Review Article

Lennart Hardell* and Michael Carlberg

Lost opportunities for cancer prevention: historical evidence on early warnings with emphasis on radiofrequency radiation

https://doi.org/10.1515/reveh-2020-0168
Received December 16, 2020; accepted January 30, 2021; published online February 15, 2021

Abstract: Some historical aspects on late lessons from early warnings on cancer risks with lost time for prevention are discussed. One current example is the cancer-causing effect from radiofrequency (RF) radiation. Studies since decades have shown increased human cancer risk. The fifth generation, 5G, for wireless communication is about to be implemented world-wide despite no comprehensive investigations of potential risks to human health and the environment. This has created debate on this technology among concerned people in many countries. In an appeal to EU in September 2017, currently endorsed by more than 400 scientists and medical doctors, a moratorium on the 5G deployment was required until proper scientific evaluation of negative consequences has been made (www.5Gappeal.eu). That request has not been taken seriously by EU. Lack of proper unbiased risk evaluation of the 5G technology makes adverse effects impossible to be foreseen. This disregard is exemplified by the recent report from the International Commission on non-ionizing radiation protection (ICNIRP) whereby only thermal (heating) effects from RF radiation are acknowledged despite a large number of reported non-thermal effects. Thus, no health effects are acknowledged by ICNIRP for non-thermal RF electromagnetic fields in the range of 100 kHz–300 GHz. Based on results in three case-control studies on use of wireless phones we present preventable fraction for brain tumors. Numbers of brain tumors of not defined type were found to increase in Sweden, especially in the age group 20–39 years in both genders, based on the Swedish Inpatient Register. This may be caused by the high prevalence of wireless phone use among children and in adolescence taking a reasonable latency period and the higher vulnerability to RF radiation among young persons.

Keywords: asbestos; cancer prevention; DDT; dioxins; early warnings; glyphosate; phenoxyacetic acids; radiofrequency radiation; tobacco.

Introduction

The International Agency for Research on Cancer (IARC) at the World Health Organization (WHO) initiated in 1969 a program to evaluate human cancer risks of chemicals. It was later expanded to include chemical mixtures, radiation and viruses. So far, this program has resulted in 125 Monographs. Mostly, as the history shows, it has taken a long time between the first reports of increased cancer risk and cancer classification of the agent. Thereby preventive measures have not been taken in due time with high costs to society as a consequence in terms of increased numbers of cases with diseases leading to suffering and costs for treatment, loss of professional activity and eventually premature deaths [1–3]. Thus, early warnings should not be neglected. In fact, false positives on environmental risks are extremely rare [4]. In the following some historical examples are discussed, followed by a review of the current controversy on radiofrequency (RF) radiation and cancer. These examples serve as lessons for early warnings [5, 6].

No doubt the reports from the European Environment Agency on late lessons from early warnings may serve as important documents for the precautionary approach. Volume 1 was published in 2001 [5]. It dealt with 12 key lessons on health and environmental hazards. The 2013 volume on late lessons was grouped into five parts including e.g., health, ecosystems, justice, and governance [6]. Both volumes give examples on action that could have

***Corresponding author: Lennart Hardell**, MD, PhD, Professor, Department of Oncology, Faculty of Medicine and Health, Örebro University, SE-701 82 Örebro, Sweden; and Present address: The Environment and Cancer Research Foundation, Studievägen 35, SE-702 17 Örebro, Sweden,
E-mail: lennart.hardell@environmentandcancer.com
Michael Carlberg, The Environment and Cancer Research Foundation, Örebro, Sweden,
E-mail: michael.carlberg@environmentandcancer.com

been taken to prevent harm. In the following some examples are discussed partly based on our own research experiences.

Examples of early warnings on cancer risks

The first history on occupational diseases was written by the Italian physician Bernardini Ramazzini in his book "De morbis artificum" (Diseases of Workers) printed in Modena, Italy 1700. He is regarded to be the 'father of occupational medicine'. A second extended version was printed in Padua 1713. In the book 53 chapters deal with different occupations and diseases occurring in these occupations [7].

Regarding specific occupational exposures the English physician Percival Pott was the first to describe that men working as chimneysweeps, and thereby exposed to soot, had an increased risk for scrotal cancer. He published his findings in 1775 [8]. This disease was known as chimney-sweepers' cancer. It is regarded to be the first report of an environmental factor causing cancer. It took a long time of campaigning to stop little boys being used to clean chimneys by climbing up them. More than 200 years later soot was classified as a human carcinogen Group 1 (carcinogenic) by IARC in 1985 [9].

Asbestos

Another both occupational and environmental toxic substance is asbestos. Already in 1899, a UK Factor Inspector observed the sharp glass-like jagged nature of asbestos particles [10]. The author noted asbestos dust in the air of the factory rooms and that "*the effects have been found to be injurious*". Numerous reports have since then described increased risks primarily of lung cancer and mesothelioma. Already in 1935, a man with asbestosis and lung cancer was reported [11]. In 1953 it was reported that a man who had worked with asbestos died of pleural mesothelioma [12]. South African researchers published in 1960 a report on increased risk for mesothelioma for both occupational and environmental exposure to asbestos [13]. The American physician Dr. Irving Selikoff gave to a broader public insight into a dramatic increased cancer mortality among American insulation workers exposed to asbestos. Also, that environmental exposure increased the risk of mesothelioma [14]. This started a long-standing battle between a multinational industry defending its product, and public health and regulatory bodies [15, 16]. Asbestos was in 1977 evaluated by IARC to be carcinogenic to humans, Group 1 [17]. This was almost 20 years since the clear evidence of cancer risks was published in the early 1960s. Years were lost for prevention and yielded increased numbers of deaths.

Tobacco

Tobacco has a long history of reported adverse health effects. When first introduced in Europe smoking was recommended for medical purposes, in fact as prophylaxis for many diseases. In 1604 King James I of United Kingdom wrote against the use of tobacco [18]. Sömmering stated in a thesis in 1795 that tobacco pipes induced an increased risk for lip cancer [19]. Cancer of the tongue was described some 100 years later in 1890 [20]. A high proportion of diseases including lung cancer among cigar makers and sellers, waiters, and innkeepers was reported in 1914 [21]. A clearly increased incidence of lung cancer was first reported by Müller in 1940 [22]. This evidence and other cancer studies in the 1940s in Germany [23] and in the Netherlands [24] were mainly disregarded thereby omitting the possibility of early prevention. It was not until the 1950s when more studies showed health risks from tobacco, primarily for diseases such as cancer of the lung, myocardial infarction, peripheral vascular diseases, and chronic obstructive lung disease. Tobacco was in 1986 classified by IARC as a human carcinogen, Group 1 [25]. No doubt the history of smoking shows that early warnings were mainly neglected. Greenwashing by industry and its allied experts has a history of counteracting preventive measurements [26].

DDT

The marine biologist Rachel Carson was the first to write a general picture of chemical damage to the environment, human and animal health in her book Silent Spring published in 1962 [27]. She gave the first comprehensive description of the bioaccumulation of the insecticide DDT (*para*,*para*′-DDT –1,1′-(2,2,2-trichloro-ethylidene)bis (4-chloro benzene)). DDT was discovered in 1939 by the Swiss researcher Paul Müller. For that he received the Nobel Prize in medicine in 1948. No doubt the book by Rachel Carson was opposed by the chemical industry that even tried to stop the publication. In fact, DDT was defended by the American Medical Association and the US Nutrition Foundation unified with 54 companies in the food, chemical and allied industries [28]. The main human studies on human carcinogenicity of DDT and its main metabolite DDE (1,1'-(2,2-dichloroethenylidene)- bis(4-chlorobenzene)) were performed from the 1990s and onward [29].

The Stockholm Convention on Persistent Organic Pollutants was adopted in 2001. It provided initially evidence for the elimination of 12 chemicals, one of which was DDT [30]. The use of DDT was banned in most countries in the 1970s [31]. In 1972, the US EPA issued a cancellation order for DDT [32]. DDT was evaluated by IARC in 2018 to be probably carcinogenic to humans, Group 2A [29]. It had previously been evaluated as a possibly human carcinogen, Group 2B [33]. One of the main toxic issues is the bioaccumulation of DDT and its metabolites with long half-time in the environment [27]. DDT is still used in some countries, e.g. for malaria control. Due to its chemical behavior its metabolites can be found in human tissue [34, 35].

Phenoxyacetic acids

In 1977, a report was published on a series of patients who had been spraying phenoxy herbicides for the Swedish Forestry and who subsequently developed soft-tissue sarcoma [36]. Herbicides of this type include 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). 2,4,5-T was contaminated by 2,3,7,8,tetrachlorodibenzo-p-dioxin (TCDD), one of the most toxic chemicals in the world. This clinical observation was the first to indicate a possible increased cancer risk for these chemicals. Based on that report an increased risk for soft-tissue sarcoma was found both for these phenoxy herbicides and the chemically related chlorophenols, mostly exposure to pentachlorophenol, in a following case-control study [37]. These results were corroborated in further studies by our research group and others, for an overview see [2].

Another set of studies included malignant lymphoma, also initiated by a clinical observation [38]. This clinical observation resulted in further studies. An increased risk was found for both non-Hodgkin lymphoma (NHL) and Hodgkin's disease for persons exposed to phenoxy herbicides or chlorophenols [39]. Also, the increased lymphoma risk was confirmed in other studies, for overview see [2, 40].

One of the main types of chlorophenols, pentachlorophenol, was classified by IARC in 2019 to be carcinogenic to humans, Group 1 [41]. The phenoxy herbicide 2,4-D was in 2018 classified by IARC as possibly carcinogenic to humans, Group 2B [29]. It was the same classification as in 1977 including also 2,4,5-T [42].

Dioxins

The phenoxy herbicides 2,4-D, 2,4,5-T and chlorophenols were contaminated with dioxins. Of large concern was TCDD that contaminated 2,4,5-T and trichlorophenol. The initial Swedish results on cancer risks from this group of chemicals were followed by studies in other countries that confirmed the findings, for overview see [2,40]. Vietnam veterans exposed to the defoliating agent Agent Orange, including 2,4-D and 2,4,5-T, with TCDD contamination suffering from soft-tissue sarcoma or malignant lymphoma were in 1991 judged to be eligible for service-related compensation [43].

In 1976 an accident occurred in a chemical plant at Seveso, Italy producing 2,4,5-trichlorophenol. Thereby the surrounding area was contaminated with dioxins and the general population was exposed to TCDD. In the aftermath an increased incidence in malignant diseases, notably soft-tissue sarcoma and hematolymphatic malignancies was found in the population [40, 44].

Various *ad hoc* explanations were postulated by the chemical industry and its allied experts to discredit the cancer risks [2]. However, in 1997 IARC classified TCDD as a human carcinogen, Group 1 [45]. It had previously been evaluated in 1977 by IARC to be a possibly human carcinogen, Group 2B [42]. This was about two decades after the first epidemiological publications on increased cancer risk for TCDD contaminated herbicides.

Glyphosate

In the case-control studies by the Hardell group on risk factors for NHL exposure to all types of herbicides was assessed. In addition to phenoxyacetic acids also glyphosate turned out to increase the risk [46, 47]. Hairy cell leukemia (HCL) is regarded to be a subtype of NHL. In a separate study on HCL glyphosate was a risk factor also for that malignancy [48]. Similar results were also found in other studies [49, 50].

Glyphosate was in 1970 tested as herbicide and was patented by Monsanto [51]. It was registered for use in USA in 1974 with the trade name 'Roundup'. Since the patent has expired it is produced nowadays by many manufactures. In 1996 genetically engineered glyphosate tolerant crops were introduced (Roundup Ready) and since then the global use has increased 15-fold. Glyphosate has in recent years been the most widely used pesticide [52].

IARC at WHO evaluated glyphosate in March 2015 and classified it as a Group 2A, a probable human carcinogen [53, 54]. This was based on "limited" evidence of cancer in humans (from real-world exposures that occurred) and "sufficient" evidence of cancer in experimental animals (from studies of "pure" glyphosate). IARC also concluded

that there was "strong" evidence for genotoxicity, both for "pure" glyphosate and for glyphosate formulations.

The European Food Safety Authority (EFSA) is the EU agency for risk assessment regarding food safety. In October 2015, that is seven months after the IARC evaluation, EFSA published its own evaluation [55]. In summary EFSA dismissed without clear explanation any association of glyphosate with cancer. All findings on carcinogenesis in animal studies were incorrectly discarded as chance findings. Mechanistic evidence on genotoxicity was ignored. Oxidative stress was confirmed but dismissed as a ground for carcinogenesis [56]. It should be noted that EFSA did not reveal the names of the authors of the chapters and references were redacted.

Monsanto, the main glyphosate producer, hired a panel of scientists to defend glyphosate. Thus, in 2016 a 17-page article was published in Critical Reviews in Toxicology, known to be an industry friendly product defense journal [57]. It was concluded that "*In summary, the totality of the evidence, especially in light of the extensive testing that glyphosate has received, as judged by the Expert Panels, does not support the conclusion that glyphosate is a "probable human carcinogen" and, consistent with previous regulatory assessments, the Expert Panels conclude that glyphosate is unlikely to pose a carcinogenic risk to humans.*"

This review was made by four expert panels. In the initial publication no conflicts of interest were stated. All but six of the 16 authors appeared with their university or hospital affiliation. During lawsuits in USA on glyphosate exposure and NHL it was revealed that the authors were not independent, and that Monsanto was deeply involved in organizing, reviewing and editing the review. In fact, Monsanto paid the authors through a consulting firm, *Intertek* [58].

As a consequence Critical Reviews in Toxicology was forced to make a Corrigendum two years later: "*When this article was originally published on 28th September 2016, the contributions, contractual status and potential competing interests of all authors and non-author contributors were not fully disclosed to Critical Reviews in Toxicology. Specifically, the Acknowledgments and Declaration of Interest were not complete. After further clarification from the authors, these sections are corrected to reflect the full contributions, contractual status and, potential competing interests of all authors and non-author contributors and read as follows … This overview paper (paper) is part of a supplement, the preparation of which was coordinated by Intertek Scientific & Regulatory Consultancy (Intertek) under the leadership of Ashley Roberts. It was prepared subsequent to completion of the four manuscripts as an overview and presented the opinions and conclusions of four groups of the expert panel. The expert panels were organized and supported administratively by Intertek. Funding was provided to Intertek by Monsanto Company, which is a primary producer and marketer of glyphosate and related products. All the expert panelists other than John Acquavella and Larry D. Kier were compensated through a contract with Intertek. John Acquavella and Larry D. Kier were compensated through existing consulting contracts with Monsanto Company*" [59].

Product defense by downplaying risk seems to have been one of Monsanto's strategies [60].

The German chemical company Bayer purchased Monsanto in 2018. It is facing a magnitude of lawsuits on NHL and glyphosate exposure. So far in three lawsuits about 200 million USD have been awarded by the juries [58]. No doubt the use of glyphosate is of large economic importance both for the producers and the agriculture. In 2017 the EU Commission extended the use of glyphosate until 2022 [61].

Radiofrequency radiation

In 2011 radiofrequency electromagnetic fields (RF-EMF) in the frequency range 30 kHz–300 GHz were evaluated by IARC at WHO to be possibly carcinogenic to humans, Group 2B [62, 63]. This was based on evidence of increased risk for glioma and acoustic neuroma in human epidemiology studies on use of mobile and/or cordless phone (DECT) [64–69]. The increased cancer risk was supported by laboratory studies [70, 71].

Extremely low frequency (ELF)-EMF was in 2001 evaluated by IARC to be a possible human carcinogen, Group 2B [72]. This was the first time that non-ionizing radiation at low intensity levels can be a possible cause of cancer. It predated the IARC finding for RF-EMF by a decade.

Since then the evidence on RF-EMF carcinogenesis has strengthened based on further human studies on use of wireless phones, as reviewed [73, 74]. Also animal studies show increased cancer risk, both near field RF-EMF exposure [75–77] and far field exposure [78, 79]. Mechanistic studies show increase of reactive oxygen species (ROS) [80] as well as DNA damage [81]. These results give support to the increased cancer risk in humans and laboratory tested animals for RF radiation. In fact, RF-EMF may now be classified as a human carcinogen, Group 1 [82, 83]. However, such classification can only be made by IARC.

Of course, these well documented health hazards from RF-EMF are not well accepted by the telecom industry and its allied experts. Several methods are used to create doubt.

Studies are discredited, only partly cited, or even not cited at all [84–86]. Thereby the uniformed reader gets the wrong information on actual risks. This includes also regulatory agencies and policy makers. Even agencies aimed at setting exposure guidelines may include pro-industry and biased scientists that obscure the true risks [87, 88].

ICNIRP

The International Commission on Non-Ionizing Radiation Protection (ICNIRP) is a private non-governmental (NGO) organization registered in Munich, Germany. ICNIRP appoints its own members and is closed to transparency. It was started in 1992 with the biophysicist Michael Repacholi as the first chairman, now emeritus member. ICNIRP has published three articles with guidelines on RF-EMF exposure [86, 89, 90]. Only thermal (heating) effects from RF radiation are recognized, thereby excluding all studies showing harmful effects at lower non-thermal intensities. In contrast to ICNIRP, some other expert panels such as European Academy of Environmental Medicine [91], the Bioinitiative group [92], and the Russian Commission for Protection from Non-Ionizing Radiation [93], take into account non-thermal RF effects and suggest much lower guidelines for RF exposure.

ICNIRP has managed to get collaborative status with WHO, as discussed previously [88]. The aim is to harmonize the RF-radiation guidelines all over the world. For that purpose ICNIRP has been successful. The guidelines are set to allow very high exposure levels so that the deployment of this technology is not hampered, in favor for industry but at disadvantage to human health and environment. In fact, the ICNIRP guidelines have never been challenged by industry in peer-reviewed articles, which must be taken as a green card for acceptance by industry.

Attributable fraction

The attributable fraction (AF), sometimes also called the etiologic fraction, is the number of cases in which exposure played an etiologic role. This is the preventable fraction if exposure would not be present. In Belpomme et al. [73] we published meta-analyses for longest cumulative use of mobile phones with odds ratio (OR) and 95 % confidence interval (CI), both for total and for ipsilateral wireless phone use. Note that only the Hardell group assessed also use of cordless phones (DECT). We present here AF based on statistically significant increased risks in the meta-analyses. AF is the proportion of cases that can be attributed to the particular exposure. This is calculated as the exposed case fraction multiplied by [(OR-1)/OR].

As displayed in Table 1 the AF for glioma was calculated to 4.88%, 95% CI = 2.44–6.57%, corresponding to 211 preventable cases, 95% CI = 105–284 cases in the longest time for all cumulative use of wireless phones. Regarding ipsilateral use of the wireless phone AF was 6.03%, 95% CI = 4.51–7.12%, yielding 150 cases; 95% CI 112–177 to be preventable.

For meningioma AF = 1.75%, 95% CI = 0.39–2.73 corresponded to 39 cases, 95 % CI = 9–61 cases for ipsilateral use of the wireless phone was calculated. Calculation of AF for acoustic neuroma yielded 4.63%, 95% CI = 3.07–5.63% corresponding to 42 cases, 95% CI = 28–51 cases for ipsilateral use of the phone.

Table 1: Attributable fraction (AF) based on meta-analyses of case-controls studies on use of wireless phones with statistically significant increased risk. For details see Belpomme et al. [73]. Odds ratio (OR), 95% confidence interval (CI), and numbers (n) are given.

	Cases		Meta-analysis		AF		AF, corresponding cases	
	Total n	Exposed n	OR	95% CI	AF, %	95% CI (%)	N	95 % CI
Glioma[a]								
Longest[b] cumulative use ≥ 1640 h	4,319	445	1.90	1.31–2.76	4.88	2.44–6.57	211	105–284
Longest[b] cumulative use, ipsilateral ≥ 1640 h	2,484	247	2.54	1.83–3.52	6.03	4.51–7.12	150	112–177
Meningioma[a]								
Longest[b] cumulative use, ipsilateral ≥ 1640 h	2,241	119	1.49	1.08–2.06	1.75	0.39–2.73	39	9–61
Acoustic neuroma[c]								
Longest cumulative use, ipsilateral ≥ 1640 h	899	66	2.71	1.72–4.28	4.63	3.07–5.63	42	28–51

[a]Based on Interphone [67], Coureau et al. [101], Hardell and Carlberg [104], Carlberg and Hardell [102]. [b]Coureau et al. [101] ≥896 h. [c]Based on Interphone [68], Hardell et al. [108].

Rates of brain tumors in the Swedish National Inpatient Register ICD-code D43

Rates of brain tumors of unknown type, D43, were studied using the Swedish Inpatient Register (IPR) without any personal identification information [94]. It was established in 1964 and has complete national coverage since 1987 [95]. Register data on D43 are available from 1998. Currently more than 99% of hospital discharges are registered. For outpatients the data are less reliable due to missing information. The reporting of outpatients has increased during more recent years so these time trends may give spurious results, thus we omitted outpatients from the analysis.

Data were analyzed for the time period 1998–2019. Age-standardized rates are not available in the register. Instead numbers of patients per 100,000 inhabitants are reported. The Joinpoint Regression Analysis program version 4.1.1.1 was used to examine numbers of patients per 100,000 in inpatient care and incidence per 100,000 person-years in the Swedish Inpatient Register, by fitting a model of 0–3 joinpoints using permutation tests with Bonferroni correction for multiple testing to calculate the number of joinpoints that best fits the material [96]. When joinpoints were detected annual percentage changes (APC) and 95% CIs were calculated for each linear segment. Average annual percentage changes (AAPC) were also calculated for the whole time period using the average of the APCs weighted by the length of the segment. To be able to calculate APC and AAPC the data was log-transformed prior to analysis. Thus, it was not possible to perform joinpoint regression analysis when there were years with no cases during that time period. Since the data do not include any personal identification no ethical approval was needed.

In men AAPC increased during 1998–2019 with +1.77%, 95% confidence interval (CI) –0.02, +3.58%, Table 2; Figure 1. The increase was highest in the age group 20–39 years, +2.90%, 95% CI +1.66, +4.16 %, Figure 2. AAPC increased statistically significant in all age groups, except 0–19 years.

Similar results were found in women with AAPC +1.70%, 95% CI +0.38, +3.05% during 1998–2019, Table 3; Figure 3. Also in women the highest increase of AAPC was found in the age group 20–39 years, +2.89%, 95% CI + 1.54, +4.27%, Figure 4. AAPC increased statistically significant in all age groups except 0–19 years and 80+ years. Especially high increase of APC was seen in women aged 60–79 years during 2005–2019, and women aged 80+ years during 2010–2019.

Table 2: Joinpoint regression analysis of brain tumor rates (numbers per 100,000) in men in the Swedish Inpatient Register 1998–2019, ICD-10 code D43 (https://sdb.socialstyrelsen.se/if_par/val.aspx).

ICD-10	Joinpoint location	APC 1 (95% CI)	APC 2 (95% CI)	APC 3 (95% CI)	AAPC (95% CI)
D43					
All men (n=10,540)	2008; 2011	+0.13 (–0.85, +1.12)	+8.95 (–3.99, +23.64)	+1.22 (–0.16, +2.63)	+1.77 (–0.02, +3.58)
0–19 years (n=662)	No joinpoint detected	–	–	–	+1.83 (–0.13, +3.82
20–39 years (n=1,117)	No joinpoint detected	–	–	–	+2.90 (+1.66, +4.16)
40–59 years (n=2,799)	No joinpoint detected	–	–	–	+1.61 (+0.88, +2.36)
60–79 years (n=4,867)	No joinpoint detected	–	–	–	+1.67 (+0.99, +2.36)
80+ years (n=1,095)	No joinpoint detected	–	–	–	+1.40 (+0.11, +2.70)

APC, annual percentage change (APC 1, time from 1998 to first joinpoint; APC 2, time from first joinpoint to 2019 or to second joinpoint; APC 3, time from second joinpoint to 2019); AAPC, average annual percentage change

Discussion

No doubt there are historical examples of late lessons from early warnings on health risks whereby preventive measurements have been neglected. Some of the examples here clearly show that if the scientific evidence on cancer risks had been taken seriously lives could have been saved.

Tobacco is a good example of cancer risks that were disregarded for decades since clear evidence of increased risk. It was not until 1986 that IARC classified tobacco as a human carcinogen, Group 1 [25]. The strategies by the tobacco industry to sow doubt on the risks include e.g., to fund research that supports their position, to hide their involvement, to promote 'no risk' studies, to criticize research that shows risk, and to disseminate data and their interpretation of the results to the press and layman, for further details see Bero [98].

In fact, these strategies by the tobacco industry to obscure scientific facts seem to be textbook examples on product defense that may be used by different industries.

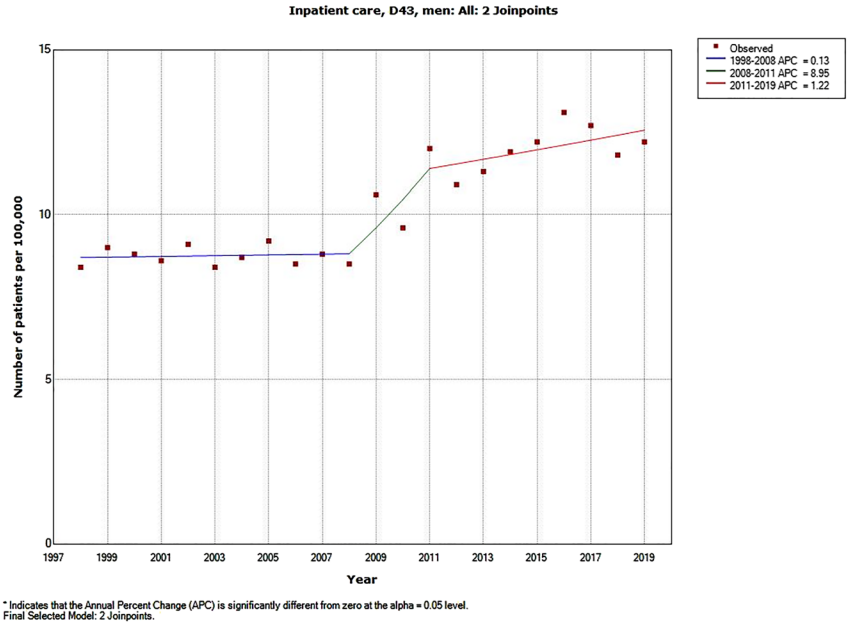

Figure 1: Joinpoint regression analysis of number of patients per 100,000 inhabitants. According to the Swedish National Inpatient Register for men, all ages during 1998–2019 diagnosed with D43 = tumour of unknown type in the brain or CNS. Note that in Sweden 1G (NMT, Nordic mobile telephone System) operated during 1981–2007. 2G (GSM) started 1991, 3G UMTS) started 2003, 4G started 2015, and DECT started 1988 [97].

One current controversy is cancer risks from RF radiation. No lessons on prevention of cancer risks seem to have been learned in spite of decades of publications on adverse health risks. In fact, early prevention is usually very cost effective [2, 99]. The issue on RF radiation risks is on-going and in fact increasing despite decades of research showing adverse effects on human health, plants, insects and birds. It seems as if the industry view of no risk dominates on national level [84], among many countries [85], also at EU level (www.5gappeal.eu), and even within WHO [88]. Notably such industry organizations and nations have the power and economic resources to suppress scientific evidence on risks and have access to mainstream media to propagate their views, may it be for political or economic reasons.

RF radiation is a current controversy regarding cancer risks. The 2011 IARC evaluation on carcinogenesis [62, 63] has been downplayed and detracted by industry and captured agencies from the very beginning in spite of increasing evidence on harmful effects. However, IARC has decided that a new evaluation of cancer risks is top priority within a few years [100].

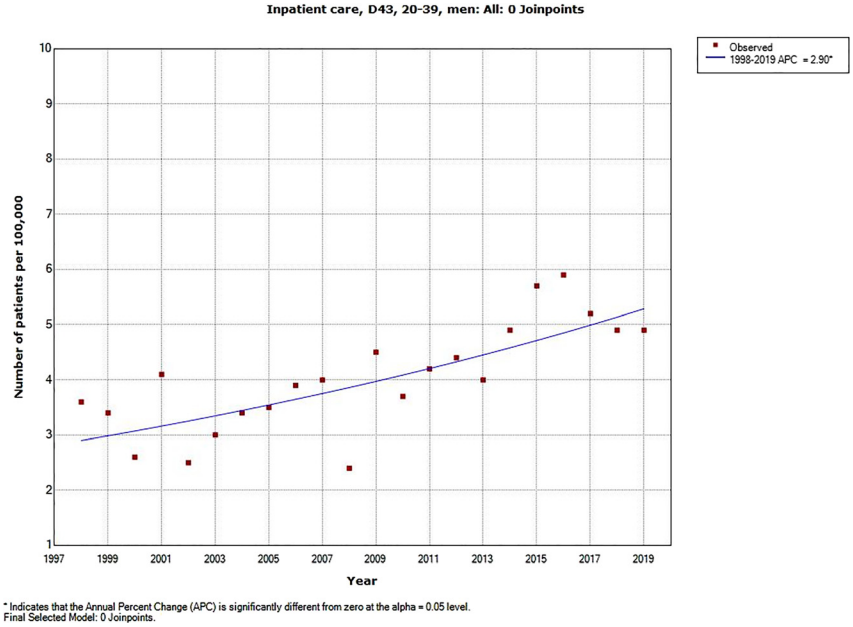

Figure 2: Joinpoint regression analysis of number of patients per 100,000 inhabitants. According to the Swedish National Inpatient Register for men aged 20–39 years during 1998–2019 diagnosed with D43 = tumour of unknown type in the brain or CNS.

Table 3: Joinpoint regression analysis of brain tumour rates (numbers per 100,000) in women in the Swedish Inpatient Register 1998–2019, ICD-10 code D43 (https://sdb.socialstyrelsen.se/if_par/val.aspx).

ICD-10	Joinpoint location	APC 1 (95% CI)	APC 2 (95% CI)	APC 3 (95 % CI)	AAPC (95% CI)
D43					
All women (n=9,611)	2008; 2017	+0.24 (−0.75, +1.24)	+4.77 (+3.32, +6.24)	−4.35 (−15.82, +8.68)	+1.70 (+0.38, +3.05)
0–19 years (n=570)	No joinpoint detected	–	–	–	+0.86 (−0.55, +2.28
20–39 years (n=907)	No joinpoint detected	–	–	–	+2.89 (+1.54, +4.27)
40–59 years (n=2,509)	No joinpoint detected	–	–	–	+1.91 (+0.80, +3.02)
60–79 years (n=4,307)	2005	−0.95 (−4.07, +2.27)	+3.45 (+2.30, +4.62)	–	+1.96 (+0,73, +3.21)
80+ years (n=1,318)	2010	−0.66 (−3.11, +1.84)	+5.49 (+1.51, +9.63)	–	+1.93 (−0.11, +4.02)

APC, annual percentage change (APC 1, time from 1998 to first joinpoint; APC 2, time from first joinpoint to 2019 or to second joinpoint; APC 3, time from second joinpoint to 2019); AAPC, average annual percentage change.

In this article we give some further data on the RF carcinogenesis. The attributable fraction gives the number of cases that could have been prevented if no risk exists for a specific exposure. Based on results in case-control studies from three study groups that have shown statistically significant increased risk for glioma and acoustic neuroma 211 glioma cases (all exposure) and 42 acoustic neuroma cases (ipsilateral exposure) would have been preventable in the longest cumulative exposure group. The preventable fraction was 4.88 and 4.63%, respectively. Highest preventable fraction was found for glioma with ipsilateral wireless phone use, 6.03% corresponding to 150 cases. Lower AF was calculated for meningioma, 1.75%, yielding 39 preventable cases (ipsilateral exposure). As displayed in Belpomme et al. [73] these results were based on Interphone [67], Coureau et al. [101], and Carlberg, Hardell [102], each without statistically significant increased risk. However, meta-analysis of these studies yielded, OR = 1.49, 95% CI = 1.08–2.06.

We have previously published results on increasing rates of tumors of unknown type in the brain or CNS both in the Swedish Inpatient Register and Causes of Death Register during 1998–2013 [103]. There was a clear increasing trend in both genders during that time period, especially during more recent years with AAPC +1.78 %, 95% CI + 0.76, 2.81% for both genders combined. A joinpoint was found in men in 2007; time period 2007–2013 APC +4.95%, 95% CI +1.59, +8.42%. Similarly, in women a joinpoint was detected in 2008; time period 2008–2013 APC +4.08%, 95% CI +1.80, +6.41%.

We have now extended the time period up to 2019. Thus, we report increasing AAPC in both genders during 1998–2019 of similar magnitude as previously. In men the result was of borderline significance although the AAPC

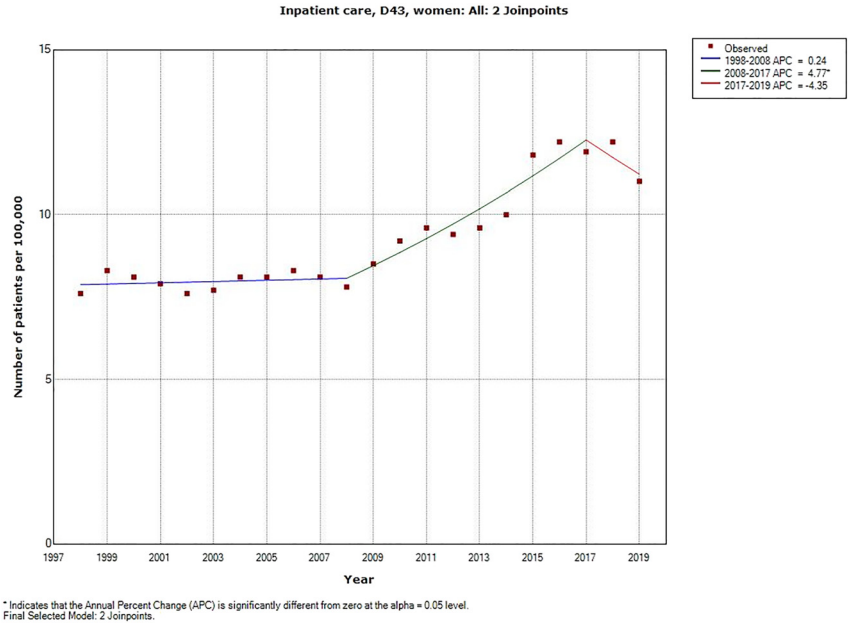

Figure 3: Joinpoint regression analysis of number of patients per 100,000 inhabitants. According to the Swedish National Inpatient Register for women, all ages during 1998–2019 diagnosed with D43 = tumour of unknown type in the brain or CNS. Note that in Sweden 1G (NMT; Nordic mobile telephone System) operated during 1981–2007. 2G (GSM) started 1991, 3G (UMTS) started 2003, 4G started 2015, and DECT started 1988 [97].

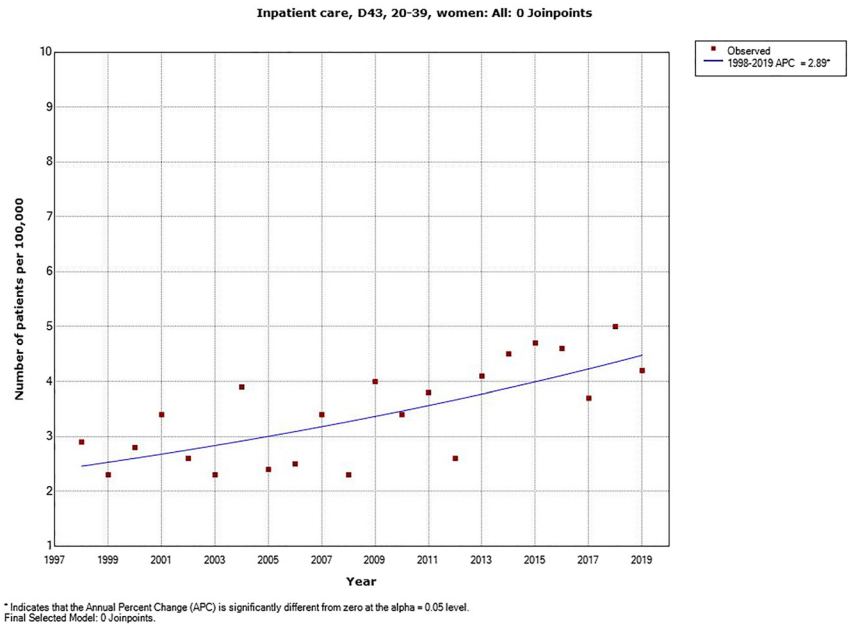

Figure 4: Joinpoint regression analysis of number of patients per 100,000 inhabitants. According to the Swedish National Inpatient Register for women aged 20–39 years during 1998–2019 diagnosed with D43 = tumour of unknown type in the brain or CNS.

overlapped previous findings. Lower APC was found during more recent years in both men and women, see Figures 1 and 3. This may reflect a better diagnostic procedure and thus decreasing numbers of unknown brain tumor type. A delay in reporting to the register during recent years may also have an impact on the results.

It is noteworthy that we found highest AAPC in the age group 20–39 years in both men and women, Tables 2 and 3. We found in our case-control study on glioma a median latency period for use of mobile phone of 9.0 years (mean 10.1 years). The corresponding results for cordless phones (DECT) were 7.0 and 8.0 years, respectively [104]. In a population-based study during 2005–2006 on use of mobile and cordless phones among Swedish children aged 7–14 years 79.1% reported access to mobile phone and use of cordless phone was reported by 83.8% [105]. Thus, our current findings with increasing numbers of brain tumors in the age group 20–39 years may be consistent with use of wireless phones taking a reasonable latency period. Moreover, our previous results showed highest risk for subjects that started the use of mobile or cordless phone before 20 years of age [104]. That age groups would also be more vulnerable to RF radiation [106]. In legends to Figures 1 and 3 we report the history for wireless phone use in Sweden. Figure 5 displays the number of out-going mobile phone minutes in millions during 2000–2019 in

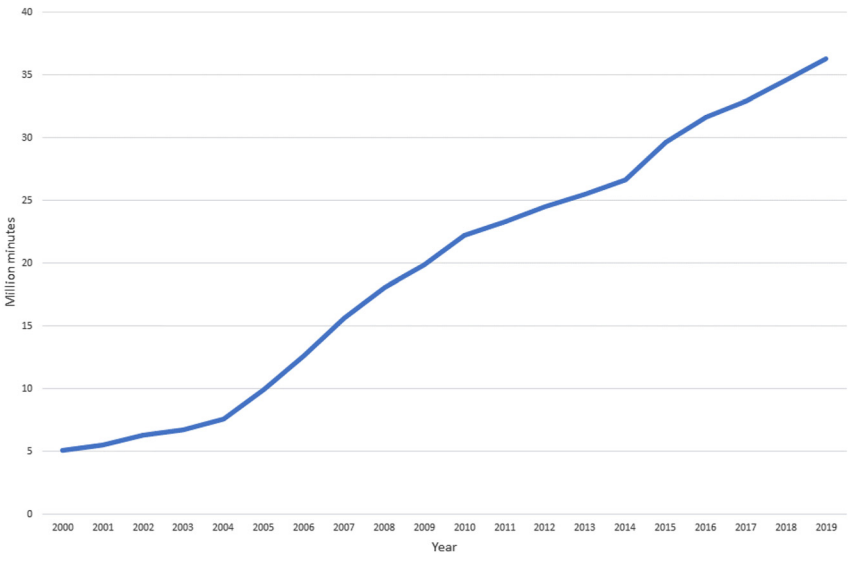

Figure 5: Number of out-going mobile phone minutes in millions during 2000–2019 in Sweden according to post-och Tele-styrelsen [The Swedish post and telecom Authority (PTS)]. Available from: https://statistik.pts.se/svensk-telekommarknad/tabeller/mobila-samtals-och-datatjanster/tabell-13-trafikminuter-utgaende/.

Sweden. The major increase is since early 21st century and may be associated with our findings of increasing numbers of brain tumors of unknown type considering a reasonable latency time.

As we have discussed elsewhere the Swedish Cancer Register is not reliable to study the incidence of brain tumors [103, 107]. The register is mainly based on reporting of cases with histopathological diagnosis. Now diagnosis may be based on CT and/or MRI without further investigations especially of patients with poor outcome. Biopsy or operation may be difficult to perform due to tumor location, age and co-morbidity. In the Swedish Cancer Register about 90% of the cases are diagnosed with cytology or histology, a number that has increased somewhat during recent years [107]. This fact indicates that brain tumors of unknown type are under-reported to the Cancer Register.

This review gives insight into missed opportunities for cancer prevention exemplified by asbestos, tobacco, certain pesticides and now RF radiation. No doubt economic considerations are favored instead of cancer prevention. The cancer victim is the loser in terms of suffering, life quality and shorter life expectancy. Also the life for the next-of-kin is affected. A strategy to sow doubt on cancer risks was established decades ago and is now adopted and implemented in more sophisticated way by the telecom industry regarding RF-EMF risks to human beings and the environment. Industry has the economic power, access to politicians and media whereas concerned people are unheard.

Acknowledgments: The authors want to thank The Electromagnetic Safety Alliance, Inc. USA for general support.
Research funding: None declared.
Author contributions: LH and MC contributed to the conception, design and writing of the manuscript. Both authors read and approved the final manuscript. MC made the statistical analyses.
Competing interests: Authors state no conflict of interest.
Informed consent: Informed consent is not applicable.
Ethical approval: Not applicable.

References

1. Hardell L, Walker MJ, Walhjalt B, Friedman LS, Richter ED. Secret ties to industry and conflicting interests in cancer research. Am J Ind Med 2002;50:227–33.
2. Hardell L. Pesticides, soft-tissue sarcoma and non-Hodgkin lymphoma–historical aspects on the precautionary principle in cancer prevention. Acta Oncol 2008;47:347–54.
3. Gee D. More or less precaution? In: European Environment Agency: late lessons from early warnings: science, precaution, innovation, Publications Office of European Union. EEA Report No 1. Luxembourg: European Environment Agency; 2013: 643–69 pp.
4. Hansen SF, Tickner JA. The precautionary principle and false alarms – lesson learned. In: Late lessons from early warnings: science, precaution, innovation, Publications Office of European Union. EEA Report No 1. Luxembourg: European Environment Agency; 2013:17–45 pp.
5. European Environment Agency. Late lessons from early warnings: the precautionary principle 1896-2000. Luxembourg: European Environment Agency. Environmental issue report 2001; No. 22.
6. European Environment Agency. Late lessons from early warnings: science, precaution, innovation. Luxembourg: Publications Office of European Union. EEA Report; 2013. No. 1.
7. Ramazzini B. De morbis artificum diatriba [diseases of workers]. 1713. Am J Public Health 2001;91:1380–2.
8. Pott P. Chirurgical observations relative to the cataract, the polypus of the nose, the cancer of the scrotum, the different kinds of ruptures, and the mortification of the toes and feet. London: Hawes, Clarke and Collins; 1775:63–8 pp.
9. IARC. Monographs on the evaluation of carcinogenic risks to humans polynuclear aromatic compounds, Part 4, Bitumens, coal-tars and derived products, shale-oils and soots. Lyon: IARC; 1985, vol 35.
10. Deane L. Report on the health of workers in asbestos and other dusty trades. In: HM Chief Inspector of Factories and Workshops. Annual Report for 1898. London: HMSO; 1899:171–2 pp.
11. Lynch KM, Smith WA. Pulmonary asbestosis III: carcinoma of lung in asbesto-silicosis. Am J Cancer1935;24:56–64.
12. Weiss A. Pleurakrebs bei lungenasbestose, in vivo morphologisch gesichert [Cancer of pleura in pulmonary asbestosis determined morphologically in vivo]. Medizinische 1953;6:93–4.
13. Wagner JC, Sleggs CA, Marchand P. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Ind Med 1960;17:260–71.
14. Selikoff IJ, Churg J, Hammond EC. Asbestos exposure and neoplasia. J Am Med Assoc 1964;188:22–6.
15. Tweedale G. Secret ties in asbestos – downplaying and effacing the risks of toxic minerals. In: Walker MJ, editor. Corporate ties that bind. New York: Skyhorse Publishing; 2017:136–51 pp.
16. Ruff K. Serving industry, promoting skepticism, discrediting epidemiology. In: Walker MJ, editor. Corporate ties that bind. New York: Skyhorse Publishing; 2017:119–35 pp.
17. IARC. Monographs on the evaluation of carcinogenic risks to humans asbestos. Lyon: IARC; 1977, vol 14.
18. James A. Counterblaste to Tobacco (Published anonymously; republished, 1954). London: The Rodale Press; 1604.
19. Sömmering ST. De morbis vasorum absorbentium corporis humani. Frankfurt: Varrentrapp & Wenner; 1795.
20. Anon. Cancer and smoking. Br Med J 1890;1:748.
21. Brinkmann H. Statistiche Übersicht über 108 Fälle von Primären Bronchial- und Lungencarcinom, nebst einem Fall von Bronchuscarcinom mit Tödlicher Arrosion der Arteria Pulmonalis [Statistical Overview of 108 Cases of Primary Bronchial and Lung Carcinoma together with One Case of Carcinoma of the Bronchus with Fatal Erosion of the Pulmonary Artery]. Leipzig: Verlag Emil Lehmann; 1914.
22. Müller FH. Tabakmißbrauch und Lungencarcinom [Tobacco abuse and lung carcinoma]. Z Krebsforsch 1940;49:57–85.

23. Schairer E, Schöniger E. Lungenkrebs und Tabakverbrauch [Lung cancer and tobacco consumption]. Z Krebsforsch 1944;54:261–9.
24. Wassink WF. Distribution of lung cancer by occupation (Dutch). Ned Tijdschr Geneeskd 1948;92:3732–47.
25. IARC. Monographs on the evaluation of carcinogenic risks to humans tobacco smoking. Lyon: IARC; 1986, vol 38.
26. Walhjalt B, Greenwashing. The Swedish experience. In: Walker MJ, editor. Corporate ties that bind. New York: Skyhorse Publishing; 2017:96–108 pp.
27. Carson R. Silent spring. Boston: Houghton Mifflin; 1962.
28. Walker MJ. A dark culture – the history and literature of health-damaging production, its exposure, and its corporate defense. In: Walker MJ, editor. Corporate ties that bind. New York: Skyhorse Publishing; 2017:1–40 pp.
29. IARC. Monographs on the evaluation of carcinogenic risks to humans DDT, Lindane, and 2,4-D. Lyon: International Agency for Research on Cancer; 2018, vol 113.
30. UNEP. The Stockholm convention on persistent organic pollutants. Geneva: United Nations Environment Programme; 2002. Available from: www.pops.int.
31. Turusov V, Rakitsky V, Tomatis L. Dichlorodiphenyltrichloroethane (DDT): ubiquity, persistence, and risks. Environ Health Perspect 2002;110:125–8.
32. EPA. DDT - a brief history and status. Washington (DC), USA: United States Environmental Protection Agency; 2015. Available from: http://www2.epa.gov/ingredients-used-pesticide-products.
33. IARC. Monographs on the evaluation of carcinogenic risks to humans some organochlorine pesticides. Lyon: IARC; 1974, vol 5.
34. Liljegren G, Hardell L, Lindström G, Dahl P, Magnuson A. Case-control study on breast cancer and adipose tissue concentrations of congener specific polychlorinated biphenyls, DDE and hexachlorobenzene. Eur J Cancer Prev 1998;7:135–40.
35. Hardell L, van Bavel B, Lindström G, Björnfoth H, Orgum P, Carlberg M, et al. Adipose tissue concentrations of p,p'-DDE and the risk for endometrial cancer. Gynecol Oncol 2004;95:706–11.
36. Hardell L. Maligna mesenkymala mjukdelstumörer och exposition för fenoxisyror - en klinisk observation [Malignant mesenchymal soft tissue tumours and exposure to phenoxyacetic acids – a clinical observation]. Lakartidningen 1977;77:2753–4.
37. Hardell L, Sandström A. Case-control study: soft-tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. Br J Cancer 1979;39:711–7.
38. Hardell L. Malignant lymphoma of histiocytic type and exposure to phenoxyacetic acids or chlorophenols. Lancet 1979;1:55–6.
39. Hardell L, Eriksson M, Lenner P, Lundgren E. Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: a case-control study. Br J Cancer 1981;43:169–76.
40. Hardell L, Eriksson M. Epidemiological studies on cancer and exposure to dioxins and related compunds. In: Schecter A, editor. Dioxins and health including other persistent organic pollutants and endocrine disruptors. New Jersey: Wiley; 2012:303–58 pp.
41. IARC. Monographs on the evaluation of carcinogenic risks to humans pentachlorophenol and some related compounds. Lyon: IARC; 2019, vol 117.
42. IARC. Monographs on the evaluation of carcinogenic risks to humans some fumigants, the herbicides 2,4-D and 2,4,5-T, chlorinated dibenzodioxins and miscellaneous industrial chemicals. Lyon: IARC; 1977, vol 15.
43. Hammond S, Schecter A. Agent Orange. Health and environmental issues in Vietnam, Cambodja, and Laos. In: Schecter A, editor. Dioxins and health including other persistent organic pollutants and endocrine disruptors. New Jersey: Wiley; 2012:469–520 pp.
44. Pesatori AC, Bertazzi PA. The Seveso accident. In: Schecter A, editor. Dioxins and health including other persistent organic pollutants and endocrine disruptors. New Jersey: Wiley; 2012: 445–68 pp.
45. IARC. Monographs on the evaluation of carcinogenic risks to humans polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans. Lyon: IARC; 1997, vol 69.
46. Hardell L, Eriksson M. A case-control study of non-Hodgkin lymphoma and exposure to pesticides. Cancer 1999;85:1353–60.
47. Eriksson M, Hardell L, Carlberg M, Akerman M. Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis. Int J Cancer 2008;123: 1657–63.
48. Nordström M, Hardell L, Magnuson A, Hagberg H, Rask-Andersen A. Occupational exposures, animal exposure and smoking as risk factors for hairy cell leukaemia evaluated in a case-control study. Br J Cancer 1998;77:2048–52.
49. McDuffie HH, Pahwa P, McLaughlin JR, Spinelli JJ, Fincham S, Dosman JA, et al. Non-Hodgkin's lymphoma and specific pesticide exposures in men: cross-Canada study of pesticides and health. Cancer Epidemiol Biomarkers Prev 2001;10:1155–63.
50. De Roos AJ, Zahm SH, Cantor KP, Weisenburger DD, Holmes FF, Burmeister LF, et al. Integrative assessment of multiple pesticides as risk factors for non-Hodgkin's lymphoma among men. Occup Environ Med 2001;60:E11.
51. Portier CJ. A comprehensive analysis of the animal carcinogenicity data for glyphosate from chronic exposure rodent carcinogenicity studies. Environ Health 2020;19:18.
52. Benbrook CM. Trends in glyphosate herbicide use in the United States and globally. Environ Sci Eur 2016;28:3.
53. Guyton KZ, Loomis D, Grosse Y, El Ghissassi F, Benbrahim-Tallaa L, Guha N, et al. Carcinogenicity of tetrachlorvinphos, parathion, malathion, diazinon, and glyphosate. Lancet Oncol 2015;16: 490–1.
54. IARC. Monographs on the evaluation of carcinogenic risks to humans some organophosphate insecticides and herbicides. Lyon: IARC; 2017, vol 112.
55. EFSA (European Food Safety Authority). Conclusion on the peer review of the pesticide risk assessment of the active substance glyphosate. EFSA J 2015;13:4302.
56. Portier CJ, Armstrong BK, Baguley BC, Baur X, Belyaev I, Bellé R, et al. Differences in the carcinogenic evaluation of glyphosate between the International agency for Research on cancer (IARC) and the European Food Safety Authority (EFSA). J Epidemiol Community Health 2016;70:741–5.
57. Williams GM, Aardema M, Acquavella J, Berry SC, Brusick D, Burns MM, et al. A review of the carcinogenic potential of glyphosate by four independent expert panels and comparison to the IARC assessment. Crit Rev Toxicol 2016;46(supp1):3–20.
58. Michaels D. The triumph of doubt. Oxford: Oxford University Press; 2020:157–60 pp.
59. Williams GM, Aardema M, Acquavella J, Berry SC, Brusick D, Burns MM, et al. Corrigendum. Erratum for glyphosate rodent

carcinogenicity bioassay expert panel review. Crit Rev Toxicol 2016;48:902. Crit Rev Toxicol 2018.
60. Gillam C. Revealed: Monsanto predicted crop system would damage US farms. The Guardian; 2020. Available from: https://www.theguardian.com/us-news/2020/mar/30/monsanto-crop-system-damage-us-farms-documents.
61. European Commission. Glyphosate. Status of glyphosate in the EU. Available from: https://ec.europa.eu/food/plant/pesticides/glyphosate_en.
62. Baan R, Grosse Y, Lauby-Secretan B, El Ghissassi F, Bouvard V, Benbrahim-Tallaa L, et al. Carcinogenicity of radiofrequency electromagnetic fields. Lancet Oncol 2011;12:624–6.
63. IARC. Monographs on the evaluation of carcinogenic risks to humans, non-ionizing radiation, part 2: radiofrequency electromagnetic fields. Lyon: International Agency for Research on Cancer; 2013, vol 102.
64. Hardell L, Carlberg M, Hansson Mild K. Pooled analysis of two case-control studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997–2003. Int Arch Occup Environ Health 2006;79:630–9.
65. Hardell L, Carlberg M, Hansson Mild K. Pooled analysis of two case-control studies on the use of cellular and cordless telephones and the risk of benign brain tumours diagnosed during 1997–2003. Int J Oncol 2006;28:509–18.
66. Hardell L, Carlberg M, Hansson Mild K. Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects. Int J Oncol 2011;38:1465–74.
67. Interphone Study Group. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. Int J Epidemiol 2010;39:675–94.
68. Interphone Study Group. Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. Cancer Epidemiol 2011;35:453–64.
69. Cardis E, Armstrong BK, Bowman JD, Giles GG, Hours M, Krewski D, et al. Risk of brain tumours in relation to estimated RF dose from mobile phones: Results from five Interphone countries. Occup Environ Med 2011;68:631–40.
70. Repacholi MH, Basten A, Gebski V, Noonan D, Finnie J, Harris AW. Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 900 MHZ electromagnetic fields. Radiat Res 1997;147:631–40.
71. Tillmann T, Ernst H, Streckert J, Zhou Y, Taugner F, Hansen V, et al. Indication of cocarcinogenic potential of chronic UMTS-modulated radiofrequency exposure in an ethylnitrosourea mouse model. Int J Radiat Biol 2010;86:529–41.
72. IARC. Monographs on the evaluation of carcinogenic risks to humans, non-ionizing radiation, part 1: static and extremely low-frequency. (ELF) Electr Magn Fields; 2002, vol 80.
73. Belpomme D, Hardell L, Belyaev I, Burgio E, Carpenter DO. Thermal and non-thermal health effects of non-ionizing radiation: an international perspective. Environ Pollut 2018;242:643–58.
74. Miller AB, Sears ME, Morgan LL, Davis DL, Hardell L, Oremus M, et al. Risks to health and well-being from radio-frequency radiation emitted by cell phones and other wireless devices. Front Public Health 2019;7:223.
75. National Toxicology Program. NTP technical report on the toxicology and carcinogenesis studies in B6C3F1/N mice exposed to whole-body radio frequency radiation at a frequency (1,900 MHz) and modulations (GSM and CDMA) used by cell phones. NTP TR 596, March 26–28; 2018. Available from: https://ntp.niehs.nih.gov/ntp/about_ntp/trpanel/2018/march/tr596peerdraft.pdf.
76. National Toxicology Program. NTP technical report on the toxicology and carcinogenesis studies in Hsd:Sprague Dawley sd rats exposed to whole-body radio frequency radiation at a frequency (900 MHz) and modulations (GSM and CDMA) used by cell phones. NTP TR 595, March 26–28; 2018. Available from: https://ntp.niehs.nih.gov/ntp/about_ntp/trpanel/2018/march/tr595peerdraft.pdf.
77. Hardell L, Carblerg M. Comments on the US National Toxicology Program technical reports on toxicology and carcinogenesis study in rats exposed to whole-body radiofrequency radiation at 900 MHz and in mice exposed to whole-body radiofrequency radiation at 1,900 MHz. Int J Oncol 2019;54:111–27.
78. Falcioni L, Bua L, Tibaldi E, Lauriola M, De Angelis L, Gnudi F, et al. Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8 GHz GSM base station environmental emission. Environ Res 2018;165:496–503.
79. Hardell L, Carblerg M, Hedendahl LK, Koppel T, Ahonen M. Environmental radiofrequency radiation at the Järntorget Square in Stockholm Old Town, Sweden in May, 2018 compared with results on brain and heart tumour risks in rats exposed to 1.8 GHz base station environmental emissions. World Acad Sci J 2019;1:47–54.
80. Yakymenko I, Tsybulin O, Sidorik E, Henshel D, Kyrylenko O, Kyrylenko S. Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation. Electromagn Biol Med 2016;35:186–202.
81. Smith-Roe SL, Wyde ME, Stout MD, Winters JW, Hobbs CA, Shepard KG, et al. *E*valuation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure. Environ Mol Mutagen 2020;61:276–90.
82. Carlberg M, Hardell L. Evaluation of mobile phone and cordless phone use and glioma risk using the Bradford Hill viewpoints from 1965 on Association or Causation. Bio Med Res Int 2017;2017:9218486.
83. Peleg M, Nativ O, Richter ED. Radio frequency radiation-related cancer: assessing causation in the occupational/military setting. Environ Res 2018;163:123–33.
84. Hardell L. Comment. Notes on parliament hearing in Tallinn, Estonia June 4, 2019 as regards the deployment of the fifth generation, 5G, of wireless communication. World Acad Sci J 2019;1:275–82.
85. Hardell L, Nyberg R. Appeals that matter or not on a moratorium on the deployment of the fifth generation, 5G, for microwave radiation. Mol Clin Oncol 2020;12:247–57.
86. International Commission on Non-Ionizing Radiation Protection (ICNIRP). Guidelines for limiting exposure to electromagnetic Fields (100 kHz to 300 GHz). Health Phys 2020;118:483–524.

87. Starkey SJ. Inaccurate official assessment of radiofrequency safety by the Advisory Group on non-ionising Radiation. Rev Environ Health 2016;31:493–503.
88. Hardell L. World Health Organization, radiofrequency radiation and health - a hard nut to crack (Review). Int J Oncol 2017;51: 405–13.
89. International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Phys 1998;74:494–522.
90. International Commission on Non-Ionizing Radiation Protection. ICNIRP statement on the "Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Phys 2009;97:257–8.
91. Belyaev I, Dean A, Eger H, Hubmann G, Jandrisovits R, Kern M, et al. EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses. Rev Environ Health 2016;31:363–97.
92. Bioinitiative: Bioinitiative. A rationale for biologically-based exposure standards for low-intensity electromagnetic radiation; 2012. Available from: https://bioinitiative.org/.
93. Grigoriev Y. Russian National Committee on non-ionizing radiation protection and EMF RF standards. New conditions of EMF RF exposure and guarantee of the health to population. Available from: https://www.radiationresearch.org/wp-content/uploads/2018/06/021235_grigoriev.pdf.
94. The National Board of Health and Welfare. Available from: Statistikdatabaser - Diagnoser - Val (socialstyrelsen.se.
95. Ludvigsson JF, Andersson E, Ekbom A, Feychting M, Kim JL, Reuterwall C, et al. External review and validation of the Swedish national inpatient register. BMC Public Health 2011;11:450.
96. Kim HJ, Fay MP, Feuer EJ, Midthune DN. Permutation tests for joinpoint regression with applications to cancer rates. Stat Med 2000;19:335–51.
97. Hardell L, Carlberg M. Mobile and cordless phone use and brain tumor risk. In: Rosch PJ, editor. Bioelectromagnetic and subtle energy medicine. Boca Raton, FL: CRC Press; 2015:539–58 pp.
98. Bero LA. Tobacco industry manipulation of research. In: Late lessons from early warnings: science, precaution, innovation, EEA Report. Luxembourg: European Environment Agency; 2013: 151–78 pp.
99. Skou Andersen M, Owain Chubb D. Understanding and accounting for the costs of inacation. In: Late lessons from early warnings: science, precaution, innovation. Luxembourg: European Environment Agency; 2013:564–80 pp.
100. IARC Monographs Priorities Group. Advisory group recommendations on priorities for the IARC monographs. Lancet Oncol 2019;20:763–4.
101. Coureau G, Bouvier G, Lebailly P, Fabbro-Peray P, Gruber A, Leffondre K, et al. Mobile phone use and brain tumours in the CERENAT case-control study. Occup Environ Med 2014;71: 514–22.
102. Carlberg M, Hardell L. Pooled analysis of Swedish case-control studies during 1997-2003 and 2007-2009 on meningioma risk associated with the use of mobile and cordless phones. Oncol Rep 2015;33:3093–8.
103. Hardell L, Carlberg M. Increasing rates of brain tumours in the Swedish national inpatient register and the causes of death register. Int J Environ Res Publ Health 2015;12:3793–37813.
104. Hardell L, Carlberg M. Mobile phone and cordless phone use and the risk for glioma – analysis of pooled case-control studies in Sweden, 1997-2003 and 2007-2009. Pathophysiology 2015;22: 1–13.
105. Söderqvist F, Hardell L, Carlberg M, Hansson Mild K. Ownership and use of wireless telephones: a population-based study of Swedish children aged 7-14 years. BMC Public Health 2007;7: 105.
106. Morgan LL, Kesari S, Davis DL. Why children absorb more microwave radiation than adults: the consequences. J Microsc Ultrastruct 2014;2:197–204.
107. Hardell L, Carlberg M. Mobile phones, cordless phones and rates of brain tumors in different age groups in the Swedish National Inpatient Register and the Swedish Cancer Register during 1998-2015. PLoS One 2017;12:e0185461.
108. Hardell L, Carlberg M, Söderqvist F, Hansson Mild K. Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997-2003 and 2007-2009 and use of mobile and cordless phones. Int J Oncol 2013;43:1036–44.