Mobile Telecommunications and Health

Review of the current scientific research in view of precautionary health protection

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Review of the Current Scientific Research in view of Precautionary Health Protection

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1 Introduction

1.1 New Technologies and Precautionary Health Protection

No technology covering virtually entire countries with its emissions has ever been rolled out as quickly as mobile telecommunications. At the same time, there are only few direct studies of the potential health risks of typical mobile telecommunications frequencies and modulations for the exposed population. Also, many of the existing studies worked with high intensities, which will only be found in rare cases in the real environment. High intensities of high frequency electromagnetic fields can heat the absorbing tissue and trigger stress reactions in the body and thus with rising temperatures lead to thermal damage. Effects from high intensity high frequency EMFs, also know as thermal effects, on the central nervous system, the immune system, the cardio-vascular system and the reproductive system including teratogenic effects, have been proven for mammals with a multitude of experiments.

The results of studies of the thermal effects of high frequency EMFs form the basis of the recommendations of the International Commission on Non-Ionizing Radiation Protection (ICNIRP), which, in the past, were the basis for the guidelines set by the government in Germany and many other countries. The base guideline was an upper limit on the Specific Absorption Rate (SAR), i.e. the amount of energy absorbed by the body from the field within a given unit of time.

According to ICNIRP, thermal damage will not occur at SAR values of under 4 W/kg and exposure levels of 0.4 W/kg for professional exposures and 0.08W/kg for the general population are considered safe.

Parallel to the experiments examining thermal effects, there have been a growing number of studies examining the effects on the body of HF EMFs at sub-thermal intensities. We now have a plethora of experimental studies examining a variety of effects on all levels of the organism, ranging from effects on single cells to effects which manifest themselves as reactions of the entire body. In addition to the experimental studies, there have been a number of epidemiological studies in order to establish the possible causal correlations between higher exposures to HF EMFs, for example as found near base stations, and health damage amongst the population groups with higher exposures.

The mobile telecommunications situation reflects, once again, the dilemma already known from chemical toxicology: The study of potential health effects cannot generally compete with the speed of technical development and the roll out of the product. The extremely fast roll out of the mobile telecommunications technology and the accompanying public fear of the potential danger of this technology have stimulated research insofar that now we have more studies examining the effects of frequencies and modulations as used in mobile telecommunications on biological systems. There are also a growing number of experiments using lower intensities, reflecting the real conditions of exposure in the vicinity of base stations and equipment, so that effects found in the studies can be extrapolated into real life conditions. The number of studies which examine the

physiological effects of real mobile exposures is still very low, compared to the degree of penetration achieved by the technology and the number of (potentially) exposed persons. The WHO amongst others, have only recently begun to develop targeted strategies to examine the potential health risk from mobile telecommunications and results can earliest be expected within several years.

In the meantime, it is only possible to assess the potential dangers of mobile telecommunications using the results generated by uncoordinated research, which is still mainly orientated towards topics and criteria of relevant to science only, rather than addressing the requirements of society as a whole.

Faced with a state of incomplete scientific research it is necessary to chose between two fundamentally different assessment theories when planning to assess the potential health risks of new technologies:

The first theory is based on the (without doubt correct) scholarly understanding that is practically impossible to prove the 'non-harmfulness' to human health or the environment of a technology, a material or a product. This understanding is interpreted in such a way that a presupposition of 'not guilty' is adopted and any risks have to be unequivocally proven.

'Unequivocal proof' in this context means the consistent evidence for a biological-physiological or an ecological chain of effects, from the biophysical or biochemical primary effect through to the physiological effects and the resulting illness or, if applicable, the ecological damage.

This theory, which is firmly based in scientific conservatism, has the advantage that it will stand up in court and will not hinder the introduction of new technologies. It is methodologically simple, since it is sufficient to examine studies which are presented as 'proof' with regards to their methodological correctness and their validity and then to put all these reviewed pieces of evidence together like a jigsaw to produce a whole picture. The complete whole picture finally constitutes the scientific proof required by the legislators and courts.

The disadvantage of this theory is obviously the length of time necessary to obtain enough knowledge for a completed chain of proof, during which many facts will be created, which may later prove irreversible or only reversible with very high costs attached, such as investments and irreversible damage to health and the environment.

The second theory solves the dilemma of the time delay. It is based on the assessment of the potential risks of a technology on the basis of existing knowledge. If there are sufficient indications that there may be damaging effects, the precautionary principle for the protection of health and the environment will apply and avoidable exposures will be avoided until such time when there is enough knowledge for a wider introduction of the technology in question. This theory draws its justification not least from the experiences with the introduction of technologies and products (such as asbestos, DDT, CFCs, formaldehyde, wood preservatives, mass X-ray screenings etc.), which were widely used, even many years after the first clear indications of health and ecological damage had appeared. When finally sufficient scientific proof for the health and ecological damage

was provided, it took many more years until the further use was finally reduced and banned through the courts and international negotiations.

The advantage of the precautionary principle is of course primarily medical and ecological, since exposures are initially limited to a level recognised as safe under the precautionary principle. But it can also offer economical advantages, because firstly, it may prevent potentially highly risky investments, but also secondly, because the commitment to and observance of the precautionary principle will create trust within the general population and thus increase acceptance for the placing of emitting equipment.

On the other hand, it will be the industry – as the owner of emitting equipment – who has to bear the disadvantage of this principle, when it becomes clear that, for precautionary reasons, an economically and technically perfectly-suited site can't be approved, or maybe even an entire technology has to be abandoned.

Furthermore, the methodological difficulties of this theory must not be underestimated, since it is not enough to prove the reliability of single scientific studies, which is just as essential under this premise as under the first theory. The ultimate goal however is – to remain with the jigsaw analogy – to put the existing jigsaw pieces together and recognise early on which pictures might appear once the work is completed.

1.2 Terms of Reference and Structure of the Review

The aim of this study was the assessment of the potential risks of electromagnetic fields as they are used for mobile telecommunications with respect to precautionary health protection. To this aim, the scientific literature was reviewed with regards to study results which might be of importance to the assessment of potential health risks from mobile telecommunications.

To create a base for later scientific discussion, a list of studies which are particularly important in this respect should be created. On the basis of these papers, the health risk from exposure to electromagnetic fields from mobile telecommunications should be assessed. Finally, recommendations for future scientific studies should be formulated.

The methodological aspects of this examination are presented in Chapter 2. This is followed by a review of the current scientific knowledge of the effects of high frequency electromagnetic fields. This review is structured according to the different levels of effects:

- biophysical and biochemical primary effects of HF fields on organic matter as a whole or at the level of cells and membranes (Chapter 3)
- primary biological effects on the cellular level, i.e. on the genetic substance and on intracellular processes as well as cell transformation and cell proliferation (Chapter 4)
- patho-physiological effects, i.e. physiological effects with possible but not certain negative health implications (Chapter 5)
- pathological effects, which means manifested illness and other effects such as the damage of cognitive functions, which have been found in epidemiological or experimental studies (Chapter 6).

The conclusions of all findings are drawn in Chapter 7. In Chapter 8, we make recommendations for precautionary health protection with regards to exposures to the electromagnetic fields of mobile telecommunications and for focal points for further research.

2 Collating and Interpreting the Scientific Data (Methodology)

2.1 Criteria for the Selection of Papers

In order to include a maximum of relevant literature, we analysed the literature we have catalogued in our own database, EMFbase, as well as exploring the three following paths:

- research in other relevant scientific databases
- complete sifting of at least the last two full years' issues of all relevant scientific journals available in the Central Library of Medicine in Cologne, the Technical Information Library in Hanover, and the Library of the Medical University of Hanover
- evaluation of all existing monographs, reviews and conference reports related to the subject matter

The basic literature research was finished in February 2000.

Literature databases are a convenient research tool, but their value in assessing the current scientific knowledge in a subject matter is limited by the number of registered publications, inconsistent use of keywords, the changing understanding of certain procedures, effects etc. and last but not least, due to long time delays between the time of publication and availability in the database. Furthermore, databases usually only keep abstracts of papers, and those differ often from the full text with regards to the presentation and interpretation of the results. Our research for this review confirmed this observation, reflecting the results of a study of Pitkin et al. (1999) according to which almost 40% of all papers published in the six largest medical journals contained inaccuracies and mistakes in the abstracts. To be at the cutting edge of scientific knowledge, it is necessary to research current scientific journals and find older papers via monographs and reviews. Reviews are only useful to gain an overview over a subject matter and as a source for literature leads. It is inappropriate to use assessments or interpretations of a review study since some authors of reviews will have based their conclusion on abstracts rather than the full texts of the papers they discuss.

2.2 Assessment Criteria

One sub-goal of the present paper was to identify those scientific papers which are particularly interesting for the assessment of potential health risks caused by the electromagnetic emissions of mobile telecommunications. (Extracts from our database EMFbase with a summary of the results of these papers can be found in Annex E. In the source references, these papers carry an asterisk*). Only peer reviewed papers published in scientific journals were included in our review. We also accorded weight to the 'Impact Factor', which is calculated by the Institute for Scientific Information in Philadelphia. This factor is a rough measure for the amount of importance and reputation attributed to a scientific journal in its subject matter.

The papers able to pass this first filter were subsequently interpreted according to the following criteria:

- carrier frequency or frequency range
- manner of modulation
- modulation frequency or frequency range
- power flux density
- specific Absorption Rate
- electric filed strength
- duration of exposure
- other parameters of exposure (such as other fields [incl. ELF], ambient and if applicable body temperature, particular forms of modulation)
- source of exposure or environment of the exposure (such as antenna emitting freely, anechoic chamber, transmission line)
- object of experiments (human, animal, cell system)
- examined pathological results (manifested illness and other effects on the whole body)
- examined patho-physiological effects (physiological effects with a potential for health damage)
- examined biological effects (mostly on the cellular level)
- examined biophysical and biochemical processes (primary effects on the level of molecules, membranes etc.)
- methodology of the experiments (procedures used)
- results (including a mention if our own interpretations differ from those of the author)
- statistical significance of the results
- appropriateness of the model (with regards to the statements made about effects on humans)
- appropriateness of the methodology (methodical weakness analysis)
- documentation of the conditions of the experiments (completeness, reproducibility)
- context of other experiments (mention of experiments with the same or contradicting results)
- meaning (Main conclusions drawn from the results, importance for the assessment of health risks for humans)

Because of the delay of science with regards to the electromagnetic frequencies emitted by mobile telecommunications, a risk analysis cannot be limited to the frequencies and

modulations actually used by this technology. Therefore, we have included all papers examining carrier frequencies from 100MHz to 10GHz. In the experiments at cellular level, but also in animal experiments, effects have been found that only appear at certain modulations or are a lot stronger at these modulations (chapter 3 and 4). At this point in time it is not possible to determine whether the majority of the found effects are caused by the HF carrier wave or the modulation. This is why we have included all forms of modulation into this review. Because of the nature and the importance of the so-called 'thermal effects' (chapter 3.1) we have not set an exclusion limit for power flux density and Specific Absorption Rate. However, we did not include papers, in which the EMF exposure led to a considerable rise in body temperature (>1ºC) of the animals or human subjects.

When evaluating the papers, we kept making the following observations:

- important single results are 'masked' for example when data are 'pooled'
- certain observations are dismissed by the authors as 'blips' if they don't fit the (expected/otherwise observed) general trend, without sufficient explanation being offered for this dismissal
- single results are not taken into account for statistical reasons, but a common trend is not recognised or not sufficiently acknowledged.

In such cases, whenever this was possible based on the existing data, we proceeded to make our own interpretations. Where our evaluation differed from the main statements of the authors, it will be noted.

3 Primary Reciprocal Effects between High Frequency Electromagnetic Fields and Biological Systems (Biophysical and Biochemical Processes)

3.1 Thermal Effects

3.1.1 Effects of Homogenous Warming

HF electromagnetic fields are absorbed depending on the frequency and polarisation of the fields on the one hand and the dimensions and material characteristics of the biological system on the other hand. They cause electric currents (dominant in the range under 1 MHz), polarisation effects and potential differences on cell membranes (in the range between 1 MHz and 100 MHz) or trigger rotational oscillations of polar molecules (mainly within the GHz range). All these processes can lead to a warming of the biological material if the intensity is sufficient (Ohmic losses in the low frequency range and dielectrical losses in the GHz range). The avoidance of health-damaging warming is the base of the concept of SAR, expressed by limiting the specific absorption rate, measured as the energy absorption per unit, to a rate which will exclude overheating based on the body's own thermo-regulative processes. For humans, a whole body exposure of 0.4 W/kg corresponds approximately to half the metabolic base rate. In absence of heat conduction or other thermal dissipation, a SAR of 0.4 W/kg will lead to a temperature rise of 10^{-4}K/sek (Foster 1996) in soft tissue like muscles or the brain.

3.1.2 Microthermal Effects

The warming through microwaves is fundamentally different from the warming through a water bath for example. In the latter case the energy is transmitted by stochastic collisions. In microwave heating it is in the simplest case the electrical component which puts polar molecules within the medium collectively in vibration (3.2.1). Because of 'friction' with the dense ambient medium, the energy is quickly transmitted to this medium and further dissipated by collisions. When corresponding inner molecular degrees of freedom exist, the microwave energy can also be absorbed as a quantum and, in a large molecule, stored (3.3.). Compared to conventional warming, the absorption of a microwave quantum is a singular process, which can lead to localised warming if the absorbing molecules are suitably distributed. Liu & Cleary (1995) show in a theoretical model that at the cellular level, membrane-bound water can lead to frequency dependent spatial discrepancies in dissipation of the SAR and the induced HF fields.

Microthermal effects can also be caused by the non-uniformity of thermal conductivity of tissue at microscopic level, especially when the warming is short, strong and local. This is of importance mainly for the evaluation of pulsed fields, because in such fields, even at a low average power flux density, the energy absorbed during a pulse can be very high. Radiation in the form of short pulses can lead to a very high rate of temperature rise, which can itself trigger thermoelastic waves, a phenomenon, which is inked to the

acoustic perception of microwaves. A high peak-SAR can also trigger thermally-induced membrane phenomena (Foster 1996).

3.2 Direct Field Effects

3.2.1 Effects from the Electrical Component of the Electromagnetic Field

The electric component of the electromagnetic field exerts a force on electrical charges, permanent dipole moments, induced dipole moments and higher multipole moments. The forces on charges create currents, however these only play a role in the lower HF range, causing changes in membrane potentials (stimulation) or thermal effects (see above).

Permanent charge distributions in biomolecules and cells lead to permanent dipole (or higher multipole) moments. The electrical field exerts a torque on dipoles, which tries to align the dipole moment parallel to the field. In alternating fields with not too high frequencies, the interactions lead to oscillations of the dipoles. In dense media, these oscillations are hindered by interactions with the surrounding particles, which lead to heating (see above). If the particles are too large or the surrounding particle density is too high or if the frequency of the field is too high, the oscillations cannot develop.

The threshold field strengths for the orientation of dipolar cells and other objects of similar size (radius of approx. 1 μm) are at 100 V/m, the cut-off frequencies in water (temperature 300K) are at circa 0.05Hz, hence far outside the HF range. DNA molecules and other bio-polymers can be put into oscillation by fields with frequencies in the kHz range. Spherical protein molecules (radius approx. 5nm) can still follow fields with frequencies up to 400 kHz, however this requires field strengths of 10^6V/m (Foster 1996). Such field strengths are not usually reached in the environment.

The interaction between a field and a particle with an induced dipole moment depends on the field strength to the power of 2, that means, a continuous electrical alternating field influences the particle via a constant torque, however the torque of a modulated field follows the modulation. There is no limitation through a cut-off frequency for the interaction between a field and an induced dipole moment, however for frequencies over 1 MHz, the forces exerted on the cells are very small unless field strengths of several thousand V/m are used. With such field strengths however, strong dielectrophoretic forces appear, which can lead to cell deformations, to the orientation of non-spherical cells and to the so-called coin roll effect, a stringing together of cells. Since the induced dipole moment depends on the polarizability of the particle and the latter on the size of the particle, even higher field strengths are needed for smaller bodies than cells (biopolymers).

Electric fields can induce electrical potentials on cell membranes. The size of these potentials depends on the electric field strength, the dimensions of the cell, the frequency of the field, the electrical conductivity within and outside of the cell as well as the capacitance of the cell membrane.

With frequencies above 1 MHz the membrane is practically short-circuited and the induced membrane potentials become very small. However, theoretical rectification processes and non-linear phenomena at the cell membrane have been discussed, and these could lead to an intensification of the effect and to membrane potentials that have an effect on cell physiology.

3.2.2 Effects from the Magnetic Component of the Electromagnetic Field

With some exceptions, biological tissue is not magnetic and the mutual effects between the magnetic component of an electromagnetic field and tissue are generally small. However, the presence of magnetite crystals, which have a strong capacity to absorb the frequency range of 0.5 to 10 GHz which is relevant for mobile telecommunications, has been found in the human brain as well as in the tissue of many animals (*Kirschvink 1996). Under exposure to amplitude modulated or pulse modulated microwaves, the frequency of the crystal vibrations varies according to the modulation frequency, and thus transmits it, for example in the form of an acoustic wave onto the ambient medium and the cell membrane, which possibly leads to changes of the permeability of the membrane (*Kirschvink 1996). Theoretical calculations show that magnetite transmitted effects can only occur at high densities of superparamagnetic particles (*Dobson & St. Pierre 1998).

3.3 Quantum Effects

The quantum energy from radio and microwaves in the frequency range between 100 MHz to 10 GHz is far too low to break ionic, covalent or hydrogen bonds. Bohr et al. (*1997) have however shown theoretically, that wring resonances can be triggered in chain molecules. The frequencies of these resonances are in the range from 1 to 10 GHz for proteins and 10 MHz to 10 GHz for DNA molecules. The wring modes of molecules manifest themselves as 'torsions' in the molecule chain, which can lead to structural changes.

The influences of microwaves on structural changes in molecules have been found in experiments using the protein ß-Lactoglobuline (*Bohr & Bohr 2000). The triggering of resonant wring modes can even lead to chain breaks, since due to White's Theory, the added energy can be concentrated in a very limited part of the molecule during structural changes (*Bohr et al.). In this part, the chain can break.

3.4 Other Effects

Resonance Phenomena

When the frequency of the electromagnetic wave meets the natural vibrations in the cell structures or in tissue, it can lead to resonances. Rhythmical fluctuations of signal substances, matter-exchange-processes and Ion-conductivity can be found in many neurones, receptors and cell types. These oscillations can influence the membrane potentials and switch certain stimuli on and off. An external field – according to theory – can imprint an external oscillation frequency onto these structures. Neurones which have

been modified in this way can even synchronise the following neurones in the same way. This external synchronisation would even remain after the disappearance of the external stimulus.

Indirect Effects

In addition to the previously described triggering of wring resonances, microwaves can possibly damage the genetic substance via the formation of hydroxyl radicals. The input energy of microwaves is sufficient to raise the ratio of oxidants to anti-oxidants, a self-accelerating chain reaction of free radicals can lead to damage of the DNA (Scott 1992, see also Maes et al. 1995).

3.5 Particular Properties of Pulsed Electromagnetic Fields

In an evaluation of circa 40 studies, in which the biological effects of pulsed high frequency fields were directly compared to those of continuous fields of the same median power density, Postow & Swicord (1996) concluded that in half of the studies, the biological effectiveness of pulsed fields was significantly higher. Only in a few studies were the continuous fields more effective and in the remainder of the studies the effectiveness of both was practically the same. The studies which are mainly discussed in chapter 4 and 5 convey a similar picture.

At first glance, the higher biological effectiveness of pulsed electromagnetic fields in comparison to continuous fields at the same median power flux densities could have an almost trivial cause:

The individual pulses of pulse modulated fields have a higher amplitude than the continuous fields; the possible threshold for the triggering of biological reactions could therefore be passed in these fields during the duration of the pulse.

However, numerous experiments found that the biological response depends in a complicated manner on the duration of the pulse and its frequency. Given that some effects have only been observed at certain pulse frequencies, we presume that in addition to the described effect, there are others which can be originally attributed to the low frequency modulation (see also chapter 4).

4 Biological Primary Effects of High Frequency Electromagnetic Fields Effects on Cellular Level

At the cellular level, it is possible that there may be direct effects of the EM field on the genetic material, which we have collated under the heading Gene Toxicity and which will manifest as mutations if the cell's own repair mechanisms fail. On the other hand, it is also possible that the fields influence cellular processes such as gene-transcription and gene-translation. Furthermore it is possible that the fields can impact on the cell membranes, the intracellular processes of signal transmission and not least the cell cycle. Just like direct damage of the genetic substance, a disruption of these processes can lead to a transformation of the cell, to disruptions of inter-cellular communication and to a changed rate of cell division, which can lead to a slower – or very importantly with respect to a potential carcinogenic effect – faster growth.

4.1 Gene Toxicity

A basic question for the evaluation of the potential dangers of mobile telecommunication is whether the electromagnetic fields used are genotoxic. If the fields had the potential to damage genetic substance directly, they would not only amplify the effects of other carcinogenic teratogenic or mutagenic substances, but they would induce these effects themselves. A direct genotoxic effect of electromagnetic fields with frequencies as they are used for mobile telecommunications has been thought to be not likely in the past (Brusick et al. 1998, Moulder et al. 1999, Repacholi 1997, Repacholi 1998, Saunders et al.1991, Verschaeve 1995, Verschaeve & Maes 1998). The reasons for this assumption were on the one hand that the quantum energy contained in EM field in the radio and microwave range was not sufficient to break molecular bonds. This assumption is no longer tenable after the experiments of Bohr et al. (*1997) and Bohr & Bohr (*2000) (see also chapter 3.3). On the other hand, it was argued that there was a large number of experiments showing no genotoxic effects. Our list of papers in Annex A, Table A.1 shows however, that the much debated findings of the work of Lai & Singh (*1995), in which the direct damage of DNA (single strand and double strand breaks) has been proven, have been confirmed by a whole range of other studies, some by the same laboratory, but also by other groups (*Lai & Singh 1996, 1997, *Phillips 1998, *Sarkar 1994). A study by Varma & Traboulay (1977) on the effect of HF fields on pure DNA had already resulted in hints of direct genotoxic effects, however, this experiment used a relatively high power flux density and therefore significant warming may have occurred, at least locally. Lai and Singh (*1997) found that the dispensation of melatonin and N-Tert-Butylalpha-Phenylnitron (PBN) before the EMF exposure prevented the occurrence of DNA breaks. Melatonin captures free radicals and for PBN it has been proven that it protects cells from cell death induced by free radicals.

In Appendix Table A.1 we also list the experiment of Meltz et al. (*1987) and Stagg et al. (*1997) which examined the influences of EMF fields on the DNA repair mechanisms and the DNA synthesis.

The term chromosome aberration sums up all anomalies of the DNA double strand level with respect to chromatids and chromosomes. Examples for structural chromosome aberrations are: chromatid and chromosome breaks, chromatid gaps, acentric fragments as well as di- and tetracentric chromosomes.

Chromosome aberrations have been observed in a multitude of experimental conditions, *in vivo* as well as *in vitro* (Table A.1). Maes et al.(*1997) found a rise of chromosome aberrations in human lymphocytes in workers who were professionally exposed to radiation from mobile equipment, but also in experiments with human blood under controlled exposure conditions (GSM base station, 15 W/m², exposure time of 2 hours). However, this was the only study so far which used the actual fields of a real base station.

The incidence of micronuclei indicates whether the distribution of chromosomes into the daughter nuclei after a cell division has been normal and complete. A number of studies have proven a higher incidence of micronuclei under the influence of HF EMF fields, which is interpreted as an indication for chromosome damage (Table A.1). With one exception, the frequencies were all over 1 GHz and in most cases the intensities were relatively high.

For the incidence of sister chromatid exchange as a measure for damage at DNA single strand level, only very few studies using typical mobile frequencies and intensities have been done so far (Table A.1). Maes et al.(*1996) found that the radiation of a GSM base station (954 MHz, 217 Hz, duration of exposure: 2 hours) raises the genotoxic effects of Mitomycin C significantly, demonstrated via the sister chromatid exchange.

Genetic damage can lead to cell mutation with possibly damaging effects for the living organism. Mutations which promote faster cell division will be discussed in chapter 4.3. Table A.1 shows in its last block some studies which focussed on the evidence of changes in the genetic materials which manifest themselves as changed properties within the organism.

4.2 Cellular Processes

4.2.1 Gene-Transcription and Gene-Translation

The code of the DNA controls protein synthesis in the ribosomes via the RNA. The creation of RNA, i.e. the imprinting of genetic information happens in the cell nucleus (transcription). The encoded information is transported via messenger-RNA (M-RNA) to the ribosomes and is read there with the help of Transfer RNA (t-RNA). According to the transmitted code, proteins are subsequently synthesized. This process of synthesis is called translation. Since one m-RNA chain can be used by several ribosomes, the rate of synthesis of the corresponding protein can be a lot higher than that of the m-RNA. Mistakes made during the genetic transcription can thus be ‘raised to a higher power’ at the protein level.

In the first block of Appendix Table A.2, we list several recent studies which demonstrated changes of gene transcription and translation under the influence of electromagnetic fields of mobile telecommunications. Fritze et al. (*1997) observed

changed gene transcription in certain areas of the brains of rats which had been exposed to the field of a GSM phone for four hours.

In an *in vitro* experiment, Ivaschuk et al. (*1997) exposed cells to a pulse modulated HF field (836.55 MHz, TDMA 50Hz) and afterwards extracted and analysed the entire cellular RNA.

This showed statistically significant changes with regards to the transcription of the response gene *c-jun* (90W/m², duration of exposure: 20 minutes), however no changes with regards to *c-fos*. The results of the experiments by Goswami et al. (*1999) found a evidence for an influence on the transcription of the response gene *c-fos* by a similar field, whilst for *c-jun* and *c-myc*, no statistically significant effect was observed. The intensities at which effects on gene translation had been observed were well below the values at which thermal effects would occur in mammals.

4.2.2 Membrane Function

There is a large number of experimental evidence that high frequency fields, non-pulsed and pulsed can affect different properties of the ion channels in cell membranes, for example in the form of a lowering of the rate of channel formation or the reduction of frequency of the opening of individual channels (Repacholi 1998). The frequency of openings of ion channels which are activated by acetylcholine was significantly lowered by a microwave field (10.75 GHz) with a power flux density of a few μW/cm². (*D'Inzeo et al.1988). Changes of the membranes as a whole have also been observed under the influence of weak fields. Thus, Phelan et al. (*1992) observed that a 2.45 GHz field, with a pulse modulation of 100 Hz could trigger a phase transition from liquid to solid in melatonin containing cells after an exposure of 1 hour at a SAR of 0.2 W/kg.

4.2.3 Signal Transduction

Ca^{2+}

The divalent Calcium cation Ca^{2+} plays an important role in the cell-signal-transduction: regulating the energy output, the cellular metabolism and the phenotypical expression of cell characteristics.

The signal function of the Ca^{2+} is based on a complicated network of cellular channels and transport mechanisms, which maintains the Ca^{2+} concentration within the cell at a lower level than outside, but which is also linked to dynamic reservoirs. This allows the transduction of extracellular signals (hormones, growth factors) as Ca^{2+} peaks in the cytosol, transmitting information encoded in their intensity and frequency. It is known that this signal process can be disrupted by a variety of toxic chemicals in the environment, which can lead to cell damage and even cell death (Kass & Orrenius 1999).

Studies by Bawin et al. (*1975) and Blackman et al. (*1979) showed very early on *in vitro* experiments that the Ca^{2+} balance of nerve cells and brain tissue can be disrupted by HF fields with low frequency amplitude modulations.

Both studies worked with amplitude modulated 147 MHz fields (with intensities ranging from 5 to 20 W/m^2). The maximum effect occurred at a modulation frequency of 16 Hz. Experiments conducted by Dutta et al. (*1984 *1989) and Lin-Liu & Adey (*1982) also showed significant dependence on the modulation frequencies, in some cases at Specific Absorption Rates of as low as 0.5 W/kg. Equally, Somosy et al. (*1993) found that an effect on the distribution of Ca in intestinal cells is only possible within a field modulated with a low frequency. Wolke et al. (*1996) observed in their experiment on myocytes that exposure to fields with mobile-like carrier frequencies of 900 MHz and 1800 MHz resulted in lower intracellular concentrations of Ca^{2+} for all modulation frequencies (16 Hz, 50 Hz, 217 Hz, 30 KHz) compared to exposures to a continuous 900 MHz field or no exposure at all. A statistically significant effect was only found with the combination of a carrier wave of 900MHz and a modulation frequency of 50 Hz. The Specific Absorption Rate for this experiment was between 0.01 and 0.034 W/kg, far below the range which might be relevant for 'thermal' effects.

Enzymes

Protein kinases are enzymes with the property to phosphorylate other enzymes or proteins. Phosphorylation, a covalent modification by addition of a phosphate group, changes the activity or function of a protein. The protein kinases play an important role in the transmission of information from the membrane receptors for hormones and cytokines into the interior of the cell, and thus in the regulation of many intracellular processes such as glucose and lipid metabolisms, protein synthesis, membrane permeability, enzyme intake and transformation by viruses.

An amplitude modulated 450 MHz field is capable of decreasing the activity of protein kinases which are not activated by cyclical Adenosine monophosphate. Byus et al. (*1984) showed that the degree of inactivity depended on the exposure time as well as the modulation frequency. Maximum effects occurred at exposure times of 15 to 30 minutes with a modulation frequency of 16 Hz.

The enzyme ornithine decarboxylase (ODC) determines the speed of the biosynthesis of polyamines. Polyamines are needed for DNA synthesis and cell growth. ODC is also activated in relation to carcinogenesis. The control of OCD activity from the exterior is facilitated via processes on the cell membrane. Byus et al. (*1988) exposed three different cell types (rat hepatoma cells, egg cells of the Chinese hamster, human melanoma cells) for one hour to a 450 MHz field with a 16 Hz amplitude modulation and a power flux density of 10W/m^2. The exposure raised ODC activity by a little more than 50%. The heightened ODC activity remained for several hours after the exposure. Similar fields with a 60 Hz and a 100 Hz modulation had no effects. Another study (*Penafiel et al. 1997) observed heightened ODC activity after the radiation of L929-cells of mice with a 835 MHz field which had been amplitude modulated at 60Hz or pulse modulated at 50Hz. No effects whatsoever were observed with an analogue mobile phone, a frequency modulation at 60 Hz and a speech amplitude modulation. This last finding confirms other results by the same group, according to which a minimum coherence time of 10 seconds of the field needs to be present for an effect on ODC activity to manifest (*Litovitz et al.1993, 1997, see also Glaser 1998 and Litovitz 1998). The coherence time of speech modulated fields however is shorter than a second.

Further important proof that low frequency modulation has a determining influence on the effects of electromagnetic fields on enzyme activity was found by Dutta et al. (*1994): They compared the effects of a low frequency modulated 147 Hz field (0.05 W/kg) and a combined low frequency electric and magnetic field (ELF EM, 21.2V/97nT). A continuous high frequency field only had a small effect (3.6 per cent) on the activity of enolase in *Escheria Coli*, a 16 Hz modulated field led to an increase in activity of nearly 62 per cent, a 60 Hz modulated field led to a decrease of activity of 28.5 per cent. At ELF-EM a similar response could be observed: increase of enzyme activity by more than 59 per cent at a frequency of 16 Hz and decrease of 24 per cent at 60 Hz. The results of the experiments by Behari et al.(*1998) point in the same direction. They found that a 30 to 35 day long exposure of rats to amplitude modulated fields (6.11 – 9.65 W/kg) led to a significant increase in Na^+-K^+-ATPase activity, which was independent from the carrier frequency, but characteristically dependent on the modulation frequency, because the effect was always stronger at a 16 Hz modulation than at a 76 Hz modulation.

4.2.4 Cell Cycle

An undisrupted signal transduction or efficient cell cycle control mechanisms which are capable of correcting false information or facilitating repairs are the prerequisite for cell cycle progression if the genomic integrity of the cell is to be maintained (Shackelford et al. 1999). Disturbances of the DNA replication can lead to detrimental mutations and as a consequence to cell death or in multicellular organisms to cancer. The causes for irregularities in the course of the cell cycle are almost always to be found in mistakes during signal transduction and/or the failure of control mechanisms.

In Appendix Table A.2. we list studies which examined disruption of the cell cycle. The only *in vivo* experiment is the one by Mankowska et al. (*1979) which also used intensities as they are found in the environment of real emitting equipment. Statistically significant increases of disrupted metaphases with uni-, quadri- and hexavalencies were demonstrated in this study from a power flux density of 5 W/m^2.

Cleary et al. (81996) found in their experiment that 2.45 GHz fields are roughly twice as effective as 27 MHz fields when it comes to the triggering of cell cycle disturbances. Whilst the 27 MHz fields had no influence on the G2/M phase of egg cells of the Chinese hamster, disturbances of all phases were observed in a 2.45 GHz field.

4.3 Cell Transformation and Cell Proliferation

In vitro experiments of the effects of high frequency fields on the rate or division or the rate of proliferation of cells, expressed in the proliferation rate and the (neoplastic) transformation of cells can offer important findings with regards to possible carcinogenic effects of the fields. The adverse influences of the fields which could not be prevented by the cell's own repair mechanisms manifest themselves in disrupted cell proliferation and cell transformation rates.

Table A.3 gives an overview of the studies, in which the effects of high frequency fields on cell transformation and cell proliferation rates were the focus of the examinations.

4.3.1 Cell Transformation

Balzer-Kubiczek & Harrison (*1985, *1989, *1991) found an increase in neoplastic transformations in cells which had been exposed *in vitro* to a high frequency field with a low frequency pulse. The effect depended on intensity, but was only observable, if a tumour promoter (TPA) was added after the exposure.

Czerska et al. (*1992) found that low frequency pulsed microwave radiation (2.45 GHz) increased the rate of transformation of small inactive lymphocytes into large activated lymphoblasts. Continuous radiation could trigger this effect only at power flux densities that also led to measurable warming.

However, the experiments with pulsed radiation which triggered the cell transformation at power flux densities, for which a homogenous warming can be ruled out, showed that homogenous warming cannot be responsible for this effect.

4.3.2 Cell Communication

Disrupted communication between transformed cells and normal cells plays an important role in tumor promotion. Cain et al. (*1997) co-cultivated transformed cells with normal cells. The co-culture was exposed for 28 days to a TDMA (50Hz) modulated 836.55 MHz field as well as to the tumor promoter TPA in various concentrations. At power flux densities of 3 and 30 W/m^2, which corresponded to Specific Absorption Rates of 1.5 and 15 mW/kg, they did not find a statistically significant difference of focus formation between the exposed and the control cultures for any of the TPA concentrations. The data for the lowest intensity (0.3 W/m^2/0.15 mW/kg) show for two of the three TPA concentrations that there was a small but statistically significant difference in the number of foci, and for the lowest TPA concentration also for the surface and density of the foci.

4.3.3 Cell Proliferation

Anderstam et al. (*1983) found in their experiments with bacteria that some strains reacted to the exposure with an amplitude modulated 2.45 GHz field (500Hz, 35 to 100 W/kg) with an increased proliferation. Also for some species, the number of mutations and the frequency of mutations were increased. These results were confirmed by Hamnerius et al. (*1985) amongst others. Grospietsch et al. (1995) found similar results for 150 MHz fields with several amplitude modulations.

Cleary et al. (*1990 a,b) demonstrated on human lymphocytes and on Glioma cells that the rate of cell division was increased after exposure with a continuous 2.45 GHz field. In a newer experiment, the same effect could be observed for exposures with a pulse modulated field of the same carrier frequency (*Cleary et al. 1996).

In the first of the two experiments which were conducted with fields displaying all the characteristics of real pulsed mobile emissions (see also Table A.3), an increased DNA synthesis rate was observed, but no faster proliferation of the examined cells was found. (*Stagg et al. 1997). In the second experiment, at similarly low intensities (0.0021 W/kg) however, transmitted by a GSM modulated 960 MHz wave, an increase of the cell

proliferation rate was found (*Velizarov et al. 1999). The EMF exposure in this experiment was conducted at two different temperatures, which also applied to the relating control cultures. The increase of the proliferation rate only happened in the exposed cell cultures. Similar experiments to prove that microwaves and 'conventional' heat have different effects, were conducted by La Cara et al. (*1999) on a thermophile bacterium, in which the radiation with a 10.4 GHz field led to an irreversible inactivation of the thermostable enzyme ß-galactosidase, whilst heating in a water bath had no effect. This result confirmed the results of Saffer & Profenno (*1992) which had worked with frequencies in the lower GHz range.

5 Patho-Physiological Effects

5.1 Immune System

The immune system plays a central role in the protection against infectious micro-organisms in the environment and, also, against several kinds of cancer cells. Experiments on hamsters, mice and rats found, amongst other things, that there was a reduction in the activity of natural killer cells and an increase in macrophage activity (see e.g. Yang et al. 1983; Ramo Rao et al. 1983; Smialowicz et al. 1983). However, the majority of experiments on living animals were carried out at power flux density levels that produced an increase in body temperature of more than 1°C. On the other hand, it was observed in parallel *in vitro* experiments, that *in vitro* heating of macrophages did indeed lead to increased activity; the effect was, however, weaker than that of the *in vivo* radiation which produced the same temperature (Ramo Rao et al. 1983).

Elekes et al. (*1996) observed that, after exposing mice for a period of 3 hours per day over several days using microwaves (2.45 GHz) with a power flux density of 1W/m² (SAR = 0.14 W/kg), there was an increase in antibody-producing cells in the spleen of about 37% with continuous radiation and around 55% with amplitude-modulated radiation.

In contrast to the *in vivo* experiments, numerous *in vitro* experiments were carried out with intensities at which an effect due to warming can be excluded. Thus, Lyle et al. (*1983) observed an inhibition of cytotoxicity of T-Lymphocytes in the mouse with a 450 MHz field that was amplitude modulated with various frequencies in the range between 3Hz to 100 Hz. The effect that was demonstrated with a relatively low power flux density of 15 W/m² was greatest at the 60 Hz modulation. The inhibition of cytotoxic effectiveness of the irradiated lymphocytes declined continually for both the lower and higher modulation frequencies.

The tables in Appendix A list further experiments with (human) leucocytes in which damaging effects were proven at non-thermal power flux density levels, especially also with low frequency amplitude modulated fields.

The work of Maes et al.(*1995) deserves special consideration. In an *in vitro* experiment with human leucocytes at a GSM base station and also in the examination of the lymphocytes in the blood of workers who were exposed to the fields of the mobile phone base stations during maintenance work, they found that there was an increase in chromosome damage (chromatid breakage, acentric fragments and some chromosome breaks).

5.2 Central Nervous System

5.2.1 Blood Brain Barrier

The brain of mammals is protected from potentially dangerous materials in the blood by the blood brain barrier, a specialized neurovascular complex. The blood brain barrier

functions as a selective hydrophobic filter that can only be easily passed through by small fat-soluble molecules. Other non fat-soluble molecules, e.g. glucose, can pass through the filter with the help of carrier proteins that have a high affinity for specific molecules.

It is known that a large number of disorders of the central nervous system are caused by disturbances of the barrier function of the blood brain barrier (*Salford et al. 1994).

Severe warming of the brain can lead to an increased permeability of the blood-brain barrier for those materials whose passage should actually be prevented. The results of first experiments with high frequency fields of high intensity, which led to a higher permeability of the blood brain barrier, were then interpreted as a consequence of warming by the HF radiation.

However, Appendix Table B.1 lists a whole series of studies in which a greatly increased permeability of the blood brain barrier was produced through pulsed high frequency fields of very low intensity (*Oscar & Hawkins 1977, *Neubauer et al. 1990, *Salford et al.1994, *Fritze et al.1997) amongst others with carrier frequencies and modulation frequencies which corresponded to those of mobile telephony (GSM).

5.2.2 Neurotransmitters

Pulsed and continuous high frequency fields of low intensity may lead to chemical changes in the brain. Inaba et al. (*1992) exposed rats to a continuous 2.45 GHz field with a power flux density of between 50 to 100 W/m2 and found a significant reduction in the Noradrenalin content of the Hypothalamus, whilst the two other neurotransmitters Dihydroxyphenylacetic acid and 5-Hydroxyindolacetic acid were found in the pons and medulla oblongata in significantly increased concentrations. The radiation did not produce significant changes in the dopamine or serotonin concentrations.

Lai et al. (*1987, 1989 a, b, see above Lai et al. 1988) found also in experiments using rats that a 2.45 GHz field modulated with 500 Hz pulse-modulation influences brain activity, especially in the frontal cortex and the hippocampus, via the most important parasympathetic neurotransmitter acetylcholine. It could be demonstrated that the effect was related to the exposure duration. A 45 minute exposure duration led to significant reductions in choline-uptake, the reduction to 20 minutes exposure produced a significant increase. A similar behaviour was found in animals also as a reaction to stress through the reduction of the freedom of movement and through acoustic white noise.

5.2.3 Electroencephalogram (EEG)

In contrast to the neuroendocrine effects, which can barely be measured directly in the brain of humans, EEG studies can be carried out relatively easily. Several valid studies of that kind do now exist.

Most animal experiments have limited validity, since they were carried out with relatively high power flux density values (see e.g. Chizhenkova 1988: 2.397 MHz, cw, 400 W/m^2, Chizhenkova & Safroshkina 1996: 799 MHz, cw, 400 W/m^2, Thuroczy et al. 1994; 2.45 GHz, AM 16 Hz, 100 W/m^2).

One of the few exceptions are the studies by Vorobyov et al. (*1997), who observed an increase on the left-right symmetry in the EEG in rats that were exposed to a 945 MHz field (AM, 4Hz, 1 to 2 W/m^2, within the first 20 seconds after the start of the exposure.

Early experiments by von Klitzing (1995) with EEG recording during the exposure of subjects to pulsed high frequency fields, that were similar to those of mobile telephone fields (150 MHz, 217 Hz, power flux density in the pulse in the brain at a 6 cm depth below 10^{-2} W/m^2), found changes in the awake EEG, these were called into question because of insufficient documentation.

In later experiments however, a clear effect was demonstrated in the awake and sleeping EEGs.

Reiser et al. (*1995) observed, both with exposures to a 150 MHz field (modulated frequency 9.6 Hz, peak power 0.5 mW, 4 cm distance, near-field conditions) and also in the field of a mobile telephone (902 MHz, modulation frequency 217 Hz, peak power 8W, 40 cm distance), a significant increase in the energy in the EEG frequency bands - Alpha, Beta 1 and Beta 2.

Experiments by Röschke & Mann (*1997) resulted in no significant difference in the EEGs for exposed and sham-exposed subjects under short exposure conditions (3.5 minutes, 900 MHz, GSM, 0.5 W/m2). However, the peak of approx. 9Hz in the presented averaged power density spectra of exposed subjects was clearly lower and narrower than for non-exposed subjects. The same authors (*Mann & Röschke 1996) demonstrated again in the field of a GSM mobile telephone (8W, distance 40 cm power flux density 0.5 W/m^2), a reduction of the time taken to fall asleep and a statistically significant reduction of the duration and the proportion of the REM sleep. Furthermore, the spectral analysis revealed an increased power density of the EEG signal during REM sleep above all in the 'Alpha' frequency band. The REM suppressive effect and the reduction of the time taken to fall asleep were also confirmed by the same research team (*Mann et al.1997, *Wagner et al. 1998). The study carried out in 1997 also found a significant increase in the cortisol concentration in the blood of humans exposed to a 900 MHz/217 Hz field with a power flux density value of 0.2 W/m^2. Systematic deviations were also observed for the Growth Hormone and Melatonin levels, but these did not reach significance level.

Whilst in the previously cited studies, changes in the sleep EEG could be demonstrated only as a consequence of the influence of mobile telecommunications fields for several hours, Borbély et al. (1999) were able to demonstrate that changes in sleep were already occurring after 15 to 30 minutes exposure. This research team used also a 900 MHz field, which could be selectively pulse-modulated with either 2, 8, 217 or 1736 Hz. As in the other experiments, a statistically significant reduction in the proportion of REM sleep was found at a Specific Absorption Rate of less than 1W/kg. In addition, the waking-up phase was noticeably reduced.

Freude et al. (*1998, see also Henschel et al. 1999) examined the effect of the radiation from mobile telephones on slow brain potentials.

Slow brain potentials are event-correlated brain potentials that arise during the preparation for motor action and/or information processing. Changes in the slow brain

potentials give an indication about the influences on specific aspects of human information processing. Freude et al. found that the fields of a mobile telephone (916.2 MHz, 217 Hz, SAR 0.882-1.42 W/kg, exposure time 3 to 5 minutes) led to a statistically significant decrease of the slow readiness potentials for specific tasks, in specific brain areas.

5.2.4 Cognitive Functions

Impairments of the brain, e.g. by modification of the choline-uptake, can be expected to cause learning deficits. These were demonstrated in many learning experiments, in which rats were previously exposed to pulsed microwave fields (*Lai et al. 1989, 1994; *Wong & Lai 2000, see above D'Andrea 1999 for older studies). In the study by Lai et al. (*1994), rats were exposed for 45 minutes to a 500 Hz pulsed 2.45 GHz field with a power flux density of 10 W/m^2. This intensity resulted in a mean whole body SAR of 0.6 W/kg. Following the exposure, the starved rats were placed in a labyrinth with several arms in which food was placed. The researchers measured how effectively the 'exposed rats' and the 'sham-exposed rats' searched the labyrinth for food. For the 'exposed' group, significantly more failed attempts were observed, i.e. searching already emptied labyrinth arms. The authors attributed the low performance of the 'exposed' rats to deficits in spatial memory. The 'handicap' of the EMF exposure could be levelled out in a follow-up experiment, in which the rats were given either the acetylcholine agonist Physostigmin or the opiate antagonist Naltrexone before their exposure. According to the authors, these findings are confirmation of their results from previous studies (see above), in which they had found that high frequency electromagnetic fields influence cholinergic and endogenous opioid neurotransmitter systems in the brain and that this effect can lead to memory deficits. In the meantime, the effect has been confirmed by other experiments (Mickley & Cobb 1998).

In a further experiment (*Wang & Lai 2000), rats were trained over several sessions to find a platform situated just under the water surface inside a round water basin. Subsequently, they were exposed to pulsed microwave radiation for an hour (2.45 GHz, 500 pulses per second, mean power flux density 2W/m^2, mean whole-body SAR 1.2 W/kg). Testing was then carried out to determine how long the 'exposed rats' needed to find the platform from different starting positions, compared to the 'non-exposed rats' or 'sham-exposed rats'. The 'exposed rats' clearly required longer for this, as they spent significantly less time in the correct quadrant of the water basin. Finally, the recorded traces of the swimming lanes used by the 'exposed animals' differed from those of the control groups, this suggests that different strategies were used when searching for the platform. This result confirms the findings from other studies that pulsed high frequency fields can influence specific aspects of memory performance.

The effects of a 600 MHz field on the memory of rats were also demonstrated by Mickley et al. (*1994). In this experiment, the capacity of the animals to recognize familiar objects was measured in relation to the radiation they received. Whilst the 'non-exposed control animals and also the animals who were exposed to a SAR of 0.1 W/kg occupied themselves for longer with a novel object compared to a familiar object, the higher exposed animals spent just as much time examining an actually familiar object as with a

novel object. The limit for this exposure dependent change in behaviour was between 0.1 and 1.0 W/kg

The lowest SAR so far which has been shown to have an effect on cognitive functioning in rats was 0.072 W/kg. However, in this experiment, pulses with a peak of more than 700 MW (megawatts) were used (Raslear et al. 1993). The low SAR in this case resulted only from averaging over time with a very low pulse repetition rate of 0.125 pulses per second and a pulse width of only 80 nsec.

It has been shown in experiments by Preece et al. (*1999) that fields like those used in mobile telephony can influence cognitive functions of the brain. In this study, 36 subjects were subjected to a 915 MHz field of a simulated mobile telephone. The field was overlaid either with a 217 Hz sinusoidal modulation or a 217 Hz pulse modulation. In the analogue simulation the net forward power was about one Watt, and in the digital simulation it was 0.125 Watt. Under the conditions 'Exposure to analogue field', 'Exposure to digital field' or 'Sham exposure without any field', each of the test persons had to carry out several tests to measure ability to react and various tests of memory performance. In both exposed groups there was a slight but statistically significant decrease in reaction time, which was more marked for 'Analogue exposure' than for 'Digital exposure'.

5.3 Hormone Systems

5.3.1 Stress Hormones

Environmental pollution can act as a stressor on the body, like physical and mental stressors, and cause 'alarm reactions'. Such reactions are associated with hormonal changes. The presence of a stress-situation can be proved by the presence of hormones like adrenocorticotropin [the adrenocorticotrophic hormone] (ACTH), cortisol and corticosterone in the blood, and also to a lesser extent by changes in the concentration of prolactin and growth hormone.

Electromagnetic fields can clearly cause stress reactions in animals used for experiments. Thus, the experiment by Imaida et al. (*1998a) on rats that were exposed for a duration of 90 minutes daily over a period of 6 weeks to a field with a carrier frequency of 929.9MHz and a 50 Hz pulse modulation, showed a statistically significant increase in the ACTH and corticosterone levels. The whole-body SAR value in this experiment was between 0.58 and 0.8 W/kg. The exposure in the 1.439 GHz field, equally with a 50 Hz pulse modulation and a SAR value between 0.453 and 0.680 W/kg had the same effect (*Imaida et al. 1998b).

Chou et al.(*1992) exposed rats in a long-term experiment (25 months) to 800 MHz pulse-modulated 2.45 GHz field that led to a Specific Absorption Rate of 0.15 to 0.4 W/kg. Alongside other physiological parameters the corticosterone profile was regularly measured for the first half year of the experiment. Whilst the hormone profile of the exposed animals and the non-exposed animals were practically identical in the later stages of the experiment, with the exception of a slight increase in the sham-exposed group of animals in the third phase of the experiment, the first examination after 6 week's exposure showed a statistically significant increase in the corticosterone profile in the blood of the exposed animals.

The authors report that their attempt to replicate this effect produced no statistically significant results, however, only 20 animals were tested in this second experiment whilst the actual series of experiments contained 200 animals.

A similarly extensive experiment on rats like that of Chou et al. However, with an unmodulated 435 MHz field showed no difference in the concentration of the hormones ACTH, corticosterone and prolactin between the exposed animals and the non-exposed animals (Toler et al. 1988).

The few experiments previously carried out on humans do not yet produce a clear picture. Mann et al. (*1998) exposed 24 volunteer subjects whilst asleep to the field of a mobile telephone that was transmitted from a separate antenna (900 MHz, 217 Hz, 0.2 W/ m^2). Blood samples were withdrawn via a catheter whilst the subjects were asleep and they were analysed for, amongst other things, cortisol and growth hormone concentrations. There were systematic differences between the 'exposed subjects' and the 'sham-exposed subjects' during the course of the night for both hormones, which only reached statistical significance levels for cortisol.

De Seze et al. (*1998) examined the effect of a GSM mobile telephone (900MHz, 217 Hz) on subjects who were exposed to the field for 2 hours per day, 5 days per week for over a month. Based on nine blood sample withdrawals per week; amongst other things, the change in the concentrations of ACTH, growth hormone and prolactin were determined over time.

The authors' evaluation of their studies was that at one month, intermittent exposure in the radio-frequent field from the mobile telephone had no lasting or accumulative effects on the hormone secretions from the anterior lobe of the pituitary gland. In their data, it is however noticeable that that ACTH and prolactin follow a quite similar profile over time: the concentrations started at high initial values at the start of the exposure and then decreased in the following 3 weeks, and they then rose slightly again. The growth hormone concentrations are very high for the first measurements during the exposure period, they then fall to the pre-exposure concentration levels and maintain these levels until the end of the experiment. Possibly, these measurements show a temporary stress reaction, which reduced in the following weeks.

5.3.2 Melatonin

The hormone melatonin, which is produced in the pineal gland, functions as a regulating hormonal signal that synchronizes the endocrine rhythms of all the hormone glands. It regulates, amongst other things, the daily cycles of ACTH and the cortisol-release and thereby regulates the daily rhythms of many metabolic processes.

Melatonin also exerts influences (inhibitory) on sex hormones and it has a stimulatory effect on the immune system. Melatonin also influences specific cancer illnesses via the regulation of the release of the sex hormones. In addition, melatonin is a free radical scavenger, inactivating radicals such as OH, which amongst other things can be dangerous for the genetic material. Furthermore, during *in vivo* experiments, it was demonstrated that melatonin hinders changes in DNA produced by chemical carcinogens

and it protects lymphocytes from chromosome damage in high frequency electromagnetic fields (*Lai & Singh 1997).

In the previously described experiments carried out by Imaida et al.(*1998 a, b), it was found that the experimental animals that were exposed to a pulse-modulated high frequency field had a reduced melatonin concentrations in the blood. This finding could not be confirmed by Heikkinen et al. (1999), who exposed mice for 17 months to a 900 MHz field with a 217 Hz GSM pulse modulation (SAR: 0.35 to 1.5 W/kg). Studies by Vollrath et al. (1997) using rats and hamsters with a 900 MHz field (217 Hz GSM, SAR: 0.04 to 0.36 W/kg) could not contribute much to the clarification of the problem, since in several sub-sets of the experiment statistically significant differences between 'exposed animals' and 'non-exposed animals' had been found, but according to the authors these resulted from mistakes in the experimental order.

In experiments by Mann et al. (*1997 see above), the stress hormones were measured as well as the serum melatonin profile. This showed, in the case of the exposed humans, that for a period of between 3 to 4 hours in the middle of the night there was an increase compared to the control values, but these were not statistically significant according to the evaluation of the authors.

6 Pathological Effects

6.1 Results of Experimental Studies

6.1.1 Cancer

Carcinogenesis

Carcinogenesis is a multi-layered process, at the beginning of which is a certain impact on the level of the genetic material. This can be a direct impact (for example ionising radiation) or an indirect action via the product of a reaction (for example OH radicals). A direct or indirect interaction with DNA can lead to damage of the DNA or the chromatin structures (see also Chapter 3). If those damages are not repaired by endogenous processes, the damage will be permanent. Thus, the initiated cell can, if the immunological control fails, under the influence of hormones and promoters develop into a pre-neoplastic focus, which can then lead to a malignant tumor. The different steps of carcinogenesis are summarised in three phases:

- Initiation: Triggering of damage on the DNA and mutations on critical genes
- Promotion: Increased rate of DNA synthesis and proliferation of transformed cells
- Progression: Transition of a pre-neoplastic focus to a malignant tumor

A physical or chemical pollutant can in principle be effective in all three phases of carcinogenesis.

- Initiation: Triggering of direct DNA damage or of a substance which causes DNA damage, disruption of repair processes of the DNA
- Promotion: Promotion of the proliferation of transformed cells
- Progression: Suppression of immune-reactions and promotion of tumor growth

Results from Animal Experiments

In vivo experiments using animals with an inbred genetic predisposition for certain tumor illnesses or in which animals were injected with cancer cells, yielded very different results (see Appendix C, Table C.1). In the majority of the studies, no cancer promoting effect of high frequency electromagnetic fields could be found, or effects were only observed under certain conditions of exposure (marked in the Table with 'partly'), and even in those cases they were often not statistically significant. However, it needs to be noted that many studies with negative results had very short exposure times and durations of the study itself (for example Chagnaud et al. 1999: 2 weeks, Salford et al. 1993: 2 to 3 weeks) and hence they do not have much relevance to answer the question whether high frequency electromagnetic fields have carcinogenic potential.

Some long-term studies have yielded results which indicate a carcinogenic or co-carcinogenic effect of electromagnetic fields with mobile telecommunications frequencies

if the animals are exposed over a long period of time. (*Repacholi et al. 1997, *Szmigielski et al. 1982 and *Szudinski et al. 1983). Important in this context is also the study of Chou et al. (*1992). This study did not find a statistically significant rise in tumors in a particular organ. However, the exposed group developed not only a higher number of tumors in total, but also the number of primary malignant and metastatic malignant neoplasms was significantly higher in the exposed animals. In their discussion of the results, the authors point to the fact that the number of the primary malignant neoplasms in the exposed group compared to the control group is four times higher and that this finding is statistically significant, but then go on to undermine their finding by quoting literature, according to which the tumor incidence of the exposed group should still be within the normal range.

The experiment of Toler et al. (*1997) using animals with a predisposition for chest tumors did not result in a higher incidence of these, but the number of ovarian tumors was significantly higher in the exposed group compared to the controls.

The intensities at which an increase in tumors was found in animals were one to two powers of ten below the values at which one would expect a triggering of 'thermal' effects. According to the presenting results, low frequency modulation does not seem to be responsible for the carcinogenic effect.

6.1.2 Infertility and Teratogenic Effects

Teratogenesis

Teratogenic effects of a pollutant can – as with the carcinogenic effect – either be caused by the triggering of a genetic defect or a harmful impact on the foetal development. The formation of a genetic malformation during its initiation phase is analogous to carcinogenesis, i.e. teratogenic effects are also caused by direct or indirect impact on the DNA and disruptions of the endogenous repair mechanisms. Later damages of the foetus can either be caused by direct effects of the pollutant on the foetus or by reactions to the pollutant within the mother's organism, which would then be passed on to the foetus.

Results from Animal Experiments

A multitude of studies have demonstrated that high body temperatures in mammals lead to a spermatotoxic and teratogenic effect. Since many studies examining such effects from high frequency electromagnetic fields worked with intensities that were capable of significantly raising body temperature, it cannot be excluded that the observed spermatotoxic and teratogenic effects were caused by a thermal effect, (see for example Berman et al. 1982, 1983, Berman & Carter 1984, Jensh et al. 1983a,b, Kowalczuk et al. 1983, Lary et al. 1983, Nawrat et al. 1985, Saunders et al. 1981, 1983, for the results of older studies, see O'Connor 1980). The results of these studies do not always appear consistent, however, this can possibly be explained by a different thermal susceptibility of the different animal species used. In rats for example, a loss of thermally damaged embryos is often observed, whilst the birth of malformed animals is rare. Other mammals show a wider bandwidth between teratogenic and lethal exposures. (Verschaeve & Maes 1998).

However, there are some indications in the literature for teratogenic effects at intensities that cause no (or, if at all very small) rises in temperature. Magras & Xenos (1997) exposed mice during six months to a real transmitter. The mice had offspring five times during this period and a continuous decrease in offspring was found down to irreversible infertility. The exposure consisted of several radio and TV transmitters in the VHF and UHF bands and measured between 0.00168 and 0.01053 W/m^2. A repetition of this study would be desirable in order to exclude that the effect was due to problems with the maintenance of the animals or the screening of the control group.

Khillare and Behari (*1998) found that male rats that had been exposed to a 200 MHz field (power flux density:14.7W/m^2, SAR:1.65 to 2.0W/kg) during a period of 35 days for six days per week and two hours per exposure day and which were afterwards mated with unexposed females, produced significantly less offspring that the males in the unexposed control group.

In an experiment by Akdag et al. (1999) male rats were exposed one hour every day to a 9.45 GHz field (power flux density:2.5W/m^2, SAR:1.8 W/kg) during different periods of 13, 26, 39 or 52 days corresponding to one, two, three and four cycles of the seminal epithelium.

At the end of each exposure period the following data were measured and compared to an unexposed control group: number of sperm in the epididymides, morphology of the sperm and weight of the testicles, epididymides, seminal vesicles and prostate.

They found amongst other effects a decrease in the number of sperm (statistically significant in the group exposed for 53 days) and an increase of abnormal sperm (statistically significant in the groups exposed for 26, 39 and 52 days).

A co-teratogenic effect under non-thermal exposures with power flux densities of 10 to 100 W/m^2 in combination with cytosine arabinoside (CA) was found in a study by Marcickiewicz et al. (*1986). In the experiment, mice were exposed in utero for two hours a day to 2.45 GHz from the first to the 18th day of the pregnancy. The field, which alone was not teratogenic, significantly increased the teratogenic effect of CA. A direct teratogenic effect of microwave radiation with a frequency of 2.45 GHz on the brains of newborn rats was found by Inalösz et al. (*1997). However the authors declared that the SAR of 2.3W/kg led to a rise of rectal temperature of 1.0ºC.

6.2 Results of Epidemiological Studies

Methodological Requirements

In principle, epidemiological studies are an effective instrument to prove potential health risks of a pollutant under real environmental and exposure conditions. Usually, they are carried out by comparing statistical data about the incidence of an illness in an exposed population as opposed to the incidence of this illness in an unexposed population. The exact classification of exposure would require the metrological recording of the pollutant for all participants (exposed and unexposed) during the entire latency period of the illness. This is often not practicable and for long latency periods, which can usually only be addressed via retrospective studies, inherently impossible. Under such circumstances it

has to suffice that surrogates are used, for example having a profession which is linked to a certain exposure or the proximity of the home to an emitting installation. In some cases, if the emitting installations have been used for a long time in the same mode, it is possible to extrapolate past exposures from current measurements.

The quality of the exposure classification determines the validity of an epidemiological study. Possible weaknesses, which can lead to wrong results, are:

- People are classified as 'exposed' or 'strongly exposed' although in fact there is no or only little exposure. An example with regards to high frequency fields is the often-used exposure classification on the basis of professional categories, such as radar operators or telecommunications engineers, for whom it cannot be excluded that the main occupation is a desk job without exposure.

- It is assumed that the control group is completely unexposed, although the pollutant is actually ubiquitous, which will lead to smaller but still potentially significant exposures in the control group. One known example are mains frequency magnetic fields, which affect the immediate neighbours of power supply equipment, but still exist at non-negligible strengths in houses which are further away from such equipment.

Both effects lead to a levelling out between the exposed and unexposed group and hence to an underestimation of the real health risk posed by the pollutant in question.

Another weakness of epidemiological studies can be the presence of unrecognized confounders, i.e. other influences, which also affect the groups studied and influence the development of the illness. This can be environmental factors, such as exposures to other pollutants, but also socio-economic and behavioural factors. If not all potentially relevant confounders are factored in, the results can be distorted, either towards an overestimation or an underestimation of the real risk.

The fast development of mobile technology has lead to a double dilemma with regards to the study of potential risks through epidemiological studies:

- For illnesses like cancer with latency periods of many years it is still too early to expect valid results. If mobile telecommunications are indeed linked to a higher incidence of cancer, the illness will only have manifested in a few people so far. This should at least be valid for the part of the population whose exposures are from base stations only. Potentially it could be different for direct mobile phone users, since these are generally exposed to significantly higher intensities. But also for this group, at this moment in time, we would expect results from epidemiological studies to underestimate the real risk.

- In some years epidemiological studies will hit a different obstacle: once base stations cover the entire country and a large proportion of the population use a mobile phone, it will become difficult to find the necessary unexposed control groups.

Given this dilemma, epidemiological studies carried out in the past have a certain validity, even if the exposures are not exactly the same as they would be today and the studies do not always correspond to today's quality standards.

The Selection of Studies

At the time of finishing this present report there were only two epidemiological studies of health risks in relation to actual existing mobile telecommunications exposures (*Rothman at al. 1996, *Hardell et al. 1999). However there are a much larger number of studies available, in which the health effects of high frequency electromagnetic fields in humans were examined (see also Appendix D, Table D.1). Just under a quarter of all results relate to exposures with low frequency pulse or amplitude modulated high frequency fields, such as they are used for mobile telecommunications, even if the carrier and modulation frequencies are in most cases not identical with those of mobile telecommunications.

In Appendix Table D.1, the examined illnesses are listed with their evaluated end point (incidence or mortality), data describing the exposure situation is given and the quality of the exposure classification is assessed. Finally, the result of the study is evaluated as 'Relative Risk' (RR) which includes the relevant risk factors in the form of standardised mortality rates, standardised morbidity rates and odds ratios, and the statistical significance is assessed. For each study we list the value for the highest exposure class or if there was a further differentiation of the examined groups, for example according to occupational groups, the highest found value.

Values are considered statistically significant (s.s.) if the value RR=1 outside of the 95% confidence interval or if $p<0.05$.

A statistical evaluation of the results presented in Table D.1 can be found in Table 6.1. Here, we list for every illness how many studies or separate results are available, how many of these show a relative risk RR >1 and how many are statistically significant.

Almost all the studies, in which the total cancer risk without any differentiation according to tumor form were examined, showed a risk factor of RR>1. Half of the studies resulted in statistically significant risk factors with a maximum value of 2.1, which corresponds to a doubling of the statistical risk to develop cancer from exposure to high frequency electromagnetic fields.

A similar picture was found in relation to tumors of the nervous system, especially brain tumors. Here, the maximum value for relative risk found was 3.4. Eleven of the total of 15 studies yielded a positive result, more than half of which were statistically significant.

The incidence of breast cancer in relation to high frequency fields must be examined separately for men and women. All three studies relating to the breast cancer incidence in women yielded risk factors greater than 1, the statistically significant values were 1.15 and 1.5. For men, risk factors of up to 2.9 were found; however, not all were statistically significant.

Of the total of 16 results for leukaemia without further differentiation of the illness, 13 were positive (RR>1), more than half of these results were statistically significant. The highest statistically significant value for the relative risk was 2.85. Amongst the results of the differentiated studies, the following are notable: lymphatic leukaemia (7 results, 5 positive, 4 statistically significant, RR maximum value: 2.74) and acute myeloic leukaemia (4 different studies, 3 positive results, 2 statistically significant, maximum RR value: 2.89).

With regards to the correlation of high frequency electromagnetic fields from radar and other sources and testicular cancer, three studies have been conducted. All lead to statistically significant risk factors with a maximum value of 6.9.

The studies regarding cardio-vascular diseases did not result in a clear picture, not least because of the multitude of the symptoms examined.

All four studies of fertility problems in relation to the exposure of men to microwaves indicate increased risk. In two studies statistically significant risk factors of up to 2.7 were found.

With regards to irregular courses of pregnancies and malformations in children of mothers which had been exposed to high frequency fields, there are a large number of studies with positive results, of which only two fit into the frequency range relevant to our report. Both of these studies found statistically significant positive results with risk factors of up to 2.36.

Of the studies of cancer risk of children whose fathers had been exposed to electro-magnetic fields, only two correspond to the quality criteria required for inclusion into this report. Both indicate an increased risk, but only one result is statistically significant at a value of RR=2.3. (With regards to the cancer risk of children in correlation to the exposure of their parents, see also Colt & Blair 1998).

Regarding the disruption of motor functions as well as psychological functions and well-being, there is only one valid study for the frequency bands relevant to this report, which yielded a slightly increased risk factor. However since other studies of transmitters with frequencies below 100 MHz resulted in serious indications of increased risk, indicating that this problem should be given more attention in the future, we also included the study of Zhao et al. (1994), although it didn't meet our quality standards with regards to the statistical evaluation.

Unfortunately, the majority of the studies do not state the actual strength of the exposures. Measurements are only available for the radio and television transmitter used for the studies of Hocking et al. (1996) and McKenzie et al. (1998). The mean power flux densities for all 16 municipalities affected by this transmitter were $3.3\ 10^{-3} W/m^2$ within the range from $2.6\ 10^{-4}$ to $1.46\ 10^{-2} W/m^2$ (McKenzie et al. 1998). The ICNIRP guidelines for the general population recommend a maximum value of 2 to 2.51 W/m^2 for the range of frequencies emitted by this transmitter (64.25 to 527.25MHz). This means that the exposures in these studies were below the German guidelines by a factor of 10^{-4}.

Table 6.1
Overview over the results of epidemiological studies with regards to the health risks of high frequency electromagnetic exposures (see also Appendix D, Table D.1)

Illness	Number of studies (results)	Studies (results) with RR>1	Statistically significant results
All illnesses	2	0	0
Cancer, unspecified	6 (7)	5 (6)	3
Brain tumours unspecified and tumours of the nervous system unspecified	14 (21)	10 (15)	6 (7)
Cancer (eyes)	1	1	1
Cancer of the respiratory organs, lung cancer	5	2	1
Chest cancer, men	2	2	0
Breast Cancer, women	3	3	2
Cancer of the lymphatic and blood forming system unspecified	4	4	1
Leukaemia unspecified	12 (16)	9 (13)	5 (7)
Acute leukaemia unspecified	4	4	0
Lymphatic leukaemia unspecified	4 (7)	2 (5)	1 (4)
Acute lymphatic leukaemia	2	2	0
Chronic lymphatic leukaemia	4	4	1
Leukaemia, non lymph. non-myelo	1 (4)	1 (4)	1 (2)
Lymphoma, Hodgkin-Syndrome	5 (7)	3 (4)	1
Testicular cancer	3 (5)	3 (5)	3 (4)
Uterine cancer	1	1	1
Skin cancer	4	3	1
Cardio-vascular diseases	4 (5)	3 (4)	1
Infertility, reduced fertility, men	4 (7)	4 (7)	2 (4)
Infertility, reduced fertility, women	1	1	0
Miscarriages, stillbirths, malformations and other birth defects	2 (3)	2 (3)	2
Cancer, offspring (parental exposure)	2	2	1
Neurodegenerative diseases, Alzheimer's	1	1	0
Disruptions of motor and psychological functions and well-being	2 (9)	2 (9)	1 (7)

7 Health Risks to Humans Resulting from Exposure to the Electromagnetic Fields of Mobile Telecommunications

The triggering of an illness caused by an (environmental) pollutant and the development of this illness are a multi-phased process, which begins with a biological, biochemical or biophysical primary interaction of the pollutant with the biological system and ends with the manifestation of the illness. During the different phases of the process, the body's own repair mechanisms can intervene and impede the further development of the illness. An assessment of the potential health risks of electromagnetic fields as they are used for mobile telecommunications should therefore be mainly based on studies conducted directly on humans, because extrapolations from animal studies or even *in vitro* studies on cell cultures only have limited validity for effects in humans, due to the difference in susceptibilities and the lack of organic interactions in cell cultures. However, due to the ethical limits to the research on humans, it is unavoidable to use results from experiments with animals, single organs or cells in order to discover the biological and physiological mechanisms.

Cancer

Given the results of the present epidemiological studies, it can be concluded that electromagnetic fields with frequencies in the mobile telecommunications range do play a role in the development of cancer. This is particularly notable for tumours of the central nervous system, for which there is only the one epidemiological study so far, examining the actual use of mobile phones. The most striking result of this study was an obvious correlation between the side at which the phone was used and the side at which the tumour occurred. The brain tumour incidence however was only slightly increased. A (hypothetical) explanation of such a finding could for example be that mobile fields have a promoting effect on previously initiated (multiple) tumours, triggering a defence mechanism in the body which is capable of suppressing unpromoted tumours.

Higher risks were also demonstrated for several forms of leukaemia.

Although the studies in relation to testicular cancer were examining particular exposure conditions (emitting equipment worn partly on the body at hip level), given the high risk factor found, a possible risk cannot be excluded, especially not for mobile users wearing the devices in standby mode on their belts. The epidemiological findings for testicular cancer also need to be interpreted in conjunction with the results of the studies of fertility problems occurring in relation to high frequency electromagnetic fields.

The risk factors for cancers other than testicular cancer are only moderately increased, but not negligible, considering this technology will potentially reach full coverage of the entire population.

Reliable conclusions about a possible dose-response-relationship cannot be made on the basis of the present results of epidemiological studies, but an increase of cancer risk cannot be excluded even at power flux densities as low as 0.1 W/m².

In long-term animal experiments, the carcinogenic effect of pulse modulated high frequency fields was demonstrated for power flux densities of circa 3W/m² (mouse, exposure duration 18 months, 30 minutes per day, SAR (mouse) circa 0.01 W/kg).

On the cellular level, a multitude of studies found the type of damage from high frequency electromagnetic fields which is important for cancer initiation and cancer promotion:

Direct damage on DNA as well as influences on DNA synthesis and DNA repair mechanisms were demonstrated in *in vivo* and *in vitro* experiments for continuous and pulsed fields at power flux densities from 10W/m² and 9W/m² respectively.

Chromosome aberrations and micronuclei occurred at power flux densities from 5 W/m².

Neoplastic cell transformation and an enhanced cell proliferation were demonstrated for Specific Absorption Rates of below 0.5W/kg, and individual studies demonstrated that the obvious disturbance of the communication between cells, which is a prerequisite for the uninhibited proliferation of cells that is characteristic for cancer development, occurs at just a few W/m².

Conclusion:

The results of the studies for all stages of cancer development from the damage of the genetic material via the uninhibited proliferation of cells and debilitation of the immune system (see below) up to the manifestation of the illness prove effects at power flux densities of less than 1 W/m². For some stages of cancer development, intensities of 0.1 W/m² or even less may suffice to trigger effects.

Debilitation of the Immune System

Damaging effects on the immune system which can aid the development of illnesses were demonstrated in animal experiments at power flux densities of 1 W/m² (mouse, exposure duration 6 days, 3 hours per day, SAR (mouse) 0.14W/kg). In *in vitro* experiments on lymphocytes, defects of the genetic material were demonstrated at power flux densities of circa 10 W/m². The presence of stress hormones, which when permanent can debilitate the immune system, was found to be increased in human experiments from power flux densities of 0.2W/m². In animal experiments (rat) a similar effect was observed at a Specific Absorption Rate of circa 0.2 W/kg.

Conclusion:

Experiments on animals prove harmful effects on the immune system from circa 1 W/m²; at power flux densities of 0.2 W/m² higher secretions of stress hormones in humans have been demonstrated.

Influences on the Central Nervous System and Cognitive Function

The effects of pulsed and continuous high frequency fields on the blood-brain-barrier and the activity of neurotransmitters were demonstrated in animal experiments for power flux densities of 3 and 10 W/m^2 respectively.

In humans, influences on the slow brain potentials were found at SAR values of 0.882 to 1.42W/kg, i.e. well below the current guidelines for partial body exposure of 2 W/kg.

Changes in the sleep EEG of humans, which showed a shortening of the REM sleep phase occurred at intensities as low as 0.5 W/m^2.

In animal experiments, changes in the EEG were demonstrated at power flux densities of 1 to 2W/m^2.

Impairment of cognitive functions was found in animal experiments at power flux densities of 2W/m^2. In humans, there are indications that brain functions are influenced by fields such as they occur when using a mobile telephone.

An epidemiological study of children who had been exposed to pulsed high frequency fields, found a decrease in the capability to concentrate and an increase in reaction times.

Conclusion:

Effects of high frequency electromagnetic fields on the central nervous system are proven for intensities well below the current guidelines. Measurable physiological changes have been demonstrated for intensities from 0.5 W/m^2. Impairments of cognitive functions are proven for animals from 2W/m^2.

Electrosensitivity or Electromagnetic Hypersensitivity

The terms 'electrosensitivity' or 'electromagnetic hypersensitivity' describe disturbances of well-being and impairments of health, such as they are suffered by certain sensitive people when working with or being in the presence of devices and equipment emitting electrical, magnetic or electromagnetic fields. The sensitivity manifests in a variety of symptoms including:

- nervous symptoms such as sleep disturbances, headaches, exhaustion, lack of concentration, irritability, anxiety, stress
- cardio-vascular complaints
- disruptions of hormones and metabolism
- skin complaints

The composition and strength of the complaints varies enormously in different individuals. The correlation of the complaints with electromagnetic exposures and other environmental influences seems to vary strongly not only between affected persons but also in time, a fact that has so far impeded the conclusive scientific proof of a cause-effect-relationship in provocation studies. The present results of scientific studies are often not conclusive and partly contradictory. On the other hand, however, there is a wealth of data

collected by the self-help organisations of affected people, which has not yet been explored.

Conclusion:

On the basis of current knowledge it is impossible to estimate the risk of electrosensitive reactions or to make recommendations for guidelines designed to avoid such a risk for the general population, which is composed of sensitive and non-sensitive persons.

8 Recommendations

8.1 Precautionary Health Protection in Relation to Exposures to Electromagnetic Fields of Mobile Telecommunications

With mobile telecommunications we have to differentiate to exposure situations:

- exposure of residents near base stations
- exposure of mobile users when using the devices

To limit exposure to an acceptable degree, if this is possible at all, there need to be different strategies for the two different exposure groups.

Exposures from Base Stations

In humans, harmful organic effects of high frequency electromagnetic fields as used by mobile telecommunications have been demonstrated for power flux densities from 0.2W/m² (see Chapter 7). Already at values of 0.1 W/m² such effects cannot be excluded. If a security factor of 10 is applied to this value, as it is applied by ICNIRP and appears appropriate given the current knowledge, the precautionary limit should be 0.01W/m². This should be rigorously adhered to by all base stations near sensitive places such as residential areas, schools, nurseries, playgrounds, hospitals and all other places at which humans are present for longer than 4 hours.

We recommend the precautionary limit of 0.01 W/m² independent of the carrier frequency. The rough dependency on frequency with higher limits outside of the resonance range, as it is applied in the concept of SAR, is not justifiable given the results of the scientific studies which conclusively prove non-thermal effects of high frequency fields. Also, the current allowed higher exposures for parts of the body, as long as they refer to the head or thorax are not justifiable.

Exposures of Mobile Phone Users

Given the state of technology now and in the foreseeable future, it is currently technically impossible to apply the recommended maximum value for mobile base stations also to the use of mobile phones. However, a lowering of the guidelines to a maximum of 0.5 W/m² should urgently be considered.

A particular problem in this exposure group is posed by children and adolescents, not only because their organism is still developing and therefore particularly susceptible, but also because many adolescents have come to be the most regular users of mobile phones.

Advertising towards this population group should be banned. Furthermore, particular efforts should be made to lower the exposures during calls. It would be recommendable to conduct (covert) advertising campaigns propagating the use of headsets. It would also be important to develop communications and advertising aiming at minimising the exposures created by carrying mobile phones in standby mode on the body.

8.2 Scientific Studies Regarding the Health Risk of Mobile Telecommunications

The precautionary limits recommended in Chapter 8.1 are based on the current scientific knowledge. This is, however, still incomplete and in the case of this technology, which is exposing the entire population to its emissions, further research efforts are needed to create a base for the setting of truly reliable guidelines. Based on the scientific knowledge presented in this report, the further research requirements are mainly for studies on living organisms (humans or animals):

Epidemiological studies

- studies that metrologically record the exposure on existing radio transmitters (USW), TV transmitters and longer-established radio communications and paging networks. (The emissions of this type of equipment with regards to the modulation frequencies may not be directly comparable to those of mobile telecommunications, but such studies could nevertheless offer important indications for the assessment of the exposure risks of high frequency electromagnetic fields; the studies should focus on cancer and illnesses of the central nervous system including neurodegenerative diseases as well as cardio-vascular diseases and any diseases caused by a disruption of the immune system; such studies should also address potential clusters of unspecified symptoms and impairments of well-being (electrosensitivity)).
- a meta-study with retrospective dosimetry for the studies which examined the residents near emitting base stations (see Appendix D) with the help of measured data from comparable sites
- a cohort study examining the health (see above) of mobile users and residents near mobile base stations
- epidemiological animal studies on pets

Experimental long-term studies

Studies of the chronic effects of the fields emitted by mobile telecommunications

- on the central nervous system (preferably on humans)
- on the immune and endocrine system (preferably on humans, but further animal experiments at low intensities would also be helpful for example with regards to EMF-induced stress)
- on the cardio-vascular system (variability of heartbeat rates, blood pressure, etc., on humans and on animals)

Experimental short-term studies

Studies of the acute effects of the fields emitted by mobile telecommunications

- on the brain in various rest and stress situations (preferably making use of EEG and similar methods)

Beyond these suggestions, it would be important to develop a strategy for the research of the 'electrosensitivity' phenomenon and its incidence, which would acknowledge the failure of traditional scientific methods to address the problem and allow the inclusion of the data available from the self-help groups and associations of the affected.

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Repacholi M.H.	1998	Low-level exposure to radiofrequency electromagnetic fields: health effects and research needs	Bioelectromagnetics	19	1-19
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Salford L.G., Brun A., Persson B.R.R. & Eberhardt J.	1993	Experimental studies of brain tumour development during exposure to continuous and pulsed 915 MHz radiofrequency radiation	Bioelectrochem. Bioenerg.	30	313-318
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Törnqvist S., Knave B., Ahlbom A. & Persson T.	1991	Incidence of leukemia and brain tumors in some 'electrical occupations'	Br. J. Ind. Med.	48	597-603
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Tynes T., Hannevik M., Anderson A., Visnes A.I. & Haldorsen T.	1996	Incidence of breast cancer in Norwegian female radio and telegraph operators	Cancer Causes Contr.	7	197-204
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Verschaeve L.	1995	Can non ionizing radiation induce cancer?	Cancer Journal 8:237	8	237-249
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Vijayalaxmi, Mohan N., Meltz M.L. & Wittler M.A.	1997 b	Proliferation and cytogenetic studies in human blood lymphocytes exposed *in vitro* to 2450-MHz radiofrequency radiation	Int. J. Radiat. Biol.	72	751-757
Vollrath L., Spessert R., Kratzsch T., Keiner M. & Hollmann H.	1997	No short-term effects of high-frequency electromagnetic fields on the mammalian pineal gland	Bioelectromagnetics	18	376-387

Vorobyov V.V., Galchenko A.A., Kukushkin N.I. & Akoev I.G.	1997	Effects of weak microwave fields amplitude modulated at ELF on EEG of symmetric brain areas in rats	Bioelectromagnetics	18	293-298
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Wolke S., Neibig U., Elsner R., Gollnick F. und Meyer R.	1996	Calcium homeostasis of isolated heart muscle cells exposed to pulsed high-frequency electromagnetic fields	Bioelectromagnetics	17	144-153
Wu R.Y., Chiang H., Shao B.J., Li N.G. & Fu Y.D.	1994	Effects of 2.45 GHz microwave radiation and phorbol ester 12-o-tetradecanoylphorbol-13-acetate on dimethylhydrazine-induced colon cancer in mice	Bioelectromagnetics	15	531-538
Yang H.K., Cain C.A., Lockwood J. § Tompkins W.A.F.	1983	Effects of microwave exposure on the hamster immune system. I. Natural killer cell activity	Bioelectromagnetics	4	123-139
Yao K.T.S.	1978	Microwave radiation-induced chromosomal aberrations in corneal epithelium of Chinese hamsters	J. Hered.	69	409-412
Yao K.T.S.	1982	Cytogenetic consequences of microwave irradiation on mammalian cells incubated *in vitro*	J. Hered.	73	133-138
Zhao Z., Zhang S., Zho H., Zhang S., Su J., & Li L.	1994	The effects of radiofrequency (<30 MHz) radiation in humans	Rev. Environ. Health	10	213-215

Appendix A

Studies of the effects of high frequency electromagnetic fields on the cellular level

Abbreviations

neg.	negative finding
n.s.	not statistically significant
pos.	positive finding
s.s.	statistically significant
partly	some findings
#	disagreement with the conclusions of the authors
?	unknown; not provided; unreliable

Table A.1 Genotoxic effects of high frequency electromagnetic fields

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
Direct DNA damage						
2,45 GHz	cw	10 – 20 W/m^2 0,6 – 1,2 W/kg	2 h	Rat, in vivo	pos., s.s.	Lai & Singh 1995
2,45 GHz	PM, 500 Hz	10 – 20 W/m^2 0,6 – 1,2 W/kg	2 h	Rat, in vivo	pos., s.s.	Lai & Singh 1995
2,45 GHz	PM, cw	20 W/m^2 1,2 W/kg	2 h	Rat, in vivo	pos., s.s.	Lai & Singh 1996
2,45 GHz	PM, 500 Hz	20 W/m^2 1,2 W/kg	2 h	Rat, in vivo	pos., s.s.	Lai & Singh 1996
2,45 GHz	PM, 500 Hz	20 W/m^2 1,2 W/kg	2 h	Rat, in vivo	pos., s.s.	Lai & Singh 1997
2,45 GHz	cw	0,7 – 1,9 W/kg	2 h – 24 h	Glioblastoma Cells (Human), in vitro	neg.?	Malyapa et al. 1997a
2,45 GHz	cw	0,7 – 1,9 W/kg	2 h – 24 h	Fibroblasts (Mouse), in vitro	neg.?	Malyapa et al. 1997a
836 – 848 MHz	cw, FM, PM	0,6 W/kg	2 h – 24 h	Glioblastoma Cells (Human), in vitro	neg.?	Malyapa et al. 1997b
836 – 848 MHz		0,6 W/kg	2 h – 24 h	Fibroblasts (Mouse), in vitro	neg.?	Malyapa et al. 1997b
2,45 GHz	cw	1,2 W/kg	2 h	Rat (Brain), in vivo	?	Malyapa et al. 1998
814 – 837 MHz	PM, TDMA, 50 Hz	8 – 90 W/m^2 2,4 – 26 W/kg	1 h – 10,67 h	T-Lymphoblasten	pos., s.s.	Phillips et al. 1998
2,45 GHz	cw	10 W/m^2 1,18 W/kg	120 d, 2 h/d – 200 d, 2 h/d	Mouse (Brain, Testicles), in vivo	pos., s.s.	Sarkar et al. 1994
1,7 GHz	cw	500 W/m^2	30 min	Mouse (Testicles), in vivo	pos.	Varma & Traboulay 1977

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
Influences on DNA synthesis and repair						
350 MHz	cw	10 – 100 W/m^2 0,039 – 4,5 W/kg	1 h – 3 h	Fibroblasts (Human), in vitro	unclear, partly pos.	Meltz et al. 1987
350 MHz	PM, 5,0 Hz	10 – 100 W/m^2 0,039 – 4,5 W/kg	1 h – 3 h	Fibroblasts (Human), in vitro	unclear, partly pos.	Meltz et al. 1987
850 MHz	cw	10 – 100 W/m^2	1 h – 3 h	Fibroblasts (Human), in vitro	unclear, partly pos.	Meltz et al. 1987
850 MHz	PM, 5,0 Hz	10 – 100 W/m^2	1 h – 3 h	Fibroblasts (Human), in vitro	unclear, partly pos.	Meltz et al. 1987
1,2 GHz	cw	10 – 100 W/m^2	1 h – 3 h	Fibroblasts (Human), in vitro	unclear, partly pos.	Meltz et al. 1987
1,2 GHz	PM, 80 kHz	10 – 100 W/m^2	1 h – 3 h	Fibroblasts (Human), in vitro	unclear, partly pos.	Meltz et al. 1987
836,55 MHz	PM, TDMA, 50 Hz	0,9 – 90 W/m^2 0,00015 - 0,059 W/kg	4 h – 14 d	Glioma-Cells (Rat), in vitro	pos., s.s.	Stagg et al. 1997
Chromosome aberrations						
2,45 GHz	cw	?	?	Mouse (bone marrow), in vivo	pos.	Banerjee et al. 1983
2,45 GHz	cw	400 W/m^2	6 d, 30 min/d	Rat, in vivo	pos.	Beechey et al. 1986
7,7 GHz	cw	300 W/m^2	15 - 60 min	Fibroblasts (Chin. Hamster), in vitro	pos., s.s.	Garaj-Vrohac et al. 1990
7,7 GHz	cw	5 W/m^2	15 - 60 min	Fibroblasts (Chin. Hamster), in vitro	pos., s.s.	Garaj-Vrohac et al. 1991
7,7 GHz	cw	5 – 300 W/m^2	10 min	Lymphocytes (Human), in vitro	pos., s.s.	Garaj-Vrohac et al. 1992
0,4 MHz – 20 GHz	cw, AM, PM			Human, in vivo	pos.#, n.s.	Garson et al. 1991
2,45 GHz	PM, 25 kHz	490 W/m^2 33,8 W/kg	2 h	Egg Cells (Chinese Hamster)	pos., partly s.s.	Kerbacher et al. 1990

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
2,45 GHz	cw	104 – 193 W/kg	20 min	Lymphocytes (Human), in vitro	neg.	Lloyd et al. 1984
2,45 GHz	cw	4 – 200 W/kg	20 min	Lymphocytes (Human)	neg.	Lloyd et al. 1986
2,45 GHz	cw	75 W/kg	30 – 120 min	Lymphocytes (Human), in vitro	pos.	Maes et al. 1993
954 MH	PM, 217 Hz, GSM	Occupational exposure		Lymphocytes, Human, in vivo	pos., s.s.	Maes et al. 1995
954 MHz	217 Hz, GSM	15 W/m^2 1,5 W/kg	2 h	Lymphocytes (Human), in vitro	pos., s.s.	Maes et al. 1995
935,2 MHz	PM/GSM, 217 Hz	0,3 – 0,4 W/kg	2 h	Lymphocytes (Human), in vitro	pos., n.s.	Maes et al. 1997
9,4 GHz	PM, 1000 Hz	1 – 100 W/m^2	2 w, 3 d/w, 1 h/d	Mouse, in vivo	pos., s.s.	Manikowska et al. 1979
2,45 GHz	cw	0,05 – 20 W/kg	2 w, 6 d/w, 30 min/d	Mouse, in vivo	pos., s.s.	Manikowska-Czerska et al. 1985
2,55 GHz	cw	2W/kg	20 min	DNA (E.coli), in vitro	pos.	Sagripanti & Swicord 1986
2,0 – 8,75 GHz	cw	10 W/kg	5 min – 25 min	DNA, in vitro	pos., s.s.	Sagripanti et al. 1987
2,45 GHz	cw	100 W/m^2	120 d 6 h/d	Spermatogonia (Mouse), in vivo	neg.	Saunders et al. 1988
2,45 GHz	cw	50 W/m^2 12,46 W/kg	90 min	Lymphocytes (Human), in vitro	pos., n.s.	Vijayalaxmi et al. 1997
2,45 GHz	cw	750 W/m^2	5 – 30 min	Chinese Hamster (Corneal Epithelium), in vivo	pos, s.s.	Yao 1978
2,45 GHz	cw	15,2 W/kg		RH5- and RH16-Cells (Kangaroo-Rat), in vitro	pos., s.s.	Yao 1982

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
Micronuclei						
154 – 162 MHz	PM, 24,4 Hz	Changing exposures on the pastures		Cow (Erythrocytes) in vivo	pos., s.s.	Balode 1996
2,45 GHz	CW	530 W/m^2 90 W/kg	10 min	Lymphocytes (Human), in vitro	pos., partly s.s.	d'Ambrosio et al. 1995
2,45	AM, 50 Hz, sin	530 W/m^2 90 W/kg	10 min	Lymphocytes (Human), in vitro	pos., partly s.s.	d'Ambrosio et al. 1995
1,25 – 1,35 GHz	?PM	0,1 – 200 W/m^2	Occupational exposure	Lymphocytes (Human), in vivo	pos.	Fucic et al. 1992
7,7 GHz	cw	5 W/m^2	15 - 60 min	Fibroblasts (Chin. Hamster), in vitro	pos., s.s.	Garaj-Vrohac et al. 1991
7,7 GHz	cw	5 – 300 W/m^2	10 min	Lymphocytes (Human), in vitro	pos., s.s.	Garaj-Vrohac et al. 1992
2,45 GHz	cw	75 W/kg	30 – 120 min	Lymphocytes (Human), in vitro	pos.	Maes et al. 1993
9,0 GHz	cw	70 W/kg	10 min	Lymphocytes (bovine), in vitro	pos., s.s.	Scarfi et al. 1996
2,45	cw	50 W/m^2 12,46 W/kg	90 min	Lymphocytes (Human), in vitro	pos.#, n.s.	Vijayalaxmi et al. 1997 b
2,45	cw	1,0 W/kg	18 mon	Erythroczyten (Mouse blood / bone marrow)	pos, s.s.	Vijayalaxmi et al. 1997 a
Sister chromatid exchange						
380 MHz	PM, 17,65 Hz	80 W/kg	?	Lymphocytes (Human), in vitro	neg.	Antonopoulos et al. 1997
900 MHz	PM/DCS, 217 Hz	208 W/kg	?	Lymphocytes (Human), in vitro	neg.	Antonopoulos et al. 1997
1,8 GHz	PM/GSM, 217 Hz	1700 W/kg	?	Lymphocytes (Human), in vitro	neg.	Antonopoulos et al. 1997
2,45 GHz	cw	?	?	Mouse (bone marrow), in vivo	neg.	Banerjee et al. 1983

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
2,45 GHz	PM, 25 kHz	490 W/m^2 33,8 W/kg	2 h	Egg Cells (Chinese Hamster), in vitro	neg.	Ciaravino et al. 1987
2,45 GHz	PM, 25 kHz	490 W/m^2 33,8 W/kg	2 h	Egg Cells (Chinese Hamster), in vitro	neg.	Ciaravino et al. 1991
2,45 GHz	cw	104 – 193 W/kg	20 min	Lymphocytes (Human), in vitro, Add. caffeine	pos., s.s.#	Lloyd et al. 1984
2,45 GHz	cw	75 W/kg	30 – 120 min	Lymphocytes (Human), in vitro	neg.	Maes et al. 1993
954 MHz	PM/GSM, 217 Hz	1,5 W/kg	2 h	Lymphocytes (Human), in vitro	pos., s.s.	Maes et al. 1996
935,2 MHz	PM/GSM, 217 Hz	0,3 – 0,4 W/kg	2 h	Lymphocytes (Human), in vitro	pos., partly s.s.	Maes et al. 1997
2,45 GHz	cw	100 W/m^2	120 d 6 h/d	Spermatogonia (Mouse), in vivo	neg.	Saunders et al. 1988
Mutations						
2,45 GHz	AM, 100 Hz	40 – 80 W/kg	2 h –6 h	Escherichia coli, in vitro	partly pos., s.s.	Anderstam et al. 1983
2,45 GHz	AM, 100 Hz	40 – 80 W/kg	4 h – 7 h	Salmonella typhimurium, in vitro	neg.	Anderstam et al. 1983
3,07 GHz	PM, 500 Hz	95 W/kg	1 h	Escherichia coli, in vitro	neg.	Anderstam et al. 1983
3,07 GHz	PM, 500 Hz	75 - 100 W/kg	2 h – 2,5 h	Salmonella typhimurium, in vitro	neg.	Anderstam et al. 1983
9,4 GHz	cw	600 W/m^2 23 W/kg	30 – 120 min	Escherichia coli, in vitro	neg.	Dardalhon et al. 1981
9,4 GHz	cw	600 W/m^2 23 W/kg	30 – 120 min	Saccharomyces cerevisiae, in vitro	partly pos., s.s.	Dardalhon et al. 1981
9,4 GHz	cw	10 – 600 W/m^2	330 min	Saccharomyces cerevisiae, in vitro	pos.	Dardalhon et al. 1985

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
2,45 GHz	AM, 100 Hz	130 W/kg	5,7 h	Salmonella typhimurium, in vitro	partly pos, s.s.	Hamnerius et al. 1985
3,10 GHz	PM, 500 Hz	90 W/kg	6 h	Salmonella typhimurium, in vitro	partly pos., n.s.	Hamnerius et al. 1985
2,45 GHz	AM, 100 Hz	110 W/kg	6 h	Drosophila melanogaster, in vivo	neg.	Hamnerius et al. 1985
3,10 GHz	PM, 500 Hz	60 W/kg	6 h	Drosophila melanogaster, in vivo	neg.	Hamnerius et al. 1985
2,375 MHz	cw	150.000 – 250.000 W/m^2	25 – 300 min	Drosophila melanogaster, in vivo	partly pos., s.s.	Marec et al. 1985
2,45	PM, 25 kHz	480 W/m^2 30 W/kg	bis 63 h	Leukaemia-Cells (Mouse), in vitro	pos./neg., partly s.s.	Meltz et al. 1989
2,45 GHz	PM, 25 kHz	650 – 870 W/m^2 40 – 40,8 W/kg	4 h	Leukaemia-Cells (Mouse), in vitro	neg.	Meltz et al. 1990

Table A.2 Effects of high frequency electromagnetic fields on cellular processes

Frequency	Modulation	Power Flux Density SAR	Exposure Duration	Examines Subject Method	Result	Ref.
Gene transcription and gene translation						
890 – 915 GHz	PM/GSM, 217 Hz	0,3 – 7,5 W/kg	4 h	Brain (Rat), in vivo	pos., partly s.s.	Fritze et al. 1997 a
835,62 MHz	FM/cw	0,6 W/kg	4 d	Fibroblasts (Mouse), in vitro	partly pos., s.s.	Goswami et al. 1999
847,74 MHz	PM/CDMA, 50 Hz	0,6 W/kg	4 d	Fibroblasts (Mouse), in vitro	partly pos., s.s.	Goswami et al. 1999
836,55 MHz	PM/TDMA, 50 Hz	0,9 – 90 W/m^2 0,00026 – 0,026 W/kg	20 – 100 min	Pheochromocytoma Cells (Rat), in vitro	pos., s.s.	Ivaschuk et al. 1997
Cell-Cycle						
380 MHz	PM, 17,65 Hz	80 W/kg	?	Lymphocytes (Human), in vitro	neg.	Antonopoulos et al. 1997
900 MHz	PM/DCS, 217 Hz	208 W/kg	?	Lymphocytes (Human), in vitro	neg.	Antonopoulos et al. 1997
1,8 GHz	PM/GSM, 217 Hz	1700 W/kg	?	Lymphocytes (Human), in vitro	neg.	Antonopoulos et al. 1997
2,45 GHz	PM, 25 kHz	490 W/m^2 33,8 W/kg	2 h	Egg Cells (Chinese Hamster), in vitro	neg.	Ciaravino et al. 1991
2,45 GHz	cw	5 – 25 W/kg	2 h	Egg Cells (Chinese Hamster), in vitro	pos., s.s.	Cleary et al. 1996
9,4 GHz	PM, 1,0 kHz	1 – 100 W/m^2	2 w 5 d/w 1 h/d	Mouse, in vivo	pos., s.s.	Manikowska et al. 1979
2,45 GHz	cw	100 W/m^2	6x1 h	Lymphocytes (Human), in vitro	neg.	Pazmany et al. 1990
2,45 GHz	cw	100 W/m^2	3x1 h	Lymphocytes (Human), in vitro	neg.	Pazmany et al. 1990
2,45 GHz	cw	100 W/m^2	5 h	Lymphocytes (Human), in vitro	pos., s.s.	Pazmany et al. 1990

Table A.3 Effects of high frequency electromagnetic fields on cell transformation and cell proliferation

Frequency	Modulation	Power Flux Density SAR	Exposure Duration	Studied Subject Method	Result	Ref.
Cell Transformations (including neoplastic)						
2,45 GHz	PM, 120 Hz	4,4 W/kg	24 h	Fibroblasts (Mouse), in vitro	partly pos., s.s.	Balcer-Kubiczek & Harrison 1985
2,45 GHz	PM, 120 Hz	4,4 W/kg	24 h	Fibroblasts (Mouse), in vitro	partly pos., s.s.	Balcer-Kubiczek & Harrison 1989
2,45 GHz	PM, 120 Hz	0,1 – 4,4 W/kg	24 h	Fibroblasts (Mouse), in vitro	partly pos., s.s.	Balcer-Kubiczek & Harrison 1991
2,45 GHz	cw	0,8 – 12,3 W/kg	5 d	Lymphocytes (Human), in vitro	neg.	Czerska et al. 1992
2,45 GHz	PM, 1000 Hz	0,8 – 12,3 W/kg	5 d	Lymphocytes (Human), in vitro	pos., s.s.	Czerska et al. 1992
2,45 GHz	cw	50 W/m^2		Lymphocytes (Mouse)	pos.	Smialowicz et al. 1979
Cell Communication						
836,55 MHz	PM, TDMA, 50 Hz	0,3 – 30 W/m^2 0,00015 - 0,015 W/kg	28 d	Fibroblasts (Mouse), in vitro	partly pos., s.s.	Cain et al. 1997
Cell Proliferation						
2,45 GHz	AM, 100 Hz	40 – 80 W/kg	2 h –6 h	Escherichia coli, in vitro	partly pos., s.s.	Anderstam et al. 1983
2,45 GHz	AM, 100 Hz	40 – 80 W/kg	4 h – 7 h	Salmonella typhimurium, in vitro	partly pos., s.s.	Anderstam et al. 1983
3,07 GHz	PM, 500 Hz	95 W/kg	1 h	Escherichia coli, in vitro	partly pos., s.s.	Anderstam et al. 1983
3,07 GHz	PM, 500 Hz	75 - 100 W/kg	2 h – 2,5 h	Salmonella typhimurium, in vitro	partly pos., s.s.	Anderstam et al. 1983
900 MHz	PM/GSM, 217 Hz	0,55 – 2,0 W/m^2 0,075 – 0,270 W/kg	10 d 2 h/d	Lymphocytes (Rat, (Sprague-Dawley), in vivo	neg.	Chagnaud & Veyret 1999
2,45	cw	5 – 50 W/kg	2 h	Blut (Human), Lymphocytes, in vitro	pos., s.s.	Cleary et al. 1990 a
2,45	cw	5 – 75 W/kg	2 h	Glioma-Cells, in vitro	pos. s.s.	Cleary et al. 1990 b

Frequency	Modulation	Power Flux Density SAR	Exposure Duration	Studied Subject Method	Result	Ref.
2,45 GHz	cw	5 – 50 W/kg	2 h	T-Lymphocytes (Mouse, CTLL-2), in vitro	pos., s.s.	Cleary et al. 1996
2,45 GHz	PM/PCS, 50 Hz	5 W/kg	2 h	T-Lymphocytes (Mouse, CTLL-2), in vitro	pos., s.s.	Cleary et al. 1996
2,45 GHz	?	?	15 s – 5 h	Myeloma- and Hybridoma-Cells (Mouse), in vitro	?, Methode fragwürdig	Dorp et al. 1998
150 MHz	AM, 72 Hz, 217 Hz, 1100 Hz	1,6 kV/m 5,4 µT		Escherichia coli, in vitro	pos., partly s.s.	Grospietsch et al. 1995
2,45 GHz	AM, 100 Hz	130 W/kg	5,7 h	Salmonella typhimurium,	pos, s.s.	Hamnerius et al. 1985
3,10 GHz	PM, 500 Hz	90 W/kg	6 h	Salmonella typhimurium,	pos., s.s.	Hamnerius et al. 1985
836,55 MHz	PM, TDMA, 50 Hz	0,9 – 90 W/m^2 0,00015 - 0,059 W/kg	4 h – 14 d	Glioma-Cells (Rat), in vitro	neg.	Stagg et al. 1997
960 MHz	PM, GSM, 217 Hz	0,0021 W/kg	30 min	transform. Epithel-Amnion-Cells (Human), in vitro	pos., (s.s)	Velizarov et al. 1999

Appendix B

Studies of the effects of high frequency electromagnetic fields on the central nervous system (Blood-Brain-Barrier)

Abbreviations

neg.	negative finding
n.s.	not statistically significant
pos.	positive finding
s.s.	statistically significant
partly	some findings
#	disagreement with the conclusions of the authors
?	unknown; not provided; unreliable

Table B.1 Effects of high frequency electromagnetic fields on the central nervous system

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
2,8 GHz	cw	100 W/m^2	2 h	Rat (Wistar)	pos.	Albert 1979
2,45 GHz	cw	100 W/m^2 2,5 W/kg	2 h	Hamster (Chin.)	pos., s.s.	Albert & Kerns 1981
900 MHz	PM/GSM, 217 Hz	0,3 – 7,5 W/kg	4 h	Rat (Wistar)	pos., partly s.s.	Fritze et al. 1997 b
2,8 GHz	cw	100 – 400 W/m^2	4 h	Rat (Tac:N(SD)sBR)	partly pos, n.s.	Gruenau et al. 1982
2,8 GHz	PM, 500 Hz	10 – 150 W/m^2	4 h	Rat (Tac:N(SD)sBR)	partly pos, n.s.	Gruenau et al. 1982
2,45 GHz	PM, 100 Hz	100 W/m^2 2 W/kg	30 min – 2 h	Rat (Sprague Dawley)	pos., s.s.	Neubauer et al. 1990
1,3 GHz	cw	3 – 30 W/m^2	20 min	Rat (Wistar)	pos., s.s.	Oscar & Hawkins 1977
1,3 GHz	PM, 5 Hz	0,3 – 0,5 W/m^2	20 min	Rat (Wistar)	pos., s.s.	Oscar & Hawkins 1977
1,3 GHz	PM, 1000 Hz	1 – 10 W/m^2	20 min	Rat (Wistar)	pos., s.s.	Oscar & Hawkins 1977
2,45 GHz	cw	1,0 – 300 W/m^2	30 min	Rat (Sprague-Dawley)	partly pos., s.s.	Preston et al. 1979
915 MHz	cw	0,3 – 5,0 W/kg	2 h	Rat (Fischer 344)	pos., s.s.	Salford et al. 1994
915 MHz	PM, 8 Hz	0,016 – 0,16 W/kg	2 h	Rat (Fischer 344)	pos., s.s.	Salford et al. 1994
915 MHz	PM, 16 Hz	0,03 – 2,1 W/kg	2 h	Rat (Fischer 344)	pos., s.s.	Salford et al. 1994
915 MHz	PM, 50 Hz	0,3 – 5,0 W/kg	2 h	Rat (Fischer 344)	pos., s.s.	Salford et al. 1994
915 MHz	PM, 200 Hz	0,4 – 2,9 W/kg	2 h	Rat (Fischer 344)	pos., s.s.	Salford et al. 1994

Appendix C

Studies of the Carcenogenic Effects of High Frequency Electromagnetic Fields in Animal Experiments

Abbreviations

neg.	negative finding
n.s.	not statistically significant
pos.	positive finding
s.s.	statistically significant
partly	some findings
#	disagreement with the conclusions of the authors
?	unknown; not provided; unreliable

Table C.1 Animal experiments regarding the carcenogenic effects of high frequency electromagnetic fields

Frequency	Modulation	Power Flux Density / SAR	Exposure duration	Studies subject / Method	Result	Ref.
836,55 MHz	PM/TDMA, 50 Hz	0,74 – 1,6 W/kg	24 mon 4 d/w 2 h/d	Rat (Fischer 344)	neg.	Adey et al. 1999
900 MHz	PM/GSM, 217 Hz	0,55 – 2,0 W/m^2	2 w, 2 h/d	Rat, Cancer, total	neg.	Chagnaud et al. 1999
2,45 GHz	PM, 800 Hz	0,15 – 0,4	25 mon	Rat, Cancer, total	pos., s.s.	Chou et al. 1992
2,45 GHz	cw	0,3 W/kg	18 mon, 7 d/w, 20 h/d	Mouse (C3H/HeJ), Cancer, total	neg.	Frei et al. 1998 a
2,45 GHz	cw	1,0 W/kg	78 w, 7 d/w, 20 h/d	Mouse (C3H/HeJ), Cancer, total	partly pos., n.s.	Frei et al. 1998 b
835,62 MHz	FM, cw	0,75 W/kg	150 d, 5 d/w, 4 h/d	Rat (Fischer 344), B16 Melanoma	partly pos., n.s.	Higashikubo et al. 1999
835,62 MHz	PM/CDMA, 50 Hz	0,75 W/kg	150 d, 5 d/w, 4 h/d	Rat (Fischer 344), B16 Melanoma	neg.	Higashikubo et al. 1999
929,2 MHz	PM/TDMA, 50 Hz	0,58 – 0,8	6 w, 5 d/w, 90 min/d	Rat (Fischer 344), Liver cancer	neg.	Imaida et al 1998 a
1,439 GHz	PM/TDMA, 50 Hz	0,453 - 0,680 W/kg	6 w, 5 d/w, 90 min/d	Rat (Fischer 344), Liver cancer	neg.	Imaida et al. 1998 b
900 MHz	PM/GSM, 217 Hz	2,6 – 13 W/m^2 0,008 – 4,2 W/kg	18 mon 30 min/d	Mouse (transgenic Eμ-Pim1), Lyphomas	pos., s.s.	Repacholi et al. 1997
915 MHz	PM, 4 – 217 Hz	0,0077 – 1,0 W/kg	2-3 w 5d/w 7h/d	Rat (Fischer 344), Brain Tumor	partly pos., n.s.	Salford et al. 1993
2,45 GHz	cw	10 W/m^2 1,2 W/kg	max. 46 w, 6 d/w, 2,5 h/d	Mouse (C57BL/6J), B16 Melanoma	partly pos., n.s.	Santini et al. 1988
2,45 GHz	PM, 25 Hz	10 W/m^2 1,2 W/kg	max. 46 w, 6 d/w, 2,5 h/d	Mouse (C57BL/6J), B16 Melanoma	partly pos., n.s.	Santini et al. 1988
2,45 GHz	cw	50 – 150 W/m^2 2 – 8 W/kg	12 mon 6d/w, 2h/d	Mouse (C3H/HeA), Cancer, total	pos., s.s.	Szmigielski et al. 1982
2,45 GHz	cw	50 – 150 W/m^2 2 – 8 W/kg	5 mon 6d/w, 2h/d	Mouse (Balb/c), Skin Cancer	pos., s.s.	Szmigielski et al. 1982

2,45 GHz	cw	50 – 150 W/m^2	6 mon, 2 h/d	Mouse (Balb/c), Hautcancer	pos., s.s.	Szudinski et al. 1982
435 MHz	PM, 1,0 kHz	10 W/m^2 0,32 W/kg	21 mon	Mouse (C3H/HeJ), Chest tumors, Ovarian tumors	partly pos., s.s.	Toler et al. 1997
2,45 GHz	cw	100 W/m^2 11 W/kg	5 mon, 6 d/w, 3 h/d	Mouse (Balb/c), Intestinal cancer	partly pos., n.s.	Wu et al. 1994

Appendix D

Epidemiological Studies of the health Risks of HF EMFs

Table D.1 Overview of the results of epidemiological studies regarding exposures in the high frequency spectrum and health risks

Column 1: studied illness

Column 2: Exposure situation

Column 3: Reliability of the exposure classification:

- 3: Source of exposure and quantity clearly identified,
- 2: Method of exposure clearly identified
- 1: HF-exposure probable

Column 4: Relative Risk (R.R.), Explanations see text

Column 5: Statistical significance of the findings:

- s.s.: statistically significant(R.R.=1 outside of 95 %-trust interval, or. $p<0,05$
- n.s.: statistically not significant

Column 6: Literatur reference

Column 7: Comments:

- R: Values in the Column R.R. obtained by conversion (reciprocal value, proportion) of other numerical values or via the interpretation of diagrams
- *: Paper listed in the literature references of Appendix E

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
All Illnesses						
All illnesses, morbidity	MW, Radar, Military	2	1,18	n.s.	Robinette et al. 1980	R*
All Illnesses, morbidity	MW, mobile telecommunications	3	0,93	n.s.	Rothman et al. 1996	
Cancer, total						
Cancer, total, morbidity	MW, Radar, Military	2	1,50	n.s.	Robinette et al 1980	R*
Cancer, total, Incidence	RF, Radio, women	2	1,2	s.s.	Tynes et al. 1996	*
Cancer, total, Incidence	RF/MW, Military	2	2,07	s.s.	Szmigielski 1996	*
Cancer, total, Incidence	HF, Radio and TV transmitters, local residents	3	1,09	s.s.	Dolk et al. 1997 a	*
Cancer, total, Incidence	HF, place of work	1	2,0	n.s.	Lagorio et al. 1997	
Cancer, total, Incidence	RF/MW, Radar and Radio, Police	2	0,96	n.s.	Finkelstein 1998	*
Multiple Myelome	HF, Radio and TV transmitter, local residents	3	1,23	n.s.	Dolk et al. 1997 a	*
Brain tumors, total and tumors of the nervous system, total						
Brain-Tumors, total, Morbidity	HF, Place of work	1	1,54	n.s.	Lin et al. 1985	
Brain-Tumors, Glioblastomas and Astrocytoma, Morbidity	HF, Place of work	1	2,15	s.s.	Lin et al. 1985	
Brain-Tumors, total, Morbidity	HF, Place of work, Men	1	0,38	n.s.	Milham 1985	
Brain-Tumors, total, Morbidity	RF/MW, Place of work, Men	2	2,3	s.s.	Thomas et al. 1987	*
Brain-Tumors, total, Morbidity	RF, Amateur Radio Users	2	1,39	n.s.	Milham 1988	
Brain-Tumors, total, Incidence	HF, Place of work	1	2,9	s.s.	Törnqvist et al. 1991	
Brain-Tumors, Glioblastomas, Incidence	HF, Place of work	1	3,4	s.s.	Törnqvist et al. 1991	
Brain-Tumors, total, Incidence	RF, Place of work, Men	2	0,61	n.s.	Tynes et al. 1992	
Brain-Tumors, total, Incidence	RF, Radio, Women	2	1,0		Tynes et al. 1996	*
Brain-Tumors, total, Incidence	HF, Place of work, Men	1	2,4	s.s.	Beall et al. 1996	
Brain-Tumors, total, Incidence	RF/MW, Military	2	1,39	s.s.	Grayson 1996	*

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Brain-Tumors, total, Morbidity	HF/MW, TV transmitters and others residents (adults)	3	0,89	n.s.	Hocking et al. 1996	*
Brain-Tumors, total, Incidence	HF/MW, TV and other transmitters, Local residents/ Adults	3	0,82	n.s.	Hocking et al. 1996	*
Brain-Tumors, total, Morbidity	HF/MW, TV and other transmitters, Local residents/child.	3	1,0		Hocking et al. 1996	*
Brain-Tumors, total, Incidence	HF/MW, TV and other transmitters, Local residents/child.	3	1,3	n.s.	Hocking et al. 1996	*
Tumors des Nervensystems einschl. Hirntumors, Incidence	RF/MW, Military	2	1,91	s.s.	Szmigielski 1996	*
Brain-Tumors, total, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,29	n.s.	Dolk et al. 1997 a	*
Brain-Tumors, maligne, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,31	n.s.	Dolk et al. 1997 a	*
Brain-Tumors, total, Incidence	RF/MW, Radar and Radio, Police	2	0,84	n.s.	Finkelstein 1998	*
Brain-Tumors, total, Incidence	MW, Mobil telecommunications, Mobile phones	3	1,20	n.s.	Hardell et al. 1999	*
Brain-Tumors, Expos.seite, Incidence	MW, Mobilradio, Handy	3	R 2,45 L 2,40	n.s. n.s.	Hardell et al. 1999	*
Cancer, Eyes						
Melanome, Augen, Incidence	MW, Radar, Military	1	2,1	s.s.	Holly et al. 1995	
Cancer of the respiratory system, lung cancer						
Cancer der Atmungsorgane, Morbidity	MW, Radar, Military	2	2,59	s.s.	Robinette et al. 1980	R*
Lungencancer, Morbidity	HF, Place of work, Men	1	0,80	n.s.	Milham 1985	
Lungencancer, Incidence	RF, Radio, Women	2	1,2	n.s.	Tynes et al. 1996	*
Lungencancer, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,01	n.s.	Dolk et al. 1997 a	*
Lungencancer, Incidence	RF/MW, Radar and Radio, Police	2	0,66	s.s.	Finkelstein 1998	*
Chest cancer, Men						
Brustcancer, Mönner, Incidence	HF, Place of work	1	2,9	n.s.	Demers et al. 1991	

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Brustcancer, Men, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,64	n.s.	Dolk et al. 1997 a	*
Breast cancer, Women						
Brustcancer, Women, Morbidity	HF, Place of work	2	1,15	s.s.	Cantor et al. 1995	*
Brustcancer, Women, Incidence	RF, Radio, Women	2	1,5	s.s.	Tynes et al. 1996	*
Brustcancer, Women, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,08	n.s.	Dolk et al. 1997 a	*
Cancer of the lymphatic and blood forming systems, total						
Cancer des lymphat. and des blutbild. Systems, Morbidity	MW, Radar, Military	2	1,98	n.s.	Robinette et al. 1980	R*
Cancer des lymphat. and des blutbild. Systems, Morbidity	HF, Place of work, Men	1	1,37	n.s.	Milham 1985	
Cancer des lymphat. and des blutbild. Systems, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,21	n.s.	Dolk et al. 1997 a	*
Cancer des lymphat. and des blutbild. Systems, Incidence	RF/MW, Military	2	6,31	s.s.	Szmigielski 1996	*
Leukaemia, total						
Leukaemia, total, Morbidity	HF, Place of work	1	1,11	n.s.	Milham 1982	
Leukaemia, total, Morbidity	RF, Amateur radio user	2	1,91	s.s.	Milham 1985 a	
Leukaemia, total, Morbidity	HF, Place of work, Men	1	1,02	n.s.	Milham 1985 b	
Leukaemia, total, Morbidity	RF Amateur radio user	2	1,24	n.s.	Milham 1988	
Leukaemia, total, Incidence	HF, Military	1	2,4	s.s.	Garland et al. 1990	
Leukaemia, total, Incidence	HF, Place of work	1	0,8	n.s.	Törnqvist et al. 1991	
Leukaemia, total, Incidence	RF, Place of work, Men	2	2,85	s.s.	Tynes et al. 1992	
Leukaemia, total, Incidence	RF, Radio, Women	2	1,1	n.s.	Tynes et al. 1996	*
Leukaemia, total, Morbidity	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,17	n.s.	Hocking et al. 1996	*
Leukaemia, total, Morbidity	RF/MW, TV and other transmitters, Local residents/ children.	3	2,32	s.s.	Hocking et al. 1996	*
Leukaemia, total, Incidence	RF/MW, TV and other transmitters , Local residents/	3	1,24	s.s.	Hocking et al. 1996	*

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
	Adults					
Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents/ Children.	3	1,58	s.s.	Hocking et al. 1996	*
Leukaemia, total, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,83	s.s.	Dolk et al. 1997 a	*
Leukaemia and Non-Hodgkin-Lymphoma, total, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	1,25	n.s.	Dolk et al. 1997 a	*
Leukaemia, total, Incidence	RF/MW, Radar and Radio, Police	2	0,6	n.s.	Finkelstein 1998	*
Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents/ children.	3	1,47	n.s.	McKenzie et al. 1998	*
Acute Leukaemia, total						
Acute Leukaemia, total, Morbidity	HF, Place of work	1	2,39	n.s.	Milham 1982	
Acute Leukaemia, total, Morbidity	HF, Place of work, Men	1	2,12	n.s.	Milham 1985	
Acute Unspez. Leukaemia, Morbidity	RF, Amateur radio users	2	1,76	n.s.	Milham 1988	
Acute Leukaemia, total, Incidence	HF, TV and Radio transmitters, Local residents	3	1,86	n.s.	Dolk et al. 1997 a	*
Lymphat. Leukaemia, total						
Lymphat. Leukaemia, total, Morbidity	RF, Amateur radio users	2	0,77	n.s.	Milham 1985	
Lymphat. Leukaemia, total, Morbidity	RF, Amateur radio users	2	1,03	n.s.	Milham 1988	
Lymphat. Leukaemia, total, Morbidity	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,39	s.s.	Hocking et al. 1996	*
Lymphat. Leukaemia, total, Morbidity	RF/MW, TV and other transmitters, Local residents/ children.	3	2,74	s.s.	Hocking et al. 1996	*
Lymphat. Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,32	s.s.	Hocking et al. 1996	*
Lymphat. Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents/ children	3	1,55	s.s.	Hocking et al. 1996	*

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Lymphat. Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents /children.	3	1,53	n.s.	McKenzie et al. 1998	*
Acute Lymphat. Leukaemia						
Acute Lymphat. Leukaemia, Morbidity	RF, Amateur radio users	2	1,20	n.s.	Milham 1988	
Acute Lymphat. Leukaemia, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	3,57	n.s.	Dolk et al. 1997 a	*
Chron. Lymphat. Leukaemia						
Chron. Lymphat. Leukaemia, Morbidity	RF, Amateur radio users	2	1,43	n.s.	Milham 1985	
Chron. Lymphat. Leukaemia, Morbidity	RF, Amateur radio users	2	1,09	n.s.	Milham 1988	
Chron. Lymphat. Leukaemia, Incidence	HF, Place of work	1	1,3	n.s.	Törnqvist et al. 1991	
Chron. Lymphat. Leukaemia, Incidence	HF, Sender Radio and Fernsehen, Local residents	3	2,56	s.s.	Dolk et al. 1997 a	*
Myelo. Leukaemia, total						
Myelo. Leukaemia, total, Morbidity	RF, Amateur radio users	2	2,81	s.s.	Milham 1985	
Myelo. Leukaemia, total, Morbidity	RF, Amateur radio users	2	1,40	n.s.	Milham 1988	
Myelo. Leukaemia, total, Morbidity	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,01	n.s.	Hocking et al. 1996	*
Myelo. Leukaemia, total, Morbidity	RF/MW, TV and other transmitters, Local residents/child.	3	1,77	n.s.	Hocking et al. 1996	*
Myelo. Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,09	n.s.	Hocking et al. 1996	*
Myelo. Leukaemia, total, Incidence	RF/MW, TV and other transmitters, Local residents/child.	3	1,73	n.s.	Hocking et al. 1996	*
Acute Myelo. Leukaemia						
Acute Myelo. Leukaemia, Morbidity	RF, Amateur radio users	2	2,89	s.s.	Milham 1985	
Acute Myelo. Leukaemia, Morbidity	RF, Amateur radio users	2	1,76	s.s.	Milham 1988	
Acute Myelo. Leukaemia, Incidence	HF, Place of work	1	2,1	n.s.	Törnqvist et al. 1991	
Acute Myelo. Leukaemia, Incidence	HF, Radio, TV, Local residents	3	1,02	n.s.	Dolk et al. 1997	*

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Chron. Myelo. Leukaemia						
Chron. Myelo. Leukaemia, Morbidity	RF, Amateur radio users	2	2,67	s.s.	Milham 1985	
Chron. Myelo. Leukaemia, Morbidity	RF, Amateur radio users	2	0,86		Milham 1988	
Chron. Myelo. Leukaemia, Incidence	HF, Radio and TV transmitters, Local residents	3	1,23	n.s.	Dolk et al. 1997	*
Leukaemia, non-lymph. and non-myelo.						
Leukaemia, non-lymph. and non-myelo., Morbidity	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,57	s.s.	Hocking et al. 1996	*
Leukaemia, non-lymph. and non-myelo., Morbidity	RF/MW, TV and other transmitters, Local residents/child.	3	1,45	n.s.	Hocking et al. 1996	*
Leukaemia, non-lymph. and non-myelo., Incidence	RF/MW, TV and other transmitters, Local residents/ Adults	3	1,67	s.s.	Hocking et al. 1996	*
Leukaemia, non-lymph. and non-myelo., Incidence	RF/MW, TV and other transmitters, Local residents/child.	3	1,65	n.s.	Hocking et al. 1996	*
Lymphomas, Hodgkin-Syndrome						
Lymphosarkome, Morbidity	HF, Place of work, Men	1	0,73	n.s.	Milham 1985	
Lymphome, excl. Lymphosarkoma, Morbidity	HF, Place of work, Men	1	3,42	n.s.	Milham 1985	
Hodgkin-Syndrome, Morbidity	RF, Amateur radio users	2	1,23	n.s.	Milham 1988	
Other malignant illness of the lymphat. tissues, Morbidity	RF, Amateur radio users	2	1,62	s.s.	Milham 1988	
Lymphomas, total, Incidence	RF, Radio, Women	2	1,3	n.s.	Tynes et al. 1996	*
Hodgkin-Syndrome, Incidence	RF/MW, Radar and Radio, Police	2	0,84	n.s.	Finkelstein 1998	*
Non-Hodgkin-Lymphoma, Incidence	HF, Radio and TV transmitters, Local residents	3	0,66	n.s.	Dolk et al. 1997 a	*
Testicular cancer						
Testicular cancer, Incidence	RF/MW, Place of work	2	3,1	s.s.	Hayes et al. 1990	
Germ cell-Carcinoma, Seminoma	RF/MW, Place of work	2	2,8	n.s.	Hayes et al. 1990	
Germ cell-Carzinome, others	RF/MW, Place of work	2	3,2	s.s.	Hayes et al. 1990	

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Testicular cancer, Incidence	MW, Radar, Police	2	6,9	s.s.	Davis & Mostofi 1993	*
Testicular cancer, Incidence	RF/MW, Radar and Radio, Police	2	1,33	s.s.	Finkelstein 1998	*
Cancer of the uterus						
Cancer of the uterus, Incidence	RF, Radio, Women	2	1,9	s.s.	Tynes et al. 1996	*
Skin cancer						
Skin Cancer, Malignant Melanoma, Incidence	RF, Radio, Women	2	0,9	n.s.	Tynes et al. 1996	*
Skin Cancer, total, Incidence	RF/MW, Military	2	1,67	n.s.	Szmigielski 1996	*
Skin cancer, Malignant Melanoma, Incidence	HF, Radio and TV transmitters, Local residents	3	1,43	n.s.	Dolk et al. 1997 a	*
Skin cancer, Malignant Melanoma, Incidence	RF/MW, Radar and Radio, Police	2	1,37	s.s.	Finkelstein 1998	*
Heart and cardio vascular diseases						
Cardio vascular diseases, Morbidity	MW, Radar, Military	2	1,09	n.s.	Robinette et al. 1980	R*
Cardio vascular diseases, Morbidity	RF, Amateur radio users	2	0,70	s.s.	Milham 1988	
Abnorm. Hearbeat rate variability	RF/AM, Radio transmitters, Place of work	2	1,6	s.s.	Bortkiewicz et al. 1996	*
Abnormal ECG	MW	2	2,9	?	Zhao et al. 1994	R
Cardio vascular complaints	MW	2	3,2	?	Zhao et al. 1994	R
Infertility, reduced fertility, Men						
reduced Fertility, reduced Sperm count	MW, Place of work	2	1,20	s.s.	Lancranjan et al. 1975	R
reduced Fertility, immob. Spermatozoa	MW, Place of work	2	1,39	s.s.	Lancranjan et al. 1975	R
reduced Fertility, normal Spermatozoa	MW, Place of work	2	1,18	s.s.	Lancranjan et al. 1975	R
reduced. Fertility, reduced Sperm count	MW, Military	2	2,70	s.s.	Weyandt et al. 1996	R
reduced Fertility, reduced sperm count	MW, Radar	2	1,54	n.s.	Hjollund et al. 1997	R
reduced Fertility, immob. Spermatozoa	MW, Radar	2	1,58	n.s.	Hjollund et al. 1997	R
reduced Fertility, reduced Sperm count	MW, Radar	2	1,10	n.s.	Schrader et al 1998	R

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Infertility, reduced. fertility, Women						
reduced. fertilityt	KW, Place of work, Physiotherapie, Mothers	2	1,7	n.s.	Larsen et al. 1991 a	*
Miscarriages, Stillbirths, Malformations and other abnormalities of newborns						
Malformations and perinatal death	KW, Place of work, Mother	2	2,36	s.s.	Källén et al 1982	
Miscarriage	MW, Place of work, Mother	2	1,28	s.s.	Ouellet-Hellstrom & Stewart 1993	*
Miscarriage	KW, Place of work, Mother	2	1,07	n.s.	Ouellet-Hellstrom & Stewart 1993	*
Cancer, Offspring (parental exposure)						
Tumors of the nervous system	HF, Place of work, fathers	1	2,01	n.s.	Cole Johnson & Spitz 1989	
Cancer, total, Incidence	Radar, Place of work, fathers	2	2,3	s.s.	Smulevich et al. 1999	*
Neurodegenerative Diseases						
Alzheimer's, Morbidity	HF, Place of work	1	1,5	n.s.	Savitz et al. 1998	*
Parkinson's Disease	HF, Place of work	1	-		Savitz et al. 1998	*
Amyotrophic Lateral Sklerosis	HF, Place of work	1	-		Savitz et al. 1998	*
Disturbances of motoric and psychological reactions, Unwellness						
Reduced stamina, Boys	MW, Radar	2	1,38	s.s.	Kolodynski & Kolodynska 1996	R*
Reduced stamina, Girls	MW, Radar	2	1,38	s.s.	Kolodynski & Kolodynska 1996	R*
reduced memory, Boys	MW, Radar	2	1,09	s.s	Kolodynski & Kolodynska 1996	R*
Reduced memory, Girls	MW, Radar	2	1,12	s.s	Kolodynski & Kolodynska 1996	R*
Reduced concentration, Boys	MW, Radar	2	1,23	s.s.	Kolodynski & Kolodynska 1996	R*

Illness	Exposure	Exp. class.	R.R.	stat. Sign.	References	C
Reduced Concentration, Girls	MW, Radar	2	1,20	s.s.	Kolodynski & Kolodynska 1996	R*
Extended reaction time, Boys	MW, Radar	2	1,07	n.s.	Kolodynski & Kolodynska 1996	R*
Extended reaction time, Girls	MW, Radar	2	1,12	s.s.	Kolodynski & Kolodynska 1996	R*
Unwellness. ('Neurosis')	MW	2	3,2	?	Zhao et al. 1994	R

Appendix E (only available in German)

Extracts of our database (EMFbase)

Important research papers relevant to the assessment of health risks resulting from exposure to the electromagnetic fields of mobile telecommunications under the aspect of precautionary health protection