Bioelectroclzemistry and Bioenergetics, 29 (1993) *255 276*
Elsevier Sequoia S.A., Lausanne

JEC BB 01580

Review

Electromagnetic fields, cancer, and the theory of neuroendocrine-related promotion

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(Received 16 July 1992; in revised form 6 August 1992)

Abstract

Environmental electromagnetic fields were predicted to increase the risk for cancer in chronically exposed human subjects because of impaired immunosurveillance mediated by the neuroendocrine system. This theory was examined by evaluating the human observational studies involving EMF-exposed subjects, and it was determined that the risk of cancer is greater when EMFs are added to the environment, at least for children and white males. The inference of risk obtained from the studies supports the theory of neuroendocrine-related progression of cancer but does not prove it because the studies provide no basis to exclude other possible mechanisms such as EMF-induced changes in ornithine decarboxylase, melatonin, or ion-resonance interactions.

INTRODUCTION

In 1972, Becker expressed concern regarding possible health consequences due to long-term exposure to electromagnetic fields (EMFs) in the residential and occupational environments [1]. The concern was based on laboratory observations indicating that induced electrical signals might compromise regulatory systems in the body [2-4]. We reviewed the evidence for the existence of biological effects in living organisms caused by non-thermal EMFs, and suggested that the effects were mediated by the neuroendocrine system [5-7].

The neuroendocrine theory (NET) for explaining EME-induced biological effects is depicted in Fig. 1. The EMF is detected by cells in the nervous system, and the information is transmitted to the hypothalamus which then orchestrates electrical and hormonal responses to oppose the randomizing effects of the EMF on the electrical interactions that mediate life processes such as transcription, translation, and antibody interactions. The magnitude and direction of each of the measurable parameters that constitutes the response

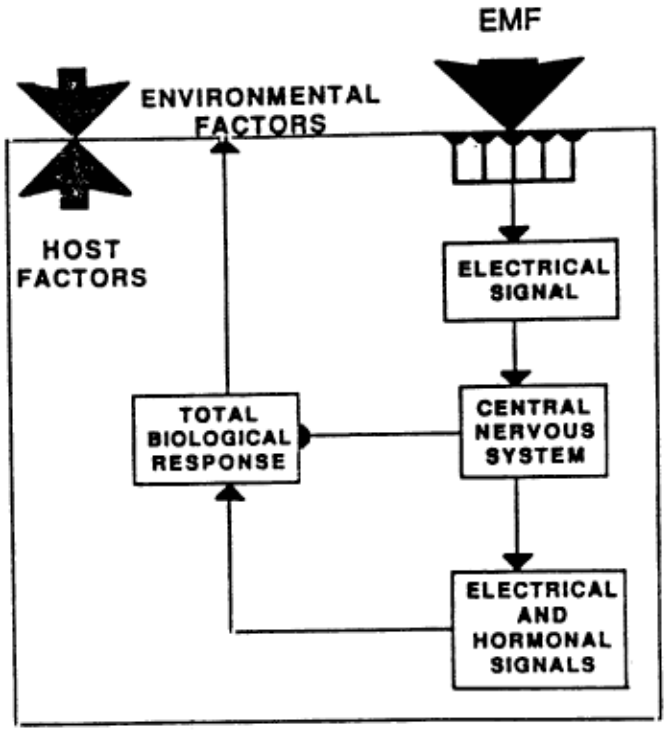

Fig. 1. The neuroendocrine theory for mediating EMF-induced biological effects. The field is detected and transduced into a biological signal which is received in the central nervous system. The resulting hormonal and electrical signals to the various body systems initiate adaptive physiological responses that are partly determined by host and environmental factors.

cascade are influenced by host factors, and factors in the host s environment. The defining characteristics of the NET are as follows: (1) the primary effect produced by the EMF in the detecting cell is a rapid functional change not involving protein synthesis or altered production of mRNA; (2) direct physical interaction between the EMF and the cell type whose behavior is characterized by the dependent variable in an experiment is not necessarily part of the causal chain leading to the bioeffect; (3) the magnitude and direction of the effects produced are determined jointly by the EME and other host and environmental factors; (4) there is no stoichiometric relationship between the energy of the EMF and the effect observed. The NET is consistent with the mutually unrelated laboratory studies involving EME exposure, including both the positive and negative studies (studies in which the null hypothesis was rejected and accepted respectively), and also with the positive studies that were subsequently followed by negative reports specifically disputing the original publication. Some such conflicts resulted from bias or differing experimental conditions between studies. Beyond these factors, however, valid issues of repeatability and consistency remain to be addressed and accommodated by a proper theory; the NET provides a tenable framework for understanding and addressing these issues.

Having adopted the view that the EMF is a stressor (Fig. 1), we may inquire about the likelihood for development of pathological processes in chronically exposed

human subjects. Chronic exposure of healthy animals to mild stressors does not generally produce permanent changes in endocrinological endpoints. Even so, the physiological state produced by chronic exposure can be differentiated experimentally from the state not involving exposure: chronic exposure can alter growth [8 10], produce changes in functional measures of reserve capacity [11], and worsen manifestations of disease in animals [12 28] and human subjects [29 351. Since chronic stressors can promote disease, and since EMFs are stressors, higher disease levels may be expected in the EMF-exposed human population [36]. That is, people who have been exposed to EMFs because of where they live or work will exhibit higher disease levels compared with appropriate control groups wherein such exposure did not occur.

Cancer is a good choice for use in evaluating the public-health implications of NET because (1) animal models have been developed to facilitate detailed studies, (2) a mechanism whereby stress may promote cancer has been identified, (3) cancer is a common cause of death and death certificates often list the decedent s address and occupation, thereby providing analyzable public-health records.

THE NEUROENDOCRINE THEORY AND CANCER

Experimental neoplasms in rats and mice are responsive to both host and environmental conditions. Mice injected with tumor cells and subjected to amputation [161 or tumbling [17] exhibited higher numbers of lung colonies of dancer cells. Environmental stressors altered the latent period for mammary tumors in mice infected with the Bittner oncogenic virus [20]. Female mice injected with murine sarcoma virus and subjected to daily electric shock showed a significant increase in maximum tumor size [121. Application of a partial body cast altered the incidence of tumors in mice that were inoculated with a sarcoma virus 3 days later [13]. EMFs hastened development of benzopyrene-induced skin cancer in mice [14,15] and altered tumor growth in mice that develop spontaneous viral mammary carcinoma [37]. Exposure to stressors can also retard tumor development: immobilization, sound, electrical shock and restraint stressors each slowed the rate of a cancer growth in rats [25-28]. Thus there is ample evidence to suggest that stressors such as EMFs can modify the dynamics of tumor growth in animals.

Stress leads to changes in many humoral and cellular immune mechanisms in laboratory animals and human subjects [38-41], particularly natural killer (NK) cells (large granular non-B non-T lymphocytes). NK cells are not MHC restricted and act as effector cells to mediate natural immunity; they also regulate and influence other components of the immune system via production of lymphokines [421. NK-cell activity is altered by surgical stress [43], electric shocks [44], cold [45], restraint [46], and rotation [47]. The suppressive effect of stress on NK cells is greater in older animals, compared with younger animals [48]. Stress can cause release of opioid peptides and corticosteroids, which in turn affect IL-2 and interferon [49,50], both of which are involved in NK function.

Much evidence suggests that NK cells form an important part of host defense against cancer [51]. NK cells appear to play a role in inhibition of tumor metastasis [52] and in the destruction of circulating tumor emboli [531. Immunosuppressive therapy in transplant patients depresses NK activity and increases the risk of cancer [54]. Patients with immunodeficiencies and depressed NK activity have a high incidence of cancer [55]. Low NK activity in normal individuals is associated with familial cancer [56]. These observations suggest that NK cells participate in immunosurveillance against neoplastic cells, and that the efficiency of immunosurveillance is reduced when the NK cell is impaired. Thus an association between EMFs and cancer is predicted on the basis of the NET as arising from EMF-impaired immunosurveillance.

Beginning in the late 1970s, many controlled human observational studies involving EMFs and cancer appeared, and they permit evaluation of the hypothesis of a link between EMFs and cancer. My purpose here is to review these studies.

DESIGNS FOR HUMAN OBSERVATIONAL STUDIES

The ideal study

The hypothesis of neuroendocrine-related promotion (NERP) of cancer is that a subject exposed to EMFs is more likely to develop the disease than would have been the case with all other environmental, genetic, and psychological factors remaining the same, except for the absence of the EMF. For a group of EMF-exposed subjects, the hypothesis is that their cancer rate will be higher, compared with non-exposed subjects. The hypothesis could be directly tested in a controlled study of EMF-exposed subjects such as residents near powerlines, users of electric blankets, amateur radio operators, or servicemen who operate electromagnetic weapons and communications systems. From among a group of healthy subjects; individuals would be randomly assigned to either the exposed or non-exposed groups, with appropriate balance for all known or suspected risk factors for all diseases. If the source of exposure were powerlines, the subjects in the exposed group would live beside powerlines that operated in a normal fashion, but those in the control group would live beside non-energized powerlines. For occupationally exposed individuals, the comparison would be between those receiving occupational exposure and those receiving sham exposure in the occupational setting. If the study subject were followed for 10-20 years, the relative risks for cancer and for each cancer subtype could be computed.

Ethical and practical factors preclude performance of such a study: (1) no authority exists capable of randomly assigning subjects to EMF or non-EMF groups; (2) the putative study is inherently inefficient statistically because great numbers of subjects must be followed to ascertain the existence of a relatively small number of subjects that develop cancer; (3) EMF dosage is the independent variable in the study but no practical method exists to characterize dosage in both groups, thus an unambiguous analysis of cancer rate in relationship to dosage

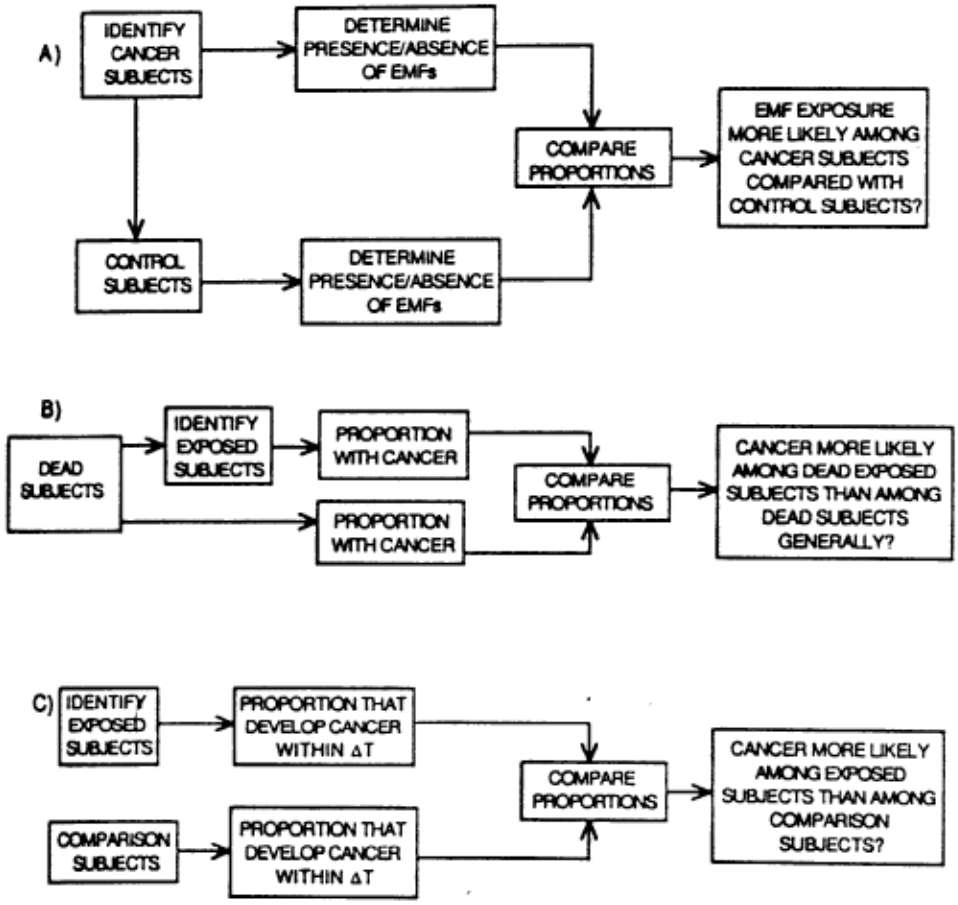

Fig. 2. Designs of human observational studies pertinent to evaluation of the NERP hypothesis: (A) case-control study, (B) proportional mortality (or morbidity) study, (C) standardized mortality (or morbidity) study.

would not be possible; (4) although some risk factors for cancer are known, other risk factors are presently unascertained, thus a null result could be equally explained by a failure to control for a pertinent risk factor or the non-significance of EMF exposure.

Possible inferences from practical designs

Several designs, less logically powerful but more practical than the ideal human study, have been used extensively in EMF observational studies (Fig. 2). In these cases the investigator neither randomizes the experimental subjects nor exerts the control that is routinely exercised by laboratory investigators. Consequently, the EMF is not restricted to the treatment group and the comparability of the groups with respect to some potentially pertinent factors cannot be guaranteed.

In a case-control study, subjects having the cancer or cancer subtype chosen for study are identified, and the proportion of the diseased subjects that had EMF exposure is determined (Fig. 2(A)). When a control group is chosen, the proportions of exposed

subjects in the two groups may be compared to determine whether EMF exposure was more likely among the cancer subjects. If so, one reasonable inference is that, in the future, similarly exposed subjects will be more likely to develop cancer compared with similar controls. If the control subject were disease free, then the hypothesis tested would be whether EMF exposure was more likely among cancer subjects compared with healthy subjects; thus, the potential biasing effect of EMFs toward the occurrence of cancer in healthy subjects which is the basic issue of concern would be assessable. If the study subjects had a particular type of cancer, say leukemia, and the control subjects had non-leukemia cancer, then the hypothesis actually tested would be whether EMF exposure was more likely among leukemia subjects compared with subjects having other forms of cancer. The assumption cannot be made that the subjects who developed non-leukemic cancer provided an unbiased estimate of the prevalence of EMF exposure among the non-diseased population because, under the NERP hypothesis, a diseased control group will contain a higher proportion of EMF-exposed subjects. Since the estimate of relative risk in an observational study involves comparison of the risks in the cases and controls, the use of a diseased control group can lead to an underestimation of the risk of EMF exposure in the healthy population [57,58].

A proportional mortality (or morbidity) study (PMR) (Fig. 2(B)) permits a determination of whether cancer or a cancer subtype was more likely among dead exposed subjects, than among dead subjects generally. Since a PMR study includes only dead subjects, and not subjects who were at risk of dying, no direct inferences are possible regarding similarly exposed but healthy subjects; within a particular study, one could justifiably conclude only that the association between EMFs and cancer was stronger than the association between EMFs and other causes of death. This is a well recognized inferential limitation of the PMR design [59].

The case control study employing a control group consisting of normal subjects is a more probative practical study design for evaluating association between EMFs and cancer, compared with either employing diseased subjects or with the PMR design. The standardized mortality (or morbidity) study (SMR) is a third practical design (Fig. 2(C)). Subjects exposed to EMFs are identified and followed to determine the proportion that develop cancer; a comparison with the corresponding proportion in another group permits assessment of whether cancer was more likely among the exposed subjects. In the SMR design, information is obtained about the population at risk for developing cancer in the sense that a higher incidence of EMF-related cancer would be observed as an increased proportion of the disease among the exposed subjects. However, an unascertained number of subjects in the comparison group wilt also have been exposed to EMFs, and those subjects might contribute disproportionately to the fraction of comparison-group subjects that develop cancer within the study time period. It cannot be presumed that the prevalence of EMF exposure among those in the comparison group who died was identical with that of the healthy population. There are no rules of inference or routines of thought for evaluating practical human observational studies that are more specific or reliable than those described in Fig. 2 [60-64].

The case-control studies that used a normal comparison group and the SMR studies will be analyzed together because they are more relevant to the association between EMFs and the risk of cancer in healthy subjects, compared with the case-control studies that used diseased comparison groups and the PMR studies. This choice for classification of SMR studies had no significant effect on the outcome of the analysis.

OBSERVATIONAL STUDIES OF HUMAN SUBJECTS EXPOSED TO EMFs

Introduction

This is a post hoc analysis to test a hypothesis not considered by the original investigators. To guard against a selection bias in favor of studies that supported the hypothesis, essentially every published EMF human study and every statistical comparison within a particular study that was pertinent to the NERP hypothesis were considered. Only some of the comparisons matter at all with regard to the NERP hypothesis, and few matter very much; those that do are included here. The number of associations examined or examinable in a study has no relationship to the plausibility of the NERP hypothesis as to warrant statistical adjustments for multiple tests because its validity does not depend on what else has been examined
[65,66].

It was sometimes necessary to calculate risk estimates that were not performed by the original investigator; in these instances, the χ^2 test was used, and 95% confidence intervals (CI) were calculated using the method of Haenszel et al. [67]. Disagreement exists regarding the relative roles of significance-testing and the confidence interval in evaluating epidemiological studies [65,66,68]. Significance-testing permits objective decision-making, and it will be employed here; the CI is also given because it defines the values for an association that are within a plausible range [66].

In describing the studies, particular attention was paid to the nature of the control group and the means by which the investigator determined the actual occurrence of EMF exposure. The former consideration determines the nature of the possible conclusion of the study and its relevance to the hypothesized association between EMFs and cancer; the latter factor is similarly crucial because the association cannot be tested in a study that does not involve an EMF-exposed group.

Some investigators evaluated their observations on the basis of what they believed to be a gradient in EMF dose. In my view, all such attempts were unconvincing, and those distinctions have not been respected in this analysis. If EMF exposure is a biologically significant factor, then different doses will surely have different consequences, as is the case for all other biologically significant factors. It is quite another matter, however, to prove dose-dependence in an observational study because the requisite characterization of EMF dosage is a presently unsolved problem. Some have argued that proof of dependence of risk

TABLE 1

Risk estimates for the occurrence of cancer, based on comparisons with normal subjects; OR odds ratio, SMR standardized mortality (or morbidity) ratio, CL confidence limits; where necessary, confidence intervals were calculated using the reported data; the time interval listed is that within which subjects were entered into the study

Reference	Place and Time	Parameter evaluated [a]	Surrogate for EMF exposure	Measure Of risk	Estimate of risk (95% CL) [b]
69	Denver, CO, 1950-1973	Childhood cancer	WL codes	OR	*2.3 (1.6-3.5)
70	Denver, CO, 1976-1983	Childhood cancer	WL codes	OR	*1.5 (1.0-2.3)
71	Seattle, WA, 1981-1984	Acute non-lympho-cytic leukemia	WL codes	OR	1.5 (0.8-2.9)
73	Rhode Island, 1964-1978	Childhood leukemia	Modified WL codes	OR	1.1 (0.7-1.6)
74	Los Angeles, CA, 1980-1987	Childhood leukemia	WL codes	OR	*1.7 (1.1-2.5)
		Childhood leukemia	24 h measurements (dichotomized at 1.18 mG)	OR	1.2 (0.7-2.1)
		Childhood leukemia	Spot measurements (dichotomized at 1.24 mG)	OR	1.1 (0.4-2.6)
75	Stockholm, 1958-1973	Childhood tumors	Spot measurements (dichotomized at 3 mG)	OR	*2.1 (1.1-4.2)
76	England, 1971-1983	Cancer	Residence near EMF source	SMR	1.0 (0.8-1.2)
		Lung cancer (female)	Residence near EMF source	SMR	*1.8 (1.1-2.7)

77	Sweden, 1960-1973	Cancer (males)	Job title	SMR	*1.15 (1.1-1.2)
		Cancer (females)	Job title	SMR	*1.08 (1.01-1.15)
80	Polish military, 1971-1980	Cancer	Job title	SMR	*3.0 (2.6-3.4)
		Leukemia and lymphoma	Job title	SMR	*6.9 (5.1-9.1)
81	New Hampshire, 1952-1977	Leukemia	Job title	OR	*3.4(1.4-8.2)
82	US Navy, 1974-1984	Leukemia	Job title	SMR	*2.4 (1.0-5.0)
83	Canada, 1965-1973	Leukemia	Job title	SMR	*3.5 (1.5-6.9)
84	Sweden, 1961-1973	Leukemia	Job title	SMR	1.0 (0.5-1.8)
85	Washington, California, 1979-1984	Acute myeloid leukemia	Amateur-radio operation	SMR	*1.8 (1.0-2.8)
86	Sweden, 1977-1982	Acute myeloid leukemia	Job title	OR	*3.8 (1.5-9.5)
87	USA, 1983-1987	Male breast cancer	Job title	OR	*1.8 (1.0-3.7)
88	Montreal, 1976-1983	Malignant melanoma	Job title	SMR	*2.7 (1.3-5.0)

*$P < 0.05$

[a] Endpoint of occurrence or death.

[b] Based on birth residences.

TABLE 2

Power-frequency magnetic fields measured at residences coded for EMF using the WL codes; the codes have been collapsed as follows: end pole + OLCC = Control; OHCC + VHCC = EMF; *N,* number of residences

Reference	Location		Magnetic field (mG)	
			EMF residences	Control residences
95	Denver, Co	Median	1.6 *(N* 190)	<0.5 *(N* = 227
70	Denver, CO	Mean	1.3 *(N* = 100)	0.6 *(N* = 334)
		Median	1.0 *(N* = 100)	0.4 *(N* = 334)
72	Seattle, WA	Mean	1.4 *(N* = 13)	0.5 *(N* = 26)
74	Los Angeles, CA	Mean	0.9 *(N* = 326)	0.6 *(N* = 345*)*
		Median	0.8 *(N* = 326)	0.5 *(N* = 345)

on EME dose is a condition precedent to the acceptable inference of a risk relationship, but the reverse is true: proof of a relationship between EMF exposure and risk is the necessary first step.

Comparisons involving normal subjects

In a study by Wertheimer and Leeper (WL), the cases were children who died with cancer and the controls were normal subjects identified from birth certificates [69]. The relationship between various predetermined classes of powerlines and the birth and death residences of the two groups was determined by inspection and measurement (the WL codes), and more than the expected number of cancer cases occurred among the subjects who lived near powerlines (Table 1); a similar result was found for leukemia. Evidence of the validity of WL codes as a surrogate for EMF exposure was provided by measurements showing a relation between field strength and the coding system (Table 2). In an independent study designed to test the validity of the original observations, Savitz et al. [70] coded blindly (resulting in a reproducibility of coding of *95%,* compared with 91% for Wertheimer and Leeper), and found similar results as reported previously (Table 1). Savitz et al. [70] also measured the magnetic field and provided independent verification that the coding system actually discriminated between the relative presence and absence of magnetic fields (Table 2). When the measurements were dichotomized at 2-3 mG, no correlation was observed with cancer; however, 64% of the cancer residences were not measured.

The WL codes were used in three locations other than Denver. In Seattle [71], the codes were validated [72] (Table 2), and no significant relation with acute non-lymphocytic leukemia (ANL) was observed (Table 1). In Rhode Island, an unvalidated version of the WL codes was used, and no link was found between EMFs and childhood leukemia [73]. In a Los Angeles study [74], childhood leukemia was considered in relation to magnetic fields as

indexed by the WL codes, 24 h measurements and spot measurements. An association with magnetic fields as indexed by the codes was observed, but no correlation was seen between leukemia and either mean 24 h or spot measurements of the field. Again, the WL codes were shown to delineate consistently residences with relatively high magnetic fields (Table 2).

In a study involving children from Stockholm who were diagnosed with tumors (92% malignant), matched controls were chosen from birth records [75], and the magnetic field at each residence was measured; more tumors than expected were associated with EMFs greater than 3 mG (Table 1).

MeDowall [76] identified more than 7600 persons within a study area in England who lived within 15 m of a transformer in 1971. Between 1971 and 1983 approximately 10% of the subjects died, and the observed number of deaths with cancer was that which was expected on the basis of national cancer rates (Table 1); the lung cancer rate among women, however, was significantly elevated. In Sweden [77], men and women in the electronics or electrical industry were at greater risk for cancer than the general working population (Table 1). The incidence of cancer among Swedish powerline workers during 1961-1979 was the same as that in other blue-collar workers [78], but the powerline workers had a significantly increased risk of chronic lymphocytic leukemia [79].

Szmigielski et al. [80] studied the incidence of cancer among Polish military career personnel who had occupational exposure to EMFs prior to 1980. The total population of career servicemen was analyzed, and about 3% were identified on the basis of job titles as having occupational exposure; they included personnel working on production, repair, and use of EMF devices, and those engaged in teaching and research with EMFs. Typical exposures were estimated at below 0.2 mW cm^2 for 4-8 h daily, with incidental exposures up to 1 mW cm^2, and short-lasting exposures as high as 20 mW cm^2. All cases of cancer diagnosed during the study period were evaluated, and the relative risks for cancer and for cancer of the blood-forming system were significantly higher among the exposed personnel (Table 1).

Shipyard workers did not have an increased risk for leukemia compared with the general population [81], but electricians had a significantly elevated risk compared with the general shipyard workforce; the finding of risk persisted after (he data were adjusted for potential confounders using a conditional logistic regression model (Table 1). Similar results were found for electrical welders (OR = 3.2, CI = 1.1-9.4). No significant relationships were seen with exposure to chemicals or ionizing radiation, as indexed by either job title.

Of *95* occupations in the US Navy, only electrician s mates had an increased incidence of leukemia during 1974-1984 (Table 1); electrician s mates work in an environment that contains elevated 60 Hz EMFs [82]. Among Canadian workers [83], death with leukemia was more likely to occur among telephone and powerline servicemen compared with the general Canadian workforce (Table 1); a similar result was found for intestinal cancer (OR = 2.4, CI = 1.0-5.0).

Telephone operators in Sweden [84] had no higher risk of leukemia during 1961-1973, compared with national incidence rates (Table 1), but increased risks were observed in American men as a result of hobby-related EMF exposure [85]. In the latter study, the OR for death from all causes among government-licensed amateur radio operators was 0.71, but the OR for death from cancer was 0.89. Thus, although the radio operators were healthy overall, they did less well with regard to cancer; several specific categories of leukemia were significantly elevated, including acute myeloid leukemia (AML) (Table 1).

Cases of AML were identified from the catchment area of four Swedish cities, and EME exposure was determined on the basis of job titles obtained from questionnaires [86]. A disproportionate amount of EMF exposure among the AML cases compared with normal subjects was observed (Table 1).

The effect of EMF exposure on breast cancer has been studied only in males [87]; the cases were identified through a cancer registry, and matched to normal controls. Job titles as determined during interviews were used as surrogates for EMF exposure. and men with occupational exposure were found to be more likely to develop breast cancer (Table 1). The incidence of malignant melanoma among male workers in a telecommunications industry [88] was greater than that of the residents of Montreal (Table 1).

American servicemen who graduated from US Navy technical schools during the Korean War were divided in half, and the mortality rates in the two groups through 1974 were found to be identical [89]. Despite a strong healthy worker effect [90], the proportion of study subjects all of whom were heavily exposed to EMFs that died with nervous-system malignancies was more than twice as great as expected based on the rates among US males aged 20 64 years in 1970 *(P* < 0.01) [91].

Several ecological human studies suggested that cancer occurs more often when EMFs are added to the environment [92-94]. Cancer incidence in census tracts in Honolulu, Hawaii having television or radio broadcast antennas was significantly higher among men in eight of the nine affected tracts, and among women in two tracts [92]. Census tracts that did not contain antennas had no elevated incidence of cancer [92]. In Wichita, Kansas, the relationship between cancer incidence and the probability for radar exposure was studied [93]. The authors determined the cancer incidence for 76 census tracts in Wichita during 1975-1977, and hypothesized that it was positively correlated with the probability of exposure to the air-traffic-control and weather-surveillance radars at Wichita s two airports, particularly along the terrain crests that interrupted the line-of-sight projection of the radar beams. A mathematical formula was used to characterize the relative radar exposure of each census tract, based on its relative elevation, distance from the radars, and the presence of intervening hills that tended to shield the census tract. A negative value of the computed parameter indicated that that census tract was shielded from the corresponding radar by intervening hills. Some tracts were unshielded, some had one shield, and some were shielded from both radars. The hypothesis was that cancer incidence would be highest in the unshielded tracts, and lowest in the tracts with two shields. Using a two-step multiple

regression analysis, cancer morbidity was found to be significantly related to the amount of radar exposure, as indexed by the shield. When the morbidity data were indexed **by** body site a significant result implicating the shields was found for the diagnostic category that included breast cancer [93].

The 91 counties in the United States that contained the city nearest each US Air Force base had significantly higher cancer death rates for both men and women during 1950 1969, when compared with population-matched counties without an Air Force base [94].

Comparisons involving diseased subjects

One study used diseased and normal subjects as the comparison group in a case-control design involving EMF exposure and cancer [95]. One case group consisted of subjects who died with cancer, and the comparison group was chosen from among those who died with any other condition. The second case group was 5-year cancer survivors; their controls were chosen from random telephone surveys. The case groups and matched controls were indexed for EMF exposure according to the WL codes, and a significant association between EMF exposure and cancer was seen (Table 3).

In an English study [96], subjects with lymphomas or leukemias were matched with patients recently discharged from hospitals, and no association with EMF exposure was found, as indexed by residing within 50 m of a powerline (Table 3). Coleman et al. [97] identified leukemia cases from another English study area, and found no significant increase in the odds of exposure (defined as residence near transformers), compared with subjects having other forms of cancer (Table 3).

Underground coal miners in the US were exposed to low-frequency EMFs from power distribution lines strung overhead in the mines [98]. When EMF exposure was dichotomized at 25 years of experience (those with fewer than 25 years underground work were considered to be unexposed to EMFs), the risk of death due to leukemia rather than other diseases was greater in the exposed group (Table 3).

In a French hospital-based case-control study [99], subjects with leukemia were about four times more likely to have had occupational exposure to EMFs, compared with subjects that had diseases other than leukemia (Table 3). A significant correlation with occupational benzene exposure was also reported (OR = 2.8), suggesting that their method of blindly coding occupational-exposure questionnaires was reasonably sensitive for purposes of risk analysis.

Using job titles as surrogates for EMF exposure, men exposed in New Zealand during 1980-1984 [100] were more likely to have leukemia than other forms of cancer (Table 3). The risk among electrical workers of dying in England and Wales with AML was elevated, compared with the risk of dying from other causes (Table 3) [101].

TABLE 3

Risk estimates for the occurrence of cancer, due to residential exposure to power frequencies, based on comparisons with dead or diseased subjects; PMR proportional mortality (or morbidity) ratio, SMR standardized mortality (or morbidity) ratio, OR odds ratio, CL confidence limit; where necessary, confidence intervals were calculated using the reported data; the time interval listed is that within which subjects were entered into the study

Reference	Place and time	Parameter evaluated [a]	Surrogate for EMF exposure	Measure of risk	Estimate of risk (95% CL)
95	Greater Denver, CO, 1967-1977	Cancer [b]	WL codes	OR	*1.3 (1.1-1.5)
96	England, 1983-1985	Leukemia, lymphoma	Residence near EMF	OR	1.2 (0.9-1.5)
97	London, 1965-1980	Leukemia	Residence near EMF source	OR	1.2 (0.8-2.0)
98	USA	Leukemia	Job title	OR	*2.5 (1.8-3.4)
99	France, 1984-1988	Leukemia	Job title	OR	*4.0 (1.3-12.9)
100	New Zealand, 1980-1984	Leukemia	Job title	OR	*1.6 (1.0-2.5)
101	England and Wales, 1973	Acute myeloid leukemia	Job title	OR	*2.3 (1.4-3.7)
102	USA, 1985-1986	Brain cancer	Job title	OR	*1.4 (1.1-1.7)
91	Maryland, 1969-1982	Brain cancer	Job title	OR	*2.2 (1.1-4.1)

103	USA, 1978-1981	Brain cancer	Job title	OR	*2.3 (1.3-4.2)
104	East Texas, 1969-1978	Brain cancer	Job title	OR	*4.6 (1.5-14.5)[c]
105	Washington, 1950-1979	Leukemia	Job title	PMR	*1.4 (1.1-1.6)
106	Washington, California, 1971-1983	Leukemia	Amateur-radio operation	PMR	*1.9 (1.6-2.2)
107	London, 1961-1979	Leukemia	Job title	PMR	*1.2 (1-1.4)
108	Los Angeles, CA, 1972-1979	Leukemia	Job title	PMR	1.3 (0.9-1.8)
109	Wisconsin, 1963-1978	Leukemia	Job title	PMR	1.0 (0.8-1.3)
101	England and Wales, 1970=1972	Leukemia	Job title	PMR	1.0 (0.8-1.1)
110	Finland	Leukemia	Job title	PMR	1.2 (0.7-2.0)

* $P < 0.05$.

[a] Endpoint of occurrence or death.

[b] Reduced representation of lung cancer.

[c] Definite + probably compared with possible + none.

During 1984-1985, 16 states supplied data to the US National Center for Health Statistics regarding more than 400 000 men who died with known occupations. The OR for mortality among workers in 15 electrical occupations were estimated, compared with those from subjects who died from any cause except brain cancer or leukemia. The age- and race-adjusted OR for brain cancer, but not leukemia, was elevated [102]. Three other studies linked occupational EMF exposure in men and brain cancer (Table 3). In a Maryland study [91], the risk of dying with brain cancer was greater among white males with putative occupational exposure to EMFs, compared with the risk of dying without cancer. In a second study [103], the authors considered the possibility that exposure to petrochemical pollutants might be involved in an association between EMFs and brain cancer, and they chose their case and comparison groups from regions containing petrochemical industries. Brain cancer cases among white men were identified from hospital records; those who were ever exposed to EMFs had a higher risk of brain cancer, compared with men who died from other causes. Speers et al. [104] compared white men who died with brain tumors and white men who died from all other causes during 1969-1978, and found that individuals with occupational exposure were more likely to be among the cases than the controls.

In Washington [105,106] and London [107], leukemia occurred more often than expected among subjects with various electrical occupations, but not in Los Angeles [108], Wisconsin [109], England [101], or Finland [110] (Table 3). In most of the studies, however, various leukemia subgroups and specific occupations had elevated PMRs.

In some studies the likelihood of EME exposure was too low, the presence of EMFs was inextricably confounded with a chemical agent, or the study contained too few subjects or a peculiar control group to warrant consideration [111-120].

DISCUSSION

When EMF exposure as indexed by residence, spot measurements, or job titles was compared in cancer and normal subjects, associations between the exposure index and the disease were usually observed (Table 1). Similarly, when the cancer rate in exposed subjects was compared with that in putatively unexposed subjects, associations between exposure and cancer were usually observed (Table 1). In all studies in which an association between EMFs and cancer occurred, the effect of the EMF was always to increase the relative risk estimate for cancer. No single report is conclusive in itself, but the Denver studies [69,70] are particularly supportive of an inference of association between EMFs and cancer because (1) the endpoint chosen was all forms of cancer, with no restriction to specific cancer subtypes, (2) the comparisons were based on normal subjects, and (3) the WL codes that were used as a surrogate of EMF exposure have been consistently validated as a true index of EME exposure.

The studies listed in Table 1 that failed to find an association between EMFs and cancer do not provide strong evidence against the inference of such an association.

Severson et al. [71] studied the association between EMFs and ANL, but it was unlikely that an increased incidence of such a rare disease [121] could be detected in a small study. Moreover, the control group was not representative because it had fewer *(P* < 0.1) smokers, non-whites, and poor people. The principal weakness of the Rhode Island study [73] was the uncertainty of EMF exposure.

A relationship between measured EMFs and cancer risk was not observed in the Los Angeles study [74], but (1) measurements were made for only about 61% of the study subjects, compared with about 92% of the subjects that were indexed using the WL codes, (2) the validity of neither spot nor 24-h-averaged measurement as surrogates for historical exposure was evaluated, (3) the variance of the EMF measurements was not reported, (4) the time of the year at which the EMF measurements were made was apparently not controlled, and hence the data may contain bias due to seasonal variations in currents in the powerlines near the subjects homes, *(5)* no realistic attempt was made to correlate the actual measured fields and the level of current loading on the powerlines adjacent to the subjects homes, (6) magnetic fields at homes of the cases that resided outside Los Angeles were not measured, (7) the local power companies were apparently not blinded as to the days and locations at which fields in the homes were measured, and (8) some measurements were arbitrarily excluded from the analysis.

All five studies that used unique and unvalidated surrogates for EMF exposure based on distance from powerline equipment [73,75,76,96,97] failed to find an association with cancer. The most likely explanation for this result is the lack of sensitivity and specificity of the exposure index used in each study the subjects that were classified as exposed were simply not exposed, compared with the subjects that were classified as not exposed. In England, most powerlines are underground, and thus the contribution of visible powerlines to the ambient EMF levels is less than in the United States. In the Swedish study [75] the investigators considered distances as great as 150 m to be within the zone of influence of powerline equipment, but the mean field strength at the residences labeled as exposed was the same as that at the control residences (1.0 mG), indicating that many residences were incorrectly classified in the exposed category.

In the absence of direct evidence that validates the index used to classify subjects as exposed or not exposed to EMFs, probably the best indication of reliability of an index is whether an association reported in one study using the index can be used in another study at a different location to find a similar association. EMF exposure indexed by one or more of a group of job titles has been found with remarkable consistency to be significantly associated with cancer and various cancer subtypes (Table 1). What are the possibilities of error in these studies? A repeated finding of statistical significance based solely on chance is not a plausible explanation for the observed results, and consequently bias in the classification of study subjects, imprecision in characterization of EMF exposure, or the presence of confounding factors are the only reasonable alternatives to the conclusion that the observed associations are real. Since there is no basis in any study to suspect that misclassification bias occurred differentially in the cases and controls, the only effect of this bias which

undoubtedly occurred in all the studies was toward the null hypothesis [122]. The indexed occupational groups were heterogeneous with regard to the occupations themselves, the work actually performed, and the meaning of the same job title in different countries. The major consequence of this heterogeneity was that the sensitivity and specificity of subject classification based on job titles was compromised, and therefore the studies were biased toward underestimation of the risk of EMF exposure. The heterogeneity also makes it unlikely that EMF-related job title was actually a surrogate for a workplace chemical factor that actually mediated the link with cancer, because no known factors other than EMFs were routinely present in each workplace studied. A consistently observed increased risk of a given magnitude cannot be ascribed to an unascertained factor whose prevalence varies in each study group [60]. It seems clear, therefore, that the possibilities for error in the occupational studies do not invalidate the conclusion that EMF exposure increased the risk for cancer. However, the findings do not mean that workers in all of the listed occupations have an increased risk of 2-3 times as a result of EMF exposure because it may be true that they were exposed to EMFs and other physical agents that combined to produce the observed associations. The evidence indicates that EMF exposure is a contributing factor in the production of excess cases of cancer. It does not indicate that it operates in the absence of other factors.

The risk estimates among occupationally exposed subjects as coded by job titles averaged about twice those observed in children who resided near powerlines, even though (1) the healthy worker effect was frequently observed among the entire study group in the occupational EMF studies, and (2) children are a more biologically vulnerable population. Among the possible explanations are the following: (1) EMFs at the higher frequencies (which were often present in the job-title studies but not in the childhood cancer studies) were more potent promoters of cancer, compared with power-frequency EMFs; (2) the presence of non-EMF factors in the workplace (not present in homes) contributed to the observed difference in risk; (3) the two kinds of studies differed in EMF dose because of differences in the average duration of exposure to EMFs; (4) the discrimination between exposed and unexposed subjects was greater when exposure was coded by job title.

The case-control studies listed in Table 3 show that an association between EMFs and cancer in diseased subjects can occur; this association makes it more likely that an association exists between EMFs and healthy subjects, compared with the likelihood that would pertain in the absence of the studies. Elevation of the risk of brain cancer (compared with the risk for other cancers) among electricians and engineers was reported in 1982 [123]. The same result was then reported in the four subsequent case control studies that compared the risk for brain cancer with the risk for other cancers associated with EMFs as indexed by job titles (Table 3). This indicates that EMF exposure promotes brain cancer more than it promotes other forms of cancer.

The likely effect of EMF exposure was to make the class of dead subjects larger than it

otherwise would have been, because EMF exposure increased the risk of dying. Since the PMR is computed after the population of dead subjects has been ascertained, the methodology is inherently incapable of directly addressing the link between EMF exposure and likelihood of disease. Nevertheless, almost every PMR study contains data indicating a significantly increased risk in one or more sub-groups.

The word *cause* is sometimes used in connection with the relationship between EMF and cancer, but the essential meaning of the term is a *necessary antecedent factor* [124], which does not apply to the EMF-cancer studies because not all subjects exposed to EMFs developed cancer and not all subjects that developed cancer had a history of excessive EMF exposure. Since EMFs are not a necessary antecedent factor for cancer, use of *cause* was avoided here and *association* was employed to characterize the link between EMFs and diseases that can be discovered in the human observational studies. One appropriate meaning of *association* is that the two events (cancer and EMF exposure) happen to occur during the same interval of time, as in the ecological studies [92-94], but the association referred to here is that which occurred between EMF exposure and cancer in the subject that actually developed the cancer.

CONCLUSION

The human observational studies, particularly those listed in Table I support the inference that the risk of all forms of cancer is increased when subjects are exposed to EMFs, as predicted by the NERP hypothesis. The studies involve mostly children and white males, and consequently the effects of EMF exposure on females is unknown, although it seems implausible to expect that females might somehow be spared the increased risk observed in males. The inference of risk at least for males and children obtained from the human studies supports the NERP hypothesis but does not prove it because the studies provide no basis to exclude other possible mechanisms. Laboratory studies involving human subjects have shown that EMFs induce electrical changes in the central nervous system [125-127], but the predicted immune-system consequences have not been demonstrated. Such evidence is necessary to prove that promotion of human cancer by EMFs is mediated by the neuroendocrine system.

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