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Environmental Electromagnetic Energy and Public Health

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INTRODUCTION

Electromagnetic fields (EMFs) are real, physical, incorporeal entities that arise from the existence and motion of atomic charges. Man-made EMFs became increasingly common constituents of the general and workplace environments early in the 20th century. Some life-styles and occupations are associated with more than the average amount of exposure to EMFs. People who live near high-voltage powerlines, for example, experience strong electric and magnetic fields that are not usually present in neighborhoods without powerlines. People who use electric blankets similarly experience stronger fields for longer durations compared to the general population. Navy shipboard personnel are exposed to EMFs from many shipboard radars, and in this regard their work environment differs significantly from that of other young men. People living near airports or antenna farms are exposed to radar beams or broadcast radiation, and such residential areas therefore differ from other socio-economically comparable areas with regard to the content of the electromagnetic background. Amateur radio operators experience more EMF exposure than the general population because of their proximity to radiating antennas.

Many patterns of living, working, playing, or resting can be identified with increased intensity and duration of exposure to EMFs. The existence of groups in the general population that experience increased exposure raises the question of whether these groups exhibit an increased incidence or prevalence of disease that is linked to EMF exposure.

In the USA this issue first surfaced in the early 1970's when U.S. powerline engineers heard Soviet reports of adverse health effects among Soviet powerline workers (1). The EMF hazards issue was debated in lengthy hearings in New York, and in two hearings in California, in the context of proposed power lines. The U.S. Department of Defense and Energy became involved as the questions widened to include high frequencies. Now, near the second decade of interest in health hazards of environmental EMFs, the data is sufficiently crystallized to permit a realistic assessment of the extent of the problem. This is my purpose.

I begin with an effort to cultivate appreciation for the nature and extent of artificial EMFs in the environment. Next, bioassay studies performed in the laboratory are described. They are important because they convince us that EMFs have physiological significance, and hence have at least a possibility of promoting disease. Equally as important, the bioassay studies tell us of the general nature of EMF-induced bioeffects by keying the EMF studies into a larger and more

established literature — that dealing with chronic stressors. Chronic stressors promote disease, and it is the fact that environmental EMFs are chronic stressors that makes them determinants of human disease. Following my discussion of these points, I describe the reports of the epidemiology of environmental EMFs. It is lamentable that society requires actual evidence of overt disease in innocent people — dead bodies — before considering protective steps. But such steps are expensive, and they arguably imperil public order and national security. I do not comment on these arguments, but my views on the public-health significance of environmental EMFs are given in the concluding section.

This Chapter is devoted to an attempt to organize data into a rational framework. I have focused on the reports that are particularly pertinent to the mainstream public-health issues of environmental EMFs. I have not omitted data merely because it does not conform to my (or anyone else's) opinion, and I have tried not to use my opinion to gloss over the absence of data.

EMFs IN THE ENVIRONMENT

The artificial electromagnetic environment of the USA is a superposition of contributions from many sources having diverse operating characteristics. They include high- and low-power emitters that can be omnidirectional or directional, and that can operate continuously or intermittently. At high frequencies, the general EMF background consists predominantly of the AM radio band (0.535–1.604 MHz) and the FM and television band (54–806 MHz). There are approximately 5000 FM and 5000 AM broadcast stations in the USA, and more than 1200 television stations (2). At any given moment about half the U.S. population is exposed to these sources at levels above 0.005 μW/cm2, and about 1% is exposed above 1 $\mu W/cm^2$ (3).

EMFs emanating from the electrical power system (60 Hz in the USA, 50 Hz in Europe and the USSR) constitute most of the artificial low-frequency electromagnetic background. The average background 60-Hz electric field is about 1 V/m, and the average background magnetic field is about 800 μGauss.

EMFs much greater than the background are found in the vicinity of specific sources. The power density from a typical 50,000-watt AM radio station does not decrease below 1 μw/cm2 within a radius of about 3280 ft (4). FM radio stations vary in strength and antenna design, but 193 of 2750 such stations in the USA probably have levels exceeding 1000 $\mu w/cm^2$ within 200 ft of the antenna (5). In large urban areas, the elevation necessary for transmission of radio and television signals is sometimes obtained by mounting the antennas atop a tall building. This produces high EMF levels in nearby buildings (6).

When antennas are grouped, they produce relatively intense EMF levels over broad areas. Mount Wilson, California, for example, has 27 radio and television antennas, and they produce strong EMFs in both public and private areas (7). The Sentinel Heights area south of Syracuse, New York contains about a dozen transmitters and they result in essentially ambient levels of about 1 $\mu W/cm^2$ throughout an area of several square miles (8). Only a few of the radiation

hotspots have actually been measured. The Environmental Protection Agency (EPA) measured the radiofrequency radiation levels at Honolulu, Hawaii in 1975 (9), and returned 9 years later to make additional measurements (10). They found large fields in homes and businesses near various radio towers. Another hotspot was reported near an antenna farm on Cougar Mountain outside Seattle, Washington, where readings up to 700 $\mu W/cm^2$ were recorded in areas accessible to the public (11). Healy heights near Portland, Oregon exhibited levels in excess of 100 $\mu W/cm^2$ in public areas and in private homes (12). In Denver, Colorado, measurements in a public area near the antenna farm that services Denver showed levels as high as 1000 $\mu W/cm^2$. Most indoor levels were less than 50 $\mu W/cm^2$, but some were as high as 580 $\mu W/cm^2$ (13).

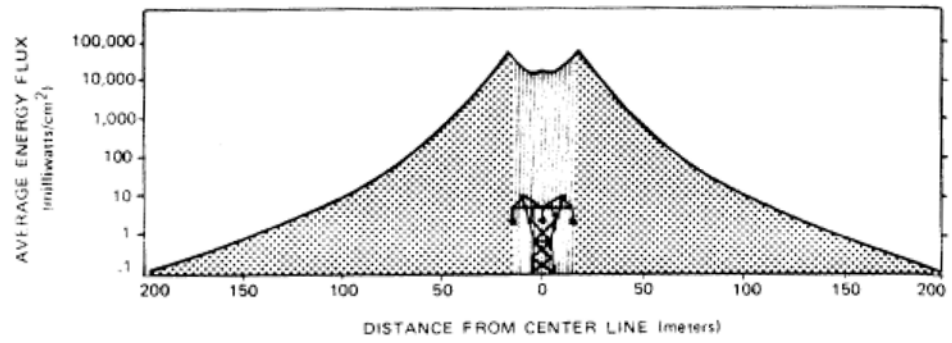

Figure 1. Calculated average ground-level energy flux of a typical 765-kV powerline as a function of lateral distance from the center-line (18).

Because of stray radiation from radar, exposure levels near airports and military bases can be in the range of 10–100 $\mu W/cm^2$ at distances up to 0.5 miles. The Pave Paws 420–450 MHz radar on Cape Cod (1 of 4 such installations in the USA) produces 0.03–3700 $\mu W/cm^2$, depending on location (14).

Microwave-relay antennas are used for long-distance telephone service and for private communications. A 10-ft diameter antenna positioned 100 ft above the ground produces ground level EMFs of approximately 0.03–7.5 $\mu W/cm^2$ within 376 ft of the tower (15). There are several thousand microwave-relay towers in the USA, each with 2 or more antennas.

Mobile communications equipment and hand-held walkie-talkies are relatively low- power sources, but they account for significant exposure levels because the radiating antenna is ordinarily close to the user. A walkie-talkie operating at 165 MHz, with an output of 1.8 watts results in 144–12,000 $\mu W/cm^2$ in the vicinity of the head of the user (16).

Starting and stopping of trains in the Bay Area Rapid Transit System in California produces low-frequency EMFs throughout the entire San Francisco Bay area (17).

Powerlines transport electrical energy, and they are usually built overhead rather than underground. The energy carried by a powerline actually moves through the space that surrounds

the wires (18) (Figure 1), and consists of an electric and magnetic field. Various design considerations such as line geometry, phase spacing, and operating voltage materially affect field strength at various lateral distances (19). Electric fields associated with powerlines having different voltages are shown in Figure 2. Figure 3 shows the spatial distribution of the magnetic field of a 345-kV powerline.

Power-frequency magnetic fields measured over 223 km of roads in Quebec showed that the fields were greater than 400 μGauss more than 90% of the time, greater than1600 μGauss 50% of the time, greater than 5000 μGauss 10% of the time, and greater than 10,000 μgauss 1% of the time (20). The distribution of power-frequency magnetic fields that we measured in a region in the English midlands is shown in figure 4. Readings were taken at the domiciles of suicide victims and at an equal number of appropriately chosen control addresses (21). A total of 1184 addresses were measured, yielding a mean of about 800 μGauss (range, 10–15,000 μGauss).

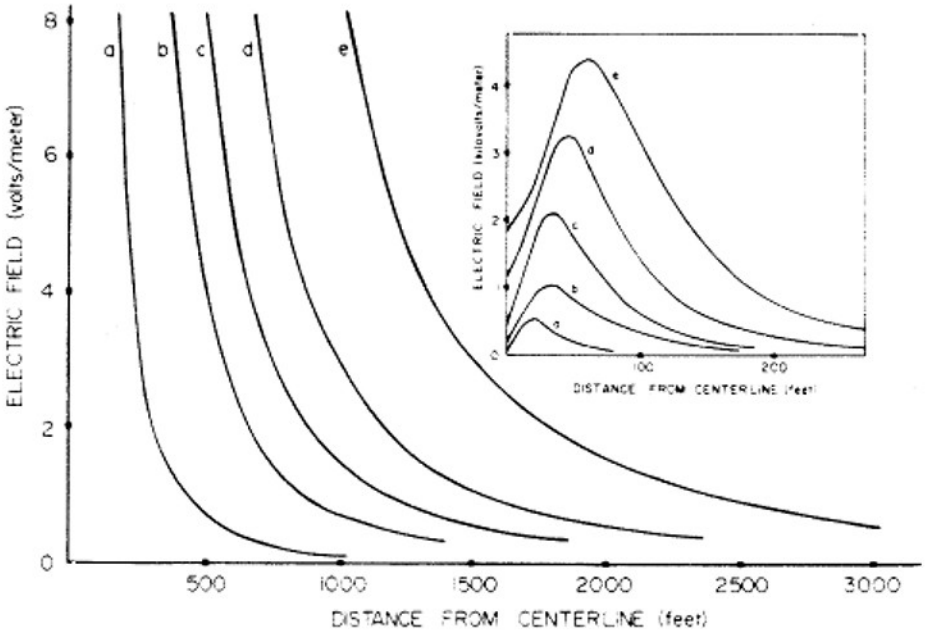

Figure 2. Ground-level electric fields of high-voltage powerlines, calculated by the method of images (18). (a), 115 kV; (b), 230 kV; (c), 345 kV; (d), 500 kV; (e), 765 kV.

An electric field of 2900 V/m was measured from an electric blanket (22). The 60-Hz magnetic field from an electric blanket and from other common electrical devices are shown in Table 1. in addition to line-frequency fields, line-operated devices can produce significant fields at harmonics up to 1 kHz, and beyond (23).

Table 1. Electric Field at 60 Hz Associated with Common Electrical Devices. The measurements were made using a magnetic flux probe (Magnetek Model 1846) calibrated by the use of a uniform magnetic field of known magnitude and direction (224).

	Magnetic Field (mGauss) at Indicated Distance (cm) from Source		
Source	**10**	**20**	**30**
Electric Blanket* (Northern Style 13, 180W)	13.8	6.2	2.8
Electric Razor (Remington Model GMF 1)	580	110	40
Electric Wire 18gauge (5A)			
Parallel conductors	2.8	1	0.5
Separated conductor	100	55	36
Light Bulb (Sylvania)			
60 W (0.42 A)	1.02	0.56	0.25
100 W (0.75 A)	1.50	0.85	0.46
Electric Iron (GE Catalog 16F66)	44	11	3.7
Electric Typewriter (IBM Selectric II)	2.8	1.2	0.56
Electric Motor (Fischer No. 1907501, 1/6 HP)	46	10	3.7
Electric Motor (GE Model 5KC42JG14EX, ½ HP)	355	14	6.3
Fluorescent Light (Westinghouse, 15W (2))	5.2	3.4	2.65
Soldering Iron (Weller Model WTCPN)	185	54	23
Refrigerator (GE Model TBF 21 DW)	4.8	3.6	2.65

*230, 120, 30 mGauss at the surface, 1 cm and 5 m respectively

Human exposures to magnetic fields associated with medical imaging, and some occupational activities, have been summarized by Stuchly (24).

Video display terminals produce environmental electric fields of 10 Hz–200 MHz. An electric field in excess of 300 V/m was measured 20 cm from a terminal (25). The electric field exceeded 5 V/m along more than 120° of arc at a radius of 60 cm from the terminal.

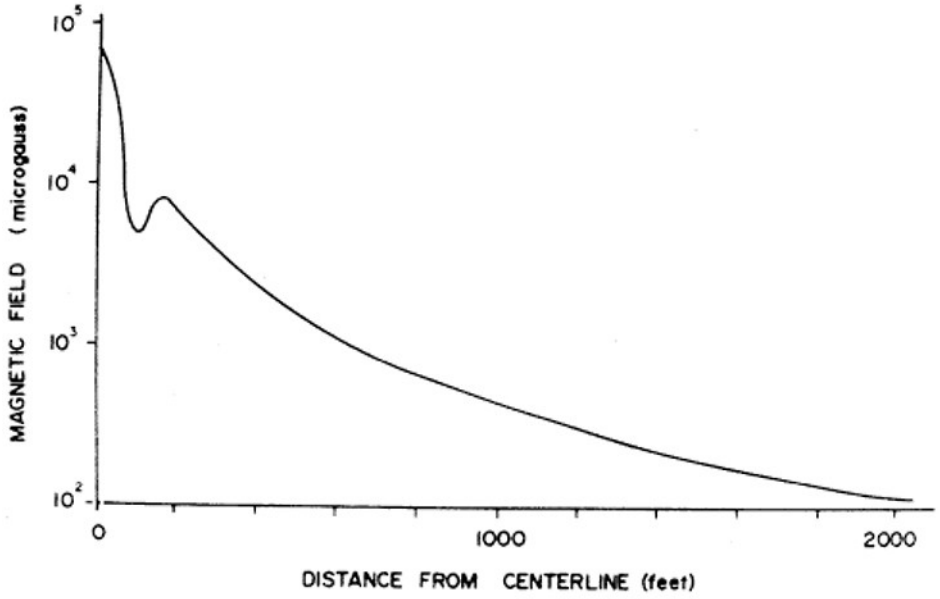

Figure 3. Distance dependence of the magnetic field of a twin-circuit vertical 345-kV powerline, calculated by the method of images (18). Each phase (phase relationship 1, 2, 3:3, 1, 2) consists of two conductors (1.08 in, diameter) spaced 1.5 ft apart. The assumed line and phase spacings are 28.5 and 24 ft, respectively. The height of the lowest phase in each line is 63.1 ft.

Data regarding occupational exposure to EMFs is scanty (26), and that regarding exposure of military servicemen is essentially non-existent.

There are no U.S. federal standards to protect the general population from overexposure to any frequency of nonionizing radiation. The EPA has authority to promulgate high-frequency standards but has not done so. The agency has declared that the sole authority to set standards at low frequencies rests with the several states (none of which has taken any significant action). The Occupational Safety and Health Administration has a 10,000 $\mu W/cm^2$ standard, but the courts have ruled it unenforceable. The Soviet general population exposure standard for radiofrequency and microwave radiation is 10 $\mu W/cm^2$ for 300 MHz–300 GHz, and 3–25 V/m for 30 kHz–300 MHz (27). The standard at low frequencies has been described in the previous Chapter.

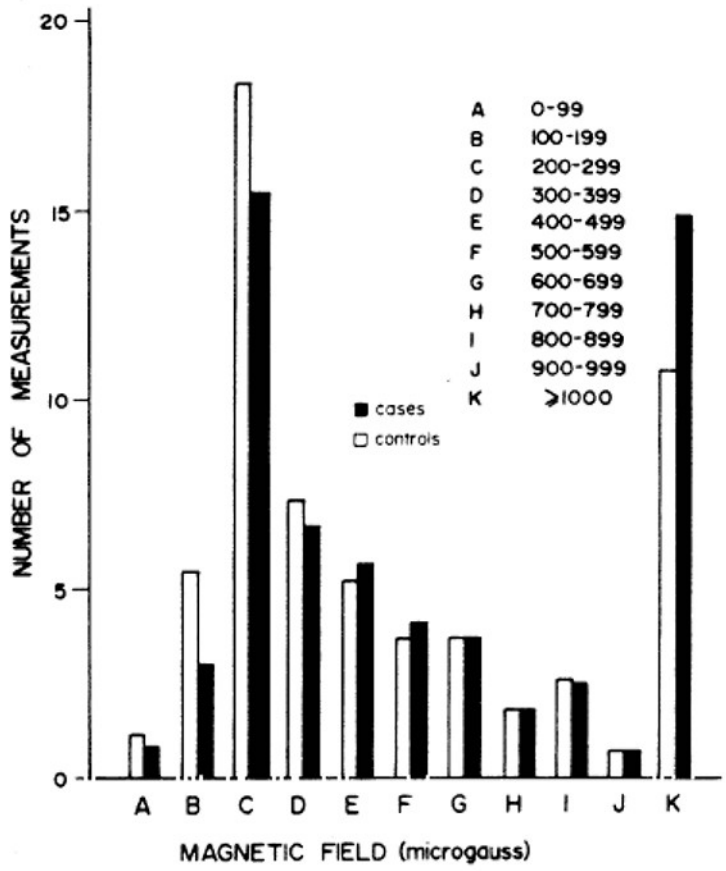

Figure 4. Distribution of Power Frequency Magnetic Fields in a Study District in the English Midlands. The readings were taken with an FDM-100-10-53 meter (Deno) with the search coil mounted 1 meter above the ground, and 0.5 m from the center of the front door of the residence. Data is taken from a case-control study of the correlation between suicide and power-frequency magnetic fields (21). The number of domiciles of suicide cases and controls having a magnetic field within the indicated interval is shown.

LABORATORY STUDIES OF EMF BIOEFFECTS

INTRODUCTION

The threshold public-health question regarding environmental EMFs is whether their presence in human living space is detected by the subjects. The body has a specialized detector, the eye, for receiving EMFs with a narrow frequency range. If human beings have no detector for other environmental EMFs, then the body would remain ignorant of their presence and they could not produce changes in the function of the body — pathological changes or otherwise. There are different ways to approach this pertinent question. One could search for the detector in model systems, or search for biological effects in animal systems (on the certain inference that if there were an EMF-induced bioeffects, the EMF must have been detected). Historically, only the latter approach has been successful, and it is this data that will be reviewed here.

TRANSIENT EFFECTS IN ANIMALS

Some of the earliest studies were performed in the Soviet Union, and it apparently was Soviet scientists who first recognized the compensatory nature of EMF-induced bioeffects (28). Kholodov studied the effects of magnetic-field exposure on the electroencephalogram (EEG) in rabbits, and found alterations in delta waves and spindles (brief bursts of 8–12 Hz waves) due to 1-3 minutes' exposure at 200–1000 Gauss (29). These reactions usually occurred after a latent period of about 10 seconds, and they lasted at least 30 seconds in about half the animals tested. Sometimes, desynchronization (an abrupt change in the main rhythm) occurred 2–10 seconds after the field was turned on (14% of the cases) or off (24%). Similar changes in EEGs due to EMFs at 50 Hz, and 3 GHz have been reported (28, 30).

Lott and McCain measured the total integrated EEG in rats before, during, and after exposure to a DC field of 10 kV/m (31). They found a transient increase associated with either the application or removal of the field, a steady response that persisted during application of the field, and an after-effect. A 640-Hz field, 40 V/m, also increased the total integrated EEG, particularly for readings from the hypothalamic region.

At high frequencies, a different effect on the total integrated electrical activity was observed. Rabbits exposed for 5 minutes to 700-2800 $\mu W/cm^2$, 9.3 GHz exhibited no EEG changes during the exposure period (32). But commencing 10 minutes after exposure, an interval of decreased EEG activity occurred that persisted for up to 15 minutes.

Friedman and Carey measured the corticoid production in monkeys exposed to a 200-Gauss DC magnetic field for 4 hrs/day (33). An increase in urinary corticoids lasting 6 days was observed, followed by a return to baseline despite continued exposure.

Several endocrinological parameters in rats (serum corticoids, pituitary ACTH, and ACTH releasing factor in the hypothalamus) were increased following brief exposure to 10–1000 $\mu W/cm^2$ at 2.4 GHz (34). At 3 GHz, rats exposed to 5–10 $\mu W/cm^2$, 8 hrs/day, had elevated levels of excreted corticoids after 1–3 months of exposure (35). Mice exposed to 25 or 50 kV/m, 60 Hz, exhibited increased corticosterone levels upon application of the field, and the levels returned to baseline despite continued application of the field (36). EMF effects on adrenal tissue *in vitro* have been reported (37).

The adrenal corticoid response to EMF stimulation is time dependent, and it varies nonlinearly with dose (38). When groups of rats were exposed to 500, 1000, 2000, and 5000 V/m at 50 Hz, the average urine corticoid level of the latter two groups changed similarly during the 4-month exposure period: approximately the same maximum value was achieved in both groups and they exhibited increased corticoid levels as compared to the controls. The 1000-V/m group, however, exhibited lower corticoid levels for the first 2 months of the exposure period followed by a rise above the control level during the last half of the exposure period; at 500 V/m the pattern of corticoid excretion was identical to that of the controls. The biological response was

reversible in the sense that when the field was removed, the corticoid level returned to normal within 2 months.

At 3 GHz, 153 μW/cm^2, an increase in thyroid weight was found after 2 weeks' exposure, but after 5 months' exposure the weights were normal (39). Ossenkopp et al. Found that both male and female rats exposed *in utero* to 0.5 Hz, 0.5–30 Gauss had increased thyroid weights at 105–130 days of age (40).

Fischer et al. Exposed rats to 50 and 5300 V/m, 50 Hz, and observed bradycardia at both field strengths beginning 15 minutes after commencement of exposure (41). At the lower field strength an 8% decrease occurred, and it remained statistically significant after 2, 10, 21 and 50 days of continuous exposure. At 5300 V/m the decrease in heart- rate after 15 minutes' exposure was about 16%: it was not seen following 2, 10, or 21 days' exposure, but it was present (about 5%) after 50 days. Bradycardia was also reported in rabbits following exposure to 50 Hz, 1000 V/m (42); the heart-rate decreased by about 9% after 30–60 days. Microwave EMFs also produced bradycardia (43); the effect was seen after 2 weeks' but not after 2 months' exposure.

Table 2. Percent Change in Hematological parameters in Mice (45). RBC, red blood cell concentration; Hct, hematocrit; Hb, hemoglobin; MCV, mean cell volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration. A, no change in exposure conditions; B, change in exposure condition as indicated. NM, not measured.

		Percentage Change					
Experiment	**Condition**	**RBC**	**Hct**	**Hb**	**MCV**	**MCH**	**MCHC**
A							
Male Control	nF ➤➤ nF	1.7	2.0	-4.5*	1.0	-6.2*	-6.3*
Female Control	nF ➤➤ nF	3.9*	4.1*	1.7	0.2	-5.0*	-5.1*
B							
Male I	F ➤➤ nF	-4.7*	-5.1	NM	0	NM	NM
	nF ➤➤ F	-5.2*	-4.9	NM	0.2	NM	NM
Male II	F nF	-9.0*	-9.1*	-3.3*	-0.4	5.7*	6.0*
	nF F	-6.5*	-7.0*	-2.4	-0.7	3.9*	6.1*
Female I	F ➤➤ nF	-4.1*	-4.6*	-4.2*	-1.2	0.5	1.2
	nF ➤➤ F	-6.4*	-6.7*	-3.4*	-0.5	3.8	4.8
Female II	F ➤➤ nF	-5.3*	-6.0*	3.4	-1.2*	8.3*	10.0*
	nF ➤➤ F	-7.1*	-9.2*	3.5	-2.3*	11.0*	13.6*

*$P < 0.05$

In preliminary studies, dogs were exposed to 15 kV/m, 60 Hz, for 5 hours to determine whether such exposure altered the physiological response to a controlled hemorrhage (10 ml/kg, over a 3-minute period) (44). The cardiovascular changes at the end of the hemorrhage were: mean arterial pressure fell an average of 5.9 mmHg in the control group and 16 mmHg in the exposed group; arterial pulse pressure fell 0.9 mmHg in the control group and 10.9 mmHg in the exposed group; average heart-rate decreased 9.3 beats per minute in the control group, but increased 57.5 beats per minute in the exposed group. The data was rejected by the sponsor (1).

To study transient responses to EMFs, we looked for changes in hematological parameters of mice due to a 60 Hz electric field of 5 kV/m (45). There were four consecutive experiments, two with males and two with females. In each there were two groups: one for which the control period preceded the exposure period (nF ➤➤ F), and one in which the pattern was reversed (nF ➤➤ F). On "day 1" of each experiment the mice were divided into the two groups and the electric field was applied to one-half the population. On "day 3" the blood parameters were measured in each mouse and immediately thereafter the exposed and non-exposed groups were interchanged. On "day 5" the blood parameters were measured again and the mice were killed.

Blood was collected from ophthalmic vessels and it was therefore necessary, before applying the field, to determine the influence of the first blood collection procedure on the values measured after the second such procedure. We measured the blood parameters in the two groups of mice, one male and one female, under conditions that were identical in all respects to those employed during the field-exposure portion of the study, and we found that the method of blood collection had a tendency to produce higher RBC, Hct, and MCV values and lower values of Hb, MCH, and MCHC (Table 2).

The results obtained in connection with the application of the electric field are shown in Table 2. In each experiment, RBC on "day 5" was significantly less than on "day 3," regardless of whether the interval between "day 3" and "day 5" was an exposure period or a non-exposure period. A decline in Hct paralleled the RBC changes, but Hb showed no consistent changes. MCV showed a tendency to decrease, but the other computed indices both increased.

The trends in the computed indices, and especially the changes in RBC and Hct, were opposite to those induced by our method of blood collection alone. It follows, therefore, that the applied electric field had a physiological impact. The unique feature of the observed responses is that, for each parameter, a change in the same direction occurred with both the F ➤➤ nF and nF ➤➤ F groups. An analysis of variance confirmed that in all four experiments there was an effect associated with time but not with the order of field application. This indicated that the animals responded to the change in their electrical environment, not to the electric field itself.

Szmigielski et al. (46) studied the action of an EMF on the granulopoietic reaction in rabbits that had been subjected to an acute staphylococcal infection. Rabbits were exposed to 3000 $\mu W/cm^2$, 3 GHz, 6 hrs/day, for 6 or 12 weeks, and then were infected intravenously with bacteria. Four to six days after infection in the 6-weeks exposed animals displayed stronger granulocytosis than did the control animals, but this was reversed by the end of the observation

period. These changes were accompanied by a consistent reduction in the bone-marrow reserve pool, and a depressed lysozyme activity. Animals exposed for 3 months displayed consistently depressed granulocytosis after the infection, and both the bone-marrow reserve pool and the blood serum lysozyme activity were lowered during the entire postinfection period. The results were interpreted to mean that the EMF-exposed animals lacked the reserve capacity to adapt to the infection as efficiently as the control animals: fewer granulocytes could be mobilized, and there was a resulting decline in lysozyme activity.

Shandala and Vinogradov studied the effect of an EMF (1–500 μW/cm^2, 2.4 GHz, for 30 days) on the phagocytic action of neutrophils in peripheral blood of guinea pigs (47). They found that the percent of killed microbes increased following exposure to 1– 10 μW/cm^2, and decreased at 50 and 500 μW/cm^2; the most pronounced effects occurred at 1 μW/cm^2. EMF-induced alterations in the complement titer in blood serum were also found. Both immunological indicators returned to normal within two months of the cessation of irradiation.

Shandala et al. Reported a significant disturbance in the immunological system of rats exposed intermittently to 500 μW/cm^2 for 30 days (48); blast cells in peripheral blood, and rosette-forming cells in the spleen and thymus were both affected following EMF exposure.

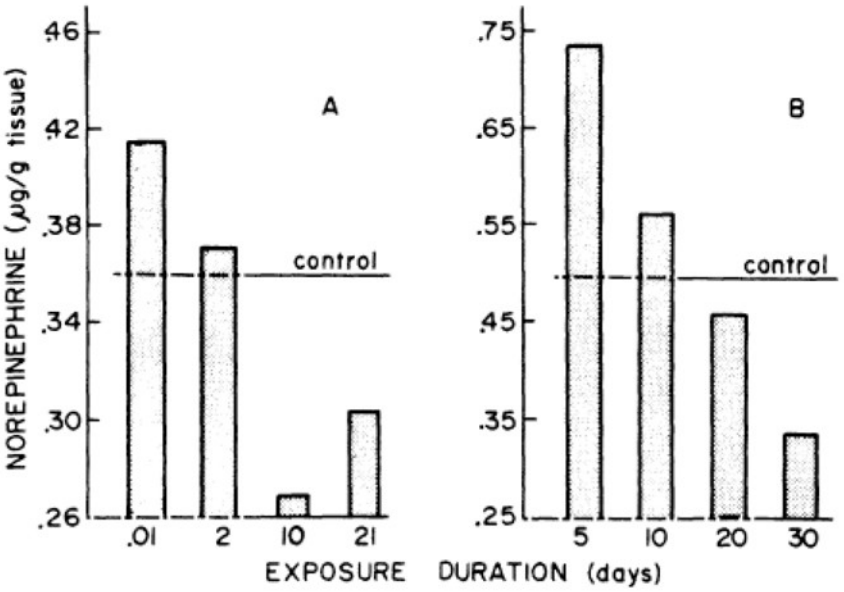

Figure 5. Norepinephrine levels in rat brain following exposure to EMFs. A, 5300 V/m, 50 Hz (54); B, 500 μw/cm2, 2.4 GHz (55).

The threshold for changes in mitochondrial enzymes in murine brain tissue was 100–500 μW/cm^2 at 3 GHz (49). EMF-drug interactions have been reported (50,51). Merely shielding animals from ambient fields can produce biological changes (52,53).

Fischer et al. Found that 50 Hz, 5300 V/m, resulted in an initial rise of norepinephrine in rat brain, and a subsequent decline above the control level (54); Grin observed the same sequence of changes at 2.4 GHz, 500 $\mu W/cm^2$ (55) (Figure 5).

It is apparent from the reported data that all types of EMFs can elicit biological responses. Generally, the responses are transient and the measured parameters return to baseline despite continued application of the EMF.

HUMAN EXPERIMENTS

Soviet scientists had concluded in the 1930's that EMFs altered the human nervous system (56), but human studies were apparently not performed elsewhere until several decades later. In an experimental design in which each subject was exposed to two frequencies in the 2–12 Hz range, at 4 V/m, Hamer found a longer reaction time at the higher frequency (57). Friedman et al. Applied magnetic fields of 0.1 and 0.2 Hz to separate groups of male and female subjects, and for both groups found a longer reaction time at the higher frequency compared to the lower frequency (58). Persinger et al. found no difference in the mean reaction time in either males or females due to 0.3–30 v/m, 3–10 Hz, but he reported a significant difference between the sexes in response variability (59). A 60 Hz, 1 Gauss field altered the ability to concentrate (60). All 6 experimental subjects demonstrated a decline in performance (addition of two- digit numbers) in the second test session of the exposure period, and all 6 improved in the first test session of the post-exposure period (60). In contrast, the control subjects showed no consistent changes.

Dumanskiy et al. reported an increase in blood glucose in humans following exposure to 15 kV/m, 50 Hz, 1.5 hrs/day for 6 days (61).

Volunteers confined for up to 7 days were exposed to a 1-Gauss magnetic field, 45 Hz, for 24 hours: they did not know which 24-hour period during their confinement would be chosen for the application of the EMF. Serum triglycerides in 9 of 10 exposed subjects reached a maximum value 1–2 days after EMF exposure; similar trends were not seen in any of the control subjects (62).

A 5 Hz magnetic field (about 10 Gauss) altered the ability of subjects to process and remember verbally delivered information (63).

Wever studied circadian rhythms using an elaborately constructed underground bunker which provided complete isolation from all environmental time cues (64–67). One of the two suites in the bunker was shielded against all electric and magnetic fields of terrestrial or atmospheric origin. In addition, the room contained built in facilities for introducing a range of artificial fields. In the second suite, the Earth's normal fields were continuously present. The rooms were built in such a way that the subjects could not distinguish between them. Subjects were isolated in the bunker for 3–8 weeks, and various circadian rhythms including activity and body temperature were recorded. Wever found that 34 subjects who lived in the non-shielded room had a body temperature rhythm with a mean period of 24.87 ± 0.44 hrs, while 50 subjects

who lived in the shielded room had a body temperature rhythm of 25.26 ± 0.85 hrs ($P < 0.01$). In 15 subjects who lived in the shielded room, internal desynchronization occurred. That is, while the body temperature rhythm continued to maintain a circadian rhythm near 25 hours, the period of the activity changed, either lengthening or shortening. Thus, the normal synchronization between the rhythms was destroyed. Internal desynchronization was not observed in the non-shielded room in which the natural fields of the Earth were present.

In another study, an artificial electric field (10 Hz, 2.5 V/m) was switched on and off in a changing temporal sequence. No subject knew when the field was present, and each subject acted as his own control. Wever found that the presence of the EMF reversed the effects found previously. That is, within the field present, the 10 subjects showed lower values of the period of the body temperature rhythm, and in no case did internal desynchronization occur. Moreover, when the field was switched on with the subject in a state of internal desynchronization, the desynchronization was stopped. Wever concluded that the artificial electric field on one hand, and the total of the normal EMFs on the other, had similar influences on circadian rhythms.

Workers in Italian state electric-train substations are exposed to maximum electric and magnetic fields at 50 Hz of 5 kV/m and 150 mG, respectively (68). Substation workers were divided into 4 groups depending on their average weekly exposure to the maximum electric field, namely 0 (controls), 1, 10 and 20 hours/week. The employees in each group were subjected to a clinical examination and laboratory tests. The studies showed a correlation with duration of exposure to the maximum electric field: compared to the controls, the clinical values for most parameters in the 1 and 10 hr/week groups were elevated (or depressed), whereas the values in the 20 hr/week group were normal.

Table 3. Changes in Average Body Weights of Rats Exposed to 45-Hz Vertical Electric Fields (70). The control rats were housed in a field-free environment.

Experiment	Field (V/m)	No. of Rats	Exposure Time (days)	Weight Gain (gm)
1	25–100	143	36	142 ± 14*
	Control	47	36	209 ± 20
2	10–50	47	40	150 ± 19*
	Control	16	40	215 ± 11
3	2–10	94	32	131 ± 12*
	Control	32	32	166 ± 12
4	0.5–2	32	30	131 ± 11*
	Control	32	30	170 ± 11

*$P < 0.001$

In the USA, the rules applicable to human experimentation have become strict, and experiments involving voluntary human exposure to EMFs are presently not being performed.

ADVERSE EFFECTS

Altered spermatogenesis occurred in rats following exposure to 5000 V/m, 50 Hz, for up to 4.5 months (69). After 1.5 months' exposure, the number of atypical sperm cells was significantly greater in the exposed animals (30.7% vs. 15.9%, $P < 0.01$); the percentage of pathological forms increased with the duration of exposure and reached 36.8% after 4.5 months. The exposed rats also produced fewer sperm cells and exhibited a higher ratio of living to dead cells; both effects became significant after 3.5 months.

In a Navy study, exposure to 0.5–100 V/m, 45 Hz consistently produced depression of the body weights of the exposed animals (70) (Table 3). The results of the study were disavowed by the sponsor (but not the investigators), apparently because they control rats had been maintained under Faraday-cage conditions. Low-frequency electric and magnetic fields also produced growth depression in 25-day-old chicks (71).

We performed midshaft fractures on rats, following which half the group was exposed to 5 kV/m, 60 Hz, and half was maintained as a control (72). The extent of bone healing was evaluated at 14 days postfracture on the basis of blind scoring of serial microscopic sections. In two replicate studies, we found a highly significant retardation in healing; the fractures in the exposed rats exhibited the development normally seen in a 10-day fracture. We found no effect on fracture-healing following exposure at 1 kV/m. the adverse effect of a 60-Hz electric field on fracture healing in the rat was confirmed in three replicate studies (73) (the paper must be read carefully before the point is appreciated).

Grissett et al. exposed 30 monkeys to 20 V/m and 2 Gauss at 76 Hz (74). After 1 year, the field-exposed males were significantly heavier than the control males.

When chickens were irradiated for more than 200 days at 0.19–360 $\mu W/cm^2$ (7 GHz), the irradiated animals exhibited a doubled mortality rate, and a deterioration in health (75).

Hansson described histopathological changes in the brains of rabbits that had been exposed to 50-Hz electric fields, 15 kV/m (76). The observed changes included the formation of numerous lamellar bodies in the endoplasmic reticulum in Purkinje cells. Hansson suggested to me that the experiment be repeated using a different animal species. I exposed mice to 9.8 kV/m for 60 days, and Hansson subjected the brains of the exposed and control animals to the same kind of analysis previously performed for the rabbits. The same histopathological changes previously seen in rabbit brain were observed in the brain tissue from the exposed mice (77).

Investigators exposed pregnant rats to 100 μW/cm2 at 27.1 MHz for up to 20 days (78). The frequency was chosen because of its widespread industrial use in radiofrequency heaters and heat sealers. One group was exposed for 20 days, and other groups were exposed for 5 and 10 days. The rats exposed for the longest time gained weight the slowest. The rats in the exposed group experienced significantly greater fetal resorption rates. Among their viable offspring, there

was a significantly higher incidence of delayed development (higher incidence of incomplete cranial ossification) (78).

IN VITRO STUDIES

In vitro studies have demonstrated EMF-induced changes in growth rate (79,80), respiration (81), metabolism (82, 83), membrane receptors (84, 85), immune response (86, 87), and morphology (88), but the actual mechanism of detection of EMFs has not yet been discovered.

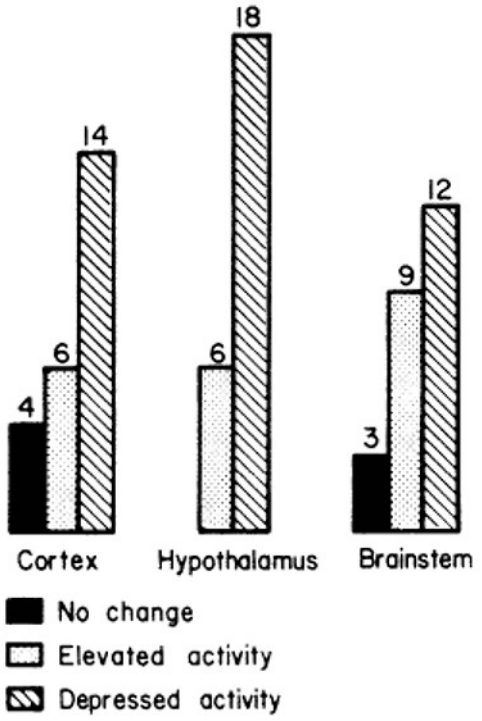

Figure 6. EEG response to 100 μW/cm^2, 3 GHz (95). The numbers indicate rabbits with a given response.

Beyond the phenomenon of detection, the significance of *in vitro* studies with respect to intact organism remains dubious. The point is illustrated by the work of Adey and his colleagues. It was found that EMFs altered behavior in humans and monkeys and cats (57, 89, 90). The idea evolved that EMFs could alter neuronal excitability, if they were in the frequency range of the EEG. An *in vitro* system involving calcium-binding to brain tissue was designed under the belief that the process was important. A complex series of results were then obtained concerning the levels of pre-incubated calcium that were released into solution from live or dead brain tissue (91–93): at 147 MHz, there was an increase when the EMF was modulated at 6–10 Hz, but no increase at 0.5–3 or 25–35 Hz; with EMFs of 6 and 16 Hz, there was a decrease at 10 and 56 V/m, but not at 5 or 100 V/m; there was no change at 1 Hz or 32 Hz, at either 10 or 56 V/m; at

450 MHz, modulated at 16 Hz, there was an increase. Nothing corresponding to these unusual features has been seen in *in vivo* studies.

Table 4. Biological Effects of Exposure to a 15-kV/m 60-Hz Electric Field (97). The average values were determined following 30 days of continuous exposure. There were no statistically significant changes in water consumption during the first 14 days of any experiment.

Experiment	Number of Rats	Average Water Consumed (ml)	Pituitary Weight (µg/g)	Serum Corticoids (µg/100 ml)
1	15 experimental	846 ± 68*	38.7 ± 3.2*	6.8 ± 0.8*
	18 control	940 ± 142	35.2 ± 3.8	8.7 ± 1.2
2	14 experimental	749 ± 80*	43.9 ± 4.1*	7.2 ± 1.5
	20 control	891 ± 93	40.6 ± 3.1	7.6 ± 2.1
3	19 experimental	819 ± 83*	32.9 ± 3.1	
	21 control	890 ± 83	35.2 ± 2.6	
4	16 experimental	901 ± 50*	38.0 ± 2.4	6.0 ± 0.7
	14 control	1054 ± 84	39.0 ± 2.6	6.4 ± 0.6
5	20 experimental	1003 ± 82*	31.4 ± 2.4*	9.1 ± 2.0*
	20 control	1099 ± 117	29.4 ± 2.9	16.3 ± 3.8
6	14 experimental	1143 ± 157	31.2 ± 1.8	9.5 ± 2.0
	16 control	1202 ± 107	30.6 ± 1.8	9.7 ± 4.0

*$P < 0.05$

ROLE OF ENVIRONMENTAL FACTORS

Twenty-four rabbits were exposed to 100 µW/cm^2, 3 GHz, for 30 minutes, after which their EEG slow-wave activity in different parts of the brain was measured (94). The data (Figure 6) indicated that the rabbits responded differently from one another. If the results of the study had been presented as the average behavior of an experimental compared to control group, it would have been concluded that the fields produced no EEG changes.

Freeman and Carey exposed rabbits to 11–210 Gauss DC and 5–11 Gauss at 0.1–0.2 Hz for up to 60 hrs (95). Four of the 12 exposed rabbits exhibited some histopathological changes which were attributed to exacerbation of a sub-clinical encephalitozoonosis by a stressor effect of the magnetic field (95). Histopathological changes were also observed in the adrenal cortex of 70% of mice chronically exposed to strong static magnetic fields (96). The changes were attributed to the direct stressor effect of the field — akin to Selye's diseases of adaption (109).

We exposed individually caged rats to 15 kV/m, 60 Hz, for 30 days and measured pituitary weight, serum corticoids, and water consumed (Table 4) (97). In the first replicate, each of the measured indices was significantly different in the treated group. In the sixth replicate none of the indices differed between the groups, and in replicates 2–5 various combinations of the indices differed between the groups. Numerous sponsors in the USA commissioned attempts to (loosely speaking) replicate this humble study, thereby generating fodder for ten years of argument (1, 106, 233).

Table 5. Average Glucose Levels in Three Replicate Experiments. (A), reported data analysis (99); (B), analysis of actual data (100) taking into account the 60-Hz background fields (101).

		Serum Glucose Levels (mg/dl)					
Experiment		**Control**	**2 V/m**	**10 V/m**	**20 V/m**	**50 V/m**	**100 V/m**
(A)	1	281.1 ± 83.8	176.3 ± 74.4	259.4 ± 124.6	218.9 ± 100	286.0 ± 156.9	235.8 ± 49.3
	2	210.4 ± 55.4	237.3 ± 62.2	259.4 ± 95.9	241.6 ± 149.1	256.2 ± 118.6	269.4 ± 95.9
	3	187.2 ± 34.4	199.0 ± 30.8	199.1 ± 34.3	201.3 ± 40.9	199.1 ± 34.2	232.3 ± 44.0

		Serum Glucose Levels (mg/dl)		Statistical Significance
		Control +2 V/m	**50 V/m +100 V/m**	
(B)	1	228.7 ± 94.4	260.9 ± 117.2	$P = 0.23$
	2	223.9 ± 59.5	284.2 ± 116.0	$P < 0.02$
	3	193.7 ± 32.7	219.7 ± 42.4	$P < 0.01$

Guinea pigs were exposed to 3 GHz, 10 min/day, for 30 days (98), and both the irradiated and the sham-exposed animals were sampled before and after each daily exposure bout. The sham-exposed group revealed no significant changes, but animals exposed to 25 or 50 $\mu W/cm^2$ exhibited EMF-induced alterations with time dependencies that differed with each animal. For a given exposure duration, the WBC was above the normal level in some animals, and below it in others; as a result, the average values varied little during the study. At 500 $\mu W/cm^2$, however, even on the average there was a pronounced leucopenia and lymphocytosis.

In an Army study, rats were exposed for 28 days to 2, 10, 20, 50, and 100 V/m, 45 Hz, in three replicate experiments, following which complete blood chemistries were performed (99); the serum glucose levels are listed in Table 5A. although some differences between the control

and exposed groups were seen, no linear dose-effect relationship was manifested, and consequently the authors regarded the data as having failed to show a biological effect of the EMF (99). But the 60-Hz electric field in the test cages was 0.18–9.15 V/m (100). In my view, the 2-V/m group should have therefore been considered a control group in relation to the 50–100 V/m exposed groups. When we did this (101), the data revealed increases in serum glucose in each replicate (Table 5B). (This approach to the Army data also suggests the existence of effects on other parameters, including triglycerides.)

By the mid-1970's, no studies had been done to assess the possible impact on successive generations of animals of the continuous presence of a low-frequency EMF; we therefore undertook such a study (102). Initially, mature male and female mice were split into horizontal, vertical, and control groups. Mice in the horizontal group were allowed to mate, gestate, deliver, and rear their offspring in a horizontal 60-Hz electric field of 10 kV/m. At maturity, randomly selected individuals from the first generation were similarly allowed to mate and rear their offspring while being continuously exposed. Randomly selected individuals from the second generation were then mated to produce the third and final generation. A parallel procedure was followed for the vertical group wherein three generations were produced in a 60-Hz vertical electric field of 15 kV/m, and for the control group wherein three generations were produced in the ambient laboratory electric field. In the first and second generations, males and females reared in both the horizontal and vertical electric field were significantly smaller than the controls when weighed at 35 days after birth. In the third generation, the only group whose body weights were significantly affected were the males exposed to the vertical field. In both the second and third generation, a large mortality rate in the vertical-field mice was seen during the 8–35 day postpartum period.

We repeated the multi-generation study at 3.5 kV/m, using an improved exposure system (103). In the first generation, no consistent effect on body weight attributable to the EMF was seen throughout a 63-day observation period. In both the vertical and horizontal groups, however, mortality among newborns was increased. In the vertical-control group 48 animals (about 17%) died between birth and weaning. In the vertical-exposed group, if the electric field was not a causative factor, a 17% mortality rate should also have been seen. However, that group exhibited a 31% mortality — 82 animals died and not the expected 44. thus, 38 animals, about 16% of those born, failed to live to weaning because of the electric field. A similar result was obtained in the horizontal-exposed group — about 11% of the animals born failed to live to weaning because of the electric field.

In the second generation, no pattern regarding body weight attributable to the EMF was seen throughout a 108-day observation period. The vertical-exposed, group, however, again exhibited a higher mortality; about 6% of the animals alive at weaning failed to live to the final day of observation due to the presence of the EMF. In the third generation, the exposed animals had higher body weights, particularly in the horizontal-exposed group. At 49 days after birth, the males and females in each exposed group were significantly heavier than their respective

controls. At 119 days after birth, only the females in the horizontal-exposed group were significantly heavier, but this was part of a consistent trend for that group. Again we saw an increased mortality in the vertical-exposed group — 10% of the weaned animals failed to survive to the end because of the electric field.

Following the publication of our first multi-generation study (102), investigators at Battelle Northwest laboratories, Richland, Washington were commissioned to replicate the work. They first developed an exposure system that was unexcelled with regard to field homogeneity and reproducibility of electrical environment. Every aspect of the animals' physical environment — light, temperature, humidity, presence of pathogens in the air, air flow, for example — was rigorously monitored and controlled by automatic equipment. The investigators then constructed two complete exposure facilities: each consisted of a completely characterized exposure unit, an identical unit for sham-irradiation, and a completely controlled environment suitable for housing both units.

The multi-generation study was begun in the first exposure facility, and 3 weeks later a replicate study was begun in the second facility; both replicates were done double blind. At the end of the study, the males and females in the first replicate were statistically significantly smaller than the controls, but in the second replicate they were significantly larger (104). The data appears in the report to the sponsor (not in the literature), and the reader must perform his own statistical analysis to support this conclusion.

ANALYSIS

The reports can be summarized this way (166):

1) Exposure to EMFs can result in alterations in all body systems, including the nervous, endocrine, cardiovascular, hematological, immune, and reproductive systems;

2) The effects manifested in each tissue or system are largely independent of the type of electromagnetic field in the sense that common physiological responses are produced by spectrally different electromagnetic fields;

3) An organism's response to an electromagnetic field is determined by a combination of factors including its physiological history, genetic predisposition, and the totality of prevailing environmental conditions;

4) EMF-induced biological effects in animals are best characterized as adaptive or compensatory because the fields present the organism with an environmental factor to which it must accommodate. Linear dose-response relationships are generally not observed.

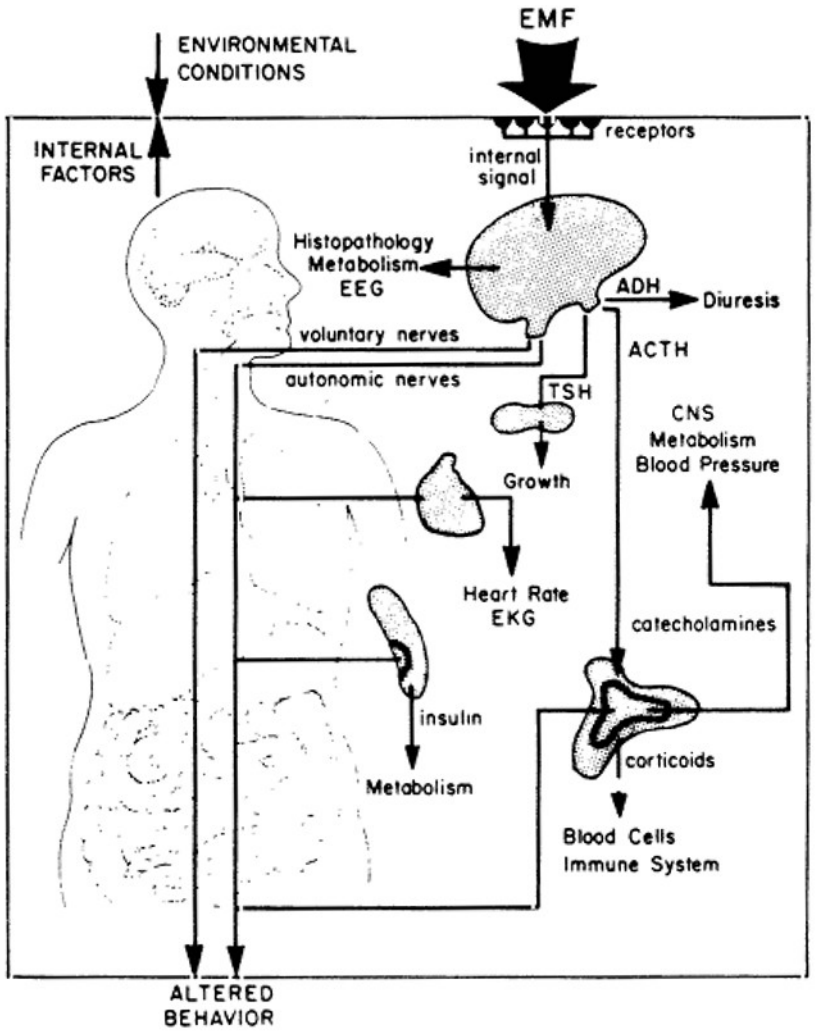

Figure 7. Hypothesis regarding the sequence of physiological events underlying the chronic stressor effect of environmental EMFs.

My hypothesis for the mechanism underlying EMF-induced bioeffects is depicted in Figure 7. the EMF is detected by the peripheral or central nervous system, and the fact of its presence is communicated to the parts of the CNS that initiate and control compensatory responses. The nervous system, both directly and through its rich connections with the endocrine and immune systems modulates the dynamics of the body's various organs and systems in a manner best suited to protect the integrity of the organism. It is the latter transient changes that are detected in most experiments. Their magnitude and direction are simultaneously influenced by other factors in the organ's environment that also impact on the body's compensatory apparatus. Finally, although it is easy to overlook the point, every organism differs from all other organisms of its kind (even though they may look similar) in ways that can affect the outcome of an experiment.

Perhaps the most important lesson of the literature is the guidance that it provides regarding how future studies should be designed. Most studies have been in the nature of bioassays

intended to determine the broad parameters of EMF bioeffects. Replicate studies have generally failed to confirm the initial observations in the sense that the specific changes reported in the initial studies were not observed in the replicates. The Battelle attempt (104) to replicate our multi-generation studies (102, 103) is a good example. Putative replicate studies are frequently used in argument to deprecate the reliability of the initial reports (105, 106). Scientists who come to the EMF bioeffects literature for the first time are consequently presented with a confused picture which perhaps suggests that the area is not worthy of effort. This is not the case, and I hope that the reader will consider my explanation for the essential cause of the apparent confusion.

The precise numerical values that we observed (Table 4) cannot be exactly duplicated because the environmental factors present in our laboratory in Syracuse, New York in 1976 had a significant impact on the specific observations, and those conditions cannot be precisely duplicated (because they were not all catalogued). Similar comments can be made about the work of most of the investigators who first reported EMF bioeffects. If the conditions of an effect cannot be specifically duplicated, the effect cannot be specifically duplicated.

One must recognize exactly what was concluded in most of the original studies. On the basis of the data in Table 4, for example, we concluded that the presence of an electric field was correlated with a change in the average physiological status of the test groups, and therefore that the electric field was detected by the animals. We made no assertion that other investigators would see exactly the same magnitude or direction of change in specific physiological variables. At the time most bioassay studies were performed, the question under consideration was whether any effect occurred, and no investigator then (or now (104)) had the facilities to control for every possible factor that could affect the magnitude or direction of an effect: Again, the question was *is* there an effect.

Sponsors have not looked beyond the numbers in the Tables, and seen what actually was reported. Having failed to recognize what properly was a candidate for replication, it is not surprising that they failed to recognize that the data from replicate studies was actually consistent with the results of the original work. The assertion of inconsistency or conflict (106, 233) is a chimera.

The first decade of sustained laboratory studies of EMF-induced bioeffects is now behind us, and there is no need to repeat the original studies. It is clear to all reasonable investigators that EMFs can affect physiology. What remains is to frame laboratory experiments that have a specific purpose. We have had our pioneers — now, we need settlers.

Irrespective of the hypothesis depicted in Figure 7, actual laboratory data from numerous experiments shows that simulated environmental EMFs produce biological effects, particularly compensatory reactions. If the data is accepted, we must consider the public-health significance of such reactions.

Even if a reaction to environmental EMFs occurs transiently and then evaporates and can no

longer be measured using traditional endpoints, there is a rational basis for expecting EMF-linked pathological changes to occur in exposed subjects. This basis will now be considered.

ENVIRONMENTAL FACTORS AND DISEASE

INTRODUCTION

Many of the physical properties of the fluid matrix of the mammalian body remain remarkably constant despite wide variations in characteristics of the organism's environment. This stable state was termed homeostasis by Cannon (107) who was a student of the physiological mechanisms by which the stability of "this personal, individual sack of salty water, in which each of us lives and moves and has his being" was maintained. His idea was that environmental factors such as temperature or oxygen level constituted a stress on the organism that produced an internal strain which could be pictured as a deviation from a normal level. The CNS and the adrenal gland then act in concert to negate the strain and restore homeostasis. Thus, in an individual undergoing a heat stress, the pores open to facilitate heat loss, whereas a heat-producing response (shivering) occurs in an individual undergoing a cold stress. This use of physical terminology and concepts in a physiological context was an effective descriptive device, even though there existed no means by which to actually measure physiological strain.

In 1936, Selye reported a syndrome in rats produced following exposure to acute agents including cold, surgical injury, muscular exercise, and various injected substances (108). The syndrome consisted of an enlarged adrenal cortex, shrunken thymus, spleen, and lymph nodes, and the appearance of stomach ulcers. He subsequently found that many other acute stimuli produced the syndrome, that it occurred in many species including man, that there existed many other biochemical and physiological indices of the syndrome, and that it was mediated primarily by the anterior pituitary and the adrenal cortex (109). Selye described the syndrome as being a normal physiological response when it was initiated by tolerable levels of the stimuli, but as a pathological process when either the stimulus intensity was too high, or when the organism itself was unable to exhibit a normal response. In these latter cases, diseases of adaption could develop (hypertension, nephrosclerosis, periarteritis nodosa, for example). Selye originally employed the term "stress" as a synonym for the external stimuli or noxious agents, or injected substances that were observed to elicit the syndrome (109,110). Subsequently, he employed the term "stressor" for the external factor, and defined stress in terms of the response of the organism (111). Considered as an internal state variable of the organism, Selye, like Cannon, could provide no means to measure stress, nor even to define it uniquely.

The concept of stress as a special variable — a variable distinct from quantifiable parameters such as temperature, enzyme concentration, and so forth — also arose in the context of clinical medicine. Merely from taking a good history, the clinician could observe that various factors in a patient's life had a predisposing influence on the course of the patient's disease. Death of a loved one, loss of a job, an unhappy marital situation, poor diet, or chronic exposure

to various physical or chemical factors could exacerbate the clinical course of patients with ulcers, high blood pressure, asthma, and many other diseases. The clinical observation was that the so-called stresses and strains of life could markedly and adversely affect the course of a disease. The life stresses observed by the clinician are historically unrelated to an adaption syndrome.

The idea that human disease is somehow related to stress sprang from these roots. It is obviously an important and popular concept in physiology and medicine. In the laboratory, the adaption syndrome is a useful model for fundamental studies in neuroendocrinology. In clinical medicine, psychosomatic influences on human disease are being increasingly recognized, and explored. Despite this, the concept of stress has led to much confusion, and most modern authorities have recommended that the term itself, or the Cannon/Selye–clinical conception of it be eliminated because, as an organismal state, stress can neither be measured nor defined (112–115). This view seems to me to be the correct one, and will be followed here. As I hope the reader will ultimately agree, the decision to forego an attempt to understand the relationship between environmental factors and disease in terms of an internal state variable called stress is helpful in understanding the influences of environmental EMFs.

Although we shall avoid the term “stress”, the term “stressor” is quite useful and will be employed to denote the external stimulus. Specifically, by this term I mean any somatic or neurogenic factor that can elicit an acute adrenal-cortical response. A change in environmental temperature or EMF level, or death of a loved one are examples of stressors, the color of one’s socks is an example of a factor that is not (normally) a stressor. The factor can, but need not necessarily, elicit a response. Thus, if a temperature change $\Delta T = T_2 - T_1 = 1°F$ elicits a measurable response in an individual during a reasonable number of independent trials, or in an appreciable percentage of the population of a test species, then that factor can be a stressor. It is not necessary to specify whether T_1 or T_2 are normal or abnormal — it is enough that they are different. Characterization of a stimulus as a stressor always requires a frame of reference. Thus $\Delta T = 1°F$ may be a stressor for $T_1 = 70°F$, but not for $T_1 = 20°F$. Put another way, a change in temperature can be, but is not always a stressor. Similarly, environmental EMFs can be but are not always stressors (Figure 7).

CHRONIC STRESSORS AND ENDOCRINOLOGICAL ENDPOINTS

Application of acute stressors produces many complicated interrelated, time-dependent changes in adrenal-cortex secretions and other endocrinological endpoints (116–118). These endpoints tend to return to normal levels even with continued application of the stressor. Rats briefly subjected to 85-db noise exhibited a 9-fold increase in serum corticosterone (119). When subjected to the noise for 1 hr, a 4-fold increase was found, and when the stressor was applied continuously for 7 hr, only a 2-fold increase was observed (120).

Rats were placed in an immobilization apparatus that prevented any gross body movements, and plasma corticosterone levels were determined at various times after initiation of confinement

(using separate groups of animals at each time point) (121). At 20–200 min after the beginning of confinement, the corticosterone levels in the confined animals were significantly greater than those of the control (not confined) animals. About 24 hrs after initiation of confinement however, the corticosterone levels were identical in the two groups. Adrenal exhaustion could not explain the data (121).

Rats exposed to chronic crowding (0.1 in^2 of floor space per gram of body weight) or a cold stress (6°C) exhibited increased plasma levels of corticosterone after 1 week, but thereafter, at 2 and 4 weeks, the plasma levels were normal (122).

Human subjects exhibit significant adrenal–cortical activation when exposed to real or imagined important changers in their environment (123). Typically, there is a rapid loss of endocrine responsivity to the initial provocative stimulus, probably reflecting some form of adaption. Studies of plasma cortisol in paramedics, fire fighters, air-traffic controllers, and pilots have failed to demonstrate any long-lasting adrenal–cortical responsivity (124).

In addition to being inherently transitory, the magnitude of the changes in endocrinological endpoints depends strongly on many factors, only some of which can be satisfactorily quantified. These factors include circadian rhythm and environmental lighting (125), the nature of the stressor (126, 127), age (128–130), housing conditions (131), species and strain (132), and psychosomatic factors (123, 133).

Since neither man nor animals exhibit altered endocrine activity following repeated or chronic exposure to non-exhausting stressors, it seems clear that endocrinological endpoints are neither the mechanism nor the harbinger for an increased risk of future illness. Despite this, it is important that the reader recognize the importance of endocrinological endpoints in the evaluation of the health implications of stressors such as environmental EMFs. Changes in endocrinological endpoints permit characterization of a factor as a stressor. (They do not, however, permit risk evaluation or even the relative comparison of the magnitude of two different stressors — say 15 min exposure to 0°C compared to undergoing preparatory procedures in a hospital for coronary bypass surgery.) All chronically applied stressors have a non-zero probability of affecting an individual's risk for disease.

STRESSORS AND DISEASE

Extensive clinical observations have implicated stressors as risk factors for cardiovascular disease (134–137), diabetes (138), depression (139), allergies (140), and cancer (141, 142). Although the risk of disease associated with chronic exposure to stressors cannot be expressed in endocrinological terms, it can be studied and measured in the laboratory in other ways.

Animal models have been developed to facilitate controlled exploration of the link between chronic stressors and disease or other adverse physiological consequences. One method involves study of the effect of a chronic stressor on a non-endocrinological endpoint. Parameters involving growth, development, or healing may be sensitive to chronic stressors because they

tend to integrate small effects over time. For example, when pregnant rats were subjected to surface illumination (4280 lm/m^2, 35 min/day, on days 14–22 of gestation) the female offspring later experienced fewer conceptions, more spontaneous abortions, longer pregnancies, and fewer viable offspring than did the control rats (143). Beginning at 1–5 kV/m, power-frequency electric fields retarded fracture healing in rats (72, 73).

Another approach involves a functional measurement that samples reserve capacity (144). Mice were exposed to a regimen of chronic stressors administered over 2 weeks. The stressors included tail pinch (1 min duration), cold swim (3 min at 4°C), electric shock (30 min), shaker (30 min), water deprivation (24 hr), and isolation (48 hr). The individual stressors were randomly administered over a 2 week period, following which the chronically stressed animals were assigned to one of 3 experimental groups: one group was sacrificed and used for determination of plasma corticosterone levels, a second group was subjected to behavioral testing (gross locomotor activity), and a third group was exposed to an acute noise/light stressor prior to behavioral testing. Following testing, the mice were sacrificed and the corticosterone levels were determined. The corticosterone levels in the chronically stressed mice were normal, but their basal behavioral activity and their behavioral activation response to the acute stressor were each significantly reduced. The corticosterone response in the mice subjected to behavioral testing was significantly greater than that of the comparable control group.

A third approach involves the use of an animal model for a particular disease. The studies involving cancer models illustrate this approach, and will be considered here.

In these studies, the animal is injected or implanted with cancer cells that ordinarily will lead to its death within a reasonable period for laboratory investigation. Parameters that characterize the growth or development of the cancer including growth rate, tumor size, propensity for metastasis, duration of survival, and mortality can be directly measured. The pertinent question in such experiments is: Does the addition of a chronic stressor alter the course of the cancer in the host? We are interested in studies in which the stressor can be reasonably characterized as innocuous because, either by measurement or assumption, it transiently activates the adrenal–cortical system but produces no direct organic damage. (I do not include, for example, full-body exposure to X-rays.) Further, application of the innocuous stressor is continued throughout the period of observation so that we can validly speak of the simultaneous presence of the stressor and the disease. The control for these studies is the subsequent course of parameters that characterize the cancer in a group of animals that do not receive the innocuous stressor.

Female mice were injected with murine sarcoma virus and beginning on the day of inoculation, were subjected to 4 hrs of electric shock (100 10-sec treatments at 2–4 mA, DC) for 3 successive days (145). The shocked animals showed a significant increase in maximum tumor size. Application of a partial body cast altered the incidence of tumors in mice that were inoculated with a sarcoma virus 3 days later (146). Of 24 mice that received the lower inoculum range, only 8 of the control (not casted) but 19 of the casted mice developed tumors. Chronic exposure to microwave EMFs significantly hastened development of benzopyrene-induced skin

cancer in mice (147, 148).

Mice were injected with fibrosarcoma cells in the hind footpad, and the injected limb or the contralateral limb subsequently were amputated (149). Fourteen days after surgery the mice were killed, and the number of pulmonary metastases was determined. Groups that received the ipsilateral or the contralateral amputation both showed significantly increased pulmonary metastases (149). Similar results were seen in mice that were subjected to tumbling, immobilization or drugs that induced seizures (150). The mice subjected to these stressors exhibited higher numbers of lung colonies of cancer cells following injection of cancer cells.

More complicated experimental designs have yielded even more subtle insights into the nature of chronic stressors. Mice that received inescapable foot shocks exhibited accelerated tumor growth and decreased survival compared to unshocked animals (151). The ability to cope prevented both effects: in animals that received escapable foot shocks, both tumor endpoints were the same as those of the controls. Another report (152) described modification of the antitumor action of an immunomodulator by a stressor. The implanted tumor grew at a known rate in host animals, but did not grow in animals that also received the immunomodulator. If the animals were subjected to a stressor, however, the effect of the immunomodulator was overcome, resulting in tumor growth.

Riley studied the effect of environmental stressors on the latent period for mammary tumors in mice infected with the Bittner oncogenic virus (153). Under usual housing conditions, 80–100% of the female mice developed mammary tumors within 8–18 months after birth. In a normal mouse colony, the animals are exposed to dust, odor, noise, pheromones, and other potential stressors. In a group of mice housed under conditions that reduced exposure to these stressors, the specially-caged group had a median tumor latent period of 566 days, compared to 358 days for the group exposed to the physical stressors (153).

The Riley study brings into focus an inherent uncertainty in stressor studies. Some stressors may have a beneficial effect on an animal's ability to resist development of disease, and the investigator usually does not know which conditions should be associated with a beneficial effect — he can only establish that some conditions are different than others, and that, speaking anthropomorphically, some conditions are preferable to others. Each of the factors in the colony that Riley labeled "normal" was likely capable of producing a physiological response, and the signs, symptoms, and biochemical indices exhibited by the animals were each the sum of the constituent responses to the various stressors. Removing one factor (say, the dust) or adding a factor (say, doubling the pheromone concentration) might increase or decrease the value of any particular independent variable. Studies involving the effect of caging density of animals on various tumor endpoints are a good example of this complicated interplay. Such studies generally show that isolated animals are more prone to exhibit tumor growth (154–157), but it is not established whether the exacerbation of the cancer should be viewed as associated with the application of a chronic neurogenic stressor (isolation), or the removal of a protective stressor (pheromones, for example).

Immobilization, sound, and electrical shock stressors, each slowed the growth rate of a mammary carcinoma in rats (158). Inhibition of tumor development has also been reported by Newberry et al. When a shock stressor was administered to different groups of rats for 25, 40, or 85 consecutive days, the exposed animals exhibited fewer tumors after 40 or 85 days, compared to the controls (159). Rats subjected to a restraint stressor (5 or 10 hrs/day for 73 days) exhibited significantly reduced average number of tumors (160). The same result was observed when the restraint stress was administered for 12 hrs/day (161).

It seems clear that experimental neoplasms in rats and mice (and other disease states in animal models) are responsive to the environmental conditions of the host. That is, chronic innocuous stressors have a measurable impact on cancer growth. There is, however, no general formulation that permits us to predict outcome in any given set of circumstances. The nature and duration of the stressor, the extent of the exposed organism's control over it, housing conditions, and probably many other factors play an important role in the elaborated response.

MECHANISMS

The physiological details that constitute the link between chronic stressors and disease are peripheral to my purpose here, but they merit some consideration because even a brief discussion increases the plausibility of the link. Electric shocks depressed the cytotoxicity of natural killer (NK) cells in rats (162). A chronic immobilization stressor selectively affected different components of the cellular immune system in rats (163). After 11 days of exposure to the stressor, the number of total T cells was significantly decreased, but after a 12-day recovery period T-cell number was significantly increased. Total leukocytes, T-cell subpopulations, and NK-cell activity were similarly selectively affected by the restraint stress. Okimura et al. also observed suppression of various kinds of cell mediated immunity in mice subjected to a restraint stressor (164).

Stress-induced immunosuppression is not necessarily restricted to the period of exposure to the stressor (165). Rats were exposed to a foot shock, 2–4 hrs/day over a period of 6 months. One month after termination of exposure, the ability of splenic T lymphocytes to respond to a mitogen was suppressed. Thus, the rats apparently had a non-endocrinological memory of their recent history. Even adrenalectomized animals exhibit immunosuppression following chronic exposure to stressors (167, 168).

The effect of a chronic stressor on the immune system can be modulated by psychological factors (169). Rats given electric shocks exhibited immunosuppression (determined by the ability of their lymphocytes to respond to T-cell mitogens). But when the experimental animals were provided with the ability to avoid the stressor (although they received the same intensity, duration, and amount of electric shock as the first group), immunosuppression was avoided. Thus, the neurogenic factor modified the effect of the physical stressor.

For many years it has been recognized that there are nerve endings in the various organs and tissues of the immune system (thymus gland, bone marrow, spleen, and lymph nodes). Recent

evidence suggests that the two systems also have an intimate functional relationship in which lymphokines and neuroendocrine peptide hormones can be secreted and detected by the cells of either the immune or neuroendocrine systems (170).

ANALYSIS

Chronic exposure to stressors generally does not alter endocrinological endpoints. Despite this, it is clear that the physiological state of a chronically exposed organism can be differentiated experimentally from that of other similar organisms not exposed to the stressor. Chronic exposure to a stressor can alter an ongoing physiological response such as healing, and it can be manifested by changes in functional measures of reserve capacity. Chronic exposure to stressors can also alter, and worsen, manifestations of the disease state. In all cases, both in the laboratory and in life, an organism is responding to myriad external stimuli of various magnitudes and durations, only some of which are considered and analyzed as stressors. At any instant, each physiological parameter in the organism exhibits a value which is essentially an algebraic sum of the effects associated with each of the factors in the organism's environment. No particular parameter can even theoretically characterize either the state of the organism, nor the quantum of the impact associated with a particular stressor.

Chronic exposure to stressors can exacerbate the disease state, but psychological factors can modulate the response, and sometimes confer a protective benefit. Chronic exposure to stressors also affects the immune system, suggesting that impaired immune surveillance may be the mechanism underlying stressor-related effects.

If we apply the research — which was conducted mostly on rodents — to human physiology, what then can we reasonably expect will occur in a human population chronically exposed to an electromagnetic stressor? Since chronic stressors promote disease, and since EMFs are stressors, I conclude that higher disease levels will be found in the exposed population. That is, people who have been exposed to a chronic electromagnetic stressor by virtue of where they live (near a high-voltage powerline), work (electrical trades, military weapons operator), sleep (electrically heated waterbed, electric blankets), or play (amateur radio operators) will exhibit higher disease levels compared to appropriately matched control groups wherein such exposure does not occur. Becker reached this conclusion almost 15 years ago (171, 172). Its importance can be gauged by the virulence and source of its opposition. He based his view, in 1974, on scanty evidence, instinct and judgment, and consequently could have been wrong. Now, the view is based on voluminous data, and is inescapable.

We have no *a priori* or experimentally-based reason for expecting that one disease or another will be singled out from among the range of non-traumatic human diseases. Indeed, the generalized clinical experience is that all human diseases can be exacerbated by chronic exposure to stressors. Practically, however, cancer is the likely disease for study in exposed populations because it kills many people, and death certificates often list the decedent's address and occupation. Consequently, analyzable public-health records already exist.

EMFs AND DISEASE

INTRODUCTION

In therapeutic medicine, the physician's goal is the diagnosis and treatment of the patient's disease. In public health, however, the doctor is the government and the patient is the public at large. Consequently, the attitude towards public health varies with the political texture of the society. Concern for the well-being of its citizens has not been a dominant theme in the history of English-speaking people. Malthus viewed public health matters as nature's way of weeding out the genetically inferior individuals. Huxley equated attempts at promoting public health with interference in the survival and propagation of the fittest, and hence an ultimate deterrent to the human species. This attitude became incorporated in state and federal common law in the USA. State health departments were confined primarily to dealing with infectious diseases, filth, and other acute determinants of disease. At the federal level responsibility became split among several federal agencies within the Executive Department of government — which is to say that their vigor is under the direct political control of the President. As a consequence of this tradition, there has been little governmental (and no industry) interest in electromagnetic-field epidemiology. Despite the general absence of official support however, a broad array of epidemiological studies have been performed.

OCCUPATIONAL EXPOSURE

Zaret reported several small clusters of cancer among occupationally-exposed men (173). These included 2 cases of brain tumors among 18 workers servicing microwave communications equipment, 5 cases of cancer among a group of 17 men who worked on a weapons system involving electromagnetic pulses, and 3 cases of cancer among 8 men employed as repairmen for airborne navigation systems.

During a study of occupational mortality in the state of Washington that involved 438,000 deaths during 1950–1979 (174), Milham found a disproportionately large number of deaths from leukemia among aluminum workers (20 deaths as opposed to the expected mortality of 10.6). Since strong magnetic fields are created as part of the aluminum-manufacturing process, Milham chose job classifications as a surrogate for occupational exposure to EMFs. He found more observed than expected instances of leukemia in 10 of the 11 occupations considered including electricians, aluminum workers, linemen, power-station workers, and electrical engineers (Table 6). Chlorine manufacturing also involves exposure to magnetic fields, but a cohort of 157 exposed men (40–290 Gauss, 1 year or greater) had a normal cancer rate compared with that of Swedish men (175).

Wright et al. used Milham's occupational designations and studied the possible link to leukemia incidence (1972–1979) for white males in Los Angeles County (176). There were no cases in 2 categories, but the number of observed cases exceeded the number of expected cases in 7 of the remaining 9 categories. One of the two occupations that did not show an increase in

leukemia (welders and flame-cutters) was the same occupation that did not show an increase in the Milham study. An overall increase in acute leukemia was found (Table 6).

Coleman et al. examined the incidence of leukemia among men who were occupationally exposed to EMFs in southeast England (177). The 10 electrical occupations studied were essentially equivalent to the 11 categories used to classify American workers. They found a 17% excess of leukemias in the electrically-exposed occupations (Table 6). For 8 of the 10 occupations examined, more leukemias were observed than expected.

In studies that only analyze death certificates, an increase in the frequency of one disease may occur as a result of a decrease in the frequency of another disease. Thus, a higher than expected frequency does not necessarily mean that the true relative risk for the disease was increased. More confidence can be placed in the interpretation of the increased frequency as a true indicator of risk if the frequency is greatly increased (by more than a factor of three, for example) or if more than a handful of studies uncover an increased frequency.

Table 6. Leukemia Incidence (Mortality) in Men Occupationally Exposed to Electromagnetic Fields

		Leukemia		Acute Leukemia	
Investigator	**Study Area**	**Observed**	**Expected**	**Observed**	**Expected**
Milham (174)	Washington	(136)*	(92)	(60)*	(36.7)
Wright (176)	Los Angeles	35	27.2	23**	13.3
Coleman (177)	England	113**	96.5	45	35

* $P < 0.01$

** $P < 0.05$

Another concern relating to the validity of the EMF–cancer reports involves the occupational factors actually linked with the cancer. Chemicals or other factors might be wholly responsible, thereby acquitting EMFs as etiological factors. The occurrence of a multiplicity of studies (involving different designs and performed in widely separated locations) pointing to the same link is required to overcome this concern. Studies from disparate industries are particularly valuable.

Underground coal miners work in relatively close proximity to electrical power distribution lines that are strung overhead in the mines. In general, coal miners are not at increased risk for leukemia or other cancers (178). In a case-control study comparing 40 coal workers who died from leukemia with 160 controls who died from causes other than cancer or accidents, the odds ratio for chronic leukemia, chronic lymphocytic leukemia, and myelogenous leukemia were 8.2, 6.3, and 4.7, respectively (179). All these increased risk indices were statistically significant.

The incidence of cancer among white males who worked at the Portsmouth Naval Shipyard between 1952 and 1977 was studied in a case-control investigation (180). Fifty-three individuals who died with leukemia were each matched to 4 controls. No associations were found with ionizing radiation or solvent exposure, but significant associations were found for 2 occupations — electrician (odds ratio 3.0, 95% confidence interval 1.2–6.98), and welder (odds ratio 3.83 for myeloid leukemia, confidence interval 1.28–11.46).

Flodin et al. sought a link between background ionizing radiation and acute myeloid leukemia but, instead, found that electrical technicians, electrical welders and computer and telephone mechanics had a greater than average risk of developing the disease (odds ratio 3.8) (181).

McDowall reported on a case-control study involving 537 deaths in England and Wales (males greater than 15 years of age) that died from acute myeloid leukemia in 1973 (182). The control group included all causes of death except leukemia. They found a consistently increased relative risk for the occupations that involved exposure to EMFs: occupationally-exposed men had a relative risk of acute myeloid leukemia of 2.3 ($P < 0.05$). In a similar case-control study Pearce et al., reported that electrical workers in New Zealand were at increased risk of leukemia (183–184). Wisconsin electrical workers had a normal rate of leukemia mortality between 1963 and 1978, but individual occupations including electrical engineers and radio and telegraph operators exhibited significantly higher rates of leukemia than were expected (185).

In 1971–83, 1,691 deaths occurred in Washington and California among male members of the American Radio Relay League, a group of amateur radio operators. Twenty-four deaths from leukemia were observed, compared to the expected 12.6 (186) ($P < 0.01$). Employment-related exposure did not explain the leukemia excess.

Robinette et al., studied the effects of EMF exposure among Navy servicemen who graduated from training schools and served aboard ships during 1950-1954 (187). One group (20,781 men) consisted of subjects who worked as radiomen, radarmen, and aviation-electricians' mates, and the second group (19,649 men) consisted of aviation, electronics, or fire-control technicians. The men in both groups were heavily exposed, both occupationally (1000 $\mu W/cm^2$) and after work hours (because the ship's radars operated more than 8 hours per day), compared to typical exposure levels experienced by the general public in the USA. The chief difference between the groups was the possibility that some men in the smaller group were occasionally exposed to 100,000 $\mu W/cm^2$. No differences in variety of mortality and morbidity indices were found by the investigators that could be attributed to the difference in exposure, but there were 16 deaths in the combined groups from eye, brain, and other nervous-system neoplasms. This represented 7.9% of all malignant deaths as compared with the rate of 3.8% for the general population ($P < 0.01$) (188).

The choice of an appropriate control group is a significant inherent difficulty in studies with control cohort. If the true relative risk for disease is increased in both the case and control groups, as may be true in the Robinette report (187), then the study would actually be only a second-order study of differentially increased relative risk. Such a design has a built-in

assumption that relative risk is proportional to a surrogate for magnitude or duration of exposure. If the surrogate is invalid or the effect is not proportional to dose, then the validity of the study is destroyed. Another U.S. government-sponsored study with a dubious control group involved the foreign-service officers at the U.S. Embassy in Moscow who were subjected to microwave EMFs from sources outside the Embassy building (223). The control subjects were foreign-service officers who served in Eastern Europe and who may also have been subjected to microwave EMFs. This realistic possibility undercuts the conclusion (cancer among foreign-service officers in Moscow not related to microwave EMFs) of the study.

Lin et al. studied the relationship between occupation and brain-tumor mortality that occurred among white male Maryland residents between 1969-1982 (188). A total of 951 cases were included, consisting of 370 gliomas, 149 astrocytomas, and 432 brain tumors of unspecified type. Preliminary analysis showed more deaths among occupationally-exposed workers (electricians, electrical engineers, linemen) than expected (from 1970 Maryland Census data): 50 deaths from glioma and astrocytoma were observed (18 expected), 28 deaths from brain tumors of unspecified type (14.7 expected), and both differences were significant ($P < 0.01$). But a possible bias was introduced because the comparison involved mortality data from a 14-year span and occupation prevalence based on only one year. To overcome the problem, a case-control study was performed in which the control group consisted of white males who died from causes other than malignant neoplasms, matched on age and date of death. It was found that patients in occupations involving exposure to electromagnetic fields exhibited more glioma and astrocytomas than the controls: electricians (13 vs. 10), engineers (18 vs. 6) and utility company employees (19 vs. 11). The patients who died from gliomas or astrocytomas were younger by an average of 5.1 years compared to the controls.

The relative risk for brain cancer among English electrical workers was elevated (189). A Swedish study reported that powerline workers have a slightly elevated risk of leukemia and brain tumors, but that power station operators have normal cancer rates (222).

During 1964–1978, 157 cases of neuroblastoma occurred in children under 15 years of age in Texas for which adequate records could be obtained (190). A control group was formed from randomly selected birth certificates, and data on parental occupation was analyzed based on a system in which occupational exposures were classified according to presumed chemical exposures. Cluster 7 (formed on the basis of presumed moderate exposure to hydrocarbons) was associated with an increased risk of neuroblastoma (odds ratio 3.17). Cluster 7 included occupations that both involved (electricians) and did not involve (printers) exposure to EMFs. When the data was reanalyzed to include only Group 1 occupations (electricians, electric and electronic workers, linemen, welders, and utility employees) the odds ratio for neuroblastoma was 2.14. when Group 1 was expanded to include parents who sold or serviced electrical equipment, the odds ratio was 2.13 ($P = 0.05$). When only electronics workers were evaluated, the odds ratio was 11.75 ($P < 0.05$).

The authors did not specify explicitly whether it was the occupations of the fathers or

mothers that were being evaluated. If the link with neuroblastoma actually involved EMFs, it may have resulted from parental exposure of the mother. Perhaps the more intriguing possibility for the observed association (190) is that it was linked to EMF exposure of the children's fathers. There is some confirmatory support for this possibility: retinoblastoma occurred more often among children whose fathers were radio and television repairmen (191).

During 1968–1975, the annual rate of eye cancer among men who worked in the electrical and electronics industry in England and Wales was consistently greater than that of the general work force (192). None of the other 25 occupational groups showed consistent increases of comparable magnitude.

Vagero and Olin studied whether cancer cases in Sweden reported in 1961–1973 contained more cases of cancer among men or women aged 15–64 who were classified as working in the electronics industry (the particular occupations were not specified) (193). They found a 15% excess of cancers among men, and an 8% excess of cancers among women for workers in the electronics industry. Canadian high-voltage powerline workers exhibited more than a three-fold increase in cancer of the intestine ($P < 0.01$) (194).

EXPOSURE IN THE GENERAL ENVIRONMENT

In 1977 Becker reported a cancer cluster among residents of a rural area south of Syracuse, New York that was traversed by high-voltage powerlines and contained 20 antennas (195). The cancer incidence during 1974–1977 was almost double the rate expected in the county as a whole.

In 1979, Wertheimer and Leeper reported the first controlled study of a potential link between EMFs in the general environment and human disease (196). They asked whether children who lived in the greater Denver area, and who died of cancer in 1950–1973, lived near powerlines more commonly than did normal children. Their definitions of both "powerline" and "near" were arbitrary, and were rooted in their idea that any nexus between powerlines and cancer was mediated by the magnetic field of the powerlines. Their definitions were applied to the addresses of both the children who died from cancer, and to the addresses of an appropriately chosen control group. Roughly twice the expected death rates from leukemia, lymphoma, and nervous-system tumors were found in subjects living near powerlines. They performed a similar study among adults who died (or recovered) from cancer in 1967–1977, and again found an association between living near powerlines and cancer (197). Data for both studies is shown in Table 7.

Table 7. Incidence of Cancer Reported by Wertheimer and Leeper (16, 17). Children, $\chi^2 = 18.20$, $P < 0.001$. Adults, $\chi^2 = 7.94$, $P < 0.01$.

	Children†		Adults*	
	Cancer Cases	Controls	Cancer Cases	Controls
Near Powerlines	101	55	438	372
Away from Powerlines	171	217	741	807

†Birth addresses: all cases

*Wiring subcategories combined

Fulton et al. studied the possible association of childhood leukemia with powerlines in Rhode Island (198). The study was similar in some ways to that of Wertheimer and Leeper, and differed mainly in the manner in which nearness was defined. Using their definition, Fulton et al., could not demonstrate a link between childhood leukemia and powerlines. Their data was subsequently reanalyzed by Wertheimer and Leeper, and again an association with powerlines was not found (199).

It is difficult to characterize an ambient electromagnetic environment in engineering terms because of the spatial, spectral, and temporal variations of the fields. In epidemiological studies, this difficulty can sometimes be overcome by relating features of the environment, as opposed to actual EMF measurements, to cancer incidence. But perception of features of an environment is a subjective process, and divisions and classifications may no be made in the same fashion by different groups of investigators. Also, the design and density of powerlines varies significantly throughout the country, and therefore a classification system that indexes exposure in one area may not do so in another area. The validity of the scheme adopted by Wertheimer and Leeper as a surrogate for magnetic-field exposure was independently confirmed (200).

Recently, an exhaustive review and replicate study of the work linking childhood cancer and powerlines (196) was performed under the auspices of public-health and power-company officials in New York, with the result that the conclusion of a link between childhood cancer and powerlines was confirmed. Even more recently, the Electric Power Research Institute commissioned a second replicate study of the first replicate study.

A partial characterization of the EMF environment at two suburban elementary schools in northwest Oregon revealed power-frequency electric fields of 3–4.5 V/m inside the schools, and 3–170 V/m outside one school, and 1–18 V/m outside the other school (202). The magnetic fields inside the schools were less than 1 mGauss, but outside the school they were as high as 75 mGauss at one school and 35 mGauss at the second school. The power density in various frequency ranges between 1 kHz and 15.5 GHz did not differ between the two sites. A cluster of 4 cases of childhood cancer in a fourth grade class occurred within a 9-month period at the school with the higher outdoor power-frequency fields (202). The authors concluded that the

measurements indicated that the EMFs were not responsible for the cancer, but the opposite conclusion is equally valid based on the data presented.

In a case-control study that involved 716 tumors (660 malignant, 56 benign) in Stockholm County (1958–1973) in patients 0–18 years of age (203), the authors asked: (1) did more than the expected number of tumors occur in children who lived near 200,000-volt powerlines; (2) did more than the expected number of tumors occur in people who lived in regions with high magnetic fields? Among the tumor cases, they found 32 dwellings at which a 200,000-volt powerline was visible, but only 13 such dwellings were found in the control group ($P < 0.05$). Of the 48 dwellings that exhibited a high magnetic field (3 mG or greater) 34 were tumor cases and 14 control ($P < 0.05$).

The two major airports in Wichita, Kansas, use radars to control approaches and landings, and their beams blanket the city. Lester and Moore (204) asked whether the geographic pattern of cancer in Wichita was a complicating factor because the hills interrupted the line-of-sight beam of the radars thereby creating a shield from one or both radars in various parts of the city. A three-tiered measure of exposure was derived consisting of areas with the highest, intermediate, and lowest amounts of radar exposure. It was found that cancer incidence in Wichita residents in 1975–1977 (3,004 cases) was related to the amount of radar exposure ($P < 0.05$) after correcting for age, economic stratification, male/female ratio, and race. Those census tracts with the highest shield showed the lowest cancer incidence, and the tracts with the lowest shield had the highest incidence of cancer.

Counties in the USA that had an Air Force base had a significantly higher cancer mortality during 1950–1969 than did control counties without an Air force base (205–207).

A cluster of five cases of a rare ovarian tumor diagnosed over a 4-year period was reported in Florida (208). Three potential environmental risk factors were identified in the neighborhood where the children lived: proximity to a major highway, a lead smelter, and powerlines. The children lived 14–592 feet from a 69,000-volt powerline for an average of 7.8 years prior to diagnosis.

Electromagnetic fields have been linked with suicide (21), polycythemia (209), nervous system disorders (210, 211), sexual dysfunction (212), reproductive hazards (213), abnormal fetal development (214, 215), amyotrophic lateral sclerosis (216), heart disease (217, 218), and subjective complaints (219).

ANALYSIS

Leukemia has been linked to many occupations involving electricity. Such workers are exposed to many chemicals including diphenyls, naphthalenes, phenols, epoxys, oils, and solvents, any one or combination of which may have mediated the observed link. But most of the electricity-related occupations listed within the individual studies reported more cancers than expected (25 of 32 occupations described in the studies in Table 6). These occupations, which

included electrician, aluminum worker, power-station operator, powerline worker, and telegraph operator, have no discernible chemical factor in common. Furthermore, a link to leukemia has appeared in a group formed on the basis of hobby interests (186), thereby lending more credibility to a non-chemical hypothesis. Increased leukemia has been found in children who lived near powerlines (196). Powerlines can produce ozone, but this seems an unlikely explanation for the observed correlation because there were few addresses near the type of high-voltage powerlines that produce ozone. The evidence of a link between leukemia and EMFs has emerged from many different places: California, Denver, England, Los Angeles, New Zealand, Wales, and Washington.

The only common factor that linked cancer with the subjects' environment was the electromagnetic field. Since the frequency of cancer was increased when the electromagnetic field was added to the environment, the electromagnetic field was a risk factor for cancer.

Although the evidence of a link is reasonably clear, it is unsatisfactory in several salient aspects, the most important of which is that of the quantum of impact associated with specific patterns of exposure. If a child lives in a power-frequency magnetic field of 0.5 Gauss for 5 (10, or 15) years, what is his relative risk for leukemia? Heart disease? To what extent are the clinical signs and symptoms exhibited by the aged who use electric blankets associated with chronic exposure to the fields? To specifically what increased relative risks are individuals who live near airport radars or antenna farms exposed? These answers cannot be distilled from the present literature, but the reason for this inability merits our attention. Conclusive epidemiology (by which I mean the strongest possible statement that can be made linking a disease and a determinant within the intrinsic limitations of epidemiology) occurs in only two situations. Sometimes, the determinant is strongly and uniquely associated with a disease, such as malaria with mosquitos. Conclusive epidemiology can also result when the determinant is weaker but society's desire to know is strong, as manifested by its decision to fund the requisite good-faith research. The link between cancer and cigarette smoking is a good example. EMF epidemiology presently falls in neither category.

Infectious disease epidemiology involves the study of the dynamics of an offending microorganism, and its effects on human health. Good (mankind) and evil (microorganisms) are clearly distinguishable, and we have no difficulty in choosing sides in the conflict. EMF epidemiology is a more complicated matter because the offending fields in the environment are attributable to specific sources and their owners and operators. Thus, unlike the man–against–nature conflict in infectious disease epidemiology, we have a three-cornered conflict. Alas, it is the third corner that has the funds for the requisite studies.

If we choose to force an evaluation of the degree of risk associated with exposure to environmental EMFs from the existing epidemiological literature, then it seems to me that its adumbrations are truly ominous. The design of most epidemiological studies tended to make them relatively insensitive to finding a link between a surrogate for exposure, and disease, because most studies have been done cheaply. An insensitive study detects only the strongest

effects. Academic criticism has been directed toward the relatively primitive nature of the epidemiological studies (220), but in my view such criticism is largely misplaced because it is not reasonable to expect cheap studies to provide measures of relative risk or dose/effect. Further, such criticism generally relates to second- order concerns, and not to the basic public-health issues raised by environmental EMFs.

EMFs AND PUBLIC HEALTH

PUBLIC-HEALTH ASPECTS

The prototypical action of stressors is to promote (not initiate) disease, and this is the role that environmental fields play in the incidence and prevalence of human disease. No specific agent can accurately be said to cause any chronic human disease, and EMFs in the environment should therefore be viewed as one of a range of factors that can tax a subject's adaptive capacity. When the total body load of environmental stressors exceeds an individual's capacity, its immune surveillance mechanisms are impaired and disease occurs. Electromagnetic fields are one such disease-promoting factor. No other view in the literature fits all the data.

The most important deficiency in the existing data is our ability to actually measure the risk associated with specific patterns of environmental exposure in comparison to other accepted risk factors for disease such as cigarette smoking, sex, age, cholesterol level, and so forth. Although many aspects of the mechanisms of interaction will ultimately be deduced from relevant animal and tissue studies, such studies are inherently unable to provide data regarding quantification of the relative importance of the risk. Only human beings eat, work, play, sleep, and exercise like human beings.

The molecular mechanism involved in the actual detection by the body of the electromagnetic field remains purely speculative. None of the old theories (166) have born fruit. Recent reports by Liboff and colleagues (220) describe EMF-induced bioeffects whose physical basis may be understood. For now, I believe that it is better to state a general model based on hypothesis. Consequently, my summary evaluation of the literature is that it establishes that chronic exposure to environmental EMFs taxes adaptive capacity and thereby promotes human disease.

COUNTER-ARGUMENTS

In 1974 (1, 225) I first expressed judgments that subsequently evolved into the views expressed here (166, 201, 226–229). It was difficult, at that time, to truly accept the idea that such a pervasive entity as the non-thermal electromagnetic field could be harmful. My conclusion was worded delicately, with room for retreat in the face of definitive contrary data. But no data requiring a contrary conclusion has appeared, and I am now convinced that none will appear. Several arguments, however, have been made during the intervening 13 years whose thrust tends to oppose, weaken, or trivialize the conclusion that chronic exposure to

environmental EMFs is a risk factor for human disease. The historical details regarding these arguments — who spoke them, when and where — is described elsewhere (1, 230–232). Here, I intend only to briefly list and describe the principal counter-arguments, and to tell the reader why I have dismissed them. The lesson of the literature concerning the EMF-related health risk is contained in several bodies of data which must be integrated and connected to rationally support the conclusion. One must be aware that prior to the present broad-scale use of EMFs in the environment, there was no serious consideration given to the possible adverse consequences of human exposure. Thus, the present exposure patterns are built on assumptions of safety, and not actual evidence thereof. One must be aware of the laboratory work involving the exposure of numerous animal species to simulated environmental EMFs. If one neglects to consider all the pertinent work, it is possible to promote a prejudice against subsequent epidemiological studies showing apparent links between exposure and disease. One must also be familiar with the literature regarding the disease-promoting nature of chronic stressors. Again, if one does not admit the numerous studies that underlie this concept, then the epidemiological data seems wholly unsupported and slippery. One must be familiar with the human epidemiology of EMFs, and with the inherent limitations of that science — it forever deals in shades of grey, and never provides an unassailable conclusion. Finally, one must have a realistic notion of how science is done in the USA, and who pays for it. If the individual links in the chain are not connected, then the conclusion cannot be sustained for simple ignorance. But it is not ignorance that I wish to consider under the rubric of counter-arguments. Rather, it is affirmative statements in the literature that run counter to the judgments of risk that I have made.

The argument has been made that there simply are no biological effects due to EMFs, and consequently there can be no hazard associated with exposure to EMFs. This argument was first made in New York in 1975 (1), and subsequently has been repudiated by essentially every investigator in the field. It is, however, of more than historical interest because today, in the bulk of the present litigation in U.S. courts regarding risks of environmental EMFs, this remains one of the chief arguments advanced by the polluter.

A second argument is that several blue-ribbon government and industry panels have concluded that no significant health risk due to exposure to environmental EMFs has been proved. Such arguments are not *bona fide* opinion evidence because they are invariably collective judgments of individuals chosen by the polluter (106, 233). Such a procedure is simply a matter of the fox being on the jury at a goose's trial (1, 234). Blue-ribbon panels make it impossible to pin a given view on a given person, and to ascertain a specific basis for the view.

The biophysical argument is that EMFs can affect biological systems only by stimulating nerves or depositing heat, and that since environmental EMFs do neither, they cannot be a health risk. There is no official U.S. exposure standard for environmental EMFs, but there is an unofficial standard, and it is founded directly on this argument. The proponent of such a view assumes that nothing can happen to a biological system other than via processes that he first accepts as proven. Thus, the argument is invalid because it is unscientific.

It has been argued that power and communications companies have received no reports of illness among people living or working near EMF sources, and that one may visually inspect such premises and observe nothing untoward. The arguments, which appear in essentially all judicial and administrative proceedings in the USA regarding the EMF issue, are both self-serving and irrelevant.

It has been argued that there is much negative literature, and therefore that the reports are in conflict, or are contradictory, and that definitive statements regarding risk cannot be made. The argument is a shibboleth because the negative EMF literature establishes essentially nothing. There is not a single such study that has taught us anything worthwhile about nature. Anyone can drill a hole and fail to strike oil, and the existence of an empty hole is the meanest evidence imaginable that oil does not exist. To weight such studies against actual observations is illogical.

There is one further argument, a powerful one that may prevail. Whatever the magnitude of the risk of environmental EMFs, we must accept it because the alternative would cost too much, disrupt society, and endanger national security. Continued discussion is therefore pointless.

INVOLUNTARY HUMAN EXPERIMENTATION

Power companies, electric-blanket manufacturers, and other organizations produce products that liberate electromagnetic fields into the environment, but they are generally silent about this fact and most subjects are unaware of the presence of the EMFs. Because the various devices directly result in human exposures that are significantly above the ambient, the question arises whether the situation constitutes involuntary human experimentation. In New York, experimentation is defined in terms of physical intervention upon a subject that is not required for the direct benefit of the subject (235). The law provides “no human research may be conducted in this state in the absence of voluntary informed consent subscribed to in writing by the human subject…” (236). U.S. federal regulations governing human research are vastly more detailed, but also require voluntary written informed consent (except in special circumstances) (237). All modern authority opposes involuntary intervention upon a subject (238–240). My opinion is that many present-day exposure patterns, such as living near a powerline, are exactly the kind of physical intervention upon subjects that is proscribed by law and applicable ethical principles. In the USA, civil remedies exist (at least for non-military subjects) to counter such activity, including the law of nuisance, battery, personal injury, and inverse condemnation. Although some of these actions are presently encumbered with significant evidentiary problems for the plaintiff, the problems become fewer with time because of the tide of reports, latches of the defendants, and other factors.

SUMMARY

Chronic exposure to a biological stressor is a risk factor for disease. Laboratory studies show that electromagnetic fields can be biological stressors. Such fields, when present in the environment, are therefore risk factors for disease. The emergence of direct evidence of a

link between electromagnetic fields and one class of diseases — cancer — has been facilitated by the availability of cancer demographic data, and dose not imply that electromagnetic fields have a particular propensity to promote cancer as opposed to heart disease, psychiatric disorders, or other maladies. Controversy, or at least the appearance of controversy, regarding the health risks associated with environmental electromagnetic fields has developed because the emerging scientific picture runs markedly counter to the long-standing interests of some industries and government agencies in unbridled use of the electromagnetic spectrum. The existence of a link between electromagnetic fields in the environment and disease has been established despite the fact that many important details regarding it remain undiscovered.

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