Environmental Health Issues of Radiofrequency and Microwave Exposure*

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Introduction

Although during World War II evidence about acute health effects of high-frequency (HF) EMF exposure was collected, no urgent need for exposure restrictions was recognized. Primarily, persons exposed occupationally or at military applications were considered at risk. Consequently the first exposure standards were issued for limiting the exposure at the work place (ANSI 1966).
During the 50ies and 60ies high-powered sources of RF radiation were installed all over the world due to the increase in radio and television broadcasting. However, because of the usually rather far distances to inhabited areas and the height of the antennae again the main concern was related to occupational exposure.

Because of the increased usage of microwave ovens in the late 60ies possible risks for the general public from leakage of radiation were addressed.

Generally in the Western hemisphere the aspect of tissue heating of exposed subjects from absorption of electromagnetic energy was the primary interest and a considerable amount of research was directed towards a better understanding of the underlying mechanism and of the parameters responsible for variations in the amount of energy absorbed. At the same time dosimetry was developed.

In the late 60ies and early 70ies several Eastern countries set up exposure limits for occupational exposure. These limit values were considerably lower as compared to Western standards. The basis of the derivation of these guidelines was not confined to temperature increase due to absorption of electromagnetic energy. Effects on wellbeing and a wide range of effects studied in animal experiments were considered.

In 1971 WHO recognized the need for international collaboration and convened a working group meeting in The Hague which recommended that protection of men from hazards due to radiofrequency and microwave exposure should be an issue of priority in the field of non-ionizing radiation protection. In 1973 a symposium sponsored by WHO concluded that an international organization should coordinate research on the biological effects of HF EMFs and should develop a non-ionizing protection program. The International Radiation Protection Association (IRPA) became responsible. A joint WHO/IRPA task group issued the first Environmental Health Criteria on radiofrequency and microwaves in 1981 (UNEP/WHO/IRPA, 1981). In 1992 an independent NGO succeeded the IRPA/INIRC: The International Commission on Non-Ionizing Radiation Protection (ICNIRP). ICNIRP dominates the issue of radiation protection and standard setting all over the world. Although an independent NGO, WHO and ILO have in fact delegated the issue of non-ionizing radiation protection to ICNIRP.
ICNIRP adopted the proposal of IRPA from 1984 to use only short-term effects due to absorption of electromagnetic energy and conversion to heat as a basis for the set-up of limit values in the frequency range of 10 MHz to 10 GHz. The basic restriction of a whole-body specific energy absorption

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rate (SAR) derived from a SAR of 4 W/kg already established in 1984 has remained unchanged, despite the growing evidence of biological and health relevant effects at considerably lower levels and the collected epidemiological and experimental data pointing to long-term adverse effects.

Critique of the Strategy of ICNIRP

In order to defend the decision once made to use short-term effects from tissue heating as the sole basis for the derivation of guidelines, ICNIRP seeks refuge in a scientifically and ethically questionable strategy. The reason for these desperate efforts might be found in the intimidating complexity of the exposure conditions which have to be covered by such guidelines.

It is known since decades that exposure to high levels of HF EMFs leads to an increase in body temperature due to the fact that biological systems are lossy dielectrics. Electromagnetic waves propagating through tissues interact with the molecules of the medium and energy transfer occurs. This results in an attenuation of the field and in an increase of the kinetic energy of the molecules, which is reflected in an increase of temperature. It has been established that high levels of exposure lead to adverse effects either directly due to heating (burns, hemorrhage, tissue necrosis) or due to a breakdown of local or systemic temperature regulation. A thorough investigation of the regulatory response and of behavioral effects revealed that a whole-body temperature increase exceeding about 1 °C due to exposure to HF EMFs would increase the probability of immediate thermal effects. Such a temperature increase would occur if a resting adult is exposed to a SAR of about 4 W/kg for 30 minutes. Therefore it seems a reasonable approach to use a SAR of 4 W/kg as a basis for the derivation of guidelines. The advantage of this philosophy is the immediate solution to the problem of the complexity of exposure conditions. This is because in its essence the thermal effects principle is an equal energy principle. Irrespective of the diverse exposure parameters it is assumed that different exposure conditions leading to the same SAR will exert the same effects. Although compliance with the limit values has to be established through measurement of SAR, for practical purposes reference levels derived from basic restrictions (formulated in terms of SAR) and the functional relationship between exposure parameters (e.g. frequency of the electromagnetic waves) and SAR can be used as a surrogate.

However, one has overlooked several inherent problems:

- First of all, SAR is an effect metameter which in practice can not be measured in the exposed subjects but only in phantoms or using mathematical models.
- The definition of SAR as the time derivative of the incremental energy absorbed by, or dissipated in an incremental mass contained in a volume of a given density only makes sense if this derivative exists. However, this is, strictly speaking, not the case. Reducing the volume element beyond limits would result in a transition to quantum mechanical or quantum electrodynamical conditions. On the other hand assuming a volume element of constant density and non-quantum dimensions is biologically unsound. At higher frequencies it has been shown that due to the heterogeneity of tissue structures SAR would be extremely different in neighboring parts of tissues. Measurements in phantoms do not reflect these differences and computer simulation would soon face its limits due to accumulation of computation inaccuracies.
- Furthermore, SAR is a rate! A rate as a basis for limiting exposure makes only sense if short-term effects are considered. At the time of introduction of the SAR principle there was not a single study answering the problem of long-term exposures. Even if only thermal effects are considered, long-term exposure might lead to adverse effects. Since energy is deposited at a constant rate, equilibrium can only be maintained by regulatory processes. The involved thermoregulatory mechanisms could be exhausted or the strain induced might have some adverse effects in the long run. Apparently, at that time no one could imagine long-term exposure conditions at fairly high levels.

- Another problem results from the operational definition of SAR. It includes averaging over time. Complex conditions with intermittent or time-varying exposure is implicitly assumed to have the same effect as a continuous constant exposure of the same average SAR.

Hence, in addition to the assumption that the thermal effects principle is valid, there are a number of additional assumptions necessary to utilize the proposed concept:

- the short-term exposure principle
- the time-dose reciprocity principle
- the continuous wave principle

and to account for combined exposures to several sources of radiation

- the additive effects principle.

The latter states that the effects of a combined exposure to different EMFs results in the sum of the effects of exposure to each EMF presented singly. Although this assumption is biologically naive it might hold around threshold values. However, due to non-linearity of the interaction of EMFs with tissues it might be utterly wrong. There are no investigations systematically investigating the validity of the additive effects assumption.

As stated above, exposures leading to a temperature increase exceeding about 1 °C in a resting adult might lead to adverse health effects. This is the law of thermal effects. And virtually all scientists in the field agree that such levels (or equivalent ones in situations where temperature regulation is stressed by environmental conditions, work load etc. or for subjects with compromised thermoregulation) should be avoided. However, the law of thermal effects has been extended to the thermal effects principle, which states that a necessary and sufficient condition for adverse health effects to occur is a relevant increase in temperature due to absorption of electromagnetic energy. And it is this principle which is severely challenged by a number of scientists. Clearly, demonstrating that there are in fact health relevant effects below the level of relevant tissue heating would not only result in a reduction of exposure limits but would immediately lead to a breakdown of the total architecture of most present day exposure standards and in particular of that presented by ICNIRP (1998). Hence every effort is taken by ICNIRP to refute challenging evidence. This is done by the following strategy:

- epidemiological evidence is not taken seriously because of ‘poor exposure assessment’ (which might be an argument against studies reporting no effect, because exposure misclassification rather leads to wrong negative as to wrong positive results!)
- some studies are falsely claimed to show no effect (e.g. Källen et al. 1982; Beall et al. 1996)
- animal experiments are either discussed in an exceptionally unsound manner (e.g. Chou et al. 1992; with a completely irrelevant reference to benign tumors) or are dismissed due to alleged methodological deficiencies (e.g. Lai & Singh, 1995, 1996) or are wrongly claimed to having been challenged by unsuccessful attempts to replicate the findings (e.g. Kues et al. 1985) or are simply not considered because the studies have not yet been replicated (e.g. Repacholi et al. 1997)
- generally the evidence from different areas of research (epidemiological, in vivo and in vitro studies) is separated and no attempt is made to assess the implications the different studies have on each other
- the presence of positive as well as negative results is taken as an indication of no effect thereby violating fundamental principles of methodology and generally none of the scientifically established methods to aggregate results are used to arrive at an overall assessment

- finally it is said that the findings from experimental studies are difficult to assess since their implications to human health are unclear.

Basically this is an immunization strategy, because no possible result could eventually lead to a falsification of the applied principle(s): Of every experimental result it could be said that its implications to human health are unclear (experimentation in humans is limited for ethical reasons) and epidemiological studies can never establish a causal relationship between exposure and outcome (prospective cohort studies having under ideal conditions the virtue of establishing such a relationship would show results not earlier than in 15 to 30 years, facing the risk that the exposure conditions change essentially during this period compromising the potential to pin down the causal factor). Although ICNIRP states that there is 'no convincing evidence that typical exposure levels lead to adverse reproductive outcomes or an increased cancer risk in exposed individuals' (ICNIRP, 1998, p.507) it does not indicate what would be considered a convincing evidence. Without such a statement about evidence to be considered as a falsification of the fundamental principles applied by ICNIRP, the procedure has to be viewed as scientifically invalid.

An Alternative Approach

It is interesting to note that not only ICNIRP and other standards setting bodies but several reviews from a number of independent scientists (e.g. Verschaeve & Maes, 1998; Hermann & Hossmann, 1997) although pointing at some important results with possible implications to health, have concluded that exposures to HF EMFs seem not to confer an elevated risk. Such statements are possibly due to the expectation that HF EMFs could be assessed in analogy to chemical substances. However, there is little justification for the assumption that the variety of different exposure conditions of HF EMFs could be viewed as a single entity. A toxicological assessment of all hydrocarbons as one entity would be meaningless. Even one single chemical compound might have grossly different effects due to isomerism (e.g. benzpyrene, tetrachlorodibenzodioxin).

The assumption that exposures differing in frequency, modulation type, modulations parameters and a variety of other aspects should exert similar effects and thus should demonstrate consistent results across different studies possibly investigating different endpoints in different systems (which would again introduce new parameters due to specific interaction with the field conditions) seems a little far-fetched. The predominance of the thermal effects principle has resulted in a main-stream of scientific work which might have been governed by the wrong questions derived from possibly futile concepts.

The main question should have been: If it is not the energy of the field propagating through the tissues which is hazardous (at low intensities) what else could it be? It would soon have been recognized that the experimental paradigms used so far are more or less insufficient to answer this question. If the results from epidemiological studies, consistently showing a small but elevated risk for different types of cancer, would have been taken seriously, it would have begun to dawn that real-world exposure conditions are far from being reflected in experimental set-ups used all over the world. There are only a few studies using in situ field exposure conditions (e.g. Haider et al. 1994; Magras & Xenos, 1997). It should be noted that exposure-omission studies (systematically leaving out one component after the other of a complex combined condition) are by far more indicative of a causal relationship than exposure studies (see e.g. the investigations into the contribution of the earth magnetic field summarized by Blackman in this volume).

Instead of considering all HF EMFs as one entity we, as a first approximation, should divide the exposures into meaningful subsets, as it is done in toxicology for chemical substances. Such subsets could be ELF amplitide modulated fields in the different frequency bands used for radio and television broadcasting, modern mobile telecommunication frequencies, microwave fields at 2.45 GHz (microwave ovens), radar exposures and different conditions found in occupational exposures (e.g. shortwave diathermia).

To derive reference values the evidence related to these different conditions could then be evaluated and subjected to a process widely used in environmental health: It consists of several steps starting from a LOAEL (lowest observed adverse effect level) or a NOAEL (no observed adverse effect level) and proceeds by application of several empirically determined or convention based factors accounting for differences between and within species, the possibility of continuous and combined exposures and possible non-threshold conditions. The result of such a procedure could immediately be traced back to the evidence and could be changed easily along with growing knowledge.

If this procedure is applied to the derivation of preliminary exposure limits for protection of the general public from potential adverse effects of emissions from mobile phone base stations we are faced with the problem that there is not a single study, experimental or other, which investigated health effects of such exposures (only one in vitro short term exposure study by Maes et al. 1996 was done at a base station). However, some studies, devoted to the effects of mobile phones, could with caution be used for this assessment due to the fact that the exposures were in the far field.

Another problem arises from the uncertainty about the underlying mechanism, which makes a judgment about endpoints used in human studies, whether or not they can be considered as adverse effects, difficult. Consider for example the studies on effects of pulsed HF EMFs on human sleep (Mann & Röschke 1996; Wagner et al. 1998; Borbely et al. 1999): Although the reported effects are definitely not directly related to health (we cannot conclude, due to the short duration of the experiments, that any health relevant chronic sleep disturbances may occur) and are similar to those observed after mild barbiturate or anti-depressive therapy, the occurrence of the effect could be attributed to an interaction of the field with basic functions of the central nervous system and therefore viewed as an indication of a possible hazard, because this interaction will not be confined to structures guiding the architecture of sleep. Hence the interpretation with regard to health will depend on the details of the unknown mechanism and not on the observed endpoint.

Hundreds of millions of people all over the world are now exposed to microwaves from mobile phones and base stations. Compared to the enormous success of telecommunication industry and the still continuing growth, there is definitely too little research into possible biological and health effects of exposure to EMFs emitted by mobile phones and especially of base stations. There are only about 40 studies published so far on this issues. Many of these studies, however, can not be included into an assessment due to severe deficiencies. Among these deficiencies poor dosimetric standards and not rigorously controlled exposure conditions are the least problem.

Due to the short exposure duration it is too early for epidemiological studies to demonstrate an effect (Rothman et al. 1996). With regard to the older analog mobile phone system Hardell et al. (2000) found an association between the location of brain tumors and the side of the head the mobile phone was predominantly used. However, it is too early to draw a conclusion on this issue, except that it underlines the recommendation of prudent usage of this technology. A generalization to effects of emissions from base stations is not possible.

A number of experimental studies in humans revealed both positive and negative results. All of these studies were about effects of short-term exposure. The effects investigated were related to cognitive performance, EEG parameters, sleep, blood pressure and hormones. Animal studies and in particular cancer related studies are difficult to assess because it is not known at which stage of carcinogenesis exposure might have an effect. Only one study has been conducted so far without a restriction to a certain stage (Repacholi et al. 1997). Chagnaud et al. (1999) investigated effects on tumor progression, Imaida et al. (1998a,b) on tumor promotion, and Adey et al. (1999) both on transformation and on tumor development. Furthermore, the latter studies, with the exception of Chagnaud et al. (1999), were done under near-field conditions and can therefore not be used for the assessment of base stations.

The application of the procedure to derive exposure reference levels described above will be exemplified using the study of Repacholi et al. (1997): The result obtained in this investigation was a more than two-fold increase in lymphoma in the exposed transgenic mice as compared to sham exposed.

It is definitely a health relevant effect, although the types of lymphoma developed in mice are different to those in humans. Considering the time course of the development of lymphoma, an effective level of 0.5 W/kg is estimated. The study provides no dose effect relationship. Hence it does not directly allow calculation of a NOAEL. In such cases conventionally the NOAEL is set at the square root of 10 down from the LOAEL. Because the effect observed was more than a doubling of the lymphoma incidence (hence not the lowest increase of incidence detectable in such an experiment) the square root of 100 has to be chosen. Conventionally an interspecies factor (accounting for the fact that in animal studies effects on wellbeing are not included) - usually set at a value of 3 – would be introduced. However, this additional factor was omitted, accounting for the lower metabolic rate in mice. Because a whole body SAR of 0.5 W/kg at 900 MHz is equivalent to about 2.8 mW/cm² (28 W/m²) exposure level in humans, we arrive at a NOAEL of 0.28 mW/cm² (2.8 W/m²). Again due to convention, inter-individual differences are taken into consideration by applying an intra-species factor of 10, resulting in a NOAEL for single and time-limited exposure of sensitive subjects of 0.028 mW/cm² (0.28 W/m²). In cases where there are no data available on the relationship between exposure duration and effect, a linear function is assumed. In the present case (with one hour exposure) this factor amounts to 24. The result is a NOAEL for unlimited exposure of 1.167 µW/cm² (11.67 mW/m²). Possible combined exposures are accounted for by a further factor conventionally set to 10. The net result is a reference level of 0.117 µW/cm² (1.17 mW/m²) for the general public. Similar calculations could be done for several other studies resulting in reference levels of 0.1 to 1 µW/cm² (1 to 10 mW/m²).

Final Remarks

The studies conducted so far do not allow a final conclusion. And any suggestion for a reference level has to be considered preliminary. A main problem is the unsystematic and erratic nature of scientific investigations. Despite the plea of WHO to coordinate research on an international level, neither ICNIRP nor the EMF model project of WHO have served this purpose. The research needs identified by these bodies are just reactions to intriguing results already reported and not strategic and proactive. This seems to be due to a disbelief into all effects not in line with the thermal effects principle. Hence all the energy is put into demonstrating that these results are wrong. Scientific progress is not likely to emerge from such a defensive strategy. All our strengths should be unified by the challenging issue of the nature of interactions between EMFs and the human body. I have a feeling that the most important questions have not even been formulated let alone that they have been answered.

References

- Adey, W. R.; Byus, C. V.; Cain, C. D.; Higgins, R. J.; Jones, R. A.; Kean, C. J.; Kuster, N.; MacMurray, A.; Stagg, R. B.; Zimmerman, G.; Phillips, J. L.; Haggren,W. (1999). Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats chronically exposed to 836 MHz modulated microwaves. Radiat Res, 152:293-302.
- ANSI (1966). Safety level of electromagnetic radiation with respect to personnel. New York (USAS C. 95.I-1966)
- Beall, C., Delzell, E., Cole, P. & Brill, I. (1996). Brain tumors among electronics industry workers. Epidemiology 7:125-130.
- Borbely, A. A.; Huber, R.; Graf, T.; Fuchs, B.; Gallmann, E.; Achermann, P. (1999). Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. Neurosci Lett 275:207-210.
- Chagnaud, J.-L.; Moreau, J.-M.; Veyret, B. (1999). No effect of short-term exposure to GSM-modulated low-power microwaves on benzo(a)pyrene-induced tumours in rat. Int. J. Radiat. Biol. 75:1251-1256.
- Chou, C.-K.; Guy, A. W.; Kunz, L. I.; Johnson, R. B.; Crowley, J. J.; Krupp, J. H. (1992). Long-term, low-level microwave irradiation of rats. Bioelectromagnetics 13:469-496.

- Haider,Th.; Kansmüller, S.; Kundi, M.; Haider, M. (1994). Clastogenic effects of radiofrequency radiation on chromosomes of Tradescantia. Mutat. Res. 324:65-68.
- Hardell, L.; Nasman, A.; Pahlson, A.; Hollquist, A. (2000). Case-control study on radiological work, medical X-ray investigations, and use of cellular telephones as risk factors for brain tumors. MedGenMed, May 4, 2000.
- Herrman, D.M.; Hosmann, K-A. (1997). Neurological effects of microwave exposure related to mobile communication. J. Neurol. Sci. 182:1-14.
- ICNIRP (1998). Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Physics 74(4):494-522.
- Imaida, K.; Taki, M.; Yamaguchi, T.; Ito, T.; Watanabe, S.; Wake, K.; Aimoto, A.; Kamimura, Y.; Ito, N.; Shirai, T. (1998a). Lack of promoting effects of the electromagnetic near-field used for cellular phones (929.2 MHz) on rat liver carcinogenesis in a medium-term liver bioassay. Carcinogenesis, 19(2), 311-314.
- Imaida, K., Taki, M., Watanabe, S., Kamimura, Y., Ito, T., Yamaguchi, T., Ito, N. and Shirai, T. (1998b). The 1.5 GHz electromagnetic near-field used for cellular phones does not promote rat liver carcinogenesis in a medium-term liver bioassay. Jpn J Cancer Res, 89(10), 995-1002.
- IRPA (1984). Interim guidelines on limits of exposure to radiofrequency electromagnetic fields in the frequency range from 100 kHz to 300 GHz. Health Physics 54, 115-123.
- Källen, B.; Malmquist, G.; Moritz, U. (1982). Delivery outcome among physiotherapists in Sweden: Is non-ionizing radiation a fetal hazard? Arch. Environ. Health 37:81-85.
- Kues, H. A.; Hirst, L. W.; Lutty, G. A.; D'Anna, S. A.; Dunkelberger, G. R. (1985). Effect of 2.45-GHz microwaves on primate corneal endothelium. Bioelectromagnetics 6:177-188.
- Lai, H.; Singh, N. P. (1995). Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics 16:207-210.
- Lai, H.; Singh, N. P. (1996). Single-and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int J Radiation Biol 69:513-521.
- Maes A.; Collier M.; Slaets D.; Verschaeve L. (1996). 954 MHz microwaves enhance the mutagenic properties of mitomycin. Environ. Mol. Mutagen. 28:26-30.
- Magras, I.N.; Xenos, T.D. (1997). RF radiation-induced changes in the prenatal development of mice. Bioelectromagnetics 18:455-461.
- Mann, K.; Röschke, J. (1996). Effects of pulsed high-frequency electromagnetic fields on human sleep. Neuropsychobiology 33:41-47.
- Repacholi M.H.; Basten A.; Gebski V.; Noonan D.; Finnie J.; Harris A.W. (1997). Lymphomas in Eμ-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. Radiation Res. 147:631-640.
- Rothman, K. J.; Loughlin, J. E.; Funch, D. P.; Dreyer, N. A. (1996). Overall mortality of cellular telephone customers. Epidemiology 7:303-305.
- UNEP/WHO/IRPA (1981). Radiofrequency and microwaves. Geneva: World Health Organization; Environmental Health Criteria 16.
- Verschaeve, L.; Maes, A. (1998). Genetic, carcinogenic and teratogenic effects of radiofrequency fields. Mutat. Res. 410:141-165.
- Wagner, P.; Röschke, J.; Mann, K.; Hiller, W.; Frank, C. (1998). Human sleep under the influence of pulsed radiofrequency electromagnetic fields: A polysommnographic study using standardized conditions. Bioelectromagnetics, 19, 199-202.