close to the towers, i.e. inside 2 km there were 101 adults and 10 children 1with leukaemia. The expected incidence was 94.17 and 8.94 respectively. This gives O/E = 1.073, 95%CI: 0.81-1.42 for adults and O/E = 1.12, 95%CI: 0.61-2.06 for children.

This the childhood leukaemia rate is elevated around the 21 TV transmission towers in the United Kingdom, more so than the adult rate, but the small numbers mean the elevation is not significant.

For brain tumours, the rate is elevated within 10 km of the towers, for Malignant and Benign Brain Tumour O/E = 1.06, 95%CI: 0.93-1.20, n=224, and for Malignant Brain Tumours, O/E = 1.03, 95%CI: 0.90-1.18. It would have been interesting to observe the radial cancer rates for these tumours for the numbers involved are much higher than for the Sutra Tower Study. The equivalent data from the North Sydney Study is for Childhood Leukaemia O/E = 1.8, 95%CI: 1.2-2.5, n=33 and for Brain Tumour O/E = 1.3, 95%CI: 0.7-2.3, n=12. Hocking et al. use a 4 km radius whereas the UK data is for a 10 km radius. There is a different mix of TV and FM signals and signal strengths in the North Sydney Study than in the UK Study. Accepting these differences they all show elevated leukaemia and brain tumor. The very large output power of the Sutra Tower results in far higher childhood cancer rates.

15.3.6 Conclusion:

If the authors had known about and applied the available engineering knowledge about radial exposure patterns, they would have concluded that there was a significant dose-response relationship between a number of adult cancers and RF exposure from TV/FM transmission towers. There is complete internal consistency once the different radiation patterns are recognized. The study also shows elevations in cancers from sites all over the body, with dose-response relationships being evident for those presented. Hence the observations are consistent with the data in Selvin et al.(1992) data and analysis of Robinette

et al. (1980), Milham (1985,1988) and Szmigielski (1996). This shows a causal relationship between RF exposure and cancer.

Excesses in cancer are still seen at 10 km, where the direct exposure is 5 to 10 times less than the UHF peak of 1.3(W/cm2, i.e. between 0.13 and 0.26(W/cm2. Applying the lifetime mean residential exposure factor of 0.15 reduces the exposure associated with these adverse health effects to the range 0.02 to 0.034 (W/cm2.

The data in Dolk et al. is internally consistent, shows elevated childhood leukaemia and brain tumor, and a set of dose-response relationships which are likely to be highly significant, if related to realistic radial RF patterns, for cancer at a wide range of body sites including All Cancer, Leukaemia, Non-Hodgkin's Lymphoma, Brain Cancer, Bladder Cancer, Prostate Cancer, Skin Melanoma, Male and Female Breast Cancer and Colorectal Cancer. This is also consistent with Robinette et al. (1980), Szmigielski (1996) and Milham (1985, 1988).

ICNIRP's Cancer Assessment - Conclusion:

Discounting the two inappropriately included studies, Barron and Barraf (1958) and Rothman et al. (1996a), all of the remaining studies report significant increases in cancer incidence and mortality from RF/MW exposure, or total mortality. Significant dose-response relationships are reported by Robinette et al. (1980) for Respiratory Cancer and Beall et al. (1996) for Brain Cancer; are contained in the data of Selvin et al. (1992) for All Childhood Cancer, Leukaemia, Lymphoma and Brain Cancer; of Dolk et al. (1997a,b) for Adult Leukaemia, Bladder Cancer, Melanoma; and in the extended analysis of Hocking et al. (1996, 1998) through McKenzie, Yin and Morrell (1997) and of Szmigielski (1996, 1998). A dose response relationship for total mortality is also shown by Rothman et al. (1996b).

The three challenges in this report of the ICNIRP assessment, of using the wrong methodology and of using Constructive Dismissal to defend their flawed methodology, and neglecting a large body of epidemiological research, have been proven.

The first is proven by comparison with the approaches and levels of evidence used for chemicals and air pollution. The second is proven by a detailed analysis of the research results cited by the ICNIRP. The Reproductive and Cancer Assessments of the ICNIRP are very limited in their scope and very selective in the studies chosen. In both cases inappropriate studies have been included to incorrectly bolster ICNIRP's case that there is no reliable evidence of adverse effects. In both cases published material and author's conclusions have been misquoted with a bias towards finding no effects. In both assessments the cited papers include elevated, significantly elevated and dose-response increases in miscarriage and cancer. In the cancer case it is sufficient to establish cause and effect with the material cited. This is strongly confirmed when the very large number of studies which are available are included. For Reproductive Effects, the cited material is indicative, but when the additional available studies are included and the biological mechanisms are considered, consistent with the cancer assessment and evidence of neurological and hormonal influences of EMR, a causal relationship between low level EMR exposure and reproductive effects is established.

The statements in opposition to the adoption of the ICNIRP Guideline for use a national standards is proven to be fully justified. The ICNIRP guideline is many orders of magnitude above the levels at which known adverse human health effects occur. Hence the world's population is being put severely at risk by the campaign to adopt the flawed ICNIRP Guidelines globally.

17. Additional studies not cited by ICNIRP:

Studies cited in the WHO (1993) review:

There is a large body of epidemiologic scientific literature that is relevant to the assessment of RF/MW exposures risk of cancer. Almost all of these studies have not been referenced in the WHO/UNEP/IRPA review, WHO (1993), that is cited by ICNIRP to be one of the "more detailed reviews". In fact the ICNIRP review covers more published studies than does the WHO/UNEP/IRPA review, but both ignore most of the published epidemiological studies. Three of the studies cited by WHO (1993) are omitted by ICNIRP. They are the case-study by Archimbaud et al (1989), and Air Force Base studies of Lester and Moore (1982) and Lester (1985) and Amateur Radio Study of Milham (1985). WHO (1993) omits the Wichita Kansas Study of Lester and Moore (1982a) and the Operator Electrical Workers Study of Milham (1985) and the Amateur Radio Operators study, Milham (1988).

WHO (1993) and ICNIRP (1998) share many of the flawed methodological approaches and the assumption of the RF-thermal effect that the only RF/MW effect is heating of human tissues. The review teams were chaired by the same person during most of the 1990's, Dr Michael Repacholi.

WHO (1993) states that no significant effects were found in Lilienfeld et al. (1978). This has been proved here to be wrong. The U.S. Air Force Bases Studies are described as "contradictory" because Polson and Merritt (1985) correctly criticize Lester and Moore for relating cancer rates in counties to the existence of Air Force bases in those counties when many cities which are close to Air Force bases are in adjacent counties without Air Force bases. Lester (1985) adjusted the analysis accordingly and concludes:

"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 the county containing the city nearest the base, which would be expected to be a truer indicator of the effect produced by some factor emanating from the base than would a county in which the base is situated but in which the nearest city is farther away."

The Lester (1985) updated result was in the same journal issue as Polson and Merritt (1985) and yet WHO (1993) ignores the existence and significance of the correction. This again reveals the bias towards dismissing evidence of effects.

WHO (1993) acknowledges that Szmigielski et al. (1988) and Archimbaud et al.(1989) show a relationship between RF/MW and increased risk of cancer, including Acute Myelogenous Leukaemia. In the case of Milham (1985) the increase in Leukaemia is acknowledged. However, the result is questioned because it is noted that many of the Amateur radio operators are also employed in the "Electrical Industries". Hence they are exposed to PCBs, solvents, fumes, and 50/60 Hz magnetic fields and not 300Hz-300GHz radiation. This claim is challenged by evidence which supports the EMR Spectrum Principle because many ELF powered appliances also emit RF/MW radiation which is much more bioelectrically active.

The overall WHO (1993) conclusions include the statements:

"In summary, the epidemiological and comparative clinical studies do not provide clear evidence of detrimental health effects in humans from exposure to RF fields".

And

"The question of whether RF might act as a carcinogen should be further evaluated in epidemiological studies."

If the studies available to it had all be included and the proper epidemiological assessment principles had been followed, then the WHO review should have concluded that RF radiation is a probable human

carcinogen, because they had more and stronger evidence than the U.S.E.P.A. review team in 1990, and they concluded that RF/MW was a possible human carcinogen.

Lester and Moore (1982a) is their initial study which tested the hypothesis that radar might increase the risk of cancer by noting that Wichita Kansas had radar sets on Air Force Bases on two opposite sides of Wichita. The tested the hypothesis by separating populations which were exposed to no radar signals, living in valleys, one radar signal, on one or other hill slope, and two radar signals by living on ridges. The cancer incidences are 303, 429 and 470 per 100,000 (1.00:1.42:1.55). The dose-response association persisted through age, sex, race and socio-economic adjustments.

Dr Sam Milham's two other studies not cited in WHO (1993) are Milham (1985a), a large study of Electrical Workers in Washington, and Milham (1988) and updated Amateur Radio Study covering California and Washington. Milham (1985a) studied cancer rates in 486,000 adult male workers who were in occupations in Washington State which had potential exposures to electromagnetic fields. This showed elevated and significantly elevated cancer rates in many body organs. The results are summarized in Table 23.

Table 23: Summary of all site cancers from Robinette et al. (1980), using AT/ET except for Brain cancer (FT/ET), Milham (1985a), Szmigielski (1996) and for Dolk (1997a,b) using the maximum and/or significant result in the radial patterns.

	Robinette		Milham	Szmigielski		Dolk(a)	Dolk(b)	
Exposure Regime			RF/MW		Mixed	RF/MW	RF/MW	RF/MW
	High	Mod.	High	Low	Low			
Relationship	RR		PMR	RR	O/E	O/E		
Sample Size(N)			202	2649	55,500	17409	13372	
Symptoms								
All Malignant Neoplasms				1.66*	106**	2.07*	1.20*	
Esophageal and Stomach						3.24**		
Respiratory Tract, Lung				1.75	114**	1.06		
Colorectal/ bladder (1)					3.19**	1.36/1.76		1.10
Liver, pancreas				117*	1.47			
Skin, Melanoma			2.66		1.67*	2.39*	1.11	
Thyroid				1.54				
Brain, CNS (2)	2.39		143**	1.91*	1.31	1.06		
Leukaemia	2.22*		136*	6.31***	1.74*	1.15		
Non-Hodgkins Lymphoma					164**	5.82***	1.30*	
Acute Leukaemia (Lympho)					162**	5.75*	3.57	1.04
Acute Myeloblastic Leuk.						8.62***	1.02	1.17
Chronic Myelocytic Leuk.						13.90***		1.23
Chronic Lymphoblastic Leuk						3.68**	2.56*	1.20

p-values: * <0.05; ** <0.01; *** <0.001

Note (1): Colorectal for Szmigielski and the left Dolk(a) and bladder for the right Dolk(a) and Dolk(b).
Note (2): In Milham 16 of the unspecified neoplasms were brain tumors which have been added to this group.

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 elevated but not significant (SMR = 124). However, mortality from acute myeloid leukemia was significantly elevated (SMR = 176). Elevated cancer rates were found for Esophagus, SMR = 113 (71-172); Large Intestine, SMR = 111 (89-137); Prostate, SMR = 114 (90-142); Brain, SMR = 139 (93-200), Lymphoma + Leukaemia, SMR = 123 (99-152); Hodgkin's Disease, SMR = 123 (40-288); Leukaemia, SMR = 124 (87-172) and Other Lymphatic Tissue, SMR = 162 (117-218).

Table 23 shows a great deal of consistency between several large studies which stand as proof, backed by many dose-response relationships, even at residential exposure levels, that RF/MW increases the risk of cancer over the whole body. This stands in strong contrast with the ICNIRP and WHO review conclusions.

17.2 Studies not cited by WHO nor ICNIRP:

The following is a brief summary of a number of relevant epidemiological studies which have been omitted by both WHO (1993) and ICNIRP (1998).

A broad Summary:

It is not widely recognized that ELF epidemiological studies have relevance to RF/MW assessments of effects. There are two primary reasons for this. High voltage power lines are sources of RF radiation, especially in the 3 to 30 MHz range, Vignati and Giuliani (1997). This is why you often hear a buzz on your radio as you drive under a powerline. This is outlined as part of the EMR Spectrum Principle. This evidence proves that epidemiological studies of RF/MW and ELF show elevated and significantly increased cancer in many body organs, but especially brain cancer, leukaemia and breast cancer.

More neurasthenic symptoms (chronic mental and physical weakness and fatigue) in group exposed to radar (Djordevic et al., 1979).

Higher frequency of increase in red blood cells (polycythemaemia) with microwave exposure (Friedman, 1981).

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. A significant dose-response relationship was found: No exposure: 1.00; Possible exposure OR = 1.44(1.06-1.95); Probable exposure, OR = 1.95 (0.94-3.91), and Definite exposure, OR = 2.15 (1.10-4.06).

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.

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.

Navy Electrician's Mates have an excess risk of leukaemia, RR=2.4 (1.0-5.0), Garland et al. (1990)

Savitz and Chen (1990) show significant increased risk of childhood cancer (Neuroblastoma (OR=11.8*), Brain Tumour (OR=2.7*) and CNS tumors (OR=1.7)) associated with parents who work in electrical and electronic industries.

Increased risk for all brain tumours (RR=2.9 (1.2-5.9)) and glioblastomas (RR=3.4 (1.1-8.0)) for assemblers, and repairmen in the radio and TV industry, Tornqvist et al. (1991)

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).

Floderus, Tornqvist and Stenlund (1994) found a significant increase in Brain Tumours, RR = 12.2, 95%CI:2.8-52.5; Breast Cancer, RR = 4.9, 95%CI:1.6-11.8 and pituitary tumours, RR = 3.2, 95%CI:1.6-6.2, for electrical railway workers younger than 30 yrs.

Loomis, Savitz and Ananth (1994) found significant increases in Female Breast Cancer in Electrical Workers, OR = 1.38 (95%CI: 1.04-1.82). Adjusted Ors for electrical engineers, OR = 1.73 (95%CI: 0.92-3.75); for electrical technicians, OR = 1.28 (95%CI:0.79-2.07) and for telephone installers, repairers and line workers, OR = 2.17 (95%CI:1.17-4.02).

Women working in the Telephone Industry showed many excess cancers. Among white women (age <49) Rectum Cancer MOR = 3.3 (1.2-8,7); connective tissue MOR = 4.4 (2.2-8,2); Breast Cancer MOR = 1.6 (1.2-2.1), corpus uteri MOR = 3.3 (1.5-7.5, Ovary MOR = 2.1 (1.3-2.5); and Brain MOR = 2.1 (1.2-3.7). Excess risks of connective tissue cancer among engineers and technicians; office workers; telephone operators; and mechanics and repairers, MOR = 8.5, 4.9, 1.7, and 4.4 respectively, Dosemeci and Blair (1994).

Significantly increased and dose-response increases of total Mortality, All Cancer, Leukaemia and Brain Tumour were found in US Utility Workers (1950-1988) and sub-set of occupations, Savitz and Loomis (1995).

Increased risk of female breast cancer with exposure to radiofrequency EMF, RR=1.15 (1.1-1.2), Cantor et al. (1995).

Tynes et al. (1996) observed an excess risk of Female Breast Cancer for female radio and telegraph operators exposed to RF (405 kHz-25 MHz), SIR = 1.5 for > 50 yr of age.

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 physical and scholastic performance of children in the open field exposure range of 0.0008-0.41 (W/cm2, mean measured level in the range 0.0028- 0.039(W/cm2, Kolodynski and Kolodynska (1996).

A 6-fold increase in broken chromosomes in the peripheral erythrocytes of the exposed cows ($p<0.01$). The measured exposure is in the range 0.042 to 6.6(W/cm2 placing the mean exposure in the range 0.157 to 0.63(W/cm2, Balode (1996).

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(W/cm2, a mean open field exposure of 0.039(W/cm2 and measured distance exposure of 0.0027(W/cm2 (for the radar signal). A probable biological mechanism was identified through observed changes in physiological conditions, Balodis et al. (1996),

Chromosome and reproductive damage in plants exposed RF/MW in the range 0.042 to 6.6(W/cm2. Magone (1996).

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(W/cm2, below 0.1(W/cm2. and even below 0.01(W/cm2.

Stenlund and Floderus (1997) identified increased Testicular Cancer in middle ELF exposure (>0.28(T) and high exposure (>0.4(T) compared with low exposure ((0.15(T) with OR = 1.3 (CI:0.7-2.5) and OR = 2.1 (CI:1.0-4.3) for those (60 years and OR = 1.9 (CI:0.8-4.4) and OR = 3.9 (CI:1.4-11.2) for men (40 years. These produce significant ($p<0.05$) dose-response relationships.

Brain tumour studies:

The Bioelectromagnetic principles include the EMR sensitivity of the human brain. This suggests that brain cancer is a probable result of EMR exposure. In section 16.2 several studies show significant increases in brain cancer. A literature search, assisted by MEDLINE, identified over 60 studies showing increased rates of brain cancer, and over 30 showing significant increases in brain cancer in EMR exposed populations. The following studies show significant increases in brain tumours for children and adults, in ELF to RF/MW exposures in residential, occupational, commercial and military situations.

Sweden, Power lines, Children RR = 3.9, $p<0.05$, Tomenius (1986),

Denver, Residential, children, 2-level wire code Savitz et al. (1988)
OR = 2.04 (1.11-3.76)

U.S., prenatal electric blanket, Savitz, John and Kleckner (1990)
Brain cancer in children , OR = 2.5 (1.1-5.5)

Finland, power lines Verkasalo et al. (1993)
CNS tumours in boys SIR = 4.2, (1.4-9.9)
>0.2(T or > 0.4(T-years

Denmark, High voltage installations Olsen et al. (1993)

Children, >0.4(T Significantly increased cancer, including CNS tumours.

Denver, Residence Savitz and Kaune (1993)
Children, Wire Code OR = 2.5 (1.1-5.5)

U.S. Meta Analysis of 13 studies Washburn et al. (1994)
Residential Children, CNS RR = 1.89 (1.34-2.67)

Washington State, Electrical Workers Milham (1985)
All Groups PMR = 123, p<0.05) n=101
Electrical and electronic technicians PMR = 134 n=7
Power Station Operators PMR = 130 n=3
Electricians PMR = 155 p<0.01) n=46

Maryland, U.S. electrical industries Lin et al. (1985)
Brain tumours Died significantly earlier n=951

United States, 16 States (Mortality) Loomis and Savitz (1990)
Electrical engineers and technicians OR = 2.7 (2.1-3.4)
Telephone workers, OR = 1.6 (1.1-2.4)
Electric power workers OR = 1.7 (1.1-2.7)
Electrical workers in
manufacturing industries OR = 2.1 (1.3-3.4)

Finland, Occupational exposures Juutilainen, Laara and Pukkala (1990)
CNS tumour, 25-64 year old male
Probable exposure RR = 1.3 (0.7-2.3)
Possible exposure RR = 1.3 (1.0-1.6)

Germany, electrical workers Schlehofer et. al. (1990)
Women RR = 5.2 (1.4-20.1)
Men RR = 0.9 (0.2-2.3)

Aerospace electromechanical workers Park et al. (1990)
All workers PMR = 4.2 (p<0.0001) n=583
For hourly workers with up to
20 years of work PMR = 8.7 (p=0.000003).

Sweden, Occupational exposure Tornqvist et al. (1991)

Assemblers and repairmen in radio and TV industry
All brain tumours SMR=2.9, (1.2-5.9)
Glioblastomas SMR=3.4, (1.1-8.0)

All welders SMR=1.3, (1.0-1.7)
Iron/steel industry SMR=3.2, (1.0-7.4)
For glioblastomas SMR=1.5 (1.1-2.1).

Norway, Occupational electric Tynes, Andersen and Langmark (1992)
and magnetic exposures.

Overall brain tumour SIR = 1.09 (0.9-1.41) n=77

ISCO code weak magnetic/
electric exposure SIR = 2.20 (1.01-4.18)

Tram drivers SIR = 2.04 (0.42-5.98)
Radio/telegraph operators SIR = 1.20 (0.25-3.49)
Electricians, installation SIR = 1.23 (0.67-2.07)
Electricians, power supply SIR = 1.16 (0.43-2.53)
Power plant operators SIR = 1.15 (0.24-3.36)
Power line workers SIR = 1.51 (0.78-2.64)
Telephone installers SIR = 1.22 (0.49-2.51)
Railway track walkers SIR = 2.20 (1.1-4.18)
Total for these groups SIR = 1.14, (0.9-1.42)

New Zealand, electrical workers Preston-Martin et al. (1993)
Electricians OR = 4.6 (1.7-12.2)
Electrical engineers OR = 8.2 (2.0 - 34)

Sweden, Occupational exposure Floderus et al. (1993)
Low exposure RR = 1.0 (0.7-1.6)
Moderate Exposure RR = 1.5 (1.0-2.2)
High Exposure RR = 1.4 (0.9-2.1) n=261

United States, Telephone industry Dosemeci and Blair (1994)
Women from 24 states OR = 2.1 (1.2-3.7)

England, Electrical workers Fear et al. (1996)
PPR = 118 (103-136)

Brazil, San Paulo, Utility workers Mattos and Koifman (1996)
PCMR = 7.7 (1.02-9.65)

France electrical utility workers Guenel et al. (1996)
Brain tumour OR = 3.06 (1.08-8.74) n=69
5 year latency OR = 3.69 (1.10-12.43)

Telephone and radio operators, electricians, Kaplan et al. (1997).
All Brain Tumours OR = 1.2 (0.2-5.2)
Malignant Brain Tumours OR = 1.4 (0.2-8.7)

Danish Utility Workers Johansen and Olsen (1998)
Women SIR = 1.3 (0.7-2.2) n=15
Women >10yrs < 0.09(T SIR = 1.9 n=4
0.1-0.29(T SIR = 9.2 p<0.05 n=2

United States, Occupational Cocco, Heineman and Dosemeci (1999)
CNS cancer RR=1.2 to 1.3 p<0.05 n =12980

United States: Microwave repair workers Zarek (1977)
RR = 74.1 (15.0-367), p<0.001 n=2

United States Embassy in Moscow: Lilienfeld et al. (1978)
Radar RF/MW exposure

Males Working in the Embassy SMR = 20 (2.4-72.2) , p<0.01 n=2

Navy, Korean War, FT/ET groups, RF/MW Robinette et al. (1980)
For brain, eye and CNS RR = 1.66 (1.06-2.60) n=8

New Zealand, electrical workers Pearce, Reif and Fraser (1989)
Radio and TV repair OR = 7.86 (2.2-28.1) n=2
Electricians OR = 1.68 (0.75-3.79) n=6
Total electrical workers OR = 1.62 (1.04-2.52) n=21

Canada, British Colombia pilots Salisbury et al. (1991)
Elevated PMRs for brain cancer and nervous system cancer.

Canada, Commecial pilots Band et al. (1990)
ELF/RF SMR = 4.17 (1.4-9.5), p=0.017 n=4
SIR = 3.45 (1.2-7.9), p=0.03 n=4

England, British Airways pilots Irvine and Davies (1992)
ELF/RF PMR = 2.68, p<0.05

U.S. Air Force Grayson and Lyons (1996)
Aircrew compared OR = 1.77(1.17-2.68) n=37
with non-flying crew
Adjusted for rank, socioecom. OR = 1.22 (0.76-1.95)

Polish Military Szmigielski (1996)
Mortality RR=1.9 (1.08-3.47), p<0.05 n=55,500

U.S. Air Force personnel Grayson (1996)
Age-race-adjusted odds ratios
ELF fields OR = 1.28 (0.95-1.74)
RF/MW fields OR = 1.39 (1.01-1.90)

Brazilian Naval Personnel Santana, Silva and Loomis (1999)
Aged <56 yrs OR = 4.63 (2.54-8.45) n=40
Unmarried men OR = 3.18 (1.69-5.99)

This lists 34 separate studies, covering over 56 occupational or residential groups which have been exposed to EMR and more than 40 show significant increases in brain cancer incidence or mortality.

Thirteen studies show dose-response relationships for EMR exposure and brain tumor:

Denver, United States, power lines Wertheimer and Leeper (1979)
Childhood) Birth Address RR = 1.83, p=0.04 n=22
CNS tumors) Death Address RR = 1.76, p=0.017 n=30
Dose related for children living at same address.

Eastern U.S. Electronic Industries Thomas et al. (1987)
Astrocytic brain tumours RR=4.9 (1.9-13.2)
Duration Employed (yr)
Unexposed <5 5-19 (20
RR 1.0 3.3 7.6 10.4

Solder fume adjusted RR 1.0 1.65 3.8 5.2
(trend p<0.05)

East Texas, Males Glioma Speers, Dobbins and Miller (1988)
n=202
Transportation, communication
and utilities industries OR = 2.26 (1.18-4.32)
Electricity or electromagnetic fields OR = 3.94 (1.52-10.20) Trend: p<0.01

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

Glioma OR=1.8 (0.8-4.3) p for trend =0.05
Astrocytoma, >5 years empl. OR=4.4 (1.2-15.6)

Los Angeles Country, electrical industry Mack et al. (1991)
n=272
Astrocytomas RR = 10.3 (1.3-80.8) Trend, p=0.01

San Francisco, Sutra Tower (FM/TV) Selvin et al. (1992)
Children < 21 yrs RR = 2.87, (1.30-6.32), p<0.01 n=35
Comparing <4.5km and >4.5 km Trend p <0.0001

Quebec, Ontario and French Utility workers Theriault et al. (1994)
>=90th percentile, years of exposure

(5 years OR = 0.81 (0.3-2.21)
(20 years OR = 1.78 (0.69-4.62)
>=20 (20 years OR = 5.90 (0.37-94.4)

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

U.S. Electrical Workers Savitiz and Loomis (1995)
Mortality Dose-response OR = 1.94 per (T-yr

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

U.S. Computer exposures Beall et al. (1996)
Computer Programmers (>10 yrs) OR = 2.8 (1.1-7.0) Trend p = 0.04
Engineering/Technical (>10 yrs) OR = 1.7 (1.0-3.0) Trend p = 0.07
Glioma, All subjects, 5yr programme OR = 3.9 (1.2-12.4) Trend p = 0.08

Ontario Hydro male employees (Adjusted ORs) Miller at al. (1996)
Brain Tumour Mod. Field OR = 1.27 (0.32-5.41)
High Field OR = 1.33 (0.52 -10.8) Both show trends.
Benign Brain Mod. Field OR = 5.38 (0.42-69.3)
Tumour High Field OR = 5.64 (0.3-105)

Norway Tynes and Haldorsen (1997)

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

Of the thirteen studies showing dose-response relationships for EMR exposure and brain cancer, at least seven show significant dose-response relationships. The majority of studies involve industrial exposures mainly to ELF fields but many appliances, like power lines, also produce a spectrum of RF signals. One, Beall et al. (1996), involves VDT exposure that is mixed ELF/RF. Two of the significant dose-response studies involve RF/MW, Thomas et al. (1987) and Selvin et al. (1992), and Thomas et al. for workers.

17.5 Leukaemia Overview:

The most frequently identified cancer associated with ELF EMR and RF/MW EMR is childhood and adult leukaemia. This is unremarkable in the light of the sensitivity of the whole body to EMR and the established biological effect of altered cellular calcium ion homeostasis with the strong implications towards impairment of the immune system from this. The ICNIRP reviews these studies, and in a very limited selection of the literature, find that Relative Risks are consistently in the range 1.5 to 3. The studies suffer generally from small numbers. The ICNIRP notes that a common cut-off point at 0.2(T is emerging but from their limited research selection, it is not strong enough, in "the absence of experimental research to form a basis for setting exposure guidelines". ICNIRP is consistent. Their inverted priorities place biological mechanism above epidemiological evidence.

The ICNIRP finds the evidence for melatonin reduction insufficient to convince them though a very selective citing of the literature. Hence they retain a guideline based on limiting induced current. At 50/60 Hz it is close to 100(T.

As with the RF/MW assessment, there is a large body of literature not cited by ICNIRP that strongly confirms the 0.2(T cut-off for residential studies of childhood cancer. All of the studies cited by the ICNIRP have mean annual exposures less than 1-10 (T for occupational studies and <0.6(T for residential studies. Hence in a Public Health Protection Approach a cut-off of 0.2(T would be identified and then a safety factor would be applied to allow for the uncertainties produced by small sample sizes and the extent of the exposure risk, i.e. the whole population. This demonstrated again how severely and consistently flawed the ICNIRP approach is.

Based on the strongly supported EMR Spectrum Principle, if ELF exposures are consistently shown to increase leukaemia then RF/MW exposures are highly likely to also produce leukaemia, and at lower exposure intensities. Table 24 summarizes RF/MW studies showing increases in adult Leukaemia.

When the RF/MW studies which have identified significant increases in Adult Leukaemia are ranked from residential, recreational, occupational and military exposures, they form a global dose-response relationship, Table 24.

All of these studies involve non-thermal exposures. Together they confirm a causal relationship between RF/MW exposure and adult leukaemia.

17.6 Summary:

The vast majority of these epidemiological studies are not cited by ICNIRP. They confirm the EMR Spectrum Principle that health effects occur across the spectrum from ELF to RF/MW and in mixed exposures. Leukaemia, Brain Tumour and Breast and other hormonal related cancers are most commonly associated with EMR exposure. Compared to chemical assessments, the strength of evidence that there is a cause and effect relationship between EMR and cancer exceeds that of most substances that are classified as human carcinogens.

Table 24: A summary of epidemiological studies involving adult leukaemia mortality or incidence, ranked by probable RF/MW exposure category.

Study	Reference	Exposure Category	Exposure Type	Leukaemia Risk Ratio	95% Conf. Interval
Polish Military (Mortality)	Szmigielski et al., 1996	High	ALL	5.75	1.22-18.16
			CML	13.90	6.72-22.12
			CLL	3.68	1.45-5.18
			AML	8.62	3.54-13.67
			All Leuk.	6.31	3.12-14.32
Korean War (Mortality)	Robinette et a. (1980)	High	All Leuk.	2.22	1.02-4.81
Amateur Radio (Mortality)	Milham (1988)	Moderate	AML	1.79	1.03-2.85
UK TV/FM (Incidence)	Dolk et al. (1997a)	Mod/Low	Adult Leuk.	1.83	1.22-1.74
			CML	1.02	0.28-2.60
			AML	1.86	0.89-3.42
			ALL	3.57	0.74-10.43
			CLL	2.56	1.11-5.05
North Sydney TV/FM towers (Mortality)	Hocking et al. (1996)	Low	All Leuk.	1.17	0.96-1.43
			ALL+CLL	1.39	1.00-1.92
			AML+CML	1.01	0.82-1.24
			Other Leuk	1.57	1.01-2.46
UK TV/FM (Incidence)	Dolk et al. (1997b)	Low	Adult Leuk.	1.03	1.00-1.07
			CML	1.16	
			AML	1.17	
			ALL	1.04	
			CLL	1.20	

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

The studies cited by ICNIRP contain sufficient evidence to conclude cause and effect between RF/MW and cancer across many body organs, especially leukaemia and brain tumour, and at chronic lifetime exposures showing dose-response relationships pointing to a zero level.

This is confirmed by more than three as many studies as are cited by ICNIRP(1998), thus confirming the cause and effect relationship between RF/MW exposure and cancer. Support also comes from ELF studies and those involving "Electrical or Utility" Occupations because of the validity of the EMR Spectrum Principle.

The ICNIRP's conclusion is:

“Overall, the results from a small number of epidemiological studies published provide only limited information on cancer risk.”

This is demonstrably wrong and grossly misleading.

18. Recommended Public RF/MW Exposure Standard:

ICNIRP's thermally based approach has been proven many times to be wrong in terms of scientific evidence and public health standard methodology. There is sufficient epidemiological evidence to establish a cause and effect relationship between chronic low level EMR exposure and many adverse health effects, including cardiac, neurological, reproductive and cancer effects. The dose response relationships indicate a cancer and reproductive problem threshold near zero.

Hence the target for the recommended standard is 10nW/cm2.

This is despite the fact that, consistent with the Bioelectromagnetic Principle 2 which identifies the sensitivity of the brain to interference by EMR, and there is confirmation from the Swiss, Schwarzenburg Study. This study identified adverse effects on sleep and a number of other serious health effects, down to mean RF levels of 0.4nW/cm2.

Significant learning difficulties were measured in children in a school in exposed to the pulsed radar signal at Skrunda, Latvia, compared to unexposed schools, Kolodynski and Kolodynska (1996). The RF-exposed children live within a 20 km radius of the radar. At 3.7 km from the radar the measured field was 0.32 (W/cm2. Assuming an inverse square law between 3.7 and 20 km from the radar, at 10 km the exposure is approximately 0.04 (W/cm2 and 0.01 (W/cm2 at 20 km. Hence the children are showing significant physical and intellectual impairment when chronically exposed in the range 10 to 40 nW/cm2.

Wever (1974) and Konig (1974) proved that human brain detect and use the Schumann Resonance signals for synchronization and maintaining the homeostatic relationship during diurnal variations. Cherry (2000) shows that the Schumann Resonance signal is a biological mechanism to many human significant adverse health effects observed in relation to geomagnetic activity variation. This shows that brains detect the Schumann Resonance signals intensity and frequency and uses hormones such as melatonin to communicate with organs and cells to maintain diurnal regulation. Since the mean Schumann Resonance intensity is close to 0.1 pW/cm2, and adverse health effects are observed for higher and lower intensity levels, then endogenous ELF and RF/MW modulated signals can interfere with hearts, brains and cells at extremely low intensities, approaching zero exposure. Hence minimization of exposure is needed to protect workers and public health.

The background RF/MW levels in Western cities are already in the range 1nW/cm2 - 5nW/cm2, except near cell sites and radio and TV towers. A practical option to avoid these demonstrated effects is to set the initial public exposure limit at
nW/cm2 (0.01(W/cm2).

In all cases all practical attempts should be undertaken to keep exposures as low as possible, below 10 nW/cm2.

A primary means of achieving public health protection is a strong move away from "wireless" technology. If major cities moved to fiber-optic cables for all telephone, fax, radio, TV and internet services, removing the need for broadcast transmission sites, the mean public exposure could be kept below 10 nW/cm2. Mobile phones, couple with their base stations, pose the highest risk in their present modes of

introduction. The usage of mobile phones should be minimized and discouraged, and the base station placement should be remote from where people live and work, from hospitals and schools.

19. Achieving the 10 nW/cm2 standard:

A cell site policy which limits the peak exposure to 1(W/cm2 at the boundary of the nearest dwelling property, is likely to have a mean weekly exposure of 0.3 to 0.6(W/cm2 because of the phone traffic going through the site. At the house the exposure is smaller, and inside it is about 1/20th to 1/40th of the outside value, i.e. 0.007 to 0.03(W/cm2. Allowing for an 75% home/away ratio, this becomes 6 to 23 nW/cm2. Away from the centre of the radiated beams the mean exposure is much lower. Hence the peak boundary exposure limit of 1(W/cm2 achieves a lifetime mean residential exposure of 10nW/cm2 for almost all of the population. This shows that the proposed standard is more achievable than it initially seems.

Note: Cherry, N.J., 2000: "Schumann Resonances, a biological mechanism for human health effects of Geomagnetic Activity". Is under peer-review for publication in the journal, Natural Hazards.

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