

Jürgen Breckenkamp · Gabriele Berg · Maria Blettner

Biological effects on human health due to radiofrequency/microwave exposure: a synopsis of cohort studies

Received: 13 May 2003 / Accepted: 22 July 2003 / Published online: 24 September 2003
© Springer-Verlag 2003

Abstract We evaluated the methods and results of nine cohort studies dealing with the biological effects on human health from exposure to radiofrequencies/microwaves, published between 1980 and 2002. The size of the cohorts varied between 304 (3,362 person years) and nearly 200,000 persons (2.7 million person years). As exposures were defined: dielectric heaters in a plastic manufacturing plant, working with radio devices (professional and amateur), production of wireless communication technologies, radar devices of the Canadian police, radar units used by the military as well as artificially produced electromagnetic pulses similar to those after a nuclear explosion. In all studies (except one that used a qualitative job-exposure-matrix) either the duration of occupational work as an approximation to actual exposure was determined or a simple yes/no differentiation was used based on a definition of high-exposed and/or low-exposed (occupational) groups. Either total mortality, cancer mortality, cancer incidence or other outcomes were estimated. In most of the studies, an increased risk for various types of cancer was found in exposed study participants, although in different organs. The overall results were, however, inconsistent. The most important limitations of the studies were the lack of measurements referring to past and current exposures and, thus, the unknown details on actual exposure, the use of possibly biased data as well as the lack of adjustment for potential confounders and the use of indirect standardization techniques. Due to these limitations and the inconsistencies of the results it has to be concluded that the studies give no evidence of high frequency emissions causing cancer.

Introduction

With increasing application of electrical systems in households, during leisure time and at work places, the environment of humans has changed fundamentally. Due to the widespread use of cellular phones the portion of the population exposed to high frequency electromagnetic fields, such as radiofrequencies (RF) and microwaves, has increased rapidly. The possible effects of high frequency electromagnetic fields on the human organism are controversially discussed.

Although the proportion of occupationally exposed persons appears to be small, an increase in the number of exposed persons can be anticipated, in particular outside of the industrial production facilities (by use of cellular phones, wireless local area networks and/or bluetooth technologies).

There is a need to study the health risks of electromagnetic fields because of the ever-increasing sources of high frequency radiation (e.g. cellular telephones, cordless telephones, wireless local area networks, industrial machines) and the corresponding number of persons exposed. There is also a need to respond to the increasing public concern of potential health effects. So far, only a few epidemiological cohort studies investigating the effects of high frequency electromagnetic fields on the health status have been conducted. Concerning direct health effects of high frequency electromagnetic fields, these studies indicate an enhanced cancer risk, in particular brain tumors, leukemias and breast cancer. Regarding the incidence of cardiovascular diseases, no conclusions can be drawn from the cohort studies.

Apart from epidemiological studies on cancer risk within groups occupationally exposed to RF radiation, larger studies were carried out in populations living near transmitting towers. Such population-based studies were performed by Maskarinec et al. [1] in Hawaii, by Altpeter [2] in Switzerland, by Dolk et al. [3, 4] in Great Britain and by Hocking et al. [5] in northern Sydney. Some of these ecological studies found significantly enhanced rates of leukemia in the exposed populations.

J. Breckenkamp (✉) · G. Berg · M. Blettner
Faculty of Health Sciences Department 3 Epidemiology
and Medical Statistics,
University of Bielefeld,
PO Box 10 01 31, 33501 Bielefeld, Germany
e-mail: juergen.breckenkamp@uni-bielefeld.de
Tel.: +49-521-1065166
Fax: +49-521-1066009

This review is restricted to potentially high-exposed (occupational) groups, thus the comparatively low exposure of RF radiation from cellular phones was not considered. According to our definition, a cohort is based on historical and/or prospective individual data. Therefore our review does not include any ecological studies, but only those studies that are based on individual data.

The electromagnetic spectrum

The physical term electromagnetic field (EMF) covers the frequency range from 0 cycles per second up to 300 GHz which includes e.g. the static fields in nuclear spin tomography, the low frequency electric and magnetic fields and the high frequency electromagnetic fields used in industrial dielectric procedures. The most important applications in the high frequency range are radio, television and mobile communication technologies. The frequency of 30 kHz is generally used as a demarcation value between static fields/low frequency and high frequency fields, however, an international definition for the demarcation does not exist. The internationally accepted names commonly used for different coverages of frequencies are specified in Table 1.

Static fields occur in application areas of medicine, galvanotechnics, high energy accelerator technology and metallurgy. Low frequency fields occur within 50/60 Hz ranges of power supplies and railway current lines as well as in the area of warmth induction. Welding, melting, broadcasting and radio navigation are assigned to the frequency ranges “very low frequency” (VLF), “low frequency” (LF) and “medium frequency” (MF). High frequencies (HF) in the range of 3–30 MHz are typically found in industrial processes, such as warming up, drying, welding, gluing, polymerizing and sterilizing as well as in areas such as agriculture, medicine, radio astronomy and broadcasting. Areas of application within the range of ‘very high frequency’ (VHF) are found in industry, broadcasting and medicine as well as in television technology, air traffic control, radar and radio navigation. The “ultra high frequency” (UHF) band is utilized as mobile communication, television, radar, radio relay link,

portable radio, telemetry, medicine, microwave ovens and for procedures in food industry. Applications using the range of “super high frequency” (SHF) and “extremely high frequency” (EHF) are altimeters, radar, navigation, radio relay link, satellite, radio astronomy, radio meteorology, space research and radio spectroscopy [6].

Theoretical framework

Since currently it is not clear what parameters of the electromagnetic field have an influence on the biological effects in tissues, the rate of energy absorption per unit mass or specific absorption rate (SAR) is used, expressed as watts per kilogram (W/kg). Recommendations regarding limit values for the population are expressed by the International Commission on Non-Ionizing Radiation Protection (ICNIRP). One SAR of 0.08 W/kg body weight [7, 8] is considered as the limit value for whole-body irradiation in the general population. For vocational activities higher limit values are given and the limit value for whole-body irradiation is 0.4 W/kg body weight. Considering the fact that the threshold level for thermal effects according to current knowledge is 4 W/kg, the limit value for occupational exposure is based on a safety factor of 10 [8].

So far, it is not clear by which mechanisms high frequency electromagnetic fields with an intensity below the limit values could affect biological systems [9]. Higher intensities are well known to lead to thermal effects. In contrast to ionizing radiation, the energy of high frequency electromagnetic fields is not high enough to be able to ionize atoms or molecules or to open chemical bonds. Chemical changes may, however, occur in cells, at least with high intensities.

Experimental studies show that the exposure to high frequency fields has no carcinogenic effect in the sense of initiating a tumor cell, but exposure to high frequency fields may indirectly promote tumor growth or facilitate the absorption of carcinogenic substances into the cell [10, 11]. Theoretical approaches to biochemical mechanisms of tumor induction initiated by high frequency fields assume that proteins changed by heat shock may work as tumor promoters [12]. In experimental studies with cell cultures and on animals, the use of electromagnetic energies leading to an increase in cell or body temperature demonstrated a number of further effects [13]. Among these effects are changes of neural and neuromuscular functions, higher permeability of the blood-brain barrier, degradation of sight, stress-induced changes in the immune system, changes in the hematopoietic system, in reproduction ability, in cell morphology, and in water and electrolyte equilibrium as well as in the function of cell membranes. There is a series of experimental studies giving evidence that some of the effects indicated above may be initiated by low intensities of pulsed or continuous high frequency fields, such as elevated permeability of the blood-brain barrier, changes in the immune system or infertility [14, 15, 16].

Table 1 Ranges of radiofrequencies (Brüggemeyer H et al. [6])

International designation	Range	
	From	To
Sub ELF (sub-extremely low frequency)	0 Hz	30 Hz
ELF (extremely low frequency)	30 Hz	300 Hz
VF (voice frequency)	300 Hz	3 kHz
VLF (very low frequency)	3 kHz	30 kHz
LF (low frequency)	30 kHz	300 kHz
MF (medium frequency)	300 kHz	3 MHz
HF (high frequency)	3 MHz	30 MHz
VHF (very high frequency)	30 MHz	300 MHz
UHF (ultra high frequency)	300 MHz	3 GHz
SHF (super high frequency)	3 GHz	30 GHz
EHF (extremely high frequency)	30 GHz	300 GHz

Methods

Literature search

We searched for cohort studies on the subject “health risk by exposure to high frequency electromagnetic fields”. The search was performed using the literature data bases Medline, CancerLit and HealthStar and the EMF data base of the WHO. Different search words were used, among others: “electromagnetic fields, radiation, radiation/adverse effects, cancer, cohort study”. In Medline the filters “human” and “English” were set. In HealthStar and CancerLit the filter “non Medline” was also used.

In addition, a search was conducted in Medline with given medical subject headings (MeSH), i.e. “electromagnetic fields/adverse effects, radiation non-ionizing/adverse effects”. For this search the methodical filter “cohort study” was supplementary used.

All searches were restricted to publications in English language and to the time period 1970–2002. We did not define a minimum size for the cohorts to be included. Overall 10 publications from 9 cohort studies were found.

Criteria for evaluation

The criteria for evaluation of available cohort studies were:

- Size of the cohort
- Completeness of reporting other important characteristics of the cohort (age, sex distribution)
- Exposure (potential exposure)
- Exposure measurements
- Confounding factors
- Incidence/mortality
- Quality of follow-up (length, completeness).

In fact, the weaknesses of the studies outlined in this paper were partly discussed by the respective authors themselves or already referred to in other reviews [17, 18].

Results

The results of the nine cohort studies investigated were published between 1980 and 2002 (Table 2). The size of the cohorts varied between 304 participants with 3,362 person years [19] and nearly 200,000 with 2.7 million person years [20]. As exposures, both pulsed and non-pulsed high frequency electromagnetic fields from different sources were defined.

Table 3 summarizes all the results for all cancers. Furthermore, some selected results, mostly increased standardized mortality ratios (SMR) and standardized incidence ratios (SIR) are presented in the text. The results of some subcohort comparisons presented in this Results section might be biased due to the use of indirect standardization. This problem is dealt with in the Discussion section.

In order to achieve better legibility, most of the confidence intervals are given in Table 3. An asterisk (*) is used to indicate statistically significant results and *n.s.* for indication of insignificant results.

Italian plasticware workers (I)

Lagorio et al. [21] examined the influence of high frequency radiation of dielectric heat sealers on cancer mortality in persons employed in an Italian plasticware manufacturing plant. A total of 201 men and 481 women, who had been employed between 1 October 1962 (beginning of factory production) and 30 September 1992 were concluded to be qualified for the study. The calculation of person years was based on the period between the entry into the study and the end of observation, 30 December 1992. The evaluation was limited to 481 women (10,609 person years) who contributed more than three-quarters of all person years. Men were excluded because only four men worked as sealer operators, with the majority working in technical and servicing jobs.

Exposure was defined by length of time employed and by three occupational groups (RF sealer operators, other laborers, white-collar workers). The sub-cohort of white-collar workers was ignored as it comprised only 29 women (for an overview see Table 2). From the study population 302 females were exposed to RF radiation.

Compared to the regional mortality rates, an increased mortality was found in the exposed sub-cohort for malignant neoplasms (SMR 2.0^{n.s.}), accidents (SMR 2.4^{n.s.}) and mortality of all causes (SMR 1.4^{n.s.}). The SMR of all causes of death did not differ from the corresponding SMR of women not working on RF heat sealers (SMR 1.3^{n.s.}).

Both the minor size of the cohort and the small number of deceased female employees ($n=14$) contributed to the limited power of the study.

Five female employees (1% of the study population) lost to follow-up were considered to be alive by the end of the study, an assumption which possibly biases the results as it cannot be excluded that one or more of these females died from cancer after having been lost to follow-up. In addition, this fact causes a slight overestimation of person-years at risk.

No information was given about the completeness of personnel files, providing personal data and data of employment.

The study gives no information about individual exposure to RF radiation and, as stated above, job title and time in the job were used instead. An exposure to the carcinogenic vinyl chloride monomer with an average of 37 $\mu\text{g}/\text{m}^3$ was given for sealer operators in 1983, but no information was available for the other years of the follow-up period. The authors did not consider this exposure when data analyses were performed.

The study reports only cancer mortality but it would have been useful to report cancer incidence too.

Table 2 Cohort studies on radiofrequency and microwave emissions

Study ¹	Cohort	Exposure	Outcome	Method
Groves et al. 2002, USA [30]	Vb US Naval personnel, who served during Korean War period, 40,890 men, 1950–1997	Microwaves (radar equipment) Frequency range: not stated Exposure intensity: mean exposure <1 mW/cm ² ; infrequently exposure >100 mW/cm ² (assumptions) High and low exposed group, defined by job title No information about individual exposure	Mortality by cause of death	SMR, RR, internal and external comparisons (with age-specific white male death frequencies)
Morgan et al. 2000, USA [20]	IX Employees of Motorola, 195,775 persons, 2.7 million person years, 1976–1996	Radio frequencies (wireless communication technologies) Frequency range: not stated Exposure intensity: not stated Qualitative job exposure matrix: 4 groups by job title: background, low, moderate, high No information about individual exposure	Mortality: all-cause, all cancers, brain cancer, all lymphatic/hematopoietic system cancers combined, leukemia	SMR, RR, internal and external comparisons
Finkelstein 1998, Canada [28]	IV Ontario police officers, 22,197 persons, 1970–1995	Microwaves (traffic radar units) Frequency range: 10.525 GHz (early devices), 24.15 GHz (since 1975), 35.0 GHz (devices introduced in mid of the 1990s, but not widespread) Exposure intensity: not stated Years from employment or department entry date (duration only) No information about individual exposure	Cancer incidence: testis, leukemia, brain, eye, skin	SIR, external comparisons
Lagorio et al. 1997, Italy [21]	I Plasticware workers, 481 women, 10,609 person years, 1962–1992	Radio frequencies (dielectric heat sealers) Frequency range: not stated, but presumable 27.12 MHz Exposure intensity: max. >10 W/m ² in the mid-1980s (no metal-shielding, no earthing) Job title and time in the job; 3 sub cohorts, 1 ignored due to 29 women only No information about individual exposure	Total mortality, cancer mortality	SMR, internal and external comparisons
Tynes 1996, Norway [23]	III Female radio and telegraph operators working at sea, 2,619 women, 72,105 person years, 1961–1991	Exposure to light at night, radio frequency and to some extent, extremely low frequency fields Frequency range: 405 kHz–25 MHz Exposure intensity: below detection level at operators desk; 0.5 m from the front of the tuner and 1.5–2 m above floor level 70–200 V/m and 0.1–0.5 A/m; close to unshielded antenna 1,400 V/m and 7.5 A/m Spot measurements of radio frequency fields, time in the job No information about individual exposure	Breast cancer (and cancer of other tissues)	SIR, OR, internal and external comparisons
Szmigielski 1996, Poland [31]	VI Military career personnel, 128,000 persons a year, 1971–1985	Exposure to radio frequencies/microwaves (pulse-modulated high frequency electromagnetic fields) Frequency range: 150 MHz–3.5 GHz Exposure intensity: 85% of posts <2 W/m ² , other posts 2–6 W/m ² , incidental exceeding 6 W/m ² High and low exposed group, defined by professional activity No information about individual exposure	Cancer morbidity	OER, internal comparisons
Muhm 1992, USA, [19]	VII Workers in an electromagnetic pulse test program, 304 men, 3,362 person years, 1970–1986	Nuclear-exposition-related electromagnetic pulses Frequency range: 10 kHz–100 MHz Exposure intensity: not stated Potential exposure ≥30 days/6 months No information about individual exposure	Each cause of death	SMR, external comparisons
Tynes et al. 1992, Norway [32]	VIII Norwegian electrical workers, 37,945 men, 824,321 person years, 1961–1985	Extremely low frequencies and radio frequencies Frequency range: not stated Exposure intensity: not stated Job description from census data, occupations classified into five categories of exposure No information about individual exposure	Cancer incidence (leukemia and brain tumors)	SIR, internal and external comparisons

Table 2 (continued)

Study ¹	Cohort	Exposure	Outcome	Method
Milham 1988, USA [22]	II Amateur radio operators, 67,829 persons, 232,499 person years, 1979–1984	Radio frequencies Frequency range: not stated Exposure intensity: not stated License as indicator of possible exposure No information about individual exposure	Mortality by cause of death	SMR, external comparison
Robinette et al. 1980, USA [29]	Va US Naval personnel, who served during Korean War period, 40,890 men, 1950–1974	Microwaves (radar equipment) Frequency range: not stated Exposure intensity: mean exposure <1 mW/cm ² ; infrequently exposure >100 mW/cm ² (assumptions) High and low exposed group, defined by job title No information about individual exposure	Mortality by cause of death, hospitalization during military service, later hospitalization in Veterans Administration facilities	MR (observed/expected), internal comparisons

¹ Roman numerals indicate the order of the studies as being discussed in the text.
1 W/m²=0.1 mW/cm².

U.S. Amateur radio operators (II) / Norwegian female radio and telegraph operators (III)

In two cohort studies the possible effects of high frequency electromagnetic fields on radio operators was investigated [22, 23].

In a cohort of 67,829 amateur radio operators (232,499 person years), registered in Washington State or California, 2,485 men were identified to have died within the period 1979–1984. Women were excluded from the study as only a few of them had radio licenses. The period between the day of licensing and 31 December 1984 or the day of death was used to determine person years at risk. All persons with a valid license were regarded as exposed. Causes of death of the deceased male radio operators were compared with the US death rates.

The number of the cases was sufficient for the differentiation of malignant neoplasms according to the organ/tissues concerned. The observed overall mortality in radio operators was clearly lower than the expected mortality, based on the US death rates (SMR 0.71*). Increased mortality was found for neoplasms of the lymphatic and hematopoietic systems (SMR 1.23^{n.s.}) as well as in the sub-groups leukemia (SMR 1.24^{n.s.}), acute myeloid leukemia (SMR 1.76*) and for neoplasms of other lymphatic tissues (SMR 1.62*), only.

Unfortunately, the author gave no information on the mean age of the cohort members at study entry even though the dates of birth were available from the amateur radio operator files used. He only cited the results of another publication which indicated that the average amateur radio operator was a 46-year-old male first licensed in 1963 who spent 6.1 h per week on his hobby [24].

A differentiation regarding license class was used as a crude estimator of years operating, but not published as part of the study. Results showed lowest SMRs for the license class “novice”, the entry level for amateur radio operators. With an average age of 38.4 years, the license

holders in this class were substantially younger than the average license holders in the other four classes [25].

An exposure to RF radiation was assumed for persons licensed as amateur radio operators. The proportion of amateur radio operators who did not send but only received radio traffic is however unclear. This is an important question as no exposure is experienced when only receiving radio traffic. Also no information was available about the year a person was first licensed or years of operating.

Some 31% of the Washington State amateur radio operators worked in occupations with exposure to RF radiation or power frequency electromagnetic fields (EMF) as radio operator, television repairman, electronics technician, etc. However, these jobs were listed only in a fraction of 3% of the Washington State male death certificates. This difference could lead to an underestimation of exposure to electromagnetic fields for amateur radio operators. Consequently, the results may be biased by an overestimation of the possible effects due to exposure. It cannot be excluded that the higher SMRs of neoplasms of the lymphatic and hematopoietic system were due to an effect of EMF exposure. EMF exposure may result in a depression of the melatonin production, which diminishes the assumed oncogenic effect of melatonin [26, 27].

An internal comparison of occupationally exposed and non-exposed radio operators could have given some information about the possible overestimation of effects due to leisure time RF radiation exposure.

In the second study [23] the incidence of breast cancer was analyzed in women who worked professionally at sea as radio or telegraph operators and were additionally exposed to light at night.

The study design is somewhat complicated as three different cohorts were described and matched in parts. A nested case-control study was also described in the same publication. The following description refers only to the so-called telecom cohort (TC), the most complete cohort, and the nested case-control study.

Table 3 Radiofrequency/microwave emissions and morbidity/mortality

Outcome	Classification	Exposure	Results			Study		
			Measure	CI*				
All diseases	ICD 8 (000–796)	Radar	MR	0.96	–	Robinette et al. 1980 ¹ [29]	Va	
	ICD 9 (001–999)	Radar	SMR	0.69	0.67–0.71	Groves et al. 2002 ² [30]	Vb	
	ICD 9 (001–799)	Radar	SMR	0.65	0.63–0.67	Groves et al. 2002 [30]	Vb	
	ICD 9 (001–999)	RF	SMR	1.4	0.7–2.7	Lagorio et al. 1997 [21]	I	
	ICD 8 (000–999)	RF	SMR	0.71	0.69–0.74	Milham 1988 [22]	II	
	ICD 9 (001–999)	EMP	SMR	0.56	0.31–0.95	Muhm 1992 [19]	VII	
Malignant neoplasms	ICD 8 (140–209)	Radar	MR	01. Apr	–	Robinette et al. 1980 [29]	VA	
	ICD 9 (140–208)	Radar	SMR	0.73	0.69–0.77	Groves et al. 2002 [30]	Vb	
	ICD 9 (140–208)	RF	SMR	2.0	0.7–4.3	Lagorio et al. 1997 [21]	I	
	ICD 8 (140–209)	RF	SMR	0.89	0.82–0.95	Milham 1988 [22]	II	
	ICD 9 (140–208, 238, .4, .6, 289.8 EX 202, .2, .3, .5, .6)	EMP	SMR	0.32	0.04–1.15	Muhm 1992 [19]	VII	
	Not defined	Radar/RF	OER**	2.7	1.12–3.58	Szmigielski 1996 [31]	VI	
	ICD 9 (140–208)	Radar	SIR	0.90	0.83–0.98	Finkelstein 1998 ³ [28]	IV	
	Not defined	RF	SIR	1.2	1.0–1.4	Tynes et al. 1996 [23]	III	
	ICD 7 (140–204)	RF/ELF	SIR	1.6	1.03–1.09	Tynes et al. 1992 [32]	VIII	
	Oral cavity	not defined	Radar/RF	OER	0.71	0.42–1.32	Szmigielski 1996 [31]	VI
		ICD 9 (140–149)	Radar	SIR	0.71	0.45–1.06	Finkelstein 1998 [28]	IV
	Pharynx	Not defined	Radar/RF	OER	1.08	0.82–1.24	Szmigielski 1996 [31]	VI
Buccal cavity and pharynx	ICD 9 (140–149)	Radar	SMR	0.49	0.32–0.76	Groves et al. 2002 [30]	Vb	
	ICD 7 (140–148)	RF/ELF	SIR	0.91	0.76–1.09	Tynes et al. 1992 [32]		
Esophagus	ICD 9 (150.0–150.9)	Radar	SMR	1.08	0.82–1.42	Groves et al. 2002 [30]	Vb	
	ICD 8 (150)	RF	SMR	1.13	0.71–1.72	Milham 1988 [22]	II	
	ICD 7 (150)	RF/ELF	SIR	0.93	0.66–1.27	Tynes et al. 1992 [32]	VIII	
Stomach	ICD 8 (151)	RF	SMR	1.02	0.68–1.45	Milham 1988 [22]	II	
	ICD 7 (151)	RF	SIR	0.4	0.1–2.0	Tynes et al. 1996 [23]	III	
	ICD 7 (151)	RF/ELF	SIR	1.08	0.97–1.20	Tynes et al. 1992 [32]	VIII	
Esophagus and stomach	Not defined	Radar/RF	OER	3.24	1.85–5.06	Szmigielski 1996 [31]	VI	
Large intestine (colon)	ICD 8 (153)	RF	SMR	1.11	0.89–1.37	Milham 1988 [22]	II	
	ICD 7 (153)	RF	SIR	1.3	0.6–2.6	Tynes et al. 1996 [23]	III	
	ICD 7 (153)	RF/ELF	SIR	1.13	1.01–1.26	Tynes et al. 1992 [32]	VIII	
Rectum	ICD 8 (154)	RF	SMR	0.77	0.42–1.29	Milham 1988 [22]	II	
	ICD 7 (154)	RF	SIR	1.8	0.7–3.9	Tynes et al. 1996 [23]	III	
	ICD 7 (154)	RF/ELF	SIR	1.1	0.87–1.17	Tynes et al. 1992 [32]	VIII	
Colorectal	Not defined	Radar/RF	OER	3.19	1.54–6.18	Szmigielski 1996 [31]	VI	
Liver	ICD 8 (155)	RF	SMR	0.65	0.33–1.17	Milham 1988 [22]	II	
	ICD 7 (155)	RF/ELF	SIR	1.11	0.74–2.66	Tynes et al. 1992 [32]	VIII	
Gall bladder	ICD 7 (156)	RF/ELF	SIR	0.71	0.37–1.24	Tynes et al. 1992 [32]	VIII	
Pancreas	ICD 8 (157)	RF	SMR	0.64	0.42–0.94	Milham 1988 [22]	II	
	ICD 7 (157)	RF	SIR	0.6	0.0–3.5	Tynes et al. 1996 [23]	III	
	ICD 7 (157)	RF/ELF	SIR	1.19	1.01–1.38	Tynes et al. 1992 [32]	VIII	
Liver, pancreas	Not defined	Radar/RF	OER	1.47	0.76–3.02	Szmigielski 1996 [31]	VI	
Digestive system	ICD 9 (150–159)	Radar	MR	1.14	–	Robinette et al. 1980 [29]	Va	
	ICD 8 (150–159)	Radar	SIR	0.92	0.77–1.09	Finkelstein 1998 [28]	IV	
Larynx	ICD 9 (161)	Radar	SIR	0.98	0.52–1.68	Finkelstein 1998 [28]	IV	
	ICD 7 (161)	RF/ELF	SIR	1.39	1.08–1.76	Tynes et al. 1992 [32]	III	
Trachea, bronchus, lung	ICD 9 (162.0–162.9)	Radar	SMR	0.64	0.58–0.70	Groves et al. 2002 [30]	Vb	
	ICD 9 (162)	Radar	SIR	0.66	0.52–0.82	Finkelstein 1998 [28]	IV	
	ICD 7 (162)	RF	SIR	1.2	0.4–2.7	Tynes et al. 1996 [23]	III	
	ICD 7 (162)	RF/ELF	SIR	1.9	1.00–1.19	Tynes et al. 1992 [32]	VIII	
Laryngeal, lung	Not defined	Radar/RF	OER	1.06	0.72–1.56	Szmigielski 1996 [31]	VI	
Pleura	ICD 7 (163)	RF/ELF	SIR	1.88	1.13–2.93	Tynes et al. 1992 [32]	VIII	
Respiratory system	ICD 8 (160–163)	Radar	MR	1.14	–	Robinette et al. 1980 [29]	Va	
	ICD 8 (160–163)	RF	SMR	0.66	0.58–0.76	Milham 1988 [22]	II	
Bones	Not defined	Radar/RF	OER	0.67	0.36–1.42	Szmigielski 1996 [31]	VI	
	ICD 9 (170)	Radar	SIR	0.82	0.10–3.00	Finkelstein 1998 [28]	IV	
Soft tissue	ICD 9 (171)	Radar	SIR	1.12	0.45–2.31	Finkelstein 1998 [28]	IV	
	ICD 7 (197)	RF/ELF	SIR	1.36	0.93–1.91	Tynes et al. 1992 [32]	VIII	

Table 3 (continued)

Outcome	Classification	Exposure	Results			Study	
			Measure	CI*			
Melanoma	Not defined	RF	SMR	1.13	–	Morgan et al. 2000 [20]	IX
	ICD 9 (172)	Radar	SIR	1.45	1.10–1.88	Finkelstein 1998 [28]	IV
	ICD 7 (190)	RF	SIR	0.9	0.4–1.7	Tynes et al. 1996 [23]	III
	ICD 7 (190)	RF/ELF	SIR	1.09	0.91–1.45	Tynes et al. 1992 [32]	VIII
Non-melanoma skin cancer	ICD 7 (191)	RF/ELF	SIR	1.00	0.80–1.23	Tynes et al. 1992 [32]	VIII
Skin, including melanomas	Not defined	Radar/RF	OER	1.67	0.92–4.13	Szmigielski 1996 [31]	VI
Breast (male)	ICD 9 (175.0–175.9)	Radar	SMR	1.05	0.26–4.20	Groves et al. 2002 [30]	Vb
	(female)	ICD 7 (170)	RF	SIR	1.50	1.10–2.00	Tynes et al. 1996 [23]
(male)	ICD 7 (170)	RF/ELF	SIR	2.07	0.94–1.10	Tynes et al. 1992 [32]	VIII
Cervix	ICD 7 (171)	RF	SIR	1.0	0.6–1.7	Tynes et al. 1996 [23]	III
Ovary	ICD 7 (175)	RF	SIR	0.8	0.3–1.6	Tynes et al. 1996 [23]	III
Uterus	ICD 7 (172)	RF	SIR	1.9	1.0–3.2	Tynes et al. 1996 [23]	III
Prostate	ICD 8 (185)	RF	SMR	1.14	0.90–1.42	Milham 1988 [22]	II
	ICD 9 (185)	Radar	SIR	1.16	0.93–1.43	Finkelstein 1998 [28]	IV
	ICD 7 (177)	RF/ELF	SIR	1.02	0.94–1.10	Tynes et al. 1992 [32]	VIII
Kidney and prostatic	Not defined	Radar/RF	OER	0.86	0.54–1.67	Szmigielski 1996 [31]	VI
Testis	ICD 9 (186.0–186.9)	Radar	SMR	0.60	0.25–1.43	Groves et al. 2002 [30]	Vb
	ICD 9 (186)	Radar	SIR	1.30	0.89–1.84	Finkelstein 1998 [28]	IV
	ICD 7 (178)	RF/ELF	SIR	0.83	0.59–1.12	Tynes et al. 1992 [32]	VIII
Bladder	ICD 8 (188)	RF	SMR	0.66	0.38–1.08	Milham 1988 [22]	II
	ICD 9 (188)	Radar	SIR	0.93	0.63–1.33	Finkelstein 1998 [28]	IV
	ICD 7 (181)	RF	SIR	0.6	0.0–3.6	Tynes et al. 1996 [23]	III
	ICD 7 (181)	RF/ELF	SIR	1.23	1.10–1.38	Tynes et al. 1992 [32]	VIII
Kidney	ICD 8 (189)	RF	SMR	0.94	0.57–1.48	Milham 1988 [22]	II
	ICD 9 (189)	Radar	SIR	0.96	0.61–1.44	Finkelstein 1998 [28]	IV
	ICD 7 (180)	RF	SIR	1.6	0.3–4.8	Tynes et al. 1996 [23]	III
	ICD 7 (180)	RF/ELF	SIR	1.09	0.92–1.28	Tynes et al. 1992 [32]	VIII
Brain	ICD 9 (191.0–191.9)	Radar	SMR	0.71	0.51–0.98	Groves et al. 2002 [30]	Vb
	ICD 8 (191)	RF	SMR	1.39	0.93–2.00	Milham 1988 [22]	II
	ICD 9 (191)	Radar	SIR	0.84	0.48–1.36	Finkelstein 1998 [28]	IV
	ICD 7 (193)	RF	SIR	1.0	0.3–2.3	Tynes et al. 1996 [23]	III
	ICD 7 (193)	RF/ELF	SIR	1.09	0.90–1.41	Tynes et al. 1992 [32]	VIII
Nervous system, including brain tumors	Not defined	RF	SMR	0.53	0.21–1.09	Morgan et al. 2000 [20]	IX
	Not defined	Radar/RF	OER	1.91	1.08–3.47	Szmigielski 1996 [31]	VI
Unspecified organs	ICD 7 (199)	RF/ELF	SIR	0.91	0.76–1.07	Tynes et al. 1992 [32]	VIII
Thyroid	Not defined	Radar/RF	OER	1.54	0.82–2.59	Szmigielski 1996 [31]	VI
	ICD 9 (193)	Radar	SIR	0.86	0.32–1.87	Finkelstein 1998 [28]	IV
Hematopoietic	ICD 9 (200–208, 238.4, .6, 289.8, EX 202.2, .3, .5, .6)	EMP	SMR	3.31	0.40–11.96	Muhm 1992 (2 cases only) [19]	VII
Lymphatic and hematopoietic system	ICD 8 (200–209)	Radar	MR	1.18	–	Robinette et al. 1980 [29]	Va
	ICD 8 (200–209)	RF	SMR	1.23	0.99–1.52	Milham 1988 [22]	II
	Not defined	RF	SMR	0.54	0.33–0.83	Morgan et al. 2000 [20]	IX
	Not defined	Radar/RF	OER	6.31	3.12–14.32	Szmigielski 1996 [31]	VI
Lymphosarcoma/reticulosarcoma	ICD 8 (200)	RF	SMR	0.47	0.15–1.10	Milham 1988 [22]	II
Lymphoma	ICD 9 (200)	EMP	SMR	10.87	0.28–60.56	Muhm 1992 (1 case only) [19]	VII
Lymphoma and multiple myeloma	ICD 9 (200–203)	Radar	SMR	0.89	0.72–1.09	Groves et al. 2002 [30]	Vb
Non-Hodgkins lymphoma	ICD 7 (200–202)	RF/ELF	SIR	0.77	0.60–0.98	Tynes et al. 1992 [32]	VIII

Table 3 (continued)

Outcome	Classification	Exposure	Results		Study		
			Measure	CI*			
Hodgkins disease	ICD 8 (201)	RF	SMR	1.23	0.40–2.88	Milham 1988 [22]	II
	Not defined	RF	SMR	1.11	0.23–3.24	Morgan et al. 2000 [20]	IX
	ICD 9 (201)	Radar	SIR	0.84	0.36–1.66	Finkelstein 1998 [28]	IV
	ICD 7 (201)	RF/ELF	SIR	0.85	0.56–1.24	Tynes et al. 1992 [32]	VIII
Multiple myeloma	ICD 7 (203)	RF/ELF	SIR	1.02	0.79–1.30	Tynes et al. 1992 [32]	VIII
Leukemia	ICD 9 (204–208)	Radar	SMR	1.14	0.90–1.44	Groves et al. 2002 [30]	Vb
	ICD 8 (204–207)	RF	SMR	1.24	0.87–1.72	Milham 1988 [22]	II
	Not defined	RF	SMR	0.77	0.38–1.38	Morgan et al. 2000 [20]	IX
	ICD 9 (204–208, 202.4, 203.1)	EMP	SMR	4.37	0.11–24.33	Muhm 1992 (1 case only) [19]	VII
	ICD 9 (204–208)	Radar	SIR	0.60	0.31–1.05	Finkelstein 1998 [28]	IV
	ICD 7 (204)	RF/ELF	SIR	1.1	0.1–4.1	Tynes et al. 1996 [23]	III
Other lymphatic tissue	ICD 7 (204)	RF/ELF	SIR	1.08	0.89–1.31	Tynes et al. 1992 [32]	VIII
	ICD 8 (202, 203)	RF	SMR	1.62	1.17–2.18	Milham 1988 [22]	II
Lymphocytic leukemia	ICD 9 (204.0–204.9)	Radar	SMR	1.12	0.69–1.83	Groves et al. 2002 [30]	Vb
Non-lymphocytic leukaemia	ICD 9 (205.0–207.7, 207.9)	Radar	SMR	1.24	0.90–1.69	Groves et al. 2002 [30]	Vb
Circulatory diseases	ICD 8 (390–458)	Radar	MR	0.93	–	Robinette et al. 1980 [29]	Va
	ICD 9 (390–459)	Radar	SMR	0.65	0.62–0.69	Groves et al. 2002 [30]	Vb
	ICD 8 (390–458)	RF	SMR	0.70	0.66–0.74	Milham 1988 [22]	II
Respiratory diseases	ICD 9 (460–519)	Radar	SMR	0.51	0.44–0.60	Groves et al. 2002 [30]	Vb
	ICD 8 (460–519)	RF	SMR	0.50	0.42–0.60	Milham 1988 [22]	II
Accidents and violence	ICD 9 (800–999)	Radar	SMR	0.79	0.73–0.85	Groves et al. 2002 [30]	Vb
	ICD 9 (800–999)	RF	SMR	2.4	0.3–8.7	Lagorio et al. 1997 [21]	I
	ICD 8 (E800–E999)	RF	SMR	0.64	0.52–0.77	Milham 1988 [22]	II

¹ MR of the total high-exposed group, standardized for year of birth.

² External comparison with the high exposed subcohort (morbidity).

³ We assume that the coding of Finkelstein is based on ICD 9, since classification is restricted to 140–208. Finkelstein used one-tailed statistical tests and computed 90% confidence intervals.

* CI Confidence Interval.

** Observed/expected ratio of incidence.

The TC cohort consisted of 2,619 women (72,105 person years), who were certified between 1920 and 1980. During the follow-up period from 1961 to 1991, 140 new cancers occurred. Increased SIR values were found for all cancers (SIR 1.2^{n.s.}) and for breast cancer (SIR 1.5*). The calculated SIR values were based on the Norwegian female population as reference.

In the context of a nested case-control study, internal comparisons were accomplished. A total of 50 women with cancer diagnosis were matched with 4–7 controls. Detailed job histories were collected from the Norwegian seamens registry.

The duration of occupational activity and night-shift were defined as exposure, and in each case divided into three categories. For women 50 years and older, a dose-response relationship was found between the risk of breast cancer and the duration of occupation (odds ratios 1.0, 1.9, 5.9, *p*-value for trend 0.02) as well as between the risk of breast cancer and shift work (odds ratios 1.0, 3.3, 6.1, *p*-value for trend 0.01). However, after adjustment for each other, the trend was statistically insignificant.

The authors gave no information about the mean age of the participants at study entry, but mentioned that the mean age at certification was 23 years. There was also no

information regarding the extent to which radio operating devices were used in leisure time.

The authors had no information about individual exposure, therefore spot measures were performed in operator rooms of ships equipped with older radio operating devices.

The possible influence of electromagnetic field exposure and exposure to light at night, especially the influence of combined exposures on a reduced production of melatonin was formulated as a hypothesis that should be evaluated by further studies.

Canadian police officers (IV) / U.S. Navy technicians I (Va) / U.S. Navy technicians II (Vb)

Three studies into health effects of radiation within the microwave range (radar) are presented. The Canadian study [28] examined the risk for cancer among police officers exposed to radiation from radar devices for speed measurement. The study population consisted of 1,596 female and 20,601 male police officers. The admission into the follow-up, running from 1970 to 1995, took place either on the basis of date of employment or at the time at

which a police department could identify the cohort completely. For police officers in departments that could supply only data of current employees, the entrance date was specified as 1 January 1992. Due to the unequal distribution of the sexes the evaluation was limited to the male participants only.

Population-based data were used as a reference to determine SIR. The vocational activity was considered as exposure, a measurement or estimation of exposure was not available.

A total of 561 cases of malignant disease were observed during follow-up and based on the Ontario population disease rates, this was less than expected (SIR 0.90*). The authors attributed this result to the healthy worker effect, as among other things the proportion of smokers among police officers is lower than in the general population. In this study an increased risk was found for malignant neoplasms of the prostate (SIR 1.16^{n.s.}), for testicular cancer (SIR 1.3^{n.s.}) and for melanoma (SIR 1.45*).

A limitation might be that not all invited police departments participated in the study (self-selection). Firstly, there was no information about the number of police officers employed in these departments, and secondly, the individual use of radar might be less or more frequent in those departments which did not participate.

No categorization of the participating police officers into those who used the radar device frequently, occasionally or never, was done. The authors stated rather generally that the “use of radar speed-measuring devices increased rapidly” during the 1970s and that radar units, in many cases, were used almost daily.

As stated before, the individual exposure was determined in the context of a case-control study. It has also been stated that there was no biological model explaining the increased incidence for most of the cancers because the maximum penetration of energy is about 1 cm.

“Since the Ontario cancer registry counts multiple primaries in the same individual, officers were not withdrawn from follow-up on the date of diagnosis of a first tumor, but were at risk of the diagnosis of multiple primaries during the follow-up period” [28]. This procedure does not permit a comparison with the results of the other incidence studies described here, as these studies defined the first diagnosed tumor as outcome.

Robinette et al. [29] accomplished a study with 40,890 members of American naval personnel, who had been in military service during the Korean War. The cohort was followed up from 1950 to 1974.

Based on measurements which the Navy had performed on ships, two sub-cohorts were formed to determine the effects of an exposure to microwaves (radar). A minimum and a maximum exposed cohort was formed, each comprising approximately 20,000 persons. Three military occupational classes, i.e. electronics technicians, fire control technicians, and aviation electronics technicians, were classified as potentially highly exposed

and radio operators, radar operators and aviation electrician’s mates were classified as potentially least exposed.

Potential exposure was estimated by job category and by a so-called hazard number, which was constructed on the basis of individual records using “the sum of all power ratings of all fire control radars aboard the ship, or search radars aboard the aircraft to which the technician was assigned, multiplied by the number of months of assessment”. The assessment of the hazard number was done for those 435 men who died and for a 5% randomly selected sample of living men.

A comparison within the potentially high exposed group regarding hazard numbers (less than or more than 5,000 hazards) showed a significant difference for malignant neoplasms of the respiratory tract with a corresponding mortality ratio of 2.20; $p < 0.05$ (10 cases).

A comparison with external data (causes of death) was not done, therefore combined mortality ratios were used as standard for the computation of mortality ratios from each of the two sub-cohorts. The results, limited to internal comparisons, did not show unfavorable effects in the highly exposed group: in the case of malignant neoplasms (altogether 202 cases) the mortality ratio (MR) was 1.04 in the maximum exposed and 0.96 in the minimum exposed cohort. Due to the fact that some comparison with an unexposed group was missing the results presented here can be interpreted with reservation only.

Assessment of exposure was limited to the time period from 1950 to 1954. The study gives no information about a possible exposure of participants after the Korean War period, neither in the Navy nor in the civilian field. Possible further occupational or environmental exposure to chemicals etc. were unknown, date of birth and year of graduation were incomplete for many subjects, and the use of job titles gave only a crude estimate of exposure.

In 2002, the results of the extended follow-up period of more than 40 years (1950 through 1997) in the cohort of the Navy personnel were published by Groves et al. [30]. Contrary to the work of Robinette et al. [29], nine possible confounders and effect modifications by age at cohort entry were assessed and an external comparison regarding causes of death was processed.

For most of the diseases, the potentially high-exposed stratum had lower mortality rates than the potentially low-exposed stratum, with the exception of higher rates that were statistically significant in all leukemias (RR 1.48, 95% CI 1.01–2.17) and in the sub-cohort “non lymphatic leukemia” (RR 1.82, 95% CI 1.05–3.14). Some higher rates were additionally found in “all external causes of death”, primarily due to “accidents involving air transportation” and “injuries resulting from operations of war”.

The standardized mortality ratios (external comparison) showed lower values being statistically significant or values near 1 (not significant) for the potentially high-exposed stratum.

Information about a possible exposure of participants to microwave radiation after the Korean War period—in the Navy or the civilian field—was not available. Other

limitations were the unawareness of further exposures (occupational or environmental exposure to chemicals etc.), the unawareness of date of birth and year of graduation for many subjects, and the use of job titles [see 30].

One aspect not discussed by the authors themselves is the use of the age-specific mortality rates of US Caucasian males for external comparisons, whereby it can be assumed that the cohort did not consist of Caucasian Americans only. However, the extent of a possible inaccuracy resulting from this procedure cannot be estimated.

Polish military personnel (VI)

In the Polish study, all cancers that occurred in military career personnel between 1971 and 1985 were registered [31]. From the (on average) 128,000 persons each year approximately 3,700 were exposed to pulse-modulated 150–3,500 MHz radiofrequency/microwave radiation. Data of exposure to radiation-emitting equipment on jobs were documented by the military and could be made available. However, it was not possible to determine the individual exposure of the military personnel. Instead, the study worked with a yes/no assumption.

Analyses were limited to comparison of the exposed to the non-exposed group within the cohort. Observed/expected ratios (OER) were calculated using the non-exposed group as reference. The OER thus corresponds to the odds ratio. An increased morbidity was found for malignant neoplasms (OER 2.07*), among them colorectal neoplasms (OER 3.19*), malignancies of the esophagus and stomach (OER 3.24*), neoplasms of the nervous system including brain tumors (OER 1.91*) and malignancies of the haematopoietic system and lymphatic organs (OER 6.31*).

Results of the study may be biased by the fact that the study population differed from year to year, because military personnel left or entered the army. Cancers occurring after the military service were not detectable with this study design. Additionally, the exact age distribution was unknown. The authors stated that “The exact age distribution is still classified information and therefore, the results may be given only in the form of incidence rates and odds ratios”.

Another problem already mentioned by Elwood [17], has to do with the fact that “a serviceman who developed cancer had more sources of information on possible RF exposures compared to a serviceman who did not develop cancer”. While for servicemen who did not develop cancer the service occupational records were available to obtain information on the exposure, for servicemen that developed cancer additional information regarding exposure was collected from the records of military hospitals and the central military medical board. Thus it is more probable that an exposure is documented. This may explain the increased OER of different neoplasms compared to other studies.

Study results were not compared to the expected cancer morbidity in the Polish population. Therefore a possible “healthy worker effect” in both the exposed and the unexposed military personnel cannot be determined. This effect could lead to an overestimation of cancer occurrence, while the actual result would show lower OERs for all localizations of cancer.

Workers in an electromagnetic pulse test program (EPTP) (VII)

Muhm [19] conducted a cohort study among 304 men (3,362 person years) employed in a company that performed tests regarding the simulation of electromagnetic impulses, similar to those resulting from an “interaction between nuclear explosion and the surrounding atmosphere”.

Exposed employees were not identified directly. Participation on special mandatory health surveillance examinations for employees working in the testing program, was used as an indirect marker of exposure. Only those employees who underwent such an examination between 1970 (beginning of examinations) and 31 December 1986 were included. Female employees were excluded as they represented less than 2% of the exposed study population.

SMRs were computed using age, sex, year and race-specific mortality rates in the USA as reference. Results should be interpreted with caution, because only 2 of the 14 deaths were attributed to malignant neoplasms. For all causes of death the SMR (0.56*) was lower than expected. For neoplasms of the hematopoietic system (2 cases) the SMR (3.31^{n.s.}) was higher than expected.

The small number of reported deaths ($n=14$), the fact that the vital status of 10 former employees could not be determined and the incomplete ascertainment of other health-endangering exposures can all lead to the results being biased. An exposure to chemicals and ionizing radiation exceeding that of the general population was known for 46 and 73 men, respectively. Whether or not there was an exposure to chemicals is unknown in 226 cases, and for 78 cases the exposure status regarding ionizing radiation was not available.

The completeness of exposed employees is at least questionable. The author identified 233 employees, that had undergone the health examinations, but an external institute reported about health examinations of 400 employees in 1976. Another critical aspect is that the health examination was only “required for anyone who was expected to be exposed to EPTP for at least 30 days during a 6-month period”. It is not stated whether one health examination was considered as a criteria to be included into the study group or the examinations had to be repeated regularly to be considered as exposed. Thus it is possible that for example employees regularly exposed for 15 days in a 6-months period for years, were excluded from the study although they were the group with the highest exposure.

Norwegian electrical workers (VIII)

In Norway the incidence of leukemias and brain tumors was determined in a cohort of 37,945 persons (824,321 person years) employed in the electrical branch [32]. Information on occupational activity was available from the 1960 and 1970 censuses.

The study included all men working in occupations with a possible exposure to electromagnetic fields in 1960. Analyses were performed with the data of the entire cohort (follow-up 1961–1985, 3,806 cancers) as well as with the data of a sub-cohort (follow-up 1970–1985, 2,065 cancers). The sub-cohort (group 2) consisted of those men who were also occupationally active in 1970. The authors assumed that the employees economically active in 1970 had been continuously employed in the same job from 1960 to 1970 (group 2).

Exposure was classified into five categories: (1) weak magnetic fields, (2) intermediate magnetic fields, (3) radiofrequency, (4) weak magnetic and electric fields, and (5) heavy magnetic and electric fields. The occupations were assigned to each group respectively. The cohort members were linked to the Norway cancer registry by their personal identification number. For the computation of the expected number of cancers the occupationally active male population served as reference.

Analysis of the complete cohort showed the highest SIR for leukemia (SIR: 2.56; 95% CI: 0.94–5.58) in radio/television repairmen. Analysis of the subcohort again showed the highest SIR (3.18; 95% CI: 1.03–7.43) for leukemia in radio/television repairmen. One occupational group had remarkable high values regarding brain tumors in the sub cohort: The authors calculated a SIR of 2.20 (95% CI: 1.10–4.18) for railway track walkers (9 cases).

Additionally, data were evaluated according to the type of the exposure (see above). An exposure to high frequency or to heavy magnetic and electrical fields resulted in higher risks of leukemia with SIR values of 2.85 (95% CI: 1.30–85.41) and, 1.79 (95% CI: 1.09–2.76), respectively. Exposure to weak magnetic and electrical fields was associated with an SIR of 2.20 (95% CI: 1.01–4.18) for brain tumors.

The use of the job description from the 1960 census data as an indirect indicator of exposure to electromagnetic fields conceals the danger of a low accuracy. The reasons are that the percentage of exposed employees in the jobs of interest, as well as the duration and the level of exposure had been estimated. Therefore the results of this study should be interpreted with caution.

Motorola employees (IX)

From 1976 to 1996, Morgan et al. [20] performed a follow-up on 195,775 employees of Motorola, with a total of 2.7 million person years. The aim of the study was to investigate the number of deaths from brain tumors and from malignant neoplasms of the lymphatic and haematopoietic system: 6,269 employees died during

follow-up. In 52 cases, the cause of death was a brain tumor and in 193 cases malignant neoplasms of the lymphatic and haematopoietic system.

Using a qualitative job-exposure-matrix and the assistance of experts to classify relative exposures, the 9,724 job titles were assigned to the groups: high exposure, moderate exposure, low exposure, and background exposure.

All employees who had worked for a period of 6 months or longer at Motorola and had worked at least 1 day during the follow-up period of 1976–1996 were included in the cohort. The rates of the entire cohort and the exposed sub cohort (moderate or high exposure) were compared both internally and with the mortality data of the States of Arizona, Florida, Illinois and Texas. The above mentioned States were used as reference because most of the Motorola plants are located in these States.

For the entire cohort most of the computed SMRs were near to 1 (external comparison). A comparison of the rates of the exposed sub-cohort with the external rates gave similar results. The internal comparisons showed no increased risk for the exposed group, with the exception of Hodgkins disease (SMR 2.25; 95% CI: 0.4–10.4) during the period 1975–1985.

No individual measurements were performed, instead a qualitative job-exposure-matrix was used. The estimation of possible exposure was made more difficult by the fact that current and past exposures were different in many jobs.

Score values for the RF exposure groups were derived from an exposure validation study. However, very little information is given on this validation study.

Cellular phone use of employees was not addressed as an additional source of exposure. It is however possible that domestic as well as vocational cellular phone use began earlier and is higher in employees of a company manufacturing wireless communication technologies than in the general population. This aspect should be considered in view of the low SMR (0.60) for cancers of the central nervous system (CNS), of which 51 of the 53 CNS malignancies were brain cancers.

Discussion and conclusions

Measurement of exposure

The current measurement of exposure as done in the cited studies was subject to substantial problems. On the one hand, it is almost impossible to determine the individual exposure in more than a few hundred persons. On the other hand, the size of a cohort consisting of e.g. 300 persons is too small to achieve a statistically significant estimate of comparatively rare exposure effects.

A possible solution may be to determine the exposure at least in one single sample in the group of exposed persons or to perform a nested case-control study. The latter is recommended because it may be possible to determine exposure and co-variables in all participants.

A retrospective assessment of exposure seems to be difficult since precise job descriptions and measurements have to be retrospectively available for a substantial period of time.

A second problem is the mixture of exposures in the cohort studies available. Radiofrequencies as well as microwaves were defined as exposure. Some studies dealt with combined exposures or defined groups exposed to extremely low frequency (ELF) fields. It is known that ionizing radiation is produced by certain construction units of radar devices. This fact might, therefore, play a role as soon as repair is being carried out by persons and if the units have been kept connected.

Those studies that are analysing large cohorts pose a third problem, i.e. the difficulty to identify actually exposed persons rather than occupational groups.

In summary, it can be stated that all attempts to improve measurement of exposure have not been successful in the studies described in this paper.

Outcomes

Statistically significant results have been found for some of the outcomes. Two studies reported lower SMR values for respiratory mortality and three studies reported lower SMR values for circulatory mortality in the exposed participants. Lower ratios can be interpreted as indicating a 'healthy worker effect', which may well be the case here.

The results regarding malignant neoplasms are inconsistent. Three studies reported increased SIR and SMR values to be statistically significant, three further studies found significantly lower levels of disease or mortality in comparison with the corresponding reference cohorts. The remaining three studies showed results with ratios near 1, which is insignificant in terms of statistics.

Malignant neoplasms of the large intestine (colon) have been reported more often for the exposed (sub)cohorts. The same is true for prostate cancers, but none of the results turned out to be statistically significant.

Differences/changes in the coding between the various revisions of the international classification of diseases (ICD) need to be taken into account when comparing the studies described here.

A further problem in interpreting the results is the definition of different outcomes (mortality vs. incidence). While six studies used mortality as endpoint (outcome) of interest, four studies used disease incidences as endpoints. The advantage is that incidences are not affected by any type of treatment. On the other hand, it can be assumed that reported mortality may be of more accuracy than incidence finding and that comparison data are readily available.

Most of the studies used the indirect standardization technique to compare the outcomes of the study population (observed cases) with the number of cases in the general population (expected cases) or to compare exposed and unexposed subcohorts.

Lagorio et al. [21] for example stated in their abstract section: "The all-cancer SMR was higher among women employed in the sealing department, where exposure to RF occurred, than in the whole cohort". However, comparison of SMR in the exposed group with the whole cohort has to be interpreted with caution, as SMRs "do not share a common set of weights" [33], in other words: rates were taken from the general population but weights (age distributions) were taken from each cohort. If the weights differ between the cohorts, the SMR are not comparable.

The same problem may occur in the study of Milham (see Table 1 of [22]) and Robinette et al. [29], the latter analysed sub-cohorts using SMR. The difference in portions of high-exposed technicians, born 1926 or earlier, from 8.0% (electronics technicians) to 14.6% (fire control technicians) and 18.8% (aviation electronics technicians) may be a reason for incomparability between the SMR of electronics technicians and the other two technicians groups combined (Table 2 in [29]).

Tynes et al. [32] compared the SMR of different occupational groups. There is no comment on whether or not the age distribution differs from group to group.

Finkelstein [28] did not discuss the results of his sub-cohort analysis and only the external comparison was indicated for his analysis. The results of the sub-cohort analysis are probably incomparable, as it can be assumed that the mean age in the sub cohort "10-60 years from hire" is higher than in the second sub-cohort, hence, the age distributions differ from one another. However, interpretation of the study results is difficult due to the absence of information on individual exposure and the partially small number of cases [19, 21, 32], the unawareness of possible exposures after serving on naval ships [29, 30], the absence of biological models [28] as well as the occupational exposure to high frequency electromagnetic fields within approximately 30% of the amateur radio operators [22].

Outcomes may be affected (or even biased) by other environmental (e.g. chemical) exposures to various extents, which explains some of the inconsistencies.

In our paper, 10 publications were reviewed, most of them using more than 4 or 5 tumor classifications. Up to six subgroups were analysed and in some of the studies a differentiation was made regarding age groups. Therefore, some of the unexpected findings may have been accidental (multiple testing).

In the investigated cohort studies, cancer morbidity and mortality due to exposure to high frequency electromagnetic fields belonged to the outcomes most frequently defined. Also, in 14 cohort studies examining health risks of an exposure to low frequency electrical and magnetic fields, the total numbers of death and cancer were of major interest [34, 35, 36, 37, 38, 39, 40, 41, 42, 43].

So far, questions on particular diseases of the circulatory system [44], on further diseases, such as multiple sclerosis [45], dementia, Parkinson or Alzheimer disease [46], on subjective aspects of individual health (no proof)

or suicide [47] and on the effects of exposure during pregnancy [48] were only rarely taken up.

In principle, the results from cohort studies including persons that were exposed to high frequency fields could be important for assessing health risks of mobile phones. If in high-exposed groups a health effect is observed, it appears to be justified to extrapolate to persons that have been exposed to a lower degree.

However, it is still unclear whether or not there is a health risk for persons that have been exposed over a long period. Only minor conclusions can be drawn from the material for persons using mobile phones.

- In summary, only few cohort studies have up to now been accomplished regarding subsequent health effects of high frequency electromagnetic fields.
- In the available studies, total mortality, partially differentiated by important groups of diseases and/or cancer incidence or cancer mortality, especially brain tumor and leukemia, were defined as outcomes.
- In future cohort studies, some additional outcomes should be specified in order to record further diseases, which may be associated with an exposure to RF radiation. Among others these are the incidences of heart disease and subjective health complaints, which can be regarded as predictors of future diseases.
- When selecting a cohort, attention has to be paid to the size of the study population which should be appropriate with regard to the questions of research.
- Most of the exposure assessments in the cohort studies are not satisfying due to the lack of individual exposure profiles.
- Apart from external comparisons, internal comparisons should also be done to get an estimation of the influence of the healthy worker effect on the study results.
- Altogether, the use of the indirect standardization as the only method is not adequate. Analyses should be completed either by a direct standardization to minimize false conclusions or advanced statistical/epidemiological techniques should be used for analyses. An advantage of the latter methods is the possible adjustment for potential confounders.
- Implementation of a nested case-control study might be helpful, because it is impossible to measure individual exposure and covariates in a study population, such as Motorola. It might be possible to get more precise information about past exposure status.
- If a new cohort study is planned in Europe, the personnel of AM broadcast transmitter stations should be discussed as an interesting one, because exposure assessment is relatively easy to perform.

References

1. Maskarinec G, Cooper J, Swygert L (1994) Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations. *J Environ Pathol Toxicol Oncol* 13:33–37
2. Altpeter ES (1995) Study on health effects of shortwave transmitter station of Schwarzenberg, Berne, Switzerland. BEW Publication Series No 55, University of Berne
3. Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt I, Elliott P (1997) Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Cornfield transmitter. *Am J Epidemiol* 145:1–9
4. Dolk H, Elliott P, Shaddick G, Walls P, Thakrar B (1997) Cancer incidence near radio and television transmitters in Great Britain. II. All high power transmitters. *Am J Epidemiol* 145:10–17
5. Hocking B, Gordon IR, Grain HL, Hatfield GE (1996) Cancer incidence and mortality and proximity to TV towers. *Med J Aust* 165:601–605
6. Brüggemeyer H, Eichhorn K-F, Eggert S, Förster H-J, Heinrich W, Krause N, Kunsch B (2002) Elektromagnetische Felder. Frequenzbereich 0 Hz–300 GHz. www.eev.e-technik.uni-erlangen.de/ak-nir/lf90nir2.pdf
7. ICNIRP—International Commission on Non-Ionizing Radiation Protection (1996) Health issues related to use of hand-held radiotelephones and base transmitters. *Health Phys* 70:587–593
8. ICNIRP—International Commission on Non-Ionizing Radiation Protection (1998) Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). *Health Phys* 74:494–522
9. Blettner M, Berg G (2000) Are mobile phones harmful? *Acta Oncol* 39:927–930
10. Verschaevae L, Maes A (1998) Genetic, carcinogenic and teratogenic effects of radiofrequency fields. *Mutat Res* 410:141–165
11. Stuchly MA (1998) Biomedical concerns in wireless communications. *Crit Rev Biomed Eng* 26:117–151
12. French PW, Penny R, Laurence JA, McKenzie DR (2001) Mobile phones, heat shock proteins and cancer. *Differentiation* 67:93–97
13. Michaelson SM, Elson EC (1996) Interaction of non-modulated radiofrequency fields with living matter: experimental results. In: Polk C, Postow E (eds) *Handbook of biological effects of electromagnetic fields*, 2nd edn. CRC Press, Boca Raton, pp 435–533
14. Neubauer C, Phelan AM, Kues H, Lange DG (1990) Microwave irradiation of rats at 2.45 GHz activates pinocytotic-like uptake of tracer by capillary endothelial cells of cerebral cortex. *Bioelectromagnetics* 11:261–268
15. Akdag MZ, Celik S, Ketani A, Nergiz Y, Deniz M, Dasdag S (1999) Effect of chronic low-intensity microwave radiation on sperm count, sperm morphology, and testicular and epididymal tissues of rats. *Bioelectromagnetics* 18:133–145
16. Lyle DB, Schechter P, Adey WR, Lundak RL (1983) Suppression of T-lymphocyte cytotoxicity following exposure to sinusoidally amplitude-modulated fields. *Bioelectromagnetics* 4:281–292
17. Elwood JM (1999) A critical review of epidemiologic studies of radiofrequency exposure and human cancers. *Environ Health Perspect* 107 [Suppl 1]:155–168
18. Goldsmith JR (1997) Epidemiologic evidence relevant to radar (microwave) effects. *Environ Health Perspect* 105 [Suppl 6]:1579–1587
19. Muhm J (1992) Mortality investigation of workers in an electromagnetic pulse test program. *J Occup Med* 34:287–292
20. Morgan RW, Kelsh MA, Zhao K, Exuzides KA, Heringer S, Negrete W (2000) radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems. *Epidemiology* 11:118–127

21. Lagorio S, Rossi S, Vecchia P et al. (1997) Mortality of plastic-ware workers exposed to radiofrequencies. *Bioelectromagnetics* 18:418–421
22. Milham S (1988) Increased mortality in amateur radio operators due to lymphatic and hemotopoietic malignancies. *Am J Epidemiol* 127:50–54
23. Tynes T, Hannevik M, Andersen A, Vistnes AI, Haldorsen T (1996) Incidence of breast cancer in Norwegian female radio and telegraph operators. *Cancer Causes Control* 7:197–204
24. Amateurradio (1980) A look at ham radio as we enter the eighties. In: Amateurradio Survey. American Radio Relay League, Newington, CT
25. Milham S (1988) Mortality by license class in amateur radio operators. *Am J Epidemiol* 128:1175–1176
26. Davis S, Kaune WT, Mirick DK, Chen C, Stevens RG (2001) Residential magnetic fields, light-at-night, and nocturnal urinary 6-sulfatoxymelatonin concentration in women. *Am J Epidemiol* 154:591–600
27. Levallois P, Dumont M, Touitou Y et al. (2001) Effects of electric and magnetic fields from high-power lines on female urinary excretion of 6-sulfatoxymelatonin concentration in women. *Am J Epidemiol* 154:601–609
28. Finkelstein MM (1998) Cancer incidence among Ontario police officers. *Am J Ind Med* 34:157–162
29. Robinette CD, Silverman C, Jablon S (1980) Effects upon health of occupational exposure to microwave radiation (radar). *Am J Epidemiol* 112:39–53
30. Groves FD, Page WF, Gridley G et al. (2002) Cancer in Korean Navy technicians: mortality survey after 40 years. *Am J Epidemiol* 155:810–818
31. Szmigielski S (1996) Cancer mortality in subjects occupationally exposed to high-frequency (radiofrequency and microwaves) electromagnetic radiation. *Sci Total Environ* 180:9–17
32. Tynes T, Andersen A, Langmark F (1992) Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields. *Am J Epidemiol* 136:81–88
33. Checkoway H, Pearce NE, Crawford-Brown DJ (1989) Research methods in occupational epidemiology. Oxford University Press, New York Oxford
34. Savitz DA, Loomis DP (1995) Magnetic field exposure in relation to leukemia and brain cancer mortality. *Am J Epidemiol* 141:123–134
35. Sorahan T, Nichols L, Tongeren M van, Harrington JM (2001) Occupational exposure to magnetic fields relative to mortality from brain tumors: updated and revised findings from a study of United Kingdom electricity generation and transmission workers, 1973–97. *Occup Environ Med* 58:626–630
36. Harrington JM, Nichols L, Sorahan T, Tongeren M van (2001) Leukemia mortality in relation to magnetic field exposure: findings from a study of United Kingdom electricity generation and transmission workers, 1973–97. *Occup Environ Med* 58:307–314
37. Sahl JD, Kelsh MA, Greenland S (1993) Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers. *Epidemiology* 4:104–114
38. Spinelli JJ, Band PR, Svirchev LM, Gallagher RP (1991) Mortality and cancer incidence in aluminum plant workers. *J Occup Med* 33:1150–1155
39. Rønneberg A, Haldorsen T, Romunstad P, Andersen A (1999) Occupational exposure and cancer incidence among workers from an aluminum smelter in western Norway. *Scand J Work Environ Health* 25:207–214
40. Toernqvist S, Knave B, Ahlhorn A, Persson T (1991) Incidence of leukemia and brain tumors in some “electrical occupations”. *Br J Ind Med* 48:597–603
41. Floderus B, Stenlund C, Persson T (1999) Occupational magnetic field exposure and site-specific cancer incidence: a Swedish cohort study. *Cancer Causes Control* 10:323–332
42. Gruénel P, Raskmark P, Andersen JB, Lynge E (1993) Incidence of cancer in persons with occupational exposure to electromagnetic fields in Denmark. *Br J Ind Med* 50:758–764
43. Milham S (1996) Increased incidence of cancer in a cohort of office workers exposed to strong magnetic fields. *Am J Ind Med* 30:702–704
44. Savitz DA, Liao D, Sastre A, Kleckner RC, Kavet R (1999) Magnetic field exposure and cardiovascular disease among electric utility workers. *Am J Epidemiol* 149: 135–142
45. Johansen C, Koch-Henrikssen N, Rasmussen S, Olsen JH (1999) Multiple sclerosis among utility workers. *Neurology* 52:1279–1282
46. Johansen C (2000) Exposure to electromagnetic fields and risk of central nervous system disease in utility workers. *Epidemiology* 11:539–543
47. Järholm B, Stenberg A (2002) Suicide mortality among electricians in the Swedish construction industry. *Occup Environ Med* 59:199–200
48. Li DK, Odouli R, Wi S, Janevic T, Golditch I, Bracken TD, Senior R, Rankin R, Iriye R (2002) A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of miscarriage. *Epidemiology* 13:9–20