Fig. 1.10 Distribution of 32 837 base stations in the United Kingdom according to average antenna height and total radiated power

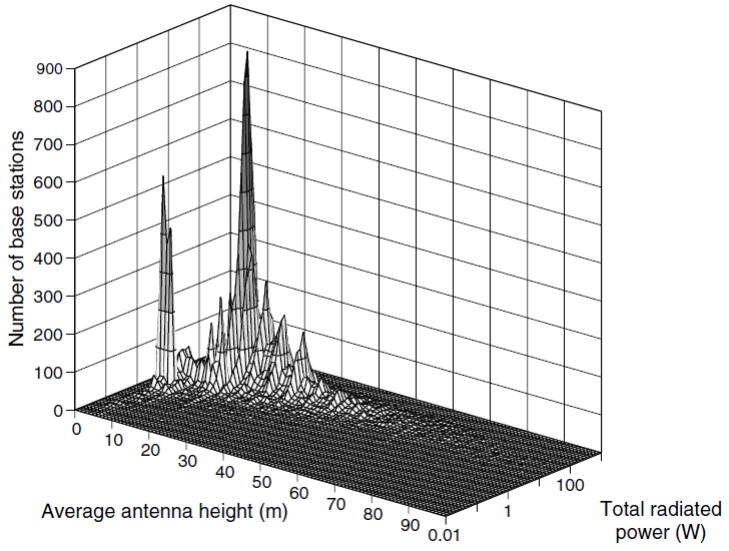


Table 1.9 Estimated minimum, maximum and average exposures in the brain from various sources of radiofrequency radiation

Source	Frequency (MHz)	Exposure			
		Average	Minimum	Maximum	Unit
FM transmitter	100	0.02	0.01	0.07	V/m
TV station	700	0.02	0.001	0.05	V/m
GSM900 base station	950	0.05	0.001	4	V/m
GSM1800 base station	1850	0.05	0.001	6	V/m
DECT base station	1890	0.1	0.03	1	V/m
UMTS 1950 base station	2140	0.05	0.001	6	V/m
WLAN base station	2450	0.03	0.007	1	V/m
WLAN base station	5200/5800	0.01	0.001	1	V/m
GSM900 mobile phone	900	50	0.2	250	mW
GSM1800 mobile phone	1750	40	0.1	125	mW
DECT cordless phone	1890	10	3	20	mW
UMTS mobile phone	1950	1	0.0003	200	mW
WLAN cordless phone	2450	10	3	20	mW

Note: Far-field exposures are estimated in terms of incident-field values and exposures from handsets are calculated from time-averaged output power.
Compiled and calculated by the Working Group from Kühn *et al.* (2010)

Fig. 1.12 Variation in the whole-body specific absorption rate (SAR) produced per unit power density as a function of frequency in the adult male phantom NORMAN, and child phantoms of three different ages, standing on a conductive floor (grounded) and insulated

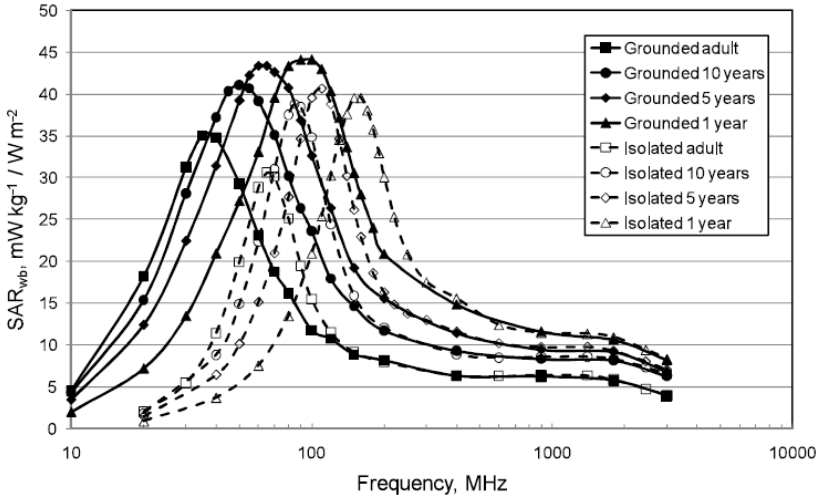


Table 1.8 Depth of penetration of muscle and fat by radiofrequency fields at typical telecommunication frequencies

Frequency (MHz)	Muscle			Fat		
	Relative permittivity	Conductivity (S/m)	Penetration depth (mm)	Relative permittivity	Conductivity (S/m)	Penetration depth[a] (mm)
400	57.13	0.80	52	5.58	0.041	310
900	55.03	0.94	42	5.46	0.051	244
1800	53.55	1.34	29	5.35	0.078	158
2450	52.73	1.74	22	5.28	0.105	116
5200	49.28	4.27	8.8	5.01	0.255	47

[a] Penetration depths have been calculated based on the equation given in the Glossary.
MHz, megahertz; mm, millimetre; S/m, siemens per metre
Compiled by the Working Group from *Tissue Properties Database: Dielectric Properties* by IT'IS Foundation: http://www.itis.ethz.ch/itis-for-health/tissue-properties/database/dielectric-properties/

Fig. 1.17 Mobile-phone subscriptions per 100 people in high-, middle-, and low-income countries, 2000 and 2007

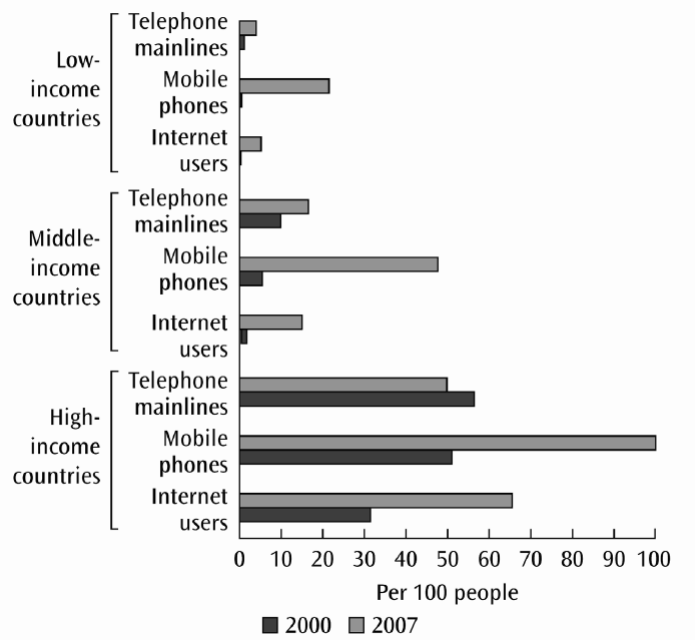
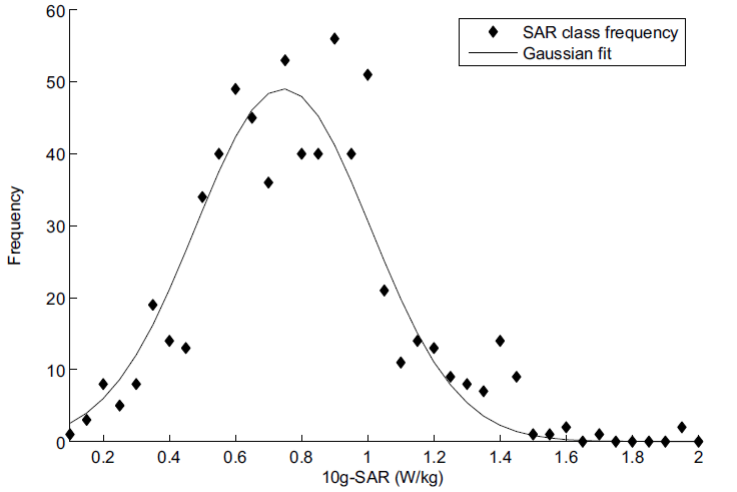


Fig. 1.18 Statistical distribution of maximum psSAR-10 g measured for 668 mobile phones, according to standard EN50361 (CENELEC, 2001)



Data from German Federal Office for Radiation Protection, in Kühn & Kuster (2007)

Table 2.11 (continued)

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Deltour *et al.* (2009)	Denmark, Finland, Norway, Sweden	Unclear	Use increased from zero in the mid-1980s to 'widespread' in the early 1990s to 'sharply increased' in the mid-1990s.	Glioma and meningioma	1974–2003	Incidence rates from Nordic National Cancer Registries	Very slight increases in incidence from 1974 to 1997; no change after 1998	No apparent impact of mobile-phone use on incidence of cancer of the brain. High-quality registration. Up to 10 yr potential latency
Hardell & Carlberg (2009)	Sweden	None	Presumably sharp increases between 1980s and 2000	Brain, age > 19 yr Acoustic neuroma, age > 19 yr	1970–2007	Incidence rates from Swedish Cancer Registry	Changing annual incidence: 1970–79 (+0.15%) 1980–89 (+1.54%) 1990–99 (–0.25%) 2000–07 (+1.26%) 1970–79 (–1.66%) 1980–89 (+4.86%) 1990–99 (+0.66%) 2000–07 (–7.08%)	No evidence of an impact of mobile-phone use on the risk of acoustic neuroma. No or very weak evidence of an effect of phone use on risk of tumours of the brain. Slightly stronger evidence for increased risk of astrocytoma in the most recent period
Inskip *et al.* (2010)	USA (SEER Program); nine state or regional population-based cancer registries	Number of mobile-phone subscribers in USA by year	From very few in 1990 to 25 million in 1995; 100 million in 2000 and 200 million in 2005	All brain, excluding meningioma and lymphoma	1977–2006	Incidence rates from SEER	Gradual increase in risks from 1977 to 1985; since 1986 the pattern is flat or slightly decreasing. Some age/sex subgroups show increasing trends in some subtypes	No apparent impact of mobile-phone use on incidence of cancer of the brain. Very large numbers of cases. Up to 10 yr of potential latency

Caso- controle celulares

Table 2.13 Case–control studies of glioma and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell *et al.* (1999) Sweden, 1994–96	136	Two controls per case	Population	Self-administered standardized questionnaire	48 glioblastoma, 46 astrocytoma, 19 oliodendro-glioma, 3 ependymoma, 16 mixed glioma, and 4 other malignant tumours	Never use of mobile phone Ever use	 53	1.0 1.0 (0.6–1.5)	Age, sex, SEI, and year of diagnosis	
Muscat *et al.* (2000) USA, 1994–98	469	422	In-patients from five USA academic medical centres. Controls from the same hospitals as cases, from daily admission rosters	In-person interviews, history of mobile-phone use	Brain cancer (191.0–191.9)	Ever use Cumulative use (h): 0 > 0 to ≤ 8.7 > 8.7 to ≤ 60 > 60 to ≤ 480 > 480	NR 17 12 19 14	0.7 (0.5–1.1) 1.0 1.0 (0.5–2.0) 0.6 (0.3–1.3) 0.9 (0.5–1.8) 0.7 (0.3–1.4)	Age, education, sex, race, study centre, proxy, year of interview	Analyses showed no associations by year of use. Few subjects with long-term heavy exposure. Response rates were 82% for cases and 90% for controls.
	108	422			Temporal lobe	Ever use	108	0.9 (0.5–1.7)		
	60	422			Parietal lobe	Ever use	60	0.8 (0.3–2.0)		
	354	422			Astrocytic	Ever use	41	0.8 (0.5–1.2)		

Caso- controle celulares

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell *et al.* (2011a) Sweden, 1997–2003	1148	2438	Population	Self-administered standardized questionnaire	Glioma	Never use of mobile/ cordless phone		1.0	Sex, age, SEI, and year of diagnosis	Pooled analysis of case–control data for living cases ascertained from 1997–2000, and 2000–03, as well as case–control data for deceased cases 1997–2003.
						Ever use (mobile phone)	529	1.3 (1.1–1.6)		
						Time since start of use (yr)				
						> 1–5	250	1.1 (0.9–1.4)		
						> 5–10	156	1.3 (1.0–1.6)		
						> 10	123	2.5 (1.8–3.3)		
						Cumulative call time, mobile phone (h)				
						1–1000	427	1.2 (1.03–1.5)		
						1001–2000	44	1.8 (1.2–2.8)		
						> 2000	58	3.2 (2.0–5.1)		

Caso- controle celulares

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell *et al.* (2006a,c) Sweden, 2000–03	317	1990	Population	Self-administered standardized questionnaire	248 astrocytomas, and 69 other malignant tumours of the brain	Never use of mobile/ cordless phone	63	1.0	Age, sex, SEI, and year of diagnosis	
						Ever use, analogue	68	2.6 (1.5–4.3)		Analogue phone: Ipsilateral use: 3.1 (95% CI, 1.6–6.2); contralateral use: 2.6 (95% CI, 1.3–5.4)
						Ever use, digital	198	1.9 (1.3–2.7)		Digital phone: Ipsilateral use: 2.6 (95% CI, 1.6–4.1); contralateral use: 1.3 (95% CI, 0.8–2.2)
						Time since start of use, analogue (yr)				
						> 1–5	0	–		
						> 5–10	20	1.8 (0.9–3.5)		
						> 10	48	3.5 (2.0–6.4)		
						Time since start of use, digital (yr)				
						> 1–5	100	1.6 (1.1–2.4)		
						> 5–10	79	2.2 (1.4–3.4)		
						> 10	19	3.6 (1.7–7.5)		

Caso- controle celulares

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
INTERPHONE Study Group (2010) Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, United Kingdom, 2000–04	2708	2972	Population (except United Kingdom: GP patients)	Interviewer-administered standardized questionnaire	Glioma (D33.0, D43.0–43.9, C71.0–71.9)	Never regular use of mobile phone	1042	1.0 (ref.)	Sex, age, study centre, ethnicity (in Israel) and education	OR highest in short-term users (start of mobile phone use, 1–4 yr before reference date) (OR, 3.77; 95% CI, 1.25–11.4, based on eight cases)
						Regular use	1666	0.81 (0.70–0.94)		
						Time since start of use (yr)				
						1.5	156	0.62 (0.46–0.81)		
						2–4	644	0.84 (0.70–1.00)		
						5–9	614	0.81 (0.60–0.97)		
						≥ 10	252	0.98 (0.76–1.26)		
						Cumulative call time with no hands-free devices (h)				
						< 5	141	0.70 (0.52–0.94)		
						5–12.9	145	0.71 (0.53–0.94)		
						13–30.9	189	1.05 (0.79–1.38)		
						31–60.9	144	0.74 (0.55–0.98)		
						61–114.9	171	0.81 (0.61–1.08)		
						115–199.9	160	0.73 (0.54–0.98)		
						200–359.9	158	0.76 (0.57–1.01)		
						360–734.9	189	0.82 (0.62–1.08)		
						735–1639.9	159	0.71 (0.53–0.96)		
						≥ 1 640	210	1.40 (1.03–1.89)		

IARC – celulares e RF

6. EVALUATION

6.1 Cancer in Humans

There is *limited evidence* in humans for the carcinogenicity of radiofrequency radiation. Positive associations have been observed between exposure to radiofrequency radiation from wireless phones and glioma, and acoustic neuroma.

6.2 Cancer in Experimental Animals

There is *limited evidence* in experimental animals for the carcinogenicity of radiofrequency radiation.

6.3 Overall Evaluation

Radiofrequency electromagnetic fields are *possibly carcinogenic to humans (Group 2B).*

6.4 Rationale of the evaluation of the epidemiological evidence

The human epidemiological evidence was mixed. Several small early case–control studies were considered to be largely uninformative. A large cohort study showed no increase in risk of relevant tumours, but it lacked information on level of mobile-phone use and there were several potential sources of misclassification of exposure. The bulk of evidence came from reports of the INTERPHONE study, a very large international, multicentre case–control study and a separate large case–control study from Sweden on gliomas and meningiomas of the brain and acoustic neuromas. While affected by selection bias and information bias to varying degrees, these studies showed an association between glioma and acoustic neuroma and mobile-phone use; specifically in people with highest cumulative use of mobile phones, in people who had used mobile phones on the same side of the head as that on which their tumour developed, and in people whose tumour was in the temporal lobe of the brain (the area of the brain that is most exposed to RF radiation when a wireless phone is used at the ear). The Swedish study found similar results for cordless phones. The comparative weakness of the associations in the INTERPHONE study and inconsistencies between its results and those of the Swedish study led to the evaluation of *limited evidence* for glioma and acoustic neuroma, as decided by the majority of the members of the Working Group. A small, recently published Japanese case–control study, which also observed an association of acoustic neuroma with mobile-phone use, contributed to the evaluation of *limited evidence* for acoustic neuroma.

There was, however, a minority opinion that current evidence in humans was *inadequate*, therefore permitting no conclusion about a causal association. This minority saw inconsistency between the two case–control studies and a lack of exposure–response relationship in the INTERPHONE study. The minority also pointed to the fact that no increase in rates of glioma or acoustic neuroma was seen in a nationwide Danish cohort study, and that up to now, reported time trends in incidence rates of glioma have not shown a trend parallel to time trends in mobile-phone use.

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NON-IONIZING RADIATION, PART 1: STATIC AND EXTREMELY LOW-FREQUENCY (ELF) ELECTRIC AND MAGNETIC FIELDS

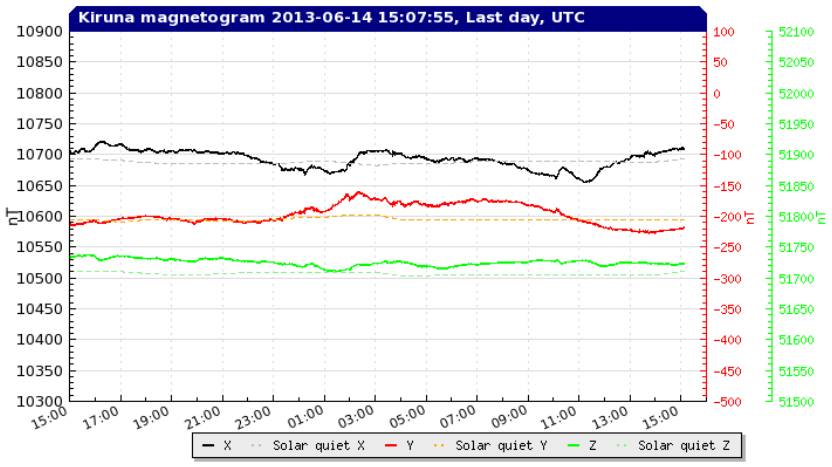
IARC – estáticos e ELF

Table 2. Conductivities of various tissues assumed for power-frequency electric and magnetic fields

Tissue	σ (S/m)	Tissue	σ (S/m)
Bladder	0.2	Heart	0.5
Blood	0.7	Kidney	0.09
Bone (cancellous)	0.08	Liver	0.04
Bone (compact)	0.02	Lungs	0.07
Brain (white)	0.06	Muscle	0.24
Cerebrospinal fluid	2.0	Skin	0.04
Eye sclera	0.5	Spinal cord	0.07
Fat	0.02	Testes	0.42

From Gandhi *et al.* (2001)

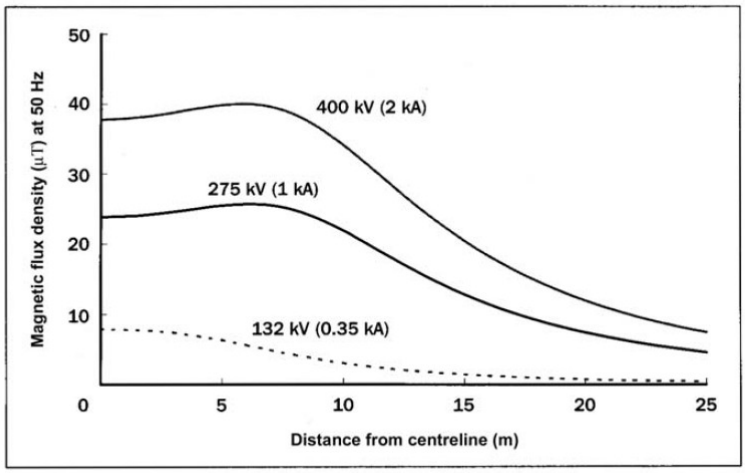
Campo geomagnético



Mais informações: http://www.irf.se//Observatory/?link[Magnetometers]=Description

IARC – estáticos e ELF

Figure 3. Magnetic fields from high-voltage overhead power lines



From National Radiological Protection Board (2001)

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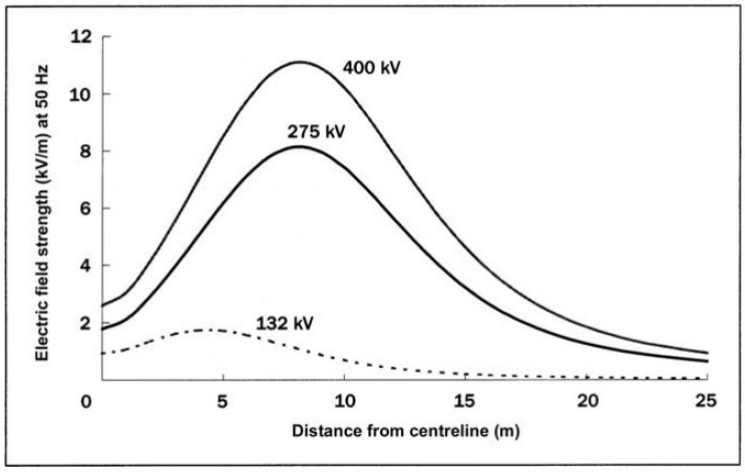
Figure 2. Electric fields from high-voltage overhead power lines



From National Radiological Protection Board (2001)

IARC – estáticos e ELF

Table 10. Calculated electric fields (mV/m) in a vertical uniform electric field (60 Hz, 1 kV/m) induced in a model of a grounded adult human body[a]

Tissue/organ	E_{avg}	$E_{99\ percentile}$	E_{max}
Blood	1.4	8.9	24
Bone marrow	3.6	34	41
Brain	0.86	2.0	3.7
Cerebrospinal fluid	0.35	1.0	1.6
Heart	1.4	2.8	3.6
Kidneys	1.4	3.1	4.5
Lungs	1.4	2.4	3.6
Muscle	1.6	10	32
Prostate	1.7	2.8	3.1
Spleen	1.8	2.6	3.2
Testes	0.48	1.2	1.6

Modified from Kavet *et al.* (2001)
[a] Corresponding current densities can be computed from tissue conductivity values (see Table 2, General Introduction)

IARC – estáticos e ELF

Table 12. Calculated electric fields (μV/m) in a uniform magnetic field (60 Hz, 1 μT) oriented front-to-back induced in a model of an adult human

Tissue/organ	E_{avg}	$E_{99\ percentile}$	E_{max}
Blood	6.9	23	83
Bone marrow	16	93	154
Brain	11	31	74
Cerebrospinal fluid	5.2	17	25
Heart	14	38	49
Kidneys	25	53	71
Lungs	21	49	86
Muscle	15	51	147
Prostate	17	36	52
Spleen	41	72	92
Testes	15	41	73

Modified from Kavet *et al*. (2001)

Table 18. Cohort study of childhood cancer and exposure to ELF magnetic fields

Study size, number of cases	Exposure	SIR (95% CI) by cancer site									
		Leukaemia	No. of cases	CNS	No. of cases	Lymphoma	No. of cases	Other sites	No. of cases	All cancers	No. of cases
68 300 boys, 66 500 girls, aged 0–19 years; 140 incident cancer cases diagnosed 1970–89	**Calculated historical magnetic fields**										
	< 0.01 μT (baseline)	1.0		1.0		1.0		1.0		1.0	
	0.01–0.19 μT	0.89 (0.61–1.3)	32	0.85 (0.59–1.2)	34	0.91 (0.51–1.5)	15	1.1 (0.79–1.4)	48	0.94 (0.79–1.1)	129
	≥ 0.2 μT	1.6 (0.32–4.5)	3	2.3 (0.75–5.4)	5	0 (0.0–4.2)	0	1.2 (0.26–3.6)	3	1.5 (0.74–2.7)	11
	Calculated cumulative magnetic fields (μT–years)										
	< 0.01 (baseline)	1.0		1.0		1.0		1.0		1.0	
	0.01–0.39	0.90 (0.62–1.3)	32	0.82 (0.56–1.2)	32	0.88 (0.48–1.5)	14	1.1 (0.80–1.4)	47	0.93 (0.78–1.1)	125
	≥ 0.4	1.2 (0.26–3.6)	3	2.3 (0.94–4.8)	7	0.64 (0.02–3.6)	1	1.0 (0.27–2.6)	4	1.4 (0.77–2.3)	15

From Verkasalo *et al.* (1993), Finland
SIR, standardized incidence ratio; CI, confidence interval; CNS, central nervous system
Expected numbers calculated in sex-specific five-year age groups; no further adjustments. SIRs for highest exposure categories for CNS tumours are questionable, since one boy with three primary tumours was counted three times.

Table 19 (contd)

Reference, area	Study size (for analyses)	Exposure	No. of cases	Risk estimates: odds ratio (95% CI)	No. of cases	Risk estimates: odds ratio (95% CI)	Comments
Linet *et al.* (1997), nine mid-western and mid-Atlantic states, USA	*Wire code*: 408 cases, 408 controls, aged 0–14 years; *24-h measurements*: 638 cases, 620 controls	**Time-weighted average (24-h bedroom measurement plus spot measurements in two rooms)**		Unmatched		Matched	Unmatched analysis additionally adjusted for age, sex, mother's education and family income; information on a variety of potential confounding factors was available; wire coding of subjects who refused to participate; relatively low response rates for the measurements in controls; only acute lymphoblastic leukaemia; children with Down syndrome excluded from this study (Schüz *et al.*, 2001a)
		< 0.065 µT (baseline)	267	1.0	206	1.0	
		0.065–0.099 µT	123	1.1 (0.81–1.5)	92	0.96 (0.65–1.4)	
		0.100–0.199 µT	151	1.1 (0.83–1.5)	107	1.2 (0.79–1.7)	
		≥ 0.200 µT	83	1.2 (0.86–1.8)	58	1.5 (0.91–2.6)	
		Wire code					
		UG/VLCC (baseline)			175	1.0	
		OLCC			116	1.1 (0.74–1.5)	
		OHCC			87	0.99 (0.67–1.5)	
		VHCC			24	0.88 (0.48–1.6)	

UG, underground wires; VLCC, very low current configuration; OLCC, ordinary low current configuration; OHCC, ordinary high current configuration; VHCC, very high current configuration; LCC, low current configuration; HCC, high current configuration; UKCCSI, UK Childhood Cancer Study Investigators

[a] In these tables, only studies that contributed substantially to the overall summary were considered; only results that were part of the analysis strategy defined above are presented; exposure metrics and cut-points vary across studies, for a better comparison, please refer to Table 23.

IARC – estáticos e ELF

5.5 Evaluation

There is *limited evidence* in humans for the carcinogenicity of extremely low-frequency magnetic fields in relation to childhood leukaemia.

There is *inadequate evidence* in humans for the carcinogenicity of extremely low-frequency magnetic fields in relation to all other cancers.

There is *inadequate evidence* in humans for the carcinogenicity of static electric or magnetic fields and extremely low-frequency electric fields.

There is *inadequate evidence* in experimental animals for the carcinogenicity of extremely low-frequency magnetic fields.

No data relevant to the carcinogenicity of static electric or magnetic fields and extremely low-frequency electric fields in experimental animals were available.

Overall evaluation

Extremely low-frequency magnetic fields are *possibly carcinogenic to humans (Group 2B)*.

Static electric and magnetic fields and extremely low-frequency electric fields are *not classifiable as to their carcinogenicity to humans (Group 3)*.

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