Radiofrequency Field Exposure and Cancer: What Do the Laboratory Studies Suggest?

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Significant concern has been raised about possible health effects from exposure to radiofrequency (RF) electromagnetic fields, especially after the rapid introduction of mobile telecommunications systems. Parents are especially concerned with the possibility that children might develop cancer after exposure to the RF emissions from mobile telephone base stations erected in or near schools. These questions have followed scientific reports suggesting that residence near high voltage power lines may to be associated with an increased childhood leukemia risk. Epidemiologic studies have been plagued by poor RF exposure assessment and differences in methodology. There are no high-quality epidemiologic studies that can be used to evaluate health risks from RF exposure. Laboratory studies in this area have been somewhat confusing. Some animal studies suggest that RF fields accelerate the development of sarcoma colonies in the lung, mammary tumors, skin tumors, hepatomas, and sarcomas. A substantial RF-induced increase in lymphoma incidence in transgenic mice exposed for up to 18 months has also been reported. In contrast, other studies have not found carcinogenic effects. These conflicting results indicate the need for more well-conducted studies on laboratory animals, supplemented with high-quality *in vitro* studies to identify effects that need further research *in vivo*, and to characterize any acting mechanisms, especially at low RF field levels. This paper provides a review of the laboratory studies and indicates what conclusions about RF-induced cancer can be drawn. — *Environ Health Perspect* 105(Suppl 6):1565–1568 (1997)

Key words: radiofrequency fields, cancer, health effects, laboratory studies

Introduction

Radiofrequency (RF) fields are part of the electromagnetic spectrum and have many of the same properties as infrared radiation (heat), except that RF fields have a lower frequency. Electromagnetic fields are composed of electric and magnetic waves, and depending on the wavelength of the RF field and distance from the source, measurement of both the electric and magnetic fields may be needed to properly characterize the RF source. RF fields have frequencies between 300 Hz and 300 GHz (*1*). Microwaves (300 MHz–300 GHz) are a subset of the RF spectrum and can be called either RF or microwaves.

Electromagnetic fields of extremely high frequencies (i.e., high energy), such as X-rays, are able to break molecular bonds in cells to produce ionization (positively and negatively charged molecules). The radiant energy component of a field needed to break the weakest macromolecular bond (hydrogen bond) is approximately 0.1 eV; a single covalent bond is 3.6 eV (*2*). RF fields at frequencies between 300 MHz and 300 GHz have radiant energies between 1.24 and 1240 eV—hundreds of times less energy than is needed to break molecular bonds and cause ionization. This is why RF fields are called nonionizing radiation (NIR).

The intensity (or power density) of RF fields is measured in watts per square meter (W/m^2) or in microwatts per square meter (W/m^2) for very weak fields. The units and symbols used are as follows: 1 hertz (Hz) = 1 cycle per second; 1 megahertz (MHz) = 1 million Hz; and 1 gigahertz (GHz) = 1 thousand MHz. 1 W/m^2 = 1,000,000 W/m^2. The specific absorption rate (SAR) is a measure of the rate of RF energy absorption within tissue. The unit for SAR is watts per kilogram (W/kg).

Reviews of the epidemiologic studies on both occupational and general public RF exposure have concluded that there is no consistent evidence of a carcinogenic hazard (*1,3–6*). Inconsistencies among recent studies have not shed any further light (*7–13*). Overall, epidemiologic studies suffer from inadequate assessment of exposure and confounding, and poor methodology (*5,14*). Further epidemiologic studies are needed, particularly to investigate populations of mobile telephone users, among which accurate RF exposure assessments are now possible.

Results from current laboratory investigations of biological effects of exposure to RF fields are consistent with responses to induced heating, which causes a rise in tissue or body temperature of greater than 1 °C (*1*). Most studies have examined end points other than cancer, such as physiologic and thermoregulatory responses, effects on behavior, induction of lens opacities (cataracts), and adverse reproductive outcomes following acute exposure to relatively high levels of RF fields. Very few studies are relevant to the evaluation of low-level RF exposure and its effects on the development of cancer in humans.

This paper reviews the published laboratory studies, both *in vitro* and *in vivo*, that relate to cancer causation from low-level RF field exposure in various biological systems and draws conclusions about possible cancer risk.

Laboratory Studies

The laboratory studies dexcribed in this paper fall into two categories:

a) In vitro studies are conducted on isolated components of biological systems such as solutions of molecules (e.g., DNA), cultures of cells, or pieces of tissue. These studies are important for determining possible mechanisms by which RF fields interact with biological systems and for identifying appropriate end points and exposure conditions to be tested in whole animals. It is important to determine mechanisms of interaction in order to understand how RF fields act at the molecular or cellular level and extrapolate *in vitro* results to the *in vivo* level. Studying simple systems allows interactions to be detected that may be masked in the complexity of interactions that occur normally within the whole animal. Thus, biological effects found *in vitro* must still be tested *in vivo*.

This paper is based on a presentation at the International Conference on Radiation and Health held 3–7 November 1996 in Beer Sheva, Israel. Abstracts of these papers were previously published in *Public Health Reviews* 24(3–4):205–431 (1996). Manuscript received at *EHP* 7 April 1997; accepted 27 June 1997.

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Abbreviations: CW, continuous wave; NIR, nonionizing radiations; ODC, ornithine decarboxylase; RF, radiofrequency; SAR, specific absorption rate; TPA, 12-*O*-tetradecanoylphorbol-13-acetate.

b) In vivo studies are conducted on complete biological systems such as laboratory animals. The great advantage of these studies is that they are conducted under laboratory conditions with environmental and exposure parameters that can be carefully controlled. The only difference between exposed and unexposed animals should be their actual exposure to RF fields. Because experiments cannot normally be conducted on humans, animal studies are very useful for making health risk assessments. However, the results of animal studies are only applicable to humans if the observed effects occur in various types of animals; one animal model may be extremely sensitive to a particular end point and display reactions not observed in humans (*15,16*).

Cancer

In considering the laboratory evidence, each component in the carcinogenesis process is addressed: initiation, promotion, and progression.

Initiation Studies

Most *in vitro* studies have reported a lack of RF-induced DNA damage [for reviews, see (*1,5,6,14,17*)]. A lack of effect from RF exposure has also been reported on mutation frequency in yeast (*18,19*) on mouse leukemia cells (*20*), or on chromosome aberration frequency in human lymphocytes (*21,22*).

However, two rodent studies suggest that RF fields may affect DNA directly. When mice were exposed to 2.45-GHz fields at an SAR of 1.18 W/kg for 2 hr/day for 120, 150, and 200 days, structural genomic rearrangements were found in brain and testes cells (*23*). Lai and Singh (*24,25*) reported that rats exposed to pulsed (2 sec pulses, 500 pulses/sec) or continuous wave (CW) 2.45-GHz fields with SARs of 0.6 or 1.2 W/kg for 2 hr increased the number of single and double-strand breaks in brain cell DNA when assayed 4 hr after RF exposure. Lai and Singh (*26*) also reported that treatment of rats immediately before or after exposure with either melatonin (1 mg/kg) or PBN (*N-tert*-butlylphenylnitrone, 100 mg/kg) blocks the formation of DNA breaks by RF fields. These experiments challenge the belief that RF fields are unable to break molecular bonds. Epigenetic events may be involved; therefore, it is important to replicate these studies and determine dose–response relationships before they can be used in health risk assessments, especially given the weight of evidence suggesting that RF fields are not genotoxic.

Promotion and Progression Studies

Studies have tested the possibility that RF fields may influence tumor promotion through increases in the rate of cell proliferation, or via effects mediated through changes in proliferative signaling pathways, leading to enhanced transcription and DNA synthesis, with conflicting results (*27,28*). Ion fluxes through the cell membrane constitute important signaling mechanisms. A number of reports suggest that RF fields may be capable of affecting ion fluxes via effects on ion pumps such as Na^+K^+-ATPase in human red blood cells exposed to RF and microwave radiation (*29,30*).

Athermal effects on gross transcription, as measured by incorporation of the specific RNA precursor ^{3}H-uridine, have been reported following the exposure of glioma cells to CW RF and microwave radiation (*31*). Similar effects on cellular proliferation, assayed as the incorporation of the specific DNA precursor ^{3}H-thymidine, were also reported following exposure of human lymphocytes (*32*) or glioma cells (*31*). Both transcription and proliferation were elevated at an SAR of 25 W/kg but appeared to be unchanged or even depressed at higher SARs. RF exposure has also been reported to induce activity of the enzyme ornithine decarboxylase (ODC), levels of which are often elevated during cell growth and tumor promotion. The exposure of mouse fibroblasts to amplitude-modulated microwaves at an SAR of 3 W/kg increased ODC activity (*33*), but to a much lower level than treatment with a chemical promoter. It should be noted, however, that changes in the level of this enzyme are not necessarily indicative of tumor promotion (*34*).

Assays of cell transformation are used to detect changes consistent with tumorigenesis but do not provide information on the nature of the damage giving rise to the change. An increased rate of *in vitro* transformation has been reported (*35–37*) in a chromosomally abnormal cell line. Enhanced transformation rates were found in $C3H10T_{1/2}$ cells exposed to combined amplitude-modulated microwaves (4.4 W/kg) and X-rays followed by treatment with the chemical promoter 12-*O*-tetradecanoylphorbol-13-acetate (TPA), compared with cells exposed only to X-rays and TPA (*35*). Similar levels of enhanced transformation rates were found after exposure to microwaves and/or X-rays (1.5 Gy), followed by treatment with the promoter (*36*). However, there are inconsistencies between these two studies. In the first study, microwave exposure resulted in a 50% reduction in plating efficiency, whereas in the second study no such effect was observed. Further, although the data from the second study were consistent with an additive effect of microwaves and X-rays when followed by TPA treatment, unlike the first study, this effect was not statistically significant.

Balcer-Kubiczek and Harrison (*37*) also reported that exposure to microwaves at SARs between 0.1 and 4.4 W/kg, followed by TPA treatment, resulted in a dose-dependent induction of transformation. In addition, microwave exposure slightly enhanced the effects of X-irradiation and TPA on transformation rate. These studies are important, but their relationship to carcinogenesis *in vivo* is unclear. $C3H10T_{1/2}$ cells are highly abnormal chromosomally and their response to proliferative stimuli may be atypical. In addition, transformation studies tend to be susceptible to a variety of experimental confounding factors (*4*).

Szmigielski et al. (*38*) and Szudzinski et al. (*39*) report that chronic exposure of mice to RF (2.45 GHz, 2–3 or 6–8 W/kg, 2 hr/day, 6 days/week for up to 12 months; 40 or 100 animals/group) accelerated the development of sarcoma colonies in the lung after subcutaneous injection of sarcoma cells; mammary tumors in mice having a normally high incidence of these tumors; and skin tumors that were chemically induced in mice by painting the skin with the carcinogen 3,4-benzo[*a*]pyrene. Szmigielski et al. (*40*) also reported that exposure to RF fields (2.45 GHz CW, 4–5 W/kg, 2 hr/day, 5–6 days/week for a few months; 40 animals/group) increased the number of chemically induced hepatomas and sarcomas and increased the number of skin tumors in mice given a subcarcinogenic dose of benzo[*a*]pyrene. The authors suggested that the acceleration of tumor development may have resulted from a direct effect on immunocompetent cells. However, the possibility that heating and cage stress may have influenced the progress of these tumors cannot be dismissed.

In contrast to the reports of Szmigielski et al. and Szudzinski and co-workers, Salford et al. (*41*) report no effect on the progression of tumors cells injected into rat brain after exposure to continuous or pulsed 915-MHz RF fields (CW exposure,

1 W/pulse, 1.67 W/kg; 0.41 W/kg for 217 Hz modulation, pulse width 0.52 msec, 2 W/pulse; 62 animals/group). Santini et al. (*42*) found that the progression of subcutaneously implanted melanoma cells in mice was unaffected by daily exposure to pulsed or CW exposure to RF fields (2.45 GHz CW and pulsed, 10 W/m^2, 1.2 W/kg, 2.5 hr/day, 6 days/week, for the lifetime of the animal). Also, Wu et al. (*43*) report no effect of RF exposure (2.45 GHz exposed 3 hr/day, 6 days/week for 5 months; average SAR 10–12 W/kg) on chemically induced colon cancer in mice. Rotkovska et al. (*44*) found that exposure of mice to low-level RF fields from a police radar (34 GHz) did not affect biological parameters that could initiate any pathologic process.

One hundred rats were exposed to RF fields (2.45 GHz, 800 pulses/sec, 10-sec pulse width, average SAR 0.15–0.4 W/kg, depending on the size of the rat, 21.5 hr/day for 25 months) and compared with 100 sham-exposed controls (*45*). Tests of more than 155 parameters were negative for effects on general health, longevity, cause of death, or lesions associated with aging or benign neoplasms. The authors reported that no single type of malignant tumor was enhanced by exposure. The incidence of primary malignancies in the exposed group was significantly higher than in controls, but was similar to the levels of primary malignancies reported elsewhere in this strain of rat.

Repacholi et al. (*46*) report a 2.4-fold increase in the incidence of lymphomas in *Eμ-pim1* transgenic mice exposed for up to 18 months to 900-MHz fields pulsed at 217 Hz with pulse widths of 0.6 sec. The time to lymphoma development was also shorter in the RF-exposed animals. This is the first and only study using transgenic animals exposed to RF fields. Transgenic animals provide a very sensitive model for the end point to be tested. If the study had produced no RF-induced increase in lymphoma, it would have provided substantial evidence that RF fields do not influence lymphomagenesis. Fewer transgenic than normal animals are needed to detect a given effect promoted by RF fields. It should be remembered that the mice were genetically engineered for a predisposition to lymphoma by insertion of extra *pim1* oncogenes into the DNA. Thus the relevance of results found in a very sensitive animal model to possible carcinogenesis in humans still needs to be determined. However, since the only difference between transgenic and normal mice is the insertion of the oncogene, this study could provide an *in vivo* model for determining how RF fields interact at the molecular level. Further research is needed to replicate and extend the results to determine if there is a dose–response relationship and to determine the applicability of results found in transgenic mice to possible effects in humans.

Overall, the evidence suggests that RF exposure is not mutagenic and is therefore unlikely to initiate cancers. The evidence for a co-carcinogenic effect or an effect on tumor promotion or progression is suggestive but not substantive. The few studies conducted to date are sufficiently indicative to merit further investigation.

Amplitude-modulated Radiofrequency Effects

Several research groups have noted effects from exposure to very low levels of amplitude-modulated RF fields. Such exposure, at levels too low to involve heating, reportedly altered electrical activity of the brain in cats and rabbits; activity of the enzyme ODC, levels of which may be elevated during tumor promotion; and calcium ion mobility in brain tissue *in vivo* and *in vitro* (*1,34*). Effective SARs *in vitro* were less than 0.01 W/kg, occurring within modulation frequency windows (usually between 1 and 100 Hz,) and sometimes within power density windows. These changes in calcium ion mobility have not been easy to corroborate and challenge the conventional assumption that the likelihood or severity of an effect increases as a function of dose. Further, they are not sufficiently well established nor are their implications for human health understood (*14*).

Conclusions

Reviewers evaluating possible links between RF exposure and excess risk of cancer have concluded that there is no clear evidence for such a link (*1,3–6,14*). The United Kingdom NRPB Advisory Group on Non-ionising Radiation concluded that there is no firm quantitative evidence of a carcinogenic hazard from electromagnetic field exposures for the general public and workers in the electrical, electronics, and telecommunications industries (*4*). Studies published since this review was completed have not shed any further light on the possibility that RF exposure poses a carcinogenic risk. After a detailed review of the scientific literature, the International Commision on Non-ionizing Radiation Protection (ICNIRP) (*5*) concluded that "there is no substantive evidence that adverse health effects, including cancer, can occur in people exposed to levels at or below the limits on whole body average SAR recommended by INIRC (*47*), or, at or below the ICNIRP limits for localised SAR...."

Few studies have been conducted that specifically address the issue of cancer and RF exposure. More multidisciplinary focused studies are needed to address this. This is one of the goals of the International Electromagnetic Fields Project (*48*). The first scientific review meeting to reach interim conclusions on health risk and identify research needs, was held in Munich in November 1996. The results of this review will be available shortly (*14*). The conclusion was that "although hazards from exposure to high-level (thermal) RF fields were established, no known health hazards were associated with exposure to RF sources emitting fields too low to cause a significant temperature rise in tissue. Biological effects from low-level RF exposure were identified needing replication and further study." High priority research needs were epidemiology and animal carcinogenesis studies and *in vitro* studies identifying effects that can be tested *in vivo*.

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