A Critical Review of Epidemiologic Studies of Radiofrequency Exposure and Human Cancers

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This paper reviews studies that have assessed associations between likely exposure to radiofrequency (RF) transmissions and various types of human cancer. These studies include three cluster investigations and five studies relating to general populations; all of these studies consider place of residence at the time of cancer diagnosis in regard to proximity to radio or television transmitters. There are also five relevant occupational cohort studies and several case–control studies of particular types of cancer. These studies assessed a large number of possible associations. Several positive associations suggesting an increased risk of some types of cancer in those who may have had greater exposure to RF emissions have been reported. However, the results are inconsistent: there is no type of cancer that has been consistently associated with RF exposures. The epidemiologic evidence falls short of the strength and consistency of evidence that is required to come to a reasonable conclusion that RF emissions are a likely cause of one or more types of human cancer. The evidence is weak in regard to its inconsistency, the design of the studies, the lack of detail on actual exposures, and the limitations of the studies in their ability to deal with other likely relevant factors. In some studies there may be biases in the data used. — *Environ Health Perspect* 107(Suppl 1):155–168 (1999). *http://ehpnet1.niehs.nih.gov/docs/1999/Suppl-1/155-168elwood/abstract.html*

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Radiofrequency radiation (RFR) occupies the range from 100 kHz to 300 GHz in the electromagnetic spectrum. RFR is a higher frequency (shorter wavelength) than the extremely low frequency (ELF) radiation used in electric power sources, and a lower frequency than infrared radiation. This range is used for radiofrequency (RF) communications and microwave sources, including in approximate order by increasing frequency, amplitude modulation (AM) radio, frequency modulation (FM) radio, very high frequency (VHF) radio and television (TV), ultrahigh frequency (UHF) TV and cellular telephone transmissions, and microwave ovens, radar, and satellite communications. Natural RFR is negligible, so population exposure is a phenomenon of the current century, and has increased greatly in recent years. Public concern tends to focus on new types of emitters, and there have been public concerns about radio and TV transmitters, TV receivers, and microwave ovens coincident with their development and utilization.

Recently in several countries there has been considerable public concern and legal proceedings about cellular telephone systems (cell phones) in regard to potential risks both to users of cell phones and from population exposure to cell phone transmitters. Although cell phone users have a much higher potential dose exposure because the device is held close to the head (*1,2*), public concern has often been greater regarding cell phone transmitters, where although the potential dose exposure is much less, the exposure is seen as involuntary; health concerns may also be raised as a support for aesthetic and other objections.

International guidelines for RF exposures have recently been revised by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) (*3*). These guidelines for body exposure were set on the basis of avoiding thermal effects. The basic restriction for whole-body exposure is a specific energy absorption rate (SAR) of 0.4 W/kg for occupational exposure and 0.08 W/kg for general population exposure. The ICNIRP review of human and animal data for RF ranges shows that the threshold for irreversible effects in even the most sensitive tissues is > 4 W/kg under normal environmental conditions (*3*). As a result, the occupational exposure restriction is based on a safety factor of 10, and the general population basic restriction on a further reduction factor of 5, resulting in 0.08 W/kg. These reference levels in equivalent power densities are 200 $\mu W/cm^2$ at 10 to 400 MHz; $f/2$ $\mu W/cm^2$ at 400 to 2000 MHz, where f = frequency in MHz; and 1000 $\mu W/cm^2$ at 2 to 300 GHz. Some national standards hold to 200 $\mu W/cm^2$ throughout this range. Power outputs from cell phone transmitters are low and exposure levels decline with the square of distance from the source, so that even at distances as short as 30 m, exposure levels are likely to be < 5% of the public exposure limit. Thus, interest in potential human effects from cell phone transmitter exposures depends on the existence of relevant biologic effects at levels much below those producing thermal effects—so-called athermal effects. Higher limits were set for exposure of smaller body parts: the occupational localized SAR limit for exposure of the head was set at 10 W/kg, averaged over any 10-g mass of tissue and over any 6-min period; the general public exposure limit for the head is 2 W/kg averaged similarly. These levels are relevant to cell phone users.

The ICNIRP report (*3*), which also reviewed several other expert reports (*1,4–9*), concluded that exposure to these fields is therefore unlikely to initiate carcinogenesis. These expert reports note many negative results from *in vitro* studies on DNA damage, mutation frequency, and chromosome aberration frequency. In addition there are data suggesting biologic effects, some of which are potentially relevant to cancer causation, at low exposure levels. Strand breaks in DNA in rodent tissues have been described at SAR levels around 1 W/kg (*10–12*), although the methodology of these studies has been questioned (*13*). Excesses of malignancies have been noted in rats exposed to microwaves (*14*). Some studies suggested promotion effects on preinitiated cells (*15*)

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Abbreviations used: ALL, acute lymphoblastic lymphoma; ELF, extremely low frequency; EMF, electromagnetic field; FM, frequency modulation; ICNIRP, International Commission on Non-Ionizing Radiation Protection; LGAs, local government areas; OR, odds ratio; PEMFs, pulsed electromagnetic fields; RF, radiofrequency; RFR, radiofrequency radiation; RR, relative risk; SAR, specific energy absorption rate; TV, television; UHF, ultrahigh frequency; VHF, very high frequency.

but did not clearly exclude thermal effects, whereas other studies at athermal levels reported no effects (*16,17*). An important recent study showed an increase (RR = 2.4, 95% confidence limits 1.3–4.5) in lymphoma incidence in genetically predisposed mice compared to controls at dosage levels of pulsed 900 MHz fields, which for most animals would have been below thermal levels (*18*).

There is evidence of effects of athermal levels of RFs on calcium release or surface binding in cells (*19,20*)—with some negative reports (*21*)—and on brain electrical activity (*19*), T-lymphocyte cytotoxic activity (*22*), changes in non-cyclic-AMP-dependent kinases (*23*), and on orthonine decarboxylase activity (*24,25*), although there are also many negative reports (*3*). Increased neoplastic transformation in cells also treated with a chemical promoter has been noted (*26*).

Thus there are various experimental results that are consistent with biologic effects, some of which might be related to carcinogenic mechanisms, of RFs at strengths below those that produce thermal effects. However, the results are inconsistent and no clear mechanism has been shown consistently in a variety of cell systems and animals. As a result the consideration of the epidemiologic literature relating RFs to cancer occurrence in humans must be balanced against this uncertain background: There is no clear evidence for a carcinogenic action relevant to intact humans, but similarly, it is difficult to argue that a carcinogenic mechanism can be ruled out.

Neither the ICNIRP (*3*) nor any earlier major reports discussed epidemiologic studies in any detail. The most interesting epidemiologic studies of general population RF exposure have been published recently and were not included in the epidemiologic review by Bergqvist (*27*). Therefore, although the ICNIRP and earlier reports concluded that there was no good evidence for an epidemiologic association, continuing public concern and the recent epidemiologic studies stimulated the current review.

Methods

Published reports on studies of RFs and cancer in humans were identified by a literature search using Medline (*28*) to search from 1988 to June 1998, by searching references given by the major reviews (*1,3–9*), and by other studies and reviews. The studies used were limited to studies of individuals or communities classified as likely to have been exposed to RF emissions; studies of ELF radiation or electromagnetic fields (EMFs) in general, where the main exposure was likely to be to ELF, were not included. Nonhuman studies, laboratory studies, individual case reports, and studies without any comparison group were not considered.

Further calculations were needed to obtain results from Robinette et al. (*29*) in a format similar to that of the others: 95% confidence limits of the given observed to expected ratios were calculated, and a trend test was applied using the method of Breslow and Day (*30*).

Results

The epidemiologic studies dealing with RF emissions and human cancer fall into four groups: studies of clusters of cases; studies of general populations exposed to TV, radio, and similar emissions; studies of occupational groups with exposures to such emissions; and case–control studies.

Cluster Studies

A cluster study is based on the unplanned observation of an apparent excess number of cases of a disease in a particular place and time period. The occurrence of clusters of any uncommon disease is an expected phenomenon due to chance variation and small numbers. There is no statistical or other method to determine, once a cluster has occurred, whether or not it is due to chance; investigations of clusters are often unhelpful, and are often done only as a public relations effort to allay community concern (*31*). The appropriate approach to a cluster is to treat it as raising a new hypothesis to be tested in independent data.

Three cluster investigations of cancer and RF radiation have been reported in peer-reviewed literature (Table 1).

A cluster of 12 cases of acute leukemia in children in Hawaii led to a case–control study of a slightly larger number of cases (*32*), which showed an excess among those living within 2.6 miles of radio towers. This was not significant (odds ratio [OR] = 2.0, 95% confidence limits 0.06–8.3). The authors concluded that "the clustering may

Table 1. Radiofrequency emissions and cancers: cluster studies.

	Cluster studies			
	Hawaii (*32*)	U.S. policemen (*33*)	Sutton Coldfield (*34*)	
Study characteristic				
Exposure	Residence within 2.6 miles of radio towers	Use of hand-held radar gun	Residence close to TV or radio transmitters	
Ascertainment	Residence	Self-report	Residence	
Exposed group	General population	U.S. policemen	General population	
Study type	Case–control: 14 cases	Cohort: 6 cases	Small area incidence analysis	
Type of cancer	Acute leukemia, children	Testicular	Various	
Cancer type/OR/test for trend				
All leukemia	—	—	1.83 (1.22–2.74)[a,b,*]	0.001[a,*]
Acute leukemia	2.0 (0.06–8.30)[c]	—	1.86 (0.89–3.42)[a,b]	0.004[a,*]
Acute myeloid leukemia	—	—	1.02 (0.26–2.60)[a,b]	0.045[a,*]
Acute lymphatic leukemia	—	—	3.57 (0.74–10.4)[a,b]	NS[a]
Chronic myeloid leukemia	—	—	1.23 (0.15–4.43)[a,b]	NS[a]
Chronic lymphatic leukemia	—	—	2.56 (1.11–5.05)[a,b,*]	0.007[a,*]
Non-Hodgkin lymphoma	—	—	0.66 (0.28–1.30)[a,b]	NS[a]
Multiple myeloma	—	—	1.54 (0.74–2.83)[a,b]	NS[a]
Testis	—	Only shared risk factor[a]	—	—
All cancer	—	—	1.09 (1.01–1.17)[a,b,*]	NS[a]

NS, nonsignificant, $p > 0.05$; —, no information was given in the study; OR, odds ratio. [a]Adult only. [b]OR within 2 km. [c]Child only. *Statistically significant, $p < 0.05$.

have been a chance event, but because of its particular characteristics, we feel it should be noted" (*32*).

Another cluster investigation followed the observation of what appeared to be a high number of cancers of the testis (six cases) among 340 U.S. policemen who used radar guns and often kept them on their lap in their patrol car (*33*). This study showed a positive association, but because it was based on a cluster situation it cannot be interpreted further.

An appropriate type of cluster investigation is the study of a suspected cluster of leukemias and lymphomas in adults living close to the Sutton Coldfield TV and radio transmitter near Birmingham, UK (*34*). The authors used data over a 12-year period to compare the residential postcode of patients with cancer and the census number of residents in that postcode area (allowing adjustment for age, gender, regional variations within the country, and an index of socioeconomic level).

For the types of cancer suspected on the initial cluster, there was an excess of total adult leukemia within 2 km, with the risk declining from there out to the edge of a 10-km circle. Similar results were seen for some subtypes of leukemia. Part of the trend of decreasing risk from distance from the transmitter was because the observed number of cases in people living close to the 10-km boundary was less than expected. For lymphomas—also part of the initial cluster—there was an excess risk within the 10-km circle, but the risk was less in those within the inner 2-km circle. The authors appropriately concluded that "no causal implications regarding radio and TV transmitters can be drawn from this finding, based as it is on a single cluster investigation" (*34*).

Studies of General Populations Exposed to Radio and Transmitter Television Transmissions

Five studies look at general population groups living close to RF transmitters (Table 2).

Sutton Coldfield Study. In the further analysis of the Sutton Coldfield study, several other types of cancer in adults were assessed—types that were not part of the initial cluster (*33*). Of 11 types of cancer assessed, two showed a significant trend in incidence with distance from the transmitter (melanoma and bladder cancer). Although not affected by the clustering bias, the difficulty here is one of multiple testing; some positive findings will arise by chance. These associations are new observations not based on a prior hypothesis; therefore they require further assessment.

The results for childhood cancer can also be assessed because the original cluster was related to adult cancer. There was no significant trend with distance for all cancer or for leukemia in children.

Study of Twenty-One U.K. Transmitters. Dolk et al. (*35*) then identified all 21 radio and TV transmitters in Britain with transmission power of over 500 kW for TV or 250 kW for FM radio, including Sutton Coldfield. They again assessed an inner circle of 2 km, an outer circle of 10 km of residence, and the trend in risk with distance from the transmitter up to 10 km. They used data for up to 12 years, which included 3300 adult leukemia cases and were based on a total population living within 10 km of any transmitter of 3.39 million people. The associations suggested by the Sutton Coldfield study (with adult leukemia, skin melanoma, and bladder cancer) were tested by a new analysis based only on all the other transmitters, excluding Sutton Coldfield. For childhood cancer, where there was no significant excess in Sutton Coldfield, the data from all 21 transmitters were combined.

The most important aspect of the study was to assess if the increase in adult leukemia seen near the Sutton Coldfield transmitter was also seen near the other transmitters. That was not shown. The total number of cases living within the inner 2-km circle from the other 20 transmitters was 3% less than expected, whereas the number living within the overall 10-km circle was 3% more than expected. The detailed results for all adult leukemias are complex, however. Close to the transmitter the observed-to-expected ratio was < 1, although it was based on a small number of cases. The ratio rose to 1.15 at distances of 2 to 3 km, then gradually decreased to become close to 1 at distances beyond 7.5 km. Because there were few cases close to the transmitter, the statistical test used shows a weak decline in risk due to the risk falling from distances of 2 to 3 km out to the edge of the circle, with borderline significance ($p = 0.05$).

These results do not confirm the findings in Sutton Coldfield. The result is what we would expect if the Sutton Coldfield results were influenced by the fact that that study was based on an initial cluster of cases close to the transmitter. Similarly, for most subtypes of leukemia and lymphoma, the risk in those living closest to the transmitter (within 2 km) was less than expected. Only one type, chronic lymphatic leukemia, showed a pattern of some increased risk closer to the transmitter, but this was nonsignificant.

Results for individual transmitters showed significant declines in risk of adult leukemia with increasing distance for three transmitters; results from two of these transmitters showed that the risks at the edge of the 10-km circle were less than expected. The overall declining trend with distance was mainly due to the data for the Crystal Palace transmitter near London; it had most of the observed cases because of the high population. The Crystal Palace transmitter is a high-powered TV transmitter (> 870 kW) without FM transmission; the other two transmitters showing trends in adult leukemia were combined TV transmitters (500 and 287 kW) plus FM transmissions of 250 kW. The Sutton Coldfield transmitter transmits both TV and FM frequencies. The authors grouped transmitters by whether they carried TV or FM emissions, and by power, but no clear differences emerged (*35*).

The authors suggest three possible explanations of the trend of reducing risk from 2 to 3 km out to 10 km from the transmitters. It may be due to chance or to "other unmeasured sociodemographic or environmental factors" (*35*). Alternatively, if reduced risk is causally related to the transmissions, the usual exposure model on which exposure declines with the square of distance would not explain the results; the authors state: "if there were a true association with radio transmission, the lack of replication of the pattern and magnitude of excesses near Sutton Coldfield may indicate that a simple radial decline exposure model is not sufficient" (*35*). The authors conclude that in general their study is negative, and "at most, gives no more than very weak support to the Sutton Coldfield findings" (*35*).

Of the two other cancer types related to distance in the Sutton Coldfield study, the results for the other 20 transmitters showed no trend with distance for bladder cancer, and only a small irregular variation in melanoma. Leukemias and brain cancers in childhood were also examined using data from all 21 sites. There was no significant increased risk in either disease within the inner 2-km circle or in the overall 10-km circle, and no regular variation with distance.

Study in Sydney, Australia. This study (*36*) assessed cancer incidence and

Table 2. Radiofrequency emissions and cancers: general population studies.

	General population studies							
	Sutton Coldfield (*34*)		U.K. 21 transmitters (*35*)		Sydney 1 (*36,37*)	Sydney 2 (*40*)	Sydney 2 (*40*)	San Francisco (*41*)
Study characteristic								
Exposure	TV, radio		TV, radio		TV	TV	TV	TV, radio
Ascertainment	Residence		Residence		Residence	Residence	Residence	Residence
Frequency, MHz	30–1000		30–1000		60–215	60–215 All areas	60–215 Excluding Lane Cove	—
Cancer type/risk/test for trend								
Adult								
All leukemia	—	—	0.97 (0.78–1.21)[a]	0.052	1.18 (0.98–1.42)[b]	—	—	—
Acute leukemia	—	—	0.94 (0.67–1.31)[a]	NS	—	—	—	—
Acute myeloid leukemia	—	—	0.77 (0.50–1.19)[a]	NS	—	—	—	—
Acute lymphatic leukemia	—	—	0.90 (0.39–2.11)[a]	NS	1.32 (1.09–1.59)[b,*]	—	—	—
Chronic myeloid leukemia	—	—	0.63 (0.30–1.29)[a]	NS	1.09 (0.91–1.32)[b]	—	—	—
Chronic lymphatic leukemia	—	—	1.20 (0.83–1.46)[a]	NS	—	—	—	—
Other leukemia	—	—	—	—	1.67 (1.12–2.49)[b,*]	—	—	—
Adult								
Multiple myeloma	1.54 (0.74–2.83)[a]	NS	—	—	—	—	—	—
Brain cancers	1.31 (0.75–2.29)[a]	NS	—	—	0.89 (0.71–1.11)[b]	—	—	—
Melanoma skin	1.43 (0.83–2.44)[a]	0.018*	1.11 (0.84–1.46)[a]	NS	—	—	—	—
Bladder	1.52 (1.13–2.04)[a,*]	0.04*	1.08 (0.94–1.24)[a]	NS	—	—	—	—
Melanoma eye	0 cases	NS	—	—	—	—	—	—
Male breast	1.64 (0.04–9.13)[a]	NS	—	—	—	—	—	—
Female breast	1.08 (0.90–1.31)[a]	NS	—	—	—	—	—	—
Lung	1.01 (0.84–1.21)[a]	NS	—	—	—	—	—	—
Colorectal	1.13 (0.94–1.35)[a]	NS	—	—	—	—	—	—
Stomach	0.75 (0.54–1.06)[a]	NS	—	—	—	—	—	—
Prostate	1.13 (0.82–1.55)[a]	NS	—	—	—	—	—	—
Child								
All leukemia	—	NS	1.12 (0.61–2.06)[a]	NS	1.58 (1.07–2.34)[b,*]	1.38 (0.99–1.91)[c]	0.90 (0.56–1.44)[c]	0.73 NS[d]
Lymphatic leukemia	—	—	—	—	1.55 (1.00–2.41)[b,*]	—	—	—
Myeloid leukemia	—	—	—	—	1.73 (0.62–4.81)[b]	—	—	—
Other leukemia	—	—	—	—	1.65 (0.33–8.19)[b]	—	—	—
All cancer	1.8[e]	NS	—	—	—	—	—	—
Brain	—	—	0.50 (0.10–1.46)[a]	NS	1.10 (0.59–2.06)[b]	—	—	1.16 NS[d]
Hodgkin	—	—	—	—	—	—	—	1.23 NS[d]
Non-Hodgkin lymphoma	—	—	—	—	—	—	—	1.03 NS[d]
Lymphoma and leukemia	—	—	—	—	—	—	—	0.89 NS[d]
Acute lymphatic leukemia	—	—	—	—	—	1.45 (0.96–2.19)[b]	0.83 (0.45–1.55)[c]	—

—, No information was given in the study; NS, not significant; RR, relative risk. [a]OR within 2 km. [b]RR: high to low exposure areas. [c]RR: regression, for 1 μW/cm^2 exposure change. [d]RR for residence within 3.6 km of tower. [e]Confidence limits are not given. *Statistically significant, $p<0.05$.

mortality around three TV transmitters in northern Sydney. There was no suggestion of a cluster of disease. The three towers emit TV transmissions in the range of 60 to 215 MHz with one additional channel at approximately 500 MHz. The maximum power density was estimated as 8 μW/cm^2 at approximately 1 km from the towers, reducing to 0.2 μW/cm^2 at 4 km; estimates at 8 km were approximately 0.05 μW/cm^2 and were substantially less at 12 km. The analysis compared an inner area within 4 km with an outer area that extended from approximately 4 km to 15 km away. Incidence and mortality data from leukemia in total, by subtype, and from brain tumors were collected from 1972 to 1990. These data were subdivided by gender and by broad age groups.

For adult cancers (15+ years of age) (*37*) there was a small nonsignificant increase in the incidence of total adult leukemia (RR = 1.18, 95% confidence limits 0.98–1.42) and no significant increase in leukemia mortality.

There was an increased incidence of childhood leukemia (RR = 1.58, 95% confidence limits 1.1–2.3, based on 134 cases) (*36*). This excess was seen separately for lymphatic, myeloid, and other leukemias in childhood, although the latter two are based on small numbers and are not significant. The mortality rates for leukemia in children were increased, more so than for incidence (RR = 2.3, limits 1.4–4.0). There was no excess of brain tumors in adults or children.

Hocking et al. (*36*) comment that control for other factors was limited. They claim that the socioeconomic distribution of the inner and outer areas is generally similar. The broad age range used raises the possibility of age confounding; a younger age distribution within the group 0 to 15 years of age would confer a higher leukemia risk. They point out that the inner area has higher traffic density, which could be related to increased benzene exposure; the inner area also has higher population density, but they note there is a hypothesis that leukemia in children might be increased in sparsely populated areas into which many people have recently moved (*38*). Hocking et al. (*36*) suggest that the increase could be related to the modulations at 50 Hz to 5 MHz as well as to the carrier wave.

The Hocking et al. (*36*) study is considerably weaker than the British studies (*34,35*): It deals only with one area. It makes only one comparison—between areas defined at one arbitrary distance from the transmitter—rather than exploring the overall relationship with place of residence.

There is little control for other potentially relevant factors. The results, showing a stronger association for childhood leukemia than for adult leukemia, contrast with the British data, which show no clear excess in children. The authors conclude "more detailed studies (e.g., relating cases to power density contours) are required to replicate any association and to look for dose–response relationships before any conclusions can be drawn" (*36*).

Comparisons between U.K. and Sydney Studies. The authors of the Sydney study (*37*) have commented that their study found an excess of childhood leukemia, which was not found in the UK study. Hocking et al. (*37*) point out that the UK transmitters were 3 to 10 times more powerful than those used in Australia but generally used UHF (430–890 MHz), whereas the Sydney transmitters used VHF (63–217 MHz). Although it would be expected that any effect should be greater in the UK, Hocking et al. (*37*) hypothesize a window effect, where biologic effects might show a complex relationship with strength of exposure.

In reply, the principal author of the UK study notes that "our results around multiple transmitters were more equivocal than is reflected in the letter by Hocking et al." (*39*). She emphasizes that the UK study found no excess of leukemia in those living closest to the transmitters, and although they found some decline in incidence with increasing distance, they could not distinguish whether this was associated with either TV or FM transmission. They found a weak trend near the Crystal Palace transmitter, which does not transmit FM, as well as near FM transmitters. In regard to the window effect, Dolk (*39*) queries the suggestion of Hocking et al. (*37*) and comments that "when assessing the evidence from just two studies, it is easy to make the theories fit the data post hoc and very premature to conclude that the two studies 'suggest' such effects" (*39*).

Further Analysis of Leukemia Rates in North Sydney in Children and Likely Exposure to Radiofrequency Emissions from Transmitters. A further analysis of the data from North Sydney has been published (*40*). McKenzie et al. (*40*) point out that Hocking et al. (*36*) compared an inner group of three aggregated local government areas (LGAs) close to the transmission tower with an outer ring of aggregated LGAs. Hocking et al. (*36*) excluded on socioeconomic grounds some other LGAs also close to the towers, although they stated that there was little evidence of a relationship between socioeconomic levels and acute lymphoblastic leukemia (ALL) in New South Wales, Australia. Therefore, the new study examined 16 LGAs: 3 closest to the towers (Willoughby, Lane Cove, and North Sydney), as used by Hocking et al. (*36*), 7 in North Sydney, and 6 in other areas of Sydney; the distances of these LGAs are comparable to the outer ring used in the Hocking et al. study. This study and the study of Hocking and his co-workers used data collected from 1972 to 1990.

McKenzie et al. (*40*) calculated the signal strengths by a formula dependent on the distance from the tower and the angle of depression from the horizontal, and also carried out site measurements using a Holaday Instruments broad band isotropic field strength meter. The calculated signal strengths showed reasonable correlation for free space conditions at roof height, with other sites subject to shadowing having lower observed strengths, and two points near the base of the antennae having greater emissions than calculated. Average RFR exposure levels for the LGAs are presented based on calculated signal strengths for a random sample of 20 residences in the high signal strength areas, and at other distances by an average of fewer measurements. Signal strengths in the three closest LGAs were 1.46 $\mu W/cm^2$ in Lane Cove, 1.40 $\mu W/cm^2$ in Willoughby, and 0.66 $\mu W/cm^2$ in North Sydney. Most other areas showed average strengths of < 0.25 $\mu W/cm^2$, apart from Hunters Hill, which showed 0.46 $\mu W/cm^2$. Maximum levels in streets immediately below the antennae were up to 100 $\mu W/cm^2$.

The incidence rate of ALL was highest in Lane Cove, a high exposure area, but the rates in North Sydney and Willoughby, the other two high exposure areas, were similar to those for the less-exposed areas. The regression trend for ALL incidence against estimated RFR exposure based on all LGAs gave a relative risk of 1.45 (95% confidence limits 0.96–2.19) for a change in RFR exposure of 1 $\mu W/cm^2$; that is, an almost significant positive association. For total childhood leukemia the result was similar, with a relative risk of 1.38 (95% confidence limits 0.99–1.91). However, the exclusion of Lane Cove removed this trend, giving relative risks of 0.83 (95% confidence limits 0.45–1.55) for ALL and 0.90 (95% confidence limits 0.56–1.44) for total leukemia.

McKenzie et al. (*40*) concluded that leukemia rates vary in different localities in Sydney, and in particular, the leukemia incidence in Lane Cove was abnormally high during the years studied. This is unlikely to be due to RFR exposure because the other two areas with high RFR exposures, Willoughby and North Sydney, did not show any increase in leukemia incidence. The relationship reported by Hocking et al. (*36*) depends on this excess in Lane Cove. The excess in Lane Cove must be due either to chance or to some factor apart from RF radiation. For the excess to be related to RFR exposure, actual exposures of children who live in Lane Cove would have to be much higher than those estimated; correspondingly, actual exposures in Willoughby would have to be much lower than those calculated. The authors suggest that further studies are needed to examine the excess rates in Lane Cove (*40*).

The second North Sydney study (*40*) is an improvement on the first North Sydney study by Hocking and colleagues (*36*) because the extra detail gives greater ability to assess a regular association between leukemia occurrence and estimated RF exposures. The estimation of exposure is still based primarily on distance, although the measurements performed confirm that the three areas closest to the transmitters have higher exposure levels when measured at a few open air sites than do the other areas. The second study (*40*) still shows an association between leukemia incidence and estimated RFR exposure if all areas are included. However, of three areas with high RFR exposure levels, only one of these three has an elevated risk. This makes it more likely that some factor other than RFR is responsible for the excess.

San Francisco Study. Another study of general population exposures assessed cancers in those younger than 21 years of age in San Francisco between 1973 and 1988 (*41*). This study assessed leukemia, lymphatic cancer, and brain cancer by examining the distribution of cases in terms of residence in relationship to the Sutro Tower, a large tower that carries TV and radio transmitters. No information on emission levels is included. Adjustments for population density in small areas were made. The results showed no evidence that cancers were more common in those who lived close to the TV and radio emissions tower. The risk ratio for residence within 3.5 km was 0.73 for all leukemia ($p = 0.86$).

Honolulu Study. A further study noted by Dolk et al. (*35*) was an unpublished

report. It showed an increase in adult leukemias in areas of Honolulu with broadcasting antennae, but Dolk et al. (*35*) comment that "interpretation of that study was complicated both by the ecologic nature of the design and the problem of potential confounding."

Studies of Occupational Groups

The third set of studies applies to occupational exposures (Table 3). The results are complex because in cohort studies all types of cancer can be assessed and the categorization of cancer type varies greatly.

Polish Military Study. This cohort study (*42*) assessed military personnel in Poland from 1971 to 1985, and used military records to divide subjects into those likely to have been exposed to RFs or microwaves and those not exposed. It is estimated that in the great majority of the exposed situations, the fields were pulse modulated emissions at 150 to 3500 MHz and were less than 2 $\mu W/cm^2$, with a minority of exposures at higher levels.

An essential characteristic of a cohort study is that the information sources used to document the exposure are identical for subjects who develop the outcome of interest (cancer) and for those who do not. This information was collected from the records of central and regional Polish military hospitals and the central military medical board, and it is stated that information on exposure to possible carcinogenic factors was collected from these sources as well as from the service occupational records (*42*). Thus a serviceman who developed cancer had more sources of information on possible RF exposures compared to a serviceman who did not develop cancer. This raises the possibility of systematic bias; such a bias would be expected to produce an increased relative risk for all types of cancers.

The results show an excess of all cancers (RR = 2.1, 95% confidence limits 1.1–3.6). For all leukemias and lymphomas, there were 24 cases observed compared to approximately 3.8 expected, giving a risk ratio of 6.3 (95% confidence limits 3.1–14.3). There were also considerable excesses of cancers of the esophagus, stomach, colon, and rectum. These cancer types, unlike the leukemias and lymphomas, have rarely been implicated in connection with nonionizing radiation exposure.

The overall doubling of total cancer rates is inconsistent with other reports. It raises the question of whether cancer occurrence is better recorded in those who were exposed, perhaps because of variations in their military rank, length of service, or type of posting. Szmigielski (*42*) states that "it is not possible to offer a reasonable explanation for the 3-fold increase in the rate of stomach and colorectal adenocarcinoma" and that "surprisingly there was no difference seen in the most common cancer, lung cancer." No information is presented on cancer mortality. Although mortality may be sometimes of less interest as it is affected by treatment, it has the advantage of often being more reliably recorded than cancer incidence. In this situation mortality information would be a useful addition to the study.

U.S. Navy Study. The Polish study results (*42*) contrast with those of an earlier cohort study of U.S. naval personnel, in which 20,000 men with maximum opportunity for exposure to radar emissions were identified and compared to a similar number of 20,000 subjects with a lower potential for exposure (*29*). Those with maximum exposure opportunity were involved in electronic equipment repair; those with lower potential exposure were involved in equipment operation. All subjects had graduated from U.S. Navy technical schools between 1950 and 1954, and had served on U.S. Navy ships at the time of the Korean war. As with the Polish study, exposure was based on service records. The outcome was cancer deaths, ascertained up to 1974. Mortality may be a less sensitive indicator of a hazard but is more likely to be consistently recorded. The extent of potential exposure was assessed in two ways: by job category and by hazard number—a measure of potential exposure based on a review of individual records for all men who died from disease and for a 5% sample of the others.

Confidence limits and a test of trend over categories of exposure have been applied to the Robinette et al. (*29*) published data because the original did not present such measures (see "Methods"). The total mortality rate and total cancer mortality rate were nonsignificantly increased in the highest exposure group. For cancer of the respiratory tract, the added analysis shows a significantly increased risk in the highest exposure group defined by hazard number (RR = 2.2, 95% confidence limits 1.1–4.1), although a test of trend over the four exposure groups specified is not significant (chi-square 3.1, $p > 0.07$) and the analysis by job category shows no significant excesses. No significant excesses were seen for cancers of the digestive organs, leukemias and lymphomas, or other cancers. Other causes of death including heart disease and other diseases were also assessed and no increases were seen. Total admission rates to Navy hospitals up to 1959 were assessed in addition to deaths, which gives some measure of cancer incidence. Total cancer admissions were slightly lower in the high exposure group.

Robinette et al. (*29*) conclude that "the results demonstrate that in a large group of men, many of whom may have received substantial exposures, any health effects which occurred were insufficient to be clearly perceptible at the level of mortality or hospital morbidity at the time of exposure." One weakness of this study is that it compares two groups with high and low levels of exposure, respectively; whether the mortality rates in the low-exposure group are different from those of an unexposed group is unknown.

Study of U.S. Amateur Radio Operators. In a study in the United States, Milham (*43*) identified radio operators in Washington State and California from 1979 to 1984 and linked their names with death records up to 1984. Radio operators were identified from federal amateur radio operator licenses. The study was restricted to men, although women were excluded only on the basis of name because no gender information was available. No information on other exposures was available, although the author noted that these subjects were likely to be exposed to electric shock, soldering fumes, and degreasing agents. The occupation (as given on the death certificate) was an electrically related job for 31% of the Washington State group, so exposures to electric power frequencies were also likely. The results showed significantly lower death rates than expected from all causes and from all cancers. There was a significantly increased risk of one of nine types of leukemia reviewed (acute myeloid leukemia) and also of cancers of other lymphatic tissue. There were statistically significant reductions in death rates from cancer of the respiratory system and of the pancreas. The lack of information on other relevant exposures limits any firm interpretation of this study.

Study of Breast Cancers in Norwegian Female Radio and Telegraph Operators. Cancer occurrence was studied in a group of 2600 Norwegian female radio and telegraph operators who were certified to work as radio and telegraph operators between 1920 and 1980 and who worked on merchant ships at sea (*44*).

Table 3. Radiofrequency emissions and cancers: occupational studies and study of amateur radio operators.

	Occupational/amateur radio operator studies				
	Polish military (*42*)	U.S. Navy (*29*)	U.S. amateur radio operators (*43*)	Norwegian female ship radio operators (*44*)	Canada/France electric utility workers (*46*)
Study characteristics					
Exposure	RF/microwave	Microwave (radar)	Radio operation	Radio operation	PEMF
Ascertainment	Service records	Service records	Radio operators license records	Employment records	Employment records and field measurements
Exposed group	Military, male	Military, male	Amateur radio operators, male	Radio operators on ships, female	Electric utility workers
Frequency, MHz	150–3500	—	—	0.4–25	5–20, possibly up to 300
Outcome data	Incidence	Mortality	Mortality	Incidence	Incidence
Exposed group for RR values	—	Hazard no. 5001 +	—	—	≥90th percentile
Outcome					
Total deaths, all causes	—	1.23 (0.98–1.52)[a]	0.71 (0.69–0.74)*	—	—
All cancer	2.07 (1.12–3.58)*	1.44 (0.96–2.07)[a]	0.89 (0.82–0.95)*	1.2 (1.0–1.4)*	1.39 (1.05–1.85)*
Lymphatic and hematopoetic	6.31 (3.12–14.32)*	1.64 (0.70–3.25)[a]	1.23 (0.99–1.52)	—	0.96 (0.48–1.90)
All leukemia	—	—	1.24 (0.87–1.72)	1.1 (0.1–4.1)	0.80 (0.19–3.36)
Acute myeloid leukemia	8.62 (3.54–13.67)*	—	1.76 (1.03–2.85)*	—	1.02 (0.08–13.04)
Acute lymphatic leukemia	5.75 (1.22–18.16)*	—	1.20 (0.26–3.81)	—	—
Acute nonlymphoid	—	—	—	—	1.91 (0.19–19.39)
Chronic myeloid leukemia	13.90 (6.72–22.12)*	—	0.86 (0.17–2.50)	—	—
Chronic lymphatic leukemia	3.68 (1.45–5.18)*	—	1.09 (0.40–2.38)	—	2.98 (0.21–41.57)
Hodgkin disease	2.96 (1.32–4.37)*	—	1.23 (0.40–2.88)	—	1.33 (0.23–7.68)
Non-Hodgkin lymphoma	5.82 (2.11–9.74)*	—	—	—	1.80 (0.62–5.25)
Lymphoma/lymphosarcoma	5.82 (2.11–9.74)*	—	0.47 (0.15–1.1)	1.3 (0.4–2.9)	—
Other lymphatic	—	—	1.62 (1.17–2.18)*	—	—
Lung	—	—	—	1.2 (0.4–2.7)	3.11 (1.60–6.04)*
Larynx, lung	1.06 (0.72–1.56)	—	—	—	—
Respiratory tract	—	2.20 (1.05–4.06)[a]	0.66 (0.58–0.76)*	—	—
Other respiratory	—	—	—	—	1.20 (0.13–11.28)
Oral cavity	0.71 (0.42–1.32)	—	—	—	—
Pharynx	1.08 (0.82–1.24)	—	—	—	—
Lip, oral cavity, pharynx	—	—	—	—	4.98 (0.73–34.00)
Esophagus	—	—	1.13 (0.71–1.72)	—	—
Stomach	—	—	1.02 (0.68–1.45)	0.4 (0.1–2.0)	2.16 (0.22–20.88)
Esophagus, stomach	3.24 (1.85–5.06)*	—	—	—	—
Colon	—	—	1.11 (0.89–1.37)	1.3 (0.6–2.6)	1.35 (0.43–4.19)
Rectum	—	—	0.77 (0.42–1.29)	1.8 (0.7–3.9)	1.54 (0.42–5.61)
Colorectal	3.19 (1.54–6.18)*	—	—	—	—
Digestive organs	—	0.78 (0.15–2.31)[a]	—	—	—
Liver	—	—	0.65 (0.33–1.17)	—	—
Pancreas	—	—	0.64 (0.42–0.94)*	0.6 (0.0–3.5)	—
Liver, pancreas	1.47 (0.76–1.56)	—	—	—	—
Other gastrointestinal	—	—	—	—	1.47 (0.47–4.54)
Prostate	—	—	1.14 (0.90–1.42)	—	0.78 (0.23–2.56)
Kidney	—	—	0.94 (0.57–1.48)	1.6 (0.3–4.8)	—
Kidney, prostate	0.86 (0.54–1.67)	—	—	—	—
Bladder	—	—	0.66 (0.38–1.08)	0.6 (0.0–3.6)	—
Urinary tract	—	—	—	—	1.02 (0.34–3.07)
Female breast	—	—	—	1.5 (1.1–2.0)*	—
Cervix	—	—	—	1.0 (0.6–1.7)	—
Uterus (endometrium)	—	—	—	1.9 (1.0–3.2)*	—
Ovary	—	—	—	0.8 (0.3–1.6)	—
Bone	0.67 (0.36–1.42)	—	—	—	—
Skin	1.67 (0.92–4.13)	—	—	—	—
Melanoma	—	—	—	0.9 (0.4–1.7)	0.31 (0.03–2.82)
Thyroid	1.54 (0.82–2.59)	—	—	—	—
Multiple myeloma	0	—	—	—	0.20 (0.03–1.39)
Brain/nervous system	1.91 (1.08–3.47)*	—	1.39 (0.93–2.00)	1.0 (0.3–2.3)	1.90 (0.48–7.58)
Brain, astrocytoma	—	—	—	—	6.26 (0.30–132.2)
Brain, glioblastoma	—	—			0.57 (0.08–3.91)
Other cancers	—	1.17 (0.50–2.32)[a]		0.7 (0.3–1.3)	

—, No information was given in the study. [a]Robinette et al. (*29*): data are observed/exposed ratios for the highest exposure group (hazard function 5001 +) compared to all subjects, with 95% confidence limits; test of trend also applied, all results nonsignificant. *Statistically significant, $p < 0.05$.

The incidence of all cancers was modestly increased, although this was significant (RR = 1.2, 95% confidence limits 1.0–1.4). An excess risk was seen for breast cancer (RR = 1.5, 95% confidence limits 1.1–2.0) and also for uterine cancer (RR = 1.9, 95% confidence limits 1.0–3.2). There was no significant excess of leukemias, lymphoma, brain tumors, or of several other types of cancer, although the results were based on small numbers.

The authors discussed exposures to shift work, time changes, and to light-at-night, as well as to RF emissions (405 kHz–25 MHz) and ELF fields (50 Hz). The breast cancer association was further explored in a nested case–control study, showing an association with shift work in women over age 50. There are many established risk factors for breast cancer, including several aspects of reproductive history, alcohol consumption, obesity, and a family history of breast of other cancers. Cancer of the uterus shares several of these known associations with reproductive and dietary factors (*45*). The increased risks for both breast and uterine cancer, with no excesses in leukemia or similar cancers, are more suggestive of a relationship to reproductive or other lifestyle factors than to an association with RF emissions. An analysis adjusted for age at first birth (a major risk factor for breast cancer), however, still gave an RR value of 1.5 for the association between breast cancer and work as a radio operator. Tynes et al. (*44*) performed some spot measurements of fields in ships still equipped with older equipment and found that "measured values at the operator's desk were below the detection level for both electric and magnetic fields"; however, measurements 0.5 m in front of the radio tuner showed magnetic fields at frequencies above 8 MHz, which exceeded occupational exposure limits. This study presents interesting observations that require further research.

Study of Electric Utility Workers: Short-Duration Pulsed Electromagnetic Fields. Armstrong et al. (*46*) carried out a nested case–control study within cohorts of electric utility workers in Quebec, Canada, and in France, in whom 2679 incident cases of all types of cancer were identified. The exposure assessed was short-duration pulsed EMFs (PEMFs) generated mainly by dielectric switching operations. Exposures in cancer cases and in comparison subjects were assessed by a job exposure matrix based on electric field measurements carried out on approximately 1300 workers for 1-week periods in 1991 to 1992 to assess the proportion of subjects in each job who had a weekly mean exposure of > 100 ppb. That is, for > 100 ppb of time, the electric field was > 200 V/m in the 5 to 20 MHz frequency band; this is equivalent to 14.4 milliseconds/40-hr week. However, the meters also responded to RF fields of approximately 150 to 300 MHz and to radio transmissions; thus, some of the high recorded exposures could be due to RF fields (*46*).

The study has extensive data on other related factors such as smoking and other occupational exposures. There were no associations seen between exposure to these EMFs and cancers that had been suggested to be associated with EMF exposure, including leukemia, lymphomas, brain cancer, and melanoma. The results for cancers of the lip, mouth, and pharynx (together) showed a nonsignificant increase based on small numbers. Stomach cancer results showed a nonsignificant increase, restricted to France and with no clear dose–response relationship; for all cancer there was a significant increase for workers at or above the 90% percentile of exposure (RR = 1.39, 95% confidence limits 1.05–1.85) but no significant increase (RR = 1.03) based on the median dose. A significant association was seen with lung cancer: RR = 3.11, 95% confidence limits 1.60–6.04 based on the 90% percentile, and RR = 1.27 (95% confidence limits 0.96–1.68) based on the median. This was seen almost exclusively in the Quebec cohort; the levels of exposure to PEMFs were higher in Quebec. The risk increased with duration of exposure, and was seen after adjustment for smoking and other occupational exposures. The authors note that the findings were unexpected and arose after assessment of 30 cancer sites and three types of exposure—approximately 100 comparisons. They note: "However, several factors limit the strength of the evidence for a causal relation; lack of precision in what the meters measured, little previous evidence for this association; and no elevated risk for lung cancer in the utility workers overall in comparison with the general population" (*46*).

This study is methodologically the strongest of the studies reviewed here. The work histories were detailed and the results were based partially on field measurements of exposures, with good information on other relevant factors. The fields, however, may not be relevant to the major sources of RFR because they relate to a specific type of emission in a lower frequency range of 5 to 20 MHz. The study was negative in regard to leukemias and similar cancers, which were elevated in the Polish military study (*42*) and in the first Sydney study (*36*). The Armstrong et al. (*46*) study found an unexpected association with lung cancer, which is one of the few sites not elevated in the Polish military study and has not been suggested in any other study of EMF exposures.

Case–Control Studies

Several case–control studies with specific mention of RF emissions have been published (Table 4).

Brain Cancer in U.S. Air Force Personnel. In this study (*47*), U.S. Air Force servicemen who served between 1970 and 1989 and who developed brain cancer were identified from service records, and each was compared to four comparison subjects (unaffected Air Force personnel of the same age and ethnic group). Occupational histories were taken from personnel records and reviewed to assess likely exposure to both low frequency and RF microwave fields. This assessment used a job-exposure matrix developed by an expert group including a radiation physicist, an occupational health specialist, and an industrial hygienist.

The authors found that the risk of developing a brain tumor increased considerably with increasing military rank, and they could not suggest a good explanation of that relationship. In addition, they found a small association with electromagnetic radiation, which was statistically significant for RF microwave radiation (RR = 1.39, 95% confidence limits 1.01–1.90) and was somewhat lower for ELF radiation (RR = 1.28, 95% confidence limits 0.95–1.74). This study did not include men who had left the service; a bias could arise if any subjects left the service because of health problems that were later diagnosed as brain cancer.

Brain Cancer in U.S. Civilians with Occupational Exposures to Radiofrequency Emissions. This study (*48*) shows the complexity of exposures experienced through occupation. Cases were men who died of brain or other related cancers in three areas of the United States; controls were men who died from causes excluding brain cancer, epilepsy, stroke, suicide, or homicide, matched on age, year of death, and area of residence. Information on occupation was collected by interviews with the next of kin, with response rates of

Table 4. Radiofrequency emissions and cancers: case–control studies.

	Case–control studies					
	Brain tumors	Brain tumors, fatal	Testicular tumors	Breast cancer in men	Breast cancer deaths, women	Eye melanoma
Study characteristics						
Reference	Grayson, 1996 (*47*)	Thomas et al., 1987 (*48*)	Hayes et al., 1990 (*49*)	Demers et al., 1991 (*50*)	Cantor et al., 1995 (*51*)	Holly et al., 1996 (*52*)
Exposure	RF/microwave	Occupations with RF exposure	RF/microwave	Radio communication work	RF fields	Microwaves, radar
Ascertainment	Service records: job-exposure matrix	Job title: two exposure assessments	Job title and self report	Job title	Job, on death certificate	Interview
Exposed group	U.S., military	U.S., men > age 30, white	U.S., mainly military	U.S., civilian	U.S., women	U.S., general population
Cancer type, adult						
Brain cancers	1.39 (1.01–1.90)*	1.0 (0.5–1.9)[a]	—	—	—	—
Male breast	—	—	—	2.9 (0.8–10.0)	—	—
Testis	—	—	3.1 (1.4–6.9)[b,*]	—	—	—
Testis	—	—	1.1 (0.6–2.1)[c]	—	—	—
Female breast, white	—	—	—	—	1.14 NS[d]	—
Female breast, black	—	—	—	—	1.34 NS	—
Eye melanoma	—	—	—	—	—	2.1 (1.1–4.0)*

—, No information given in the study. [a]For RF exposure, not in electrical jobs. [b]By self-reported exposure. [c]By assessment from job titles; any exposure. [d]Maximum level of exposure. *Statistically significant, $p < 0.05$.

74% for the cases and 63% for the controls. Occupations were classified in terms of likely exposures by two methods: one based on a predesigned list of occupations likely to involve RF radiation, and one based on a review of each job by a certified industrial hygienist.

The single-factor analysis showed an excess risk among men who had ever had a job in which they were likely to be exposed to RF radiation (RR = 1.6, 95% confidence limits 1.0–2.4). However, this risk was only seen in those whose potential exposure to RFR was in an electrical or electronic occupation, where the relative risk was 2.3. By comparison, those exposed to RFR without working in such a job had a relative risk of 1.0, and the risk was increased in electronics workers with no exposure to RFR.

Thus the increased risk was due to some other aspect of work in an electrical or electronics occupation. Further analysis showed increases in risk with jobs involving exposure to soldering fumes, lead, and organic solvents. The authors conclude that "that pattern of excess brain tumor risk among electrical and electronics workers, and not among others exposed to radiofrequency radiation, suggests that simple exposure to radiofrequency radiation is not the responsible agent" (*48*).

Study of Testicular Cancer. A study in the United States (*49*) assessed 271 men 18 to 42 years of age with testicular cancer and 259 controls, identified at three medical centers, two of which were military hospitals. Exposure to microwave and other radio waves was assessed in two ways: by an analysis based on job title and by self-report. An excess risk was seen with the self-reported exposure (RR = 3.1, 95% confidence limits 1.4–6.9). However, no association was seen with the analysis based on job title (RR = 1.1); job categories classified as likely to have had the heaviest exposure had RR values = 0.8. Therefore, the results are inconsistent.

Male Breast Cancer Study. Occupational exposure to EMFs and breast cancer in men (a rare disease) has been studied (*50*) based on 227 cases from 10 areas of the United States, as compared to 300 controls. Exposure status was defined as ever having been employed in a job that had been classified as involving potential exposure to EMFs. The risks seen were highest among electricians, telephone linemen, and electric power workers. Radio and communications workers had a nonsignificant increase in risk (RR = 2.9, 95% confidence limits 0.8–10.0) based on seven cases. Risk did not vary with duration of exposed employment. In this study the participation rates were low, especially in the controls. The results can be regarded only as a preliminary observation requiring further research.

Case–Control Study of Breast Cancer Deaths. Cantor et al. (*51*) performed a case–control study of women who died from breast cancer between 1984 and 1989 in 24 states in the United States, matching each with controls randomly selected from noncancer deaths and matched for age, gender, and race; over 33,000 cases and 117,000 controls. Occupational data were limited to that recorded on the death certificate. A job-exposure matrix was used to estimate the probability and level of 31 workplace exposures, and the analyses were carried out after adjusting for socioeconomic status. The authors conclude that "suggestive associations for probability and level of exposure were found for styrene, several organic solvents,...and several metals/metal oxides and acid mists. Because of the methodologic limitations of this study, its primary value is in suggesting hypotheses for further evaluation" (*51*).

RF EMF exposure was one of the exposures studied. It was classified in five groups ranging from nonexposed through four increasing levels of estimated exposure probability. For white subjects, a significant OR of 1.15 (95% confidence limits 1.1–1.2) was reported for the fourth of the five levels, but there was no excess in the highest level of exposure probability (OR 0.99) and no regular trend. An analysis using exposure level, that is, estimated intensity, classified as nonexposed and three levels, showed significant increases at level 1 and level 3 (OR 1.15 and 1.14, respectively) but a reduced risk at level 2 (OR 0.95, 95% confidence limits 0.9–1.0), and thus again no regular trend. Results for African–American women were somewhat similar, with some significant elevations in individual categories but no regular trend on either analysis.

The authors' discussion takes into account the issues of multiple testing and the lack of a dose–response trend, and their conclusion was "in this investigation, we found no association with either ionizing or non-ionizing radiation" (*51*).

Case–Control Study of Intraocular Melanoma: Microwaves and Radar. Holly et al. (*52*) conducted a case–control study of intraocular melanoma based on 221 male white patients residing in the western United States and seen at one center in San Francisco, compared to 447 controls of similar age in the same geographical area as identified through random digit dialing. A large number of occupational groups were assessed, with significant increased risks found in chemists, sailors or fishermen, welders, and health-related occupations. In terms of specific exposures, significant excess risks were seen with exposures to formaldehyde, pesticides, carbon tetrachloride, asbestos, and a marginally significant excess risk seen with exposure to antifreeze. Only results for the exposures for which there was an increased risk are presented, and these are presumably a subset of a large number of different exposures that were assessed, of which the total number is not given.

The finding of an elevated risk for ever exposure to microwaves or radar, with an OR of 2.1, 95% confidence limits 1.1–4.0, based on 21 of the 221 patients being exposed, must be taken in this context. The authors do not mention this result in their summary, and they point out that the data on recall of specific exposures "are more subject to recall bias than the major findings based on the occupational groups" (*52*). They state that the association with health-related occupations, which might be the one most readily associated with microwaves, "could have resulted from referral bias" (*52*), given that this study was carried out in only one referral center.

Other Human Studies Relevant to Cancer Causation. Garson et al. (*53*) carried out chromosomal studies on 38 radio linemen employed by Telecom Australia and who had exposure to 400 kHz to 20 GHz radio frequencies at or below occupational limits, and 38 age-matched controls who were clerical staff with no exposure to RFs. Some 200 metaphases from each subject were studied and scored by an observer who was blind as to exposure status. A monitoring committee including representatives of Telecom Australia and the relevant union monitored the study and all data were coded blind until the study was finalized. The results showed virtually identical frequencies in a range of specified aspects of chromosomal damage, with the overall risk ratio of aberrant cells being 1.0 (95% confidence limit 0.8–1.3). The authors state that this was the first study of chromosome damage in workers exposed to RFs.

In an *in vitro* experiment using high dosages of high frequency microwave radiation, samples of human whole blood were exposed to 7.7 GHz radiation at power densities from 0.5 to 30 mW/cm^2. This produced an increase in chromosomal aberrations and micronuclei (*54*).

A few other studies assessed blood counts, which could be relevant to cancer but are also influenced by many other factors. Two studies found no effects in workers with occasional high exposures to radar (*55,56*).

Discussion

Methods

For this review, a systematic search procedure has been used to identify all major published studies dealing specifically with RFs and human cancer. Many other studies relate to exposures to EMFs with no further specification. These may include RF exposures but, in the absence of any specification, the major exposures are more likely to be ELFs. Such studies have been examined in major reviews of ELF exposure. They include cohort studies of telecommunication workers (*57,58*), workers in electrically related jobs (*59*), and studies assessing exposures to visual display terminals (*60*). Studies based only on national routine data sets of incidence or mortality with occupation as recorded on the death certificate or hospital record were excluded because they suffer from several problems: the quality of the data is limited; multiple testing is a major difficulty; and such reports, because of their size, are often unpublished or are published only as government documents. Selected items from them published in the scientific literature are particularly open to publication bias.

Although only studies published in scientific journals have been included in this review, the report by Lilienfeld et al. (*61*) deserves attention because it has been given publicity by others. According to Bergqvist (*27*), this study compared employees at the U.S. Embassy in Moscow with employees of U.S. embassies in other eastern European countries because RF at 0.5 and 10 GHz of up to 5 μW/cm^2 for 9 hr/day was detected at the Moscow Embassy between 1963 and 1975, but such radiation was not detected at other embassies. The study of embassy employees reviewed medical records for 71%, and used a questionnaire with a response rate of 42%, which is low. Bergqvist (*27*) quotes a relative risk for all cancers of 0.9 (95% confidence limits 0.5–1.4) and increased risks of leukemia and of breast cancers, each nonsignificant and based on only two cases, comparing the Moscow Embassy staff with U.S. national rates. For both leukemia and breast cancer, the rates were lower for Moscow Embassy staff than for the staff of other eastern European embassies with no recorded RF exposure. Goldsmith (*62*) also comments on this study and presents data showing excess risks of leukemia, brain tumors, and breast cancers in embassy staff compared to expected values, which are not specified but are presumably based on U.S. rates, but the data show lower risks in Moscow staff than in other embassy staff. Goldsmith also notes that studies of chromosome changes and hemologic measures were also conducted, which he claims shows changes in the embassy staff as compared to foreign service examinations carried out in Washington, although he quotes an expert panel that concluded that "no valid conclusions could be drawn from this study" (*62*). Goldsmith implies that the results suggest there must have been similar radiation exposures at the other eastern European embassies, but there is no evidence for this. Overall, the results of this study appear negative.

Types of Epidemiological Study. The relative importance of epidemiologic studies can be referred to as a hierarchy of studies (*63,64*). The strongest evidence to assess a cause-and-effect relationship comes from an experimental study, but obviously this cannot be applied to potential hazards in a general population. The best studies to assess such potential hazards are studies based on individuals with good information on the suspected causal factor, the disease outcome, and on other relevant factors related to the outcome. Such cohort and case–control studies are the method by which most recognized causes of human cancer have been identified, such as tobacco smoking, exposure to asbestos, or exposure to ionizing radiation. Usually many such studies need to be done before a consensus can be reached (*63*).

Limitations of Studies of Radiofrequencies and Health Effects: The Ecological Fallacy. The type of evidence available on health effects of RF is not comparable to the types of evidence available for such well-established links as those between smoking and lung cancer, asbestos and mesothelioma, or thalidomide and birth defects. For RF, the studies of individuals

are limited to small case–control studies with poor estimation of RF exposure and to cohort studies of certain groups (such as military personnel) whose exposure levels are likely to be different from those of the general population. Even these individually based studies do not show consistent results.

The studies of general populations are not based on individuals but are based on comparing community groups with hypothesized different levels of exposure determined, for example, by residence. These are ecological studies. It is fallacious to assume that an association seen in an ecological study indicates a cause-and-effect relationship between individuals (*65*); this error has been referred to as the ecological fallacy. Thus, many ecological studies demonstrated relationships between heart disease frequency in different geographic areas and various characteristics in drinking water, but further studies have not shown any important relationship at the individual level (*66*). The explanation is that geographical areas with different water supplies have many other differences that are more directly related to heart disease.

The ecological fallacy can only be demonstrated when hypotheses, created on the basis of comparisons between groups, are tested in high-quality studies of individuals, with good measurement of individual exposure and individual health outcome. A recent example is a major case–control study to test the hypothesis that childhood cancer was related to radon gas concentrations (*67*). Radon levels in the current and previous homes of children who later developed leukemia, and in comparison children, were directly measured in a study involving nearly 1000 children. The mean radon levels were lower for children who developed leukemia than for controls; the authors conclude: "In contrast to prior ecological studies, the results from this analytic study provide no evidence for an association between indoor radon exposure and childhood leukemia" (*67*).

Criteria Used in Assessing Causality. Criteria have been developed that are generally and internationally accepted for the assessment of epidemiologic evidence from an individual study, and from the totality of evidence derived from a number of studies. There are two sets of criteria. The first set comprises three factors that could explain an apparent association between an exposure and a disease, apart from a cause-and-effect relationship (*63*). These factors are as follows:

- Bias in the observations that are made. For example, in a study based on an interview recall of exposures, people affected with cancer may more readily recall and report a previous exposure than people who have not had cancer.
- The effect of other relevant factors, known as confounding. Thus there may be an association between ice cream sales and drownings in coastal areas due to a third related factor, that of good weather.
- Apparent associations may be due to chance variation. This is assessed by statistical methods.

The first process in assessing whether a particular study gives a valid cause-and-effect assessment is therefore to see if these alternative explanations can be reasonably excluded.

The next process is to look for the specific features expected if a biologic cause-and-effect relationship applies. These criteria, often referred to as the Bradford Hill criteria (*68*), are accepted by many multidisciplinary international groups in the assessment of cause and effect in cancer and in other major diseases (*69*). They are useful but are not prescriptive or absolute criteria, and their application must take into account measurement errors and probable heterogeneity of both exposure and outcome. The criteria are as follows: The time relationship must be clear; the suspected cause must clearly precede the development of the disease. The relationship should be strong, that is, there should be a substantially higher rate of disease in subjects exposed to the potential causal factor than in those not exposed. There should be a dose–response relationship, that is, subjects with greater exposure should have greater disease outcome rates. Specificity may be helpful, although it is not always applicable. This means that a particular causal agent causes a particular disease rather than a whole range of diseases. Plausibility may be a useful criterion but is often open to varied interpretations. This means that a mechanism for the relationship between the causal agent and the disease outcome can be recognized based on knowledge of the underlying biology of the situation. A related criterion that is sometimes added is analogy—that the relationship is similar to some other established relationship—but this is merely an aspect of plausibility. Coherence implies that there should be an overall association seen between the general distribution of the causal agent and the distribution of the disease that it causes. It is a less useful criterion because it holds only if the causal relationship is strong. Consistency is the most important criterion. Consistency is assessed first as consistency within a study. For example, if radiation causes a particular cancer, we would expect to see that relationship in men and women, in different social groups, geographical areas, and so on. The most important criterion is consistency among various studies (*63*). In the great majority of situations the development of a consensus on whether a particular agent causes (for example) cancer is based on a consideration of the consistency of evidence from a large number of studies, of different designs and in different populations that overall give a substantial body of evidence.

Application of Criteria for Causality. The available studies on RF and human cancer have been assessed in regard to these criteria.

The study designs used in general are weak. This applies particularly to the assessment of likely exposure. In all the general population studies, the measure of potential exposure used has been the place of residence at the time of diagnosis of the cancer or at the time of death. This is only an approximate indicator of the level of exposure to the relevant wavelengths of RF transmissions at the critical time for the causation of the cancer, which will be months or years before diagnosis. Also, information on other relevant factors at the individual level has not been collected and cannot be adjusted for. Where area of residence is defined down to a small unit, as in the U.K. studies (*34,35*), there is some ability to control for other factors by using variables related to that small area of residence, but where the area is larger, as in the first Sydney study (*36*), this is less easily done.

The occupational cohort studies are somewhat stronger. However, although these are studies of individuals in terms of cancer occurrence, data on exposure to RFs are indirect, being based on job title and other details such as military posting, with either assumptions or limited field measurements available, not for all the individuals in the study, but for other samples of those in particular jobs or of the environment of particular jobs.

In the case–control studies, estimates of likely exposure in the past have been based either on job titles or on self-report; none of these studies are convincing. Therefore all of these studies are relatively weak, which means that issues of observation bias

and the influence of other relevant factors cannot be easily discounted.

In terms of criteria for positive aspects of causality, we can accept the time criterion that exposure was likely to precede the effect. None of these studies give enough detail to assess the likely time interval between exposure and an increase in risk.

In regard to strength, the strongest associations were seen in the Polish military study (*42*). The difficulty is that this study showed an excess of all cancer and of many types of cancer, including gastrointestinal tract cancer, which has not been related to radiation in any other major study. It seems likely that there is a bias in that study toward a general excess of disease being recorded in association with higher exposure. A strong association with lung cancer was seen in the cohort study of electrical utility workers exposed to PEMFs, but the authors themselves regarded this with considerable doubt. The associations were not strong in any of the other studies. In the Sydney study (*36*) the association with the incidence of childhood leukemia had an odds ratio of 1.6. In the more extensive and powerful UK study, even the equivocal results on total leukemia are based on a maximum excess of only 15% seen only in some localities (*35*).

Few of these studies showed any consistent dose–response assessment. The utility workers (*46*) and the U.S. military study (*29*) had some measures of different likely exposure dosages but did not show consistent results. The Sutton Coldfield and UK studies (*34,35*) were based largely on assessing a measure of dose response, that is, a gradation of risk with distance of residence from the transmitter, and did not give consistent positive results for any cancer site.

The criteria on which these studies clearly break down is in specificity to cancer types and in consistency. If there is a real effect of RF on cancer, we would expect to see it consistently on certain types of cancer in various studies. However, this is not found. Figure 1 summarizes the results for adult and childhood leukemias. The strongest individual results for RF in a general population were the first Sydney results for childhood leukemia (*36*). However, the Sutton Coldfield and U.K. 20 transmitter studies showed no clear excess of childhood leukemia (*34,35*), nor did the San Francisco study (*41*). For adult leukemias of all types, there was a weaker but still positive relationship in the Sydney study (*36*). The results for the U.K. 20 transmitter studies gave some evidence of a decreasing trend with residence, although the effect was small and there was no excess in those who live closest to the transmitter (*35*). The results for all adult leukemias were weak and equivocal in the U.S. Navy study (*29*). The Polish military study showed an excess of leukemia but also an excess of several other cancers that were not shown in any other study (*42*).

In adults, cancers of the central nervous system and the brain gave positive results in the Polish military study (*42*) and in the small case–control study of brain tumors in U.S. military (*47*) but showed no association in the study of electric utility workers (*46*) or the Sydney study (*36*), and no clear association in the Sutton Coldfield study (*35*). There was no association with cancers of the brain in childhood in any of the three studies that assessed it (*35,36,41*).

The criterion of coherence, that is, whether the rates of these diseases vary in time and place with RF emissions, is only relevant if it is claimed that these associations are strong and if this exposure is the major cause of these diseases. This criterion is not helpful.

The final criterion is that of plausibility, which relates to whether an established mechanism relating in biologic terms this exposure to cancer production is accepted. The experimental evidence, both in human cell systems and in the nonhuman situations, is in itself controversial. There is no consensus from major interdisciplinary review groups that the evidence clearly suggests a potential biologic mechanism for cancer causation, although a minority of results suggest potential effects in terms of initiation or promotion actions; therefore, this criterion is also unhelpful.

Conclusions

Several studies have assessed associations between likely exposure to RF and various types of human cancer.

The studies individually are weak and, as a consequence, the results cannot be easily interpreted in terms of cause and effect. The major impression from these studies is their inconsistency. There is no type of cancer that has been consistently associated with RF exposures.

The epidemiologic evidence falls short of the strength and consistency of evidence that is required to come to a reasonable conclusion that RF emissions are a likely cause of one or more types of human cancer. The evidence is weak in regard to its inconsistency, the weak design of the studies, the lack of detail on actual exposures, the limitations of the studies in their ability to deal with other likely factors, and in some studies there may be biases in the data used. Whereas the current epidemiologic evidence justifies further research to clarify the situation, there is no consistent evidence of any substantial effect on human cancer causation.

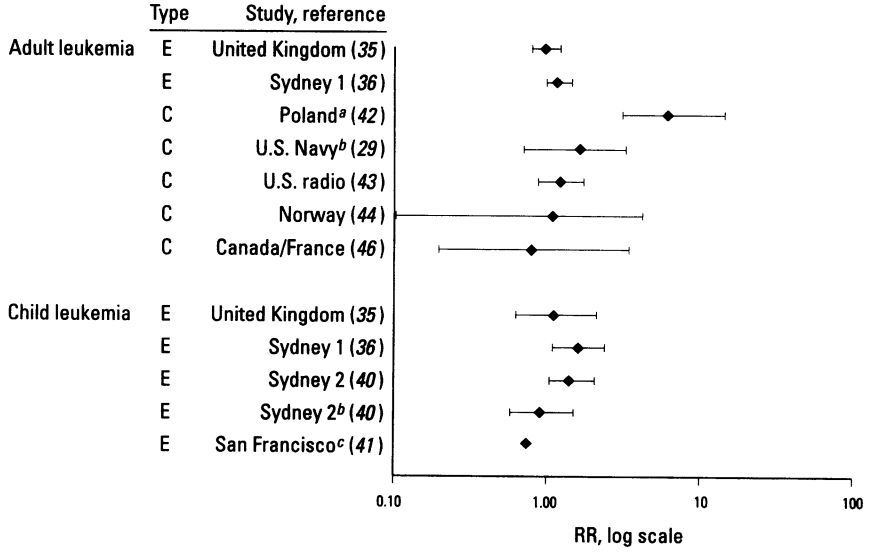

Figure 1. Relative risks and 95% confidence limits for studies of leukemia in adults and in children. Type of study: C, occupational cohort; E, ecological. [a]All lymphatic and hematopoietic—total leukemia not given. [b]Excluding Lane Cove area. [c]No confidence limits given; nonsignificant.

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REFERENCES AND NOTES

1. International Commission on Non-Ionizing Radiation Protection. Health issues related to the use of hand-held radiotelephones and base transmitters. Health Phys 70:587–593 (1996).
2. Rothman KJ, Chou C, Morgan R, Balzano Q, Guy AW, Funch DP, Preston-Martin S, Mandel J, Steffens R, Carlo G. Assessment of cellular telephone and other radio frequency exposure for epidemiologic research. Epidemiology 7:291–298 (1996).
3. International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Phys 74:494–522 (1998).
4. United Nations Environment Programme/World Health Organization and International Radiation Protection Association. Electromagnetic Fields (300 Hz to 300 GHz). Environmental Health Criteria Series 137. Geneva:World Health Organization, 1993.
5. National Radiological Protection Board. Biological Effects of Exposure to Non-Ionising Electromagnetic Fields and Radiation. III: Radiofrequency and Microwave Radiation. R-240. Chilton, UK:National Radiological Protection Board; 1991.
6. McKinlay AF, Andersen JB, Bernhardt JH, Grandolfo M, Hossmann K, Mild KH, Swerdlow AJ, Van Leeuwen M, Verschaeve L, Veyret B. Radiotelephones and Human Health - Proposal for a European Research Programme. Brussels: European Commission Directorate General XIII, 1996.
7. Cridland NA. Electromagnetic fields and cancer: a review of relevant cellular studies. NRPB-R256. Chilton, UK:National Radiological Protection Board, 1993.
8. Polk C, Postow E. Biological Effects of Electromagnetic Fields. 2nd ed. Boca Raton, FL:CRC Press, 1996.
9. Repacholi MH. Low-level exposure to radiofrequency electromagnetic fields: health effects and research needs. Bioelectromagnetics 19:1–19 (1998).
10. Sarkar S, Ali S, Behari J. Effect of low power microwave on the mouse genome: a direct DNA analysis. Mutat Res 320:141–147 (1994).
11. Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics 16:207–210 (1995).
12. Lai H, Singh NP. Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int J Radiat Biol 69:513–521 (1996).
13. Williams GM. Comment on "Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells" by Henry Lai and Narendra P. Singh. [Letter]. Bioelectromagnetics 17:165 (1996).
14. Chou C, Guy A, Kunz LI, Johnson RB, Crowley JJ, Krupp JH. Long-term, low-level microwave irradiation of rats. Bioelectromagnetics 13:469–496 (1992).
15. Szmigielski S, Szudinski A, Pietraszek A, Bielec M, Wrembel JK. Accelerated development of spontaneous and benzopyrene-induced skin cancer in mice exposed to 2450-MHz microwave radiation. Bioelectromagnetics 3:179–191 (1982).
16. Santini R, Hosni M, Deschaux P, Packeco H. B16 melanoma development in black mice exposed to low-level microwave radiation. Bioelectromagnetics 9:105–107 (1988).
17. Salford LG, Brun A, Eberhardt JL. Experimental studies of brain tumour development during exposure to continuous and pulsed 915 MHz radiofrequency radiation. Bioelectrochem Bioenerg 30:313–318 (1993).
18. Repacholi MH, Basten A, Gebski V, Noonan D, Finnie J, Harris AW. Lymphomas in Eμ-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. Radiat Res 147:631–640 (1997).
19. Bawin SM, Kaczmarek LK, Adey WR. Effects of modulated VHF fields on the central nervous system. Ann NY Acad Sci 274:74–81 (1975).
20. Blackman CF, Elder JA, Weil CM, Benane SG, Eichinger DC, House DE. Induction of calcium-ion efflux from brain tissue by radiofrequency radiation: effects of modulation frequency and field strength. Radio Sci 14:93–98 (1979).
21. Albert EN, Slaby F, Roche J, Loftus J. Effect of amplitude modulated 147 MHz radiofrequency radiation on calcium ion efflux from avian brain tissue. Radiat Res 109:19–27 (1987).
22. Lyle DB, Schechter P, Adey WR, Lundak RL. Suppression of T-lymphocyte cytotoxicity following exposure to sinusoidally amplitude-modulated fields. Bioelectromagnetics 4:281–292 (1998).
23. Byus CV, Lundak RL, Fletcher RM, Adey WR. Alterations in protein kinase activity following exposure of cultured human lymphocytes to modulated microwave fields. Bioelectromagnetics 5:341–351 (1984).
24. Byus CV, Kartun K, Pieper S, Adey WR. Increased orthinine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumour promoters. Cancer Res 48:4222–4226 (1988).
25. Litovitz TA, Krause D, Penafiel M, Elson EC, Mullins JM. The role of coherence time in the effect of microwaves on orthinine decarboxylase activity. Bioelectromagnetics 14:395–403 (1993).
26. Balcer-Kubiczek EK, Harrison GH. Neoplastic transformation of C3H/10T1/2 cells following exposure to 120 Hz modulated 2.45 GHz microwaves and phorbol ester tumour promoter. Radiat Res 126:65–72 (1991).
27. Bergqvist U. Review of epidemiological studies. In: Mobile Communications Safety (Kuster N, Balzano Q, Lin JC, eds). London:Chapman & Hall, 1997;147–170.
28. Ovid: Medline [database on CD-ROM]. Version 3.0. Bethesda, MD: National Library of Medicine, 1966-. Updated monthly (Optional).
29. Robinette CD, Silverman C, Jablon S. Effects upon health of occupational exposure to microwave radiation (radar). Am J Epidemiol 112:39–53 (1980).
30. Breslow NE, Day NE. Statistical Methods in Cancer Research. Volume 2: The Design and Analysis of Cohort Studies. IARC Sci Publ No. 82. Lyon:International Agency for Research on Cancer, 1987.
31. Rothman KJ. A sobering start for the cluster busters' conference. Am J Epidemiol 132(Suppl.1):S6–S13 (1990).
32. Maskarinec G, Cooper J, Swygert L. Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations. J Environ Pathol Toxicol Oncol 13:33–37 (1994).
33. Davis RL, Mostofi FK. Cluster of testicular cancer in police officers exposed to hand-held radar. Am J Ind Med 24:231–233 (1993).
34. Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt L, Elliott P. Cancer incidence near radio and television transmitters in Great Britain. 1: Sutton Coldfield transmitter. Am J Epidemiol 145:1–9 (1997).
35. Dolk H, Elliott P, Shaddick G, Walls P, Thakrar B. Cancer incidence near radio and television transmitters in Great Britain. 2: All high power transmitters. Am J Epidemiol 145:10–17 (1997).
36. Hocking B, Gordon IR, Grain HL, Hatfield GE. Cancer incidence and mortality and proximity to TV towers. Med J Aust 165:601–605 (1996).
37. Hocking B, Gordon I, Hatfield G, Grain H. Re: Cancer incidence near radio and television transmitters in Great Britain. I: Sutton Coldfield transmitter. II: All high power transmitters. [Letter]. Am J Epidemiol 147:90–91 (1998).
38. Greaves MF. Speculations on the cause of childhood acute lymphoblastic leukemia. Leukemia 2(2):120–125 (1988).
39. Dolk H. Reply: Re: Cancer incidence near radio and television transmitters in Great Britain. I: Sutton Coldfield transmitter. II: All high power transmitters. [Letter]. Am J Epidemiol 147:91 (1998).
40. McKenzie DR, Yin Y, Morrell S. Childhood incidence of acute lymphoblastic leukaemia and exposure to broadcast radiation in Sydney—a second look. Aust NZ J Public Health 22:360–367 (1998).
41. Selvin S, Schulman J, Merrill DW. Distance and risk measures for the analysis of spatial data: a study of childhood cancers. Soc Sci Med 34:769–777 (1992).
42. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. Sci Total Environ 180:9–17 (1996).
43. Milham S. Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies. Am J Epidemiol 127:50–54 (1988).
44. Tynes T, Hannevik M, Andersen A, Vistnes AI, Haldorsen T. Incidence of breast cancer in Norwegian female radio and telegraph operators. Cancer Causes Control 7:197–204 (1996).
45. Kvåle G, Heuch I, Nilssen S. Reproductive factors and cancers of the breast and genital organs—are the different cancer sites similarly affected? Cancer Detect Prev 15:369–377 (1991).

46. Armstrong B, Theriault G, Guenel P, Deadman J, Goldberg M, Heroux P. Association between exposure to pulsed electromagnetic fields and cancer in electric utility workers in Quebec, Canada, and France. Am J Epidemiol 140:805–820 (1994).
47. Grayson JK. Radiation exposure, socioeconomic status, and brain tumour risk in the US Air Force: a nested case-control study. Am J Epidemiol 143:480–486 (1996).
48. Thomas TL, Stolley PD, Stemhagen A, Fontham ETH, Bleeker ML, Stewart PA, Hoover RN. Brain tumour mortality risk among men with electrical and electronic jobs: a case-control study. J Natl Cancer Inst 79:233–238 (1987).
49. Hayes RB, Brown LM, Pottern LM, Gomez M, Kardaun JWPF, Hoover RN, O'Connell KJ, Sutzman RE, Javadpour N. Occupation and risk of testicular cancer: a case-control study. Int J Epidemiol 19:825–831 (1990).
50. Demers PA, Thomas DB, Rosenblatt KA, Jimenez LM, McTiernan A, Stalsberg H, Sternhagen A, Thompson WD, McCrea Curnen MG, Satariano W, et al. Occupational exposure to electromagnetic fields and breast cancer in men. Am J Epidemiol 134:340–347 (1991).
51. Cantor K, Stewart P, Brinton L, Dosemeci M. Occupational exposures and female breast cancer mortality in the United States. J Occup Environ Med 37:336–348 (1995).
52. Holly EA, Aston DA, Ahn DK, Smith AH. Intraocular melanoma linked to occupations and chemical exposures. Epidemiology 7:55–61 (1996).
53. Garson OM, McRobert TL, Campbell LJ, Hocking B, Gordon I. A chromosomal study of workers with long term exposure to radiofrequency radiation. Med J Aust 155:289–292 (1991).
54. Garaj-Vrhovac V, Fucic A, Horvat D. The correlation between the frequency of micronuclei and specific aberrations in human lymphocytes exposed to microwave radiation *in vitro*. Mutat Res 281:181–186 (1992).
55. Barron CI, Baraff AA. Medical considerations of exposure to microwaves (radar). JAMA 168:1194–1199 (1958).
56. Djordjevic Z, Kolak A, Stojkovic M, Rankovic N, Ristic P. A study of the health status of radar workers. Aviat Space Environ Med 50:396–398 (1979).
57. De Guire L, Theriault G, Iturra H, Provencher S, Cyr D, Case BW. Increased incidence of malignant melanoma of the skin in workers in a telecommunications industry. Br J Ind Med 45:824–828 (1988).
58. Vågerö D, Ahlbom A, Olin R, Sahlsten S. Cancer morbidity among workers in the telecommunications industry. Br J Ind Med 42:191–195 (1985).
59. Törnqvist S, Knave B, Ahlbom A, Persson T. Incidence of leukaemia and brain tumours in some "electrical occupations." Br J Ind Med 48:597–603 (1991).
60. Beall C, Delzell E, Cole P, Brill I. Brain tumours among electronics industry workers. Epidemiology 7:125–130 (1996).
61. Lilienfeld AM, Tonascia J, Tonascia S, Libauer CA, Cauthen GM. Foreign Service Health Status Study—Evaluation of Health Status of Foreign Service and Other Employees from Selected Eastern Posts. NTIS PB-288163. Washington: State Department, 1978.
62. Goldsmith JR. Epidemiologic evidence of radiofrequency radiation (microwave) effects on health in military, broadcasting, and occupational studies. Int J Occup Med Environ Health 1:47–57 (1995).
63. Elwood JM. Critical Appraisal of Epidemiological Studies and Clinical Trials. 2nd ed. Oxford:Oxford University Press, 1998.
64. U.S. Preventive Services Task Force. Guide to Clinical Preventive Services: An Assessment of the Effectiveness of 169 Interventions. Baltimore:Williams & Wilkins, 1989.
65. Morgenstern H. Uses of ecologic analysis in epidemiologic research. Am J Public Health 72:1336–1344 (1982).
66. Last JM, ed. A Dictionary of Epidemiology. 3rd ed. New York:Oxford University Press, 1995.
67. Lubin JH, Linet MS, Boice JD, Buckley J, Conrath SM, Hatch EE, Kleinerman RA, Tarone RE, Wacholder S, Robison LL. Case-control study of childhood acute lymphoblastic leukemia and residential radon exposure. J Natl Cancer Inst 90:294–300 (1998).
68. Hill AB. The environment and disease: association or causation? Proc R Soc Med 58:295–300 (1965).
69. Weed DL. Causal and preventive inference. In: Cancer Prevention and Control (Greenwald P, Kramer BS, Weed DL, eds). New York:Marcel Dekker, 1995;285–302.