Nonionizing Electromagnetic Fields and Cancer: A Review

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Low-frequency electromagnetic radiation had previously been thought to cause human injury only by generation of excess heat or by shock from direct contact with electric current. Information accumulating over the past few

Introduction

Oncologists are being asked to evaluate the possibility that a patient's malignancy, or a malignancy cluster, is causally related to exposure to nonionizing electric and magnetic fields. The answer to this question is unresolved, due to an incomplete and inconclusive preliminary research data base. There is a deceiving quietness about these fields compared with known carcinogenic agents like chemicals, which are generally easier to detect and more amenable to experimentation. Dose-response studies of chemical and physical carcinogens usually show a linear dose response, a characteristic that may not be true of electromagnetic fields. Some studies have suggested distinctive frequency zones of effect instead of linearity [1].

Although we are constantly bathed in electromagnetic fields, especially 60-Hz fields (Hz = cycles per second) related to electric power, field strengths generally reach high levels before we are aware of interaction with our physiology. Magnetic fields can induce electric currents in the body, but those too may be undetected. The human body generates its own internal electric currents and fields, measured routinely by ECG and EEG. The brain generates measurable electrical activity at four frequency ranges: alpha at 8-13 Hz; beta at greater than 13 Hz; theta at 4-7 Hz; delta at less than 4 Hz.

Nonionizing electromagnetic fields have been used in medical practice for many years; examples include ultrasound therapy of musculoskeletal disease [2], radiofrequency radiation ablation of abnormal heart rhythms [3], pulsed electromagnetic field treatment of orthopedic diseases [4], and radiofrequency- or microwave frequency-induced hyperthermia in antineoplastic therapy, either alone or in conjunction with ionizing radiation therapy [5] or chemotherapy [6].

Recent attention has focused on nonionizing, nonthermal electromagnetic fields as an environmental etiology of human disease. Research suggests that electromagnetic fields may influence the immune system [7], behavior [8], pregnancy [9], chronobiology [10], and cancer [11]. A number of case reports of diseases or symptoms related to exposure to these fields have been discussed [12]. Reaction to available information by the scientific-medical community and the public has been mixed, generating legal and regulatory activity and, within some groups, a rapid acceptance of the idea that these fields fit into the carcinogenic cascade.

In the United States and other countries, safe exposure guidelines for the fields generated by high-voltage electric power transmission lines, as well as other nonionizing electromagnetic fields, are available, but these are revised periodically [13]. Through the courts, legal action has attempted to associate exposure to nonionizing electromagnetic fields with the development of leukemia, lymphoma, and primary brain neoplasm. There are no good theoretical correlates at present to explain this wide range of neoplasias believed to be related to electromagnetic field exposure. Although most researchers in the field believe that electromagnetic fields are not a neoplastic initiator, considerable research has focused on whether these fields are involved in the promotional stages of human carcinogenesis [14].

Nonionizing Electromagnetic Fields: Background

Living organisms have evolved on the Earth while exposed to nonionizing electromagnetic fields produced by the Earth's natural conditions. The Earth generates a geoelectrostatic field of 130 to 150 volts/meter (V/m), which can vary by about 15%; much larger changes occur during geoelectrostatic disturbances [15]. Atmospheric disturbances may produce fields increasing to thousands of volts per meter [16]. The Earth also normally generates a weak static (non-time
varying) magnetic field with a fairly stable magnetic flux density of 30 to 50 microtesla (mcT),
varying by only about 0.1% daily [15,16]. Clinicians routinely expose patients to magnetic fields of 1
to 2 T during diagnostic magnetic resonance imaging, and these are considered safe [17].
Of the electromagnetic fields studied, those of 60-Hz electric power transmission frequency have
received considerable attention because of their omnipresence and epidemiologic studies suggesting
a relationship between these fields and the development of cancer [18,19]. The spectrum of
frequencies comprising electromagnetic radiation extends from the lowest frequencies, represented
by electric power transmission at 50 (Europe) and 60 (USA) Hz, to higher-end frequencies of ionizing
radiation at 10²ºHz)(Table 1).
Ionization potential indicates that the radiation possesses sufficient energy to cause ejection or
displacement of orbital electrons from atoms or molecules [20], and the energy content of
electromagnetic radiation ("photon energy") is directly proportional to its frequency [21]. At
frequencies greater than 1 million gigahertz, electromagnetic radiation is ionizing. Table 2 shows the
relationship between electromagnetic frequency and another measure of the energy content of the
electromagnetic radiation, electron-volts (eV).
The amount of electron-volt energy of the electric power transmission frequencies and microwaves is
minuscule when compared with that of ionizing radiation or higher frequency nonionizing
electromagnetic radiation. This is one reason that nonionizing electromagnetic fields are not
considered to be carcinogenic initiators.
Common daily exposure to low-frequency electromagnetic fields occurs not only from electric power
transmission lines and electric wiring inside and outside the home, but from in-home and
occupational devices and appliances that use 60-Hz electric power frequencies, such as electric
blankets, kitchen and bathroom appliances, computers and video display terminals (VDTs), and
telephones (cellular, cordless). Although these sources generate both electric and magnetic fields, it
is the magnetic field component that researchers believe may be involved in the promotional stage
of carcinogenesis. The strength of a magnetic field is commonly measured in milligauss (mG) or
microtesla (Table 3).
Table 4 shows a representative sampling of magnetic fields, measured in microtesla, produced by
various devices and appliances at two distances. The important point to be gleaned from Table 4 is
how rapidly field strength falls off when moving away from the field source. The number of persons
actually exposed to the very highest kilovolt (kV) electric power transmission lines is low. Table 5
shows the rapid fall off of magnetic field strength in milligauss and electric field strength in
kilovolt/meter from high-voltage electric power transmission lines carrying different voltages.
A well-known effect of the application of electromagnetic fields in science and clinical medicine is the
generation of heat. This is particularly important in deep heating treatment for physical therapy and
in the generation of antineoplastic hyperthermia. Although this is accomplished at frequencies much
greater than the 60-Hz electric power transmission frequencies, it is believed that the possible
involvement in the carcinogenic process by electromagnetic fields is in the nonthermal mode.
Ionizing radiation is either an electromagnetic wave-like radiation or particulate radiation. Particulate
radiations, including charged particles such as electrons, protons, alpha particles, or heavy ions,
appear to ionize directly; ionizing electromagnetic radiations (ie, x-rays and gamma radiation)
energize orbital electrons, producing fast recoil electrons and resulting in ionization [20]. Studies of
mutational spectra have shown that the greatest percentage of changes induced by ionizing
radiation are deletions [22].

Carcinogenesis

Initiation/Promotion
Carcinogenesis at the chromosomal-molecular level has two essential steps:
Initiation, through which a genetic mutation of some type occurs (eg, ionization radiation can cause
deletions, chemicals can cause transversions).
Promotion, during which epigenetic changes occur.
Each of these, in turn, may have multiple steps. Tumor initiation begins through mutations from
exposure to carcinogens, with cells displaying altered responsiveness to the microenvironment, and
promotion results in further clonal expansion and proliferation of initiated cells [22]. Initiation creates
a heritable change in the cell, which it does not express, with no change in the rate of DNA
synthesis, cell division, or enzyme activity [23]. It is known that a delay in time between initiation
and application of a promoter will still result in production of tumors (Figure 1, line 5).
Since nonionizing electromagnetic fields, especially those generated at 60 Hz, have such low energy
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It is unlikely that these fields are able to create mutational changes that would initiate cells. It has been suggested that nonionizing electromagnetic fields instead enter the carcinogenic pathway at the promotion stage [24]. One group has suggested that microwave fields may couple directly to electrostatic fields on cell membrane surfaces [25]. A series of studies with nonionizing electromagnetic fields has shown no genotoxicity in various cellular systems [26-30].

Promoting agents in chemical carcinogenesis are well-defined compounds that have very weak or no carcinogenic activity when tested alone, but markedly enhance tumor yield when applied repeatedly following a low or suboptimal dose of a carcinogenic initiator [31]. The major effect of promoters appears to be the specific expansion of an initiated cell population in target tissue such as the skin [32].

Promotion appears, at least in the initial stages, to be a reversible step in carcinogenesis, and may itself have more than one stage. Stage I promoters appear to require only one dose, and after this dose, the effect is irreversible for 4 to 6 weeks [32]. Because stage I is partially irreversible, there is a significant decrease in tumor production when more than 10 weeks (at least in the skin model) separates the application of stage I and II promoters. The two stages of promotion show a good dose-response relationship [33].

Much of what is currently known about the initiation-promotion scheme of carcinogenesis comes from the mouse skin model. A schematic representation of the timing and results of initiation and promotion is shown in Figure 1. In this scheme, the promotional agent is well controlled with respect to timing and dose. In Figure 1, substituting nonionizing electromagnetic fields for a known promoter like 12-o-tetradecanoylphorbol-13-acetate (TPA, an effective stage I promoter) implies, at least from a clinical standpoint, that the human subject interacts with a magnetic field of approximately the same strength at about the same time for the same amount of time, and with the whole or same area of the body.

Applying the promotional concepts found in skin carcinogenesis to nonionizing electromagnetic fields, it is clear that similar repetitive interaction with the initiated target would be required. However, it is not clear that a human subject comes into contact with a nonionizing electromagnetic field in the same way with each contact, making the concept of promoter dose application in the field, versus the laboratory, most difficult to interpret, and making time between contacts with the electromagnetic field a critical point. If nonionizing electromagnetic fields, even after one application, induce a retained change specific for electromagnetic field interaction upon which future application of the field can be built, the time between contacts with the field may be less important. The concept of dose is more difficult to apply to electromagnetic fields than to chemical promoters, since parameters needed to define the dose of nonionizing electromagnetic fields are being determined. The difficulty inherent in quantifying the actual dose received by an individual or group of individuals is one of the criticisms of some epidemiologic studies [11]. We are used to a model of the carcinogenic process implying that increasing doses of carcinogen result in greater risk of neoplasia. This may be true of chemical and physical carcinogens generally, but it is unproven in nonionizing electromagnetic fields. There are studies linking electromagnetic fields and their cellular effects with specific frequencies, above and below which the effects taper off [34-36]. This has been called a frequency window or tuning curve [1].

Ornithine Decarboxylase and Protein Kinase C

Ornithine decarboxylase is a cell-cycle-regulated enzyme with high activity in the G1 period of the cell cycle. It is useful as a marker because its activity can change markedly and rapidly in response to extracellular signals [37]. Ornithine decarboxylase is a controlling enzyme in the polyamine biosynthetic pathway, and an increase in its activity represents a cell’s change from quiescence to active growth [38].

Rapidly growing cells, or those stimulated to grow, show high ornithine decarboxylase levels. The loss of the simplicity of the development of a neoplastic cell from a nonneoplastic cell has prompted the appearance of models of tumorigenesis that are cascades of steps involving genetic and epigenetic mechanisms, all contributing to this transformation. Castagna et al [39] have shown that TPA appears to bind and activate the protein kinase C (PKC) enzyme. The PKC enzyme appears to be activated by diacylglycerol, a chemical that has a structure similar to that of TPA, although with a lower overall affinity; there is a good correlation between relative tumor-producing ability of various phorbol esters and capacity to activate PKC [33].

Protein kinase C itself has multiple forms, and these forms show differential tissue activity, although the individual functions of these forms are not exactly clear. Some work has shown that certain oncogenes alter expression of some of the isoforms [40,41], and individual isoforms of PKC have distinct biologic effects in the same cell system [31]. The PKC receptor protein location in the cell

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membrane may allow it to be the receptor for a variety of tumor promoters, transducing this signal to the interior of the cell [33]. Experimental data for the interaction of electromagnetic fields with ornithine decarboxylase and PKC are reviewed below in the section on cellular studies.

The Cell Cycle/Transcription

Cell cycle dynamics and control of the normal cell cycle play an important role in the neoplastic process. Three groups of cells are thought to be representative of the cell cycle at any one time:

1. Cells that have achieved a final differentiated form from which they will carry out a specific task.
2. Persistently proliferating cells, which are required to replace tissue systems in constant need of replacement.
3. Recruitable cells, which exist in the steady state phase of carrying out a specific task but can, when properly stimulated, enter active cell cycling.

The cell cycle follows a series of steps, including the S phase during which chromosomal replication, size doubling, and mitosis occur. Cell surface receptors and ligands have a significant role in regulating the extent of cellular proliferation; the IGF-1 receptor may play such a role [42].

Several nuclear tumor-suppressor genes produce proteins affecting the cell cycle. The p53 and Rb genes produce proteins that probably curb cell proliferation by interrupting the G1 period of the cell cycle [43]. The genetic effect of mutational change has become a well-defined avenue in the carcinogenic pathway, with work identifying oncogenes, proto-oncogenes, and tumor-suppressor genes. A normal cellular proto-oncogene can be converted into a virulent oncogene by a single-base change (point mutation) [43]. The protein product of the p53 gene is, by itself, a transcription factor that binds to a specific DNA sequence and activates the transcription of a nearby gene [43].

Cellular transcription occurs through the activation of DNA-dependent RNA polymerases, of which there are three in the cell nucleus. RNA polymerase II, transcribing the messenger RNA that encodes proteins, is important in gene regulation [44], Goodman et al [45,46], have shown that cellular transcription can be induced or enhanced by low-frequency pulsed magnetic fields, which has implications not only in carcinogenesis but also in medical treatment. However, this work has not yet been duplicated.

Pineal Output of Melatonin

The pineal gland, located in the brain as an integral part of the optic pathway, was at one time considered to be an additional eye. The hormone melatonin, a major product of the pineal gland, is an integral part of the human circadian rhythm system. Circulating melatonin concentration in humans exhibits a circadian rhythm: Levels are low in the daytime and begin increasing a few hours after the onset of darkness, with peak values reached at 2 am to 4 am [47]. Melatonin also varies during the menstrual cycle, with the lowest peak values during days 13 to 17 of a 28-day cycle [47].

In 1980, the pineal gland was shown to be sensitive to fluctuations in magnetic fields by a group who theorized that birds used the Earth's magnetic field for navigation. These researchers showed that in birds the relationship between light and dark and melatonin was abolished when either the pineal was removed or the magnetic field was changed [48]. When guinea pigs were subjected to a changing magnetic field, electrophysiologic monitoring of the pineal gland showed that this organ's electrical activity was the sole brain area responding to the magnetic field [48].

The presence of melatonin appears to protect humans against neoplastic development and propagation, and one group of investigators has proposed that low melatonin levels may be a factor in the genesis of breast cancer [49]. Others have suggested that extremely low frequency nonionizing electromagnetic fields do change pineal function and decrease the synthesis and release of melatonin [50]. The sum of this information has led to the hypothesis that electromagnetic fields, by possibly decreasing pineal output of melatonin, may provide a mechanism for breast cancer development [51].

Experimental Studies

Cellular Studies

Balcer-Kubiczek and Harrison [52] exposed C3H/10T1/2 cells to 24-hour 2,450-MHz microwave radiation accompanied by benzopyrenes simultaneously or by x-rays sandwiched between the microwave radiation. X-ray groups were also treated with TPA postirradiation. Cultures with the same parameters exposed to sham microwaves served as controls. The data suggest that microwave frequency irradiation, without inducing thermal change, enhanced transformation frequency of the cells in this system, if cells previously exposed to microwave frequency and x-rays were subsequently cultured with TPA.

The authors suggest two possible hypotheses:
1. Microwave frequencies are independent tumor initiators.
2. Microwave frequencies interfere with repair of ionizing/chemical damage.

In a further study by this group [53], this cell system was exposed to 2,450-MHz microwave frequencies for 24 hours and/or 1.5-Gy 238-kV(p) [kilovolt peak] x-rays at 3.75 Gy/min. Transformation frequency and cell survival were measured with and without postirradiation addition of TPA. Microwave frequencies had no effect on the survival of x-ray irradiated cells, and TPA increased cell transformation by x-ray by about three times. There was a significant increase in transformation frequency with microwaves and TPA in postirradiation groups, but no transformed foci observed after microwave exposure of the postirradiation groups and no TPA. The authors felt that overall, the results confirmed the enhancing effects of microwave frequencies on neoplastic transformation, without specifying microwaves as a primary carcinogenic agent.

Byus et al [38] noted a 30% increase in ornithine decarboxylase activity in a hepatoma cell line exposed to an electric field of 0.1 mV/m for 1 hour. This group also measured ornithine decarboxylase levels in hepatoma, CHO, and 294 T melanoma cell lines after exposure to a 450-MHz microwave radiation, modulated with 16 Hz amplitude modulated signal at 1.0 mW/cm². The three cell lines were exposed to this signal for 1 hour, and the data indicated an increase in ornithine decarboxylase activity in all three cell lines. In two of the lines, hepatoma and CHO, stimulation of ornithine decarboxylase by TPA was enhanced by the 1-hour field exposure.

Cossarizza et al [54] have looked at whether 50-Hz nonionizing electromagnetic fields could affect repair of damage to lymphocytes by gamma irradiation or of damage induced by antiproliferative agents. Cells exposed to gamma irradiation with or without electromagnetic fields showed a similar pattern of survival, and there was no worsening lymphocyte survival with combined nonionizing electromagnetic fields and bleomycin, vinblastine, or doxorubicin.

Cain et al [55] have explored the potential for electromagnetic fields to be a copromoter with a chemical tumor promoter. This work used a 60-Hz nonionizing electromagnetic field along with TPA in a C3H/10T1/2(10e) cell system. Cultures were exposed to 60-Hz fields of 1 G for 1 hour four times a day for 28 days; field exposure alone did not promote focus formation. When TPA at 50 ng/mL was added, the cells exposed to electromagnetic fields showed increased number of foci and increased total area of foci/dish. Data review suggests that electromagnetic fields plus TPA acted as copromoters to increase the number and, to a lesser extent, the size of foci. Since these results indicate that nonionizing electromagnetic fields may copromote to increase the number of foci, this may support electromagnetic fields as a promoter, since a possible function of tumor promoters is to increase the clone size of an initiated cell population.

Salvatore et al [56,57] irradiated MCF-7 breast carcinoma cells in culture with a 15-Hz, 1 G magnetic field with and without chemotherapeutic agents. Cells were cultured to a density of 250,000/mL, and wells containing 25,000 cells/well were prepared. Antineoplastic agents (methotrexate, doxorubicin, and cyclophosphamide) were added to wells in increasing concentrations, and the wells then incubated at control temperature of 37º ± 0.1ºC either with or without (control) the magnetic field. Optical density measurements were done at 96 hours using the Tetrazolium assay technique. No increase in cell proliferation was seen in MCF-7 cells exposed to the magnetic field alone, and the combination of magnetic field and chemotherapy increased cell kill over chemotherapy alone (Table 6).

**Animal Studies**

Mouse models of carcinogenesis are the basis for a number of studies seeking to elucidate the role of electromagnetic fields as promoters or copromoters. Studies have also used extremely low frequency nonionizing electromagnetic fields and microwave frequencies in models of skin, breast, and hepatic neoplasias. Other observations have been recorded in animal systems [57]. Thomson et al [58] found no increased incidence or effect on the progression of P388 leukemia in mice. Preskorn et al [59] recorded an inhibition of the growth of tumor and an increase in life span of mice after fetal irradiation with microwaves, although there was a slight increase in temperature in this system, and Mikhail and Fam [60] have suggested that these fields increased the incidence of malignant lymphoma in mice.

**Extremely Low Frequency (ELF) Studies**--Bellossi et al [61] used three extremely low frequencies-12, 100, and 460 Hz-to expose a female mouse mammary carcinoma system to nonionizing electromagnetic fields. Using tumor weight and weights of the lungs and spleen, the authors concluded that there was a differential effect on this system according to the frequency: 12 Hz may promote growth, 100 Hz showed no effect, and 460 Hz impaired growth.

McCLean et al [62] have used a 60-Hz 2-mT (20 G) electromagnetic field to expose SENCAR mice for 21 weeks. Animals were first exposed to subthermal doses of DMBA [dimethylbenz(a)-anthracene],
and beginning 1 week after this, were either exposed to the field or sham exposed. No tumors developed in these groups. Substituting TPA as the promoting agent, there was a slight but not statistically significant earlier tumor development in the magnetic field/TPA group. Measured natural killer cell activity did not differ in any of the groups.

Stuchly et al [63] have exposed SENCAR mice to a 60-Hz 2-mT field for 6 hours a day, 5 days a week, after initiation with a single application of DMBA and in conjunction with suboptimal weekly TPA. The data suggest that the field modified the time course of tumor development, with statistically significant differences in rate of tumor development, but not in the number of affected animals or in the mean number of tumors at the end of promotion. The authors feel that the findings in this study may support the concept that the interaction between magnetic fields and living systems is weak and precipitated by other agents (eg, TPA).

Rannug et al [64] used a rat hepatic model (Sprague-Dawley) to observe interaction of magnetic fields with initiators and promoters. Fifty-Hz magnetic fields at 0.5 mcT or 0.5 mT were applied during both initiation phases and continued until animal sacrifice after 12 weeks of phenobarbital exposure. This work did not suggest that magnetic fields acted as copromoters but, rather, that in some instances magnetic fields were acting to inhibit tumor formation.

Mevissen et al [65] used both a direct current (DC) magnetic field (non-time varying) and an alternating current (AC) magnetic field (time varying) to observe promotion/copromotion in a rat mammary carcinoma model initiated with DMBA. High-strength DC (15 mT) and AC (30 mT) magnetic fields appeared to exert statistically significant effects on tumor development and/or growth.

The authors interpreted the increase in tumor weight but not number by the DC magnetic field as copromotion, and the increased number of tumors per animal by the AC magnetic field as promotion, although with a low number of animals per experiment, statistical evaluation was limited. Exposure to 50-Hz magnetic field strengths of 0.3 to 1.0 mcT revealed no evidence of tumor promoting effects at these low flux densities.

**Microwaves**--Szudzinski et al [66] used Balb/c mice exposed to benzopyrene for 6 months and simultaneously irradiated with athermal (5 mW/cm²) or subthermal (15 mW/cm²) 2,450-MHz microwaves. Another animal group was preirradiated with microwaves at 10 mW/cm² for 1, 2, or 3 months, then treated with benzopyrene. Control animals were treated with benzopyrene and sham irradiation. The microwave irradiation groups showed an acceleration of development of benzopyrene-induced skin cancer and shortened life span of tumor-bearing hosts. This effect appeared greater at higher field strengths and longer exposure times, indicating a possible dose dependency. The low level, longer exposure to microwaves appeared to lead to marked suppression of delayed hypersensitivity, and this effect on the immune system may contribute to accelerated neoplastic development and growth.

The same microwave frequency (2,450 MHz) was used by another group [67] in C3H/HeA mice with a high incidence of spontaneous breast cancer and Balb/c mice treated with benzopyrene resulting in skin cancer. The irradiation protocol was carried out in an anechoic chamber at 5 or 15 mW/cm² for 2 hours a day, six sessions a week. C3H/HeA mice were irradiated from the 6th week of life up to the 6th month of life. Balb/c mice were irradiated either prior to benzopyrene (1 or 3 months) or simultaneously with benzopyrene (over 5 months). Control groups were maintained. Review of the data shows that for groups radiated at either 5 or 15 mW/cm² for 3 months, there was an increase in the mean number of neoplastic lung colonies and an acceleration in the development of benzopyrene-induced skin cancer. The C3H/HeA mice exposed to microwaves developed breast tumors earlier than controls. The accelerated cancer development in these test systems at 5 mW/cm² was similar to that caused by chronic stress related to confinement, but different from that of 15 mW/cm².

Santini et al [68] used a lower power density of 2,450 MHz microwaves (1.0 mW/cm²) 2½ hours a day, six sessions/week until death in a B16 mouse melanoma system. No increase in tumor size or decrease in survival time was seen in the treated animals.

**Summary**

In spite of the increasing exposure of the general population during much of this century to nonionizing electromagnetic fields, there does not appear to be a correlative increased incidence of neoplasias from these fields, regardless of their possible effect in the carcinogenic cascade. Although one conclusion is that nonionizing electromagnetic fields are contributing minimally to the promotion or progression of neoplasms, an alternative concept is that the effect of these fields occurs in a specified population, a group that may be more susceptible to these fields. A search for cellular or other markers may identify this property.
Physicians are in a position to recognize variations of known disease, and the appearance of new disease. Oncologists may attempt to document the nature of a patient's nonionizing electromagnetic field exposure (type, timing, field strength, others with a similar malignancy) and other factors normally considered in any patient who has developed cancer, but it is not yet clear how to apply this information. However, oncologists may find it difficult to conclude that a patient's malignancy (or malignancy in a community cluster) is causally related to exposure to nonionizing electromagnetic fields.

Federal legislation established the National Electromagnetic Field Advisory Committee to help focus future research into these fields, and although investigation of carcinogenesis is part of the research agenda, it is but one of a number of research needs to understand nonionizing electromagnetic fields and their relationship to human disease.

The Environmental Protection Agency has issued a study on research needs and priorities concerning electromagnetic fields. This work emphasized the urgent need for studying the biophysical mechanisms and exposure assessment, along with studies on health effects. There is much confusion about the carcinogenic potential of electromagnetic fields, and the representative data reviewed here do not allow us to conclude that such fields unequivocally participate in the carcinogenic cascade. Scientific experimentation continues, and the research data base that implies that nonionizing electromagnetic fields are nongenotoxic but have activity at the cell membrane, or influence biochemical processes, requires confirmation.

A number of legal outcomes have favored the view that malignancy is not causally related to nonionizing electromagnetic fields exposure, and this appears appropriate based on the current available scientific and medical data. Future funding of this research is essential to answer the questions of possible carcinogenic or other health effects of nonthermal, nonionizing electromagnetic fields.

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