RADIOFREQUENCY RADIATION FROM BROADCAST TRANSMISSION TOWERS AND CANCER: A REVIEW OF EPIDEMIOLOGY STUDIES

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EXECUTIVE SUMMARY

San Francisco's Sutro Tower is the site of the majority of the city's radio and television broadcasting transmitters. Although measures of radio-frequency radiation (RFR) in public areas around Sutro Tower, have been consistently lower than the 1997 United States Federal Communications Commission safe exposure limit, area residents have expressed concerns that exposure to radiofrequency radiation from the transmitters may harm their health. Comprehensive literature reviews, including those by the World Health Organization, the Royal Society of Canada, and the International Commission on Non-Ionizing Radiation Protection, have concluded that RFR is not harmful below established thresholds. However, prompted by residents concerns, the San Francisco Department of Public Health critiqued peer-reviewed epidemiological evidence on the relationship of cancer and RFR with regards to broadcast towers and occupational exposures.

TOWER STUDIES

Six peer reviewed published studies assessed the association between cancer rates and nearby broadcast towers (see Table II of the report). Each of the studies had limitations. Overall, among the positive studies, the findings are not internally consistent across the population studied, or across time or distance. The Great Britain and Australia studies were not also externally consistent among types of cancer. Because of weaknesses, current evidence does not suggest that living near broadcast towers would lead to an increased risk of cancer.

- Selvin et al. (1992), used 15 years of cancer registry data for San Francisco and did not find evidence to suggest that the geographical distribution of childhood cancers was concentrated around Sutro Tower.
- Maskarinic et al., (1994) found that the odds of getting childhood leukemia was 2.6-fold (95% C.I.= 0.6-8.) greater among children living within 2.6 miles of a radio broadcast tower in Oahu Hawaii,; however, leukemia rates were only elevated for a two year period after which they returned to expected levels.
- Hocking et al. (1997) found that childhood leukemia incidence was 58% greater (95% CI= 1.07-2.34) and adult leukemia rates were 24% greater (95% CI= 1.09-1.40) in three communities close to broadcast towers in Sydney, Australia in comparison to non exposed communities. However, a re-analysis by Mckenzie and Morrell (1998) using 3 additional control communities and distance-based continuous estimates of RFR-exposure, showed that the earlier results reflected high rates of leukemia in one broadcast tower community, and other similarly exposed broadcast tower communities had the expected rates of leukemia.
- Dolk et al. (1997a, 1997b) examined how cancer rates changed with distance from broadcast towers in Great Britain. The first study looked at a single tower and found rates of adult leukemia 83% higher (95% CI=1.22-2.74) for those living within 2 km of the tower. A later extension of this study, examining childhood and adult leukemia rates among 3.4 million people living near the remaining 20 broadcast transmitters in Great Britain, found that adult leukemia rates were 3% higher than expected (95% CI, 1.00-1.07) within 10 kilometers of the towers and decreased with distance. Notably, rates were not elevated in the residences closest to the transmitter. Furthermore, the authors found that most of the excess leukemia cases were found near 1 of the transmitters. Childhood leukemia rates were not elevated either in the first or second study.

OCCUPATIONAL STUDIES

We found eleven peer reviewed published studies of associations between RFR exposure at work and cancer (Table III). Many of the studies looked at more than one subtype of cancer. One should note that when many sub-types of cancer are assessed in a single study without a strong a priori hypothesis, there is a greater likelihood that a statistically significant elevation in one or more subtypes may be found

by chance alone. While several of the occupational studies had positive findings for at least one type of cancer, we noted significant problems (sometimes noted by the authors) that limited the interpretation of the results. Overall, despite some provocative findings, due to the lack of consistency among cancer subtypes and the methodological weaknesses of many of the positive studies, the occupational epidemiology literature does not provide support for a link between RFR and cancer.

- Szmiegelski (1996) found associations between RFR and several kinds of cancer; however, the study acquired more complete exposure information on subjects with cancer than subjects without cancer biasing the results toward finding an association.
- Grayson (1996) reported a 39% increase in the odds of brain cancer (95% CI = 1.01-1.90) in “ever" vs. "never" exposed among United States Air Force servicemen; however, when highly exposed servicemen were compared to those with lower exposure the evidence for an exposure-response relationship disappeared.
- Thomas (1987) found an association between brain cancer and RFR but it was limited to workers who were also exposed to extremely-low frequency radiation (also called EMF).
- Hayes (1990) found an association of occupational exposure to RFR and testicular cancer based on self-reported exposure that was not replicated using a more objective assessment of exposure. Tynes et al. (1996) studied cancer in a female cohort of radio and telegraph operator finding an elevated rate for breast cancer (SIR=1.5, 95% CI = 1.1-2.0) among exposed workers. However, when rates in this cohort were compared with other women who worked at sea but were not RFR-exposed, breast cancer incidence was also elevated, indicating that other factors such as chronobiological disturbances and delayed fertility may have better explained the elevated breast cancer rates.
- Morgan et al. (2000), a well conducted study comparing the mortality rates for brain and lymphatic and hematopoietic cancers (including leukemia) among exposed and unexposed workers using several different measures of exposure and did not find evidence of an RFR—cancer relationship among workers for any subtype of these cancers.

CONCLUSION

In summary, this review has not found substantial epidemiological evidence to support an association of radiofrequency radiation with cancer. Furthermore, two prior San Francisco childhood cancer studies found no evidence of childhood cancer clusters in the neighborhoods surrounding the tower. The association of adult and childhood cancers and broadcast tower RFR exposure do not appear to be supported by the epidemiological literature, therefore studies of neighborhoods near the tower are not warranted at this time. Further more, studies of rare cancers such as leukemia, brain cancer, and testicular cancer would be severely limited by the many years of data required, the challenge of estimating lifetime exposures among migrating populations, and the lack of historical exposure measurements. Many of these questions may be best answered by further studies in other exposed populations. To help respond to concerns of residents living near Sutro Tower, the San Francisco Department of Public Health recommends that data from the Northern California Cancer Registry and the 2000 Census be used to identify any temporal and geographical clustering of cancer in San Francisco.

OVERVIEW AND OBJECTIVES

In, 1997, the United States Federal Communications Commission set the radiofrequency radiation (RFR) standard for this country based on the threshold limit for biological thermal (heating) effects in the most sensitive tissues. This limit is currently believed to be at a specific energy absorption rate (SAR) of 4 W/kg. The occupational exposure standard (regulated by OSHA) is 10-fold lower than the threshold limit (0.4 W/kg) and whole-body general population exposures are limited to $1/50^{th}$ of the threshold (0.08 W/kg). Since each wavelength of RFR is absorbed differently by the body, power densities that correspond with an SAR of 0.08 W/kg vary by wavelength. An SAR of 0.08 W/kg is equivalent to power densities of 200 $\mu W/cm^2$ at 30 to 300 MHz, 667 $\mu W/cm^2$ at 1000 MHz and 1000 $\mu W/cm^2$ at 1.5-300 GHz. These standards are reflected in the conclusions of the American National Standards Institute and the National Council on Radiation Protection and Measurements (NCRP).

Several other agencies including the International Commission on Non-Ionizing Radiation Protection (ICNIRP), the World Health Organization, and government agencies in England, Canada and Australia have reviewed radiofrequency literature (see Appendix I for a list of these reports). All of these studies concluded that RFR is not harmful below levels that produce thermal effects. Thermal-effect based standards have provoked controversy because some scientists believe there is evidence for non-thermal effects. If non-thermal effects are demonstrated to exist, then current FCC standards would not necessarily be health protective.

This review critiques epidemiological evidence for the relationship of radiofrequency radiation (RFR) exposures below the current United States RFR standard and cancer. This review was requested by the San Francisco County Board of Supervisors Sutro Tower Task Force and residents living near Sutro Tower who have concerns that exposure to the tower might result in elevated rates of cancer in neighborhoods around the TV/radio tower.

Sutro Tower emits radiofrequency radiation (RFR). RFR is a form of electromagnetic energy characterized by the range of frequencies, 1 MHz to 3 GHz. Included in this range are AM radio transmission (~1 MHz), FM radio transmission (~100 MHz), television (54-216 MHz), cellular phones (900 MHz), PCS phones (1800-2200 MHz) radar and microwaves (2450 MHz) (see Table I)[1] (FCC Report).

Table I. Sources of extremely low frequency and radiofrequency radiation

Source	Frequency (MHz)	Type	Power Density Limit ($\mu W/cm^2$)
Video Display Terminal	0.015-0.3	ELF*	No U.S. Standard
Power Lines	0.06	ELF*	No U.S. Standard
Dielectric Heater	1-100 (usually 27.1)	RFR[1]	245
FM Radio	88-108	RFR[1]	200
VHF Television	54-72, 76-88, 174-216	RFR[1]	200
UHF Radio	470-890	RFR[1]	313-593
Dish Antenna	800-15,000	RFR[1]	533-1000
Cordless Telephone	46-49	RFR[1,2]	200
Cellular Telephone	824-850, 900	RFR[1,2]	549-600
Traffic Radar	10,500 and 24,000	RFR[1]	1000
Microwave Oven	915 and 2,450	RFR[1]	610-1000

* ICNIRP has recommended 60 Hz recommended limits for 24-hour exposures. They are: electric field, 5kV/m; magnetic field, 1 mG (IRPA/INIRC, 1990). [1] (Cleveland and Ulcek, 1999); [2] Power density limits listed are for whole-body exposures.

Radiofrequency radiation is non-ionizing. Unlike ionizing radiation such as x-rays, RFR does not have the power to break chemical bonds and damage genetic material. RFR is also very different in nature from "extremely low frequency" electromagnetic radiation (ELF), sometimes called EMF. ELF is on the least energetic part of the electromagnetic spectrum (below 1 MHz) and includes power lines, radios, televisions and many household appliances. Unlike RFR, ELF cannot heat tissue and radiates

very little energy. Because RFR has very different biophysical properties from ionizing radiation and ELF, this literature review exclusively examines epidemiology studies of the effect of radiofrequency exposure.

Concern over health effects of RFR have been associated with new technologies such as radar guns, microwave ovens and, most recently, cellular and PCS telephones and the resulting placement of cellular phone antennas in communities. Several recent studies and reviews have exclusively addressed the health effects of cellular phone use. However, RFR exposures from cellular telephones are different in a variety of ways from exposure to TV/radio waves from fixed antennas: (1) Absorption of the frequencies commonly used in cellular phones is poorer than that of TV and FM radio; (2) Cellular telephones result in short-term exposures very close to the head while broadcast tower exposures are of much longer duration and at lower levels; (3) Cellular telephone exposures are a personal choice; TV/radio antenna exposures are not.

The available literature and thus this review focuses on cancer and includes two general types of epidemiological studies: occupational studies and studies of people who reside near TV/radio antenna towers. Since the nature of cellular telephone exposure is different from that experienced occupationally or by residents near broadcast towers, studies that exclusively focus on health effects from cellular telephone use have not been included.

Other health effects that have been studied in relation to RFR exposure include heart disease, neurological, cognitive and reproductive effects. For the most part, these studies have also been negative and have mostly focused on exposures to cellular phones. They are not part of this review but several of the reviews listed in Appendix I do discuss these studies. Animal studies are briefly summarized first.

This review is restricted to the potential human health effects of RFR exposures below the current United States standard.

EXPERIMENTAL STUDIES IN ANIMALS

Recent animal and cellular studies of the carcinogenicity or genotoxicity of RFR are summarized in Appendix II. Although there are some animal toxicology studies that demonstrate carcinogenic or genotoxic effects below the threshold for thermal effects, results are inconsistent and no clear mechanism has been shown. Consistency is important because positive results can occur by chance and some species may be more prone to biologic effects than others. Examples of inconsistency are studies which have been done in cancer-prone species of laboratory animals but have not been repeated in "normal" animals. Other positive studies have results that could not be replicated. Published reviews of animal studies include Repacholi (1997, 1998), Verschave et al (1998), Brusick et al. (1998), and Valberg (1997). Szmigielski et al. (1988) in a review of the laboratory evidence for immunologic and cancer endpoints concluded, "Most of the observed effects are inconsistent, transient, and difficult to confirm and interpret." Similar conclusions have been reached by most of the above reviews.

EPIDEMIOLOGY OF CANCER DUE TO RFR FROM BROADCAST TOWERS

Several communities in the vicinity of television / radio broadcast towers have been the subject of epidemiological studies. These studies are most directly relevant to the community around Sutro Tower since frequencies were often similar to those found in San Francisco, and exposures in these studies were below current United States RFR limits. Some of these studies were prompted by cancer clusters in neighborhoods near towers. Cancer cluster investigations have been important in generating interest in potential carcinogenic effects of RFR below the current standard but are not useful, in themselves, in providing evidence for a causal association of RFR with cancer. Studies that control for potential confounding factors, consider exposure/response relationships and observe effects across a range of RFR frequencies are more useful for determining causal relationships.

Maskarinec G, Cooper J, Swygert L, et al. Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations. J Environ Pathol Toxicol Oncol 13:33-37, 1994.

This childhood leukemia case-control study was prompted by detection of a childhood leukemia cluster in the vicinity of radio broadcast towers in Oahu, Hawaii and an unpublished 1986 report of elevated rates of cancer in census tracts with broadcast towers (Anderson and Henderson, 1986). The towers in the vicinity of the cancer cluster broadcast radio signals of 23 kHz.

Twelve children under the age of 16 diagnosed with leukemia from 1979 to 1990 who lived on the Waianae Coast of Oahu were matched with 4 age- and sex-matched controls. Several risk factors were explored including distance from nearby radio broadcast towers as defined by residence within 2.6 miles of one of the towers. Interviewers were not blinded to case/control status.

The authors found that the odds of getting leukemia was 2.6-fold greater among children living within 2.6 miles of a radio broadcast tower, although the odds ratio was not statistically significant (95% C.I.= 0.6-8.3). Also, elevated leukemia rates were restricted to a 2-year period in the early 1980's, after which leukemia rates returned to expected levels. Exposures from the towers did not decrease after 1984 indicating that RFR exposures did not explain the cluster. These analyses were not adjusted for socioeconomic bias. The incidence of leukemia has been shown to increase with socioeconomic status in several other studies. Also, since interviewers were not blinded, these results are potentially biased upward.

Hocking B, Gordon I, Grain HL, Hatfield GE. Cancer incidence and mortality and proximity to TV towers. Med J Aust 1996; 165(2):601-605.

Hocking et al. (1996) examined cancer incidence and mortality from 1972 to 1990 in residential areas near 3 TV towers in Sydney. Power density based exposures were modeled using a formula recommended by the National Council on Radiation Protection. Modeled exposures were 1 $\mu W/cm^2$ at the center of the 3 towers, reached a maximum of 8$\mu W/cm^2$, and reduced to 0.2 $\mu W/cm^2$ at 4 km, well below the Australian standard of 0.2 mW/cm^2. Actual power densities as measured by the Commonwealth Department of communications were up to 5 times lower than the modeled exposures. Instead of either of these measures, residential distance from the tower was used as a proxy for exposure. The authors compared adult and childhood leukemia rates in 3 "inner" areas within a 4 km radius of the towers with six "outer" neighborhoods more distant from the towers. "Outer" communities were selected because of similar upper middle classes socioeconomic status (SES); childhood leukemia incidence is often greater in communities with higher SES. Poisson models adjusted for age group, sex, and calendar period were used to compare incidence in the tower and "control" areas. SIRs and SMRs were calculated to compare cancer incidence and mortality in the study area to all of New South Wales (NSW). Cancer rate ratios in all ages and in children 0-14 years were examined.

No increased brain cancer incidence or mortality was found. However, incidence rates of leukemia were higher in the 3 areas close to the tower. The relative rates were higher especially among children. In this group, leukemia incidence rates were 58% higher (95% CI= 1.07-2.34) with very similar findings for acute lymphocytic leukemia (1.55, 1.00-2.41), a common type of leukemia in children. In the all ages analysis, the rate ratio for total leukemia was 1.24 (95% CI,: 1.09-1.40). Other analyses found no evidence of heterogeneity between municipalities or trends in incidence across the three time periods. Results did not change when residences close to the site of a factory which had used radium were omitted.

Although the authors claim that they adjusted for age differences between "inner" and "outer" communities, only broad age groups were used (0-14, 15-69 and 70 and over). It is standard to use 10-year age groups. Socioeconomic status was a potential confounding factor not explicitly controlled for in the analysis although the authors point out that the "outer" and "inner" areas contained the same proportion of communities in the top quintile of SES, and, there was no evidence for increased rates of leukemia with higher SES in New South Wales. Furthermore, the authors did not attempt to use declining distance from the source to get a continuous or more refined measure of exposure. The

rationale for including leukemia mortality data is unclear since incidence data were available and mortality may be influenced by factors other than exposure. However, in the case of childhood leukemia, the relative rate for mortality was much higher in communities closer to the tower. Within leukemia type, there is no sensible reason why RFR would effect mortality. An alternative explanation would be poorer health access in communities closer to the tower.

McKenzie DR, Yin Y and Morrell S. Childhood incidence of acute lymphoblastic leukemia and exposure to broadcast radiation in Sydney – a second look. Aust N Z J Public Health 1998;22:360-367.

The Sydney study was reanalyzed by McKenzie and Morrell (1998) using additional communities and continuous RFR-exposure estimates, based, in part, on distance from the tower to see if a dose-response relationship existed. Hocking et al. had excluded 6 communities near the 3 towers because they were affluent. McKenzie and Morrell used as controls for the more affluent areas, 6 areas south of Sydney matched for socioeconomic status using an index constructed from several socioeconomic variables on the Australian census. Annual population estimates in five-year age groups also collected by the registry were used as denominators to calculate age-adjusted incidence rates. Hocking et al. had used only broad age group for age adjustment.

The reanalysis included descriptive graphs that looked at the change in leukemia rate with the change in exposure. These graphs showed that the earlier results were driven by one community with high RFR exposure and high rates of leukemia. Other similarly-exposed communities had rates of leukemia equivalent to those found in non-RFR exposed neighborhoods. McKenzie et al. (1999) later noted that there was statistically significant heterogeneity of effect in the exposed communities (using a chi-square test). Therefore, effects were felt to be inconsistent with an effect of radiofrequency radiation.

Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt I and Elliott P. Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield Transmitter. AJE 145(1):1-9, 1997.

Hocking et al.'s study of cancer rates around TV/radio towers in North Sydney was followed shortly by two analyses of such communities in Great Britain. Dolk et al. investigated the community around the Sutton Coldfield TV/radio transmitter in Great Britain, partly in response to a presumed adult leukemia cluster publicized by the media. Rates of several cancers were examined including: hematopoetic and lymphatic cancers, leukemia, brain, lung, breast, colorectal and prostate cancer. In children, all cancers and all leukemias were studied. Postcoded data were used to look at the rate of cancer in the residences around the tower from 1974 to 1986. Personal RFR exposure was estimated using distance from the transmitter. The study area included residences from 0.5 to 10 km from the tower using 10 bands with increasing distance from the tower. The maximum total power density summed across frequencies at only 1 measurement point at 2.5 m above the ground was 0.013 W/m^2 for television and 0.057 W/m^2 for FM radio.

Expected cancer rates were calculated from national incidence rates stratified by 5-year age group, sex, year and deprivation quintile (a measure of socioeconomic status). Stone's statistic, which assesses a decline in risk with distance from a source, was used to examine the potential effect of the tower on cancer rates in nearby communities. The background variability of leukemia in the larger region was also examined.

Childhood cancers tested were not elevated. Rates of adult leukemia were 83% higher (95% CI=1.22-2.74) in those living within 2 km of the transmitter. However rates fell below expected levels in residences from 2.1 - 10 km from the tower.. Occupation of the cases did not seem to explain the excess and rates were equally elevated when the study was divided into two 6 year periods. The authors attributed elevated rates of skin melanoma to higher affluence in the area nearest the transmitter; skin cancer is higher in affluent communities. A finding of elevated bladder cancer rates was attributed to chance. The authors concluded that the elevated adult leukemia rates were not explained by the

publicized cluster since the study period ended before the cluster was identified and, the higher rates observed near the transmitter did not change with calendar period.

Dolk H, Elliott P, Shaddick G, Walls P, Thakrar B. Cancer incidence near radio and television transmitters in Great Britain. II All high power transmitters. AJE 145(1):10-17, 1997.

The authors next repeated their study examining childhood and adult leukemia rates, brain, skin and bladder cancer among 3.4 million people living near the remaining 20 broadcast transmitters in Great Britain. The transmitters were divided into 4 transmission groups: highest power TV transmitters, all TV transmitters, all FM transmitters and all transmitters with both FM and TV transmission.

No elevations in rates for skin, bladder, or childhood cancers were found. Within 10 kilometers of the towers, adult leukemia rates were 3% higher than expected (95% CI, 1.00-1.07) and decreased with distance; however, rates were not elevated in the residences closest to the transmitter. Furthermore, the authors found that most of the leukemia cases were found near 1 of the transmitters. Finally, transmission group did not appear to be important.

The observed excess rate was much smaller than what was observed in the Sutton Coldfield study (83%). Given that the small excess rate was due largely to the number of cancers around one transmitter, the author's interpreted this study as giving at most weak support for a true association of RFR and adult leukemia.

As with the other broadcast tower epidemiology studies, distance was used as a proxy for exposures; actual exposure measurements were not used. However, several categories of distance were used around 20 transmitters to give a range of exposures and the ability to look at dose/response. Second, observations with unusual influence on the results were studied. Third, since all broadcast transmitters within Great Britain were used, the authors were able to examine potential differences in health effects within the RFR spectrum.

SAN FRANCISCO STUDIES

Selvin S, Schulman J, Merrill DW. Distance and risk measures for the analysis of spatial data: a study of childhood cancers. Social Science and Medicine 34(7):769-777, 1992.

Selvin et al. (1992) examined whether Sutro Tower explained the geographical distribution of childhood cancers in San Francisco after adjustment for population density. The authors defined someone as exposed if they lived within 3.5 km of the tower. Incident cases of leukemia, brain cancer and lymphatic cancer in children were studied. The authors did not adjust for age, sex, ethnic group or socioeconomic status and did not measure exposure levels around the tower. After adjustment for population density, the authors did not find any evidence of geographic variation of childhood cancer rates in relation to distance from the tower, i.e. no clustering of childhood cancers.

Taylor F, Study of San Francisco residents 0-4 years of age diagnosed with cancer between 1981-1987. Report to the San Francisco Department of Public Health, 12/14/89. Unpublished.

From 1973 to 1985, the San Francisco Department of Public Health observed a greater number of leukemia cases than expected among children aged 0-4 in Noe/Eureka Valley. The increase was due to an excess of leukemia in the 0-4 age group between 1973 and 1988. Data were collected for all San Francisco children aged 0-4 years diagnosed with any type of cancer between 1981 and 1987 to see where they lived from the time of their birth to diagnosis, and what risk factors they might share. Data were also collected for children aged 4 to 15 years diagnosed with cancer between 1981 and 1987 while living in the Noe/Eureka Valley area. The increased incidence of childhood cancer did not continue after 1985. The report concluded, "The results of this study do not suggest that any residential areas, pre-schools or child care facilities, out of door playing areas, or occupational sites or water supplies are associated with significant proportions of cancers diagnosed in children 0-4 years in San Francisco between 1981 and 1987". Unfortunately response rates were very low (55%).

It is important to note that a cluster of childhood cancer near the tower would have been detected in this study since all incident childhood cancers were part of the study population. In addition, since residential history for each cancer case was explored, there appears to have been no cluster of children who *lived* near Sutro Tower and then moved to another San Francisco neighborhood from 1981 to 1987. The tower had been operating for a sufficient number of years to see an effect of RFR from the tower on childhood cancer had one existed.

SUMMARY OF GENERAL POPULATION AND BROADCAST TOWER EPIDEMIOLOGY STUDIES

The tower epidemiology studies are summarized in Table II. The primary weakness of each of these studies is a paucity of RFR exposure data. Each of the authors of the broadcast tower studies have taken a similar and sensible approach to exposure estimation, using distance from a tower. Three studies used continuous exposure estimates allowing them to examine potential exposure/response relationships (Mckenzie and Morrell, 1998; Dolk et al.1997a, 1997b).

Three of the five published studies found statistically significant elevations in cancer rates in communities residing near towers. However, a comparison of these studies reveals many inconsistencies. For example, Hocking et al. found that childhood leukemia incidence was 58% greater (95% CI= 1.07-2.34) in communities close to the towers. Adult leukemia rates were 24% greater (95% CI= 1.09-1.40). Dolk et al. found only a 3% excess rate of adult leukemia (95% CI=1.00-1.07) in Great Britain communities close to TV/radio towers and no association of the towers with childhood leukemia. In addition, exposures were *higher* in the Great Britain studies than in the Australia study. Finally, in the re-analysis of North Sydney data by McKenzie and Morrell (1998), effects were found to be driven by only one of the 3 RFR-exposed areas.

Also, the three studies that were able to assess exposure/response found no such relationships. Dolk et al. (1997a,1997b) compared trends in leukemia rates with increasing residential distance from towers. In their study, adult residents living closest to the tower had lower rates than those who lived slightly further away. McKenzie and Morrell's (1998) modeled RFR exposures appeared to be unrelated to leukemia rates. Thus, while the study of all high frequency broadcast towers in Great Britain is the strongest general population study conducted so far, the exposure/response relationships are not consistent with a causal effect of RFR. Therefore, to date the general population studies indicate that RFR is associated with, at most, a very small increase in adult leukemia rates. The tower studies do not indicate that RFR is associated with elevated rates for other types of cancer.

OCCUPATIONAL STUDIES

Several occupational epidemiology studies have examined associations of RFR exposure at work with cancer later on in life. In general, occupational exposures are higher than that found in the residential setting. One potential advantage of occupational exposures is actual exposure measures or, more often, a good assessment of exposures typical of a certain occupation and information on the time spent in that occupation. This method called a job exposure matrix was used by several investigators. In occupational studies, it is important to consider other work-related exposures that could potentially confound the associations between RFR and the cancers studied as well as an examination of effects across several different RFR exposure levels.

Milham, S. "Silent keys": leukaemia mortality in amateur radio operators [letter]. Lancet 1(8432):812, 1985.

Milham (1985) studied mortality ham radio operators from 1971 to 1983 in the states of Washington and California. Since ham radios are transmitters as well as receivers, ham radio operators are exposed to radiofrequency radiation. Milham used deaths of members of the "American Radio Relay League" which were published in their monthly magazine in a section called "Silent Keys". Milham used proportionate mortality ratios (PMRs) to see if the proportion of ham radio operators dying from several causes of death was greater than that found in the general population. The percent of deaths due to

leukemia was more than double what was found in US white males. PMRs were also elevated for several subtypes of leukemia including acute and chronic myeloid leukemia. This association was not explained by occupation. The proportion was still doubled (PMR=210) when those with occupations with electrical exposure were excluded.

By their nature, PMRs will be elevated for some conditions when they are lower than usual for others. For this reason, they are difficult to interpret and are no longer generally used. Also, it is not clear from this brief report if Milham looked at causes of death other than leukemia – if so, this finding could have occurred by chance. In addition, Milham only looked at deaths that were reported in this amateur ham radio publication. Presumably, not all deaths among club members, let alone all ham radio operators were listed. Given the use of PMR, this might be a source of bias if those dying from usual causes were less likely to have obituaries in this publication. Finally, there is no potential information on the level of ham radio use both at the time of diagnosis and in the years before. Ham radio exposures also include exposures to other potential carcinogens such as degreasing agents and solvents which could also explain these associations.

Robinette CD, Silverman C and Jablon S. Effects upon health of occupation exposure to microwave radiation (radar). American Journal of Epidemiology, 112(1):39-53, 1980.

Robinette et al. looked at a wide range of health effects that could be potentially related to radar exposure including death, hospitalizations during and after service and disability compensation among Naval personnel who graduated from US Navy technical schools between 1950 and 1954. Only cancer health effects are reviewed here. The Navy has been measuring radar exposures on ships since 1957. Radar exposures were estimated for each occupation allowing for changing exposures over time with changes in equipment. Information from service records on length of time in particular occupations was used to identify 20,109 men with "maximal" potential exposure to radar emissions (those repairing radar equipment). An additional 20,781 men who were classified as having "minimum potential for exposure" (those operating radar equipment) served as a control group. The highly exposed group had infrequent exposures of more than 100 mW/cm^2, greater than exposures in any of the general population studies and double that of ICNIRP's estimated threshold for thermal effects of 50 mW/cm^2. (ICNIRP, 1996; Cleveland and Ulcek, 1999). Only individuals who died during the study period and a random sample of 5% of the remaining men had their service records examined to determine exposure. Year of birth was also only available from these two groups.

After standardizing for year of birth, the highly exposed group had higher mortality rates for two categories, respiratory tract neoplasms (MR=1.14), and cancers of the lymphatic and hematopoietic system (MR=1.18). Robinette et al. did not provide 95% confidence intervals for any of the mortality rates but according to the authors the elevated mortality rates for lymphatic and hematopoietic system were not significant.

Low exposure men were 1.3 years younger on average. Calculated mortality ratios were adjusted for year of birth, presumably based on the random sample from which birth dates were available. Since the incidence of most cancers increases with age, if their adjustment for age was ineffective this might result in a small upward bias in the exposure estimate. The high exposure group had men with more years of service and a greater proportion who began service in WWII. If other exposures potentially related to lymphatic and hematopoietic cancer incidence or survival were higher in WWII, this

Table II. General Population and Tower Epidemiology Studies

Author/Year	Study Population,	Effect Estimate	Cancers	Exposure Measure	Results (95% CI)	Comments
Hocking et al., 1996	Residents near 3 TV towers in North Sydney Comparison with outer areas All ages and children	Rate ratio for Incidence and Mortality Inner/outer	Brain cancer and leukemia (total, lymphatic, myeloid and other).	0.2 μW/cm2 at 4 km. (modeled) Residence within ~4km = "inner". Surrounding = outer.	***Incidence only presented here*** **All ages:** Brain 0.89 (0.71-1.11) Total leukemia: 1.24 (1.09-1/40) Lymphatic: 1.32 (1.09-1.59) Myeloid 1.09 (0.91-1.32) Other 1.67 (1.12-2.49) **Children:** Brain: 1.10 (0.59-2.06) Total: 1.58 (1.07—2.34) Lymphatic: 1.55 (1.00-2.41) Myeloid: 1.73 (0.62-4.81) Other: 1.65 (0.33-8.19)	Reanalyzed by Mckenzie et al. (1998)
Dolk et al., 1997b (II)	Residents near Transmitters in Great Britain Adults and children	O/E ratio	All leukemias, all acute leukemias, acute myeloid, acute lymphatic, chronic myeloid, chronic lymphatic leukemia, + melanoma and bladder cancer.	Distance, 2-10Km	**All Adults living within 10K of transmitter:** None of the outcomes are statistically - significantly elevated compared to the general population. Decline in risk with distance from 2-10K, but not among those living <2K from transmitter. **Children:** Not elevated in any analysis.	Also see Dolk et al. Part I (1997) for cluster analysis.
McKenzie et al. (1998)	Residents near TV towers in North Sydney, Australia. Adults and children	SIR along continuum of exposure level.	Total leukemia, Acute lymphocytic leukemia	Square of distance + measures used to create continuous estimate of exposure.	Rates were higher for all outcomes in areas with lower exposures. Influential observation of 1 of 3 areas seemed to explain findings in Hocking et al. (1996).	Reanalysis of Hocking et al. (1996). Added some areas. Created continuous exposure estimate for all areas.
Maskarinec et al., (1994)	Oahu, Hawaii residents near radio towers.	Case-control EOR	Childhood leukemia		OR=2.6 (0.6-8.3). No adjustment for SES. From 1985-1990 leukemia rates returned to expected levels while exposures did not change.	
Selvin et al., (1992)	San Francisco childhood cancer	Density-equalized counts	Childhood leukemia, brain cancer and lymphatic cancer	Distance from Sutro Tower.	There was no clustering of childhood cancer near Sutro Tower after density equalization.	Did not adjust for age or SES.
Taylor et al. (unpublished, 1989)	San Francisco children. Did not look at RFR.		Childhood leukemia		No association of residence with higher than expected numbers of childhood leukemia in SF. No cluster detected near Sutro.	Very low response rate.

would also bias mortality ratios upward. Furthermore, there is no mention of adjustment for smoking. Higher smoking rates among those with higher RFR exposures would also bias mortality ratios upward.

Thomas TL, Stolley PD, Sternhagen A, Fontham ETH, Bleecker ML, Stewart PA and Hoover RN. Brain tumor mortality risk among men with electrical and electronics jobs: a case-control study. JNCI 79:233-238, 1987.

Brain tumor incidence in those with potential RFR exposure was explored in a case-control study of workers in regions in 3 states – New Jersey, Pennsylvania and Louisiana. Cases were white males who died of brain cancer (or other central nervous system tumors) between 1979 and 1981. Brain cancer diagnoses were verified by a review of hospital records. One control was selected for each case matched for age at death, year of death and residential location (study region). Next of kin of 435 cases and 386 controls were interviewed to obtain information on job title, job location, employment dates, and hours worked per work as well as other factors shown to be risk factors for brain cancer in earlier studies.

Occupational RFR exposure was determined using two methods. First, occupations were classified as ever exposed or not using the method of Milham (1985). Second, an industrial hygienist assigned potential RFR exposure to each job listed in any subject's occupational history and level of exposure was determined. Using the second method, potential confounding by exposure to lead and soldering fumes was examined as well.

Men who ever had an occupation with RFR exposure as assessed using the first method had a greater risk of brain cancer (OR=1.6, 95% CI=1.0-2.4). Men in occupations with potential RFR exposure were next divided into two categories – those with and without potential ELF exposure. The elevated risks in the exposed were due to those with a job in an electrical or electronics job (OR=2.3, 95% CI=1.3-4.2). Men with these occupations for 5 years or longer had a 3-fold risk with no evidence of an exposure/response with longer exposure. In men exposed to RFR who *never* worked in electrical or electronics jobs (i.e. did not have occupational ELF exposure), the odds of brain tumor was not elevated (OR=1.0, 95% CI=0.5-1.9). Using the second method yielded similar results. Further analyses determined that the elevations were greatest in electronics manufacture and repair workers.

Since brain cancer mortality risks were elevated specifically for the group with potential ELF exposure suggests that the effects were not due to RFR radiation, especially given that RFR exposures in these occupations were probably intermittent, according to the authors. The authors did not adjust for socioeconomic status. Several studies have found that brain cancer incidence increases with social class. This might have resulted in an upward bias of effect estimates.

Milham, S. Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies. American Journal of Epidemiology 127:50-54, 1988.

Milham's observations in the brief 1985 report described above were explored further using 1984 licensing records for amateur radio station operators from the Federal Communications Commission. Records did not include information on the years of licensing or the date when the operator was first licensed. Ham radio operators from Washington State and California licensed between 1979 and 1984 were identified (n=67,829). Vital status of the licensees was determined using probability-based linkages with mortality databases in each state. Eighty-four percent of deaths were from California. Person-years at risk were determined using the current license as the estimated start of the risk period and the day of death as the end of the risk period. US mortality rates ***among males and females*** were used to calculate expected numbers of deaths. Milham does not mention if the results were age-standardized, and if so, what standard was used.

All cancers of lymphatic and hematopoietic tissue were nearly significantly elevated (SMR=123, 95% CI=99-152). This elevation is probably explained by the subtype of cancer of "other lymphatic tissues" (SMR=162, 95%CI=117-218). No other subtype was significantly elevated but SMRs exceeded 100 for leukemia and Hodgkin's disease. When leukemia deaths were analyzed by type, elevations were found only for acute myeloid leukemia (SMR=176, 95% CI=103-285). Deaths from respiratory and

circulatory diseases and those from lung cancer were much lower than expected, probably an indication of low smoking rates in this group.

In an analysis of occupational information on Washington State deaths certificates (this information was absent from California death certificates at the time of the study), Milham observed that 31% of the operators who died worked in occupations with potential for ELF exposure. These included radio operator, television repairman and electronics technician. Unfortunately, the author did not distinguish RFR and ELF exposures.

Since the two type of exposures theoretically would have different biological mechanisms, it is hard to attribute elevated SMRs to ham radio operation. Analyses stratified by potential occupational ELF exposure such as was done by Thomas et al. (1987), would have strengthened this study. Milham's (1988) use of a standard with both men and women could potentially have biased his results upward since according to 1987-1991 SEER data, white male incidence rates for both leukemia and lymphomas were 58 to 77% higher in men than women (Ries et al., 2000).

Hayes RB, Brown LM, Pottern LM, Gomez M, Kardaun JWPF, Hoover RN, O'Connell KJ, Sutzman RE and Javadpour N. Occupation and risk for testicular cancer: a case-control study. International Journal of Epidemiology 19:825-831, 1990.

Hayes et al. (1990) conducted a case-control study of risk factors for testicular cancer. Cases were men aged 18 to 42 with testicular cancer diagnosed between 1976 and June 30th, 1981 who were referred to 1 of 3 health centers, two of which were military. Controls were *other cancer patients* at the same hospital with a cancer not of the genital tract. Controls were frequency matched for age. To assess potential bias from using cancer patients as controls, analyses were redone using only controls with lymphatic and hematopoietic cancers. 308 cases and 288 controls were interviewed. Interviews included questions on exposures to ionizing radiation, microwaves, and radio waves (both in this review are defined as RFR), in part to investigate their *a priori* clinical impression that "radar and other microwave exposure were common among military testicular cancer cases". An industrial hygienist reviewed job titles to assess whether the occupation had high, medium or low exposure to RFR. Subtypes of testicular cancer (germinal cell carcinomas – seminomas and others) were also studied.

In the RFR-related analyses, the OR for self-reported radiofrequency exposure was significantly elevated for all testicular cancers combined (OR=3.1, 95% CI=1.4 to 6.9). However, when job title was used as a proxy for exposure, the odds ratio was unity overall with no evidence for a dose response across exposure levels. Self-reported and job-based RFR exposures were not well correlated.

Testicular cancer is found mostly among young men with incidence highest from ages 30 to 34. Because the study was restricted to men 18-42 years of age, most of the participants had a small duration of employment (median 3 years) before diagnosis and consequently occupational exposures of short duration. Presumably, the interval of occupational exposure and diagnosis was also short for many of the cases. If exposures of longer latency are relevant, then this could have obscured some occupational associations. Because cancer patients were used as controls, recall bias is not a good explanation of the discrepant results found when using self-reported exposures versus those categorized by job title. It is hard to know which method would have the most validity for RFR exposure assessment.

Grayson JK. Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: a nested case-control study. Am J Epidemiol 143:480-486, 1996.

This author reports on the effects of both ELF and RFR using a nested case-control design. Only the RFR results are reported here. The original cohort was composed of male US Air Force Personnel who served from 1970 to 1989. The study base consisted of approximately 880,000 male US Air Force servicemen and 11,174,248 person-years. Cases were identified by linking national hospital discharge data with a list of Air Force personnel who served during the study period. Four controls were randomly selected from each case's risk set – Air Force personnel matched for birth year and race and who were present in the USAF at the time of the case diagnosis. Personnel with diagnoses of leukemia, breast

cancer or malignant melanoma were not eligible to be cases. According to Grayson, "Follow-up of individuals who had separated from the US Air Force was not attempted." A total of 920 controls were selected for 230 cases.

Job title, age, race, rank and duration of service were abstracted from USAF service records for each case and control. Radiofrequency, ELF and ionizing radiation exposures were estimated using a job-exposure matrix for each study subject. In the United States Air Force, all incidents in which personnel are exposed to RFR above 10 mW/cm^2 are reported to a central agency. Using data from this exposure registry, jobs were classified as having no, possible or probable potential for exposure. Control occupational exposure was censored at the time of the case's diagnosis so that the case and control person-time would coincide. Potential joint effects of ELF, RFR and ionizing radiation were also investigated.

The authors found a 39% increase in the odds of brain cancer (95% CI = 1.01-1.90) in "ever vs. never" exposed using conditional logistic models adjusted for age, months of service, and military rank. However, odds ratios did not increase across 4 categories of increasing RFR exposure. Brain cancer risk did increase with increasing rank even after adjustment for age and months of service in the USAF. The association for rank was larger than that for RFR and the odds ratio was greatest when senior officers were compared to all others (OR=3.30, 95% CI=1.99-5.45). There were no apparent joint effects of RFR with ELF or ionizing radiation.

This study has relatively good exposure assessment. Exposures by job title were allowed to vary within the study period and were rated independently by 2 panels. Also, ELF and RFR exposures were considered separately. There was no exposure response relationship with RFR, however. One potential source of bias might be the requirement that cases be with the US Air Force at the time of diagnosis. If a case had to be in the USAF at the time of diagnosis, then effects could be biased downwards. Sicker cases might have been more likely to leave the service before diagnosis reducing the case rate and the risk ratio. The relationship of increased brain tumor risk with increased social class has been shown in several other studies and is probably not due to detection bias (men of higher rank are more likely to be diagnosed) because military care is available to all USAF personnel. Since rank was used as a proxy for socioeconomic status (SES) and higher SES has been associated with higher rates of brain cancer in this country, the association with rank is not surprising.

Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. The Science of the Total Environment 180:9-17, 1996.

Cancer rates in Polish military personnel were compared among those exposed or unexposed to radiofrequency radiation in the years 1971 to 1985. Exposure status was determined through military records and, according to the author, hospital records among those with an incident diagnosis of a neoplasm. Most (85%) exposures were pulse modulated emissions ranging in frequency from 150 to 3500 MHz. Annual cancer morbidity rates (1^{st} hospitalization) were calculated per 100,000 servicemen and averaged over the 15-year study period. Rates in the unexposed group were used to determine expected rates of cancer and observed/expected ratios (OERs) were calculated for each cancer type.

Szmigielski observed higher than expected numbers of cancers for nine of twelve types of cancer studied. Significant elevations were found for colorectal, stomach, skin, and brain cancer and cancers of the haematopoietic system and lymphatic organs. For all cancers combined, there was a statistically significant doubling of the rate among the exposed military personnel (OER=2.07, 95% CI=1.12 to 3.58).

Unfortunately, this study suffers from a methodologic flaw. Because Szmigielski used medical records to determine exposure status, military personnel with cancer hospitalizations had a better chance of being classified as exposed. The information bias could have substantially increased the effect sizes for all types of cancer. Potential evidence for this bias is the elevated rates for many types of cancer that have not been elevated in other studies.

Tynes T, Hannevik M, Andersen A, Vistnes AI and Haldorsen T. Incidence of breast cancer in Norwegian female radio and telegraph operators. Cancer Causes and Control 7:197-204, 1996.

Tynes et al. were interested in exploring a hypothesis that pineal gland stimulation from exposure to artificial light at night raised the risk of breast cancer. They evaluated this hypothesis using a cohort of 2619 female radio and telegraph operators who often did shift work (worked overnight) on merchant ships at sea. All female and radio telephone operators working in 1960 were included in the cohort. In addition to increased exposure to artificial light, the cohort was also exposed to both radiofrequency (405 kHz-25 MHz) and extremely low frequency radiation (50 Hz). Although historical RFR measurements were not available, the radio/telegraph equipment had remained unchanged throughout the thirty year study period. RFR measurements at the end of the study period were all below occupational guidelines recommended by the International Radiation Protection Association.

Cancer diagnoses in the cohort occurring from 1961 to 1991 were identified by linkage of the cohort with the Cancer Registry of Norway. The authors looked at several cancers including leukemia, lymphoma, lung, esophageal, colon, rectal, kidney, cervical, breast, uterine, ovary, bone, thyroid and brain cancers. An elevated risk was seen for breast cancer (SIR=1.5, 95% CI=1.1-2.0)) and uterine cancer (SIR=1.9, 95% CI= 1.0-3.2). Elevated risks were not seen for leukemia, lymphoma, or brain cancer. The highest breast cancer risk was found in radio and telegraph operators aged 50 to 54 years with an SIR of 2.5 (95% CI=1.3-4.3). The uterine cancer risk was of borderline statistical significance and was not explored further.

The breast cancer association was further examined in a nested case-control study. Four to seven controls alive at the time of case diagnosis and matched for year of birth were selected for each case. Analyses were stratified by age: those younger than 50, and those 50 years or older. The authors did find an association of shift work and duration of employment (a potential proxy for RFR/ELF exposures) in those over 50. However shift work was highly correlated with duration of employment (.79) and, according to the authors, adjusted analyses were most consistent with an effect of shift work (these analyses were not reported in the paper). In other analyses, rates in this cohort were compared to rates in women with other occupations at sea– these data were collected as part of a related occupational cancer cohort study. Breast cancer rates were elevated in female office clerks at sea (SIR=1.4, CI=1.0-2.0) and female kitchen helpers (SIR=1.4, CI=0.9-2.0) who worked onboard ships. Rates were not elevated in pursers and stewardesses (SIR=1.0, 95% CI=0.8-1.4). Since rates were elevated (although not always significantly) in women with professional or technical jobs at sea, the authors investigated the possibility that delayed childbirth might explain the higher rates observed. However, adjustment for this did not change the observed effects.

The study included several RFR measurements onboard ships. Equipment generating RFR has not changed for the three decades covered by the study period so current measurements probably are a good reflection of historical exposures. However, the authors did not attempt to develop separate exposure metrics for shift work, RFR and ELF. According to the investigators, "'Shift work' highly reflects frequent presence in the radio room both at night and during the day with possible exposure to light at night, and RF and ELF fields." In other words, all of these exposures are probably very highly correlated. Therefore, it is hard to know which exposure(s) is/are associated with the elevated rates of breast cancer. The authors speculate that there might be an interaction between shift work and ELF/RFR exposures.

Liakouris AJ. Radiofrequency (RF) sickness in the Lilienfeld Study: An effect of modulated microwaves? Archives of Environmental Health, 53:236-238, 1998.

Liakouris (1998) reinterpreted results from a government report by Lillienfeld (1976) which investigated the health impacts of microwave irradiation of the Moscow embassy between 1953 and 1976. Embassy workers were exposed to RFR for 6 to 8 hours a day and 5 days a week for 2 to 4 years on average. Exposures were between 0.6 and 9.5 GHz and had SAR of 2-28 $\mu W/cm^2$. In the original study, embassy workers were compared to other embassy workers in Eastern Europe. Although the authors of

the original study found elevated lymphocytes in the Moscow workers, they did not report on other elevations in controls, according to Liakouris. These included problems with skin (e.g. eczema, psoriasis), problems in pregnancy, neurological problems, tumors and higher rates of depression.

It is possible that findings reported here were not originally emphasized because they were either not consistent or non-specific. For example, Liakouris notes that there were elevations of benign tumors among men but malignant tumors among women. Given equivalent RFR exposures, one would not expect it to raise the risk of benign tumors in one group (men) and malignant tumors in another (women) unless, for example, the subgroup of malignant reproductive cancers was elevated. Differences in mood (irritability and depression and loss of appetite) could be due to ecologic differences in Moscow and the cities where controls resided. No adjustment for confounding in the original study was mentioned in this article. In a published commentary, Goldsmith (1997) implied that there were similar radiation exposures in the Eastern embassy workers used as controls, but he did not cite evidence for this. There has been no published follow-up of the Moscow embassy cohort.

Morgan RW, Kelsh MA, Zhao K, Exuzides A, Heringer S and Negrete W. Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems. Epidemiology 11:118-127, 2000.

While studies of cellular telephone use are not included in this report, this study of occupational exposures in those who manufacture wireless products is relevant. Furthermore, exposures were predominantly of RFR and were not as confounded by ELF as most other occupational mortality studies were. Morgan et al. studied cancer mortality rates in 195,775 Motorola employees who had worked at least 6 months total and who had been employed at least 1 day from 1976 to 1996. These workers contributed a total of 2.7 million person years. The cohort was 44% female; most other occupational studies have only included men. Mortality risks for cancer of the brain and lymphatic/hematopoietic systems were examined with follow-up of vital status to 1996. Employee deaths were determined by linking personnel records to the Social Security Administration Master Mortality file and the National Death Index. Cause of death was validated in a random sample of death certificates using nosologists blinded to the cause of death listed on the death certificate. This study was funded by Motorola, Inc.

The investigators used a job exposure matrix to categorize RFR exposures in 9,724 job titles into no-, low-, moderate- and high-exposure categories. Extensive input from both company and outside experts was used to assign jobs to categories. Most workers (72%) had no RF exposure. Only 4.5% of workers were classified as having high RF exposure. According to the authors, exposures in the RF-exposed subcohort were lower than in military and plastics manufacturing workers.

Indirect standardized mortality ratios adjusted for age, gender and race were calculated using mortality rates in the 4 states where most employees resided as a standard. Mortality rates in high and low or no-exposure groups were also compared to account for a potential healthy worker effect. Cancer mortality rates were compared using usual, peak and cumulative RFR exposures. Latencies of 5-, 10- and 20-years were considered. Twenty-four percent of workers were available for consideration in the 20-year latency analysis. Only 3.2% of the cohort died during the study period.

For the total cohort, including those exposed and unexposed to RFR, SMRs for all cancers were substantially lower than in the standard population (SMR=0.78, 95% CI=0.75-0.82), indicating presence of a healthy worker effect. SMRs for brain and lymphatic/hematopoietic cancers were also less than expected (SMR=0.77, 0.67-0.89 and SMR=0.60, 95% CI=0.45-0.78). In workers with RFR exposure, SMR patterns were quite similar to that of the total cohort. Brain and lymphatic cancer SMRs were not elevated – SMRs were 0.53 and 0.54 respectively and 95% confidence intervals did not include 1.0 .

In the internal comparison, the authors examined relative rates of brain cancer, lymphatic and hematopoietic cancer, leukemia and non-Hodgkin's lymphoma in highly exposed, moderately exposed and unexposed workers. Relative rates of mortality in the RFR-exposed subcohort did not increase by duration of exposure, with relative rates in the highest exposed group mostly <u>below</u> 1.0 (i.e. cancer mortality rates were often lower in the highest exposed group than they were in the unexposed group). In

almost all of the internal cohort analyses in this study, brain cancer risk was *lower* among exposed than unexposed and risks were never highest in the high exposure group.

The negative finding in this mortality study which included 2.7 million person-years may be explained by poor study power since the cohort was young, brain and lymphatic cancers are rare, and few workers were highly exposed. However, the exposure assessment used by the authors was much more extensive than seen in other studies. Also, if RFR-exposure were causally associated with brain and lymphatic cancer mortality, poor power would result in wide confidence intervals not effect estimates below 1.0. Therefore low power does not explain the results and this study does not provide support for an association of RFR with brain or lymphatic cancer.

SUMMARY OF OCCUPATIONAL STUDIES BY TYPE OF CANCER

The occupational studies are summarized in Table III. Two types of cancer are of special interest given the results of the tower/general population studies. They are brain/central nervous system cancers and lymphatic and hematopoietic cancers (leukemia is a subtype of this class). Testicular and breast cancer are also of interest since well designed occupational case-control studies for each cancer type have been conducted with RFR as the exposure of interest.

Evidence for an association of RFR with brain cancer

There have been six occupational studies of RFR and brain cancer. There is limited evidence from one occupational epidemiology study of an association of brain cancer and RFR among USAF servicemen (Grayson, 1996). This study found a 39% increase in the odds of brain cancer (95% CI = 1.01-1.90) in “ever vs. never" exposed, but odds ratios did not increase with increasing RFR exposure. Since brain tumor cases had to currently serve to be included in the study, effects were biased downward. This bias might have affected an exposure-response relationship. In another case-control study, an effect was found in a subset of workers exposed to both ELF and RFR. (Thomas et al., 1987), but not in workers exposed only to RFR. In the Polish military cohort Szmiegelski (1996) observed elevated rates

Evidence for an association of RFR with breast cancer

The association of RFR and breast cancer has been examined in two studies (Tynes et al., 1996; Morgan et al., 2000). In the cohort study of Morgan et al. (2000), when mortality in the cohort was compared to the general population, the SMR for breast cancer was not elevated, however this could have been explained by a healthy worker effect or the young age of the cohort. Breast cancer was not examined by level of RFR exposure within the cohort.

Tynes et al. (1996) studied a female cohort of radio and telegraph operators. In an analysis of the entire cohort, the SIR for breast cancer was elevated (SIR=1.5, 95% CI = 1.1-2.0) with the elevation strongest in the 50-54 year age group (SIR=2.5, 95% CI=1.3-4.3). However, when rates in this cohort were compared with other women who worked at sea but were not RFR-exposed, breast cancer incidence of brain cancer. However, this study was positive for several cancers including many which had not been associated with RFR in other studies and was very likely biased.

Three of four cohort studies were negative (Milham, 1988; Tynes et al., 1996; Morgan et al., 2000). The negative finding in the Motorola cohort study which included 2.7 million person-years may be explained by poor study power since brain cancer is rare, the cohort was young and only a small

Table III. Occupational Studies of RFR and Cancer

Author/Year	Study Population, N	Design / Effect Estimate	Cancers	Exposure Measure	Results	Comments
Robinette et al., 1980	U.S. Navy technicians graduating (1950-1954) n=20,109 men	Mortality ratio, Comparison of high vs. low/no exposed. Hospital admissions.	All malignant neoplasms, cancer of digestive organs, respiratory tract, L&H, other malignant neoplasms		MR lymphatic and hematopoietic=1.18	NO CI
Milham, 1985	n=1,691 ham radio operators with deaths published in club paper	PMR	Leukemia only cancer stated in report. Others may have been investigated	All exposed.	PMR=210 after exclusion of those in elect. occupations.	
Thomas et al., 1987	435 cases, 386 controls matched for age, year of death and state	Case-control. OR reported as RR. odds of electrical job	Brain	Milham's categories or JEM	Milham: OR=1.6 (1.0-2.4) JEM: OR= 2.3 (1.3-4.2) subgroup in manufacturing or repair of electronics.	
Milham, 1988	(n=67,829) licensed ham radio operators in WA and CA	SMRs (mortality)	All, esophagus, stomach, large intestine, rectum, liver, pancreas, respiratory system, prostate, urinary bladder, kidney, brain, L&H: lymphosarcoma/reticulosarcoma, Hodgkin's leukemia, other lymphatic	All exposed.	L&H, 123 (99-152) Other lymphatic (162, 117-218) AML (176, 103-285)	standard pop = US males and females results in upward bias of estimates.
Hayes et al., 1990	men 18-42 case-control cases=308 controls=288 dx 1976-1981	incidence / OR	testicular and subtypes	self-report or JEM	Self-report: 3.1 (1.4-6.9) job title (get OR, OR=1.0)	Controls were other cancer patients with cancer not of the genital tract.
Davis and Mostofi, 1993	1979-1991, 6 cases c/ 0.89 expected	OER (Cancer cluster)	Testicular	Y/N – radar	0/E=6.9	How standardized?
Grayson, 1996	Nested case-control, USAF.	OR, adjusted for age, months of service and rank	Brain	JEM + service records, no, probable, possible	ever/never: OR=3.3 (1.99-5.45), but no evidence of exposure / response	Dx of leukemia, breast cancer or melanoma not eligible.
Szmigielski (1996)	Polish Military (1971-1985)		All, Colorectal, stomach, skin, and brain cancer, L&H		all: OER=2.07 (1.12-3.58) + for 9/12 cancers including brain , H&L	information bias
Tynes et al., (1996)	Cohort + Case-Control: 2619 female radio and telegraph operators	Cancer f/u from 1961-1991; SIR	Breast & stomach, colon, rectum, pancreas, lung, cervix, uterus, ovary, kidney, bladder, malignant melanoma, brain tumor, lymphoma, leukemia, other, all cancers.		Neg. for leukemia, lymphoma, brain Breast (50-54) SIR=2.5 (1.3-4.3) OR = Shift work	Also ELF exposure.

Table III. Occupational Studies of RFR and Cancer (continued)

Author/Year	Study Population, N	Design / Effect Estimate	Cancers	Exposure/Measure	Results	Comments
Liakouris (1998)	reanalysis/reinterpretation.	Moscow embassy workers	malignant tumors, benign tumors	measurements during most of exposure period but not in offices or living areas.	elevations of benign tumors in men and malignant tumors in women.	
Morgan et al., (2000)	Motorola employees, n=195,775	SMR (mortality)	CNS, L&H (Hodgkin's, leukemia & aleuleumia), melanoma, cancers of the respiratory system, digestive organs & peritoneum (stomach and pancreas), breast, other female genital cancer, prostate, bladder and other urinary, kidney	jem to no-low- mod- and high exposed. 72% no RFR exposure	Internal cohort analysis in which exposure measured cumulatively, average exposure or highest exposure groups were compared to low/no exposed groups. For L&H and brain cancers.	Study funded by Motorola

proportion were highly exposed. However, in almost all of the internal cohort analyses in this study, brain cancer risk was *lower* among exposed than unexposed and risks were never highest in the high exposure group (Morgan et al., 2000). This is persuasive evidence that there is not an association of RFR and brain cancer.

Evidence for an association of RFR exposure with lymphatic and hematopoietic cancers including leukemia, Hodgkin's disease and non-Hodgkin's lymphoma.

Although several investigators examined the association of occupational RFR exposure and cancers of the lymphatic and hematopoietic system, many of the studies with positive findings were flawed or biased (Milham, 1985; Milham,1988; Szmiegelski, 1996). Milham (1985) found elevated PMRs for leukemia but did not reveal if any other cancers were studied. If this was the only positive finding from many cancers studied, it could be a chance finding. Also, PMRs will be elevated if rates for other causes of death are lower than expected. Finally, only selected deaths among ham radio operators were studied. In Milham's second study (1988), he observed elevated SMRs for all lymphatic and hematopoietic cancers, acute myeloid leukemia and "other lymphatic" cancers. However, he calculated SMRs using a standard population with both men and women while his cohort was largely male, potentially biasing his effects upward. In addition, his study population, ham radio operators, also had potentially high ELF exposure which was not adjusted for in analyses. Szmiegelski (1996) had better information on exposure in those with cancer than in those without cancer. Consequently, he found associations with several types of cancer that were not found in any other occupational studies.

Three other studies found either nonsignificant elevations or no association of RFR with this class of cancers. Robinette (1980) observed an 18% increase in mortality rates for lymphatic and hematopoietic cancers among those who were RFR exposed, however this elevation was not statistically significant and there were potential upward biases in effect estimates. Tynes et al. (1996) studied the association of RFR and lymphatic and hematopoietic cancers in the larger cohort study described in their report and found no association. Exposure assessment was somewhat better in this report than in most other occupational studies of RFR. Morgan et al. (2000) compared cancer mortality rates among exposed and unexposed workers using usual, peak and cumulative RFR exposures. Leukemia mortality rates were either nonsignificantly elevated or lower in the highest exposed group.

Since the studies that found an association generally had substantial biases and the studies that did not find an association were methodologically superior, the epidemiology studies, taken together do not indicate an association of occupational RFR exposure with lymphatic and hematopoietic cancers.

Evidence for an association of RFR with testicular cancer

There has been only one epidemiology study of RFR and testicular cancer (Hayes et al., 1990). In this case-control study, there was an association of occupational exposure to RFR and testicular cancer when self-report was used, but not when a job exposure matrix was used. Since this study had equivocal results, future studies of the association of testicular cancer and RFR are warranted. A testicular cancer cluster (Davis and Mostofi, 1993) reported among police officers in two adjacent United States counties using radar guns was not reviewed here since a cluster is useful for hypothesis generation but not for the actual assessment of a causal association.

Tynes et al. (1996) studied a female cohort of radio and telegraph operators. In an analysis of the entire cohort, the SIR for breast cancer was elevated (SIR=1.5, 95% CI = 1.1-2.0) with the elevation strongest in the 50-54 year age group (SIR=2.5, 95% CI=1.3-4.3). However, when rates in this cohort were compared with other women who worked at sea but were not RFR-exposed, breast cancer incidence was also elevated, indicating that chronobiological disturbances and delayed fertility are better explanations than RFR for the elevated breast cancer rates.

RELEVANCE OF AVAILABLE EVIDENCE TO THE STUDY OF RFR -- CANCER RELATIONSHIPS IN NEIGHBORHOODS NEAR SUTRO TOWER

Overall, data from the very large Great Britain study, re-analysis of the North Sydney data, and two studies of childhood cancer which included neighborhoods close to Sutro tower do not support a relationship of RFR from broadcast towers and child leukemia.

The study by Selvin et al. (1992) was designed to detect spatial clusters of incident childhood cancers in small areas should they exist. The study period was long (1973 to 1988) and all cases of leukemia, brain and lymphatic cancer in children were included. Selvin et al. specifically attempted to relate density-equalized numbers of cancer to Sutro Tower. After adjustment for population density (which allows investigation of clusters without relying on unstable rate calculations), there was no evidence of an association of childhood cancer clusters for these diagnoses with distance from Sutro Tower. Since leukemia cases comprise the bulk of childhood cancers (Lum et al., 2000) there was presumably substantial overlap between cases used in the Taylor et al. (1989) and Selvin et al. (1992) studies. A re-analysis of this relationship would add 12 years of outcome data, but the results would only be different if the relationship between tower related exposure and leukemia is different in this period than in the preceding period.

Adult leukemia has not been investigated in San Francisco. The British 20-transmitter study (Dolk et al., 1997b) was only able to detect a small effect (3% increase) on adult leukemia rates based on data for 3.4 million people (an effect driven by only one of 20 areas), and was unable to detect RFR exposure/response patterns (declining risk of leukemia with increasing distance from a broadcast tower). Though the occupational studies of adult leukemia and RFR had the advantage of greater statistical power, potentially better exposure assessment, and better characterization of potential confounders they lend little support to the hypothesized relationship between RFR and adult leukemia (Tynes et al. , 1996; Morgan et al., 2000). In conclusion, the adult leukemia findings in the general population studies show at best small effects (which imply the need for studies with very large sample sizes) and are not supported by occupational studies. For these reasons, an adult leukemia study in San Francisco would be unlikely to lead to important new information about this relationship.

Brain cancer has also not been investigated in San Francisco. Using the experience of the large British study which represented 3.4 million people, it is unlikely that a study of residents living near the tower would have the study power to detect an effect. Since brain cancer is very rare, the association of RFR and brain cancer is probably best studied using a nested case-control design from a very large occupational cohort such as the United States military.

CONCLUSION

In summary, this review has not found substantial epidemiologic evidence to support an association of radiofrequency radiation with cancer. Furthermore, two prior San Francisco childhood cancer studies found no evidence of childhood cancer clusters in the neighborhoods surrounding the tower. Therefore, there does not appear to be a basis for conducting a cancer-RFR study around Sutro Tower. Questions raised by the literature such as the relationship of RFR with rare outcomes such as testicular and brain cancer would be difficult if not impossible to meaningfully answer by a study in San Francisco because study power would be too low due to the limited size of the exposed population, migration in and out of exposed communities, and the lack of historical exposure measurement. Many of these questions may be best answered by further studies in other target populations.

GENERAL POPULATION EPIDEMIOLOGY STUDIES

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APPENDIX I.

NATIONAL AND INTERNATIONAL GOVERNMENT REPORTS/REVIEWS

United States Government Reports

Cleveland RF and Ulcek JL. Questions and answers about biologic effects and potential hazards of radiofrequency electromagnetic fields. Office of Engineering and Technology, Federal Communications Commission, OET Bulletin Number 56 (Fourth Edition), August 1999

Cleveland RF, Sylvar DM, Ulcek JL. Evaluating compliance with FCC guidelines for human exposure to radiofrequency electromagnetic fields. Office of Engineering and Technology, Federal Communications Commission, OET Bulletin Number 65, Edition 97-01, August 1997.

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International Non-Ionizing Radiation Committee (INIRC) of the International Radiation Protection Association (IRPA). Interim Guidelines on Limits of Exposure to 50/60 Hz Electric and Magnetic Fields, Health Physics 58(1):113-122, 1990.

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International Non-Ionizing Radiation Committee (INIRC) of the International Radiation Protection Association (IRPA). Guidelines on limits of exposure to radiofrequency electromagnetic fields, 3 kHz to 1 00 kHz to 3 00 GHz. Health Phys: 54: 115-23, 1998

United Nations Environmental Program (UNEP) /World Health Organization (WHO) /International Radiation Protection Association (IRPA). Electromagnetic fields (300 Hz-300 GHz). Geneva: World Health Organization; Environmental Health Criteria #137, 1993.

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Canada: Health Canada, Royal Society of Canada. A review of the potential health risks of radiofrequency fields from wireless telecommunication devices. March, 1999.

England: National Radiation Protection Board (NRPB). Advice on the 1998 ICNIRP guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz). NRPB 10, No. 2, 1999.

Australia: Australian Radiation Protection and Nuclear Safety Agency (ARPANSA). The mobile phone system and health effects.

Upcoming International Reports
2003: Cancer evaluation for RFR by International Agency for Research on Cancer (IARC)
2004: Non-cancer evaluation for RFR by World Health Organization

APPENDIX II. ANIMAL AND CELLULAR STUDIES –1997 TO 1999

Study	Type of Study	Outcome	Dosage	=/- Study
Adey, R. et al. **Spontaneous and Nitrosourea-Induced Primary Tumors of the Central Nervous System in Fischer 344 Rats Chronically Exposed to 836 MHz Modulated Microwaves**, Radiation Research, Sept. 1999, 152, 293-302	Animal Study	CNS Tumors		No tumorigenic effect, possible inhibition
Goswami, PC; Albee, LD; Parsian, AJ; Baty, JD; Moros, EG, Pickard, WF, Roti, JL; Hunt, CR **Proto-oncogene mRNA levels and activities of multi-transcription factors in C3H 10T ½ murine embryonic fibroblasts exp 835.62 and 847.74 Mhz cellular phone communication frequency radiation**, Radiation Research, 1999 Mar, 151(3):300-9	Cell study	Proto-gene oncogene expression, mRNA levels	0.6 w/kg	+/- results
Frei, MR; Jauchem, JR; Dusch, SJ; Merritt, JH, Berger, RE, Stedham, MA **Chronic, low level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves,** Radiation Research, 1998 Nov, 150(5): 568-76	Animal study	Mammary tumors	1.0 W/kg	- study
Kwee, S; Rasmark, P; **Changes in cell proliferation due to environmental non-ionizing radiation**, Bioelectrochemistry and Bioenergetics; 1998, 44 (2): 251-255	Cell study	Change in cell proliferation		Reduced cell proliferation with increased RF power
Frei, MR; Berger, RE, Dusch, SJ; Guel, V; Jauchem, JR; Merritt, JH; Stedham, MA**. Chronic exposure of cancer-prone mice to low-level 2450 Mhz radio frequency radiation**, Bioelectromagnetics, 1998, 19 (1):20-31	Mice study	Decreased latency and incidence of mammary tumors	0.3 W/kg	Negative results
Imaida, K. et al., Title unknown, Carcinogenesis, Feb 1998, 19, 311-314	Animal Study	Liver Cancer	0.58- 0.80 W/kg	Does not promote liver cancer, increase in hormone levels.
Malyapa, RS; Ahern, EW,; Straube, WL; Moros, EG; Pickard, WF; Roti; JL **Measurement of DNA damage after exposure to electromagnetic radiation in the cellular phone communication frequency band (835.62 and 847.7 Mhz**) Radiation Research, 1997 Dec, 148(6):618-27	Cell study	DNA damage	0.6 W/kg	No effects observed
Fritze, K; Wiessner, C; Kuster, N; Sommer, C; Gass, P; Hermann, DM; **Effect of Global System for mobile communication microwave exposure on the genomic response of the rat brain**. Neuroscience, 1997 Dec, 81(3):627-39	Animal/cell study	Change in messenger RNA, cell proliferation	0.3 –7.5 W/kg	No changes to changes to rat brain
Maes, A; Collier, M; Van Gorp, U; Vandoninck, S; Verschaeve, L **Cytogenic effects of 935.2 MHz (GSM) microwaves alone and in combination with mitomycin C** Mutation Research, 1997 Sept 18, 393 (1-2):151-6	Genetic study on exposed whole blood cells	Chromosome aberrations		No cytogenic effect found
Toler, JC; Shelton, WW; Frei, MR; Merritt, JH; Stedham, **MA Long-term level exposure of mice prone to mammary tumors to 435 MHz radio frequency radiation.** Radiation Research, 1997 Sept, 148(3):227-34	Animal Study	Changes in mammary tumors		No differences

Study	Type of Study	Outcome	Dosage	=/- Study
Repacholi, MH; Basten, A; Gebski, V; Noonan, D; Finnie, J; Harris, AW. **Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 90 electromagnetic fields**. Radiation Research, 1997 May, 147 (5):631-40	Animal study	Lymphomas	0.008-4.2 W/kg	Increase risk for lymphoma
Vijayalaxmi; Frei, MR; Dusch, SJ; Guel, V; Meltz, ML; Jauchem, JR **Frequency of micronuclei in the peripheral blood and bone marrow of cancer-prone chronically exposed to 2450 Mhz radiofrequency radiation**, Radiation Research 1997 Apr, 147 (4):495-500	Animal study	Genotoxicity as indicated by micronuclei in polycellular erythrocytes	1.0W/kg	Negative study
Ivaschuk, OI; Jones, RA; Ishida-Jones, T; Haggren, W; Adey, WR; Phillips, JL **Exposure of nerve growth factor-treated PC12 rat pheochromocyte cells to a modulated Rf field at 836.55; effects on c-fos expression.** Bioelectromagnetics, 1997, 18(3):223-9	Cellular study	Change in gene expression		No changes noticed
Stagg, RB; Thomas, WJ; Jones, RA; Adey, WR **DNA synthesis and cell proliferation C6 glioma and primary glial cells exposed to a 836.55 modulated radio frequency field** Bioelectromagnetics, 1997, 18(3):230-6	Cell Study	Alteration of DNA synthesis	0.15-59 muW/g	No changes noted
Nelson, BK; Conover, DL; Krieg, EF Jr.; Snyder, DL; Edwards, RM **Interactions of radiofrequency radiation-induced hyperthermia methoxyethanol teratogenicity in rats** Bioelectromagnetics, 1997, 18(5): 349-59	Animal Study	Teratogenicity	Thermal exposures	Enhanced teratogenicity with chemical
Lai, H; Singh, NP **Melatonin and spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand break brain cells** Bioelectromagnetics, 1997, 18(6):446-54	Cell study	DNA strand breaks	1.2 W/kg	DNA damage blocked by chemicals

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