Use of mobile phones in Norway and risk of intracranial tumours

Lars Klaeboe[a], Karl Gerhard Blaasaas[b] and Tore Tynes[a,c]

To test the hypothesis that exposure to radio-frequency electromagnetic fields from mobile phones increases the incidence of gliomas, meningiomas and acoustic neuromas in adults. The incident cases were of patients aged 19–69 years who were diagnosed during 2001–2002 in Southern Norway. Population controls were selected and frequency-matched for age, sex, and residential area. Detailed information about mobile phone use was collected from 289 glioma (response rate 77%), 207 meningioma patients (71%), and 45 acoustic neuroma patients (68%) and from 358 (69%) controls. For regular mobile phone use, defined as use on average at least once a week or more for at least 6 months, the odds ratio was 0.6 (95% confidence interval 0.4–0.9) for gliomas, 0.8 (95% confidence interval 0.5–1.1) for meningiomas and 0.5 (95% confidence interval 0.2–1.0) for acoustic neuromas. Similar results were found with mobile phone use for 6 years or more for gliomas and acoustic neuromas. An exception was meningiomas, where the odds ratio was 1.2 (95% confidence interval 0.6–2.2). Furthermore, no increasing trend was observed for gliomas or acoustic neuromas by increasing duration of regular use, the time since first regular use or cumulative use of mobile phones. The results from the present study indicate that use of mobile phones is not associated with an increased risk of gliomas, meningiomas or acoustic neuromas.

***European Journal of Cancer Prevention* 16:158–164**

European Journal of Cancer Prevention 2007, **16:**158–164

Keywords: acoustic neuroma, glioma, meningioma, mobile phones, odds ratio

[a]The Cancer Registry of Norway, Institute of Population-based Cancer Research, [b]Norwegian Armed Forces Joint Medical Services, Oslo and [c]Norwegian Radiation Protection Authority, Østerås, Norway

Correspondence and requests for reprints to Lars Klaeboe, MSc, The Cancer Registry of Norway, Institute of Population-Based Cancer Research, N-0310 Oslo, Norway
Tel: +47 22451300; fax: +47 22451370;
e-mail: lars.klaeboe@kreftregisteret.no

Sponsorship: This study was supported by funding from the European Union Fifth Framework Program, 'Quality of Life and Management of living Resources' (contract QLK4-CT-1999-01563) and the International Union against Cancer (UICC). The UICC received funds for this purpose from the Mobile Manufacturers' Forum and GSM Association. Provision of funds to the INTERPHONE study investigators via the UICC was governed by agreements that guaranteed INTERPHONE's complete scientific independence. These agreements are publicly available at http://www.iarc.fr/ENG/Units/Rcad.html. No conflict of interest exists.

Received 26 October 2005 **Accepted** 11 January 2006

Introduction

The use of mobile phones increased rapidly during the late 1990s, and there is increasing public concern about a possible link between mobile phone use and brain tumours. Mobile phones represent a relatively new technology, and their possible biological effects in producing neoplasms are unknown (Advisory Group on Non-ionizing Radiation, 2003). In contrast to ionizing radiation, radio-frequency fields do not have enough energy to break chemical bonds or damage DNA (Chang *et al.*, 2005). The electromagnetic fields from mobile phones are concentrated close to the antenna, so the exposure is highest in the structures closest to the surface of the head.

Over the last few years, several epidemiological studies have been published investigating the possibility of a link between mobile telephone use and the risk of brain tumours. No studies of gliomas, meningiomas or acoustic neuromas have found an overall link to mobile phone exposure in general (Boice and McLaughlin, 2002; Christensen *et al.*, 2004, 2005; Lonn *et al.*, 2004a, 2005). When we take the latency time for brain tumours into account, we expect the duration of mobile phone use to be too short for the investigation of the risks from long-term use in many of these studies.

Norway, together with the other Nordic countries, was among the first countries in Western Europe to introduce mobile phones. Norway is therefore suitable for an investigation of the risks of brain tumours in relation to long-term mobile phone use. The Norwegian population has been using mobile phones since the 1980s, and there has been a strong increase of users during the 1990s (Lonn *et al.*, 2004b). Approximately 5% of the population used mobile phones in 1990, 25% in 1995 and over 80% use them currently. Therefore, a study based on the Norwegian population will have a relatively large proportion of long-term users, which is crucial for the detection of any increased risk of tumours related to long-term mobile phone use.

This study is a part of the INTERPHONE study – an international collaborative case–control study of brain

0959-8278

tumours, acoustic neuromas and parotid gland tumours in relation to mobile phone use, co-ordinated by the International Agency for Research on Cancer (IARC) (Cardis and Kilkenny, 1999). Here we report results for gliomas, meningiomas and acoustic neuromas from Norway.

The aim of the present study was to test the hypothesis that exposure from mobile phones increases the risk of gliomas, meningiomas and acoustic neuromas.

Material and methods

Study population

This population-based case–control study was conducted among all citizens aged 19–69 years in two regions: the south/east and the western/middle parts of Norway. These areas are covered by four university hospitals with neurosurgery clinics. Table 1 presents the basic characteristics of cases and controls.

Case ascertainment

The cases were all of individuals in the age group of 19–69 years who had been diagnosed with gliomas, meningiomas and acoustic neuromas throughout the 2-year period 2001–2002 (Table 1).

Cases were identified continuously during the study period through collaboration with the neurosurgery clinics at the hospitals in the study areas. The patients were asked to give written consent before the interview was conducted. Face-to-face interviews were conducted at the time of hospitalization by nurses employed at the respective neurosurgical departments, specially trained medical students or experienced interviewers. To ensure full coverage of the cases, we searched the hospital discharge registries for cases missed at the clinics. These patients were then interviewed either at home or by telephone. Proxy respondents were interviewed for 36% of the glioma cases. None of the meningioma or acoustic neuroma patients was deceased or too ill to participate, so proxy interviews were not undertaken for these individuals. Almost half the interviews in this study were performed by telephone by experienced interviewers. Patients not histologically verified were diagnosed by computed tomography or magnetic resonance imaging. We used the date of diagnosis as the reference date for exposure calculations.

Table 1 Basic characteristics of participating cases and controls

	No. of gliomas (%)	No. of meningiomas (%)	No. of acoustic neuromas (%)	No. of controls (%)
Interviewed participants	289 (77)	207(71)	45 (68)	358 (69)
Age at reference date (years)				
19–39	67 (23)	21 (10)	7 (10)	96 (27)
40–59	139 (49)	131 (65)	30 (44)	175 (49)
60–69	83 (28)	55 (25)	8 (12)	87 (24)
Sex				
Women	119 (43)	156 (74)	23 (51)	182 (51)
Men	170 (57)	51 (26)	22 (49)	176 (49)
Education				
Below upper secondary level	57 (20)	39 (18)	6 (13)	62 (17)
Upper secondary level	145 (50)	105 (51)	22 (49)	161 (45)
Tertiary education	87 (30)	63 (31)	17 (38)	135 (38)
Region				
South/east Norway	183 (63)	132 (64)	25 (56)	224 (63)
Western/middle Norway	106 (37)	75 (36)	20 (44)	134 (37)

Controls

Controls were randomly sampled from the Norwegian Central Population Register on the basis of the unique 11-digit personal number assigned to all Norwegian residents. At the start of the study, we established a pool of 2500 potential controls with the same age and residential distribution as that of the people with brain tumour in Norway (Cancer Registry data). From this pool, we randomly selected 518 individuals who were then invited as controls.

The controls used in the analyses were stratified on age, sex, educational level and residential area. They were contacted by mail and asked to give written consent before the interview was conducted. Reference dates for the controls were calculated as the mean of the diagnosis dates for the cases for each tumour site. The controls had never been diagnosed with any of the tumour types under investigation.

Data collection

Data were collected between 1 January 2001 and 31 March 2004. The participants were asked whether they had been using mobile phones regularly, defined as an average of at least once a week for at least 6 months, and about the models and makes of the phones they had used. For each phone model, information was collected about the start and end dates of use, the average amount of time the phone was used and the average number of calls made. Any substantial change in use was reported if it lasted for more than 6 months; usage information was also collected for these periods. Data were also collected on the extent of hands-free use. Information was collected on preferred ear for telephone use, and whether the individual was left or right handed. Information on the educational level of cases and controls and their spouses/partners was obtained during the interview and was used as a proxy for socio-economic status in the analysis.

The study was approved by the Norwegian Data Inspectorate and The Regional Ethical Committee.

Exposure assessment

We defined as reference category those individuals who reported never or only occasional ('not regularly') use of mobile phones. Exposure within 1 year of the reference date was not considered. The cut points were chosen at approximately the 25th and 75th percentiles for controls. The number of years of regular mobile phone use was categorized into less than 2 years, 2–5 years and 6 years or more. The time since first regular use was also categorized into less than 2 years, 2–5 years and 6 years or more. We calculated the cumulative time of mobile phone use, categorized into less than 17, 17–424 and 425 h or more. The cumulative number of mobile phone calls was calculated and categorized into fewer than 400 calls, 400–6999 calls and 7000 or more calls. Use of analogue and digital mobile phones was also analysed separately.

Use of hands-free devices for mobile phones reduces the amount of exposure from the phone to the head (Bit-Babik *et al.*, 2003). In the subanalysis of cumulative hours of use, we reduced the cumulative time depending on estimated use of hands-free devices. Time periods for which the person reported 'almost always using a hands-free device' was taken as unexposed. For periods when a hands-free device was used during more than half the calling time, 75% of the time was excluded; when a hands-free device was used during half the calling time, 50% of the used time was excluded; and when a hands-free device was used during less than half the calling time, 25% of the time was excluded from the cumulative hours of use.

We conducted analyses of laterality as described by Lonn *et al.* (2004a). For cases, ipsilateral use was defined as either use of a mobile phone on the same side as the tumour or bilateral use. Contralateral use was defined as either use on the opposite side or bilateral use. Controls were randomly assigned as ipsilateral or contralateral users. Patients with tumours in the midsection of the brain or in an unknown location were excluded from this analysis.

Statistical analysis

We analysed associations between indicators of mobile phone use and intracranial tumours, estimated as the odds ratio (OR) with 95% confidence interval (95% CI), using logistic regression models. All analyses were adjusted for age, sex, residential area and attained educational level. We used 20-year age groups and divided residential areas into south/east and western/middle Norway. The statistical package EGRET was used for these analyses (EGRET Statistical Software, 1988).

Results

The overall participation rates for the three cancer sites investigated were 74% ($n = 541$) for cases and 69% ($n = 358$) for controls. The relative risk estimates did not differ between men and women, and the results are presented for all participants combined.

No increased risk was observed for gliomas, meningiomas (Table 2) and acoustic neuromas (Table 3) among regular mobile phone users (glioma: OR 0.6, 95% CI 0.4–0.9; meningioma: OR 0.8, 95% CI 0.5–1.1; acoustic neuroma: OR 0.5, 95% CI 0.2–1.0). In addition, no increasing trend was observed for gliomas or acoustic neuromas by increasing duration of regular use, the time since first regular use or cumulative use of mobile phones. For meningiomas, a tendency towards an increasing trend was observed for the same three indices as above; the highest risk estimate for this site was shown for duration of regular use of more than 6 years (OR 1.2, 95% CI 0.6–2.2).

Adjustment of cumulative use by hands-free devices did not change the risk estimates, for gliomas, meningiomas or acoustic neuromas.

The risk estimates obtained when analysing the subset of glioma patients interviewed by proxy were similar to the results from the overall analysis (data not shown).

For meningiomas, use of analogue telephones showed a moderately increased risk compared with digital telephones, but this was not statistically significant. The OR values for the two categories of duration of regular analogue use analysed were 1.5 and 1.2, respectively.

For ipsilateral regular use of mobile phones, ORs for gliomas and meningiomas were 1.0 (95% CI 0.7–1.4) and 0.9 (95% CI 0.6–1.3), respectively (Table 4). For acoustic neuroma, the OR was 0.7 (95% CI 0.3–1.4) for ipsilateral use (Table 5). The results for contralateral use were 0.7 (95% CI 0.5–1.1) for gliomas, 0.9 (95% CI 0.6–1.3) for meningiomas and 0.9 (95% CI 0.5–1.9) for acoustic neuromas.

Discussion

No association was found between use of mobile phone and risk of gliomas, meningiomas or acoustic neuromas. Nor did the laterality of mobile phone use correlate with the location of tumours. Finally, no clear differences in risk were observed regarding use of analogue or digital mobile phones.

This study was population based and aimed to include all patients diagnosed with gliomas, meningiomas

Table 2 Odds ratio (OR)[a] for gliomas and meningiomas according to mobile phone use, Norway, 2001–2002

	Gliomas				Meningiomas			
	No. of cases	No. of controls	OR	95% CI	No. of cases	No. of controls	OR	95% CI
No or irregular use[b]	128	131	1.0		111	131	1.0	
Regular use[c]	161	227	0.6	0.4–0.9	96	227	0.8	0.5–1.1
Duration of regular use (years)								
<2	38	61	0.6	0.4–1.0	27	63	0.6	0.4–1.1
2–5	68	105	0.6	0.4–0.9	41	114	0.7	0.4–1.1
≥ 6	55	61	0.7	0.4–1.2	28	50	1.2	0.6–2.2
Time since first regular use (years)								
<2	27	46	0.6	0.4–1.1	19	46	0.6	0.3–1.1
2–5	64	108	0.5	0.3–0.8	41	107	0.7	0.4–1.2
≥ 6	70	73	0.8	0.5–1.2	36	74	1.0	0.6–1.8
Cumulative use (h)								
<17	33	54	0.6	0.4–1.0	28	54	0.8	0.4–1.3
17–424	76	115	0.6	0.4–0.9	47	118	0.7	0.4–1.1
≥ 425	52	58	0.7	0.4–1.2	21	55	0.9	0.5–1.8
Cumulative use adjusted for hands-free (h)								
<17	33	55	0.6	0.4–1.0	31	54	0.8	0.5–1.4
17–424	79	118	0.6	0.4–0.9	47	123	0.7	0.4–1.1
≥ 425	49	54	0.7	0.4–1.3	18	49	0.9	0.4–1.7
Cumulative number of calls								
<400	37	56	0.7	0.4–1.1	32	56	0.8	0.5–1.3
400–6999	72	112	0.6	0.4–0.9	41	115	0.7	0.4–1.1
≥ 7000	52	59	0.7	0.4–1.1	23	56	1.0	0.5–1.9
Digital phones								
Regular use[c]	110	170	0.6	0.4–0.8	64	170	0.6	0.4–1.0
Time since first regular use (years)								
<2	26	46	0.6	0.3–1.0	19	46	0.6	0.3–1.1
2–5	60	98	0.5	0.3–0.8	34	97	0.7	0.4–1.2
≥ 6	24	26	0.7	0.4–1.3	11	27	1.0	0.6–1.8
Analogue phones								
Regular use[c]	47	56	0.7	0.4–1.1	31	56	1.2	0.7–2.3
Time since first regular use (years)								
<6	5	42	0.4	0.1–1.4	7	10	1.5	0.5–4.5
≥ 6	10	46	0.7	0.4–1.2	24	46	1.2	0.6–2.4

Note that the totals for variables are not equal because of missing responses to several questions.
CI, confidence interval.
[a]Adjusted for age, sex, residential area and education.
[b]Reference category.
[c]Regular use defined as use of a mobile phone on average once or more a week for 6 months or more.

and acoustic neuromas in Southern Norway. Controls were randomly selected from the population registry and reference dates were adjusted to ensure that follow-up for exposure was as close as possible for cases and controls.

A potential selection bias may have occurred as a result of a 30% non-responses from both cases and controls. If mobile phone users among the controls were more willing than non-users to participate in our study, the risk might have been underestimated. To evaluate this possibility, those who declined participation when contacted were asked to answer a short questionnaire about mobile phone use. Among the controls who refused to participate, 65% answered the short questionnaire, and of these the proportion of regular users was 54% compared with 63% of participating controls. On the other hand, only 3% of the non-participating cases answered this questionnaire, but for participating cases the proportion of regular users was 52%. Mobile phone use might have been more prevalent among those whom we were unable to contact. This selection, as a result of non-participation, might to some extent explain why the observed OR values are less than 1.0.

Recall bias is also a potential problem in this study, with regard to both quantification of use and laterality of use. One might speculate that patients are more likely to overestimate reported exposure although, on the other hand, they are more likely to report ipsilateral use. Glioma patients interviewed after surgery may have given imprecise estimates of exposure as a result of reduced memory. Recall bias concerning overestimation and ipsilateral use is therefore more likely to have occurred among cases of meningiomas and acoustic neuromas than among gliomas. With regard to laterality, our analysis revealed a slightly increased ipsilateral OR for gliomas, with a corresponding decreased OR for contralateral use of mobile phones, indicating recall bias for this site. For meningiomas, no such pattern was seen, indicating no recall bias, and for acoustic neuromas the numbers were too small to evaluate recall bias.

Table 3 Odds ratios (OR)[a] for acoustic neuromas according to mobile phone use, Norway, 2001–2002

	No. of cases	No. of controls	OR	95% CI
No or irregular use[b]	23	131	1.0	
Regular use[c]	22	227	0.5	0.2–1.0
Duration of regular use (years)				
<2	5	67	0.4	0.1–1.2
2–5	10	95	0.5	0.2–1.3
≥6	7	59	0.5	0.2–1.5
Time since first regular use (years)				
<2	4	49	0.4	0.1–1.4
2–5	10	105	0.5	0.2–1.2
≥6	8	67	0.5	0.2–1.4
Cumulative use (h)				
<17	3	55	0.3	0.1–1.0
17–424	12	110	0.6	0.2–1.3
≥425	7	56	0.6	0.2–1.8
Cumulative use adjusted for hands free (h)				
<17	4	57	0.3	0.1–1.0
17–424	12	113	0.5	0.2–1.2
≥425	6	61	0.5	0.2–1.6
Cumulative number of calls				
<400	3	57	0.3	0.1–1.0
400–6999	11	107	0.6	0.2–1.3
≥7000	8	57	0.7	0.2–1.9
Digital phones				
Regular use[c]	13	170	0.2	0.2–0.9
Time since first regular use (years)				
<2	4	49	0.5	0.1–1.5
2–5	7	92	0.4	0.2–1.2
≥6	2	23	0.5	0.1–2.4
Analogue phones				
Regular use[c]	8	56	0.8	0.3–2.2
Time since first regular use (years)				
<6	2	13	1.0	0.2–5.7
≥6	6	43	0.7	0.2–2.2

Note that the totals for variables are not equal because of missing responses to several questions.
CI, confidence interval.
[a]Adjusted for age, sex, residential area and education.
[b]Reference category.
[c]Regular use defined as use of a mobile phone on average once or more a week for 6 months or more.

Differential misclassification of the exposure is an obvious problem in this study, because mobile phone use is self-reported and recall bias is a potential problem especially for long-term users. Only minor indications, however, were found 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 portion of the risk observed with ipsilateral use.

A general concern in mobile phone studies is that the exposure time examined to date might be too short. Only the few countries where mobile phones were introduced very early have been suitable for studying use for more than 10 years. The latency time for brain tumours is unclear, and our current knowledge about development of radiation-induced cancer indicates that the period from first exposure to clinical detection of the cancer can be more than 10 years (United Nations Scientific Committee on the Effects of Atomic Radiation, 2002). Thus, it might be possible that the observation time for the participants in this study might be too short to detect possible effects of mobile telephone use.

Our finding of no increased risk of gliomas is in line with most of the previously reported studies (Boice and McLaughlin, 2002; Hardell *et al.*, 2002; Christensen *et al.*, 2005; Lonn *et al.*, 2005). An exception is, however, a Finnish study reporting an excess risk in the subanalysis of gliomas among analogue phone users (Auvinen *et al.*, 2002). No studies have reported any association between mobile phone use and meningiomas (Hardell *et al.*, 1999; Muscat *et al.*, 2000; Inskip *et al.*, 2001; Auvinen *et al.*, 2002; Christensen *et al.*, 2005; Lonn *et al.*, 2005).

Our results for acoustic neuromas are in agreement with the majority of previous studies (Inskip *et al.*, 2001; Muscat *et al.*, 2002; Christensen *et al.*, 2004). The only studies that have reported increased risks are two Swedish studies; the first reported an overall increased risk among analogue mobile phone users (Hardell *et al.*, 2002). The second, by Lonn *et al.* (2004a), reported no excess risk of acoustic neuromas among short-term users, although a significantly increased risk of acoustic neuromas associated with ipsilateral mobile phone use of at least 10 years' duration was reported.

We found no association between brain tumours and amount of use measured as cumulative number of hours or total number of calls. Evidence also exists 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 network operators is low (Parslow *et al.*, 2003).

Our analysis indicated no increased risk for use of analogue phones compared with digital telephones. Separate analyses of analogue phone users may have methodological problems, as almost all analogue users have also been users of digital phones and thereby been exposed to both digital and analogue phones. Despite the maximum power of digital phones being greater than that of analogue phones, the former operate at a lower average power (Independent Expert Group on Mobile Phones, 2000), and we would expect early users with analogue phones to carry a higher risk. In addition to the frequency and duration of telephone use, factors that can affect the level of exposure to radiofrequency fields include the distance from the base station, whether the phone is used indoors or outdoors, the design of the telephone and the position of the antenna in relation to the head (Independent Expert

Table 4 Odds ratio (OR)[a] for gliomas and meningiomas according to laterality of tumours in relation to laterality of mobile phone use, Norway, 2001–2002

	Gliomas				Meningiomas			
	No. of cases	No. of controls	OR	95% CI	No. of cases	No. of controls	OR	95% CI
Ipsilateral exposure[b]								
Reference category	176	235	1.0		136	235	1.0	
Regular use[c]	91	122	1.0	0.7–1.4	48	122	0.9	0.6–1.3
Duration of regular use (years)								
<2	22	35	0.9	0.5–1.7	16	35	0.8	0.4–1.6
2–5	39	57	0.9	0.6–1.4	18	61	0.7	0.4–1.3
≥6	30	30	1.2	0.7–2.1	14	26	1.4	0.7–2.9
Time since first regular use (years)								
<2	14	29	0.7	0.4–1.4	11	29	0.7	0.3–1.4
2–5	38	56	0.9	0.6–1.5	20	55	0.8	0.5–1.5
≥6	39	37	1.3	0.8–2.1	17	38	1.1	0.6–2.3
Contralateral exposure[d]								
Reference category	193	237	1.0		138	237	1.0	
Regular use[c]	74	120	0.7	0.5–1.1	46	120	0.9	0.6–1.3
Duration of regular use (years)								
<2	19	32	0.8	0.4–1.5	10	34	0.6	0.3–1.3
2–5	28	54	0.6	0.4–1.0	22	59	0.9	0.5–1.6
≥6	27	34	0.9	0.5–1.5	14	27	1.4	0.7–2.9
Time since first regular use (years)								
<2	15	20	1.0	0.5–2.1	6	20	0.5	0.2–1.3
2–5	27	58	0.6	0.3–1.0	22	58	0.9	0.5–1.6
≥6	32	42	0.8	0.5–1.4	18	42	1.2	0.6–2.3

Note that the totals for variables are not equal because of missing responses to several questions.
CI, confidence interval.
[a]Adjusted for age, sex, residential area and education.
[b]Exposure defined as phone use on the same side as the tumour, or on both sides, and reference category as never or rare use of any type of mobile phone and use on the opposite side to the tumour.
[c]Regular use defined as use of a mobile phone on average once or more a week for 6 months or more.
[d]Exposure defined as phone use on the opposite side to the tumour 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 tumour.

Group on Mobile Phones, 2000). Such information was not evaluated in our analysis.

Social class might be considered as a confounder and we therefore adjusted for education in all analyses. Among cases with short-term mobile phone use, the tumour could have been present before the start of mobile phone use. Considering that the bulk of new users has arisen during the last few years, it is not surprising that the overall risk for brain tumours associated with mobile phone use is close to 1.0. Even if our results do not indicate any increased risk after short-term mobile phone use, we cannot exclude the possibility that short-term exposure may have an effect that can be detected only after a long latency period. People who started to use mobile phones early also tend 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.

The aetiology of brain tumours is largely unknown; only ionizing radiation exposure is established as an exogenous risk factor for brain tumours, shown in studies of survivors of the atomic bombings in Japan (United Nations Scientific Committee on the Effects of Atomic Radiation, 2002) and of individuals going through radiation treatment for tinea capitis during childhood (Ron *et al.*, 1988). The dominantly inherited disorder neurofibromatosis type 2 is associated with brain tumours (Kleihues and Cavenee, 2000), but can only explain a small proportion of the cases.

In the absence of information on what biological mechanism might be relevant, it is unclear what aspect of exposure needs to be addressed in epidemiological studies. Heating is the only known effect of radiofrequency field exposure to cause health problems, and until now most research has assumed the unit of measurement to be a function of the specific absorption rate. Mobile phones for use in Norway are not allowed to produce specific absorption rate levels high enough to increase the temperature of nearby tissue by more than 1°C.

In conclusion, our findings do not indicate that mobile phone use increases the risk of gliomas, meningiomas or acoustic neuromas. The latency time for brain tumours is, however, unclear and the follow-up period might be too short to detect possible effects of mobile telephone use.

Table 5 Odds ratio (OR)[a] for acoustic neuromas according to laterality of tumours in relation to laterality of mobile phone use, Norway, 2001–2002

	No. of cases	No. of controls	OR	95% CI
Ipsilateral exposure[b]				
Reference category	33	237	1.0	
Regular use[c]	11	120	0.7	0.3–1.4
Duration of regular use (years)				
<2	1	37	0.2	0.0–1.4
2–5	7	54	1.0	0.4–2.6
≥ 6	3	29	0.7	0.2–2.5
Time since first regular use (years)				
<2	1	30	0.2	0.0–1.8
2–5	5	54	0.7	0.3–2.0
≥ 6	5	36	0.9	0.3–2.8
Contralateral exposure[d]				
Reference category	31	242	1.0	
Regular use[c]	14	115	0.9	0.5–1.9
Duration of regular use (years)				
<2	4	35	1.0	0.3–3.1
2–5	5	46	0.8	0.3–2.4
≥ 6	4	34	0.8	0.3–2.6
Time since first regular use (years)				
<2	3	23	1.1	0.3–4.1
2–5	6	56	0.9	0.3–2.2
≥ 6	4	36	0.8	0.2–2.5

Note that the totals for variables are not equal because of missing responses to several questions.
CI, confidence interval.
[a]Adjusted for age, sex, residential area and education.
[b]Exposure defined as phone use on the same side as the tumour, or on both sides, and reference category as never or rare use of any type of mobile phone and use on the opposite side to the tumour.
[c]Regular use defined as use of a mobile phone on average once or more a week for 6 months or more.
[d]Exposure defined as phone use on the opposite side to the tumour 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 tumour.

Acknowledgements

The authors also wish to thank all involved clinics in the involved hospitals. We also thank the interviewers for skilful work. We will also thank Dr Elisabeth Cardis, coordinating the INTERPHONE project at IARC.

References

Advisory Group on Non-ionizing Radiation (2003). Health effects from radiofrequency electromagnetic fields: report of an independent Advisory Group of Non-ionizing Radiation. NRPB, Vol. 14, pp. 1–177.

Auvinen A, Hietanen M, Luukkonen R, Koskela RS (2002). Brain tumors and salivary gland cancers among cellular telephone users. *Epidemiology* **13**:356–359.

Bit-Babik G, Chou CK, Faraone A, Gessner A, Kanda M, Balzano Q (2003). Estimation of the SAR in the human head and body due to radiofrequency radiation exposure from handheld mobile phones with hands-free accessories. *Radiat Res* **159**:550–557.

Boice JDJ, McLaughlin JK (2002). Epidemiologic studies of cellular telephones and cancer risk: a review. SSI Report. Swedish Radiation Protection Authority, Vol. 16, pp. 0–38.

Cardis E, Kilkenny M (1999). International case–control study of adult brain, head and neck tumours: results of the feasibility study. *Radiat Prot Dosim* **83**:179–183.

Chang SK, Choi JS, Gil HW, Yang JO, Lee EY, Jeon YS, *et al.* (2005). Genotoxicity evaluation of electromagnetic fields generated by 835-MHz mobile phone frequency band. *Eur J Cancer Prev* **14**:175–179.

Christensen HC, Schüz J, Kosteljanetz M, Poulsen HS, Thomsen J, Johansen C (2004). Cellular telephone use and risk of acoustic neuroma. *Am J Epidemiol* **159**:277–283.

Christensen HC, Schuz J, Kosteljanetz M, Poulsen HS, Boice JD Jr, McLaughlin JK *et al.* (2005). Cellular telephones and risk for brain tumors: a population-based, incident case–control study. *Neurology* **64**:1189–1195.

EGRET Statistical Software (1988). *Statistics and epidemiology research corporation*. Washington: SERC Inc.

Hardell L, Näsman Å, Påhlson A, Hallquist A, Mild KH (1999). Use of cellular telephones and the risk for brain tumours: a case–control study. *Int J Oncol* **15**:113–116.

Hardell L, Hallquist A, Mild KH, Carlberg M, Påhlson A, Lilja A (2002). Cellular and cordless telephones and the risk for brain tumours. *Eur J Cancer Prev* **11**:377–386.

Independent Expert Group on Mobile Phones (2000). *Mobile phones and health*. Chilton, Didcot: National Radiological Protection Board.

Inskip PD, Tarone RE, Hatch EE (2001). Cellular-telephone use and brain tumors. *N Engl J Med* **344**:79–86.

Kleihues P, Cavenee WK (2000). *Pathology and genetics of tumours of the nervous system*. Lyon: IARC Press. pp. 219–222.

Lonn S, Ahlbom A, Hall P, Feychting M (2004a). Mobile phone use and the risk of acoustic neuroma. *Epidemiology* **15**:653–659.

Lonn S, Klaeboe L, Hall P, Mathiesen T, Auvinen A, Christensen HC, *et al.* (2004b). Incidence trends of adult primary intracerebral tumors in four Nordic countries. *Int J Cancer* **108**:450–455.

Lonn S, Ahlbom A, Hall P, Feychting M, and the Swedish Interphone Study Group (2005). Long-time mobile phone use and brain tumor risk. *Am J Epidemiol* **161**:526–535.

Muscat JE, Malkin MG, Shore RE, Thompson S, Neugut AI, Stellman SD, *et al.* (2002). Handheld cellular telephones and risk of acoustic neuroma. *Neurology* **58**:1304–1306.

Muscat JE, Malkin MG, Thompson S (2000). Handheld cellular telephone use and risk of brain cancer. *JAMA* **284**:300–317.

Parslow RC, Hepworth SJ, McKinney PA (2003). Recall of past use of mobile phone handsets. *Radiat Prot Dosim* **106**:233–240.

Ron E, Modan B, Boice JD Jr, Alfandary E, Stovall M, Chetrit A, *et al.* (1988). Tumors of the brain and nervous system after radiotherapy in childhood. *N Engl J Med* **319**:1033–1039.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (2002). Sources and effects of ionizing radiation. New York: Report to the General Assembly with scientific annexes.