

Epidemiological Principles for ELF and EMR Studies

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Abstract:

Epidemiology is fundamental science and the strongest evidence for the assessment of human health effects of disease agents. Moving from a possible association to a causal effect the assessment principles are followed by the Sir Austin Bradford-Hill approach. When dealing with the health effects of electromagnetic fields and radiation some specific and important epidemiological principles must be used. Exposure assessments are vital. Electromagnetic fields and radiation are invisible, odourless, silent and tasteless, and are ubiquitous. Therefore the basic physics and engineering principles that explain the nature and strength of these fields are fundamental. The basic methods of environmental epidemiology involve identifying the disease rates in an exposed group to compare the disease rates in a non-exposed group, with no confounders to confuse the results. A major problem with EMF and EMR is that in most communities there is no non-exposed reference group because we live in homes with electromagnetic fields from electric power wires and appliances and we can receive radio, TV and cellphone signals all the times in our homes. Everyone in the world is exposed to radio short-waves and satellite microwaves. This has led to the Ubiquitous Exposure (No Non-exposed Group) Principle, an extension of the Healthy Worker Effect. For studies around Radio, TV and cell site transmission towers, the horizontal antenna patterns are used to focus most of the RF energy into beams to send them to where most of the receiving population lives. The vertical antenna patterns are a function of the frequency of the carrier signal. They have main beams and many side-lobes which produces complex radial undulating signal intensity varying with distance from the tower. For studies of people living in the vicinity of radio, TV and cellphone towers it is vital that the radiation patterns and population patterns are understood. Studies that appropriately match exposure with cancer and other health effects, show strong, consistent and significant dose-response relationships indicating causal linkage between electromagnetic fields and radiation and human health effects.

Introduction:

This report is based on the fundamental classical epidemiological approach, reinforced by a classical physics and broadcast technology understanding. The principles are summarized and the information is set out to assist the carrying out of future epidemiological studies and to assist a more appropriate interpretation of previously published studies. The primary guidance is given by three eminent 20th Century environmental epidemiologists, the late Sir Austin Bradford Hill, United

Kingdom; Professor Abraham Lilienfeld, United States; and Professor John Goldsmith, Israel.

Because of the complex nature of human bodies and human environments, careful procedures and approaches have been developed to carry out and assess human health studies. In attempting to identify the effects of a potential disease agent a careful selection of an exposed and unexposed population is carried out as the differences in the rates of illness (incidence) or death (mortality) is surveyed. Where possible a multiple gradient of exposures are identified and the related disease rates are assessed as a dose-response relationship.

Epidemiology has developed highly advanced and strongly logical approaches to identify disease agents that cause health effects in complex human populations. However few epidemiologists have understood and applied these principles to ELF and RF/MW epidemiological studies or assessments of evidence. Exceptions are Drs Nancy Wertheimer, Ed Leeper, Sam Milham and Stanislaw Szmigielski, and Professors Theo Abelin, Christoph Minder and David Savitz (and his team). Therefore the need is to combine the basic epidemiological principles with the fundamental biophysical principles and the EMF/EMR exposure patterns and assessments.

There are a wide range of exposure situations, from residential and occupational exposures to the extremely low frequency (ELF) power supply electric fields and currents that produce electromagnetic fields (EMF). There are also widespread residential and occupational exposures to radiofrequency (RF) and microwave (MW) electromagnetic radiation (EMR) exposures from radio, TV, two-way radios, radars, cordless and mobile phones, and mobile phone base stations, for example.

First Epidemiological Principle:

Epidemiological evidence is the strongest evidence of human health effects in exposed populations, Lilienfeld (1983). “The proper study of man is man”.

Because of this principle, the public and occupational health protection exposure standards for most substances, including toxic chemicals and ionizing radiation, are based on epidemiological studies. However, the public health protection standards for ELF and EMR are not.

Second Epidemiological Principle:

Statistics plays a secondary role in epidemiology:

Assessing the epidemiological evidence using a precautionary approach was promoted and guided by the eminent British epidemiologist, the late Sir Austin Bradford Hill, Hill (1965). Modern epidemiology relies very heavily on statistics. For example, some studies show highly elevated effects but they are not statistically significant and therefore the authors' conclusion is that there are no effects. A recent example of this is Johansson (2000). In contrast to this, Sir Austin Bradford Hill dismisses the use of statistical significance. For example, card room workers in

a cotton mill were chronically exposed to dust and fibre and showed elevated rates of respiratory disease. The disease rates were consistently elevated about three times higher than average, but never significantly. The evidence was strong enough that it was concluded that it was a causal effect because "The evidence was so clear cut", Hill (1965). Sir Austin addresses the question of statistical significance in the assessment of a causal relationship. He states in relation to the question of causation:

"No formal tests of significance can answer these 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 the hypothesis."

Again, modern epidemiology often uses the term "criteria" for the Bradford Hill viewpoints. This is treating this approach as a *sine qua non*, directly contrary to Sir Austin's strong statement. I believe that this is placing the public and workers at much higher risk levels by significantly raising the level of evidence thresholds for deciding about causal effects. This results in setting public health protection standards that allow exposures at levels that are extremely significantly higher than those which have been shown to cause serious health effects found from multiple independent epidemiological studies.

Third Epidemiological Principle:

Association to Causation - the Bradford Hill Approach:

To deal with the question of whether there is a causal relationship Sir Austin gives us "Here then are nine different viewpoints from all of which we should study association before we cry causation. What I do not believe - and this has been suggested - is that we can usefully lay down some hard-and-fast rules of evidence that *must* be obeyed before we accept cause-and-effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non* [essential requirement]. What they can do, with greater or less strength, is help us to make up our minds on the fundamental question – it is to 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 Bradford Hill provides a well-established and very sensible health protection approach to the assessment of the available evidence of association to causation of human exposure to a disease agent and illness. Sir Austin's approach has commonly been applied to the assessment of the effects of chemicals.

Sir Austin Bradford Hill set out his nine viewpoints as **(1) Strength; (2) Consistency; (3) Specificity; (4) Temporality; (5) Biological Gradient; (6) Plausibility; (7) Coherence; (8) Experiment and (9) Analogy**, Hill (1965).

Sir Austin discusses each of his viewpoints and gives examples to clarify the context of how the assessment was being considered. Only one, **temporality**, is essential because the exposure must take place before it can be associated with

causing the disease. For each of the other viewpoints he sets out the strengths and weaknesses. In considering all viewpoints he sets out why the viewpoint is important and why it is also important to look at the reverse side of the coin. For example, **consistency** coming from repeatedly observed similar effects by different persons in different places, circumstances and in times can be a useful viewpoint showing causation. The absence of consistency may be logical and not a reason to reject the causation hypothesis - *“there will be occasions when repetition is absent or impossible and yet we should not hesitate to draw conclusions.”*

Four of the viewpoints can individually be viewed as showing a causal effect: Strength, Biological Gradient, Specificity and Experimentation.

Strength:

“First upon my list I would put the strength of the association.” Sir Austin gives examples of studies showing relative risks of 5, 8, 20, 32 and 200 as examples of the strength of association indicating a causal relationship. The ratio of 200 was for scrotal cancer mortality in chimney sweep’s compared to the average workers scrotal cancer rate. Example of 5 came from John Snow’s classic analysis the cholera epidemic in 1854 where he found the cholera rate from two companies whose grossly polluted water produced 5 times higher cholera rate than those using a cleaner, sewage-free water supply from a rival company.

A stronger association, that is a larger relative risk, is more likely to reflect a causal relationship, Elwood (1988).

Strength can be indicated by two factors, the size of the Relative Risk and the p-value. A very large and/or very significant RR value ($p < 0.01$) can be assessed as causal. If the p-value is $p < 0.005$ or even $p < 0.001$ then the strength of the relationship is classically causal.

While strength of association can show a causal link, Sir Austin stresses further the need for consideration if there is lack of strength. *“In thus putting emphasis on strength of association we must, nevertheless, look at the obverse of the coin. We must not be too ready to dismiss a cause-and-effect hypothesis merely on the grounds that the observed association appears to be slight. There are many occasions in medicine when this is in truth so.”*

Biological Gradient: Dose-response Relationship.

Sir Austin states that when we have a dose-response curve *“we should look most carefully at it”*. He uses the example of cigarette smoking. *“The clear dose-response curve admits of a simple explanation and obviously puts the case [for a causal link] in a clear light.”* Therefore a dose-response trend is strongly indicating that it is a causal relationship. Two independent dose-response relationships or a significant dose-response is strong evidence of a causal relationship.

Specificity:

In relation to causation *“One reason, needless to say, is the specificity of the association, the third characteristic which invariably we must consider. If, as here, association is limited to specific workers in particular sites and types of disease and there is no association between the work and other modes of dying, then clearly there is a strong argument for a favour of causation. We must not, however, over-emphasize the importance of the characteristic. Even in my present example there is a cause and effect relationship with two different sites of cancer – the lung and the nose [from smoking]. Milk as a carrier of infection and, in that sense, the cause of disease can produce such a disparate galaxy as scarlet fever, diphtheria, tuberculosis,”*. *“We must also keep in mind that diseases may have more than one cause.”*

Specificity can provide a causal association. However the lack of specificity is not a reason to dismiss a casual disease agent. Disease agents that expose many parts of the body and for example if they are genotoxic, then they damage the cells that they expose, causing a wide range of diseases and cancer in many body organs. Thus it is not appropriate to dismiss the agent being a causal effect for not having a specificity disease effect.

Experiment:

“Occasionally it is possible to appeal to experimental or semi experimental evidence. For example, because of observed association some preventative action is taken. Does that in fact prevent? Here the strongest support for a causal hypothesis may be revealed.”

Of the other four viewpoints each has a potential role and a potential caution.

Plausibility

Biological plausibility can be helpful in supporting a causal relationship. For example, a genotoxic substance causes cancer, mutation and enhanced cell death rates. Sir Austin on this viewpoint states: *“It will be helpful if the causation we suggest is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends on the biological knowledge of the day.”*

The absence of the current knowledge of a biologically plausible mechanism is not an appropriate situation to dismiss epidemiological evidence of a causal effect.

Consistency:

Consistency of the observed association can show a causal relationship. Consistency is shown when the exposure is associated to disease *“by different persons, in different places, circumstances and times.”* Sir Austin uses the smoking example again. In 1964 the US Surgeon-General *“found the association of smoking with cancer of the lung in 29 retrospective and 7 prospective inquiries.”*

“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.”

Coherence:

Coherence relates to the position that *“cause-and-effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease”*. *“Nevertheless, while such laboratory evidence can enormously strengthen the hypothesis and indeed, may determine the actual causative agent, the lack of such evidence cannot nullify the epidemiological observations in man”*.

For example, the lack of understanding that electromagnetic fields and radiation can reduce melatonin, alter cellular calcium ions and damage the DNA, has led to the inappropriate dismissal of epidemiological evidence of cancer by ICNIRP (1998).

Analogy:

“In some circumstances it would be fair to judge by analogy. With the effects of the thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy.”

The strongest analogy for the electromagnetic radiation situation are the parallels with the toxic chemical industry, the nuclear industry and the tobacco industry.

Summary:

If we are to appropriately use epidemiological evidence to protect public health then we must return to the Bradford Hill approach which takes strong support for a causal relationship from the temporality, strength of the association, dose-response relationship and experiment, with some support, only when appropriate, from specificity, consistency, coherence and analogy. Biological plausibility can be helpful but the lack of understanding, and the lack of statistical significance, is no reason to dismiss the epidemiological evidence.

Implications:

The earliest two ELF residential epidemiological studies were themselves of a nature and quality that indicated a causal link between household chronic mean electromagnetic fields and childhood and adult cancer, Wertheimer and Leeper (1979, 1982). Both had highly significant dose-response relationships derived from a careful exposure assessment, and they carefully investigated and eliminated several confounders. The childhood cancer study was confirmed by an independent follow-up study Savitz et al (1988). When the growing evidence in the subsequent 20 years was summarized, Milham (1998), identified over 40 residential studies and 100 occupational studies that showed nearly 500 separate risk ratios. For every one lowered Risk Ratio there are about six elevated Risk Ratios. Milham states that a number of these studies show dose-responses

between magnetic field and cancer incidence. This is definitely causal. Milham also notes that there is now no unexposed group available.

The absence of a non-exposed reference group is termed here the Ubiquitous Genotoxic Carcinogen Effect (UGCE), resulting in grossly lowered Risk Ratios and major under-estimates of the levels of the effects in more recent studies than in the very much older studies.

Fourth Epidemiological Principle:

The Environmental Health Principle: In order to understand the biochemical and biophysical cellular impacts of external agents the internal cellular processes must be understood.

It is well understood that chemicals can interfere with the biochemical processes of cells that can cause cellular damage, DNA damage, altered cellular activity and altered cellular regulation. What is not well understood is that all cells and major organs use electromagnetic signals as part of the cellular signal transduction process, cell-to-cell communication, calcium-ion concentration and regulation, and voltage-gated ion channels in cell membranes. The major vital body organs use electromagnetic signals for the generation, detection and response to processes in the brain, the central nervous system, the heart and the motor neuron system, for example. Therefore it is biophysically plausible that external electromagnetic signals can interfere with and alter internal signals which will alter internal biochemical, neurological and cardiac functions.

Fifth Epidemiological Principle: Ubiquitous Genotoxic Carcinogen Effect:

A ubiquitous agent is one that almost continuously exposes every person in a society because of its extensive household and widespread environmental exposure sources. A ubiquitous genotoxic agent will enhance cancer and many other associated health effects in the general population. This raises the background incidence rate and masks the effects of occupational exposure by raising the rate in the "Non-exposed" reference group and reducing the Relative Risk and significance. The significance is a function of the Relative Risk and the sample size. This results in significant under reporting of occupational effects and inappropriate dismissal of evidence because of widespread lack of understanding and appreciation of this factor, largely based on the ignorance of the agent's genotoxicity and ubiquitous exposures.

The fundamental assumption of environmental epidemiological studies is the use and availability of a not exposed group so that the effect of being exposed can be assessed. An example is given in the following Figures 1 and 2, derived from a WHO textbook on basic epidemiology, Beaglehole, Bonita and Kjellström (1993). Note that both forms of study use a reference group identified as "not exposed".

A case-control study looks back at the time to see what proportion of those who have the disease were exposed and not exposed. They are then compared with a group who do not have the disease and their proportion of being exposed and not exposed, Figure 1. This creates the classical 2x2 analysis and shows the problem of having no non exposed reference groups.

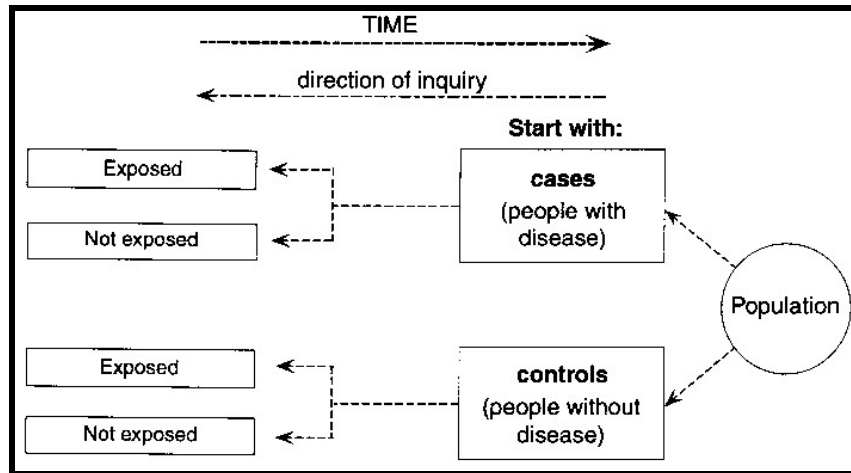


Figure 1: The basic design of a case-control study, Beaglehole, Bonita and Kjellström (1993).

Two-by-Two Tables:

Two common expressions of the comparative incidence is the ratio of ratios, called the Odds Ratio (OR) and the ratio of rates called the Relative Risk or Risk Ratio (RR). This is set out as a 2x2 table where the exposed group who have the disease are termed group A, and those who have been exposed but do not have the disease are group B. Group C were not exposed but do have the disease, while Group D were neither exposed nor have the disease, Table 1.

Table 1: The classic 2x2 table format for a case-control study.

		Disease Status		Total
		Yes	No	
Exposure Status	Yes	A	B	A+B
	No	C	D	C+D
		A+C	B+D	N

The Odds Ratio is given by: $OR = (A/B)/(C/D) = AD/BC$

The proportion of all the people who have been exposed and have the disease is $A/(A+B)$ and the proportion of the not exposed people with the disease is $C/(C+D)$.

For the Relative Risk is given by: $RR = (A/(A+B))/(C/(C+D))$

For example: If $A = 30$, $B = 970$, $C = 15$ and $D = 985$.

Then $OR = AD/BC = 30 \times 985 / (15 \times 970) = 2.03$, and
 $RR = (A/(A+B))/(C/(C+D)) = (30/(1000))/(15/1000) = 2.0$

The most used approach to calculate the statistical significance level of the increase in disease is a 95% proportional effect allowing a 5% or 1:20 being random and 19:20 that the effect is not random but is significantly confidently causal. This is normally expressed as a confidence interval around the incidence Relative Ratio for which there is a 95% occurrence of the effects levels in the range. This corresponds to a p-value of $p=0.05$. If the p-value is larger than 0.05 it is not significant and if it is 0.05 or less it is statistically significant.

For RR the 95% confidence interval (95%CI), range that contains 95% of the probability, is calculated with the following formula:

$$95\% \text{ CI} = \exp [\log_e \text{RR} \pm 1.96 \sqrt{(1/A + 1/C)}]$$

Using the example figures, $95\% \text{CI} = \exp[\log_e(2.0) \pm 1.96 \sqrt{(1/30+1/15)}]$
 $= \exp[0.693 \pm 1.96 \sqrt{(0.10)}] = \exp[0.693 \pm 0.620] = 1.08-3.72$

Hence $\text{RR} = 2.0 (1.08-3.72), p<0.025$.

For example, if the assumed not exposed people with disease were actually exposed to $1/5^{\text{th}}$ of the disease agent levels, as an example of the Ubiquitous Exposure Effect. The exposed group has 15 more disease cases, so $1/5^{\text{th}}$ is 3 cases. If the not exposed group has 3 cases removed from C it drops from 15 to 12. Then:

$$\text{RR} = 2.50 (1.29-4.85), p=0.005.$$

If the 3 cases with the disease because of the exposure are moved into A then:

$$\text{RR} = 2.75 (1.43-5.29), p=0.0016$$

This shows how the failure to accept in the Ubiquitous Exposure Effect can significantly underestimate the strength of the effect through reducing the Relative Risk and the p-value, using EPINFO 6 software.

Cohort Studies:

It is clear from Figure 2 that for an assumed not exposed group which is actually exposed, the results will be significantly underestimated from the impact of exposure. In a cohort study a particular population of people without the disease are subsequently found to have an internal group that was exposed to a substance that the majority of the group were not.

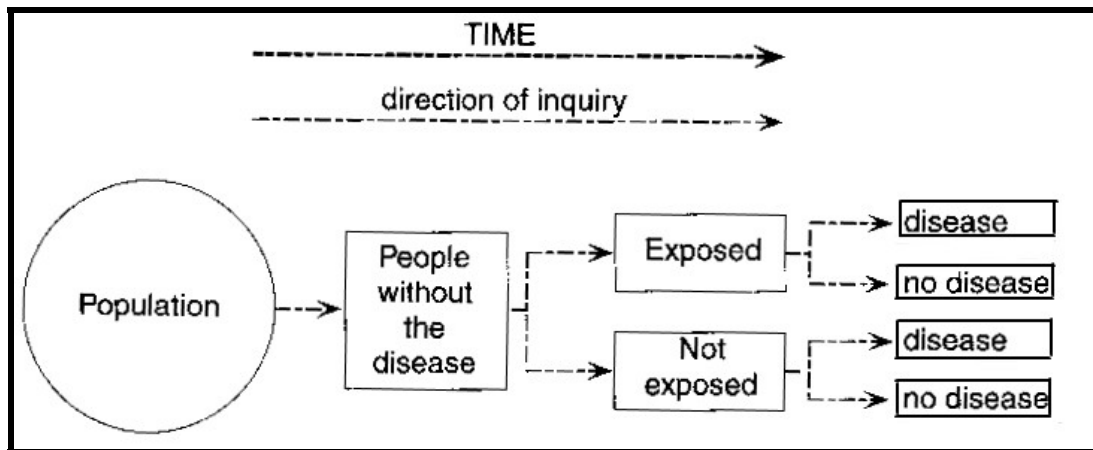


Figure 2: The basic design of a cohort study, Beaglehole, Bonita and Kjellström (1993).

By then investigating the rate of disease in the exposed group compared with the rate of disease in the remainder of the group who were not exposed, the effect of exposure can be evaluated. Initially it appears from Figure 1 that because both the case- and control- groups are compared between exposed and not exposed groups that the effect of exposure on the assumed not exposed group, could be being dealt with. However, because there is a wide range of residential and urban levels of exposures to the ELF & EMR fields, this can vary greatly from individuals and groups. This produces significant confounders in these studies.

As it has demonstrated above, the failure to adequately deal with a ubiquitous agent will place a proportion of the exposed group in the not exposed group and grossly underestimate the effects of a disease agent. Hence a more appropriate exposure assessment is vital.

When dealing with a ubiquitous agent such as electromagnetic fields and radiation, adequate exposure assessment requires extensive understanding and the development of careful methods. Otherwise, results will be very misleading through underestimating the impact of the exposure and ignoring the exposure factors within the control groups. An important factor in cohort studies is matching the characteristics of the not exposed group as much as possible to those of the exposed group so that the only difference is exposure, not age, sex, ethnicity nor income for example.

Statistical Methods:

Even though Sir Austin said that significant statistics are not required for deciding a causal relationship he agreed that they showed the play of chance and show the magnitude of the RRs. Hence an appropriate use of statistical methods is helpful.

One-tailed and two-tailed distributions:

The normal statistical method uses a one-tailed distribution test for a one direction effect and a two tailed test for a two directional effect. If you are seeking to determine whether a disease agent is going to increase an incidence of the disease then a one-tailed test is appropriate. If the influence of the agent is unknown then a

two-tailed test is appropriate. The usual 95%CI method used in the 2x2 calculation almost universally uses a two-tailed test even though it is being used for a one directional assessment in many cases. This raises the threshold of significance considerably.

The value of 1.96 is the number of standard deviations across a distribution that contains 95% of the data with 2.5 % above and below the defined interval. This gives the 95% confidence interval for a two-tail distribution. If a one tail distribution is chosen because it is a one-direction effect, then the lowest limit is 1.65 standard deviations and the upper limit is infinity. Hence for a 1-tail 95% confidence Interval lower limit is

$$95\% \text{ CI} = \exp [\log_e \text{RR} - 1.65 \sqrt{(1/A + 1/C)}]$$

For most studies the limiting factor in reaching the significance level is the small number of the incidence populations (A or C). This is illustrated by the following example:

A study involves two groups of $N_p = 1000$ person-years. One group is exposed and one is not exposed but is similar in all other aspects. The biological effect is measured in 24 of the exposed group and 12 of the unexposed group. The statistical analysis using the EPI6 program is

$N_p = 1000$ $N = 24$ $\text{RR} = 2.00$ $95\% \text{CI: } 1.01 - 3.98$ $p = 0.044$ $< 0.05^*$

It is significant because $p < 0.05$. If a 1-tail test is applied the lower 95%CI limit is 1.42 compared with 1.01, with $p < 0.001$.

The sample size has a very significant effect on the significance. It is shown in Table 2 that the 500 group has a 95%CI range of 4.53, the 1000 group 2.97 and the 2000 group 2.01.

Using a 2-tailed method, if we halve or double the total sample data with the same incidence rate, then:

Table 2: A sample of 2x2 statistical results of Relative Risk, 95% Confidence Interval, the p-value and rounded significance level, Using EPI6 software from the U.S. Centre for Disease Control. N_p is the group size (p-yrs), N_e the disease incidence in the exposed group, N_c the disease incidence in the control group which also contains a total of N_p .

N_p	N_e	N_c	RR	95%CI	p-value	Significance
500	12	6	2.00	0.76 - 5.29	0.154	n.s.
1000	24	12	2.00	1.01 - 3.98	0.044	$< 0.05^*$
2000	48	24	2.00	1.23 - 3.24	0.004	$< 0.01^{**}$
3000	72	36	2.00	1.34 - 2.97	0.0005	$< 0.001^{***}$

Sir Austin Bradford Hill warns against relying on statistical significance when considering the human health effects of epidemiological studies, Hill (1965). The results set out in Table 2 illustrate the primary role in significance of the sample

size. All have the same RR value indicating a doubling of the disease rate. The only factor that is changed is the sample size. When each group contains 500 people the result is insignificant, with 1000 it is significant ($p < 0.05$), with 2000 it is highly significant ($p < 0.01$) and with 4000 it is very highly significant. Many researchers appear to fail to understand this important aspect of statistics and the first result would be described as showing no effect because it was not a significant effect. This is an inappropriate statement if the sample size is small. With small samples it is mathematically difficult to achieve statistical significance. Another dismissive bias can occur when there is a small sample but the RR value is so high that it achieves statistical significance. Many people then dismiss the result as unlikely because of the small sample size. In fact, it is a highly significant result with a wide confidence interval only because of the small sample size.

Further Under-estimation in EMR/EMF Epidemiology:

There are two major factors that lead to almost all EMR/EMF epidemiological studies significantly underestimating the relative health effects of EMR/EMF exposures in residential and occupational studies. These factors are the ubiquitous exposure of the whole population in developed countries and the Healthy Worker Effect.

Healthy Worker Effect:

Because employed workers are on average much younger than the average whole population, and because many employment situations require a level of health and fitness, especially in uniformed public service groups such as the police, fire fighters and the military, there is a well understood and accepted Healthy Worker Effect. Beaglehole, Bonita and Kjellstrom (1993) describe this as "*an important selection bias*", because the "*working population has a lower total morbidity and mortality than the population as a whole*". They also state that "*rates among health workers are 70-90% of those in the general population.*" In fact in some circumstances that involve younger than average workers the rates can be lower than 40-50% of the general population. Lilienfeld et al. (1968) Table 5.1 reports that the average mortality rate of male US Embassy staff, employed by the State Department, was 43% of the mean US mortality rate, and 39% for non-State Department employees.

For example, the relationships outlined in Table 2 illustrating the effect of sample size on the significance level by adjusting the data for the Healthy Worker Effect, Table 3. The influence of a 33% and 50% effect can be shown by reducing the reference number by these percentages. For example if the control group has 4 diseases rather than 6 in the example, allowing for a 33% healthy worker effect. All of the RR values rise to 3.0, the lower levels of the 95%CI rise significantly taking the smallest group with $N=12$ to a p-value of 0.044. All other p-values take on the value in the larger group above except the $N=72$ group which has $p < 0.0000008$.

Table 3: A sample of 2x2 statistical results of Relative Risk, as for Table 2, with the Healthy Worker Effect Adjustment, expressed as a % reduction adjustment to the control group population.

HWE(%)	N _p	N _e	N _c	RR	95%CI	p-value
0	500	12	6	2.00	0.76 - 5.29	0.154
33	500	12	4	3.00	0.97 - 9.24	0.044
50	500	12	3	4.00	1.14 - 14.09	0.019
0	1000	24	12	2.00	1.01 - 3.98	0.044
33	1000	24	8	3.00	1.35 - 6.65	0.0094
50	1000	24	6	4.00	1.64 - 9.74	0.00093
0	2000	48	24	2.00	1.23 - 3.24	0.004
33	2000	48	16	3.00	1.71 - 5.26	0.000055
50	2000	48	12	4.00	3.13 - 7.51	0.0000028
0	3000	72	36	2.00	1.34 - 2.97	0.0005
33	3000	72	24	3.00	1.90 - 4.75	0.0000008
50	3000	72	18	4.00	2.39 - 6.69	<0.0000001

Dose-response trend lines:

“The demonstration of a clear dose-response relationship in unbiased studies provides strong evidence for a causal relationship between exposure or dose and disease”, Beaglehole et al. (1993). In epidemiology, it is the clear dose response trend that is important, not the statistical significance. For example a 3-point trend has only one degree of freedom which makes statistical significance almost impossible to achieve, but a clear rising trend shows a clear causal relationship.

If a statistical approach is taken then for a dose-response trend it may be appropriate to use a 1-tail significance test if the hypothesis is looking for a rising trend. A conservative approach is to use a 2-tail significance test because there are points above and below the line. The difference is in the P-value which for a 1-tail test is half of the P-value calculated for a 2-tail test. Plotting a dose response trend using Excel software provides the analysis of the squared correlation coefficient (r^2) as a measure of the significance of trend. (r^2) is the statistical calculation of the variance explained and associated with the trend line. The mathematical relationship between the t-test and the correlation coefficient involves a number of degrees of freedom (df), which is the number of points (N) minus 2, (df = N-2).

$$t = r \sqrt{[(N-2)/(1-r^2)]} \quad (1)$$

Table 4: An example, with a trend involving 12 points, $df = 10$. Using a table for the t-test from a statistical text book shows the following values of the p-value for a 2-tailed t-test:

	P-value			
	0.05	0.02	0.01	0.001
t-value	2.228	2.764	3.169	4.587
r^2	0.3317	0.4331	0.5011	0.6778
r	0.5760	0.6581	0.7079	0.8233

Three examples of dose-response trends are given in Figures 3-5, with a progressive high variation reduced steps.

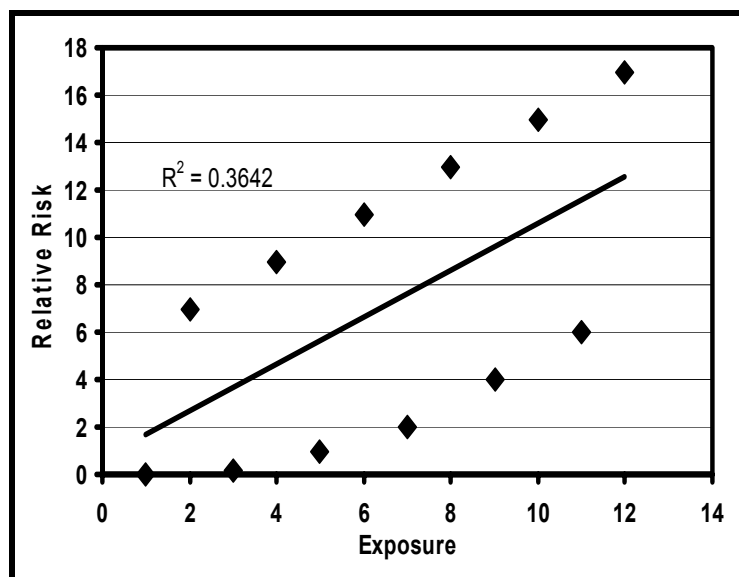


Figure 3: Dose-response trend for 12 points with a typical relative risk variance of 5.

In Figure 3 the $r^2=0.3642$ for the fitted trend line. This gives a t-test value of $t=2.393$, which is above the threshold test for $p=0.05$ at $t=2.228$. Therefore the significance of the trend is $p<0.05$.

The $r^2=0.5042$ which is a t-value of $t=3.189$, which is above the threshold test for $p=0.01$ at $t=3.169$. Therefore the significance of the trend is $p<0.01$.

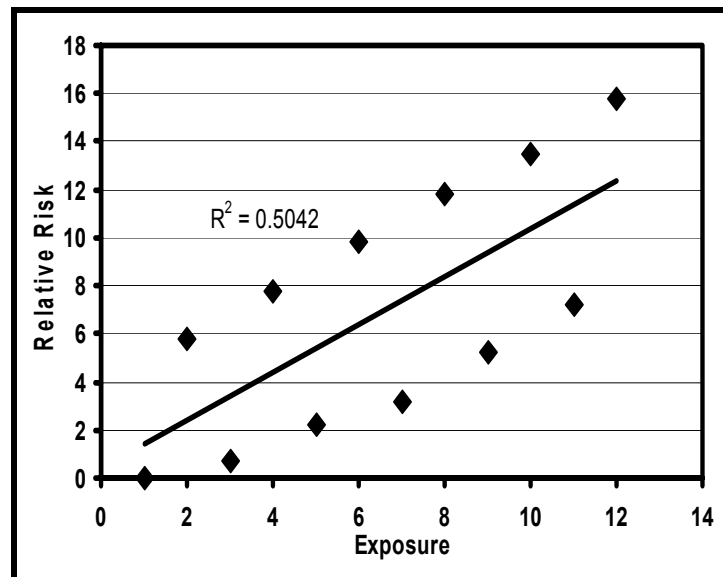


Figure 4: Dose-response trend for 12 points with a typical relative risk variance of 3.8.

On Figure 5 the trend r^2 value is 0.9306 which has a t-value of $t=11.58$, which is just above the threshold test for $p=10^{-8}$ at $t=11.56$. Therefore the significance of the trend is $p=10^{-8}$. Figures 3 and 4 show that the pattern is very similar, the gradient is the same (1.0), the only difference is the variance of the data points.

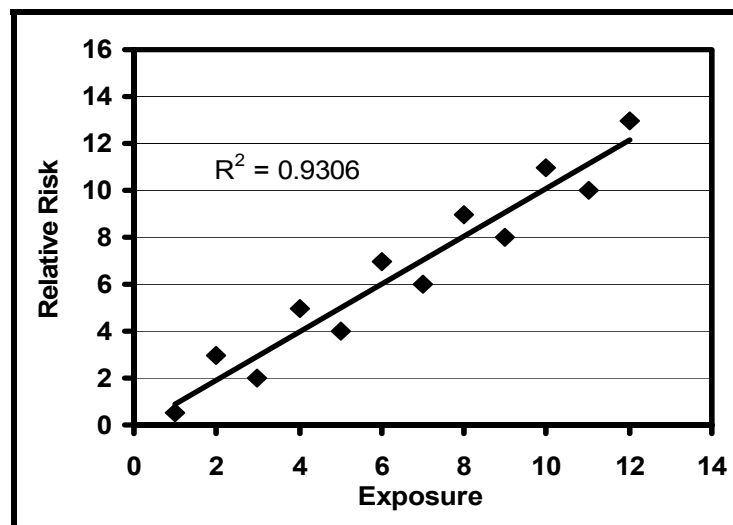


Figure 5: Dose-response trend for 12 points with a typical relative risk variance of 1.0.

Most residential and occupational epidemiological studies have 3 to 5 levels giving the degrees of freedom, $df=1$ to 3. For the t-test threshold for $p=0.05$ for $df=1$ is $t=12.706$ and for $df=3$, $t=3.182$. If the data points are less than 5 then it is probably appropriate to use the $p=0.1$ threshold, with $t=6.314$ for $df=1$; $t=2.920$ for $df=2$ and $t=2.353$ for $df=3$.

Occupational Risk Assessment:

If an occupational group is studied and a Relative Risk is determined then the probability that a given individual worker who has the disease obtained it from the occupational exposure is given by the RR. The RR is the ratio of the incidence of the disease in the exposed group vs the unexposed group. Hence if the $RR = 1$ then the disease rate is the same as for the average population. If the $RR = 2$ then the exposure has doubled the disease rate and half of the people are highly likely to have got their disease from the occupational exposure. Thus for every person the risk is 50% if no other risk factors are known. The Risk (%) = $100 (RR-1)/RR$.

Table 5: The percentage Risk for individuals having received their disease from an occupational disease agent as a function of the RR.

RR	1.5	2.0	3.0	4.0	5.0	6.0	7.0	8.0	9.0	10.0	100.0
Risk (%)	33.3	50	66.7	75	80	83.3	85.7	87.5	88.9	90	99

Hence in a given occupational situation of similar exposure levels the "balance of probabilities" or more "likely than not", requires a Relative Risk of more than 2.

On the other hand, if the epidemiological study was carried out for workers with above average exposures and the individual being considered had a higher than average exposure in the study, then the higher exposure worker would have a higher than 50% chance of receiving the disease from the occupational exposure. Dose-response trends show that the higher exposure groups sickness rates can easily be 2 to 3 times higher than the medium exposed workers.

Hence an individual occupational risk assessment involves a consideration of the Relative Risk in a given exposure situation and the exposure situation of the person being assessed.

The Value of Integration:

Classical science provides guidance to interpret a large body of complex data in this area of research. The scientific method is largely based on conceiving, stating and testing a hypothesis. This is often aimed at identifying patterns and relationships. The statement of an *a priori* hypothesis that makes sense of the data, predicts outcomes and may be confirmed by new observations is used in both reductionism and integrative science. In modern public health methods the integrative science approach is much more appropriate with the extensive research from *in vitro* and *in vivo* laboratory experiments and extensive epidemiological research.

This is the approach supported and urged by Sir Austin Bradford Hill. It is vital to recognise and accept in public health and environmental epidemiology that there is no ethically acceptable way of proving a serious and/or fatal human health effect by carrying out direct experiments. Hence a sound assessment method must be used when attempting to protect people from serious disease and death. Cancer, motor neuron disease (ALS), heart attack, miscarriage and suicide are at the centre of this consideration.

Multiple studies showing elevated Cancer, MND, Heart Attack, Miscarriage and Suicide support a causal relationship between terminal and fatal human health effects and the disease agent under consideration. Multiple significant elevation and consistent elevation is strong evidence of a causal relationship. Dose-response evidence generally puts the question of causation well beyond reasonable doubt. This is based on the approach recommended by one of the world's most eminent environmental epidemiologists, Sir Austin Bradford Hill, Hill (1965).

Biophysics Principle: EMR Spectrum Principle:

The EMR Spectrum Principle is that the higher the carrier frequency, then the higher the biological and health effects will be. It is very well established that the higher the carrier frequency is, and the shorter the wavelength is, and the higher the water content is, then the faster the RF/MW signal gets absorbed and the higher the induced electric field, the conductivity and the induced electric current is.

It is observed that both biological effects and epidemiological effects appear to be the same or very similar from ELF exposure and from much lower RF/MW intensity exposures, including calcium ion efflux, melatonin reduction, DNA strand breakage, chromosome aberrations, leukaemia, brain cancer, breast cancer, miscarriage and neurological effects.

The dielectric constant is approximately the AC equivalent of the DC Resistance. As the dielectric constant decreases the conductivity increases. The dielectric properties of biological tissue depend on the water content because of the interaction of the RF/MW signal with the tissues. Two types of effects control the dielectric constant frequency dependence. One is the oscillation of the free charges or ions and the other the rotation of the molecules at the frequency of the applied electromagnetic signal, Johnson and Guy (1972). This results in a progressive reduction in the dielectric constant with rising frequency of the electromagnetic signal, Figure 6.

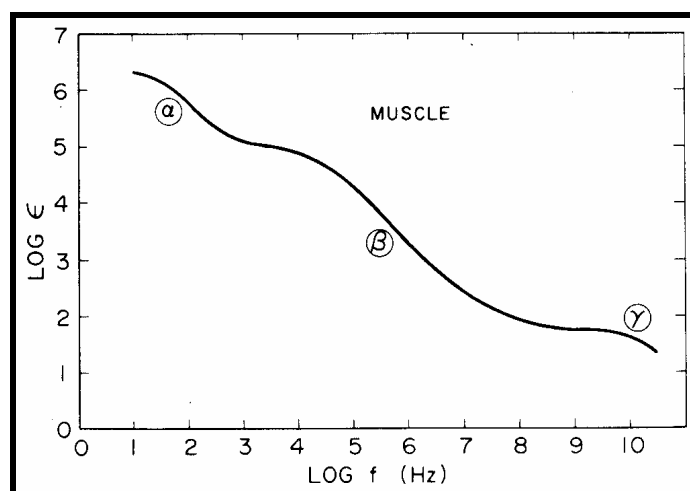


Figure 6: The dielectric constant of muscle as a function of frequency, Schwan and Foster (1980).

The significant drop in dielectric constant with increasing frequency shows a linked process across the spectrum with increasing conductivity and higher induced currents as the frequency rises, Vignati and Giuliani (1997), Figure 7.

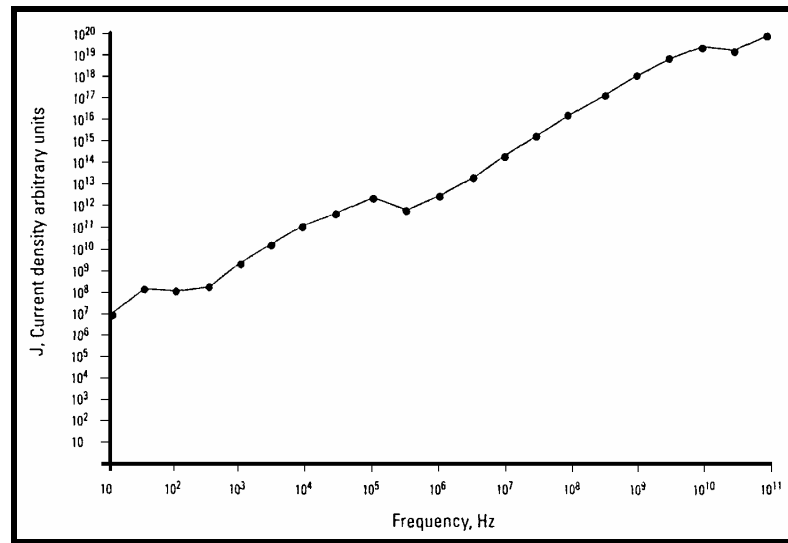


Figure 7: Capacitive induced current density in a toroid of human muscle tissue of unitary radius, exposed to a unitary magnetic field induction, Vignati and Giuliani (1997).

Figures 6 and 7 are consistent with data presented by Johnson and Guy (1972), Tables 6 and 7.

Adey (1988) Figure 8, shows that a 56V/m ELF field induces a tissue gradient of 10^{-7} V/cm. Whereas a 56V/m 147MHz signal, modulated by the same spectrum range of ELF fields, induces a tissue gradient of 10^{-1} V/cm, a million times higher. This is close to the factor given by Figure 7 between 16Hz and 147MHz.

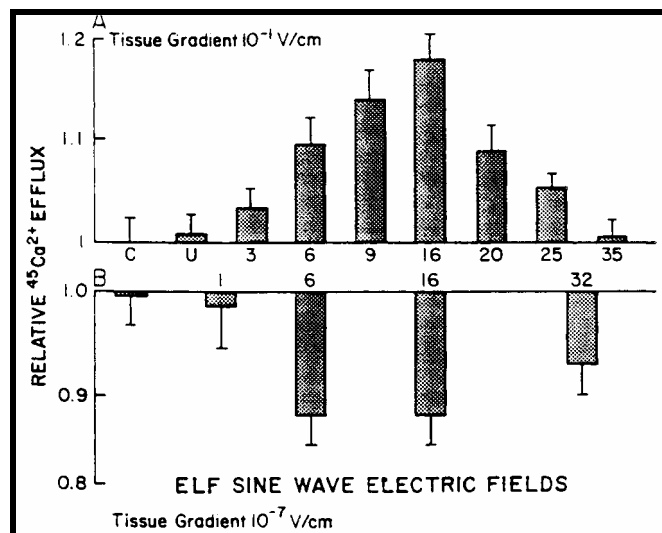


Figure 8: Relative Ca^{2+} efflux (positive and negative) from isolated chick cerebral hemisphere exposed to (A) weak RF field (147 MHz, 0.8 mW/cm^2 , 56 V/m in air), amplitude modulated at low frequencies (abscissa) and (B) ELF electric field (56 V/m in air) over the same ELF modulation frequency, Adey (1988). The tissue gradients differ by 10^6 between A and B.

**PROPERTIES OF ELECTROMAGNETIC WAVES IN BIOLOGICAL MEDIA,
(Johnson and Guy (1972))**

TABLE 6: Muscle, Skin, and Tissues with High Water Content

Frequency (MHz)	Wavelength in Air (cm)	Dielectric Constant ϵ_H	Conductivity σ_H (mho/m)	Wavelength λ_H (cm)	Depth of Penetration*
1	30000	2000	0.40	436	91.3
10	3000	160	0.63	118	21.6
27.12	1106	113	0.61	68.1	14.3
40.68	738	97.3	0.69	51.3	11.2
100	300	71.7	0.89	27.0	6.66
200	150	56.5	1.28	16.6	4.79
300	100	54	1.37	11.9	3.89
433	69.3	53	1.43	8.76	3.57
750	40	52	1.54	5.34	3.18
915	32.8	51	1.60	4.46	3.04
1500	20	49	1.77	2.81	2.42
2450	12.2	47	2.21	1.76	1.70
3000	10	46	2.26	1.45	1.61
5000	6	44	3.92	0.89	0.79
5800	5.17	43.3	4.73	0.78	0.72
8000	3.75	40	7.65	0.58	0.41
10000	3	39.9	10.30	0.46	0.34

λ_H : The wavelength inside the high water content tissue; * Depth of penetration is based on the depth at which the intensity drops to the level of $1/e$, i.e. about 36.8%.

TABLE 7: Fat, Bone, and Tissues with Low Water Content

Frequency (MHz)	Wavelength in Air (cm)	Dielectric Constant ϵ_L	Conductivity σ_L (mmho/m)	Wavelength λ_L (cm)	Depth of Penetration
1	30000				
10	3000				
27.12	1106	20	10.9-43.2	241	159
40.68	738	14.6	12.6-52.8	187	118
100	300	7.45	19.1-75.9	106	60.4
200	150	5.95	25.8-94.2	59.7	39.2
300	100	5.7	31.6-107	41.0	32.1
433	69.3	5.6	37.9-118	29.8	26.2
750	40	5.6	49.8-138	16.8	23.0
915	32.8	5.6	55.6-147	13.7	17.7
1500	20	5.6	70.8-171	8.41	13.9
2450	12.2	5.5	96.4-213	5.21	11.2
3000	10	5.5	110-234	4.25	9.7
5000	6	5.5	162-309	2.63	6.7
5900	5.17	5.05	186-338	2.29	5.2
8000	3.75	4.7	255-431	1.73	4.6
10000	3	4.5	324-549	1.41	3.4

λ_L : The wavelength inside the low water content tissue;

These biophysics relationships shown in tables 6 and 7 of tissue parameters and the carrier frequency were published in 1972 by two leaders in this area showing how they vary in low and high water tissues.

Implications of the failure to appreciate the EMR Spectrum Principle:

There is strong evidence of consistent effects across the spectrum and strong support for the biophysical evidence and principles of higher and higher carrier frequencies induced higher and higher currents induced in tissues. Therefore effects found from ELF fields are much more likely to occur at much lower mean intensities with exposure to RF/MW fields. There are over 150 epidemiological studies of ELF health effects and very fewer, less than about 25 RF/MW epidemiological studies. The RF/MW studies do show the many similar health effects but the EMR Spectrum Principle shows that all ELF associated health and biological effects are strongly likely to be associated with very low residential RF/MW exposure levels, including around radio, TV and cell phone towers.

Epidemiological evidence supports the EMR Spectrum Principle:

There is robust and extensive data supporting the EMR Spectrum Principle. While this paper is primarily about RF/MW exposures, confirmation of adverse effects is given by studies that involve mixed and ELF exposures. Astrocytomas are a subgroup of Gliomas (Brain Cancers). A group that was chronically exposed to high ELF fields in electrical utility occupations developed a high rate of Astrocytomas, Theriault et al. (1994).

$$\text{OR} = 28.48 (1.76-461.3)$$

In a 16-year data set of childhood cancer in the vicinity of the Sutro Tower derived from Selvin et al. (1992), a powerful radio and TV tower in San Francisco, out to a distance of 1 km the mean personal exposures are about $0.1\mu\text{W}/\text{cm}^2$. Three brain cancers occurred in a population of about 894 children. This gives:

$$\text{RR} = 15.5 (3.14-76.8), p=0.004$$

Within 500m of the tower there were 2 brain cancers within a population of about 144 children. Their estimated mean personal exposure was about $0.4\mu\text{W}/\text{cm}^2$. This gives:

$$\text{RR} = 64.2 (10.8-382), p=0.00103$$

Zaret (1977) reports that in a group of 18 workers who were servicing microwave communication equipment there were 2 with Astrocytoma. Allowing for a 10-year exposure and cancer development period, this gives an incidence rate of 1111 per 100,000p-yrs and a relative risk of:

$$\text{RR} = 1634 (385-6939), p<0.0000009$$

This shows that RF/MW radiation exposure produces very high increased rates of brain cancer. It is even higher for residential exposure levels for childhood brain cancer than compared to high ELF exposures in electrical occupations.

The EMR Spectrum and the data supporting it give robust support for the hypothesis is that electromagnetic radiation and ELF fields are a Universal Genotoxic Carcinogen.

It is established in toxicology that a genotoxic substance has no safe threshold level because the damage occurs cell-by-cell. This statement is contained in the UK Royal Commission on Environmental Pollution, Report No. 23, Setting Environmental Standards, UKRCEP (1998).

For genotoxic carcinogenic substances the safe threshold is zero and the approach that should be taken with genotoxic carcinogens is the *de minimis* approach.

Electrical and electronic workers are regularly and chronically exposed to elevated ELF fields and multiple independent epidemiological studies show significantly elevated brain cancer. The EMR spectrum principle predicts that cell phone users, regularly and chronically exposing their heads and bodies to radio-frequency/Microwave radiation will have highly elevated brain cancer incidence. It has already been shown that analogue cell phone users in Sweden have significantly increased the risk of brain cancer. For Astrocytomas in the temporal or occipital areas, OR=9.00 (1.14-71.0) based on 12 cases and 5 controls, Hardell et al. (2002). The EMR Spectrum Principle, along with the evidence of genotoxicity, shows that the risk of brain cancer, and many other health effects, is elevated in populations living within the vicinity of cellphone base station antennas.

Exposure Assessment:

Exposure assessment is vital for reducing or removing potential confounding factors. It is also vital to know whether for cited studies particular parts of the body were exposed or whether the whole body was exposed to the disease agent. For example for electrical occupations, residential powerline situations, radio and radar exposed commercial broadcast and military situation, far field and whole body exposures are dominant. For cell phone usage the head has been the primary target with whole body and specific organ exposures depending on the mode of use of the phone and its location when calls are not in progress. The older bag-phones had a remote aerial and separate handset, whereas modern digital phones have the aerial built into the handset.

A classical occupational exposure assessment involves detailed surveys of particular occupational positions or activities and then assessing a statistical frequency distribution of particular people or particular job descriptions in order to make careful estimates of the personal or group exposure levels. For example Robinette et al. (1980) studied military personnel involved in repairing or using radio and radar equipment on U.S. Naval ships during the Korean War. Van Wijngaarden et al. (2000) and Savitz et al. (2000) used the job exposure matrix method to assess personal exposures in electric utility workers, and refined it by using personal exposure meters for over 2800 workers.

For EMR the mean operational exposure level is a strong function of proximity or distance from the source. For occupational and military exposed to toxic substances that cause cancer it is the long-term mean exposure over months or years that related to the cancer rate because the mechanism involves cumulative cellular damage. Sometimes intermittent high peak exposure dominates the daily or monthly average, whereas in other situations the ubiquitous residential exposure can be dominant.

Exposure Comparison Example:

For example, an occupation exposure to a 2 minute peak exposure of 1000 units, with two secondary exposures of 200 units for 5 minutes and 8 hours exposure to 10 units with a mean ambient exposure of 0.5 units for 16 hours. The mean daily exposure is 6.44 units. Alternatively another occupation as longer middle exposure of 50 units for 4 hours, 4 hours at 2 units and an ambient exposure of 0.02 units for 16 hours. This has a daily mean exposure of 8.7 units. A repairman who has 2 hours of 1000 units, 4 hours of 100 units, 2 hours of 10 units and 16 hours of 1 unit, has an index of 101.5 units. The first example is typically for a military radar repair technician on a ship, the second is typical of a TV transmission station maintain technician. whereas the third is a pre-1980 radar repair person working much of the day near active radars high powered radars. A heavy cellphone user, on the phone for 3 hrs/day at 800 units, in an office with mean exposures of 10 units for 5 hours and an ambient exposure of 0.02 units for 16 hours, has a mean daily exposure index of 102.1 units. Cellphone usage produces localized head area mean exposures that are comparable with radar repair technician's whole-body exposure.

The examples above are typical high exposure days. Most days in occupations would have much lower or less high level exposures and many weeks are spent away from the moderate to high RF exposure situation. Hence their annual mean exposure is typically less than 10 times lower than the high exposure day levels.

Heavy cellphone users tend to have a dominant usage during the weak with continued but lower time-length usage over the weekend and during vacations. However, any weekend or vacation usage is exposing the user's head to "radar-like" exposure intensity levels.

Interaction of electromagnetic fields and radiation with the Human Body:

When considering the epidemiological evidence related to the health effects of electromagnetic fields and radiation it is essential to have a comprehensive understanding of the nature and characteristics of the fields and radiation patterns and intensities. There is a wide range of interactions with body tissues and parts depending on the frequency of the oscillating fields. The initial biophysical interaction of low frequency fields is to induce electric field gradients within the body, which induces an electric current which seeks to flow to "earth". This is because voltage is the electromotive potential raised above the earth's value. Therefore their release of electrical energy is produced by the current flowing back to earth. The human body is a conductor, largely made of water, so it provides a pathway for the electric current to flow to earth. The same thing happens when exposed to radio-frequency fields, but, as shown by the EMR Spectrum Principle,

for a given external field intensity the induced internal currents are much higher from the higher carrier frequencies.

Melatonin, produced by our body's pineal gland, especially at night, is a very highly potent antioxidant substance. Because of the EMF reduced melatonin mechanism, Reiter and Robinson (1995), and with young children's melatonin levels being low, additional reduction by ELF fields at night could create a large impact on cancer risk associated with the mean bedroom exposure. For some children the proximity to power lines will dominate this magnetic field exposure. Other children live further away from power lines but sleep in higher fields because of the configuration of the household wiring. For example, the child's bed is on the outside wall of the house, near to where the power cable enters the house or is joined to the power switch and fuse board, with fields in the range 4 to 12mG.

Power frequency (ELF) fields:

Power frequency (50/60 Hz, 1 Hz = 1 cycle per second) electric and magnetic fields are produced around wires, appliances and equipment powered by electricity. The electric field (E, V/m) is proportional to the voltage (V, volts) and the magnetic field (B) is proportional to the current (i, Amperes). B, in milliGauss [mG] or microTesla [μ T]; 10 mG = 1 μ T. In Figure 1 this is illustrated by showing the electric fields produced when devices are plugged in and the voltage is applied. The magnetic fields are added when the switch is turned on and the current flows to operate the appliances, Figure 9.

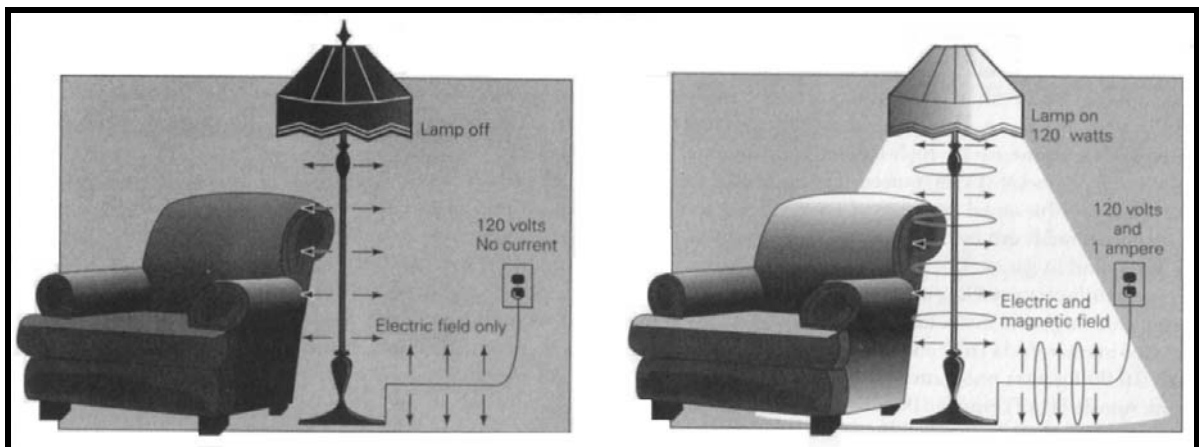


Figure 9: The electric field results from being plugged in and a voltage is applied between the plug and the "end" of the supplied device, left. When the device is turned on the oscillating at 50/60 Hz current flows, creating an electromagnetic field, Levitt (1995).

For power supplies these fields carry their energy in an oscillating current that produces oscillating fields of 50 Hz or 60 Hz. Some European electric train and tram systems use 16.7 Hz. In North America household supplies are 60 Hz and 120 V, whereas in the U.K., Europe, Australia and New Zealand, 50 Hz and 240 V power supplies are used. This shows that the North American supplies are associated with higher currents and higher magnetic fields for a given power requirement.

Almost all residential studies of electromagnetic exposures find health effects closely correlated with the magnetic fields produced by mean electric currents, and hence Wiring Current Configuration Codes or mean long-term exposures, are useful and more appropriate than spot measurements following diagnosis. There is strong evidence that ELF fields are genotoxic. This means that chronic mean cumulative exposures are correlated with chronic diseases. Another important biological fact is that sleeping within an electromagnetic field reduces the melatonin levels, significantly altering the antioxidant protection provided by Melatonin. For this reason nocturnal fields are more highly correlated with some health effects.

Some questions have been raised about the appropriateness of spot measurements and estimates of mean chronic exposures, such as Wiring Codes. In a San Francisco residential ELF associated Adult Glioma study Wrensch et al. (1999) measured residential fields and found that they correlated very well with the Wertheimer-Leeper and more weakly with the Kaune-Savitz Wire Codes.

Wertheimer-Leeper Wire Code from very low to very high					
	VLCC	OLCC	OHCC	VHCC	
Magnetic Field (mG)	0.6	1.4	2.1	2.7	Trend $p < 0.005$
Kaune-Savitz Wire Code					
	Low	Medium	High		
Magnetic Field (mG)	1.2	1.6	2.6		Trend $p = 0.055$

The Kaune-Savitz Wire Code produces a medium field strength that is rather close to the Low field, with the Low field being close to the Ordinary Low not the Very Low fields of the Wertheimer-Leeper Wire Code. The Wertheimer-Leeper Wire Code is more linear in assessing the values of the range of fields.

All homes have low level ELF fields throughout rooms, with higher local fields near some appliances, especially electric motors such as in vacuum cleaners, hair driers, sewing machines, drills, lathes, washing machines and clothes driers. All offices, shops, schools, hospitals, factories, and all other buildings with electric power supply connections have ELF fields, including 50% of readings being greater than 2mG along roads. In electric trams the median was 10.3mG, Lindgren et al. (2001). Cars have ELF fields from equipment and from the rotating steel belted radial tires Milham, Hatfield and Tell (1999).

This creates the ubiquitous ELF exposure situation. The relative personal mean magnetic field strengths for children living or going to school near high voltage power lines were measured in Norway, Figure 10, Vistnes et al. (1997).

Inside 30m from a 300kV power line the fields rose from 5 to 12mG. Living near the power line (60m) produced 2 to 20mG, averaging 4mG. The home 175m from the power line ranged from 0.3-0.8mG, averaging 0.6mG. At the school near the power line the child's fields ranged from 5 to 90mG, averaging 15mG. The school away from the power line (300m) produced personal fields in the range 0.1-2mG averaging 0.2mG. This illustrated the influence and range of internal and external magnetic field sources.

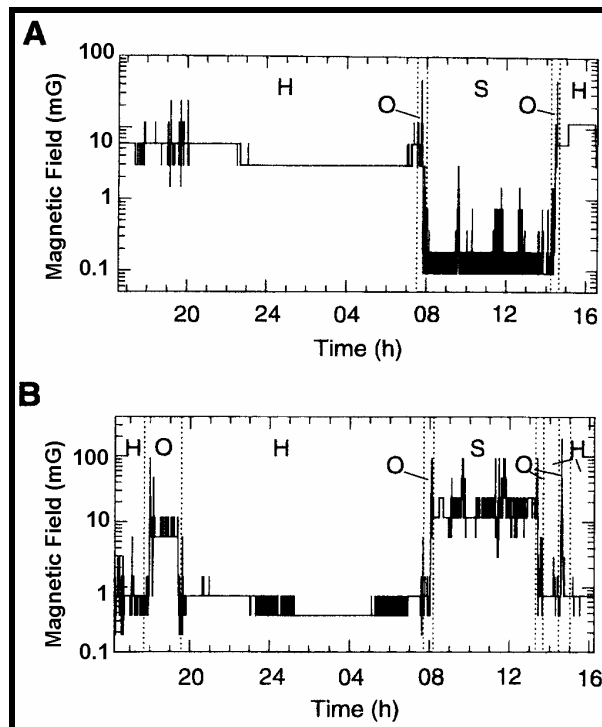


Figure 10: Examples of magnetic field recordings for child A, living 60 m from the 300 kV powerline and attending a school 300 m from the powerline, and child B who lived 175 m from the powerline and attended a school 25 m from the powerline, Vistnes et al. (1997). **H**: Home, **S**: School and **O**: Other.

In both cases the time at home is more dominant than the time at school. However, in the case of a school near the powerline, the child's average exposure by going to the school is significantly higher.

Around high voltage powerlines the electric field is more constant as it relates to the supply voltage which is more constant. The magnetic fields are more variable because they vary as the current changes to deal with the varying electric power loads being supplied. The following table gives some examples of magnetic field intensities as a function of distance from high voltage power lines in the US.

Table 8: Magnetic fields as a function of distance from power lines, based on USEPA, 1992, cited in National Research Council, 1997. Note, at peak loads the fields can be doubled.

Transmission Lines, kV	Max. magnetic field on Right-of-Way,	Representative Magnetic fields at different distances from lines, mG.					
		15.24m (50ft)	30.48m (100ft)	60.96m (200ft)	91.4m (300ft)	121.9m (400ft)	152m (500ft)
115	30	7	2	0.4	0.2	0.1	0.05
230	58	20	7	1.8	0.8	0.4	0.2
500	87	29	13	3.2	1.4	0.7	0.35
660	115	38	17	4.2	1.9	0.9	0.5
1000	150	50	22	5.4	2.4	1.2	0.6

An independent comparison is available from Norway, Vistnes et al. (1997), Figure 11.

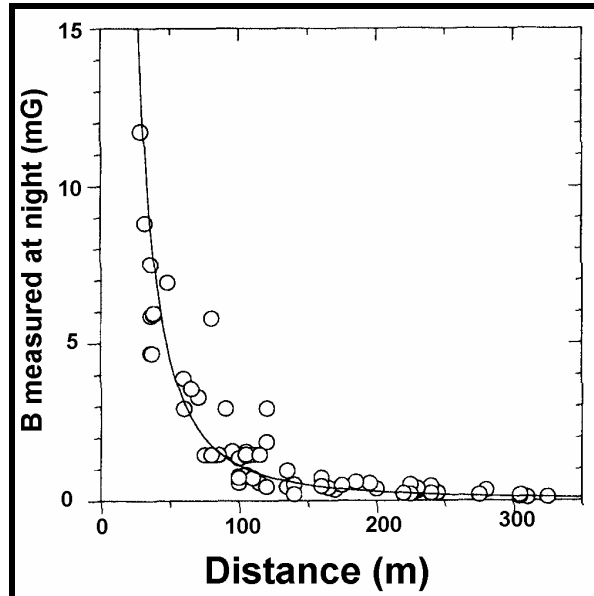


Figure 11: Geometric means of nighttime individual magnetic fields for 65 children living in the vicinity of a 300 kV powerline in Norway, Vistnes et al. (1997).

In Figure 11 the fitted line underestimates the mean nocturnal fields for some children by as much as a factor of 2 to 5 in some cases. In this survey about half of the children went to a school near the powerline and the others went to a school far from the powerline. Personal daily mean and long-term mean exposures vary with the dominant daily activity exposures. For children this is the primarily the home and school exposure regime. Examples of the typical daily exposure pattern for one of each of these cases is given in Figure 12.

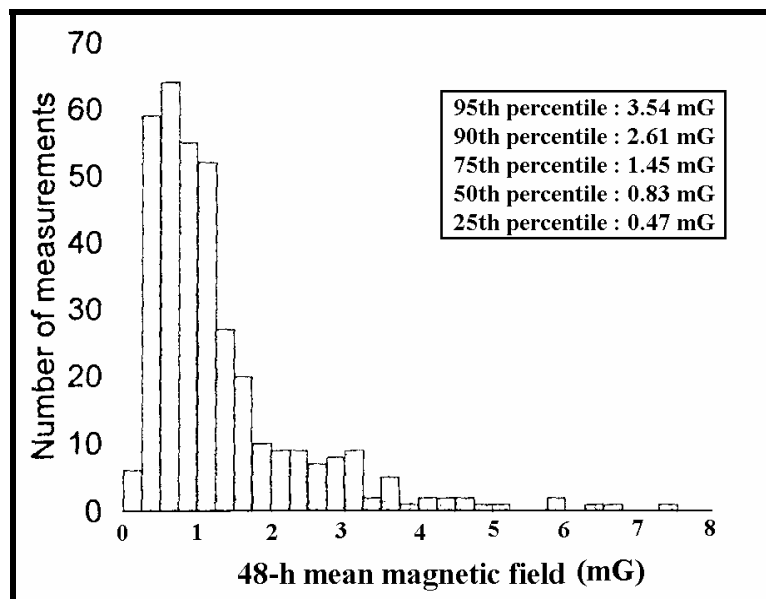


Figure 12: Frequency distribution of measured 48-hr average childhood magnetic field exposures from 382 Canadian children, Deadman et al. (1999).

A survey of Canadian homes was carried out to compare the typical and 48 hr average exposure of children across five provinces, Deadman et al. (1999). There were highly significant variations between the average home and school magnetic fields. The mean for homes was 1.41 mG, with the highest province being Quebec at 1.90 mG and the lowest being Alberta at 0.62 mG. For schools the average was 1.16 mG, with Manitoba being the highest at 1.56 mG and Alberta being the lowest at 0.70 mG. These show ranges of over a factor of 2 in the children's home and school mean magnetic field exposures.

For the surveyed Canadian children the median exposure is 0.83mG and the 95%ile is over 4 times higher at 3.54mG. The ratio between the 25%ile and the 95%ile is 7.53mG, Figure 12. An European occupational magnetic field exposure survey is given in Figure 13.

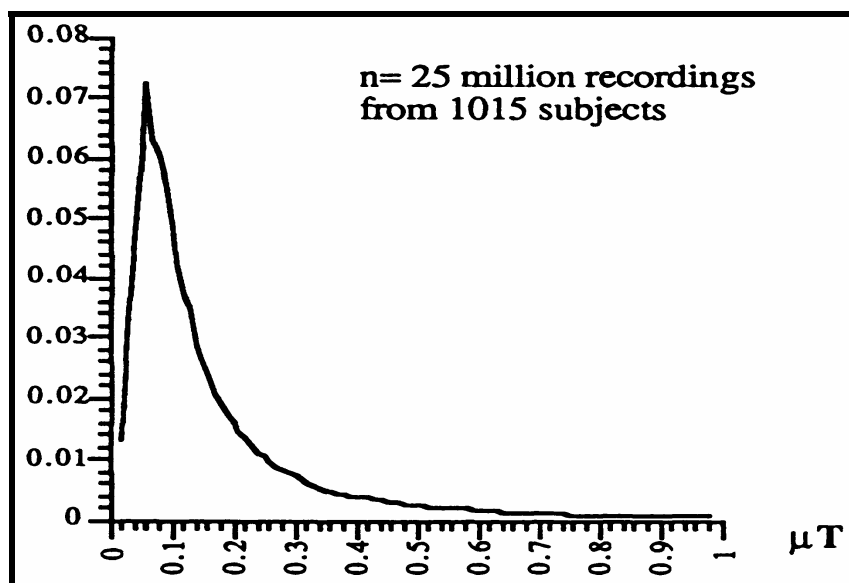


Figure 13: The Frequency distribution of occupational exposure to magnetic fields in Europe.

The median in exposure is 0.06 μ T (0.6mG) and the average is about 0.12 μ T (1.2mG) with a small proportion of high exposures above 0.6 μ T (6mG). The distribution is very close to the childhood exposure survey in Canada.

These surveys from the U.S., Norway and Canada show how varied are the mean 50/60 Hz magnetic field exposures for individual children, for homes, schools and regions. The exposure intensities vary over ranges for which extremely important and highly significant health effects are shown to occur. The ability and the need to minimize exposures is clearly evident. Keeping schools and homes well away from high voltage powerlines, keeping household wiring away from children's and adults' bed, domestic energy efficiency reducing electrical energy requirements, are a vital elements of the risk reduction strategy that is necessary to reduce the incidence and risk of serious illness and death for children and adults.

Broadcast Tower Epidemiological Study Principles:

Two fundamental study spatial patterns need to be understood and appreciated in order to carry out and to interpret the results of epidemiological studies of health effects in the vicinity of broadcast towers.

- (a) The population pattern in the vicinity of the tower.
- (b) The horizontal radiation exposure pattern, a combination of the horizontal antenna patterns and each antenna vertical radiation pattern.

Three principles are involved in the assessment of health effects around broadcast towers, Cherry (2001):

- (1) Assess the size of the population living in close proximity to the tower because to identify a high cancer rate requires a large population. If few people live near a tower then health effects cannot be detected.
- (2) The horizontal radiation pattern needs to have a high exposure level near the tower if the near tower cancer rate is to be elevated. VHF signals, generally used by AM and FM radio stations, have high exposure levels within 1 km of the tower while UHF signals generally peak outside 1.5 to 2 km from the tower.
- (3) To have a high cancer rate near the tower there needs to be an RF/MW sensitive cancer-type that has a relatively short latency and high response rate.

These three factors, a large population, a high RF exposure and an RF sensitive cancer type, must be present in order to raise the number of cases within 1 or 2 km of a broadcast tower to a detectable level.

The combination of population patterns and radiation patterns lead to two generally radial patterns, Cherry (2001), Figure 14.

Type A Pattern: Low near the tower, rising to an undulating peak between about 1.5km and 6 km. Beyond this the pattern declines with distance.

Type B Pattern: High near the tower, falling in an undulating fashion with distance from the tower.

Broadcast towers provide a unique opportunity for determining whether or not RF/MW exposures are causally related to cancer. This arises from two factors. The first is the large populations that may be exposed and the second is the particular shape of the radial RF patterns. The ground level radial RF radiation patterns are complex undulating functions of the carrier frequency, the height of the tower and the antenna horizontal and vertical radiation patterns. When rates of disease follow these patterns it excludes all other factors, removing all possible confounders.

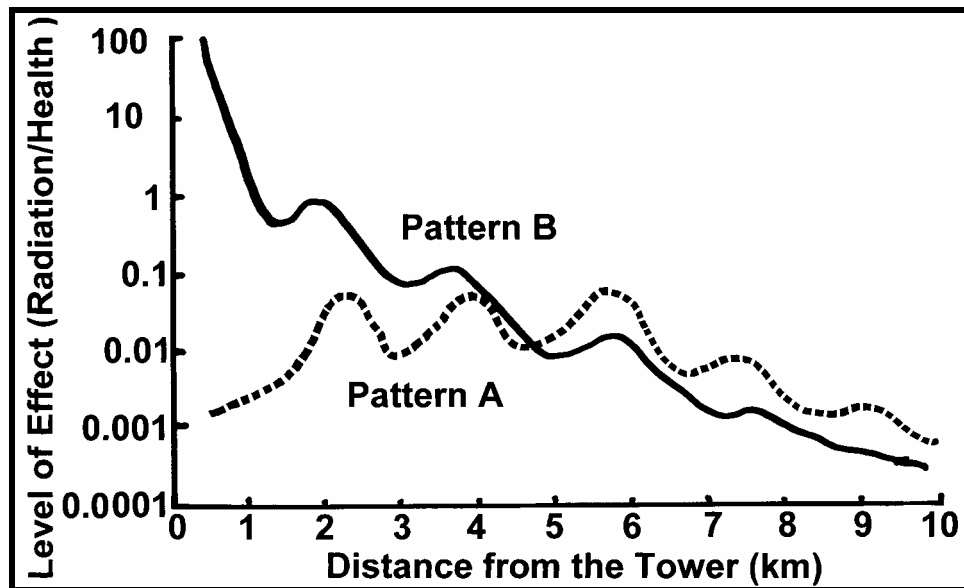


Figure 14: Typical antenna and health effect patterns around broadcast transmission towers. Pattern A (dashed) is typical of UHF antennae and health effects patterns with no VHF and/or low population numbers near the tower. Pattern B, solid line, with a high local population, powerful VHF signals and an RF sensitive cancer type.

Failure to understand these study principles and methods leads to the wrong conclusions. For example, Dolk et al. (1997a) found a Pattern B for adult Leukaemia in the vicinity of the Sutton Coldfield tower near Birmingham, which had a type B radiation pattern because it had powerful VHF stations on the broadcast mast. Leukaemia is a very RF-sensitive cancer, Szmigielski (1996).

All other cancers presented near the Sutton Coldfield tower, showed a Type A pattern, including All Cancer, Non-Hodgkin's Lymphomas, Skin Melanoma, and Bladder Cancer. Because the Leukaemia radial pattern was a Pattern B type, which the authors appear to assume all broadcast towers produce, like an inverse square law pattern, they studied 20 other sites to see if they had the same results for Leukaemia. All other sites individually and when grouped showed Pattern A types for adult Leukaemia. Figure 15.

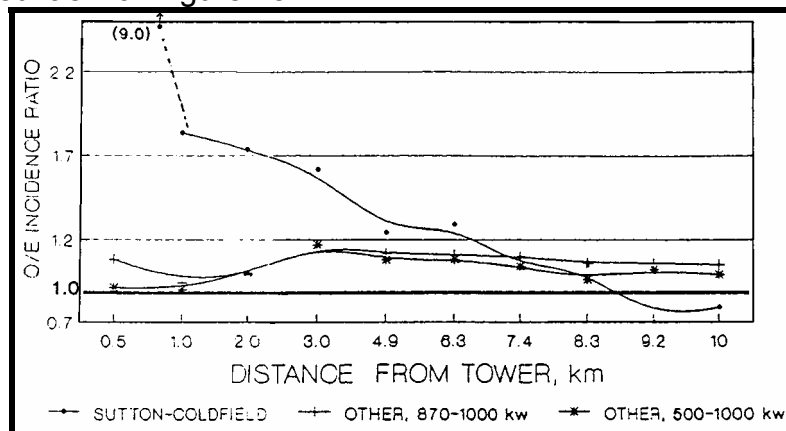


Figure 15: The radial patterns of adult cancers in Sutton Coldfield and 20 other regional TV/FM transmission sites in United Kingdom, from Goldsmith (1997) derived from Dolk et al. (1997a,b).

However, no other site than Birmingham had both a high population living close to the tower and a VHF high power emission. Hence all other sites logically produced radial Pattern A types.

Cherry (2001) appropriately concludes that this makes the chance of confounding factors vanishing small and indicates a causal relation between a range of adult cancers and chronic exposure to very low mean-intensity RF radiation at less than 0.1% of present standards.

RF/MW Exposures Surveys:

A radio antenna uses rapidly oscillating electric currents in a conductor to produce an oscillating electric field that will radiate away from the antenna and propagates through the air and vacuum at the speed of light. The oscillating electric field generates an oscillating magnetic field at right angles to the electric field. Usually the antennae are vertical so that the vertical field is electrical and the horizontal field is magnetic. This is called electromagnetic radiation. Very close to the antenna, within about one wavelength, there is an inductive component where part of the magnetic field links back to the antenna inducing a counter current. Hence close to the antenna, called the Near-Field, the electric field is dominant and not exactly proportional to the magnetic field. Beyond about 1 wavelength, in the Far-Field, the reactive induction does not occur and the balanced EMR oscillation is regular and proportional.

The wavelength (λ) is equal to the speed (c) divided by the frequency (f). Since the speed is near 3×10^8 m/s a frequency of 1 kHz has a wavelength of 300km, 1MHz it is 300m and for 1GHz it is 30cm. In almost all situations people are exposed to the Far Field of a radiofrequency/microwave (RF/MW) signal. The main exceptions are people using portable hand-held telephones where the aerial is a few cm from the head, well inside the Near Field. This creates very high intensity exposures. In addition, the head being a conductor takes part in the inductive process that occurs within the Near Field.

In the Far Field the intensity of a beam decreases as the inverse square of the radius because the energy is spread over a growing sphere. In most locations there is a direct and a reflected signal from the ground, plus other weaker reflected signals. The environmental complexity of RF/MW exposures varies with the carrier frequency. The shorter the wavelength the narrower the main beams and side-lobes are of the signals radiated by the antennae. Also the signal is reflected from solid surfaces such as walls and the ground, and the signal is scattered by the leaves and branches of trees and other complex surfaces. A formal consultants report, Hammett and Edison (1997), on the radio frequency fields around the Sutro Tower, in the heart of San Francisco, produces the formula which they derived from the Federal Communications Commission, Science and Technology Bulletin No 65, (October 1985) which gives a formula for calculating power density from individual radiation sources:

$$\text{Power Density, } S = \frac{2.65 \times 1.64 \times 100 \text{ RFF}^2 \times [0.4 \text{ VERP} + \text{AERP}]}{4\pi D^2}, \text{ in mW/cm}^2$$

where VERP = total peak visual ERP for NTSC TV stations, in kW,
 AERP = total aural (or average for DVD stations) ERP, in kW,
 RFF = relative field factor, vertical antenna pattern, at the direction to the
 actual point of calculation, and
 D = Distance from the centre of radiation to the point of calculation, in m.

The factor of 2.56 accounts for the increase in power density due to ground reflection. The factor of 1.64 is the gain of a halfway dipole relative to an isotropic radiator, part of the horizontal antenna pattern. The factor of 0.4 converts the peak visual ERP for TV stations to an average RMS value for FM radio stations and DTV (Digital TV) stations the value of the VERP is zero. This shows the vital importance of the relative field factor which deals with main beam and side lobes, Figures 16 to 18.

Around broadcast towers the ground level exposure patterns are a function of the power of the source signal and the antenna gain. The gain, expressed as a function of the Equivalent Isotropic Radiated Power (EIRP) is a function of the technology used to focus the signal. Antennae are complex elements that attempt to efficiently focus the main beam and minimize the side-lobes. The ability to do this to some extent is a function of the carrier frequency. Because of these side-lobes a complex antenna pattern is formed with undulating peaks in the 'near field' towers, which typically extends out to 5 to 6 km. VHF antenna patterns produce ground level Pattern A, with a high exposure peak within 1 km of the tower.

Vertical Antenna Patterns:

The vertical antenna pattern is a function of the antenna type and the carrier frequency. Figure 16 shows the vertical antenna pattern of an 8-dipole array for a 98 MHz FM station.

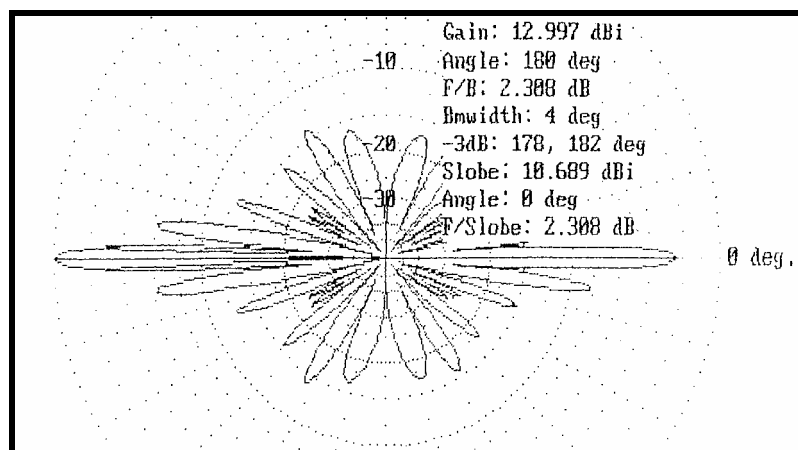


Figure 16: A typical vertical antenna pattern for a 4-element dipole array at about 98 MHz.(VHF), Units in dB.

The radial scale in Figure 16 is in dB that varies logarithmically with intensity. There is a very large difference between the intensity in the main horizontal beam (0 deg), the first minimum and the first side-lobe. These three points are -2.3, -28 and -8.1 dB respectively. These correspond to gains of 0.588, 0.00016 and

0.155, or relative gains of 1.0, 0.00027 and 0.2 respectively. The side-lobes have elevation angles of 8, 15, 40, 57 and 72 degrees. For an antenna at 500m above ground level, the ground level side-lobe peaks occur at 160, 390, 600, 1870 and 3560m from the base of the tower, with troughs of significantly 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.

RF/MW antennas, including cell phone antennas have complex horizontal and vertical radiation patterns, for example Figure 17.

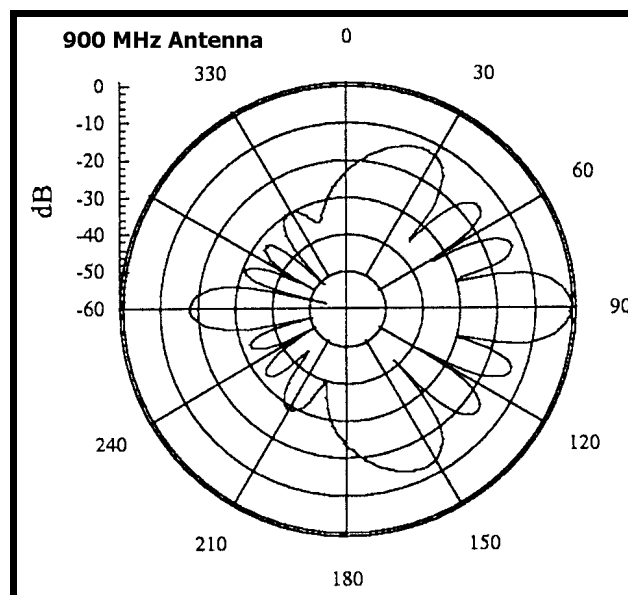


Figure 17: The antenna pattern for a 900 MHz cell phone base station transmission, Bernardi et al. (2000).

In Figure 17 note that the radial scale is in decibels (dB) which is a logarithmic scale. From the main beam peak at 90° to the minimum on either side at about 75° and 105°, the signal is 30 dB lower, i.e. 1000 times weaker. The other smaller peaks are called side-lobes. They are typically 12 to 16 dB smaller or 16 to 40 times less intense than the main beam. All antennas have main beams and side-lobes producing a complex spatial pattern that needs to be understood when carrying out health effects studies around radio/TV broadcast or cell phone transmission stations.

Figure 18 shows the dominant strength of the main beam, which is actually stronger because of the square of the relative field factor. The elevation angle of the antenna is usually slightly tilted downwards by 0.5° to point the main beam at the more remote listening or viewing audience.

The Relative Field peaks in Figure 18 are at 0.5, 3.5, 5.7, 7.9, 10.1 and 12.3°. With the assumption of the mean height of the antennae at 460m, these peaks correspond to ground level positions at 52.7, 7.5, 4.6, 3.3, 2.6 and 2.1km from the

tower. The actual exposure intensity is a function of the square of the Relative Field and the inverse square of the distance along a beam.

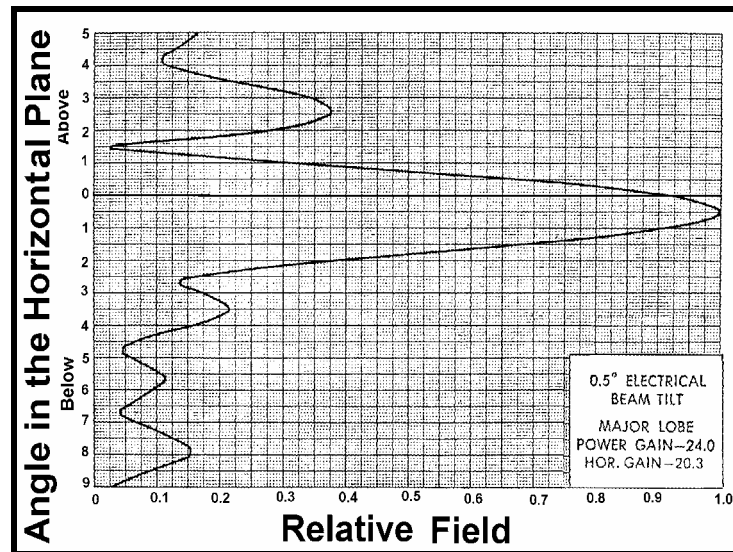


Figure 18: A typical Relative Field for a UHF RF/MW broadcast antenna from Hammett and Edison (1997). The signal intensity is a function of the square of the Relative Field.

This results in the ground level peaks being closer to the Tower, especially for the most remote peaks. These adjustments are taken into account by the radial UHF pattern in Figure 19. This shows the main beam peaks at 12.5km and the major side-lobe peaks at 6, 4.5, 3.2, 2.2 and 1.1km.

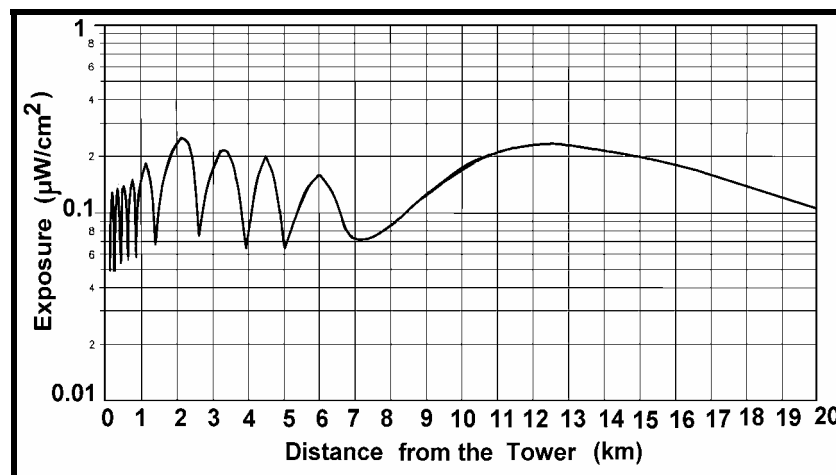


Figure 19: Ground level exposure for a typical UHF TV broadcast signal, from an antenna pattern (14), for an 18 MW EIRP transmitter at 460m AGL, for a flat horizontal surface.

Figure 19 shows that the UHF antenna produces a Type A radial exposure pattern.

In contrast to the UHF pattern Figure 20 shows typical radial VHF patterns. Figure 20a shows ground level RF intensity measurements taken in New York round the Empire State Building in the early 1930's. There were radio stations on towers at the top of the building. Figure 20b is from a broadcast engineers handbook

showing the frequency dependence of the peaks at the general and consistent declining intensity of distance in contrast to the UHF in Figure 19.

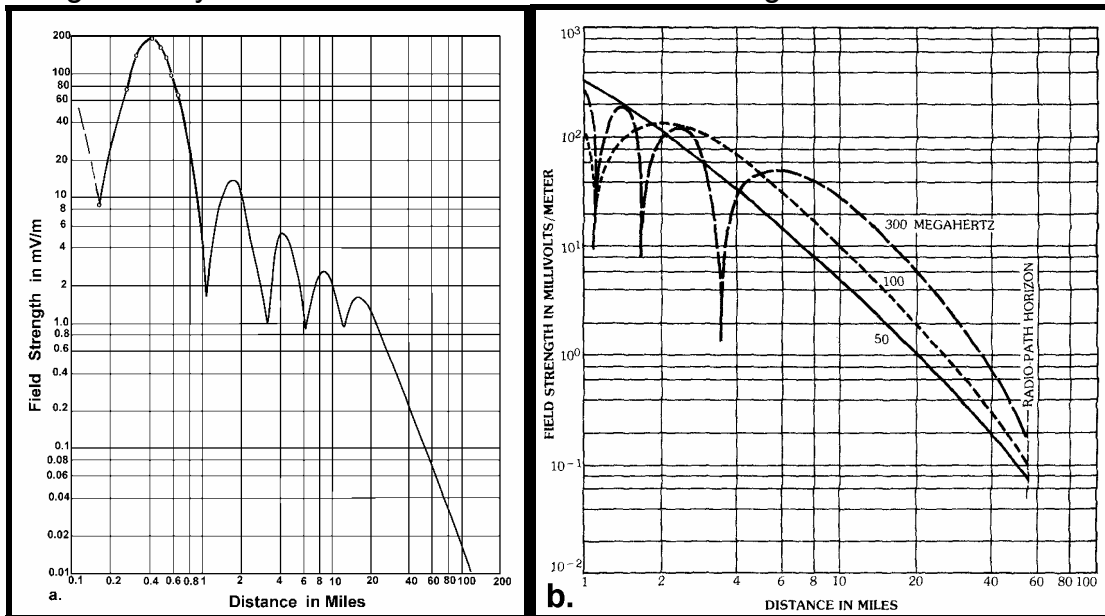


Figure 20: Ground level radiation pattern for (a) the 44 MHz (VHF) signal from the Empire State Building in New York City, Jones (1933) by merging his figures 6 and 8, and (b) a theoretical set of 1 kW antenna at a height 1000ft and a receiver at a height 30ft, Jordon (1985).

These figures show the distinct difference between the Type A pattern from the UHF signals and the Type B patterns of VHF signals used for FM radio stations.

Horizontal antenna patterns:

Antennae are not only capable of focusing RF radiant power into vertical beams but can also focus the beams in the horizontal plain to send most of the broadcast signal towards most of the listeners and viewers, Figure 21-23.

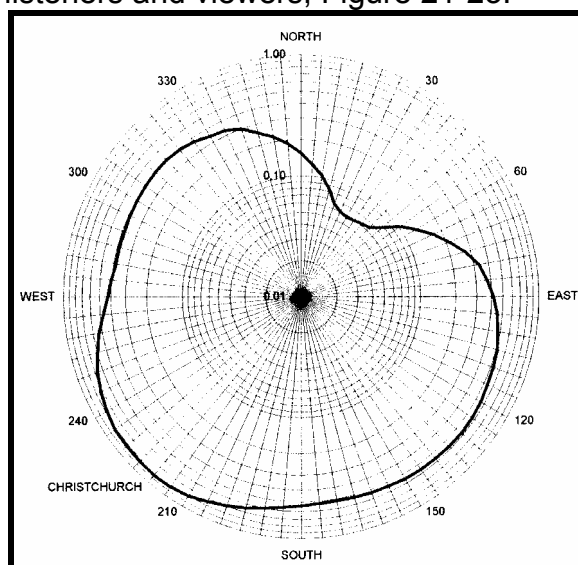


Figure 21: Horizontal antenna pattern for an 8-element dipole array for a 98 MHz FM transmission pointed towards the targeted high population city of Christchurch.

Figure 21 shows that the signals from this antenna are horizontally focused towards the city of Christchurch from the tower which is located to the northeast of the city. The second is for the UHF antenna, the vertical pattern of which is in Figure 22 for a UHF signal from the Sutro Tower in San Francisco.

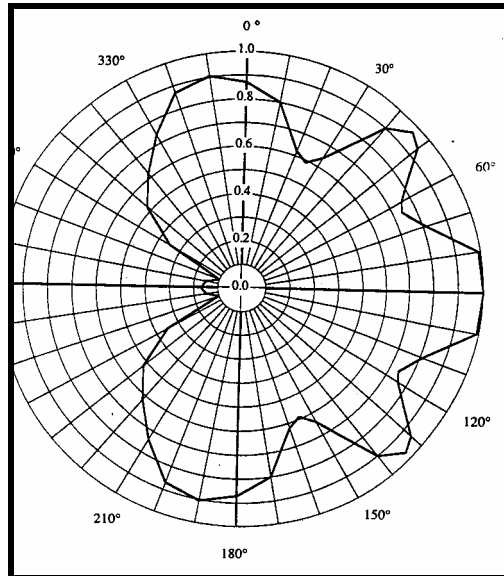


Figure 22: Horizontal antenna radiation patterns showing the relative field strength for, (a) UHF Digital TV (linear scale) from the Sutra Tower, Hammett and Edison (1997).

The Sutra tower is in the mid-western portion of the San Francisco Peninsula, with a small number of seaside suburbs behind it, most of the City of San Francisco, plus Oakland and Berkeley to the east.

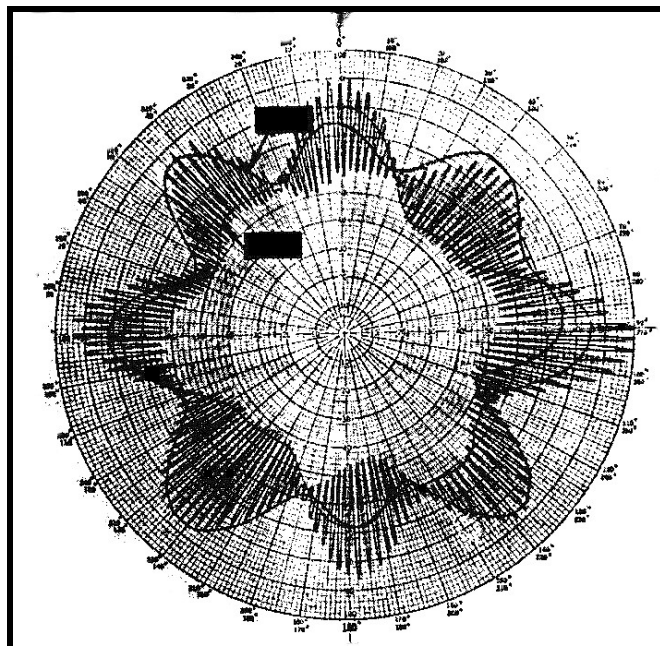


Figure 23: Detailed and smoothed horizontal antenna pattern measured from a circularly polarized FM broadcast antenna, Ben-Dov (1972).

Figure 23 shows that the generally presented 8 or 9 horizontal peaks are smoothed on a graph that actually has 15 or 16 sharp peaks forming each smoothed broad peaks.

Broadcast engineers have told me and the data confirms that they can design and build antennas with horizontal and vertical patterns to send most of the signals to where most of the receivers are. The Fourier phasing of the antenna dipoles and panels gives undulating patterns that can be focused to some extent to achieve the design objectives.

Direct measurement RF exposure surveys:

The initial global ubiquitous exposure to RF/MW fields was produced by short-wave radio and telecommunication and weather satellite signals. RF/MW fields are produced by many more sources than is commonly known, including high voltage power lines Vignati and Giuliani (1997). Many modern appliances at home and work also produce RF/MW radiation fields, including microwave ovens, computers, TVs and Play Stations, cordless and mobile phones Mild (1980) and Kraune et al. (2002). Urban and rural areas have detectable and usable RF/MW signals from many Radio and TV stations, Tell and Mantiply (1980), Mantiply et al. (1997), and cell phone base stations, Bernardi et al. (2000).

Some occupations are identified as having above average exposures. They include “electrical and electronic occupations”, radio and radar operators that include military, police officers and fire fighters, heavy computer users, ICNIRP (1998), welders (Skotte and Hjollund (1997), Kheifets et al. (1995)) and many industrial situations where workers spend long period near operating electric motors. Some office situations are worse than others because of proximity to transformers and power cables. All occupations using mobile phones or two-way radios, including police officers, security guards, commercial truck operators, and airport staff. Commercial and military pilots are exposed to a mixture of ELF and RF/MW fields from power supplies, visual displays, radios and radars (Nicholas et al. (1998, 2000)). Radar, radio and TV equipment and antenna repairmen are frequently exposed to higher than average RF/MW radiation.

An early urban RF exposure assessment was carried out by Tell and Mantiply in the late 1970s. The following graph, Figure 24, shows the spectral analysis from Portland Oregon showing a number of FM stations. They carried out measurements to survey 15 cities throughout the United States. The frequency distribution of personal exposures is given in Figure 25.

The survey of 15 US cities showed that there were scores of RF/MW sources. The mean exposure at that time was measured as $0.0048\mu\text{W}/\text{cm}^2$ ranging from $0.002\mu\text{W}/\text{cm}^2$ in Chicago and San Francisco, to $0.018\mu\text{W}/\text{cm}^2$ in Boston. However San Francisco had 2.34%, Chicago 0.4%, Boston 1.5% and Washington DC 2.8% exposed to $\geq 1\mu\text{W}/\text{cm}^2$. About 1 % of the all populations were exposed to $1\mu\text{W}/\text{cm}^2$ or more, 200 times the average.

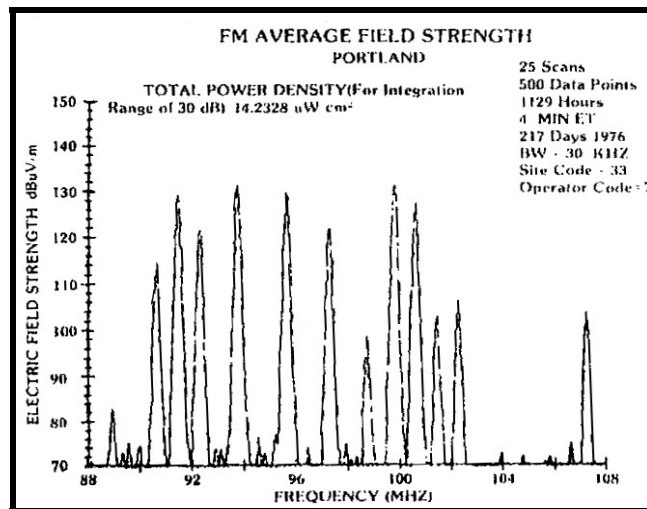


Figure 24: Measured FM radio broadcast field intensity spectrum in Portland, Oregon, 1976, Tell and Mantiply (1980).

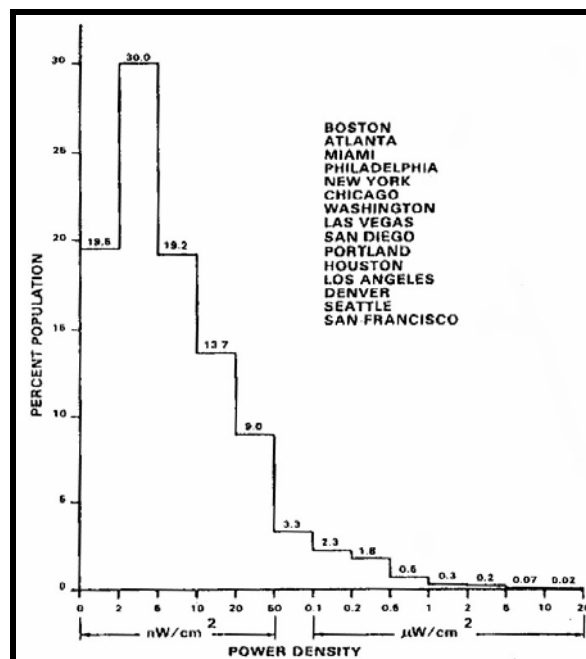


Figure 25: Differential fraction of population and 15 cities exposed to measured radio-frequency radiation, Tell and Mantiply (1980).

Installation of many cell phone base stations in Vancouver raised concerns about exposure of children's schools. In 1997 a base-station antenna was installed in the church cross located across the street from an elementary school in Vancouver. Some of the parents of the students became concerned that the RF radiation from this and other antennas could affect their children's health. In response, a survey was carried out of RF radiation in around five schools selected in consultation with the concerned parents, Thansandote, Gajda and Lecuyer (1999). The results are set out in a table, Figure 26.

The authors of the paper try to allay the parents' concerns in a statement that the exposures are well within the Canadian Standard, Code 6. However, the standard is based on avoiding acute tissue heating effects, not on avoiding cancer and many

other health effects being shown to be caused well under the reported levels, even when the peak level is divided by 200 to approximate the mean exposure level.

Frequency band	Power density, $\mu\text{W}/\text{m}^2$				
	School 1*	School 2†	School 3‡	School 4§	School 5§
Analog	0.10	25 600	2250	0.12	2.90
PCS	1620	NA	0.23	0.25	0.51
AM	442	NA	37.1	51.0	87.2
FM	6	NA	9.16	0.64	0.99
TV	0.61	NA	0.05	0.012	0.011

Note: PCS = personal communication services (digital cellular phone service); analog = first-generation cellular phone service. NA = data not available because of measurement time limitation.
 *PCS antenna across street.
 †Analog antenna on school roof.
 ‡Analog antenna across street.
 §No antenna in vicinity.

Figure 26: Maximum measured power densities of radiofrequency radiation in areas by staff and students in 5 Vancouver Schools, Thansandote et al. (1999).

The total exposure for schools 1 to 5 are $2068.7\mu\text{W}/\text{cm}^2$ ($10.3\mu\text{W}/\text{cm}^2$); $25,600\mu\text{W}/\text{cm}^2$ ($123\mu\text{W}/\text{cm}^2$); $2296.5\mu\text{W}/\text{cm}^2$ ($11.4\mu\text{W}/\text{cm}^2$); $52.02\mu\text{W}/\text{cm}^2$ ($0.26\mu\text{W}/\text{cm}^2$) and $91.6\mu\text{W}/\text{cm}^2$ ($0.46\mu\text{W}/\text{cm}^2$), with the mean exposures in brackets. All schools have an estimated mean exposure above the $0.2\mu\text{W}/\text{cm}^2$ threshold found for childhood leukaemia incidence and mortality in North Sydney, and Rome. Cell phone radiation has been shown to significantly damage DNA, $p < 0.0001$, at an exposure level of $1.2\mu\text{W}/\text{cm}^2$, a clearly non-thermal effect. This and several other studies show that non-thermal cell phone radiation is genotoxic, with a safe level of zero exposure. Therefore these children and teachers are at serious risk of getting cancer and many other serious health effects.

A recent US survey covers a different range of frequencies, Figure 27.

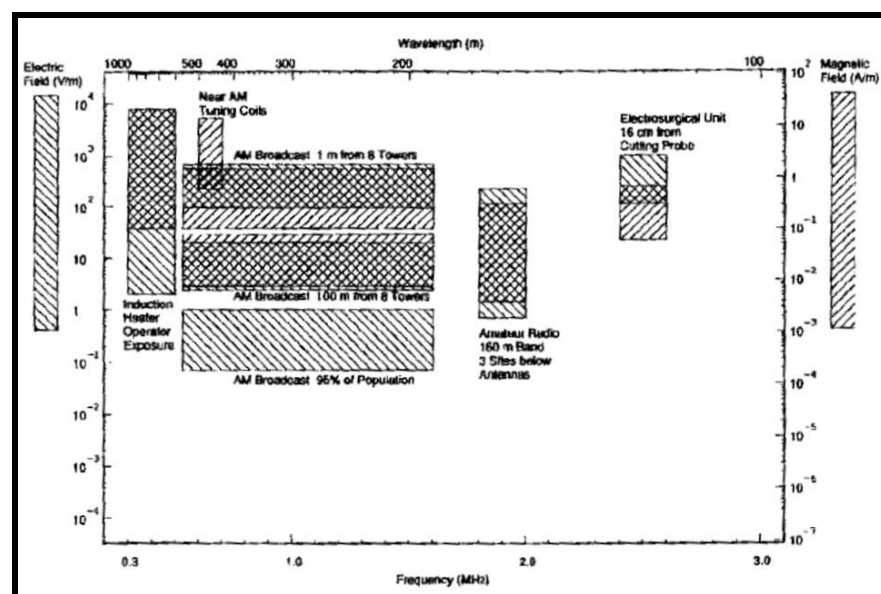


Figure 27: Middle frequency band (MHz) from 0.9 to 3.0 MHz, Mantiply et al. (1997).

The field strength of 1000V/m to 265mW/cm², 100 V/m is equal to 2653μW/cm², and 10V/m to 26.5μW/cm².

Evidence of the Ubiquitous Genotoxic Carcinogen Effect:

The evidence that the household electric wiring, that is the source of residential electromagnetic fields that did not exist in home prior to 1900, is a major cause of the cancer rate rise in the 20th century is set in context by Court-Brown and Doll (1961). They noted that there had been a massive rise in leukaemia in a wide range of age groups in England and Wales in the first half of 20th-century, Figure 28.

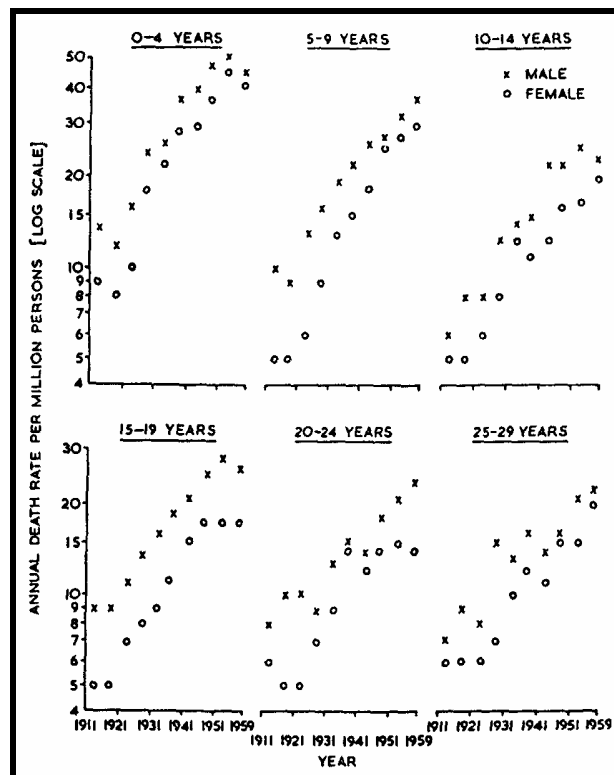


Figure 28: Trend in leukaemia mortality with time for England and Wales for 5-year age-groups by sex, from 1911-1959, Court-Brown and Hill (1961).

There has been a great deal of investigation and speculation about what the source of this early childhood leukaemia mortality is caused by. It is primarily Acute Lymphoblastic Leukaemia (ALL). It is paralleled by significant rises in all leukaemia mortality from the data presented, including all age-ranges up to 30 years, Figure 28.

Court-Brown and Hill were interested in a distinctly new pattern of early childhood leukaemia death that was evident in the 1945-59 data from England and Wales but not in the 1910-1915 data, Figure 29.

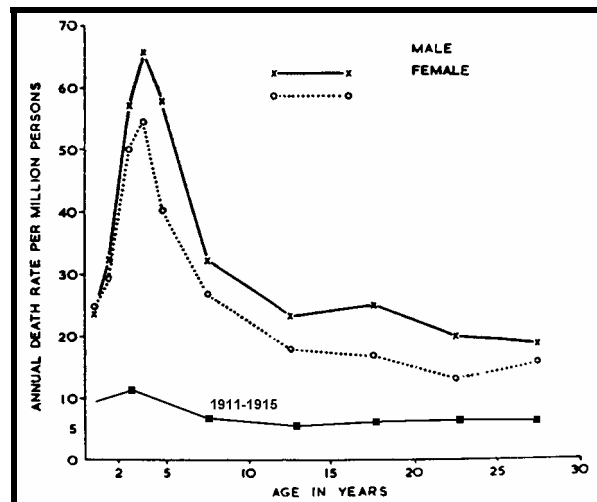


Figure 29: Age-specific death rates for leukaemia under the age of 30 years by sex in England and Wales, Court-Brown and Doll (1961), with the 1911-1915 data added from their Figure 3.

Kraut et al. (1994) showed that several childhood cancers, including brain cancer, leukaemia and lymphomas, showed a dose-response relationship to the proportion of residential electrical wiring in Canadian Provinces, Figure 30 shows the brain cancer graph.

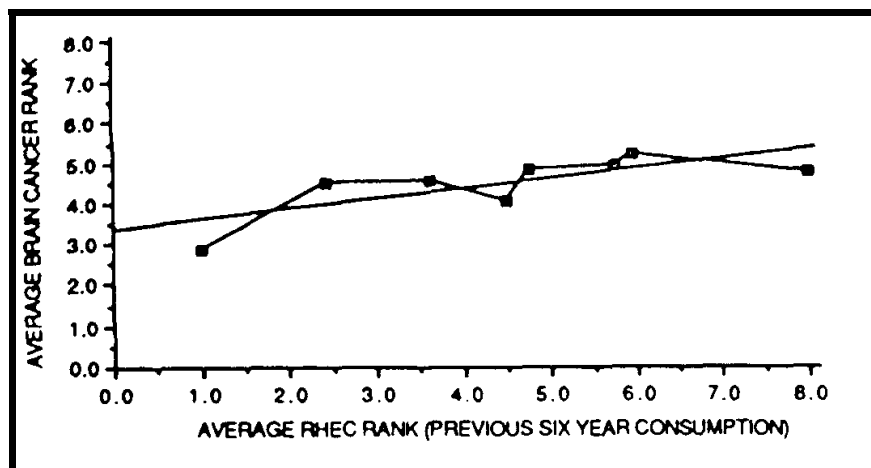


Figure 30: Scatter plot of provincial brain cancer rank and average residential hydroelectric consumption rank in the previous 6 years, Kraut et al. (1994).

The first residential childhood cancer study, Wertheimer and Leeper (1979) found a dose-response increase in All Cancer, dominated by Leukaemia and Brain Cancer for children associated with EMF. There are several other studies. Hence there are multiple studies confirming that brain cancer and leukaemia is increased in children exposed to residential ELF fields. The biological impact of RF/MW fields is much higher than ELF fields. This provides very strong support for the evidence that pulsed RF/MW fields from cellphones will significantly increase the incidence of brain cancers in cellphone users, and highly significantly in heavy cellphone users.

The biomarker cancer, ALL in very young children, is independently confirmed by several studies. A good example is Hatch et al. (1998). They found that pregnant women exposed to residential ELF fields had significantly higher incidence of ALL

in their babies, OR = 1.59, 95%CI: 1.11-2.29. The children's use of electric blankets and mattresses also significantly raised the rate, OR = 2.75, 95%CI: 1.52-4.98. The use of electronic equipment, including video games and TV watching, also raised the incidence. The data shows a regular pattern of higher ORs for the <1 year exposure than the higher age groups. This is consistent with the rapid development of ALL in young, susceptible children and the greater protection of some children and developing immune systems, leading to lower incidence rates at higher ages.

The relationship of early childhood ALL to household wiring, in a spatial geographic and temporal development pattern is robustly confirmed by Milham and Ossiander (2001), primarily based on independent US data.

Totally independent support comes from a recently published study, Li et al. (2002). They monitored the personal ELF fields of 1063 pregnant women in California. They found a significant dose-response increase in miscarriage. The maximum typical daily peak exposure of 16mG resulted in RR = 5.7, 95%CI: 2.1-15.7, a 5.7-fold increase in early miscarriage rate. A universal genotoxic substance damages the chromosomes in the fetus within the womb. Low level damage is repaired in most children but some develop cancer, especially ALL. Higher exposures increase this risk. Very high exposures produce very high chromosome aberration rates, causing malformation and initiating miscarriage.

ELF field exposure is ubiquitous and has resulted in a major contribution to the cancer rate rise over the 20th Century. Court-Brown and Doll (1961) report a 5 to 6 fold increase in leukaemia from 1911-1959. Cancer incidence rates have continued to rise in the later 4 decades of the 20th Century. A conservative estimate of the contribution of residential ELF fields to the cancer rate is a factor of 8 to 10. A childhood cancer study in New Zealand found that the adjusted childhood leukaemia rate was raised by a factor of 12, OR = 12.0, 95%CI: 1.1-137, with a threshold of 2mG at the child's bed. If the cut-off point had been 1.95mG this would have been OR = 15.5, 95%CI: 1.1-224, Dockerty et al. (1998).

Applying a low factor of 6 to Table 4.1 reduces the control group from $N_c=6$ to $N_c=1$ for $N_e=12$. This results in:

$$\mathbf{RR = 12.0, 95\%CI: 1.57-91.94, p=0.005}$$

All of the other groups have RR =12.0 with much more significant p-values. This clearly illustrates the under-estimation of the health effects by ignoring the ubiquitous nature of the EMF fields.

The mysterious childhood cancer peak for 2-4 year olds:

Through a detailed analysis of the U.S. childhood leukaemia data from the 1920's to the 1960's, an association was found with the extent of electricity reticulation in homes, country by country and state by state, Milham and Ossiander (2001).

Milham and Ossiander (2001) proposed the hypothesis that the causal agent for this new childhood cancer peak was the electromagnetic fields in the homes created by electric power domestic reticulation. This explains the time delay

between the UK and the US. It also explains the time delay between white and black households in the US. It also explains the Australian situation with the potential to apply country by country and state by state.

The 2-4 year old cALL peak did not exist before domestic electric reticulation occurred and was formed case by case after it did occur. As the proportion of homes connected increased, then the new cALL peak mortality rate rose proportionally. The formation of the year-3 peak and rise in leukaemia mortality is confirmed, Figure 31. It went higher and higher over time.

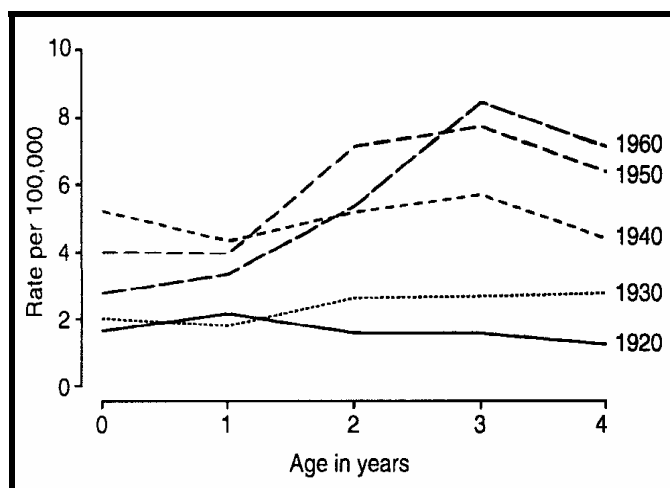


Figure 31: Childhood leukaemia mortality for the United States whites by single years of age 0-4, for each 10 years from 1920, Milham and Ossiander (2001).

By obtaining the data in state by state development of electrical reticulation the early childhood age-specific leukaemia rate plotted as a proportion of the number of homes electrified. For the 1928 to 32 period this is in Figure 32 and the period 1949-52 in Figure 33.

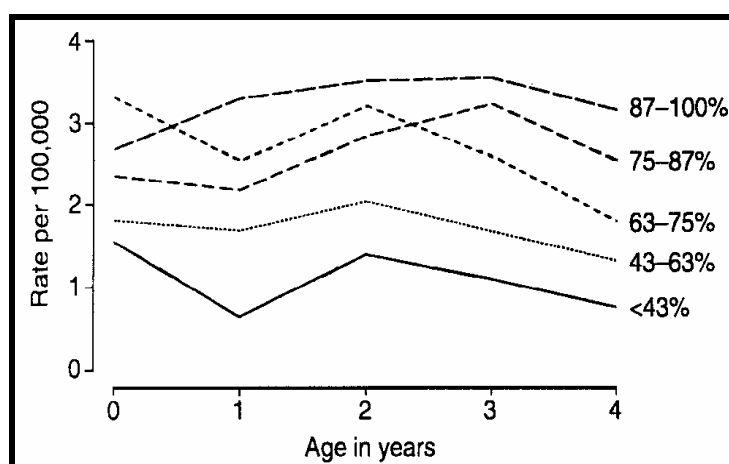


Figure 32: Childhood leukaemia mortality rates for all races 1928-32, by percent of residential electrification and age of death, Milham and Ossiander (2001).

The early period, Figure 32, shows the initial absence of the 2-4 year peak but a progressive increase in the early childhood leukaemia rate with increasing percentage of electrical reticulation.

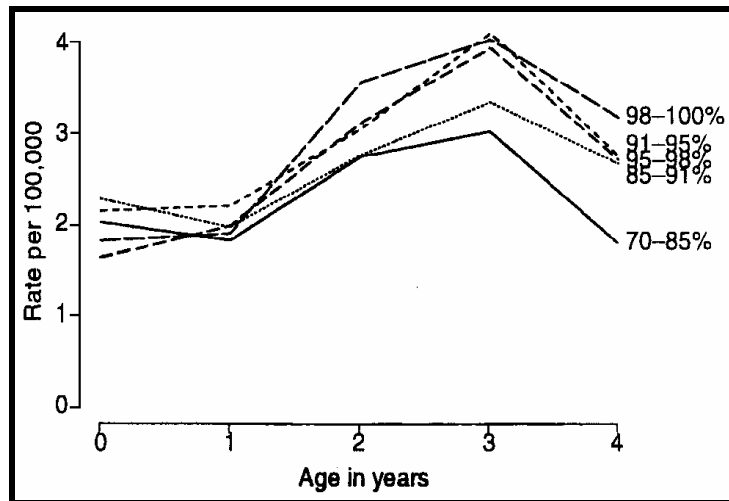


Figure 33: Childhood leukaemia mortality rates for all races 1949-51, by percent of residential electrification and age of death, Milham and Ossiander (2001).

Figure 33 shows that the 2-4 year old peak is well developed in the 1949-51 data and there remains a gradient with higher leukaemia mortality rates with the increasing proportion of residential electric reticulation. This is direct, robust, confirmation of the hypothesis. The early childhood ALL leukaemia and all leukaemia mortality is attributable to the electromagnetic fields in homes produced by electrical reticulation.

Milham and Ossiander state that worldwide occurrence of this peak of childhood leukaemia follows the introduction of electrification. For example, Ramot and McGrath (1982) found a dramatic shift from childhood lymphoma to leukaemia (ALL) that occurred in the Arab population in the Gaza Strip after the introduction of electric power reticulation to homes. Milham and Ossiander (2001) state:

"The authors conclude the childhood leukaemia peak of common Acute Lymphoblastic Leukaemia (cALL) is attributable to residential electrification. 75% of childhood cALL and 60 % of all childhood leukaemia may be preventable."

This means that the early childhood all leukaemia (<5 years) increased from 1900 to 1995, from less than 10 per million to more than 77 per million in 1995, an increase by a factor of 7.7, a factor of 4.6 is attributable to household EMF exposures. For cALL the early childhood incidence has risen from about 7 per million in 1900 to around 70 per million in the SEER data for white children in the US in 1995. This factor of 10 increase has a factor of 7.5 attributed to household 50/60 Hz fields.

After carrying out an exposure survey, Wertheimer and Leeper (1979) carefully assessed the Wiring Code Configuration for each case and control, and evaluated potential confounders relating to age, race, economic status and proximity to heavy

traffic. The cases and controls were ranked into four levels in relation to the wiring code, Very Low, Low, High and Very High. The percentage of all surveyed homes in these levels was 6.8%, 63.7%, 29% and 0.6%. This illustrates the problem of finding a non-exposed group. The cancer rates were calculated for each exposure group. A significant dose-response trend resulted, $p = 0.008$, Figure 34.

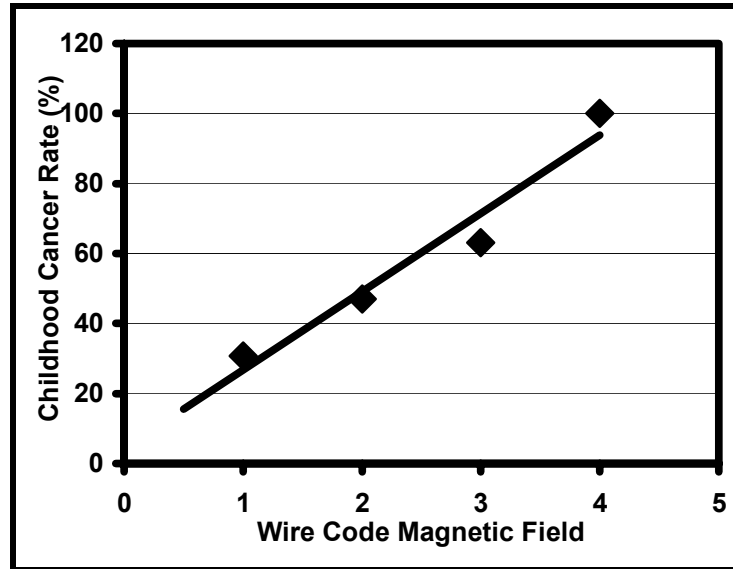


Figure 34: Childhood cancer rates in Denver 1976-79, relative to chronic electromagnetic field exposures assessed using a Wiring Code Configuration, (1). Trend $p = 0.008$.

The chronic mean magnetic field exposure associated with the Very High Wire Code (VHWC) is about 2 mG. It was observed that of the children diagnosed with cancer who were living in the VHWC fields ($n=6$), 100% of their cancers were associated with their exposure to the magnetic fields. Even in the Very Low fields, 30.8% of the cancer cases were associated with the magnetic field exposures.

A study in New Zealand involved long-term measurements (hours) of the magnetic fields in the bedrooms and play areas of children with leukaemia. Dockerty et al. (1998, 1999) found that for a cut-off point of 2 mG, after adjusting for pregnancy, income and mothers education, the Adjusted Odds Ratio was Adj OR = 12.0 (1.1-137). The middle (1-2mG) group had Adj OR = 1.75 (0.4-7.4), with <1mG being the reference group OR = 1.0. This shows a weak, non-significant trend from a small case number study. When cases were divided into size-based "thirds", the adjusted bedroom fields showed no association but the highest exposure cut-point was quite low, at 0.55mG. This illustrates the strongly skewed distribution of mean daily personal exposures. For the Dayroom exposures this produced a dose-response with middle third Adj OR = 3.8 (0.5-28.7) and Highest third Adj OR = 5.2 (0.9-30.8). When the readings were combined into a time-weighted average exposure, using the cases with <1mG as the reference group (OR = 1.0), a more uniform dose-response increase in childhood leukaemia incidence was found. For 1mG - <2 mG, Adj OR = 1.5 (0.3-7.2). For ≥ 2 mG the Adj OR = 3.5 (0.5-23.7) (26).

A similar project, with long-term mean magnetic field measurements, was carried out in Germany, Michaelis et al. (1998). They compared the Childhood Leukaemia rate with a range of mean magnetic field measurements in the child's bedroom. The

24-hr median, OR = 2.3 (0.8-6.7). For the 24-hr median for children ≤ 4 yrs, OR = 7.1 (1.4-37.2). For the nighttime only medians for all children, OR = 3.8 (1.2-11.9) and for children ≤ 4 yrs, OR = 7.4 (1.4-38.4). This confirms the stronger association with sleeping ELF exposures and leukaemia, most likely because of the magnetic field reducing melatonin. The vulnerability of younger children is also evident, also related to lower immune system competence and lower levels of melatonin in early childhood.

Savitz et al. (1988) carried out a replication of Wertheimer and Leeper (1979). They used an expanded cancer data-base and developed an alternative Wiring Code approach, using a buried wiring system as the lowest exposure situation, Figure 35.

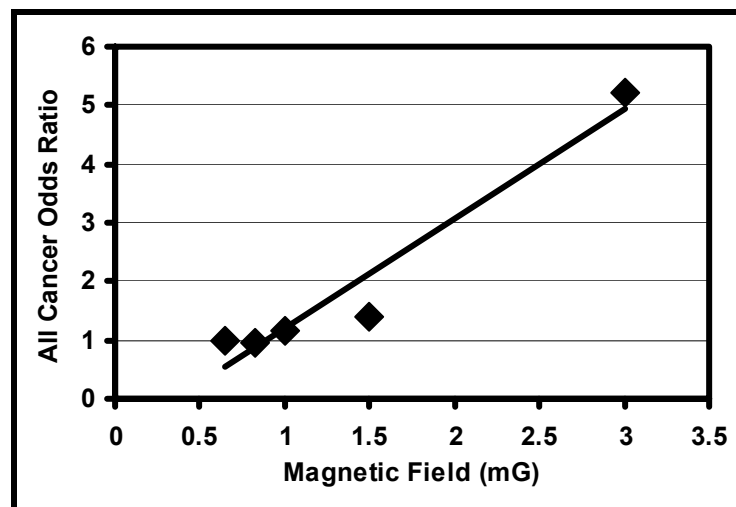


Figure 35: Childhood Cancer (0-14 years) in Denver for cases (1976-1983) and controls (1984-1985), Savitz et al (1988). Trend $p = 0.01$.

The five-level wire code cancer trend associated with fields at the time of diagnosis resulted in a significant trend, $p=0.02$, with the VHWC Cancer rate OR = 2.20 (0.93-5.21). When the wire code was related to residential exposures 2 years before diagnosis, allowing for a 2 year latency effect, the trend was more significant, $p=0.01$, Figure 2. The VHWC All Cancer rate was then raised to OR = 5.22 (1.18-23.09). Taken together Wertheimer and Leeper and Savitz et al. provide a classically causal relationship between residential electromagnetic fields and childhood cancer, including leukaemia and lymphoma, Hill (1965). They are supported and confirmed by New Zealand and German studies cited above and about 20 additional studies cited below.

There is a more modern Preconceived Dismissive Approach (PDA). As the evidence gets significantly stronger, the conclusions are getting significantly weaker, Savitz (2001). This occurs despite some of the highest quality and strongest evidence being produced by Professor Savitz's research team. The dismissive approach is putting public health seriously at risk of demonstrated adverse health effects by retaining high allowable public exposure standards of over 1000mG when the childhood cancer rate is significantly elevated, even with daily mean exposures well below 2mG.

The greater strength of evidence available now includes the genotoxic evidence and a very large body of published epidemiological studies showing elevated childhood cancer rates, in many situations they are significantly elevated and in over 20 studies with dose-response trends. In selecting evidence of dose-response trends a threshold of trend $p < 0.1$ was used because they typically have only 3 to 5 points and often have small case numbers. Many of the studies show significant trends, $p < 0.05$. One example is given from Green et al. (1999), Figure 36.

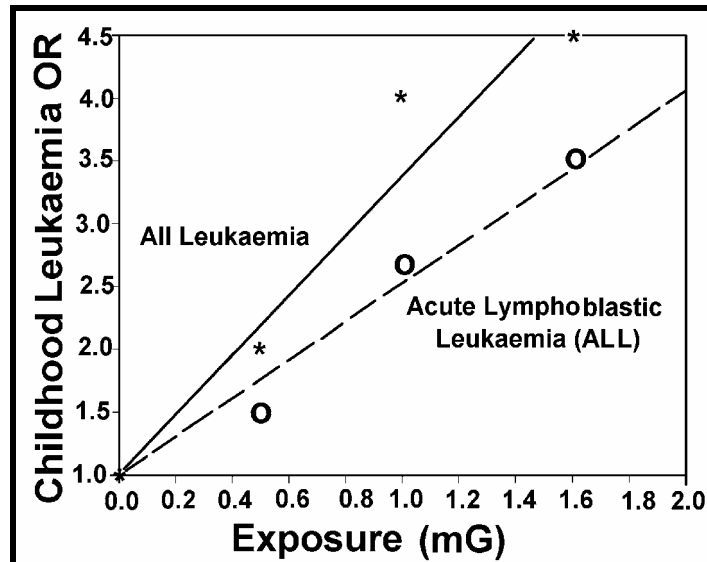


Figure 36: Childhood leukaemia and ALL dose-response relationship in the Canadian Residential related to the measured magnetic fields for the time of diagnosis, Green et al. (29). For the ALL the trend is highly significant, $p < 0.02$ and for all leukaemia, $p < 0.05$.

This definitely shows a causal link between ELF exposures and Childhood Leukaemia. Making adjustments for the Ubiquitous Genotoxic Carcinogen effect, which highlights the No-Exposure Factor, significantly strengthens the causal link. When studies use 1mG or 0.1mG as the reference cut-point they are selecting a group of people whose cancer rate has been progressively raised by living in these fields all their lives and for several generations. The historical cancer rise relationships have been well investigated.

The predominance of Acute Lymphoblastic Leukaemia (ALL) in early childhood, peaking between 2 and 4 years, has provided the proof of the source of the majority of cancer from its temporal and spatial development pattern over the 20th Century. Sir Richard Doll identified a new carcinogenic phenomenon in 1961, Court-Brown and Doll (1961). It was associated with a massive progressive rise of Leukaemia in all age groups considered and was characterized by the early childhood peak, 2-4 years, of ALL. This did not exist in 1910 but was well developed in the UK and Wales by 1930 and in the United States by the 1940's.

A great deal of research has tried to identify the cause of this "biomarker" cancer. Milham and Osslander (2001) showed that the only factor that followed the spatial and temporal development of this childhood ALL peak was the introduction of electrical wiring in homes. Every residential Childhood Leukaemia study showing elevated cancer rates confirms this conclusion. In particular, Kraut et al. (1994)

show significant dose-response increases in childhood Brain Cancer, Leukaemia and Lymphoma in proportion to the level of domestic electrical power supply in Canada. This confirms that the biophysical mechanism is genotoxic and causes a wide range of cancers.

Hatch et al. (1998) found that pregnant mothers who used electric blankets or electric heating pads during their pregnancies had significantly elevated incidence of children with ALL, OR = 1.59 (1.11-2.29), and OR = 1.46 (1.10-1.98), respectively. Hatch et al. also found that the small children's EMF/EMR exposure from the TV produced dose-response increases in ALL with distance from the TV and with hours per day of watching TV. Being less than 6 ft and more than 6 hours, OR = 4.67 (1.64-13.36). For video games connected to the TV for an hour or more a day, OR = 1.87 (1.13-3.10). This finding confirms the early initiation of the cancer *in utero* during pregnancy, and the advancement of the ALL with EMF/EMR exposures after birth. The RF/MW impacts are quite high with large and significant OR's.

Green et al. (1999) independently confirmed these observations. They found a dose-response for All Leukaemia and for ALL for children in Ontario with measured average residential magnetic fields. All Leukaemia rates were doubled from 0.5mG average fields compared with <0.3mG, OR = 2.0 (0.6-6.8). For 1mG it was significantly 4-times higher, OR = 4.0 (1.1-14.4), Figure 36. This confirms that the higher the domestic EMF fields the higher the ALL and All Leukaemia incidence rate.

Feychting and Ahlbom (1993) found a trend relationship between Childhood Leukaemia and the measured magnetic field closest to the time of diagnosis. After extensive analysis of the magnetic field data they chose <1mG as the reference group. The 1-1.9mG group has RR = 2.1 (0.6-6.1), n=4; for ≥2mG, RR = 2.7 (1.0-6.3), n=7 and for ≥3mG, RR= 3.8 (1.4-9.3), n=7. By pooling together a Danish and this Swedish study the results were strengthened Feychting et al. (1995). Retaining the 1mG reference cut-point, for ≥2mG, RR = 2.0 (1.0-4.1), n=10 and ≥5mG, RR = 5.1 (2.1-12.6), n=8, Trend p <0.0001.

Fajardo-Gutierrez et al. (1993) found in Mexico that children living near the high voltage distribution substations had significantly increased Leukaemia rates, OR = 2.63 (1.26-5.36). For children living near power lines, there was OR = 2.5 (0.97-6.67) and near the lower voltage distribution power lines, OR = 2.12 (0.79-5.85). This is effective as a weak dose-response as the higher fields produce higher childhood leukaemia rates.

Over the 20th Century the 0-5 year old cancer rate (per 100,000 p-yrs) has risen from less than 1 to over 8. For 2-3 year olds Leukaemia has risen from less than 1 to over 10. All other age group leukaemia rates have risen in parallel, along with other cancers and many other health effects. The contribution of household wiring and electromagnetic fields is at least 60% of the cancer rise, Milham and Ossiander (2001). Therefore an adjustment factor to reduce the control group rate to deal with this effect, the No-Exposure Factor (NEF) recommended is NEF = 4. For All Childhood Cancer this corresponds to a reference cancer rate of 2 per 100,000 p-

yrs. When using a risk assessment approach to identify the acceptable cancer rate of 1 in a million or 1 in 100,000, then the use of the NEF is vital.

If Hatch et al. and Green et al. had used a conservative non-exposure control rate of 2 per 100,000 p-yrs reducing their control group rate (group size) then their results would be significantly stronger. For example from Hatch et al. watching TV >6 hours at closer than 4 feet has an ALL Odds Ratio of OR = 4.39 (1.75-11.04). If adjusted for the NEF this rises to OR = 17.6. From Green et al., All Leukaemia was elevated from ≥ 1.4 mG exposure to Adj OR = 4.5 (1.3-15.9). Adjusting for the No-Exposure Factor gives OR = 18.0. Both of these papers provide multiple dose-response increases in Leukaemia from a range of ELF/RF/MW exposures, confirming the causal link.

Early Residential radar-exposure cancer studies:

In 1982 Lester and Moore published a study of radar related cancers in residential populations in Wichita, Kansas, based on a hypothesis that radar could produce cancer. This was based on the evidence of chromosome damage and the Zaret (1977) evidence of cancer rates in radar repairing workers. Because there were airport and air force base radars to the east and west of Wichita they used geographic distributions of total cancer incidence on ridges exposed to both radars, sides of hills exposed to only one radar and valleys sheltered from both radars. Mortality data was obtained from the period 1975-1977.

A significant linear trend ($p=0.034$) was found with incident rates (/100,000 p-yrs) of 470, 429 and 303 respectively from high to low RF/MW exposures, Figure 37. They concluded that their results established a correlation between radar exposure and cancer incidence, but that more research was necessary for causation. They were unaware of the Moscow Embassy and the Korean War study results that support and confirm their findings.

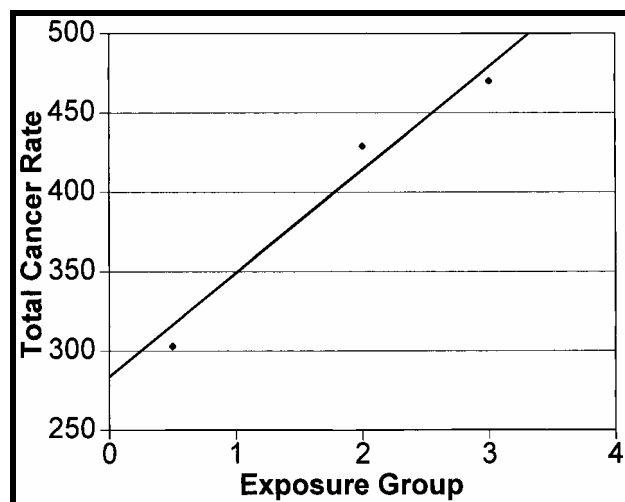


Figure 37: Cancer rates in Wichita, Kansas, for the population not exposed to a radar, exposed to one radar and exposed to two radars, at their residences, Lester and Moore (1982a), Trend $p=0.034$.

They then carried out their own follow-up study to test the hypothesis that cancer mortality is associated, in part, with the possibility of chronic exposure to radar. They studied the cancer rates in 92 counties associated with US Air Force Bases (AFBs) with radars, over the period 1950-1969. They found that counties with AFBs (and radars) had significantly higher cancer rates for males ($p=0.04$) and females ($p=0.02$), Lester and Moore (1982b).

Thus the hypothesis is strongly supported by this study with significant and dose-response increases in All Cancer mortality.

Polish Military study:

The largest and most reliable military RF/MW cancer study was carried out by Professor Stanislaw Szmigielski, Szmigielski (1996) and Szmigielski, Sobiczewska and Kubacki (2001). The Polish military established an RF/MW hygiene regime in 1972 that required RF/MW exposures to be reported and recorded. They have maintained a detailed cancer registry over a longer period than that. The reported and recorded RF/MW exposures placed the personnel into the exposed group. All of the remaining military personnel are used as the reference group.

Matching the data, 1971-1990, shows extremely high levels of leukaemia, $RR = 5.33$, $p<0.01$, and a significant elevation of brain/CNS cancer with $RR = 2.70$, $p<0.01$. With chronic exposure there is cumulative cellular damage that will normally lead to a near linear dose-response when the studied population is large enough. A prospective study was carried out within the second paper involving 36 cases. When ranked according to their peak threshold they show a significant linear trend despite the small sample size, Table 9.

Table 9: The All Cancer peak exposure dose-response from the prospective section of the Polish Military study, Szmigielski, Sobiczewska and Kubacki (2001).

Peak Exposure Threshold	Group (a+b)	Cases (a)	RR	95%CI	p-value
100 μ W/cm ²	3850	36	2.16	1.54-3.02	0.0000046
200 μ W/cm ²	1950	22	2.60	1.70-3.98	0.0000048
600 μ W/cm ²	630	13	4.76	2.76-8.21	$<10^{-7}$
1000 μ W/cm ²	280	6	4.94	2.23-10.96	0.0000015
Trend $p = 0.048$					

The relationship between peak and mean exposures is closer with military occupations than the factor of 200 found between peak and median residential exposures. A factor of 50 is more appropriate giving a range of 2 to 20 μ W/cm² from low to very high exposure in Table 9, but these mean exposures are similar to the range in schools in Vancouver, Figure 26, with the 200 factor.

Conclusions and Recommendations:

The fundamental and classical epidemiological approach recommended by Sir Austin Bradford Hill, along with appropriate consideration of a more accurate exposure assessment approach would result in very different decision outcomes. Appropriate integration of all of the laboratory evidence that electromagnetic fields and radiation are genotoxic would make scientific sense of a very large body of epidemiological studies showing elevated cancer rates, not only in occupational exposures but also in residential exposure situations. This strongly challenges and rejects the modern interpretation that the only effects are shocks and tissue heating. Accepting that epidemiological evidence is the strongest evidence of human health effects, points to the same conclusions. The failure to recognize ubiquitous exposure effect has grossly underestimated and masked the true epidemiological results. An understanding of the radial radio-frequency and microwave exposures around radio/TV towers and cell site's allows dose-response relationships to be found, and all point to a no safe threshold level, consistent with the genotoxic nature of the EMF/EMR signals. Understanding and appreciating that extremely low residential exposure levels to a genotoxic substance causes elevated cancer rates, along with many other health effects caused by mutations and enhanced cell death rates that has the effect of accelerated aging.

When setting public health standards for air pollution health effects, for example, very fine particles which are recognized by the WHO to have dose-responses with a safe level of zero exposure, then a standard authority can choose to set a standard such as $50\mu\text{g}/\text{m}^3$ as a mean daily concentration of PM10, Figure 38.

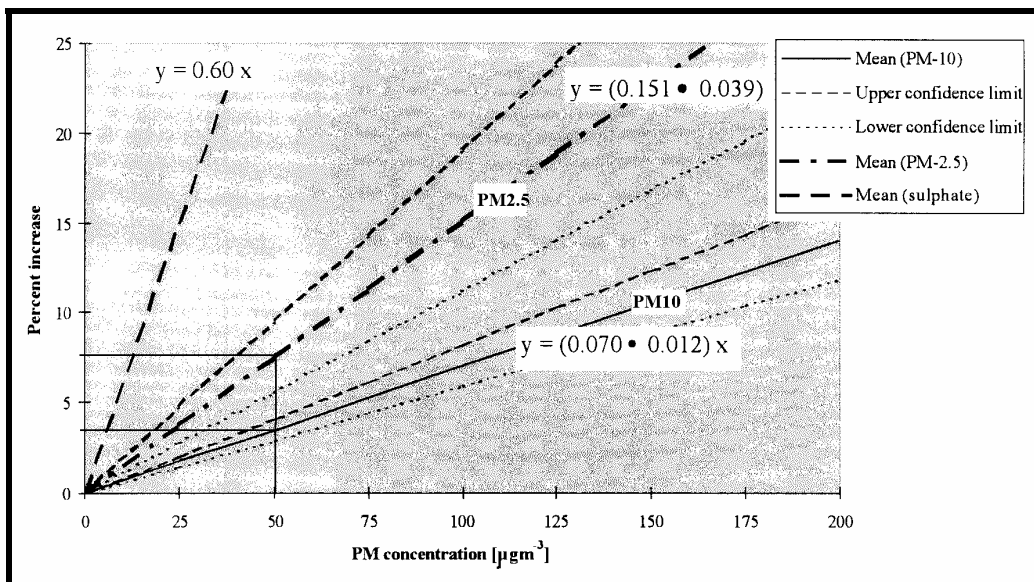


Figure 38: Increase in daily mortality as a function of PM concentration. WHO (1999).

The environmental performance indications of the desired levels can be decided by national or regional standards authorities. The New Zealand Ministry for the Environment promotes an approach set out in Table 10. Where exposure levels are regularly above a practically achievable guideline or standard, for the agents whose safe level is actually zero, then a management plan should be developed to reduce

exposures to have no exceedences of the desired target level. When the 0 exceedences level has been set then further reductions should be sought to progressively reduce the rates of the adverse health effects. Where the exposures are lower than the chosen target level then they should be maintained, and where the community desires, reduced towards the good (33%) or excellent level, 10% of the target standard.

Table 10: Environmental Performance Indicators Programme air quality categories, Ministry for the Environment.

Category	Measured value	Comment
Action	Exceeds the guideline value	Exceedences of the guideline are a cause for concern and warrant action if they occur on a regular basis.
Alert	Between 66% and 100% of the guideline value	This is a warning level, which can lead to exceedences if trends are not curbed.
Acceptable	Between 33% and 66% of the guideline value	This is a broad category, where maximum values might be of concern in some sensitive locations, but are generally <i>at a</i> level that does not warrant dramatic action.
Good	Between 10% and 33% of the guideline value	Peak measurements in this range are unlikely to affect air quality.
Excellent	Less than 10% of the guideline value	Of little concern: if maximum values are less than a 10th of the guideline, average values are likely to be much less.

There is strong and robust evidence that there is a causal relationship between chronic exposure to electromagnetic fields and radiation of human populations and elevated rates of serious health effects, because they are genotoxic. This requires major reductions in the allowable public and occupational health protection exposure standards. Their levels should be set at such values that chronic exposure does not result in detectable elevation of cancer, cardiac, reproductive and neurological health effects, once the ubiquitous exposures have been reduced.

For residential ELF exposures in the community, a desirable and achievable Standard level is 1mG (0.1µT), with a Good level of 0.33mG and an Excellent level of 0.1mG.

For residential radio-frequency and microwave exposures in the community an initial desirable and achievable direct external standard level of $0.1\mu\text{W}/\text{cm}^2$ is recommended, as it is applied in Salzburg, Austria. Because their signals are genotoxic carcinogens there is no safe threshold and the exposures need to be minimized.

An excellent level is $0.01\mu\text{W}/\text{cm}^2$. With the large reduction of indoor exposures of less than 2% of the outdoor exposure, away from windows facing a source antenna, allowing for inside and outside, and home and away activity a mean level of 5% can be achieved, which for an excellent situation would result in a mean exposure of about $0.5\text{nW}/\text{cm}^2$.

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