

# High Probability Radio Frequency Radiation Causes Brain Tumors. Expert Report

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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**



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

## 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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