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Biological effects of amplitude-modulated radiofrequency radiation

by Jukka Juutilainen, PhD,¹ René de Seze, PhD²

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Users of mobile telephones are exposed to radiofrequency radiation. One of the questions still open today is whether amplitude-modulated radiofrequency signals from digital phones exert specific bioeffects different from those of continuous (unmodulated) radiofrequency radiation. This paper reviews recent literature on the bioeffects of amplitude-modulated radiofrequency radiation, from cells to humans. The consistency of the results is discussed, and exposure parameters are compared to identify possible biologically active forms of amplitude modulation. Several studies have reported findings consistent with effects on the nervous system and cancer-related biological processes. However, the methods and exposure parameters vary widely, and no independent replications of the positive findings have been reported. The results available today fail to support the existence of well-defined modulation-specific bioeffects from exposure to radiofrequency radiation. Additional systematic studies are needed to identify possible reproducible modulation-dependent effects and biologically active modulation parameters.

Key terms cellular telephones, electromagnetic fields, microwaves, nonionizing radiation, nonthermal effects.

The bioeffects of amplitude-modulated (AM) radiofrequency radiation have been a subject of debate since early publications (1, 2) suggesting that weak radiofrequency radiation, if amplitude modulated at low frequencies, might have specific effects different from the thermal effects of strong radiofrequency energy. This discussion has been recently activated by the increasing human exposure to radiofrequency radiation due to the rapid growth in the use of mobile telephones.

Some of the new personal telecommunication systems emit AM radiofrequency electromagnetic fields. Since the modulation frequencies are in the extremely low-frequency (ELF) range (0—300 Hz), it has been hypothesized that these modulated fields might have effects similar to the purported cancer-related or other health effects of ELF magnetic fields emitted by, for example, powerlines. Although the whole-body average exposure levels caused by these devices are very low, local energy absorption maximums can be close to the exposure limits. Moreover, the high number of people exposed warrants continued research to make sure that there are no risks associated with long-term exposure to weak AM fields.

This review focuses on recently published literature on the biological effects of AM radiofrequency radiation, from cells to humans. Reviews of the older literature are

available elsewhere (3—6). The aim of this review is different from that of other recent reviews (7, 8). More than critically analyzing the details of each study, this paper attempts to address the consistency of the results with respect to the effective exposure parameters. If there are any specific effects of modulated radiofrequency radiation, it is important to try to identify the modulation frequencies, carrier frequencies, and other exposure parameters that are biologically active, as well as those that are not.

Radiofrequency radiation

Radiofrequency electromagnetic fields constitute a part of the electromagnetic spectrum (table 1). *Microwaves* are a part of the radiofrequency band. The limit between radiofrequency and infrared radiation is normally defined as 300 GHz, corresponding to a wavelength of 1 mm. The lower frequency limit is variously defined by different organizations. In this review radiofrequency refers to frequencies above 100 kHz. In a strict physical sense, a radiofrequency electromagnetic field can be called radiofrequency *radiation* only in the *far field* (ie, far enough

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Table 1. The electromagnetic spectrum.

	Wavelength (λ) ^a	Frequency (f) ^a
Ionizing radiation	<100 nm	>3·10 ¹⁵ Hz
Ultraviolet radiation	100–400 nm	800–3000 THz
Visible light radiation	400–700 nm	400–800 THz
Infrared radiation	0.7–1 mm	0.3–400 THz
Radiofrequency radiation	1 mm–3 km	0.1 MHz–300 GHz
Microwaves	1–1000 mm	0.3–300 GHz
Very low-frequency (VLF) electromagnetic fields	3–1000 km	0.3–100 kHz
Extremely low-frequency (ELF) electromagnetic fields	>1000 km	<300 Hz
Static electric and magnetic fields	∞	0

^aThe relationship between wavelength and frequency is given by $c = \lambda f$, where c is the velocity of light (and other forms of electromagnetic radiation) in free space ($3 \cdot 10^8$ m/s).

from the source). Normally, this means a distance of at least 1 wavelength. In nonphysical language, for example, in legislation, all forms of electromagnetic fields are sometimes called radiation even if they do not fulfill the physical criteria of electromagnetic radiation.

Modulation

Radiofrequency radiation is often modulated by another, lower frequency signal to make it, for example, carry information or to save energy. The most common forms of modulation used for telecommunication are amplitude modulation and frequency modulation. In amplitude

Figure 1. Schematic representation of an unmodulated (continuous) wave (A) and two forms of amplitude modulation: waves modulated with a smoothly varying (B) or pulsed (C) signal.

modulation, the amplitude of the radiofrequency *carrier wave* is modulated by the low-frequency signal (eg, voice) (figure 1). Similarly, frequency modulation involves modulation of the frequency so that it varies in a narrow band around the basic frequency. A form of amplitude modulation is *pulse modulation*. Pulse-modulated (PM) radiation with very high intensity, short duration pulses is emitted by radar. Pulse modulation is also used in digital mobile phones (9), such as the European GSM or the North American DAMPS system. The frequency of the pulses is 217 Hz with the GSM and 50 Hz with the DAMPS. Analog mobile phones, in contrast, are based on frequency-modulated (FM) signals.

Thermal, athermal and nonthermal effects

Absorbed radiofrequency energy is converted to heat in tissues. When the thermal load is such that the core temperature of the organism increases with respect to its normal value, "thermal" effects are induced. If the thermoregulatory system is able to maintain the organism at its normal temperature, "athermal" (or isothermal) effects may then be linked with chronic stress. Finally, when the thermal load is low enough not to trigger thermoregulation, "specific" or "nonthermal" effects can occur through mechanisms other than macroscopic heating of tissues.

Some of the bioeffects of pulsed fields that do not cause general heating or trigger thermoregulation may, nevertheless, be caused by local heat production. The hearing of microwave pulses is a good example. This generally accepted and physically well-understood phenomenon is based on thermoacoustic expansion, acoustic waves generated by the absorption of high-peak power microwave pulses (6). The hearing of microwave pulses can also produce behavioral changes in laboratory animals. Well-established specific bioeffects of AM radiofrequency radiation thus exist. However, the biological effects based on thermoacoustic expansion have not been included in this review. Instead the review focuses on the reported effects that may be "nonthermal", based on mechanisms other than tissue heating.

The amount of thermal energy absorbed per mass unit (watts/kilogram) is expressed by a quantity called *specific absorption rate* (SAR). It is recommended that the whole-body SAR values associated with human exposure to radiofrequency radiation should be less than 0.4 W/kg (10). The average SAR values of mobile phone users are orders of magnitude below this limit, but local maximums in the head can be higher than 1 W/kg if calculated for very small volumes of tissue. This value is not far from the limit of 1.6 W/kg (11) or 2 W/kg (12) recommended for local exposure.

Effects of amplitude-modulated radiofrequency radiation in vitro

Nerve and muscle cells

Many in vitro experiments have been performed on cardiac cells or isolated hearts. Microwaves were observed to affect aggregated cardiac cells from chicken embryos (13). Although a decrease in the interbeat interval after 190-second exposure to 2.45-GHz microwaves was consistent with a thermal effect, other results were less predictable, for example, an increase in the interbeat interval under continuous microwave exposure at 1.2—12.2 W/kg and a decrease in the interbeat interval after PM microwave exposures at 8.4—12.2 W/kg (duty cycle = 11%). In contrast, although a higher average SAR of around 80 W/kg was used (2.45 GHz, 10 μ s, 100 pps, 1/1000 duty cycle), Field et al (14) did not find any effect on the mean action-potential firing rate of spontaneously active ganglion neurons of land snails, while a significant ($P < 0.05$) increase in the mean input resistance of exposed neurons was described. Schwartz & Mealing (15) did not find any change in calcium ion fluxes or contractile force in isolated atrial stripe of frog heart exposed for 32 minutes to continuous or AM 1-GHz electromagnetic fields (0.5 Hz or 16 Hz, 3.2 μ W/kg to 1.6 W/kg). Exposure of frog hearts for 2 hours to pulsed 2.45-GHz microwaves (10 μ s, 1/1000 duty cycle, 16-Hz modulation, SAR 0.003, 2 or 6 W/kg) did not modify conduction velocity (16). Although Pakhomov et al (17) did not find any effects on the twitch rates and amplitudes of isolated *Rana temporaria* frog auricles when PM 915-MHz microwaves at SAR values below 10—20 W/kg were applied alone, they observed an increase in both these parameters after sensitization by 1-mM caffeine added to the saline flow system.

Genotoxicity

An earlier critical review on the genotoxicity of microwave radiation in vitro (18) emphasized the need for careful temperature control and concluded that microwaves are not genotoxic unless high temperatures are achieved.

The possibility that genotoxic effects are induced after in vitro exposure to radiofrequency fields was investigated by D'Ambrosio et al (19) using human whole blood exposed to 9-GHz microwaves amplitude modulated at 50 Hz (10 min, 90 W/kg). A marginally significant ($P < 0.05$) increase in the number of cells showing micronuclei was seen after the AM exposure but not after the continuous exposure. The thermal increase was 5°C, but the authors reported that a similar heating through a water bath had had no effects on micronuclei in earlier experiments.

Maes et al (20) exposed human peripheral blood lymphocytes to 2.45-GHz microwaves for 30 and 120

minutes at a constant temperature of 36.1°C. The authors found an increase in the frequency of chromosome aberrations and micronuclei. However, cell proliferation and sister chromatid exchange frequency were not affected. In a recent study by the same group (21), whole blood was exposed next (5-cm distance) to the radiating antenna of a GSM base station (49-V/m, 2-h exposure). Although some genetic damage was observed (chromosome aberrations), the authors concluded that microwaves emitted by a base station are not capable of inducing genetic effects in the general population exposed to field levels much lower than those used in these experiments.

The clastogenic effects of short-wave exposure (10—21 MHz) were tested using plant cuttings (*Tradescantia*) exposed in situ near various broadcasting antennas for 30 hours (22). There was a statistically significant 2-fold increase in the mean micronuclei frequency in the exposed samples with respect to sham-exposed (shielded) or control (100 m away) samples. The effect occurred at field levels of 27.5 V/m and 0.073 A/m, that is, at levels which should not cause any significant heating of the samples.

Studies related to cocarcinogenic effects

Genetic mutation is the first step of carcinogenesis, often called *initiation*. *Promoters* are agents that act during the later stages of carcinogenesis, leading to the development of tumors from the initiated potential cancer cells. Carcinogenesis is a complex process, and several different factors may modify the probability that genotoxic effects lead to the development of malignant tumors. The term *cocarcinogenic* is used here to refer to promoters and other agents that are carcinogenic only in combination with genotoxic exposures.

Combined effects of 954-MHz radiofrequency radiation and the chemical mutagen mitomycin C were studied using sister chromatic exchange in human lymphocytes as the end point (23). Whole blood samples from 8 donors were exposed to AM radiation from a GSM base station. The samples were kept 5 cm away from the antenna at a constant temperature of 17 (± 1)°C. The estimated SAR was 1.5 W/kg. After the radiofrequency exposure, lymphocytes were cultured for 70 hours with or without mitomycin C. The radiofrequency exposure alone did not increase the frequency of sister chromatid exchanges. However, a clear increase was seen in the cells exposed to mitomycin C, and this response was significantly enhanced in the cells exposed to microwaves.

Cain et al (24) tested the hypothesis that AM microwaves and a chemical tumor promoter, 12-O-tetradecanoylphorbol-13-acetate (TPA), are copromoters that enhance focus formation of transformed cells in coculture with parental C3H/10T1/2 fibroblasts. In this model, the parental cells normally suppress focus formation,

probably due to intercellular communication between the transformed cells and normal cells. Decreased intercellular communication is probably the main mechanism by which tumor promoters lead to focus formation in this model. Cell cultures were exposed to an 836.55-MHz field using a form of pulse modulation [time-division multiple access (TDMA)] with a pulse frequency of 50 Hz. An SAR value of 0.15, 1.5, or 15 mW/kg was used during the pulses. The cells were exposed 24 hours a day for 28 days in a repeating cycle, 20 minutes on, 20 minutes off. The radiofrequency exposures did not alter the TPA-induced focus formation in this model.

Ornithine decarboxylase (ODC) is an enzyme needed in the synthesis of polyamines. Its activity is elevated in rapidly growing tissues, including tumors. ODC activity is also strongly increased by known chemical tumor promoters such as TPA, and it is therefore a potentially useful marker of tumor promotion. Both ELF magnetic fields (25, 26) and AM microwaves (27) have been reported to affect ODC activity in vitro and in vivo, although the about 2-fold increases found in these experiments are much lower than the changes induced by, for example, TPA. The role of different modulations in the effects of 835-MHz microwaves on the activity of ODC was investigated in L929 cells at an SAR of about 2.5 W/kg (28). Exposure to continuous waves produced no effects for exposure times between 2 and 24 hours, except for a small enhancement after 6 hours of exposure. No effects were seen in cells exposed to FM signals. Exposure to AM fields, in contrast, yielded significant enhancement of ODC activity. The effect was seen at several different types of amplitude modulation, including a typical signal from a TDMA cellular telephone. The effect was frequency dependent, with statistically significant effects at modulation frequencies from 16 to 65 Hz, but little or no effects at 6 Hz and 600 Hz.

Effects of amplitude-modulated radiofrequency radiation in vivo

Nervous system

Lai (29) published an extensive review of the neurological effects of radiofrequency radiation, from the ionic or molecular level (calcium fluxes, neurotransmitters, etc) to the behavioral level (psychological effects), including anatomy, morphology, physiology, electrophysiology, and interactions with drugs. In recent years, many experiments conducted by Lai and his co-workers on neurochemical changes showed effects of acute low-level microwave radiation on the neurological system of rats, particularly modifying the pharmacologic action of drugs on phenomena such as hypo- or hyperthermia,

cataplexy, stereotypic behavior, and narcosis (30). Additional effects were seen independently of any drug on the activity of the cholinergic system in rat brain, functionally related to behavioral tasks involving learning and memory. Activation of endogenous opioids by microwaves seemed to play an important mediating role in some of these effects. Recent results by Lai indicate an important role for the cholinergic system and endogenous opioids in the microwave-induced spatial memory deficit in the radial-arm maze (31). These results suggest that the effect of microwaves on working memory also involves the cholinergic system, also modulated by endogenous opioids, and moreover that only central, and not peripheral, endogenous opioids are responsible for the effect. According to the authors, the septohippocampal pathway was much more involved in the acute effect of microwaves than the basalis-cortical pathway was.

Another effect on the cholinergic system was reported for 880-MHz microwaves pulse-modulated with 16-Hz square waves (32). A 1-hour exposure of male Wistar rats significantly decreased acetylcholinesterase activity in the neocortex by 25%. This effect was not obtained at other modulation frequencies (3, 5, 7 or 30 Hz) or with unmodulated continuous waves. The gamma-aminobutyric acid (GABA) and glutamate receptors were also affected after exposures as short as 5 minutes, at 880 MHz or 915 MHz, with a decrease in ^3H -muscimol binding and an increase in ^3H -glutamate. This effect was dose-dependent for power densities greater than $50 \mu\text{W}/\text{cm}^2$. In vitro studies on synaptosomal preparations or a model system consisting of a bilayer proteolipid membrane enriched in GABA receptors yielded similar results.

Consistent behavioral and endocrine changes were observed under various conditions of exposure to low-intensity 3-GHz continuous or 2.45-GHz modulated microwaves (33). The modulation consisted of pulse trains, with a pulse frequency of 400 Hz within each train, and the repetition frequency of the trains was 0.05, 0.28, or 0.5 Hz. The effect was different (activation or inhibition) depending on the duration of exposure: 0.5–12 hours, single or repeated (15–60 days, 7–12 hours/day), pulsed or continuous wave exposure. A threshold level was determined at $10 \mu\text{W}/\text{cm}^2$.

No effects of continuous-wave microwaves at $10 \mu\text{W}/\text{cm}^2$ were found on the total power of the electroencephalographic (EEG) spectrum from rats (34). However, an increase in the delta band and a decrease of the alpha and beta bands were observed after a 5-minute exposure to 2.45-GHz pulsed microwaves at $3 \mu\text{W}/\text{cm}^2$ (217 Hz, 1/8 duty cycle) (35).

Three-hour whole-body irradiation at an incident power density of $1 \mu\text{W}/\text{cm}^2$ to 2450-MHz microwaves amplitude modulated at 16 Hz caused changes in the ultrastructural distribution of calcium and calcium-

activated ATPase (adenosine triphosphatase) in the medial habenula of mice (36). The changes included a decreased number of synaptic vesicles containing Ca^{++} precipitates at 1 hour and 24 hours after exposure, appearance of reaction products in the synaptic clefts and on nonsynaptic surfaces of the neuronal plasma membrane, and increased Ca^{++} -ATPase activity, specific for quercetin-sensitive intracellular Ca^{++} -ATPase.

Human experiments

The effects of pulsed high-frequency electromagnetic fields similar to those emitted by mobile telephones were recently published in relation to human sleep (37). The power density was low ($50 \mu\text{W}/\text{cm}^2$) and thus mimicked exposure to a nearby base station. It was applied for 8 hours starting at light extinction at 2300. Twelve subjects spent 3 successive nights in a sleep laboratory. The first night was for adaptation, and during the 2 following nights they were either exposed or sham exposed. The significant findings included a decrease in sleep onset latency, from $12.3 (\pm 6.0)$ to $9.5 (\pm 4.4)$ minutes, and a slight alteration, from $-10.1 (\pm 1.4)$ to $-9.6 (\pm 1.6)$ dB [$0 \text{ dB} = 1 (\mu\text{V})^2/\text{Hz}$], of the mean power density of the averaged power EEG spectrum during REM (rapid eye movement) sleep. Exposure of awake subjects for 3.5 minutes to the same field did not cause any changes in the electroencephalogram (38).

Blood-brain barrier

An important characteristic of the central nervous system is the permeability of the blood-brain barrier. Exposure of Fischer 344 rats to 915-MHz microwaves at SAR values between 0.016 and 5 W/kg showed significantly more albumin leakage in animals exposed to PM (30%) or continuous fields (40%, $P=0.002$) than in control animals (8%) (39–41). Similar findings were reported by another group (42) for rats exposed to the radiofrequency, static or gradient magnetic fields associated with magnetic resonance imaging.

Genotoxic effects

When evaluated by the alkaline microgel electrophoresis ("comet") assay, acute exposure of rats to low-intensity microwaves increased DNA (deoxyribonucleic acid) single- and double-strand breaks in brain cells (43, 44). Male Sprague Dawley rats were exposed for 2 hours to pulsed microwaves (2.45 GHz, 2- μs pulse width, 500 pps, SAR of 0.6 or 1.2 W/kg) or continuous microwaves (SAR of 1.2 W/kg). Four hours after the pulsed exposure, the number of DNA breaks was statistically significantly increased. In the continuous exposure the increase was detectable immediately after the 2-hour exposure. The mechanism behind these observations is not known. The authors speculated on a possible increase in DNA breakage or a suppression of the repair processes.

Life span and cancer studies

An extensive review of long-term exposure of animals to radiofrequency radiation was written by Guy (45). Another review (46) indicated a trend towards an increase rather than a decrease in life span. Chou et al (47) exposed 100 male Sprague-Dawley rats for 25 months, 21.5 hours/day, to 2.45-GHz microwaves pulse modulated at 800 Hz. The pulsed microwaves were square-wave modulated at 8 Hz. The SAR values ranged from 0.4 to 0.15 W/kg depending on animal weight. The general health of the animals was evaluated by measuring several parameters, including behavior, immunology, hematology, blood chemistry, metabolism, organ weights, histopathology, and longevity. The total number of endpoints was 155. The findings were negative for most of the parameters measured. Positive findings of effects on corticosterone levels and the immune system at 13 months of exposure were not confirmed in follow-up studies. Differences in oxygen consumption and carbon dioxide production were seen in young rats. A statistically significant increase of primary malignant tumors was observed in the exposed versus the control rats. The authors concluded that the results did not indicate any definitive biological effects in rats chronically exposed to radiofrequency radiation at 2.45 GHz. The positive findings need independent experimental evaluation.

Transgenic mice of the strain *E μ -Pim1* were exposed to 900-MHz radiofrequency fields pulse modulated at 217 Hz, similar to the fields used in GSM phones (48). *E μ -Pim1* mice are moderately predisposed to develop lymphoma spontaneously. One hundred animals were exposed and 101 were sham-exposed for 60 minutes a day for 18 months. The specific absorption rates varied from 0.008 to 4.2 W/kg. A statistically significant doubling of lymphoma risk was found for the exposed mice in a comparison with the controls.

A study on the progression of transplanted brain tumors (49) failed to find any effects of continuous or pulsed microwaves. Several modulations were tested, including the 217-Hz modulation of GSM mobile phones.

Female Sprague-Dawley rats ($N=21$) received an injection of benzo[a]pyrene and were divided into 8 groups (50). The animals were then sham-exposed or exposed 2 hours/day to pulsed, GSM-type (217 Hz, duty factor 1/8) 900-MHz microwaves at 55 or 200 $\mu\text{W}/\text{cm}^2$. The estimated SAR values were 0.065 or 0.24 W/kg. No differences were seen in tumor development, the level of an autoantibody related to tumor development, survival, or immunologic parameters.

Epidemiologic studies

Very little epidemiologic evidence is available on AM radiofrequency electromagnetic fields (51, 52). Most of

these studies deal with radioelectronics workers or with radar- or radio-broadcasting exposure.

Slight changes in the heart rate of radiocommunications workers have been reported (53, 54) that suggest neurovegetative impairment due to radiofrequency exposure. The small size of the cohort (71 exposed persons, 22 referents) indicates that confirmation with a more extensive study is needed. Similar findings were recently published from an old study (1973–1975) using a threshold of 100 V/m between the high- and low-level exposures (55). Neurovegetative disturbances without focal central nervous system (CNS) changes were also found by Bielski (56), with abnormal EEG recordings from 29% of the workers.

The population of the Skrunda village in Latvia has been chronically exposed to PM radiofrequency radiation from a near-by radio station. The station consists of 2 radars operating in the 156- to 162-MHz range. They emit 0.8-ms pulses at 41-ms intervals (ie, the pulse frequency is 24.4 Hz). The time-average exposure levels did not exceed 100 $\mu\text{W}/\text{cm}^2$ (57), but no detailed assessment of the radiofrequency levels experienced by the “exposed” population is available. Significant differences in motor function, memory, and attention were reported in an epidemiologic study comparing schoolchildren living in the exposed area and in a nearby reference area (58).

All cancer cases among Polish military career personnel were registered during a period of 15 years (59). Each year about 3700 (3%) of the population were classified as exposed to radiofrequency radiation. Data on exposure was obtained from military safety groups, who take measurements of radiofrequency intensities around potential sources and keep health records of personnel working at these posts. The radiofrequency exposures were mostly pulse-modulated and had carrier frequencies of 150 MHz–3.5 GHz. At about 80% of the posts, the intensities were 0.1–2 W/m^2 . About 15% had intensities of 2–6 W/m^2 , and exposure exceeding 6 W/m^2 was rare. Compared with the unexposed personnel, the risk for all cancers was twice as high [relative risk (RR) 2.07, 95% confidence interval (95% CI) 1.1–3.6] among those exposed to radiofrequency radiation. The increase was mainly due to increased colorectal (RR 3.2, 95% CI 1.5–6.2) malignancies, esophagus and stomach (RR 3.2, 95% CI 1.9–5.1) malignancies, nervous system tumors (RR 1.9, 95% CI 1.1–3.5), and malignancies of the hematopoietic system and lymphatic organs (RR 6.3, 95% CI 3.1–14.3).

An association between lung cancer and exposure to “pulsed electromagnetic fields” among electric utility workers has been reported (60). The odds ratio for all cancers was also slightly increased in the highest exposure category. The exposure was evaluated by the radiofrequency channel of personal electromagnetic exposure meters (Positron). The radiofrequency channel has been

designed to detect fast-changing transients from 50- to 60-Hz electrical systems by measuring fields in the 5- to 20-MHz frequency band. It was later, however, found to respond to other frequencies, including transmissions from walkie-talkies and car and truck radios. The interpretation of these findings is difficult because it is not exactly known what the meters actually measured.

The AM electromagnetic fields emitted by the Skrunda station and the Polish military (mainly radar) equipment differ in many respects from those produced by mobile phones. As long as we do not understand the mechanisms of the possible bioeffects of AM radiofrequency radiation, the observations are of limited value in assessing the health effects of mobile communication.

Mobile phones have been used only for a relatively short period. Epidemiologic studies on the eventual health effects related to these devices are starting, and only short-term results are now available (61).

Mechanisms of interaction

There is no generally accepted theory to explain the biophysical mechanisms by which AM fields could affect living organisms unless they are strong enough to cause general or local heating. Any theory that attempts to answer this question should explain the basic physical processes by which cells detect weak signals. If the biological responses are due to the modulation frequency and not the carrier frequency, the theory should also explain how the radiofrequency signal is demodulated to produce the low-frequency signal. Many hypothetical mechanisms have been proposed (6, 62, 63). Most of the proposed mechanisms are merely qualitative speculations that cannot be verified or falsified by experimental testing. These are of little, if any, value in understanding the biophysical basis of the possible effects of modulated radiofrequency radiation. Some of the hypotheses, however, have been formulated exactly enough to produce predictions that can be tested experimentally.

As long as the interaction mechanisms remain unknown, it will be difficult to accept the existence of specific bioeffects of AM radiofrequency fields.

Discussion

Results suggesting bioeffects of weak AM radiofrequency fields have been obtained using a wide variety of biological models and end points. None of the findings described in this review have been replicated in an independent laboratory. Thus they cannot be considered as established bioeffects.

Table 2. Recent studies of the biological effects of amplitude-modulated radiofrequency radiation. [CF = carrier frequency, MF = modulation frequency, SAR = specific absorption rate, CW = continuous wave, AM = amplitude modulation, AchE = acetylcholinesterase, GABA = gamma-aminobutyric acid, EEG = electroencephalography, ATPase = adenosine triphosphatase, + = an effect has been observed, ++ = stronger effect than at the other exposure parameters, +d = the effect differs from those of the other field parameters, but it is not possible to say which effect is stronger (eg, different direction of change), - = no effect, .. = data not available]

Reference	Biological variables studied	CF (MHz)	MF (Hz)	Time-average SAR (W/kg)	Peak ^a SAR (W/kg)	Effects at CW?	Effects at AM?	Duration of exposure
<i>Nerve or muscle cells in vitro</i>								
Seaman & deHaan, 1993 (13)	Interbeat intervals in chicken cardiac cells	2450	10 000	1.2—12.2	11—112	+	+d	190 s
Field et al, 1993 (14)	Snail neurons	2450	0.8—1.7, or 16	12—43.5	24—87	+	+d	190 s
Schwartz & Mealing (15)	Firing rate	2450	100	80	80 000	..	-	45 min
	Input resistance	2450	100	80	80 000	..	+	45 km
	Atrial stripe of frog heart, Ca ⁺⁺ flux and contractile force	1000	0.5 or 16	3.2·10 ⁻⁶ —1.6	6.4·10 ⁻⁶ —3.2	..	-	32 min
Yee et al, 1994 (16)	Frog heart, conduction velocity	2450	16	0.003, 2, or 6	3—6000	..	-	2 h
Pakhomov et al, 1995 (17)	Twitch rate and amplitude of frog auricle, combined effect with caffeine	915	16 + 250	8—10	45—50	..	+	33 min
<i>Genotoxicity in vitro</i>								
d'Ambrosio et al, 1995 (19)	Human lymphocytes, micronuclei	9000	50	90	180	-	+	10 min
Maes et al, 1993 (20)	Human lymphocytes							
	Chromosome aberrations and micronuclei	2450	50	75	225	..	+	30 or 120 min
	Sister chromatid exchanges	2450	50	75	225	..	-	30 or 120 min
Maes et al, 1995 (21)	Human lymphocytes, chromosome aberrations	954	217	1.5	12	..	+	2 h
Maes et al, 1996 (23)	Human lymphocytes, combined effect with mitomycin C on sister chromatid exchanges	954	217	1.5	12	..	+	2 h
<i>Other in vitro studies</i>								
Philippova et al, 1994 (64)	Ligand-receptor binding							
	Glutamic acid, hippocampal cells	900	1—100	0.5—18	2.5—90	..	+	15 min
	Dihydroalprenolol, new born hepatic cells	900	1—100	0.5—18	2.5—90	..	+	15 min
Litovitz et al, 1993 (27)	Ornithine decarboxylase activity	915	55—65	2.5	2.63	..	+	8 h
Penafiel et al, 1997 (28)	Ornithine decarboxylase activity	835	16—65	2.5	5—7.5	-	+	2—24 h
		835	6, 600	2.5	5—7.5	-	-	2—24 h
Cain et al, 1997 (24)	Focus formation in C3H/10T1/2 cells	837	50	0.00005—0.005	0.00015—0.015	-	-	28 d
<i>Nervous system in vivo</i>								
Lai et al, 1992 (30)	High-affinity choline uptake	2450	500	0.6	600	+	+	45 min
Kolomytkin et al, 1994 (32)	Rats							
	Neocortex AchE activity	880	16	0.02—0.4	0.04—0.8	-	+	1 h
		880	3, 5, 7 or 30	0.02—0.4	0.04—0.8	-	-	1 h
	GABA and glutamate receptors	880 or 915	16	0.02—0.4	0.04—0.8	-	+	5 min
		880 or 915	3, 5, 7 or 30	0.02—0.4	0.04—0.8	-	-	5 min
Thuroczy et al, 1994 (34)	Spectral EEG of rats (↑β power)	4000	16	8.4	13	-	+	30 min
	Cerebral blood flow	4000	16	8.4	13	+	-	30 min
Thuroczy et al, 1995 (35)	Spectral EEG (↓αβ, ↑δ) and visual evoked potentials in rats	2450	217	0.3	2.5	..	+	30 min
Navakatikian & Tomashevskaya, 1994 (33)	Behavioral and endocrine changes in rats	2450(CW) or 3000 (PW)	400 and 0.05, 0.28 or 0.5	0.03—0.75	1.5—37.5·10 ³	+	+d	0.5—12 h ^b
Kittel et al, 1996 (36)	Ca ⁺⁺ distribution and Ca ⁺⁺ ATPases in mouse brain	2450	16	1 mW/cm ²	?	..	+	3 h
Mann & Rösche, 1996 (37)	Changes in human sleep	900	217	0.05	0.4	..	+	8 h
Rösche & Mann, 1997 (38)	Human EEG	900	217	0.05 mW/cm ²	0.4	..	-	35 min
Salford et al, 1993 (40), Salford et al, 1994 (41)	Permeability of blood-brain barrier in rats	915	8—200	0.016—5	3.3	+	+	2—960 min
<i>Genotoxicity in vivo</i>								
Lai & Singh, 1995 (43), Lai & Singh, 1996 (44)	DNA single and double strand breaks in rat brain	2450	500	0.6	600	..	+	2 h
				1.2	1200	+	+d	
<i>Carcinogenesis in vivo</i>								
Chou et al, 1992 (47)	Rats							
	Endpoints (N=155) measuring general health	2450	8 and 800	0.15—0.4	38—100	..	-	25 months
	Primary malignancies						+	
Salford et al, 1993 (49)	Tumor growth in rat brain after injection of glioma cells	915	4, 8, 16, 50 or 200	0.008—1.67	3.33	-	-	7 h x 9—16 d
Chagnaud et al, 1996 (50)	Tumor growth, survival, several immunologic parameters	900	217	0.065 or 0.24	0.52 or 1.92	..	-	2 h x 10 d
Repacholi et al, 1997 (48)	Lymphoma-prone transgenic mice, lymphoma development	900	217	0.008—4.2	0.064—34	..	+	18 months, 1 h/d

^a For pulsed modulations the peak value is here defined as the SAR or intensity during the pulse, and it has been calculated for this table as the time-average value (usually given in the original article) divided by the duty cycle. For 100% sinusoidal modulations, the peak value has been calculated as twice the average.

^b Single or repeated (15—60 d, 7—12 h/d).

The varying biological models and methods make it difficult to form a unified picture of the possible bioeffects of AM radiofrequency radiation. Genotoxic effects have been reported at relatively low SAR values both in vivo and in vitro. However, the whole animal models used thus far have not provided clear evidence of cancer-related effects of radiofrequency radiation. There is also some evidence that relatively weak AM fields may affect behavior and functioning of the nervous system in vivo, but there is only limited in vitro support for the effects of weak fields on nerve or muscle cells.

To evaluate the consistency of the results, the exposure parameters found to affect or not to affect organisms in recent studies are compared in table 2 on page 251.

If there are specific biological effects that depend on the low-frequency signal used for modulating the radiofrequency carrier wave, rectification or demodulation of the carrier wave must take place in cells or tissues. The speed of the demodulation mechanism sets an upper limit for the frequency of the carrier wave that can be demodulated. The data available in table 2 are very limited for evaluating the possible dependence of the effects on carrier frequency. Carrier frequencies of 2.45 GHz or around 900 MHz (880–1000 MHz) have been used in most of the studies. Effects at relatively low SAR values have been reported at both frequencies, although there is a tendency towards the use of high SAR values or, if the time-averaged SAR is low, very high peak SAR values in experiments conducted using the 2.45-GHz carrier frequency (31, 43, 44). In the only study at 9 GHz (19), a high time-averaged SAR of 90 W/kg was used.

Although several hypothetical mechanisms have been proposed to explain the effects of weak ELF fields or ELF-modulated radiofrequency fields, there is no generally accepted biophysical theory that would predict which modulation frequencies do and which do not produce biological effects. Neither do the studies reviewed in table 2 suggest a clear dependence on modulation frequency. Effects have been reported at several frequencies, mostly below 1000 Hz. However, comparing the effects of different frequencies is difficult, as most studies have used only one modulation frequency. Two studies that tested several frequencies did not find indications of frequency dependency (40, 64). Seaman & deHaan (13) found effects at all modulation frequencies, but there were some indications that different modulations might act differently. Kolomytkin et al (32) saw a response at the 16-Hz modulation frequency, but no effects at 3, 5, 7 or 30 Hz. The results of Penafiel et al (28) indicate that there might be a range of modulation frequencies from 16 to 65 Hz that are biologically active, while no significant effects have been observed at 6 Hz or 600 Hz. Navakatikian & Tomashevskaya (33) always used 400-Hz modulation within the pulse trains, but their results indicate that the

repetition frequency (0.05 to 0.5 Hz) of the pulse trains might be of importance for the biological effects. From a practical point of view, it is worth noting that effects at relatively low SAR values have also been reported at 217 and 50–60 Hz, frequencies common in the human environment.

Many studies have used only AM exposure. Whether the effects reported in these studies depend on the presence of the modulation is not known. Three studies (19, 28, 32) report effects with amplitude modulation but not with continuous radiation, and 4 studies report different effects with continuous and AM exposures (13, 33, 34, 43).

Concluding remarks

The literature relevant to the possible specific biological effects of AM radiofrequency radiation consists of scattered observations obtained using a wide variety of experimental models and exposure parameters. The results available today do not form an internally consistent picture that would point to the existence of well-defined biological effects at certain physical parameters of AM radiofrequency radiation.

A more systematic approach in bioeffects research is needed for the risk assessment of AM radiofrequency radiation. Since any single study can answer only a very limited number of questions, a systematic approach is best achieved by increased international collaboration and the coordination of research efforts.

To study the existence of modulation-dependent effects, further studies should always include both modulated- and continuous-wave exposure groups. If reproducible effects are found, additional studies should investigate the dependence of the effects on modulation and carrier frequencies.

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