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# Case-control study on the use of cellular and cordless phones and the risk for malignant brain tumours

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**Abstract.**
*Purpose*: To investigate the use of cellular and cordless phones and the risk for malignant brain tumours.
*Materials and methods*: A case-control study was performed on 649 patients aged 20–80 years of both sexes with malignant brain tumour diagnosed from 1 January 1997 to 30 June 2000. All patients were alive during the time of the study and had histopathology verified brain tumours. One matched control to each case was selected from the Swedish Population Register. The study area was the Uppsala–Örebro, Stockholm, Linköping and Göteborg medical regions of Sweden.
*Results*: Exposure was assessed by a questionnaire answered by 588 (91%) cases and 581 (90%) controls. Phone usage was defined as 'ever use' and usage starting within 1 year before diagnosis was disregarded. Overall, no significantly increased risks were found: analogue cellular phones yielded an odds ratio (OR) = 1.13, 95% confidence interval (CI) = 0.82–1.57, digital cellular phones OR = 1.13, CI = 0.86–1.48, and cordless phones OR = 1.13, CI = 0.85–1.50. For ipsilateral (same side) radiofrequency exposure, analogue mobile phones gave OR = 1.85, CI = 1.16–2.96, for all malignant brain tumours. For astrocytoma, this risk was OR = 1.95, CI = 1.12–3.39. For all malignant brain tumours, digital mobile phones yielded OR = 1.59, CI = 1.05–2.41, and cordless phones yielded OR = 1.46, CI = 0.96–2.23, in the analysis of ipsilateral exposure.
*Conclusion*: The ipsilateral use of an analogue cellular phone yielded a significantly increased risk for malignant brain tumours.

## 1. Introduction

The increasing use of cellular phones has caused concern of an increased risk for brain tumours. The use is widespread in the population and even a small increase in risk would thus have a serious impact on health. Furthermore, an increased risk would strengthen the need for precautionary principles for the use of mobile phones. Radiofrequency (RF) signals are transmitted and received in the range of 400–2000 MHz.

In Sweden, the analogue (Nordic Mobile Telephone System; NMT) was introduced in 1981 operating at 450 MHz. The analogue 900 MHz system started in 1986, but was closed in 2000 in Sweden. The digital system (Global System for Mobile Communication; GSM) started in 1991 and is now the most common mobile phone type. The first cordless phones were available in Sweden in 1988. Initially, the analogue system in the 800–900 MHz RF range was used. Now digital cordless phones that operate at 1900 MHz are available. RF signals from cellular and cordless phones fall within the microwave part of the electromagnetic spectrum.

In a previous study, we found a non-significantly increased risk for brain tumours in the temporal and occipital brain areas in patients with ipsilateral use of a cellular phone (Hardell *et al.* 1999, 2000, 2001). The risk was significantly increased after adjustment for other risk factors in the study (laboratory work and medical diagnostic X-ray investigations of the head/neck) yielding odds ratio (OR) = 2.62, 95% confidence interval (CI) = 1.02–6.71. These anatomical areas represent the parts of the brain with highest RF exposure during a phone call.

The previous study encompassed cases diagnosed during 1994–96. We now report results for malignant brain tumours from a subsequent investigation. No subjects from the previous study were included in the new study. Results for the whole study, including both malignant and benign brain tumours, are reported by Hardell *et al.* (2002). We present here results for malignant brain tumours in further detail.

## 2. Materials and methods

This paper involves a subgroup of prevalent cases with malignant brain tumours within a larger study also including cases with benign brain tumours. Only cases of both sexes with brain tumours diagnosed between 1 January 1997 and 30 June 2000 were included, aged 20–80 years at the time of diagnosis. They were reported in a consecutive way from the four regional cancer registries in the medical regions of Uppsala–Örebro, Stockholm, Linköping and Göteborg. All cases had a diagnosis of histopathology

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and all malignant codes were included (Kleihues and Cavenee 1997). The treating physicians were contacted for permission to include the patient in the study.

In total, 1111 cases with a malignant brain tumour were reported during the study period; 393 patients had died during the time until we received the report from the cancer registry. These cases were not included in this study involving only living subjects. Furthermore, four cases were not resident in the study area, 37 were not capable of participating for medical reasons as reported by the case or a relative, and 28 cases were refused by the treating physician to be included. Finally, 649 living cases were enrolled. One reason to include only living subjects was to get as reliable exposure data as possible. A disadvantage would be if an exposure gives a higher risk for a tumour with a higher degree of malignancy and thus with a bad prognosis for the patient. There is no information that this is the situation for the use of mobile phones, but we will address this topic further in a study only on deceased patients.

One control for each case was drawn from the population register. They were matched for sex and age and lived in the same geographical area (region) of Sweden as the cases. Thus, the study included 649 cases and 649 matched controls.

### 2.1. *Assessment of exposure*

Ethical committees approved the investigation. Information on exposures was assessed by a postal 21-page questionnaire sent to both cases and controls. If the answers were unclear, a nurse trained for this purpose supplemented the answers over the telephone. All study subjects were assigned an identification number, which did not show whether the subject was a case or a control.

The questionnaire contained questions on complete occupational history, exposure to different agents such as, for example, pesticides, asbestos, organic solvents, oils, smoking habits, etc. Special emphasis was put on potential exposure to ionizing radiation and electromagnetic fields. Women were asked for their reproductive history.

Questions were asked about the type of cellular phone, the years of use and brand name, and the mean number of daily calls and their duration. Cumulative use (h) for all years was then calculated. Data were also collected on phone use in a car with a fixed external antenna or a hands-free device with an earpiece outside a car, both taken as no exposure to microwaves. One question asked which ear was used most frequently during cellular phone calls, or if both sides were equally used.

For cordless phones, the years of use, mean number of minutes per day and ear used were requested for in the same way. All exposures that started within 1 year before diagnosis were disregarded. Thereby, the same year was used for the matched control as for the corresponding case: for example, subjects with time of diagnosis in 1998 and exposure starting 1997 were regarded as unexposed. The calculations were based on ever/never use of mobile or cordless phones. Cumulative exposure (h) was calculated up to the year before diagnosis for the case and the matched control.

Since the specific absorption rate (SAR; W $kg^{-1}$) differs for different types of phones, a better estimate would be SAR-hours for exposure. However, we did not have the information permitting us to make such calculations.

Histopathology was obtained from the cancer registry. The anatomical site of the tumour in the brain was determined from neuroradiology records. All coding for the anatomical area of the tumour was done without knowledge of whether the subject was exposed to cellular or cordless phones.

### 2.2. *Statistical methods*

Conditional logistic regression analysis for matched studies was used to calculate OR and 95% CI, (SAS Institute, Inc., Cary, NC, USA). Only complete pairs (1:1) were used. Thereby, the risk for the use of the analogue and digital system as well as cordless phones was calculated separately. This part of the study concerned the use of mobile or cordless phones. The results were adjusted for socio-economic status (SES). In a multivariate analysis, the different phone types and SES were included.

## 3. Results

This part of the study included 649 patients with malignant brain tumour and their matched controls. The questionnaire was answered by 588 (91%) cases and 581 (90%) controls. Of the 588 patients with a malignant brain tumour, 415 had astrocytoma including glioblastoma multiforme, six medulloblastoma, 54 oligodendroglioma, 11 ependymoma, 65 other or mixed glioma, and 37 other malignant brain tumours. The mean age for the cases was 50.2 years and for controls 50.2 years. Of the cases, 340 were men and 248 women; corresponding numbers for controls were 348 men and 233 women, respectively.

Overall, exposure to analogue phones was reported by 110 (18.7%) of the cases and 106 (18.2%) of the controls, digital of 204 (34.7%) cases and 192 (33.0%) controls, and cordless phones of 179 (30.4%) cases

versus 154 (26.5%) controls. Cumulative use (h) for analogue phones was: cases = mean 415 h, median 85 h, range 1–7848 h; controls = mean 236 h, median 101 h, range 1–3833 h; digital: cases = mean 279 h, median 64 h, range 1–3354 h; controls = mean 298 h, median 85 h, range 1–4380 h; cordless: cases = mean 850 h, median 304 h, range 7–16 425 h; controls = mean 707 h, median 183 h, range 2–10 950 h. The median latency (tumour induction) period for use of analogue phones was 7 years, for digital was 4 years and for cordless phones was 5 years for the cases.

The following results were based on 529 complete sets of cases and controls. Results are given for 'ever use', the duration of use (years) as well as cumulative use (h), as above. Ever use of analogue phones yielded OR = 1.13, CI = 0.82–1.57 (table 1). For ipsilateral use of an analogue mobile phone, a significantly increased risk for malignant brain tumours was found, OR = 1.85, CI = 1.16–2.96. No increased risk was found for contralateral use. Using a 5-year time window for exposure, >1–6 years before diagnosis, an increased risk was found as well as with a latency period >6 years.

Overall, digital phones did not increase risk, OR = 1.13, CI = 0.86–1.48 (table 1). However, for ipsilateral exposure, a significantly increased risk was found, OR 1.59, CI = 1.05–2.41. For cordless phones, an OR = 1.13, CI = 0.85–1.50 was calculated (table 1). For ipsilateral exposure, this risk increased further to OR = 1.46, CI = 0.96–2.23.

The use of analogue phones gave OR = 1.29, CI = 0.88–1.91 for astrocytoma including glioblastoma (table 2). For ipsilateral RF exposure, a significantly increased risk with OR = 1.95, CI = 1.12–3.39, was calculated. The risk was highest in the >1–6-year time window of exposure before diagnosis. No increased risk was found for contralateral exposure. For astrocytoma in the temporal or occipital areas, OR = 1.46, CI = 0.65–3.25, was calculated in total regardless of laterality of RF exposure based on 15/11 discordant case/control sets of exposure. This risk was highest in the >1–6-year period yielding OR = 9.64, CI = 1.19–78 (9/1 discordant case/control sets).

The use of digital phone gave OR = 1.13, CI = 0.82–1.56, for astrocytoma (table 2). For ipsilateral exposure, OR = 1.62, CI = 0.99–2.63, was calculated. For astrocytoma in the temporal or occipital areas, OR = 0.87, CI = 0.44–1.72, was calculated in total regardless of side for RF exposure (16/18 discordant case/control sets).

Cordless phones yielded OR = 1.26, CI = 0.90–1.76, for astrocytoma (table 2). Ipsilateral exposure gave OR = 1.66, CI = 1.03–2.68. For astrocytoma in the temporal or occipital areas, OR = 1.40, CI = 0.72–2.74, was calculated in total regardless of the side for RF exposure (21/15 discordant case/control sets).

Analysis of a dose–response effect was performed by dividing cumulative exposure (h) among controls into three equal groups. A dose–response effect was found only for cordless phones (tables 1 and 2).

Table 3 shows the multivariate analysis of the total material. The corresponding univariate results are shown in table 1. No major changes of the results were found.

## 4. Discussion

Both experimental and human data show that RF exposure may have biological effects in target cells or tissue. The Stewart Commission (IEGMP 2000) recently made a overview of experimental studies with relevance for mobile phones. It found evidence that the RF fields can affect some cellular physiological functions, but the significance of such effects for human health is uncertain. Several cancer-related experimental studies do give indications of the effects that are of importance in this respect and they recommend further research in the area to clarify the position. An increased incidence of lymphoma was found in genetically engineered mice exposed to pulsed 900 MHz RF radiation (Repacholi *et al.* 1997).

Results from epidemiological studies are discussed in Hardell *et al.* (2002). Several studies have too short an observation time (tumour induction period) or have other deficits in the design, for instance a lack of information on tumour localization and the ear used during calls. Overall, no significantly increased risks were found. Of interest, however, is the non-significant increased risk for neuroepithelioma in Muscat *et al.* (2000, 2001) and acoustic neurinoma (not significant) in Inskip *et al.* (2001). Dreyer *et al.* (1999) reported an increased standardized mortality rate for brain cancer among hand-held cellular phone users, based on low numbers. However, a significantly increased risk for uveal melanoma was found by Stang *et al.* (2001). Furthermore, of interest are results in a recently published Finnish study with OR = 2.1, 95% CI = 1.3–3.4, for astrocytoma associated with use of analogue phones (Auvinen *et al.* 2002).

Only living subjects were included in the present study and 393 patients with a malignant brain tumour that had died were excluded. This would certainly give the possibility of selection bias which could influence the results if exposure was related to the prognosis of the disease. Data on that is, however, lacking.

During a mobile phone call, the highest exposure

Table 1. Odds ratio (OR) and 95% confidence interval (CI) for malignant brain tumors.

| | Ipsilateral | | Contralateral | | Varying ipsi/contralateral | | All | |
|---|---|---|---|---|---|---|---|---|
| | Ca/Co | OR | Ca/Co | OR | Ca/Co | OR | Ca/Co | OR |
| Analogue | | | | | | | | |
| 450 MHz (ever) | 19/11 | 1.73 (0.82–3.64) | 10/16 | 0.62 (0.28–1.37) | 3/1 | 3.06 (0.32–29.5) | 34/32 | 1.06 (0.66–1.72) |
| 900 MHz (ever) | 40/18 | 2.23 (1.28–3.88) | 13/17 | 0.76 (0.37–1.56) | 3/7 | 0.42 (0.11–1.64) | 58/44 | 1.32 (0.89–1.96) |
| All (450+ 900 MHz) | 50/27 | 1.85 (1.16–2.96) | 19/30 | 0.62 (0.35–1.11) | 6/8 | 0.75 (0.26–2.17) | 79/70 | 1.13 (0.82–1.57) |
| >1–6-year latency | 23/12 | 1.93 (0.96–3.88) | 7/14 | 0.49 (0.20–1.21) | 3/4 | 0.77 (0.17–3.44) | 36/33 | 1.09 (0.68–1.75) |
| >6-year latency | 27/15 | 1.80 (0.96–3.38) | 12/16 | 0.74 (0.35–1.57) | 3/4 | 0.72 (0.16–3.27) | 43/37 | 1.17 (0.75–1.81) |
| ⩽52 h | 21/11 | 2.02 (0.97–4.21) | | | | | | |
| >52–165 h | 16/9 | 1.83 (0.81–4.14) | | | | | | |
| >165 h | 17/11 | 1.70 (0.79–3.67) | | | | | | |
| Digital | | | | | | | | |
| Total | 59/37 | 1.59 (1.05–2.41) | 32/36 | 0.86 (0.53–1.39) | 12/14 | 0.88 (0.40–1.90) | 112/99 | 1.13 (0.86–1.48) |
| >1–6-year latency | 52/34 | 1.51 (0.98–2.3) | 31/33 | 0.90 (0.55–1.48) | 9/13 | 0.69 (0.30–1.63) | 100/92 | 1.08 (0.81–1.43) |
| >6-year latency | 7/3 | 2.29 (0.59–8.93) | 1/3 | 0.30 (0.03–2.92) | 3/1 | 3.44 (0.35–34.0) | 12/7 | 1.71 (0.67–4.34) |
| ⩽43 h | 31/14 | 2.31 (1.22–4.39) | | | | | | |
| >43–342 h | 19/18 | 1.11 (0.57–2.14) | | | | | | |
| >342 h | 18/14 | 1.47 (0.72–3.02) | | | | | | |
| Cordless | | | | | | | | |
| Total | 55/37 | 1.46 (0.96–2.23) | 31/34 | 0.89 (0.55–1.46) | 11/11 | 0.96 (0.41–2.22) | 104/92 | 1.13 (0.85–1.50) |
| >1–6-year latency | 40/24 | 1.60 (0.96–2.67) | 16/27 | 0.59 (0.32–1.09) | 8/9 | 0.86 (0.33–2.23) | 68/69 | 0.97 (0.69–1.36) |
| >6-year latency | 15/13 | 1.16 (0.55–2.46) | 15/7 | 2.06 (0.83–5.09) | 3/2 | 1.43 (0.24–8.61) | 36/23 | 1.56 (0.92–2/63) |
| ⩽104 h | 14/16 | 0.94 (0.45–1.96) | | | | | | |
| >104–319 h | 18/14 | 1.34 (0.66–2.71) | | | | | | |
| >319 h | 30/14 | 2.15 (1.13–4.09) | | | | | | |

Numbers of discordant pairs with exposed case (Ca) or control (Co) are given. Matched analyses are adjusted for socio-economic status.

to microwaves occurs on the same side of the head as the phone is used. There is a rapid decline in dose and the other side of the brain is exposed to a lower degree. OR was calculated for ipsilateral, contralateral, or both ipsi- and contralateral exposure to microwaves from a mobile phone by combining data for both sides of the head. Interestingly, increased risk was found for tumours in the hemisphere with highest exposure to microwaves (ipsilateral) and no consistent risk was found for contralateral exposure. The highest risk was found for astrocytoma and ipsilateral use of an analogue phone.

Dose–effect calculations were made using hours for exposure among the controls divided in three equal groups. An effect was seen for cordless phones. This is of interest since cordless phones operate with a fairly similar effect. Regarding analogue and digital phones, exposure varies largely due to the localization of the antenna and different SAR values for different phones. Thus, cumulative exposure (h) seems not to

Table 2. Odds ratio (OR) and 95% confidence interval (CI) for astrocytoma.

| | Ipsilateral | | Contralateral | | Varying ipsi/contralateral | | All | |
|---|---|---|---|---|---|---|---|---|
| | Ca/Co | OR | Ca/Co | OR | Ca/Co | OR | Ca/Co | OR |
| Analogue | | | | | | | | |
| 450 MHz (ever) | 13/8 | 1.63 (0.67–3.94) | 8/9 | 0.87 (0.34–2.27) | 3/1 | 3.17 (0.33–30.9) | 24/20 | 1.20 (0.66–2.17) |
| 900 MHz (ever) | 30/12 | 2.50 (1.28–4.89) | 10/10 | 1.02 (0.42–2.46) | 3/5 | 0.56 (0.13–2.42) | 44/29 | 1.52 (0.95–2.43) |
| All (450+ 900 MHz) | 37/19 | 1.95 (1.12–3.39) | 14/17 | 0.81 (0.40–1.65) | 6/6 | 0.99 (0.31–3.11) | 58/45 | 1.29 (0.88–1.91) |
| >1–6-year latency | 18/7 | 2.66 (1.11–6.41) | 4/6 | 0.63 (0.18–2.26) | 3/3 | 1.04 (0.21–5.24) | 26/18 | 1.44 (0.79–2.63) |
| >6-year latency | 19/12 | 1.54 (0.74–3.18) | 10/11 | 0.91 (0.39–2.16) | 3/3 | 0.90 (0.18–4.59) | 32/27 | 1.17 (0.70–1.96) |
| ⩽52 h | 16/7 | 2.48 (1.01–6.11) | | | | | | |
| >52–149 h | 13/7 | 1.93 (0.76–4.85) | | | | | | |
| >149 h | 12/9 | 1.51 (0.62–3.69) | | | | | | |
| Digital | | | | | | | | |
| Total | 43/27 | 1.62 (0.99–2.63) | 41/42 | 0.87 (0.48–1.57) | 9/11 | 0.85 (0.35–2.06) | 80/71 | 1.13 (0.82–1.56) |
| >1–6-year latency | 37/24 | 1.54 (0.92–2.59) | 21/23 | 0.86 (0.47–1.58) | 6/10 | 0.61 (0.22–1.68) | 69/66 | 1.04 (0.73–1.46) |
| >6-year latency | 6/3 | 1.99 (0.49–8.07) | 1/1 | 1.00 (0.06–16.0) | 3/1 | 3.51 (0.35–35.2) | 11/5 | 2.26 (0.78–6.55) |
| ⩽43 h | 26/10 | 2.71 (1.29–5.70) | | | | | | |
| >43–360 h | 13/14 | 1.02 (0.47–2.22) | | | | | | |
| >360 h | 13/12 | 1.31 (0.58–2.97) | | | | | | |
| Cordless | | | | | | | | |
| Total | 46/27 | 1.66 (1.03–2.68) | 22/22 | 0.98 (0.54–1.78) | 7/7 | 0.94 (0.33–2.72) | 79/63 | 1.26 (0.90–1.76) |
| >1–6-year latency | 33/19 | 1.67 (0.94–2.95) | 10/16 | 0.60 (0.27–1.34) | 4/6 | 0.63 (0.17–2.24) | 49/47 | 1.03 (0.68–1.54) |
| >6-year latency | 13/8 | 1.59 (0.65–3.85) | 12/6 | 2.06 (0.76–5.58) | 3/1 | 2.83 (0.29–27.6) | 30/16 | 1.86 (1.01–3.41) |
| ⩽104 h | 12/12 | 1.05 (0.47–2.38) | | | | | | |
| >104–319 h | 13/11 | 1.31 (0.58–2.96) | | | | | | |
| >319 h | 26/9 | 2.85 (1.32–6.15) | | | | | | |

Numbers of discordant pairs with exposed case (Ca) or control (Co) are given. Matched analyses are adjusted for socio-economic status.

be the most appropriate method. Instead, SAR-hours would be preferable when calculating within the tumour area, but this was not possible due to the lack of SAR information for the different phones (cf. $\mu$Tesla-years for exposure to extremely low-frequency electromagnetic radiation) (Hardell *et al.* 1995). Note, however, that increasing duration of use (years) did not appear to be associated with increased risk (tables 1 and 2). Analysis of trend in ORs by duration of analogue phone use showed the highest risk for >10-year use in the total material, yielding OR= 3.20, 95% CI= 1.17–8.76, and for astrocytoma OR= 3.77, 95% CI= 1.05–13.5.

In a case-control study, it is always possible that cases report more exposure than controls. This could be the situation in a study on mobile phones and the risk for brain tumours. However, cordless phones have almost not been discussed in this context at all, so our findings of an increased risk for these may indicate that recall bias was not a major problem in the study. In addition, the finding of highest risk in the anatomical area with highest RF exposure argues

Table 3. Multivariate analysis of exposure in the whole study group.

| | Malignant | | | Astrocytoma | | |
|---|---|---|---|---|---|---|
| | Ca/Co | OR | CI | Ca/Co | OR | CI |
| Analogue | 79/70 | 1.11 | 0.80–1.54 | 58/45 | 1.26 | 0.84–1.87 |
| Digital | 112/99 | 1.09 | 0.83–1.45 | 80/71 | 1.03 | 0.73–1.45 |
| Cordless | 104/92 | 1.10 | 0.83–1.47 | 79/63 | 1.22 | 0.86–1.73 |

Numbers of discordant pairs with exposed case (Ca) or control (Co) are given.

against a recall bias. Patients usually do not have exact information of the tumour localization and the concepts of latency and dose–response effect are not well understood in the population.

Recall bias was analysed in the whole study group (Hardell *et al.* 2002). However, among cases who reported a previous cancer diagnosis, exposure frequency to cellular and cordless phones was lower than among those who did not report cancer. In the group of patients with a relative who had assisted in answering the questionnaire, the exposure frequency was lower that among the others. Only two cases in the whole study group (one with a malignant brain tumour, another with a benign tumour) expressed concern in the questionnaire about the use of a mobile phone. On the contrary, many other items were discussed by the 232 cases under the question 'Other information', such as other diseases, chemicals, extremely low-frequency electromagnetic fields, stress, ionizing radiation, heredity, etc.

Observational bias could have been introduced in the study during the supplementary telephone interviews. The answers in the questionnaire by cases and controls were compared with the final exposure category. The changes of exposure were similar among cases and controls, thus arguing against observational bias. Recall bias can, of course, never be completely excluded in a case-control study, but it seems in this study not to explain the results.

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