

Update to Expert Report on RF-EMF

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1.0 Introduction

This is a review of new literature on the effects of radiofrequency electric and magnetic fields (RF-EMF or RF) since 2021 (epidemiology and cancer studies in animals) or 2023 (mechanistic studies). The report is intended to upgrade the previous report [1] on this topic.

2.0 New Epidemiology

2.1 Studies in Children and Adolescents

Castano-Vinyals et al. (2022) [2] reported on a case-control study (MOBI-kids) conducted in 14 countries (Australia, Austria, Canada, France, Germany, Greece, India, Israel, Italy, Japan, Korea, the Netherlands, New Zealand, Spain) to determine if wireless phone use was associated with

1 increased brain tumor risk in young people. Roughly half of the 899 cases came from Spain
2 (208), Italy (160) and France (102). The study used matched hospital controls (appendicitis
3 patients) and, although the study protocol called for 2 cases per control, the authors had too
4 many cases for which controls could not be found so they used post-hoc matching to find 1-3
5 eligible controls for each case (5 cases had no eligible controls) with repeat sampling (a control
6 could be matched to more than 1 case). The matching required for the case and the control to
7 be from the same country, of the same sex, within 1 year (<17 years old) or 2 years (≥ 17 years
8 old) at date of diagnosis and difference of dates of interview within 1 year. Cases consisted of
9 patients aged 10-24 years diagnosed with a benign or malignant brain tumor (BT) between
10 2010 and 2015. Tumors originated in the middle of the brain were excluded because they were
11 assumed to have little radio-frequency (RF) energy. Patients were given face-to-face interviews
12 to assess exposures and parents were also asked to complete a questionnaire regarding
13 exposures. Two exposure metrics were applied to the data to evaluate risk as a function of
14 exposure; history of the use of wireless communication devices (time since start of use and
15 lifetime cumulative call time) and estimates of dose from the use of wireless phones including
16 both wireless phones and Digital Enhanced Cordless Telecommunications phones. Regular
17 users were defined as having made or received calls at least once per week for a period of 3
18 months or more. A latency period of 1 year before the reference date was applied to the
19 analysis. A simple imputation was used for missing data based.

20 A total of 899 cases out of 1257 eligible and 1910 controls out of 2539 eligible agreed to
21 participate in the study. The response rate was 72% for cases (ranging from 15-96% depending
22 on country) and 54% for controls (34 to 97% across countries). Differences were seen between
23 cases and controls on education levels of parents, recall of mobile phone use and how the
24 interviews were conducted although no statistical tests were presented. They focused their
25 study on neuroepithelial tumors for which they had 671 cases (551 gliomas) and 1889 matched
26 controls. The main analyses from this report are presented in Table 1. Basically, the primary
27 analyses demonstrate no change or non-significant decreasing risk with increasing dose for all
28 measures of exposure. Additional analyses looked at the sensitivity of removing each country
29 separately on the overall analysis, excluding proxy interviews, increasing the latency to 5 and 10
30 years, analyzing mobile phones and cordless digital phones separately, analyzing males and
31 females separately, changing the matching numbers, excluding imputation subjects, adjusting
32 for participation bias, quality of the mobile phone use recall, source of the interview data,
33 anatomical location of the tumor, and tumor type. None of these sub-analyses differed
34 dramatically from the primary analyses. The authors conclude that their study provides no
35 evidence of a positive causal association between wireless phone use and brain tumors in
36 young people. [The strengths of this study include the exposure metrics, the completeness of
37 the analyses presented, and the size of the study. Limitations include no analysis of ipsilateral

1 and contralateral tumors, no analyses using all brain tumors, the use of hospital-based controls
2 and the impact of potential confounders such as education level of parents, type of residency
3 (urban versus rural) and country of residence.]

4 Kojimaharar et al. (2023) [3] presented data from the MOBI-kids study mentioned earlier [2]
5 focused solely on the data derived from Korea and Japan. The cases included inpatient cases of
6 brain tumors aged 10-15 years and diagnosed between 2011 and 2015 at 15 hospitals in Korea
7 and 31 hospitals in Japan. Two controls consisting of patients diagnosed with acute
8 appendicitis from the same hospital were post-hoc matched to each case, matching to age
9 (within 1.5 years for <17 years and within 2.5 for older cases), area of residence, and sex.

10 Questionnaires and interviews matched that used in [2]. The analysis used in this study
11 included mid-brain tumors and controls from outside the Tokyo metropolitan area that were
12 excluded in [2]. Exposure was calculated as in [2] but with modifications to account for the
13 types of phones used in Korea and Japan and using a 1-year lag.

14 Cases included 52 patients from Korea matched with 104 controls and 66 patients from Japan
15 matched with 132 controls. There were significant differences in mother's education, urban
16 residency and mobile phone use between Japan and Korea for both cases and controls and
17 possibly similar differences between cases and controls although no statistical evaluation was
18 provided. Of the 118 brain tumors, 46 were gliomas. Regular mobile phone use was higher in
19 Korea than Japan. The data from Japan and Korea show a non-significant increasing trend for all
20 brain tumors with no single comparison statistically significant. No analysis was provided for
21 exposed versus unexposed. No supplemental analyses were performed. [The greatest strength
22 of this study is the care they used in developing the exposure metrics and a better evaluation of
23 confounders than [2]. Limitations include small sample size, no analysis of ipsilateral and
24 contralateral tumors, no analyses using neuroepithelial tumors (for comparison), and the use of
25 hospital-based controls.]

1

2 **Table 1:** Results from recent epidemiology studies of RF and brain tumors in children and adolescents

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Sample Size for all endpoints (% resp.)	Cases ¹	Group	OR (95% CI)	P trend	Definition
Castano-Vinyals et al. (2022) [2]	CC	2010-2015 Australia, Austria, Canada, France, Germany, Greece, India, Israel, Italy, Japan, Korea, the Netherlands, New Zealand, Spain	10-24, Both	Neuroepithelial Brain Tumors	671 cases 1889 matched controls	590/81 217/70 208/9 165/2	Regular Use 10-14 years 15-19 years 20-24 years	0.85 (0.62-1.18) 0.87 (0.61-1.25) 1.06 (0.19-5.83)		Calls once per week for 3 consecutive months, 1 year lag
						81 165 283 142	Time since start of use All users (<1 yr ref) 1-4 years 5-9 years 10+ years	1.0 0.88 (0.63-1.24) 0.83 (0.58-1.19) 0.75 (0.50-1.13)		
						70 106 93 18	10-14 yr (<1 yr ref) 1-4 years 5-9 years 10+ years	1.0 0.82 (0.55-1.20) 0.98 (0.65-1.49) 0.82 (0.43-1.55)		
						9 53 116 39	15-19 yr (<1 yr ref) 1-4 years 5-9 years 10+ years	1.0 0.79 (0.32-1.94) 0.55 (0.23-1.34) 0.45 (0.18-1.33)		
						2 6 74 85	20-24 yr (<1 yr ref) 1-4 years 5-9 years 10+ years	1.0 1.76 (0.25-12.5) 0.97 (0.17-5.43) 1.02 (0.18-5.81)		
						204 141 97 114 113	Cumulative calls All users (first q ref) 2 nd Q 3 rd Q 4 th Q 5 th Q	1.0 1.13 (0.85-1.48) 0.71 (0.53-0.96) 0.85 (0.63-1.18) 0.84 (0.61-1.15)		
						110 51 35 40 49	10-14 yr (<1 yr ref) 2 nd Q 3 rd Q 4 th Q 5 th Q	1.0 1.04 (0.68-1.59) 0.72 (0.45-1.15) 0.88 (0.56-1.38) 1.09 (0.67-1.76)		
						60 48 36 45 28	15-19 yr (<1 yr ref) 2 nd Q 3 rd Q 4 th Q 5 th Q	1.0 0.94 (0.57-1.54) 0.62 (0.37-1.05) 0.78 (0.46-1.30) 0.47 (0.26-0.84)		
						34	20-24 yr (<1 yr ref) 2 nd Q	1.0 1.55 (0.87-2.78)		

¹ Entries are exposed cases / unexposed cases for exposed versus unexposed, cases only in each exposure group when exposure is broken down into categories

						42 26 29 36	3 rd Q 4 th Q 5 th Q	0.87 (0.46-1.63) 0.92 (0.49-1.72) 1.15 (0.61-2.15)		
							Cumulative call time All users (first q ref)	1.0		
						226	2 nd Q	0.90 (0.68-1.19)		
						127	3 rd Q	0.73 (0.55-0.98)		
						105	4 th Q	0.68 (0.51-0.92)		
						102	5 th Q	0.74 (0.55-0.99)		
						111				
							10-14 yr (<1 yr ref)	1.0		
						115	2 nd Q	1.04 (0.68-1.57)		
						52	3 rd Q	0.76 (0.48-1.21)		
						34	4 th Q	0.90 (0.58-1.42)		
						42	5 th Q	0.84 (0.53-1.34)		
						44				
							15-19 yr (<1 yr ref)	1.0		
						61	2 nd Q	0.79 (0.48-1.30)		
						45	3 rd Q	0.67 (0.39-1.13)		
						40	4 th Q	0.52 (0.30-0.91)		
						32	5 th Q	0.61 (0.36-1.04)		
						39				
							20-24 yr (<1 yr ref)	1.0		
						50	2 nd Q	0.77 (0.43-1.38)		
						30	3 rd Q	0.70 (0.40-1.21)		
						31	4 th Q	0.57 (0.32-1.03)		
						28	5 th Q	0.65 (0.36-1.17)		
						28				
Kojimaharra et al. (2023) [3]	CC	2010-2015, Korea and Japan	10-24, both	Brain tumors, Gliomas	132 Cases 264 Controls	29 27 29 33	Cumulative call time – Brain Tumor Non-user 0.1-54.8 hr 56.1-254.5 hr 258.1-18,780 hr	1.0 1.10 (0.58-2.08) 1.32 (0.62-2.81) 1.61 (0.72-3.60)	0.389	Most adjusted analyses, 1 year lag
						29 29 29 31	Tumor RF Energy – Brain Tumor Non-user Tertile 1 Tertile 2 Tertile 3	1.0 1.14 (0.51-2.23) 1.25 (0.60-2.61) 1.33 (0.61-2.89)	0.523	
						12 14 10 10	Cumulative call time – Glioma Non-user 0.1-54.8 hr 56.1-254.5 hr 258.1-18,780 hr	1.0 1.18 (0.37-3.77) 0.64 (0.16-2.54) 0.70 (0.16-3.03)	0.200	
						12 15 11 8	Tumor RF Energy – Glioma Non-user 0.1-54.8 hr 56.1-254.5 hr 258.1-18,780 hr	1.0 1.38 (0.38-4.97) 0.92 (0.25-3.40) 0.45 (0.09-2.29)	0.149	

2.2 Studies in Adults

2.2.1 cross-sectional/ecological studies

Robiei et al. (2023) [4] conducted a cross-sectional study on 2,796 employees (of Shiraz University of Medical Sciences (SUMS), Shiraz, Iran. Participants, through face-to-face interviews, provided information on sociodemographic data (e.g., gender, age, nationality, and education level) smartphone usage, history of diabetes, hypertension, cardiac ischemia, myocardial infarction, renal failure, fatty liver, hepatitis, chronic lung disease, thyroid disease, kidney stone, gall bladder stone, rheumatoid disease, epilepsy, and chronic headache. No information was provided on the sampling scheme used for the selection of participants. The study included 52% female and 48% male participants. The authors contend that their study demonstrated no link between mobile phone Internet usage and the risk of the listed health problems. No details or justification is provided for this conclusion.

Moon (2023) [5] (with corrigendum [6, 7]) conducted a ecologic population-based study in South Korea to examine the trends of incidence in malignant neoplasm of the brain (MNB) in association with mobile phone usage. The study included annual incidence data from the Korean Central Cancer Registry for a period of 20 years (1999-2018). Annual mobile subscription data was obtained from the International Telecom Union were analyzed via Pearson correlation. They obtained mixed results with some tumor classifications showing a correlation with subscriptions and others not. The main classification for gliomas (C71.9) demonstrated a significant negative correlation with mobile phone subscriptions. De Vocht (2023) [8] disagreed with the conclusions and argued there was no correlation at all due to rises and falls in different categories that related to better diagnosis and not mobile phone subscriptions.

Uddin et al. (2023) [9] conducted a ecologic population-based study in Taiwan to examine the trends of incidence and deaths due to malignant neoplasm of the brain (MNB) in association with mobile phone usage. The study included annual incidence and annual mortality data from the Taiwan Cancer Registry for a period of 20 years (January 2000-December 2019). Annual mobile phone users data was obtained from the National Communications Commission. Correlations were analyzed via Pearson correlation, regression analysis, and join-point regression analysis. The authors concluded that “no significant correlation was observed between MNB incidences and deaths and mobile phone usage “. This conclusion is in direct contradiction of their findings in Tables 2, 3 and 4 of their report. As with any ecologic study, this study is very limited in utility because detailed characteristics of the individuals are unavailable so it is impossible to control for other factors that might affect the observed correlations.

Galceran et al. (2025) [10] described trends in the incidence of central nervous system (CNS) cancers in Spain and its possible relationship with mobile phone use. Adjusted incidence rates of CNS cancers by sex, age (adults and children), site, and histological type from 1985 to 2015 were assessed using data from 14 general and pediatric cancer registries. The study included 20,325 CNS malignancies in adults and 2,372 in children. Of these, 14,693 (adults) and 2118 (children) were gliomas and 283 (adults) and 9 (children) were malignant meningiomas with 25.3% tumors of unspecified histology. Linear assessment over time showed a significant increase in annual percentage change (APC) in the incidence of gliomas in adults (2.0, 1.7-2.2) but not children (-0.1, -0.6-0.5) and no significant change in APC for meningiomas in adults (0.5, -0.5-1.6) with insufficient data to evaluate changes for children. A join-point analysis also demonstrated an increasing APC for gliomas in adults with a high APC (4.3, 3.5-5.1) until 1998 followed by a much slower increase (0.9, 0.5-1.3) from 1998 on. The data in children yielded similar results for the earlier years (6.0, 1.2-11.0) with a change point in 1991 and a non-significant decrease after 1991. The authors conclude that “These results do not support the hypothesis of a possible correlation between the use of MPBs and the incidence of malignant brain tumours”. However, this conclusion does not seem to apply to trends in adults for gliomas where increasing trends are evident.

2.2.2 Cohort studies

Schuz et al. (2022) [11] updated the Million Women Study [12, 13] to extend the original follow-up. This study was previously described in the earlier expert report [1]. In brief, 1.3 million women born between 1935-1950 were recruited into the study during 1996-2001. As described earlier, cellular telephone use was assessed by questionnaire and asked 1999-2005 and again in 2009 and 2010-2013. For this follow-up, all study participants were followed via record linkage to National Health Services databases on deaths and cancer registrations (including nonmalignant brain tumors) up until the earlier of death, cancer diagnosis or December 31, 2017. The main results concerning brain tumors are summarized in Table 1. All comparisons for the different exposure questionnaire times were against never users (see definitions in Table 1) and none were statistically significant from 1. Follow-up was from the time of the questionnaire so there were fewer tumors in the 2010-2013 respondents compared with the 1999-2005 respondents. For most comparisons, using exposures from the 2010-2013 respondents resulted in higher relative risks with all above 1.0 for gliomas and glioblastomas as compared to the 1999-2005 exposures which saw most relative risks below 1.0. The main weaknesses of this study include exposure information from 1996-2005 for the full analysis (as noted in the earlier review, cellular phone usage in the UK increased dramatically during the actual study period as well as the recruiting period with rates per 100 people of 9.901 (1995), 12.473 (1996), 78.281 (2001), 108.598 (2005), 121.73 (2009), 121.2 (2015), and 117.3 (2020), 122.8 (2023) [14] so some of the cases with no exposure are likely to have been exposed, and the smaller

number of cases (shorter follow-up) when exposure from 2010-2013 is used. Note that the study population has doubled the rates of glioma; the original evaluation's seven year average follow-up saw ~82 gliomas per year while from that point to now (December 31, 2009 to December 31, 2015 or 6 years), there have been an additional 996 tumors or 166 gliomas/year.

Zhang et al. (2023) [15] used data from the UK Biobank prospective cohort study to explore the relationship between cell phone usage and 25 site-specific cancers. The study included a total of 431,861 persons aged 38-73 without prior cancers recruited between 2006 and 2010. Cell phone usage was determined at baseline based on the following question, "For approximately how many years have you been using a mobile phone at least once per week to make or receive calls?", and five valid options were given to select: "Never used mobile phone at least once per week", "One year or less", "Two to 4 years", "Five to 8 years", and "More than 8 years" (there was no follow-up). Mobile phone users were also asked about weekly usage of mobile phones, use of hands-free devices, and the usual side of head for phone usage. All covariates were also obtained at baseline. The follow-up date for cancer was not given; the average follow-up time was 10.7 years with an interquartile range of 9.8 to 11.5 years. A total of 35,401 men and 30,865 women developed overall cancers. The main results related to total cancers and brain tumors are presented in Table 2. The authors reported a significant increase in risk for all cancers with exposure to cell phones with a demonstrated exposure-response for length of use in years for both males and females. There was no association of cellular phone use with brain tumors for males or females (exposure-response was not presented). The strengths of this study are its size, the cohort design and the ability to carefully evaluate the impact of covariates. The main weakness of the study is the use of cellular phone use data at baseline with no follow-up; cellular phone use has changed dramatically since 2010 and this could lead to exposure misclassification and attenuation to the null. Also, lack of information on other mobile phone use (texting, videos, etc.) could alter exposure categories and change the risks.

Feychting et al. (2024) [16] conducted the Cohort Study on Mobile Phones and Health (COSMOS) specifically to overcome differential recall, selection bias and crude exposure assessment they claim exist in other studies. Participants at least 18 years old were recruited in Denmark, Finland, the Netherlands, Sweden, and the UK from 2007 to 2012. The different countries used different strategies for identifying and contacting participants (Table 2 in [16] presents cohort size by country). Demographic data and mobile phone use were assessed using a baseline questionnaire. Participants were followed through population-based cancer registers to identify glioma, meningioma, and acoustic neuroma cases during follow-up. The end of follow-up differed by country and in some cases, by sub-cohort (Denmark: end of November 2016; Finland: end of 2015; Sweden: end of 2017; Netherlands: AMIGO cohort 7th November 2017, EPIC cohort end of 2014, Nightingale cohort 14th June 2017; UK: Scotland end of 2015, England and Wales 1st April 2020). Non-differential exposure misclassification was reduced by

adjusting estimates of mobile phone call-time through regression calibration methods based on self-reported data and objective operator-recorded information at baseline. Hazard ratios (HR) and 95% confidence intervals (CI) for glioma, meningioma, and acoustic neuroma in relation to lifetime history of mobile phone use were estimated with Cox regression models with attained age as the underlying time-scale, adjusted for country, sex, educational level, and marital status.

The cohort consisted of 264,574 participants who accrued 1,836,479 person-years of follow-up. The median follow-up time was 7.12 years. There were 149 glioma, 89 meningioma, and 29 incident cases of acoustic neuroma diagnosed during follow-up. A Cox proportional hazards model was used to analyze the data. Table 2 shows the results for gliomas. The adjusted HR per 100 regression-calibrated cumulative hours of mobile phone call-time was 1.00 (95 % CI 0.98–1.02) for glioma, 1.01 (95 % CI 0.96–1.06) for meningioma, and 1.02 (95 % CI 0.99–1.06) for acoustic neuroma. For glioma, the HR for ≥ 1908 regression-calibrated cumulative hours (90th percentile cut-point) was 1.07 (95 % CI 0.62–1.86). Over 15 years of mobile phone use was not associated with an increased tumor risk; for glioma the HR was 0.97 (95 % CI 0.62–1.52). The authors concluded that cumulative mobile phone use is not associated with the risk of developing glioma, meningioma, or acoustic neuroma. The strengths of this study are its design (a large multinational cohort) and careful evaluation of exposure. The major limitations include the small number of cancers detected (low statistical power) and heterogenous response for different countries.

Kundi (2024) [17] commented on the conclusions of the study and made three basic points. He argued the follow-up time was too short based on what was known about brain tumors and any tumor seen in this study was likely to be overtly growing before the study began. Second, he felt the use of only baseline exposure from 2007-2012 introduces exposure misclassification since exposure changes from the beginning of follow-up are likely to be higher and more widespread. Finally, he argues the power was inadequate and uses an example of a 20% proportionate change in baseline risk to demonstrate this point. The authors responded [18] arguing that the latency was not too short if non-ionizing radiation was a promoter and not an initiator and argued that cancers can occur earlier, even for initiators and solid tumors.

Moskowitz et al. (2024) [19] described five separate areas of concern they saw with this paper. First, they criticized the exposure assessment for no recent follow-up of phone usage and for ignoring other sources of exposure such as DECT phones, wifi, and cellular towers. Their second criticism dealt with outcome assessment where they argue that brain tumors in cancer registries in some of the countries included in the cohort have demonstrated problems and that the observed incidence of gliomas in this study is unusually high and the observed incidence of meningioma is too low and this could impact the significance of any observed exposure-related effects. Third, they criticized the findings as being too short after exposure to be able to

1 demonstrate a risk. Fourth, they criticized the paper based on statistical issues questioning the
2 power of the study, the heterogeneity between countries and data availability. Finally, they
3 argued that the some of the funding sources created a conflict of interest and the firewalls put
4 in place to avoid that were inadequate. The authors responded [18] addressing each issue in
5 the letter from Moskowitz et al. (2024), in most cases by noting they had already addressed this
6 in the paper or through discussions of additional research findings from other authors.

7 *2.2.3 Case-control studies*

8 Turuban et al. (2024) [20] evaluated the potential association between occupational RF-EMF
9 exposure and brain tumor risk in the multi-country INTEROCC case-control study. This study is
10 not included since it did not evaluate cellular phone usage and the RF-EMF fields encountered
11 in the workplace are different from cellular network fields.

1

2 **Table 2:** Results from recent epidemiology studies of RF and brain tumors in adults

Author (year)	Study Type	Years, Country	Age at recruitment (years), sex	Tumor Type	Sample Size for all endpoints (% resp.)	Cases ²	Group	OR (95% CI)	P trend	Definition
Schuz et al. (2022) [11]	Cohort	Recruit 1996-2001, Cell phone usage obtained in 1999-2005 and 2010-2011, British Million Women Study	Middle aged females	All brain tumors, Glioma, Glioblastoma, Meningioma, Pituitary, Other/unspecified	776,156 (65%)	937	Glioma (from 2001 exposure)			Ever use and everyday use were direct questions
						120	Ever-Never use	0.89 (0.80-0.99)		
						540	Daily Use	0.87 (0.71-1.07)		
							10-year use	0.89 (0.78-1.02)		
						281	Glioma (from 2011 exposure)			
						94	Ever-Never use	1.05 (0.86-1.29)		
						205	20+ min/week	1.12 (0.86-1.46)		
							10+ years of use	1.11 (0.85-1.44)		
						702	Glioblastoma (from 2001 exposure)			
						92	Ever-Never use	0.93 (0.82-1.05)		
						405	Daily Use	0.92 (0.73-1.17)		
							10-year use	0.91 (0.78-1.06)		
Zhang et al. (2023) [15]	Cohort	Recruit 2006-2010, follow-up date not given (mean 10.7 years) England	38-73	25 most common site-specific cancers	431,861 201,868 Male 229,993 Female	28,644	Any cancers			<0.001
						903	Males	1.09 (1.06-1.12)		
						4,972	Length of use			
						9,229	≤1 yr	1.0 (ref)		
						13,540	2-4 yr	0.98 (0.92-1.06)		
							5-8	1.01 (0.94-1.08)		
							>8 yr	1.06 (0.99-1.04)		
						25,507	Females	1.03 (1.00-1.06)		

² Entries are exposed cases / unexposed cases for exposed versus unexposed, cases only in each exposure group when exposure is broken down into categories, cases in exposed users in cohort studies

						933 6,492 10,396 7,686	Length of use ≤1 yr 2-4 yr 5-8 >8 yr	1.0 (ref) 1.02 (0.96-1.10) 1.06 (0.99-1.04) 1.06 0.99-1.13)	0.025	
						423 316	Brain/CNS Tumors Males Females	1.01 (0.80-1.27) 0.92 (0.71-1.20)		
Feychting et al. (2024) [16]	Cohort	Recruit 2007-2012, Follow-up by country, Denmark: end of November 2016; Finland: end of 2015; Sweden: end of 2017; Netherlands: AMIGO cohort 7th November 2017, EPIC cohort end of 2014, Nightingale cohort 14th June 2017; UK: Scotland end of 2015, England and Wales 1st April 2020	≥18	Glioma, Meningioma, Acoustic Neuroma	264,574	66 36 38 72 37 31 47 39 58	Glioma Cumulative Hrs Exposure Calibrated <464 464-1061 ≥1062 Uncalibrated <301 301-962 ≥963 Years of Use 0-9 10-14 ≥15	1.0 (ref) 0.99 (0.64-1.52) 0.92 0.58-1.44) 1.0 (ref) 0.98 (0.64-1.51) 0.77 (0.49-1.22) 1.0 (ref) 0.81 (0.51-1.28) 0.97 (0.62-1.52)	1.0 (0.98-1.02) 1.00 (0.98-1.02)	

2.3 Conclusions from New Studies in Humans

The evidence on an association between cellular phone use and the risk of brain tumors in children is still moderate showing a statistical association for which chance, bias and confounding could not be completely ruled out. The information on gliomas alone is insufficient to make any determination.

One new study [2] and one sub-cohort study [3] are available regarding the relationship between brain tumor risks and exposure to RF from cellular phone usage. Figure 1 summarizes the information from all of the available studies and shows several meta-analyses evaluating the general trend seen in these studies. Figure 1 basically shows a tendency for the studies to produce a positive finding when aggregated. Using the data from the recent study by Kojimaharar et al. (2023) [3] instead of the main study by [2] yields a statistically significant meta-analysis. The data is for brain tumors in general with very limited information on gliomas alone.

The evidence on an association between cellular phone use and the risk of glioma in adults is still strong.

Figure 2 provides a series of meta-analyses using the information from the newer studies included with the older studies. This can be compared with the meta-analyses in Figure 2 in the previous Expert Review [1]. The new meta-analyses have clearly reduced the magnitude and significance of the findings from the previous analyses that do not include these new studies. While there is still considerable difference from study to study on ever versus never usage of cellular phones, 6 of the 8 meta-analyses in Figure 2 are still positive and 1 analysis is significantly positive.

One of the new studies, Schuz et al. (2022) [11], is an update of a previous study by Benson et al. (2013) [13] and, as such, suffers from all of the limitations of that study previously described. These include the exposure misclassification when using the original phone usage statistics. As noted, participants early on in the study will likely have cell phone usage that severely underscores their exposure. As discussed above, cell phone usage in the UK stabilized after 2009 and was fairly stable from 2005 onward. Thus, the numbers provided for 2010 to 2013 are more likely to truly represent exposure during the entire timeframe than the original exposures. In interpreting the data, one must choose between the two main analyses in Schuz et al. (2022) (full data set, early exposures only; reduced data set, newer exposure measures). The entries for L1 and L2 in Figure 2 illustrate the differences between these two analyses and the impact they have on the meta-analyses. The authors might have seen stronger positive results had they used the 2010-2013 exposure data for the entire span of the study [This would be difficult to do with tumors and deaths affecting who can answer the survey from 2010 to 2013.]

1 All three of the new studies are cohort studies that use Cox proportional hazards model as the
2 basis for their analyses. All three of these studies report relative risk estimates at or below 1 for
3 most exposure categories. Note that Feychting et al. (2024) [16], Zhang et al.; (2023) [15], and
4 Frei et al. (2011) [21] did not provide ever/never use so a meta-analysis was performed to get
5 the value used in Figure 2. Frei et al. (2011) [21] used linear Poisson regression in their analysis.
6 Schuz et al. (2022) specifically stated they used time since entry into the cohort as the
7 underlying time variable in the model. It is not clear what underlying time variable was used by
8 Zhang et al. (2023). Feychting et al. (2024) used age as the underlying time variable in the
9 analysis. The use of age as the time variable creates a problem with the overall analysis
10 because of the assumption that the additional hazard caused by any covariate (e.g. cell phone
11 usage) then becomes proportional to the age of the participant rather than the time on study.
12 To understand the nature of this problem, let's consider a simple example using the approach
13 by Schuz et al. as compared to Feychting et al. Suppose a 70-year old person and a 25 year-old
14 person enter the study on the same day. Using time since entry into the study as the underlying
15 time variable, at the end of 10 years follow-up, they both had used a phone for 550 hours, their
16 risk would be the same because their time variable would be 10 years for both and the
17 additional hazard from exposure would be proportionate to the underlying hazard for people
18 that long on the study. However, using the method used by Feychting et al. (2024) this would
19 not be the case. When using age as the underlying time variable, the underlying hazard for the
20 25-year old would be different than that of the 70-year old but the proportional impact on their
21 underlying hazards is set to be the same for the 550-hour group (A central assumption of the
22 Cox model is that the hazard ratio between groups remains constant over time.). The
23 consequence of this is that even though the 70-year old has the same exposure as the 25-year
24 old, the actual amount of risk for the 2 examples would be different. Looking at Table 1 in
25 Feychting et al. (2024), we see that there is a bias for older people to have shorter exposures
26 and younger people to have longer exposures. This creates a problem in the evaluation if the
27 actual risk is additive (as in Poisson regression) or not proportional to age.

28 For adults, although the data has changed somewhat and has overall relative risks that are
29 smaller, I find that the new studies are problematic and not sufficient to convince me that
30 previous studies aren't better. Given their limitations and the differences in analysis methods,
31 there is no reason to dramatically change my previous opinion. In conclusion, an association has
32 been established between the use of cellular telephones and the risk of gliomas and chance, bias
33 and confounding are unlikely to have driven this finding. The ecological studies are of insufficient
34 strength and quality to alter the findings from the observational studies.

Figure 1: Forest plot and meta-analyses of regular use or ever use of cellular telephones and the risk of brain tumors/glioma [studies with a solid blue square either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are individual studies or smaller pooled studies; red diamonds are meta-analyses] ^a

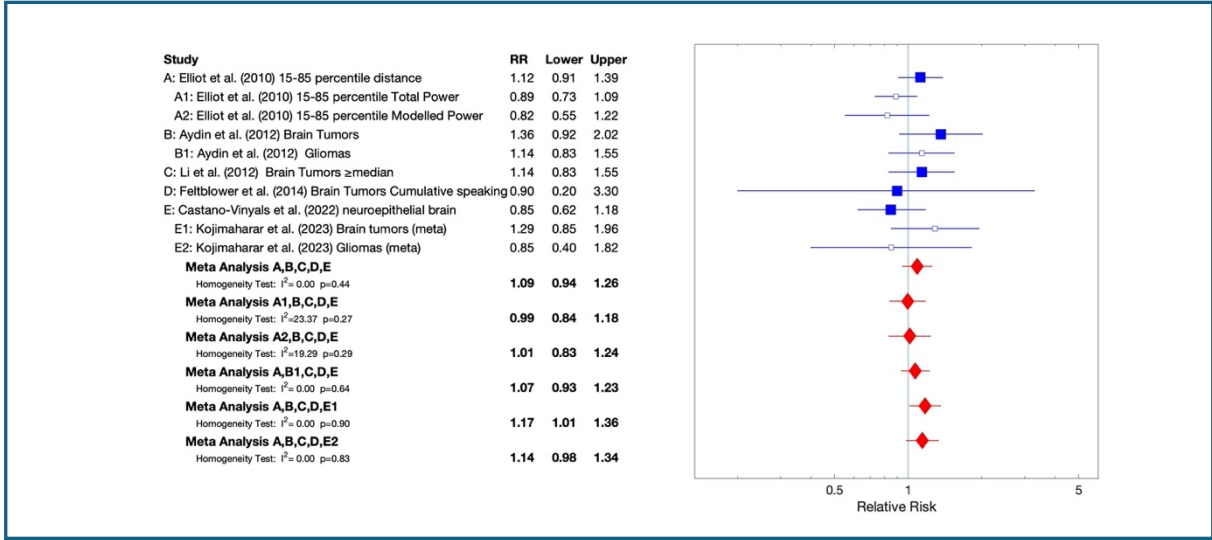
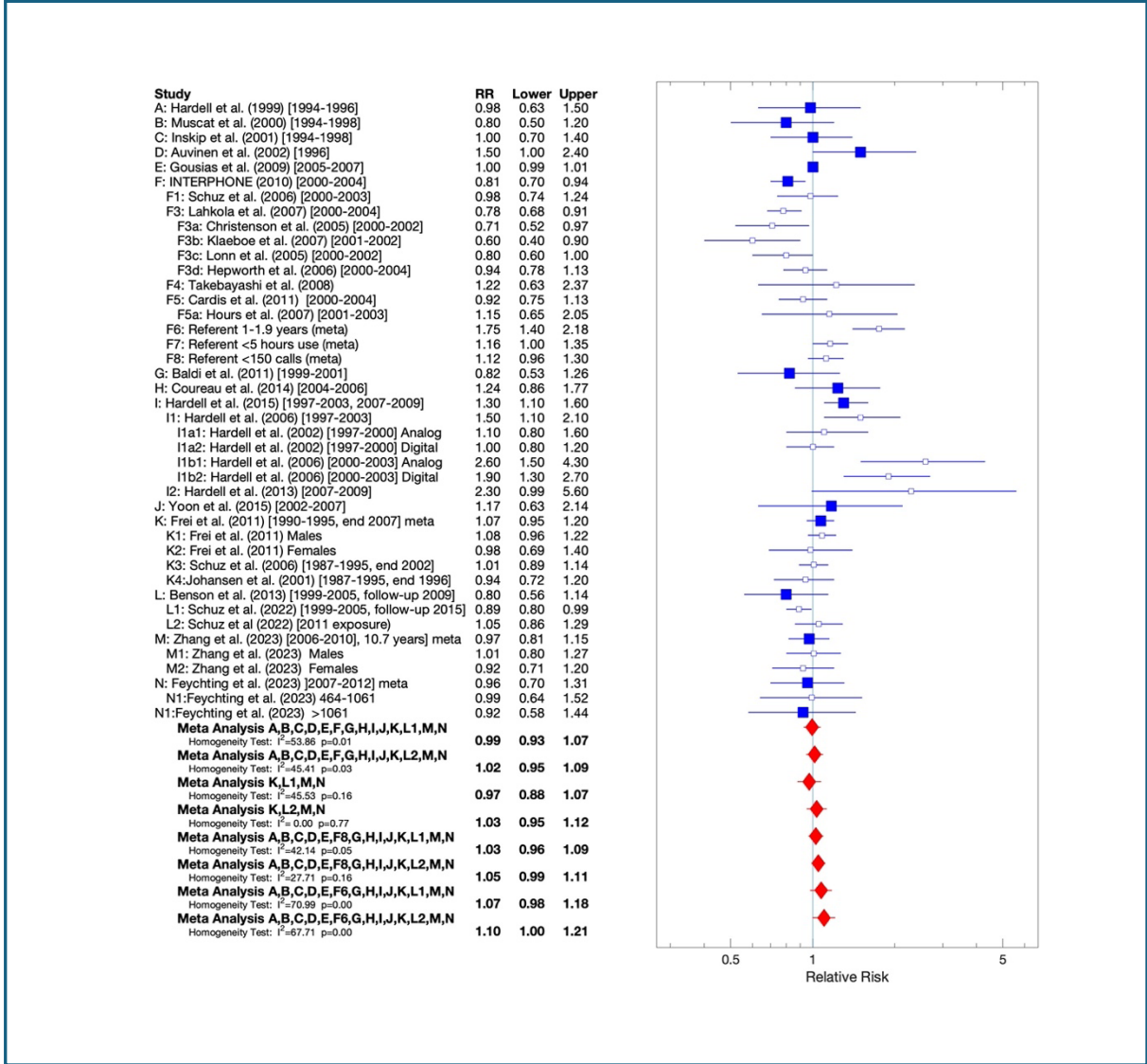


Figure 2: Forest plot and meta-analyses of regular use or ever use of cellular telephones and the risk of glioma [studies with a solid blue square either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are individual studies or smaller pooled studies; red diamonds are meta-analyses] ^a



The column labeled “Study” provides the reference to the publication and the years in which cases and controls were collected for case control studies and the years when phone use information were collected for cohort studies and the year in which follow-up ended. Some studies are pooled evaluations of multiple other studies, so the other studies are indented. For example, the Interphone study (Study F) is the pooled analysis of studies from 13 countries. Lahkola et al. (2007) (Study F3) is a pooled analysis of the data from 5 of those countries and Christenson et al (2005) (Study F3a) is the publication for data from one of those 5 countries. The column labeled “RR” is the risk ratio (OR, RR or mRR) from the study, “Lower” and “Upper” are the lower and upper bound on a 95% confidence interval around the RR. The graphic on the right simply plots the RR as a square or diamond with the “whiskers” (blue line running through the box) showing the width of the 95% confidence interval. The vertical line passing through 1 represents no effect. If the box and both whiskers are to the right of this line (greater than 1) and not touching it, this finding is statistically significant with a positive effect; if they fall completely to the left of the vertical line (below 1), then the risk is significantly reduced. The blue boxes that are filled in are major studies, the blue boxes that are

white in the middle are the sub-studies and the red diamonds are all meta-analyses. “Homogeneity Test” provides the I^2 statistic and the p-value for the Q-test

3.0 Laboratory Cancer Studies

Brooks et al. (2024) [22] used gliomas (12) and cardiac schwannomas (9) obtained from the Ramazzini study in rats [23] compared to non-tumor brain tissue from RF-exposed rats and spontaneous gliomas (2 control animals) to identify genetic alterations through molecular characterization of important cancer genes relevant for human gliomagenesis. The top 23 orthologous human glioma-related genes were used for the analysis. The analyses suggest that rat gliomas resulting from life-time exposure to RFR histologically resemble low grade human gliomas but surprisingly no mutations were detected in rat gliomas that had homology to the human IDH1 p.R132 or IDH2 p.R172 that are common in human gliomas.

As seen above, the only study in this section is evaluating the molecular signatures of the tumors seen in the Raamazzini study and is not a new study. Thus there is no new literature in this section and my prior evaluation still holds:

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.

4.0 Mechanisms Related to Carcinogenicity

There are many studies that explore various theories of why and how RF-EMF interrupts normal cell function in ways that could lead to carcinogenicity. This review concentrates on updating my previous review of studies addressing oxidative stress and genotoxicity using only the newest studies from the last 3 years. The studies cited below cover an extensive range of the literature from 2023 to 2025 but are not exhaustive and there are certainly studies that will have been missed in this review.

To identify which publications to review, a list of all studies reviewed by a Swiss expert group on electromagnetic fields and non-ionizing radiation between the years 2023 and 2025 [24] was obtained. This constituted 766 references and associated articles. From these, I removed all articles regarding electric and magnetic fields below 800 MHz and above 3GHz (the upper bound excludes most 5G studies, but this review is intended to relate to predominantly past exposures). In addition, epidemiologic studies and animal carcinogenicity studies were included in the previous sections. From the remaining studies, any that did not directly measure markers

of oxidative stress or genetic toxicity were excluded. The final list included the 45 references that were considered in this review and are described below³.

4.1 Oxidative Stress

4.1.1 Studies of oxidative stress in humans

Kadhim et al. (2023) [25] investigated oxidative stress, antioxidant and thyroid function in the serum of workers at electrical substations and cellular communication towers in Baghdad. The study included 40 men from electrical substations (aged 19-60), 40 men from cellular communication towers (aged 26-55), and 40 men as controls for the study (aged 21-55).

Collection occurred between November 2021 and January 2022. The authors measured total oxidant status (TOS), MDA, total antioxidant capacity (TAC) and GSH. Thyroid function was evaluated using thyroid stimulating hormone (TSH), triiodothyronine (T3) and thyroxine (T4). They also obtained data on BMI and smoking. Substation workers had greater oxidative stress markers and lower antioxidant markers compared to tower workers ($p<0.001$) and controls ($p<0.001$). Tower workers had greater oxidative stress markers and lower antioxidant markers compared to controls ($p<0.001$). TSH was significantly higher in electrical substations workers compared to the other two groups but T3 and T4 were unchanged. [BMI and smoking percentage were quite high in all groups. No information was given on smoking frequency, duration, etc.]

Özen et al. (2023) [26] investigated the effects of mobile phone use during pregnancy on cord blood oxidant-antioxidant levels. Cell phone usage of 67 healthy pregnant women who delivered by normal spontaneous vaginal birth without additional risk factors were obtained. Total antioxidant status, total oxidant status (TOS), ischemia modified albumin (IMA), total thiol, native thiol, disulfide levels and disulfide/total thiol, disulfide/native thiol, native thiol/total thiol ratios were evaluated in umbilical cord blood from these births. A negative correlation was found between daily talking duration (≤ 30 min vs >30 min) by mobile phone and IMA levels; a positive correlation was found between daily talking duration and native thiol and total thiol levels ($p<0.05$). TOS, native thiol and total thiol levels were higher in the mothers who have another mobile phone in their bedroom at night ($p<0.05$). [There was no correction for any other factors such as duration of birth contractions, sex of child].

Rangesh et al. (2024) [27] Evaluated the metabolites in first urines of 60 men naturally exposed to high and low levels (30 per group) of cellular RF radiation in microenvironments defined by sampling small communities in India using a NARDA-brand Field Strength Analyzer. The high exposure group, Marwari Awas Compound, had an average of 126.8 mW/m^2 and the low

³ Summaries are derived from the abstracts provided for each individual study with modifications to shorten their presentation and provide my views on the findings from the studies.

exposure group, Village Kamarchinva Done, had an average of $45.17 \mu\text{W}/\text{m}^2$, ~2800 times less than the high exposure group. The two groups were age-matched adults over 19 years of age and were recruited from November 2019 to July 2022. Some potential participants were excluded for a variety of reasons (page 55 of report). Their analysis of the metabolomics data resulted in 10 upregulated metabolites and 19 downregulated metabolites in the high exposure group compared to the low exposure group (their heatmap only shows 11 upregulated and 15 downregulated). The authors describe their findings as preliminary and that they suggest a connection between oxidative stress and gut microbiota imbalance. [No covariates were used in any analyses, there were no descriptive statistics on the study subjects except for age and the presentation of the results is somewhat difficult to understand with numbers between groups of genes not exactly matching the text.]

4.1.2 Studies of oxidative stress in laboratory mammals

Goudarzi et al. (2023) [28] exposed forty-two male Wistar rats to RF-EMF and randomly divided them into 6 groups. Group 1 (controls) received 5 mL of normal saline via gavage, Group 2 received 915 MHz radiation, Group 3 received 2450 MHz radiation, Group 4 received rosmarinic acid (RA) plus 915 MHz radiation, Group 5 received RA plus 2450MHz radiation, and Group 6 received oral RA (5 mg/kg). Treatment and radiation (1 hour per day) continued for up to 30 days. All markers were assessed in heart cells. RF-EMF significantly reduced the activities of SOD, catalase (CAT), and glutathione peroxidase (GPx), GSH, and total antioxidant capacity (TAC) and significantly increased oxidative stress indices, such as the levels of malondialdehyde (MDA) and nitric oxide (NO), and the content of protein carbonyl (PC). In contrast, RA significantly elevated TAC level (all groups), GSH content (group 4), GPx activity (group 5), SOD activity (all groups), and CAT activity (group 5) and conversely reduced MDA level (all groups), NO level (all groups), and PC content (all groups). RF-EMF showed clear indications of oxidative stress and toxicity in heart cells. [There is no group without vehicle for comparison to the exposure without RA.]

Karadayi et al. (2023) [29] evaluated oxidative stress and histopathological changes in testicular tissue of 45-day old male Wistar rats exposed to electromagnetic radiation at varying electric field intensities within the 2.45 GHz radiofrequency (RF) range. The study used six (6) experimental groups including a sham control group (9 rats) and groups (8 animals per group) exposed to electric field strengths (EFS) of 0.6 V/m, 1.9 V/m, 5 V/m, 10 V/m, and 15 V/m, respectively. Exposure started in-utero after fertilization and lasted for 45 days after birth with exposures of 1 hour per day. All rats in the RF radiation-exposed groups exhibited elevated MDA values and decreased GSH values in testicular tissue, showing a dose-response pattern (untested) with only significance in the highest exposure group ($p < 0.05$). A comparative analysis between the microwave radiation-exposed group and the control group revealed alterations in seminiferous tubular sections again showing dose-response (not tested) and a significant

change in the highest exposure group ($p < 0.001$ against all other groups). [There is some vagueness as to the exact experimental design with no discussion of the number of dams studied, how fertilization was determined, etc.]

Khayat et al. (2023) [30] investigated the effect of RF-EMF exposure during pregnancy on Matrix metalloproteinase 2 (MMP-2) and (MMP-9) 9 expressions after neonatal hypoxia-ischemia (HI) in rats. Groups of 10 female Wistar rats were either sham exposed or exposed to 900 MHz mobile phone radiation for 12 hours per day at 2 watts/kg. Forty male offspring from each group of dams were divided into 2 groups of 20 with one group serving as control and the other receiving induced HI (90 minutes at 8% oxygen). This resulted in 4 groups, sham exposed, EMF exposed, sham exposed + HI, and EMF-exposed + HI. Seven days after HI induction, neurobehavioral tests were performed, and then brain tissue was taken from the skull to measure MMP-2 and MMP-9 expressions, inflammation, oxidative stress, infarct volume and cerebral edema. EMF exposure without HI altered oxidative stress/inflammation markers (TNF- α , TAC, and TOC), but not significantly. EMF-exposure + HI altered oxidative stress/inflammation markers ($p < 0.01$, TNF- α , TAC, and TOC).

Maalouf et al. (2023) [31] analyzed the expression of thermoregulatory genes and the involvement of mitochondria in adipose tissue metabolism after RF exposure. Thirty-six (36) male C57BL/6J mice were assigned to either exposed or control groups. The exposed groups were subjected to RF fields at 900 MHz, with specific absorption rates (SAR) of 0.1 W/kg or 0.4 W/kg, either for three or seven consecutive days (final group sizes are not provided). The findings indicate that RF exposure leads to changes in adipose tissue markers, with some effects being dose-dependent and time-dependent. In brown adipose tissue (BAT), after 3 days of RF exposure, thermogenesis is reduced, mitochondrial activity in BAT decreases, and an increase in gene expression, responsible for balancing the regulatory and damaging effects of reactive oxygen species (ROS), was observed. This effect was partially compensated after 7 days of exposure. In white adipose tissue (WAT), RF exposure results in reduced fatty acid oxidation, impaired energy production, and hindered adipocyte differentiation. Notably, no effects of RF on mitochondrial biogenesis in WAT were observed. These findings contribute to understanding the effects of RF exposure on adipose tissue metabolism and thermoregulation, highlighting dose-dependent and time-dependent responses.

Men et al. (2023) [32] evaluated oxidative stress in the testicles of groups of 20 male Wistar rats exposed to 2.856 GHz microwave radiation (30 mW/cm²) for 6 weeks, 5 days/wk, 15 min/day. Their goal was to see if metformin (a well-known antioxidant) could reduce any oxidative stress induced by the RF exposure. Their results indicated that testis exposed to RF had reduced GSH ($p < 0.01$), increased MDA ($p < 0.01$) and reduced HO-1 expression ($p < 0.05$) which could then be alleviated by Metformin.

Salameh et al. (2023) [33] exposed 26 pregnant female Sprague-Dawley rats to 900 MHz GSM with an SAR estimated in the liver to be 0.768 W/kg. Whole rat embryos were extracted at gestational days 7.5, 11.5, 15.5 and 19.5. Oxidative stress markers were evaluated in either whole body (7.5 days) or liver (all other days). A significant decrease in MDA was observed for days 7.5, 11.5 and 19.5 with a significant increase at 15.5. Mitochondrial SOD was increased after 11.5 days, Cytosolic SOD after 15.5 days, CAT increased at the three earliest times and decreased at 19.5.

Serin et al. (2023) [34] examined changes in 12 gene regions in thyroid tissue of female Wistar albino rats. Ten (10) rats were exposed to a Wi-Fi signal (Zyxel NBG-418N-V2 wireless Access Point/Router) for 12 h/day, 30 days with an appropriate control group of 10 animals. Exposed rats had significant increases in gene expression levels of many gene regions investigated in the study ($P < 0.05$). Among the study groups, 12 different genes known to be related to autophagy and oxidative stress were significantly altered by Wi-Fi exposure. [It is not possible to determine exposure from this study]

Zufry et al. (2023) [35] exposed Wistar rats (sex not given) in groups of 10 to cellular signals from GSM modules (1.8 GHz, receiving and sending calls) for 60, 150 or 180 minutes/day for 12 weeks. An appropriate control group also contained 10 animals. TSH, T4, and MDA concentrations in serum and monocarboxylate transporter 8 (MCT8) concentrations in brain were measured following exposure. TSH and T4 were significantly lower in all exposure groups and MDA was significantly increased in all exposure groups. MCT8 was significantly reduced for the 150 min/day and 180 min/day exposure but not for the 60 min/day exposure.

Fatah et al. (2024) [36] evaluated the effectiveness of rosmarinic acid (RA) against oxidative stress caused by RF exposure in rats. Male Wistar rats six groups, 7 animals per group) were given 5 mL normal saline (0.9% NaCl) or RA (20 mg/kg) by gavage, and either no RF-EMF exposure, 915 MHz cell phone exposure, or 2.450 GHz RF exposure. After thirty days of 1 hr/day RF exposure, biochemical and histopathological parameters of their testes were measured. Both RF exposures significantly ($p < 0.001$) diminished the activity of antioxidant enzymes (SO, CAT, GPx and glutathione content). Higher frequency exposure significantly ($p < 0.001$) increased oxidative stress indices (NDA, NO, protein carbonyl content) and both exposures significantly reduced the number of Sertoli cells, spermatogonia, primary spermatocyte, epithelial height, and seminiferous tubular and luminal diameters ($P < 0.001$). RA reversed the above-mentioned harms and moderated the adverse effects of 2.450 GHz RF on the testes of rats by significantly diminishing the oxidative stress indices and antioxidant enzyme rise and improving the histological parameters ($P < 0.001$).

Jaffar et al. (2024) [37] assessed the oxidative stress status in testicles of six groups of male Sprague-Dawley rat pups exposed to W-Fi (2.45 GHz TP-LINK AC750 Wireless Dual Band Wi-Fi

Router Archer C20, 24 hours/day) and/or edible bird's nest (EBN) for 14 consecutive weeks. Adequate controls (n=6 animals) were also included. There was a significant increase in total oxidant status (TOS), a significant decrease in total antioxidant status (TAS), and a significant increase in the expression of 8-hydroxy-2'-deoxyguanosine (8-OHdG) in the testis of the Wi-Fi group. Sperm chromatin integrity, morphology, concentration, and motility were among the parameters that significantly decreased. EBN had little effect of the oxidative stress caused by the RF signal.

Keles et al. (2024) [38] exposed 6 21-day old male Sprague-Dawley rats to RF-EMF (900 MHz, 0.242 W/m²) for 25 days (7 d/wk, 1 hr/day) with an appropriate control. The RF group had significantly increased (p=0.001) kidney tissue volume and weight and total body weight. Lipid peroxidation, GSH, CAT and SOD were also significantly increased (p=0.001) in the kidney tissue of the RF-exposed rats. Histopathological evaluation also revealed cortical/medullary bleeding/obstruction and widespread fibrosis, dilatation, vacuolization, and degeneration in distal and proximal tubules, decreased and atypical parietal cells, degeneration in epithelial cells, dilated and degenerated glomeruli in the Malpighian body, Bowman's membrane degeneration and degeneration in the vascular pole, podocyte, pedicel and mesangial cells following EMF exposure. They concluded long-term exposure to 0.9 GHz EMF can activate the renin-angiotensin system in the rat kidney resulting in structural, histopathological, and biochemical changes.

Mohamed et al. (2024) [39] investigated the potential histological, morphological and biochemical effects on the hippocampus of electromagnetic fields (EMFs) disseminated by mobile phones and the roles of baobab (*Adansonia digitata*) (AD) and black seed (*Nigella sativa*) (BS) in mitigating these effects. Fifty-six male, 12-week-old Wistar albino rats were divided into eight groups of seven animals each (control, AD only, BS only, EMF only (900 MHz 1 hr/day, 28 days, SAR not given), EMF + AD, EMF + BS, EMF + AD + BS). No statistically significant differences between the EMF and control or sham groups in terms of pyramidal neuron numbers (p>0.05) were detected. Exposure to 900-MHz EMF produced adverse changes in the structures of neurons at histopathological analysis and no changes in CAT or SOD.

Ozergin Coskin et al. (2024) [40] investigated the effects of the 1800 MHz electromagnetic field (average SAR of 0.117 W/kg) created by a generator on the parotid gland in rats. A total of three groups (control, 6 hours/day exposure and 12 hours/day exposure for 30 days), each with 7 male Sprague-Dawley rats, were included in the study. Histopathology showed an increase in degeneration in the parotid gland in rats exposed to electromagnetic fields for 6 and 12 hours a day, and immunohistochemical analysis showed an increase in the apoptotic index in both study groups (P = .001, P < .001). TOS and calcium were significantly increased in both exposure

groups ($p < 0.05$ for 6 hr/day and $p < 0.01$ for 12 hr/day) and TAS was similarly significantly decreased.

Pang et al. (2024) [41] exposed groups (10 animals per group) of 8-week old male Wistar rats to 2.856 GHz RF (testicular SAR of 34.2 W/kg) alone and with blueberry anthocyanins (3 doses). An appropriate control was used. RF exposure caused a significant decrease (p ranging from < 0.05 to < 0.001) of sperm motility, sex hormone levels and testicular tissue structure damage, increased SIRT1 expression and decreased FoxO1 expression, increased GSH/GSSG, and SOD, inhibited MDA, increased LDH, SDH and ATP synthase and decreased Caspase-3 expression. Blueberry anthocyanins inhibited the oxidative stress induced by the RF. [This experiment was done with a very strong exposure to RF]

Pardhiya et al. (2024) [42] studied the biocompatibility of Bovine serum albumin (BSA) conjugated manganese dioxide nanoparticles (MNP*) to counteract the impact of RF on the male Wistar rats. For the RF part of this complicated set of studies, 6 10-week old male Wistar rats were exposed to 2.002 GHz RF (SAR 1.2 W/kg) for 2 hr/day, 8 weeks with an appropriate control. RF significantly ($p < 0.05$) reduced SOD activity, did not alter CAT and significantly increased ($p < 0.05$) MDA in blood of exposed rats. RF also significantly ($p < 0.01$) increased DNA damage levels (comet assay) in peripheral blood cells.

Yardim et al. (2024) [43] exposed six (6) separate groups of six (6) male Wistar Albino rats to RF-EMF (2.6 GHz, SAR 0.297 W/kg, 30 min/d, 5 d/wk, 4 weeks) and/or melatonin (10mg/kg/day for 4 weeks). The groups included untreated control, sham RF controls, RF-only exposure, melatonin-only controls, and melatonin and RF simultaneous exposure. Biochemical analyses were performed on testicular tissue by measuring malondialdehyde, total nitric oxide, glutathione, and glutathione peroxidase levels. Results showed a significant increase in MDA and total NO and a significant decrease in GSH and GSHPx following RF-only exposure. Melatonin attenuated these effects. [The conclusions match what is written but the main findings in Figure 6 cannot be accurately interpreted due to errors in the footnote].

Abdel Rahman et al. (2025) [44] exposed Sprague-Dawley rats (sex not given) to RF-EMF (no description) and/or avocado extract to evaluate oxidative stress and apoptotic activity in the tongue and parotid gland. [Study is too poorly described to be evaluated appropriately].

Akbari et al. (2025) [45] studied the effect of moderate aerobic exercise (AE, using a complicated schedule of increasingly more exercise) on oxidative stress (OS) and cardiomyocyte damage resulting from cell phone RF (Huawei Honor 7, SAR 0.52 W/kg, 15 cm from cages, 0.9-1.8 GHz, 3 hr/d for 6 weeks) in male Wistar rats. Rats were randomly allocated to 4 groups with 8 animals each: control (C), RF only, AE only, and RF+AE. The heart was collected 48 hours after the last AE session to assess OS, histopathological damage, and left ventricular structure.

Tissue sections revealed that the RF-only group had significantly more heart tissue damage (increased apoptotic cells and collagen deposition) than the control group, and adding AE mitigated the damage ($P < 0.05$), but not back to control levels. In the RF-only group, in comparison to the RF+AE, control, and AE groups, the activity of SOD, CAT, and GPX significantly decreased, and the concentration of malondialdehyde MDA significantly increased ($p < 0.05$). Exercise training in both the AE and RF+AE groups resulted in a significant reduction in SOD, CAT, and GPX activity and a significant increase in MDA ($P < 0.05$). RF+AE significantly attenuated the effects of RF only, but not back to control baseline.

Gao et al. (2025) [46] studied the role and regulatory mechanisms of ferroptosis in RF-induced reproductive damage. Three groups of 20 each male C57 mice were exposed to 2.856 GHz of RF radiation at 0, 13 and 26 W/kg testis for 30 min and followed for 2 weeks with multiple sampling times. The exposed rats showed decreased sperm motility, induced morphological changes, damaged testicular tissue and mitochondrial morphology, increased MDA and decreased GSH/GSSG. It is likely that increased Fe(2)(+) levels increased SLC7A11 and decreased GPX4 to cause the oxidative stress. In a follow-up experiment, mouse spermatocyte cells (GC-2 cell line) were pretreated or not with 1 μ M Fer-1 (a ferroptosis inhibitor), and exposed to the same exposures of RF as the rats. The same changes in oxidative stress were seen in the exposed GC-2 cells and this was attenuated by the Fer-1. Nrf2, Keap1, SLC7A11, GPX4 and HO-1 expressions were down-regulated by RF exposure and significantly up-regulated following the addition of the Fer-1 inhibitor. The results confirmed that the Nrf2 signaling pathway can regulate ferroptosis of oxidative stress. This study suggests that RF exposure affects mouse reproductive function by enhancing oxidative stress, inducing ferroptosis by inhibiting the Nrf2 signaling pathway and modifying SLC7A11 and GPX4 protein expressions. [Ferroptosis is a unique iron-dependent form of cell death that is distinct from apoptosis, necrosis and autophagy.]

Gunay et al. (2025) [47] exposed groups of 6 male Wistar rats to 2.1 GHz RF (electric field 15 V/m) for 15 minutes daily and 4 hours daily for 3 weeks. The rats were divided into eight groups: normal diet and untreated control, Sham1 (15 min/d sham RF exposure), Sham2 (4 hr/day sham RF exposure), RFF1 (14 min/day RF), RFF2 (4 hr/day RF), high fat diet and control (HFD), Sham+HFD (4 hr/day), and HFD+RFF (4 hr/day). [Significant changes in TOS, TAS and TNF-alpha were noted by the authors, but it was not possible to determine what comparison groups these tests were referencing.]

4.1.3 Studies of oxidative stress in mammalian cells

Bertuccio et al. (2023) [48] investigated the effects of 2.45 GHz electromagnetic radiation on cell redox status in SH-SY5Y neuroblastoma cells, which were differentiated to neuronal-like cells, and peripheral blood mononuclear cells (PBMCs). Cells were exposed to 2.45 GHz

electromagnetic radiation for 2, 24, and 48 h. Cell viability and mitochondrial activity alterations were measured by reactive oxygen species (ROS), mitochondrial transmembrane potential (DeltaPsim), NAD⁺/NADH ratio, mitochondrial transcription factor A (mtTFA), and superoxide dismutase 1 (SOD1) gene transcript levels. Apoptosis and autophagy were also investigated by evaluating B-cell lymphoma 2 (BCL2), BCL2-associated X protein (BAX), and microtubule-associated protein 1A/1B-light chain 3 (LC3) gene transcript levels. Cell viability was significantly reduced after 24-48 h of exposure to radiation. ROS levels significantly increased in radiation-exposed cells, compared with controls at all exposure times. DeltaPsim values decreased after 2 and 24 h in exposed SH-SY5Y cells, while in PBMCs, values decreased soon after 2 h of exposure. Alterations were also found in the NAD⁺/NADH ratio, mtTFA, SOD1, LC3 gene expression, and BAX/BCL2 ratio.

Joushomme et al. (2023) [49] exposed live human keratinocytes and fibroblasts cells to continuous or intermittent (5 min ON/ 10 min OFF) exposure to 5G 3.5 GHz signals at specific absorption rates (SAR) up to 4 W/kg for 24 h. Their goal was to assess the impact on basal or chemically-induced activity of Heat Shock Factor (HSF), Rat Sarcoma virus (RAS) and Extracellular signal-Regulated Kinases (ERK) kinases, and Promyelocytic Leukemia Protein (PML). These are all molecular pathways involved in environmental cell-stress responses. They saw a significant decrease of the HSF1 basal BRET signal when fibroblasts cells were exposed at the lower SARs tested (0.25 and 1 W/kg), but not at the highest one (4 W/kg), and a slight decrease of As(2)O(3) maximal efficacy to trigger PML SUMOylation when fibroblasts cells, but not keratinocytes, were continuously exposed to the 5G RF-EMF signal. [Authors failed to show the results; just indicated non-significant or gave the exact value. Trend could not be assessed.]

Koohestanidehaghi et al. (2023) [50] collected the MII oocytes in NMRI mice and used *in vitro* fertilization to generate zygotes. Zygotes were divided into control (363 zygotes) and exposed (363 zygotes) groups. Exposed zygotes were exposed to a mobile phone for 30 minutes with frequency 900-1800 MHz RF-EMR. All zygotes were incubated for 5 days. Intracellular ROS, morphokinetic, embryo viability rate, and gene expression were evaluated. Exposure of zygotes to RF-EMR induced ROS ($p=0.0001$) and resulted in a significant decrease in blastocyst viability (87.85 ± 2.86 versus 94.23 ± 2.44), delay in cleavage development ($t(3)-t(12)$) and also increased the time (in hours) to reach the blastocyst stage (97.44 ± 5.21 versus 92.56 ± 6.7). A significant increase ($p=0.0001$) was seen in mRNA levels of Hsp70 and a decrease ($p=0.0001$) in SOD gene expression. [Using a cell phone for exposures introduces uncertainty in the level of exposure]

Yadav et al. (2023) [51] examined how exposure of human THP-1 monocytic cells to 15, 30, 45, 60, 90, and 120 min of 2.318 GHz RFR using a fourth generation mobile phone (Redmi Note 7) at a stated power density of 0.224 W/m² would impact innate immune responses. Superoxide

production was significantly increased at 15,30 and 45 minutes but not later. Nitric oxide release was increased at 30 minutes, 60 minutes and 90 minutes but was normal at all other times. MTT and TNF-alpha levels did not change over the course of the experiment. After 30 min of exposure, the RFR dramatically enhanced level of the pro-inflammatory cytokine IL-1 alpha. Overall, the results suggested that RFR can affect the function of the human innate immune system. . [Using a cell phone for exposures introduces uncertainty in the level of exposure]

Bertuccio et al. (2024) [52] evaluated the toxic effects caused exposure of SH-SY5Y neuronal-like cells and PBMCs from blood donors to 2.45 GHz EMR (24 hour, 1.80 V/m electric field) and the efficacy of sulforaphane in preventing this toxicity. Different concentrations of sulforaphane were used (5-10-25 microg/mL). Cell viability was significantly reduced by RF-EMF in SH-SY5Y cells, but reduced (non-significantly) in PBMCs and fully alleviated by 5 µg/ml SFN. ROS was significantly enhanced by RF exposure in both cell lines and only partially alleviated by SFN. Mitochondrial activity, the transcription and protein levels of redox markers, and apoptosis-related genes were similarly affected by exposure to RF-EMF and partially or fully corrected by exposure to SFN.

Sannino and colleagues [53-59] have conducted a number of assays using various cell lines, exposure times, cellular markers, etc. to evaluate the ability of RF-EMF to protect cells from genotoxicity and/or oxidative stress by first exposing the cells to RF-EMF followed by a toxicant with known ability to cause genotoxicity and/or/oxidative stress. In general, they were able to show such protective effects and in some, but not all, they were able to show similar effects by RF alone.

4.2 Genotoxicity (*in-vivo* and *in-vitro* in mammals)

4.2.1 Studies of genotoxicity in humans

Sangle et al. (2023) [60] evaluated micronuclei (MN) frequency in exfoliated oral mucosal cells in high (>5 years, >10 hours per week) vs low (<5 years, <4-5 hrs/week) mobile phone users. The study included 50 male and 50 female users equally split between low and high users. The authors noted significant increases in MN frequency in high users, non-users of headphones, CDMA (vs GSM) users and for the same side vs opposite side of cell phone use buccal samples. [This study is somewhat suspicious since all comparisons balanced out to 50 versus 50, even for the side of the mouth analysis.]

Kundi et al. (2024) [61] exposed volunteers to low and high doses of a UMTS signal (n = 20 at 0.1 W/kg and n = 21 at 1.6 W/kg SAR) for 2 h on 5 consecutive days using headphones that allowed separate exposures to each side of the head. Before and three weeks after the exposure, buccal cells were collected from both cheeks and MN and other nuclear anomalies

reflecting mitotic disturbance and acute cytotoxic effects were scored. They found no evidence for induction of MN or nuclear buds but did find a significant increase of binucleated cells and karyolytic cells on the exposed side only. Their conclusions state that, under the present experimental conditions, RF fields did not cause acute chromosomal damage in oral mucosa cells but did disturb cell cycle and increase cytotoxicity. [It is important to consider that it takes between 7–21 days for a cell with an MN in the basal layer to emerge to the surface of the buccal mucosa, sometimes longer depending upon age and other factors. [62]]

4.2.2 Studies of genotoxicity in laboratory mammals

Pei et al. (2023) [63] investigated the potential impact of mobile phone radiation on bone marrow hematopoietic stem cells in 160 male Balb/C mice. Four groups of 40 mice were exposed to cellular phones as blank control (no phone), control (phone, no card), TD-SCDMA phone and LTE-Advanced phone. TD-SCDMA and LTE-Advanced groups saw an increase ($p < 0.05$) in the percentage of micronuclei in hematopoietic stem cells after 6 weeks of exposure and an increase in apoptosis in the same time frame. . [Using a cell phone for exposures introduces uncertainty in the level of exposure]

4.2.3 Studies of genotoxicity in mammalian cells

Misik et al. (2023) [64] exposed pooled peripheral blood mononuclear cells (PBMC) from three groups (young normal weight, young obese and older age normal weight individuals) exposed to different doses of 1.95 GHz RF-EMF (0.25, 0.5 and 1.0 W/kg, UMTS) and simultaneously or sequentially to different chemicals which can cause DNA damage via different molecular mechanisms (CrO₃, NiCl₂, benzo[a]pyrene diol epoxide (BPDE), and 4-nitroquinoline 1-oxide (4NQO)). They found no difference with regard to the background values for DNA damage in the three groups and no difference in young participants PBMC, but a significant increase of DNA damage (81% without and 36% with serum) in cells from older participants after 16 h radiation. In combined treatment experiments they found no impact of the UMTS-signal on chemically induced DNA damage in the different groups in general. However, a moderate decrease of DNA damage was seen in simultaneous treatment experiments for some chemicals.

Ravaioli et al. (2023) [65] analyzed DNA-methylation profiles of cervical cancer and neuroblastoma cell lines (HeLa, BE(2)C and SH-SY5Y) exposed to 900 MHz GSM-modulated RF-EMF. RF-EMF influenced DNA-methylation of LINE-1 and ribosomal repeats in terms of both average profiles and organization of methylated and unmethylated CpG sites, in different ways in each of the three cell lines studied.

Sannino et al. (2023) [56] investigated the effects of 1950 MHz LTE RF-EMF administered alone and in combination with mitomycin-C (MMC), a well-known cytotoxic agent, on Chinese hamster lung fibroblast (V79) cells at specific absorption rate (SAR) of 0.3 and 1.25 W/kg. No changes in chromosomal damage (MN formation), oxidative stress (ROS), and cell cycle

progression were detected following radiofrequency exposure alone, for all the experimental conditions tested and biological endpoints investigated. When radiofrequency exposure was followed by MMC treatment (20 h pre-exposure at 1.25 W/kg) there was a significant reduction of MMC-induced damage. No other significant changes were evident. [The authors suggest this demonstrates a protective effect against MN of pre-exposure to RF-EMF; the results do not support this conclusion]

Massaro et al. (2024) [66] evaluated cytotoxicity and genotoxicity of continuous or pulsed 1.6 GHz RF (SAR 0.4 W/kg, 2 h exposures) in primary human dermal fibroblasts (HDF). Biological effects were measured immediately after exposure or 24 hours later by analyzing the effects on cell cycle, ultrastructure, protein expression, mitotic spindle, CREST-stained micronuclei, chromosome segregation and gamma-H2AX/53BP1 foci. RF EMF modified morphological and phenotypical markers in the cells as well as the expression of different heat-shock proteins and phospho-AKT. No significant effect was observed on cell cycle, chromosome segregation, CREST-stained micronuclei and gamma-H2AX/53BP1 foci. Mitotic spindle anomalies were increased following exposure. [Although not explicit, it appears there were only 4 replicates per group, a small sample size].

Worel et al. (2024) [67] studied the impact of RF-EMF (1950 MHz, UMTS signal, 0.25, 0.5 and 1 W/kg) on DNA stability in an astrocytoma cell line (1321N1) treated simultaneously or sequentially with 4-nitroquinoline 1-oxide (4NQO), benzo[a]pyrene diol epoxide (BPDE), NiCl₂, or CrO₃. The compounds cause DNA damage via different molecular mechanisms. DNA damage was measured in single cell gel electrophoresis (comet) assays. The authors saw no significant alterations of chemically induced DNA migration by RF-EMF under all experimental series. They found a moderate reduction of basal and 4NQO-induced DNA damage from exposure to 0.25 W/kg but not at higher doses.

4.3 Other

Huang et al. (2023) [68] exposed OLN-93 rat oligodendroglia cells, HT-22 mouse hippocampal cells, and BV2 microglial cells continuous wave (CW-RF) or a pulse-modulated wave (PW-RF, 50 Hz pulse frequency, 1/3 duty cycle) pattern. RT-qPCR, and Western blot were used to examine the expression of C/EBP β and its related genes, markers of inflammation. The authors demonstrated that PW-RF significantly increased the mRNA level of C/EBP β in oligodendroglia but not in other types of cells. In addition, the expression of three isoforms and several interacting proteins and targeted genes of C/EBP β were markedly changed after 6-h PW-RF but not for CW-RF. [Increased inflammation markers could be the result of either oxidative stress or genotoxicity, but that cannot be confirmed here.]

4.4 Summary for Mechanisms of Carcinogenicity

The results of recent studies of changes in oxidative stress do not dramatically differ from the studies in my previous review in terms of design, methods or results. While some of the recent oxidative stress studies are negative, the majority are showing changes in oxidative stress or oxidative stress pathways. The same holds true for studies of genotoxicity. RF exposure emitted from commonly used devices is capable of influencing ROS production, mitochondrial imbalance and overall genotoxicity, but it must be considered that these effects may vary based on the experimental parameters. In fact, they may depend on the time of exposure to electromagnetic fields and on the animal type, tissue type, and cells origin.

Thus, my previous conclusions in these two areas remain the same:

There is sufficient evidence in the literature to conclude that oxidative stress is a possible mechanism by which RF causes cancer in humans.

Overall, there is sufficient evidence to suggest that genotoxicity, probably due to oxidative stress, is caused by RF and could be a mechanism by which cancer is induced by RF.

5.0 Overall Summary

There is no change to my overall opinion.

RF exposure probably causes gliomas 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.

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9 *Frequencies in Oligodendroglial Cells*. Int J Mol Sci, 2023. **24**(13).

10

Appendix I: Curriculum Vitae Christopher J. Portier, Ph.D.

CURRICULUM VITAE

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Education:
1981 Ph.D. (Biostatistics), University of North Carolina, Chapel Hill
1979 M.S. (Biostatistics), University of North Carolina, Chapel Hill
1977 B.S. (Mathematics), summa cum laude, Nicholls State University

Employment:
2021-present Member, Advisory Board, Animal-free Safety Assessment of Chemicals: Project Cluster for Implementation of Novel Strategies, European Commission H2020
2020-present **Chair**, Stakeholder Advisory Group, PrecisionTox, European Commission H2020 Program Grant Agreement No. 965406
2020-present **Member**, Executive Board, PrecisionTox, PrecisionTox, European Commission H2020 Program Grant Agreement No. 965406
2018-2020 **Scientific Advisor**, World Health Organization, Environment Program - Europe
2016-present **Scientific Advisor on Pesticide Policies**, multiple European Non-Government Organizations
2013-present **Consultant**, CJP Consulting LLC, Seattle, Washington
2013-2020 **Consultant** to various governmental agencies (multiple countries)
2013-2014 **Senior Visiting Scientist**, International Agency for Research on Cancer, Lyon, France
2013-present **Senior Contributing Scientist**, Environmental Defense Fund, New York City, NY
2010-2013 **Director**, National Center for Environment Health, Centers for Disease Control and Prevention, Atlanta, GA
2010-2013 **Director**, Agency for Toxic Substances and Disease Registry, Atlanta, GA
2009 – 2010 **Senior Advisor to the Director**, National Institute of Environmental Health Sciences and National Toxicology Program, Research Triangle Park, North Carolina.
2009 – 2010 **Visiting Scientist**, National Research Centre for Environmental Toxicology (EnTox), Queensland, Australia
2006 - 2009 **Associate Director**, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.
2006 - 2009 **Director, Office of Risk Assessment Research**, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.

1	1993 – 2010	Head, Environmental Systems Biology (originally Stochastic Modeling), Laboratory of
2		Molecular Toxicology, National Institute of Environmental Health Sciences, Research
3		Triangle Park, North Carolina.
4	2000 - 2006	Associate Director, National Toxicology Program , National Institute of
5		Environmental Health Sciences, Division of Intramural Research, Research Triangle
6		Park, North Carolina.
7	2000 - 2006	Director, Environmental Toxicology Program , National Institute of Environmental
8		Health Sciences, Division of Intramural Research, Research Triangle Park, North
9		Carolina.
10	2006-2007	Scientific Advisor to the Director , Public Health and the Environment Department,
11		World Health Organization, Geneva, Switzerland (detail from NIEHS – four months)
12	1993 - 2005	Chief, Laboratory of Computational Biology and Risk Analysis (originally the
13		Laboratory of Quantitative and Computational Biology), National Institute of
14		Environmental Health Sciences, Division of Intramural Research, Research Triangle
15		Park, North Carolina.
16	1996 - 2000	Associate Director for Risk Assessment , Environmental Toxicology Program National
17		Institute of Environmental Health Sciences, Division of Intramural Research,
18		Research Triangle Park, North Carolina.
19	1990 - 1993	Head, Risk Methodology Section , National Institute of Environmental Health
20		Sciences, Division of Biometry and Risk Assessment, Research Triangle Park, North
21		Carolina.
22	1987, 1992, 1990	Guest Scientist , German Cancer Research Center, Heidelberg, Germany.
23	1978 - 1990	Principal Investigator , National Institute of Environmental Health Sciences, Division
24		of Biometry and Risk Assessment, Research Triangle Park, North Carolina.
25	1977	Mathematician , Computer Sciences Division, Oak Ridge National Laboratory, Oak
26		Ridge, Tennessee.
27	1976	Undergraduate Research Trainee , Neutron Physics Division, Oak Ridge National
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29		
30	University Affiliations:	
31	2014 – 2021	Visiting Professor, Department of Toxicogenomics, Maastricht University, The
32		Netherlands
33	2013 – 2016	Honorary Professor, National Research Centre for Environmental Toxicology,
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35	2011 – present	Adjunct Professor, Department of Environmental Health, Emory University, Atlanta,
36		GA, USA
37	2009 – 2010	Visiting Professor, University of Queensland, Brisbane, Australia
38	1986 - 2007	Adjunct Professor of Biostatistics, University of North Carolina, School of Public
39		Health, Chapel Hill, North Carolina.
40	1990-1992	Adjunct Professor of Statistics, University of Waterloo, Waterloo, Ontario, Canada
41		
42	Honors & Awards:	
43		2016 Elected Fellow, Collegium Ramazzini
44		2013 President's Dream Green Team Award for "A Human Health Perspective on
45		Climate Change"
46		Fellow, World Innovation Foundation, 2006
47		Society of Toxicology, Risk Assessment Specialty Section, Paper of the Year, 2006
48		Society of Toxicology, Risk Assessment Specialty Section, Paper of the Year, 2005

Outstanding Risk Practitioner Award, International Society for Risk Analysis, 2000.
 Elected Fellow, International Statistical Institute, 2000.
 Outstanding Performance Award, National Institute of Environmental Health Sciences, numerous dates.
 Commendation for Sustained High Quality Work Performance, National Institute of Environmental Health Sciences, numerous dates.
 Merit Award, National Institute of Health, 1998.
 Board of Publications, Best Paper Award, Society of Toxicology, 1995.
 Distinguished Achievement Award, Section on Statistics and the Environment, American Statistical Association, 1995.
 Spiegelman Award presented by the American Public Health Association to the most outstanding public health statistician under the age of 40, 1995.
 Best-applied statistics paper, Centers for Disease Control, 1993.
 Elected Fellow, American Statistical Association, 1992.
 Elected Foreign Correspondent, Russian National Academy of Natural Sciences, 1992.
 First recipient of the James E. Grizzle Distinguished Alumnus Award, The Department of Biostatistics, The University of North Carolina, 1991.

Professional Societies Membership:

Society of Toxicology, American Public Health Association, International Statistics Institute, Bioelectromagnetics Society

Editorial Activities:

Editor in Chief - The Open Environmental Journal (2008 to 2010)
 Associate Editor – Frontiers in Predictive Toxicity (2010 to present)
 Associate Editor - Environmental Health Perspectives (1987-2006)
 Associate Editor - Risk Analysis: An International Journal (1989-2003)
 Editorial Board – Environmental and Ecological Statistics (2004-2007)
 Associate Editor – Statistics in Medicine (1998-2002)
 Associate Editor - Biometrics (1997-99)
 Editorial Board Member/Reviewer (different dates): Biometrika, Cancer Research, Communications in Statistics, Fundamental and Applied Toxicology, Journal of Applied Toxicology, Journal of the American Statistical Association, Journal of Toxicology and Environmental Health, Science, Mathematical Biosciences, Journal of Mathematical Biology, Carcinogenesis, Science, PNAS, Toxicological Sciences and others

Advisory & Review Committees:

2019-present	Member, UCSF PHRE Science Response Network
2016-2021	Member, World Health Organization Regional Office Europe, Setting research priorities in environment and health
2015 – 2016	Member, Committee to Review the Draft Interagency Report on the Impacts of Climate Change on Human Health in the United States, National Research Council, National Academy of Sciences, USA
2010 – 2016	Member, Science Advisory Group on Electromagnetic Fields and Health, Netherlands Organisation for Health Research and Development
2009 – 2010	Coordinating Lead Author, Interagency Working Group on Climate Change and Health

1	2009 – 2013	Member, Institute of Medicine Roundtable on Environmental Health Sciences
2		Research and Medicine
3	2009 – 2012	Member, National Academies of Science Roundtable on Science and Technology for
4		Sustainability
5	2009	Member, WHO Advisory group on the health implications of the use of DDT to
6		reduce risks of malaria.
7	2005 – 2010	Chair, Subcommittee on Toxics and Risk, President's National Council on Science and
8		Technology
9	1997 - 2012	Advisor, <i>World Health Organization</i> , International Program on Chemical Safety, EMF
10		Project.
11	2008 – 2010	Member, Environmental Protection Agency, Science Advisory Board
12	2007 – 2010	Member, International Life Sciences Institute, Health and Environmental Sciences
13		Institute, Subcommittee on Susceptible Populations
14	2008	Center Review Committee, Canadian National Science and Engineering Research
15		Council Chair in Risk Assessment
16	2008	Chair, International Agency for Research on Cancer Monographs Advisory Group,
17		Lyon, France
18	2008	Advisory Group, Center for Environmental Oncology, University of Pittsburgh
19		Cancer Institute
20	2007.	Chair, WHO Workshop on Low Cost Options for Reducing Exposures to ELF-EMF,
21		Geneva
22	2007.	Invited Participant, International Program on Chemical Safety Workshop on
23		Aggregate and Cumulative Risk Assessment, Washington, DC.
24	2006	Rapporteur, International Agency for Research on Cancer, Scientific Advisory Group
25		to Plan Volume 100 of the IARC Monograph Series
26	2005	Chair, International Agency for Research on Cancer, Scientific Advisory Board on the
27		Preamble to the Cancer Monograph Series
28	2005	Chair, World Health Organization Expert Panel on Health Criteria Document for
29		Extremely Low Frequency Electric and Magnetic Fields
30	2003 – 2005	Co-Chair, Subcommittee on Health and Environment, President's National Council on
31		Science and Technology
32	2003	Ad-Hoc member, EPA Science Advisory Board, Review of Children's Cancer Risk
33		Assessment Supplement to Cancer Guidelines
34	2002 – 2006	Co-Chair, Subcommittee on Mercury, President's National Council on Science and
35		Technology
36	2000 – 2007	Member, Finish Academy of Sciences Centers of Excellence Program Science Advisory
37		Committee
38	2000	Reviewer, <i>Congressional Research Service, Library of Congress</i> ; Research needs
39		relevant to children's environmental health risks.
40	1998 - 2004	Member and Chair, <i>Environmental Protection Agency</i> , FIFRA Science Advisory Panel.
41	1997 - 2006	Member, National Occupational Research Agenda Team, <i>National Institute of</i>
42		<i>Occupational Safety and Health</i> .
43	1995 - 2000	Advisor, <i>Australian Health Council</i> , Risk Assessment Methodology, Member <i>NHMRC</i>
44		Steering Committee on Cancer Risk Assessment Guidelines.
45	1992 - 2000	Member, <i>EPA Dioxin Reassessment Working Group</i> .
46	1985 - 2007	Thesis director for graduate students, Department of Biostatistics, <i>University of</i>
47		<i>North Carolina - Chapel Hill, North Carolina</i> .
48	1997	Advisor, <i>Netherlands National Health Council</i> , Risk Assessment Methodology.

1 1997 Reviewer, *Air Force Office of Scientific Research*.
2 1996 - 1997 Temporary Advisor, *World Health Organization*, Expert Committee on Food
3 Additives.
4 1996 Advisor, *Environmental Protection Agency*; Evaluation of the benchmark dose
5 methodology.
6 1996 Advisor, *Environmental Protection Agency*; Evaluation of risks from exposure to PCBs.
7 1996 Expert Review Committee, *Environmental Protection Agency*; Cancer dose-response
8 for PCB's.
9 1995 - 1996 Member, *California Environmental Protection Agency*, Risk Assessment Advisory
10 Committee.
11 1994 - 1997 Science Advisory Panel, *Public Broadcasting System Production "Poisons in the
12 Womb"*.
13 1991 - 1995 Ad-Hoc Member, *Environmental Protection Agency*, Science Advisory Panel.
14

15 **Legislative Hearings:**

16 Pesticide Presentation, European Parliament, Brussels, May 2023
17 Glyphosate Hearing, European Parliament, Brussels, October, 2017
18 Glyphosate Carcinogenicity, European Parliament, Brussels, December 2015
19 Glyphosate Carcinogenicity, German Parliament, Berlin, July 2015
20 Lead and Children's Health, Senate Committee on Environment and Public Works,
21 July, 2012
22 Asthma and Children's Health, Senate Committee on Environment and Public Works,
23 May, 2012
24 Contaminated Drywall, Senate Committee on Commerce, Science and
25 Transportation, December, 2012.
26 Camp Lejeune Contaminated Drinking Water, House Committee on Science and
27 Technology, September, 2010.
28 Autism and Vaccines, House Committee on Government Reform, December, 2002.
29

30 **US Government Service Activities:**

31 Member, President's Task Force on Environmental Justice 2010-2013
32 Member, President's Task Force on Children's Environmental Health 2009-2013
33 Member, National Toxicology Program Executive Committee 2010-2013
34 Financial Support and International Press Conference for research on "The Health
35 Benefits of Tackling Climate Change" appearing as a series in *Lancet*, November 25,
36 2009
37 Organizing Committee, White House Stakeholder briefing on Climate Change and
38 Human Health, Old Executive Office Building, November 2009.
39 Member, US Delegation, World Climate Congress, Geneva (September 2009)
40 Member, US Delegation, Global Risk Communication Dialogue (2008-2009)
41 Member, NIEHS Corrective Action Plan Management Committee (2008-2009)
42 Primary focus, all interagency activities on hazards and risk (2006 to present)
43 Co-Organizer, NIEHS/EPA Workshop on Children's Environmental Health, RTP, NC,
44 January, (2007)
45 Co-Organizer, NIEHS/NTP Workshop on the Identification of Targets for the HTS
46 Roadmap Project (2007)
47 Coordinator, NIEHS/EPA Review of the Children's Environmental Health Centers
48 Program (2006-2007)

Organizing Committee, Global Environmental Health Initiative, NIEHS (2006 to 2009)
 NIEHS Leadership Council (2005 to 2009)
 Organizer, formal collaborative agreements between NTP and Ramazzini Foundation
 (2001 to 2006)
 Organizer, formal collaborative agreements between NTP and Korean NTP (2002 to
 2006)
 NIEHS Title 42 Review Committee (2003 to 2004)
 NIEHS Executive Committee and Operations Update Committee (2000 to 2005)
 NIEHS Leadership Retreats, DERT Retreats, DIR Retreats (all years since 1997)
 Presenter, NIEHS-sponsored National Academy of Sciences Committee on Emerging
 Issues in Environmental Health, November, 2001
 Organizer and presenter, National Toxicology Program Executive Committee
 Meetings (multiple dates since 2000)
 Organizer and presenter, National Toxicology Program Board of Scientific Counselors
 (multiple dates since 1998)
 Organizer, Joint NIEHS/US Geological Survey Interagency Program on Exposure
 Assessment, April 2001 to present)
 Organizer, US-Vietnam Scientific Conference on the Health and Environmental
 Effects of Agent Orange/Dioxin in Vietnam, March, 2002
 Organizing Committee, National Toxicology Program/EPA/FDA Scientific Conference
 on the Allergenicity of Genetically Modified Food, November, 2001
 NIEHS Town Hall Meeting, Los Angeles California, November, 2001
 NTP Research Directions, NAEHSC, Research Triangle Park, NC. May, 2001.
 NCI Study Section Center Presite Meeting, Seattle, Washington, January, 2001.
 Program committee member, *NIEHS/Colorado State University* conference on the
 Application of Technology to Chemical Mixture Research, 2001.
 Coordinating Core Committee, National Center for Toxicogenomics, NIEHS, 2000 to
 present
 Organizer, Joint US-Vietnam Consultation on Research on Agent Orange Health
 Effects in Vietnam. Singapore, 2000
 ICCVAM/NICEATM, Up-and-Down Procedure Peer Review Meeting, 2000.
 Chairman, *NIEHS* Risk Assessment Research Committee, 1995-present.
 Discussant, *NIEHS/PNNL* Workshop on Human Biology Models for Environmental
 Health Effects, 2000.
 Risk Assessment Coordinator, *NIEHS* US *RAPID* Program for the Evaluation of Health
 Risks from Exposure to Electric and Magnetic Fields, 1996-99.
 Organizer and Chair, Four Public Comment Sessions on the report of the *NIEHS/DOE*
 Working Group on the Health Effects of Exposure to Electric and Magnetic Fields,
 1998.
 Organizer and Co-Chair, *NIEHS/DOE* Working Group on the Health Effects of
 Exposure to Electric and Magnetic Fields, 1998.
 Scientific Organizing Committee, *NIEHS* Workshop on Risk Assessment Issues
 Associated with Endocrine Disrupting Chemicals, 1998.
 Organizer, *NIEHS/DOE* Science Research Symposium on the Health Effects of
 Exposure to Electric and Magnetic Fields I: Biophysical Mechanisms and *In Vitro*
 Experimentation, 1998.
 Organizer, *NIEHS/DOE* Science Research Symposium on the Health Effects of
 Exposure to Electric and Magnetic Fields II: Epidemiological Findings, 1998.

Organizer, *NIEHS/DOE Science Research Symposium on the Health Effects of Exposure to Electric and Magnetic Fields III: In Vitro and Clinical Research Findings*, 1998.
 Head, Toxicokinetics Faculty, *NIEHS*, 1994-97.
 Coordinator/Director, *NIEHS/ATSDR Interagency Course on Mechanistic Modeling in Environmental Risk Assessment*, 1996.
 Organizer, *NIEHS/EPA Workshop on Research Priorities for New Risk Assessment Guidelines*, 1996.
 Co-Organizer, *National Institute of Statistical Sciences, NIEHS/EPA Workshop on Mechanistic Modeling in Risk Assessment*, 1995.
 Scientific Coordinator and Mission Director, *NIEHS "Mission to Vietnam"* to assess the potential for scientific collaboration on the impact of Agent Orange on the Vietnamese Population, 1995.
 Chairman, *NIEHS Computer Science Focus Group*, 1995.
 Discussant, National Toxicology Program Workshop on Mechanistic Modeling in Toxicology, *NIEHS*, 1995.
 Discussant, National Toxicology Program Workshop on Mechanisms of Carcinogenesis, *NIEHS*, 1995.
 Co-Organizer, International Conference on The Role of Cell Proliferation in Carcinogenesis, co-sponsored by *NIEHS, The Chemical Industry Institute of Toxicology, The International Life Sciences Institute and The American Industrial Health Council*, 1992.
 Organizer and Director, Scientific Basis of Animal Carcinogenicity Testing, Moscow, Russia, co-sponsored by the *International Agency for Research on Cancer, NIEHS, Health and Welfare Canada and The All-Union Cancer Research Center*, 1991.
 Chairman, Computer Technology Advisory Forum, *NIEHS*, 1989.
 Organizer and Director, Design and Analysis of Long-Term Animal Carcinogenicity Experiments, Lyon, France, co-sponsored by the *International Agency for Research on Cancer and the NIEHS*, 1988.

Extramural Activities:

Member, NRC Committee to review the Draft Interagency Report on the Impacts of Climate Change on Human Health in the United States, Washington, DC, 2015
 Expert Scientist, International Agency for Research on Cancer Monograph Meeting on Some Organophosphate Pesticides and Herbicides, Lyon, France, March, 2015
 Overall Chair, International Agency for Research on Cancer Monograph Meeting on Diesel and Gasoline Engine Exhausts and related compounds, Lyon, France, June, 2012
 Advisor to Wellcome Trust at "International Research Futures Symposium on Global Change, Economic Sustainability, and Human Health", London, England, March, 2012.
 Expert Panel Member for review of Hollings Marine Laboratory, National Oceanographic and Atmospheric Agency, Charleston, USA, February, 2012.
 Chair, Mechanism Subgroup, International Agency for Research on Cancer Monograph Meeting on Radiofrequency Electric and Magnetic Fields, Lyon, France, May, 2011
 Advisor, Greek Ministry Health, Working group on hexavalent chromium in the environment, January, 2011

Member, WHO Consultation on Human Health Risks from DDT, Geneva, Switzerland,
 November, 2010
 Associate Editor, *Frontiers in Predictive Toxicity*, 2010 – 2011
 Scientific Advisor, Health Investigation Levels Workshop, Canberra, Australia,
 January, 2010
 Chair, IARC Working Group, IARC Monograph 100-G, Lyon, France, October, 2009
 Scientific Organizing Committee, VII World Congress on Alternatives and Animal Use
 in Life Sciences, Rome, Italy, September, 2009
 Chair, Research Directions Working Group, World Health Organization Consultation
 on Global Research on Climate Change and Health, October, 2008.
 Editor-in-Chief, *The Open Environment Journal*, May 2008-August, 2010
 Member, EPA Science Advisory Board, July, 2008-present
 Working Group Member, IARC Monograph 98 - Fire-fighting, Painting and Shift-
 work, Lyon, France, November, 2007
 Chair, WHO Extremely Low Frequency Magnetic and Electric Fields Workshop on
 Intervention Strategies, June, 2007
 Special Advisor to the Director, Program on Public Health and the Environment,
 WHO, Geneva, May-July, 2007
 Member, International Life Sciences Institute Working Group on Susceptible
 Populations, March, 2007 – present
 Special Advisor to the Director, Program on Public Health and the Environment,
 WHO, Geneva, November, 2006-January, 2007
 Breakout Group Chair, International Workshop on Uncertainty and Variability in
 PBPK Modeling, RTP, NC USA, October, 2006
 Member, Health Effects Sciences Institute Committee on Sensitive Subpopulations
 and Groups, Washington, DC, 2006 to present
 Rapporteur, Steering Committee for developing the 100th Monograph of the
 International Agency for Research on Cancer, Lyon, France, September, 2006
 Co-Organizer, parallel workshops on the advancement of PBPK modeling in risk
 assessment, Research Triangle Park, November, 2006, Corfu, Greece, April, 2007.
 Organizer, *Alternative Models in Developmental Neurotoxicity*, Alexandria, Virginia,
 March, 2006.
 Organizer, NTP High Throughput Screening Workshop, Washington, DC, December,
 2005
 Organizer, IS RTP Meeting on Alternative Methods in Toxicology, Baltimore,
 Maryland, November, 2005
 Organizer, NTP 25th Anniversary Meeting, Washington, DC, May, 2005
 Organizer, IPCS/WHO Workgroup on Dose-Response Modeling, Geneva,
 Switzerland, September, 2004
 Organizer, Consultation on harmonization of toxicological research between the NTP,
 Ramazzini Foundation and the European Union, European Congress of Toxicology,
 Florence, Italy, September, 2003.
 Member, WHO Workgroup on the epidemiology of cellular phone toxicity, Tsukuba,
 Japan, September, 2003.
 Program Committee, 12th International Conference on Global Warming, Boston,
 Massachusetts, May 2003
 Program Committee, International Conference on Cancer Risk Assessment, Athens,
 Greece, August, 2003
 Chair, WHO Public Consultation on Risk Communication, Luxembourg, February,
 2003.

Chair, WHO Committee on Establishing a Plan for Implementation of the
 Precautionary Principle in Risk Management. Luxembourg, February, 2003.
 Presenter (on behalf of US Government), National Academy of Sciences Panel on the
 Use of Third Party Toxicity Research with Human Research Participants, December,
 2002
 Member, US Science Delegation, United Nations Environmental Program
 Consultation on Organic Mercury, September, 2002
 Science Panel Member, IARC Carcinogenicity Review of ELF-EMF, Lyon, France, June,
 2001.
 Reviewer, Finish Ministry of Health Centers of Excellence Program, Helsinki, April,
 2001.
 EPA dioxin reassessment peer review workshop and public comment session,
 Washington, DC, 2000.
 Organizer: Dioxin Dose-Response Working Group Meeting, Fort Collins, Colorado,
 February, 2000.
 Chair, Spiegelman Award Committee, *American Public Health Association*, 1998.
 Chair, *Bioelectromagnetics Society* Symposium on the use of Transgenic Animals in
 Evaluating Health Risks from Exposure to Cellular Phones, St. Petersburg, Florida,
 1998.
 Member, *World Health Organization* International Program on Chemical Safety,
 Workshop on Issues in Cancer Risk Assessment, 1998.
 Advisor, *Joint Committee on Food Additives, World Health Organization/Food and
 Agriculture Organization*. Evaluation of certain food additives and contaminants
 Member, US Government Methylene Chloride Risk Characterization Science
 Committee, 1996-1998.
 Scientific Organizing Committee, *Colorado State University* Workshop on Biomedical
 Advances on Chemical Mixtures, 1997.
National Academy of Sciences, Institute of Medicine, Committee on Funding Future
 Agent Orange Research in Vietnam, 1996.
 Discussant, Workshop on the role of Endocrine Disruptors in Human Health, 1995.
 Advisor to *Australian Health Council* on Risk Assessment Methodology, Member
NHMRC Steering Committee on Cancer Risk Assessment Guidelines
 Participant, International Program on Chemical Safety of the *World Health
 Organization* Workshop on Chemical Risk Assessment, London, England, 1995.
 Participant, *IARC* Workshop on Receptor-Mediated Carcinogenesis, Lyon, France,
 1994.
 Co-Organizer, Symposium on Quantitative Risk Assessment, *German Cancer Research
 Center*, Heidelberg, Germany, 1993.
 Participant, *IARC* Monograph on Risk Assessment Methodology, *International Agency
 for Research on Cancer*, Lyon, France, 1993.
 Thesis advisor for graduate student, *University of Waterloo*, Waterloo, Ontario,
 Canada. 1991-93.
 Co-Organizer, *Russian Academy of Sciences* Informatics and Cybernetics Research
 Award, 1992.
 Official Observer, *IARC* Monograph on the Biological Effects of Ultraviolet Radiation,
International Agency for Research on Cancer, Lyon, France, 1992.
 Member, *International Life Sciences Institute*, Dose-Response Working Group, 1991.
 Participant in Banbury Conference on Human Health Risks from Exposures to Dioxins,

Banbury Conference Center, Cold Spring Harbor, New York, 1990.
Co-Chairman, Session on Biostatistical Developments in Cancer Research, 15th
International Cancer Congress, Hamburg, Germany, 1990.
Participant in Environmental Protection Agency Workshop on Risk Assessment
Guidelines, Virginia Beach, Virginia, 1989.

Invited Presentations (present-1999)

“A Comprehensive Analysis of Animal Carcinogenicity Data for Glyphosate”, Coalition on Health and
the Environment, Webinar, May 13, 2020
“Data Pertaining to Glyphosate Carcinogenicity“, University of Washington, Seattle, Washington,
November, 2018
“From Here to There: How does a Biostatistician Become Director of a National Health Agency”,
University of Washington, Seattle, Washington, November, 2018
“Introduction to Cancer Modeling”, Maastricht University, June, 2018
“Data Pertaining to Glyphosate Carcinogenicity“, Utrecht University, April, 2018
“Glyphosate Cancer Risks and Failure of the Pesticide Regulatory Process”, Utrecht University, April
2018
“In Vitro to In Vivo Concordance for Toxicity Prediction and Use in Safety Assessments”, Washington,
DC, October 2017
“Glyphosate Cancer Classification”, EAPCCT Meeting, Basel, Switzerland, May, 2017
“Hide and Seek: Glyphosate Cancer Risks and the European Pesticide Regulatory Process”, Maastricht
University, Netherlands, May, 2017
“Carcinogenicity of Glyphosate: A Systematic Review of the Available Evidence”, European Chemical
Agency, November 2016
“Glyphosate Carcinogenicity”, Swiss Society of Toxicology, Basel, November, 2016
“Evaluating Health Effects at Toxic Dump Sites”, COST Meeting, Budapest, Hungary, March, 2016
“Systems Toxicology; Real World Applications”, SwissTox Meeting, Diablerettes, Switzerland, January,
2016
“Glyphosate Carcinogenicity”, Concerned Scientists of Switzerland Annual Meeting, Zurich, December,
2015
“Lunch debate in the EU Parliament on Glyphosate”, EU Parliament Building, Brussels, Belgium,
December, 2015
“Should the precautionary principle be invoked for RF-EMF”, BIOEM 2015, Asilomar, CA, USA, June
2015
“IARC Monograph Review Process and Glyphosate”, Deutscher Bundestag, Berlin, Germany, June 2015
“The Exposome: Why does it matter for public health?”, Association of Public Health Laboratories, Dr.
Katherine Kelly Distinguished Lecture, Indianapolis, USA May 2015
“A bioinformatics/biostatistics approach to systems toxicology”, Maastricht University, Maastricht,
Netherlands, March, 2015
“Mechanistic Data, Cellular Pathways, and Cancer Classification”, 39th Annual Toxicology Forum
Meeting, Washington D.C., USA, January, 2015
“Current Issues in Environmental Public Health”, Nicholls State University, Thibodaux, LA, April, 2014
“Review of Approaches to the Quantification of Risks”, International Agency for Research on Cancer,
Lyon, France, November, 2013
“The Gene-Environment-Disease Interactome”, Maastricht University, September, 2013
“Toxicogenomics and Electromagnetic Fields”, BEMS Annual Meeting, Thessaloniki, Greece, June 2013
“Toxicogenomics and Risk Assessment”, International Symposium on Toxicogenomics and Human
Health, Paris, France, May, 2013

1 “Extreme Weather, Climate and Health: Putting Science into Practice”, Climate and Health Conference,
2 Washington, DC, February, 2013

3 “Biofuels and Human Health: CDC’s activities”, Our Energy Future and Health, National Academy of
4 Sciences, Washington, DC, USA, January, 2013

5 “Global Environmental Health”, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD,
6 USA, January, 2013

7 “Addressing Superfund Sites”, SBRP Annual Meeting, Washington, DC, October, 2012

8 “The future of cancer risk assessment”, 6th NCC International Symposium- Management of Carcinogenic
9 Hazard: Recent Progress and Future Perspectives, Seoul, Korea, June, 2012

10 “ATSDR Reorganization and New Directions”, National Toxicology Program Executive Committee,
11 Washington, DC, May, 2012

12 “Hydraulic Fracturing, National Concerns”, Institute of Medicine Roundtable on Environmental Health,
13 Washington, DC, April, 2012.

14 “Using Health Impact Assessment to Guide National Policy”, National Health Impact Assessment
15 Meeting, Washington, DC, April, 2012

16 “The Great Debate”, Society of Toxicology Meetings, San Francisco, USA, March, 2012.

17 “CDC and Environmental Health”, Society of Toxicology Meetings, San Francisco, USA, March, 2012.

18 “Keynote Address”, National Climate Assessment Initial Workshop, Charleston, SC, February, 2012.

19 “Integration of Climate, Weather and Health”, American Meteorological Society, New Orleans, USA,
20 January, 2012.

21 “Biomonitoring and Environmental Health”, CINVESTAV, Mexico Department of Health, Mexico City,
22 Mexico, January, 2012.

23 “What is health risk assessment”, International Conference on EMF and Health, Brussels, Belgium,
24 November, 2012.

25 “Risks from multiple chemicals in polluted communities”, International Conference on Chemical
26 Mixtures, Wasington, USA, October, 2011.

27 “Healthy Homes: The Scientific Support”, National Healthy Homes Conference, Denver, CO, June, 2011

28 “Climate Change and Human Health”, Keynote Address, Yale School of Public Health Annual Alumni
29 Day, New Haven, Ct, June, 2011

30 “Environmental Public Health”, Keynote Address, International Conference on Sustainable Remediation,
31 Amherst, MA, June, 2011

32 “The gene-environment disease interactome for humans”, Society of Toxicology Annual Meeting,
33 Washington, DC, March, 2011

34 “Environmental Exposure Science in the 21st Century”, NAS Committee on Human and Environmental
35 Exposure Science in the 21st Century, Washington, DC, February, 2011

36 “Human Health Effects and the Impact of Climate Change on Our Oceans”, Annual National Conference
37 on Science, Policy, and the Environment, Washington, Dc, January, 2011

38 “A Vision for the National Center for Environmental Health and Agency for Toxic Substances and
39 Disease Registry”, NAS Committee on Emerging Issues in Environmental Health, Washington, DC,
40 December, 2010

41 “Predicting Health Risks Using Gene-Expression Data”, EPA NextGEN Workshop, Research Triangle
42 Park, NC, October, 2010

43 “Building Sustainable Environments”, NAS Workshop: Pathways to Sustainable Development, Atlanta,
44 GA, September, 2010

45 “Future Directions in Environmental Public Health”, State Environmental Health Directors Annual
46 Meeting, Portland, OR, September 2010

47 “The gene-environment disease interactome for humans”, AACR Workshop on The Future of Molecular
48 Epidemiology, Miami, Fl, June, 2010

49 “Emerging methods for determining chemical hazards”, Keynote Address, Human Health Hazard
50 Indicators Workshop, Sacramento, March, 2010.

1 “Dose-response and risk assessment considerations of melamine in infants”, Society of Toxicology
 2 Annual Meetings, Sal Lake City, March, 2010
 3 “The gene-environment disease interactome for humans”, University of Queensland Centre for Clinical
 4 Research, Brisbane, Australia, February, 2010
 5 “Research Needs for Climate Change and Human Health”, Queensland Health and ENTOX, February,
 6 2010
 7 “Using systems biology to develop the gene-environment disease interactome for humans”, ENTOX,
 8 Brisbane, Australia, December, 2009
 9 “Quantifying the health risks of dioxins and dioxin-like compounds”, ENTOX, January, 2010
 10 “Epigenetics and its use in toxicology and risk assessment”, Keynote Address, Australian College of
 11 Toxicology and Risk Assessment Annual Meeting, Canberra, December, 2009.
 12 “The changing shape of regulatory toxicology in the United States”, Office of Chemical Safety &
 13 Environmental Health, Canberra, Australia, December, 2009
 14 “The changing shape of regulatory toxicology in the United States”, NICNAS, Sydney, Australia,
 15 December, 2009
 16 “Development and Use of the Gene-Environment-Disease Interactome for Humans”, 60th Anniversary of
 17 the Department of Biostatistics, University of North Carolina, Chapel Hill, NC, October, 2009
 18 “Using systems biology to develop the gene-environment disease interactome for humans”, Seventh
 19 World Congress on Alternatives and Animal Use in Life Sciences, Rome, Italy, September, 2009
 20 “Use of ‘Omics’ Technologies to Enhance Risk Assessment”, International Workshop on Genomics in
 21 Cancer Risk Assessment, Venice, Italy, August, 2009.
 22 “Proteomics, Genomics and the Human Gene-Environment-Disease Interactome”, First International
 23 Radiation Proteomics Workshop, Munich, Germany, May, 2009.
 24 “Molecular Biology and Regulatory Toxicology”, Society of Toxicology, Annual Meeting, Baltimore,
 25 Maryland, March, 2009.
 26 “Using Modern Research Tools to Enhance Regulatory Toxicology”, SOT Satellite Meeting on
 27 Development Of Toxicological And Environmental Public Health Infrastructures In Africa:
 28 Understanding The Premise And Mapping The Approach, Baltimore, MD, March 2009.
 29 “Health Implications of Climate Change”, Navy and Marine Public Health Conference, Norfolk, Va.,
 30 March, 2009.
 31 “Climate Change and Minority Health”, Second Annual National Conference on Health Disparities,
 32 University of the Virgin Islands, St. Croix, December, 2008.
 33 “Climate Change and Human Health”, the Environmental Mutagen Society Annual Meetings, Puerto
 34 Rico, October, 2008.
 35 “Climate Change and Human Health”, The Puerto Rico Chamber of Commerce, October, 2008.
 36 “Using Stem Cells in Environmental Health Research”, California Stem Cells and Predictive Toxicology
 37 Initiative Workshop, Berkeley, CA, July, 2008.
 38 “Developing the Disease-Environment Interactome”, IMBA-FGF Workshop on Developing Omics
 39 Technologies to Assess Unclear Risks”, Berlin, Germany, May, 2008
 40 “Environmental Systems Biology”, 40th Annual Interface Symposium on the interface between computer
 41 science and statistics, Durham, NC, May, 2008
 42 “Environmental causes of childhood leukemia and modern environmental health research”, Summary
 43 Address, ICNIRP/WHO/BS Symposium on Research Advances in Childhood Leukemia, Berlin,
 44 Germany, May, 2008.
 45 “Using systems biology as a tool to understand environmental health”, Atlantic Coast Symposium on the
 46 Mathematical Sciences in Biology and Medicine, Raleigh, NC, April, 2008.
 47 “Biological networks and high-throughput screening”, Society of Toxicology, Seattle, Washington,
 48 March, 2008.
 49 “Environment and cancer: strategies for identifying new hazards”. Workshop on the Environment and
 50 Cancer, American Cancer Society, Atlanta, Georgia, USA. January, 2008.

1 “A systems approach to human health risk assessment”, Environmental Health Sciences Decision
 2 Making: Risk Management, Evidence and Ethics. US National Academy of Sciences, Washington,
 3 DC, USA. January, 2008.
 4 “Finding targets for high-throughput screening linking genetics, genomics, pathways and human disease”.
 5 NTP Biological Screening Program Seminar Series. January, 2008.
 6 “The forest for the trees: A systems approach to environmental health research”. Keynote Address, KTL-
 7 DEH Center of Excellence Program, Lamalo, Finland. December, 2007.
 8 “Toxicology for the 21st Century”, ECNIS Research Colloquium, Engelheim, Germany, October, 2007.
 9 “Evidence-Based Decision Making in Public Health”, First International Workshop on Evidence-Based
 10 Toxicology, Cuomo, Italy. October, 2007.
 11 “Genes, Pathways and Diseases”, World Health Organization Headquarters, Geneva, Switzerland.
 12 September, 2007
 13 “Pharmacokinetics and dynamics of ketamine in horses”, University of Bern, June, 2007
 14 “Uncertainty and Variability in Risk Assessment”, NAS Workshop on Characterizing Uncertainty:
 15 Subgroup on Uncertainty in Estimating Low-Dose Risk from High-Dose Data, June, 2007
 16 “The utility and interpretation of high-throughput screening data in risk assessment with focus on the
 17 NTP programs”, International Workshop on “Evaluating upstream endpoints for improved decision
 18 making and risk assessment”, Berkeley, Ca. May, 2007
 19 “Future directions of risk assessment at the World Health Organization”, International Program for
 20 Chemical Safety Harmonization Steering Committee Meeting, Berlin, Germany, May, 2007
 21 “Chipping away at environmental health risk assessment”, GEMS annual meeting, Research Triangle
 22 park, NC, April, 2007
 23 “The forest for the trees: A systems approach to environmental health”, National Conference on Science,
 24 Policy and the Environment, Washington, DC, February, 2007
 25 “Emerging technologies and their application in risk assessment”, National Research Council Standing
 26 Committee on Risk Analysis Issues and Reviews, Washington, DC, December, 2006
 27 “Stochastic systems biology modeling”, International Congress on Systems Biology, Tokyo, Japan,
 28 October, 2006
 29 “Acceptable risk: environmental health research and public health”, National Symposium on Acceptable
 30 Risk in Clinical Medicine, RTP, NC, September, 2006
 31 “Stochastic Systems Biology”, Society for Industrial and Applied Mathematics Annual Meeting, Raleigh,
 32 NC, August, 2006.
 33 “Identifying and Quantifying Gene Interaction Networks”, Academia Sinica, Tapei, Taiwan, April, 2006.
 34 “Estimating Health Risks from Environmental Exposures”, Medical School, Cheng Kung National
 35 University, Taiwan, April, 2006.
 36 “Systems Biology and Environmental Health Research”, National Institute of Environmental Studies,
 37 Tsukuba, Japan, April, 2006.
 38 “Cancer Research and Risk Assessment: Looking to the Future”, National Cancer Institute, Japan, April,
 39 2006
 40 “Cancer Research and Risk Assessment: Looking to the Future”, Keynote Address, Cancer and the
 41 Environment Symposium, Duke University, March, 2006
 42 “Environmental Systems Biology”, Mount St Mary’s College, Maryland, March, 2006.
 43 “Bioinformatics in High Throughput Screening: A proposal”, ILSI Annual Meeting, Puerto Rico,
 44 January, 2006.
 45 “Gene Regulatory Networks in Cancer Risk Assessment, German Cancer Research Center, Heidelberg,
 46 Germany, December, 2005.
 47 “Alternative Methods in Toxicology; Problems and Solutions”, Keynote Address, Workshop on
 48 Alternative Methods in Toxicology, Baltimore, Maryland, November, 2005
 49 “Future Directions of the National Toxicology Program”, American Public Health Association Annual
 50 Meeting, New Orleans, Louisiana, November, 2005.
 51 “Risk Assessment”, A Mini-Course, University of Finland, Kuopio, Finland, October, 2005.

1 “Mechanism-Based Modeling as an Alternative to Animals in Toxicology”, 5th World Conference on
 2 Alternatives To Animals Used in the Life Sciences, Berlin, Germany, August, 2005.
 3 “Environmental Systems Biology”, Toxicology Forum, Aspen, Colorado, July, 2005.
 4 “Mechanistic Implications of Modifiers of Chemical Toxicity”, Keynote Lecture, Workshop on Modifiers
 5 of Chemical Toxicity: Implications for Human Health Risk Assessment, Poros, Greece, June, 2005.
 6 “Future Directions in Screening for Toxicants”, Committee on the Future of Toxicology, National
 7 Academy of Sciences, Washington, DC, May, 2005.
 8 “Toxicogenomics and the Future of Cancer Risk Assessment”, International Agency for Research on
 9 Cancer, Lyon France, May, 2005.
 10 “Statistical Contributions to Federal Advisory Committees”, Eastern North American Region of the
 11 Biometrics Society Annual Meeting, Austin, Texas, March, 2005.
 12 “The Future of Long-Term Animal Carcinogenicity Studies”, Invited Debate, Society of Toxicology
 13 Annual Meetings, New Orleans, Louisiana, March, 2005.
 14 “Future Directions of the National Toxicology Program”, Society of Toxicology Annual Meetings, New
 15 Orleans, Louisiana, March, 2005.
 16 “Role of the National Toxicology Program in Risk Assessment”, Federal-State Oncology and Risk
 17 Analysis Committee, Madison, Wisconsin, October, 2004.
 18 “Identifying and Quantifying Cancer Risks using Mechanistic Data”, Cancer and The Environment: NCI
 19 Science Writers Seminar, National Institutes of Health, Bethesda, Maryland, October, 2004.
 20 “Toxicogenomics and the Future of Risk Assessment”, Office of Science and Technology Policy,
 21 Washington, DC, August, 2004
 22 “A Vision for the National Toxicology Program”, Toxicology Forum, Aspen, Colorado, June, 2004
 23 “Health and Environment”, Joint Program on Climate Variability and Human Health, Atlanta, Georgia,
 24 March, 2004
 25 “Dose-Response Analysis of Toxicogenomic Data”, NIEHS Toxicogenomics Faculty, Research Triangle
 26 Park, NC, December, 2003.
 27 “Dose-Response Analysis of Toxicogenomic Data”, DERT Retreat, Southern Pines, NC, December,
 28 2003.
 29 “Toxicogenomics and the Research Directions of the National Toxicology Program”, Keynote Address,
 30 Conference on Toxicology in the 21st Century, Korean Food and Drug Administration, Seoul, Korea,
 31 November, 2003.
 32 “Dioxin and Agent Orange: What is known and what is suspected”, Keynote Address, NCST/NIEHS
 33 Conference of Environmental Mediation for Dioxin Soil Contamination, Hanoi, Vietnam,
 34 November, 2003.
 35 “Dose-Response Analysis of Toxicogenomic Data”, NCSU/NIEHS Workshop on Bioinformatics and
 36 Risk Assessment, Research Triangle Park, NC, October, 2003.
 37 “Health Effects of Electric and Magnetic Fields: Current Research Directions”, Joint WHO/Japanese
 38 Ministry of Health Public Meeting on Cellular Radiation, Tokyo, Japan, September, 2003.
 39 “Future Directions in Toxicology and the National Toxicology Program”, NIEHS Public Liaison
 40 Meeting, New York City, September, 2003.
 41 “Chipping Away at Risk Assessment: Genomics, Proteomics, Metabonomics and Cancer Risk
 42 Assessment”, Society of Toxicologic Pathology, Savannah, Ga. June, 2003.
 43 “Cancer Modeling: An Overview”, Statistical Methods in Cancer Research, Radiation Effects Research
 44 Foundation, Kyoto, Japan, March, 2003 (presented by H. Toyoshiba due to war in Iraq and US Govt.
 45 responsibilities)
 46 “Statistical methods for evaluating population exposures using CDC’s Environmental Report Card”,
 47 CDC, Atlanta, Ga. March, 2003.
 48 “Bystander Effects in Carcinogenesis”, Society of Toxicology Meetings, Salt Lake City, Utah, March,
 49 2003
 50 “Validation and use of genetically-modified mouse models as alternatives in carcinogenicity testing”,
 51 ILSI Workshop on Genetically Modified Mouse Models, Alexandria, Va., February, 2003.

1 “The Future of Genomics in Toxicology”, Mississippi State University, Oxford, Mississippi, February,
 2 2003
 3 “NIEHS priorities in ecological research”, USGS Director’s Retreat, Washington, DC. January, 2003.
 4 “The US National Toxicology Program”, Keynote Lecture at Opening Ceremonies for the Korean
 5 National Toxicology Program, Seoul, Korea, November, 2002
 6 “The NIEHS/NTP Initiatives on Alternative Models in Toxicology”, Science Advisory Board Meeting of
 7 the Center for the Advancement of Alternatives in Toxicology, Johns Hopkins University,
 8 Baltimore, Maryland, November, 2002
 9 “The Analysis and Interpretation of Two-Stage Liver Bioassays in the Rat”, [Workshop on Hepatic](#)
 10 [Preneoplasia: Quantitative Evaluation in Carcinogenesis Bioassays and Relevance for Human](#)
 11 [Hepatocarcinogenesis, Heidelberg](#), Germany, June, 2002
 12 “Chipping Away at Risk Assessment: Genomics, Proteomics, Metabonomics and Cancer Risk
 13 Assessment”, Office of Environmental Health Hazard Assessment, California Department of Health,
 14 Sacramento, California, June, 2002
 15 “Mechanistic Modeling of Stochastic Endpoints”, NIEHS Conference on Future Directions in
 16 Biostatistics, Research Triangle Park, North Carolina, May, 2002
 17 “Mechanistic Models of Skin Carcinogenesis”, North Carolina State University, Raleigh, North Carolina,
 18 May, 2002
 19 “Endocrine Dismodulation and Cancer”, Workshop on Light, Endocrine Systems and Cancer, University
 20 of Cologne, Cologne, Germany, May, 2002
 21 “Mechanism-Based Quantitative Analysis of Carcinogenesis Data”, New York Academy of Sciences,
 22 April, 2002.
 23 “Emerging Issues in Cancer Risk Assessment”, Sunrise Expert Seminar, American Association for
 24 Cancer Research Annual Meetings, San Francisco, California, April, 2002
 25 “Quantitative Evaluation of Health Risks from Dioxin”, Joint US-Vietnam Scientific Conference on the
 26 Health and Environmental Effects of Agent Orange/Dioxin in Vietnam, Hanoi, Vietnam, March,
 27 2002
 28 “Cancer Risk Assessment”, Hanoi Medical School/Vietnam Environmental Protection Agency/Vietnam
 29 Ministry of Health Joint Seminar, Hanoi Vietnam, January, 2002
 30 “Mechanism-Based Modeling of Genomics Data”, University of Berne, Switzerland, October, 2001.
 31 “Multistage Models of Carcinogenesis”, International Biometrics Conference Satellite Meeting, Fukuoka,
 32 Japan, September, 2001. (given by H. Toyoshiba due to conflict in scheduling)
 33 “Discussion of CDC’s Report on Levels of Environmental Contaminants in Human Tissues”, National
 34 Public Radio Talk of the Nation Science Friday, March, 2001.
 35 “Mixtures and Models in Environmental Health Risk Assessment”, *NIEHS/Colorado State University*
 36 conference on the Application of Technology to Chemical Mixture Research, January, 2001
 37 “Biological and biophysical research at extremely low- and radio-frequencies”, Forschungsgemeinschaft
 38 Funk, Bad Münstereifel, Germany, December 2000.
 39 “Harmonization of study evaluation and health risk assessment”, 2nd International EMF Seminar, Chinese
 40 Ministry of Health, WHO and ICNIRP, Xian, China, October 2000.
 41 “QRA: extrapolations (animals to humans; high dose low dose)”, First International Course on Scientific
 42 Basis of Carcinogen Assessment – Quo Vadis?, NIVA, Naantali, Finland, September 2000.
 43 “Statistical and biological models and toxicological issues in risk assessment”, Center for Environmental
 44 Health Risk Assessment (CERA) workshop on Future Needs in Risk Assessment, Stockholm,
 45 Sweden, September 2000.
 46 “Innovative use of mechanistic considerations in forming quantitative risk assessment models”,
 47 Workshop on Future Research For Improving Risk Assessment Methods, National Institute for
 48 Occupational Safety and Health, Aspen, Colorado, August 2000.
 49 “Latest scientific findings on dioxin”, Dioxin Summit, University of California at Berkeley, California,
 50 August 2000.

1 “Stochastic modeling in carcinogenesis and development”, Virtual Human Workshop, Research Triangle
2 Park, NC. June 2000.

3 “Evaluating health risks; methods and examples”, Federal Office for Public Health, Bern, Switzerland.
4 May 2000.

5 “Decisions about environmental health risks: What are the key questions and how does this apply to
6 melatonin”, Low frequency EMF, visible light, melatonin and cancer”, International Symposium,
7 University of Cologne and the German Research Society, Cologne, Germany. May 2000.

8 “Children's environmental health-risk assessment issues and challenges”, California Environmental
9 Protection Agency, Office of Environmental Health Assessment, Oakland, CA. April 2000.

10 “Children’s environmental health risks”, WHO/NIEHS Pacific Rim Meeting on Children’s Health,
11 Manila, April, 2000.

12 “Toxicology and environmental health seminar series 2000”, University of Florida, Gainesville, Florida.
13 April 2000.

14 “Biologically based population health risk assessment models”, University of Ottawa, Department of
15 Epidemiology and Community Medicine and the Institute of Population Health, Ottawa, Canada.
16 March 2000.

17 “Molecular epidemiology: a new tool in cancer prevention”, Applying science to regulatory policy and
18 public health, Keystone Symposium, Taos, New Mexico. February 2000.

19 “Statistical methods used for evaluating chemical safety in the environment”, Mathematical Research
20 Institute, Oberwolfach, Germany. February 2000.

21 “Mechanism-based mathematical modeling in health risk assessment” NC State University, January,
22 2000.

23 “Methods for analyzing and quantifying EMF health risks”, WHO/ICNIRP scientific meeting, Erice,
24 Sicily, Italy. November 1999.

25 “Harmonization of cancer and non-cancer risk assessment”, The Society of Toxicology, Washington, DC.
26 November 1999.

27 “Risky business: evaluating the safety of environmental exposures”, Illinois Environmental Health
28 Association, 1999 Annual Education Conference, Peoria, IL. October 1999.

29 “Challenges in the use of experimental and epidemiological data in health risk assessment”, Symposium
30 on “Statistical methods in epidemiology and demography”, Department of Biostatistics at the
31 University of North Carolina in Chapel Hill, Chapel Hill, North Carolina. October 1999.

32 “Linking toxicokinetics and toxicodynamics in biologically-based dose response models”, Dioxin 99, 19th
33 International Symposium on Halogenated Environmental Organic Pollutants and Persistent Organic
34 Pollutants (POPs), Venice, Italy. September 1999.

35 “How did WHO decide on a TDI for dioxin”, Faculty of Environmental Studies in Nagasaki University
36 and the Department of Medical Informatics in Kyushu Medical School, Nagasaki, Japan. September
37 1999.

38 “PBPK Models for Estrogen”, Workshop To Evaluate Research Priorities For Endocrine Active
39 Compound Risk Assessment Methods, CMA, EPA, NIEHS, Research Triangle Park, North
40 Carolina. August 1999.

41 “Risk assessment activities in the National Toxicology Program; risk assessment methodology, and
42 mechanistic modeling of endocrine disruptors and dioxins”, Harvard Symposium on Persistent
43 Organic Pollutants (POPs): A Public Health Perspective, Boston, Massachusetts. June 1999.

44 “Do extremely low frequency electric and magnetic fields pose a health risk?”, 21st Annual Meeting of
45 the Bioelectromagnetics Society, Long Beach, California. June 1999.

46 “Probabilistic assessment of cancer dose response data”, Probabilistic Risk Assessment Workshop,
47 Sarasota, Florida. February 1999.

48 “Linking toxicology and epidemiology: the role of mechanistic modeling”, CDC and ATSDR
49 Symposium on Statistical Methods, Emerging Statistical Issues In Public Health For The 21st
50 Century, Atlanta, Georgia. January 1999.

“Evaluation Of Health Risks From Exposure To Electric And Magnetic Fields; Completion Of A 2-Year Review Process”, Toxicology Round Table, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, Virginia. January 1999.

Interagency Agreements and Intramural Research Grants

US Environmental Protection Agency (1993-1994). Development and implementation of a mechanistic model for 2,3,7,8-TCDD. \$25K.

Agency for Toxic Substances and Disease Registry (1994-1996). Study of the relationship between chemical structure/activity and dose-response shape and magnitude for carcinogens and development of physiologically based pharmacokinetic models for risk assessment. \$796K.

NIEHS, Intramural Research Grant (1995-1998). Development of a mechanistic model of the impact of electromagnetic fields on melatonin. \$180K.

NIEHS, Interagency Coordinator for Risk Assessment, EMFRAPID Program (1996-1998). Evaluation of potential risks from exposure to electric and magnetic fields. \$2 million.

US Environmental Protection Agency (1999). Update to Dose-Response Chapter, Dioxin Reassessment. \$25K.

NIH, Office of Research on Minority Health (1999-2002) GIS/Resampling method for evaluating data on environmental justice. \$70K.

Direction of Ph.D. Theses:

A Bailer. *The effects of treatment lethality on tests of carcinogenicity*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1986.

P Williams. *Estimating tumor incidence rates using the method of moments and maximum likelihood estimation combined*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1989.

G Carr. *The analysis of data on adverse reactions to chemicals in developmental toxicology*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1989.

S Liu. *Estimating parameters in a two-stage model of carcinogenesis using information on enzyme-altered foci from initiation-promotion experiments*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1993.

CD Sherman. *Multipath/multistage models of carcinogenesis*. Department of Statistics and Actuarial Sciences, University of Waterloo, Waterloo, Ontario, Canada, 1994.

C Lyles. *Cell labeling data: Models and parameter estimation*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1995.

F Ye. *The equal slopes test for benchmark doses*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 2001

S Whitaker. *Development of a biologically-based mathematical model of fetal development*. Department of Mathematics, North Carolina State University, Raleigh, North Carolina, 2000.

R Helms. *Homeostatic feedback control of growth on multistage cancer models*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 2001.

Journal Articles: (peer reviewed)

1. London, L., et al., *A call from 40 public health scientists for an end to the continuing humanitarian and environmental catastrophe in Gaza*. *Environmental Health*, 2024. **23**(1): p. 59.

2. Chartres, N., et al., *Conducting evaluations of evidence that are transparent, timely and can lead to health-protective actions*. *Environ Health*, 2022. **21**(1): p. 123.

- 1 3. Danforth, C.G., et al., *Development and demonstration of a data visualization platform of*
2 *short-term guidelines for ambient air levels of benzene during disaster response in*
3 *Houston, TX*. Integr Environ Assess Manag, 2023, July 18, doi: 10.1002/ieam.4814.
- 4 4. Wai, T.H., et al., Insights from application of a hierarchical spatio-temporal model to an
5 intensive urban black carbon monitoring dataset. Atmospheric Environment, 2022. **277**: p.
6 119069.
- 7 5. Miller, D.J., et al., Characterizing Elevated Urban Air Pollutant Spatial Patterns with
8 Mobile Monitoring in Houston, Texas. Environ Sci Technol, 2020. **54**(4): p. 2133-2142.
- 9 6. Portier CJ: A comprehensive analysis of the animal carcinogenicity data for glyphosate
10 from chronic exposure rodent carcinogenicity studies. *Environmental*
11 *Health* 2020, 19(1):18.
- 12 7. Robinson, C., Portier, C., Čavoški, A., Mesnage, R., Roger, A., Clausen, P., Whaley, P.,
13 Muilerman, H., Lyssimachou, A.: **Achieving a High Level of Protection from Pesticides in Europe:**
14 **Problems with the Current Risk Assessment Procedure and Solutions**, *European Journal of Risk*
15 *Regulation* 2020, 1-31
- 16 8. Krewski D, Rice JM, Bird M, Milton B, Collins B, Lajoie P, Billard M, Grosse Y,
17 Coglianò VJ, Caldwell JC *et al*: **Concordance between sites of tumor development in**
18 **humans and in experimental animals for 111 agents that are carcinogenic to humans.**
19 *Journal of toxicology and environmental health Part B, Critical reviews* 2019, **22**(7-
20 8):203-236.
- 21 9. Alexeeff, S. E., A. Roy, J. Shan, X. Liu, K. Messier, J. S. Apte, C. Portier, S. Sidney and
22 S. K. Van Den Eeden (2018). "High-resolution mapping of traffic related air pollution with
23 Google street view cars and incidence of cardiovascular events within neighborhoods in
24 Oakland, CA." *Environ Health* **17**(1): 17-38.
- 25 10. Messier, K. P., Chambliss, S. E., Choi, J. J., Roy, A., Marshall, J. D., Brauer, M., Szpiro,
26 A. A., Portier, C. J., Lunden, M. M., Kerckhoffs, J., Vermeulen, R. C. H., Hamburg, S. P.,
27 Apte, J. S., Mapping Air Pollution with Google Streetview Cars: Efficient Approaches
28 with Mobile Monitoring and Land Use Regression, *Environmental Science and*
29 *Technology*, October, 2018
- 30 11. Espín-Pérez, A., Portier, C. J., Chadeau-Hyam, M., van Veldhoven, K., Kleinjans, J., de
31 Kok, T., Comparison of statistical methods and the use of quality control samples for batch
32 effect correction in human transcriptome data, *PLOS One* 13(8), 2018
- 33 12. Apte, JS, Messier, KP, Gani, S, Brauer, M, Kirchstetter, TW, Lunden, MM, Marshall, JD,
34 Portier, CJ, Vermeulen, RCH, Hamburg, S., High-Resolution Air Pollution Mapping with
35 Google Streetview Cars: Exploiting Big Data, *Environmental Science and Technology*
36 2017, **51** (12) 6999-7008
- 37 13. Sand S, Parham F, Portier CJ, Tice RR, Krewski D. Comparison of Points of Departure for
38 Health Risk Assessment Based on High-Throughput Screening Data. *Environ Health*
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- 1 14. Cote I, Andersen ME, Ankley GT, Barone S, Birnbaum LS, Boekelheide K, et al. The
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3 Applications to Risk Assessment, and Future Directions. *Environ Health Perspect* (2016)
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5 PMCID: PMCPMC5089888.
- 6 15. Parham F, Portier CJ, Chang X, Mevissen M. The Use of Signal-Transduction and
7 Metabolic Pathways to Predict Human Disease Targets from Electric and Magnetic Fields
8 Using in vitro Data in Human Cell Lines. *Frontiers in public health* (2016) **4**:193. doi:
9 10.3389/fpubh.2016.00193. PubMed PMID: 27656641; PubMed Central PMCID:
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- 11 16. Portier CJ, Armstrong BK, Baguley BC, Baur X, Belyaev I, Belle R, et al. Differences in
12 the carcinogenic evaluation of glyphosate between the International Agency for Research
13 on Cancer (IARC) and the European Food Safety Authority (EFSA). *Journal of*
14 *epidemiology and community health* (2016) **70**(8):741-5. doi: 10.1136/jech-2015-207005.
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17 exposure to non-carcinogenic chemical mixtures. *Archives of toxicology* (2016) **90**(3):513-
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20 Characteristics of Carcinogens as a Basis for Organizing Data on Mechanisms of
21 Carcinogenesis. *Environ Health Perspect* (2016) **124**(6):713-21. doi:
22 10.1289/ehp.1509912. PubMed PMID: 26600562; PubMed Central PMCID:
23 PMCPMC4892922.
- 24 19. McPartland, J., Dantzker, H.C., Portier, C. J. Building a robust 21st century chemical
25 testing program at the U.S. Environmental Protection Agency: recommendations for
26 strengthening scientific engagement, *Environ Health Perspect* 2015. 123 (1): p. 1-5.
- 27 20. Smith, M.T., Gibbons, C.F., Fritz, J.M., Rusyn, I., Lambert, P., Kavlock, R., Hecht, S.S.,
28 Bucher, J., Caldwell, J.C., Demarini, D., Cogliano, V., Portier, C., Paan, R., Straif, K.,
29 Guyton, K.Z., Key Characteristics of Carcinogens and an Approach to using Mechanistic
30 Data in their Classification, *Environ Health Perspect* 2015 (in press)
- 31 21. Portier CJ, Goldman LR, Goldstein BD. Inconclusive Findings: now you see them, now
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33 10.1289/ehp.1408106.PMID: 24486734
- 34 22. Thomas, R., Thomas, R.S., Auerbach, S. S., Portier, C. J., Biological networks for
35 predicting chemical hepatocarcinogenicity using gene expression data from treated mice
36 and relevance across human and rat species. *PLoS One*, 2013. **8**(5): p. e63308.
- 37 23. Scinicariello, F., Buser, M.C., Mevissen, M., Portier, C.J., Blood lead level association
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- 40 24. Thomas R, Portier CJ., Gene Expression Networks, *Methods Mol Biol.* 2013;930:165-78.

25. Aylward LL, Kirman CR, Schoeny R, Portier CJ, Hays SM., Evaluation of Biomonitoring Data from the CDC National Exposure Report in a Risk Assessment Context: Perspectives across Chemicals. *Environ Health Perspect.* 2012 **121** (3)
26. Sand, S., Portier, C.J., Krewski, D. A Signal-to-noise crossover dose as the point of departure for risk assessment. *Environmental Health Perspectives.* 119(12):1766-74, 2011
27. Gohlke, J.M., Thomas, R., Woodward, A., Campbell-Lundrum, D., Pruss-Ustun, A., Hales, S., Portier, C.J. Estimating the global public health implications of electricity and coal consumption. *Environmental Health Perspectives* 2011 119 (6): 821-6
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30. Luke, N.S., DeVito, M.J., Portier, C.J., El-Masri, H.A., Employing a mechanistic model for the MAPK pathway to examine the impact of cellular all-or-none behavior on overall tissue response, *Dose-Response* 2010 8(3): 347-67.
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33. Hines RN, Sargent D, Autrup H, Birnbaum LS, Brent RL, Doerrner NG, Cohen Hubal EA, Juberg DR, Laurent C, Luebke R., Olejniczak K, Portier CJ, Slikker W. Approaches for assessing risks to sensitive populations: lessons learned from evaluating risks in the pediatric population. *Tox. Sci.* 2010 **113** (4), 4-26.
34. Portier, C. Toxicological decision making on hazards and risks – status quo and the way forward: current concepts and schemes of science-driven decision making – an overview. *Human and Experimental Toxicology* 2009 **28**(2-3), 123-125
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Appendix II: Previous Cases Resulting in Depositions and Court Appearances

Glyphosate multidistrict litigation under Judge Vince Chhabria. MDL 2741, Case 3:16-md-02741-VC, US District Court, Northern District of California

Edwin Hardeman (plaintiff) v. Monsanto Company (defendant), MDL 2741, Case 3:16-cv-00525-VC, US District Court, Northern District of California

Edwin Hardeman (plaintiff) v. Monsanto Company (defendant), MDL 2741, Case 3:16-cv-00525-VC, US District Court, Northern District of California

Alva and Alberta Pilliod (plaintiffs) v. Monsanto Company (defendant), Alameda County Superior Court, Case A158228

Walter Winston et al. (plaintiffs) v. Monsanto Company (defendant), Circuit Court of the City of St. Louis, State of Missouri, Case No. 1822-CC00515

Depositions from Winston v. Monsanto were also to be used for the following:

Bellah v. Monsanto, Lake C., CA

Caballero v. Monsanto, Alameda County, CA

Bargas v. Monsanto, Alameda County, CA

Wade v. Monsanto, St. Louis City, MO

Stevick v. Monsanto, San Francisco, CA
Seitz v. Monsanto, St. Louis City, MO

Kane v. Monsanto, St. Louis City, MO

Bogner v. Monsanto, St. Louis County, MO

Neal v. Monsanto, St. Louis City, MO

Ranitidine Products Liability Litigation, County of Alameda, CA, JCCP 5150

Appendix III: Compensation

Billing is at \$500.00 per hour in 30-minute increments for all activities including depositions and trial testimony with the exception of travel time which will be billed at \$200.00 per hour with a maximum of 8 hours per day. Reasonable expenses incurred including transportation costs, hotels and meals will be reimbursed.

1 **Certification**

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3 I hereby certify that this report is a complete and accurate statement of all of my opinions, and
4 the basis and reasons for them, to which I will testify under oath.
5 12/1/2025
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7 

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9 _____
10 Christopher J. Portier

December 1, 2025

Date