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

**Volume 2 - Appendices**

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# **Appendix 1.**

## **Geomagnetic Fields, their Fluctuations and Health Effects.**

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Fluctuations in the level of the Earth's Geomagnetic Field (GMF), a quasi static magnetic field, and geomagnetic storms have been associated with a number of health effects and disorders in scientific literature spanning more than 50 years.

Presented below is a summary of some of the key health effects and disorders that have been associated with these fluctuations and other changes in the GMF.

- A1. Melatonin synthesis disruption.**
- A2. Suicide.**
- A3. Depression and other mental disorders.**
- A4. Heart rate/disease and blood pressure.**
- A5. Light sensitivity.**
- A6. SIDS**
- A7. Other.**

A summary of some of the key findings in each of these areas is presented over the following pages. In addition there is an explanation of the relevant geomagnetic indices and a list of references for all the papers cited in this document.

## A1. Melatonin synthesis disruption

**Bartsch *et al.* (1994).** Seasonal changes of nocturnal urinary 6-sulfatoxymelatonin excretion in Rats demonstrated peak levels in summer despite constant photoperiods. The authors hypothesise that the horizontal component H of the geomagnetic field may act as a seasonal Zeitgeber because H shows a similar seasonal rhythm, and changes in in the direction and intensity of H can affect pineal activity.

**Burch *et al* (1999).**“...reductions in nocturnal 6-OHMS excretion were higher on days when geomagnetic activity and 60Hz MF exposures were both elevated, *suggesting a common biological mechanism*”. Authors also report that mean overnight 6-OHMS excretion was lower on days when the 36-h Ap or aa values exceeded 30 nT.

**Juutilainen *et al.*(2006).** With a small sample group of 60 women, the authors tested the hypothesis that the inconsistencies seen in studies on the effect of MF's upon melatonin production in humans are due to interaction with light in pineal response to MF's. The authors, with a retrospective analysis based on a questionnaire and actual MF measurements, split the sample group into four groups based on whether they had been exposed to MF's and/or Light-At-Night (LAN) and compared the levels of 6-OHMS excretion for each subgroup. The lowest excretion of 6-OHMS was observed in the group of women who were exposed to both MF and LAN, and the differences between the groups were significant ( $p < 0.0001$ ). The study supports the hypothesis that daytime occupational exposure to MF enhances the effects of nighttime light exposure on melatonin production.

**Weydahl (2001).** A circadian rhythm was found to characterize both melatonin and K index, the peak in K index (23:24) preceding that of melatonin (06:08). During the span of investigation, a circannual variation also characterized both variables. Correlation analyses suggest that changes in geomagnetic activity had to be of a certain magnitude to affect the circadian amplitude of melatonin. If large enough ( $>80\text{nT}/3\text{h}$ ), changes in geomagnetic activity were reported to significantly decrease salivary melatonin concentration.

## A2. Suicide

**Berk *et al* (2006).** Authors report that suicide amongst females increased significantly in autumn during concurrent periods of geomagnetic storm activity ( $p = 0.01$ ). This pattern was not observed in males ( $p = 0.16$ ).

**Gordon and Berk (2003).** The authors found a correlation between suicides and average storm activity in South Africa between January 1980 and December 1992. The effect was shown to be stronger in females ( $p < 0.005$ ) than males ( $p < 0.025$ ).

**Partonen *et al* (2004).** High levels of solar radiation activity were associated with the increased risk of suicide ( $p = 0.00001$ ), but the effect of geomagnetic activity was weak.

### **A3. Depression and mental disorders**

A four year study (**Becker *et al* 1961, Friedman *et al* 1963**) reported a positive correlation between the monthly sums of geomagnetic K indices from Fredericksburg and the monthly admissions to two mental hospitals in New York.

Results in this study suggest that statistically significant low to marked linear relationships exist between geomagnetic parameters and a gross measure of human disturbance. These relationships are evident when the measure of the geomagnetic parameter is restricted to those periods of higher disturbance which can be categorized as magnetic storms.

**Dimitrova *et al* (2002)**. The authors' investigations suggest that most of the people examined in their study during the autumn and spring equinox, irrespective of their health status could be sensitive to geomagnetic changes, which have a direct influence upon self-confidence and working ability.

**Kay (1994)**. The hypothesis that geomagnetic storms may partly account for the seasonal variation in the incidence of depression by acting as a precipitant of depressive illness in susceptible individuals is supported by a statistically significant 36.2% increase in male hospital admissions with a diagnosis of depressed phase, manic-depressive illness in the second week following such storms compared with geomagnetically quiet control periods.

**Krivelyova and Robotti (2003)**. The authors found that, for most of the countries in their sample, the previous week's unusually high levels of geomagnetic activity have a negative and statistically and economically significant impact on today's stock returns. The results are consistent with changes in risk-taking behavior caused by depressive disorders, since geomagnetic storms have been found to substantially increase the incidence of depression and other psychological disturbances among people.

**Persinger (1987)**. The author reports that geomagnetic variations were found to be correlated with enhanced anxiety, sleep disturbances, altered models and greater incidences of psychiatric admissions.

**Raps (1999)**. A pilot study indicates two statistically significant correlations between the numbers of first admissions (psychiatric) per month and the level of solar radioflux in the corresponding month, ( $p < 0.05$ ) and sudden magnetic disturbances of the ionosphere ( $p < 0.01$ ).

### **A4. Heart rate/disease and blood pressure**

**Belov *et al* (1998)**. The authors found a positive correlation of the EEG (electroencephalogram) data with the geomagnetic activity. The strongest correlations were found in the frontal and central areas of the brain. The degree of synchronisation of the spontaneous EEG seems to reflect sensitivity of the human nervous system to the Earth's magnetic field. A stressor response to strong short-term disturbances in the geomagnetic field reveals itself in the form of enhancement of the EEG global synchronisation.

**Chernouss *et al* (2001).** “A correlation between geomagnetic disturbances and heart rate was calculated and different reactions of people on geophysical impact were shown. The special group of ‘Aurora Disturbance Sensitive People’ (ADSP) was revealed. An international study, aimed at evaluation of the impact on human health due to exposure of northern populations to the geophysical risk factor (GRF) in the Circumpolar areas located under the Aurora Belt, is needed. “

**Dimitrova *et al* (2004).** Arterial blood pressure (bp) was found to increase significantly with the increase of the geomagnetic activity (GMA) level, and systolic and diastolic bp were found to increase significantly from the day before till the second day after the geomagnetic storm. These effects were present irrespective of sex and medication.

**Otto *et al* (1982).** In a survey of 66,900 cases of death from ischaemic heart disease increased geomagnetic field, and increased geomagnetic storms were positively and significantly related to death from ischaemic heart disease.

**Srivastava *et al* (1980).** For the monthly geomagnetic mean data for 1972 there was a statistically significant correlation coefficient of  $r=+0.59 \pm 0.20$  with myocardial infarction. A 50% increase in hospital admissions for myocardial infarction was observed during 1978 compared with 1972, suggesting a relationship high sunspot numbers.

## **A5. Light sensitivity**

**Cremer-Bartels *et al* (1983).** The authors found a relationship between the coincidence of static magnetic field variation at near GMF strength and a decrease in human night vision acuity. Suggest that the inhibition of methoxylation of indolamines is involved in the magnetic field sensitivity in humans.

**Partonen (1998).** Author reports that under exposure to rotating magnetic field, the size of pinealocytes is bigger at night than during the day in spring, but there is no day-night difference in autumn. Specialized photo-receptors amplify the influence of a weak magnetic field in patients with winter seasonal affective disorder (SAD), modulating the response of the photoreceptive system to light and the pineal response to a magnetic

**Thoss *et al* (2000).** Authors show that the visual sensitivity of man is influenced by periodic sinusoidal inversion of the vertical component of the geomagnetic field. This effect indicates a visual fixation in the north-south direction, showing a pronounced resonance at a period duration of 110s.

**Thoss *et al* (2002).** Reports the existence of a weak influence of the static field on visual sensitivity in man. If the directions of view line and field vector coincide the perception threshold of alight stimulus is slightly but significantly increased. This significance will be lost if view line deviates by 10 degrees from the field direction.

**Thoss *et al* (2003).** The results from this report imply that looking in the direction of the magnetic field reduces the visual discrimination threshold (by about 4%,  $p < 0.02$ ).

## **A6. SIDS**

**Weissbluth & Weissbluth (1994).** SIDS exhibits circannual, circadian, and ontogenetic features which may reflect an impaired maturation of the photoneuroendocrine system caused by a genetic absence or mutation of the enzyme NAT. The failure of normal pineal gland development and subsequent impaired production of its main secretory product, melatonin, may cause a lethal imbalance in the chemical interactions among serotonin, progesterone, and catecholamines. The result of this chemical imbalance, culminating in SIDS, involves the neurotoxic and cardiomyotoxic effects of abnormally elevated catecholamines and intracellular calcium ions.

The human embryonic pineal glands, at 120 days exhibit two distinctive types of cells observed by light microscopy, and at 150 days, in only some of these cells, the photosensitive pigment melanin appears.

The similarity between fetal and neonatal pinealocytes and retina photosensory cells suggests that the pineal gland is capable of detecting changes in light in utero and during the early postpartum period (Altar, 1982).

Factors which influence pineal maturation include intensity of light or directionality of the photoperiod, that is, whether the duration of day light is seasonally shortening or lengthening.

In humans, the pineal gland is influenced by environmental light and initially contains the capability to function as a photoreceptor and a neuroendocrine organ. It develops into only a neuroendocrine organ at about the third month of life.

Sparks & Hunsaker have confirmed is an extension of their original study, the findings that pineal glands are smaller in SIDS infants than in controls ( $p < 0.005$ ) and that pineal glands are larger in summer than winter.

SIDS infants' pineal glands however did not grow larger in the summer months and perhaps is therefore less responsive to photoperiod stimulating effects compared to normal infants.

In adults, intra-individual variations of 08:00 hr plasma melatonin concentration occur with peak values in January and July (20-22  $\text{pg ml}^{-1}$ ) and troughs in May and October (12-13  $\text{pg ml}^{-1}$ ) and these seasonal differences are statistically significant (Arendt *et al.*, 1979).

Changes in the direction of photoperiod duration shortly after the winter and spring solstice may account for the seasonal peak values, but seasonal temperature differences may also be important. Another characteristic feature of melatonin secretion is that of pulsatility (Reiter, 1991). Discrete burst or episodes of melatonin released during the night are detected if the frequency of sampling is high. Melatonin

receptor sites have been identified in the fetus (Yuan *et al.*, 1991). High melatonin concentrations derived from the mother are present in newborn infants, but they fall rapidly to barely detectable levels within about 1 week. Subsequently, melatonin concentrations remain low until 3-4 months of age, and after this age, the concentrations gradually increase.

Catecholamines – light is received by the eyes, and the photoreceptors in the retina produce a neuronal signal which is transmitted through the suprachiasmatic nucleus (SCN) to ganglionic fibers of the peripheral sympathetic nervous system. The chemical messenger which is released from the post-ganglionic sympathetic nerve-endings terminating on the pineal gland is the catecholamine norepinephrine (NE). Neural responses from the retina during daylight inhibit the release of NE; darkness releases this system from inhibition which permits NE stimulation of the pineal gland to occur. One of the major biochemical responses of NE stimulation of the pineal gland is an increase in the intracellular concentration of calcium ion.

High progesterone levels may be associated with SIDS as they are associated with low levels of melatonin (in rates high levels inhibit the release of melatonin from the pineal).

In 1990 another paper was published which showed that among infants 3 months of age or less, cerebral spinal-fluid melatonin concentrations in SIDS infants were statistically significantly lower than in control infants. Also, lower concentrations of melatonin were detected in the blood of SIDS infants compared to controls.

Serotonin concentrations in the pineal gland exhibit seasonal variation with the highest concentration during the month of December and the lowest concentration during the month of June. In the blood, serotonin concentrations are highest at night. In the newborn infant serotonin concentrations are high and gradually fall over several months. Statistically significant circadian rhythms are well developed immediately after the infant is born.

The absence of the biologically expected nocturnal surge of melatonin in the evening hours, during the first few months of life, especially in the winter when serotonin activity is peaking is the exact congruence of circannual, circadian and ontogenetic features which would be associated with maximum serotonin-induced increases in  $[Ca^{2+}]$ , in the heart and brain.

Progesterone concentrations which are negatively correlated with melatonin concentrations, would also be high causing further increases in  $[Ca^{2+}]$ .

Norepinephrine activity would also be increasing during the first few months causing toxic effects on the heart and brain. The combination of elevated Norepinephrine and depressed melatonin would cause a reduction in the amount of brown fat around the adrenal gland.

The clinical picture of SIDS is that of a weak or mild temperament baby who may appear sleepy, drowsy, or listless; these behavioural features could result from elevated progesterone or calcium. These babies sleep well, do not cry much, but die silently at night, mainly during the winter.

**Leach *et al.* (1999).** Many of the epidemiologic features that characterize SIDS infants and families have remained the same, despite the recent decrease in SIDS incidence in the UK. These included the same characteristic age distribution, few deaths in the first few weeks of life or after 6 months, with a peak between 4 and 16 weeks, a higher incidence in males, lower birth weight, shorter gestation, and more neonatal problems at delivery.

The majority of the SIDS deaths occurred during the night sleep (83%) and there was no particular day of the week on which a significantly higher proportion of deaths occurred.

There has however been a reduction in the previous high winter peaks of death and a shift of SIDS families to the more deprived social grouping.

Just more than one quarter of the SIDS deaths (27%) occurred in the 3 winter months (dec-feb) in the three years of the study.

The report lends weight to the mounting evidence that the association between smoking and SIDS may be part of a casual mechanism.

**Eckert (1992).** Author constructs hypothesis based upon: a) observed clustering of Sudden Infant Death Syndrome (SIDS) cases at places with abnormal geomagnetic fields (GMF) and/or electromagnetic fields (EMF); b) recorded GMF pulsations matching the breathing frequencies of infants; c) the reported immature development of increased dendritic spine density in the brain stem of SIDS cases and; d) the increased dendrite arborisation in the brains of rats exposed to magnetic fields (MF).

The hypothesis consists of two parts:

1. A disturbed GMF in the residence or surroundings of a pregnant woman may interrupt the normal development of the central organ which controls respiration (brain stem) of the fetus. This is termed the 'Selection Factor'.
2. If such an infant with a functional disturbance of the control organ is then exposed to a GMF or EMF with pulsations similar to his own breathing frequency, but inverted in phase, value form etc then the vital nerve impulses from the respiration control organ to the breathing organs may be disturbed or blocked with fatal effect. This is termed the 'Trigger Factor'.

**Persinger & O'Connor (2001).** The author hypothesises that if geomagnetic-mediated stimuli trigger many sudden infant deaths, then the days in which they and hospital admissions for cardiac arrhythmias for adults occur should share a similar source of variance.

Factor analyses of the days in which a sudden infant death occurred in Ontario or adults were admitted for one of eight categories of cardiac crisis in the Sudbury (Ontario) region for the year 1984 supported each shared about 40% of their variance, also shared about 40% of the variance with pulsations (0.2 Hz to 5 Hz) was associated.

The results are consistent with important role of geomagnetic variables in the occurrence of transient electrical anomalies in brain function rather than cardiac blood flow.



**Grainger *et al.* (2000).** Power frequency magnetic fields have been implicated in SIDS. Through the use of a case-control study measuring 50 Hz electric and magnetic fields at the SIDS baby's last head position, no association could be found between SIDS and either electric ( $p = 0.327$ ) or magnetic ( $p = 0.827$ ) 50 Hz fields.

**Sparks and Hunsaker (1988).** The authors observed, through a small (7 SIDS, 5 control) sample post-mortem analysis, a statistically significant ( $p < 0.005$ ) reduction in the anatomic size of the pineal gland in SIDS infants, as compared to age-matched controls.

97% of all respiratory disturbances in infants at risk for SIDS occur during sleep, with a high preponderance occurring during non-REM sleep.

**Sparks and Hunsaker (1997).** The authors found a highly significant ( $p < 0.0001$ ) decrease in the size of the pineal gland in the SIDS population ( $18.2 \pm 0.9 \text{mm}^2$ ) compared with the control population ( $28.0 \pm 2.5 \text{mm}^2$ ). At the same time there was no difference in the mean age, brain weight, or body weight between the control and the SIDS populations as a whole.

**Sparks and Hunsaker (2002).** On the basis of current data, the authors postulate that apoptotic neurodegeneration constitutes the anatomic substrate accounting for the pathophysiologic mechanism and proximate cause of SIDS.

“...the data clearly suggest that there is a neurodegenerative process occurring in SIDS, that a marker of this neurodegenerative process can be useful in diagnosing SIDS, and that, therefore, the syndrome of sudden infant death is a neurodegenerative disorder.”

**Sturner *et al.* (1990).** The authors found a significant correlation for melatonin levels in different body fluids from the same individual. Age adjusted Cerebrospinal Fluid (CSF) melatonin levels were significantly lower among the SIDS infants than among those dying of other causes. A similar, but non-significant, trend was also noted in blood and vitreous humour. Diminished melatonin production may be characteristic of SIDS and could represent an impairment in the maturation of physiologic circadian organization.

...most SIDS deaths occur during sleep and have a distinctive age distribution, reaching a peak at 3 months. Maturation of the normal pattern of sleep behaviour with sleep-time concentrated during the night, takes place during the second month of life. ...studies have demonstrated that the characteristic daily rhythm, in melatonin secretion, with peak values occurring at night, matures with a similar time course, and is detectable in infants at approximately 3 months of age. **The concurrence of these maturation processes and the age incidence of SIDS deaths may thus be related.**

Autopsies were performed within 28hr of death.

**Kennaway *et al.* (1996).** Rhythmic excretion of the urinary melatonin metabolite 6-sulfatoxymelatonin appears between 49-55 weeks postconception (9-15 weeks of age) in singleton babies born at term in hospital.

The authors tested the following hypotheses: that the delay in the appearance of a melatonin rhythm in premature infants is influenced by the underlying reason for the prematurity, that siblings of SIDS victims have a delayed appearance of melatonin rhythmicity, and that continuous lighting in the neonatal nursery contributes to the delayed appearance of melatonin rhythmicity in premature infants.

They confirmed earlier research that infants are born with a functionally immature SCN-pineal gland axis that does not establish significant hormonal rhythmicity until 9-12 weeks of age in full-term infants.

**Tryba *et al.* (2006).** The authors' data suggest an intriguing possibility that, in SIDS victims, disturbances in the serotonergic system may underlie the failure to gasp appropriately and autoresuscitate despite increased release of 5-HT during hypoxia. Serotonin acting via 5-HT<sub>2a</sub> receptors can modulate the respiratory rhythm generating network *in vitro* and respiratory motor neuron activity, including the recruitment and maturation of phrenic motoneurons during development.

\*5-HT receptors are receptors for the neurotransmitter and peripheral signal mediator serotonin, also known as 5-hydroxytryptamine (5-HT).

These receptors are located on the cell membrane of nerve cells and other cell types in animals and mediate the effects of serotonin as the endogenous ligand.

The 5-HT<sub>2a</sub> receptors perform action upon the muscle (contraction, vasoconstriction/dilation) and platelets (aggregation).

\*Hypoxia – this is when the infant is deficient in oxygen. It can be caused by inadequate oxygen transport, the inability of tissues to use oxygen and the reduction in partial pressure of oxygen.

<sup>1</sup>Although the incidence of SIDS varies among countries and ethnic groups, there are always three epidemiological characteristic features:

- (i) A circannual pattern with an excess of deaths due to SIDS in the winter months.
- (ii) A circadian rhythm of SIDS occurring at night.
- (iii) An ontogenetic feature of SIDS occurring after the first few weeks, and during the first 3-4 months of age.

**Goldwater (2003).** – SIDS was first defined in 1969 as “the sudden death of any infant or young child, which is unexpected by history, and which is a thorough post-mortem examination fails to show an adequate cause of death.” (Bergman 1970).

This has since been refined to include only infants dying suddenly under one year of age.

## A7. Other

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<sup>1</sup> Weissbluth & Weissbluth 1993

**Aldrich, Andrews and Liboff (2001).** The authors designed a metric which incorporates the orientations of power lines relative to geomagnetic north. Odds Ratio between DIP angle and incidence of brain cancer 4.86 (95% CI = 1.13 - 20.96). The results from this analysis offer evidence that a metric of EMF's, incorporating a geomagnetic component, may finally provide some clarification on the protracted quandary about EMFs and cancer risk.

**Dupont, M. J. *et al.* (2005).** 32 pregnant rats were exposed for to three days before expected parturition either to a coil that generated 0.5 Hz sine-wave, 5 to 10 nT magnetic fields, or to a reference coil (< 1 nT). Litters born to exposed mothers contained significantly fewer pups than those exposed to the control conditions. There were significantly fewer males and fewer females in litters exposed to the fields generated in the E-W and N-S directions, respectively. The authors suggest these result support the hypothesis that a specific temporal configuration of brief periods of geomagnetic activity can produce an increased incidence of nonvital fetuses, neonates, or infants.

**O'Connor, R. P. and Persinger, M. A. (1997).** The authors hypothesised that sudden decreases in nocturnal melatonin by a specific range of geomagnetic activity would precipitate sudden infant death. They report a correlation of 0.90 between the number of cases of Sudden Infant Death Syndrome (SIDS) and an increase in numbers of days per month with average geomagnetic activity between 11 and 20 nT and 31 through 40 nT, but also a decrease in the number of days with values between 21 and 30 nT.

**Wever (1979).** Author reports that when subjects were placed in a non EMF shielded room compared to a shielded room, and all other factors identical, the period of their autonomous circadian rhythm was shorter, and the tendency towards internal desynchronization decreased.

**Yaga *et al.* (1993).** The authors found that rats exposed to pulsed static magnetic fields during mid or late dark phase significantly suppressed pineal NAT activity, the rate limiting enzyme in melatonin synthesis, as well as the melatonin content in the pineal gland. However, these parameters were not influenced by MF's when the exposure occurred early in the dark phase or during the day. These results suggest the responsiveness of the pineal gland to magnetic field perturbations change throughout the photoperiod.

## Geomagnetic Activity Indices<sup>1</sup>

<b>Kp Index</b>	<b>Ap Index</b>	<b>Magnetic Field level, nT</b>	<b>Geomagnetic Field Conditions</b>	<b>Storm category/ average frequency of occurrence</b>
<b>0</b>	<b>0-2</b>	<b>0-5</b>	<b>Very Quiet</b>	<b>-</b>
<b>1</b>	<b>3-5</b>	<b>5-10</b>	<b>Quiet</b>	<b>-</b>
<b>2</b>	<b>6-9</b>	<b>10-20</b>	<b>Quiet</b>	<b>-</b>
<b>3</b>	<b>12-18</b>	<b>20-40</b>	<b>Semi-Quiet</b>	<b>-</b>
<b>4</b>	<b>22-32</b>	<b>40-70</b>	<b>Unsettled</b>	<b>-</b>
<b>5</b>	<b>39-56</b>	<b>70-120</b>	<b>MINOR STORM</b>	<b>G1/~2.4 days</b>
<b>6</b>	<b>67-94</b>	<b>120-200</b>	<b>MAJOR STORM</b>	<b>G2/~6.8 days</b>
<b>7</b>	<b>111-154</b>	<b>200-330</b>	<b>SEVERE STORM</b>	<b>G3/~20 days</b>
<b>8</b>	<b>179-236</b>	<b>330-500</b>	<b>SEVERE STORM</b>	<b>G4/~40.5 days</b>
<b>9</b>	<b>300-400</b>	<b>&gt;500</b>	<b>EXTREMELY SEVERE</b>	<b>G5/~2<sup>3</sup>/<sub>4</sub> years</b>

<sup>1</sup>“playing the Field: Geomagnetic Storms and International Stock Markets”. Anna Kriveloya and Cesare Robotti, 2003.

<sup>1</sup>The subscript “p” means planetary and designates a global magnetic activity index. The **K Index** is a three hour range index. K indices isolate solar particle effects on the earth’s magnetic field; over a 3-hour period, they classify into disturbance levels the range of variation of the more unsettled horizontal field component. Each activity level relates almost logarithmically to its corresponding disturbance amplitude. Three-hour indices discriminate conservatively between true magnetic field perturbations and the quiet-day variations produced by ionospheric currents.

K indices range in 28 steps from 0 (quiet) to 9 (greatly disturbed) with fractional parts expressed in thirds of a unit. A k-value equal to 27, for example, means 2 and 2/3 or 3-; a K-value equal to 30 means 3 and 0/3 or 3 exactly; and a K-value equal to 33 means 3 and 1/3 or 3+. The arithmetic mean of the K values scaled at 13 observatories globally gives Kp.

Equivalent Amplitude- The a-index ranges from 0 to 400 and represents a K-value converted to a linear scale in gammas (nanoTeslas) – a scale that measures equivalent disturbance amplitude of a station at which K=9 has a lower limit of 400 gammas.

**aa index.**<sup>2</sup> A daily and half daily index of geomagnetic activity determined from the k indexes scaled at two nearly antipodal stations at invariant magnetic latitude 50 degrees (Hartland, England, and Canberra, Australia). The aa values are in units of 1 nT. The index is available back to 1868, and is provided by the Institut de Physique du Globe de Paris, France.

**Ak index**<sup>3</sup>. A daily index of geomagnetic activity for a specific station or network of stations (represented generically here by k) derived as the average of the eight 3-hourly ak indexes in a Universal Time day.

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<sup>1</sup> [ftp://ftp.ngdc.noaa.gov/STP.GEOMAGNETIC\\_DATA/INDICES/KP\\_AP/](ftp://ftp.ngdc.noaa.gov/STP.GEOMAGNETIC_DATA/INDICES/KP_AP/)

<sup>2</sup> <http://www.saunalahti.fi/fmbb/astro/indices.htm>

<sup>3</sup> <http://www.sci.fi/~fmbb/astro/indices.htm>

**National Oceanic & Atmospheric Association Space Weather Scale  
for Geomagnetic Storms**<sup>1</sup>

Category		Effect (some or all of these are possible)	Physical measure	Average Frequency (1 cycle = 11 yr)
G 5	Extreme	<p><b>Power systems:</b> grid systems can collapse and transformers experience damage.</p> <p><b>Spacecraft operations:</b> extensive surface charging, problems with orientation, uplink/downlink, and tracking satellites.</p> <p><b>Other systems:</b> pipeline currents reach hundreds of amps, HF (high frequency) radio propagation impossible in many areas for one to two days, satellite navigation degraded for days, low-frequency radio navigation out for hours, and the aurora seen as low as the equator.</p>	Kp = 9	4 storm events per cycle at this Kp level (4 storm days per cycle)
G 4	Severe	<p><b>Power systems:</b> possible voltage stability problems, portions of grids collapse and protective devices trip.</p> <p><b>Spacecraft operations:</b> experience surface charging and tracking problems, orientation problems need corrections.</p> <p><b>Other systems:</b> induced pipeline currents affect preventive measures, HF radio propagation sporadic, satellite navigation degraded for hours, low-frequency radio navigation disrupted, and the aurora seen as low as the tropics.</p>	Kp = 8, including a 9-	100 per cycle (60 days per cycle)
G 3	Strong	<p><b>Power systems:</b> voltage corrections required, false alarms triggered on protection devices, and high 'gas-in-oil' transformer readings likely.</p>	Kp = 7	200 per cycle (130 days per cycle)

<sup>1</sup> <http://geology.about.com/library/bl/blgeomagstormscale.htm>

		<p><b>Spacecraft operations:</b> surface charging on satellite components, increased drag on satellite, and orientation problems need corrections.</p> <p><b>Other systems:</b> intermittent satellite navigation and low-frequency radio navigation problems, HF radio intermittent, and the aurora seen as low as mid-latitudes.</p>		
G 2	Moderate	<p><b>Power systems:</b> high-latitude power systems affected.</p> <p><b>Spacecraft operations:</b> corrective actions are required by ground control; changes in drag affect orbit predictions.</p> <p><b>Other systems:</b> HF radio propagation fades at higher latitudes, and the aurora seen as low as 50 degrees.</p>	Kp = 6	600 per cycle (360 days per cycle)
G 1	Minor	<p><b>Power systems:</b> weak power grid fluctuations.</p> <p><b>Spacecraft operations:</b> minor impact on satellite operations.</p> <p><b>Other systems:</b> the aurora seen at high latitudes (60 degrees); migratory animals begin to be affected.</p>	Kp = 5	1700 per cycle (900 days per cycle)

## A8. References

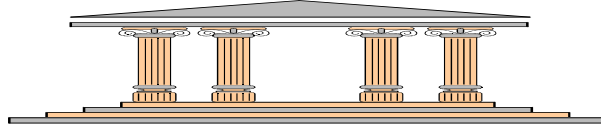
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## Appendix 2

### Health effects of exposure to power frequency electric fields



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#### SCIENTIFIC BACKGROUND

##### Epidemiology

Most of the 20 or so epidemiological studies to date report a modest elevation of childhood cancer incidence (roughly  $<2.0$ ) at field strengths only seen in a very small percentage of homes. This nevertheless is sufficiently persistent to persuade IARC to classify ELF magnetic fields as possibly carcinogenic (Class 2B). IARC does not have sufficiently adequate evidence to pronounce about ELF electric fields however.

It is right to separate out the two components (electric and magnetic) since

- a) they are entirely unrelated at power frequencies
- and
- b) they have very different characteristics.

Only when an electromagnetic wave has reached plane wave conditions can one deduce the strength of magnetic fields from the accompanying electric component, since at say 50 Hz the wave length is some 6000 kilometres long and no plane wave can be formed adequately inside one sixth of that distance, so exposes are in the radiating near field. In essence then, and unlike RF/MW studies, no studies calculating or measuring the ELF magnetic component can say anything at all about the electric component's effects. At higher frequencies the electric component increasingly carries most of the energy, so routinely meters collect the electric component when measuring such sources as RF masts or equipment.

For reasons set out below most of the ELF epidemiology has focused on *magnetic* fields. There are however eight residential studies in the literature addressing the ELF *electric* field, and several occupational studies. These are set out chronologically in Table 1.

**Table A2.1: Residential ELF electric field exposure studies in childhood leukaemia homes**

Study	Country	Subjects	Residence examined	Exposure assessment method		Diagnostic group	Result	
Savitz et al 1988	USA (Denver, CO)	356 cases (103 leukaemia) (128 measured) from cancer registry and hospitals, controls from random digit dialling	diagnosis	three orthogonal single-axis, spot measurement	centre of three different rooms including bedroom, meter in stand, average of different rooms	all cancer 0-14	>14 v <6	0.90 (0.43-1.88)
							9-14	1.23 (0.68-2.22)
							6-9	0.88 (0.49-1.58)
Coghill, Steward and Philips	UK	56 cases from media advertising and personal introduction. 56 controls suggested by parents of cases.		Single-axis (vertical), 24 hours but results presented for 12 hours night-time,	variable position in bedroom (on bed or on stand beside it)	leukaemia	>20 v <5	4.69 (1.17-27.78)
							>10 v <5	2.86 (1.16-8.00)
London et al 1991	USA (Los Angeles, CA)	232 cases (136 measured) from cancer surveillance	House in study area occupied longest by	three orthogonal single-axis spot measurements,	centres of 3 or 4 rooms including bedroom and	leukaemia 0-10	>90 v <50%	0.44 (0.19-1.01)
							75-89	1.11 (0.58-2.56)

		programme; controls from friends or random digit dialling	case during life		living room, results presented for bedroom only, normal power		50-74	0.66 (0.36-1.21)
Tynes and Haldorsen 1997	Norway	cases from cancer registry, controls from population registry and census	whole life	Calculation of unperturbed field from powerline	closest corner of residence to powerline	all cancers 0-14	“electric fields were not significantly associated with cancer”	
McBride et al 1999	Canada	449 cases from treatment centres and cancer registries; controls from health insurance rolls	diagnosis	48 hour Personal monitoring, meter worn in backpack		leukaemia 0-14	>20 v <20	0.80 (0.50-1.27)
Green et al 1999	Canada	88 cases from hospital records, controls from telephone marketing lists		Personal monitoring, meter by child, or worn in backpack or pouch	exposure only during time meter was worn, ie not at night	leukaemia 0-14	>11.6 v <5.6	0.5 (0.1-4.7)
							7.9-11.6	0.3 (0.1-0.9)
							5.6-7.9	0.3 (0.1-1.2)
						ALL 0-14	>11.6 v <5.6	0.4 (0.1-0.9)
							7.9-11.6	0.3 (0.1-1.5)
							5.6-7.9	0.4 (0.1-2.3)

Dockerty et al 1998	New Zealand	344 cases 0-14 from national registries, controls from birth records	House lived in at time of interview	single axis (unspecified direction), 24 hours	Daytime room and bedroom (under bed)	leukaemia 0-14	>10.75 v <3.64	bedroom: 2.3 (0.4-12.7) daytime room: 2.5 (0.3-18.4)
UKCCS 2002	UK	273 cases from paediatricians and oncologists, controls from health service registers	during one year prior to diagnosis	Single-axis (vertical), 48 hour	next to bed, meter in stand	all cancer 0-14	Results not presented	
				Three orthogonal single-axis, 2 sets of 3 minute measurements 48 hours apart	in bedroom (side of bed and on bed) and living room; results presented for mean of two on-bed positions	all cancer 0-14	>20 v <10	1.26 (0.77-2.07)
							10-20	1.35 (0.91-2.00)
						leukaemia 0-14	>20 v <10	1.32 (0.73-2.39)
							10-20	1.42 (0.88-2.27)
Calculation from HV power line (3380 cases)		all cancer	>20 v <10	1.18 (0.69-2.00)				
		leukaemia	>20 v <10	1.12 (0.58-2.17)				

Table A2.1 was constructed by John Swanson of the National Grid, who argues that this background does not encourage interest in the ELF electric field. However if the table is rearranged to reflect the order of *nocturnal exposure strength at the child's bedplace*, a clear dose response pattern is seen:

**Table A2.2: Bedplace ELF electric field exposure in childhood cancer homes**

Study	Exposure measures	Odds ratio (OR) or relative risk (RR)	95CI%
Coghill, Steward et al, 1996	12 hours TWA, bedplace only	<b>4.69</b>	1.17-27.0
Dockerty, Elwood et al, 1997	24 hours TWA, bedroom and one other room	<b>2.3</b>	0.4 – 12.7
Skinner Mee et al., 2002	Spot, 48 hours apart, bedplace and one other room	<b>1.42</b>	0.88-2.27

Incidentally, when we took out a subset in our study of measurements at the child's pillow only, the RR rose to over eightfold (not published). The remaining five studies used protocols unable properly to characterise the electric field exposure, and accordingly did not show any differences between cases and controls:

**Table A2.3: Residential ELF electric field studies with poor characterisation**

Study	Year	Deficiency
Savitz, Wachtel et al, *	1988	room centres only
Green et al,	1999	daytime personal monitor
McBride, Gallagher et al.,	1999	48hrs personal monitor
Tynes and Haldorse	1997	room corner nearest HT line
London, Thomas et al,	1991	spot measurements only

\* abandoned a third way through the collection.

It is my contention that the three studies actually measuring fields at that place and time where the child is most likely to be, namely the bedplace between say 2000hrs and 0800hrs the next day, show firmly that there is a stronger association between electric fields and childhood leukaemia than that seen in the magnetic field studies.

It would be possible for the Skinner, Mee et al data to be reanalysed to extract the data relating to nocturnal exposure, as a test of this hypothesis. In their study of course the 48 hours data still dilutes the nocturnal bedplace exposure to about a third, since the children would have only been in bed for two periods of about 8 hours (say 16hrs in total) during the 48 hrs period.

The Bradford Hill criteria for proving causation of any disease include other kinds of scientific study, since epidemiology alone can only show association, not causation. Another requirement is to demonstrate and replicate a plausible biological mechanism of interaction. This has never been satisfactorily done for the magnetic component, but the literature is

heavy with example of ELF electric field effects, not only in the case of the leukaemias but also a number of other disorders.

### **Cell studies**

In early work, Suzanne Bawin and Ross Adey at Loma Linda showed in 1976 that electric fields replicably caused an efflux of calcium from live cat brains, not only at RF but also at ELF frequencies. Since calcium acts as a first and second messenger for instructing cells to execute vital life processes, its presence in the cell cytosol (interior fluid) is jealously avoided by means of calmodulin, so that of the biological ions calcium is by far the least concentrated. Calcium has a number of other important roles including those within the mitochondria where adenosine triphosphate (ATP) is synthesised. There it potentiates nitric oxide synthase (mNOS), a newly discovered enzyme controlling the rate of ATP synthase.

In consequence that ELF electric fields can disturb the sequestration of calcium is a worrisome finding. To avoid a long explanation the quotation below from a recent website (<http://www.icswebsite.com/emf/emfissues/emfissues9.html>) sets out the background in brief:

“One of the most experienced researchers in the field, Dr. Ross Adey, in 1988 [ref. 17] presented a three step model involving calcium ions which could explain observed EMF induced biological effects. Key to the model is the activation of intracellular messenger systems (adenylate cyclase and protein kinase) by calcium in a stimulus amplification process across the cell membrane.

Numerous scientific studies have demonstrated the physiological importance of calcium [ref. 13]. Calcium functions as a ubiquitous intracellular messenger. For example, in 1947 it was shown that an intracellular injection of a small amount of calcium causes a skeletal muscle to contract. In recent years it has become clear that calcium acts as an intracellular messenger in a wide variety of cellular responses, including secretion and cell proliferation. In nerve cells, calcium influx has been shown to be involved in the initiation of neurotransmitter secretion; the calcium enters the cells through voltage-gated ion channels that open when the plasma membrane of the nerve terminal is depolarized by an invading action potential. Another function of calcium in all cells is to regulate metabolic processes in conjunction with the calcium-binding protein calmodulin. Many enzymatic processes are regulated by calcium. Calcium has been shown to modify gene transcription.

Thus, induced alteration of intracellular calcium concentrations which disrupt the homeostasis of the cell, has serious consequences for the health and future development of the cell. Calcium ions in partnerships with biomolecules have been shown to control the proliferation of non-tumorigenic cells in vitro and in vivo. The evidence points to calcium and a biomolecule called AMP being co-generators of the signal committing the cell to DNA synthesis. Calcium influx in a cell stimulates proliferation, whereas calcium efflux does the opposite.

Balcer-Kubiczek (1994) [ref. 18] linked intracellular calcium levels to the future of damaged cells between becoming transformed (cancer) or dying by apoptosis (the healthy situation). Mattana et al. (1997) [ref. 19] described the importance of calcium ions for cell homeostasis which controls a variety of cellular responses determining the health of the cell. Hence, reductions in intracellular calcium have a very important effect.

Calcium ions are involved in the function of gap junctions or protein structures which link adjacent cells and provide a channel for the passing of messenger molecules. The gap junction can open and close to control the flow. The opening and closing is regulated by calcium ion concentration. Thus, calcium plays another key role in maintaining or interrupting the communication mechanisms for maintaining the health of cells because gap junctions are used to sense differences between cells and to initiate corrections in regulatory behavior as necessary.

It is widely accepted that calcium plays a central role in the development of the immune system response. An elevation of calcium ions is a nearly universal feature associated with activation of cells of the immune system. Using T-cell human leukemia cells, Lindstrom et al. (1995) [ref. 20] replicated and extended the research of

other scientists and showed, that oscillating low level EMFs produce the same calcium ion reaction as does an antibody.

Numerous scientific studies [ref. 13] have demonstrated that EMFs can alter the membrane ion pumps responsible for pumping calcium, sodium and potassium in and out of the cells. Effects have been shown at low current densities, thousands of times lower than currents induced by MW fields. ELF fields have been shown to have the same effect. **Reference is made to a scientific paper from 1992 [ref. 21] mentioning 10 different laboratories which have demonstrated these effects of calcium.**

Dr. Cherry of Lincoln University, New Zealand [ref. 13] concludes: There is extremely strong evidence that both ELF and ELF modulated RF/MW radiation causes calcium ion efflux from cells which significantly alters the intracellular calcium concentrations, reducing the efficacy of lymphocytes in the immune system, participating in the alteration of transformation of pineal serotonin to melatonin and altering the damaged cells likelihood of becoming neoplastic (cancerous) or dying by apoptosis.

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It should be clear from the above that the calcium efflux research is well established by many laboratories, and that this is primarily an electric field effect.

Another well established effect is the inhibition of lymphocyte response to mitogenic challenge in the presence of both RF and ELF electric fields. This work was pioneered by Dan Lyle in the early 1980s, who reported that the cells were not able to respond after exposure for as little as four hours (Lyle, Ayotte et al., 1983; Lyle, Schechter et al, 1988). The same sort of effect was reported by Italian researchers (Conti, Giganti et al). Parallel work by Liburdy at UCLA also found that the electric field not the magnetic was the active parameter in inhibiting the beneficial effects of Tamoxifen on breast cancer cell lines, and this was subsequently repeated at four other laboratories, though not well reported in the peer reviewed literature, only at conferences.

Martin Blank and colleagues at Columbia have shown that the ELF electric field also acts as a stressor with effects on heat shock proteins, particularly HSP 70 where its gene expression is upregulated in a manner similar to that in thermal insult.

Finally in our own lab we have recently demonstrated that exposure of aerated double distilled and HPLC grade water to ELF electric fields causes the formation of nitrites and nitrates, again a worrisome feature since these even at micromolar concentrations can deaminate DNA and inhibit ATP synthesis as reported in well conducted peer reviewed studies from other labs.

***In short, there is ample and reproducible evidence at a cellular level that the ELF electric field can act as an important adverse factor in the maintenance of cell processes and viability, particularly lymphocytes, which are the cell of interest in childhood leukaemia.***

### **Animal Studies**

Live animal studies are arguably of some use in identifying biological responses to EMF exposure, but also suffer disadvantages in that one cannot always argue from them that the same applies to human beings. For example the percentage of lymphocytes in mice differs significantly from the human level, in that whereas in mice 70 percent of white blood cells are lymphocytes, they are less than 20 percent in human beings. Physical morphology also obvious is not comparable.

Nevertheless there are numerous animal studies recording bioeffects from exposure to ELF electric fields, probably the most famous of which are the early sensitivity studies by Kalmijn on elasmobranch fishes, who showed *inter alia* that sharks can sense electric fields as low as a quarter millionth of a volt/metre. The 1950s *Gymnarchus Niloticus* studies of Lishman are another (previous) example of this extreme sensitivity.

I have already mentioned the 1970s Bawin and Adey studies of electric field effects on living cat brains. A 1989 statistical analysis of some 40 animal experiments from six laboratories by Morris, Kimball et al. concluded that ELF electric fields have an adverse effect on lymphocyte function. Hannsen in 1981 reported that small mammals grazed under powerlines exhibited damage to their brains' Purkinje cells. Saunders and McCaig, by contrast, reviewed the literature concerning developmental effects of weak electric fields but mainly in relation to DC rather than ELF. They did not discount the possibility that significant perturbation of the organism's normal endogenous electric field could result in developmental anomalies at field strengths above the present ICNIRP guidelines, and recommended that further studies be carried out.

Animal studies involved electric field exposure on the whole are not numerous, nor do they particularly concentrate on the electric component: an early study on hens (Krueger, Giarola et al 1975) found that exposure to an ELF electric field lowered fertility. Frank Prato's group in 1999 reported an involvement of nitric oxide in the responses of land snails to heat in the presence of ELF EM fields, and though the finding is significant, their study design did not segregate the electric from the magnetic field.

In a series of experiments at Battelle Pacific North West Laboratories in the 1980s Wilson and colleagues found that ELF electric fields disrupted melatonin synthesis and NAT activity (NAT is the rate limiting enzyme in melatonin synthesis) in rats (Wilson, 1988).

Studies of effects on larger mammals include those on primates, cattle and sheep. At SRI a long series of studies found effects of ELF electric fields on operant behaviour, but only at around 30kV/m (Rogers, Orr et al., 1995). Studies exposing the primates to rapid onset electric fields however had a profoundly disrupting effect on melatonin synthesis (Rogers, Reiter et al, 1995). Hefeneider McCoy et al in 2001 reported positive effects on interleukin 1 (an indicator of T cell responsiveness) in a series of two studies on ewe lambs, but found that age and using lower field strength might be responsible for a negative result in their third investigation. Studies of cattle grazed under powerlines are also somewhat inconclusive: there seem to have been no adverse effects on endogenous melatonin levels (Lee, Stormshak et al., 1995), but a more recent study (Rodriguez, Petitcherc et al, 2003) reported lengthened estrous and luteal cycles in cows exposed to 10kV/m.

In short the evidence from animal studies is patchy, but does not allow one to rule out the possibility that ELF electric fields have adverse effects, particularly on melatonin synthesis.

### **Human Studies**

These have inherent difficulties for ethical reasons. Early studies found that electric field exposures caused alterations in circadian rhythm (Wever, 1973) triglycerides (Beischer, 1977), and cognitive effects (Trimmel and Schweiger 1998). None of these studies segregated the electric component effectively however.

This evident dearth of research attention in critical areas is unfortunate since it has precluded regulatory authorities in the West from evidence based standard setting except at the much higher thermal levels, despite much evidence that biological effects are reported well below these. The same conclusions were not shared in Eastern bloc countries, where the view is that mankind has always lived in a magnetic field, and "if anything is novel in the environment since the advent of electricity it is the electric component" (Shandala, Dumanskyi et al., 1988). From the outset their own studies reported electric, not magnetic field effects among switchyard workers in the former Soviet Union (Asanova and Rakov, 1958).

### **Mechanisms of interaction**

If the evidence of electric field bioeffects from cellular, live animals and human studies is patchy, the reverse is true when mechanisms of interaction between electric fields and organisms are reviewed. Whereas hypotheses embracing the magnetic component such as ion cyclotron resonance have been the subject of much controversy and lack of replication, mechanisms of electric field effects are by and large robust and well accepted by the scientific community.

The major endogenous fields in any multicellular organism are electric fields arising from the sino-atrial node of the heart and the cortical polar tracts and their commissures in brain, and both these functions are accepted as vital indicators. Development of the organism moreover is well accepted as subject to endogenous electric fields, as is the regeneration of damaged limbs and the repair of soft and hard tissue wounds.

Moreover, the way organic energy is produced by the synthesis of adenosine triphosphate (ATP) relies upon electron transport and a potential difference across the inner mitochondrial membrane, and this can be depolarised by electric fields. These established mechanisms probably owe their evolution to the almost total absence of any alternating electric fields in the environment before electricity.

Because electric fields are used for so many life processes, there are an equal number of ways these can be disrupted, given that electric fields are superpositive with no lower limit. Any charged particle will disrupt cell stability, the clearest example being free radicals, which are unpaired electrons at their simplest level. Accordingly the body has developed free radical scavengers such as catalase, superoxide dismutase and glutathione to quench these.

At night however when the body's essential cell repair by mitosis is largely carried out (some 500 million cells are lost each daily in a normal adult), molecular oxygen may not be sufficient to act as an adequate final electron acceptor in the electron transport chain, so additional assistance is provided by melatonin. If this process is disrupted or not able to quench additional free radicals resulting from electric field insult, then a large and widespread cascade of adverse effects are likely.

Of these we have now shown in our laboratory that ELF electric fields create nitric oxide and its more stable daughters nitrite and nitrate when HPLC grade water is aerated in their presence, and at field strengths routinely found near appliances and powerlines. Given the exquisite conductivity of body fluids, composed as they partly are of sodium cations and chlorine anions (see Foster, 2003) these radicals can permeate the organism with some speed. We showed in 1996 that electric fields were associated with childhood leukaemia. It is therefore no surprise to note that the level of nitrite in their serum is four times normal, and the level in their cerebrospinal fluid is two orders of magnitude higher than that of normal healthy controls.

Since it is now generally accepted that nitrite at micromolar levels is sufficient to deaminate DNA and inhibit ATP synthesis, this discovery builds the bridge between two well accepted biological mechanisms and thereby provides a complete explanation of how weak ELF electric fields can exert an adverse effect on health.

The effect is not confined to childhood leukaemia, though I accept this may be the concern of interest to the charity and the Commission alike. Table 4 below lists a number of disorders already linked to EMF by studies in the literature, and shows how in each case there are also studies linking the same disorders to excess nitrite/nitrate concentrations in the patient.

**Table A2.4: Health Disorders associated both with EMF and NOx**

Condition	EMF - implicating studies	NOx - implicating studies
Alzheimers	Qiu, Fratiglioni et al 2004	Coyle & Puttfarcken, 1993
Arthritis	De Mattei, Pellati et al 2004	Boveris, Alvarez et al 2002
Bradycardia	Asanova & Rakov 1958	Sanhueze, Riquelme et al 2005
Breast cancer	Wilson, Stevens et al 1997	Nasamura, Yasuoka et al

		2006
Cancer	Nichols and Sorahan 2005	Masri, Comhair et al 2005
Diabetes	Oztas, Kalkan et al 2004	Parslow, McKinney et al 1997
Epilepsy	Fuller, Wilson et al 2003	Faradji, Rousset et al 2000
Immune system dysfunction	Rosenspire, Kindzelshii et al 2005	Bonhomme-Faivre et al 2003
Leukaemia	Draper, Vincent et al 2005	Kellner & Zunino 2004
Myalgic Encephalopathy	Coghill, 2006	Pall, 2000
Myocardial Infarction	Savitz, Liao et al 1999	Alonso & Radomski, 2003
Myopathies	Asanova & Rakov 1966	Jeyarasasingam, et al 2000
Neurodegenerative (ALS)	Hakansson, Gustavsson et al 2004	Moisse & Strong 2006
Parkinsons	Sandyk, 1993	Pall, 2002
Sudden Infant Death	Eckert, 1976	George, Wiklund et al 1997

A Japanese study in 2000 confirmed the ability of weak ELF magnetic fields (0.1mT) to enhance endotoxin-induced nitric oxide in biological fluids *in vivo* in mice, but did not recognise the need to segregate the electric component (Yoshikawa, Tanigawa et al., 2000).

In conclusion, this promising line of research has still to be followed up, though its wider implications have been in evidence for perhaps several decades (it was first reported in 1958 by Reiter and Reiter, though not in the context of biology, only in meteorology), and certainly since Furchgott reported the role of NO as a muscle relaxant in the 1980s. However, the nitrite/nitrate connection with EMF exposure now underpins with experimental evidence the important role of environmental ELF electric fields in disease states in general and leukaemia in particular.