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High Probability Radio Frequency Radiation Causes Brain Tumors. Expert Report

By Dr. Christopher J. Portier
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Region: USA
Theme: Intelligence, Science and Medicine

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Christopher J. Portier, Ph.D., former director of the National Center for Environmental Health at the Centers for Disease Control and Prevention (CDC) and the Agency for Toxic Substances and Disease Registry (ATSDR), and a scientific advisor for the World Health Organization (WHO), recently completed an expert report on brain tumor risk from exposure to radio frequency (RF) radiation used in cellphone technology.

After completing a comprehensive review of the scientific literature, Dr. Portier concluded:

> "In my opinion, RF exposure probably causes gliomas and neuromas and, given the human, animal and experimental evidence, I assert that, to a reasonable degree of scientific certainty, the probability that RF exposure causes gliomas and neuromas is high."

In 2011, Dr. Portier was selected to represent the CDC on an expert working group convened by the WHO International Agency for Research on Cancer (IARC) to review the carcinogenicity of RF radiation. Based upon recommendations of the expert panel, the IARC declared RF radiation "possibly carcinogenic to humans" (Group 2B) and the following year issued a **monograph** summarizing the evidence. Because the preponderance of the peer-reviewed research published since 2011 supports the need to upgrade this classification, the IARC has prioritized a **new review** to be conducted by 2024.

Dr. Portier's 176-page expert report including 443 references was prepared for the plaintiffs in a major product liability **lawsuit,** Murray et al. v Motorola, Inc. et al., filed in the Superior Court for the District of Columbia against the telecommunications industry. The report appears as Exhibit 3 in a recent filing with the Court.

Christopher J. Portier. Expert Report. Exhibit C. Murray et al. v. Motorola, Inc. et al. Superior Court for the District of Columbia. March 1, 2021. pp. 1-176.

The report can be downloaded here.

Summary Statements from the Expert Report

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4.1.5 Conclusions for Gliomas (p. 51)

> "The evidence on an association between cellular phone use and the risk of glioma in adults is quite strong. While there is considerable difference from study to study on ever versus never usage of cellular phones, 5 of the 6 meta-analyses in Figure 1. are positive and two are significantly positive. Once you consider latency, the meta-analyses in Figure 2 clearly demonstrate an increasing risk with increasing latency. The exposure response meta-regressions in Table 10 and Table 11 clearly indicate that risk is increasing with cumulative hours of exposure, especially in the highest exposure groups. There is a strong tendency toward gliomas appearing on the same side of the head as the phone is generally used and the temporal lobe is strongly suggested as a target. These findings do not appear to be due to chance. The cohort studies appear to show less of a risk than the case-control studies, but one study is likely to be severely impacted by differential exposure misclassification (Frei et al., 2007) and the other (Benson et al., 2012) is likely to have a milder differential exposure misclassification. The case-control studies are possibly impacted by recall bias although that issue has been examined in a number of different evaluations. Selection bias could have been an issue for the Interphone study, but their alternative analysis using different referent groups reduces that concern. Confounding is not an issue here. In conclusion, an association has been established between the use of cellular telephones and the risk of gliomas and chance, bias and confounding are unlikely to have driven this finding. The ecological studies are of insufficient strength and quality to fully negate the findings from the observational studies.
>
> The data in children is insufficient to draw any conclusions."

Table 3: Results from epidemiology studies for duration (cumulative hours) of use of a cellular telephone and the risk of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Cumulative use	Exposed Cases	OR (95% CI)	P Trend	Comparison group
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Glioma	<13 hours	55	0.8 (0.4-1.4)	ND	Any use
					13-100 hours	58	0.7 (0.4-1.3)		2+ calls/w
					>100 hours	54	0.9 (0.5-1.6)		
					>500 hours	27	0.5 (0.2-1.3)		
Spinelli et al. (2009)	CC	2005, France	≥18, Both	Glioma	≤48 (converted from hour years)	8	0.86 (0.3-2.44)	ND	Used a phone, cumulative use based upon subscription limits of hours/month
					48-432	58	1.45 (0.75-2.80)		
					≥432	13	1.07 (0.41-2.82)		
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Glioma	<5 hours	141	0.70 (0.52-0.94)		Avg 1 call per week for 6 mo (lag 1 yr), no hands-free
					5-12.9 hours	145	0.71 (0.53-0.94)		
					13-30.9 hours	189	1.05 (0.79-1.38)		
					31-60.9 hours	144	0.74 (0.55-0.98)		
					61-114.9 hours	171	0.81 (0.61-1.08)		
					115-199.9 hours	160	0.73 (0.54-0.98)		
					200-359.9 hours	158	0.76 (0.57-1.01)		
					360-734.9 hours	189	0.82 (0.62-1.08)		
					735-1639.9 hours	159	0.71 (0.53-0.96)		
					≥1640 hours	210	1.40 (1.03-1.89)		
					Using<5 hours referent				
					5-12.9 hours	92	0.88 (0.56-1.39)		Restricted to ever regular users
					13-30.9 hours	127	1.37 (0.87-2.14)		
					31-60.9 hours	108	1.13 (0.72-1.77)		
					61-114.9 hours	121	1.06 (0.68-1.67)		
					115-199.9 hours	129	1.13 (0.71-1.78)		
					200-359.9 hours	116	1.00 (0.63-1.58)		
					360-734.9 hours	142	1.17 (0.74-1.84)		
					735-1639.9 hours	126	1.09 (0.69-1.72)		
					≥1640 hours	160	1.82 (1.15-2.89)		
Coureau et al. (2014)	CC	2004-2006, France	≥16, Both	Glioma	<43	24	0.83 (0.48-1.44)	0.02	Avg 1 call per week for 6 mo
					43-112	20	0.77 (0.42-1.41)		
					113-338	28	1.07 (0.60-1.90)		
					339-895	28	1.78 (0.98-3.24)		
					≥896	24	2.89 (1.41-5.93)		
					Exclude proxies (weighted)				
					<29	19	0.73 (0.39-1.35)	0.03	Weighted for shared use and hands-free use
					29-86	20	0.97 (0.52-1.78)		
					87-326	31	1.56 (0.86-2.83)		
					377-835	22	1.62 (0.84-3.14)		
					≥836	18	2.83 (1.30-6.27)		
Hardell et al. (2015)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Glioma	Per 100 cumulative hours of use	NA	1.013 (1.009-1.017		>1 year
					Cumulative use	340		<0.0001	
					1-122	198	1.3 (1.05-1.5)		
					123-511	179	1.3 (1.02-1.6)		
					512-1486	228	1.4 (1.04-1.8)		
					>1486		2.2 (1.7-2.9]		
Yoon et al. (2015)	CC	2002-2007, Korea	15-69	Glioma	<300	97	1.25 (0.64-2.45)	ND	>1 year (maybe also non-regular user)
					300-900	70	1.59 (0.72-3.21)		
					>900	70	0.64 (0.30-1.34)		
					Excluding proxies				
					<300	73	0.99 (0.46-2.12)		
					300-900	61	1.17 (0.53-2.57)		
					>900	55	0.62 (0.27-1.43)		

4.2.5 Conclusions for Acoustic Neuromas (p. 72)

“The evidence on an association between cellular phone use and the risk of acoustic neuromas [ANs] in adults is strong. While there is considerable difference from study to study on ever versus never usage of cellular phones, 3 of the 4 meta-analyses in Figure 3 are above 1 although none-significantly. The meta-analyses in Figure 4 demonstrate an increased risk in the highest 2 latency groups for the case-control studies that gets slightly higher when the cohort studies are added. For latency >=5 years, the mRRs are significantly elevated for the case-control studies and the combined case-control and cohort studies. The exposure response meta-regressions in Table 19 indicates that risk is increasing with cumulative hours of exposure, especially in the highest exposure groups. This finding, however, is sensitive to the inclusion of the Hardell et al. (2013) [160] study. There is a strong tendency toward ANs appearing on the same side of the head as the phone is generally used, especially as the exposure increases. These findings do not appear to be due to chance. The cohort studies appear to show less of a risk than the case-control studies, but one study is likely to be severely impacted by differential exposure misclassification (Schuz et al. (2011) [99]) and the other (Benson et al. (2013) [102]) is likely to have a milder differential exposure misclassification. Both studies have very few cases. The case-control studies are possibly impacted by recall bias and this cannot be ruled out for the ANs. Selection bias could have been an issue for Interphone (2010) [67], and, unlike their analysis of the glioma data, they have not looked at an alternate referent population for their analyses of AN. Confounding is not an issue here. In conclusion, an association has been established between the use of cellular telephones and the risk of ANs and chance and confounding are unlikely to have driven this finding. Potential recall bias and selection bias may still be an issue with some of these findings.”

Table 14: Results from epidemiology studies for duration (cumulative hours) of use of a cellular telephone and the risk of acoustic neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Cumulative use	Exposed Cases	OR (95% CI)	P Trend	Comparison group
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic neuroma	<13 hours	5	0.7 (0.2-2.3)	ND	Any use
					13-100 hours	8	1.2 (0.5-3.1)		2+ calls/w
					>100 hours	9	1.4 (0.6-3.5)		
					>500 hours	1	0.4 (0.0-3.3)		
Muscat et al. (2002)	CC	1997-1999, New York City	≥18, Both	Acoustic neuroma	1-60 hours	9	0.9 (0.3-3.1)	0.53	Referent was asked if they were a regular
					>60 hours	9	0.7 (0.2-2.6)		user
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Acoustic neuroma	1-year lag				Avg 1 call per week for 6, no hands-free
					<5 hours	58	0.77 (0.52-1.15)		
					5-12.9 hours	63	0.80 (0.54-1.18)		
					13-30.9 hours	80	1.04 (0.71-1.52)		
					31-60.9 hours	66	0.95 (0.63-1.42)		
					61-114.9 hours	74	0.96 (0.66-1.41)		
					115-199.9 hours	68	0.96 (0.65-1.42)		
					200-359.9 hours	50	0.60 (0.39-0.91)		
					360-734.9 hours	58	0.72 (0.48-1.09)		
					735-1639.9 hours	49	0.48(0.30-0.78)		
					≥1640 hours	77	1.32 (0.88-1.97)		
					5-year lag				
					<5 hours	42	1.07 (0.69-1.68)		
					5-12.9 hours	30	1.06 (0.60-1.87)		
					13-30.9 hours	40	1.32 (0.80-2.19)		
					31-60.9 hours	36	0.86 (0.52-1.41)		
					61-114.9 hours	21	0.63 (0.35-1.13)		
					115-199.9 hours	22	0.71 (0.39-1.29)		
					200-359.9 hours	29	0.83 (0.48-1.46)		
					360-734.9 hours	26	0.74 (0.42-1.28)		
					735-1639.9 hours	22	0.60 (0.34-1.06)		
					≥1640 hours	36	2.79 (1.51-5.16)		
Pettersson et al. (2014)	Case-Control	Sweden	20-69, Both	Acoustic Neuroma	<38	70	1.09 (0.73-1.62)		Avg 1 call per week for 6 mo (lag 1 yr),
					38-189	73	1.12 (0.74-1.69)		weighted hands-free
					190-679	66	1.13 (0.75-1.70)		
					≥680	89	1.46 (0.98-2.17)		
					Histologically confirmed				
					<38	30	0.97 (0.55-1.71)		
					38-189	39	0.91 (0.51-1.60)		
					190-679	34	1.03 (0.57-1.87)		
					≥680	37	1.14 (0.63-2.07)		
Hardell et al. (2013)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Acoustic Neuroma	Per 100 cumulative hours of use	NA	1.009 (1.001-1.017)		>1 year
					Quartiles				
					1-122 hours	91	1.6 (1.1-2.2)	0.052	
					123-511 hours	37	1.5 (0.9-2.3)		
					512-1,486 hours	42	2.4 (1.5-3.8)		
					>1,486 hours	30	2.6 (1.5-4.4)		

5.5. Summary and Conclusions for Laboratory Cancer Studies (p. 86-88)

“The central question to ask of animal cancer studies is “Can RF increase the incidence of tumors in laboratory animals?” The answer, with high confidence, is yes. Table 20 summarizes the findings from the chronic exposure carcinogenicity studies for RF.

For rats, the NTP (2018) [177] chronic exposure bioassay in male Sprague-Dawley rats, including in-utero exposure, is clearly positive for acoustic neuromas of the heart, malignant

gliomas of the brain and pheochromocytomas of the adrenal gland. These findings are further supported by the presence of preneoplastic lesions and tissue toxicity in the heart, brain glial cells and adrenal glands. The less convincing findings in the study by Falcioni et al. (2018) [178] of heart acoustic neuromas in male Sprague-Dawley rats and a marginal increase in malignant gliomas in females provides additional support for this finding….

In conclusion, there is sufficient evidence from these laboratory studies to conclude that RF can cause tumors in experimental animals with strong findings for gliomas, heart Schwannomas and adrenal pheochromocytomas in male rats and harderian gland tumors in male mice and uterine polyps in female mice. There is also some evidence supporting liver tumors and lung tumors in male and possibly female mice."

6. Mechanisms Related to Carcinogenicity (p. 91)

> "There is sufficient evidence to suggest that both oxidative stress and genotoxicity are caused by exposure to RF and that these mechanisms could be the reason why RF can induce cancer in humans."

7. Summary of Bradford Hill Evaluations (p. 109)

> "RF exposure probably causes gliomas and acoustic neuromas, and given the human, animal and experimental evidence, I assert that, to a reasonable degree of scientific certainty, the probability that RF exposure causes these cancers is high."

Table 22: Summary conclusion for Hill's nine aspects of epidemiological data and related science (p. 110-111)

Table 22: Summary conclusions for Hill's nine aspects of epidemiological data and related science

Aspect	Conclusion	Reason
Consistency of the observed association	Strong	Multiple studies, many are positive, meta-analyses with little heterogeneity show positive findings at higher exposures, different research teams, different continents, different questionnaires, no obvious bias in case-control studies, no obvious confounding, laterality is significant
Strength of the observed association	Strong	Significant meta-analyses

Biological plausibility	Very Strong	Multiple cancers in multiple species, same tumors as humans in male rats, not due to chance, increased risk of rare tumors, convincing evidence for genotoxicity and oxidative stress
Biological gradient	Strong	Clearly seen in some case-control studies, clearly seen in the meta-analyses and met-regressions, not seen in the cohort studies, clearly seen in animal studies
Temporal relationship of the observed association	Satisfied	Exposure clearly came before cancers
Specificity of the observed association	Strong	The only cancers linked to RF exposure are gliomas and acoustic neuromas
Coherence	Strong	Cancers seen in the rats have strong similarity to human gliomas and acoustic neuromas, laterality and brain location support coherence
Evidence from human experimentation	No data	No studies are available
Analogy	No data	No studies available in the literature

Final Conclusion (p. 111)

> "In my opinion, RF exposure probably causes gliomas and neuromas and, given the human, animal and experimental evidence, I assert that, to a reasonable degree of scientific certainty, the probability that RF exposure causes gliomas and neuromas is high."

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