DRAFT - WORK IN PROGRESS – Version 1
29/6/06

Adverse health effects associated with exposure to ELF electric and magnetic fields – assembly of scientific evidence and discussion of possible public health impact

Volume 1

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Summary

The following table gives the total impact, in thousands of pounds per year per 1,000 exposed population, from EMF exposure for five '*what-if*' scenarios, as calculated in a moderate and transparent way in section 2.3. The impact of other diseases is of the order of 100 times greater than that of childhood leukaemia alone.

	CL alone	NIEHS 2	Calif 5	Calif 11	12 diseases
With credibility factors	4	70	146	679	716
With definite causation	5	122	246	1899	2629

We do not think it is rational to base an assessment on childhood leukaemia alone, when most of the hypothesised mechanisms and their supporting evidence relates to biological systems involved in many diseases, rather than exclusively to childhood leukaemia. The decreasing Degree of Certainty with greater numbers of diseases is however reflected in the credibility factors. Therefore, while there remains considerable uncertainty and imprecision in such assessments, it seems sensible to give consideration to the above scenarios and multiple outcomes, without adopting any one as definitive.

The next table shows the numbers of ELF-EMF epidemiological studies covered in major reviews to 2002, as explained in section 2.2. This shows that, on the basis of numbers of studies and their statistical strength, there is more and stronger evidence for some other diseases than for childhood leukaemia.

Disease	Studies	Positives	Significant positives	Significant negatives
childhood leukaemia	19	16	3	0
adult leukaemia	43	32	11	0
9 other diseases	150	110	36	1

Since 2002 there have been many new studies increasing knowledge of potential mechanisms. Important earlier studies have been overlooked in the major reviews, for example the results of Schuz *et al.* (2001) showing stronger associations of childhood leukaemia with nocturnal exposure, with its implications for the melatonin hypothesis.

In addition, we note two substantial areas of established and relevant research which have also been largely overlooked: solar and geo-magnetic activity (S-GMA), which includes ELF exposure, and bio-detection of magnetic fields by migrating birds and other animals. Both give firm implications for biological effects of very low fields. The first reinforces implications of ELF EMFs for various diseases. The second reinforces implications for biological mechanisms by which this may be possible.

CONTENTS

1. Introductory comments

This document seeks to assemble the scientific evidence for a range of adverse health outcomes associated with exposure to electric and magnetic fields from the electricity supply. We first consider the main external reviews of EMF health effects, namely NIEHS 1999, IARC 2001 and the California Health Dept EMF Report of 2002, how their findings compare and how these findings impact on the risk assessment and cost-benefit analysis. We then collate the scientific evidence to take account of a wide range of available information. For each adverse health outcome we consider four categories:

1. Direct epidemiology: human epidemiology in relation to exposure to power frequency electric and/or magnetic fields.

2. Other relevant epidemiology, for example, epidemiology of health outcomes associated with fluctuations in geomagnetic fields.

3. Direct experimental, animal or other evidence of an experimental or mechanistic nature.

4. Other relevant laboratory, mechanistic or theoretical evidence.

Headings 2 and 4 are particularly aimed at accessing potentially important evidence which might otherwise fall outside the terms of reference of official committees investigating EMFs and health. An example here concerns the literature on adverse health effects relating to natural fluctuations in the Earth's (the geomagnetic) field.

In recent years considerable progress has been made in examining possible mechanisms by which EMF[1] exposures may result in increased risk of adverse health outcomes. For example, the early generation of *in vitro* and animal experiments tended to treat MFs[1] in the same way as conventional (e.g. chemical) carcinogens. Exposures were carried out at high dose and adverse effects including dose response effects were looked for. In general terms, this approach has not provided convincing evidence that EMFs are directly carcinogenic. However, a recent review by Juutilainen (2006) shows that with both *in vitro* and animal experiments EMFs enhance the effects of existing carcinogens.

Other models have also been developed. The hypothesis in which MF exposures disrupt the nocturnal production of pineal melatonin is interesting because it is potentially a common feature of the apparent disparate set of adverse health outcomes associated with EMF exposures. It is obviously the case that pineal melatonin was not present in the early generation of *in vitro* experiments, but where melatonin has been investigated its action has been shown to be inhibited in the presence of magnetic fields as low as 0.2 μT (see for example Ishido *et al.* 2001). The review by Henshaw & Reiter (2005) highlights strong evidence of melatonin disruption in populations

[1] EMFs = electric and magnetic fields; MFs = magnetic fields; EFs = electric fields

exposed to neighbourhood EMFs. The recent volunteer study by Davies *et al.* (2006) adds to the evidence here.

There have been important advances in the proposed radical pair mechanism in which magnetic fields may enhance the lifetime of free radicals in the body. Experimentally, the effect has been observed with fields as low as 50 μT and there is no theoretical objection to such effects occurring at fields as low as 0.4 μT. Recent work suggests that a key mechanism in navigation across a range of animal species is a radical pair mechanism operating within the eye (Wiltschko & Wiltschko 2006). Furthermore, it would appear that visible light is needed to create the initial radical pairs which are acted upon by changes in the earth's quasi-static magnetic field which are then communicated to the brain for purposes of navigation. Behavioural responses of homing pigeons indicate they can distinguish among magnetic fields that differ in intensity by about 10 - 30 nT (Beason 2005). However, this may be attributed to small particles of magnetite in the beak. The level attributed to the radical pair mechanism may be higher, in the robin radical pairs were associated with the detection of 85 nT (0.085 μT) fields (Ritz *et al*. 2004).

(Note: Historically, review bodies seem to have emphasised the energetic aspects of possible EMF effects, which led to a sceptical outlook, deeming bio-effects of ELF-EMF implausible, if not impossible. At first sight ELF-EMFs at levels typically encountered lacked both the quantum energy to damage DNA directly, because of their low frequency, and the macroscopic energy to damage the human body or its organs by induced currents, because of their low intensity.

However, information effects require only a very small amount of energy, just enough to transmit the information. For example, the bystander effect in which cells which are not themselves hit by ionising radiation express chromosomal damage in their descendants, is mediated by signalling molecules generated by the hit cell (Azzam *et al*. 2002, Mothersill & Seymour 1998). Thus, if a signal can be detected it can have an effect. Modern research is improving understanding of how and where this might occur. We think it premature to deem bio-effects implausible before understanding the mechanistic evidence and bio-detection systems. As research progresses, bio-effects seem ever more plausible despite the historical sceptical outlook.)

The following adverse health outcomes have been considered (i) childhood leukaemia; (ii) adult leukaemia; (iii) adult brain cancer; (iv) miscarriage; (v) ALS; (vi) childhood brain cancer; (vii) female breast cancer; (viii) male breast cancer; (ix) Alzheimer's disease; (x) suicide; (xi) heart disease; (xii) depression; (xiii) electro-sensitivity; (xiv) universal carcinogen and (xv) other reproductive.

2. Health impacts based on external reviews.

2.1 External reviews of causation of different diseases.

The purpose of this section is to show how the main external reviews regard the relative strength of evidence for causation of the various diseases.

Evidence for causation includes both epidemiological and mechanistic evidence. The former involves statistical incidence of specific diseases associated with exposure, so readily relates to specific diseases. The latter more often relates to biological systems and functions which might affect many diseases, so tends not to be specific to single diseases.

The IARC system of classification was designed to classify agents or exposures for carcinogenicity rather than to rate causation of a single disease. Nevertheless, some review bodies have adopted the system for specific diseases, as in Table 2.1. The class 2B is summarised as "limited evidence" for a "possible human carcinogen" or a possible cause for other diseases.

Table 2.1 Review bodies' assessments of EMF causation of various diseases.

Disease	IARC 2001 2B	NIEHS 1999 2B	Calif 2002 2B	Calif 2002 % Degree of C
1. Childhood Leukaemia	yes	yes	Yes	72
2. Adult Leukaemia		yes	Yes	57
3. Adult Brain Cancer			Yes	64
4. Miscarriage			Yes	56
5. ALS			Yes	54
6. Childhood Brain Cancer				25
7. Female Breast Cancer				28
8. Male Breast Cancer				35
9. Alzheimer's disease				26
10. Suicide				46
11. Heart Disease				33
12. Depression				- (other)
13. Electro-sensitivity				- (other)
14. Universal Carcinogen				3 (SBN)
15. Other reproductive				5 (SBN)

Many of the blank boxes in this table correspond to the review bodies not having considered or assessed causation for the specific disease, according to the California report, while in some cases IARC and NIEHS have deemed the evidence "inadequate". The latter is the IARC class 3 which means the evidence is insufficient to decide a class of causation, not that there is not a cause (which would be IARC class 4). See also the Table 21.2 from page 379 of the California DHS report 2002, reproduced elsewhere in this paper.

The California DHS report 2002 is the only review to give a quantified assessment of the possibility of causation. This was called "confidence in causality" in an earlier draft report, and the final 2002 version calls it Degree of Certainty. Assessments were made separately by three DHS expert reviewers ("active in the EMF field for more than a decade" - page 380) working to a comprehensive framework to consider a wide range of epidemiological and mechanistic evidence as well as the earlier reviews by other bodies. Each reviewer gave a "best-judgment" value and a range. The average of the three best-judgment values is given in the right hand column in Table 2.1 above.

Depression was not evaluated in this way; we give it a low value of 0.05 to avoid exaggeration.

Diseases 12 and 13, marked "other" in the right hand column, appear in Chapter 19 (Other adverse non-cancer health outcomes) and are not given a Degree of Certainty. The last two (classes of) diseases, 14 and 15, are given Degrees of Certainty of 5% and 3% and are described by "strongly believe not", indicated by SBN in the Table.

2.2 Epidemiological evidence for different diseases.

This section shows and compares the epidemiological evidence for different diseases. The evidence is as reviewed by the main review bodies and listed in the latest of them, the California 2002 report. There is further evidence from more recent studies, which would generally strengthen the case for causation, but we do not include it here as it may risk selectivity on our part.

Table 2.2 shows the numbers of studies, positive results and statistically significant positive results. These are taken from the summary tables in the California report, taking results with OR > 1 as positive and those with the lower CL > 1 as significant positives. Results with OR given as 1.00 are counted as half negative and half positive, and similarly with lower CL given as 1.00 as half significant and half not significant.

We then calculate p values for finding such numbers of positive and of significant results. The p values are the probabilities of such results (or stronger) occurring by chance (under the null hypothesis, i.e. assuming there is no causation). The p values are mostly below 0.05, showing that such numbers of results are themselves statistically significant and unlikely to occur by chance. Indeed several are very strongly significant. However, that only relates to chance or random error. There remains the possibility of bias (especially where studies are small and/or poorly designed) and confounding (especially where studies are large in the general population and with low odds ratio).

Table 2.2. Numbers of reviewed epidemiological studies and of positive and significant results.

Disease	Number of studies	Positive studies: number, p value*		Significant positives: number, p value*		Meta-analytic OR (95% CI)
1. CL	19	16	0.0014	3	0.01	1.3 (1.0 - 1.7)
2. Adult L	43	32	0.0007	11.5	<<0.00001	1.2 (1.12 - 1.24)
3. A Brain C	32	25	0.0007	6	0.0001	1.2 (1.1 - 1.3)
4. Miscarriage	37	27.5	0.0015	9	<<.00001	
5. ALS	7	6,	0.06	3	0.0004	1.5 (1.2 - 1.7)
6. C Brain C	12	6	>0.5	2	0.04	
7. F Breast C	24	17.5	0.012	5.5	0.0001	
8. M Breast C	16	11.5	0.04	no data		
9. Alzheimer's	6	4	0.34	2.5	0.001	
10. Suicide	8	6.5	0.02	3	0.0007	
11. Heart disease	8	6.5	0.02	5.5	<<0.00001	

*Null hypothesis, result occurs by chance

The p values in the table were obtained from the cumulative binomial distribution where there are fewer than 12 studies, otherwise from its normal approximation for numbers of positives (p for a positive being 0.5) and from its Poisson approximation for numbers of significant positives (p for a one-sided significant being 0.025).

The right hand column shows meta-analytic summaries from Kheifets 1995, Kheifets 1997a, Ahlbom 2001 or Wartenberg 2001, as reported in the California report.

While the 19 childhood leukaemia studies were generally residential case-control studies, the 43 adult leukaemia studies included 41 occupational and 2 residential studies (both positive, 1 significantly positive). The exposure metrics included many from wire codes for residential studies and many from job history and even job titles for occupational studies. Although these "metrics" may be correlated to field exposures, their imprecision offers scope for both confounding and for regression dilution of results, for both childhood and adult leukaemia. In general for the diseases, both residential and occupational studies showed similar positive results.

Generally, the studies counted in the lists are separate studies reported in different papers. In some cases the California list has separate entries for different exposure categories from the same study. For example, with heart disease the table has 8 results from 3 published studies. Including lower exposure level results in this way tends to dilute the strength of the findings in Table 2.2 above, but we have declined to select one result per study in order to avoid selectivity on our part. More than three studies on heart disease are discussed in the California report, but only 3 are listed in their summary table.

Some results used exposure to electrical devices, as well as residential and occupational exposure. Where they appear in the California lists, all of these types are reflected in Table 2.2 above. For example in the case of miscarriage, there are 10 results for bed heaters, clearly negative overall, which are still included though they may be irrelevant. The effect of including all the listed results from the California tables is to dilute the overall effect in Table 2.2 above, but we avoid imposing our own selection or rejection and simply take the California lists as given.

Comparing results for childhood and adult leukaemia, just on the counts of papers, positives and significant results, we see that adult leukaemia (AL) has a stronger number of positives (lower p value) and a much stronger number of significant positives. The meta-analytic summary for childhood leukaemia (CL) is also weaker than that for AL, despite its slightly higher OR, because its lower confidence limit is only 1.0.

This comparison of CL and AL is made on the raw collection of papers without selecting papers compatible for pooled analysis. When Greenland *et al.* (2000) and Ahlbom *et al.* (2000) did their pooled analysis for CL, they obtained stronger results. However, those stronger results should not be compared with the raw full set for AL. Like for like, the association for AL shows up statistically stronger than that for CL. However, there are other factors to take into account for causation, and it should be noted the California team gave a lower Degree of Certainty for AL than for CL.

Note that Adult Brain Cancer and Miscarriage, as well as Adult Leukaemia, show statistically stronger associations than does Childhood Leukaemia, on the basis of the p values shown, although the diseases in the lower half of the table do not.

2.3. Hypothetical benefit of avoided exposure.

This section estimates the hypothetical benefits of avoiding residential EMF exposures under various what-if scenarios. Table 2.3 lists 12 diseases (or adverse health outcomes) for which there are peer-reviewed scientific papers giving some support to EMFs being a possible Causal Risk Factor (CRF) and calculates a specific impact for each disease in £M per case.

This section uses data from published sources and methodologies from government guidelines, as far as available, often erring on the side of under-estimation in order to avoid exaggerated results and recognising the high level of imprecision implicit in the process.

The basic value of a life, for such hypothetical purposes, is rounded down to £1M from figures in the Treasury Guide 2005 used in health and road accident analysis. For example, page 25 of the Treasury Guide refers to a DfT benchmark of £1-1.5M in 2002 prices and its VOSL of £1.25M. Although official guidance cautions against adjusting life value for old age, to avoid exaggeration we allow only £0.1M life value for Alzheimer's disease as it is often incurred very late in life.

The impact of non-fatal cases is rounded down from Highways Economics Note 1, 2002, of £140k for serious injury and £11k for slight injury. Thus a non-fatal cancer case is rounded down to £100k and the (annual) impact of a case of depression to £10k. Depression is assessed as annual impact and other diseases as a single incidence. Miscarriage is treated as a "slight injury" although that may be an under-estimate in many cases. Slightly higher figures (£125k) are used for non-fatal childhood cancer cases in view of the impact of treatment on childhood development and on families.

Fatality rates are from sources such as Cancer Research UK, ONS and DH statistics. The statistics for risk of suicide and heart disease relate only to fatal cases, so their fatality rate is taken as 1. Depression (which always refers to that which is clinically diagnosed) is taken as non-fatal.

Table 2.3 Specific Impact per case of hypothetically associated diseases.

	Disease	life value £M	fatality rate	sensitivity	non-fatal impact £M/case	specific impact £M
1	Childhood leukaemia	1	0.3	4	0.125	1.55
2	Adult leukaemia	1	0.8	3	0.1	2.46
3	Adult brain cancer	1	0.8	3	0.1	2.46
4	Miscarriage	1	0	1.5	0.01	0.02
5	ALS	1	0.5	1.5	0.1	0.83
6	Childhood brain cancer	1	0.35	4	0.125	1.73
7	Female breast cancer	1	0.3	3	0.1	1.11
8	Male breast cancer	1	0.3	3	0.1	1.11
9	Alzheimer's	0.1	1	3	0	0.30
10	Suicide	1	1	1.5	0	1.50
11	Heart	1	1	1.5	0	1.50
12	Depression	1	0	1.5	0.01	0.02

Sensitivity factors may be more controversial, although Treasury, HSE and EU guidance clearly establish that such sensitivities should be a consideration. A quantitative precedent from HSE guidance uses a factor of 2 specifically for cancer as a “dread disease”. Two further sensitivities acknowledged as proper considerations are risks for children and risks imposed involuntarily. We use a factor of 2 for children and 1.5 for involuntary imposition, though we have not found quantitative precedents for those choices. We regard all of the EMF exposures as involuntary for the present purpose. However, we limit the combined factors to a maximum of 4, which is therefore the factor used for imposed (hypothetical) risks of childhood cancers, with 3 for imposed dread diseases for adults, and 1.5 for imposed non-dread diseases.

Table 2.4 estimates the total impact per year per thousand exposed population, consisting of 189 children aged 0-14 and 811 “adults” 15 and over (420 females and 391 males), including 160 over-65s, in proportion with ONS UK statistics. The “number exposed” column gives the relevant number of people from 1,000 general population exposed. There would typically be 17 pregnancies per year (1 per 60 whole population) according to DH statistics. Alzheimer’s disease is calculated only on the basis of over-65s, so the relevant number is 160. Heart fatality data used in EMF statistical association refer to working men, so the number exposed there is counted as 391. No heart disease effect on women is included, as we do not have usable EMF data for them.

Table 2.4 Total Impact (£k/year) per disease per 1,000 exposed population.

	disease	number of exposed people	relative risk	unexposed risk M/year	credibility of causation	total impact £k/year
1	Childhood leukaemia	189	1.44	42	0.72	4
2	Adult leukaemia	811	1.32	183	0.57	67
3	Adult brain cancer	811	1.31	131	0.64	52
4	Miscarriage	17	1.51	250000	0.56	18
5	ALS	811	1.8	20	0.54	6
6	Childhood brain cancer	189	1.26	30.7	0.25	1
7	Female breast cancer	420	1.39	1600	0.28	81
8	Male breast cancer	391	2.1	11	0.35	2
9	Alzheimer's	160	1.54	4900	0.26	33
10	Suicide	811	1.73	94	0.46	38
11	Heart	391	1.5	3900	0.33	377
12	Depression	811	1.4	150000	0.05	36

The relative risks in Table 2.4 are taken as geometric means of the odds ratios from the several studies in the summary tables for each disease given in the California 2002 report, with the exception of depression for which California does not provide such a table. The geometric mean is appropriate for combining odds ratios, which are always greater than zero and hinge on 1 as to whether they reflect a positive or negative finding. It is not true to say (as in Ahlbom 2000) that geometric means suppress outliers; relative to the arithmetic mean they suppress high outliers but emphasise low outliers, and depress the mean itself, but nevertheless they are appropriate here.

As we have in mind the benefit of avoiding residential exposure, the geometric means are taken from the odds ratios (ORs) of residential studies only, where available. Generally the ORs are similar for occupational and residential studies. For example, all the childhood studies are for residential exposures, whereas of the 43 adult leukaemia studies listed in California Table 8.1.1, 41 are occupational and 2 residential. The geometric mean OR for all 43 studies is 1.327 and that for the 2 residential studies is 1.318. Where there were no residential studies listed, that is for ALS, Alzheimer's, suicide and heart disease, the means were taken from the occupational studies.

We differentiate between taking evidence from occupational exposures into account when considering causation from EMF as a possible risk factor, and preferring residential exposure data when considering the hypothetical benefit of its removal.

For adult brain cancer, the California summary Table 9.1.1 included 29 occupational and 3 residential studies. But the more detailed Table 9.1.2 included 2 more residential studies with odds ratios. Hence we took the geometric mean from the 5

residential studies, which was 1.31, whereas from all 32 studies in Table 9.1.1 it was 1.28.

Where there were several ORs for different exposure levels from a single study, e.g. for miscarriage, we selected those nearest to TWA 0.4 μT, for comparability with options for removal of residential exposures. Where there were several ORs for different occupations from a single study, e.g. for suicide, these were reduced to a single geometric mean for the study. This avoided inflating the result by counting several higher findings from the same study. Generally, however, the California report lists only one OR per study, so there is no choice.

Note that taking the crude OR from all studies, including lower exposure levels, without matching exposure levels and weighting according to study size, leads to a smaller OR than the more discriminating pooled studies of Greenland *et al.* and Ahlbom *et al.* for childhood leukaemia. They found ORs around 2, whereas the crude geometric mean from the 19 listed studies is only 1.44. Properly matched pooled studies are not available for other diseases. Our impact assessments may therefore be under-estimated on this account, but we compare Childhood Leukaemia and other diseases on a like-for-like basis.

Figures for unexposed risk, that is from the normal incidence of the disease, were derived from ONS, DH and Cancer Research data. Where possible they are for UK, otherwise for England and Wales, although as the figures used are rates per million, calculated for the relevant population exposed, they are not likely to differ greatly. These figures could benefit from greater consistency of source, but the difference would not be substantial.

The credibility factors are taken from the California Degrees of Certainty as in Table 2.1 above, with the exception of depression as noted above. We only count this at 0.05, the same as a "strongly believe not" class of diseases, although there is significant evidence and the California report cites 6 studies and describes it as "worthy of further study".

Table 2.5 summarises the total impact accumulated over the various diseases under different 'what-if' scenarios. The five scenarios are those involving 1, 2, 5, 11 or 12 diseases as described in Table 2.1. The first row gives the total impact using the credibility factors derived from the California report. The second row gives the total impact with all credibility factors set to 1, that is what-if the exposure was a definite Causal Risk Factor (CRF) for all the diseases in each scenario, with numerical relative risk derived from published research findings as described above.

The highest figure, the impact for all 12 diseases with definite causation, is some £2.6 million per year per thousand population exposed, or some 500 times the impact of CL alone calculated on the same basis. However, that excludes non-fatal heart disease, female heart disease and other diseases which have been raised as possibly having EMF as a causal risk factor, such as electrical sensitivity, epilepsy, SIDS, AIDS, headaches and insomnia. They are excluded because we do not have data to estimate relative risk, and we prefer to use the externality of diseases and data derived from review bodies and research studies. But the highest figure should not be seen as

a maximum or upper limit to the possible impact; it is merely an estimate based on a hypothetical scenario.

Table 2.5: Total impact (£000 per year per 1,000 exposed population) from EMF exposure for five what-if scenarios.

	CL alone	NIEHS 2	Calif 5	Calif 11	12 diseases
with credibility factors	4	70	146	679	716
with definite causation	5	122	246	1899	2629

For considering potential precautionary measures, the scenarios might be a guide to the potential total impact and hence the benefit of removing exposures. The possible impact may range from zero (if there were no direct or indirect causation for any of the diseases) to the highest figure in the table if there were definite causation for all 12 diseases, and beyond that if other diseases were implicated.

To get some balance of potential impact, the use of carefully constructed credibility factors would seem helpful. In this case it places the likely impact, based on current evidence, in the lower part of the full range.

We do not think it is rational to base an assessment on childhood leukaemia alone, when most of the hypothesised mechanisms and their supporting evidence relates to biological systems involved in many diseases, rather than exclusively to childhood leukaemia. The decreasing Degree of Certainty with greater numbers of diseases is however reflected in the credibility ratings. Therefore, while there remains considerable uncertainty and imprecision in such assessments, it seems sensible to give consideration to the above scenarios and multiple outcomes, without adopting any one as definitive.

3. Review of mechanisms

Two mechanisms will be mentioned here, the melatonin hypothesis and the radical pair mechanism.

3.1 The Melatonin Hypothesis

Melatonin (N-acetyl-5-methoxytrptamine) is a hormone produced nocturnally in the pineal gland. Its production is triggered by a signal from the eye indicating that light has fallen below ~200 lux. This trigger does not involve the visual system rather recently discovered ganglion cells in the eye (Berson *et al*. 2002; Hatter *et al*. 2002). Melatonin is a powerful antioxidant and radical scavenger whose mechanisms of action, some of which are cell receptor mediated, has been extensively researched. As an antioxidant, melatonin acts as a natural anti-cancer agent but it is also known to

regulate seasonal breeding in animals and reduced melatonin is associated with depression. The decline in production of melatonin in the aged is associated with the onset of neurodegenerative diseases (see review in Reiter 1998).

Unlike other antioxidants, such as vitamins C and E and glutathione, melatonin enters cells and is able to act directly on DNA. Indeed, the concentration of melatonin in cells, tissues and organs is markedly non-uniform (see Editorial by Reiter 2003). As a result, the so-called normal physiological concentrations of melatonin need to be carefully defined. For example, melatonin concentrations in bile and in the cerebrospinal fluid of the third ventricle are orders of magnitude higher than in the blood.

The nocturnal production of pineal melatonin can be completely suppressed by exposure to light-at-night. Nowadays, compared to our forefathers, man is exposed to far more light-at-night, from street and indoor lighting, with a corresponding reduction in the total amount of nocturnal melatonin produced. The so-called melatonin hypothesis (Stevens 1987), postulates that the increased risk in breast cancer in recent decades in industrialised countries results in part from increased exposure at light-at-night (LAN). Strong support for this hypothesis comes from the increased risk of breast cancer in nightshift workers (Swerdlow 2003) and the fact that such workers who go on to contract breast cancer produce lower nocturnal melatonin (Schernhammer & Hankinson 2005). Further support comes from the recent observation that normal physiological concentrations of nocturnal melatonin in women's blood prevents the growth of human breast tumours when transplanted into rats (Blask *et al*. 2005).

The melatonin hypothesis has been applied to EMFs (indeed this was integral to the original hypothesis by Stevens, 1987). The adverse health effects ranging from childhood leukaemia through to depression and miscarriage comprises an apparent disparate set of illnesses. However, melatonin suppression or disruption could be a common factor leading to increased risk. Henshaw & Reiter (2005) review the various studies suggesting melatonin disruption by power frequency EMFs. Volunteer experiments involving short-term exposures to magnetic fields (for example, for 30 minutes) tend not to yield evidence of such disruption, although those involving longer terms exposures have shown a clear effect, for example the recent report by Davies *et al.* (2006). However, studies in animals, in volunteers with comparatively long-term exposures and populations exposed to neighbourhood fields associated with the electricity supply together provides strong evidence of melatonin disruption, including from magnetic fields as low as 0.2 μT. In the case of neighbourhood exposures, it would appear that the effect of switching of magnetic fields and/or of electric fields are an important factor in melatonin disruption. Indeed, the initial volunteer experiments indicating melatonin disruption themselves concerned electric rather than magnetic fields (Wever 1979).

Henshaw & Reiter also hypothesise that melatonin disruption could increase the risk of childhood leukaemia, citing the evidence that melatonin is highly protective of oxidative damage to the human haemopoietic system (Vijayalaxmi *et al*. 1996) and that in animals melatonin is highly protective of oxidative damage to the fetus (Wakatsuki *et al*. 1999a, b and 2001; Okatani *et al*. 1997).

3.2 The Radical Pair Mechanism

Magnetic fields are able to alter the lifetime of chemically reactive species known as *free radicals*. In the body, such radicals are capable of damaging DNA in a manner that could lead to cancer. Magnetic fields act on free radicals by altering the ratio of their spin states, that is, the ratio of triplet (T) to singlet (S) states of pairs of radicals. At relatively high levels magnetic fields act to decrease the ratio of triplets to singlets. In recent years, however, the existence of a so-called Low Field Effect (LFE) has been recognised where magnetic fields increase the ratio of triplets to singlets. In the body, the average lifetime of a given triplet is longer than that of a given singlet, therefore the low field effect results in increased availability of free radicals to cause biological damage.

Investigations into the LFE have been carried out in the field of Theoretical Chemistry where it is predicted that this effect occurs typically below 100 μT but theoretically the effect could occur with fields as low as 0.4 μT. The LFE has been investigated experimentally in laboratory systems designed to mimic the presence of free radicals in the body. Here, such effects have currently been observed with fields down to 50 μT.

As mentioned above, there is now a body of studies demonstrating that magnetic fields enhance the effects of existing carcinogens (Juutilainen 2006) and that while they do not directly address fields in the 0.4 μT regime, they lend support for the radical pair mechanism.

Our understanding of the action of magnetic fields on radical pairs has been considerably advanced in the study of animal navigation in the earth's (geomagnetic) field. Birds, for example, are able to detect changes as low as 10 – 30 nT (0.01 – 0.03 μT) in the earth's quasi-static field of around 48 μT in the UK, although the level attributable to the radical pair mechanism maybe slightly higher. These recent advances in understanding have been reviewed by Wiltschko & Wiltschko (2006). In birds, for example, the crucial processes of magnetoreception take place in the eyes and seems to be restricted to the right eye. Radical pairs are generated in the eye by the action of specific wavelength visible light on receptors. Cryptochrome, the photopigment suggested to form the crucial radical pairs, has recently been extracted from the retina of two species of migrating birds. In salamanders, however, the receptors have been found to be located in the pineal gland (Deutschlander *et al*. 1999). While this magnetic sensing facility is designed for the purposes of navigation, its presence in this species in the site of melatonin synthesis (the pineal gland) is intriguing. Overall, magneto-reception by animals, across a wise range of species, may provide important clues to in our understanding of the underlying mechanism reported health effects from power frequency magnetic field exposures.

4. Adverse health effects from fluctuations in the Geomagnetic Field.

The research literature contains a body of information reporting a range of adverse health effects associated with fluctuations in the geomagnetic field, principally in the range 0.03 to 0.10 μT. Such fluctuations, on a time scale of a few hours, arise from

electrically charged solar particles reaching the top of the earth's atmosphere. Melatonin disruption, suicide, depression and mental disorders, heart disease and blood pressure, light sensitivity and sudden infant death syndrome (SIDS) are amongst the adverse conditions associated with GM fluctuations. These are described in more detail in Appendix 1.

5. Electric fields

There is a body of information suggesting adverse health effects from power frequency electric fields. The evidence is reviewed in Appendix 2. Corona ions will not be discussed in this document but may be added at a later stage.

6. Summary of the epidemiological and mechanistic evidence

6.1 Childhood leukaemia

6.1.1 Direct epidemiological evidence

The California report cites 19 studies. These are listed in section 1 of the References. The following studies were either not included or were published later: (i) the pooled analysis of Ahlbom *et al.* (2000) indicating a doubly of risk above 0.4 μT; (ii) the pooled analysis by Greenland *et al.* (2000) indicating a 1.7-fold increase in risk above 0.3 μT; (iii) Schüz *et al*. (2001) showing a 3.21-fold increase in risk with night-time exposures above 0.2 μT with evidence for a dose-response effect; (iv) Draper *et al*. (2005) who found that compared with those children who lived >600 m from a powerline at birth, children who lived within 200 m had a relative risk of leukaemia of 1.69 (95% CI: 1.13 – 2.53); those born between 200 and 600 m had a relative risk of 1.23 (1.02 – 1.49) and (v) Kabuto *et al*. (2006) showing increased risk of childhood ALL of 4.7 (95% CI: 1.15 – 19.0) for magnetic fields above 0.4 μT compared with less that 0.1 μT.

A review of earlier studies may be found in Heath (1996).

6.1.2 Other relevant epidemiological evidence
(still to do)

6.1.3 Direct experimental evidence

Melatonin is highly protective of oxidative damage to the human haemopoietic system (Vijayalaxmi *et al*. 1995 & 1996) and shows similar protectivity in mice (Vijayalaxmi *et al*. 1999, Badr *et al*. 1999). In animals melatonin is highly protective of oxidative damage to the fetus (see for example Wakatsuki *et al*.1999a, b and 2001a, b; Okatani *et al*. 1997).

6.1.4 Other relevant experimental/theoretical evidence

Populations exposed to neighbourhood EMFs have reduced melatonin (see Henshaw & Reiter 2005). Longer term volunteer studies also show these effects (for example, Davies *et al*. 2006). The melatonin hypothesis could be applied to childhood leukaemia (see Henshaw & Reiter 2005). Also of interest: Nocturnal maternal serum melatonin levels increase significantly during pregnancy (Nakamura *et al*. 2001).

6.2. Adult leukaemia

6.2.1 Direct epidemiological evidence

Chapter 8 of the California Report considers 55 studies, although a number of these were grouped producing 43 sets of studies. Of these 32 gave odds ratios >1.0 ($p = 0.0007$) of which 11 were statistically significant ($p << 0.00001$) – see references section 8.2 below. Overall, the California Report classes magnetic fields as a class 2B carcinogen for adult leukaemia.

See also Juutilainen *et al*. (1990), Alfredsson *et al*. (1996), Heath (1996), Baris *et al*. (1996).

6.2.2 Other relevant epidemiological evidence
(Still to do)

6.2.3 Direct experimental evidence

See section 6.1.3

.

Review by Juutilainen *et al.* (2006): a pooled analysis of *in vitro* studies and short term animal studies show that MFs enhance the effects of known carcinogens. The studies reviewed cover a wide range of cell types, including human lymphocytes. Overall, the findings lend support for the radical pair mechanism.

6.2.4 Other relevant experimental/theoretical evidence

See section 6.1.4.

6.3. Adult brain cancer

6.3.1 Direct epidemiological evidence

Chapter 9 of the California Report lists 39 studies which when grouped reduces to 32 studies. Of these, 25 give an odds ratio >1.0 ($p = 0.0007$) of which 6 were statistically significant ($p = 0.0001$). Overall, the California Report classes magnetic fields as a class 2B carcinogen

for adult brain cancer. Note that for brain cancer the California Report relies heavily on the review by Kheifets (2001)

A later study by Villeneuve *et al*. (2002) found for MF exposures >0.6 μT relative to those with exposures <0.3 μT a more pronounced risk among men diagnosed with glioblastoma multiforme (OR = 5.36, 95% CI: 1.16 – 24.78).

See also the review by Heath (1996) and Baris *et al*. (1996).

6.3.2 Other relevant epidemiological evidence

Aldrich *et al.* (2001): "Brain cancer risk and EMFs: Assessing the Geomagnetic component". This paper assesses the possible role for geomagnetic fields in the incidence of brain cancer.

6.3.3 Direct experimental evidence

Wei *et al*. (2000) reported that 60 Hz magnetic fields can increase the proliferation of human astrocytoma cells and strongly potentiate the effect of two agonists. Juutilainen *et al.* (2006) carried out a pooled analysis of *in vitro* studies and short term animal studies show that MFs enhance the effects of known carcinogens. The studies reviewed cover a wide range of cell types, including brain cells. Overall, the findings lend support for the radical pair mechanism.

6.3.4 Other relevant experimental/theoretical evidence

Melatonin is highly protective of damage to fetal brain (see section 6.1.3). This may be relevant to the aetiology of adult brain cancer.

6.4. Miscarriage

6.4.1 Direct epidemiological evidence

Chapter 13 of the California Report cites 16 studies of miscarriage, 11 of which concern exposure to the earlier types of computer VDUs. The important up-to-date studies are the prospective study of Li *et al*. (2002) and the nested case-control study of Lee *et al*. (2002). Both of these studies found strong associations with maximum field and rate of change metrics of magnetic field exposures. Overall, the California Report classes magnetic fields as class 2B in the IARC system.

See earlier studies by Wertheimer & Leeper (1986 and 1989); Chernoff *et al*. (1992); Shaw & Croen (1983); Delpizzo (1994); Goddijn & Leschot (2000).

6.4.2 Other relevant epidemiological evidence

Of possible interest: there is evidence that sudden infant death syndrome (SIDS) is associated with fluctuations with the geomagnetic field. For discussion see Appendix 1, Section 6.
Melatonin is known to control seasonal breeding in animals.

6.4.3 Direct experimental evidence

High levels in the body of the luteinising hormone, LH hormone is associated with miscarriage (Regan *et al*. 1990) and melatonin significantly decreases LH levels (Voordouw *et al*. 1992).

6.4.4 Other relevant experimental/theoretical evidence

In animals melatonin is highly protective of oxidative damage to the fetus, see section 6.1.3.
Unfinished.

6.5. ALS/motor neurone disease

6.5.1 Direct epidemiological evidence

See references 8.5.1.
Unfinished.

6.5.2 Other relevant epidemiological evidence
Unfinished.

6.5.3 Direct experimental evidence
Unfinished.

6.5.4 Other relevant experimental/theoretical evidence
Reiter (1998) review of the mechanistic evidence linking reduced melatonin and neurodegenerative diseases. Melatonin may be a neuroprotector in ALS (Jacob *et al*. 2002)

6.6. Childhood brain tumours

6.6.1 Direct epidemiological evidence
Unfinished.

6.6.2 Other relevant epidemiological evidence
Unfinished.

6.6.3 Direct experimental evidence
Experiments in animals show that melatonin is highly protective of damage to fetal brain, see section 6.3.1.
Unfinished.

6.6.4 Other relevant experimental/theoretical evidence
Unfinished.

6.7. Female breast cancer

This has been widely discussed in relation to the melatonin hypothesis of Stevens (1987). Full details will follow later.

6.7.1 Direct epidemiological evidence
Check California Report plus review by Erren (2001).

6.7.2 Other relevant epidemiological evidence
MFs plus melatonin in animal experiments. Schernhammer & Hankinson 2005; Stevens 2005.

6.7.3 Direct experimental evidence
Ishido et al, 2001 (and 6 other labs) 1.2 μT MFs inhibit melatonin in suppressing growth of MCF-7 cells *in vitro*. Also, similar papers for Tamoxifen. Blask et al 2005.

6.7.4 Other relevant experimental/theoretical evidence

6.8. Male breast cancer

6.8.1 Direct epidemiological evidence
A pooled analysis by Erren (2001) gave an RR of 1.37 (95% CI: 1.11 – 1.71).
Unfinished

6.8.2 Other relevant epidemiological evidence

6.8.3 Direct experimental evidence

6.8.4 Other relevant experimental/theoretical evidence

6.9 Alzheimer's Disease

6.9.1 Direct epidemiological evidence
See references section 8.9

6.9.2 Other relevant epidemiological evidence
Unfinished.

6.9.3 Direct experimental evidence

6.9.4 Other relevant experimental/theoretical evidence
Reiter (1998) review of the mechanistic evidence linking reduced melatonin and neurodegenerative diseases.

6.10. Suicide

6.10.1 Direct epidemiological evidence
See references section 8.10.

6.10.2 Other relevant epidemiological evidence
See Appendix A2 concerning suicide and geomagnetic fields.

6.10.3 Direct experimental evidence
Unfinished.

6.10.4 Other relevant experimental/theoretical evidence
See Stoff & Mann (1997) "The neurobiology of suicide – from the bench to the clinic".
Unfinished.

Welker *et al.* (1983) Serotonin and melatonin reduced in rat pineal gland by rotating direction of earth's magnetic field

6.11. Heart disease

6.11.1 Direct epidemiological evidence
See reference section 8.11.

6.11.2 Other relevant epidemiological evidence
See Appendix A4 concerning heart rate and heart disease in relation to geomagnetic fields. See also Otsuka *et al.* (2001) electrocardiographic magnetoreception in association with geomagnetic fields.

6.11.3 Direct experimental evidence
Unfinished.

6.11.4 Other relevant experimental/theoretical evidence
Unfinished.

6.12. Depression

6.12.1 Direct epidemiological evidence
List of studies from Ahlbom review plus other papers

6.12.2 Other relevant epidemiological evidence
See Appendix A3 concerning depression and other mental disorders in relation to geomagnetic fields.

6.12.3 Direct experimental evidence
Melatonin *per se* affects depression.

Unfinished.

6.12.4 Other relevant experimental/theoretical evidence
See Wilson, 1988.

6.13. Other illnesses not considered: Parkinson's Disease, SIDS, heart rate variability, Arrhythmia, lung disease, variations in EEG signals, Neuroblastoma, Immune system effects, Endocrine system effects, developmental effects and birth defects

6.13.1 Direct epidemiological evidence
Hakansson (2002); Tynes (????) - malignant melanoma; Floderus (1999); Savitz (1999); Graham (????) - heart rate variation; Cook (????) - EEG signals; Wilkins (????) and De Roos (????) – neuroblastoma; Kleinerman (2005) – glioma; Marino (2000) and Mevissen (????) – immune system; Floderus (1999) & Marino (2001) - endocrine system, Blaasaas (2003) & (2004) – birth defects; Feychting (2005) - developmental affects.

6.13.2 Other relevant epidemiological evidence

6.13.3 Direct experimental evidence
Most papers in this area.

6.13.4 Other relevant experimental/theoretical evidence

6.14. Universal carcinogen

6.15 Other reproductive

7. Tables 2.1

Table 2.1: Taken from the California Report: Table 21.2, page 379

TABLE 21.2 A COMPARISON OF DHS REVIEWERS' DEGREE OF CERTAINTY WITH THAT OF OTHER AGENCIES

HEALTH OUTCOME	NIEHS WORKING GROUP	IARC	NRPB	DHS
Childhood leukemia	2B*	2B	Possible	2B to 1
Adult leukemia	2B (lymphocytic)	Inadequate	Inadequate	2B to 1
Adult brain cancer	Inadequate	Inadequate	Inadequate	2B
Miscarriage	Inadequate	Not Considered	Not considered	2B
ALS	Inadequate	Not Considered	Possible but perhaps due to shocks	2B
Childhood brain cancer, breast cancers, other reproductive, Alzheimer's, suicide, sudden cardiac death, sensitivity	Inadequate	Inadequate or Not Considered	No for Parkinson's disease, inadequate for Alzheimer's, other endpoints not yet considered	Inadequate

* Although the majority of scientists assembled to prepare the NIEHS Working Group Report voted for a "possible 2B" classification for these cancers, the lay person's summary submitted by the Director of NIEHS to Congress stated: "ELF-EMF exposure cannot be recognized as entirely safe because of weak scientific evidence that exposure may pose a leukemia hazard." (Final Report NIH Publication 99-4493, May 1999)

21.0 Conclusions - 379 -
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Table 6.2.1: Adult leukaemia list of studies scanned in from chapter 8 of the California Report form page 121, chapter 8.

8.1 THE PATTERN OF EPIDEMIOLOGICAL EVIDENCE

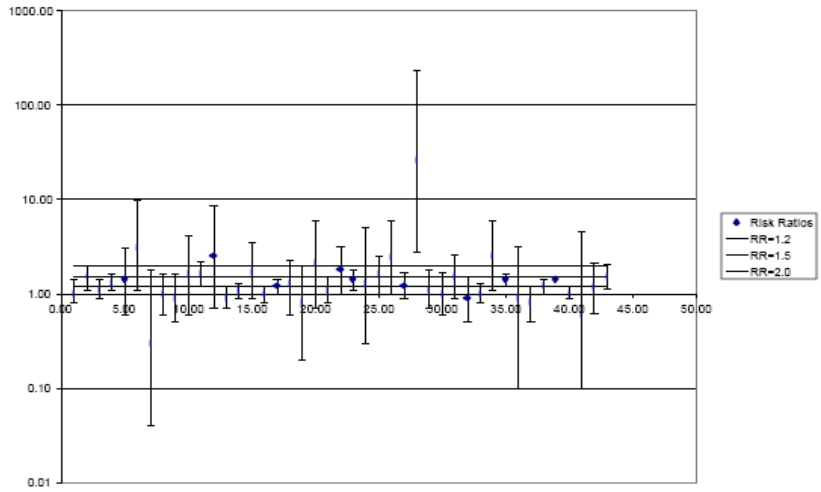

Figure 8.1.1 Studies of Adult Leukemia and EMFs Primarily Based on Kheifets (1997)

Table 6.3.1: Adult brain cancer list of studies scanned in from chapter 9 of the California Report from page 166, chapter 9.

9.1 THE PATTERN OF EPIDEMIOLOGICAL EVIDENCE

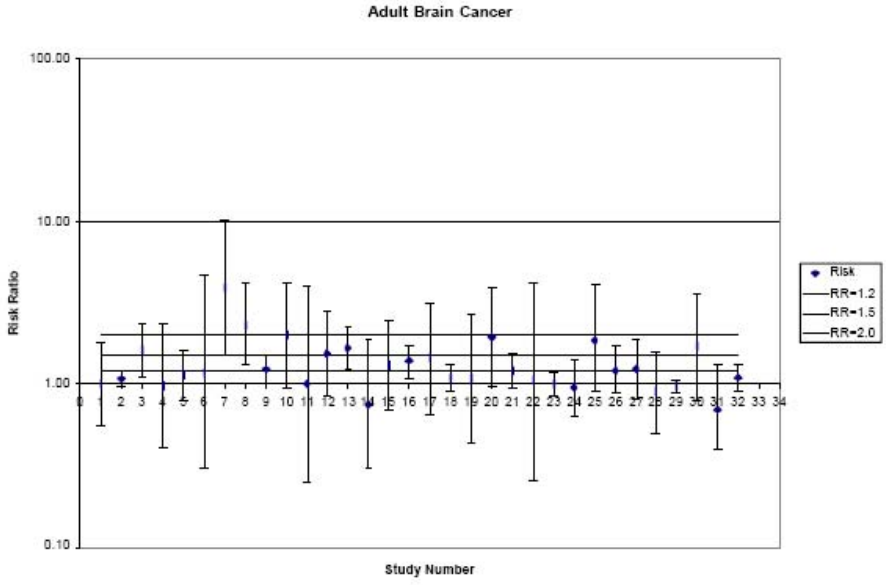

Figure 9.1.1 Studies of Adult Brain Cancer Derived Primarily from Kheifets et al. (1995)

Liz's Alzheimer's Disease and Parkinson's Disease

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8.1.1. Direct epidemiological evidence

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(still to do)

8.1.3 Direct experimental evidence

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