Report to the Workplace Safety and Insurance Appeals Tribunal

Breast Cancer and Occupational Exposure to Electromagnetic Fields

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On October 28, 2008, Mr. Gary Newhouse asked me to address the following questions:

1. What is the current level of evidence that EMF and/or ELF is cancer causing or promoting, with particular reference to breast cancer? Please explain.

2. What is your opinion on this comment from Dr. M. Bitran found at page 14 of Exhibit 20:

> *Comprehensive reviews of epidemiological and laboratory studies carried out by authoritative organizations have consistently concluded that the evidence does not substantiate a cause-effect link between ELF magnetic fields and cancer. Recent epidemiological studies on breast cancer and occupational and residential exposure to ELF magnetic fields are negative on balance. A meta-analysis found a small risk increase that may be due to artifacts. Recent reviews of epidemiological and animal data conclude that ELF magnetic fields are most likely not a risk factor in breast cancer.*

3. What is your opinion on this comment from Dr. M. Bitran found at page 23 of Exhibit 20:

> *The link between ELF magnetic field exposure and female breast cancer has become more tenuous as newer, larger epidemiological studies with better exposure assessment have become available.*

4. Please comment on the elements of average exposure, transient peak impact exposures, and cumulative dose estimates of exposure for each of the three workers in terms of the relationship between those exposures and whether or not exposure to EMF would have been a significant contributing factor in the development or onset of breast cancer in each case.

My expert testimony regarding each of these questions follows:

Question 1. What is the current level of evidence that EMF and/or ELF is cancer causing or promoting, with particular reference to breast cancer? Please explain.

1. We have evidence from **epidemiological** studies of an associated between increased breast cancer and exposure of both men and women to low frequency magnetic fields.

2. We have evidence from **in vivo** animals studies that low frequency magnetic fields are mammary tumor promoters between 1 to 500 mG and tumor inhibitors at higher magnetic fields.

3. We have evidence from **in vitro** studies that magnetic fields at 12 mG stimulate growth of breast cancer cells and reduce the effectiveness of both melatonin and tamoxifen in estrogen receptor-positive (ER+) human breast cancer cells.

4. We also have a plausible **mechanistic model** that explains the relationship between exposure to electromagnetic fields, melatonin, estrogen, and breast cancer growth in both animals and humans.

Each of these will be explained in order and supported by evidence from the scientific literature.

1.1 Epidemiological Studies

Epidemiological studies are design to show whether a statistically significant **association** exists between an **agent** (in this case, extremely low frequency (ELF) magnetic field) and an **outcome** (such as breast cancer). The strength of epidemiological studies is that they involve realistic exposures and document human responses. However, these studies may also have confounders (other factors that may affect the results) that need to be considered and eliminated where possible. Due to the genetic variability of populations and the variability of exposures these studies require statistical testing and large populations for greater accuracy. Often multiple studies need to show a similar association before they are taken seriously. One important point is that these studies show **associations** and are not designed to show **causation** (as do *in vivo* experimental studies) nor are they designed to provide information on the **mechanism** of action (as do *in vitro* studies).

To date, there are more than 18 epidemiological studies (of which I am aware) showing an association between breast cancer and occupational low frequency electromagnetic field exposure in both men (6 studies, Table 1) and women (12 studies, Table 2).

The relative risk for men (Table 1) ranges from 0.7 to 9 with one study showing a 100-fold increase in risk (Milham 2004). This last study was of 3 men who worked in a basement office near electrical switchgear and developed breast cancer (when 0.03 were expected in that population). They were exposed to 92 mG magnetic field in their office.

This form of cancer is rare among men (incidence 1/100,000 according to the Office of Epidemiology and Health Surveillance (1991) and the presence of one or two cases is likely to result in a high-risk estimate. While the risks are high in these studies, they account for a

handful of men developing this type of cancer. Erren (2001) did a meta-analysis of fifteen male breast cancer/EMF studies and reported, ‘‘A fairly homogeneous increased risk for men . . .” with a pooled relative risk of 1.37 (95% confidence interval 1.11–1.71). Despite this the author concludes that results from individual studies are variable and in part contradictory which inhibits valid conclusions about an association of breast cancer and EMF exposure.

The relative risk for women (Table 2) ranges from 0.7 to 4.6 with one study showing a value of 7.4 for women under the age of 50 with estrogen receptor positive (ER+) breast cancer. Many of studies report risk factors that are less than 2. A risk factor of 1.8 is an 80% increased risk. A risk factor of 2 is a doubling or a 100% increase risk of developing breast cancer. While these risks are low, the fact that the incidence of breast cancer among women is high indicates that this additional increased risk can account for a large population of women. In 2001, 1 in 10 women in Canada would be expected to develop breast cancer in her lifetime (Friedenreich et al. 2001). Today I believe it is 1 in 9 women.

In studies that do multiple comparisons (Van Wijngaarden et al. 2001, for example), we can tease out the importance of menopausal status and estrogen-responsive status. These data show that risk increases with years at work (10-20 years), for premenopausal women, with ER receptor positive breast cancer and that a risk is observed at levels of 1.6 mG-y exposure. Despite these data these authors conclude, “These findings give little support to the hypothesis that electromagnetic fields cause cancer of the female breast.” While this statement is technically correct -- epidemiological studies are not designed to test for causality -- this is a misleading and irresponsible conclusion since the data show a statistically significant association with exposure to magnetic fields especially for ER+ premenopausal women who have been exposed for 10-20 years.

One potential criticism of the studies selected for Tables 1 and 2 is that they do not include all studies of breast cancer and the studies missing are those that show no adverse associations.

To address this issue I would like to quote from a document written by Dr. Martin Blank at Columbia University (Blank 2007).

> *We should be reminded that ‘scientific proof’ is not symmetric (Popper, 1959).*
>
> *One cannot prove that EMF is harmless no matter how many negative results one presents. One single reproducible ([statistically] significant) harmful effect would outweigh all the negative results.*
>
> *Scientific method is not democratic. The word ‘proof’ in ‘scientific proof’ is best understood in terms of its older meaning of ‘test’. It does not rely on an adversarial ‘weight of the evidence’, where opposing results and arguments are presented and compared. Answers do not come from keeping a scoreboard of positive versus negative results* [note: positive and negative results refers to studies with and without statistically significant effects] *and merely tallying the numbers to get a score.*
>
> *The above characteristics of science are generally acknowledged to be valid as*

abstract principles, but in EMF research, it has been quite common to list positive and negative findings and thereby imply equal weights. . . .

Negative studies play an important role in science, and there is good reason to publish them when they are failures to replicate earlier positive results. This can often lead to important clarifications of the effect, the technique, etc.

However, negative studies are being used in another way. Although they cannot prove there is no positive effect, they do have an influence in the unscientific 'weight of evidence approach'. In epidemiology, where it is difficult to compare studies done under different conditions, it is common to make a table of the positive and negative results. The simple listing has the effect of a tally, and the overall score substitutes for an evaluation. In any case, one can write that the evidence is 'not consistent', 'not convincing' or claims are 'unsubstantiated' and therefore 'unproven'. The same is true in experimental studies . . . the contradictory results are juxtaposed and a draw is implied. This is a relatively cheap but effective way to neutralize or negate a positive study (Blank 2007).

I provide an analogy of what this means. Not all prospectors for gold will find gold in an area. Two things have to happen. The gold has to be present and the prospector has to find it. If gold is found in a particular location it may indicate a richer vein nearby or it may be that the gold was brought to that area by water or some other source. So one nugget of gold does not indicate a gold mine. Irrespective of how many prospectors fail to find gold, if multiple prospectors begin to find gold you can bet gold exists in that region.

We now have several studies showing a statistically significant association between breast cancer and magnetic field exposure in both men and women. These data cannot be dismissed because other studies find no association. Also, these studies do not demonstrate causation although they may imply it.

ELF magnetic fields are classified as a Class 2b carcinogen, possibly carcinogenic, based on residential exposure and the increased risk of childhood leukemia and on occupational exposure and adult leukemia. At the time the NIEHS (US National Institute of Environmental Health Sciences) and other international bodies accepted this classification for low frequency magnetic fields, there were a total of 13 childhood epidemiological studies that were deemed to be of sufficient quality to be assessed and of these 6 showed a statistically significant association of childhood leukemia and residential magnetic field exposure. There were also six meta-analyses of which five showed a statistically significant increased risk, albeit small (Havas 2000). The international consensus accepts that a magnetic field of 2-4 mG is associated with a two-fold (or 100%) increased risk of children developing leukemia. Since this classification was accepted, more studies have confirmed the link between childhood leukemia and residential magnetic field exposure.

In Tables 1 and 2, there are 18 epidemiological studies (not 6 as above for childhood leukemia) showing an increased risk of breast cancer with occupational EMF exposure either through job classifications, measurements or estimates of exposure. And unlike for childhood leukemia

where experimental studies neither support nor refute the association with magnetic fields, for breast cancer, experimental studies (both *in vivo* and *in vitro*) show a cause effect relationship and point to possible mechanisms of action (see next section).

Concluding Remarks Epidemiological Studies: Epidemiological studies show a small increased risk (median value for statistically significant association is 1.5 or 50% increase risk) for women developing breast cancer who are exposed to magnetic fields that range from less than 1.6 mG to greater than 4 mG. The risk seems to be greater with longer exposure and for premenopausal women, presumably because they are still producing estrogen. It may also be greater for women with ER+ breast cancer. Since the incidence of breast cancer is high among women, this "small" increased risk needs to be taken serious as it has the potential to adversely affect a large population of women.

For men the relative risk is much higher (median value for statistically significant association is 4 or 300% increase risk) than it is for women, although the incidence is considerably lower so fewer men are affected compared with women.

1.4 *In vivo* Studies

In vivo studies experimentally expose an organism to an agent and observe the effect. Testing is often done with laboratory animals (rats, mice, rabbits) and the results are extrapolated to humans (the organism of interest). These studies are done under controlled laboratory conditions and, if properly designed, do not have problems with confounders influencing the results. Because genetically uniform organisms are used this minimizes diversity in the response and experiments can be replicated. The more realistic the situation is the better. Sometimes, experimenters will use high exposures expecting the agent to act more quickly and to have a more profound effect. However, this is an assumption that needs testing and is not always the case, as I will show next. The weakest aspect of an *in vivo* study is linking the results to human exposure (dose). Unlike epidemiological studies that provide an association, *in vivo* studies provide evidence for a **cause-effect relationship**. As such they are powerful scientific tools for testing the toxicity of an agent or the efficacy of a drug.

In 2000, I wrote a major review of the literature dealing with low frequency EMFs. A large part of that review was a critical examination of two reports by the US National Research Council (NRC) and the US National Institute of Environmental Health Sciences (NIEHS). My report was peer-reviewed and was published in the Canadian National Research Council Journal of Environmental Reviews (Havas 2000).

As part of that review I examined the *in vivo* literature that exposed rats to magnetic fields. This research is not standardized and everyone used his or her own protocol. For this reason the studies are difficult to compare. Some show no effect of magnetic field, others show adverse effects and some show that the magnetic field is beneficial to the rats and inhibits growth of mammary tumors. Indeed the authors of the two reports were unable to come to a conclusion about what these studies show.

When I examined the studies I ranked the effects they documented and I ordered them according to the strength of the magnetic field used in the testing (Table 3). The results show a clear pattern. At magnetic field exposures of 1 to 500 mG all the studies show adverse effects. These rats were injected with tumor initiators and exposing them to a magnetic field promoted the growth of the breast cancer. Rats had a higher incidence of tumors, more tumors, and a shorter latency period (i.e. tumor developed more quickly). In two of the experiments no initiator was used and these also showed harmful effects although not as severe.

At magnetic field exposures of 1000 mG both harmful and beneficial effects of the magnetic field were document and at even higher magnetic fields (2500 to 5000) only beneficial effects were noted.

This seems totally contrary to what one might expect. If 1 mG is "harmful" then certainly 5000 mG should also be harmful and possibly more harmful. When scientists can't explain a result they may assume something is wrong with the experimental design. However, the results make perfect sense if we look at them not in terms of the organism but rather in terms of the cancer cells.

What these data show is that the low intensity magnetic fields stimulate the growth of breast cancer cells and that higher magnetic fields inhibit the growth of these cells. Indeed we now have studies testing the effectiveness of low frequency magnetic fields to treat cancer (Cameron et al. 2005). So instead of these studies being "inconsistent", "inconclusive" and "contradictory" they show that cancer cells are responding to magnetic fields. Breast cancer cells in rats are stimulated at low magnetic fields and inhibited at high magnetic fields. The end result is that magnetic fields are more harmful at low intensities then they are at extremely high intensities, where the magnetic fields may have the same effect that high frequency radiation has on cancer cell growth-namely inhibition or cell death.

The question remains to what degree can we extrapolate from rats to humans. If we can extrapolate, what these studies suggest is that ELF magnetic fields **stimulate breast cancer growth** at levels between 1 and 500 mG and as such are **promoters** of breast cancer. Evidence of ELF magnetic fields **initiating** cancer is present but weak.

1.3 *In vitro* Studies

In vitro studies use cells or subcellular components to experimentally examine mechanisms of action. Once we understand the mechanism we can manipulate the system to give the same result each time. This is why mechanistic studies are so important. Because controlled conditions are used with uniform biological material, this type of testing lends itself to independent replication by other laboratories. The weakness of an *in vitro* study is that it uses artificial conditions and eliminates the response of the rest of the organism (namely repair mechanism and other interactions within the body).

Liburdy et al. (1993) exposed human breast cancer cells (MCF-7) to magnetic fields at 2 mG (which was considered a background level) and 12 mG and tested the effectiveness of melatonin in inhibiting the growth of the breast cancer cells. Melatonin is known to have oncostatic

properties and this was demonstrated in the control cultures. Melatonin inhibited the growth of these ER+ human breast cancer cells by 18% and 27% in two replicates controls ($P<0.02$). However, melatonin had no effect on the cells exposed to 12 mG magnetic field. According to the authors, these results provide the first evidence that ELF magnetic fields can act at the cellular level to enhance breast cancer cell proliferation by blocking melatonin's natural oncostatic action. There appears to be a dose threshold between 2 and 12 mG.

In 1997, Harland and Liburdy were the first to present experimental evidence that magnetic fields modify drug interactions with human breast cancer cells. They found that 12 mG magnetic field exposure blocks the oncostatic effect of tamoxifen at pharmacological levels (10-7 M) on MCF-7 human breast cancer cells *in vitro*. Instead of a 40% decrease in cell growth (control), with magnetic field exposure cell growth was inhibited by only 17% with tamoxifen.

Together, these findings provide support to the theory that environmental level magnetic fields can modify the action of a drug or hormone on regulation of cell proliferation.

Laboratories around the world have repeated these studies with similar results (Blackman et al. 2001; Ishido et l. 2001; Hanf et al. 2004). This is the gold standard for any experiment, the ability to have it repeated independently by other labs. It is well known that most tumors become resistant to tamoxifen in the course of treatment resulting in treatment failure. Electromagnetic fields reduce the efficacy of tamoxifen similar to tamoxifen resistance (Girgert et al. 2008).

These studies demonstrate that at 12 mG the effectiveness is significantly reduced of both melatonin, which is produced by the body, and tamoxifen, which is prescribed as a drug for breast cancer. What this means for women with breast cancer is that two potential mechanisms by which they could reduce breast cancer growth no longer function optimally at an elevated magnetic field. This points to the mechanism by which magnetic fields affect breast cancer growth, inhibition of the agents that protect against cancers. Therefore women who are being treated for breast cancer with tamoxifen who are in a high magnetic field environment may not derive the full benefit of that drug.

One question that remains is at what level of magnetic field are affects on melatonin and tamoxifen observed in women as opposed to human breast cancer cell cultures? Is it within the 2 and 12 mG threshold as suggest by the *in vitro* studies?

> *An experiment on EMF stimulation of cell growth that has almost disappeared from the EMF literature is the work of Robert Liburdy (Liburdy et al, 1993). He reported that weak 60Hz fields can interfere with the ability to inhibit growth in MCF7 breast cancer cells. This finding has been replicated six times, but the original experiment and its replications have been ignored by many health oriented scientists (Liburdy, 2003), including the recent WHO review (BEMS Supplement 7, 2005). Even breast cancer researchers (e.g., Loberg et al, 1999), who have not been directly involved in the EMF debate, appear to be totally unaware of results showing the ability of weak 60Hz fields to affect cancer cell growth. It is shocking when an EMF research review by a presumably*

scientifically neutral WHO fails to even mention any of the papers that offers insight into the mechanism of a devastating disease that is so prevalent in the population Let us not forget the asymmetry in scientific proof (Popper, 1959), where a single reproducible harmful effect would outweigh all the negative results. The many replications of the Liburdy experiment have given us a crucial finding regarding the question of EMF risk, and they cannot be ignored (Blank 2007).

1.4 Mechanisms and Models EMF and Breast Cancer

Stevens et al. (1992) postulated a possible mechanism by which magnetic fields stimulate breast cancer growth. Their reasoning was based on the following facts and findings:

1. Two products of electric power, light-at-night (LAN) and electromagnetic fields (EMF), can suppress production of melatonin by the pineal gland.
2. Melatonin, in turn, has been shown to suppress mammary tumorigenesis in experimental animals.
3. Melatonin is both oncostatic and cytotoxic to breast, ovarian, and bladder cancer cell lines.
4. Conversely suppression of pineal function has been implicated in the etiology of several types of cancer including breast, prostate, ovarian, and melanoma.
5. Melatonin exerts a generally suppressive action on other endocrine glands. Reduced circulating concentrations of melatonin may result in increased prolactin release by the pituitary and increased estrogen and testosterone release by the gonads. Hence lower melatonin would lead to an increase in circulating estrogen levels and would stimulate the proliferation of ER+ breast tissue.
6. Moreover, recent epidemiological findings indicate an increased risk of breast cancer in workers occupationally exposed to EMF.

On the basis of these considerations, it is proposed that the use of electrical power accounts, in part, for the higher risks of breast cancer in industrialized societies (Stevens et al. 1992).

The fact that different cancers are associated with EMF exposure and that not all studies show a higher incidence (or odds ratio) for a particular form of cancer is not an inconsistency if EMFs promote rather than initiate cancer. For promoters the cancer has to be present before they will work. Animal studies confirm this perspective (Table 3). This might explain why a large number of studies failed to find a relationship between different types of cancer and magnetic field exposure.

1.5 Conclusions

In 1998, the NIEHS classified low frequency electromagnetic fields as a class 2b carcinogen based on epidemiological studies of residential exposure and childhood leukemia and occupational exposure and adult leukemia. At that time there were 6 studies showing an association of childhood leukemia and magnetic field exposure at values ranging from 2 to 4 mG**.** Currently there are 19 epidemiological studies showing an association between magnetic

field exposure and both male and female breast cancer at values at and above 1.6 mG (Tables 1 & 2).

In 1998, there were no *in vivo* studies either supporting or refuting the link between childhood leukemia and low frequency EMF exposure. In 1998 there were 7 studies showing promotion of mammary tumors in laboratory animals by low frequency magnetic fields at levels between 1 and 500 mG. Mammary tumors in rats respond to magnetic fields and are stimulated at levels below 500 mG and are inhibited at higher exposures (Table 3).

In 1998, there were no *in vitro* studies either supporting or refuting the link between childhood leukemia and low frequency EMF exposure. Currently there at least 6 studies (independently replicated) showing that 12 mG magnetic fields promote the growth of estrogen receptor-positive breast cancer (MCF-7, which is a human cell line). These studies also show that magnetic fields reduce the oncostatic effect of both melatonin and tamoxifen.

We still do not have evidence for a plausible mechanism that explains the relationship between ELF EMF and childhood leukemia but we do have a plausible explanation for the relationship between EMF, melatonin, estrogen, and breast cancer growth.

In my opinion, there is little or no evidence that ELF EMF initiate breast cancer. **However, there is a strong body of scientific evidence showing that ELF magnetic fields promote breast cancer growth, especially estrogen-receptor positive breast cancer and that the mechanism involves inhibiting the action of oncostatic melatonin and tamoxifen.** The scientific evidence for breast cancer is much stronger than it is for leukemia upon which the current classification of magnetic fields as a class 2B carcinogen was based.

2. What is your opinion on this comment from Dr. M. Bitran found at page 14 of Exhibit 20:

> *Comprehensive reviews of epidemiological and laboratory studies carried out by authoritative organizations have consistently concluded that the evidence does not substantiate a cause-effect link between ELF magnetic fields and cancer. Recent epidemiological studies on breast cancer and occupational and residential exposure to ELF magnetic fields are negative on balance. A meta-analysis found a small risk increase that may be due to artifacts. Recent reviews of epidemiological and animal data conclude that ELF magnetic fields are most likely not a risk factor in breast cancer.*

I would agree with part of Dr. Bitran's statement and disagree with part of it.

Agree: Comprehensive reviews of epidemiological and laboratory studies carried out by authoritative organizations have consistently concluded that the evidence does not substantiate a **cause-effect** link between ELF magnetic fields and cancer.

The word "cause" is ambiguous and is seldom used except in laboratory studies. If the "cause-effect" link that Dr. Bitran is referring to is one of "initiation" such that "ELF EMFs do not

initiate cancer" then I would agree. The evidence from laboratory studies indicates "promotion" not "initiation".

Epidemiological studies are designed to examine "associations" rather than "cause-effect" relationships. Consequently, it is "correct" but "misleading" to state that epidemiological studies do not show a "cause-effect" relationship since they are not designed to do so. Even epidemiological studies that "show an association" do not show a "cause-effect" relationship.

Disagree: Recent epidemiological studies on breast cancer and occupational and residential exposure to ELF magnetic fields are negative on balance.

Once again I assume that the term "negative" is used to indicate "no association" between ELF EMF and breast cancer risk. The assumption is that a negative study negates a "positive" study. This is not the case. See Dr Blank's quote on pages 3 and 4.

There are three possible outcomes of an epidemiological study: no significant association, a harmful association, or a beneficial association. This is true for drugs. The drug may be either harmful, beneficial or it may have no effect. Similarly exposure to EMFs may be harmful, beneficial or have no effect. If the number of studies reporting a statistically significant beneficial association is similar to the number of studies reporting a harmful association then we question whether those harmful associations are real. This is not the case with epidemiological studies of ELF EMF and breast cancer. Several studies show a harmful association whereas very few show a beneficial association. The fact that studies show no association does not negate any of the studies that show an association (either positive or negative). This is particularly true if EMFs are promoters rather than initiators of breast cancer. If EMFs promote growth of breast cancer then the cancer has to be present before the EMF has an effect.

Disagree: A meta-analysis found a small risk increase that may be due to artifacts.

This statement is a red herring. By implying a "possible" but unsupported "artifact" it dismisses the results.

Question 3. What is your opinion on this comment from Dr. M. Bitran found at page 23 of Exhibit 20:

> *The link between ELF magnetic field exposure and female breast cancer has become more tenuous as newer, larger epidemiological studies with better exposure assessment have become available.*

Disagree: Unfortunately we don't have better exposure metrics. Indeed this is a weakness in most epidemiological studies of breast cancer. Funding for research on electromagnetic fields was cut in the United States after the EMR Rapid program ended with the 1998 report. We never had any significant funding in Canada. Hence limited research had been done in recent years. The ideal type of study would be with continuous personal monitors but since breast

cancer takes years to develop the type of monitoring would need to be prolonged and in place before the breast cancer is diagnosed.

Question 4. Please comment on the elements of average exposure, transient peak impact exposures, and cumulative dose estimates of exposure for each of the three workers in terms of the relationship between those exposures and whether or not exposure to EMF would have been a significant contributing factor in the development or onset of breast cancer in each case.

In laboratory studies exposure, which includes the strength of the magnetic field and duration of exposure, is clearly documented. In epidemiological studies we do not yet know which metric (measurement) most closely is associated with an increased risk of breast cancer. For example is it the average exposure or some estimate of the peak exposure or some time-weighted average or cumulative exposure? Exposure for office workers is likely to vary during the day and the time-weighted average, maximum exposure and cumulative exposure are likely to be important.

Engineers and statisticians like to work with averages but most organisms respond to the maxima, rather than the average. This is clearly demonstrated by tomato plants that are frost-sensitive. If the temperature drops below freezing for a couple of hours, the tomato plant will die, even though the average daily temperature might be above freezing.

A more relevant example comes from the scientific literature showing the relationship between ELF magnetic field and miscarriages. Li et al 2002 found that the risk of miscarrying was associated with a peak value of 16 mG for women in their first trimester and not with a 2 mG average magnetic field, which is the cut-off value used in most studies of breast cancer.

The Tribunal determined that the appropriate estimate of mean exposure is 1.86 mG for all three women. Based on this figure and the work records for each of the three workers, the total cumulative ELF magnetic field dose estimates for the workers are (as calculated by Dr.Bitran in his August 2007 report):

P.B.	0.192 micro-Tesla years	1.92 mG years
M.S.	0.258 micro-Tesla years.	2.58 mG years
L.W.	0.308 micro-Tesla years	3.08 mG years

As I read the transcript of some of the testimony and the reports by Dr. Bitran I became concerned with how the magnetic field exposures for the three workers were derived. My concerns include the following:

1. Since magnetic fields change with current used, it is important to have equipment "working" when doing the measurements. It was not clear to me whether the computers were "processing" and whether the printers were "printing" when the measurements were taken. This is critical if you want to measure the exposure of the women working in front of these machines. A computer or printer in "idle mode" has a much lower magnetic field than one that is computing or printing. So my question is, was the equipment in Tables 1 to 6 working when the measurements were taken rather than just plugged in and turned on?

2. I was also concerned that the highest magnetic field readings were simply ignored (Bitran 2006, page 17 of 36).

   "Second, measurements that are orders of magnitude larger than most of the measurements should not be included, as they are likely produced by extraneous high current source, or the result of a transient, and would bias the average to an artificially high value. One measurement from Table 1 is in this category. It is the largest measurement taken in the whole survey, 275 mG with a rapid drop off to 6.04 mG at 1 foot."

   If the values measured are accurate and if this one piece of equipment contributed to higher exposure, then this needs to be included rather than excluded from the results since it would contribute to average exposure.

   I don't agree that these readings are necessarily due to "extraneous" high current source nor do I agree that they are a necessarily a result of a "transient".

   These peak exposures need to be factored into the overall exposure of the workers to get an accurate picture of exposure. Doing otherwise is simply "fudging" the results. They can be eliminated from calculations ONLY if they are known to be incorrect readings.

This concludes my written expert testimony.

TABLE 1 Epidemiological studies of statistically significant breast cancer in men.

	Reference country	Description	Exposure (uT)	Male Breast Tumor cases	RR	95% CI
1	Demers et al. 1991 USA	All subjects:				
		welders		4	0.8	0.2-3.1
		electric utility trades		13	**6.0**	1.7-21
		all electrical occupations		33	**1.8**	1.0-3.7
		Exposed before age 30 & for >30 years:				
		welders		3	**4.3**	0.4-43
		electric utility trades		11	**7.4**	1.6-34
		all electrical occupations		22	**3.3**	1.5-7.3
2	Matanoski et al. 1991 New York State, USA	telephone company, line jobs		2	**6.5**	0.79-24
		central office technicians	mean = 0.25			
3	Tynes et al. 1992 Norway	electrical occupations		170	**2.1**	1.1-3.6
		electric transport work		4	**4.0**	1.1-10
4	Loomis 1992 USA (24 states)	Age at death less than 65:				
		All electrical occupations		3	**2.2**	0.6-7.8
		Telephone Workers		1	**9.0**	0.9-89
5	Guenel et al. 1993 Denmark	jobs with intermittent exposure		23	**1.2**	0.77-1.8
		jobs with continuous exposure		2	**1.5**	0.16-4.9
6	Floderus et al. 1994 Sweden	Conductors		1	**2.7**	0.4-20
		Railway workers		4	**4.3**	1.6-12
		Engine drivers		2	**8.3**	2.0-34
7	Robinson et al. 1996 USA (NY State & western counties)	carpenters union: wood machinists trade, mortality			**4.69***	1.28-7.20*
8	Feychting et al. 1997 Sweden case-control	All Subjects: Quartile 2	0.16-0.19	17	**1.2**	0.6-2.7
		All Subjects: Quartile 3	0.20-0.28	17	**1.3**	0.6-2.8
		All Subjects: Quartile 4	> 0.29	11	0.7	0.3-1.9
		All Subjects: 90th percentile	> 0.41	4	0.7	0.2-2.3
		≤ 60 years old: Quartile 2	0.16-0.19	9	**2.9**	0.7-11
		≤ 60 years olds: Quartile 3	0.20-0.28	8	**2.5**	0.6-9.5
		≤ 60 years olds: Quartile 4	> 0.29	5	0.9	0.2-4.5
		≤ 60 years old: 90th percentile	> 0.41	3	**1.5**	0.3-8.3
9	Pollan et al. 2001 Sweden	Total Cohort **(203 cases)**	0.12-0.16		**1.37**	0.95-1.98
		Geometric Mean Average Work Exposure	0.16-0.22		**1.25**	0.82-1.91
		<0.12 uT used as reference	0.22-0.3		**1.64**	1.03-2.61
			>0.3		0.92	0.53-1.60
		Younger than 65 years **(124 cases)**	0.12-0.16		**1.56**	0.97-2.52
		Geometric Mean Average Work Exposure	0.16-0.22		**1.02**	0.56-1.85
		<0.12 uT used as reference	0.22-0.3		**1.99**	1.11-3.56
			>0.3		1.0	0.49-2.02
		Select Occupational Sectors **103 cases)**	0.12-0.16		**1.49**	0.88-2.53
		Geometric Mean Average Work Exposure	0.16-0.22		**1.69**	0.87-3.22
		<0.12 uT used as reference	0.22-0.3		**2.17**	1.21-3.88
			>0.3		1.0	0.51-2.02
10	Milham 2004	basement office near electrical switch gear	9.2	3	**100**	P<0.001

	statistically	association
[a] TWA: time-weighted-average; CE: cummulative exposure	significant	worse
[b] RR: relative risk	not significant	worse
* PMR x 100	not significant	better
+ cited in abstract	significant	better

TABLE 2 Epidemiological studies of statistically significant breast cancer in women.

	Reference country	Description	Exposure (uT)	Female Breast Tumor cases	RR	95% CI
1	Loomis et al. 1994b	15 electrical occupations:		68	**1.4**	1.0-1.8
	USA (24 states)	telephone installers, repairers, & line workers*		15	**2.17**	1.17-4.02
		electrical engineers*		16	**1.73**	0.92-3.25
		electrical technicians*		23	**1.28**	0.79-2.07
		jobs with potential exposure:				
		computer programmer		26	**1.1**	0.7-1.7
		telephone operator		328	0.96	0.84-1.1
		data entry keyer		77	0.75	0.42-1.3
2	Cantor et al. 1995a	White women: medium exposure		1,746	**1.1**	1.0-1.2
	USA (24 states)	White women: high exposure		123	0.97	0.8-1.2
		Black women: medium exposure		273	**1.3**	1.1-1.5
		Black women: high exposure		20	**1.2**	0.7-2.1
3	Coogan et al. 1996	High Exposure Jobs: All Women		6,851	**1.4**	0.99-2.1
	USA (4 states)	High Exposure Jobs: Premenopausal Women		1,424	**2.0**	1.0-3.8
		High Exposure Jobs: Postmenopausal Women		5,163	**1.3**	0.82-2.2
4	Feychting et al. 1998	699 cases and 699 controls, calc magnetic field				
	Sweden	all women	≥0.2		1	0.7-1.5
		< 50 years			**1.8**	0.7-4.3
		ER+	≥0.1		**1.6**	0.6-4.1
		ER+, < 50 years			**7.4**	1.0-178
5	Kliukiene et al. 1999	Hours/year above 1 mG				
	Norway	900-999 hrs: < 50 years old			**1.19**	1.10-1.29
		900-999 hrs: > 50 years old			**1.03**	0.98-1.09
		900-999 hrs: all			**1.07**	1.03-1.12
	900 h/y = 112 days (8 h/d)	1000-1999 hrs: < 50 years old			**1.21**	1.13-1.30
	= 22 weeks (5d/w)	1000-1999 hrs: > 50 years old			**1.03**	0.99-1.08
	= 45% total work (50w/y)	1000-1999 hrs: all			**1.08**	1.04-1.12
		>2000 hrs: < 50 years old			**1.2**	1.11-1.29
		>2000 hrs: > 50 years old			**1.12**	1.07-1.18
		>2000 hrs: all			**1.14**	1.10-1.19
6	Forssen et al. 2000	occupational exposure to MF	>0.25		1.0	0.6-1.7
	Sweden	less than 50 years of age			**1.5**	0.6-3.5
		less than 50 years of age with ER+			**3.2**	0.5-18.9
7	Van Wijngaarden et al. 2001		uT-y			
	Canada	total exposure: Total population	0.59-0.9	207	**1.4**	1.1-1.8
		total exposure: premenopausal	0.59-0.9	124	**1.6**	1.1-2.4
		>10-20 years: premenopausal	>0-0.16	76	**1.6**	1.0-2.6
		>10-20 years: premenopausal	>0.16-0.4	153	**1.7**	1.1-2.7
		>10-20 years: premenopausal	>0.4-0.52	70	**1.7**	1.0-2.8
		20+ years: Total Population	>0-0.19	167	**1.4**	1.0-1.9
		20+ years: Total Population	>0.19-0.76	256	**1.5**	1.1-2.0
		20+ years: postmenopausal	>0.19-0.76	164	**1.6**	1.0-2.6
		>10-20 years: premenopausal, ER+	>0-0.16	38	**2.0**	1.1-3.9
		>10-20 years: premenopausal, ER+	>0.16-0.4	73	**2.0**	1.1-3.6
		>10-20 years: premenopausal, ER+	>0.52	38	**2.1**	1.1-4.0
8	Reynolds et al. 2002	Flight attendants: SIR (standardized incidence ratio)			**1.42**	1.09-1.83
	US	note: magnetic fields range from average 8 to 13 mG				
		Nicholas et al. 2000				
9	Gardener et al. 2002	Postal Communication Workers			**2.98**	1.5-5.92
	China	telephone & telegraph Operators			**4.63**	1.85-11.6
10	Kliukiene et al. 2003	radio & telegraph operators			**1.3**	1.05-1.58
11	Labreche et al. 2003	lifetime occupational exposure to ELF EMF	median/high		**1.13**	0.94-1.35
	Canada	< age 35 years			**1.40**	0.98-2.02
		< age 35 years, progesterone receptor positive			**1.56**	1.02-2.39
12	Kliukiene et al. 2004	residential exposure to MF			**1.58**	1.3-1.92
	Norway	ER-positive breast cancer			**1.33**	0.93-1.9
		ER-negative breast cancer			**1.40**	0.78-2.5
		occupational exposure to MF			**1.13**	0.91-1.4
13	Peplonska et al. 2007	engineers	cases =	2,386	**2.0**	1.0-3.8
	Polish Women	economists	controls =	2,502	**2.1**	1.0-3.8
		special trade contractors			**2.2**	1.2-4.3
		electronic & electric equipmentmanufacturers			**1.7**	1.1-2.7
		public administrators			**2.7**	1.3-5.7
		janitors and cleaners			0.7	0.5-0.8
14	McElroy et al 2007	low EMF exposure	cases =	6,213	**1.06**	0.99-1.14
	USA	medium EMF exposure	controls =	7,390	**1.09**	0.96-1.23
		high EMF exposure			**1.16**	0.9-1.5

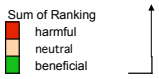
	statistically association	
[a] TWA: time-weighted-average; CE: cummulative exposure	significant	worse
[b] RR: relative risk	not significant	worse
* note: these electrical occupations were not included in the NIEHS tables 4.13-4.15.	not significant	better
	significant	better

Table 3. In vivo studies with rats. Source: Table 23 (Havas 2000). Data are ordered according to magnetic flux density.

Reference	#/grp	Initiator	Duration exp't (weeks)	Duration exp'r (h/d)	Comments on Exposure & Test Organism	Magnetic Flux Density uT	daily mT/d	total mT	Ranking incidence harm. 2 1	benef. 1 2	# tumors harm. 2 1	benef. 1 2	tumor size harm. 2 1	benef. 1 2	latency harm. 2 1	benef. 1 2	All harmful 6 5 4 3 2 1	no 0	beneficial 1 2	Sum Ranks
NTP 1998a	100	DMBA, low dose	13	18.5		500	9.25	842						0		0		2		2
NTP 1998a	100	DMBA, low dose	26	18.5		500	9.25	1684						0				1		1
NTP 1998a	100	DMBA, high dose	13	18.5		500	9.25	842										0		1
Ekstrom et al. 1988	60	DMBA, pre-exposure	25	19-21	intermittent (5s on:off)	500	5	875						0		0		2		1
Ekstrom et al. 1988	60	DMBA, pre-exposure	25	19-21	intermittent (5s on:off)	250	2.5	438						0		0		2		1
NTP 1998a	100	DMBA, low dose	26	18.5		100	1.85	337						0				1		1
NTP 1998a	100	DMBA, low dose	26	18.5	60 Hz	100	1.85	337						0				1		2
NTP 1998a	100	DMBA, low dose	13	18.5	50 & 60 Hz	100	1.85	168						0		0		2		0
NTP 1998a	100	DMBA, high dose	13	18.5		100	1.85	168										0		-1
NTP 1998a	100	DMBA, high dose	13	18.5	60 Hz	100	1.85	168										0		-1
Mevissen et al. 1998a	99	DMBA	13	24	homogeneous, no transients	100	2.4	218				0		0				2		-2
Loscher et al. 1993	99	DMBA	13	24	homogeneous, no transients	100	2.4	218										0		-3
Baum et al. 1995	99	DMBA	13	24		100	2.4	218										0		-3
Mevissen et al. 1996b	99	DMBA	13	24	homogeneous, no transients	50	1.2	109				0		0				2		-2
Beniashvili et al. 1991	25	no initiator	~ 2 yr	0.5	strain not provided	20	0.01	7		0								1		-1
Beniashvili et al. 1991	50	MNU	lifelong	0.5	strain not provided	20	0.01	7		0		0		0		0		4		0
Beniashvili et al. 1991	25	no initiator	~ 2 yr	3	strain not provided	20	0.06	44												-4
Beniashvili et al. 1991	50	MNU	lifelong	3	strain not provided	20	0.06	44						0				0		-6
Anisimov et al. 1996	40	MNU	5 mth	3	outbred white rats	20	0.06	9												-3
Mevissen et al. 1996a	99	DMBA	13	24		10	0.24	22												-1
Loscher et al. 1994	99	DMBA	13	24	gradient, no transients	0.3-1.0	0.0144	1.3		0		0		0				melatonin decr		-2
Mevissen et al. 1998a	99	DMBA	13	24	homogeneous, no transients	0.1	0.0024	0.2						0				1		-2

exp't = experiment; exp'r = daily exposure
Note: studies used female Sprague-Dawley rats and 50 Hz frequencies unless otherwise noted.
DMBA = 7, 12-dimethylbenz [a] anthracene
MNU = N-methyl-N-nitrosourea

Ranking
2 = if statisticaly significant, p≤0.05
1 = if "trend" but not statisticaly significant
# = number of times "no effect" reported



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