Dear AEMC,

I am registering my submission against the upcoming decision to roll smart meters out to all NSW homes. My reason for opposing it is because I am severely electrosensitive. Electro hypersensitivity is recognized by the World Health Organization as a functional disability. I have severe fibromyalgia and am on a disability pension due to my inability to work. EMF from wireless devices triggers this fibromyalgia into activation and can leave me in weeks of pain, sometimes needing a carer to come in to help. I live predominantly in isolation from public places and organize my life around avoidance of sources of wireless radiation. I am using a wired desktop computer so I can access online information and accounts and I don't suffer the same degree of symptoms. The home I rent is shielded, creating a faraday cage within it protecting me from the local cell tower. When I visit my children, who all have smart meters on their homes, I am limited to how much time I can spend in their homes, and I have to remediate inflammation after leaving. Installing a smart meter would greatly depreciate my health further so I would need to take any action possible to help protect myself and this may entail maintaining the digital non-smart meter I have now, and doing self-reads if necessary.

I have another friend who lives on the central coast of Sydney. She is so sensitive to electricity due to a poisoning via an MRI dye that is now no longer in use due to damages reported. She is unable to even use lighting and spends most days in pain. She had to have her fridge rewired so the wiring was deployed away from her living areas. She would be life-threateningly disadvantaged if a smart meter installation was mandatory.

I make this submission on my own behalf and on the behalf of many others who suffer from symptoms of EHS (electrohypersensitivity). We may be a small minority in contrast that recognize the effects of wireless frequencies, but I believe alternative measures for people like us would be the most humane in circumstances such as smart meter rollout.

Kind Regards

Debra Martin

Electromagnetic hypersensitivity

Backgrounder

December 2005

As societies industrialize and the technological revolution continues, there has been an unprecedented increase in the number and diversity of electromagnetic field (EMF) sources. These sources include video display units (VDUs) associated with computers, mobile phones and their base stations. While these devices have made our life richer, safer and easier, they have been accompanied by concerns about possible health risks due to their EMF emissions.

For some time a number of individuals have reported a variety of health problems that they relate to exposure to EMF. While some individuals report mild symptoms and react by avoiding the fields as best they can, others are so severely affected that they cease work and change their entire lifestyle. This reputed sensitivity to EMF has been generally termed “electromagnetic hypersensitivity” or EHS.

This fact sheet describes what is known about the condition and provides information for helping people with such symptoms. Information provided is based on a WHO Workshop on Electrical Hypersensitivity (Prague, Czech Republic, 2004), an international conference on EMF and non-specific health symptoms (COST244bis, 1998), a European Commission report (Bergqvist and Vogel, 1997) and recent reviews of the literature.

What is EHS?

EHS is characterized by a variety of non-specific symptoms, which afflicted individuals attribute to exposure to EMF. The symptoms most commonly experienced include dermatological symptoms (redness, tingling, and burning sensations) as well as neurasthenic and vegetative symptoms (fatigue, tiredness, concentration difficulties, dizziness, nausea, heart palpitation, and digestive disturbances). The collection of symptoms is not part of any recognized syndrome.

EHS resembles multiple chemical sensitivities (MCS), another disorder associated with low-level environmental exposures to chemicals. Both EHS and MCS are characterized by a range of non-specific symptoms that lack apparent toxicological or physiological basis or independent verification. A more general term for sensitivity to environmental factors is Idiopathic Environmental Intolerance (IEI), which originated from a workshop convened by the International Program on Chemical Safety (IPCS) of the WHO in 1996 in Berlin. IEI is a descriptor without any implication of chemical etiology, immunological sensitivity or EMF susceptibility. IEI incorporates a number of disorders sharing similar non-specific medically unexplained symptoms that adversely affect people. However since the term EHS is in common usage it will continue to be used here.

Prevalence

There is a very wide range of estimates of the prevalence of EHS in the general population. A survey of occupational medical centres estimated the prevalence of EHS to be a few individuals per million in the population. However, a survey of self-help groups yielded much higher estimates. Approximately 10% of reported cases of EHS were considered severe.

There is also considerable geographical variability in prevalence of EHS and in the reported symptoms. The reported incidence of EHS has been higher in Sweden, Germany, and Denmark, than in the United Kingdom, Austria, and France. VDU-related symptoms were more prevalent in Scandinavian countries, and they were more commonly related to skin disorders than elsewhere in Europe. Symptoms similar to those reported by EHS individuals are common in the general population.

Studies on EHS individuals

A number of studies have been conducted where EHS individuals were exposed to EMF similar to those that they attributed to the cause of their symptoms. The aim was to elicit symptoms under controlled laboratory conditions.

The majority of studies indicate that EHS individuals cannot detect EMF exposure any more accurately than non-EHS individuals. Well controlled and conducted double-blind studies have shown that symptoms were not correlated with EMF exposure.

It has been suggested that symptoms experienced by some EHS individuals might arise from environmental factors unrelated to EMF. Examples may include “flicker” from fluorescent lights, glare and other visual problems with

VDUs, and poor ergonomic design of computer workstations. Other factors that may play a role include poor indoor air quality or stress in the workplace or living environment.

There are also some indications that these symptoms may be due to pre-existing psychiatric conditions as well as stress reactions as a result of worrying about EMF health effects, rather than the EMF exposure itself.

Conclusions

EHS is characterized by a variety of non-specific symptoms that differ from individual to individual. The symptoms are certainly real and can vary widely in their severity. Whatever its cause, EHS can be a disabling problem for the affected individual. EHS has no clear diagnostic criteria and there is no scientific basis to link EHS symptoms to EMF exposure. Further, EHS is not a medical diagnosis, nor is it clear that it represents a single medical problem.

Physicians: Treatment of affected individuals should focus on the health symptoms and the clinical picture, and not on the person's perceived need for reducing or eliminating EMF in the workplace or home. This requires:

- a medical evaluation to identify and treat any specific conditions that may be responsible for the symptoms,
- a psychological evaluation to identify alternative psychiatric/psychological conditions that may be responsible for the symptoms,
- an assessment of the workplace and home for factors that might contribute to the presented symptoms. These could include indoor air pollution, excessive noise, poor lighting (flickering light) or ergonomic factors. A reduction of stress and other improvements in the work situation might be appropriate.

For EHS individuals with long lasting symptoms and severe handicaps, therapy should be directed principally at reducing symptoms and functional handicaps. This should be done in close co-operation with a qualified medical specialist (to address the medical and psychological aspects of the symptoms) and a hygienist (to identify and, if necessary, control factors in the environment that are known to have adverse health effects of relevance to the patient).

Treatment should aim to establish an effective physician-patient relationship, help develop strategies for coping with the situation and encourage patients to return to work and lead a normal social life.

EHS individuals: Apart from treatment by professionals, self help groups can be a valuable resource for the EHS individual.

Governments: Governments should provide appropriately targeted and balanced information about potential health hazards of EMF to EHS individuals, health-care professionals and employers. The information should include a clear statement that no scientific basis currently exists for a connection between EHS and exposure to EMF.

Researchers: Some studies suggest that certain physiological responses of EHS individuals tend to be outside the normal range. In particular, hyper reactivity in the central nervous system and imbalance in the autonomic nervous system need to be followed up in clinical investigations and the results for the individuals taken as input for possible treatment.

What WHO is doing

WHO, through its International EMF Project, is identifying research needs and co-ordinating a world-wide program of EMF studies to allow a better understanding of any health risk associated with EMF exposure. Particular emphasis is placed on possible health consequences of low-level EMF. Information about the EMF Project and EMF effects is provided in a series of fact sheets in several languages.

FURTHER READING

WHO workshop on electromagnetic hypersensitivity (2004), October 25 -27, Prague, Czech Republic,

COST244bis (1998) Proceedings from Cost 244bis International Workshop on Electromagnetic Fields and Non-Specific Health Symptoms. Sept 19-20, 1998, Graz, Austria

Bergqvist U and Vogel E (1997) Possible health implications of subjective symptoms and electromagnetic field. A report prepared by a European group of experts for the European Commission, DGV. Arbete och Hälsa, 1997:19. Swedish National Institute for Working Life, Stockholm, Sweden. ISBN 91-7045-438-8.

Rubin GJ, Das Munshi J, Wessely S. (2005) Electromagnetic hypersensitivity: a systematic review of provocation studies. Psychosom Med. 2005 Mar-Apr;67(2):224-32

Seitz H, Stinner D, Eikmann Th, Herr C, Roosli M. (2005) Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication---a literature review

published between 2000 and 2004. Science of the Total Environment, June 20 (Epub ahead of print).

Staudenmayer H. (1999) Environmental Illness, Lewis Publishers, Washington D.C. 1999, ISBN 1-56670-305-0.

Radiation and health (who.int)

International Agency for Research on Cancer

World Health Organization

PRESS RELEASE
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31 May 2011

IARC CLASSIFIES RADIOFREQUENCY ELECTROMAGNETIC FIELDS AS POSSIBLY CARCINOGENIC TO HUMANS

Lyon, France, May 31, 2011 -- The WHO/International Agency for Research on Cancer (IARC) has classified radiofrequency electromagnetic fields as **possibly carcinogenic to humans (Group 2B)**, based on an increased risk for **glioma**, a malignant type of brain cancer[1], associated with wireless phone use.

Background

Over the last few years, there has been mounting concern about the possibility of adverse health effects resulting from exposure to radiofrequency electromagnetic fields, such as those emitted by wireless communication devices. The number of mobile phone subscriptions is estimated at **5 billion globally**.

From **May 24–31 2011, a Working Group of 31 scientists from 14 countries has been meeting at IARC in Lyon, France, to assess the potential carcinogenic hazards from exposure to radiofrequency electromagnetic fields**. These assessments will be published as Volume 102 of the IARC *Monographs*, which will be the fifth volume in this series to focus on physical agents, after **Volume 55** (Solar Radiation), **Volume 75** and **Volume 78** on ionizing radiation (X-rays, gamma-rays, neutrons, radio-nuclides), and **Volume 80 on non-ionizing radiation (extremely low-frequency electromagnetic fields)**.

The IARC Monograph Working Group discussed the possibility that these exposures might induce long-term health effects, in particular an increased risk for cancer. This has relevance for public health, particularly for users of mobile phones, as the number of users is large and growing, particularly among young adults and children.

The IARC Monograph Working Group discussed and evaluated the available literature on the following exposure categories involving radiofrequency electromagnetic fields:

- occupational exposures to radar and to microwaves;
- environmental exposures associated with transmission of signals for radio, television and wireless telecommunication; and
- personal exposures associated with the use of wireless telephones.

International experts shared the complex task of tackling the **exposure data**, **the studies of cancer in humans**, the **studies of cancer in experimental animals**, and the **mechanistic and other relevant data**.

[1] **237 913 new cases of brain cancers** (all types combined) occurred around the world in 2008 (gliomas represent 2/3 of these). Source: **Globocan 2008**

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Results

The evidence was reviewed critically, and overall evaluated as being *limited*[2] among users of wireless telephones for glioma and acoustic neuroma, and *inadequate*[3] to draw conclusions for other types of cancers. The evidence from the occupational and environmental exposures mentioned above was similarly judged inadequate. The Working Group did not quantitate the risk; however, one study of past cell phone use (up to the year 2004), showed a 40% increased risk for gliomas in the highest category of heavy users (reported average: 30 minutes per day over a 10-year period).

Conclusions

Dr Jonathan Samet (University of Southern California, USA), overall Chairman of the Working Group, indicated that "the evidence, while still accumulating, is strong enough to support a conclusion and the **2B classification**. The conclusion means that there could be some risk, and therefore we need to keep a close watch for a link between cell phones and cancer risk."

"Given the potential consequences for public health of this classification and findings," said IARC Director Christopher Wild, "it is important that additional research be conducted into the long-term, heavy use of mobile phones. Pending the availability of such information, it is important to take pragmatic measures to reduce exposure such as hands-free devices or texting. "

The Working Group considered hundreds of scientific articles; the complete list will be published in the Monograph. It is noteworthy to mention that several recent in-press scientific articles[4] resulting from the **Interphone study** were made available to the working group shortly before it was due to convene, reflecting their acceptance for publication at that time, and were included in the evaluation.

A concise report summarizing the main conclusions of the IARC Working Group and the evaluations of the carcinogenic hazard from radiofrequency electromagnetic fields (including the use of mobile telephones) will be published in **The Lancet Oncology in its July 1 issue, and in a few days online**.

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[2] '***Limited evidence of carcinogenicity'***: A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

[3] '***Inadequate evidence of carcinogenicity'***: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available.

[4] a. 'Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case-control study' (the Interphone Study Group, in Cancer Epidemiology, *in press*)
b. 'Estimation of RF energy absorbed in the brain from mobile phones in the Interphone study' (Cardis et al., Occupational and Environmental Medicine, *in press*)
c. 'Risk of brain tumours in relation to estimated RF dose from mobile phones – results from five Interphone countries' (Cardis et al., Occupational and Environmental Medicine, *in press*)
d. 'Location of Gliomas in Relation to Mobile Telephone Use: A Case-Case and Case-Specular Analysis' (American Journal of Epidemiology, May 24, 2011. [Epub ahead of print].

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Link to the **audio file** posted shortly after the briefing:
http://terrance.who.int/mediacentre/audio/press_briefings/

About IARC

The International Agency for Research on Cancer (IARC) is part of the World Health Organization. Its mission is to coordinate and conduct research on the causes of human cancer, the mechanisms of carcinogenesis, and to develop scientific strategies for cancer control. The Agency is involved in both epidemiological and laboratory research and disseminates scientific information through publications, meetings, courses, and fellowships.

If you wish your name to be removed from our press release e-mailing list, please write to com@iarc.fr.

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ABOUT THE IARC MONOGRAPHS

What are the IARC Monographs?

The ***IARC Monographs*** identify environmental factors that can increase the risk of human cancer. These include chemicals, complex mixtures, occupational exposures, physical and biological agents, and lifestyle factors. National health agencies use this information as scientific support for their actions to prevent exposure to potential carcinogens. Interdisciplinary working groups of expert scientists review the published studies and evaluate the weight of the evidence that an agent can increase the risk of cancer. The principles, procedures, and scientific criteria that guide the evaluations are described in the **Preamble** to the IARC Monographs.

Since 1971, more than 900 agents have been evaluated, of which approximately 400 have been identified as **carcinogenic or potentially carcinogenic** to humans.

Definitions

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 2.

This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost *sufficient*, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (*probably carcinogenic to humans*) or Group 2B (*possibly carcinogenic to humans*) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data. The terms *probably carcinogenic* and *possibly carcinogenic* have no quantitative significance and are used simply as descriptors of different levels of evidence of human carcinogenicity, with *probably carcinogenic* signifying a higher level of evidence than *possibly carcinogenic*.

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.

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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 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.

Definitions of evidence, as used in IARC Monographs for studies in humans

The evidence relevant to carcinogenicity from studies in humans is classified into one of the following categories:

Sufficient evidence of carcinogenicity: The Working Group considers that a causal relationship has been established between exposure to the agent and human cancer. That is, a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence. A statement that there is *sufficient evidence* is followed by a separate sentence that identifies the target organ(s) or tissue(s) where an increased risk of cancer was observed in humans. Identification of a specific target organ or tissue does not preclude the possibility that the agent may cause cancer at other sites.

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Limited evidence of carcinogenicity: A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence of carcinogenicity: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available.

Evidence suggesting lack of carcinogenicity: There are several adequate studies covering the full range of levels of exposure that humans are known to encounter, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer at any observed level of exposure. The results from these studies alone or combined should have narrow confidence intervals with an upper limit close to the null value (e.g. a relative risk of 1.0). Bias and confounding should be ruled out with reasonable confidence, and the studies should have an adequate length of follow-up. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the cancer sites, conditions and levels of exposure, and length of observation covered by the available studies. In addition, the possibility of a very small risk at the levels of exposure studied can never be excluded.

In some instances, the above categories may be used to classify the degree of evidence related to carcinogenicity in specific organs or tissues.

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