ELSEVIER

Pathophysiology 16 (2009) 113–122

ISP PATHOPHYSIOLOGY

www.elsevier.com/locate/pathophys

Epidemiological evidence for an association between use of wireless phones and tumor diseases

Lennart Hardell [a,*], Michael Carlberg [a], Kjell Hansson Mild [b]

[a] *Department of Oncology, University Hospital, SE-701 85 Örebro, Sweden*
[b] *Department of Radiation Physics, Umeå University, SE-901 87 Umeå, Sweden*

Received 7 November 2008; accepted 30 January 2009

Abstract

During recent years there has been increasing public concern on potential cancer risks from microwave emissions from wireless phones. We evaluated the scientific evidence for long-term mobile phone use and the association with certain tumors in case–control studies, mostly from the Hardell group in Sweden and the Interphone study group. Regarding brain tumors the meta-analysis yielded for glioma odds ratio (OR) = 1.0, 95% confidence interval (CI) = 0.9–1.1. OR increased to 1.3, 95% CI = 1.1–1.6 with 10 year latency period, with highest risk for ipsilateral exposure (same side as the tumor localisation), OR = 1.9, 95% CI = 1.4–2.4, lower for contralateral exposure (opposite side) OR = 1.2, 95% CI = 0.9–1.7. Regarding acoustic neuroma OR = 1.0, 95% CI = 0.8–1.1 was calculated increasing to OR = 1.3, 95% CI = 0.97–1.9 with 10 year latency period. For ipsilateral exposure OR = 1.6, 95% CI = 1.1–2.4, and for contralateral exposure OR = 1.2, 95% CI = 0.8–1.9 were found. Regarding meningioma no consistent pattern of an increased risk was found. Concerning age, highest risk was found in the age group <20 years at time of first use of wireless phones in the studies from the Hardell group. For salivary gland tumors, non-Hodgkin lymphoma and testicular cancer no consistent pattern of an association with use of wireless phones was found. One study on uveal melanoma yielded for probable/certain mobile phone use OR = 4.2, 95% CI = 1.2–14.5. One study on intratemporal facial nerve tumor was not possible to evaluate due to methodological shortcomings. In summary our review yielded a consistent pattern of an increased risk for glioma and acoustic neuroma after >10 year mobile phone use. We conclude that current standard for exposure to microwaves during mobile phone use is not safe for long-term exposure and needs to be revised.
© 2009 Elsevier Ireland Ltd. All rights reserved.

Keywords: Brain tumors; Glioma; Acoustic neuroma; Meningioma; Cellular phones; Cordless phones

1. Introduction

During the last decade there has been a rapid development of wireless technology and along with that an increased use of wireless telephone communication in the world. Most persons use mobile phones and cordless phones. Additionally most populations are exposed to radiofrequency/microwave (RF) radiation emissions from wireless devices such as cellular antennas and towers, broadcast transmission towers, voice and data transmission for cell phones, pagers and personal digital assistants and other sources of RF radiation.

Concerns of health risks have been raised, primarily an increased risk for brain tumors, since the brain is the near field target organ for microwave exposure during mobile phone calls. Especially the ipsilateral brain (same side as the mobile phone has been used) is exposed, whereas the contralateral side (opposite side to the mobile phone) is much less exposed [1]. Thus, for risk analysis it is of vital importance to have information on the localisation of the tumor in the brain and which side of the head that has been predominantly used during phone calls.

Since Sweden was one of the first countries in the world to adopt this wireless technology a brief history is given in the following. First, analogue phones (NMT; Nordic Mobile Telephone System) were introduced on the market in the early 1980s using both 450 and 900 Megahertz (MHz) carrier waves. NMT 450 was used in Sweden since 1981 but closed down in December 31, 2007, whereas NMT 900 operated during 1986–2000.

* Corresponding author.
E-mail address: lennart.hardell@orebroll.se (L. Hardell).

0928-4680/$ – see front matter © 2009 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.pathophys.2009.01.003

Table 1
Odds ratios (ORs) and 95% confidence intervals (CIs) from 11 case–control studies on glioma including meta-analysis of the studies. Numbers of exposed cases and controls are given.

Author, year of publication, country, reference number	No. of cases	No. of controls	OR	95% CI
Inskip et al., 2001, USA [23]	201	358	1.0	0.7–1.4
Auvinen et al., 2002, Finland [24]	Not given	Not given	1.5	1.0–2.4
Lönn et al., 2005, Sweden [25][a]	214	399	0.8	0.6–1.0
Christensen et al., 2005, low-grade glioma, Denmark [26][a]	47	90	1.1	0.6–2.0
Christensen et al., 2005, high-grade glioma, Denmark [26][a]	59	155	0.6	0.4–0.9
Hepworth et al., 2006, UK [27][a]	508	898	0.9	0.8–1.1
Schüz et al., 2006, Germany [28]	138	283	1.0	0.7–1.3
Hardell et al., 2006, Sweden [12], all glioma	346	900	1.4	1.1–1.7
Low-grade glioma	65	900	1.4	0.9–2.3
High-grade glioma	281	900	1.4	1.1–1.8
Lahkola et al., 2006, Denmark, Norway, Finland, Sweden, UK [29]	867	1 853	0.8	0.7–0.9
Hours et al., 2007, France [30]	59	54	1.2	0.7–2.1
Klaeboe et al., 2007, Norway [31][a]	161	227	0.6	0.4–0.9
Takebayashi et al., 2008, Japan [17]	56	106	1.2	0.6–2.4
Meta-analysis	>1667[b]	>3554[b]	1.0	0.9–1.1

[a] Not included in meta-analysis because already part of pooled data in Lahkola et al., 2006 [29].
[b] Total number could not be calculated since numbers were not presented in one publication [24].

The digital system (GSM; Global System for Mobile Communication) using dual band, 900 and 1800 MHz, started to operate in 1991 and now dominates the market. The third generation of mobile phones, 3G or UMTS (Universal Mobile Telecommunication System), using 1900 MHz RF broad band transmission has been introduced worldwide since a few years, in Sweden since 2003.

Desktop cordless phones have been used in Sweden since 1988, first analogue 800–900 MHz RF fields, but since early 1990s the digital 1900 MHz DECT (Digital Enhanced Cordless Telecommunications) system is used. In our studies on tumor risk associated with use of wireless phones, we have also assessed use of cordless phones. However, most other research groups have not published such data at all, or only in a scanty way, so exposure to RF from DECT is not further discussed here. Instead the reader is referred to our previous publications on this issue [2–13].

The initial studies on brain tumor risk had too short latency periods to give a meaningful interpretation. However, during recent years studies have been published that enable evaluation of ≥10-years latency period risk, although still mostly based on low numbers [14,15]. A ≥10-years latency period seems to be a reasonable minimum period to indicate long-term carcinogenic risks from exposure to RF fields during use of mobile or cordless phones.

Table 2
Odds ratios (ORs) and 95% confidence intervals (CIs) from six case–control studies on glioma including meta-analysis of the studies using ≥10 year latency period. Numbers of exposed cases and controls are given.

Study	Total			Ipsilateral			Contralateral		
Author, year of publication, country, latency, reference number	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI
Lönn et al., 2005, Sweden, ≥10 years [25][a]	25/38	0.9	0.5–1.5	15/18	1.6	0.8–3.4	11/25	0.7	0.3–1.5
Christensen et al., 2005, Denmark, low-grade glioma, ≥10 years [26][a]	6/9	1.6	0.4–6.1	–	–	–	–	–	–
Christensen et al., 2005, Denmark, high-grade glioma, ≥10 years [26][a]	8/22	0.5	0.2–1.3	–	–	–	–	–	–
Hepworth et al., 2006, UK, ≥10 years [27][a]	66/112	0.9	0.6–1.3	Not given	1.6	0.9–2.8	Not given	0.8	0.4–1.4
Schüz et al., 2006, Germany, ≥10 years [28]	12/11	2.2	0.9–5.1	–	–	–	–	–	–
Hardell et al., 2006, Sweden, >10 years [12], all glioma	78/99	2.7	1.8–3.9	41/28	4.4	2.5–7.6	26/29	2.8	1.5–5.1
Low-grade glioma	7/99	1.5	0.6–3.8	2/28	1.2	0.3–5.8	4/29	2.1	0.6–7.6
High-grade glioma	71/99	3.1	2.0–4.6	39/28	5.4	3.0–9.6	22/29	3.1	1.6–5.9
Lahkola et al., 2006, Denmark, Norway, Finland, Sweden, UK, ≥10 years [29]	143/220	0.95	0.7–1.2	77/117	1.4	1.01–1.9	67/121	1.0	0.7–1.4
Meta-analysis	233/330	1.3	1.1–1.6	118/145	1.9	1.4–2.4	93/150	1.2	0.9–1.7

[a] Not included in meta-analysis because already part of pooled data in Lahkola et al., 2006 [29].

Table 3
Odds ratios (ORs) and 95% confidence intervals (CIs) from nine case–control studies on acoustic neuroma including meta-analysis of the studies. Numbers of exposed cases and controls are given.

Author, year of publication, country, reference number	No. of cases	No. of controls	OR	95% CI
Inskip et al., 2001, USA [23]	40	358	0.8	0.5–1.4
Lönn et al., 2004, Sweden [32][a]	89	356	1.0	0.6–1.5
Christensen et al., 2004, Denmark [33][a]	45	97	0.9	0.5–1.6
Schoemaker et al., 2005, Denmark, Finland, Sweden, Norway, Scotland, England [34]	360	1934	0.9	0.7–1.1
Hardell et al., 2006, Sweden [11]	130	900	1.7	1.2–2.3
Takebayashi et al., 2006, Japan [35]	51	192	0.7	0.4–1.2
Klaeboe et al., 2007, Norway [31][a]	22	227	0.5	0.2–1.0
Schlehofer et al., 2007, Germany [36]	29	74	0.7	0.4–1.2
Hours et al., 2007, France [30]	58	123	0.9	0.5–1.6
Meta-analysis	668	3581	1.0	0.8–1.1

[a] Not included in meta-analysis because already part of pooled data in Schoemaker et al., 2005 [34].

Long-term exposure to RF fields from mobile phones and brain tumor risk is of importance to evaluate, not the least since the use of cellular phones is globally widespread with high prevalence among almost all age groups in the population. In the following we discuss mobile phone use and the association with brain tumors, but also other tumor types that have been studied. Recently, we published a detailed review of studies on brain tumors [14] followed by meta-analyses of published studies regarding glioma, acoustic neuroma and meningioma [15]. We have now recalculated these results with the addition of two new recently published articles from the Interphone study group [16,17]. Studies from individual countries were only included in the meta-analyses if they were not also included in the joint publications for several countries. For odds ratio (OR) and 95% confidence interval (CI) we used fixed effects model as in the recent publication by Kundi [18]. The analyses were done using Stata/SE 10 (Stata/SE 10 for Windows; StataCorp., College Station, TX).

One case–control study was excluded since no separate data were presented for glioma, acoustic neuroma or meningioma [19], and another since no overall data on acoustic neuroma were published, only for some time periods without results for ≥10 year latency period [20].

Due to several methodological limitations a Danish cohort study on "mobile phone subscribers" [21] is not possible to include in the meta-analysis, and the same methodological shortcomings prevail in the published updated cohort [22]. In the following only a short overview of the results for brain tumors is given, since we have discussed these issues in more detail elsewhere [14,15]. The other tumor types that have been studied are salivary gland tumors, non-Hodgkin lymphoma (NHL), testicular cancer, eye melanoma and facial nerve tumor.

2. Glioma

Glioma is a malignant type of brain tumor and comprises about 60% of all central nervous system tumors. The highly malignant glioblastoma multiform, with poor survival, is included in this group.

Eleven case–control studies present results for glioma [12,17,23–31]. Of these eight [17,25–31] were part of the Interphone study and four of these [25–27,31] were included in a pooled-analysis with additional data for Finland [29]. The results are presented in Table 1. Overall no decreased

Table 4
Odds ratios (ORs) and 95% confidence intervals (CIs) from four case–control studies on acoustic neuroma including meta-analysis of the studies using ≥10 year latency period. Numbers of exposed cases and controls are given.

Study	Total			Ipsilateral			Contralateral		
Author, year of publication, country, latency, reference number	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI
Lönn et al., 2004, Sweden, ≥10 years [32][a]	14/29	1.8	0.8–4.3	12/15	3.9	1.6–9.5	4/17	0.8	0.2–2.9
Christensen et al., 2004, Denmark, ≥10 years [33][a]	2/15	0.2	0.04–1.1	–	–	–	–	–	–
Schoemaker et al., 2005, Denmark, Finland, Sweden, Norway, Scotland, England, ≥10 years [34]	47/212	1.0	0.7–1.5	31/124	1.3	0.8–2.0	20/105	1.0	0.6–1.7
Hardell et al., 2006, Sweden, >10 years [11]	20/99	2.9	1.6–5.5	10/28	3.5	1.5–7.8	6/29	2.4	0.9–6.3
Meta-analysis	67/311	1.3	0.97–1.9	41/152	1.6	1.1–2.4	26/134	1.2	0.8–1.9

[a] Not included in meta-analysis because already part of pooled data in Schoemaker et al., 2005 [34].

or increased risk was found for glioma in the meta-analysis; OR = 1.0, 95% CI = 0.9–1.1.

Results for 10 year latency period are presented in Table 2. Six studies [12,25–29] gave such information and three [25–27] of these were also part of the publication by Lahkola et al. [29]. The meta-analysis yielded significantly increased risk for glioma with OR = 1.3, 95% CI = 1.1–1.6 increasing to OR = 1.9, 95% CI = 1.4–2.4 for ipsilateral exposure. The latter results were based on 118 exposed cases and 145 exposed controls. Regarding contralateral exposure to microwaves from mobile phones a lower risk was calculated, OR = 1.2, 95% CI = 0.9–1.7 (n = 93 cases, 150 controls). It should be noted that in the study by Takebayashi et al. [17] analyses of maximum microwave energy absorbed at the location of the tumor gave OR = 1.6, 95% CI = 0.6–4.2 related to the highest quartile of cumulative phone time weighted by maxSAR and OR = 5.8, 95% CI = 0.96–36 for subjects with cumulative maxSAR-hour of ≥10 W/kg-h.

3. Acoustic neuroma

These tumors are benign and do not undergo malignant transformation. They tend to be encapsulated and grow in relation to the auditory and vestibular portions of nerve VIII. They are slow growing tumors initially in the auditory canal, but gradually grow out into the cerebellopontine angle, where they come into contact with vital brain stem centers.

Nine case–control studies have been published [11,23,30–36], see Table 3. Seven [30–36] were part of the Interphone study and three [31–33] were included in the publication by Schoemaker et al. [34]. Analysis of the total material yielded OR = 1.0, 95% CI = 0.8–1.1 increasing to 1.3, 95% CI = 0.97–1.9 using 10 year latency period, Table 4. For ipsilateral exposure OR increased further to 1.6, 95% CI = 1.1–2.4, whereas contralateral exposure gave a non-significantly increased risk, OR = 1.2, 95% CI = 0.8–1.9.

4. Meningioma

Meningioma arises from the pia or archnoid, which are the covering layers of the central nervous system. The majority are benign tumors that are encapsulated and well-demarched from surrounding tissue.

Regarding meningioma results have been published from nine case–control studies, Table 5 [11,16,17,23,25,26,28,30,31]. Of these, seven [16,17,25,26,28,30,31] were part of the Interphone studies. The Lahkola et al. study [16] included three separately published Interphone studies [25,26,31]. The meta-analysis in Table 5 gave a significantly reduced OR = 0.9, 95% CI = 0.8–0.9. These results were mainly caused by the findings in the Interphone study [16] with the largest numbers of cases and controls yielding OR = 0.8, 95% CI = 0.7–0.9 in that study.

Using 10 year latency period OR was close to unity and somewhat increased for ipsilateral exposure, OR = 1.3, 95% CI = 0.9–1.8, Table 6. Regarding contralateral exposure OR was non-significantly decreased to 0.8, 95% CI = 0.5–1.3. The results for laterality were based on only two studies [11,16].

5. Brain tumor risk in different age groups

We grouped cases and controls according to age when they started to use a mobile or a cordless phone [11,12]. Consistently we found the highest risk for those with first use <20 years age. Thus, for malignant brain tumors OR = 2.7, 95% CI = 1.3–6.0 was calculated for mobile phones and OR = 2.1, 95% CI = 0.97–4.6 for cordless phones. The corresponding results for benign brain tumors were OR = 2.5, 95% CI = 1.1–5.9 and OR = 0.6, 95% CI = 0.2–1.9, respectively. Previously, we published results for diagnosis of brain tumor in different age groups [37] and found highest OR = 5.9, 95% CI = 0.6–55 for ipsilateral use of analogue phones in the youngest age group 20–29 years at the time of diagnosis. Using a >5 years latency period increased the risk further.

6. Brain tumor risk for use of mobile phone in urban and rural areas

There is a difference in output power of digital mobile phones between urban and rural areas. Adaptive power control (APC) regulates power depending on the quality of the transmission. In rural areas with on average longer distance to the base station the output power level is higher than in urban areas with dense population and shorter distance to the base stations. We studied the risk for brain tumors in urban versus rural living from the data in our study with cases diagnosed January 1, 1997 to June 30, 2000 [38]. Regarding digital phones OR = 1.4, 95% CI = 0.98–2.0 was obtained for living in rural areas increasing to OR = 3.2, 95% CI = 1.2–8.4 with >5 years latency period. The corresponding results for living in urban areas were OR = 0.9, 95% CI = 0.8–1.2 and OR = 0.9, 95% CI = 0.6–1.4, respectively.

7. Salivary gland tumors

The salivary glands, especially the parotid gland, are targets for near-field microwave exposure during calls with wireless phones. A Finnish study reported OR = 1.3, 95% CI = 0.4–4.7 for those who had ever had a mobile phone subscription [24].

Results from three case–control studies have been published, one from Sweden, one from the Nordic countries and one from Israel. During the same period as our studies on brain tumors we performed a study on salivary gland tumors [39]. Our study included the whole Swedish pop-

Table 5
Odds ratios (ORs) and 95% confidence intervals (CIs) from nine case–control studies on meningioma including meta-analysis of the studies. Numbers of exposed cases and controls are given.

Author, year of publication, country, reference number	No. of cases	No. of controls	OR	95% CI
Inskip et al., 2001 (USA) [23]	67	358	0.8	0.5–1.2
Lönn et al., 2005 (Sweden) [25][a]	118	399	0.7	0.5–0.9
Christensen et al., 2005 (Denmark) [26][a]	67	133	0.8	0.5–1.3
Schüz et al., 2006 (Germany) [28]	104	234	0.8	0.6–1.1
Hardell et al., 2006 (Sweden) [11]	347	900	1.1	0.9–1.3
Klaeboe et al., 2007 (Norway) [31][a]	96	227	0.8	0.5–1.1
Hours et al., 2007 (France) [30]	71	80	0.7	0.4–1.3
Lahkola et al., 2008 (Denmark, Norway, Finland, Sweden, UK) [16]	573	1696	0.8	0.7–0.9
Takebayashi et al., 2008, Japan [17]	55	118	0.7	0.4–1.2
Meta-analysis	1217	3386	0.9	0.8–0.9

[a] Not included in meta-analysis because already part of pooled data in Lahkola et al., 2008 [16].

ulation. Cases were recruited by using the regional cancer registries, and most had a malignant disease. They were diagnosed during 1994–2000, but with some variation for the different medical regions in Sweden. Population based controls were used as reference group. The questionnaire was answered by 267 (91%) of the cases and 750 (92%) of the controls. Of the cases 245 had a cancer diagnosis. Overall no association was found; analogue phones yielded OR = 0.9, 95% CI = 0.6–1.4, digital OR = 1.0, 95% CI = 0.7–1.5 and cordless phones OR = 1.0, 95% CI = 0.7–1.4. No effect of tumor induction period was found, although regarding >10 year latency period only 6 cases had used an analogue phone, OR = 0.7, 95% CI = 0.3–1.7, whereas no case had used a digital or cordless phone with that latency period. The results did not change significantly for ipsilateral or contralateral tumors.

The Nordic part of the Interphone case–control study of an association between use of mobile phones and parotid gland tumors was published in 2006 [40]. Detailed information about mobile phone use was obtained from 60 (85%) cases with malignant tumor, 112 (88%) with benign tumor and 681 (70%) controls. Regular mobile phone use gave OR = 0.7, 95% CI = 0.4–1.3 for malignant tumors and OR = 0.9, 95% CI = 0.5–1.5 for benign parotid gland tumors. For ipsilateral mobile phone use a latency period of ≥10 year yielded OR 0.7, 95% CI = 0.1–5.7 for malignant tumors ($n = 1$) and OR = 2.6, 95% CI = 0.9–7.9 for benign tumors ($n = 6$). Contralateral use was reported by one case with benign tumor and no case with malignant tumor in the same latency group.

As part of the Interphone study results on parotid gland tumor were reported from Israel [41]. It included 402 benign and 58 malignant incident cases, total 460 (87%) of 531 eligible for the time period 2001–2003. Population based matched controls were used, in total 1266 (66%) out of 1920 eligible subjects. Thirteen cases had a latency period of ≥10 year, which gave OR = 0.9, 95% CI = 0.4–1.8. No significantly increased risk was found for duration of use; ≥10 year yielded OR = 1.0, 95% CI = 0.5–2.1. However, for cumulative number of calls >5479 OR = 1.6, 95% CI = 1.1–2.2 was found for ipsilateral and both ears used equally, whereas contralateral use gave OR = 0.8, 95% CI = 0.5–1.2. Similarly, cumulative call time >266.3 h yielded OR = 1.5, 95% CI = 1.1–2.1; contralateral use gave OR = 0.8, 95% CI = 0.6–1.3.

In the meta-analysis using 10 year latency period no overall increased risk was found, OR = 0.8, 95% CI = 0.5–1.4, but for ipsilateral use it increased to OR = 1.7, 95% CI = 0.96–2.9, whereas contralateral use gave OR = 0.4, 95% CI = 0.2–1.2, Table 7.

Table 6
Odds ratios (ORs) and 95% confidence intervals (CIs) from five case–control studies on meningioma including meta-analysis of the studies using ≥10 year latency period. Numbers of exposed cases and controls are given.

Study	Total			Ipsilateral			Contralateral		
Author, year of publication, country, latency, reference number	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI
Lönn et al., 2005, Sweden, ≥10 years [25][a]	12/36	0.9	0.4–1.9	5/18	1.3	0.5–3.9	3/23	0.5	0.1–1.7
Christensen et al., 2005, Denmark, ≥10 years [26][a]	6/8	1.0	0.3–3.2	–	–	–	–	–	–
Schüz et al., 2006, Germany, ≥10 years [28]	5/9	1.1	0.4–3.4	–	–	–	–	–	–
Hardell et al., 2006, Sweden, >10 years [11]	38/99	1.5	0.98–2.4	15/28	2.0	0.98–3.9	12/29	1.6	0.7–3.3
Lahkola et al., 2008 (Denmark, Norway, Finland, Sweden, UK) [16]	73/212	0.9	0.7–1.3	33/113	1.1	0.7–1.7	24/117	0.6	0.4–1.03
Meta-analysis	116/320	1.1	0.8–1.4	48/141	1.3	0.9–1.8	36/146	0.8	0.5–1.3

[a] Not included in meta-analysis because already part of pooled data in Lahkola et al., 2008 [16].

Table 7
Odds ratios (ORs) and 95% confidence intervals (CIs) from three case–control studies on salivary gland tumors including meta-analysis of the studies using ≥10 year latency period.

Study	Total			Ipsilateral			Contralateral		
Author, year of publication, country, latency, reference number	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI	No. of cases/controls	OR	95% CI
Hardell et al., 2004, Sweden, >10 years [39]	6/35	0.7	0.3–1.7	5/13	1.5	0.5–4.2	1/15	0.3	0.03–2.1
Lönn et al., 2006, malignant, Sweden, ≥10 years [40]	2/36	0.4	0.1–2.6	1/23	0.7	0.1–5.7	0/19	–[a]	–[a]
Lönn et al., 2006, benign, Sweden, ≥10 years [40]	7/15	1.4	0.5–3.9	6/9	2.6	0.9–7.9	1/9	0.3	0.0–2.3
Sadetzki et al., 2007, Israel, ≥10 years [41]	13/26	0.9	0.4–1.8	10/16	1.6	0.7–3.7	3/10	0.6	0.2–2.3
Meta-analysis	28/112	0.8	0.5–1.4	22/61	1.7	0.96–2.9	5/34	0.4	0.2–1.2

[a] Not included in meta-analysis because OR could not be estimated.

8. Non-Hodgkin lymphoma

The incidence of NHL increased since the 1960s in Sweden as well as in many western countries with reliable cancer registries. This trend has levelled off since the 1990s, and decreasing exposure to environmental contaminants such as PCBs and dioxins, and also certain pesticides has been postulated to be one explanation [42,43]. As part of a large case–control study on NHL, mainly on exposure to pesticides [44], also questions on the use of wireless phones were included. The study covered the time period December 1, 1999 to April 30, 2002. The questionnaire was answered by 910 (91%) cases and 1016 (92% controls). The majority of the cases had B-cell NHL and we did not find any association with use of wireless phones [45]. Regarding T-cell NHL ($n = 53$) we observed somewhat increased risks; use of analogue phone gave OR = 1.5, 95% CI = 0.6–3.7, digital phone OR = 1.9, 95% CI = 0.8–4.8 and cordless phone OR = 2.5, 95% CI = 1.1–5.6. For certain subtypes of T-cell NHL, the cutaneous and leukemia types, the risks increased further for analogue phone to OR = 3.4, 95% CI = 0.8–15, digital phone to OR = 6.1, 95% CI = 1.3–30, and cordless phone to OR = 5.5, 95% CI = 1.3–24. These results were, however, based on low numbers.

A study from USA included 551 NHL cases and 462 frequency matched controls [46]. Among regular mobile phone users NHL risk was not significantly associated with minutes per week, duration, cumulative lifetime or years of first use. However, total time >8 years gave OR = 1.6, 95% CI = 0.7–3.8. The risk increased with number of years, and was significant for the not specified group of NHL after ≥6 years use yielding OR = 3.2, 95% CI = 1.2–8.4.

9. Testicular cancer

An increasing incidence of testicular cancer has been noted in most western countries during the recent decades. It is the most common cancer type in young men and is not regarded to be an occupational disease. Cryptorchidism is an established risk factors, but also perinatal exposure to persistent organic pollutants with hormone activity has been suggested to be another risk factor [47,48]. There has been concern in the population that use of mobile phones might be a risk factor for testicular dysfunction. We performed a case–control study mainly on the use of PVC plastics as risk factor for testicular cancer [49], and included in the questionnaire also questions on the use of wireless phones. The results were based on answers from 542 (92%) cases with seminoma, 346 (89%) with non-seminoma and 870 (89%) controls [50]. Overall no association was found [50]. Only 13 cases with seminoma had used an analogue phone >10 years yielding OR = 2.1, 95% CI = 0.8–5.1 and one case with non-seminoma; OR = 0.3, 95% CI = 0.04–2.6. No case had used a digital or cordless phone with latency period >10 years. OR did not increase with cumulative use in hours for the different phone types. Regarding use of mobile phone in the stand by mode border line significance was found for seminoma, OR = 1.3, 95% CI = 1.03–1.7, but not for non-seminoma; OR = 0.9, 95% CI = 0.7–1.3. For different localisations during stand by, highest risk was found for seminoma for keeping the phone in ipsilateral trousers pocket, OR = 1.8, 95% CI = 0.97–3.4 whereas contralateral pocket gave OR = 1.0, 95% CI = 0.5–2.0.

10. Malignant melanoma of the eye

Stang et al. [51] conducted a hospital- and population-based case–control study of uveal melanoma and occupational exposures to different sources of radiofrequency radiation. A total of 118 cases with uveal melanoma and 475 controls were included. Exposure to RF-transmitting devices was rated as (a) no RF exposure, (b) possible exposure to mobile phones, or (c) probable/certain exposure to mobile phones. An elevated risk for exposure to RF-transmitting devices was reported. Exposure to radio sets gave OR = 3.0, 95% CI = 1.4–6.3 and probable/certain exposure to mobile

phones OR = 4.2, 95% CI = 1.2–14.5. The authors concluded that several methodologic limitations prevented their results from providing clear evidence on the hypothesized association.

The study was commented among others Johansen et al. [52]. In their cohort of mobile phone subscribers in Denmark no support for an association between mobile phones and ocular melanoma was found. However, as discussed elsewhere [14,15,18,55], there are several methodological limitations in the Danish cohort [21,22] that hamper the interpretation of their findings.

The paper by Stang et al. [51] has also been commented by Inskip [53] in an editorial, the main point being that missing from the paper is any consideration of occupational or recreational exposure to UV radiation.

11. Intratemporal facial nerve tumor

So far only one investigation has studied the risk of intratemporal facial nerve (IFN) tumor and the use of mobile phone [54]. A case–control approach was used with 18 patients with IFN tumors matched with controls ($n = 192$) treated for other diseases, 51 patients treated for acoustic neuroma, 72 treated for rhinosinusitis, and 69 for dysphonia and gastroesophageal reflux. Risk of facial nerve tumorigenesis was compared by extent of mobile phone use. The OR of developing an IFN tumor was 0.6, 95% CI = 0.2–1.9 with any handheld mobile phone use and OR = 0.4, 95% CI = 0.1–2.1 for regular mobile phone use. However, they concluded that the short duration of use precludes definite exclusion as a risk for IFN tumor development. Certainly the cases were too few for a sound epidemiological study and it was not correct to include patients with acoustic neuroma in the reference group.

12. Discussion

A review on use of mobile phones and the association with brain tumors included all case–control studies that we have identified in the peer-review literature. Most studies have published data with rather short latency period and limited information on long-term users.

No other studies than from the Hardell group has published comprehensive results for use of cordless phones (DECT) [2–15]. As we have discussed in our publications it is pertinent to include also such use in this type of studies. Cordless phones are an important source of exposure to microwaves and they are usually used for a longer time period on daily basis as compared to mobile phones. Thus, to exclude such use, as was done in e.g. the Interphone studies, could lead to an underestimation of the risk for brain tumors from use of wireless phones.

We have discussed shortcomings in the Interphone studies in detail elsewhere [55]. Regarding glioma the Swedish Interphone study reported 23 ORs in Table 2 in that publication [25] and 22 of these were <1.0 and one OR = 1.0. For meningioma all 23 ORs were <1.0, six even significantly so. These results indicate a systematic bias in the study unless use of mobile phones prevents glioma and meningioma, which is biologically unlikely. It should be noted that several of the overall ORs also in other Interphone studies were <1.0, some even significantly so. As an example, in the Danish Interphone study on glioma [26] all 17 ORs for high-grade glioma were <1.0, four significantly decreased. Also other Interphone studies reported ORs significantly <1.0, that is a protective effect or rather systematic bias in the studies [16,29,31].

Use of cellular telephones was mostly assessed by personal interviews in the Interphone studies. It is not described how these personal interviews were organized, a tremendous task considering that vast parts of Sweden from north to south had to be covered. In the sparsely populated and extended area in northern Sweden personal interviews must have meant lots of long distance traveling and imposed additional stress on the interviewers. No information was given in the articles on how or if this methodological problem was solved, for example were controls only included from more densely populated areas.

The interviews in the Interphone study were extensive and computer aided. It is likely that such an interview creates a stressful situation for a patient with a recent brain tumor diagnosis and operation. These patients, especially under pressure with a newly diagnosed brain tumor and possible surgery, often have difficulties remembering past exposures and inevitably have problems with concentration and may have problems with other cognitive shortcomings. In the Danish part of the Interphone study it was concluded that the patients scored significantly lower than controls due to recalling words (aphasia), problems with writing and drawing due to paralysis [26]. According to our experience a better option would have been to start with a mailed questionnaire, that can be answered by the patient during a period of more well-being, if necessary this can be complemented by a telephone interview. After surgery it is easier to answer a questionnaire at home, also with the possibility to check phone bills to verify the use. This procedure has the additional advantage that it can be accomplished without disclosure during the data collection, whether a person is a case or a control. Certainly, knowing if it was a case or a control that was interviewed in the Interphone study may have introduced observational bias.

It has been argued that recall bias might be introduced in case–control studies on cancer patients, since the patients would be more prone to find a cause for their disease than the controls. However, the contrary is often the situation since patients do not want to blame themselves for their disease. In one article we presented data on the patients own assumptions of causes of their brain tumor [5]. Of 1429 cases only two expressed concern about mobile phones and no about cordless

phones. Interestingly, cases with a previous cancer diagnosis reported lower frequency for use of wireless phones than those with no previous cancer. No interviewer bias could be demonstrated when exposure data in the questionnaire were compared before and after phone interviews [5].

The diagnosis of tumor type as well as grading is based on histopathology. X-ray investigation or MR alone is insufficient. Of the 371 cases with glioma in the Swedish Interphone study [25] histopathology examination of the tumor was available for 328 (88%) cases, and for 225 (82%) of the meningioma cases. Thus, it is possible that cases without histology confirmation of the diagnosis may have had another type of brain tumor or even brain metastases. Such misclassifications inevitably bias the result towards unity. It is remarkable that 345 glioma cases were stratified according to grade I–IV, although histopathology was available only for 328 cases. In our studies on brain tumors we have histopathology verification of all of the diagnoses. Also, the total number of included cases [25] is not completely consistent with those reported to the Swedish Cancer Registry as we have discussed elsewhere [55]. The study included cases from neurosurgery, oncology and neurology clinics as well as regional cancer registries in the study areas.

Among the controls in the glioma and meningioma study 282 (29%) refused to participate [25]. Among some of these non-responders a short interview was made and only 34% reported regular use of a cellular telephone compared with 59% of the responders. If this discrepancy extends to the total group of non-responders the true percentage of mobile phone users in controls would be approximately 52%. Hence this figure would be lower than in glioma (58% exposed) and acoustic neuroma cases (60%). Only for meningioma with 43% exposed cases a lower percentage was reported, however, considering the sex ratio (women:men) for meningioma of about 2:1 a lower percentage of mobile phone users has to be expected due to the lower rate of users among women. It should be noted that a similar procedure in another Interphone study yielded similar results regarding mobile phone use among responders and non-responders [17].

It was discussed in a medical dissertation [56] that: 'Our Swedish study, that includes a large number of long-term mobile phone users, does not support the few previously reported positive findings, and does not indicate any risk increases neither for short-term or long-term exposures.' Considering the methodological shortcomings and that in contrast to the cited assertion of 'a large number of long-term users' the study subjects included only 25 glioma and 12 meningioma cases with long-term use, its conclusion seems to be going a long way beyond what can be scientifically defended.

It might be mentioned that this area of research seems to be controversial *per se* with unfounded statements [57], easily rebutted [58] and not supported by evolving scientific evidence [59]. Statements on no risk for brain tumors based on short-time use of mobile phones [60] might be considered in a larger context [61].

We included in our studies use of mobile or cordless phone 'any time' in the exposed group and made dose-response calculations based on number of hours of cumulative use. The unexposed group included also subjects with use of wireless phones with ≤1-year latency period. On the contrary, mobile phone use in the Interphone studies was defined as 'regular use' on average once per week during at least 6 months, less than that was regarded as unexposed including also all use within <1 year before diagnosis. This definition of 'regular use' seems to have been arbitrary chosen and might have created both observational and recall bias in the interpretation of such a definition.

Use of cordless phones was not assessed or not clearly presented in the Interphone studies, e.g. [25,28]. We found a consistent pattern of an association between cordless phones and glioma and acoustic neuroma [11,12]. It has been shown that the GSM phones have a median power in the same order of magnitude as cordless phones [62]. Moreover, cordless phones are usually used for longer calls than mobile phones [11,12]. Including subjects using cordless phones in the "unexposed" group in studies on this issue, as for example in the Interphone investigations, would thus underestimate the risk and bias OR against unity.

The case participation was good in our studies, 88% for cases with benign brain tumors, 90% for malignant brain tumor cases and 89% for the controls. On the contrary case participation varied from 37% to 93% and control participation from 42% to 75% in the Interphone studies. Obviously low participation rates for cases and controls might give selection bias and influence the results in the Interphone studies.

Methodological issues in the Interphone studies have been discussed elsewhere [14,15,18,55,63–65]. It was concluded that the actual use of mobile phones was underestimated in light users and overestimated in heavy users. Random recall bias could lead to large underestimation in the risk of brain tumors associated with mobile phone use. It was further suggested that selection bias in the Interphone study resulted in under selection of unexposed controls. Refusal to participate was related to less prevalent use of mobile phones, and this could result in a downward bias in estimates of the disease risk associated with mobile phone use. As discussed by Kundi [18] there was also interview lag time between cases and controls in the Interphone studies that might have been a source of bias due to the fast increase of mobile phone use during the study period. This could have resulted in underestimation of risk.

For salivary gland tumors the results were based on three case-control studies. In the 10 year latency period the meta-analysis gave an almost significantly increased risk for ipsilateral use of mobile phones, and a non-significantly decreased risk for contralateral use. These results were based on few cases. Regarding NHL and testicular cancer some subgroup analysis yielded increased risks, but these results were based on low numbers. Use of mobile phone increased the risk significantly for melanoma of the eye. The study on intratemporal facial nerve tumors is not informative since

it was based on few cases and included acoustic neuroma patients in the control group. It is concluded that all studies were hampered by low numbers of long-term users and need to be replicated for firm evidence of an association between use of mobile phones and these tumor types.

In summary our review yielded a consistent pattern of an increased risk for acoustic neuroma and glioma after >10 years mobile phone latency. Our studies showed also an association with use of cordless phones, an issue that has not been studied at all in most investigations or only rudimentary in two studies. We conclude that current standard for exposure to microwaves during mobile phone use is not safe for long-term exposure and needs to be revised.

Acknowledgements

Supported by grants from Cancer-och Allergifonden, Cancerhjälpen and Örebro University Hospital Cancer Fund. Contribution by co-workers in the various publications is acknowledged.

References

[1] E. Cardis, I. Deltour, S. Mann, M. Moissonnier, M. Taki, N. Varsier, et al., Distribution of RF energy emitted by mobile phones in anatomical structures of the brain, Phys. Med. Biol. 53 (2008) 2771–2783.
[2] L. Hardell, A. Näsman, A. Påhlson, A. Hallquist, K. Hansson Mild, Use of cellular telephones and the risk for brain tumours: a case–control study, Int. J. Oncol. 15 (1) (1999) 113–116.
[3] L. Hardell, A. Näsman, A. Påhlson, A. Hallquist, Case–control study on radiology work, medical X-ray investigations, and use of cellular telephones as risk factors for brain tumors, MedGenMed 2 (2) (2000) E2.
[4] L. Hardell, K. Hansson Mild, A. Påhlson, A. Hallquist, Ionizing radiation, cellular telephones and the risk for brain tumours, Eur. J. Cancer Prev. 10 (6) (2001) 523–529.
[5] L. Hardell, A. Hallquist, K. Hansson Mild, M. Carlberg, A. Påhlson, A. Lilja, Cellular and cordless telephones and the risk for brain tumours, Eur. J. Cancer Prev. 11 (2002) 377–386.
[6] L. Hardell, K. Hansson Mild, M. Carlberg, Case–control study on the use of cellular and cordless phones and the risk for malignant brain tumours, Int. J. Radiat. Biol. 78 (10) (2002) 931–936.
[7] L. Hardell, K. Hansson Mild, M. Carlberg, Further aspects on cellular and cordless telephones and brain tumours, Int. J. Oncol. 22 (2003) 399–407.
[8] L. Hardell, K. Hansson Mild, M. Carlberg, A. Hallquist, A. Påhlson, Vestibular schwannoma, tinnitus and cellular telephones, Neuroepidemiology 22 (2003) 124–129.
[9] L. Hardell, M. Carlberg, K. Hansson Mild, Case–control study on cellular and cordless telephones and the risk for acoustic neuroma or meningioma in patients diagnosed 2000–2003, Neuroepidemiology 25 (2005) 120–128.
[10] L. Hardell, M. Carlberg, K. Hansson Mild, Case–control study of the association between the use of cellular and cordless telephones and malignant brain tumours diagnosed during 2000–2003, Environ. Res. 100 (2) (2006) 232–241.
[11] L. Hardell, M. Carlberg, K. Hansson Mild, Pooled analysis of two case-control studies on the use of cellular and cordless telephones and the risk of benign brain tumours diagnosed during 1997–2003, Int. J. Oncol. 28 (2006) 509–518.
[12] L. Hardell, M. Carlberg, K. Hansson Mild, Pooled analysis of two case–control studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997–2003, Int. Arch. Occup. Environ. Health 79 (2006) 630–639.
[13] L. Hardell, K. Hansson Mild, M. Carlberg, F. Söderqvist, Tumour risk associated with use of cellular telephones or cordless desktop telephones, World J. Surgical Oncol. 4 (2006) 74.
[14] L. Hardell, M. Carlberg, F. Söderqvist, K. Hansson Mild, L. Lloyd Morgan, Long-term use of cellular phones and brain tumours: increased risk associated with use for ≥10 years, Occup. Environ. Med. 64 (2007) 626–632.
[15] L. Hardell, M. Carlberg, F. Söderqvist, K. Hansson Mild, Meta-analysis of long-term mobile phone use and the association with brain tumours, Int. J. Oncol. 32 (2008) 1097–1103.
[16] A. Lahkola, T. Salminen, J. Raitanen, S. Heinävaara, M. Schoemaker, H.C. Christensen, et al., Meningioma and mobile phone use—a collaborative case–control study in five North European countries. Int. J. Epidemiol. online August 2, 2008, doi:10.1093/ije/dyn155.
[17] T. Takebayashi, N. Varsier, Y. Kikuchi, K. Wake, M. Taki, S. Watanabe, et al., Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: a case–control study, Br. J. Cancer 98 (3) (2008) 652–659.
[18] M. Kundi, The controversy about possible relationship between mobile phone use and cancer, Environ. Health Persp. 117 (2009) 316–324.
[19] J.E. Muscat, M.G. Malkin, S. Thompson, R.E. Shore, S.D. Stellman, D. McRee, et al., Handheld cellular telephone use and risk of brain cancer, JAMA 284 (23) (2000) 3001–3007.
[20] J.E. Muscat, M.G. Malkin, R.E. Shore, S. Thompson, A.I. Neugut, S.D. Stellman, et al., Handheld cellular telephones and risk of acoustic neuroma, Neurology 58 (8) (2002) 1304–1306.
[21] C. Johansen, J. Boice, J. McLaughlin, J. Olsen, Cellular telephones and cancer—a nationwide cohort study in Denmark, J. Natl. Cancer Inst. 93 (2001) 203–207.
[22] J. Schüz, R. Jacobsen, J. Olsen, J. Boice Jr., J. McLaughlin, C. Johansen C, Cellular telephone use and cancer risk: update of a nationwide Danish cohort, J. Natl. Cancer Inst. 98 (2006) 1707–1713.
[23] P. Inskip, R. Tarone, E. Hatch, T. Wilcosky, W. Shapiro, R. Selker, et al., Cellular-telephone use and brain tumors, N. Engl. J. Med. 344 (2001) 79–86.
[24] A. Auvinen, M. Hietanen, R. Luukkonen, R. Koskela, Brain tumors and salivary gland cancers among cellular telephone users, Epidemiology 13 (2002) 356–359.
[25] S. Lönn, A. Ahlbom, P. Hall, M. Feychting, Long-term mobile phone use and brain tumor risk, Am. J. Epidemiol. 161 (2005) 526–535.
[26] H. Christensen, J. Schüz, M. Kosteljanetz, H. Poulsen, J. Boice Jr., J. McLaughlin, et al., Cellular telephones and risk for brain tumors: a population-based, incident case–control study, Neurology 64 (2005) 1189–1195.
[27] S. Hepworth, M. Schoemaker, K. Muir, A. Swerdlow, M.J.A. van Tongeren, P.A. McKinney, Mobile phone use and risk of glioma in adults: case–control study, BMJ 332 (2006) 883–887.
[28] J. Schüz, E. Böhler, G. Berg, B. Schlehofer, I. Hettinger, K. Schlaefer, et al., Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany), Am. J. Epidemiol. 163 (2006) 512–520.
[29] A. Lahkola, A. Auvinen, J. Raitanen, M. Schoemaker, H. Christensen, M. Feychting, et al., Mobile phone use and risk of glioma in 5 North European countries, Int. J. Cancer 120 (2007) 1769–1775.
[30] M. Hours, M. Bernard, L. Montestrucq, M. Arslan, A. Bergeret, I. Deltour, et al., Cell phones and risk of brain and acoustic nerve tumours: the French INTERPHONE case–control study, Rev. Epidemiol. Sante Publique 55 (2007) 321–332.
[31] L. Klaeboe, K. Blaasaas, T. Tynes, Use of mobile phones in Norway and risk of intracranial tumours, Eur. J. Cancer Prev. 16 (2007) 158–164.
[32] S. Lönn, A. Ahlbom, P. Hall, M. Feychting, Mobile phone use and the risk of acoustic neuroma, Epidemiology 15 (2004) 653–659.

[33] H. Christensen, J. Schüz, M. Kosteljanetz, H. Poulsen, J. Thomsen, C. Johansen, Cellular telephone use and risk of acoustic neuroma, Am. J. Epidemiol. 159 (2004) 277–283.
[34] M. Schoemaker, A. Swerdlow, A. Ahlbom, A. Auvinen, K. Blaasaas, E. Cardis, et al., Mobile phone use and risk of acoustic neuroma: results of the Interphone case–control study in five North European countries, Br. J. Cancer 93 (2005) 842–848.
[35] T. Takebayashi, S. Akiba, Y. Kikuchi, M. Taki, K. Wake, S. Watanabe, et al., Mobile phone use and acoustic neuroma risk in Japan, Occup. Environ. Med. 63 (2006) 802–807.
[36] B. Schlehofer, K. Schlaefer, M. Blettner, G. Berg, E. Böhler, I. Hettinger, et al., Environmental risk factors for sporadic acoustic neuroma (Interphone Study Group, Germany), Eur. J. Cancer 43 (2007) 1741–1747.
[37] L. Hardell, K. Hansson Mild, M. Carlberg, A. Hallquist, Cellular and cordless telephones and the association with brain tumours in different age group, Arch. Environ. Health 59 (3) (2004) 132–137.
[38] L. Hardell, K. Hansson Mild, M. Carlberg, Use of cellular telephones and brain tumour risk in urban and rural areas, Occup. Environ. Med. 62 (2005) 390–394.
[39] L. Hardell, A. Hallquist, K. Hansson Mild, M. Carlberg, H. Gertzén, E. Schildt, et al., No association between the use of cellular or cordless telephones and salivary gland tumours, Occup. Environ. Med. 61 (8) (2004) 675–679.
[40] S. Lönn, A. Ahlbom, H. Christensen, C. Johansen, J. Schüz, S. Edström, et al., Mobile phone use and risk of parotid gland tumor, Am. J. Epidemiol. 164 (7) (2006) 637–643.
[41] S. Sadetzki, A. Chetrit, A. Jarus-Hakak, E. Cardis, Y. Deutch, S. Duvdevani, et al., Cellular phone use and risk of benign and malignant parotid gland tumors—a nationwide case–control study, Am. J. Epidemiol. 167 (4) (2008) 457–467.
[42] L. Hardell, M. Eriksson, Is the decline of the increasing incidence of non-Hodgkin lymphoma in Sweden and other countries a results of cancer preventive measures? Environ. Health Perspect. 111 (2003) 1704–1706.
[43] L. Hardell, Pesticides, soft-tissue sarcoma and non-Hodgkin lymphoma—historical aspects on the precautionary principle in cancer prevention, Acta Oncol. 47 (3) (2008) 347–354.
[44] M. Eriksson, L. Hardell, M. Carlberg, M. Åkerman, Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis, Int. J. Cancer 123 (2008) 1657–1663.
[45] L. Hardell, M. Eriksson, M. Carlberg, C. Sundström, K. Hansson Mild, Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma, Int. Arch. Occup. Environ. Health 78 (8) (2005) 625–632.
[46] M.S. Linet, T. Taggart, R.K. Severson, J.R. Cerhan, W. Cozen, et al., Cellular telephones and non-Hodgkin lymphoma, Int. J. Cancer 119 (2006) 2382–2388.
[47] L. Hardell, B. van Bavel, G. Lindström, M. Carlberg, A.C. Dreifaldt, H. Wijkström, et al., Increased concentrations of polychlorinated biphenyls, hexachlorobenzene and chlordanes in mothers to men with testicular cancer, Environ. Health Perspect. 111 (2003) 930–934.
[48] L. Hardell, B. van Bavel, G. Lindström, M. Eriksson, M. Carlberg, In utero exposure to persistent organic pollutants in relation to testicular cancer, Int. J. Androl. 29 (2006) 228–234.
[49] L. Hardell, N. Malmqvist, C.G. Ohlson, H. Westberg, M. Eriksson, Testicular cancer and occupational exposure to polyvinyl chloride plastics: a case–control study, Int. J. Cancer 109 (2004) 425–429.
[50] L. Hardell, M. Carlberg, C.G. Ohlson, H. Westberg, M. Eriksson, K. Hansson Mild, Use of cellular and cordless telephones and risk of testicular cancer, Int. J. Androl. 30 (2) (2007) 115–122.
[51] A. Stang, G. Anastassiou, W. Ahrens, K. Bromen, N. Bornfeld, K. Jöckel, The possible role of radiofrequency radiation in the development of uveal melanoma, Epidemiology 12 (2001) 7–12.
[52] C. Johansen, J.D. Boice Jr., J.K. McLaughlin, H.C. Christensen, J.H. Olsen, Mobile phones and malignant melanoma of the eye, Br. J. Cancer 86 (2002) 348–1348.
[53] P.D. Inskip, Frequent radiation exposures and frequency dependent effects: the eyes have it. (Editorials), Epidemiology 12 (2001) 1–4.
[54] H. Warren, A. Prevatt, K. Daly, P. Antonelli, Cellular telephone use and risk of intratemporal facial nerve tumor, Laryngoscope 113 (2003) 663–667.
[55] L. Hardell, M. Carlberg, K. Hansson Mild, Methodological aspects of epidemiological studies on the use of mobile phones and their association with brain tumors, Open Environ. Sci. 2 (2008) 54–61.
[56] S. Lönn, Mobile phone use and risk of intercranial tumors, Medical Dissertation, Karolinska Institute, Stockholm, Sweden, 2004.
[57] H.O. Adami, A. Ahlbom, A. Ekbom, L. Hagmar, M. Ingelman-Sundberg, Opinion—"Experts who talk rubbish", Bioelectromag. Soc. Newslett. 162 (2001) 4–5.
[58] L. Hardell, M. Walker, B. Walhjalt, L.S. Friedman, E.D. Richter, Secret ties to industry and conflicting interests in cancer research, Am. J. Ind. Med. 50 (2007) 227–233.
[59] Risk evaluation of potential environmental hazards from low frequency electromagnetic field exposure using sensitive *in vitro* methods. Final Report, Available from: <http://www.itis.ethz.ch/downloads/REFLEX_Final%20Report_171104.pdf>.
[60] D. Trichopoulus, H.O. Adami, Cellular telephones and brain tumors, N. Engl. J. Med. 344 (2) (2001) 133–134.
[61] D. Michaels, Doubt is Their Product. How Industry's Assault on Science Threatens Your Health, Oxford University Press, New York, 2008.
[62] K. Hansson Mild, L. Hardell, M. Kundi, M.O. Mattsson, Mobile phones and cancer: is there really no evidence of an association? (Review), Int. J. Mol. Med. 12 (2003) 67–72.
[63] M. Vrijheid, E. Cardis, B. Armstrong, A. Auvinen, G. Berg, K. Blaasaas, et al., Validation of short term recall of mobile phone use for the Interphone study, Occup. Environ. Med. 63 (4) (2006) 237–243.
[64] M. Vrijheid, I. Deltour, D. Krewski, M. Sanchez, E. Cardis, The effects of recall errors and of selection bias in epidemiologic studies of mobile phone use and cancer risk, J. Expo. Sci. Environ. Epidemiol. 16 (4) (2006) 371–384.
[65] M. Vrijheid, L. Richardson, B.K. Armstrong, A. Auvinen, G. Berg, M. Carrol,1, et al., Quantifying the impact of selection bias caused by non-participation in a case–control study of mobile phone use, Annals Epidemiol. 19 (2009) 33–41.