Summary of the literature: What do we know about cell phones and health?

- **Cell phones and other wireless devices emit electromagnetic fields (EMFs) that can penetrate the skin and the brain.**

  When a cell phone is held against one's ear, the head and brain can absorb low-frequency radiation, which can heat tissue. The Federal Communications Commission (FCC) sets standards to limit the maximum exposure received by the body due to the tissue absorption of radiation from cell phones. The Specific Absorption Rate (SAR) standard is intended to limit the heat generated by six minutes of exposure in a model of an adult male head, and does not take into account potential long-term damage from repeated close-range exposures, especially in children and adolescents (see below).

- **There is general concern about EMFs from multiple sources causing cancer.**

  Public concern has arisen over epidemiological studies showing associations between cancer and EMF exposure. The International Agency for Research on Cancer has classified extremely low frequency EMFs as "possibly carcinogenic to humans" based on associations of residential exposure to such EMFs (from household wiring and from proximity to overhead power lines) and increased risk of childhood leukemia.

- **A mechanism by which EMFs from cell phones could cause cancer has not been identified.**

  Non-ionizing radiation, such as EMFs from cell phones, by definition, does not have enough energy to cause the same kinds of gene mutations and tissue damage that ionizing radiation does. Evidence of DNA damage by EMFs from cell phones is mixed. While some researchers have reported biological changes associated with EMFs from cell phones, these studies have not been replicated.

- **Cell phone use is widespread and increasing**.

  Cell phone use is increasing in both the number of individuals who have a phone and the time that phones are in use. Cell phone usage in the United States has grown rapidly since the mid-1980s, with especially large annual increases in the 2000s. Americans spent a total of 2.2 trillion minutes on their mobile phones in 2008, up 100 billion minutes from the previous year, according to the Cellular Telecommunications and Internet Association. According to the recently issued President's Cancer Panel Report, cell phone use is also increasing among children.

- **There have been a number of studies on the health effects of cell phones that, in total, are inconclusive**.

  Over two dozen publications since 1999 have examined the potential relationship between cellular phone use and brain cancer. Because widespread cell phone use is a relatively recent phenomenon, most study participants had used cell phones for only a few years, which is an insufficient duration for cancer studies. The interval between exposure to any agent that can cause cancer and the clinical onset of a tumor (i.e., latency) may be many years or decades. Therefore, negative or null results from studies that do not allow for sufficient latency are uninformative at best, even if they have received considerable media attention. In most short-term studies of cell phones and brain tumors, the relative risk of cancer associated with ever having been a regular

mobile phone user was below 1.0[1], which may reflect participation bias or other methodological limitations.

- **However, epidemiologic evidence suggests that the risk of brain tumors, particularly gliomas occurring on the same side of the head as the cell phone is habitually held, may be increased after long-term use. (See Tables at the end of this document)**

  Several studies have been published recently that included participants for whom at least 10 years had elapsed since they first used a cell phone. However, the number of long-term users is small in individual studies, and therefore the latter do not have enough power to detect a statistically significant difference (with the exception of the Interphone study – see below). Five case-control studies have a combined total of fewer than 200 glioma cases among long-term users (Table 1). With relatively small numbers of study participants, the overall estimates of effect are quite variable, ranging from a decrease in risk of 52% (not statistically significant) to an increase of 170% (highly statistically significant). In a more limited analysis of two studies that examined ipsilateral use (i.e., usual use of the phone on the same side of the head where the tumor developed), the increased risk ranged from 60% to 440% (Table 1). In contrast, a longitudinal Danish study (with only 28 cases of brain and other nervous system cancers among long-term users) showed a 34% decrease in risk that was statistically significant (Table 2). The Danish study did not examine ipsilateral use, however, and included multiple tumor types, not just gliomas.

  Meta-analyses, which pool data from several studies to increase statistical power, have examined risks of glioma in long-term cell phone users. As expected, these meta-analyses show a smaller range of overall effect estimates among long-term users than the individual studies: a decrease in risk of 5% to an increase of 30% (Table 3). Focusing on ipsilateral cell phone use for at least 10 years, however, resulted in sharply higher pooled risk estimates (increases of 39% to 90%). The Interphone study, which is the largest case-control investigation to date (including subjects from Europe, Canada, Israel, Japan and New Zealand), also found an increased risk of 21% for long-term ipsilateral use, but it was not statistically significant. However, for ipsilateral use and cumulative call time in the Interphone study, the highest exposure category (more than 1640 cumulative hours, or at least 27 minutes/day over a 10-year period) was associated with a statistically significant 96% increase in risk of glioma (Table 4).

  Individual studies of acoustic neuromas (benign tumors that can affect hearing and balance and may cause partial facial paralysis) provide varied and inconclusive evidence of an increased risk (Table 5). The numbers of long-term and heavy users in individual studies are quite small (the largest study had only 20 acoustic neuroma cases among long-term users) and preclude any definitive conclusion about an association between cell phone use and the risk of acoustic neuroma. Meta-analyses show that for acoustic neuroma the effects of cell phone use among long-term (> 10 yr) users ranges from no change in risk to an increase of 60% for ipsilateral use (Table 6).

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[1] A relative risk (RR) is the risk of an outcome (developing a disease) among the exposed divided by the risk among an unexposed or less exposed group. A RR >1.0 means one is more likely to develop a disease if exposed than if unexposed. A RR < 1.0 means that one is less likely to develop a disease if exposed than if unexposed. A RR of 1.0 means there is no difference in risk between the exposed and unexposed populations.

- **There is very limited epidemiological evidence of an increased risk of parotid (salivary) gland tumors and cell phone use.**

  In a combined analysis of data from Sweden and Denmark (Lönn et al. 2006), a nonsignificantly increased risk of 160% for benign tumors was observed for ipsilateral use for 10 years or more, while a decreased risk of 70% was seen for contralateral use. In a study conducted in Israel (Sadetzki et al. 2006), where people tend to report substantially heavier use of mobile phones than elsewhere, the results suggest a relationship between heavy mobile phone use and risk of parotid gland tumors. Additional investigations of this association, with longer latency periods and large numbers of heavy users, are needed to confirm these findings.

- **Limited evidence suggests potential risk among long-term users of cordless phones.**

  Power output from cordless phones (average levels of 10 milliWatts (mW)) is similar to that from mobile phones (range of median output power 6–10 mW in cities) (Lönn et al. 2004a, 2004b). Two Swedish studies (Hardell et al. 2006b, 2006c) suggest potential risks of gliomas from cordless phones (Table 8). It is difficult to examine the effects of cordless phones and cell phones separately, however, as the majority of cordless phone users in recent studies also use mobile phones (Hardell et al. 2006, Schüz et al. 2006).

- **Limited evidence suggests that cell phone use may be potentially more hazardous for children and adolescents than for adults (Table 9).**

  EMFs can penetrate deeper into the developing brains of children and adolescents than into the brains of adults. Also, the brain is still developing into early adulthood, which may make children and adolescents more sensitive to EMF exposures. A couple of Swedish studies (Hardell et al. 2006b, 2006c) suggest increased risks of 150% to 170% for gliomas among those who first used a cell phone before age 20.

- **The age-adjusted incidence of brain tumors in the U.S, has not increased since the 1990s, according to a review of brain cancer trends from 1975-2007 in the Surveillance, Epidemiology and End Results (SEER) database (Altekruse et al. 2010). A review of California brain cancer statistics is in progress and should be completed by winter 2010.**

  The lack of an increasing trend in brain cancer incidence nationally may have multiple explanations: (i) the latency period is more than 10 years; (ii) the increased risk is too small to be observed; (iii) the increased risk is restricted to subgroups of mobile phone users; or (iv) there is no increased risk.

Tables Summarizing Studies of Cell Phone Use and Cancer

Table 1. Risk of *gliomas* [odds ratio (OR) (95% CI)] from case-control studies of more than 10 years of cell phone use.

		≥10 yrs latency			Ipsilateral use, ≥10 yrs latency		
Case Control	Grade of Glioma	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
Christensen et al. 2005	High-Grade[a]	6	9	1.64 (0.44-6.12)	-	-	-
Denmark	Low-Grade[b]	8	22	0.48 (0.19-1.26)	-	-	-
Lonn et al. 2005	All	25	38	0.9 (0.5-1.5)	15	18	1.6 (0.8-3.4)
Sweden							
Hardell et al. 2006c[c]	All	78	99	**2.7 (1.8-3.9)**	41	28	**4.4 (2.5-7.6)**
Sweden	High-Grade	7	99	1.5 (0.6-3.8)	2	28	1.2 (0.3-5.8)
	Low-Grade	71	99	**3.1 (2.0-4.6)**	39	28	**5.4 (3.0-9.6)**
Hepworth et al. 2006	All	66	112	0.90 (0.63-1.28)	-	-	**1.6 (1.02-1.52)**
UK							
Schuz et al. 2006a	All	12	11	2.2 (0.94-5.11)	-	-	**-**
Germany							

[a] High-grade gliomas are a class of fast-growing, aggressive tumors of the central nervous system.
[b] Low-grade gliomas are a class of slow-growing, less aggressive tumors of the central nervous system.
[c] Cases and controls of high-grade and low-grade gliomas were pooled in Hardell et al. 2009

Table 2. Risk of *brain and nervous system tumors* [standardized incidence ratio (SIR) (95%CI) from cohort study of more than 10 years of cell phone use.

	≥10 yrs latency				Ipsilateral use, ≥10 yrs latency		
Cohort	Cohort(N)	Cases (N)	Person Years	SIR (95% CI)	N	Person Years	SIR (95% CI)
Schuz et al. 2006b	56,648	28	169,595	**0.66 (0.44-0.95)**	-	-	-
Denmark							

Table 3. Risk of *gliomas* [odds ratio (OR) (95% CI)] from meta-analyses of case-control studies of more than 10 years of cell phone use.

	≥10 yrs latency			Ipsilateral use, ≥10 yrs latency		
Meta-Analysis	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
Lahkola et al. 2007[a]	143	220	0.95 (0.74-1.23)	77	117	**1.39 (1.01-1.92)**
Hardell et al. 2009, Khurana et al. 2009[b]	223	330	**1.3 (1.1-1.6)**	118	145	**1.9 (1.4-2.4)**
The Interphone Study Group 2010 [c]	252	232	0.98 (0.76-1.26)	108	82	1.21 (0.82-1.80)

[a] Pooled analysis of data from persons in Finland, Norway, Denmark (Christenson et al. 2005), Sweden (Lonn et al. 2005), UK (Hepworth et al. 2006)
[b] Pooled analysis of data from persons in the Nordic meta-analysis (Lahkola et al. 2007), persons in Germany (Schuz et al. 2006a), and persons in Sweden (Hardell et al. 2006a). Both Hardell et al. 2009 and Khurana et al. 2009 pooled the same data and obtained essentially identical results
[c] Pooled analysis of data from persons aged 20-59 years in 16 study centers from Australia, Canada, France, Israel, Italy, Japan, New Zealand, Germany (Schuz et al. 2006a), Finland, Norway, Denmark (Christenson et al. 2005), Sweden (Lonn et al. 2005) and the UK (Hepworth et al. 2006)

Table 4. Risk of *gliomas* [odds ratio (OR) (95% CI)] from meta-analyses of case-control studies of heavy cell phone use.

	>1640 hours			Ipsilateral use, >1640 hours		
Meta-Analysis	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
The Interphone Study Group 2010 [a]	210	154	**1.40 (1.03-1.89)**	100	62	**1.96 (1.22-3.16)**

[a] Pooled analysis of data from persons aged 20-59 years in 16 study centers from Australia, Canada, France, Israel, Italy, Japan, New Zealand, Germany (Schuz et al. 2006a), Finland, Norway, Denmark (Christenson et al. 2005), Sweden (Lonn et al. 2005) and the UK (Hepworth et al. 2006)

Table 5. Risk of *acoustic neuroma* [odds ratio (OR) (95% CI)] from case-control studies of more than 10 years of cell phone use.

	≥10 yrs latency			Ipsilateral use, ≥10 yrs latency		
Case Control	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
Christensen et al. 2004 Denmark	2	15	0.22 (0.04-1.11)	-	-	-
Lonn et al. 2004 Sweden	14	29	1.9 (0.9-4.1)	12	15	**3.9 (1.6-9.5)**
Hardell et al. 2006b Sweden	20	99	**2.9 (1.6-5.5)**	10	28	**3.5 (1.5-7.8)**

Table 6. Risk of *acoustic neuroma* [odds ratio (OR) (95% CI)] from meta-analysis of case-control studies of more than 10 years of cell phone use.

	≥10 yrs latency			Ipsilateral use, ≥10 yrs latency		
Meta-Analysis	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
Schoemaker et al. 2005[a]	47	212	1.0 (0.7-1.5)	31	124	1.3 (0.8-2.0)
Hardell et al 2009, Khurana et al. 2009 [b]	67	311	1.3 (0.97-1.9)	41	152	**1.6 (1.1-2.4)**

[a] Pooled analysis of data from persons in Finland, Scotland, England, Sweden (Lonn et al. 2004), Denmark (Christenson et al. 2004), Norway (Klaeboe et al. 2007)

[b] Pooled analysis of data from persons in the Nordic meta-analysis (Schoemaker et al. 2005) and persons in an additional study in Sweden (Hardell et al. 2006b). Hardell et al. 2009 and Khurana et al. 2009 pooled the same data and obtained essentially identical results.

Table 7. Risk of *tumors, all sites,* [odds ratio (OR) (95% CI)] from a meta-analysis of case-control studies of more than 10 years of cell phone use.

	≥10 yrs latency			Ipsilateral use, ≥10 yrs latency		
Meta-Analysis	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
Myung et al. 2009[a]	-[b]	-[b]	**1.18 (1.04 – 1.34)**	-	-	-

[a] Pooled analysis of data on various brain tumors (Hardell et al. 1999, Hardell et al. 2002, Hardell et al. 2005a, Schoemaker et al. 2005, Hardell et al. 2006a, Schuz et al. 2006a, Lahkola et al. 2007, Lahkola et al. 2008), salivary/parotid gland tumors (Hardell et al. 2004, Lonn et al. 2006, Sadetzki et al. 2008), non-Hodgkins lymphoma (Hardell et al. 2005b), testicular cancer (Hardell et al. 2007)

[b] Numbers not given, meta analysis combined results from 13 studies.

Table 8. Risk of *gliomas* and *acoustic neuromas* [odds ratio (OR) (95% CI)] in case-control-studies of more than 10 years of cordless phone use.

		Ever Used Cordless Phone			Ipsilateral use, ≥10 yrs latency		
Case Control	Brain Tumor Type	Cases (N)	Controls (N)	OR (95% CI)	Cases (N)	Controls (N)	OR (95% CI)
Hardell et al. 2006c	High-Grade Glioma	539	2162	**1.5 (1.1–1.9)**	23	45	**2.2 (1.3-3.9)**
	Low-Grade Glioma	124	2162	1.4 (0.9-3.4)	5	45	1.6 (0.5-4.6)
Hardell et al. 2006b	Acoustic Neuroma	243	2162	**1.5 (1.04-2.0)**	4	45	1.0 (0.3-2.9)

Table 9. Risk of *gliomas* and *acoustic neuromas* [odds ratio (OR) (95% CI)] in case-control studies in which age of first cordless phone use and/or cell phone use was <20 years.

			<20 years old at first use		
Case Control	Brain Tumor Type	Phone Type	Cases (N)	Controls (N)	OR (95% CI)
Hardell et al. 2006c[a]	Malignant Brain Tumors	Cell Phone	13	15	**2.5 (1.1-5.9)**
		Cordless Phone	4	16	0.6 (0.2-1.9)
Hardell et al. 2006b[a]	Benign Brain Tumors	Cell Phone	20	15	**2.7 (1.3-6.0)**
		Cordless Phone	17	16	2.1 (0.97-4.6)

[a] Participants reporting use of analog and/or digital cell phones were pooled in Hardell et al. 2009

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