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Pooled Analysis of Two Swedish Case–Control Studies on the Use of Mobile and Cordless Telephones and the Risk of Brain Tumours Diagnosed During 1997–2003

Kjell Hansson Mild

National Institute for Working Life, Umeå, Sweden
Department of Natural Sciences, Örebro University, Örebro, Sweden

Lennart Hardell

Department of Natural Sciences, Örebro University, Örebro, Sweden
Department of Oncology, University Hospital, Örebro, Sweden

Michael Carlberg

Department of Oncology, University Hospital, Örebro, Sweden

Here we present the pooled analysis of 2 case–control studies on the association of brain tumours with mobile phone use. Use of analogue cellular phones increased the risk for acoustic neuroma by 5%, 95% confidence interval (CI) = 2–9% per 100 hrs of use. The risk increased for astrocytoma grade III–IV with latency period with highest estimates using >10-year time period from first use of these phone types. The risk increased per one year of use of analogue phones by 10%, 95% CI = 6–14%, digital phones by 11%, 95% CI = 6–16%, and cordless phones by 8%, 95% CI = 5–12%. For all studied phone types OR for brain tumours, mainly acoustic neuroma and malignant brain tumours, increased with latency period, especially for astrocytoma grade III–IV.

malignant tumours benign tumours acoustic neuroma astrocytoma cellular phones

1. INTRODUCTION

The use of wireless phone communication has increased dramatically during the past decade. Today almost everyone in working life has a mobile or a cellular phone and the amount of time spent on the phone is increasing. There is concern over adverse health effects especially those caused by the use of mobile phones since the development has been technology driven rather than based on laboratory and clinical studies on potential adverse health effects. So far most human studies have been limited in their conclusions due to low numbers of long-term users. The brain is a main target organ for exposure to microwaves during the use of both mobile and desktop cordless phones. Our case–control studies on brain tumours are among the first in the world to give results for long-term users, ≥10 years, with large enough numbers of exposed subjects to estimate long-term cancer risk.

Correspondence and requests for offprints should be sent to Kjell Hansson Mild, National Institute for Working Life, SE-907 13 Umeå, Sweden. E-mail: <mild@niwl.se>.

Nordic countries were among the first in the world to introduce this new technology. Analogue (Nordic Mobile Telephone System, NMT) phones operating at 450 MHz were introduced in Sweden in 1981 but portable NMT 450 phones were first introduced in 1984. Analogue phones using 900 MHz were used in Sweden between 1986 and 2000. The digital system (GSM) started in 1991 and is presently the most common phone type. This system uses dual band, 900 and 1 800 MHz. Universal Mobile Telecommunication System (UMTS) or 3G started in Sweden in 2003 operating at 1 900 MHz.

Desktop cordless phones also use wireless technology. First the analogue system in the 800–900 MHz radiofrequency (RF) radiation was used when these phones were available in Sweden in 1988. Digital cordless telephones (DECT) that operate at 1 900 MHz have been used since 1991.

The use of mobile and desktop cordless telephones results in exposure to microwaves. The different types of phones have different output power. An NMT phone operates with a maximum power of 1 W and very seldom down regulates this; a GSM 900 phone operates with a maximum peak power of 2 W but can down regulate this to some milliwatts depending on the distance to the base station, adaptive power control (APC) and a typical value would be a few tens of milliwatts, giving a mean output power of less than 10 mW. Cordless phones lack APC and operate with a peak power of 250 mW, and with a duty cycle of 1/24 giving a mean output power of about 10 mW. The anatomical area with the highest exposure is the ipsilateral (same) side of the brain that is used during the call. If a hands-free device is used and a cellular telephone is placed at another part of the body that anatomical area receives the highest RF exposure.

We have performed six case–control studies since the 1990s on the use of mobile or cordless phones and different tumour types, i.e., brain tumours, salivary gland tumours, non-Hodgkin lymphoma and testicular cancer. Three studies concerned brain tumours and they are presented in the following with some further analysis of the study material.

The first case–control study on brain tumours was rather small [1, 2]. This was followed by two larger case–control studies on brain tumours [3, 4, 5, 6]. Here we present results from the pooled analysis of these two studies [7, 8]. In the following a short description of the studies is given; further details can be found in the various publications. In principle the same epidemiological methods were used in all studies. A summary of our six case–control studies on this topic can be found elsewhere [9].

2. MATERIALS AND METHODS

Ethical committees approved all studies. They were performed in various health service regions in Sweden and at somewhat different time periods for recruitment of cases and controls (Table 1). The cases were reported by Cancer Registries in Sweden, which has a very good coverage of all incident cancer cases. The current address was checked using the national Population Registry.

TABLE 1. Description of Case–Controls Studies by Hardell et al. [1, 2, 3, 4, 5, 6] on the Use of Mobile and Cordless Telephones and the Risk for Brain Tumour

Study	Geographical Area	Years	Included Persons	Response Rate
CNS [1, 2]	Uppsala/Örebro, Stockholm	1994–1996 1995–1996	233 cases 466 controls	209 (90%) cases 425 (91%) controls
CNS [3, 4]	Uppsala/Örebro, Stockholm, Linköping, Göteborg	Jan 1, 1997– June 30, 2000	1 617 cases* 1 617 controls	1 429 (88%) cases 1 470 (91%) controls
CNS, benign [5]	Uppsala/Örebro, Linköping	July 1, 2000– Dec 31, 2003	462 cases** 820 controls	413 (89%) cases 692 (84%) controls
CNS, malignant [6]	Uppsala/Örebro, Linköping	July 1, 2000– December 31, 2003	359 cases** 820 controls	317 (88%) cases 692 (84%) controls

Notes. CNS—central nervous system; *—one case had two benign brain tumours, **—one case had both a malignant and a benign brain tumour.

Deceased cases were excluded in order to get as good assessment of exposure as possible. The controls were drawn from the Swedish Population Registry, thereby matched to the cases on gender, age and geographical area.

3. ASSESSMENT OF EXPOSURE

Exposures to cellular and cordless phones were assessed with a mailed questionnaire including also exposure to certain chemical agents and X-ray investigations and lifetime work history whereby the socioeconomic index (SEI) was assessed since adjustment was made for SEI-code in the statistical analyses. Detailed questions were asked on the use of mobile and cordless phones including years of use, mean use per day in minutes, use of a hands-free device, external antenna in a car and ear most frequently used during phone calls. It was possible to separate the use of analogue and digital mobile phones since different prefixes are used for the phone numbers in Sweden, 010 and 07, respectively. The answers were supplemented over the phone by a trained interviewer using a structured protocol if some details were unclear. The interviews as well as the coding of the answers for statistical analyses were blinded as to case or control status. Details have been further explored in the various publications.

4. STATISTICAL ANALYSIS

Unconditional logistic regression analysis (Stata/SE 8.2 for Windows; StataCorp, College Station, TX, USA) was used to calculate odds ratios (OR) and 95% confidence intervals (CI). The unexposed category consisted of subjects who had not used cellular or cordless phones. The exposed cases and controls were divided according to phone type, analogue, digital or cordless. In the assessment of exposure the use of a mobile or cordless phone that started in the year of diagnosis (corresponding year for the matched control) was disregarded. Thereby the same year of diagnosis of the case was used for the corresponding control as cut-off for exposure. Adjustment was made in the analysis for gender, age, SEI-code and year of diagnosis [7, 8].

We used age as a continuous variable in the analysis. Latency or tumour induction period was analysed using three time periods, >1–5 years, >5–10 years and >10 years from the first use of a cellular or cordless telephone until diagnosis. Note that overall results for all latency groups were calculated in one analysis. The calculations of the combinations of lifetime use in hours (1–1 000, 1 001–2 000 and >2 000 hrs) and latency (>1–5, >5–10 and >10 years) were done separately for each latency category. Duration of use and latency period were used as continuous variables. We calculated OR and 95% CI per 100 hrs of use of the phones and also per one year of use and one-year latency period.

5. RESULTS

The pooled analysis of the two case–control studies on brain tumours was based on answers from 1 254 (88%) cases with benign brain tumour, 905 (90%) with malignant brain tumour and 2 162 (89%) controls. Details from the separate studies can be found elsewhere [3, 4, 5, 6].

Regarding meningioma the risk increased with latency period. With latency of >10 years analogue phones yielded OR = 1.6, 95% CI = 1.02–2.5, digital phones OR = 1.3, 95% CI = 0.5–3.2 and cordless phones OR = 1.6, 95% CI = 0.9–2.8. However, in the multivariate analysis adjusted for the different phone types lower odds ratios were found and none were statically significant [7] (Table 2).

All phone types increased the risk for acoustic neuroma. Regarding analogue phones odds ratio increased with latency period and was highest in the category with latency period of >15 years yielding OR = 3.5, 95% CI = 1.4–10 [7]. Increased risk was also found for digital mobile phones and cordless phones. However, in the multivariate analysis only analogue phones were significant risk factors with OR 2.2, 95% CI = 1.3–3.8 using >10-year latency period [7].

In Table 3 results are displayed per 100 hrs of use, one year of use and latency. Regarding meningioma the risk did not increase significantly per 100 hrs of use. However, per one year of use analogue phones yielded

TABLE 2. Use of Mobile and Cordless Phones and Odds Ratio (OR) and 95% Confidence Intervals (CI) for Different Tumour Types. Adjustment Was Made for Age, Gender, SEI-Code and Year of Diagnosis. Results Are Given for Different Latency Periods

	>1–5-Year Latency			>5–10-Year Latency			>10-Year Latency		
Study	Analogue OR CI	Digital OR CI	Cordless OR CI	Analogue OR CI	Digital OR CI	Cordless OR CI	Analogue OR CI	Digital OR CI	Cordless OR CI
CNS (1997–2003) [7, 8]									
All	1.3 0.9–1.7	1.1 0.97–1.3	1.2 0.97–1.4	1.4 1.1–1.9	1.4 1.1–1.8	1.4 1.1–1.7	2.1 1.5–2.9	2.1 1.1–3.9	1.6 1.1–2.4
Benign, all	1.4 0.9–2.0	1.1 0.9–1.4	1.1 0.9–1.4	1.7 1.2–2.3	1.2 0.9–1.7	1.4 1.1–1.7	1.8 1.2–2.6	1.6 0.8–3.5	1.4 0.8–2.3
Meningoma	1.2 0.8–1.8	1.0 0.8–1.3	1.0 0.8–1.3	1.2 0.8–1.8	1.1 0.8–1.6	1.3 1.01–1.8	1.6 1.02–2.5	1.3 0.5–3.2	1.6 0.9–2.8
Acoustic neuroma	2.3 1.2–4.1	1.4 1.01–2.1	1.5 1.01–2.1	3.4 2.1–5.5	1.8 1.1–3.0	1.5 0.96–2.4	3.1 1.7–5.7	0.6 0.1–5.0	1.0 0.3–2.9
Malignant, all	1.2 0.8–1.8	1.2 0.96–1.5	1.2 0.9–1.5	1.1 0.8–1.6	1.7 1.2–2.2	1.5 1.1–2.0	2.4 1.6–3.4	2.8 1.4–5.7	1.8 1.1–3.0
Astrocytoma, grade I–II	1.1 0.4–2.8	1.4 0.8–2.3	1.3 0.7–2.2	1.1 0.4–2.6	1.6 0.8–3.4	1.6 0.9–3.0	1.6 0.6–4.1	1.3 0.2–11	1.6 0.5–4.6
Astrocytoma, grade III–IV	1.3 0.8–2.2	1.3 0.97–1.7	1.2 0.9–1.7	1.3 0.8–2.0	2.2 1.6–3.1	1.8 1.3–2.5	2.7 1.8–4.2	3.8 1.8–8.1	2.2 1.3–3.9

Notes. SEI—socioeconomic index, CNS—central nervous system.

OR = 1.05, 95% CI = 1.01–1.09 and cordless phones OR = 1.04, 95% CI = 1.01–1.07. Similar results were found for latency period. For acoustic neuroma the risk increased per 100 hrs of use of analogue phones with OR = 1.05, 95% CI = 1.02–1.09. Odds ratio also increased significantly per one year of use and latency for analogue phones. Digital mobile phones and cordless phones did not increase the risk significantly in these calculations.

For astrocytoma grade I–II there was no clear trend of increasing odds ratio with increasing latency period (Table 2) and the risk was not significantly increased. Nor did odds ratio increase significantly per 100 hrs of use, one year of use or one-year latency period for any phone type (Table 3).

On the contrary, for astrocytoma grade III–IV (high grade) odds ratio increased with latency period and was highest with >10-year latency for all phone types. In that latency group analogue phones yielded OR = 2.7, 95% CI = 1.8–4.2, digital phones OR = 3.8, 95% CI = 1.8–8.1 and cordless phones OR = 2.2, 95% CI = 1.3–3.9 (Table 2). In the multivariate analysis analogue phones gave OR = 2.0, 95% CI = 1.4–2.9, digital phones OR = 2.4, 95% CI = 1.1–4.9 and cordless phones OR = 1.3, 95% CI = 0.8–2.3 [8].

The risk increased significantly for astrocytoma grade III–IV per 100 hrs of use, for analogue phones OR = 1.06, 95% CI = 1.03–1.09, digital phones OR = 1.04, 95% CI = 1.02–1.06 and cordless phones OR = 1.02, 95% CI = 1.01–1.03 (Table 3). Also per one year of use and latency period odds ratio increased significantly for all phone types.

In Table 4 analyses are presented for the three different latency periods and in each period three groups of use; 1–1 000, 1 001–2 000 and >2 000 hrs. Increased odds ratios were found for benign tumours in the latency groups of >5–10 and >10 years. These results were mainly explained by the increased risk for acoustic neuroma. There was no obvious dose-response in the three categories of cumulative hours of use, although in several calculations highest risk was found in the category with highest cumulative use in hours.

Clearly for malignant brain tumours the use of mobile phones increased odds ratio in all categories in the latency period of >10 years. All mobile phone use (analogue and digital combined) gave for 1–1 000 hrs OR = 2.0, 95% CI = 1.3–3.0 increasing to OR = 6.4, 95% CI = 3.0–14 in the group of >2000 hrs of cumulative use. Regarding cordless phones use of >1 000 hrs increased odds ratio significantly both for the latency period >5–10 and >10 years.

TABLE 3. Odds Ratio (OR) and 95% Confidence Interval (CI) per 100 hrs of Use, One-Year Use and One-Year Latency Period, Respectively, for Mobile (Analogue, Digital) or Cordless Phones in Brain Tumour Studies [7, 8]. Adjustment Was Made for Age, Gender, SEI-Code and Year of Diagnosis

	Analogue Phone		Digital Phone		Cordless Phone	
	OR	95% CI	OR	95% CI	OR	95% CI
OR per 100 hrs of use						
Benign tumour	1.03	1.003–1.060	1.00	0.98–1.03	1.01	0.998–1.020
Meningioma	1.02	0.99–1.05	0.99	0.96–1.02	1.01	0.997–1.020
Acoustic neuroma	1.05	1.02–1.09	1.03	0.998–1.060	1.01	0.997–1.020
Malignant tumour	1.05	1.02–1.07	1.03	1.01–1.05	1.01	1.01–1.02
Astrocytoma, grade I–II	1.04	0.996–1.100	1.03	0.99–1.06	1.01	0.99–1.03
Astrocytoma, grade III–IV	1.06	1.03–1.09	1.04	1.02–1.06	1.02	1.01–1.03
OR per one year of use						
Benign tumour	1.06	1.03–1.10	1.04	1.0004–1.0700	1.04	1.01–1.06
Meningioma	1.05	1.01–1.09	1.02	0.98–1.06	1.04	1.01–1.07
Acoustic neuroma	1.12	1.06–1.17	1.06	0.995–1.130	1.04	0.99–1.10
Malignant tumour	1.08	1.04–1.11	1.08	1.04–1.12	1.06	1.03–1.09
Astrocytoma, grade I–II	1.03	0.94–1.13	1.06	0.97–1.16	1.05	0.98–1.12
Astrocytoma, grade III–IV	1.10	1.06–1.14	1.11	1.06–1.16	1.08	1.05–1.12
OR per one-year latency period						
Benign tumour	1.05	1.03–1.08	1.04	1.001–1.070	1.04	1.01–1.07
Meningioma	1.03	1.004–1.060	1.02	0.98–1.06	1.04	1.01–1.07
Acoustic neuroma	1.10	1.06–1.14	1.06	0.99–1.13	1.04	0.99–1.09
Malignant tumour	1.06	1.03–1.08	1.08	1.04–1.12	1.05	1.02–1.08
Astrocytoma, grade I–II	1.03	0.96–1.09	1.06	0.97–1.16	1.04	0.98–1.11
Astrocytoma, grade III–IV	1.07	1.04–1.10	1.11	1.06–1.16	1.08	1.04–1.11

Notes. SEI—socioeconomic index.

TABLE 4. Odds Ratio (OR) and 95% Confidence Interval (CI) for Latency Periods and Cumulative Use in Hours of Mobile or Cordless Phones in Brain Tumour Studies [7, 8]. Adjustment Was Made for Age, Gender, SEI-Code and Year of Diagnosis

	>1–5-Year Latency		>5–10-Year Latency		>10-Year Latency	
	Cases Controls	OR CI	Cases Controls	OR CI	Cases Controls	OR CI
Benign tumours						
Analogue phone						
1–1 000 hrs	51 86	1.3 0.9–1.9	85 120	1.6 1.2–2.3	50 75	1.8 1.2–2.8
1 001–2 000 hrs	0 0	—	5 4	3.6 0.9–14	2 4	1.5 0.3–8.4
>2 000 hrs	1 0	—	0 3	—	5 5	2.5 0.7–8.9
Digital phone						
1–1 000 hrs	315 562	1.2 0.96–1.4	87 157	1.3 0.9–1.7	12 12	3.2 1.3–7.6
1 001–2 000 hrs	6 14	1.1 0.4–2.8	8 15	1.4 0.6–3.4	1 4	0.6 0.1–6.1
>2 000 hrs	2 5	1.1 0.2–5.7	6 5	4.0 1.2–13	0 2	—

TABLE 4. (*continued*)

	>1–5-Year Latency		>5–10-Year Latency		>10-Year Latency	
	Cases Controls	OR CI	Cases Controls	OR CI	Cases Controls	OR CI
Benign tumours (cont.)						
Mobile phone						
1–1 000 hrs	286 531	1.1 0.9–1.4	150 229	1.4 1.1–1.9	49 68	1.9 1.3–2.9
1 001–20 00 hrs	2 7	0.7 0.2–3.6	15 19	2.2 1.1–4.4	7 20	1.0 0.4–2.5
>2 000 hrs	2 3	1.7 0.3–10	6 12	1.4 0.5–3.9	8 11	2.1 0.8–5.4
Cordless phone						
1–1 000 hrs	228 399	1.1 0.9–1.4	102 166	1.4 1.02–1.8	9 34	0.8 0.3–1.6
1 001–2 000 hrs	14 26	1.0 0.5–2.0	25 23	2.3 1.2–4.1	6 3	4.3 1.03–18
>2 000 hrs	8 12	1.4 0.5–3.4	18 30	1.3 0.7–2.4	13 8	3.5 1.4–8.8
Malignant tumours						
Analogue phone						
1–1 000 hrs	39 86	1.1 0.7–1.1	54 120	1.1 0.8–1.6	54 75	1.8 1.2–2.7
1 001–2 000 hrs	0 0	—	1 4	0.5 0.1–4.9	9 4	5.7 1.7–19
>2 000 hrs	0 0	—	2 3	1.4 0.2–8.8	19 5	9.6 3.5–27
Digital phone						
1–1 000 hrs	254 562	1.2 0.96–1.5	86 157	1.5 1.04–2.0	15 12	4.1 1.7–9.7
1 001–2 000 hrs	9 14	1.5 0.6–3.5	17 15	2.7 1.3–5.6	0 4	—
>2 000 hrs	2 5	0.9 0.2–4.6	15 5	6.5 2.3–19	4 2	5.9 1.01–34
Mobile phone						
1–1 000 hrs	237 531	1.2 0.95–1.5	107 229	1.2 0.9–1.6	52 68	2.0 1.3–3.0
1 001–2 000 hrs	5 7	1.6 0.5–5.0	13 19	1.5 0.7–3.2	16 20	2.0 0.99–4.0
>2 000 hrs	1 3	0.8 0.1–7.7	9 12	1.6 0.6–3.8	28 11	6.4 3.0–14
Cordless phone						
1–1 000 hrs	173 399	1.2 0.9–1.5	79 166	1.3 0.96–1.8	13 34	1.0 0.5–2.0
1 001–2 000 hrs	12 26	1.2 0.6–2.5	20 23	2.5 1.3–4.8	10 3	11 2.9–43
>2 000 hrs	8 12	1.9 0.7–4.7	25 30	2.3 1.3–4.0	10 8	3.9 1.5–10

Notes. SEI—socioeconomic index.

6. DISCUSSION

The results in this pooled analysis were based on a fairly high number of long-term users of mobile and cordless phones. Cases were ascertained from the Swedish Cancer Registry that has a good coverage of all new cases. Controls were enrolled from the Swedish Population Registry that covers

the whole population. Thus, no selection bias was introduced in the enrolment of cases and controls in the various studies. Regarding brain tumours assessment of exposure was made about 2 months after histopathological diagnosis. One advantage was that the cases were informed about their diagnoses and that they could answer the questionnaires and phone interviews at home in a more relaxed setting than in a hospital.

A high response rate was obtained for both cases and controls. All assessment of exposures and coding of the data were made without knowing if it was a case or a control, thereby avoiding observation bias. In the statistical analysis adjustment was made for potential confounding factors such as age, gender, year of diagnosis of the case and corresponding year for the matched control, and SEI. Since the prevalence of the use of mobile and cordless phones increases over the years it was of importance to adjust for year of diagnosis. The incidence of meningioma is higher in women than in men, thus adjustment for gender was necessary.

The main result was an increased risk for acoustic neuroma and high-grade astrocytoma (grade III–IV). Especially for high-grade astrocytoma the risk increased both with latency and the number of hours of use of the studied phone types, and the results seem to be of biological relevance. Odds ratio per years of use and latency was rather similar indicating that most subjects use a phone continuously over the years, merely changing the type of the phone. Obviously the use of analogue phones has declined over the years, whereas the use of digital phones has increased in the Swedish population.

Acoustic neuroma might be a "signal" tumour type for increased brain tumour risk from microwave exposure, since it is located in an anatomical area with high exposure during calls with mobile or cordless phones. In fact, an increasing incidence of acoustic neuroma has been noted in Sweden [9]. The risk increased significantly by 5% (95% CI = 2–9%) for acoustic neuroma per 100 hrs of analogue phones. The risk increased also significantly, 12% (95% CI = 6–17%) per years of use of analogue phones, and similar results were obtained per years of latency period. However, for digital mobile phones or cordless phones the risk did not increase significantly per 100 hrs of use, years of use or latency period. Regarding the three categories of latency time that we analysed we found no clear trend. Increased odds ratio was also found in the shortest latency group, >1–5 years. This might indicate an effect in the late stage of carcinogenesis by microwaves from analogue phones. However, as we have discussed elsewhere [11], longer latency period has been found in other studies on the use of mobile phones and the risk for acoustic neuroma. Clearly further studies are necessary on brain tumours and the use of wireless communication.

Regarding meningioma and astrocytoma grade I–II (low-grade) no clear association was found. However, for astrocytoma grade III–IV (high-grade) the risk increased significantly per 100 hrs of use of all phone types, and also per years of use; 10% (95% CI = 6–14%) for analogue phones, 11% (95% CI = 6–16%) for digital phones and 8% (95% CI = 5–12%) for cordless phones. Thus, these results were similar regardless of the type of phone. As presented elsewhere [8] both analogue and digital mobile telephones were statistically significant risk factors for astrocytoma grade III–IV in the multivariate analysis.

It should be noted that the highest risk for malignant brain tumours was calculated in the >10-year latency group and >2000 hrs of cumulative use (Table 4). Obviously our results indicate a longer latency period for malignant brain tumours than for acoustic neuroma. It may depend on an effect by microwaves in different stages of carcinogenesis for these tumour types. It is certainly noteworthy and worrying that a very high risk was calculated for malignant brain tumours for use of analogue phones in the >10-year latency group and >2 000 hrs of cumulative use, OR = 9.6, 95% CI = 3.5–27. High odds ratios were also calculated for digital mobile phones and cordless phones.

Recall bias might be a problem in assessment of exposure in case–control studies. Our results with increasing risk with latency period and the time

of cumulative use, especially for astrocytoma grade III–IV, indicate that the findings are not explained by recall bias, but are of biological relevance. Furthermore, different results were found for different tumour types, which would not be expected if recall bias existed.

However, in studies of tumour risk and mobile phone use exposure assessment is a greater problem than for the acute effects since for this type of disease it is the exposure 5–10 years or more ago that is of interest. Most users of mobile phones have not been using just one single telephone. It is even more likely that if they have been using a mobile phone for more than a few years, they will also have changed their phone a few times. Many users will also have been using different phone systems such as analogue and digital, and probably many of them have also been using a cordless phone at home or at work. The problem we are facing is then how to integrate the various specific absorption rate (SAR) distributions from the different devices and add up the different times on these phones to one exposure measure? At the moment it is not clear how to combine the use of different phones with different power output, different systems, different frequencies and different anatomical SAR distribution, into one exposure and dose measure. The difficulties lie in the fact that we do not know the interacting mechanism(s) between the electromagnetic fields emitted from the phone and the biological organism.

7. CONCLUSION

In our series of studies on tumour risk associated with the use of mobile or cordless telephones the consistent finding for all studied phone types was an increased risk for brain tumours, mainly acoustic neuroma and malignant brain tumours. Using a latency period of >10 years odds ratios increased especially for astrocytoma grade III–IV. Our studies were among the first to indicate an association between the use of mobile phones and cordless phones and brain tumours. These results seem to have been corroborated in later studies from other research groups. In a recent review of currently published studies on this topic, one cohort study and 13 case–control studies, we concluded that the use of mobile phones for ≥10 years gives a consistent pattern of an increased risk for acoustic neuroma and glioma, most pronounced for high-grade glioma. The risk is highest for ipsilateral exposure [11].

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