

**Shawn E. Abrell**, WSB No. 41054, *Pro Hac Vice*  
4614 SW Kelly Avenue, Suite 200, Portland, Oregon 97239  
Tel.: 503.224.3018; Fax: 503.222.0693  
E-Mail: shawn.e.abrell@gmail.com  
*Lead Counsel for Plaintiffs*

**Tyl W. Bakker**, OSB No. 90200  
621 SW Alder, Suite 621, Portland, Oregon 97205  
Tel.: 503.244.4157; Fax: 503.220.1913  
E-Mail: tylbakker@gmail.com  
*Local Counsel for Plaintiffs*

**United States District Court**

**District of Oregon**

**Portland Division**

**AHM**, by and through  
her Guardian *ad litem* and father,  
David Mark Morrison, and  
**David Mark Morrison**, individually,

v.

**Portland Public Schools**,

Defendant.

Civil Action No. 3:11-cv-00739-MO

**Declaration of  
L. Lloyd Morgan  
Addendum I –  
Mobile Phone Use and Brain Tumors  
in Children and Adolescents**

## Re: Mobile Phone Use and Brain Tumors in Children and Adolescents: A Multicenter Case–Control Study

The data of Aydin et al. (1) contradict the authors’ conclusion that there is no association between brain tumor risk and cellphone use in children and adolescents. In addition, the study has multiple data discrepancies, and there are methodological problems in the laterality analysis.

The authors contradicted themselves when they concluded, “The absence of an exposure–response relationship ... in terms of the amount of mobile phone use ....” Their results indicated a clear exposure-response relationship and found significantly increased OR of brain cancer: 2.8 years after the first subscription for a cellphone began, using operator data, OR=2.15, 95% CI=1.07 to 4.29, combined with  $P_{\text{trend}} = .001$ , They also reported a greater than 3-fold risk OR=3.74, 95% CI=1.19 to 11.77, with >4 year duration of subscription, and nearly 3-fold increased risk for >2638 cumulative calls, OR=2.91, 95% CI=1.09-7.76. If one considers latency time as the interval between first use of a cellphone to diagnosis of brain cancer, these positive results suggest that children and adolescents may have a shorter latency time for the development of brain cancer than adults.

There are various instances in which the data are discrepant and have methodological problems. .

Table 4’s “Operator recorded use” (group 1) was from phone company records. . The “Self-reported use” (group 2) has the identical number of case and control subjects, but when case and control subjects are compared by use categories, the numbers of cases and controls differ. Table 1 reports these differences.

Billing / Memory	Exposure†	Cases	Controls	OR (95% CI)	$P_{\text{trend}}$
------------------	-----------	-------	----------	-------------	--------------------

Billing	Never	134	259	1.00	
Memory		127	245	1.00	
Billing	≤1.8 years	19	51	0.78 (0.43-1.40)	0.001*
Memory		33	62	1.09 (0.65-1.84)	0.25*
Billing	1.8-3.3 years	19	25	1.71 (0.85-3.55)	
Memory		17	25	1.47 (0.69-3.14)	
Billing	>2.8 years	24	25	2.15 (1.07-4.29)	
Memory		19	28	1.51 (0.68-3.35)	
<b>Billing Totals</b>		<b>196</b>	<b>360</b>		
<b>Memory Totals</b>		<b>196</b>	<b>360</b>		

† Years since first subscription

\*  $P_{\text{trend}}$  applied for ≤1.8, 1.8-3.3, and >2.8 years

**Table 1.** Comparison of Operator use data (Billing) with Self-reported use (Memory)

There are also discrepancies between the text and tables. Thus, the text of the paper states that operator data were available for 35% and 34% of the case and controls subjects respectively, while operator data reported Table 4 include 56.7% of the case and control subjects.

Table 5 has 3 categories “Ipsilateral use,” “Contralateral use”, and “Central or unknown location.” Ipsilateral use was defined as use “predominately on the same side as the tumor or on both sides of the head.” Contralateral use was defined as use “mostly on the side opposite to the tumor.” “No laterality was assigned if the tumor was centrally located” and there was no explanation about “unknown location.”

An analysis was performed for ipsilateral and contralateral risk, but the use of ambiguous and asymmetrically applied terms, “predominately” and “mostly,” the results of this analysis are

unclear. Also, assuming unknown location referred to the lack of identification of the tumor locations it was not possible to determine the proportion of these tumors that was ipsilateral, contralateral or centrally located and hence should have been excluded from the analysis.

The authors' definition of ipsilateral and contralateral use differed from those employed in all previous cellphone studies, as summarized in Table 1, and also differed from a dictionary definition (2).

Source	Exposure		Comments
	Ipsilateral	Contralateral	
Dictionary (2)	Situated or appearing on same side of body	Occurring or acting in conjunction with a part of the body	Does not deal with proportionality
Aydin et al. (1)	Use predominantly on same side of tumor or use on both sides	Use mostly on opposite side from tumor	Predominantly and mostly not defined. Asymmetrical definitions
Hutter et al. (3)	Exposure weighted as fraction of ipsilateral use.	Exposure weighted as fraction of contralateral use	Ideal (though subject to recall bias)
Inskip Method. (4)	$RR=(\sqrt{OR_{ipsi}+1})\div 2$	$RR=(\sqrt{OR_{contra}+1})\div 2$	Case subjects only. Ipsilateral & contralateral ORs are not defined.
Lönn et al. (5)	Same side of head as tumor or both sides of head	Opposite side of head as tumor or both sides of head [Table 3 footnote]	Underestimates true ipsilateral risk and overestimates true contralateral risk.
Hardell et al. (6)	>50% use on same side of head as tumor	<50% use on opposite side of head as tumor	Underestimates true ipsilateral risk and overestimate true contralateral risk

**Table 2.** Various study definitions of ipsilateral and contralateral use of cellphones with a dictionary definition.

It is likely these data discrepancies and methodological problems substantially contributed to the illogical findings in Table 5. Table 5 indicated that 12 of the 13 risks were higher for contralateral than ipsilateral use; that risks for “Central or unknown location tumors” were all

protective; and that 6 of 10 of these protective findings were either statistically significant, or borderline statistically significant ( $p < 0.09$ ). In addition, 3 of 4  $P_{\text{trend}}$  values indicated that statistically significant protection increased as exposure increased.

The authors reported 423 eligible case subjects and 909 eligible control subjects, with participation by 352 case subjects (83.2%) and 646 control subjects (71.0%) resulting in exclusion of 71 case subjects and 263 control subjects. However, when the reasons for exclusion were given, there were 72 case subjects (1 additional) and 280 control subjects (17 additional) that were excluded. This would result in case participation of 74% and control participation of 69%, which in turn would likely increase differential bias.

Other data discrepancies of Aydin et al are excluding of *the most common childhood brain tumor*, pilocytic astrocytoma (histology code, 9421), while including ependymoma, glioma malignant NOS, and medulloblastoma (PNET). In the United States the incidence of each of these tumors has been found to decrease with increasing age (7). Commenting on the implications of these declining rates with age, Dr. Michael Kundi, published his finding (16 months prior to Aydin et al) that, "... brain tumours with no or decreasing incidence trends for increasing age must be omitted from analysis, at least for short exposure durations." (8).

The Funding and Notes section reported several cellphone companies provided funding for this study, but there were no declarations regarding individual author's conflicts-of-interest (e.g., consulting, stock ownership, director status, etc.), nor was conflict of interest reported for the authors of the accompanying editorial (9)

The numerous discrepancies suggest a poor peer-review process and/or a rush to publish. Despite the discrepancies in the report by these authors, and contrary to the accompanying Guest Editorial in this same issue,

the findings of Aydin et al are supportive of a positive relationship between cellphone use in children and increased risk for brain tumors with shorter latency than those that have been found for adults. Further study is clearly merited on this important issue.

L. LLOYD MORGAN<sup>1</sup>  
RONALD B. HERBERMAN<sup>1,2</sup>  
ALASDAIR PHILIPS<sup>3</sup>  
DEVRA LEE DAVIS<sup>1</sup>

## Notes

**Affiliations of authors:** <sup>1</sup>Environmental Health Trust; <sup>2</sup>Intrexon Corporation; <sup>3</sup>Powerwatch

**Correspondence to:** L. Lloyd Morgan, B.S., 2022 Francisco Street, Berkeley, CA 94709 (email: Lloyd.L.Morgan@gmail.com).

## References

1. Aydin D, Feychting M, Schüz J, Tynes T, Andersen TV, Schmidt LS, Poulsen AH, Johansen C, Prochazka M, Lanngren B, Klæboe L, Eggen T, Jenni D, Grotzer M, Von der Weid N, Kuehn C, Röösl M. Mobile Phone Use and Brain Tumors in Children and Adolescents: A Multicenter Case–Control Study. *J Natl Cancer Inst.* 2011;103(16):1–13.
2. Merriam Webster's Collegiate Dictionary, 10<sup>th</sup> Edition, 1993, Merriam-Webster Inc. Springfield MA USA.
3. Hutter H-P, Moshhammer H, Wallner P, Cartellieri M, Denk-Linnert D-M, Katzinger M, Kundi M. Tinnitus and mobile phone use. *Occup Environ Med.* 2010 Dec;67(12):804-8. Epub 2010 Jun 23
4. Inskip P, Tarone RE, Hatch EE, Wilcosky TC, Shapiro WR, Selker RG, Fine HA, Black PM, Loeffler JS, Linet MS. Cellular-Telephone Use and Brain Tumors. *NEJM* 244(2) 79-86. January 11, 2001.
5. Lönn S, Ahlbom A, Hall P, Feychting M. Mobile Phone Use and the Risk of Acoustic Neuroma. *Epidemiology* 2004;15(6): 653–659.
6. Hardell L, Carlberg M, Hansson Mild K, Pooled analysis of two case-control studies on the use of cellular and cordless telephones and the risk of benign brain tumours diagnosed during 1997-2003. *Int J Oncol* 2006; 28(2): 509-518.

7. CBTRUS (2011) CBTRUS Statistical Report: Primary Brain and Central Nervous System Tumors in the United States in 2004-2007. Source: Central Brain Tumor Registry of the United States, Hinsdale, IL, USA ([www.cbtrus.org](http://www.cbtrus.org))
8. Kundi M. Essential problems in the interpretation of epidemiologic evidence for an association between mobile phone use and brain tumour. C. R. Physique 11 (2010) 556–563
9. Boice JD and Tarone RE. Cell Phones, Cancer, and Children. J Natl Cancer Inst. 2011 Aug 17;103(16):1211-3. Epub 2011 Jul 27..