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Genetic, carcinogenic and teratogenic effects of radiofrequency fields [1]

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Abstract

This paper reviews the literature data on the genetic toxicology of radiofrequency (RF) radiation. Whereas in the past most studies were devoted to microwave ovens and radar equipment, it is now mobile telecommunication that attracts most attention. Therefore we focus on mobile telephone frequencies where possible. According to a great majority of the papers, radiofrequency fields, and mobile telephone frequencies in particular, are not genotoxic: they do not induce genetic effects in vitro and in vivo, at least under non-thermal exposure conditions, and do not seem to be teratogenic or to induce cancer. Yet, some investigations gave rather alarming results that should be confirmed and completed by further experiments. Among them the investigation of synergistic effects and of possible mechanisms of action should be emphasised. © 1998 Elsevier Science B.V.

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1. Introduction

Radiofrequency fields, and especially microwaves (300 MHz–300 GHz) are a very important part of the electromagnetic spectrum with respect to their applications and possible health consequences. These non ionising radiations have relatively short wavelengths and high frequencies compared to, e.g., extreme low frequency fields, and therefore also a greater amount of energy which is sufficient to cause heating in conductive materials (Fig. 1). Close to the transmitter, these high-frequency fields may thus be harmful to human beings by producing thermal effects that may sometimes, when thermoregulation processes are insufficient, produce irreversible damage (e.g. cataract; [1,2]). This kind of effect is well known from animal experiments and for that reason does not constitute a particular health problem, as measures can be taken to prevent excessive exposure. But also low-level exposures leading to non-thermal effects are, at least according to certain investigators, possible. Non-thermal effects have been reported in cell cultures and animals, in response to exposure to low-level fields. They are not well established and therefore highly controversial.

While in the past decade people were especially concerned about the safety of microwave ovens (2450 MHz) and even before radar equipment, it is wireless communications (e.g. mobile telephones) that is par-

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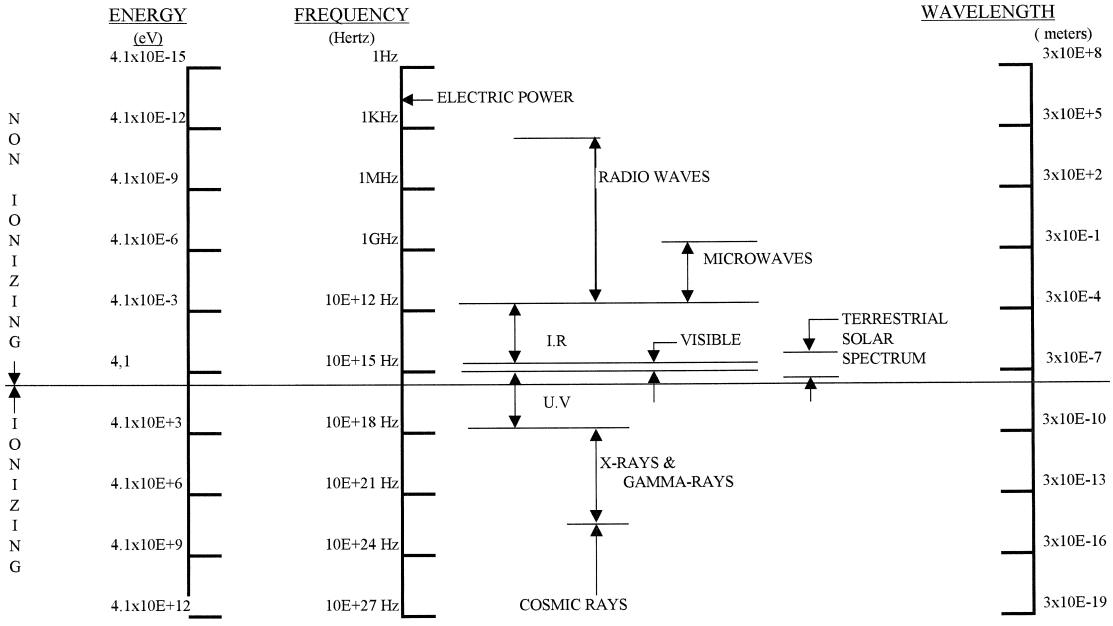

Fig. 1. The electromagnetic spectrum.

ticularly considered at present. It is now indeed generally accepted that modern microwave ovens are harmless (leakage of microwaves from ovens is insignificant) at least when properly used, whereas a number of investigations on radar operators has identified some ''thermal'' effects that lead to safety precautions minimising the hazards. But little is known about the health hazards of cellular telephones that are rapidly gaining popularity. There are several systems in use which allow many users to communicate via the system simultaneously. The dominant access technique in Europe is the so-called TDMA technique (Time Division Multiple Access) which is used in GSM (Global System for Mobile communication), DECT (Digital European Cordless Telecommunications), DCS 1800 (Digital personal Communication System) and in TETRA (Trans European Trunked RAdio) systems. The carrier frequency bands allocated for these services are set mainly in the spectrum regions of 800–900 MHz and 1.8–2.2 GHz. With regard to these (and other mobile phone) systems, one has to distinguish the possible effects emanating from transmission antennas and mobile base station antennas from the mobile phone handset itself. People may be exposed to all these sources, but it is the handset that is most often suspected of having deleterious effects. There is a concern that, as the antenna of the handset is brought close to the head when calls are made or received, there may be a thermal insult at the molecular or cellular level. According to some scientists part of the microwave-energy is absorbed by the head and may eventually produce so-called 'hot-spots' in the brain (e.g. [3,4]). Also, non-thermal effects are often anticipated. Most often headache, eye injury and cancer are mentioned as potential biological effects [5]. However, the only established possible dangerous effect of mobile phones is the interference in some circumstances with pacemakers and other apparatus prompting a warning to users from health authorities (e.g. [6]). With regard to headache, cancer or other serious illnesses, little is known for sure. Therefore there is an urgent need for the updating of our knowledge. Indeed, not only the general public, but also some of the scientists involved in research on microwave or radiofrequency bioeffects are sufficiently worried about the possible adverse health effects of present-day mobile telephones to recommend restricted use of mobile phone handsets. A few even advise not to use them at all. Although most

scientists claim that no research to date suggests serious cause for concern, they all agree that the explosive growth in use of mobile telephones (In 1995 there were over 85 million mobile telephone subscribers world-wide) justifies further research.

In this paper we review the investigations that have been performed on the genotoxic effects of radiofrequency fields, including investigations on cancer and teratogenic effects.

2. Quantities and units

Non ionising radiations are those electromagnetic radiations with a wavelength equal or higher than 10^{-7} m. The different non-ionising radiations are given in Fig. 1 where it can be seen that wavelength, frequency and photon energy are inter-related. Non-ionising radiations have photon energies lower than approximately 12 eV, which is considered the boundary with ionising radiations. This energy is too low to induce ionisations of molecules and too low to break even the weakest chemical bond. Quantities and units differ greatly between the different kinds of electromagnetic radiations as the physical, but also the biological properties differ greatly. Therefore there is no such thing as an absorbed dose or dose-equivalent that we know from ionising radiations and that is common to all non-ionising electromagnetic fields or waves. High-frequency electromagnetic fields are quantified in terms of electric field strength E, expressed as volts per meter (V/m), and magnetic field strengths H, expressed as amperes per meter (A/m). A 'dose' to which the body or tissue or cells are exposed is expressed as the specific absorption ratio (SAR). It gives an evaluation of the energy transfer to the body, tissue or cells per unit of time and per mass. It is expressed as watts per kilogram (W/kg). In biological materials only the electric field contributes to energy absorption. The specific absorption rate is given by the following formula:

$$\mathrm{SAR} = |E|^2 \, \sigma/\rho$$

where σ is the electrical conductivity of the medium (in Siemens per meter) and ρ the density of the medium (in kg/m^3). The SAR can thus be calculated by above equation or measured (by measurements of temperature increases in the exposed subject or object). However, the 'dosimetry' remains a very complicated task, among others because of the different electrical properties of different tissues [4,7]. The SAR is, e.g., dependent on the radiation source, the exposed body (or object) and its configuration. It should be noted that, whereas biological media do not have absorption mechanisms with regard to magnetic fields, rapidly varying magnetic fields might yet induce absorption through secondary induced electric currents.

Exposure to fields can be in the near field, which is the region close to the source antenna, or in the far field. In the far field the high-frequency electromagnetic field is often quantified in terms of power flux density, expressed in units of watts per squared meter (W/m^2).

3. Epidemiological investigations

Epidemiological studies, unlike most laboratory studies, tend to take several years and can only give information on exposures that have already occurred in people. Therefore, as mobile telephone bio-effects are only a very recent concern, there are until now no published epidemiological investigations on cause-specific morbidity or mortality in relation to mobile telephone use. Up to now only limited preliminary notes were published on, e.g., the methodology of epidemiological research with respect to the use of wireless communication [8–10]. However, some epidemiological investigations were conducted in the past with regard to other radiofrequency fields and applications. These studies were, among others, related to occupational exposures to radar, populations living near military installations and near broadcasting towers, amateur radio operators and users of hand-held traffic radar devices (for review, see [5,10,11]). Most studies were rather small and criticised, e.g. on grounds of wrong or insufficient data collection, the absence of any, or adequate dosimetry and the omission to investigate potential

Table 1
Overview of investigations on in vitro RF-induced genetic effects

Exposure conditions	Biological model	Biological endpoint	Results	Comments	Reference
RF-effects alone					
2.45 GHz, CW, 94 mW/cm^2	Commercialised calf thymus DNA	Analysis of thermal denaturation curves	No difference with unexposed DNA	Control and exposed DNA solutions were maintained at the same temperature	[59]
2.45 GHz, 15.2 W/kg	Rat kangaroo cells	Chromosomal aberrations	Increased incidence of unstable chromosome aberrations	The observed aberrations are more commonly associated with ionising radiations	[60]
2.45 GHz, 0–200 mW/cm^2	Chinese hamster cell lines	Cytological effects	Cytological effects (nuclear vacuoles, pycnic and decondensed chromosomes, chromosome breakage)	Effects observed under elevated temperature conditions	[21]
2.45 GHz, 15 W/kg, long-term exposure (during 320 days)	Rat kangaroo cells	Chromosome aberrations	Increased chromosome aberration frequency	A change in cell population by repeated passage of the cells and senescence may have influenced the results	[61]
2.45 GHz, CW, 20 min exposure, SAR from 104 up to 200 W/kg (mild hyperthermia)	Human lymphocytes	Chromosomal aberrations and sister chromatid exchanges	No evidence of cytogenetic damage, even under mild hyperthemia		[62,63]
7.7 GHz, 2 W max. power output, SAR not reported	V79 Chinese hamster cells	Cytogenetic effects	Increased [^{3}H]thymidine incorporation and chromosome aberrations	Thermal effect probable	[64]
7.7 GHz, 2 W max. power output, SAR not reported	V79 Chinese hamster cells	Cytogenetic effects	Effect on colony forming ability, chromosome aberrations and micronuclei	Thermal effect probable	[23]
7.7 GHz, 2 W max. power output, SAR not reported	Human lymphocytes	Cytogenetic effects	Chromosome aberrations and micronuclei	Thermal effect probable	[65]
2.45 GHz, 50 Hz modulated, 30 and 120 min exposure, 75 W/kg	Human lymphocytes	Chromosome aberrations, micronuclei and sister chromatid exchanges	Increased frequency of chromosome aberrations and micronuclei. No microwave induced sister chromatid exchange frequency or influence on the cell proliferation	Temperature computer-controlled at 36.1°C during the complete exposure time. Yet a heating effect cannot be ruled out	[22]

In situ exposure near various broadcasting antenna (10–21 MHz short waves)	Tradescantia	Micronuclei	Increased micronucleus frequency	The effect occurred at field levels of 27.5 V/m and 0.073 A/m which should not cause any significant heating of the samples	[24]
96 GHz, 50 Hz amplitude modulated, 10 min exposure, 100 W/kg	Human lymphocytes	Micronuclei	Increased micronucleus frequency	Thermal increase of 5°C	[20]
0.954 GHz from a GSM base-station, continuous, 60–120 min exposure	Human lymphocytes	Chromosome aberrations	Slight increase in chromosome aberration frequency after exposure	No increased DNA damage according to the alkaline comet assay (unpublished results)	[66]
TEM or GTEM-cells; 440, 900 and 1800 MHz, exposure at 37°C during 39 to 70 h	Human lymphocytes	Chromosome aberrations, sister chromatid exchanges, micronuclei, cell proliferation, HGPRT-mutations	No indications of any RF-induced cytogenetic damage		[67]
415 MHz, 10–30 min, 15 W	Human lymphocytes	Micronuclei	Increased micro nucleus frequency	Increase in micronucleus frequency was related to the exposure time	[68]
935.2 MHz, CW, 2 h exposure, SAR = 0.4 W/kg	Human lymphocytes	Chromosome aberrations and DNA single strand breaks (single cell gel electrophoresis assay)	No indication of direct DNA or chromosome effect		[27]
Synergisms					
2450 MHz pulsed, 49 mW/cm^2, SAR = 34 W/kg. Cells are simultaneously irradiated and MMC exposed	CHO cells	SCEs	No increased SCE frequency in cells exposed to RFs alone or with MMC compared to MMC alone		[69]
350 and 850 MHz and 1.2 GHz, pulsed 1 to 10 mW/cm^2, SAR = 0.39–4.5 W/kg. RF irradiation of the cells followed UV irradiation	Human diploid fibroblasts	DNA repair	RF alone, either continuous or pulsed, has no effect on the DNA repair. RF exposure after UV damage also had no effect		[70]
2450 MHz, pulsed, 48.8 mW/cm^2 SAR = 30 W/kg. Cells are simultaneously irradiated and MMC exposed	L5178Y mouse leukaemia cells	Forward mutation assay (thymidine kinase locus)	RF exposure alone is not mutagenic. RF does not affect either the inhibition of cell growth or the extent of mutagenesis resulting from treatment with MMC alone		[71]

Table 1 (continued)

Exposure conditions	Biological model	Biological endpoint	Results	Comments	Reference
2450 MHz, pulsed 49 mW/cm^2, SAR = 33.8 W/kg. Cells are simultaneously irradiated and adriamycin exposed	CHO cells	Cell cycle progression and SCEs		RF does not affect changes in cell progression caused by adriamycin. RF does not change the number of SCEs that were induced by adriamycin	[72]
2450 MHz, pulsed 49 mW/cm^2, SAR = 33.8 W/kg. Cells are simultaneously irradiated and exposed to MMC and adriamycin	CHO cells	Chromosome aberrations		RF alone does not enhance the chromosome aberration frequency. No alteration in the extent of chromosome aberrations for the combined treatment compared to the chemicals alone	[73]
2450 MH_2, pulsed, SAR ~40 W/kg. Cells are simultaneously exposed to Proflavin	L5178Y mouse leukaemic cells	Forward mutations at the TK-locus		RF alone is not mutagenic. No increased mutation frequency for the combined treatment compared to proflavin alone. No difference in colony size distribution of the mutant colonies	[74]
954 MHz, continuous, 15 W, 49 V/m, SAR = 1.56 W/kg. Cells exposed to mitomycin C following RF-irradiation (during lymphocyte cultivation)	Human lymphocytes	SCE		RF alone did not increase the SCE frequency. The SCE frequency for the combined treatment was always higher than for MMC alone. There was no change in cell proliferation compared to control cultures	[26]
935.2 MHz, CW, 2 h exposure, SAR = 0.4 W/kg. Cells exposed to mitomycin C following RF-irradiation (during lymphocyte cultivation)	Human lymphocytes	SCE		The combined exposure (RF + MMC) revealed a weak effect compared to cells exposed to MMC alone	[27]

confounders. This is for example illustrated in a recent review paper [5] where five studies relevant to adult cancer and RF-exposure were extensively discussed. In a study on RF-exposed US Embassy personnel in Moscow no deleterious effects were found, but the limited number of cancer cases makes this study rather non-informative in comparison with other studies [12]. Milham [13] found an increased standard mortality ratio (SMR) for certain cancer sites in radio amateurs, but there was no adjustment for confounders and most subjects were also professionally exposed to (other) electric and magnetic fields. Armstrong et al. [14] investigated electric utility workers in France and Quebec (Canada) who were exposed to different non-ionising radiation frequencies, including radiofrequencies. They especially found a relationship with lung cancer in one of the utilities (Quebec) whereas a less indicative relationship was found with stomach cancer in another utility (France). These differences from one utility to another detract somewhat from the credibility of these findings regarding the connection between electromagnetic fields and cancer. An association between lung or respiratory system cancer and RF-exposure was also found by Robinette et al. [15], but these authors did not adjust for the possible influence of smoking. Finally, Szmigielski [16] examined the cancer morbidity in Polish career military personnel. He found an excess occurrence of cancers at several cancer sites but no individual assessment was made of exposure levels or duration and, apart from age, adjustments for other factors, e.g. possible carcinogen exposures, were not made. These, and other epidemiological investigations concerned many different modes of exposure and different frequencies. For several studies, the link between the subject's occupation and actual exposure was questionable and very often exposure was also to extreme low-frequency fields and different chemicals. Therefore, taking the limited epidemiological evidence together it must presently be concluded that no definite conclusion can be drawn so far, either about the mobile telephone (no adequate data so far), or about other radiofrequency fields. A number of investigations are now being conducted with regard to cancer and other disorders, especially in the USA and Scandinavian countries, but the results of most of these studies are several years away.

4. Genetic toxicology: in vitro laboratory investigations

Possible effects on DNA or chromosome structure in somatic cells are considered to be very important as these changes could be associated with cell death or, possibly, with the development of cancer. Furthermore, such effects in male or female germ cells are important, as surviving mutations might be passed on to the next generation. Many investigations of RF-induced genetic effects in somatic- as well as in germ cells, therefore, have already been conducted in many different cell and animal systems. Again, different frequencies were investigated with emphasis on the 2450 MHz frequency of microwave ovens. A number of frequencies used in mobile telecommunication were recently also investigated, but most of these studies are still going on.

In the 1970s and 1980s, several investigations were performed on RF-induced genetic changes in microbial test systems, essentially including *Escherichia coli*, *Salmonella typhimurium* and *Saccharomyces cerevisiae*. They gave invariably negative results and will therefore not be further discussed (for a review, see e.g. [17–19]).

In vitro investigations using different eukaryotic cell systems are described in Table 1. It can be seen that both positive and negative results were obtained. Most often, when some evidence for a genetic effect was reported, this could be ascribed to hyperthermia, as the exposure conditions were clearly or most probably thermal in nature. This was, for example, clearly the case in an investigation by d'Ambrosio et al. [20] who found an increased incidence of micronuclei in microwave exposed human lymphocytes. However, the exposure was accompanied by a thermal increase of 5°C. Cytological effects were also found in Chinese hamster cells that were exposed to 2.45 GHz fields, but again these effects were observed under elevated temperature conditions [21]. Investigating the same microwave frequency, we exposed human whole blood cells in such a way that the temperature of the blood remained constant at 36.1°C. The SAR was calculated as 75.5 W/kg, which again clearly suggests that the observed chromosomal abnormalities in the lymphocytes were obtained under thermal exposure conditions [22]. In some instances a thermal effect might be anticipated

even if the exposure was described as ''being comparable to everyday environmental conditions''. SAR values or temperature measurements were missing but the data were so much resembling, e.g. thermal killing curves, that a thermal effect is more than probable (e.g. [23]). Some non-thermal positive responses were also reported (e.g. [24]), but they could possibly be due to so called sporadic positive responses. Reviews of assays used to detect DNA alterations have shown that such sporadic positive responses are indeed obtained from time to time [25]. Yet, a number of positive results remain puzzling and should be further investigated.

Because of the many negative findings (especially in bacteria and algae but also in mammalian or human cells) and as positive findings were almost invariably connected with thermal exposure conditions or with RF-independent situations, it may be concluded that in vitro RF-exposure appears not to induce any genetic damage under non-thermal conditions. Therefore we believe that synergistic investigations deserves special attention. Indeed, people are exposed to many different influences, and theoretically it may well be that a RF-exposure alone is ineffective whereas this exposure might enhance the mutagenicity, carcinogenicity or teratogenicity of chemical or physical factors. As shown in Table 1, most often no synergistic effect was found between the applied field and a number of known mutagens (e.g. mitomycin C, adriamycin, proflavin) when the exposures were simultaneous. However, when the RF-exposure precedes the mutagen, a synergistic effect is sometimes found. This was the case with mitomycin C in a recent investigation of 954-MHz waves emitted by the antenna of a GSM base station (15 W power output, SAR = 1.5 W/kg) [26]. However, when cells were exposed waves to 935.2 MHz (4.5 W) followed by mitomycin C, synergism was much less evident [27]. We are presently investigating the possible synergistic effects of 450 MHz (15 W), and 900 MHz (2–50 W) fields with mitomycin C and X-rays, but so far the results are not very clear and predominantly negative. According to another investigation microwave radiation also increases the mutagenic properties of ethyl methanesulfonate (EMS) in CHO cells [28]. The above investigations reporting a synergistic action of RF-fields with chemical or physical agents corroborates the earlier finding that microwaves enhance, in human cancers, the effects of some cytotoxic agents [29]. It should be stressed that, so far, no mechanistic studies have been conducted in order to explain the reported synergisms. As long as this is not done, the results are only of limited value.

5. Genetic toxicology: in vivo laboratory investigations

Several studies were conducted over the past 20 years using *Drosophila melanogaster* as the test organism. As for the experiments with microorganisms, they all yielded negative results. Therefore we will also refer to the earlier mentioned review papers. As seen in Tables 2 and 3, experiments in mammals gave more conflicting results. This was true as well for investigations on germ cells as for investigations on somatic cells.

5.1. Testicular function and male infertility

Most of the studies on reproduction and development of small mammals exposed to radiofrequency- and microwave radiation have shown effects that can be related to an increase in temperature. Several studies have shown, for example, that acute microwave exposure can affect spermatogenic epithelium and thus male fertility through a raising of the testicular temperature (e.g. [30,31]). The primary spermatocytes were often identified as a particularly sensitive stage and the response identified as identical to conventional heating. Some of the investigations were conducted in anaesthetised rats and mice (e.g. [30–32]) and are therefore of little value with respect to the determination of acceptable limits of human exposure. Anaesthetised animals indeed show an altered temperature regulation. The exposure of conscious animals is thus of greater relevance to exposure standards. Here, experiments have shown that acute and chronic exposure of conscious mice and rats very often do not alter the testicular function or fertility (e.g. [33]). Effects were only found in extreme conditions (e.g. extreme high increase in rectal and/or testicular temperature.

Table 2
Overview of investigations on in vivo RF-induced genetic effects in somatic cells

Exposure conditions	Animal species	Biological endpoint	Main results	Comments	References
2.45 GHz, SAR up to 21 W/kg	Chinese hamsters	Chromosomal aberrations in blood lymphocytes	No RF-effect found	Rectal temperature increase of 1.6°C in the high exposure group. Long cell cultivation time following exposure may disadvantage cells with cytogenetic damage	[75]
9.4 GHz, pulse-modulated, 1–100 W/m^2, 1 h/day over 2 weeks	Male Balb/c mice	Chromosome aberrations in male germ cells exposed as spermatocytes	SAR dependent increase in the frequency of chromosome exchanges and other cytogenetic effects		[76]
2.4 GHz, SAR = 21 W/kg, chronic exposure 8 h/day for 28 days	Mouse	Sister chromatid exchanges in bone marrow cells	No RF-effect found		[77]
2.45 GHz, CW, 2 h/day, 12, 150 and 200 days, 0.1 W/m^2, SAR = 1.2 W/kg	Male Swiss albino mice	DNA analysis with synthetic oligo probes	Altered band patterns of brain and testes DNA from exposed mice		[38]
2.45 GHz, pulsed, 2 μs puls width, 500 pps, or CW, SAR = 1.2 W/kg, 2 h exposure	Male Sprague-Dawley rats	DNA damage in brain cells by the single cell gel electrophoresis assay	Increased DNA damage for both continuous and pulsed fields		[39,40]
2.45 GHz (2 μs pulses, 500 pps), 2 h exposure at 2 mW/cm^2, SAR = 1.2 W/kg Naltrexone injections (1 mg/kg) before and after RF-exposure	Male Sprague-Dawley rats	DNA double-strand breaks in brain cells with the single cell gel electrophoresis assay	Increased DNA double-strand breaks following RF-exposure. The effect was partially blocked by treatment with naltrexone		[41]
2.45 GHz; 20 h/day, 7 days/week during 18 months; SAR = 1.0 W/kg	C3H/HeJ mice; prone to mammary tumours	Micronuclei in peripheral blood and bone marrow	No significant difference in exposed and sham-exposed animals	Also no difference in micronuclei between both groups when the animals with mammary tumours were considered separately	[125]

Table 3
Overview of investigations on in vivo RF-induced genetic effects with regard to reproduction and teratogenesis

Exposure conditions	Animal species	Biological endpoint	Main results	Comments	Reference
Acute microwave exposure of testes	Anaesthetised rats and mice	Effects on male reproductive system	Elevated testicular temperature and depletion of the spermatogonic epithelium		[30]
9.4 GHz, pulse-modulated, 1–100 W/m^2, 1 h/d over 2 weeks	Mice	Effects on male reproductive system	Chromosome aberrations in male germ cells exposed as spermatocytes	SAR-dependent increase in the frequency of chromosome exchanges and other cytogenetic effects	[76]
2.45 GHz, 30 min exposure, half-body SAR = 30 W/kg	Anaesthetised Mice	Effects on male reproductive system	Temperature dependent depletion of primary spermatocytes. Threshold at 39°C	Response identical to that of conventional heating	[32]
2.45 GHz, SAR = 44 W/kg	Anaesthetised Mice	Effects on male reproductive system	Decreased sperm count and increase in abnormal sperm	Maximal effect at spermatocyte and spermatid exposure	[31]
1.3 GHz, pulse modulated (1 μs pulses at 600 pps), whole body SAR = 6.3 W/kg; exposure 6 h/d for 9 days	Rats	Effects on male reproductive system	No statistical significant effect on daily sperm production, number of epididymal sperm, sperm morphology and mass of testes and epididymes	Body temperature raised by 1.5°C	[78]
1.3 GHz, CW, exposure 8 h at SAR = 9 W/kg	Conscious rats	Effects on male reproductive system	No significant differences in testicular function	Rectal temperature raised by 4.5°C	[79]
2.45 GHz, SAR = 5.6 W/kg, exposure for 80 h over a 4 w period	Conscious rats	Effects on male reproductive system	Transient reduction in fertility	Rectal temperature estimated to have raised to 41°C and testicular temperature to 37°C (probably no loss in fertility at lower temperatures)	[80]
2.45 GHz, 100 mW/cm^2 for 10 min; 50 mW/cm^2 (3× over 1 day) or 4× over 2 weeks	Mice	Dominant lethality	No dominant lethality, increase in mutagenicity in some instances	Males mated 6–7 weeks after exposure. Effects on mutagenicity thought to be due to a temperature increase	[81]
2.45 GHz, CW, 1.7 kW/m^2 for 70 s	Mice	Dominant lethality	Increased dominant lethality, abnormal sperm morphology and reduced male fertility	May be due to temperature changes	[82]
1.7 GHz, 50 W/kg during 30 min	Mice	Dominant lethality	Induction of dominant lethals		[83]

2.45 GHz, CW, 43 W/kg during 30 min	Mice	Dominant lethality	Reduced pregnancy rate and pre-implantation survival. No dominant lethality	Effects probably the result of a heating effect	[84]
2.45 GHz, exposure during 120 h over an 8-week period, SAR = 5 W/kg; mated after the exposure period over the following 8 weeks	Conscious mice	Dominant lethality	No effect on pregnancy rates, pre-implantation survival or testes weight.		[85]
2.45 GHz, CW, 0.05–20 W/kg, 6 h over 2 weeks	Mice	Sperm abnormalities	Increased frequency of sperm abnormalities		[86]
2.45 GHz, exposure 16 h/day for up to 30 days; SAR ≤ 20 W/kg	Conscious mice	Sperm abnormalities	No significant effect on sperm count and number of abnormal sperm	Testes temperature unaffected	[87]
2.45 GHz, exposure at SAR = ≤ 112 W/kg for up to 5 min	Mice	Embryogenesis/teratogenesis	Mainly exencephaly	Effects increased with increasing exposure	[88]
2.45 GHz, exposure 100 min/day throughout gestation at SAR = 2, 8 or 22 W/kg	Mice	Embryogenesis/teratogenesis	Significant decreased mean mass of live foetuses in the high exposure group	No abnormal raise in body temperature	[89]
2.7.12 GHz, exposure during 20–40 min/day at SAR = 11 W/kg	Rats	Embryogenesis/teratogenesis	Abnormal development, embryo/foetal deaths at all stages of development	Rectal temperature raised to 43°C. Teratogenicity directly related to the temperature of the dam during exposure and to the duration of exposure	[90–92]
6 GHz, SAR = 7 W/kg, exposure 8 h/day throughout pregnancy	Rats	Embryogenesis/teratogenesis	Slight growth retardation no evidence of increased embryo or foetal deaths or of developmental abnormalities subtle behavioural effects were noticed post-natally	Here the SAR was reported to be insufficient to raise the body temperature significantly	[93,94]
2.45 GHz, 100 min/day from day 6 to day 15 of gestation at 6 or 4 W/kg	Rats	Embryogenesis/teratogenesis	Significant mean body weight of the foetuses at 6 W/kg	Maternal temperatures raised to about 40°C (both exposure conditions)	[95,96]
2.45 GHz, 6 h/day throughout gestation; estimated SAR = 2–4 W/kg	Rats	Embryogenesis/teratogenesis	No effect	No increased rectal temperature	[97,98]

Table 3 (continued)

Exposure conditions	Animal species	Biological endpoint	Main results	Comments	Reference
915 MHz, 6 h/day throughout pregnancy, estimated SAR = 3.5 W/kg	Rats	Embryogenesis/teratogenesis	No effect	Maternal temperature unaffected	[99,100]
100 MHz, 0.4 W/kg, 6 h 40 min/day on day 6–11 of gestation	Rats	Embryogenesis/teratogenesis	No effect		[101]
27.12 MHz at 1 W/m^2 from day 0 up to day 20 of gestation, SAR = 10–4 W/kg	Rats	Embryogenesis/teratogenesis	Significant decrease in post-implantation survival and reduced cranial ossification	Normal rectal temperature results difficult to reconcile with other investigations	[102]
2.45 GHz, SAR = 7, 28 or 40 W/kg, 8 h/day for various times of gestation	Mice	Embryogenesis/teratogenesis	Significant reduction in implantation sites per litter and in foetal weight at 40 W/kg and exposure during day 1–6; significant increase in % of malformed foetuses (mainly cleft palate) for exposures at day 6–15 (same SAR); not confirmed at later experiment	Colonic temperature rose by 1 or 2.3°C in the 2 highest exposure groups; respective exposure levels assumed to be at threshold	[103,104]
2.45 GHz, CW, 100 min/day during 6–17 days of gestation, SAR = 16 W/kg	Mice	Embryogenesis/teratogenesis	Lowered foetal weight and delayed skeletal maturation persistent delay of brain maturation		[105,106]

2.45 GHz, 100 min/day from day 6–14 of gestation, SAR = 16–18 W/kg	Syrian hamster	Embryogenesis/teratogenesis	Increased foetal deaths, decreased foetal weight and decreased skeletal maturity observed at the highest SAR-values	Mean maternal rectal temperature raised 0.4 and 1.6°C resp.	[107]
2.45 GHz, 2 h/day, 7 days/week on day 1–7, 8–18 or 1–18 of gestation, SAR = 6 or 9 W/kg	Mice	Embryogenesis/teratogenesis	Significant reduction in post-implantation survival; increased intracranial and intra-abdominal bleeding; post-natally markedly decreased resistance to viral and bacterial infection	Rectal temperature increased by 1.5–2.0°C during each exposure	[108]
2.45 GHz et 10, 100 or 400 W/m^2, SAR = 0.5, 4–5 and 16–18 W/kg, 2 h/day from day 1 to 18 of gestation	Mice	Embryogenesis/teratogenesis	Enhanced effect of the chemical teratogen cytosine arabinoside (10 mg/kg on day 9); microwaves alone resulted in reduced foetal body mass ((4–5 W/kg); increased post-implantation deaths (16–18 W/kg)	2°C increased rectal temperature in highest exposure group	[109]
10 MHz, continuous waves, SAR = 6.6 W/kg 2-methoxyethanol (oral intake) administration 5 min before RF irradiation	Rats	Embryogenesis/teratogenesis	Combined exposures enhance the foetal malformation frequency as well as the severity of malformations		[110]
2.45 GHz	Mice	Sex-linked recessive lethals	No difference between sham-exposed and RF-exposed animals	Rectal temperature in the highest exposed group rose by up to 3°C	[33]

Table 4
Overview of investigations on RF-induced genetic effects in human subjects

Exposed subjects	Cells investigated	Genetic endpoint	Main results	Comments	Reference
Professionally exposed subjects; frequencies ranging from 400 kHz to 20 GHz	Human lymphocytes	Chromosome aberrations	No increase in chromosome damage found in radio-lineman who work with radiofrequencies at or below occupational exposure limits	Exposure to RF-fields is relatively pure (no known other (professionally) exposures	[44]
Professionally exposed subjects (10 $\mu W/cm^2$), frequencies ranging from 1250 to 1350 MHz	Human lymphocytes	Micronuclei	Increased micronucleus frequency in exposed subjects compared to controls	Workers exposed to other environmental influences?	[43]

Table 5
A few in vitro cancer related studies

Exposure conditions	Biological model	Biological endpoint	Main results	Comments	Reference
Cells in test-tubes exposed for 3–5 days in anechoic chamber to pulsed 10 cm microwaves from a box horn antenna	Human lymphocytes	Lymphoblastoïd transformation	Induction of lymphoblastoïd transformation by the microwave radiation	According to control experiments the effect was not due to a heating effect	[111]
Amplitude modulated 450 MHz waves, 10 W/m^2, eventually after TPA application	Reuber H35 hepatoma cells, CHO cells and 294 T human melanoma cells	ODC activity	Increased ODC activity		[48]
Amplitude modulated microwaves, SAR = 3 W/kg	Mouse fibroblasts	ODC activity	Increased ODC activity	Increase is at much lower level than treatment with a chemical promoter	[49,112]
Amplitude modulated microwaves, SAR = 0.1–4.4 W/kg, eventually combined with X-rays, followed by treatment with TPA	C3H10T1/2 cells	in vitro cell transformation	Enhanced cell transformation	Some inconsistency was found between the different studies. Furthermore, C3H10T1/2 cells are chromosomally abnormal and their response to proliferative stimuli may be atypical	[113–115]
2.45 GHz	Human red blood cells	Na^+,K^+-ATPase activity	Inhibition of Na^+,K^+-ATPase activity		[45]
2.45 GHz, CW, SAR = 5–20 W/kg	Glioma cells or human lymphocytes	RNA precursor [^{3}H]uridine or DNA precursor [^{3}H]thymidine incorporation	Elevated transcription and proliferation at SAR = 25 W/kg but unchanged at higher SARs		[46,47]
27 MHz or 2.45 GHz, CW, SAR = 5 or 25 W/kg	CHO cells	Cell cycle alterations	Cell cycle alterations found at both frequencies, 2.45 GHz being more effective in inducing alterations	Exposure under ''isothermal'' conditions (37 ± 0.2°C)	[116]

Table 6
Review of some cancer studies in RF or microwave exposed laboratory animals

Exposure conditions	Biological model	Biological endpoint	Results	Comments	References
9.27 GHz, 2 μs pulses at 500 pps, 1000 W/m^2, 4.5 min/day, 5 days/week for 59 weeks	Swiss mice	Post-mortem analysis	Microwave-induced leucosis	The investigation was severely criticised for several reasons	[117]
800 MHz, 2 h/day; 5 days/week over a period of 25 weeks. SAR up to 1.5 W/kg	RFM adult mice	Effect on longevity	Non-significant increase in the life-span of the exposed mice compared to the controls. No significant changes in mean red and white blood cells		[118]
5 Hz pulses of 447 kV/m peak electric field strength; 5 days/week for 33 weeks	AKR/J male mice	Leukaemia	21% of exposed mice had leukaemia at the end of the exposure compared to 46% in the sham-exposed group	Absence of complete analysis of leukaemia incidence precludes any definite conclusion	[119]
2.54 GHz pulsed at 800 Hz; SAR = 015–0.4 W/kg	Sprague-Dawley rats	General health and longevity	Significant raise in overall incidence of malignancies	The biological significance of the results was questioned by the authors because the higher incidence level of specific malignancies was similar to what was previously reported as being normal for untreated rats	[120]
2.45 GHz, 35 W/kg; 20 min/day during days 11–14 of gestation	CFW mice exposed in utero. Mice injected with lymphoreticular sarcoma cells at day 16 of age and further irradiated or not	Tumour incidence	Significantly lower tumour incidence in mice irradiated in utero and irradiated or sham-irradiated post-natally. Mice irradiated in utero and followed for 36 months had initially a lower tumour development rate, but then the rate increased to become similar		[121]

2.45 GHz, 500 W/m^2, SAR = 25 W/kg; 2 h/day for 7 days	BALB/c mice s.c. injected prior to irradiation with mouse sarcoma cells	Tumour growth and lung metastases	Temporary tumour regression followed by renewed growth 12 days later. More lung metastases in the exposed group compared to controls but mean survival time greater in exposed animals (53 days vs. 38 days)		[122]
2.45 GHz, 500 W/m^2, SAR = 25 W/kg; 2 h/day for 7 days	Idem but irradiation prior to injection of sarcoma cells	Tumour growth and lung metastases	Significantly accelerated tumour growth and increase in the number of lung metastases. Mean survival time shortened in irradiated mice		[122]
2.45 GHz, 50 or 150 W/m^2, SAR up to 3 and 8 W/kg resp.; 2 h/day, 6 days/week	C3H/He mice (irradiated at age 6 weeks to 12 months) or BALB/C mice irradiated 1 or 3 months prior to or simultaneously with exposure to benzo[*a*]pyrene	Effect of microwaves on tumour growth and lung cancer colony assay	SAR-dependent acceleration of both mammary tumours in C3H/HeA mice and skin tumours in BALB/c mice. Microwave exposure resulted in an increase in the number of L1-sarcoma cell colonies in the lungs of irradiated mice	C3H/HeA mice are genetically predisposed to mammary tumours. BABL/c mice exposed to 3,4-benzopyrene by skin painting (5 months treatment) 2–3 W/kg gave results similar to the effect of overcrowding (chronic stress)	[54]
2.45 GHz, CW or PW (10 μs pulses for 10 ms repeated at 25 Hz); SAR = 1.2 W/kg	C57BL/6 J mice, irradiation 15 days exposure prior to s.c. injection of 3×10^6 B16 melanoma cells and during subsequent tumour development	Effect of microwaves on tumour progression	No effect on the rate of development of melanoma or on the mean survival time (25 days)		[52]
Low level pulsed 2.45 GHz waves, SAR up to 0.4 W/kg; exposure from 2 to 27 months of age	Sprague-Dawley rats	Life time study including determination of cause of death, frequency and site of neoplastic and non-neoplastic lesions	No clear evidence of an increase in tumour incidence as a result of exposure to microwaves	Data should eventually be re-analysed	[123,124]

Table 6 (continued)

Exposure conditions	Biological model	Biological endpoint	Results	Comments	References
915 MHz, CW, 1 W and modulated with 4,8,16 and 200 Hz in 0.5 ms pulses, and 50 Hz in 6 ms pulses (2 W per pulse), 7 h/day, 5 days/week during 3 weeks	Fisher 344 rats, injected with rat glioma cells	Histopathological brain examination	No significant difference in tumour size between experimental and control animals		[53]
2.45 GHz, 10 mW/cm^2 in anechoic chamber, 3 h/day, 6 days/week over a 5-month period. SAR = 10–12 W/kg	BALB/c mice, 4 weeks old. Animals injected with dimethylhydrazine (DMH) once per week during the course of the microwave treatment. A group DMH-treated animals were also exposed to TPA once per week for 10 weeks from the third week on after initial treatment	Colon tumour incidence	The 2.45 GHz microwave radiation at 10 mW/m^2 did not promote DMH-induced colon cancers in young mice		[51]
900 MHz pulsed at 217 Hz and pulse width of 0.6 ms. Two half-hour exposure periods per day for 18 months. SAR = 0.13–0.4 W/kg	Eμ-Pim1 transgenic mice	Lymphoma	Lymphoma risk was found to be significantly higher in the exposed mice than in the controls	This study does not indicate that RF-field exposure would be specifically lymphomagenic in normal mice. So far the significance of the finding for mobile phone-linked human health is not clear	[55]

5.2. Teratogenicity

Several studies have shown that sufficiently elevated body temperatures are teratogenic to a number of mammalian species, including primates (e.g. [34–36]).

As microwave (or RF)-induced teratogenic effects were usually accompanied by a significant rise in maternal body temperature, they hence could be ascribed to this thermal effect (see [37] for a review). Yet, the results of studies on microwave induced foetal loss and developmental malformations were sometimes inconsistent with others. This is most probably due to species differences with regard to their thermo-susceptibility; rats appear more likely to lose heat-damaged embryos than to give birth to malformed young [34], whereas in other mammals a wider range is observed between a teratogenic exposure and a lethal exposure.

Overall, the conclusion should be that the evidence suggests that only exposures that have an appreciable heating effect are likely to affect the embryo adversely.

5.3. Somatic cell genetic effects

Most studies that were published so far did not demonstrate convincingly any direct DNA damage after acute or chronic exposure of biological systems to RF-fields (e.g. [17,19]), in particular when temperatures were maintained within normal physiological limits. However, especially two recent investigations have suggested that RF-fields yet can affect DNA (see Table 2). In the first, Sarkar et al. [38] found evidence of an alteration in the length of a DNA microsatellite sequence in cells from brain and testis of mice exposed to 2.45-GHz fields. In another series of experiments, Lai and Singh [39,40] demonstrated that acute exposure to low-intensity radiofrequency radiation increased DNA strand breaks in brain cells of the rat. Furthermore, DNA double-strand breaks were lower in animals to which the opioid antagonist naltrexone was injected immediately before and after the RF-exposure [41]. These data support the hypothesis that radiofrequency radiation activates endogenous opioids in the brain which in turn cause biological effects [42]. Before speculating about the possible consequences of these findings, especially with regard to the use of cellular phones (which was abundantly done in the media), it should be realised that the data were obtained for more than a worst-case exposure situation and for 2450-MHz fields that do not correspond to mobile phone frequencies. The significance of these data, therefore, remain unclear to date. These experiments should also be replicated before the results can be used in any health risk assessment, especially given the weight of evidence suggesting that RF fields are not genotoxic.

In humans only few studies have been performed (Table 4). An increased incidence in micronucleated white blood cells from professionally exposed subjects was found in one investigation [43], but it is not clear to what extent the subjects were also exposed to other environmental influences. A previous cytogenetic investigation of radio-linemen that were almost solely exposed to RF-fields gave negative results [44]. We are presently performing another study on maintenance workers being professionally exposed to microwaves of different frequencies, and so far we have not found a cytogenetic effect in these subjects as compared to non-exposed control subjects. However, the study is not yet completed and in particular some (in vitro) synergy experiments should be repeated.

According to above (and other) cytogenetic investigations, it must be clear that caution must be exercised in making any general conclusion on the clastogenicity, genotoxicity and heritable effects of radiofrequency fields and microwaves.

6. Cancer-related in vitro investigations

As stated above, the evidence for a clastogenic or genetic effect of microwaves is rather contradictory, although overall it may be concluded that RF-fields/microwaves are not genotoxic under non-thermal conditions. Therefore it may also be concluded that RF-fields or microwaves are not tumour initiators and that, if they are somehow related to carcinogenicity, this should be by some other mechanism (e.g. by influencing tumour promotion or by increasing the uptake of carcinogens in cells). Table 5 summarises most relevant investigations on in vitro cancer related RF-effects. It shows that RF-fields

may affect ion fluxes through cell membranes (important signalling mechanisms) via effects on ion pumps such as Na^+,K^+-ATPase [45] in human red blood cells exposed to RF and microwave radiation. Non-thermal effects were also reported on gross transcription as measured by incorporation of specific RNA or DNA precursor [^{3}H]uridine or [^{3}H]thymidine [46,47]. Cell transformation was shown to be induced in a dose-dependent way (increase with increasing SAR value) and according to a series of papers, low-level Hz-modulated microwave radiation may affect intracellular activities of enzymes involved in neoplastic promotion without measurable influence on the overall DNA synthesis. For example, a number of investigations brought some evidence of an effect on intracellular levels of ornithine decarboxylase (ODC) which is an enzyme usually implicated in tumour promotion [48,49]. Tumour promoters do increase the ODC synthesis. This was also found for microwaves although the microwave effect was much weaker and occurred only for certain modulations of the carrier wave.

To date it is not very clear what these results mean in terms of in vivo carcinogenesis.

7. Cancer-related in vivo investigations

A relatively large number of in vivo investigations, mainly on 2450-MHz waves but also on other frequencies, have already been performed (Table 6 and [50]). Results of many cancer investigations (especially when ongoing and so far unpublished investigations are included) do not support the suggestion that even an extensive daily exposure to EMF causes tumour growth or tumour promotion (e.g. [51–53]). Overall, the evidence for a co-carcinogenic effect or a RF-induced influence on tumour progression is not substantive. Yet, there were a few positive results that are sufficiently indicative to merit further investigation. This is the case for the investigation of Szmigielski et al. [54] who observed faster development of benzo[*a*]pyrene-induced skin tumours in mice that were exposed for some months to sub-thermal 2450-MHz microwaves. Also, a very recent investigation on transgenic mice expressing an activated Pim1 oncogene in their lymphoid cells furthermore turned up to be probably the most significant finding yet of adverse effects [55]. The mice were exposed to pulsed digital (GSM-type) phone radiation at a power density roughly equal to a cell-phone transmitting for two half-hour periods each day. A significant increase in B-cell lymphoma was evident early in the experiment, but the incidence continued to rise over the 18-month exposure period. The experiment was conducted in the ''far field'' at distances greater from the mice than the cell phone is normally held from the head. It is not yet clear what these results mean in terms of public health, but it seems clear that they should be taken seriously and prompt further research.

8. Other cancer-related studies

The above-mentioned investigations may be considered more or less directly related to cancer. Other investigations on other physiological processes may of course also be related to cancer. This is especially true for effects on the immune system. It is indeed well known that the immune system plays an important role in controlling the proliferation of cancer cells. Therefore, numerous studies have also been performed at various power levels and frequencies. However, these investigations are well beyond the scope of this report and will therefore not be discussed in detail. The interested reader can find some excellent review papers on this subject (e.g. this issue and [18,56–58]).

9. Conclusion

Today, it is still not very clear whether proper use of radiofrequency fields and microwave-emitting devices may be harmful to human beings. Many investigations have been performed in the past and many different end-points have been studied. Investigations on genetic- or cancer-related effects are among them. It was almost invariably found that biological effects of acute exposure to these fields were consistent with responses to induced heating, resulting in frank rises in cellular, tissue or body temperature of 1°C or more. This is consistent with a SAR-value above 1–2 W/kg. Most often, experimental (thermal) con-

ditions are not encountered in normal life situations or correspond to a worst-case situation that may occur for short periods of time. However, in the case of low-level exposures, the site of energy deposition might be such that micro-heating could occur. Some data are rather puzzling, and effects obtained under non-thermal conditions should require further investigations. There is a need for better understanding of this type of interaction. In particular, synergistic effects and mechanistic investigations should be highlighted. With regard to potential health effects of mobile phones, especially long term effects, the available data are at present too scarce. Further studies are needed before any valid conclusion can be drawn.

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