

# Mobile Phone Use and the Risk of Acoustic Neuroma

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**Background:** Radiofrequency exposure from mobile phones is concentrated to the tissue closest to the handset, which includes the auditory nerve. If this type of exposure increases tumor risk, acoustic neuroma would be a potential concern.

**Methods:** In this population-based case-control study we identified all cases age 20 to 69 years diagnosed with acoustic neuroma during 1999 to 2002 in certain parts of Sweden. Controls were randomly selected from the study base, stratified on age, sex, and residential area. Detailed information about mobile phone use and other environmental exposures was collected from 148 (93%) cases and 604 (72%) controls.

**Results:** The overall odds ratio for acoustic neuroma associated with regular mobile phone use was 1.0 (95% confidence interval = 0.6–1.5). Ten years after the start of mobile phone use the estimates relative risk increased to 1.9 (0.9–4.1); when restricting to tumors on the same side of the head as the phone was normally used, the relative risk was 3.9 (1.6–9.5).

**Conclusions:** Our findings do not indicate an increased risk of acoustic neuroma related to short-term mobile phone use after a short latency period. However, our data suggest an increased risk of acoustic neuroma associated with mobile phone use of at least 10 years' duration.

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If radiofrequency exposure from mobile phone use increases the risk of cancer, acoustic neuroma would be of potential concern. The exposure from mobile phones is concentrated in the head close to the handset; exposure is relatively high only for the glial and meningeal tissue closest to the surface of the

head, the parotid gland, and the vestibular portion of the eighth cranial nerve where acoustic neuromas arise.<sup>1,2</sup> Six studies have investigated the association between mobile phone use and acoustic neuroma, with inconsistent results.<sup>3–8</sup> All available studies are limited by a small number of exposed cases and a short follow-up since the time hand-held mobile phones first became available.

In Sweden, mobile phone use became common in the general population relatively early; handheld mobile phones were introduced at the end of the 1980s with an exponential increase of users during the 1990s.<sup>9</sup> Slightly less than 6% of the population used mobile phones in 1990, 23% in 1995, and over 80% today. Therefore, a study based on the Swedish population will have a large proportion of long-term users, which is crucial for the possibility of detecting any increased risk of tumors related to long-term mobile phone use. The study reported here is part of the INTERPHONE study,<sup>10</sup> an international collaborative case-control study of brain tumors, acoustic neuroma, and parotid gland tumors in relation to mobile phone use. We report here results for acoustic neuroma; results for the other outcomes will be reported separately.

## Study Population

A population-based case-control study was conducted including all persons age 20 to 69 years who were residents of 3 geographical areas covered by the regional Cancer Registries in Stockholm, Göteborg, and Lund (a population totaling 3.1 million). The study was approved by the Ethical Committee at Karolinska Institutet.

## Case Ascertainment

Eligible cases were all subjects diagnosed with acoustic neuroma (ICD-10 C72.4 and ICD-O-2 9560.0) during the period from 1 September 1999 to 31 August 2002 in the areas covered by the Lund and Göteborg Cancer Registries and from 1 January 2000 to 31 August 2002 in the Stockholm Cancer Registry area.

Cases were identified continuously during the study period through collaboration with the neurosurgery, oncology, neurology, and otorhinolaryngology clinics at all hospitals in the study areas. Trained research nurses and a psychologist visited these clinics every week to assist in the identification of eligible patients. For each case, the date

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when the case was identified was registered. To ensure full coverage of cases, we searched the regional cancer registries approximately every 3 months for cases missed at the clinics. We identified a total of 160 eligible acoustic neuroma cases, with 10% ( $n = 16$ ) identified from the cancer registry.

Medical records for all cases were examined to confirm the diagnosis to establish date of diagnosis (defined as the first medical examination leading to the diagnosis, usually when the first x-ray was taken), and to determine on which side of the head the tumor occurred. This was made without knowledge about the subjects' mobile phone habits. We used date of diagnosis as the reference date for exposure calculations.

### Control Selection

Controls were randomly selected from the study base stratified on age (in 5-year groups), sex, and residential area (4 regional cancer registry areas). Approximately every 2 months during the study period we selected controls from the continuously updated registry of the Swedish population. The required number of controls per case was stipulated by the common core protocol for the INTERPHONE study (1 per brain tumor case, 2 per acoustic neuroma case, and 3 per parotid gland tumor case); we used the entire set of controls in the present study. In total 838 controls were identified. We defined the reference date for controls as the date when the control was identified, adjusted for the average time difference between date of diagnosis and date of identification of the cases within the same matching stratum to ensure a comparable length of follow-up for cases and controls.

For both cases and controls, we excluded subjects who were completely deaf prior to the reference date or who did not possess the intellectual and language skills necessary to complete an interview.

### Data Collection

Data collection began in September 2000, which means that cases occurring during the first year (6 months in Stockholm) were identified retrospectively. Before contacting the patients, we sought permission from the physician responsible for the treatment of the patient or from the head of the clinic. Two cases died before the first contact, and for those we asked for permission to contact the closest relative as a proxy respondent.

All interviews and contacts with cases and controls were made by nurses or a psychologist employed for this purpose. Most cases and controls were contacted by phone to arrange a time for a personal interview. Some cases were contacted in person directly at the clinic. Study subjects were approached as soon as possible after being identified. Subjects who could not participate in a personal interview were offered a telephone interview instead. Those who refused participation in any kind of interview were asked to answer a

mailed questionnaire. The 16-page mailed questionnaire included selected parts of the personal interview, sufficient to perform analyses of mobile phone exposure with proper confounding control.

The computer-guided interview collected information about environmental exposures (including mobile phone use). The interviewer entered the responses directly into the computer. All interviewers were provided with cards displaying photographs of mobile phones with information about make, models, and year of introduction. Interviews lasted on average 46 minutes.

### Exposure Assessment

Study subjects were asked whether they had ever used a mobile phone, and if they were "regular" users (defined as use of a mobile phone on average once per week during 6 months or more). Regular users were also asked about how many different mobile phones they had used. For each phone, questions were asked about the dates they started and stopped using the phone, the make and model, operator, the duration and number of calls, and changes in their habits of use. The subjects were further asked about use of hands-free equipment and where the majority of calls were made (urban, suburban, or rural areas). The interview also included questions about which side of the head the subject generally held the mobile phone, and about which hand they typically used.

We defined as unexposed those subjects who reported never or only occasionally ("not regularly") using a mobile phone. Exposure within one year of the reference date was not considered. The number of years of regular mobile phone use was categorized into less than 5 years, 5–9 years, and 10 years or more. The same categorization was used for time since first regular use. We calculated cumulative time of mobile phone use, categorized into less than 30 hours, 30–449 hours, and 450 hours or more (cut points at approximately the 25<sup>th</sup> and 75<sup>th</sup> percentile for controls). The cumulative number of mobile phone calls was calculated and categorized into less than 625 calls, 625–7349 calls, and 7350 calls or more (cut points at approximately the 25<sup>th</sup> and 75<sup>th</sup> percentile for controls). Use of analog and digital mobile phones was also analyzed separately.

Use of hands-free devices for mobile phones reduces the amount of exposure from the phone to the head. In our analysis of cumulative hours of use, we reduced the cumulative time depending on estimated use of a hands-free device. Time periods for which the person reported "almost always using a hands-free device" was considered as unexposed. For periods when a hands-free device was used during more than half of the calling time, 75% of the time was excluded; when a hands-free device was used during half of the calling time, 50% of the used time was excluded; when a hands-free device was used during less than half of the calling

time, 25% of the time was excluded from the cumulative hours of use.

Mobile phones were more frequently used on the right side of the head; among the controls, 52% generally held the mobile phone on the right side, 39% on the left side, and approximately 10% reported use on both sides. To analyze the possible association between laterality of phone use and laterality of tumors, the left and right sides were considered separately. Cases were divided into a left-side and a right-side group depending on the localization of the tumor. Controls were randomly assigned, within each strata of the stratification variables (age, sex, residential area), to either the left or the right side group. For both cases and controls, exposure was defined as ipsilateral phone use or use of the phone on both sides, whereas contralateral use was considered unexposed. Side-specific relative risk estimates were calculated, which then was pooled into one relative risk estimate. To test for potential recall bias that would occur if cases overestimate ipsilateral use, similar analyses were made where contralateral phone use or use on both sides was considered exposed and ipsilateral use was considered unexposed.

In addition to mobile phones, we analyzed whether the use of DECT phones (Digital European Cordless Communication) increases the risk of acoustic neuroma. These are cordless phones that communicate with antennas located within ranges up to 500 meters. Regular DECT phone use was defined using the same criteria as regular mobile phone use.

### Confounders and Effect Modifiers

All analyses were adjusted for age, sex, residential area, and educational level categorized into 4 groups: compulsory school (up to 9-year education), vocational or secondary school, upper secondary school, and university. Furthermore, adjustment was made for hearing loss or tinnitus 5 years before the reference date; we also made separate analyses according to hearing ability.

The radiofrequency exposure from a mobile phone is directly related to the output power level used by the phone to communicate with the base station. There are indications that output power levels are higher in rural than in urban areas.<sup>11</sup> Therefore, separate analyses were performed for subjects reporting that they mainly used the mobile phone in urban areas, mainly in rural areas, and in both urban and rural areas.

### Statistical Analysis

We estimated associations between indicators of radiofrequency exposure from mobile phone use and acoustic neuroma as relative risks, calculated as odds ratios (ORs) using unconditional logistic regression models,<sup>12</sup> with 95% confidence intervals (CIs).

## RESULTS

Table 1 presents basic characteristics of cases and controls. Participation rates were 93% (n = 148) for cases and 72% for controls (n = 604). Face-to-face interviews provided exposure information for the majority of cases and controls; 5% of both cases and controls were interviewed over the phone, and 1% of cases and 7% of controls answered the mailed questionnaire. Results were unchanged after excluding answers through mailed questionnaires (data not shown). Reasons for nonparticipation included refusal (cases 4%; controls 16%), illness (cases 1%; controls 2%), and failure to contact the study subjects (cases 2%; controls 11%). Fifty-eight cases (39%) were histologically verified from histopathological reports. Cases that were not histologically verified were diagnosed by CT or MRI.

Table 2 displays the results for various categories of mobile phone use. Odds ratios did not differ between men and women, and therefore results are presented for all subjects combined. For regular use, regardless of duration, the relative risk was estimated to be 1.0 (95% CI = 0.6–1.5). We found a modest increased risk for mobile phone use of at least 10 years duration, and when phone use started at least 10 years before diagnosis. Risk estimates were close to 1.0 for regular use of less than 10 years, or when mobile phone use started less than 10 years before diagnosis.

For ipsilateral mobile phone use (Table 3) of at least 10 years since first use the odds ratio for acoustic neuroma was 3.9 (1.6–9.5). The corresponding result for contralateral use was 0.8 (0.2–2.9). Similar results were found for at least 10

**TABLE 1.** Basic Characteristics of Acoustic Neuroma Cases and Controls

	Cases (n = 148) %	Controls (n = 604) %
Age at reference date		
20–39	18	21
40–59	57	52
60–69	24	27
Sex		
Female	46	52
Male	53	48
Education*		
Compulsory school	20	22
Vocational/secondary school	24	27
Upper secondary school	18	20
University	37	30
Unknown	1	1

\*The highest education completed, as translated from the Swedish educational system.

**TABLE 2.** Association of Acoustic Neuroma With Various Characteristics of Mobile Phone Use\*

	Cases No. (n = 148)	Controls No. (n = 604)	OR (95% CI)
Frequency of use			
Never or rarely <sup>†</sup>	59	248	1.0
Regular use <sup>‡</sup>	89	356	1.0 (0.6–1.5)
Duration of regular use (years)			
<5	47	214	0.8 (0.5–1.3)
5–9	30	107	1.1 (0.7–2.0)
≥10	11	26	1.6 (0.7–3.6)
Time since first regular use (years)			
<5	44	205	0.8 (0.5–1.3)
5–9	30	113	1.1 (0.6–1.8)
≥10	14	29	1.9 (0.9–4.1)
Cumulative use (hours)			
<30	18	93	0.8 (0.4–1.4)
30–449	41	163	1.0 (0.6–1.6)
≥450	25	84	1.1 (0.6–2.1)
Cumulative use adjusted for hands-free use (hours)			
<30	17	99	0.7 (0.4–1.2)
30–449	44	155	1.1 (0.7–1.7)
≥450	21	72	1.1 (0.6–2.1)
Cumulative no. of calls			
<625	18	86	0.8 (0.4–1.4)
625–7349	38	168	0.9 (0.6–1.5)
≥7350	28	87	1.2 (0.7–2.2)
Digital phones			
Regular use <sup>‡</sup>	84	343	0.9 (0.6–1.4)
Time since first regular use (years)			
<5	54	242	0.8 (0.5–1.3)
≥5	29	93	1.2 (0.7–2.1)
Analog phones			
Regular use <sup>‡</sup>	32	85	1.6 (0.9–2.8)
Time since first regular use (years)			
<5	4	18	1.1 (0.4–3.6)
5–9	13	39	1.3 (0.6–2.9)
≥10	14	29	1.8 (0.8–4.3)

\*Adjusted for age, sex, residential area, and education.

<sup>†</sup>Reference category.<sup>‡</sup>Regular use defined as use of a mobile phone on average once per week or more, during 6 months or more.**TABLE 3.** Association of Acoustic Neuroma With Laterality of Mobile Phone Use, by Laterality of the Tumor\*

	Cases No. (n = 138)	Controls No. (n = 601)	OR (95% CI)
Ipsilateral exposure <sup>†</sup>			
Reference category	90	409	1.0
Regular use <sup>‡</sup>	48	192	1.1 (0.7–1.6)
Duration of regular use (years)			
<5	22	117	0.8 (0.5–1.4)
5–9	17	57	1.4 (0.7–2.6)
≥10	9	12	3.1 (1.2–8.4)
Time since first regular use (years)			
<5	20	110	0.8 (0.5–1.4)
5–9	16	63	1.1 (0.6–2.2)
≥10	12	15	3.9 (1.6–9.5)
Contralateral exposure <sup>§</sup>			
Reference category	94	404	1.0
Regular use <sup>‡</sup>	44	197	0.9 (0.6–1.4)
Duration of regular use (years)			
<5	25	113	0.9 (0.5–1.5)
5–9	14	64	0.9 (0.5–1.8)
≥10	4	16	0.9 (0.2–3.1)
Time since first regular use (years)			
<5	24	109	0.9 (0.5–1.5)
5–9	15	67	1.0 (0.5–1.8)
>10	4	17	0.8 (0.2–2.9)

\*Adjusted for age, sex, residential area, and education. One case and 3 controls did not state on which side of the head they generally held the phone; these subjects and was therefore excluded from the analysis. Subjects holding the phone on both sides are considered as exposed in both ipsilateral and contralateral analyses.

<sup>†</sup>Exposure defined as phone use on the same side as the tumor, or on both sides, and reference category as never or rare use of any type of mobile phone and use on the opposite side of the tumor.<sup>‡</sup>Regular use defined as use of a mobile phone on average once per week or more during 6 months or more.<sup>§</sup>Exposure defined as phone use on the opposite side of the tumor or on both sides, and reference category as never or rare use of any type of mobile phone and use on the same side as the tumor.

4.8 (1.1–20.1) for the left side and 3.8 (95% CI 1.2–11.6) for the right side. The risks were close to 1.0 for both ipsilateral and contralateral use of mobile phones when duration of phone use was not taken into account.

The risk estimates did not increase with amount of use estimated as either cumulative number of hours or cumulative number of calls (Table 2). The risk for use of digital phones was close to 1.0, whereas for use of analog phones the odds ratio was 1.6 (0.9–2.8). Adjustment for hearing loss or tinnitus did not change any of the risk estimates, and therefore hearing impairment is not included in the final analyses presented here. Furthermore, results did not differ between

years' duration of use. Side-specific estimates were unstable because of a small number of cases on each side, but risk was increased for tumors on both the left and the right side; for at least 10 years since first use, the estimated relative risk was



subjects reporting hearing loss or tinnitus 5 years before the reference date and those without hearing impairment (results not shown).

For subjects using mobile phones mainly in rural areas, the risk estimate was 0.7 (0.3–1.6), mainly in urban areas 1.4 (0.9–2.3), and in both urban and rural areas 0.7 (0.4–1.2). Similar results were found for 5 years' duration of use (results not shown).

Regular use of DECT phones was associated with an odds ratio for acoustic neuroma of 0.7 (0.4–1.2).

## DISCUSSION

In this study we observed an increased risk of acoustic neuroma for mobile phone use of at least 10 years' duration. The observed association was strongest for tumors on the same side of the head as the phone was normally used. For short-term mobile phone use and a short latency period our results do not indicate any risk increase, regardless of tumor or mobile phone laterality.

Our results for short-term use are in agreement with the majority of previous studies.<sup>3–6,8</sup> The only study<sup>7</sup> that has reported an increased risk of acoustic neuroma among short-term mobile phone users has been criticized for limitations in methods, analysis, and presentation of the study.<sup>13</sup> All previous studies have few subjects with long term exposures, including the recently published Danish case-control study,<sup>8</sup> which is also part of the INTERPHONE project. Table 4 displays the results and number of exposed cases in previous studies for the longest duration of exposure reported. Our study and the Danish case-control study used the same study protocol, and the results from the 2 studies are very similar for short-term mobile phone use. However, the Danish study had few subjects with at least 10 years since first exposure. Thus, none of the previous studies has had sufficient power to study effects of long-term mobile phone use or a long latency. The 2 US studies<sup>4,6</sup> found slightly increased risks in the group

with longest duration of phone use, on the same order of magnitude as in this study, but these results are less stable, and laterality analyses did not take duration of exposure into account. The hospital-based control selection in these studies is also a limitation.

The Danish study<sup>8</sup> and the 2 US studies<sup>4,6</sup> used a laterality analysis restricted to cases only when describing the association between laterality of the tumor and laterality of phone use. Such an analysis assumes that tumors are equally likely to occur on the left and the right side in the absence of mobile phone exposure.<sup>4</sup> In our study, however, 59% of the acoustic neuroma cases were right side tumors; other data also indicate an uneven distribution. For this reason we randomly distributed our controls into 2 control groups, and analyzed left side and right side tumors separately. Thus, these laterality analyses can be viewed as 2 separate case-control studies, where exposure in one study was defined as mobile phone use on the left side of the head, and in the other defined as right side use. The results from the 2 studies were pooled into one risk estimate.

We observed indications of an increased risk with use of analog phones, whereas results for digital phones showed no risk. Because analog phones are the only type of phone that have been used for more than 10 years, it was not possible to separate the effect of type of phone and duration of use. Furthermore, separate analyses of analog phone users have methodological problems, because almost all analog users are also users of digital phones and thereby exposed to radiofrequency fields from both digital and analog phones.

There are alternative explanations for the positive findings. Differential misclassification of the exposure is an obvious problem since mobile phone use is self-reported, and recall bias is a potential problem especially for long-term users. However, there are no indications of recall bias in the analyses of laterality of phone use; the slightly reduced risk for contralateral mobile phone use can explain only a small

**TABLE 4.** Results and Number of Exposed Cases in Previous Studies\* for the Longest Duration of Exposure Reported

Study	RR (95% CI)	No. of Exposed Cases	Duration of Exposure
Hardell et al., 1999 <sup>3</sup>	0.8 (0.1–4.2)	5	Ever exposed
Inskip et al., 2001 <sup>4</sup>	1.9 (0.5–5.1)	5	>5 yr
Johansen et al., 2001 <sup>5</sup>	0.6 (0.3–1.3)	7 <sup>†</sup>	Ever mobile phone subscriber
Muscat et al., 2002 <sup>6</sup>	1.7 (0.5–5.1)	11	3–6 yr
Hardell et al., 2002 <sup>7</sup>	3.5 (0.7–16.8)	7 <sup>‡</sup>	>10 yr
Christensen et al., 2004 <sup>8</sup>	0.2 (0.0–1.1)	2	>10 yr
Present study	1.9 (0.9–4.1)	14	≥10 yr

\*Warren et al. 2003<sup>14</sup> is not included because acoustic neuroma cases were originally selected as controls for another case group and it is impossible to evaluate the completeness of the ascertainment of acoustic neuroma cases.

<sup>†</sup>All cranial nerve sheath tumors. Only ~2% of exposed population had >10 yr since first subscription.

<sup>‡</sup>Discordant pairs.

portion of the risk observed with ipsilateral use. If one case is moved from ipsilateral to contralateral use, the relative risk for contralateral use would be close to 1.0. Thus, random variation is an alternative explanation for the reduced risk associated with contralateral mobile phone use.

There is also a possibility of differential misclassification of the disease. A common symptom of acoustic neuroma is hearing impairment, and mobile phone use on the same side of the head as the tumor occurred might lead to an earlier detection of the tumor because of difficulty hearing when talking on the phone. However, this would also affect mobile phone users with a shorter duration of mobile phone use; to explain the increased risk among long-term mobile phone users in this study some additional factor would have to be in operation.

We found no association between acoustic neuroma and amount of use measured as cumulative number of hours or total number of calls. There was a poor correlation between the number of years since first use and amount of use; many individuals who started to use a mobile phone during recent years are heavy users. There is also evidence that people tend to overestimate their amount of use, and the correlation between subjective reports about amount of use and what was registered by the operator is low.<sup>15</sup> Therefore, there is probably substantial nondifferential misclassification of the measures of amount of use.

This study is population-based, with a rapid ascertainment of cases through active participation by all clinics involved in the treatment of acoustic neuroma. Control selection continuously throughout the study period and adjustment of their reference dates ensured that controls did not have a longer opportunity for exposure than cases. The participation rate was lower among controls compared with cases, which could have introduced selection bias. If mobile phone users are more willing to participate than nonusers, the risk might be underestimated. To test this possibility, subjects who declined participation when contacted by phone were asked if they had regularly used a mobile phone, and among controls in this group the proportion of regular users was 33% compared with 59% among participating controls. On the other hand, only 16% of the nonparticipants answered this question. Among those who we were unable to contact, mobile phone use might be more prevalent; these subjects were either not at home when we on numerous occasions tried to reach them or had unlisted telephone numbers. The phone directory used for the study includes both ordinary phone numbers and mobile phones. However, people tend to change their mobile phone number more often, and therefore the information for mobile phones may not be as up-to-date. The effect of selection bias due to nonparticipation is likely to be marginal.

The etiology of acoustic neuroma is largely unknown, and only a few epidemiologic studies are available. The

dominantly inherited disorder neurofibromatosis type 2 is associated with acoustic neuroma,<sup>16</sup> but can only explain a small minority of the cases. Ionizing radiation exposure is the only established exogenous risk factor for acoustic neuroma, shown in studies of survivors of the atomic bombings in Japan<sup>17</sup> and of subjects going through radiation treatment of tinea capitis during childhood.<sup>18</sup> Other suggested causes include an increased risk associated with extremely loud noise at work<sup>19</sup> and female hormones.<sup>20</sup> Exposure to loud noise is a potential confounder in a study of mobile phone use because hearing loss could be related to an early diagnosis of the tumors and also related to mobile phone use. However, adjustment for hearing loss or tinnitus in our analyses did not change any of the risk estimates, and the results did not differ between subjects reporting hearing loss or tinnitus 5 years before the reference date and those without hearing impairment. Moreover, social class might be considered as a confounder and we therefore adjusted for education in all analyses.

Acoustic neuroma is a slow-growing benign tumor, and it is likely that many of the cases had the tumor several years before first clinical diagnosis. The mean delay from the appearance of the first symptom until the diagnosis has been reported to be more than 5 years.<sup>21</sup> Our data with higher frequency of hearing loss 5 years before diagnosis among cases (30%) than controls (20%) support this. It is therefore very difficult to predict the actual length of a latency period. Among cases with short-term mobile phone use, the tumor could have been present before the start of mobile phone use. Considering that the bulk of new users have come during the last few years, it is not surprising that the overall risk for acoustic neuroma associated with mobile phone use is close to 1.0. Even if our results do not indicate any risk increase after short-term mobile phone use, we cannot exclude the possibility that short-term exposure has an effect that can be detected only after a long latency period. People who started to use a mobile phone early tend also to be long-term users, and therefore we cannot separate the effect of short-term use with a long latency period from the effect of long-term use.

In conclusion, our findings do not indicate any increased risk of acoustic neuroma related to short-term mobile phone use after a short latency period. However, our data suggest an increased risk of acoustic neuroma for mobile phone use of at least 10 years' duration.

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## REFERENCES

- Rothman KJ, Chou CK, Morgan R, et al. Assessment of cellular telephone and other radio frequency exposure for epidemiologic research. *Epidemiology*. 1996;7:291–298.
- Dimbylow PJ, Mann SM. Characterisation of energy deposition in the head from cellular phones. *Radiat Prot Dosim*. 1999;83:139–141.
- Hardell L, Näsman Å, Pahlson A, Hallquist A, Mild KH. Use of cellular telephones and the risk for brain tumours: A case-control study. *Int J Oncol*. 1999;15:113–116.
- Inskip PD, Tarone RE, Hatch EE, et al. Cellular telephone use and brain tumors. *N Engl J Med*. 2001;344:79–86.
- Johansen C, Boice JD, McLaughlin JK, Olsen JH. Cellular telephones and cancer—a nationwide cohort study in Denmark. *J Natl Cancer Inst*. 2001;93:203–207.
- Muscat JE, Malkin MG, Shore RE, et al. Handheld cellular telephones and risk of acoustic neuroma. *Neurology*. 2002;58:1304–1306.
- Hardell L, Hallquist A, Mild KH, Carlberg M, Pahlson A, Lilja A. Cellular and cordless telephones and the risk for brain tumours. *Eur J Cancer Prev*. 2002;11:377–386.
- Christensen HC, Schüz J, Kosteljanetz M, Poulsen HS, Thomsen J, Johansen C. Cellular telephone use and risk of acoustic neuroma. *Am J Epidemiol*. 2004;159:277–283.
- Lonn S, Klæboe L, Hall P, et al. Incidence trends of adult primary intracerebral tumors in four Nordic countries. *Int J Cancer*. 2004;108:450–455.
- Cardis E, Kilkenny M. International case-control study of adult brain, head and neck tumors: results of the feasibility study. *Radiat Prot Dosim*. 1999;83:179–183.
- Lönn S, Forssén UM, Vecchia P, Ahlbom A, Feychting M. Output power levels from mobile phones in relation to the geographic position of the user. *Occup Environ Med*. 2004;61:769–772.
- Breslow NE, Day NE. *Statistical Methods in Cancer research*. Vol. 1. The Analysis of Case-Control Studies. IARC scientific Pub. No. 32. Lyon: International Agency for Research on Cancer; 1980.
- NRPB (2003). Health Effects from Radiofrequency Electromagnetic Fields: Report of an independent Advisory Group on Non-ionising Radiation. DocNRBP 2;14:119–129. Available at: [http://www.nrpb.org/publications/documents\\_of\\_nrpb/pdfs/doc\\_14\\_2.pdf](http://www.nrpb.org/publications/documents_of_nrpb/pdfs/doc_14_2.pdf)
- Warren HG, Prevatt AA, Daly KA, Antonelli PJ. Cellular telephone use and risk of intratemporal facial nerve tumor. *Laryngoscope*. 2003;113:663–667.
- Parslow RC, Hepworth SJ, McKinney P. Recall of past use of mobile phone handsets. *Radiat Prot Dosim*. 2003;106:233–240.
- Lanser MJ, Sussman SA, Frazer K. Epidemiology, pathogenesis, and genetics of acoustic tumors. *Otolaryngol Clin North Am*. 1992;25:499–520.
- Preston DL, Ron E, Yonehara S, et al. Tumors of the nervous system and pituitary gland associated with atomic bomb radiation exposure. *J Natl Cancer Inst*. 2002;94:1555–1563.
- Ron E, Modan B, Boice JD Jr, et al. Tumors of the brain and nervous system after radiotherapy in childhood. *N Engl J Med*. 1988;319:1033–1039.
- Preston-Martin S, Thomas DC, Wright WE, Henderson BE. Noise trauma in the aetiology of acoustic neuromas in men in Los Angeles County, 1978–1985. *Br J Cancer*. 1989;59:783–786.
- Schlehofer B, Blettner M, Wahrendorf J. Association between brain tumors and menopausal status. *J Natl Cancer Inst*. 1992;84:1346–1349.
- Thomsen J, Tos M. Acoustic neuroma: clinical aspects, audiovestibular assessment, diagnostic delay, and growth rate. *Am J Otol*. 1990;11:12–19.