Cellular/Mobile Phone Use and Intracranial Tumours

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Background

Cell phone use is increasingly prevalent in Canada. Public concerns of a potential associated risk with intracranial tumours have been raised. Further, recent media attention has focused on disparate precautionary policies implemented across jurisdictions in Canada and world-wide. In light of this and emerging epidemiological evidence, we review what is known about cell phone use and intracranial tumours.

Intracranial tumours

In 2008, the Canadian standardized incidence rate for brain cancer is estimated to be 8 per 100,000 for males and 6 per 100,000 for females. These rates have not increased compared to 1980 levels; in fact among females a decline in incidence of 3.6% per year since 2000 has been observed[1].

Three types of intracranial tumours are discussed in this review. Glioma is a malignant tumour with a high case fatality rate; acoustic neuroma and meningioma are benign and can usually be treated with early diagnosis.

Meta-analyses

Three meta-analyses published since 2006 and two pooled INTERPHONE studies were reviewed (Tables 1 and 2). These studies pertain to intracranial tumours in adults only.

All three meta-analyses used a random effects model, which does not assume homogeneity of effects across the studies being pooled; however, Lahkola et al (2006) also used a fixed effect model (assumes homogeneity) for selected analyses[2]. Hardell et al (2008) provided little information on the methods used while Kan et al (2008) and Lahkola et al (2006) examined heterogeneity between studies, conducted sensitivity analyses and tested for publication bias; none was identified[3,4,2].

The two most recent meta-analyses (Hardell et al 2008, Kan et al 2008) were restricted to case-control studies and are based on the same set of articles with more recent publications added to Hardell et al[3,4]. The sole exception is the exclusion of Muscat et al (2000) by Hardell et al (2008) with the rationale that results were not presented separately for glioma, acoustic neuroma and meningioma[3]. Lahkola et al (2006) included several additional older studies; Johansen et al (2001), a Danish cohort study, and three studies by Hardell et al[2]. The latter were presumably not included in the other meta-analyses due to methodological considerations; these studies have been criticized for biases in participant recruiting, exposure assessment and unclear reporting.

Summary of meta-analysis results

- Meta-analyses based on ≥ 10 years duration of use have detected a slightly increased risk (OR: 1.25, 95%CI: 1.01-1.54) for all intracranial tumours (Kan et al 2008)[4]. Pooled analyses using shorter duration did not indicate an association (Lahkola et al 2006)[2].
- Restricting the analyses to ≥ 10 years and ipsilateral use (cell phone use on the same side as the tumour), the risk increased and was significantly associated for glioma (OR: 2.0, 95%CI: 1.2-3.4) and acoustic neuroma (OR: 2.4, 95%CI: 1.1-5.3, but not for meningioma (OR: 1.7, 95%CI: 0.99-3.1) (Hardell et al 2008)[3].

Interpretation of results

- There is insufficient evidence to indicate a causal association. The evidence is most suggestive for ipsilateral tumours occurring with ten or more years of use.
- Associations were only observed for regular cell phone use of ≥ 10 years. This exposure duration/latency is appropriate for cancer studies. Previous 'negative' studies may have failed to detect an effect due to an insufficient duration of exposure or latency period. The number of study subjects for which these exposure metrics are available is relatively small.

- Type of phone (analogue/digital) may influence this association. Earlier models (analogue phones) were likely used by long-term cell phone users. These phones emit greater radiation and may be associated with an increased risk of tumour development than the newer digital phones. Individual studies have examined the odds of using analogue phones but collectively no increased risk has been observed (Kan et al 2008, Lahkola et al 2006)[4,2].
- Years of exposure is just one exposure metric; the two most recent meta-analyses did not explore other indices of exposure, e.g., hours of cumulative use and cumulative number of calls, although this information has been reported in individual studies.
- The observed strengthening of the odds with ≥ 10 years and ipsilateral use increases the biological plausibility of the association; this suggests that the greatest risk is on the most exposed side of the brain.
- Results may indicate a true increase in risk with cell phone use; however, they may also indicate recall bias. Cases may have disproportionately recalled ipsilateral cell phone use if they believed it to be related to the development of the tumour; this is plausible given media attention to the issue. If true, this would inflate the calculated risk. This appears to have occurred in some individual studies where ipsilateral use significantly increases the risk whereas contralateral use provides a protective effect suggesting information bias (Hepworth et al 2006)[5].
- Other methodological constraints involving individual studies included in the meta-analysis may have influenced the overall results. For example, the use of proxy respondents, for deceased or very ill cases, may have introduced misclassification or bias in exposure assessment. This is of particular concern for glioma where there is a high case fatality proportion.
- Given the low and stable/declining Canadian brain cancer incidence rates, any increased risk attributable to cell phone use is very small.
- Existing research is limited to adults; little is known about potential risks to children. A large cohort study is underway to examine potential risks in this population group (Feyching 2006)[6].

Summary

There is insufficient evidence to indicate a causal association between cell phone use and intracranial tumours. There is weak evidence supporting an increase in odds of glioma, acoustic neuroma, and meningioma in adults with regular, ipsilateral use for 10 years or longer. Existing findings are suggestive but preliminary because they are based on few studies with small numbers and potential biases.

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Appendix 1: Summary of Meta-Analyses of Cellular Phone Use and Intracranial Tumours

Description of Meta-Analysis					Results of Meta-Analysis					
	Studies included in Meta-Analysis	**Study Design**	**Cases/ Controls**	**Study Period**	**Tumour Type**	**Exposure (years)**	**Digital/ Analog**	**Ipsilateral**	**Cases/ Controls**	**Odds Ratio (95% CI)**
Hardell et al. 2008	Schlehofer et al. 2007 Klaeboe et al. 2007 Hours et al. 2007 Lahkola et al. 2007 Takebayashi et al. 2000 Hardell et al. 2006 Schuz et al. 2006 Hepworth et al. 2006 Christensen et al. 2005 Schoemaker et al. 2005 Lonn et al. 2005 Christensen et al. 2004 Lonn et al. 2004 Auvinen et al. 2002 Inskip et al. 2001	All case - control studies		n/s	glioma	All	n/s	n/s		0.9(0.8-1.1)
					glioma	≥10	n/s	n/s	338 / 511	1.2(0.8-1.9)
					glioma	**≥10**	**n/s**	**ipsilateral**	**>133/>163**	**2.0(1.2-3.4)**
					glioma	≥10	n/s	contralateral	>104/>175	1.1(0.6-2.0)
					acoustic neuroma	All	n/s	n/s	824/4261	0.9(0.7-1.1)
					acoustic neuroma	≥10	n/s	n/s	83/355	1.3(0.6-2.8)
					acoustic neuroma	**≥10**	**n/s**	**ipsilateral**	**53/167**	**2.4(1.1-5.3)**
					acoustic neuroma	≥10	n/s	contralateral	30/151	1.2(0.7-2.2)
					meningioma	**All**	**n/s**	**n/s**	**870/2,331**	**0.8(0.7-0.99)**
					meningioma	≥10	n/s	n/s	61/152	1.3(0.9-1.8)
					meningioma	**≥10**	**n/s**	**ipsilateral**	**20/46**	**1.7(0.99-3.1)**
					meningioma	≥10	n/s	contralateral	15/52	1.0(0.3-3.1)
Kan et al. 2008	Schuz et al. 2006* Hepworth et al. 2006* Christensen et al. 2005* Lonn et al. 2005* Schoemaker et al. 2005 Lonn et al. 2004 Auvinen et al. 2002 Inskip et al. 2001 Muscat et al. 2000	All case-control studies	5259/ 12074	2000-2006	all intracranial tumours	n/s	n/s	n/s	10 studies	0.90(0.81-0.99)
					all intracranial tumours	**≥10**	**n/s**	**n/s**	**5 studies**	**1.25(1.01-1.54)**
					all intracranial tumours	n/s	digital	n/s		0.86(0.68-1.09)
					all intracranial tumours	n/s	analog	n/s		1.13(0.83-1.54)
					high-grade glioma	n/s	n/s	n/s	5 studies	0.86(0.70-1.05)
					low-grade glioma	n/s	n/s	n/s		1.14(0.91-1.43)
					acoustic neuroma	n/s	n/s	n/s	3 studies	0.96(0.83.10)
					meningioma	n/s	n/s	n/s	5 studies	0.64(0.56-0.74

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Lahkola et al. 2006	Hardell et al. 2006 Shoemaker et al. 2005 Christensen et al. 2005 Lonn et al. 2005 Muscat et al. 2002 Hardell et al. 2002 Auvinen et al. 2002 Johansen et al. 2001 Inskip et al. 2001 Muscat et al. 2000 Hardell et al. 1999	11 case-control studies and one cohort study	2780 cases	1966 - 2005	all intracranial tumours	ever to >5 yrs	n/s	n/s	12 studies	0.98(0.83-1.16)
					all intracranial tumours	**>1 yr**	**n/s**	**ipsilateral**	**8 studies**	**1.36(0.99-1.87)**
					all intracranial tumours	>1 yr	n/s	contralateral	5 studies	1.02(0.78-1.35)
					glioma	ever to >5 yrs	n/s	n/s	9 studies	0.96(0.78-1.18)
					acoustic neuroma	ever to >5 yrs	n/s	n/s	6 studies	1.07(0.89-1.30)
					meningiomas	ever to >5 yrs	n/s	n/s	8 studies	0.87(0.72-1.05)

Appendix 2: Pooled Interphone Studies

Description of Pooled Analysis						Results of Pooled Analysis			
Study	**Country**	**Cases/ Controls**	**Study Period**	**Age**	**Tumour Type**	**Exposure Definition**	**Cases / Controls**	**Odds Ratio (95% CI)**	**Comments**
Lahkola et al. 2007	Denmark Finland Norway Sweden England	1,521/ 3,301	2000-2004	18-69	glioma	Regular use	867/1853	0.78(0.68-0.91)	Increasing trend with years since first use on ipsilateral side (p=0.04)
						≥10 yrs since first use, contralateral	67/121	0.98(0.71,1.37)	
						≥10 yrs since first use, ipsilateral	**77/117**	**1.39(1.01-1.92)**	
Shoemaker et al. 2005	Denmark Finland Norway Sweden England	678/3553	1999-2004	18-69	acoustic neuroma	Regular use		0.9(0.7-1.1)	No association reported with increasing number years of use (p=0.7)
						≥10 yrs lifetime use, contralateral		0.9(0.5-1.8)	
						≥10 yrs lifetime use, ipsilateral		**1.8(1.1-3.1)**	

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