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Exposure Limits to Radiofrequency EMF Do Not Account for Cancer Risk or Reproductive Toxicity Assessed from Data in Experimental Animals

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Abbreviations

BMD	Benchmark dose
BMDL	Benchmark dose lower limit
CDMA	Code Division Multiple Access
CoE	Certainty of evidence
FCC	Federal Communications Commission
GSM	Global System for Mobile Communications
IARC	International Agency for Research on Cancer
ICBE-EMF	International Commission on the Biological Effects of EMF
ICH	International Council for Harmonization
ICNIRP	International Commission on Non-Ionizing Radiation Protection
LOAEL	Lowest observable adverse effect level
MA	Meta-analysis
NOAEL	No-observed adverse effect level
NTP	National Toxicology Program
OEHHA	Office of Environmental Health Hazard Assessment
RF-EMF	Radiofrequency electromagnetic fields
RFR	Radiofrequency radiation
RI	Ramazzini Institute
SR	Systematic review
SAR	Specific absorption rate
UF	Uncertainty factor
US EPA	US Environmental Protection Agency
WHO	World Health Organization

Abstract

Background: Recent WHO-commissioned systematic reviews have concluded with “high certainty” that exposure to radiofrequency electromagnetic fields (RF-EMF) increases cancer risk and reduces male fertility in experimental animals.

Methods: We performed benchmark dose (BMD) analyses on experimental cancer data to estimate exposure levels associated with cancer risk of $1x10^{-5}$ (1 in 100,000). Due to the lack of an established non-linear mode of action for RF-EMF-induced tumor responses, we utilized linear low-dose extrapolation from 1% BMD values. In addition, we applied traditional uncertainty factors to the reported linear potency value of 0.03 per W/kg for male reproductive toxicity to derive health-protective exposure limits.

Results: The derived dose per hour (expressed as the specific absorption rate, SAR) at $1x10^{-5}$ cancer risk ranges from about 0.8 to 5 mW/kg. It should be noted that cancer risk increases with increasing time of exposure to RF-EMF. For protection of male fertility due to exposure to RF-EMF, the estimated SAR exposure limit was 3.3 to 10 mW/kg. These health protective whole-body exposure values are significantly lower than the current whole-body exposure limit value of 0.08 W/kg (80 mW/kg) established by ICNIRP and the FCC for the general public.

Conclusions: For the general public, current regulatory limits to RF-EMF are 15- to 900-fold higher than our estimates of exposure levels associated with

cancer risk of $1x10^{-5}$ (depending on the duration of daily exposure), and 8- to 24-fold higher than levels that are protective of male reproductive health. Thus, we strongly recommend an independent re-evaluation of RF-EMF exposure limits, integrating scientific data accumulated over the past 30 years and applying rigorous health-protective methodologies.

Background

Current exposure limits to radiofrequency electromagnetic fields (RF-EMF) in the United State are based on guidelines from the Federal Communications Commission (FCC) [1]. In many other nations, the exposure limits are based on guidelines from the International Commission on Non-Ionizing Radiation Protection (ICNIRP) [2]. However, those exposure limits were established from inadequate data and by methods that are inconsistent with procedures used by public health agencies to set exposure limits to toxic or carcinogenic environmental agents [3-6]. Everyday RF-EMF sources include cell phones, cordless phones, Wi-Fi routers and cell towers.

Exposure limits to RF-EMF were established by the FCC and ICNIRP in the late 1990s [7,8] based on results from behavioral studies conducted in small groups of rats and monkeys in the 1980s [9,10]. Those studies involved exposing food-deprived rats (N=8) or food-deprived monkeys (N=6) to 40- or 60-minute sessions to different frequencies and power densities of RF-EMF and determining the power level at which the lever-pressing response rates for the delivery of food pellets were significantly reduced compared to sham exposure sessions. No other endpoint or exposure duration was evaluated. Based on those studies, a specific absorption rate (SAR) of 4 W/kg was identified as the threshold for adverse health effects induced by RF-EMF. In a separate study in monkeys, an increase in mean body temperature of 0.7 °C was associated with a whole-body SAR of 4 W/kg (11]. Consequently, behavioral disruption associated with an increase in body temperature of approximately 1.0 °C was assumed by the FCC and ICNIRP to be the most sensitive measure of harmful effects from RF-EMF exposure [7,8]. Dosimetry of RF-EMF is the estimate of radiofrequency electromagnetic energy absorbed in biological tissues from emitting sources.

Based on the presumed threshold whole-body SAR dose of 4 W/kg, both the FCC [1,7] and ICNIRP [2,8] set exposure limits for controlled occupational exposures to 0.4 W/kg SAR averaged over the whole body by applying an arbitrary 10-fold uncertainty factor. For the general public, the FCC and ICNIRP set an exposure limit of 0.08 W/kg SAR averaged over the whole body by applying an additional 5-fold uncertainty factor. These exposure limits were expected to protect against thermal effects in humans that might occur from short-term acute exposures to RF radiation (RFR). While limited health effects data were available in the 1990s when these exposure limits were first established by the FCC and ICNIRP, the same values were reaffirmed two decades later [1,2], despite hundreds of studies showing adverse health effects at exposures well below the putative threshold level of 4 W/kg and with pulse modulations, indicative of non-thermal effects. The International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF) described how scientific studies published over the past 25 years invalidated 14 health assumptions that underlie the FCC and ICNIRP exposure limit determinations for RFR [12].

The studies from the 1980s that served as the basis for those exposure limit determinations by the FCC and ICNIRP provided an incomplete characterization of the health effects of RF-EMF and lacked information about effects due to chronic exposures. Consequently, the application of arbitrary uncertainty/safety factors to the assumed threshold dose provides no assurance of safety.

The findings of increased tumor incidence (heart schwannomas and brain gliomas) in rats exposed to RF-EMF in studies conducted by the National Toxicology Program (NTP) [13] and by the Ramazzini Institute (RI) [14] supported the findings of increased tumor incidences in case-control studies of mobile phone users [15-20]. Results from two large human case-control studies served as the major reason for the classification of RFR as “possibly carcinogenic to humans” by the International Agency for Research on Cancer (IARC) [21]. The human studies reported increases in risk of gliomas and acoustic neuromas (vestibular schwannomas) that were associated with increases in call time and latency; further increases in risk were detected when side of head use was also analyzed. Human cohort studies available at that time were judged by IARC [21] to be uninformative because exposure was based on subscriptions to mobile phone providers resulting in “several potential sources of misclassification of exposure,” The results from the NTP and RI carcinogenicity studies were not available at the time of the IARC evaluation.

Although the human studies show an increase in cancer risk after 500 to 1000 hours of call time [22-24], the exposure data lacked sufficient information on the intensity or dose of RFR associated with cancer risk.

Consequently, animal cancer data are used here to estimate doses of RFR associated with $1x10^{-5}$ extra cancer risk (i.e., the probability of an additional cancer per hundred thousand people exposed to the particular agent compared to the rate in an unexposed population), a risk level frequently used by the US Environmental Protection Agency (EPA) to set exposure limits to environmental carcinogens according to a non-threshold model.

The authors of the WHO-commissioned systematic review (SR) on the effects of exposure to RF-EMF on cancer induction in experimental animals concluded there was “high certainty of evidence” (CoE) for increased incidence of schwannomas of the heart in experimental rats exposed to RF-EMF and “moderate to high” CoE for glial cell tumors [25]. High CoE indicates the carcinogenic effect is highly likely to be due to exposure to RF-EMF. In addition, the authors reported 1% benchmark dose (BMD_{01}) values for glioma and heart schwannoma for those studies in which a significant positive trend was identified. BMD_{01} is the dose expressed as the whole-body SAR at the lower end of the experimental range that is estimated to result in 1% extra risk; it serves as the starting point (i.e., point of departure) for extrapolation of cancer risk to lower doses [3]. However, because of differences in survival between the sham group and RF-EMF exposure groups in the NTP study, we reran the benchmark dose-response analyses using poly-3 adjusted tumor rates.

To estimate exposure limits with *de minimis* human risk for non-cancer effects in the absence of sufficient information on the probability distribution of characteristics of human variability, regulatory agencies have traditionally applied uncertainty factors to the no-observed adverse effect levels (NOAEL) or to the lowest observable adverse effect levels (LOAEL) reported in experimental animal studies [4-6] (https://www.epa.gov/risk/conducting-human-health-risk-assessment) and using a threshold model. Uncertainty factors account for interspecies differences (extrapolating toxicity data from animal models to humans), intraspecies differences (due to interindividual differences in susceptibility), and adjustment if only a LOAEL is available. In the WHO-commissioned systematic review/meta-analyses (SR/MA) of experimental studies on the effects of RF-EMF exposure on male fertility, Cordelli et al. [26,27] concluded there was "high certainty of evidence that RF-EMF exposure reduces the rate of pregnancy," and the adverse effect on male fertility fit a linear dose-response relationship with a potency of 0.03 per W/kg, i.e., 3% per W/kg. We used this information to determine an exposure limit that would be reasonably protective of the effects of RF-EMF on male fertility.

Methods

Quantifying cancer risk.

Mevissen et al. [25] reported benchmark dose values for tumor responses in the studies on RF-EMF by the NTP [13] and the RI [14] in which there was a significant positive trend. Our focus is on heart schwannomas because that response was the most potent and was rated as high CoE by the authors of the systematic review on effects of RF-EMF on cancer in laboratory animals.

We also used EPA's Benchmark Dose Tools (https://www.epa.gov/bmds) to calculate BMD_{01} values for the dose-related survival-adjusted incidences of heart schwannomas and the combination of heart schwannomas and brain gliomas in male rats exposed to 900 MHz CDMA- and GSM-modulated RFR [13], as well as for the heart schwannoma data in the Ramazzini Institute study [14]. For 2-year carcinogenicity studies, the US EPA and California EPA recommend using BMDs obtained from multistage cancer dose response models provided in EPA's Benchmark Dose Software. The poly-3 adjusted tumor rates and the results of our BMD analyses are shown in Supplementary Data Files. The poly-3 survival method adjusts tumor rates by accounting for differences in mortality between control and exposure groups during the course of a carcinogenicity study.

In the current analyses we focused on $BMDL_{01}$ values since the US EPA [3] emphasizes the lower bound estimate to account for uncertainty in the true value of the BMD. Linear low-dose extrapolation from those values provides estimates of whole-body SARs associated with extra cancer risk of $1x10^{-5}$.

Determination of a whole-body SAR that minimizes adverse effects on male fertility

We used results from the systematic review by Cordelli et al. [26] on effects of RF-EMF exposure on male fertility to estimate an SAR value that could minimize those effects. Those authors reported that the relationship between SAR and reduced pregnancy in laboratory animals was linear, with a potency value of 3.0% per W/kg. Because there is insufficient information on probability distributions of human variability characteristics and interspecies differences in reproductive susceptibility to RFR exposures, uncertainty factors (Table 1) were applied to the potency value reported by Cordelli et al. [26]. Additional rationale for the use of the specified uncertainty factors is provided in Table 1.

Table 1.

Uncertainty factors applied to the 0.03 per W/kg response rate for reduced male fertility

Uncertainty Factor	**Rationale**
Interspecies: 10X (animal to human)	Based on reports that human sperm counts are declining [28] and because sperm production per gram of testicular tissue is much lower in humans compared to rats [29], an uncertainty factor of at least 10x is necessary.
Intraspecies: 10X	In addition to physiological and genetic differences, individuals differ in their exposure and response to other agents that can affect male fertility, such as endocrine-disrupting chemicals [30].

No NOAEL: 3X	A 3% response rate is not a NOAEL, it is equivalent to a LOAEL [31].

Results

Cancer risk from exposure to RF-EMF based on animal carcinogenicity data

In Table 2 are the BMD$_{01}$ values from the multistage 1 dose-response model and the corresponding 95% lower confidence limit values (BMDL$_{01}$) for heart schwannomas from the NTP study (CDMA- and GSM-modulated RFR), the Ramazzini Institute study, and the combination of heart schwannomas and brain gliomas in the NTP study in male rats. For mutagenic agents or those in which the mode of action of cancer causation is unknown, the BMDL value is the starting point for linear low-dose extrapolation of cancer risk.

Table 2.

BMD$_{01}$ for heart schwannomas based on results from the NTP and Ramazzini Institure cancer studies in male rats and heart schwannomas or brain gliomas in the NTP studies

Tumor, study	BMD$_{01}$ (W/kg)	BMDL$_{01}$ (W/kg)
Heart schwannoma		
NTP, CDMA	0.667	0.422
NTP, GSM	1.0003	0.590
Ramazzini Inst	0.097	0.037
Heart schwannoma or brain glioma		
NTP CDMA	0.538	0.344
NTP, GSM	0.494	0.336

Reduction of the BMDL01 by 10^3 yields the RF-EMF dose associated with cancer risk of $1x10^{-5}$; this is equivalent to changing the BMD units from W/kg to mW/kg. Thus, cancer risk of $1x10^{-5}$ is estimated to be associated with a daily SAR dose of 0.422 mW/kg based on results from the NTP CDMA study, 0.59 mW/kg based on results from the NTP GSM study, 0.037 mW/kg based on results from the RI study, 0.344 mW/kg and 0.336 mW/kg based on the combined rates of heart schwannoma and brain glioma from exposure to CDMA- and GSM-modulated RF-EMF, respectively, in the NTP study.

Daily exposure to RF-EMF was 9 hr/day in the NTP study [13] and 19 hr/day in the RI study [14]. Based on the BMDL01 values calculated from the multistage 1dose-response model, the per hour SAR at 10^{-5} risk is 3.8 mW/kg for the NTP CDMA results, 5.31 mW/kg based on the NTP GSM results, 0.70 mW/kg for the RI results, and 3.1 mW/kg and 3.02 mW/kg for the combination of heart schwannomas and brain gliomas from the NTP's studies of CDMA- and for GSM-modulated RF-EMF (Table 3).

The ICNIRP [2] and the FCC [1] exposure limit for the general public exposed to RF-EMF is 0.08 W/kg = 80 mW/kg, and 0.4 W/kg (400 mW/kg) for workers exposed occupationally to RF-EMF. Table 4 shows the ratio of the current ICNIRP/FCC exposure limit compared to the derived SAR values associated with increased cancer risk of $1x10^{-5}$ in relation to the number of hours per day of exposure to RF-EMF. Based on the results from carcinogenicity studies in experimental animals, it is apparent that extra cancer risk of $1x10^{-5}$ in the general public is increased by 15- to 114-fold with

1 hr/day of exposure to RF-EMF at the ICNIRP/FCC whole-body exposure limit, and by 60- to 444- fold for 4 hr/day of exposure. For workers, the cancer risk is 5 times greater than the risk for the general public.

Table 3.

The SAR (mW/kg) associated with 1x10^{-5} risk for heart schwannomas in the carcinogenicity studies by the NTP and the RI and for heart schwannomas or brain gliomas in the NTP studies, based on the number of hours per day of exposure

	Heart Schwannomas			**Heart Schwannomas or Brain gliomas**	
Exposure (hr/day)	**NTP (CDMA)**	**NTP (GSM)**	**Ramazzini Inst**	**NTP (CDMA)**	**NTP (GSM)**
	SAR (mW/kg) associated with 1x10^{-5} risk				
1	3.80	5.31	0.70	3.10	3.02
2	1.90	2.66	0.35	1.55	1.51
4	0.95	1.33	0.18	0.77	0.76
8	0.47	0.66	0.088	0.39	0.38

Table 4.

The ratio of the ICNIRP/FCC whole-body exposure limit for RF-EMF (80 mW/kg for the general public) compared to the exposure value associated with 1x10^{-5} risk for heart schwannomas in the carcinogenicity studies by the NTP and the RI and for heart schwannomas or brain gliomas in the NTP studies

	Heart Schwannomas			Heart Schwannomas or Brain gliomas	
Exposure (hr/day)	**NTP (CDMA)**	**NTP (GSM)**	**Ramazzini Inst**	**NTP (CDMA)**	**NTP (GSM)**
	Ratio of ICNIRP/FCC exposure limit to SAR (mW/kg) associated with 1x10^{-5} cancer risk				
1	21	15	114	26	26
2	42	30	229	52	53
4	84	60	444	104	105
8	170	121	909	205	211

Reduction of RF-EMF exposure limits to minimize loss of male fertility

To obtain a more protective exposure limit for male fertility, the following uncertainty factors were applied to the potency value of 0.03 per W/kg that was reported by Cordelli et al. [26] in their SR on the effects of RF-EMF exposures on male fertility in experimental animals:

10X for extrapolation from animals to humans
10X for differences between individual humans
3X if a NOAEL was not identified

Based on the cumulative uncertainty factor of 300 (when a NOAEL has not been identified), the dose associated with a 3% response (i.e., 1 W/kg) is divided by 300 to yield an exposure limit of 3.3 mW/kg for male reproductive toxicity (Table 5). However, if 1 W/kg is a NOAEL, then the cumulative uncertainty factor is 100 and the exposure limit for an adverse effect on male fertility would be 10 mW/kg. Cordelli et al. [26] never specified a

NOAEL or threshold response for any of the male reproductive parameters in their SR.

To achieve an exposure limit of 3.3 mW/kg, the ICNIRP/FCC limit would need to be reduced by 24-fold (80/3.3 = 24) for the general public and about 120-fold for occupational exposures (400/3.3 ~ 120). To achieve an exposure limit of 10 mW/kg, the ICNIRP/FCC limit would need to be reduced by 8-fold (80/10 = 8) for the general public and about 40-fold for occupational exposures (400/10 = 40).

Table 5.

The ratio of the ICNIRP/FCC whole-body SAR exposure limit for RF-EMF (80 mW/kg for the general public or 400 mW/kg for workers) compared to the calculated exposure limit needed to minimize adverse effects of RF-EMF on male fertility

Dose, SAR	Cumulative Uncertainty Factor	Calculated exposure limit, mW/kg	Ratio ICNIRP/FCC limit to calculated SAR, General public	Ratio ICNIRP/FCC limit to calculated SAR, Workers
1 W/kg ≠ NOAEL	300	3.3	24	120
1 W/kg = NOAEL	100	10	8	40

Discussion

Although the current ICNIRP and FCC exposure limits to RF-EMF are based on the application of arbitrary “safety factors” to insufficient health effects information, two recent WHO-commissioned SRs reported “high certainty of evidence” that exposures to RFR are associated with increased cancer risk [25] and decreased male fertility [26,27]. In this paper we used EPA’s Benchmark Dose Tools to estimate cancer potencies for the increased incidences heart schwannomas in the NTP and Ramazzini Institute studies on RF-EMF, and to calculate whole-body SARs that would be associated with extra cancer risk of $1x10^{-5}$. In addition, we applied uncertainty factors typically used by regulatory agencies to the reported potency value for male reproductive toxicity to estimate SAR values that would minimize adverse effects on male fertility.

Cancer risk was estimated by linear extrapolation from $BMDL_{01}$ values for the increased incidences of heart schwannomas and the combined rates of heart schwannomas and brain gliomas from the studies on RF-EMF by the NTP [13] and for the increase incidences of heart schwannomas in the Ramazzini Institute study [14]. Linear low-dose extrapolation is used when the mode of action of tumor induction is unknown [3]. While there are no studies showing that the mode of action of tumor response in animals exposed to RF-EMF would not occur in humans, numerous studies have reported genotoxic effects from exposures to RF-EMF [32-33].

To quantify human cancer risk in the absence of sufficient human dose-response data, health agencies rely on animal cancer data to establish exposure limits that minimize human cancer risk: 1 per hundred thousand ($1x10^{-5}$) or 1 per million ($1x10^{-6}$). This is because processes of disease induction are similar in humans and rats; every known human carcinogen is carcinogenic in experimental animals when adequately tested; and controlled exposures in experimental studies eliminate potential effects of confounders [34]. In addition, well-conducted animal studies can eliminate the need to wait for the availability of sufficient human cancer data before implementing public health protective strategies. Although case-control studies found increased cancer risk among mobile phone users with increased call time, latency, and laterality, the exposure data lacked sufficient information about the intensity or dose of RFR associated with cancer risk. This limitation is overcome by studies in laboratory animals. Cancer risk is a function of the RFR intensity and number of hours per day that an individual is exposed to RF-EMF. To achieve an exposure limit associated with cancer risk of $1x10^{-5}$ the FCC and ICNIRP exposure limits for RFR would need to be reduced by about 15- to 114-fold for exposures of only 1 hr/day and by 121- to 909-fold for exposures of 8 hr/day. At current exposure limits, cancer risks in the general public are much too high even for exposures of only 1 hr/day.

The identification of an environmental agent as a likely or probable human carcinogen is required by regulatory agencies before they conduct a dose-response analysis and a quantitative cancer risk assessment. In 2011, an IARC expert panel classified RF-EMF as *possibly carcinogenic to humans* based on *limited evidence* in humans and in experimental animals [21]. Limited evidence in humans, based on increased risk of gliomas in both the 13-nation INTERPHONE study and the Swedish case-control studies, means that "a positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence." The evaluation of limited evidence in experimental animals was based largely on positive finings in some initiation-promotion studies and the lack of adequate data from conventional long-term carcinogenicity studies [21].

Since the time of the IARC assessment, another large study (the French multicenter case-control study) found a significant increase in the risk of gliomas, and the results of the carcinogenicity studies of RF-EMF by the NTP and the Ramazzini Institute were published. A determination that there is now *sufficient evidence* for the carcinogenicity of RF-EMF in animals, plus an evaluation of *limited evidence* in humans is consistent with the IARC classification of *probably carcinogenic to humans*, and could lead to the EPA and/or the FCC conducting a quantitative risk assessment on RF-EMF by methodology similar to that used in this paper.

The authors of the WHO systematic review on cancer risk of RF-EMF in the general public concluded "For near field RF-EMF exposure to the head from mobile phone use, there was moderate certainty evidence that it likely does not increase the risk of glioma" [23]. However, several critical flaws in that review have been identified l35], including: 1) heavy reliance on the Danish cohort study that IARC judged to be uninformative because exposure was based on subscriptions to mobile phone providers resulting in “several potential sources of misclassification of exposure,” 2) the cited studies lacked sufficient follow-up time to detect late-developing tumors, 3) the metrics of exposure (ever vs never, and time since start of use) for the cohort studies, which dominated the conclusions of this systematic review, lacked information on the actual extent of use or of RF energy absorption, and 4) most of the meta-analyses combined data from studies that employed different methodologies (e.g., cohort vs case-control) which contributed to high between study heterogeneity. In addition, their dose-response analysis of the glioma case-control data, showed a nearly linear but nonsignificant increase in risk with cumulative call time above about 500 hr. Based on the same primary studies, a meta-analysis of the glioma case-control data by Moon et al. [24] found a statistically significantly increase in relative risk of brain gliomas at cumulative call times greater than 896 hr. Using Bradford Hill criteria to evaluate the scientific evidence on glioma risk from case-control studies of mobile phone users, Carlberg and Hardell [36] reported that the nine criteria for causation by RF radiation were fulfilled. Thus, there is sufficient evidence for regulatory action that would involve the conduct of a dose-response analysis and quantitative cancer risk assessment on RF-EMF; this action is especially needed since current exposure limits to RF-EMF are based on inadequate and outdated health effects information.

The application of uncertainty factors, which are typically used by regulatory agencies for toxic agents, to the potency value of 0.03 per W/kg reported by Cordelli et al. [26] for reduced fertility in laboratory animals exposed to RF-EMF resulted in an exposure limit value of 3.3 or 10 mW/kg. These values are 8- to 24-times lower than the ICNIRP and FCC exposure limits for the general public. In addition to effects of RF-EMF on male fertility, Cordelli [26] reported statistically significant adverse effects on most male reproductive endpoints evaluated, including decreases in sperm count, sperm vitality (immobile or dead sperm), testis or epididymis weight, sperm production and testosterone level, and increases in sperm DNA/chromatin alterations, testicular histological alterations and testicular cell death. In the SR/MA on the effects of RF-EMF exposure on pregnancy and birth outcomes in experimental animals, Cordelli et al. [35] reported statistically significant adverse effects including increases in resorbed and dead fetuses, and fetal malformations, and decreases in fetal weight and fetal length. Similar to the effects of RF-EMF exposure on male fertility, Cordelli et al. [37] reported that the increased incidence of resorbed and dead fetuses fit a linear dose-response relationship with a potency value of 0.03 per W/kg. Thus, there is a serious concern for potential adverse effects of RF-EMF exposures on male fertility and birth outcomes.

Uche and Naidenko [38] used a similar approach to derive a whole-body exposure limit to RF-EMF based on increased cardiomyopathy reported in the NTP study [13] of RFR in rats. Their calculation showed that the current ICNIRP and FCC exposure limit of 0.08 W/kg is 20- to 40-fold too high to be adequately protective of induced cardiomyopathy. Thus, there are now at least three adverse effects (cancer, male infertility, and cardiomyopathy) of RF-EMF exposure in which significant risks occur at SARs that are lower than the ICNIRP and FCC exposure limits. In addition, these effects demonstrate that the behavioral effects of RF-EMF reported in the 1980s by De Lorge and Ezell [9] and by De Lorge [10], and which serve as the basis for current limits to RFR, are certainly not the most sensitive health effects from RF-EMF exposures.

Most assumptions included in our analyses are based on guidelines published by public health agencies for setting health protective exposure limits to hazardous environmental agents. These include: 1) animal data are reliable for setting exposure limits to identified hazardous agents when sufficient information is not available from studies in humans; 2) exposure limits based on the most potent response in the most sensitive animal model are likely to be protective for most humans; 3) linear low-dose extrapolation is appropriate when there is insufficient information to establish a non-linear mode of action at low doses; 4) exposure estimates at cancer risk levels that are acceptable according to agency policy can be obtained by linear low-dose extrapolation from the BMD obtained by fitting empirical dose-response models to experimental cancer data; 5) low-dose extrapolation from the BMDL value accounts for uncertainty in the true value of the BMD; 6) for non-cancer effects, the appropriate exposure limit is based on a threshold dose-response model; 7) agency-recommended uncertainty factors adequately account for interspecies and intraspecies differences in sensitivity, as well as for the lack of an identified no-observed adverse effect level; and 8) for RF-EMF, health risks need to be based on SAR values since current exposure limits are based on that dose metric.

In addition to these assumptions, there are limitations in the values presented in this paper that might underestimate the true risk, including: 1) the utilization of SAR as the appropriate dose metric in these analyses of cancer risk does not account for EMF interactions with biological tissues at the molecular level due to near-field emissions from wireless devices located next to one's body; 2) low-dose extrapolation for evaluating cancer risk does not account for interindividual differences in susceptibility due to differences in genetic factors (e.g., DNA repair), health status, lifestyle, exposures to other cancer-causing agents, etc.

Conclusions

Based on the findings from two of the WHO-commissioned SRs that there is "high certainty of evidence" that exposures to RFR are associated with increased cancer risk and decreased male fertility, as well as benchmark dose analyses, we found that the ICNIRP and FCC exposure limits to RF-EMF are inadequate for protecting human health. Those limits need to be markedly reduced to be consistent with how public health

agencies set exposure limits to reduce health risks in the general public from exposures to hazardous environmental agents. To reduce extra cancer risk from exposure to RF-EMF to $1x10^{-5}$, the whole-body exposure limit would need to be reduced by 15- to more than 900-fold, with exposures that vary from 1 to 8 hr/day. To reduce the risk of reduced male fertility in the general public, the ICNIRP and FCC limit would need to be reduced by 8- to 24-fold. An independent re-evaluation of RF-EMF exposure limits based on scientific knowledge gained over the past 30 years and the application of health protective methodologies is long overdue.

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