



EVALUATION OF CARCINOGENIC EFFECTS OF ELECTROMAGNETIC FIELDS (EMF)

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ABSTRACT

The purpose of this study was to investigate the carcinogenic effects of electromagnetic fields on human. There are many effects of electromagnetic fields on human such as cancer, epidemiology, acute and chronic effects. These effects vary according to the field strength and environmental conditions. There have been many instances of harmful effects of electromagnetic fields from such seemingly innocuous devices as mobile phones, computers, power lines and domestic wiring. The balance of epidemiologic evidence indicates that mobile phone use of less than 10 years does not pose any increased risk of brain tumour or acoustic neuroma. For long-term use, data are sparse, and the following conclusions are therefore uncertain and tentative.

KEY WORDS: electromagnetic field, cancer

INTRODUCTION

International Agency for Research on Cancer (IARC) classification of Extremely Low Frequency (ELF) magnetic fields as “possibly carcinogenic to humans” is based upon all of the available data prior to and including 2001 (IARC, 2002). Generally, studies of the effects of ELF field exposure of cells have shown no induction of genotoxicity at fields below 50 μ T. The notable exception is evidence from recent studies reporting DNA damage at field strengths as low as 35 μ T; however, these studies are still being evaluated and our understanding of these findings is incomplete (1,2,3). There is also increasing evidence that ELF magnetic fields may interact with DNA-damaging agents (4,5). There is no clear evidence of the activation by ELF magnetic fields of genes associated with the control of the cell cycle (6). However, systematic studies analysing the response of the whole genome have yet to be performed. Many other cellular studies, for example on cell proliferation, apoptosis, calcium signalling and malignant transformation, have produced inconsistent or inconclusive results (7-12). There have been many studies suggesting that exposure to weak electromagnetic fields is associated with a small but significant increase in the risk of getting cancer (13,14). This could be caused by gene mutations resulting from DNA damage. A gene is a section of DNA containing the information needed to make a particular protein or enzyme. There is also a section that can turn the gene on or off in response to outside signals. The growth of an organism from a fertilised egg involves a hugely

complex pattern of switching genes on and off that regulates growth, cell division and differentiation into specific tissues. DNA damage can sometimes give unregulated growth to form tumours. However, the effect may not be immediate. The affected cells seem to go through several stages of ever increasing genetic and molecular anarchy before they finally reach the point of unstoppable growth and division. When assessing any carcinogenic effects of electromagnetic exposure, we must bear in mind that there may be a similar delay. It may be some years before we know the full carcinogenic effects of the recent explosive growth in the use of mobile phones.

CANCER AND EMF

In total, about 30 papers of original studies on mobile phone use and cancer were published in the last five years. Results are summarized in Table 1. for brain tumours and in Table 2. for acoustic neuroma. All but one study were case-control studies, mostly on brain tumours, some on salivary gland tumours or uvula melanoma. One was a large cohort study of all Danish mobile phone subscribers between 1982 and 1995 who were followed up for a variety of cancers; no increased risk for any cancer was observed but follow up time was short (15). The Interphone study is a multinational case-control study coordinated by the IARC. It is a population-based study with prospective ascertainment of incident cases and face-to-face interviews for exposure assessment. With regard to brain tumours, results from the first four components of the Interphone study sug-

Brain Tumours		Brain Tumours Short Latency		Brain Tumours Longer Latency	
No. Exp. Cases	RR ¹⁶ estimate (95%CI ¹⁷)	No. Exp. Cases	RR estimate (95%CI)	No. Exp. Cases	RR estimate (95%CI)
78	1,0 (0,7-1,4)	78	1,0 (0,7-1,4) >1 yr	34 16	(0,5-1,4)>5 yr 1,2 (0,6-2,6) >10 yr
66	0,8 (0,6-1,2)	28	1,1 (0,6-2,0) 2-3 yr	17	0,7 (0,7-1,4) \geq 4yr
139	0,8 (0,6-1,1)	51	1,0 (0,6-1,6) 0,5-3 yr	54 22	(0,6-1,6) \geq 3 yr 0,7 (0,4-1,4) \geq 5 yr
154	1,0 (0,8-1,1)	87	(0,9-1,3) 1-4 yr	24	1,0 (0,7-1,6) >5 yr
40 analogue	1,3 (0,9-1,8)	15 analogue	(0,7-2,0)	17 analogue	1,5 (0,9-2,5) >2yr
16 digital		11 digital	1-2 yr	1 digital	
188 analogue	1,3 (1,0-1,6)	188 analogue	(1,0-1,6) > 1 yr	46 analogue	1,3 (0,8-2,3) >10 yr
224 digital	1,0 (0,8-1,2)	224 digital	1,0 (0,8-1,2) >1 yr	33 digital	0,9 (0,6-1,5) >5 yr
214 glioma	0,8 (0,6-1,0)	112	0,8 (0,6-1,1) 1-4 yr	25	(0,5-1,5) \geq 10 yr
118 meningioma	0,7 (0,5-0,9)	64	0,6 (0,4-0,9) 1-4 yr	12	0,9 (0,4-1,9) \geq 10 yr
47 low-grade glioma	1,1 (0,6-2,0)	19	0,9 (0,4-1,8) 1-4 yr	6	(0,4-6,1) \geq 10 yr
59 high-grade glioma	0,6 (0,4-0,9)	24	0,6 (0,3-1,0) 1-4 yr	8	0,5 (0,2-1,3) \geq 10 yr
67 meningioma	0,8 (0,5-1,3)	35	0,8 (0,5-1,3) 1-4 yr	6	1,0 (0,3-3,2) \geq 10 yr

¹⁶ RR- Relative Risk ¹⁷ CI- Confidence Interval

TABLE 1. Results of epidemiological studies on mobile phone use and brain tumours (19,29,46,47,48).

Acoustic Neuroma		Acoustic Neuroma Short Latency		Acoustic Neuroma Longer Latency	
No. Exp. Cases	RR ¹⁸ (95%CI ¹⁹)	No. Exp. Cases	RR (95%CI)	No. Exp. Cases	RR (95%CI)
5	0,8 (0,1-4,2)				
22	1,0 (0,5-1,9)	8	1,8(0,7-4,5) 0,5-2 yr	5	1,9 (0,6-5,9)
7	0,6 (0,3-1,3)				
		7	0,5 (0,2-1,3) 1-2 yr	11	1,7 (0,5-5,1) 3-6 yr
38* analogue	3,5 (1,8-6,8)	12* analogue	3,0 (1,0-9,3) 1-5 yr	7* analogue	3,5 (0,7-16,8) >10 yr
23* digital	(0,7-2,2)	21* digital	1,2 (0,6-2,2) 1-5 yr	2* digital	2,0 (0,2-22,1) >5 yr
89	(0,6-1,5)	44	0,8 (0,5-1,3) 1-4 yr	14	1,9 (0,9-4,1) ≥10 yr
45	0,9 (0,5-1,6)	23	0,9 (0,5-1,6) 1-4 yr	2	(0,0-1,1) ≥10 yr
20 analogue	4,2 (1,8-10) 2,0 (1,0-3,8)	2 analogue	9,9 (1,4-69) 1-5 yr	11 analogue	5,1 (1,9-14) 5-10 yr
53 digital		29 digital	1,7 (0,9-3,5) yr	7 analogue	2,6 (0,9-8,0) >10 yr
				23 digital	2,7 (1,3-5,7) 5-10 yr
360	0,9 (0,7-1,1)	174	0,8 (0,7-1,0) 1,5-4 yr	139	0,9 (0,7-1,2) 5-9 yr
				47	(0,7-1,5) ≥10 yr

* Discordant pairs ¹⁸ RR- Relative Risk ¹⁹ CI- Confidence Interval

TABLE 2. Results of epidemiological studies on mobile phone use and acoustic neuroma (16, 18, 19, 22, 45, 46, 47)

gest no risk increase for meningioma or glioma. This is consistently so among subjects with less than 10 years of use. For regular mobile phone users of 10 years or more, no indications of risk increases were seen in three out of four components, namely in Sweden, Denmark and the UK (16) but the German component does see a somewhat raised relative risk estimate for glioma (17). This increase, however, is based on small numbers and due to the wide confidence interval the result is not in contradiction with the other Interphone components. In contrast, a Swedish group not participating in the Interphone-study, conducting several case-control studies using self-administered questionnaires for exposure assessment, has repeatedly observed increased relative risk estimates for brain tumours and is the only group that observed such an increase already after few years of use of a mobile phone (18,19).

Acoustic neuromas, benign tumours that develop very slowly, arise from the Schwann cells, which enfold the vestibulocochlear nerve. They are of particular interest because of their location. The Hardell-group from Sweden has in several studies reported raised relative risk estimates for acoustic neuroma and also with very short induction periods (19). Two of the Interphone components, Denmark and Sweden, have reported their country specific acoustic neuroma results (20,21). Lönn et al. reported a doubling of the relative risk estimate after ten years of regular mobile phone use compared to subjects who never used a mobile phone regularly. This association became stronger when the analysis was restricted to preferred phone use at the same side as the tumour. Christensen's result did not support this, but it was based on fewer long-term users. Six of thirteen components of Interphone (including Sweden and Denmark) were pooled for a joint analysis to examine the associa-

tion between mobile phone use and risk of acoustic neuroma (22). While no overall association was seen among all long-term users, the data suggest that there may be an increased risk when the preferred side of the head of use is considered in the analysis. For 10+ years of use of mobile phones, the relative risk for acoustic neuroma at the preferred side of use was 1,8. Because of methodological differences between studies, it would have been of considerable interest to compare the results across the six studies, but small numbers in most of the centres preclude that analysis. However, in an attempt to separate the effect in the four additional studies, the Danish and the Swedish studies were excluded from the pooling, which resulted in an increase of the pooled relative risk estimate. This indicates that the association seen by Schoemaker was not only driven by the Swedish data. All those studies are facing limitations in their exposure assessment, which was either a list of subscribers from the operators or self-reported mobile phone use. While the first method is an objective measure, it has limitations because subscription predicts use of a mobile phone only to some extent. Recent validation studies in volunteers comparing current self-reported use with traffic records from network operators show a moderate agreement, but it cannot be excluded that agreement is worse with respect to past mobile phone use or among patients with brain tumours. Especially patients with high stage glioma showed some memory performance problems in the Danish Interphone study (20). Childhood brain cancer studies have shown inconsistent results. As with childhood leukaemia, a pooled analysis of childhood brain cancer studies should be very informative and is therefore recommended. A pooled analysis of this kind can inexpensively provide a greater and improved insight into the existing data, including the possibility of selection bias and, if the studies are

sufficiently homogeneous, can offer the best estimate of risk (23,24). For adult breast cancer more recent studies have convincingly shown no association with exposure to ELF magnetic fields. Therefore further research into this association should be given very low priority (25-27). For adult leukaemia and brain cancer the recommendation is to update the existing large cohorts of occupationally exposed individuals. Occupational studies, pooled analyses and meta-analyses for leukaemia and brain cancer have been inconsistent and inconclusive. However, new data have subsequently been published and should be used to update these analyses (20,29,30). The priority is to address the epidemiological evidence by establishing appropriate *in vitro* and animal models for responses to low-level ELF magnetic fields that are widely transferable between laboratories. Transgenic rodent models for childhood leukaemia should be developed in order to provide appropriate experimental animal models to study the effect of ELF magnetic field exposure (21, 31,32). Otherwise, for existing animal studies, the weight of evidence is that there are no carcinogenic effects of ELF magnetic fields alone. Therefore high priority should be given to *in vitro* and animal studies in which ELF magnetic fields are rigorously evaluated as a co-carcinogen. With regard to other *in vitro* studies, experiments reporting the genotoxic effects of intermittent ELF magnetic field exposure should be replicated (14,33). Three independent large-scale studies of rats provided no evidence of an effect of ELF magnetic fields on the incidence of spontaneous mammary tumours (34-35). Most studies report no effect of ELF magnetic fields on leukaemia or lymphoma in rodent models. Several large-scale long-term studies in rodents have not shown any consistent increase in any type of cancer, including haematopoietic, mammary, brain and skin tumours (36-38). A substantial number of studies have ex-

amined the effects of ELF magnetic fields on chemically-induced mammary tumours in rats (39,40). Inconsistent results were obtained that may be due in whole or in part to differences in experimental protocols, such as the use of specific sub-strains. Most studies on the effects of ELF magnetic field exposure on chemically-induced or radiation-induced leukaemia/lymphoma models were negative. Studies of pre-neoplastic liver lesions, chemically-induced skin tumours and brain tumours reported predominantly negative results. One study reported an acceleration of UV-induced skin tumourigenesis upon exposure to ELF magnetic fields (37). Two groups have reported increased levels of DNA strand breaks in brain tissue following *in vivo* exposure to ELF magnetic fields. However, other groups, using a variety of different rodent genotoxicity models, found no evidence of genotoxic effects. The results of studies investigating non-genotoxic effects relevant to cancer are inconclusive. Overall there is no evidence that exposure to ELF magnetic fields alone causes tumours. The evidence that ELF magnetic field exposure can enhance tumour development in combination with carcinogens is inadequate.

The power and mobile phone companies, hoping to avoid litigation, often assert that because the energy of the fields is too low to give significant heating, they cannot have any biological effect (41,42). However, the evidence that electromagnetic fields can have "non-thermal" biological effects is now overwhelming (43-44). In addition, it is also recommended that the existing pooled analyses be updated, by adding data from recent studies and by applying new insights into the analysis. For new epidemiological studies to be informative they must focus on new aspects of exposure, potential interaction with other factors or on high exposure groups, or otherwise be innovative in this area of research (Table 3).

Cancer	Recommendation
Update existing pooled analyses of childhood leukaemia with new information	High
Pooled analyses of existing studies of childhood brain tumour studies	High
Update existing pooled and meta-analyses of adult leukaemia and brain tumour studies and of cohorts of occupationally exposed individuals	Medium
Development of transgenic rodent models of childhood leukaemia for use in ELF studies	High
Evaluation of co-carcinogenic effects using <i>in vitro</i> and animal studies	High
Attempted replication of <i>in vitro</i> genotoxicity studies	Medium

TABLE 3. Recommendations for further research sources, measurements and exposures

CONCLUSION

Consequently, in this review, we may assert that there are significant effects of EMF on the cancer development. In addition more comprehensive future investigations are required to determine definite effects of EMF on human diseases.

List of Abbreviations

EMF	-	Electromagnetic Field
ELF	-	Extremely Low Frequencies
IARC	-	International Agency for Research on Cancer

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