

# Epidemiological Studies of Radio Frequency Exposures and Human Cancer<sup>†</sup>

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Epidemiological studies of radio frequency (RF) exposures and human cancers include studies of military and civilian occupational groups, people who live near television and radio transmitters, and users of mobile phones. Many types of cancer have been assessed, with particular attention given to leukemia and brain tumors. The epidemiological results fall short of the strength and consistency of evidence that is required to come to a conclusion that RF emissions are a cause of human cancer. Although the epidemiological evidence in total suggests no increased risk of cancer, the results cannot be unequivocally interpreted in terms of cause and effect. The results are inconsistent, and most studies are limited by lack of detail on actual exposures, short follow-up periods, and the limited ability to deal with other relevant factors. In some studies, there may be substantial biases in the data used. For these same reasons, the studies are unable to confidently exclude any possibility of an increased risk of cancer. Further research to clarify the situation is justified. Priorities include further studies of leukemia in both adults and children, and of cranial tumors in relationship to mobile phone use. *Bioelectromagnetics Supplement 6:S63–S73, 2003.* © 2003 Wiley-Liss, Inc.

**Key words:** occupational exposures; military exposures; broadcast transmitter; mobile telephones; leukemia

## INTRODUCTION AND METHODS

This review addresses whether the available epidemiological studies show that exposure to radio frequencies causes cancers in humans. It is based on published epidemiological studies of radio frequency (RF) emissions and human cancer, identified by literature searches using *Medline* from 1988 to November 2002, supplemented by references found in previous reports and other studies. It excludes case reports, studies with no comparison group, and studies based only on large routinely collected data sets. This paper is based on two previously published works [Elwood, 1999; Australian Radiation Protection and Nuclear Safety Agency, 2002] but includes more recent studies. Other reviews have been assessed, including those by the International Committee on Non-Ionizing Radiation Protection (ICNIRP) [International Commission on Non-ionizing Radiation Protection (ICNIRP), 1998], the Royal Society of Canada [Royal Society of Canada, 1999; Krewski et al., 2001], the UK's Stewart committee [Independent Expert Group on Mobile Phones, 2000], the French Director General of Health [Directeur General de la Sante, 2001], a review commissioned by the Swedish Radiation Protection Authority [Boice and McLaughlin, 2002], and reviews by Moulder et al. [1999] and Bergqvist [1997].

## EPIDEMIOLOGICAL STUDIES OF CANCER UP TO 1999

These studies have been reviewed in more detail previously [Elwood, 1999], so only a brief summary will be given here. A study of cancer in people living within 10 km of any of 20 high power radio and TV transmitters in the UK showed generally negative results, although a weak trend of borderline statistical significance toward a decrease in rates of adult leukemia with increasing distance from the transmitters was seen [Dolk et al., 1997a]. This followed an observed excess of adult leukemia near one transmitter, located at Sutton Coldfield, following the observation of a cluster of cases of leukemia and lymphoma; no causal inference can be

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drawn from a cluster investigation alone [Dolk et al., 1997b]. In Sydney, Australia [Hocking et al., 1996], increased incidence and mortality rates of childhood leukemia in areas close to a VHF-TV transmitter were shown, but a further analysis showed that the excess applied only to one of the three areas close to the transmitter [McKenzie et al., 1998]. An earlier study of childhood cancer in San Francisco showed no geographical association with a transmitter described as a microwave tower [Selvin et al., 1992].

A cohort study in the Polish military showed substantial excesses of total cancer and of several types of cancer [Szmigielski, 1996], but there is likely to be severe bias in the exposure information in that study [Bergqvist, 1997; Elwood, 1999; Independent Expert Group on Mobile Phones, 2000]; the results are inconsistent with those of other studies. A study of US Navy servicemen showed no clear increase in cancer in personnel likely to have had substantial exposure to radar [Robinette et al., 1980]; this study has recently been updated and is discussed later. Studies of US male amateur radio operators showed an excess in one of nine types of leukemia assessed, although these men had other exposures, which may be relevant, to electric shock, soldering fumes, and degreasing agents; and many had occupational exposures [Milham, 1988]. A study of female radio and telegraph operators working at sea showed an excess of breast cancer and uterine cancer, but other confounding factors are relevant [Tynes et al., 1996]. A detailed study of electrical workers in Quebec and France showed an excess of lung cancer, but their exposures were not primarily to radio frequencies [Armstrong et al., 1994]. In a small study [Lagorio et al., 1997] based on women working with RF sealers in Italy, an excess in cancers was seen, but it was nonsignificant and based on only six cases. Many studies have reported on occupations likely to be exposed mainly to ELF fields, but who may have had exposure to radio frequencies in addition; these are difficult to interpret in terms of RF effects.

In case-control studies of particular types of cancer, radio frequencies have been one of a number of exposure factors assessed. An association was seen between likely RF exposures and brain cancers in US Air Force personnel [Grayson and Lyons, 1996]. A study in US civilians showed an excess of brain tumor deaths only for the combination of RF exposures and other electrical or electronic job exposures, not for radio frequencies alone [Thomas et al., 1987]. A case-control study of brain cancer in Israel [Kaplan et al., 1997] showed some associations with electrical jobs, but had no specific information on radio frequencies. Other studies of various cancers show excesses which vary with the method of collecting the information, are non-

significant, or are open to the problem of multiple testing [Hayes et al., 1990; Demers et al., 1991; Cantor et al., 1995; Holly et al., 1996].

## **EPIDEMIOLOGICAL STUDIES OF CANCER PUBLISHED SINCE 1999**

### **Studies of Cancer in Relationship to Radio and Television Transmitters**

A further study of cancer incidence in residents living close to the Sutton Coldfield transmitter in England used cancer data for 1987–1994 [Cooper et al., 2001]. For children, a marginally significant decline in leukemia incidence with increasing distance from the transmitter was seen for boys, based on small numbers, but there was no trend for girls. In adults, there were no significant declines in risk with distance, although several types of leukemia showed increased risks in the whole area studied.

The mortality from adult leukemia and the incidence of childhood leukemia were assessed in Rome residents in relationship to the Vatican radio station [Michelozzi et al., 2002]. This has short (4–22 MHz) and medium wave (500–1600 kHz) radio transmitters of up to 600 kW power. There are no VHF or television transmissions. There was a statistically significant decline in leukemia mortality with increasing distance from the transmitters for men, but no association for women, and a nonsignificant decrease in risk for both sexes combined. For childhood leukemia, based on eight cases, there was a significant trend of risk decreasing with increasing distance. The authors concluded that “Although the study adds evidence of an excess of leukemia in a population living near high-power radio transmitters, no causal implications can be drawn.”

### **Occupational Studies: Cohort Study of Mortality of US Motorola Employees**

A cohort study of mortality of US Motorola employees included 195775 workers, of whom 44% were women and of whom 6296 died during the follow up period [Morgan et al., 2000]. Likely RF exposures were estimated for each of 9724 job titles and classified into background, low, moderate, and high, with relative levels of RF exposure estimated as 0, 1, 6, and 100. For the whole workforce compared to a general US population, the standardized mortality ratio (SMR) for all causes was 0.66, and for all deaths from cancer, it was 0.78, both significantly reduced. This is characteristic of the healthy worker effect. Of 60 specific causes of death assessed, only five SMRs were greater than one and the highest SMR was 1.28. For all employees, risks for cancers of the lymphatic/hemopoietic system and the

central nervous system (CNS) were both significantly reduced, with SMRs of 0.77 (95% confidence limits 0.67 – 0.89) and 0.60 (limits 0.45 – 0.78), respectively. Analyses for the 24621 subjects classified as having moderate to high RF exposure by peak exposure classification showed lower SMRs for all lymphomas and leukemias (SMR 0.54, limits 0.33–0.83) and for cancers of the CNS (SMR 0.53, limits 0.21–1.09).

The more informative analyses are within the Motorola workforce, comparing employees with higher RF exposures to those in the lower exposure or unexposed categories. Detailed analyses for cancers of the brain, all lymphatic and hemopoetic cancers, leukemia, nonHodgkin's lymphoma, and Hodgkin's disease showed no increased risks. The relative risk (RR) for the high exposure category, based on usual exposure, for brain cancer was 1.07 (95% confidence limits 0.32–2.66), for lymphatic and hemopoetic cancers was 0.70 (limits 0.27–1.47), for leukemia was 0.99 (limits 0.39–2.09), and for nonHodgkin's lymphoma was 0.58 (limits 0.12–1.74). For Hodgkin's disease, there were no cases in the highest exposure category, but for those in the high and moderate exposure categories for usual exposure the RR was 1.37 (limits 0.36–3.94) based on three cases. There was no excess risk comparing those above the median exposure with those with no exposure (RR 0.95).

As the authors point out, this study is limited by the use of a qualitative job exposure matrix rather than the ideal of having actual exposure measurements on each subject, and by the relatively young age of the cohort. The findings are not compatible with RRs of 3 or greater for brain cancers, lymphomas, or leukemias, and the authors note, "We did not observe indications of excess RR, but we cannot rule out the possibility of potential effects in the range of 1.5–2.0 RR." The results show no evidence of an increase in any specific cancer, although a small increase (or decrease) cannot be excluded. The exposure information is limited; the likely exposures of the various groups of workers are not defined, and no estimates of levels of exposures are given in the paper. If the maximum levels of exposure were well within current standards, the negative result is less informative. If an effect were specific to a particular type of RF exposure, the study would have less ability to detect it. These limitations are shared by all other studies yet done. The study is sufficiently powerful to reasonably exclude a substantial excess of leukemia or lymphoma in about 10 years from first occupational RF exposure in these workers. This time interval is not long enough to exclude an incidence effect, but it does provide substantial evidence against short term promotion effects, which have been suggested from some animal experiments.

### Further Follow Up of US Navy Cohort

A further follow up [Groves et al., 2002] of the cohort study of US naval personnel included 20000 men with maximum opportunity for exposure to radar emissions and 20000 subjects with a lower potential for exposure [Robinette et al., 1980]. All had graduated from US Navy technical schools in 1950–1954, and had served on US Navy ships at the time of the Korean War. The jobs of radioman, radarman, and aviation electrician's mate were assessed as likely to have relatively low radar exposures; the highly exposed groups were electronics technicians, aviation electronics technicians, and fire control technicians. Fire control and electronics technicians repaired and maintained radar related to gun fire control and search capacities, with the potential for exposures exceeding 100 mW/cm<sup>2</sup>, although their usual exposures were well below 1 mW/cm<sup>2</sup>. All the subjects were also exposed to 60 Hz fields from electrical equipment; electrician's mates, who repaired wiring, could have had high levels of exposure to such ELF fields. In addition, the three groups of technicians with high potential radar exposure and the aviation electrician's mates were involved in the maintenance and repair of electrical equipment and would likely have had considerable exposure to solder fumes, chlorinated solvents, and oils and greases. Aviation electronics technicians and aviation electrician's mates were involved in the maintenance and use of aircraft radar and electric equipment, and they may have had flying duties. For the entire cohort, compared to US white male death rates, the overall mortality ratio (SMR) was significantly reduced at 0.74 (95% confidence limits 0.73–0.76). Death rates were similarly reduced for most diseases and most cancers. This represents a healthy worker (or healthy sailor) effect.

The critical comparison is between the high radar and the low radar exposure groups. The men in the high radar exposure group had significant reductions in total mortality (RR 0.87), deaths from cancer (RR 0.80), and within cancers, deaths from lung and related cancers (RR 0.73). They also showed significantly reduced death rates from diabetes, heart disease, and noncancerous lung disease. Increased rates were seen in deaths related to war injuries and air accidents. Amongst the cancers, there was a significant excess death rate from all leukemias combined (RR 1.48, with confidence limits of 1.01–2.17), while deaths from brain cancer were nonsignificantly reduced (RR 0.65). These findings contrast to those of the first study, where total mortality, total cancer mortality, and deaths from all lymphatic and hematopoetic diseases were nonsignificantly increased, and there was a significant excess of

deaths from respiratory tract cancer (RR 2.2). Further analysis showed that the reductions in cancers were seen in each of the three high exposure groups, being significant in electronic technicians and aviation electronics technicians. The increase in total leukemias was restricted to the aviation electronics technicians, where it was significant with a RR of 2.6, and the increased risk was seen in most subtypes of leukemia.

In this study, the actual exposure levels are unknown, and RF exposures may have been substantial in both groups. There is no information available on lifestyle factors such as smoking or on the exposures or occupations of this group after their service experience. The increased leukemia mortality in the high exposure group contrasts with the overall results; it was virtually confined to aviation electronic technicians. The authors comment that aviation electronics technicians may have had more inadvertent or accidental exposure, as they dealt primarily with radar units in aircraft, and may have been more likely to get into the beam path of an operating radar than were men dealing with shipboard installations. Despite this, they regard the failure to see an excess of leukemia in either of the other two highly radar exposed groups as evidence against there being a causal link, and also caution about multiple comparisons. Their overall conclusion was "Radar exposure had very little effect on mortality in this cohort of US Navy veterans" (p. 810).

#### **Study of Occupational Exposures of Parents Related to Neuroblastoma in Offspring**

In a case-control study of parental exposures to radio frequencies [De Roos et al., 2001], information was obtained from 537 mothers of children with neuroblastoma and 503 control mothers in the US and Canada. There were no significant associations seen with maternal occupational exposures to RF sources. There were nonsignificant increased risks seen with occupations for which an industrial hygienist classified RF exposure as "probable" (odds ratio, OR, 2.8, 95% limits 0.9 to 8.7) and for cell phone exposure (OR 2.1, 95% limits 0.4–11.0, based on only seven exposed mothers). Data on 405 fathers of affected children and 302 controls also showed no significant associations with occupational exposures to RF. Data for individual types of equipment showed odds ratios over 2.0 for some paternal exposures, such as radio transmitters of under 7 W power (but not higher power transmitters), radar, and high frequency arc welding machines. These were all based on small numbers and were not statistically significant; also relevant is the issue of multiple exposures being assessed. ELF exposure was assessed in more detail, without any clear associations being seen.

## **STUDIES OF CANCER IN ASSOCIATION WITH THE USE OF CELLULAR TELEPHONES**

### **Overall Mortality of Cell Phone Users**

In the US, a group of over 255000 personal customers of a phone company in 1993–1994 in four urban areas were identified from phone company records [Rothman et al., 1996b]. Deaths in one year, 1994, were obtained by data linkage. For customers with accounts at least 3 years old, the ratio of mortality rates in 1994 for users of hand held cellphones, compared with users of the older large portable phones carried in a bag, was 0.86 (90% confidence interval 0.47–1.53). There was no increased risk of brain tumors or leukemias with greater use of hand held phones [Dreyer et al., 1999]. The short follow up time does not allow assessment of longer term effects.

### **General Population Cohort Study of Cell Phone Users in Denmark**

Johansen et al. [2001] carried out a prospective cohort study in Denmark, using the computerized files of the two Danish cell phone companies. Excluding corporate customers, there were 420 095 cell phone subscribers identified, with subscriptions starting since 1991. Of these, 58% used a digital Global System for Mobile communication (GSM) system at first subscription, with the remainder having an analogue Nordic Mobile Telephone (NMT) system. The standardized incidence ratio for all cancers was 0.86 (95% confidence limits 0.83–0.90) in men, and 1.03 (confidence limits 0.95–1.13) in women. For men, the incidence ratios of most smoking related cancers were reduced, while testicular cancer was nonsignificantly elevated (incidence ratio 1.12, 95% limits 0.97–1.30). For women, there were no significant differences; the incidence ratio for breast cancer was 1.08 (limits 0.91–1.26). Tumors of the CNS and leukemia were examined in more detail. There were no increased risks seen, and no trends apparent, with latency up to 5 or more years or with age at first subscription, and no differences were seen between analogue and digital telephones. There was no association with site of tumor within the brain, with incidence ratios for tumors of the temporal lobe of 0.86, frontal lobe 1.11, and parietal lobe 0.48, all nonsignificant. There was no increase in salivary gland tumors or leukemia. The pattern of incidence ratios is consistent with a distribution of mobile phone use characterized by higher socioeconomic status, and as a correlate, a lower rate of smoking. The study was not able to assess intensity of use and the average period of follow-up was only 3.1 years. However, the study provides considerable evidence against any large increase in risk within a few years of use [Johansen et al., 2001].

### Case-Control Studies of Brain Tumors and Cell Phone Use

**First study in Sweden: Hardell et al.** This first Swedish study [Hardell et al., 1999, 2000] included 209 subjects with brain tumors (malignant or benign) diagnosed in 1994–1996 and 425 population-based controls. Exposure was assessed by questionnaires supplemented by telephone interviews. Ever-use of a cell phone showed no association, odds ratio 0.98, 95% confidence limits 0.69–1.41. Dose-response assessment and the use of different tumor induction periods gave no associations, even at the highest levels of use and latency period (over 968 h of use, and over 10 years). In a later paper based on the same study [Hardell et al., 2000], a marginally significant increased risk for tumors in the temporal, occipital, or temporoparietal regions was shown where cell phone use was on the same side: RR 2.62 (95% confidence limits 1.02–6.71) after multivariate analysis. This result is based on one of many comparisons and on only 13 of the 209 subjects. The Stewart report [Independent Expert Group on Mobile Phones, 2000] concludes that the results of this study could easily have occurred by chance. Several other factors gave statistically significant increases in risk: occupation as a physician, in laboratory work, or in the chemical industry, and exposure to diagnostic radiology of the head and neck region. An increased risk was found only for analogue phones, but there were few data on digital phones. Hardell et al. [1999] quote high response rates to the questionnaire in this study: 90% for cases, 91% for controls, but comparison with the cancer registry for the area suggests that only 233 of 862 registered cases were approached [Ahlbom and Feychting, 1999]. Some of this discrepancy is because subjects who had died were excluded.

**Study in the US: Muscat et al.** Muscat et al. [2000] compared 469 patients with primary malignant brain cancers identified at five referral centers in the US to 422 inpatient controls in the same hospital with either benign conditions or cancer, excluding lymphoma or leukemia. The primary question was whether patients had ever used a hand held cell phone on a regular basis, defined as having had a subscription to a cell phone service. Analyses by years of use (up to 4 or more), hours of use per month (up to 10 or more), and cumulative hours (up to 480 or more), showed no excess risks and no significant trends. The RRs in the highest exposure groups, by each measure of exposure, were each 0.7. In this study, 80% of cell phones used were analogue. There were no significant associations with any brain site, with the RR for occipital lobe tumors being 0.8, temporal lobe 0.9, parietal lobe 0.8, and

frontal lobe 1.1. Subdivision by pathological type showed no significant associations, although the risk for neuroepitheliomatous tumors was 2.1 (95% limits 0.9–4.7), based on 35 cases. Of 41 cases who specified laterality of phone use and had a localized tumor, 25 reported ipsilateral relationships (i.e., phone use on the same side as the tumor) and 15 contralateral relationships ( $P = .06$ ). Of the 14 cases with temporal lobe cancer who used cell phones, five were ipsilateral and nine contralateral ( $P = .33$ ). This study shows no excess risks, even for the specific locations of tumors that were highlighted in the previous Swedish case-control study.

**Further US Study: Inskip et al.** A larger US case-control study involved 782 patients with malignant or benign brain tumors and 799 hospital controls with nonmalignant conditions [Inskip et al., 2001]. The RR associated with use of a cell phone for more than 100 h was 1.0 (95% limits 0.6–1.5) for all brain cancers, and 0.9 for glioma, 1.4 for acoustic neuroma, and 0.7 for meningioma; all nonsignificant. There was no evidence that the risks were higher with use of 1 h or more per day, or use for 5 or more years. There was no association with laterality, no increased risk for temporal, parietal or frontal lobe tumors, and no increased risk with specific subtypes of tumors. In contrast to the study by Muscat et al. [2000], the risk for neuroepitheliomatous tumors was 0.5 (95% limits 0.1–2.0), based on 25 cases. The authors conclude, “These data do not support the hypothesis that the recent use of hand held cellular phones causes brain tumors, but they are not sufficient to evaluate the risks among long-term, heavy users and for potentially long induction periods.” An accompanying editorial [Trichopoulos and Adami, 2001] comments that the limitations to the study are that the findings apply to predominantly analogue phones, do not assess risks which may occur after a considerable latency period, and cannot confidently exclude small increases such as RRs less than 1.5.

**Second case-control study in Sweden.** Hardell et al. [2002a] performed a further case-control study of patients with malignant or benign cranial tumors diagnosed in Sweden from 1997 to 2000, who were still alive at the time of the study (unstated), and population based controls, using a postal questionnaire supplemented by phone interviews. Response rates of 88% in cases and 91% in controls are given in the summary, but this only relates to questionnaire response. Of 2253 eligible cases, 1617 were approached (most of the rest, 540, being deceased) and 1429 responded to the questionnaire; the results are based on 1303 cases and their matched controls, representing 58% of the eligible cases identified. Information on cell phones was subdivided

into analogue (450 or 900 MHz), digital, and cordless phones. Multiple comparisons make the interpretation of this study difficult. The analysis assesses type of phone, hours of use, time since first use, and site, histology, and laterality of tumor, giving many comparisons; moreover, a further presentation of the results for malignant tumors is reported in another paper [Hardell et al., 2002b].

A small, but statistically significant, increased risk of any type of brain tumor was seen with the use of analogue phones, odds ratio 1.3 (95% confidence limits 1.02–1.6), increasing to 1.4 with more than 5 years latency and 1.8 with over 10 years latency. There was a trend toward higher risk with greater total hours of use at greater than 10 years latency, but the trend was in the opposite direction at over 5 years latency. For digital phones, there was no increased risk seen even at 5 years latency (OR 0.9, limits 0.6–1.5), with no data for 10 years latency. For cordless phones there was no overall association (OR 1.0, limits 0.8–1.2).

For analogue phones, the highest risk was seen for temporal lobe tumors (OR 2.0, limits 1.3–3.1); the OR was 1.9 at over 5 years latency and was 2.6 for over 10 years latency, both nonsignificant. For digital phones there was no increase for the temporal region overall (OR 1.0, limits 0.7–1.4), but the OR was 3.0 (limits 0.8–11.1) for over 5 years latency, based on only nine cases and three controls. Cordless telephones showed no associations.

For all sites, the risks were higher with ipsilateral use, with odds ratios of 1.8, 1.3, and 1.3 for analogue, digital, and cordless phones, respectively. The risks for contralateral use were 0.9, 0.8, and 0.7, respectively. If exposures from phones increase cancer risk, a stronger association with ipsilateral use would be expected, but there should be no decrease in risk with contralateral use. Indeed, some increase in the odds ratio with contralateral use might be expected because of misclassification of phone use: even those who report one sided phone use are likely to use the phone on the other side sometimes. This pattern of increased risks with ipsilateral use and decreased risks with contralateral use is what would be expected from recall bias. The case subjects would know they have a brain tumor, know which side it is on, and may preferentially report phone use on that side. The phone use data seem to be based on a single question, with no checks for reproducibility. Also, from exposure dose considerations, no effect would be expected with cordless phones and little or no effect would be expected in areas other than the temporal lobe. The data show, however, that the increased odds ratios with ipsilateral use are seen also for areas other than the temporal lobe, although for analogue phones the risk is highest for temporal lobe tumors.

An analysis by pathological subtype for temporal area tumors showed an association between acoustic neuromas and analogue phones with an odds ratio of 3.5, limits 1.8–6.8. The risk of meningiomas was similarly increased (OR 4.5, limits 1.0–20.8). There was no trend with latency period for either type. There was no association with malignant temporal area tumors (OR 0.8, limits 0.4–1.6).

In a subsequent paper [Hardell et al., 2002b] based on a slightly modified analysis of the data for malignant tumors already presented in the previous paper [Hardell et al., 2002a], the authors conclude that a significantly increased risk was seen with ipsilateral use of analogue cellular phones. This is based on a further analysis of laterality. Again, there are increased risks for ipsilateral phone use; odds ratios were 1.85, 1.59, and 1.46 for analogue, digital, and cordless phones, respectively. The odds ratios for contralateral use are 0.62, 0.86, and 0.89, with the association with total phone use being 1.13, nonsignificant and the same for each of the three phone types. These results again are consistent with recall bias in the assessment of laterality of phone use. Yet another paper again reports the data on both benign and malignant brain tumors from this study [Hardell et al., 2003b] with a slightly different analysis. The data on acoustic neuroma presented in Hardell et al. [2002a] is also repeated in another paper [Hardell et al., 2003a], which also shows a greater increase in the incidence of acoustic neuroma than of other brain tumors in the Swedish cancer registry between 1980 and 1998.

**Other studies of acoustic neuroma.** Muscat et al. [2002], in the context of their case-control study, identified 90 patients with acoustic neuroma and 86 controls; the participation and response rates are not detailed. There was no elevation in risk of phone use up to over 2.5 h per month or up to more than 60 total hours of use (OR 0.7, limits 0.2–2.6). With total years of use, in the maximum category of 3–6 years the OR was 1.7 (limits 0.5–5.1), but the trend was irregular and nonsignificant. Of 18 tumors with known laterality, five were ipsilateral and 13 contralateral.

Inskip et al. [2001] included data on 96 patients with acoustic neuromas. There was no association with hand held phone use, even in the maximum categories of average daily use or total cumulative use. Increased risks were seen with duration of regular use greater than 6 months (OR 1.8), and with first use prior to 1994, but these were nonsignificant and there were no regular trends. There were six ipsilateral cases and eight contralateral cases.

In the Danish cohort study [Johansen et al., 2001] there were seven cranial nerve tumors, most of which

would be acoustic neuromas, compared to an expected number of 11, so no increased risk was seen.

**Study of brain and salivary gland cancers in Finland.** In a case-control study using record linkage, 398 subjects with newly diagnosed brain tumors diagnosed in 1996 were compared to population controls [Auvinen et al., 2002]. Computerized linkage for personal subscriptions to the two cellular network operators in Finland in 1996 provided the exposure information, but no information on actual use of phones was obtained. Potential confounders of place of residence, socioeconomic status, and occupation were assessed. There was a nonsignificant odds ratio of 1.3 (95% limits 0.9–1.8) for ever having held a cell phone subscription. For the longest duration of subscription, over 2 years, the odds ratio was 1.5 (limits 0.9–2.5). Fifty percent of the brain tumors were gliomas, which showed a higher and statistically significant association with analogue phone subscription (odds ratio 2.1, limits 1.3–3.4). Subscriptions for analogue phones averaged 2–3 years. For digital, the average was less than 1 year; there were no increases in risk seen with digital phones. The results are consistent with a small increased risk for brain cancer, but the associations are weak and could be due to chance variation, misclassification, or uncontrolled confounding. However by being registry based and using record linkage rather than interviews, this study does avoid the problems of incomplete response to interviews and recall bias.

The study also included 34 subjects with salivary gland cancers, but this is too few for any meaningful results. The odds ratio with having had a phone subscription was 1.3, with wide confidence limits of 0.4–4.7.

**Studies of ocular melanoma and mobile phone use.** A case-control study of uveal melanoma in Germany assessed occupation in terms of likely RF exposure [Stang et al., 2001]. There was a significant association with radio sets or mobile phones, odds ratio 3.0 (95% confidence limits 1.4–6.3). Occupations were categorized as having ‘possible,’ or ‘probable or certain,’ mobile phone exposure. The risk for the ‘probable or certain’ category was 4.2 (95% confidence limits 1.2–14.5), but this was based on only six cases. The odds ratio for those exposed to radio sets was 3.3 (95% confidence limits 1.2–9.2) based on nine cases. There was no consideration of exposure to ultraviolet radiation, which is a risk factor for ocular melanoma [Inskip, 2001].

The hypothesis raised by this study was assessed in the Danish cohort study [Johansen et al., 2002]. Based on eight cases, the RR of ocular cancer was 0.59, with 95% confidence limits of 0.25–1.17, so no increase was seen. The authors also showed that the

incidence of ocular melanoma in Denmark from 1943 to 1996 had been relatively stable, with no increase following the rapid growth of cell phone use since 1982. Dolk et al. [1997b] assessed eye melanoma around the Sutton Coldfield transmitter, finding no trend with distance, based on 20 cases.

## DISCUSSION

The epidemiological evidence does not give clear or consistent results which indicate a causal role of RF exposures in human cancer. On the other hand, the results cannot establish the absence of any hazard, although some studies are powerful enough to exclude reasonably small increased risks in the health effects assessed [Elwood, 1999]. None of the studies give good information on individual levels of exposure. The studies of general populations living near radio or television transmitters relate to RF exposures likely to be below currently accepted standards, while the studies of military personnel and occupational groups may include some higher exposures.

The general population study in the UK [Dolk et al., 1997a] is sufficiently strong to reasonably exclude a geographical pattern with an excess of human cancers in subjects living close to large television and radio transmitters, although there is still a possible question in regard to adult leukemia. The Sydney and Vatican studies, while weaker, add other evidence on leukemias. The Motorola employees’ study [Morgan et al., 2000] is sufficiently powerful to reasonably exclude a substantial excess of leukemia or lymphoma in about 10 years from first occupational RF exposure in these workers. This follow up is not long enough to exclude an incidence effect, but it does provide evidence against a short term promotion effect. However there is no estimate of the actual exposures; the negative results are reassuring if the exposures are substantial, but not if the exposures are low. The further extension of the US Navy study gives data for up to 40 years after likely radar exposures, which may have been quite high, and shows no increase in most cancers, but a small increase in leukemia in one of the three high exposure groups [Groves et al., 2002]. Again, the lack of more precise exposure data is the main limitation. The ‘control’ group in this study may also have had levels of RF exposure higher than a general population. The large, population based study of mobile phone subscribers in Denmark [Johansen et al., 2001] also gives substantial evidence against there being any short term increases in cancer with typical levels of phone use experienced by residential subscribers. None of these large studies can provide good information on the intensities of exposure experienced by the people studied.

There are now five case-control studies and one cohort study published on brain tumors in relationship to personal use of mobile phones, showing mixed results (Table 1). The main limitation of these studies is the short follow up period between first exposure to mobile phones and incidence or mortality events. Consideration of the energy distribution within the head from use of hand held cell phones shows that energy absorption would be largely restricted to the side where the phone is held and close to the surface [Rothman et al., 1996a]. This area includes the skin, salivary gland, the nerve to the ear which is the site of origin of acoustic neuroma, and a small area of the surface and tissue of the brain, sites of origin of meningiomas and gliomas, respectively. The lobes of the brain (temporal, parietal, frontal, and occipital) are large areas; and energy absorption would be maximal in some areas of the temporal, but also adjacent areas of other lobes. The rate of energy absorption (specific absorption rate, SAR) for analogue phones is higher than for the more recent digital phones, and both are substantially higher than cordless phones. However, as analogue phones were used earlier, all available studies have considerably more information available on analogue phone use, making interpretation of differences between type of phone more difficult.

In terms of these considerations, the available studies show no consistent pattern. The second Swedish study [Hardell et al., 2002a] does show an overall increase in risk of brain tumors which is maximal for analogue phone use, temporal lobe tumors and (within the temporal area) acoustic neuromas, and with phone use on the same side, which are all consistent with exposure intensity considerations. However, all these associations are based on small numbers. The data on laterality of use are based on a single question about predominant use; the reductions in risks seen with contralateral use in the Swedish study are consistent with an effect of recall bias.

The only other study showing an overall increase in risk, although nonsignificant, is the Finnish study, which lacks any precise exposure information. The two US studies, which have the most detailed exposure information show no overall increase, and neither does the Danish cohort study, which has the advantage of a prospective design. Neither of the US studies nor the Danish prospective study show any higher risk with temporal tumors or with acoustic neuroma. Neither did they show any association with phone use on the same side; in fact for acoustic neuromas there is a trend towards a stronger association with contralateral phone use. Muscat et al. [2002] point out that acoustic neuromas grow slowly and that there could be hearing loss in the affected ear in the years preceding

TABLE 1. Studies of Brain Cancer in Users of Mobile Telephone

First author	Reference	Year	Numbers of		Overall association; odds ratio, 95% limits			Analogue phones; odds ratio, 95% limits			Association for high exposure; odds ratio, 95% limits			Temporal lobe tumors; odds ratio, 95% limits				
			Cases	Controls	OR/RR	Lower	Upper	OR	Lower	Upper	OR/RR	Lower	Upper	OR/RR	Lower	Upper		
Hardell	(Hardell et al., 1999, 2000)	1999, 2000	209	425	1.0	0.7	1.4	0.9	1.4	0.6	1.4	Latency > 10 years; use > 968 h	1.1	0.3	3.4	1.55	0.8	3.0
Muscat Inskip	(Muscat et al., 2000) (Inskip et al., 2001)	2000 2001	469 782	422 799	0.85 1.0	0.6 0.6	1.2 1.5	0.88% analogue (Primarily analogue)				> 480 h > 500 h	0.7 0.7	0.3 0.2	1.4 1.1	0.9 0.8	0.5 0.5	1.7 1.4
Johansen	(Johansen et al., 2001)	2001	Cohort		0.95	0.79	1.12				> 5 years subscript (both sexes)	1.0	0.7	1.6	0.9	0.4	1.5	
Auvinen	(Auvinen et al., 2002)	2002	135	19	1.03	0.62	1.61				> 2 years	1.5	0.9	2.5				
Hardell	(Hardell et al., 2002a) Analogue phones	2002	398	1990	1.3	0.9	1.8	1.6	1.1	2.3								
			1303	1303	1.2	1.0	1.6	1.2	1.0	1.6								
	Digital phones				1.1	0.8	1.2											
												Latency > 10 years; use > 85 h	1.9	1.1	3.2	2.0	1.3	3.1
												Latency > 5 years; use > 55 h	1.1	0.6	1.9	1.0	0.7	1.4

diagnosis, perhaps leading to phone use in the other ear. Better case-control studies with great attention to the estimation of exposure and documentation of other relevant factors are now in progress to assess further any link between brain cancer and cell phone use, including those in the database of the World Health Organization International EMF project: ([www.who.int/peh-emf](http://www.who.int/peh-emf)).

The two types of tumor which have been of most concern with respect to radio frequencies are brain tumors (benign and malignant) and leukemias. In addition to the cell phone studies, increased risks of tumors of the brain in association with RF exposure in adults were shown in the Polish military study and in the small case-control study in the US military. No association was found in the study of electric utility workers or the Sydney study, and no clear association was seen in the Sutton Coldfield study. There was no association

with tumors of the brain in childhood in any of the three studies which assessed this.

The epidemiological studies of leukemia and RF exposures are difficult to interpret. The results show little consistency, but several show some association (Fig. 1). There have been to date no detailed case-control studies on leukemia in regard to radio frequencies using adequate measures of exposure. For low frequency ELF fields, it is the case-control studies of childhood leukemia, for example [Lin et al. 1997; UK Childhood Cancer Study Investigators, 1999], which have been most informative, even though there are still difficulties in estimating past exposure. The case-control study of neuroblastoma is a high quality study using good methods, but the published paper concentrates on ELF fields and has much less information on radio frequencies. If there is indeed a real association between childhood leukemia and ELF fields, an apparent association

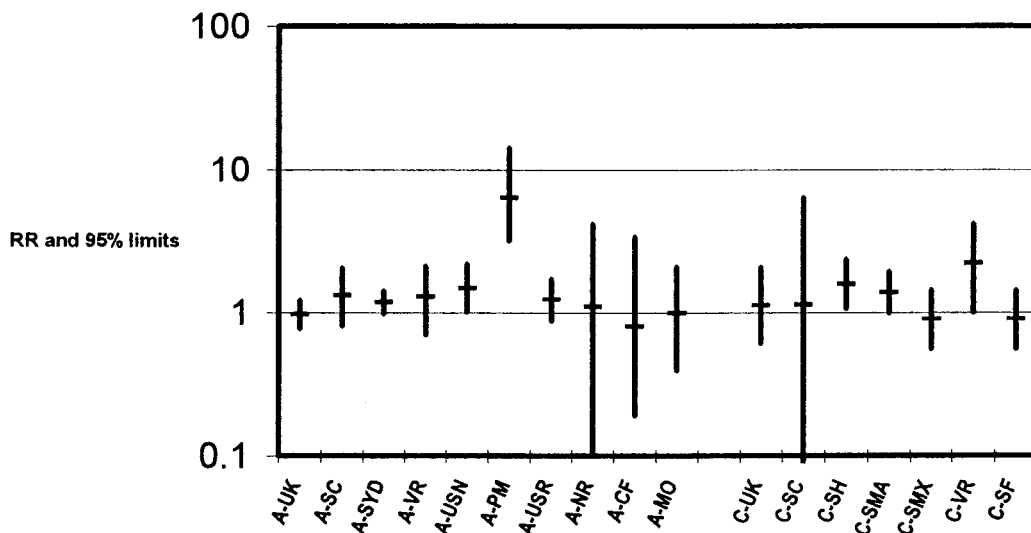


Fig. 1. Summary of results (relative risk or odds ratio and 95% confidence limits) of studies assessing leukemia in association with radio frequency exposure. Results are shown for the highest exposure group in each study.

Key to studies:

- A, adults
- A-UK, UK 21 transmitter studies [Dolk et al., 1997a]
- A-SC, Sutton coldfield 1987–1994 [Cooper et al., 2001]
- A-SYD, Sydney [Hocking et al., 1998]
- A-VR, Vatican (6 km) [Michelozzi et al., 2002]
- A-USN, US Navy [Groves et al., 2002]
- A-PM, Polish military [Szmigielski, 1996]
- A-USR, US amateur radio operators [Milham, 1988]
- A-NR, Norwegian radio operators [Tynes et al., 1996]
- A-CF, Canada–France utility workers [Armstrong et al., 1994]
- A-MO, Motorola employees [Morgan et al., 2000]

- C, children
- C-UK, UK 21 transmitter study [Dolk et al., 1997a]
- C-SC, Sutton coldfield 1987–1994 [Cooper et al., 2001]
- C-SH, Sydney study [Hocking et al., 1996]
- C-SMA, Sydney study (all areas) [McKenzie et al., 1998]
- C-SMX, Sydney study (excluding Lane Cove) [McKenzie et al., 1998]
- C-VR, Vatican (6 km) [Michelozzi et al., 2002]
- C-SF, San Francisco [Selvin et al., 1992].

with RF fields may occur simply from a correlation between exposures to ELF fields and to radio frequencies.

### Implications of Epidemiological Studies for Exposure Standards

Epidemiological studies primarily relate to the question of whether there is or is not an increased risk of disease in human populations exposed to the suspected causative agent. The available epidemiological work is not very helpful in defining a particular level of RF exposure that could be hazardous. Equally, the epidemiological evidence does not lead to an argument for any particular changes in currently accepted exposure standards.

The epidemiological studies reviewed here do not suggest that currently accepted exposure standards, such as that of ICNIRP, need to be revised downwards. The overall conclusion from the literature is that no detrimental health effects have been observed consistently in studies which are assessing exposure levels which are likely to be within the current standards or which may have occasionally been beyond those standards, for example in the occupational studies. As is expected in any area of work where there are numbers of studies, some making multiple observations, there are some positive associations reported: but the lack of consistency shows that these are more likely to be due to chance variation, biases in the observations made in the study, or the effects of other related factors, than due to a causal association with RF exposures. The generally negative experimental evidence on markers of serious effects, for example in vivo and in vitro indicators of carcinogenesis, also argue against there being any cancer causing effects at very low levels of exposure [Royal Society of Canada, 1999; Independent Expert Group on Mobile Phones, 2000]. The exposures to the head in users of mobile phones are higher than general environmental exposures, but the current epidemiological studies do not show any consistently increased risk of brain tumors. However, they are based on short follow up times, and include few tumors in the small exposed area of the brain.

### CONCLUSION

The epidemiological results fall short of the strength and consistency of evidence which is required to come to a conclusion that RF emissions are a cause of human cancer. Although the epidemiological evidence in total suggests no increased risk of cancer, the results cannot be unequivocally interpreted in terms of cause and effect. The results are inconsistent; and most studies are limited by lack of detail on actual exposures, short follow-up periods, and the limited ability to deal with other relevant factors. In some studies, there may be substantial

biases in the data used. For these same reasons, the studies are unable to confidently exclude any possibility of an increased risk of cancer. Further research to clarify the situation is justified. Priorities include further studies of leukemia in both adults and children, and of cranial tumors in relationship to mobile phone use.

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