

A Summary of the Current Understanding of the Risk of Exposure to ELF magnetic fields.

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Introduction

This document represents a *summary* of my evaluation of the scientific evidence of the risk posed by exposure to environmental magnetic fields resulting from the generation, transmission, distribution and use of electric power. This evaluation is based on almost twenty years of experience in this field, both as a researcher and as a public health scientist charged with analyzing and interpreting the relevant evidence. In the latter role, I participated directly in two recently evaluations, conducted respectively by the International Agency for Research on Cancer and by the California Department of Health Services. I was also intimately involved in the evaluation conducted by the US National Institute of Environmental Health Sciences.

Although this document is factually and scientifically accurate, supported by appropriate scientific references, I have tried to use a narrative form of expression and to avoid scientific jargon. To this extent, the language may be, at times, somewhat ‘unscientific’. For example, the acronyms ELF and EMF are used interchangeably to denote *magnetic fields of extremely low frequencies*.

Is the belief in the hazards of EMF unfounded?

Absolutely not. The evidence of risk is getting stronger to the point where the leading international authority on cancer, the International Agency for Research on Cancer (IARC), an agency of the World Health Organization (WHO), has formally classified ELF-EMF as ‘possibly carcinogenic to humans’ [IARC 2002], the same category which includes marine diesel fuel, engine exhaust, gasoline, residual heavy fuel oils, gasoline, and polybrominated biphenyls (PCBs). Ironically, engine exhaust and PCBs (used as dielectric fluids in transformers) were suggested by skeptics as possible explanations for the observed association between residence near power lines and cancer.

Over the past few years, the position of governmental agencies of the US and UK has shifted from being skeptical to admitting the possibility of a real risk. In 1997 the US National Research Council (an organ of the National Academy of Science), in its report “Possible Health Effects of Exposure to Residential Electric and Magnetic Fields” found an association between “surrogate measurements of exposure and childhood leukemia” [NAS 1997]. The fact that the association was found with *surrogate* measurements (the so-called ‘wire code paradox’) was regarded as arguing against a true causal link. However in 1998 a Working Group convened by the US National Institute of Environmental Health Sciences (NIEHS) classified EMF as a possible carcinogen [NIEHS 1998]. In 2000 the ‘wire code paradox’ was proven illusory by Greenland et al [2000]. In 2001 the UK National Radiological Protection Board (NRPB) concluded that more recent studies “suggest that relatively heavy average exposure of 0.4 microtesla [4

mG] or more are associated with a doubling of the risk of leukemia in children under 15 years of age.” And that “unless...further research indicates that the finding is due to chance or some currently unrecognized artifact, the possibility remains that intense and prolonged exposure to magnetic fields can increase the risk of leukemia in children.” [Doll et al 2001]

In the fall of 2002, the Department of Health Services (DHS) of the State of California released a Risk Evaluation [DHS 2002] carried out on behalf of the California Public Utilities Commission which stated:

- To one degree or another, all three of the DHS scientists [who authored the report] are inclined to believe that EMFs can cause some degree of increased risk of childhood leukemia, adult brain cancer, Lou Gehrig’s Disease, and miscarriage.
- They strongly believe that EMFs do not increase the risk of birth defects, or low birth weight.
- They strongly believe that EMFs are not universal carcinogens, since there are a number of cancer types that are not associated with EMF exposure.
- To one degree or another they are inclined to believe that EMFs do not cause an increased risk of breast cancer, heart disease, Alzheimer’s Disease, depression, or symptoms attributed by some to a sensitivity to EMFs.
- All three scientists had judgments that were "close to the dividing line between believing and not believing" that EMFs cause some degree of increased risk of suicide.
- For adult leukemia, two of the scientists are "close to the dividing line between believing or not believing" and one was "prone to believe" that EMFs cause some degree of increased risk.

In *my own* professional judgement, I strongly believe in an increase in risk the of childhood leukemia. I am also inclined to believe that EMFs pose an increased risk of adult leukemia, adult brain cancer, Lou Gehrig’s disease and miscarriage.

How strong needs the evidence to be?

This question was specifically addressed by the California EMF Program in its innovative evaluation. We called this thought process a ‘qualitative Bayesian evaluation’, since it was inspired by a statistical tool, Bayes’ theorem. We realized that experts in this field could reach different conclusions for two reasons (or a combination of the two):

1. They were *a priori* more or less likely to regard exposure to EMF as a risk
2. They regarded the evidence as more or less convincing.

By *a priori* belief, we mean an opinion based on general scientific knowledge, but not considering the evidence derived by studies targeted specifically to address this issue. Some reviewers, particularly those with a physical or engineering background, have an *a priori* belief of virtually zero, because they argue that the environmental fields are so weak that they cannot be perceived above the endogenous electromagnetic forces existing in the living body. Consequently, they remain skeptical even in the face of strong evidence and they try to explain it away as due to traffic fumes or PBCs from transformers (neither of which is a proven carcinogenic, as noted above), socio-economic status or other unspecified reasons.

However, this argument is flawed. Power line fields are qualitatively different from endogenous currents. They are temporally and spatially coherent, that is, their oscillations are 'in step' and the length of the step (period) is uniform and constant. This means that millions of cells throughout the body can be stimulated at the *exact same time*. It also means that each cell is stimulated repeatedly at a very regular time interval. Neither of these phenomena occurs with endogenous, naturally occurring fields. Therefore, it is perfectly possible for the organism to distinguish one type of fields from the other, even if one is much weaker than the other. Man achieves a similar result everyday through man-made signal-to-noise enhancers.

In fact, even *before* examining any direct evidence of the hazards of ELF fields, we have reasons to be at least a little wary of them. Once we realize that weak man-made fields can be perceived and distinguished from natural electric signals, we must allow for three possibilities: exposure is beneficial, exposure is indifferent or exposure is harmful. The probability that environmental EMFs are beneficial is very small because of the extraordinary coincidence that would be required for a complex organism to benefit from something that was totally absent during its evolutionary development. The probability that extraneous electrical signals leave an organism totally unperturbed also is very small, since the organism depends on electrical signals for its proper functioning.

Then there is the question of dose and size of effect. If there is a disruption of the normal electrical signals between cells, can its effects be repaired before a macroscopic effect ensues? I have no basis to believe that repair mechanisms against an unknown and totally alien agent may have evolved by accident. However, if the dose and the resulting response are small and easily tolerated, then pathological results could be seen only in a very few subjects who, either by chance or extraordinary vulnerability, are not able to tolerate these small effects. (This is analogous to saying that exposure to a common cold virus carries a very small risk of death).

How should we regard the dose received by living near powerlines? The dose may be considered in relative terms. One argument is that exposure levels are low, compared to the range of possible values that the field may assume. Another is to believe that, since ELF fields are virtually all man made, an increase from virtually zero to several mG represents a massive increase in dose that is not easily tolerated. Therefore, it is not obvious that environmental exposure is necessarily low, simply because it takes sensitive instruments to measure it. Admittedly, the introduction of electricity has not caused large

scale epidemics of cancer and other diseases, therefore the effects are either weak or limited to the areas very close to the sources or to individuals particularly sensitive. But, *even if we had no direct evidence of a risk*, we have reasons to believe that there is a small but non-negligible probability (say about 10%) that EMF poses a moderate health risk. Therefore, I strongly disagree with the proposition that the hypothesis the environmental EMF is extraordinary, and that it requires extraordinary evidence to prove it. I believe that the evidence we have is stronger than that normally required to be *at least* concerned about an environmental pollutant.

What is the major evidence in support of this hypothesis?

IARC, NIEHS, NRPB and the California DHS agree that the major evidence comes from epidemiological studies. The most recent and, therefore, the most complete formal review of this evidence is in the California EMF Program's Final Report [DHS 2002].

The epidemiological evidence became much more convincing when two independent teams of eminent epidemiologists pooled together data from the single studies on childhood leukemia into large data bases that provide more statistically stable results. Both of these 'pooled analyses' [Greenland et al 2000, Ahlbom et al 2000] have concluded that there is a consistent elevation in the risk of leukemia in children exposed to magnetic fields of 3-4 mG or more. Neither of these studies included all the data available, therefore they are not replication of each other and they reinforce each other's findings.

Is this evidence convincing?

I believe it is, because the alternative explanations are not. Other than causation, the possible explanations for the observed association between ELF exposure and cancer are chance or bias.

The two pooled analyses and many meta-analyses (a statistical method to combine the results studies from separate studies) have ruled out chance as a plausible explanation.

A special kind of bias is called confounding, that is the fact that, if two exposures are commonly found together (e.g., power lines and traffic), one may be assumed erroneously to be the cause of a disease, while the other is the real cause. Most of the studies on ELF and childhood leukemia have controlled for all known and suspected confounding factors and have concluded that these could not explain the association.

Both confounding and bias could operate in either direction. All studies suffer from a form of bias (exposure misclassification) that tends to *reduce* the estimate of risk (rather than inflate it or artificially create an association). This has been acknowledged in the IARC report [IARC 2002, pg 333].

Two common causes of bias are subject selection and subject non-participation. For example, if subjects are recruited by phone, some demographic groups are less likely to be contacted. Some demographic groups may be less inclined to participate because of

lack of time or interest. If the study groups are not representative of the general community, the study's results could be biased. However, not all childhood leukemia studies used the same methods to recruit participants or ensure their participation, yet the results are remarkably consistent. Furthermore, the effect of known or suspected biases in these studies has also been evaluated. In *some* studies, it has been determined that bias can account for *part* of the association [Hatch et al 2000], but in no case has the study been completely explained by bias.

The IARC report [IARC 2002] concludes: "It cannot be excluded that a combination of selection bias, some degree of confounding and chance could explain the results". I regard this as an extremely unlikely possibility. The *existence* of bias due to *unknown and unsuspected* factors cannot be ruled out and I agree that if we had *one* single large study we could not rule out that a number of factors could combine to create a misleading result. However, we do not have a single study on childhood leukemia; we have about 20 studies, conducted by different investigators, at different times, in different countries. There is no reason to believe that they are all affected by the same bias (that nobody can identify) or that independent, different biases would all tend systematically to increase the risk. If we look at the pattern of the results of all the childhood study published until the year 2000 (see Table 1) we note a consistency that cannot be reasonably attributed to unidentified random factors.

Table 1 – Synopsis of childhood leukemia studies published between 1979 and 2000.

Study #	Country	Author	Risk estimate	Binary outcome for exposure > 3 mG
1	UK	Coghill	no controls	?
2	New Zealand	Dockerty	no controls	+
3	Sweden	Feychting	4.44	+
4	USA	Linnet	1.51	?
5	USA	London	1.53	+
6	Canada	McBride	1.42	+
7	Germany	Michaelis	2.48	+
8	Denmark	Olsen	2.00	+
9	USA	Savitz	3.87	+
10	Sweden	Tomenius	1.41	+
11	Norway	Tynes	no cases	?
12	Finland	Verkasalo	2.00	+
13	Canada	Green	1.23	+
14	UK	UK	0.97	-
Non-measurement studies			Risk Estimate for <i>high</i> vs. <i>low</i> exposure	Binary outcome for <i>high</i> vs. <i>low</i> exposure
15	Wertheimer	USA	>1	+
16	Fajardo	Mexico	>1	+
17	Coleman	UK	>1	+
18	Petridou	Greece	>1	+

We can use a statistical tool called binomial analysis to verify if such a consistent pattern is consistent with what epidemiology calls the “null hypothesis”. The “null hypothesis” is a jargon expression to mean that ‘nothing is going on’. If we had a very large number of epidemiological studies, all perfectly free from bias and we had an approximately equal number of studies showing an increase in risk as there are studies *not* showing an increase in risk, we can conclude that the “null hypothesis” is verified. If we only have a small number of studies, we can expect that the pattern of result to be skewed one way or another. Mathematically, we can calculate the likelihood of a very skewed pattern of results *still consistent with the null hypothesis*. In some cases, the skew is so extreme that, under the laws of statistics, *we must reject* the null hypothesis.

It is useful to compare the epidemiological studies on childhood leukemia to gambling dice and the risk of disease to the risk of ‘being cheated’. The *null hypothesis* is that these dice are ‘fair’. One alternative hypothesis is that we are being cheated. (In the analogy, this is the equivalent to the hypothesis that EMF is hazardous). Yet another alternative hypothesis is that nobody is cheating us, but that the dice are cheaply made and this makes the playing odds somewhat skewed (this is equivalent to saying that EMF is not hazardous, but the biases in the studies make the pattern of result skewed).

Table 2 – Analogy between epidemiological studies and a game of chance

	Null hypothesis	Altern. Hypothesis 1	Altern. Hypothesis 2
Epidemiological studies	There is no problem	EMF is hazardous	The studies are flawed
Gambling dice	There is no problem	The dice have been deliberately <i>loaded</i>	The dice are flawed, but not <i>loaded</i>

Under the null hypothesis, if I throw a die a large number of times, I expect that half the time the die would land on half the times on an even number and half the time on an odd number. If the number of throws is small, we may expect the results to be less evenly divided.

If I throw one die a very large number of times and I observe an extreme number of throws returning an even number, I reject the null hypothesis. Something is going on, but I cannot decide if I am being deliberately cheated or I am using a cheap, unreliable die. But suppose I ask for a new die after each throw and I throw each die once and only once. If they are cheaply made, I still expect to see an approximately equal number of odds and even numbers because I do not expect random irregularities to ‘conspire’ against me. If an unusually high number of them land on an even number, I have reason to believe that they have been deliberately tampered with, i.e., I believe that Alternative Hypothesis 1 is verified.

In the case of the childhood leukemia studies, we have one study showing no risk increase, but 14 showing a risk increase (three studies were too small to estimate a risk). Statistics tell us that the chances of a pattern of results as, or more extreme than this is less than 5 in 10,000. Therefore, *remembering that biases can affect the results either*

way, it is unreasonable to still believe that each study is affected by some unidentified flaw and that, *by chance*, they all act in the same direction to create an association that does not exist.

One can also explore some worst-case scenario and still conclude that the association between exposure and disease stands up to any reasonable scrutiny. For example, let us concede (despite evidence to the contrary) that *all* the measurement-based US studies can be totally explained by selection and non-participation bias (this assumption goes well beyond the conclusion of Hatch et al, [2000]). Therefore let us lump them all together and regard them as a single, negative study. Then, we would have a total of 13 studies with eleven showing a risk increase and two not showing it. This is still a very extreme pattern ($p < 0.0112$, i.e., there is only a 1% chance of getting a pattern more extreme than this). Nobody has been able to suggest a quantitative scenario which shows that bias and/or chance is a more likely reality than causation.

Finally, a study (Schuz 2001) published too late to be included in the pooled analyses [Greenland 2000, Ahlbom 2000] not only reinforces the pattern of positive results summarized in Table 1, but also offers evidence in favor of causality and against a biased result: the study shows a clearer and stronger association when subjects are classified according to the exposure measured at night, rather than the 24-h average home fields. This is easily explained by the hypothesis that magnetic fields are responsible for an increase in risk: during the day, the children are not always in the home, therefore their exposure is not well characterized; hence the association is weakened [Flegal 1991]. At night, the exposure of the child is well measured by measurements in the bedroom, therefore the classification of exposed vs unexposed children is sharper and the risk estimate is stronger and clearer.

However, not even the author of the study could think of a reason why any bias could have affected night-time measurements more than 24-h measurements [Schuz, personal communication]

What is the major evidence against the hypothesis? Is it convincing?

Traditionally, cancer risk assessors have had more animal evidence to evaluate than human evidence. This is not the case with ELF and cancer. This is not to say that there are not animal studies supporting the hypothesis of risk. During the deliberations of the IARC Working Group in June 2001, one third of the members stated that 'there was limited animal evidence of carcinogenicity'. However, the other members disagreed and the majority conclusion was that the animal evidence was inadequate. This decision was probably the main reason why IARC [2002] classified ELF as a 'possible' rather than a 'probable' carcinogen.

The California EMF program took a different point of view: the California reviewers realized that some lines of research are prone to false positives and some are prone to false negatives. It is very likely that in a complex experiment about a poorly understood disease, like cancer, and a very complex agent, as an electromagnetic field, not all

conditions reproducing the real world can be met. To begin with, the right animal model must be found. In the famous case of smoking and lung cancer, numerous experiments failed to induce lung cancer in the typical experimental animals (rhesus monkeys, baboons, golden hamsters, rats) [Stewart et al 1962, Coggins 2002], but the experiments were eventually successful when a very unusual animal model was used, namely baby beagles [Auerbach 1970].

Second, environmental ELF waves are as complex as the sound waves emitted by a stereo system. To characterize them by measuring the average intensity is equivalent to describing a piece of music by measuring the average sound volume. Therefore, the fields used in laboratories, even if of similar intensity as the environmental fields, may be quite different in many other respects. Another health-related example is the role of diet in increasing or reducing the cancer risk. We know from observational studies that the Mediterranean diet is generally healthier than the American diet, but, if we were to translate this into an animal experiment, we don't know how to replicate these diets. Not all fats are equal, not all alcoholic beverages are equal, and not all dietary fibers are equal and not all 3 mG ELF fields are equal.

A glaring example of the difficulties facing animal experimenters is posed by the comparison of the studies on EMF and breast cancer in rats carried out by Dr. Losher and associates in Germany [Losher et al 1993, 1994, 1997, 1998] and the Battelle team in the US [Anderson et al 1999]. After a lengthy debate, scientists from both teams co-authored a paper examining the reasons for the differences in their results [Anderson et al 2000]. The possible explanations they offer include the use of different substrains of Sprague-Dawley rats (the U.S. rats were more susceptible to DMBA than the European rats), different sources for diet and DMBA, differences in environmental conditions, and differences in MF exposure metrics.

In summary, while the few positive animal results cannot be easily explained away, it is easy to find limitations in the studies that could not find an effect. In view of these arguments, the California DHS reviewers decided that the paucity of animal results was not a valid argument to refute the epidemiological evidence [DHS 2002, pg 19 and following].

Is childhood leukemia the only health risk?

The evidence of an increased risk of other adverse health effects is more sparse, but not necessarily weaker. For these other diseases, we do not have anything comparable to the pooled analyses recently conducted using the childhood leukemia data. However in some cases there are enough studies to see a consistent pattern of results, virtually ruling out chance as an explanation. Unlikely chemical carcinogens, which are restricted to specific biological pathways, magnetic fields reach all parts of the body and, therefore, they may affect many different biological processes. Once one accepts that ELF may cause an increase in childhood leukemia, evidence of other health effects becomes more credible. Both the NIEHS and the California DHS report acknowledge that magnetic fields may be

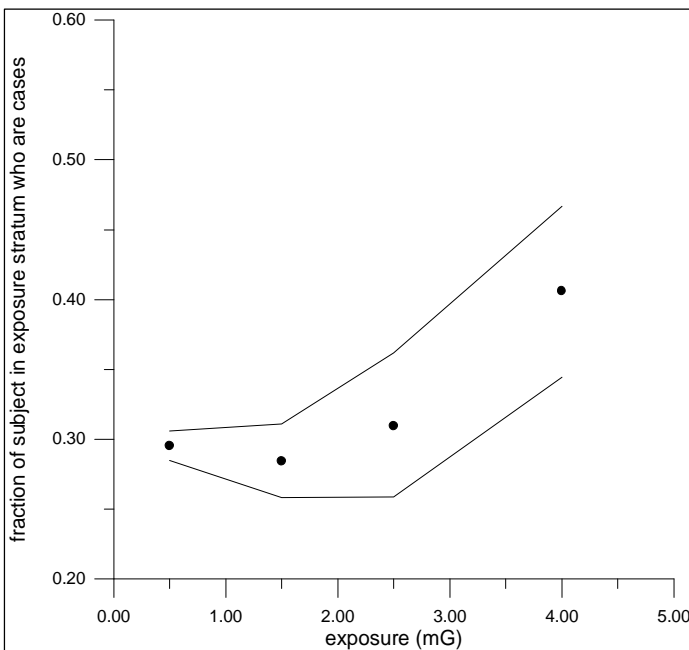
responsible for other health risks, while IARC regards the evidence as inadequate to reach a conclusion.

What is a safe level of exposure?

According to both the pooled analyses, the leukemia risk appears in children resident in houses where a field of 3-4 mG was measured. This does not mean that there is a sharp threshold between safe and hazardous levels.

Merging data from the UK study with the pooled data of the Greenland analysis [Greenland 2000], I produced the following *approximate* plot. The vertical axis displays the fraction of study subjects who are cancer cases. This is a measure of risk as a function of exposure: if there were no risk, the fraction would stay the same irrespective of exposure. Instead we see that this ratio begins to increase from about 2.5 mG. The dots represent point estimates; the continuous lines denote the 95% uncertainty limits.

I must stress that in this representation, subjects with exposure between two values are all assigned the midpoint exposure between those two values (for example subjects with exposure between 2 and 3 mG are grouped together and assigned a nominal exposure of 2.5 mG). Therefore this is an intrinsically approximate representation. Nevertheless, it suggests that it is prudent to avoid exposure above 2 mG.



These data refer to childhood leukemia. We do not have adequate data for other diseases. In the absence of more specific evidence, it is reasonable to assume *tentatively* that a similar exposure-response relationship may exist for the other conditions that epidemiological data have associated with ELF exposure.

Are there guidelines or standards we can rely on?

The current guidelines published by the International Commission on Non-Ionizing Radiation

Protection ICNIRP were published in 1998 and did not consider any study published after 1997 (note also that all of the so-called *recent* reviews quoted by ICNIRP (NRPB 1992, 1994b, NAS 1996, CRP 1997) have been superseded by more recent evaluations reaffirming the validity of the epidemiological evidence). Even then, they acknowledged the existence of epidemiological studies on EMF and cancer but concluded that this evidence “is not strong enough in the absence of support from experimental research to

form a scientific basis for setting exposure guidelines.” In its own most recent review of this evidence [ICNIRP 2001], ICNIRP’s standing committee on Epidemiology concludes that “despite 20 years of extensive epidemiologic investigation of the relation of EMF to risk of chronic disease, there are still epidemiologic questions that need to be resolved” and that the association between EMF and childhood leukemia is “unlikely to be due to chance but *may be, in part*, due to bias” (emphasis added). Therefore, since the commission admits that there is some evidence that cannot be explained away, but that is not sufficient to set safety guidelines, it is difficult to see how ICNIRP decided to issue guidelines anyway or how they intend these guidelines should be used. The ICNIRP guidelines (adopted by the Australian NHMRC) are based purely on well understood immediate biological effects resulting from acute exposure to electric or magnetic fields. Therefore, while they are an excellent guide to avoid immediate dangers, they do not offer any reassurance regarding the long term effects of chronic exposure. To this extent I agree with the statement of the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA): “There are currently no Australian standards regulating exposure to these fields. The National Health and Medical Research Council has issued *Interim guidelines on limits of exposure to 50/60 Hz electric and magnetic fields*. These guidelines are aimed at preventing *immediate* health effects resulting from exposure to these fields” (ARPANSA Web page. emphasis added).

In summary, the only way to minimize the risks of chronic EMF exposure is to use common sense prudence. This approach may well result in increased costs that need to be balanced against the benefits of possible mortality and morbidity avoided. Other benefits to be considered are the avoidance of possible future liability, increased property values and resulting revenues etc. The California EMF Program has produced two thorough analyses of the policy options, supported by comprehensive computer models [DHS 2002b]. To my knowledge, these are the most up-to-date and all-inclusive tools on this topic available to the decision makers.

Regarding the specific situations of the Benson family, I offer the following comments:

- Personally, I would be concerned to spend repeated, prolonged periods of time in fields greater than 2 mG. I would not let children play in areas where such fields exist. Such fields will exist in the Benson property if the power line is built.
- Assuming the calculations carried out by PowerLink are correct, the Bensons’ house, at its nearest side of the easement boundary, will be exposed to EMFs higher than at present. Given the well founded concerns about EMF exposure, I do not think this is a desirable prospect. The ICNIRP argument cuts both ways: the evidence we have is not sufficient to determine what levels are safe – by the same token, it is not sufficient to determine what levels are hazardous.
- Since the human epidemiological evidence was collected using average fields as a marker for exposure, PowerLink’s practice of averaging the yearly fields

can be regarded as a reasonable approach to predict risk. This is no more exact or no more reassuring than saying that, based on observations, reducing fats in our diet decreases the risk of cardiovascular diseases. It probably will, but lumping all fats together in one category is a coarse approximation. Some fats are more harmful than others and some fats may actually decrease the risk. We don't know enough about the effects of the many factors that constitute the "EMF mixture" to decide what and how should be measured to determine what is safe and what is not. For example, a recent study [Li, et al 2002] has found an association between maximum daily exposure and miscarriage.

- Individuals with average exposures above about 2 mG have been found to be at increased risk of childhood leukemia. According to the information provided, levels exceeding this level will exist in the Benson land.
- I disagree with the assertion by Powerlink that the strength of the MFs in the Bensons' house generated by the transmission of electricity over of power lines is likely to be comparable with the strength of MFs already existing in the Bensons' house. In my experience, fields in a house away from power lines are very low, lower than 0.4 mG. Higher fields may exist near appliances and some types of wiring, but these are localized fields, unlikely to affect the long-term exposure of the occupants. In any event, adding another source of exposure is never an attractive proposition.
- If PowerLink's calculations are correct, the line itself would not cause, in the house, exposures similar to those that have been associated with an increase in the leukemia risk. However, the power line fields will be in addition to those already existing in the dwelling. If localized fields already exist at critical points in the house (eg, near a bed), the contribution of the power line may well be crucial. In determining alternative solutions (eg, undergrounding, rerouting) one should also consider the potential health benefits that may be achieved in view of future development of the land.

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