Effetti dei campi elettromagnetici nei lavoratori professionalmente esposti: le evidenze scientifiche, le decisioni della magistratura

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Università degli Studi di Modena e Reggio Emilia

AMBIENTE LAVORO
CONVENTION NAZIONALE

CAMPI ELETTROMAGNETICI:

Gli effetti sulla salute:

I risultati della ricerca scientifica

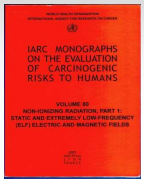
www.emf-portal.de

Database of publications:

May 2013: 17.315
May 2015: 21.231
May 2017: 24.812

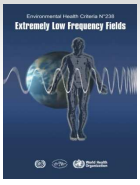
Principali rassegne sugli effetti dei CEM

ELF



IARC 2002

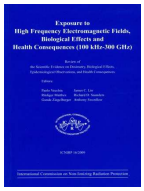
ICNIRP 2003



WHO 2007

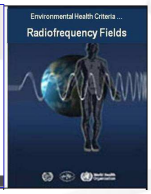
ICNIRP 2010

RF



ICNIRP 2009

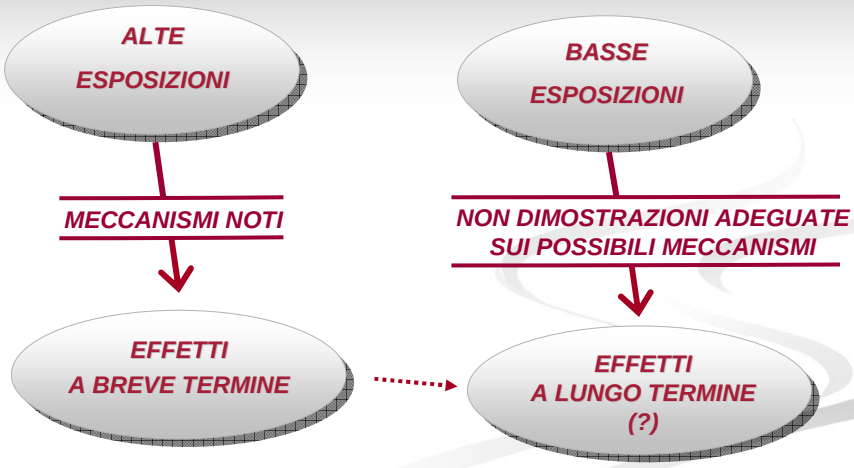
IARC 2013



WHO ?

ICNIRP ?

CAMPI ELETTROMAGNETICI: CLASSIFICAZIONE GENERALE DEGLI EFFETTI



Al momento attuale, i meccanismi "certi" in grado di indurre effetti avversi sui quali esiste accordo sono:

- *induzione magnetica e l'effetto magnetomeccanico per i campi statici*
- *induzione di flussi di ioni per le basse frequenze*
- *deposizione di energia per le radiofrequenze*

Questi meccanismi possono indurre effetti a breve termine diretti e indiretti (es. interferenza, effetto propulsivo, scosse ed alcuni altri)

NB: TUTTI QUESTI EFFETTI SONO INDOTTI SOLO DA ALTE ESPOSIZIONI

(EMF-NET 2008, ICNIRP, 2009; R. Matthes, 2009)

DIRECT SHORT TERM EFFECTS:
LOW FREQUENCIES

MECHANISM: INDUCED CURRENTS (main effect up to 100 kHz)

(THRESHOLDS: induced E-field: V/m, B-field: mT)

- ***PERCEPTION*** *(e.g. alternating electric charge causing body hair to vibrate)*
- ***EFFECTS ON NERVOUS SYSTEM*** *(E.G. MAGNETOPHOSPHENES, VERTIGO, NAUSEA, OTHER SENSORY SYMPTOMS) (< 0.1 V/m)*
- ***PERIPHERAL NERVE AND MUSCLE STIMULATION***
  CONTRACTION (3 V/m, 30 mT)
- ***EFFECTS ON CARDIAC FUNCTION*** *(EXTRASYSTOLES, CARDIAC FIBRILLATION (10-25 V/m; 100 - 250 mT)*

RELAZIONI TRA DENSITA' DI CORRENTE INDOTTA/INDUZIONE MAGNETICA ED EFFETTI BIOLOGICI

Dens. di corrente (mA/m ^{2})	EFFETTI BIOLOGICI	Induzione magnetica (mT)
<1	Assenza di effetti apprezzabili	< 0,5
1-10	Effetti biologici minori	0,5-5
10-100	Magnetofosfeni e possibili effetti sul sistema nervoso	5-50
100-1000	Alterazioni dell'eccitabilità sistema nervoso; rischi sanitari possibili	50-500
> 1000	Extrasistolia e fibrillazione ventricolare; rischi sanitari certi	> 500

da Bernhardt J.H., 1988)

DIRECT SHORT TERM EFFECTS:
HIGH FREQUENCIES

MECHANISM: ENERGY ABSORPTION (thermal effect)

- **not significant <100 KHz**
- **100 kHz-300 MHz: significant absorption, not uniform (head>trunk)**
- **above 10 GHz: absorption primarily at the body surface**

EFFECT: HEATING OF TISSUES (formal threshold > 1°C)

- ***LOADING OF THERMOREGULATION, HEAT EXHAUSTION***
- ***PAIN, BURNS***
- ***EYE*** *cataract*
- ***TESTICLE*** *adverse effects to spermatogenesis, etc.*
- ***OTHER***

NB: **some subjects (e.g. hearth diseased) possibly more susceptible**

Thresholds for thermal RF-EM effects (> 100 kHz)

Effect	Whole body SAR (W/kg)	Local SAR (W/kg)	Notes
Pain		120	3 GHz, 2 min, 10 kW/m^2
Cataract		100	
Heat perception (local exposure)		8	2.45 GHz, 10 s, 630 W/m^2
Heat exhaustion and stroke	> 4		
Loading of thermoregulation	4		
Active reduction of heat load	1.2		
Passive reduction of metabolic heat	0.5–1.5		

INDIRECT SHORT TERM EFFECTS

CONTACT CURRENTS INDUCTION

Near contact of parts of the body with an object at a different electric potential *(not direct contact with power cable); spark discharge*

- ***UP TO 100 kHz about:*** *perception, pain, painful shock , muscular contraction, burns, severe shock, breathing difficulties, etc.; 10 e 100 Hz: lower threshold;*
- ***Above 100 kHZ:*** *main effect perception, pain*

INTERFERENCE ***(EFFECTS ON MEDICAL DEVICES*** *implanted or worn, e.g. pacemakers, insulin pumps or others),*

LIVELLI DI ESPOSIZIONE AI CAMPI ELETTROMAGNETICI

ALTE ESPOSIZIONI

NORMALMENTE RARE ***(es. in prossimità di apparati per la generazione, trasformazione distribuzione della corrente elettrica, alimentazione di impianti industriali, apparecchiature per riscaldamento/saldatura ad induzione, varie apparecchiature elettromedicali quali la RM, apparati per saldatura, galvanica ecc.)***

COMUNEMENTE RILEVABILI IN CONDIZIONI NORMALI NELLA GRANDE MAGGIORANZA DEGLI AMBIENTI DI LAVORO

BASSE ESPOSIZIONI

EMF EFFECTS (HUMANS)

LONG TERM EFFECTS

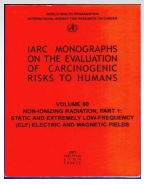
Related to low exposure levels, currently found in usual conditions in most workplaces

No agreement on biological/biophysical mechanism(s)

Scientific evidence of a causal relationship currently considered not conclusive

Principali rassegne sugli effetti dei CEM

ELF



IARC 2002

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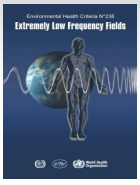

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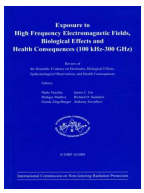

ICNIRP 2009

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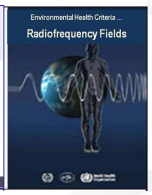


WHO ?

ICNIRP ?

EFHRAN – Report D2, 2012

Classification	*Necessary inclusion criteria*
Sufficient evidence	• when a positive relationship is observed between the exposure and the effect investigated • when the effect is replicated in several studies by independent investigators or under different protocols, and when there is a consistent exposure-response relationship • when confounding factors could be ruled out with reasonable confidence
Limited evidence	• when the evidence of the effect is restricted to a few studies, or when there are unsolved questions regarding the adequacy of the design, conduct or interpretation of the study • when confounding factors could not be ruled out in the studies with reasonable confidence
Inadequate evidence	• when the studies are of insufficient quality, consistency or statistical power to permit a conclusion
Evidence suggesting a lack of effects	• when no effects are reported in several studies by independent investigators under different protocols involving at least two species or two cell types and a sufficient range of field intensities

Outcome	Strength of evidence
Cancer outcomes	
Leukaemia in children	Limited
Brain tumours in children	Inadequate
Brain tumours in adults	Inadequate
Breast cancer in adults	Lack of effect
Other cancer (children or adults)	Inadequate
Neurodegenerative diseases	
Alzheimer's disease	Inadequate
Amyotrophic lateral sclerosis (ALS)	Inadequate
Other neurodegenerative diseases	Inadequate
Reproductive outcomes	
All outcomes	Inadequate
Cardiovascular diseases	
All diseases	Lack of effect
Well-being	
Electrical hypersensitivity (EHS)	Lack of effect
Symptoms	Inadequate

Table 2. The strength of evidence for any health outcome being associated with exposure to low frequency magnetic fields as suggested by EMF-NET (2009) and SCENIHR (2009a) and modified by the results of more recent research.

EFHRAN 2012

Outcome	Strength of evidence
Cancer outcomes	
Leukaemia in children	Inadequate
Brain tumours in children	Inadequate
Brain tumours in adults	Limited
Breast cancer in adults	Inadequate
Other cancer (children or adults)	Inadequate
Neurodegenerative diseases	
Alzheimer's disease	Inadequate
Amyotrophic lateral sclerosis (ALS)	Inadequate
Other neurodegenerative diseases	Inadequate
Reproductive outcomes	
All outcomes	Inadequate
Cardiovascular diseases	
All diseases	Inadequate
Well-being	
Electrical hypersensitivity (EHS)	Lack of effect
Symptoms	Inadequate

Table 4. The strength of evidence for any health outcome being associated with exposure to RF fields as suggested by EMF-NET (2009) and SCENIHR (2009a) and modified by the results of more recent research.

EFHRAN 2012

EFFETTI DA BASSE ESPOSIZIONI (EFFETTI A LUNGO TERMINE)

- LA INSUFFICIENTE CONCLUSIVITA'/MANCANZA DI ACCORDO DERIVA DA UNA INSUFFICIENTE BASE DI DATI SCIENTIFICI SUGLI EFFETTI AVVERSI?

www.emf-portal.de

Database of publications:

May 2013: 17.315
May 2015: 21.231
May 2017: 24.812

British Journal of Cancer (2000) **83**(5), 692–698
© 2000 Cancer Research Campaign
doi: 10.1054/ bjoc.2000.1376, available online at http://www.idealibrary.com on IDEAL®

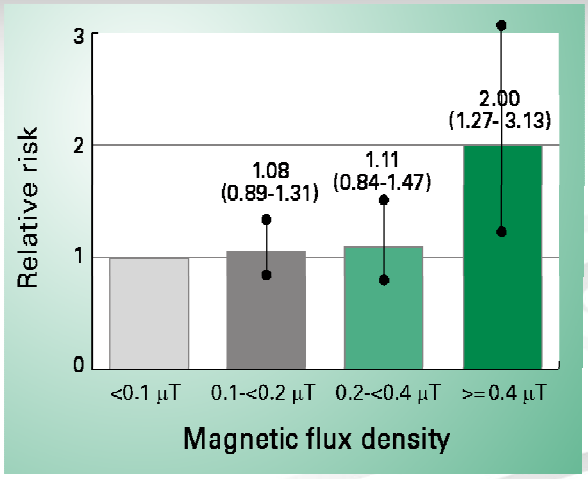
A pooled analysis of magnetic fields and childhood leukaemia

A Ahlbom[1], N Day[2], M Feychting[1], E Roman[3], J Skinner[2], J Dockerty[4], M Linet[5], M McBride[6], J Michaelis[7], JH Olsen[8], T Tynes[9] and PK Verkasalo[10,11,12]

[1]Division of Epidemiology, National Institute of Environmental Medicine, Karolinska Institute, Sweden; [2]Strangeways Research Laboratory, University of Cambridge, UK; [3]Leukaemia Research Fund Centre for Clinical Epidemiology, University of Leeds, UK; [4]Childhood Cancer Research Group, University of Oxford, UK; [5]Division of Cancer Epidemiology and Genetics, National Cancer Institute, USA; [6]Cancer Control Research Programme, British Columbia Cancer Agency, Canada; [7]Institute of Medical Statistics and Documentation, University of Mainz, Germany; [8]Institute of Cancer Epidemiology, Danish Cancer Society, Denmark; [9]Institute of Epidemiological Cancer Research, Norway; [10]Department of Public Health, University of Helsinki, Finland; [11]Finnish Cancer Registry; [12]Department of Public Health, University of Turku, Finland

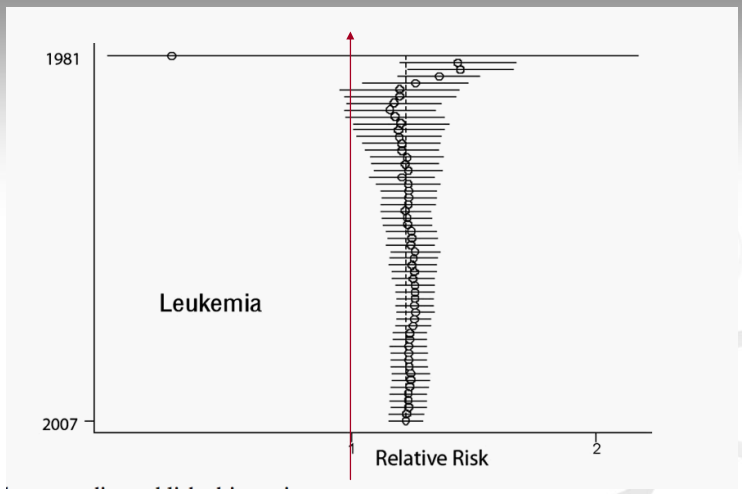
Summary Previous studies have suggested an association between exposure to 50–60 Hz magnetic fields (EMF) and childhood leukaemia. We conducted a pooled analysis based on individual records from nine studies, including the most recent ones. Studies with 24/48-hour magnetic field measurements or calculated magnetic fields were included. We specified which data analyses we planned to do and how to do them before we commenced the work. The use of individual records allowed us to use the same exposure definitions, and the large numbers of subjects enabled more precise estimation of risks at high exposure levels. For the 3203 children with leukaemia and 10 338 control children with estimated residential magnetic field exposures levels < 0.4 μT, we observed risk estimates near the no effect level, while for the 44 children with leukaemia and 62 control children with estimated residential magnetic field exposures ≥ 0.4 μT the estimated summary relative risk was 2.00 (1.27–3.13), *P* value = 0.002). Adjustment for potential confounding variables did not appreciably change the results. For North American subjects whose residences were in the highest wire code category, the estimated summary relative risk was 1.24 (0.82–1.87). Thus, we found no evidence in the combined data for the existence of the so-called wire-code paradox. In summary, the 99.2% of children residing in homes with exposure levels < 0.4 μT had estimates compatible with no increased risk, while the 0.8% of children with exposures ≥ 0.4 μT had a relative risk estimate of approximately 2, which is unlikely to be due to random variability. The explanation for the elevated risk is unknown, but selection bias may have accounted for some of the increase. © 2000 Cancer Research Campaign

Increase of childhood leukemia near power lines



Ahlbom et al. 2000

ELF: OCCUPATIONAL EXPOSURE



Pooled risk estimate of past and new studies published in a given year
n.56 studies included (1981-2007)
Combined RR high exposure : 1.16 (IC 95% : 1.11-1-22
(Kheifets et al. 2008)

IARC (2002):

ELF magnetic fields: possibly carcinogenic (2B)

ELF electric fields: not classifiable (3)

Overall, extremely low frequency magnetic fields were evaluted as possibly carcinogenic to humans (2B), based on the statistical association of higher level residential ELF magnetic field and increased risk for childhood leukemia

International Agency for Research on Cancer:

Programma "IARC Monographs on the Evaluation of Carcinogenic Risks to Humans", avviato nel 1969, finalizzato a identificare le cause di cancro nell'uomo

Scopo:
preparare, tramite dei gruppi di lavoro formati da esperti, delle rassegne critiche e delle valutazioni delle evidenze di cancerogenicità di un ampio spettro di esposizioni umane da pubblicare sotto forma di Monografie.

Evidenze considerate

- **Dati di esposizione**
- **Studi sulla cancerogenicità nell'uomo**
- **Studi sulla cancerogenicità negli animali**
- **Dati sui meccanismi e altri dati rilevanti**

Valutazione delle evidenze

(a) evidenza di cancerogenicità nell'uomo

- sufficiente
- limitata
- inadeguata
- evidenza che suggerisce l'assenza di cancerogenicità

(b) evidenza di cancerogenicità negli animali

- sufficiente
- limitata
- inadeguata
- evidenza che suggerisce l'assenza di cancerogenicità

Sulla base delle valutazioni

- sulle evidenze di cancerogenicità dagli studi sull'uomo e dagli studi sugli animali
- considerando altri dati rilevanti, come quelli sui meccanismi

si giunge ad una valutazione complessiva (5 gruppi di classificazione IARC).

Classificazione IARC (voll.1-119, june 2017)

1	L'agente è cancerogeno per l'uomo (120)
2A	L'agente è probabilmente cancerogeno per l'uomo (81)
2B	L'agente è possibilmente cancerogeno per l'uomo (299)
3	L'agente non è classificabile in relazione alla sua cancerogenicità nell'uomo (502)
4	L'agente è probabilmente non cancerogeno per l'uomo (1)

Group 1: The agent is carcinogenic to humans.

***This category is used when there is sufficient evidence of carcinogenicity in humans**. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than sufficient but there is sufficient evidence of carcinogenicity in experimental animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.*

Group 2A: The agent is probably carcinogenic to humans.

This category is used when there is **limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals**. In some cases, an agent may be classified in this category when there is **inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans**. **Exceptionally, an agent may be classified in this category solely on the basis of limited evidence of carcinogenicity in humans.** An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

Group 2B: The agent is possibly carcinogenic to humans.

This category is used for agents for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals. It may also be used when there is inadequate evidence of carcinogenicity in humans but there is sufficient evidence of carcinogenicity in experimental animals. In some instances, an agent for which there is inadequate evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Group 2A: The agent is probably carcinogenic to humans.

This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. In some cases, an agent may be classified in this category when there is inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans. Exceptionally, an agent may be classified in this category solely on the basis of limited evidence of carcinogenicity in humans. An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

Group 2B: The agent is possibly carcinogenic to humans.

This category is used for agents for which there **is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals**. It may also be used when there is inadequate evidence of carcinogenicity in humans but there is sufficient evidence of carcinogenicity in experimental animals. In some instances, an agent for which there is inadequate evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Gruppo 2A:
agenti probabilmente cancerogeni per l'uomo

Questa categoria è utilizzata quando c'è limitata evidenza di cancerogenicità nell'uomo e sufficiente evidenza di cancerogenicità negli animali da esperimento. Alcune eccezioni

Gruppo 2B: agenti possibilmente cancerogeni per l'uomo

Questa categoria è utilizzata per agenti per i quali vi è limitata evidenza di cancerogenicità nell'uomo e meno che sufficiente evidenza di cancerogenicità negli animali da esperimento. Alcune eccezioni

Group 3: **The agent is *not classifiable as to its carcinogenicity to humans.***

This category is used most commonly for agents for which the evidence of carcinogenicity is *inadequate* in humans and *inadequate* or *limited* in experimental animals.

Exceptionally, agents for which the evidence of carcinogenicity is *inadequate* in humans but *sufficient* in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans.

Agents that do not fall into any other group are also placed in this category.

An evaluation in Group 3 is not a determination of non-carcinogenicity or overall safety. It often means that further research is needed, especially when exposures are widespread or the cancer data are consistent with differing interpretations.

Group 4: **The agent is *probably not carcinogenic to humans.***

This category is used for agents for which there is *evidence suggesting lack of carcinogenicity* in humans and in experimental animals. In some instances, agents for which there is *inadequate evidence of carcinogenicity* in humans but *evidence suggesting lack of carcinogenicity* in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data, may be classified in this group.

EMF: ELF

Main cancer studied: leukemia, brain tumors, breast cancer (both male and female), other;

IARC (2002):

ELF magnetic fields: possibly carcinogenic (2B)

ELF electric fields: not classifiable (3)

Overall, extremely low frequency magnetic fields were evaluted as possibly carcinogenic to humans (2B), based on the statistical association of higher level residential ELF magnetic field and increased risk for childhood leukemia (Ahlbom et al 2000, Greenland et al 2000)

Studi su animali da esperimento:
non consistente effetto cancerogeno o co-cancerogeno delle esposizioni ai campi magnetici ELF : evidenza inadeguata

Varie ipotesi avanzate per spiegare possibili effetti cancerogeni dei campi magnetici ELF, ma non individuata alcuna spiegazione scientifica attendibile su cui esista un accordo.

I campi magnetici ELF sono stati classificati dall'International Agency for Research on Cancer come possibilmente cancerogeni (Gruppo 2B).

A causa dell'insufficienza dei dati, i campi magnetici statici e i campi elettrici statici ed ELF non possono essere classificati in relazione alla loro cancerogenicità (Gruppo 3).

Progress in Biophysics and Molecular Biology 107 (2011) 339–342

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Progress in Biophysics and Molecular Biology

journal homepage: www.elsevier.com/locate/pbiomolbio

Review

Exposure to extremely low-frequency magnetic fields and the risk of childhood cancer: Update of the epidemiological evidence

Joachim Schüz*

A B S T R A C T

There is an ongoing scientific controversy whether the observed association between exposure to residential extremely low-frequency magnetic fields (ELF-MF) and the risk of childhood leukaemia observed in epidemiological studies is causal or due to methodological shortcomings of those studies. Recent pooled analysis confirm results from previous studies, namely an approximately two-fold risk increase at ELF-MF exposures ≥0.4 μT, and demonstrate consistency of studies across countries, with different design, different methods of exposure assessment, and different systems of power transmission and distribution. On the other hand, recent pooled analyses for childhood brain tumour show little evidence for an association with ELF-MF, also at exposures ≥0.4 μT. Overall, the assessment that ELF-MF are a possible carcinogen and may cause childhood leukaemia remains valid. Ongoing research activities, mainly experimental and few new epidemiological studies, hopefully provide additional insight to bring clarity to a research area that has remained inconclusive.

European Commission

Scientific Committee on Emerging and Newly Identified Health Risks

SCENIHR

Opinion on

Potential health effects of exposure to electromagnetic fields (EMF)

SCENIHR adopted this Opinion at the 9th plenary meeting on 27 January 2015

The new epidemiological studies are consistent with earlier findings of an increased risk of childhood leukaemia with estimated daily average exposures above 0.3 to 0.4 μT. As stated in the previous Opinions, no mechanisms have been identified and no support is existing from experimental studies that could explain these findings, which, together with shortcomings of the epidemiological studies prevent a causal interpretation.

Bioelectromagnetics 37:183–189 (2016)

Comment

Extremely Low-Frequency Magnetic Fields and Risk of Childhood Leukemia: A Risk Assessment by the ARIMMORA Consortium

In conclusion, the ARIMMORA risk assessment considers evidence on ELF-MF and childhood leukemia as being consistent with the classification of possibly carcinogenic to humans (IARC Group 2B). The continuing existence of major scientific uncertainty since 2001 is a dissatisfactory situation in terms of public health and prevention [Maslanyj et al., 2010] as well as for science, given the large number of studies and the large bulk of additional scientific data collected over the last decades. It

Associazione tra esposizione ad ELF e leucemia infantile: possibili spiegazioni alternative per le associazioni epidemiologiche osservate

Difficoltà interpretative

- Scarsa evidenza di relazione dose-risposta
- Inadeguato/carente supporto sperimentale
- Inadeguato/carente conoscenza su meccanismo(i) d'azione

Spiegazioni non-causali

- Casualità?
- Misclassificazione esposizione?
- Fattori di confondimento ignoti/non controllati?
- Bias di selezione (controlli)?
- Insieme di distorsioni di tipo differente?

Da: Lagorio S, modificata

European Health Risk Assessment Network on Electromagnetic Fields Exposure

Risk analysis of human exposure to electromagnetic fields

Deliverable Report D2 of EHFRAN project

Actual date of submission July 2010, last revision 2012

Draft prepared by Zenon Sienkiewicz, Joachim Schüz, Aslak Harbo Poulsen, Elizabeth Cardis

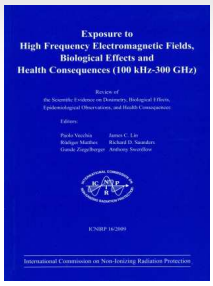
EFHRAN D2 - MF & leucemia infantile

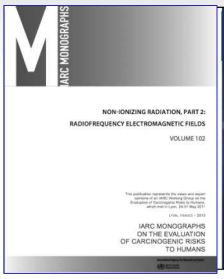
Non esistono spiegazioni meccanicistiche, e nessuna delle ipotesi proposte ha ricevuto un supporto convincente dai dati di letteratura.

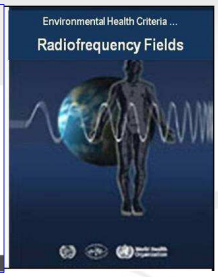
Nel complesso, gli studi sperimentali non forniscono prove che i campi magnetici a bassa frequenza siano cancerogeni.

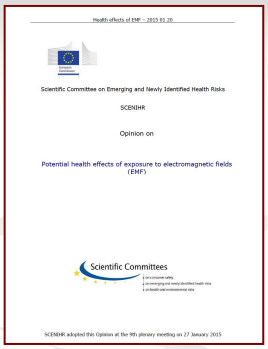
Una combinazione di probabilità, distorsioni e fattori confondenti potrebbe aver prodotto una associazione spuria negli studi epidemiologici

RADIOFREQUENCY (RF) FIELDS

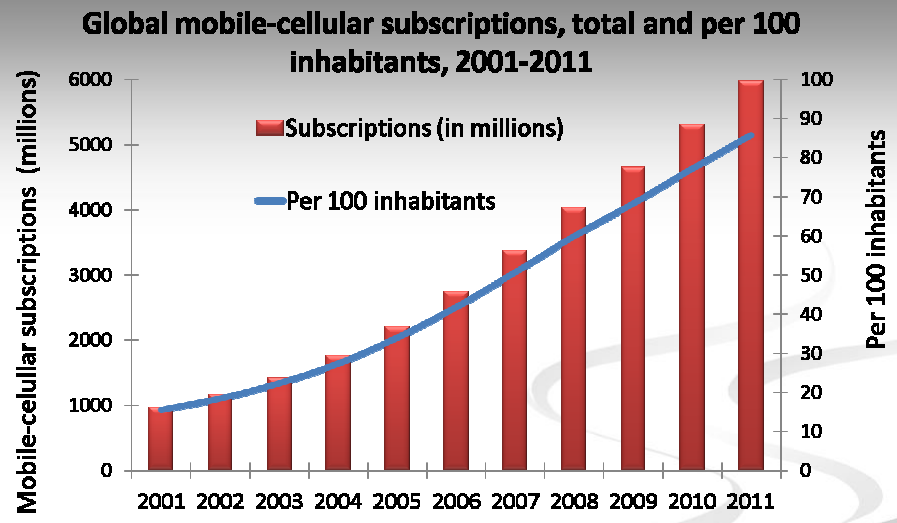








ICNIRP 2009 *IARC 2013* *WHO ?*



Source: ITU World Telecommunication /ICT Indicators database

Courtesy: S Lagorio

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International Journal of Epidemiology 2010;**39**:675–694
doi:10.1093/ije/dyq079

THEME: CANCER

Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case–control study

The INTERPHONE Study Group*

Corresponding author. Elisabeth Cardis; CREAL, Doctor Aiguader 88, 08003 Barcelona, Spain. E-mail: ecardis@creal.cat
*List of members of this study group is available in the Appendix.

Background The rapid increase in mobile telephone use has generated concern about possible health risks related to radiofrequency electromagnetic fields from this technology.

Methods An interview-based case–control study with 2708 glioma and 2409 meningioma cases and matched controls was conducted in 13 countries using a common protocol.

Results A reduced odds ratio (OR) related to ever having been a regular mobile phone user was seen for glioma [OR 0.81; 95% confidence interval (CI) 0.70–0.94] and meningioma (OR 0.79; 95% CI 0.68–0.91), possibly reflecting participation bias or other methodological limitations. No elevated OR was observed ⩾10 years after first phone use (glioma: OR 0.98; 95% CI 0.76–1.26; meningioma: OR 0.83; 95% CI 0.61–1.14). ORs were <1.0 for all deciles of lifetime number of phone calls and nine deciles of cumulative call time. In the 10th decile of recalled cumulative call time, ⩾1640 h, the OR was 1.40 (95% CI 1.03–1.89) for glioma, and 1.15 (95% CI 0.81–1.62) for meningioma; but there are implausible values of reported use in this group. ORs for glioma tended to be greater in the temporal lobe than in other lobes of the brain, but the CIs around the lobe-specific estimates were wide. ORs for glioma tended to be greater in subjects who reported usual phone use on the same side of the head as their tumour than on the opposite side.

Conclusions Overall, no increase in risk of glioma or meningioma was observed with use of mobile phones. There were suggestions of an increased risk of glioma at the highest exposure levels, but biases and error prevent a causal interpretation. The possible effects of long-term heavy use of mobile phones require further investigation.

Published by Oxford University Press on behalf of the International Epidemiological Association
© The Author 2010; all rights reserved. Advance Access publication 17 May 2010
International Journal of Epidemiology 2010;**39**:675–694
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THEME: CANCER

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*List of members of this study group is available in the Appendix.

KEY MESSAGE

- INTERPHONE is the largest case–control study of mobile phone use and brain tumours yet and includes the largest numbers of users with at least 10 years of exposure. A reduced OR for glioma and meningioma related to ever having been a regular mobile phone user possibly reflects participation bias or other methodological limitations. No elevated OR for glioma or meningioma was observed ⩾10 years after first phone use. There were suggestions of an increased risk of glioma, and much less so meningioma, in the highest decile of cumulative call time, in subjects who reported usual phone use on the same side of the head as their tumour and, for glioma, for tumours in the temporal lobe. Biases and errors limit the strength of the conclusions that can be drawn from these analyses and prevent a causal interpretation.

Evidence in humans

Baan et al. Lancet Oncology, June 22 2011

- Exposure index in the cohort study could have resulted in considerable exposure misclassification
- Three early case-control studies encompassed a period of low MPh use, users had low cumulative exposures, time since first use was short, effect measures were imprecise
  ⇨ less informative
- In **Interphone** and the **Hardell's studies** increased risk of **glioma** among heaviest and longer-term users were observed
- Although both studies are susceptible to bias (recall error and selection for participation) the WG concluded that the findings could not be dismissed and that a causal interpretation is possible
- Similar conclusion drawn from these two studies for **acoustic neuroma** (but smaller number of cases)

IARC – Classificazione delle RF

Baan et al. Lancet Oncology, June 22 2011

RF classificate come "possibilmente cancerogene per l'uomo" (gruppo 2B), sulla base di una limitata evidenza epidemiologica di associazione tra uso di telefoni cellulari e rischio di glioma, limitate evidenze sperimentali negli animali ,e deboli indicazioni di un meccanismo patogenetico.

Alcuni membri dei gruppi di lavoro hanno considerata inadeguata l'evidenza scientifica nell'uomo.

I dati relativi ad altre sorgenti di RF, e all'induzione di altri tumori sono stati considerati inadeguati.

6.1 Cancer in Humans

There is limited evidence in humans for the carcinogenicity of radiofrequency radiation. Positive associations have been observed between exposure to radiofrequency radiation from wireless phones and glioma, and acoustic neuroma.

6.2 Cancer in Experimental Animals

There is limited evidence in experimental animals for the carcinogenicity of radiofrequency radiation

6.3 Overall Evaluation

Radiofrequency electromagnetic fields are possibly carcinogenic to humans (Group 2B).

IARC Monograph 102, 2013

Valutazioni IARC relative alle NIR

1992 Radiazione solare e ultravioletta (vol. 55, 1992): **Gruppo 1**

2001 Campi elettrici e magnetici statici e ELF (vol. 80, 2002): **Gruppo 2B**

2009 Radiazione solare e ultravioletta (vol. 100D, “Radiazioni”, 2012): **Gruppo 1**

2011 Campi elettromagnetici a radiofrequenza (vol. 102, 2013): **Gruppo 2B**

Bioelectromagnetics 32:334–339 (2011)

Time Trends (1998–2007) in Brain Cancer Incidence Rates in Relation to Mobile Phone Use in England

Frank de Vocht,[1]* Igor Burstyn,[2] and John W. Cherrie[3]

[1]*Centre for Occupational and Environmental Health, School of Community Based Medicine, Manchester Academic Health Sciences Centre, The University of Manchester, Manchester, UK*
[2]*Department of Environmental and Occupational Health, School of Public Health, Drexel University, Philadelphia, Pennsylvania,*
[3]*Institute of Occupational Medicine, Edinburgh, UK*

Mobile phone use in the United Kingdom and other countries has risen steeply since the early 1990's when the first digital mobile phones were introduced. There is an ongoing controversy about whether radio frequency (RF) exposure from mobile phones increases the risk of brain cancer. However, given the widespread use and nearly two decades elapsing since mobile phones were introduced, an association should have produced a noticeable increase in the incidence of brain cancer by now. Trends in rates of newly diagnosed brain cancer cases in England between 1998 and 2007 were examined. There were no time trends in overall incidence of brain cancers for either gender, or any specific age group. Systematic increases in rates for cancers of the temporal lobe in men (0.04 new cases/year) and women (0.02/year) were observed, along with decreases in the rates of cancers of the parietal lobe (−0.03/year), cerebrum (−0.02/year) and cerebellum (−0.01/year) in men only. The increased use of mobile phones between 1985 and 2003 has not led to a noticeable change in the incidence of brain cancer in England between 1998 and 2007. The observed increase in the rate of cancers in the temporal lobe, if caused by mobile phone use, would constitute <1 additional case per 100,000 people in that period. These data do not indicate a pressing need to implement a precautionary principle by means of population-wide interventions to reduce RF exposure from mobile phones. Bioelectromagnetics 32:334–339, 2011.

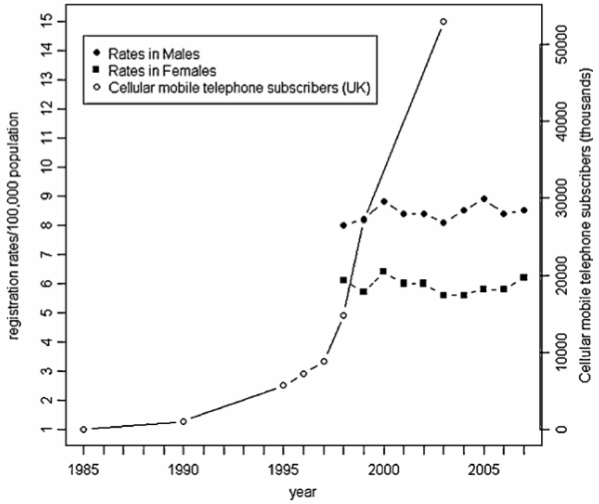
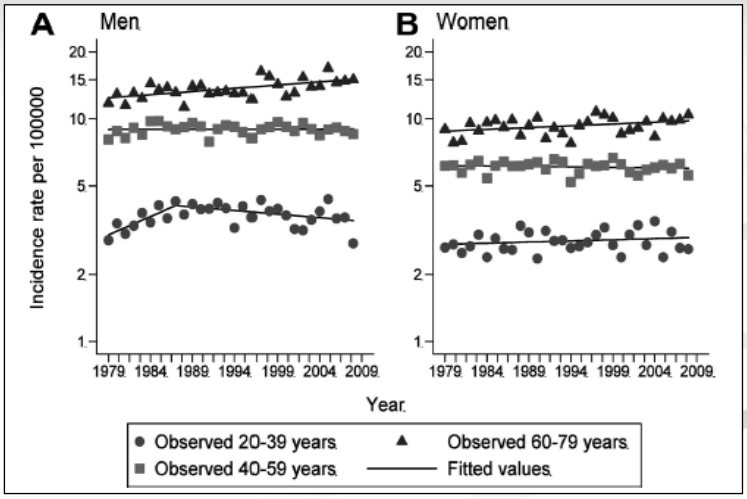


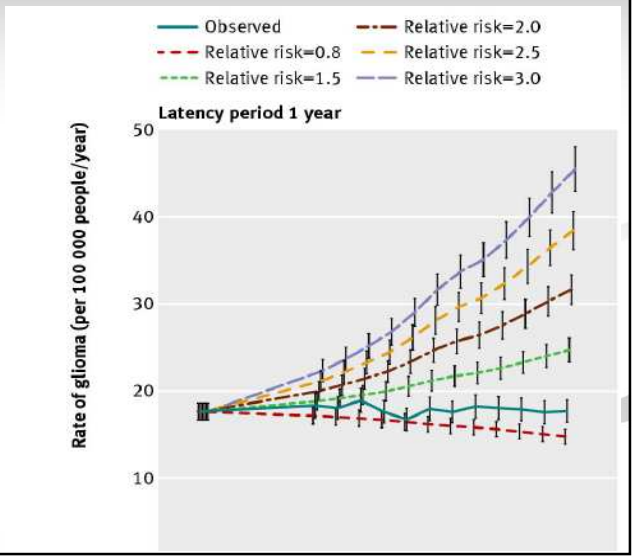
Fig. 1. Brain cancer incidence rates between 1998 and 2007 per 100,000 people in England and number of mobile phone subscribers in the UK between 1985 and 2003 [ITU, 2010].

Incidence of adult glioma in Nordic Countries from 1979 to 2008

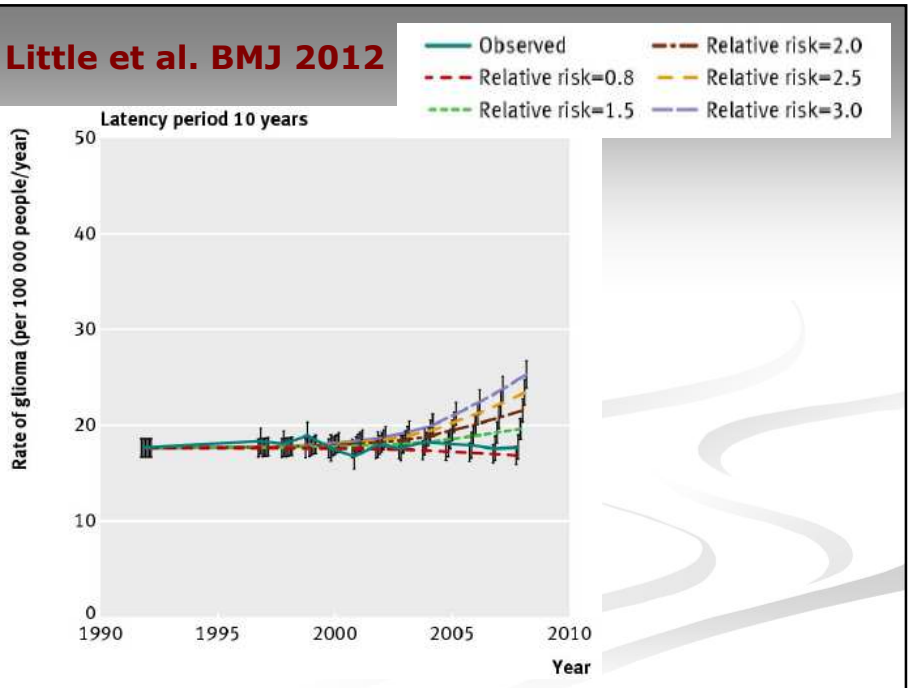
[Deltour et al. *Epidemiology* 2012]



Incidenza di glioma nella popolazione adulta USA 1997 – 2008) (Little et al. BMJ 2012]



Little et al. BMJ 2012



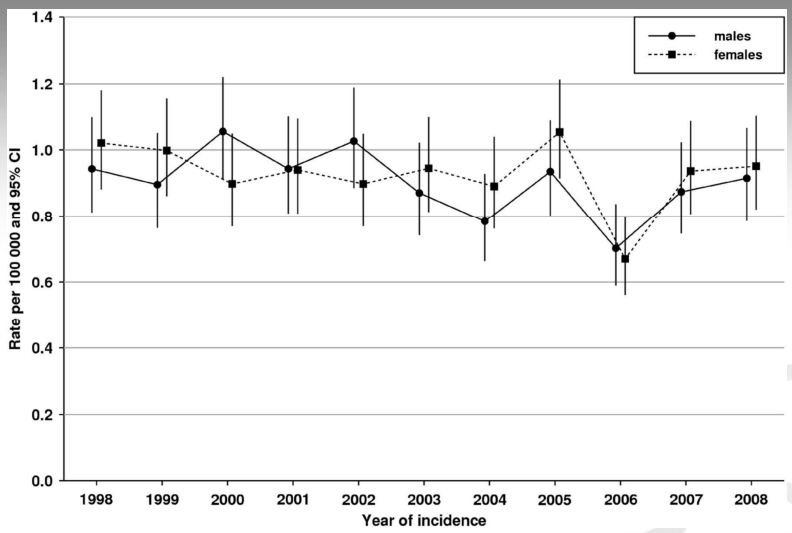
Benson V et al.
Mobile phone use and risk of brain neoplasms and other cancers: prospective study
Int J Epidemiol. 2013;42(3):792-802. doi:10.1093/ije/dyt072
.

Abstract
Background
Results from some retrospective studies suggest a possible increased risk of glioma and acoustic neuroma in users of mobile phones.

Methods
The relation between mobile phone use and incidence of intracranial central nervous system (CNS) tumours and other cancers was examined in 791 710 middle-aged women in a UK prospective cohort, the Million Women Study. Cox regression models were used to estimate adjusted relative risks (RRs) and 95% confidence intervals (CIs). Women reported mobile phone use in 1999 to 2005 and again in 2009.

Results
During 7 years' follow-up, 51 680 incident invasive cancers and 1 261 incident intracranial CNS tumours occurred. Risk among ever vs never users of mobile phones was not increased for all intracranial CNS tumours (RR = 1.01, 95% CI = 0.90–1.14, P = 0.82), for specified CNS tumour types nor for cancer at 18 other specified sites. For long-term users compared with never users, there was no appreciable association for glioma (10+ years: RR = 0.78, 95% CI = 0.55–1.10, P = 0.16) or meningioma (10+ years: RR = 1.10, 95% CI = 0.66–1.84, P = 0.71). For acoustic neuroma, there was an increase in risk with long term use vs never use (10+ years: RR = 2.46, 95% CI = 1.07–5.64, P = 0.03), the risk increasing with duration of use (trend among users, P = 0.03).

Conclusions
In this large prospective study, mobile phone use was not associated with increased incidence of glioma, meningioma or non-CNS cancers.



From: Mobile phone use and risk of brain neoplasms and other cancers: prospective study
Int J Epidemiol. 2013;42(3):792-802. doi:10.1093/ije/dyt072

Published by Oxford University Press on behalf of the International Epidemiological Association
International Journal of Epidemiology 2014;**43**:275

Authors' response to: The case of acoustic neuroma: comment on mobile phone use and risk of brain neoplasms and other cancers

From VICTORIA S BENSON,[1]* KIRSTIN PIRIE,[1] JOACHIM SCHÜZ,[2] GILLIAN K REEVES,[1] VALERIE BERAL[1] and JANE GREEN[1]

[1]Cancer Epidemiology Unit, University of Oxford, Richard Doll Building, Roosevelt Drive, Oxford, OX3 7LF, UK and [2]International Agency for Research on Cancer (IARC), Section of Environment and Radiation, 150 Cours Albert Thomas, 69372 Lyon Cedex 08, France

NB: Analyses repeated with extended data, updated 2011

Table 1 Adjusted relative risks[a] (95% confidence intervals) for all intracranial central nervous system (CNS) tumours and by tumour type, in users vs never users of mobile phones, overall and by duration of use

	All CNS tumours (n = 1727)	Glioma (n = 875)	Meningioma (n = 397)	Acoustic neuroma (n = 126)
Ever use	0.94 (0.85–1.04)	0.86 (0.75–0.99)	1.01 (0.82–1.25)	1.19 (0.81–1.75)
<5 years use	0.99 (0.83–1.17)	0.96 (0.75–1.23)	0.90 (0.63–1.28)	0.94 (0.53–1.66)
5-9 years use	0.93 (0.82–1.06)	0.86 (0.72–1.02)	1.04 (0.80–1.34)	1.46 (0.94–2.27)
10+ years use	0.90 (0.77–1.05)	0.77 (0.62–0.96)	1.08 (0.78–1.49)	1.17 (0.60–2.27)

[a]Relative risks are stratified by socioeconomic status, region and age at baseline, and adjusted for height, body mass index, smoking status, alcohol intake, strenuous exercise and use of menopausal hormonal therapy.

G Model
CANEP 1003 No. of Pages 7
ARTICLE IN PRESS
Cancer Epidemiology xxx (2016) xxx–xxx
Contents lists available at ScienceDirect
Cancer Epidemiology
The International Journal of Cancer Epidemiology, Detection, and Prevention
journal homepage: www.cancerepidemiology.net

Has the incidence of brain cancer risen in Australia since the introduction of mobile phones 29 years ago?

Simon Chapman[a,*], Lamiae Azizi[a], Qingwei Luo[a,b], Freddy Sitas[a,c]

[a] School of Public Health, University of Sydney, Australia
[b] Cancer Council NSW, Sydney, Australia
[c] School of Public Health and Community Medicine, University of New South Wales, Australia

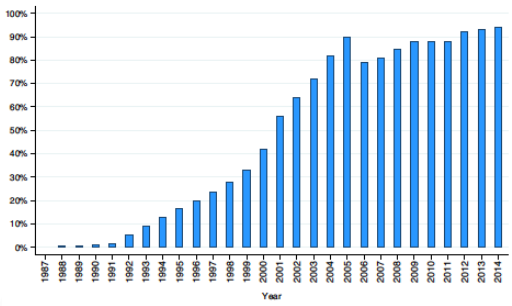
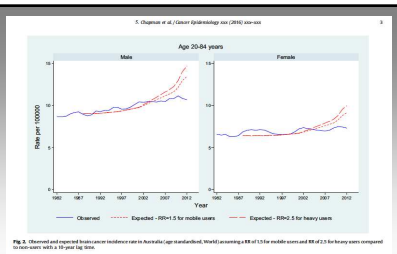
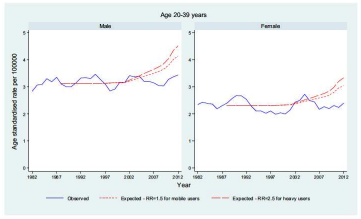
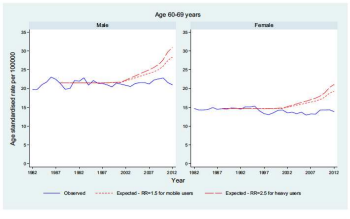
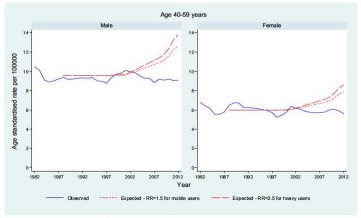


Fig. 1. Percentage of Australians with mobile phone accounts.









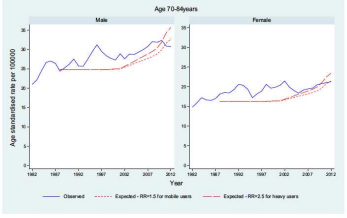


Fig. 3. Observed and modelled expected estimates of brain cancer incidence in Australia, in four age groups, assuming a RR = 1.5 for mobile users and RR = 2.5 for heavy users compared to non-users with a 10-year lag time.

Fig. 3. (Continued)

ABSTRACT

Background: Mobile phone use in Australia has increased rapidly since its introduction in 1987 with whole population usage being 94% by 2014. We explored the popularly hypothesised association between brain cancer incidence and mobile phone use.

Study methods: Using national cancer registration data, we examined age and gender specific incidence rates of 19,858 male and 14,222 females diagnosed with brain cancer in Australia between 1982 and 2012, and mobile phone usage data from 1987 to 2012. We modelled expected age specific rates (20–39, 40–59, 60–69, 70–84 years), based on published reports of relative risks (RR) of 1.5 in ever-users of mobile phones, and RR of 2.5 in a proportion of 'heavy users' (19% of all users), assuming a 10-year lag period between use and incidence.

Summary answers: Age adjusted brain cancer incidence rates (20–84 years, per 100,000) have risen slightly in males ($p < 0.05$) but were stable over 30 years in females ($p > 0.05$) and are higher in males 8.7 (CI = 8.1–9.3) than in females, 5.8 (CI = 5.3–6.3). Assuming a causal RR of 1.5 and 10-year lag period, the expected incidence rate in males in 2012 would be 11.7 (11–12.4) and in females 7.7 (CI = 7.2–8.3), both $p < 0.01$; 1434 cases observed in 2012, vs. 1867 expected. Significant increases in brain cancer incidence were observed (in keeping with modelled rates) only in those aged ≥70 years (both sexes), but the increase in incidence in this age group began from 1982, before the introduction of mobile phones. Modelled expected incidence rates were higher in all age groups in comparison to what was observed. Assuming a causal RR of 2.5 among 'heavy users' gave 2038 expected cases in all age groups.

Limitations: This is an ecological trends analysis, with no data on individual mobile phone use and outcome.

What this study adds: The observed stability of brain cancer incidence in Australia between 1982 and 2012 in all age groups except in those over 70 years compared to increasing modelled expected estimates, suggests that the observed increases in brain cancer incidence in the older age group are unlikely to be related to mobile phone use. Rather, we hypothesize that the observed increases in brain cancer incidence in Australia are related to the advent of improved diagnostic procedures when computed tomography and related imaging technologies were introduced in the early 1980s.

Chapman et al, Canc Epidemiol, 2016

Trends in incidence of primary brain cancer in New Zealand, 1995 to 2010

Stella J-H Kim,[1] Sally J. Ioannides,[1] J. Mark Elwood[1]

Australian and New Zealand Journal of Public Health 2015 VOL. 39 NO. 2

Abstract

Objective: Case-control studies have linked mobile phone use to an increased risk of glioma in the most exposed brain areas, the temporal and parietal lobes, although inconsistently. We examined time trends in the incidence rates of brain malignancies in New Zealand from 1995 to 2010.

Methods: Data from the New Zealand Cancer Registry was used to calculate incidence rates of primary brain cancer, by age, gender, morphology and anatomical site. Log-linear regression analysis was used to assess trends in the annual incidence of primary brain cancer; annual percentage changes and their 95% confidence intervals were estimated.

Results: No consistent increases in all primary brain cancer, glioma, or temporal or parietal lobe glioma were seen. At ages 10-69, the incidence of all brain cancers declined significantly. Incidence of glioma increased at ages over 70.

Conclusion: In New Zealand, there has been no consistent increase in incidence rates of primary brain cancers. An increase in glioma at ages over 70 is likely to be due to improvements in diagnosis. As with any such studies, a small effect, or one with a latent period of more than 10 to 15 years, cannot be excluded.

Key words: cancer epidemiology, brain cancer, trends, mobile phones

Environment International 97 (2016) 100–107

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journal homepage: www.elsevier.com/locate/envint

Inferring the 1985–2014 impact of mobile phone use on selected brain cancer subtypes using Bayesian structural time series and synthetic controls

Frank de Vocht

A B S T R A C T

Background: Mobile phone use has been increasing rapidly in the past decades and, in parallel, so has the annual incidence of certain types of brain cancers. However, it remains unclear whether this correlation is coincidental or whether use of mobile phones may cause the development, promotion or progression of specific cancers. The 1985–2014 incidence of selected brain cancer subtypes in England were analyzed and compared to counterfactual 'synthetic control' timeseries.

Methods: Annual 1985–2014 incidence of malignant glioma, glioblastoma multiforme, and malignant neoplasms of the temporal and parietal lobes in England were modelled based on population-level covariates using Bayesian structural time series models assuming 5,10 and 15 year minimal latency periods. Post-latency counterfactual 'synthetic England' timeseries were nowcast based on covariate trends. The impact of mobile phone use was inferred from differences between measured and modelled time series.

Results: There is no evidence of an increase in malignant glioma, glioblastoma multiforme, or malignant neoplasms of the parietal lobe not predicted in the 'synthetic England' time series. Malignant neoplasms of the temporal lobe however, have increased faster than expected. A latency period of 10 years reflected the earliest latency period when this was measurable and related to mobile phone penetration rates, and indicated an additional increase of 35% (95% Credible Interval 9%:59%) during 2005–2014; corresponding to an additional 188 (95%CI 48–324) cases annually.

Conclusions: A causal factor, of which mobile phone use (and possibly other wireless equipment) is in agreement with the hypothesized temporal association, is related to an increased risk of developing malignant neoplasms in the temporal lobe.

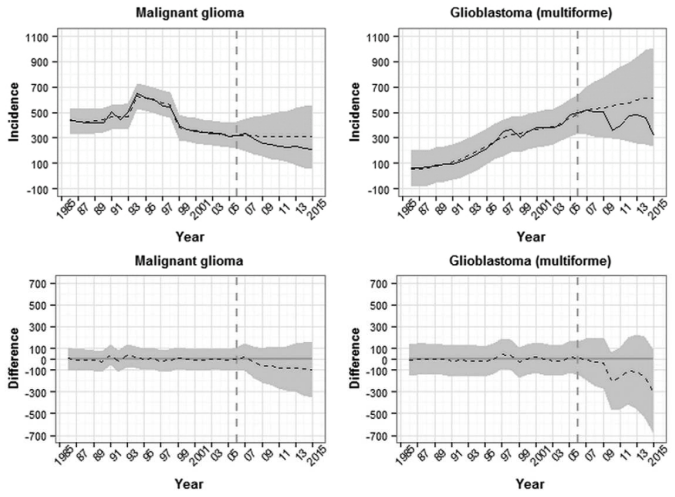

Fig. 2. Measured (solid) and modelled (dashed) incidence trends (top) and pointwise difference (bottom) for histology-based tumours; implied 10-year lag. Grey areas correspond to 95% Credible Intervals.

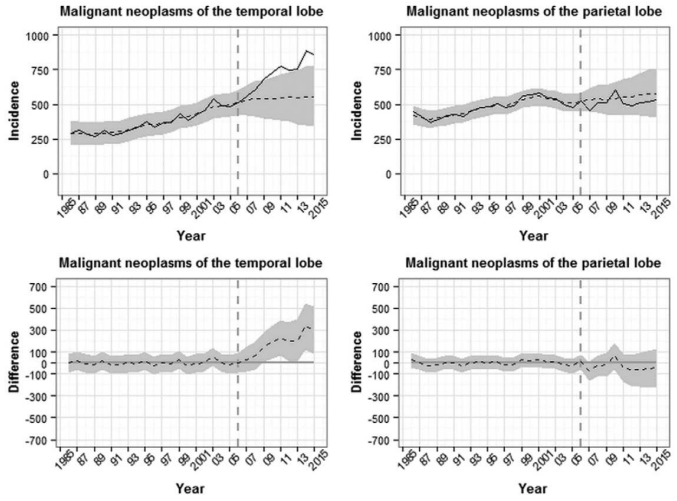

Fig. 3. Measured (solid) and modelled (dashed) incidence trends (top) and pointwise difference (bottom) for location-based brain tumours; implied 10-year lag. Grey areas correspond to 95% Credible Intervals.

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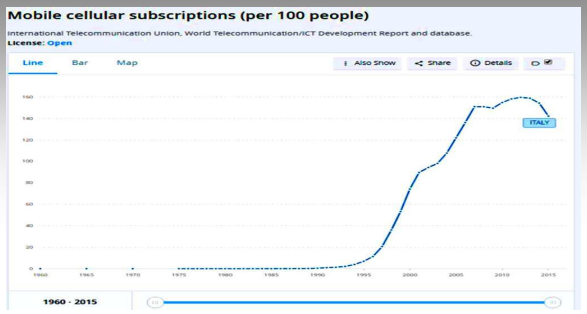
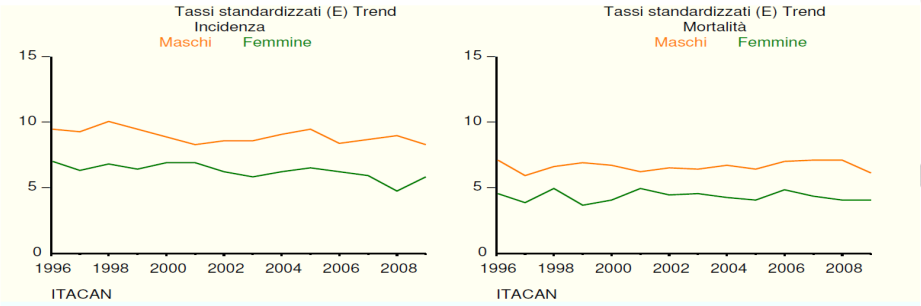
Tumori del Cervello e del Sistema Nervoso Centrale (AIRTUM)





Nei mesi scorsi la stampa ha segnalato (con una certa evidenza) alcune recenti sentenze (finora di primo grado), di riconoscimento di un rapporto di causalità tra l'esposizione professionale a CEM ed alcune forme di tumore, che sono stati riconosciuti come malattia professionale.


Torino

Ivrea, tumore al cervello per uso eccessivo del telefonino: Inail condannata a pagare, "l'uso scorretto provoca il cancro"

Un dipendente Telecom colpito da neurinoma dopo aver utilizzato il cellulare più di tre ore al giorno. Il tribunale riconosce la correlazione e la rendita vitalizia per malattia professionale. La perizia choc: "Si effetto cancerogeno"

di SARAH MARTINENGHI

Si era ammalato di cancro per colpa del telefono cellulare usato per 15 anni per più di tre ore al giorno, e ora l'Inail è stata condannata pagare la rendita perpetua per il danno sul lavoro subito da un dipendente della Telecom. "La sentenza riconosce il legame causale un tumore al cervello e l'uso di un telefono cellulare" spiegano gli avvocati Renato Ambrosio e Stefano Bertone che hanno vinto la Il verdetto è stato emesso dal giudice Luca Fadda del tribunale di Ivrea l'11 aprile.


CORRIERE DELLA SERA

Il Tribunale di Ivrea: «L'uso scorretto del cellulare ha causato un tumore»

ELISA SOLA

Tumore per uso del cellulare, «Quattro ore di cellulare al giorno per...

L'utilizzo prolungato e continuativo del cellulare, usato senza auricolari né vivavoce, è stato riconosciuto dal giudice di Ivrea Laura Fadda come possibile causa dell'insorgenza di un tumore. La vittima, che ricorreva contro l'Inail, è un tecnico della sede di Torino di una grande azienda di telefonia italiana. Si chiama Roberto Romeo, ha 57 anni e gli mancano pochi anni alla pensione. Gli è stato riconosciuto un danno biologico del 23 percento.


EL MUNDO

Un tribunal italiano concede una pensión vitalicia a un trabajador que tuvo un tumor por usar mucho el móvil

Nei mesi scorsi la stampa ha segnalato (con una certa evidenza) la notizia di alcune recenti sentenze, finora di primo grado, di riconoscimento di un rapporto di causalità tra l'esposizione professionale a CEM ed alcune forme di tumore, che sono quindi stati riconosciuti come malattia professionale.

Tali sentenze fanno seguito ad una precedente di alcuni anni fa, confermata in tutti i gradi di giudizio

I media hanno riportato notizie relative a sentenze che si sono pronunciate a favore dell'esistenza di un nesso di causalità. Tuttavia, in realtà, esistono altri procedimenti nei quali la magistratura si è espressa in modo opposto, negando un rapporto di causalità.

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Sia la revisione della IARC che tutte le rassegne di finora pubblicate da parte di autorevoli organismi internazionali, prodotte sulla base di pareri di gruppi multidisciplinari di esperti, non sono andati oltre il concetto di "**possibilità**" di un'associazione tra specifiche esposizioni a tipi specifici di CEM e alcuni specifici tipi di cancro.
In tutte le valutazioni i dati non sono invece stati considerati indicativi di una "**probabilità**".

Su queste basi, almeno da un punto di vista rigorosamente scientifico **non sembra condivisibile l'opinione di ritenere adeguatamente provata su un singolo caso una relazione che non è "probabile" nemmeno su gruppi.**

DECRETO LEGISLATIVO 1 agosto 2016, n. 159

Attuazione della direttiva 2013/35/UE sulle disposizioni minime di sicurezza e di salute relative all'esposizione dei lavoratori ai rischi derivanti dagli agenti fisici (campi elettromagnetici) e che abroga la direttiva 2004/40/CE. (16 G00172)

Pubblicato sulla GU n.192 del 18-8-2016

2 artt. (Art. 1 Modifiche al decreto legislativo 9 aprile 2008, n. 81
e Art. 2 Clausola di invarianza finanziaria)
e 1 allegato

In vigore dal: 2-9-**2016**

Titolo VIII, Capo IV – CAMPI ELETTROMAGNETICI
Dal D.Lgs. 159/2016, recepimento della Direttiva 2013/35/CE
(Art 1), che modifica gli attuali artt. 206-219)

Quali rischi sono presi in considerazione:

1. Il presente capo determina i requisiti minimi per la protezione dei lavoratori contro i rischi per la salute e la sicurezza derivanti dall'esposizione ai campi elettromagnetici (da 0 Hz a 300 GHz), come definiti dall'articolo 207, durante il lavoro. Le disposizioni riguardano la protezione dai rischi per la salute e la sicurezza dei lavoratori dovuti agli effetti biofisici diretti e agli effetti indiretti noti provocati dai campi elettromagnetici. (Art. 206, comma 1, sostituisce il precedente)

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2. I valori limite di esposizione (VLE) stabiliti nel presente capo riguardano soltanto le relazioni scientificamente accertate tra effetti biofisici diretti a breve termine ed esposizione ai campi elettromagnetici (Art. 206, comma 2, sostituisce il precedente).

Fabriziomaria Gobba,
Università di Modena e Reggio Emilia

Quali rischi non riguarda:

3. Il presente capo non riguarda la protezione da eventuali effetti a lungo termine e i rischi risultanti dal contatto con i conduttori in tensione (Art. 206, comma 3, era il precedente comma 2)

Direttiva 2013/35/UE

Preambolo:

(7) This Directive does not address suggested long-term effects of exposure to electromagnetic fields, since there is currently no well-established scientific evidence of a causal relationship

ASSOCIAZIONE ITALIANA PER LA RICERCA SUL CANCRO

Rendiamo il cancro sempre più curabile.

L'uso dei cellulari può causare un tumore al cervello?

No, le prove disponibili non sono sufficienti per affermare che vi sia un nesso, in particolare per quel che riguarda i cellulari di nuova generazione a basse emissioni di onde a radiofrequenza. Un lieve aumento di rischio è stato segnalato da alcuni studi solo per il neurinoma, un tumore benigno del nervo acustico.

Health Council of the Netherlands

Mobile phones and cancer

June 1°, 2016

formulate its conclusions. The Committee feels that it is not possible to state that there is a proven association between long-term and frequent use of a mobile telephone and an increase in the risk of tumours in the brain and head and neck region in humans. Based on the strength of the evidence it can only be concluded that such an association cannot be excluded. The Committee considers it unlikely that exposure to radiofrequency fields, which is associated with the use of mobile telephones, causes cancer. The animal data indicate a possibility of a promoting effect, but it is not clear whether this could explain the increased risk for tumours in the brain, head and neck that has been observed in some epidemiological studies. The Committee feels it more likely that a combination of bias, confounding and chance might be an explanation for the epidemiological observations.

National Cancer Institute (NCI):

"Studies thus far have not shown a consistent link between cell phone use and cancers of the brain, nerves, or other tissues of the head or neck. More research is needed because cell phone technology and how people use cell phones have been changing rapidly

National Institute of Environmental Health Sciences (NIEHS)*(is conducting studies of the possible health effects of cell phones):*

"Current scientific evidence has not conclusively linked cell phone use with any adverse health problems, but more research is need

Centers for Disease Control and Prevention (CDC):

"At this time we do not have the science to link health problems to cell phone use. Scientific studies are underway to determine whether cell phone use may cause health effects."

Federal Communications Commission (FCC):

"There is no scientific evidence that proves that wireless phone usage can lead to cancer or a variety of other problems, including headaches, dizziness or memory loss. However, organizations in the United States and overseas are sponsoring research and investigating claims of possible health effects related to the use of wireless telephones."

Food and Drug Administration (FDA),

(which regulates the safety of radiation-emitting devices such as cell phones in the United States):

"The majority of studies published have failed to show an association between exposure to radiofrequency from a cell phone and health problems."

NHS UK

"most current research suggests it's unlikely that radio waves from mobile phones or base stations increase the risk of any health problems"

PuntoSicuro
https://www.puntosicuro.it/sicurezza-sul-lavoro-C-1/tipologie-di-rischio-C-5/rischi-campi-elettromagnetici-C-39/le-sentenze-riguardanti-il-rischio-da-uso-dei-telefoni-per-lavoro-AR-17111/ Ultimo accesso: 6 sett 17

Posizione ANMIL:
Un approfondimento dell'Avv. Mauro Dalla Chiesa, Consulente Legale ANMIL

L'orientamento giurisprudenziale richiamato evidenzia due azioni immediate: la prima è quella di ***invitare l'INAIL a considerare i tumori collegati all'uso di cellulari e cordless in caso di intensa esposizione lavorativa, quale malattia professionale tabellata,*** nonché, ad intraprendere studi ed indagini epidemiologiche tese ad indagare quali siano gli effettivi limiti non nocivi dell' esposizione alle radiazioni elettromagnetiche dei cellulari e degli impianti wi-fi ad alta potenza presenti sui luoghi di lavoro.

La seconda azione è rivolta, invece, ad ***una valutazione del rischio nei documenti di valutazione aziendale previsti dal decreto legislativo 81/2008 e successive modifiche.***

CONCLUSIONI

ESPOSIZIONE A CAMPI ELETTROMAGNETICI E TUMORI PROFESSIONALI:

Alcuni magistrati, basandosi sulle conclusioni dei propri consulenti, hanno recentemente emesso delle sentenze che non sembrano coerenti con i dati della ricerca, e le conclusioni della comunità scientifica.

Tuttavia:

1) Va evidenziato che **è inappropriato assumere queste sentenze alla stregua di una "dimostrazione" dell'esistenza di un rapporto di causalità accertato, che, come visto, non trova invece supporto nei dati della ricerca e non è coerente con le indicazioni della comunità scientifica.**

CONCLUSIONI

2): E' largamente auspicabile che il ruolo centrale nella -individuazione dei reali rischi per la salute e per la sicurezza di lavoratori,
-delle basi scientifiche di tali rischi,
-delle opzioni per la gestione di tali rischi,
sia riconosciuto alla Ricerca Scientifica (i cui metodi hanno delle basi ormai largamente testate e condivise), attraverso le indicazioni fornite da **autorevoli organi scientifici internazionali e nazionali**

Health effects of solar UV radiation in workers: Effective prevention needed

Joint meeting of two ICOH scientific committees

Two ICOH scientific committees - EOHS together with Radiation and Work - welcome you to join our joint meeting in Helsinki in December.

Programme to be finalised by September 1.

Twitter: @EOHS_ICOH