Original Paper

Neuro
epidemiology

Neuroepidemiology 2010;35:109–114
DOI: 10.1159/000311044

Received: December 19, 2009
Accepted: March 22, 2010
Published online: June 15, 2010

Mobile Phone Use and the Risk for Malignant Brain Tumors: A Case-Control Study on Deceased Cases and Controls

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

[a]Department of Oncology, University Hospital, Örebro, and [b]Department of Radiation Physics, Umeå University, Umeå, Sweden

Key Words
Glioma · Astrocytoma · Oligodendroglioma · Cellular phone · Cordless phone

Abstract
We investigated the use of mobile or cordless phones and the risk for malignant brain tumors in a group of deceased cases. Most previous studies have either left out deceased cases of brain tumors or matched them to living controls and therefore a study matching deceased cases to deceased controls is warranted. Recall error is one issue since it has been claimed that increased risks reported in some studies could be due to cases blaming mobile phones as a cause of the disease. This should be of less importance for deceased cases and if cancer controls are used. In this study brain tumor cases aged 20–80 years diagnosed during 1997–2003 that had died before inclusion in our previous studies on the same topic were included. Two control groups were used: one with controls that had died from another type of cancer than brain tumor and one with controls that had died from other diseases. Exposure was assessed by a questionnaire sent to the next-of-kin for both cases and controls. Replies were obtained for 346 (75%) cases, 343 (74%) cancer controls and 276 (60%) controls with other diseases. Use of mobile phones gave an increased risk, highest in the >10 years' latency group yielding odds ratio (OR) = 2.4, and 95% confidence interval (CI) = 1.4–4.1. The risk increased with cumulative number of lifetime hours for use, and was highest in the >2,000 h group (OR = 3.4, 95% CI = 1.6–7.1). No clear association was found for use of cordless phones, although OR = 1.7, 95% CI = 0.8–3.4 was found in the group with >2,000 h of cumulative use. This investigation confirmed our previous results of an association between mobile phone use and malignant brain tumors.

Introduction

Almost everyone has a mobile (cellular) telephone nowadays and there may be more than one phone per person in many countries. The proliferation of use has not been tempered with health concerns, rather on the technical development in this area.

Since the brain is the organ with the highest near-field exposure to microwaves during the use of both mobile and desktop cordless phones an increased risk for brain tumors has been of concern. Several studies have shown an association, and our studies were among the first to clearly indicate an increased risk for both malignant brain tumors and acoustic neuroma for long-term use of

KARGER

Fax +41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

0251–5350/10/0352–0109$26.00/0

Accessible online at:
www.karger.com/ned

Prof. Dr. Lennart Hardell, MD, PhD
Department of Oncology
University Hospital
SE–701 85 Örebro (Sweden)
Tel. +46 19 602 1000, Fax +46 19 10 1768, E-Mail lennart.hardell@orebroll.se

wireless phones. The risk was most pronounced for ipsilateral tumors after a latency period of >10 years. These case-control studies have been published previously [1–8], including literature review and meta-analysis of long-term use of mobile phones [9, 10]. The aim now is not to make a thorough review of this research area since several such articles have been published [11, 12].

Our studies were based on a questionnaire that was sent home to cases and population-based controls. However, we excluded cases that had died before it was possible to collect high-quality data by interview, e.g. which ear had mostly been used during the phone calls. This was important since there is a gradient of microwave exposure to the brain depending on the ear that has been used during the call [13].

However, exclusion of deceased cases was suggested to bias our study results in a review commissioned by the former Swedish Radiation Protection Agency, now called the Swedish Radiation Safety Authority [14]. The reason why such exclusion would bias the results was not given. Since we found an increased risk for malignant brain tumors and acoustic neuroma a decreased risk for deceased cases would be necessary so as to balance the increased risk for living cases.

We decided now to proceed with a case-control study on brain tumor cases that had died before we could interview them, usually about 2 months after diagnosis. Since most of the deceased cases had a malignant brain tumor and overall no clear association was found for meningioma, the most common benign type in our case-control studies, we decided only to include deceased cases with a malignant brain tumor. The regional ethics committee approved the study.

Materials and Methods

The study included cases with a histopathological diagnosis of brain tumor during 1997–2003 aged 20–80 years at the time of the diagnosis. Details have been published previously [6, 7]. Between January 1, 1997 and June 30, 2000 the study area covered Uppsala-Örebro, Stockholm, Linköping and Göteborg medical areas in Sweden, whereas between July 1, 2000 and December 31, 2003 Uppsala-Örebro and Linköping regions were included. Cases were enrolled after we had received copies of the reports to the regional cancer registries.

Controls were selected from the Death Registry in Sweden. Two groups were included: one group consisted of controls that had died from other types of malignant diseases than brain tumor and one group with controls that had died from other diseases than cancer. They were extracted from the Death Registry matched for year of death, gender, age ±5 years and medical region. In total 5 controls in each group were selected for every case. Finally, the control in each group nearest in age to the respective case was chosen for the study.

Relatives to both cases and controls were identified through the Swedish Population Registry at the Swedish Tax Agency. They were identified as wife or husband, child, parent, sibling or other relative.

In this study all diseased cases with a malignant brain tumor diagnosed during the study period 1997–2003 were included. Since the Death Registry at the start of this investigation was not updated for 2003, cases that had died during 2003 were excluded. In total 535 deceased cases with a malignant brain tumor remained in the study. For 64 cases no relative could be identified in the Population Registry and for 7 cases no control fulfilling the matching criteria could be found. Thus, the final study encompassed 464 cases. For 1 case a control that had died from a non-malignant disease could not be found so the control group consisted of 464 controls that had died from a malignant disease and 463 from other causes.

Assessment of Exposure

Exposure was assessed by a questionnaire that contained lifetime working history so as to be able to classify socioeconomic index (SEI) for cases and controls. Use of mobile phones included a question about the type of phone, i.e. analogue prefix 010 or digital prefix 07. Mean number of minutes per day and time period of use were assessed to be able to calculate cumulative lifetime use in number of hours. We asked also questions on telephone use in a car with external antenna and a hands-free device taken as no exposure. Similarly regarding cordless phone the years of use and the mean number of minutes per day were asked for. Since relatives were interviewed it was not meaningful to ask for the ear that had mostly been used during wireless phone calls since the data quality was judged to be too low. Two reminders were sent but no interviews over the phone were performed if the study subject had not answered after these reminders. If necessary data for the responders were supplemented over the phone by a trained interviewer or by letter. Exposure was assessed from November 2006 to August 2008.

Exposure during the year preceding diagnosis or the reference date for matched controls was disregarded, and cases and controls with a year or less of exposure to mobile or cordless phones were considered unexposed. In this study of deceased cases and controls, it was not possible to determine the side of the head on which the mobile or cordless phone was used most often.

Statistical Methods

Unconditional logistic regression analysis was used to calculate odds ratios (OR) and 95% confidence intervals (CI). All analyses were done using Stata/SE 10.1 (Stata/SE 10.1 for Windows; StataCorp., College Station, Tex., USA). The unexposed category consisted of subjects who reported no use of mobile or cordless phones. The exposed cases and controls were divided according to phone type: analogue, digital, and cordless. Analogue and digital phones were also analyzed combined (i.e. mobile phone).

Adjustment was made for sex, age (as a continuous variable), SEI code and year of diagnosis. The same year as for the case was used for the corresponding control. Adjustment for year of diagnosis was made in order to avoid bias in exposure. We used also conditional logistic regression, but the results were similar as for unconditional logistic regression. In order to increase statistical

Table 1. OR and 95% CI for malignant brain tumors (346 cases, 619 controls)

	>1–5 years latency		>5–10 years latency		>10 years latency		Total >1 year latency	
	Ca/Co	OR (95% CI)	Ca/Co	OR (95% CI)	Ca/Co	OR (95% CI)	Ca/Co	OR (95% CI)
Analogue	10/13	1.5 (0.6–3.5)	14/23	1.2 (0.6–2.4)	37/31	2.3 (1.3–4.0)	61/67	1.7 (1.1–2.7)
≤149 h	7/11	1.2 (0.4–3.1)	8/13	1.2 (0.5–2.9)	5/10	1.0 (0.3–3.0)	20/34	1.1 (0.6–2.1)
>149 h	3/2	3.2 (0.5–20)	6/10	1.2 (0.4–3.5)	32/21	3.1 (1.6–5.8)	41/33	2.4 (1.4–4.2)
Digital	35/58	1.2 (0.7–1.9)	46/51	1.7 (1.02–2.7)	2/0	–	83/109	1.4 (0.97–2.1)
≤183 h	21/37	1.1 (0.6–2.0)	14/18	1.4 (0.7–3.1)	1/0	–	36/55	1.2 (0.7–2.0)
>183 h	14/21	1.4 (0.7–3.0)	32/33	1.8 (1.02–3.3)	1/0	–	47/54	1.7 (1.03–2.8)
Mobile phone	33/62	1.0 (0.6–1.7)	35/57	1.2 (0.7–1.9)	38/31	2.4 (1.4–4.1)	106/150	1.3 (0.9–1.9)
≤176 h	21/43	1.0 (0.5–1.7)	16/25	1.2 (0.6–2.3)	4/7	1.0 (0.3–3.7)	41/75	1.0 (0.7–1.6)
>176 h	12/19	1.4 (0.6–3.0)	19/32	1.1 (0.6–2.2)	34/24	2.9 (1.6–5.3)	65/75	1.7 (1.1–2.7)
Cordless phone	29/65	0.9 (0.5–1.4)	39/62	1.2 (0.8–1.9)	14/24	1.1 (0.5–2.2)	82/151	1.1 (0.7–1.5)
≤548 h	21/46	0.8 (0.5–1.5)	17/32	1.2 (0.6–2.2)	4/7	1.1 (0.3–3.9)	42/85	1.0 (0.6–1.5)
>548 h	8/19	0.8 (0.3–1.9)	22/30	1.5 (0.8–2.8)	10/17	1.1 (0.5–2.6)	40/66	1.2 (0.7–1.8)
Mobile + cordless phones	41/92	0.9 (0.6–1.3)	56/93	1.1 (0.8–1.8)	47/51	1.7 (1.1–2.8)	144/236	1.1 (0.8–1.5)
≤410 h	30/70	0.8 (0.5–1.3)	21/36	1.2 (0.6–2.1)	11/12	1.8 (0.7–4.2)	62/118	1.0 (0.7–1.4)
>410 h	11/22	1.1 (0.5–2.3)	35/57	1.2 (0.7–1.9)	36/39	1.8 (1.05–3.1)	82/118	1.3 (0.9–1.9)

Numbers of exposed cases (Ca) and controls (Co) are given. Adjustment was made for age, sex, SEI code, and year of diagnosis. Cumulative lifetime hours of use divided by median number in the control group.

power we used unconditional logistic regression analysis adjusting for the matching variables.

Cases were analyzed versus all controls and versus controls without/with cancer. Latency, defined as the number of years from first use of a wireless phone until the year of diagnosis (corresponding year was used for the matched control), was analyzed using three time periods, >1–5, >5–10, and >10 years. In the dose-response calculations, median cumulative lifetime use in number of hours among all controls was used as cutoff. Note that overall results for all latency groups were calculated in one analysis, whereas dose response was analyzed separately for each latency category. Lifetime use in hours was also divided into three groups, 1–1,000, 1,001–2,000 and >2,000 h, to further explore the dose-response relations.

Results

The causes of death for controls without a malignant disease (n = 276) were cardiovascular diseases (n = 187), neurologic diseases (n = 24), lung diseases (n = 16), gastrointestinal diseases (n = 14), infection (n = 11), diabetes (n = 10) and miscellaneous (n = 14).

Replies from relatives were obtained for 346 (75%) cases and 619 (67%) controls in total. Somewhat more relatives to cancer controls (n = 343, 74%) participated than relatives to controls with other diseases (n = 276, 60%). Husband or wife answered the questionnaire for 175 (51%) cases and 340 (55%) controls, son or daughter for 160 (46%) cases and 268 (43%) controls, whereas other relatives (parent or sibling) answered for 11 (3%) cases and 11 (2%) controls.

Use of mobile phones yielded for the whole group of malignant brain tumors OR = 1.3, 95% CI = 0.9–1.9, increasing to OR = 1.7, 95% CI = 1.1–2.7 in the group with use >176 h (i.e. more than the median lifetime use among the controls, table 1). Clearly, the risk was highest in the >10 years latency group (OR = 2.4, 95% CI = 1.4–4.1). Increased risk was found both for analogue and digital phones, although few subjects had a latency period >10 years for digital phones.

OR increased with the cumulative number of hours for mobile phone use with a statistically significant trend (p = 0.02, table 2). Use for >2,000 h gave for analogue phones OR = 5.1, 95% CI = 1.8–14 and for digital phones OR = 3.4, 95% CI = 1.5–8.1.

Regarding the use of cordless phones no statistically significantly increased risk was found (table 1). Cumulative use >2,000 h yielded OR = 1.7, 95% CI = 0.8–3.4 with no statistically significant trend (p = 0.37, table 2).

We also analyzed the risk using controls without cancer and controls with cancer separately. As can be seen in

Table 2. OR and 95% CI for cumulative lifetime use in hours of different types of wireless phones

	1–1,000 h			1,001–2,000 h			>2,000 h		
	Ca/Co	OR	95% CI	Ca/Co	OR	95% CI	Ca/Co	OR	95% CI
All controls (n = 619)									
Analogue	41/52	1.5	0.96–2.5	5/9	1.1	0.3–3.3	15/6	5.1	1.8–14
Digital	58/93	1.2	0.8–1.8	8/6	2.6	0.9–8.0	17/10	3.4	1.5–8.1
Mobile phone	71/119	1.2	0.8–1.7	12/17	1.4	0.6–3.1	23/14	3.4	1.6–7.1
Cordless phone	55/111	1.0	0.7–1.4	11/21	1.0	0.4–2.1	16/19	1.7	0.8–3.4
Mobile + cordless phone	92/166	1.1	0.8–1.5	14/37	0.7	0.4–1.4	38/33	2.2	1.3–3.9
Controls without cancer (n = 276)									
Mobile phone	71/52	1.2	0.8–2.0	12/4	2.9	0.9–9.6	23/7	3.2	1.3–8.3
Cordless phone	55/44	1.2	0.7–1.9	11/8	1.1	0.4–3.0	16/9	1.6	0.7–3.8
Mobile + cordless phone	92/70	1.2	0.8–1.8	14/12	1.1	0.5–2.5	38/13	2.7	1.3–5.4
Controls with cancer (n = 343)									
Mobile phone	71/67	1.1	0.7–1.7	12/13	1.0	0.4–2.3	23/7	3.4	1.3–8.7
Cordless phone	55/67	0.8	0.5–1.3	11/13	0.8	0.4–2.0	16/10	1.7	0.7–3.9
Mobile + cordless phone	92/96	1.0	0.7–1.4	14/25	0.6	0.3–1.1	38/20	1.9	1.02–3.6

Numbers of exposed cases (Ca) and controls (Co) are given. Adjustment was made for age, sex, SEI code, and year of diagnosis. Results are given for using all controls, controls without cancer and controls with other cancer, respectively.

table 2, similar results were obtained with statistically significantly increased OR for mobile phone use in the high exposure group (>2,000 h cumulative use). Thus, using controls without cancer yielded OR = 3.2, 95% CI = 1.3–8.3 for mobile phone use. Using controls with cancer gave OR = 3.4, 95% CI = 1.3–8.7.

Most of the 346 cases with a malignant brain tumor had a glioma (n = 314). Of these cases 267 had astrocytoma grade III–IV. The association pattern did not differ regarding tumor type and was similar to that for the whole study group (data not given). Few cases were diagnosed with oligodendroglioma (n = 9). Formally, a high OR was calculated for oligodendroglioma in the >10 years latency group (OR = 10, 95% CI = 1.1–89), but based on only 2 exposed cases. Other malignant brain tumors constituted 32 cases. In the >10 years latency group, OR = 1.9, 95% CI = 0.5–7.6 was calculated. No statistically significantly increased OR was found in any subgroup of malignant brain tumors for cordless phones (data not given).

Discussion

In our previous case-control studies we excluded all cases that had died [6, 7]. The reason for this was to obtain as high a quality as possible in the assessment of exposure. Thus, the ear mostly used during calls with a mobile or cordless phone would not be easy for a relative to answer. Handedness is not a good predictor for such preference. Since the highest exposure to the brain occurs on the side where the phone has been held during calls it is important to assess laterality of tumor and phone use for risk calculation. In fact, meta-analysis of all epidemiological studies on mobile phone use showed a consistent pattern of increased risk for ipsilateral glioma and acoustic neuroma using a 10-year latency period [10].

It has been claimed that exclusion of deceased cases would bias our results [14]. The reason for that statement is unclear and not explained, it merely seems to be an ad hoc statement. Thus, a decreased risk for brain tumor among deceased cases would be necessary so as to balance an increased risk among living cases. Certainly this would not apply for acoustic neuroma due to the good prognosis for cases with that tumor type. Since we found an increased risk both for malignant brain tumors and acoustic neuroma, with quite different prognosis, that discussion [14] does not seem to have scientific relevance. Moreover, we did not find a statistically significant association for meningioma, so in the same study the results differed for different tumor types.

In the current study we included the deceased cases with a malignant brain tumor. Most of them had a glioma. Since only a few cases with a benign tumor had died

it was not meaningful to include them in the study. Two groups of controls were included, one group that had died from other cancer types than brain tumor and another that had died from other diseases. The rationale for this was to analyze recall bias depending on whether the control had died from cancer or another type of disease. Using cancer controls, relatives might be similarly inclined as those of brain tumor cases to report mobile phone use as a cause of disease. Therefore, cancer controls could be seen as adjusting for attribution bias.

In the present study the response rate for cases and controls was lower than in our previous studies (90% for cases with malignant brain tumors and 89% for controls). As discussed elsewhere this response rate was much higher than in most studies from different countries in the so-called Interphone study group, that varied from 37 to 93% among cases and from 42 to 75% among controls [15]. Also in the present study the response rate of 75% for cases and 67% for controls was higher than in several of the Interphone studies. In contrast to previously and for ethical reasons, this time we did not perform telephone interviews if a relative had not answered after two reminders.

As in previous studies we blinded case or control status in order to minimize observational bias in the assessment of exposure. Consequently this has been one of the items that contributed to the fact that our studies were judged to have high quality in a recent evaluation of this research area [12].

In order to use the same methods for assessment of exposure only deceased cases were included. Relatives were interviewed for both cases and controls in a defined order. The same questions on use of mobile and cordless phones were used as in our previous studies. It was, however, considered that the validity of the reporting of the ear mostly used for wireless phones would be low. Thus, no such question was included in the questionnaire. Information on the whole working history was assessed since social class (i.e. SEI code) might be a predictor of brain tumor risk, i.e. higher risk in so-called white-collar jobs [16].

Unconditional logistic regression analysis was used adjusted for age, gender, and SEI code. Adjustment was also made for year of diagnosis (the same year for the corresponding control), since the use of both mobile and cordless phones has changed rapidly during the most recent decade with increasing prevalence.

The main finding in this study was an association between the use of mobile phones and malignant brain tumors. This result was similar as in our previous studies [6–8]. In these studies the risk increased further for ipsilateral exposure, indicating that the risk would even be higher in the present study if laterality of brain tumors and exposure would have been obtained. All exposure that had started ≤1 year before diagnosis, or the corresponding year for the matched control, was disregarded as in all of our previous studies.

Using latency periods of >1–5 years and >5–10 years did not increase the risk statistically significantly for mobile phone use. As in our previous studies the highest OR was calculated in the >10 years latency group, increasing further in the group with the highest number of hours for cumulative use. Thus, for living cases using a >10 years latency period we calculated OR = 2.4, 95% CI = 1.6–3.4 for analogue mobile phones and OR = 2.8, 95% CI = 1.4–5.7 for digital mobile phones [7]. In the present study with deceased cases the corresponding result was OR = 2.3, 95% CI = 1.3–4.0 for analogue phones, whereas for digital mobile phones 2 cases and no control were exposed in the same category (table 1). The similar results do not indicate that the use of mobile phones promotes the death rate. Furthermore, we did not find any statistically significant difference in survival for cases with ipsilateral or contralateral use of a mobile or cordless phone [8].

In contrast to the findings for mobile phones no statistically significantly increased risk was found for cordless phones. This is in contrast with our previous results [6, 7] and there is no clear explanation for this, although it could be due to random variation. However, it should be noted that the risk was increased in the group with >2,000 h cumulative use, although not statistically significantly so. Of course the interpretation of these results is hampered by relatively few exposed subjects in the high-exposure category.

Deceased controls were used so that assessment of exposure was made among relatives to both cases and controls. Of course validation of exposure by e.g. access to data on use from telecom operators would have been of value. However, our experience since our previous studies in this area is that such data cannot easily be obtained. Furthermore information on the use of cordless phones is lacking among operators. Interviewing relatives of both cases and controls could equally influence the quality of assessment of exposure. Relatives were defined in a strict order and the distribution of types of relatives who were interviewed was similar among cases and controls. Furthermore, during assessment of exposure (telephone interviews) and data coding for the statistical analysis the questionnaires were blinded as to case or control status.

One possibility of recall bias might be that having a cancer diagnosis might lead to overreporting of certain exposures. However, having a cancer diagnosis might also lead to underreporting of exposures so as not to feel guilty of the cancer caused by one's own behavior. We used two control groups, one with controls that had died with a cancer diagnosis other that brain tumor and another with other diseases. We found an increased risk for malignant brain tumors regardless of which control group was used. However, the risk estimate was somewhat lower using cancer controls. No cancer control had a neck and head cancer or a non-Hodkgin's lymphoma [10].

In summary, this study showed as in our previous studies, an increased risk for malignant brain tumors for use of mobile phones. The risk increased with latency and was statistically significant in the >10 years latency group. Cumulative use in hours yielded the highest risk in the >2,000 h group. We could not identify any special group of malignant brain tumors with increased risk, although most tumors were of the astrocytoma type. No statistically significantly increased risk was found for use of cordless phones, although an increased risk was found in the highest exposure group. Our findings were similar using cancer controls or controls that had died from other diseases.

Acknowledgments

Supported by grants from Cancer- och Allergifonden, Cancerhjälpen, Fondkistan and Örebro University Hospital Cancer Fund. Ms. Iréne Larsson contributed to the data collection.

References

1 Hardell L, Näsman A, Påhlson A, Hallquist A, Hansson Mild K: Use of cellular telephones and the risk for brain tumours: a case-control study. Int J Oncol 1999;15:113–116.

2 Hardell L, Hansson Mild K, Påhlson A, Hallquist A: Ionizing radiation, cellular telephones and the risk for brain tumours. Eur J Cancer Prev 2001;10:523–529.

3 Hardell L, Hansson Mild K, Carlberg M: Further aspects on cellular and cordless telephones and brain tumours. Int J Oncol 2003;22:399–407.

4 Hardell L, Carlberg M, Hansson Mild K: Case-control study on cellular and cordless telephones and the risk for acoustic neuroma or meningioma in patients diagnosed 2000–2003. Neuroepidemiology 2005;25:120–128.

5 Hardell L, Carlberg M, Hansson Mild K: Case-control study of the association between the use of cellular and cordless telephones and malignant brain tumors diagnosed during 2000–2003. Environ Res 2006;100:232–241.

6 Hardell L, Carlberg M, Hansson Mild K: 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 2006;28:509–518.

7 Hardell L, Carlberg M, Hansson Mild K: 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 2006;79:630–639.

8 Hardell L, Carlberg M: Mobile phones, cordless phones and the risk for brain tumours. Int J Oncol 2009;35:5–17.

9 Hardell L, Carlberg M, Söderqvist F, Hansson Mild K, Lloyd Morgan L: Long-term use of cellular phones and brain tumours: increased risk associated with use for ≥10 years. Occup Environ Med 2007;64:626–632.

10 Hardell L, Carlberg M, Hansson Mild K: Epidemiological evidence for an association between use of wireless phones and tumor diseases. Pathophysiology 2009;16:113–122.

11 Kundi M: The controversy about a possible relationship between mobile phone use and cancer. Environ Health Perspect 2009;117:316–324.

12 Myung SK, Ju W, McDonnell DD, et al: Mobile phone use and risk of tumors: a meta-analysis. J Clin Oncol 2009;27:5565–5572.

13 Cardis E, Deltour I, Mann S, et al: Distribution of RF energy emitted by mobile phones in anatomical structures of the brain. Phys Med Biol 2008;53:2771–2783.

14 Boice JD, McLaughlin JK: Epidemiologic studies of cellular telephones and cancer risk: a review. SSI Publication 2002;16. Accessed at http://www.stralsakerhetsmyndigheten.se/Publikationer/Rapport/Stralskydd/2002/200216/.

15 Hardell L, Carlberg M, Hansson Mild K: Methodological aspects of epidemiological studies on the use of mobile phones and their association with brain tumors. Open Env Sci 2008;2:54–61.

16 Brownson RC, Reif JS, Chang JC, Davis JR: An analysis of occupational risks for brain cancer. Am J Public Health 1990;80:169–172.