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The roles of intensity, exposure duration, and modulation on the biological effects of radiofrequency radiation and exposure guidelines

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ABSTRACT

In this paper, we review the literature on three important exposure metrics that are inadequately represented in most major radiofrequency radiation (RFR) exposure guidelines today: intensity, exposure duration, and signal modulation. Exposure intensity produces unpredictable effects as demonstrated by nonlinear effects. This is most likely caused by the biological system's ability to adjust and compensate but could lead to eventual biomic breakdown after prolonged exposure. A review of 112 low-intensity studies reveals that biological effects of RFR could occur at a median specific absorption rate of 0.0165 W/kg. Intensity and exposure duration interact since the dose of energy absorbed is the product of intensity and time. The result is that RFR behaves like a biological "stressor" capable of affecting numerous living systems. In addition to intensity and duration, man-made RFR is generally modulated to allow information to be encrypted. The effects of modulation on biological functions are not well understood. Four types of modulation outcomes are discussed. In addition, it is invalid to make direct comparisons between thermal energy and radiofrequency electromagnetic energy. Research data indicate that electromagnetic energy is more biologically potent in causing effects than thermal changes. The two likely functionthrough different mechanisms. As such, any current RFR exposure guidelines based on acute continuous-wave exposure are inadequate for health protection.

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Radiofrequency radiation (RFR); intensity; duration of exposure; modulation; specific absorption rate (SAR); biological effects

Introduction

The exposure guidelines for radiofrequency radiation (RFR) that have been adopted by the two major exposure guidelines-setting organizations – the U.S. Federal Communications Commission (Federal Communications Commission (FCC) 1997, Federal Communications Commission (FCC) 2019) and the International Commission on Non-Ionizing Radiation Protection (International Commission on Non-Ionizing Radiation Protection (ICNIRP) 1998). International Commission on Non-Ionizing Radiation Protection (ICNIRP) 2020) in Europe are widely seen as too narrowly focused on only one form of established biological effect: thermal tissue heating. The subject of RFR's thermal versus non-thermal effects in guideline setting is an old discussion dating back to the origins of nonionizing radiation's use in military radar during World War 2 as seen in early papers (Adey 1979, 1981a, 1984, 1993; Frey 1971, 1988, 1990). There are now over 50 years of literature on the inadequacy of how these increasing exposures are regulated, yet little has changed. The same arguments existed at the genesis of the issue back in the 1940s (Steneck 1985; Steneck et al. 1980).

The U.S. FCC was the first government entity to adopt exposure guidelines in 1968 for RFR. Thereafter, the fundamental acute threshold model never fully evolved with the science, although changes were added to clarify that singular model over the decades as new dosimetry measurements improved. As better understanding developed of how RFR couples with living systems, there were new additions to the guidelines regarding specific absorption rates (SARs, i.e., the rate of RF energy absorbed per unit mass of tissue); maximum permissible exposures (MPEs), different averaging times, whole-body as well as limited-body absorption allowances; wider frequency inclusions, and two-tiered allowances for the general population and occupational exposures, among others. But that has all been used within the framework of a basic acute threshold model.

The most recent FCC limits (Federal Communications Commission (FCC) 2019) for MPE to RFR essentially reaffirmed their 1997 guidelines (Federal Communications

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Commission (FCC) 1997). Exposure limits protect against adverse effects that can occur from acute short-term RFR exposures, and have been maintained in their present iteration by the FCC for the past 25 years. The same occurred with the International Commission on Non-ionizing Radiation reaffirming their 1998 guidelines (International Commission on Non-Ionizing Radiation Protection (ICNIRP) 1998) in 2020 (International Commission on Non-Ionizing Radiation Protection (ICNIRP) 2020) with minor alterations more in line with FCC's regulations on time-averaging. Both entities largely regulate for SAR based on a determination that potentially harmful biological effects can occur at a SAR level of 4.0 W/kg as averaged over the whole body.

Over the last 25–30 years, significant information has been published that in other regulated areas would have resulted in re-examination and adjustments to allowable exposure limits. This has not been the case with these two groups which adhere to a model based on obsolete scientific evidence, especially in light of the new 5G network that uses higher frequencies and novel modulation forms that have never been used before in broad civilian telecommunications and which are poorly studied.

RFR is a complex entity. Its biological effects depend on many of its physical properties, including frequency, signal characteristics, direction of the incident waves relative to the exposed object, dielectric properties, and the size, shape, species, and type of the exposed object, as well as the polarization of the waves, among other parameters. It is therefore unlikely that one can easily extrapolate the effects from one form of RFR to another. The supposition that 3G radiation is safe does not necessarily imply that 5G radiation is also safe as is the current conjecture in the guidelines today. Each one has to be investigated separately. The FCC and ICNIRP guidelines are not only obsolete; they are also inaccurate and incomplete regarding today's chronic, long-term, low-level, simultaneous, multi-frequency exposures. This has been voiced repeatedly by scores of authors over the years, now far surpassing the point of redundancy.

This paper focuses on three complex and interrelated exposure parameters – intensity, duration of exposure, and modulation – that are inherent in the FCC/ICNIRP guidelines for RFR allowances, highlighting studies that demonstrate the fallacy of such a limited approach. RFR – the electromagnetic energy used in all wireless communication – indisputably affects many biological functions in humans and non-humans alike, at all intensities and frequencies of exposures thus far studied (Levitt and Lai 2010; Levitt et al. 2021a, 2021b). Effects depend on many factors that affect energy absorption and features intrinsic to the radiation, the three most important of which are discussed below.

Intensity of exposure

Over the past several decades, there have been heated debates on the threshold intensity that can affect biological functions which are used in the setting of exposure guidelines. Few realize that the results of only two sets of experiments – De Lorge and Ezell (1980) and De Lorge (1984), – formed the backbone for most of the international exposure guidelines today. Although much work on this aspect since the 1980s continued throughout the ensuing years, the fundamental premise upon which the original SAR was formulated has become entrenched, even though the original studies were limited in scope.

In the de Lodge studies, based on 'work-stoppage' behavior in rats and monkeys, i.e., the animals ceased performing tasks they had previously been trained to do with food rewards, the threshold was found to be at a SAR of 4 W/kg. In the monkey model, it also corresponded to a rise in whole body temperature of 1°C as measured by rectal thermometers. Different versions of exposure guidelines used in different countries are basically variations of this threshold. The question is: Is this SAR level still valid based on recent research? And more to the point – was it ever valid to begin with? A closer analysis of these two studies is described below.

Specific absorption rate (SAR)

The SAR is the essential biological metric used in the current FCC/ICNIRP exposure guidelines. As mentioned above, the entire basis for the 4 W/kg SAR allowance is based literally on just two studies of observed animal behavior – De Lorge and Ezell (1980) and De Lorge (1984). SARs are almost impossible to accurately study in living systems and are therefore typically computer modeled or conducted on phantom models. In human studies, SAR can only be determined using computer and/or phantom models/calculations. (Practically, incident power density is a poor predictor of energy absorption.) But such simulations leave much to be desired regarding accuracy once transferred to far more complex living systems.

Historically, a toxicology model has been used to develop and set RFR guidelines. But as far back as 1990, the accuracy of that approach – which has been used for all EMF funding, study design, and analysis of experimental research – was called into question by Frey (1990). Toxicology models work from dose–response relationships – the greater the dose, the greater the effect. Many EMF studies have found nonlinear effects, e.g., low dose/intensity EMF exposures have shown higher effects than higher dose. It is well known that biological responses are nonlinear with respect to dosage

(e.g., see Calabrese and Baldwin 2001; Diamond 2005; Salehi et al. 2010). Adey (1984) proposed a nonlinear interaction of EMF with the cell membrane. Related to this is that Selye (1951) proposed a "general-adaptation-sydrome" of organisms in response to stressors. The stages of the general adaptation syndrome are: alarm response (a response to a stimulus); adaptation (a feedback adjustment to the stimulus); and exhaustion (deletion of adjustment that could lead to dire consequences). The timelines of the stages of response could depend on the duration of exposure and the strength of the stimulus. Thus, if responses to RFR exposure behave this way, both exposure duration (acute/chronic) and intensity would affect the observed outcome. To continue to use a classic toxicology model may be inappropriate and a more accurate biological model should be formulated.

Most biological studies do, however, require an understanding of the dose–response relationship. SAR is the rate used as a measurement of dosing (the total absorption is specific absorption (SA) = SAR × time). It is not a perfect metric, but so far, there is no better way to gauge the dosage of RFR.

SAR is considered acceptable as a dosage measurement of RFR absorption in stationary objects, e.g., cell phone usage, but is questionable in moving objects since the pattern of absorption changes with the orientation of the object. (It has been shown since the 1980s that effects of RFR depend on the orientation of exposure, among many other variables, e.g., Lai et al. 1984.) In actively moving experimental animals, the SAR would average out; whole-body averaged SAR can therefore be considered reliable in some circumstances. However, exposure of animals in groups can pose problems. For instance, rodents tend to congregate together – with the shape of a group itself then becoming an uncontrollable variable.

In vitro studies have problems too. In *in vitro* studies, the type of cells, e.g., in suspension or monolayer, becomes an important consideration. Coupling between RFR and the medium is generally poor, so high power densities are used in order to achieve certain SARs. Absorption patterns of cell cultures are far from uniform (Guy et al. 1999), but generally average SAR in the culture should be used. Also, meniscus formed by the culture medium and container surface can act like an antenna to concentrate RFR energy.

SARs in certain body organs should be given special attention. These include the brain, ear, eye, salivary glands, and skin. It is not logical to separate the body into "essential" and "non-essential" organs as in most existing RFR exposure guidelines today with different classifications for "appendage" versus "non-appendage" that allow significantly higher SARs to human arms, legs, ears and other body areas (Federal Communications Commission (FCC) 2019; International Commission on Non-Ionizing Radiation Protection (ICNIRP) 2020).

RFR effects have been observed at low intensities (<0.4 W/kg) – a list of which is included in Supplement 1 – far below the guidelines. This points to both the nonlinearity of how living systems couple with nonionizing radiation as well as the inadequacy of acute thresholds. The studies encompass many different biological effects to myriad systems, including: apoptosis induction, adrenal gland activity, blood–brain barrier permeability, brain transmitter levels, calcium concentration in heart muscle, calcium efflux, calcium movement in cells, cell growth, cognitive functions, cellular damage in liver, decreased cell proliferation, embryonic development, endocrine changes, enolose activity, genetic effects, hippocampal neuronal damage, immunological functions, kidney development, memory functions, latency of muscular contraction, membrane chemistry, nerve cell damage, metabolic changes, neural electrical activity, oxidative stress, plant growth, prion level, protein changes, renal injury, serum testosterone concentration, heat-shock protein induction, testis morphology, testosterone synthesis, thymidine incorporation, and ultrastructural alteration in cell cytoplasm. In fact, there are not many physiological functions in humans, animals, or plants that are not affected by low-level RFR.

As reflected in Supplement 1, SARs at which effects were observed were available from 112 studies. Of these, 75 (67%) were *in vivo* exposure studies with whole body/organ SARs available. The other 37 (33%) studies were *in vitro* experiments. Thus, the SARs used can be considered as the averaged SARs of the exposed objects (i.e., animal or cell culture). Most of the studies were carried out with RFR of <2500 MHz. There were several studies with millimeter waves. In addition, 52 (46%) of the studies were acute-exposure (i.e., one-time) experiments and 60 (54%) were chronic/repeated-exposure experiments. These data give a median level of 0.0165 W/kg. (The mean is 0.044 W/kg.) (Median intensity instead of mean is more appropriate here because distribution of the data points is not normal (Shapiro-Wilk Test)). It must be pointed out that the SARs reported in the analysis are those chosen to be used by the researchers and are not based on a dose–response study. Therefore, they are not the threshold SARs of responses. The data simply indicate that biological effects can occur at a level which is much lower than most current international RFR exposure guidelines.

The level at which biological effects occur represents data from *in vivo* and *in vitro* and acute and chronic/repeated-exposure experiments. There is a very wide range of effects seen. With an exposure that induces a SAR of 0.0165 W/kg, and using a ten-fold protection, the SAR would be 0.00165 W/kg (i.e., 1.65 mW/kg). For rate of energy absorption in body organs, 0.00165 W/kg is far below the maximum level allowed in the guidelines (whether over 1 or 10 gm of tissue as per FCC/ICNIRP allowances). Given the large body of work as illustrated in Supplement 1, the SAR at, or below, 4 W/kg as a safe threshold is insupportable.

Duration of exposure

The duration of exposure is another important factor in biological effects. Other than demarcations for whole body exposures averaged over 30 minutes and local body areas averaged over 6 minutes, neither FCC nor ICNIRP address duration, especially pertaining to long-term and low-level RFR exposures. These are prevalent in both near-field exposures to people with WiFi routers, for example, as well as cell phones, and far-field exposures from infrastructure that have created chronic rising ambient background levels (Levitt et al. 2021a). The guidelines are written only for short-term acute durations.

In determining the 4 W/kg SAR limit, FCC and ICNIRP assume that the exposure parameters of just two studies in which duration plays a role – De Lorge and Ezell (1980) for 40 min, and De Lorge (1984) for 60 min – were enough to extrapolate to all other exposures regarding safety limits. This is insupportable given the adverse effects associated with RFR at much lower intensities and significantly longer exposure durations.

A close examination of the two 1980′s de Lodge studies (De Lorge 1984; De Lorge and Ezell 1980), that were used to determine the current effective SAR limit at 4 W/kg contain not only problems inappropriately used to set limits but also the complexities of duration in general. For instance, the animals used in the de Lodge studies were actually exposed to RFR many times at different intensities, i.e., in the De Lorge and Ezell (1980) rat model study, each episode was of 40 min duration; and in the De Lorge (1984) monkey model (*macaca mulatta*) study, each episode was of 60 min duration. The same test animals were used repeatedly during different sessions over many days. But since we do not know if animals "remember" or "forget" previous exposures and simply adjust temporarily, we can't even be sure that the behavioral effects seen were due to acute exposures. Animals may have thermoregulated in idiosyncratic ways per animal, per species, and at different times. The De Lorge (1984) monkey study concluded that core body rise of 1°C was a better predictor of behavior disruption than SAR or power density. The role of duration is completely unclear given adaptation response. These two studies therefore should not be the basis used for SAR or duration extrapolations.

A list of long-term/repeated and short-term exposure duration studies are contained in Supplement 2. The table contains several relevant studies in which different durations of RFR exposure were studied on various and different biological endpoints. The majority of the studies, as expected, show that long-term exposure is more effective in causing effects than short-term exposure; exceptions were caused by adaptation/adjustment of the biological system studied. Since the dosage (i.e., total energy deposited in the body by RFR) is the product of SAR and time of exposure (e.g., SAR × exposure duration), a long-term exposure paradigm is like drug infusion versus medicinal pills that can allow time for an organism to adapt/adjust to the drug's effects but also makes the response more complicated.

This is observed in several studies listed in Supplement 2: Balakrishnan et al. (2014) and Eghlidospour et al. (2017) showed that some effects appeared not to be exposure-duration dependent, which could be due to a fast adaptation process, while Hidisoglu et al. (2016), Kumar et al. (2016), Sefidbakht et al. (2014), Shahi et al. (2021), and TsybuTsybulin et al. (2013) reported an opposite effect (different from acute exposure) after long-term exposure, which could be an over-compensation by activation of secondary mechanisms; and Hou et al. (2015) reported a diminished effect after longer term of exposure.

It is not unusual to see changes in effects in research after longer term exposures since feedback mechanisms in all living organisms play a critical role in homeostasis. Effects based just on duration are complex; simple exposure start-and-end points do not paint a clear picture of when effects begin or necessarily end. There are three basic phases of response to stressors – alarm, adaptation, and exhaustion – proposed by Selye (1951). For example, a response at even shorter duration of exposure may have occurred and gone unnoticed, after which the system adjusted, compensated and returned to normal after a longer period of exposure. But if exposures continue or are repeated, systems can break down and effects are then observed. Another factor is the sensitivity of the research technique used – e.g., a response may be present after short-term exposure but have gone undetected due to the limit of assay sensitivity. Thus, change occurance regarding duration is highly unpredictable, with the basic physiology of the system being studied also playing a principal role.

What we do know is that the supposition that all exposures are the same above and below the SAR threshold set by FCC/INCIRP is fundamentally flawed in light of the most current research. One feasible and logical solution to such uncertainties regarding duration as an exposure factor would be to adopt an SAR level commensurate with the studies summarized in Supplement 1 at no higher than 0.00165 W/kg, no matter the exposure conditions.

Modulations

Information-carrying technology – meaning all TV/radio/telecommunications transmission, etc. – requires modulation to function. Modulation entails the transmission carrier-wave (which is generally in the form of continuous-wave radiation) that is used to get from the transmitter to the user-destination being altered in some form, otherwise carrier-waves would just sound like static. Carrier-wave radiation is therefore encrypted with content/information via the way it is modulated, imposing/altering some aspect of a signal or waveform – such as frequency, amplitude, shape, phase, and/or combinations of these – onto the carrier-wave which can then be extracted (demodulated) and used at the receiver-end within its transmission or delivery range. This is what allows one to see a picture on a screen or hear a voice over a cell phone or radio. The problem is research shows living cells can demodulate the signals too, i.e. act as a 'receiver' and take information from modulation not unlike a radio that demodulates signals to enable listeners to hear voice or music (Silny 2007).

Modulation has become increasingly complex over the decades. It has been argued that the modulation process itself so alters the carrier-wave that only the modulation characteristics really matter. Plus, modulation of carrier-waves (in all its forms) often involves extremely low frequency (ELF) components, especially in today's broadband applications (Panagopoulos 2019; Panagopoulos et al. 2021). But both modulation and carrier-waves are biologically active as separate entities and/or when combined, and thus both are important biological factors in guidelines setting.

The long-debated question is whether the many forms of modulation are more – or differently – biologically active than the carrier wave alone? And do they act synergistically in ways that are greater than the sum of their component parts? FCC/INCIRP exposure guidelines only take carrier waves into consideration and have long been criticized for not considering modulation as a separate entity with effects of its own.

It is generally believed that modulated RFR is more biologically active than continuous-wave (CW) radiation, i.e., the carrier-wave. To understand the biological and possible hazardous health effects of RFR, it is therefore important to understand modulation effects. Below we discuss what is known about modulation from the research literature (mostly from 1990 to date) and examine the claim that modulation makes RFR more biologically significant. Studies of modulation effects are predictably contradictory, but enough research exists to indicate exposure guidelines that do not take modulation into consideration are insufficient. This could be especially true with 5G on the immediate horizon using signaling characteristics – such as complex phasing, beam steering, and 'MassiveMimo' (multiple-in, multiple-out sourcing) – and frequency ranges (in high millimeter wave ranges) that have never been used before in broad civilian-based communications. 5G requires dense small-cell infrastructure mounted on utility poles close to the population (Levitt et al. 2021a). There are presumptions of safety under current exposure guidelines regarding 5G that are alarming many experts (Blackman and Forge 2019; Hardell et al. 2021; Levitt et al. 2021a). The majority of papers written on modulation conclude that its role in guideline setting may be underestimated.

Continuous-wave (CW) RFR causes effects without modulation

The logical place to investigate the topic is an examination of effects of CW-RFR without modulation. There is research showing no significant biological effects of CW-RFR (Table 1a) but there are also studies that reported CW-RFR effects too (Table 1b). The reason why CW-RFR produced effects in some studies but not others is unknown. Both types of studies (with "effect" and "no effect" outcomes) involved many different biological endpoints, exposure intensities, and duration of exposure – with no discernible differences. A possible explanation is that different tissue types respond differently to CW-RFR. But that just adds another level of inquiry. One of the most puzzling observations is when CW caused an effect but modulation did not (e.g., Kubinyi et al. 1996; Luukkonen et al. 2009). In some studies, a modulated field produced an effect that was not produced by CW. These observations may indicate that the CW carrier-wave itself and modulation act on different mechanisms.Studies on Effects of RFR Modulation.(*CW and modulation produce different effects; **Different modulations producedifferent effects: ***modulation produces effect, but not CW; # modulation andCW have different potencies.)Study subjectWaveform and exposure conditionsResults*Arber and Lin (1985)

Table 1. (a and b) Studies on Effects of Continuous-Wave RFR.

Table 1a – *CW caused no effect*	
Bolshakov and Alekseev (1992)	CW 900-MHz RFR (0.5 W/kg) did not affect bursting responses of Lymnea neurons.
Campisi et al. (2010)	Rat neocortex astrocytes ROS and DNA damage, CW at power density of 0.026 mW/cm^2
d'Ambrosio et al. (2002)	1748-MHz CW RFR (15 min, ~5 W/kg) did not cause micronucleus formation in peripheral human blood.
Dawe et al. (2008)	Caenorhabditis elegans exposed to CW 1800-MHz RFR (2.5 h; 1.8 W/kg) did not show expression of Hsp16-1 heat shock gene.
Franzellitti et al. (2010)	CW 1800-MHz RFR (2 W/kg) did not cause DNA damage in human trophoblast HTR-8/SVneo cells.
Hirose et al. (2006)	Exposure of human glioblastoma A172 cells (24 or 48 h; 0.08 W/kg) or human IMR-90 fibroblasts (28 h; 0.08 W/kg) did not induce p53-dependent apoptosis, DNA damage, or other stress response.
Höytö et al. (2008)	Human SH-SY5Y neuroblastoma and mouse L929 fibroblast cells exposed to 872-MHz RFR for 1 or 24 h at 5 W/kg showed no oxidative effects.
Huber et al. (2002)	CW 900-MHz RFR (30 min; 1 W/kg) did not affect sleep and wake EEG in human.
Lim et al. (2005)	Exposure of human leukocytes to 900-MHz CW RFR (20 min; 1 or 4 h, 0.4, 2, 3.6 W/kg) did not affect expression of HSP70 and HSP27.
Markkanen et al. (2004)	CW 2450-MHz RFR (1 h, 0.4–3 W/kg) did not affect UV-induced apoptosis in yeast cells.
Nakamura et al. (2003)	CW 915-MHz RFR exposure (90 min; 0.4 W/kg) did not affect blood estradiol and progesterone, on splenic natural killer cell activity, on the uteroplacental circulation of pregnant rats.
O'Connor et al. (2010)	CW 900-MHz RFR exposure (30 min; 0.012–2 W/kg) did not affect cellular Ca^{2+} signal in here types of cells.
Platano et al. (2007)	CW 900-MHz RFR 1–3 periods of 90s; 2 W/kg) did not affect Ba^{2+} currents through voltage-gated calcium channels in rat cortical neurons.
Roux et al. (2011)	Exposure to CW 900-MHz RFR (10 min; 0.0026 or 0.073 W/kg) did not affect gene expression in human keratinocytes.
Sakuma et al. (2006)	CW 2142.5-MHz RFR (2 and 20 h; 0.8 W/kg) produced no DNA strand breaks in human glioblastoma A172 cells and normal human IMR-90 fibroblasts from fetal lungs.
Sakurai et al. (2011)	CW 2450-MHz RFR (1, 4, 24 h; 1, 5, 10 W/kg) did not affect gene expression in human glial cells.
Salford et al. (1997)	Rats inoculated with rat glioma cells and exposed to CW 915-MHz RFR(starting from day 5 after inoculation for 7 h/day; 5 days per week for 2–3 weeks) did not affect tumor size.
Schwartz et al. (1990)	CW 240 MHZ RFR did not affect calcium efflux from frog heart (30 min; 0.00015–0.003 W/kg)
Schwartz and Mealing (1993)	CW 1000 MHz RFR (32 min; 0.0032 – 1.6 W/kg) did not affect calcium movement and contractile force of frog heart atrial strips.
Sekijima et al. (2010)	Human A172 (glioblastoma), H4 (neuroglioma), and IMR-90 (fibroblasts from normal fetal lung) cells exposed to CW 2142.5 MHz RFR (96 h; 0.08. 0.25, 0.8 W/kg) showed no significant changes in cell growth and viability, and gene expression.
Simko et al. (2006)	Human Mono Mac 6 cells exposed to CW 1800-MHz RFR (60 min; 2 W/kg) did not show increase in free radicals and Hsp 70 expression.
Somosy et al. (1991)	CW 2450-MHz RFR (0.0024–2.4 W/kg) did not change cell surface free negative charges in mouse embryo 3T3 fibroblasts.
Somosy et al. (1993)	Exposure to CW 2450-MHz RFR (0.5 and 1 mW/cm^2) did not affect pyroantimonate precipitable calcium content of mouse intestinal epithelial cells.
Speit et al. (2007)	Human fibroblasts (ES1 cells) exposed to CW1800-MHz RFR (1–24 h; 2 W/kg, intermittently). No effects on DNA damage, micronucleus formation were observed. V79 Chinese hamster cells also showed no response.
Speit et al. (2013)	Human HL-60 cells exposed to CW 1800-MHz RFR (24 h; 1.3 W/kg 5 min ON'10 min OFF) showed no genetic effects.
Takeda et al. (2010)	Human A172 (glioblastoma), H4 (neuroglioma), and IMR-90 (fibroblasts from normal fetal lung) exposed to CW 2142.5 MHz RFR (up to 96 h; 0.08. 0.25, or 0.8 W/kg) showed no effects on cell proliferation and gene expression.
Thorlin et al. (2006)	Rat astroglial (24 h; 27 W/kg) and microglial (8 h, 3 W/kg) exposed to CW 900-MHz RFR showed no effects on two pro-inflammatory cytokines interleukin 6 (Il 6) and tumor necrosis factor-alpha (Tnfa) and morphology.
Valbonesi et al. (2014)	Exposure to CW1800 MHz RFR (4, 16, or 24 h; 2 W/kg) did not affect Hsp70 transcription in PC12 cells.
Wang et al. (2005a)	Exposure to CW 2450-MHz RFR (2 h; 5–200 W/kg) did not induce cancer-like changes or initiate malignant or synergistic transformation in mouse C3H10T1/2 cells.
Zeni et al. (2003)	Human lymphocytes exposed to a CW 900-MHz RFR (6 min followed by a 3-h pause (14 on/off cycles); 1.6 W/kg) showed no effect on micronucleus formation and proliferation index.
Zmyslony et al. (2004)	Rat lymphocytes exposed to CW 930-MHz RFR (5 or 10 min; 1.5 W/kg) did not show significant changes in free radicals, (But, RFR acts synergistically with $FeCl_2$ to enhance free radical production).
Table 1b- ***CW caused effects***	
Detlavs et al. (1996)	Rat with dermal wounds exposed to CW 53.53 and 42.19 GHz RFR (30 min/day, first 5 days after wound infliction; 10 mW/cm^2) showed decreased inflammation exudation.
Elekes et al. (1996)	CW 2450 MHz RFR (3 h/day for 6 days; 0.14 W/kg) increased antibody producing cells in spleen of male mice.
Grasso et al. (2020)	CW 900-MHz RFR (20 min; 7 V/m) changed viability, apoptoic pathway, skeletal pathway in olfactory ensheathing cells.
Houston et al. (2018)	CW 1800-MHz RFR (4 h; 0.15 or 1.5 W/kg) induced reactive oxygen species and oxidative DNA damage in mouse spermatozoa.
Luukkonen et al. (2009)	CW 872-MHz RFR (1 h; 5 W/kg) produced oxidative effects and DNA damage in human SH-SY5Y neuroblastoma cells.
Marinelli et al. (2004)	CW 900-MHz RFR exposure (2–12 h; 0.001 W/kg) caused DNA damage and activation of apoptotic pathway in human T-lymphoblastoid leukemia cells. Longer exposure (24–48 h) caused silencing of pro-apoptotic signals and activation of genes involved in both intracellular and extracellular pro-survival signaling.
Mazor et al. (2008)	CW 800-MHz RFR exposure (72 h; 2.9 or 4.1 W/kg) increased aneuploidy in human lymphocytes.
Misa-Agustiño et al. (2015)	Thymus of rat exposed to CW 2450-MHz RFR (30 min; mean thymus SAR 0.046–0.482 W/kg) showed changes in the endothelial permeability and vascularization of the thymus, and is a tissue-modulating agent for Hsp90 and glucocorticoid receptors.
Miyakoshi et al. (2005)	CW 1950-MHz RFR (1–2 h; 1, 2, 10 w/kg) inhibited phosphorylation of Hsp27 in MO54 human glioma cells
Pavicic and Trosic (2008)	V79 Chinese hamster lung fibroblasts exposed to CW .864-MHz (0.08 W/kg) and 935-MHz (0.12 W/kg) RFR (1,2, or 3 h) showed exposure time-dependent increases in growth rate.
Sagioglou et al. (2016)	Drosophila melanogaster exposed to CW 100, 395, 682, 900 MHz RFR (6 or 60 min on the 6th day or daily for the first 6 days of their life; 0.0003–0.4 W/kg) showed increased apoptotic cell death.
Salford et al. (1994)	Rats exposed to CW 915-MHz RFR (2 h; 0.016–5 W/kg) showed effects on blood–brain barrier.
Shahin et al. (2013)	Exposure to CW 2450-Mz RFR for 2 h/day for 45 days at 0.023 W/kg caused oxidative stress and affected implantation and pregnancy in mice.

(*Continued*)

Table 1. (Continued).

Table 1a – *CW caused no effect*	
Sukhotina et al. (2006)	Hamster pineal exposed to CW 1800-MHz RFR (7 h; 0.008, 0.08, 0.8, or 2.7 W/kg) showed increased release of melatonin at 0.8 W/kg.
Sun et al. (2017)	Human HL-60 cells exposed to CW 900-MHz RFR at 120 μW/cm^2, 4 h/day for 5 days induced mitochondrial oxidative DNA damage.
Tattersall et al. (2001)	Exposure to 700-MHz CW RFR (5–15 min; 0.0016 and 0.0044 W/kg) affected excitability of rat hippocampal slices.
Testylier et al. (2002)	Exposure to CW 2450 MHz RFR (1 h; 6.52 W/kg) caused a decrease in acetylcholine release from the hippocampus of the rat.
Tkalec et al. (2013)	Earthworms exposed to 900-MHz CW RFR for 2 h at 0.00013–0.00933 W/kg caused oxidative stress and DNA damage.
Wang et al. (2005b)	Rat cerebral cortical neurons exposed to CW 900-MHz RFR (12 h or 2 h/day for 6 days; 0.0015–0.003 W/kg) showed an increased expression of GABA receptors.
Xie et al. (2021)	Mouse bone marrow stem cells exposed to CW 900 MHz RFR (4 h/day for 5 days; SAR 0.00025 W/kg) showed increased free radicals and mitochondrial unfolded protein.
Yang et al. (2001)	Pig retinal ganglion cells exposed to CW 2450-MHz RFR (1 h; 30 mW/cm^2) showed intracellular morphological changes and apoptosis.

Helex aspersa neuronal electrical activity2450-MHz CW (12.9 W/kg) or noise-amplitude-modulated (20% AM at 2 Hz-20 KHz; 6.8–14.4 W/kg) RFR for 60 minAt 21°C, CW RFR inhibited spontaneous activity and reduced input activity, whereas modulated field caused excitatory responses by increasing membrane resistance. **Bachmann et al. (2006)Changes in EEG rhythm energy and dynamicsHuman subjects exposed to 7, 14, 21, 40, 70, 217, or 1000 Hz-modulated 450 MHz RFR at scalp power density 0.16 mW/cm^2 (SAR 0.35 W/kg)Exposure caused increases in EEG energy levels; more intense at higher modulation frequencies and higher EEG rhythms. **Barbault et al. (2009)Different types of tumors in human patients27.12 MHz RFR amplitude modulated at 0.01 Hz to 150 KHz using an intrabuccal applicator; 60 min 3 times a day, SAR in head 0.0001–0.1 W/kgBeneficial tumor-specific AM frequencies observed. Behari et al. (1998)Na^+–K^+-ATPase in brainRats exposure for 30–35 days (3 h/day; 6.11–9.65 W/kg) to 147-MHz and sub-harmonics of 73.5 and 36.75 MHz amplitude-modulated at 16 and 76 Hz.Increased Na^+-K^+-ATPase activity was observed in both modulations independent of carrier-wave frequency compared to sham-control. No significant difference between the two types of modulation. ***Bolshakov and Alekseev (1992)Electrical activity of Lymnea stagnalis neuronsNeurons exposed to a 900 MHz RFR CW or pulse-modulated at rates ranging from 0.5 to 110 pps.Rapid, burst-like changes in the firing rate of neurons occurred at a threshold of 0.5 W/kg with exposure to the modulated field, not with CW. Effect was independent of modulation frequency.***Campisi et al. (2010)Rat neocortex astrocytes oxidative effectsAstrocytes exposed to CW or amplitude-modulated at 50-Hz 900-MHz RFR for 20 min at power density of 0.026 mW/cm^{2}Increased level of reactive oxygen species and DNA fragmentation after exposure to the modulated field, no effect with CW field. **Croft et al. (2010)Resting alpha EEG in humans2G and 3G2G affected EEG in young adults, but no effect with 3G exposure.***Czerska et al. (1992)Transformation of human lymphocytesLymphocytes exposure to CW or pulsed (1 μs pulses at pulse repetition rate of 100–1000 pps) 2450-MHz RFR for 5 days; 12.3 W/kg.At 37°C, pulsed radiation enhanced transformation, whereas CW did not. ***d'Ambrosio et al. (1995)Micronucleus formation in peripheral human bloodBlood samples exposed to 9000 MHz CW or 50-Hz amplitude modulated RFR, 10 min; 90 W/kg. A significant increase in micronuclei was found following AM RFR exposure, butnot with CW.***d'Ambrosio et al. (2002)Micronucleus formation in peripheral human bloodBlood samples exposed to 1748-MHz CW or phase modulated fields for 15 min; ~ 5 W/kg.Phase-modulated, but not CW, caused an increase in micronucleus.*, ***Detlavs et al. (1996)Wound inflammation exudation in ratsWounds exposed to CW 53.53 or 42.19 GHz or 42.19 GHz RFR with frequency modulation band 200-MHz wide (30 min daily on the first 5 days after wound infliction).CW exposure decreased whereas modulated field exposure increased inflammatory exudation.

Modulated field elevated RNA level, CW was without effect.**Diem et al. (2005)DNA damage in human fibroblasts and rat granulosa cellsCells exposed to 1800 MHz RFR with amplitude-modulation at 217 Hz, or GSM-talk mode, intermittent (5 min on/10 min off) or continuously, for 4. 16, or 24 h; 1.2 or 2 W/kgDNA damage found after 16 h of

exposure. Intermittent showed a stronger effect than continuous exposure, modulations produced the same effect as intermittent exposure.**,***Dutta et al. (1994)Enolase in *E. coli*Escherichia coli cultures containing a plasmid with a mammalian gene for enolase were exposed for 30 min to 147-MHz carrier wave amplitude-modulated at 16 or 60 Hz, 0.05 W/kgModulation at 16 Hz increased and at 60 Hz decreased enolase activity. Exposure to 16- and 60-Hz fields caused increase and decrease in activity,

Table 2. Studies on Effects of RFR Modulation.(*CW and modulation produce different effects; **Different modulations producedifferent effects: ***modulation produces effect, but not CW; # modulation andCW have different potencies.)

	Study subject	Waveform and exposure conditions	Results
*Arber and Lin (1985)	Helex aspersa neuronal electrical activity	2450-MHz CW (12.9 W/kg) or noise-amplitude-modulated (20% AM at 2 Hz-20 KHz; 6.8–14.4 W/kg) RFR for 60 min	At 21°C, CW RFR inhibited spontaneous activity and reduced input activity, whereas modulated field caused excitatory responses by increasing membrane resistance.
**Bachmann et al. (2006)	Changes in EEG rhythm energy and dynamics	Human subjects exposed to 7, 14, 21, 40, 70, 217, or 1000 Hz-modulated 450 MHz RFR at scalp power density 0.16 mW/cm^2 (SAR 0.35 W/kg)	Exposure caused increases in EEG energy levels; more intense at higher modulation frequencies and higher EEG rhythms.
**Barbault et al. (2009)	Different types of tumors in human patients	27.12 MHz RFR amplitude modulated at 0.01 Hz to 150 KHz using an intrabuccal applicator; 60 min 3 times a day, SAR in head 0.0001–0.1 W/kg	Beneficial tumor-specific AM frequencies observed.
Behari et al. (1998)	Na^+–K^+-ATPase in brain	Rats exposure for 30–35 days (3 h/day; 6.11–9.65 W/kg) to 147-MHz and sub-harmonics of 73.5 and 36.75 MHz amplitude-modulated at 16 and 76 Hz.	Increased Na^+-K^+-ATPase activity was observed in both modulations independent of carrier-wave frequency compared to sham-control. No significant difference between the two types of modulation.
***Bolshakov and Alekseev (1992)	Electrical activity of Lymnea stagnalis neurons	Neurons exposed to a 900 MHz RFR CW or pulse-modulated at rates ranging from 0.5 to 110 pps.	Rapid, burst-like changes in the firing rate of neurons occurred at a threshold of 0.5 W/kg with exposure to the modulated field, not with CW. Effect was independent of modulation frequency.
***Campisi et al. (2010)	Rat neocortex astrocytes oxidative effects	Astrocytes exposed to CW or amplitude-modulated at 50-Hz 900-MHz RFR for 20 min at power density of 0.026 mW/cm^2	Increased level of reactive oxygen species and DNA fragmentation after exposure to the modulated field, no effect with CW field.
**Croft et al. (2010)	Resting alpha EEG in humans	2G and 3G	2G affected EEG in young adults, but no effect with 3G exposure.
***Czerska et al. (1992)	Transformation of human lymphocytes	Lymphocytes exposure to CW or pulsed (1 μs pulses at pulse repetition rate of 100–1000 pps) 2450-MHz RFR for 5 days; 12.3 W/kg.	At 37°C, pulsed radiation enhanced transformation, whereas CW did not.
***d'Ambrosio et al. (1995)	Micronucleus formation in peripheral human blood	Blood samples exposed to 9000 MHz CW or 50-Hz amplitude modulated RFR, 10 min; 90 W/kg.	A significant increase in micronuclei was found following AM RFR exposure, butnot with CW.
***d'Ambrosio et al. (2002)	Micronucleus formation in peripheral human blood	Blood samples exposed to 1748-MHz CW or phase modulated fields for 15 min; ~ 5 W/kg.	Phase-modulated, but not CW, caused an increase in micronucleus.
*, ***Detlavs et al. (1996)	Wound inflammation exudation in rats	Wounds exposed to CW 53.53 or 42.19 GHz or 42.19 GHz RFR with frequency modulation band 200-MHz wide (30 min daily on the first 5 days after wound infliction).	CW exposure decreased whereas modulated field exposure increased inflammatory exudation. Modulated field elevated RNA level, CW was without effect.
**Diem et al. (2005)	DNA damage in human fibroblasts and rat granulosa cells	Cells exposed to 1800 MHz RFR with amplitude-modulation at 217 Hz, or GSM-talk mode, intermittent (5 min on/10 min off) or continuously, for 4. 16, or 24 h; 1.2 or 2 W/kg	DNA damage found after 16 h of exposure. Intermittent showed a stronger effect than continuous exposure, modulations produced the same effect as intermittent exposure.
,*Dutta et al. (1994)	Enolase in *E. coli*	Escherichia coli cultures containing a plasmid with a mammalian gene for enolase were exposed for 30 min to 147-MHz carrier wave amplitude-modulated at 16 or 60 Hz, 0.05 W/kg	Modulation at 16 Hz increased and at 60 Hz decreased enolase activity. Exposure to 16- and 60-Hz fields caused increase and decrease in activity, respectively. Sham and CW 147-MHz RFR (at 0.5 W/kg) had no significant effect on enolase activity.
#Elekes et al. (1996)	Antibody producing in spleen	Mice exposed to 2450 MHz RFR (CW or amplitude modulated with 50- Hz square-wave (3 h/day for 6 days; 0.14 W/kg).	AM field more potent than CW field. Increased antibody producing cells in male mice only. No effect observed in female mice.

(*Continued*)

Table 2. (Continued).

	Study subject	Waveform and exposure conditions	Results
***Franzellitti et al. (2010)	DNA damage in human trophoblast HTR-8/SVneo cells	Cells were exposed to CW, GSM-217, or GSM-talk (5 min On/ 10 min off) 1800 -MHz RFR; 2 W/kg.	Increased DNA damage was observed with modulated field exposure, but not with CW field.
***Gapeyev et al. (2014)	Mouse leukocytes H_2O_2 production and DNA damage	Cells exposed to 42.2 GHz RFR; 0.1 mW/cm^2, 1 Hz modulation frequency; 20 min).	Modulated field increased H_2O_2 production; reduced X-ray-induced DNA damage.
*Grasso et al. (2020)	Cytoskeleton proteins in olfactory ensheathing cells	Cells exposed to 900-MHz CW or 50-Hz sinusoidal amplitude-modulated fields (10, 15, or 20 min; 7 V/m (0.013 mW/ cm^2)).	CW and amplitude-modulated fields produce different patterns of responses on viability and apoptotic pathways and cell markers.
**Gulati et al. (2020)	DNA damage in human lymphocytes	Cells exposed to UMTS signals at different frequency channels used by 3 G mobile phone (1923, 1947.47, and 1977 MHz) for 1 or 3 h; 0.04 W/kg.	DNA damage found only in cells exposed to 1977-MHz field.
*#Halgamuge et al. (2015)	Growth of soybean seedlings	Seedlings exposed to GSM-900 ($2.6–4.8 \times 10^4$ mW/kg) or CW 900 MHz RFR for 2 h; 0.39–2 mW/kg.	Epicotyl outgrowth reduced more by modulated radiation than CW, and hypocotyl outgrowth only reduced by CW at low intensity.
**Hinrikus et al. (2008)	EEG rhythms	Human subjects exposed to 450-MHz RFR pulse-modulated at 7, 14, and 21 Hz scalp power density 016 mW/cm^2 (0.303 W/kg).	RFR exposure modulated at 14 and 21 Hz enhanced the EEG power in the alpha and beta frequency bands, whereas no enhancement occurred at 7-Hz modulation frequency.
***Höytö et al. (2008)	Oxidative stress and cell death	Human SH-SY5Y neuroblastoma and mouse L929 fibroblast cells exposed to 872-MHz RFR either CW or GSM signal-modulated (1 or 24 h; 5 W/kg)	Drug-induced lipid peroxidation and caspase 3 increased with the modulated field and not with CW.
***Huber et al. (2002)	Sleep and waking EEG in humans	Pulsed modulated 900 MHz RFR (at 2, 8, 217, 1736 Hz) for 30 min at spatial peak SAR of 1 W/kg; and CW 900 MHz RFR	Pulsed-modulated field induced changes in sleep and waking EEG; no significant effect with CW field.
**Huber et al. (2005)	Regional cerebral blood flow	Human subjects exposed unilaterally to 900-MHz (30 min; 1 W/kg) "base-station -like" or "hand-set-like" signals. The latter has stronger low-frequency components.	Increased blood flow was observed in ipsilateral dosolateral prefrontal cortex only after "hand-set-like" signal exposure.
**Hung et al. (2007)	EEG-determined sleep onset	Human subjects exposed to GSM 900 signals modulated at 2, 8, or 217 Hz ("talk", "listen", and "standby" modes) and sham-exposure for 30 min.	Post-exposure, sleep latency after talk mode exposure was markedly and significantly delayed beyond listen and sham modes. Different modulation frequencies may differentially affect sleep onset.
*Kubinyi et al. (1996)	Aminoacyl-tRNA synthetase in brain and liver	Mice exposed in utero (100 min per day during 19 days of gestation) to CW or AM (50 Hz rectangular wave, 50%/50% On-Off ratio) 2450-MHz; 4.23 W/kg.	At postnatal day 24, Aminoacyl-tRNA synthetase was decreased in the brain after CW exposure whereas no effect was seen with AM RFR exposure. Aminoacyl-tRNA synthetase acivity increased in liver for both types of RFR.
**Kumar et al. (2021)	Epigenetic modulation in the hippocampus of Wistar rats	Rats exposed to 900-MHz, 1800-MHz, and 2450-MHz RFR for 2 h per day for 1-month, 3-month, and 6-month periods; 5.84×10^{-4} W/kg, 5.94×10^{-4} W/kg, and 6.4×10^{-4} W/kg, respectively.	Significant epigenetic modulations were observed in the hippocampus; larger changes with increasing frequency and exposure duration.
**Kunjilwar and Behari (1993)	AChE activity in brain	Male rats exposed to 147, 73.5 and 36.75 MHz RFR amplitude modulated at 16 and 76 Hz (3 h/day for 30–35 days; 0.1–0.14 W/kg).	Decrease in AChE activity observed in all conditions independent of carrier frequency. Effect slightly higher in 16 Hz than 76 Hz modulation.

(*Continued*)

Table 2. (Continued).

	Study subject	Waveform and exposure conditions	Results
**Lerchl et al. (2008)	Body weight	Adult male Djungarian hamsters (Phodopus sungorus) were exposed 24 h/day for 60 days to RFR at 383, 900, and 1800 MHz, modulated according to the TETRA (383 MHz) and GSM standards (900 and 1800 MHz), respectively; 0.08 W/kg.	At 383 MHz, exposure resulted in a significant transient increase in body weight, while at 900 MHz body weight increase was more pronounced and not transient. At 1800 MHz, no effect on body weight was seen.
***Lin (2021)	Microwave auditory effect		Caused by pulsed but not CW RFR
**Lin et al. (2013)	Pain relief effect	Rat dorsal root ganglia exposed using a bi-polar electrode to 500-KHz RFR with 25 msec pulse duration, 2.5/1.25 V amplitude, 5 min.	Sinusoidal wave has higher responses than square wave.
***Liu et al. (2021)	Wakefulness in mouse	Mice exposed for 9 days to 2400-MHz RFR modulated by 100-Hz square pulses (1/8 duty cycle).	Increased time of wakefulness with decreased times of non-rapid and rapid eye movement.
*López-Martín et al. (2009)	c-Fos expression in brain of picotoxin-induced seizure-prone rats	Pulse-modulated GSM and unmodulated signals; 2 h; mean SAR in brain 0.03–0.26 W/kg.	GSM-modulated and unmodulated signals produced different responses in different regions of the brain.
***Luukkonen et al. (2009)	Human SH-SY5Y neuroblastoma cells; DNA damage and oxidative effects	Cells exposed to CW or GSM-modulated (217 Hz) 872-MHz RFR for 1 h; 5 W/kg.	CW increased DNA damage and reactive oxygen species in cells treated with menadione. Modulated field had no significant effect.
***Markkanen et al. (2004)	Apoptosis in mutant yeast cells	Yeast cells exposed to 900 or 872 MHz RFR (CW or pulse modulated at 217 Hz, 0.577 ms) (1 h; 0.4 or 3.0 W/kg).	Amplitude-modulated RFR enhanced UV-induced apoptosis. No effect from CW field exposure.
**Markova et al. (2005)	Human lymphocyte chromatin conformation and ϒ-H2AX foci	905 and 915 MHz GSM900 mobile phone signals (577 μs pulses, inter-pulse waiting time of 4039 μs), for 1 h; 0.037 W/kg.	Effects observed were carrier-frequency dependent.
**Mohammed et al. (2013)	Latency of REM sleep in rats	Rats exposed to 900-MHz RFR (CW or modulated at 8 or 16 Hz) for 1 h/day for 1 month (peak SAR 0.245 W/kg).	Latency of REM sleep increased in rats exposed to field with 16-Hz modulation.
Nikolova et al. (2005)	Apoptosis-related gene transcription and DNA damage in embryonic stem cell-derived neural progenitor cells.	Cells exposed to 1710-MHz GSM signal (217 Hz rectangular pulses; width 0.576 ms) (5 min on/30 min off) for 6 or 48 h; 1.5 W/kg.	Increase in DNA damage after 6 h, but not 48 h, exposure. 48 h exposure affected gene expression.
**Nylund and Leszczynski (2006), (2010))	Proteome response in two types of human primary brain microvascular endothelial cells	900 MHz and 1800 MHz GSM signals for 1 h; 2.8 and 2 W/kg, respectively.	900 MHz RFR affected protein expression but not with 1800 MHz exposure; the two cell lines responded differently to the 900 MHz RFR.
**Ozgur et al. (2014)	Proliferation of human hepatocarcinoma cells	Cells exposed to 1, 2, 3, or 4 h (15 min on/15 min off) to 900- or 1800-MHz RFR; 2 W/kg.	1,800-MHz RFR had a larger impact on cell viability and cell injury than 900-MHz. Four hour exposure produced more pronounced effect.
**Panagopoulos and Margaritis (2010)	Reproductive capacity of Drosophila melanogaster	Drosophilia exposed to GSM 900 or DCS 1800 MHz signals for 1–21 min at 10 μW/cm^2.	Reproductive capacity decreased almost linearly with increasing exposure duration to both GSM 900 and DCS 1800 radiation. GSM 900 MHz radiation is slightly more bioactive than DCS 1800 MHz radiation.
#Panagopoulos et al. (2004)	Reproductive capacity of Drosophila melanogaster	Drosophilia exposed to GSM 900 phone modulated by human voice (0.436 mw/cm^2) or unmodulated (0.041 mW/cm^2) for 6 min per day during the first 2–5 days of their adult lives.	Decrease in reproductive capacity in both male and female flies observed; unmodulated field was less effective compared to sham-exposure.
#Penafiel et al. (1997)	Ornithine decarboxylase activity in L929 cells	L929 murine cells exposed to 835-MHz RFR (up to 24 h; 2.5 W/kg) with different types of amplitude modulation.	Effects are much more robust when the modulation causes low-frequency periodic changes in the amplitude of the carrier wave. CW is less effective.
#Persson et al. (1997)	Blood–brain barrier in rats	Rats exposed to 915-MHz RFR pulse modulated (217 Hz or 50 Hz) or CW for 2 to 960 min.	RFR caused pathological changes in the blood–brain barrier. (Effect showed at >1.5 J/kg). CW is more potent.

(*Continued*)

Table 2. (Continued).

	Study subject	Waveform and exposure conditions	Results
*Philippova et al. (1994)	Cell receptor binding	CW or rectangular wave modulated (1, 6, 16, 32, 75, or 100 pps) (15 min; 0.5–18 W/kg).	CW (1 W/kg) affected binding depending on the ligand and cell type studies; effect not dependent on modulation.
**Phillips et al. (1998)	DNA strand breaks	Human Molt-4 cells exposed to pulsed iDEN (813.5625 MHz, 2.4 or 24 μW/kg) or TDMA (836.55 MHz, 2.6 or 26 μW/kg) for 2 or 21 h.	Increase or decrease in DNA strand breaks occurred depending on intensity of exposure and the type of modulation.
*Poque et al. (2020)	RAS/MAPK activation in HuH7 human hepatocellular carcinoma cells	Cells exposed to 1800-MHz CW or GSM-modulated signals (24 h; 2 W/kg).	Modulated signal decreased phorbol-12-myristate-13-acetate maximal efficacy to activate RAS- and ERK-kinase activation. CW only decreased efficacy to activate ERK, but not RAS.
#Sagioglou et al. (2016)	Apoptosis in Drosophila melanogaster	D. melanogaster exposed to frequency modulated (50 kHz FM) or CW 100, 395, 682, 900 MHz RFR (6 or 60 min on the 6th day or daily for the first 6 days of their life; 0.0003–0.4 W/kg) showed increased apoptotic cell death.	Apoptotic cell death was observed in all conditions, FM signal has a stronger effect than CW.
#Salford et al. (1994)	Blood–brain barrier	Rats exposed to CW or pulse-modulated (8, 16, 50, and 200 per sec) 915-MHz RFR (2 h; 0.16–5 W/kg).	Significant increase in blood–brain barrier observed for both types of radiation.
**Sarimov et al. (2004)	Chromatin conformation in human lymphocytes	Cells exposed to GSM900 signals (577 μs pulses, 4039 μs between pulses) at 895, 900, 905, 910, and 915 MHz for 0.5–1 h; 0.0054 W/kg.	RFR effects differ at various GSM frequencies and vary between donors.
**Schmid et al. (2012)	EEG power during sleep in the spindle frequency range (approximately 11–15 Hz)	Human subjects exposed to 900 MHz RFR modulated at 14 or 217 Hz for 30 min; 2 W/kg.	EEG power during sleep in the spindle frequency range was increased during non-rapid eye movement sleep following the 14-Hz pulse-modulated condition. No significant effect was found following exposure to 217-Hz modulated field.
**Schneider and Stangassinger (2014)	Social memory performance in rats	Rats exposed to 900 MHz GSM or 1966 MHz UMTS RFR; 6 months; 0.4 W/kg.	At 6 months, male rats exposed to GSM, but not UMTS signal, showed a memory deficit; no significant effect on female rats.
**, ***Schwartz et al. (1990)	Calcium efflux from frog heart	Frog hearts exposed to 240-MHZ RFR CW or sinusoidally modulated at 0.5 or 16 Hz (30 min; 0.00015–0.003 W/kg).	Movement of calcium affected only with 16-Hz modulation at 0.003 and 0.00015 W/kg. No effect with CW and 0.5 Hz modulation.
**Seaman and DeHaan (1993)	Inter-beat intervals of aggregated cardiac cells from chicken embryos	19 sec exposure to 2450-MHz RFR at CW, pulsed modulated (duty cycle ~ 11%), or square-wave modulation (duty cycle 50%); 1.2–86.9 W/kg.	Decrease and increase in inter-beat intervals were observed. SAR, modulation, and the modulation-SAR interaction were all significant factors in altering the inter-beat interval. Some effects were probably nonthermal.
Semin et al. (1995)	Secondary structure of DNA	DNA exposed to RFR (4- to 8 GHz, 25 ms pulses, 0.4 to 0.7 mW/cm^2 peak power, 1- to 6-Hz repetition rate).	Irradiation at 3 or 4 Hz and 0.6 mW/cm2 peak power increased the accumulated damage to the DNA secondary structure; changing the pulse repetition rate to 1, 5, 6 Hz, as well as changing the peak power to 0.4 or 0.7 mW/cm^2 produced no effect.
Sirav and Seyhan (2011) (CW effect)	Blood brain barrier in rats	Rats exposed to CW 900 MHz (0.00426 W/kg) or 1800 MHz (0.00146 W/kg) for 20 min.	Increased blood–brain barrier permeability in male rats for both frequencies; no significant effect on female rats.
**Sirav and Seyhan (2016)	Blood brain barrier in rats	Rats exposed to 900- and 1800-MHz RFR (20 min; 0.02 W/kg) pulse modulated at 217 Hz, 577 μs.	Both types of RFR increased blood–brain barrier permeability in male rats, 1800-MHz was more potent. In female rats, only the 900-MHz field caused an effect.
*, #Somosy et al. (1991)	Morphological cell changes in mouse embryo 33T3 fibroblasts	Fibroblasts exposed to 2450-MHz CW or 16-Hz square-wave modulated RFR (0.0024–2.4 W/kg)	Modulated field more potent than CW in some effects. Some effects of modulated field not observed in CW exposure.
***Somosy et al. (1993)	Pyroantimonate precipitable calcium content of mouse intestinal epithelial cells	16-Hz square-wave modulated 2450-MHz RFR (0.5 and 1 mW/cm^2).	Rapid distribution of pyroantimonate precipitable calcium content was observed. No effect with CW exposure.
*Sukhotina et al. (2006)	Secretion of melatonin from pineal glands	Hamster pineal exposed to CW or GSM modulated 1800-MHz RFR (7 h; 0.008, 0.08, 0.8. 2.7 W/kg).	Increase in melatonin release observed for both fields at 0.8 W/kg; AT 2.7 W/kg, melatonin levels were elevated in the CW, but suppressed in the GSM exposure. (There was a 1.2°C increase in temperature.)

(*Continued*)

Table 2. (Continued).

	Study subject	Waveform and exposure conditions	Results
**Tkalec et al. (2005)	Growth of duckweed (Lemna minor L)	Plants exposed to 400, 900, or 1900 MHz electric field (CW or 80% AM at 1 kHz sinusoidal) (2–14 h; 10–390 V/m).	Effect (decreased growth) depended on frequency, duration and strength of exposure, and modulation.
**Tkalec et al. (2007)	Oxidative stress in duckweed (Lemna minor L)	Plants exposed to 400 and 900 MHz (CW or modulated) (2–4 h; 10–120 V/m).	Effect depended on frequency, duration and strength of exposure, and modulation.
**Tkalec et al. (2009)	Mitotic aberrations in root meristematic cells of Allium cepa	Seeds were exposed for 2 h to 400- or 900-MHz RFR at 0.03, 0.14, 4.2, or 38.2 mW/cm^2, or 80% AM 1 KHz-modulated.	The observed effects were markedly dependent on the field frequencies applied as well as on field strength and modulation.
#Tkalec et al. (2013)	Earthworm (Eisenia fetida) DNA damage and oxidative effects	Earthworms exposed to CW or modulated (80% AM at 1 kHz sinusoidal) 900-MHz RFR for 2 h; 0.00035 W/kg.	CW and modulated fields produced similar oxidative effects and DNA damage. Modulation is more potent.
#Trillo et al. (2021)	Neonatal human fibroblast Hsp47 and Hsp27 expression, and cell proliferation	Fibroblasts exposed to 448-KHz CW or 20-KHz AM 448-KHz carrier wave, 4 h; 100 μA/mm^2.	Both signals equivalently increased Hsp47 expression; AM signal more efficient in inducing Hsp27 and promoting cell proliferation.
,*Valbonesi et al. (2014)	Heat shock protein-70 expression in rat PC-12 cells	Cells exposed to 1800-MHz CW, GSM217Hz and GSM-talk signals for 4, 16, or 24 h; 2 W/kg.	Exposure to the GSM-217 Hz signal for 16 or 24 h increased HSP70 transcription, whereas no effect was observed in cells exposed to the CW or GSM-Talk signals.
**Veyret et al. (1991)	Antibody production	Mice exposed to pulsed 9400 MHz (1 ms pulses, 1000/s) RFR with or without AM by a sinusoidal frequency between 14 and 41 MHz; 10 h/day for 5 days, ~0.015 W/kg.	Pulsed field alone produced little effect; AM caused a frequency-dependent augmentation or weakening of immune responses.
*Vilić et al. (2017)	Oxidative effects and DNA damage in honey bee (Apis mellifera) larvae	Honey bee larvae were exposed to 900-MHz at different field levels for 2 h.	Catalase activity and the lipid peroxidation level decreased after exposure to unmodulated field at 0.027 mW/cm^2. DNA damage increased after exposure to modulated (80% AM 1 kHz sinusoidal) field at 0.14 mW/cm^2. Modulated RFR produced higher effects than the corresponding unmodulated field.

respectively. Sham and CW 147-MHz RFR (at 0.5 W/kg) had no significant effect on enolase activity.#Elekes et al. (1996)Antibody producing in spleenMice exposed to 2450 MHz RFR (CW or amplitude modulated with 50-Hz square-wave (3 h/day for 6 days; 0.14 W/kg).AM field more potent than CW field. Increased antibody producing cells in male mice only. No effect observed in female mice. ***Franzellitti et al. (2010)DNA damage in human trophoblast HTR-8/SVneo cellsCells were exposed to CW, GSM-217, or GSM-talk (5 min On/10 min off) 1800 -MHz RFR; 2 W/kg.Increased DNA damage was observed with modulated field exposure, but not with CW field.***Gapeyev et al. (2014)Mouse leukocytes H_2O_2 production and DNA damageCells exposed to 42.2 GHz RFR; 0.1 mW/cm^2, 1 Hz modulation frequency; 20 min).Modulated field increased H_2O_2 production; reduced X-ray-induced DNA damage.*Grasso et al. (2020)Cytoskeleton proteins in olfactory ensheathing cellsCells exposed to 900-MHz CW or 50-Hz sinusoidal amplitude-modulated fields (10, 15, or 20 min; 7 V/m (0.013 mW/cm^2)).CW and amplitude-modulated fields produce different patterns of responses on viability and apoptotic pathways and cell markers. **Gulati et al. (2020)DNA damage in human lymphocytesCells exposed to UMTS signals at different frequency channels used by 3 G mobile phone (1923, 1947.47, and 1977 MHz) for 1 or 3 h; 0.04 W/kg.DNA damage found only in cells exposed to 1977-MHz field. *#Halgamuge et al. (2015)Growth of soybean seedlingsSeedlings exposed to GSM-900 (2.6–4.8 × 10^4 mW/kg) or CW 900 MHz RFR for 2 h; 0.39–2 mW/kg. Epicotyl outgrowth
reduced more by modulated radiation than CW, and hypocotyl outgrowth only reduced by CW at low intensity. **Hinrikus et al. (2008)EEG rhythmsHuman subjects exposed to 450-MHz RFR pulse-modulated at 7, 14, and 21 Hz scalp power density 016 mW/cm^2 (0.303 W/kg).RFR exposure modulated at 14 and 21 Hz enhanced the EEG power in the alpha and beta frequency bands, whereas no enhancement occurred at 7-Hz modulation frequency. ***Höytö et al. (2008)Oxidative stress and cell deathHuman SH-SY5Y neuroblastoma and mouse L929 fibroblast cells exposed to 872-MHz RFR either CW or GSM signal-modulated (1 or 24 h; 5 W/kg)Drug-induced lipid peroxidation and caspase 3 increased with the modulated field and not with CW.***Huber et al. (2002)Sleep and waking EEG in humansPulsed modulated 900 MHz RFR (at 2, 8, 217, 1736 Hz) for 30 min at spatial peak SAR of 1 W/kg; and CW 900 MHz RFRPulsed-modulated field

induced changes in sleep and waking EEG; no significant effect with CW field.**Huber et al. (2005)Regional cerebral blood flowHuman subjects exposed unilaterally to 900-MHz (30 min; 1 W/kg) "base-station-like" or "hand-set-like" signals. The latter has stronger low-frequency components.Increased blood flow was observed in ipsilateral dosolateral prefrontal cortex only after "hand-set-like" signal exposure.**Hung et al. (2007)EEG-determined sleep onsetHuman subjects exposed to GSM 900 signals modulated at 2, 8, or 217 Hz ("talk", "listen", and "standby" modes) and sham-exposure for 30 min.Post-exposure, sleep latency after talk mode exposure was markedly and significantly delayed beyond listen and sham modes. Different modulation frequencies may differentially affect sleep onset.*Kubinyi et al. (1996)Aminoacyl-tRNA synthetase in brain and liverMice exposed in utero (100 min per day during 19 days of gestation) to CW or AM (50 Hz rectangular wave, 50%/50% On-Off ratio) 2450-MHz; 4.23 W/kg.At postnatal day 24, Aminoacyl-tRNA synthetase was decreased in the brain after CW exposure whereas no effect was seen with AM RFR exposure. Aminoacyl-tRNA synthetase acivity increased in liver for both types of RFR.**Kumar et al. (2021)Epigenetic modulation in the hippocampus of Wistar ratsRats exposed to 900-MHz, 1800-MHz, and 2450-MHz RFR for 2 h per day for 1-month, 3-month, and 6-month periods; 5.84×10^{-4} W/kg, 5.94×10^{-4} W/kg, and 6.4×10^{-4} W/kg, respectively. Significant epigenetic modulations were observed in the hippocampus; larger changes with increasing frequency and exposure duration.**Kunjilwar and Behari (1993) AChE activity in brainMale rats exposed to 147, 73.5 and 36.75 MHz RFR amplitude modulated at 16 and 76 Hz (3 h/day for 30–35 days; 0.1–0.14 W/kg).Decrease in AChE activity observed in all conditions independent of carrier frequency. Effect slightly higher in 16 Hz than 76 Hz modulation.**Lerchl et al. (2008)Body weightAdult male Djungarian hamsters (Phodopus sungorus) were exposed 24 h/day for 60 days to RFR at 383, 900, and 1800 MHz, modulated according to the TETRA (383 MHz) and GSM standards (900 and 1800 MHz), respectively; 0.08 W/kg.At 383 MHz, exposure resulted in a significant transient increase in body weight, while at 900 MHz body weight increase was more pronounced and not transient. At 1800 MHz, no effect on body weight was seen.***Lin (2021)Microwave auditory effectCaused by pulsed but not CW RFR**Lin et al. (2013)Pain relief effectRat dorsal root ganglia exposed using a bi-polar electrode to 500-KHz RFR with 25 msec pulse duration, 2.5/1.25 V amplitude, 5 min.Sinusoidal wave has higher responses than square wave.***Liu et al. (2021)Wakefulness in mouseMice exposed for 9 days to 2400-MHz RFR modulated by 100-Hz square pulses (1/8 duty cycle).Increased time of wakefulness with decreased times of non-rapid and rapid eye movement.*López-Martín et al. (2009)c-Fos expression in brain of picotoxin-induced seizure-prone ratsPulse-modulated GSM and unmodulated signals; 2 h; mean SAR in brain 0.03–0.26 W/kg.GSM-modulated and unmodulated signals produced different responses in different regions of the brain.***Luukkonen et al. (2009)Human SH-SY5Y neuroblastoma cells; DNA damage and oxidative effectsCells exposed to CW or GSM-modulated (217 Hz) 872-MHz RFR for 1 h; 5 W/kg.CW increased DNA damage and reactive oxygen species in cells treated with menadione. Modulated field had no significant effect. ***Markkanen et al. (2004)Apoptosis in mutant yeast cellsYeast cells exposed to 900 or 872 MHz RFR (CW or pulse modulated at 217 Hz, 0.577 ms) (1 h; 0.4 or 3.0 W/kg).Amplitude-modulated RFR enhanced UV-induced apoptosis. No effect from CW field exposure.**Markova et al. (2005)Human lymphocyte chromatin conformation and ϒ-H2AX foci905 and 915 MHz GSM900 mobile phone signals (577 μs pulses, inter-pulse waiting time of 4039 μs), for 1 h; 0.037 W/kg.Effects observed were carrier-frequency dependent.**Mohammed et al. (2013)Latency of REM sleep in ratsRats exposed to 900-MHz RFR (CW or modulated at 8 or 16 Hz) for 1 h/day for 1 month (peak SAR 0.245 W/kg).Latency of REM sleep increased in rats exposed to field with 16-Hz modulation.Nikolova et al. (2005)Apoptosis-related gene transcription and DNA damage in embryonic stem cell-derived neural progenitor cells.Cells exposed to 1710-MHz GSM signal (217 Hz rectangular pulses; width 0.576 ms) (5 min on/30 min off) for 6 or 48 h; 1.5 W/kg.Increase in DNA damage after 6 h, but not 48 h, exposure. 48 h exposure affected gene expression. **Nylund and Leszczynski (2006), (2010))Proteome response in two types of human primary brain microvascular endothelial cells900 MHz and 1800 MHz GSM signals for 1 h; 2.8 and 2 W/kg, respectively.900 MHz RFR affected protein expression but not with 1800 MHz exposure; the two cell lines responded differently to the 900 MHz RFR.**Ozgur et al. (2014)Proliferation of human hepatocarcinoma cellsCells exposed to 1, 2, 3, or 4 h (15 min on/15 min off) to 900- or 1800-MHz RFR; 2 W/kg.1,800-MHz RFR had a larger impact on cell viability and cell injury than 900-MHz. Four hour exposure produced more pronounced effect.**Panagopoulos and Margaritis (2010)Reproductive capacity of Drosophila melanogasterDrosophilia exposed to GSM 900 or DCS 1800 MHz signals for 1–21 min at 10 $\mu W/cm^2$. Reproductive capacity decreased almost linearly with increasing exposure duration to both GSM 900 and DCS 1800 radiation. GSM 900 MHz radiation is slightly more bioactive than DCS 1800 MHz radiation.#Panagopoulos et al. (2004)Reproductive capacity of Drosophila melanogasterDrosophilia exposed to GSM 900 phone modulated by human voice (0.436 mw/cm^2) or

unmodulated (0.041 mW/cm^2) for 6 min per day during the first 2–5 days of their adult lives.Decrease in reproductive capacity in both male and female flies observed; unmodulated field was less effective compared to sham-exposure. #Penafiel et al. (1997)Ornithine decarboxylase activity in L929 cellsL929 murine cells exposed to 835-MHz RFR (up to 24 h; 2.5 W/kg) with different types of amplitude modulation.Effects are much more robust when the modulation causes low-frequency periodic changes in the amplitude of the carrier wave. CW is less effective.#Persson et al. (1997) Blood–brain barrier in ratsRats exposed to 915-MHz RFR pulse modulated (217 Hz or 50 Hz) or CW for 2 to 960 min.RFR caused pathological changes in the blood–brain barrier. (Effect showed at >1.5 J/kg). CW is more potent.*Philippova et al. (1994)Cell receptor bindingCW or rectangular wave modulated (1, 6, 16, 32, 75, or 100 pps) (15 min; 0.5–18 W/kg).CW (1 W/kg) affected binding depending on the ligand and cell type studies; effect not dependent on modulation.**Phillips et al. (1998)DNA strand breaksHuman Molt-4 cells exposed to pulsed iDEN (813.5625 MHz, 2.4 or 24 μW/kg) or TDMA (836.55 MHz, 2.6 or 26 μW/kg) for 2 or 21 h.Increase or decrease in DNA strand breaks occurred depending on intensity of exposure and the type of modulation.*Poque et al. (2020)RAS/MAPK activation in HuH7 human hepatocellular carcinoma cellsCells exposed to 1800-MHz CW or GSM-modulated signals (24 h; 2 W/kg).Modulated signal decreased phorbol-12-myristate-13-acetate maximal efficacy to activate RAS- and ERK-kinase activation. CW only decreased efficacy to activate ERK, but not RAS. #Sagioglou et al. (2016)Apoptosis in Drosophila melanogasterD. melanogaster exposed to frequency modulated (50 kHz FM) or CW 100, 395, 682, 900 MHz RFR (6 or 60 min on the 6th day or daily for the first 6 days of their life; 0.0003–0.4 W/kg) showed increased apoptotic cell death.Apoptotic cell death was observed in all conditions, FM signal has a stronger effect than CW.#Salford et al. (1994)Blood–brain barrierRats exposed to CW or pulse-modulated (8, 16, 50, and 200 per sec) 915-MHz RFR (2 h; 0.16–5 W/kg).Significant increase in blood–brain barrier observed for both types of radiation.**Sarimov et al. (2004) Chromatin conformation in human lymphocytesCells exposed to GSM900 signals (577 μs pulses, 4039 μs between pulses) at 895, 900, 905, 910, and 915 MHz for 0.5–1 h; 0.0054 W/kg.RFR effects differ at various GSM frequencies and vary between donors.**Schmid et al. (2012)EEG power during sleep in the spindle frequency range (approximately 11–15 Hz)Human subjects exposed to 900 MHz RFR modulated at 14 or 217 Hz for 30 min; 2 W/kg.EEG power during sleep in the spindle frequency range was increased during non-rapid eye movement sleep following the 14-Hz pulse-modulated condition. No significant effect was found following exposure to 217-Hz modulated field. **Schneider and Stangassinger (2014)Social memory performance in ratsRats exposed to 900 MHz GSM or 1966 MHz UMTS RFR; 6 months; 0.4 W/kg.At 6 months, male rats exposed to GSM, but not UMTS signal, showed a memory deficit; no significant effect on female rats.**, ***Schwartz et al. (1990)Calcium efflux from frog heartFrog hearts exposed to 240-MHZ RFR CW or sinusoidally modulated at 0.5 or 16 Hz (30 min; 0.00015–0.003 W/kg).Movement of calcium affected only with 16-Hz modulation at 0.003 and 0.00015 W/kg. No effect with CW and 0.5 Hz modulation.**Seaman and DeHaan (1993) Inter-beat intervals of aggregated cardiac cells from chicken embryos19 sec exposure to 2450-MHz RFR at CW, pulsed modulated (duty cycle ~ 11%), or square-wave modulation (duty cycle 50%); 1.2–86.9 W/kg.Decrease and increase in inter-beat intervals were observed. SAR, modulation, and the modulation-SAR interaction were all significant factors in altering the inter-beat interval. Some effects were probably nonthermal.Semin et al. (1995)Secondary structure of DNADNA exposed to RFR (4- to 8 GHz, 25 ms pulses, 0.4 to 0.7 mW/cm^2 peak power, 1- to 6-Hz repetition rate). Irradiation at 3 or 4 Hz and 0.6 mW/cm2 peak power increased the accumulated damage to the DNA secondary structure; changing the pulse repetition rate to 1, 5, 6 Hz, as well as changing the peak power to 0.4 or 0.7 mW/cm^2 produced no effect.Sirav and Seyhan (2011) (CW effect) Blood brain barrier in ratsRats exposed to CW 900 MHz (0.00426 W/kg) or 1800 MHz (0.00146 W/kg) for 20 min. Increased blood–brain barrier permeability in male rats for both frequencies; no significant effect on female rats.**Sirav and Seyhan (2016)Blood brain barrier in ratsRats exposed to 900- and 1800-MHz RFR (20 min; 0.02 W/kg) pulse modulated at 217 Hz, 577 μs.Both types of RFR increased blood–brain barrier permeability in male rats, 1800-MHz was more potent. In female rats, only the 900-MHz field caused an effect.*, #Somosy et al. (1991)Morphological cell changes in mouse embryo 33T3 fibroblastsFibroblasts exposed to 2450-MHz CW or 16-Hz square-wave modulated RFR (0.0024–2.4 W/kg)Modulated field more potent than CW in some effects. Some effects of modulated field not observed in CW exposure.***Somosy et al. (1993) Pyroantimonate precipitable calcium content of mouse intestinal epithelial cells16-Hz square-wave modulated 2450-MHz RFR (0.5 and 1 mW/cm^2).Rapid distribution of pyroantimonate precipitable calcium content was observed. No effect with CW exposure.*Sukhotina et al. (2006)Secretion of melatonin from pineal glandsHamster pineal exposed to CW or GSM modulated 1800-MHz RFR (7 h; 0.008, 0.08, 0.8. 2.7 W/kg).Increase in melatonin release observed for both fields at 0.8 W/kg; AT 2.7 W/kg, melatonin levels were elevated in the CW, but suppressed in the GSM exposure. (There was a 1.2°C increase in temperature.)**Tkalec et al. (2005)Growth of duckweed

(Lemna minor L)Plants exposed to 400, 900, or 1900 MHz electric field (CW or 80% AM at 1 kHz sinusoidal) (2–14 h; 10–390 V/m).Effect (decreased growth) depended on frequency, duration and strength of exposure, and modulation.**Tkalec et al. (2007)Oxidative stress in duckweed (Lemna minor L)Plants exposed to 400 and 900 MHz (CW or modulated) (2–4 h; 10–120 V/m).Effect depended on frequency, duration and strength of exposure, and modulation.**Tkalec et al. (2009)Mitotic aberrations in root meristematic cells of Allium cepaSeeds were exposed for 2 h to 400- or 900-MHz RFR at 0.03, 0.14, 4.2, or 38.2 mW/cm^2, or 80% AM 1 KHz-modulated.The observed effects were markedly dependent on the field frequencies applied as well as on field strength and modulation.#Tkalec et al. (2013)Earthworm (Eisenia fetida) DNA damage and oxidative effectsEarthworms exposed to CW or modulated (80% AM at 1 kHz sinusoidal) 900-MHz RFR for 2 h; 0.00035 W/kg.CW and modulated fields produced similar oxidative effects and DNA damage. Modulation is more potent.#Trillo et al. (2021)Neonatal human fibroblast Hsp47 and Hsp27 expression, and cell proliferationFibroblasts exposed to 448-KHz CW or 20-KHz AM 448-KHz carrier wave, 4 h; 100 μA/mm^2.Both signals equivalently increased Hsp47 expression; AM signal more efficient in inducing Hsp27 and promoting cell proliferation.**,***Valbonesi et al. (2014)Heat shock protein-70 expression in rat PC-12 cellsCells exposed to 1800-MHz CW, GSM217Hz and GSM-talk signals for 4, 16, or 24 h; 2 W/kg.Exposure to the GSM-217 Hz signal for 16 or 24 h increased HSP70 transcription, whereas no effect was observed in cells exposed to the CW or GSM-Talk signals.**Veyret et al. (1991)Antibody productionMice exposed to pulsed 9400 MHz (1 ms pulses, 1000/s) RFR with or without AM by a sinusoidal frequency between 14 and 41 MHz; 10 h/day for 5 days, ~0.015 W/kg.Pulsed field alone produced little effect; AM caused a frequency-dependent augmentation or weakening of immune responses.*Vilić et al. (2017)Oxidative effects and DNA damage in honey bee (Apis mellifera) larvaeHoney bee larvae were exposed to 900-MHz at different field levels for 2 h.Catalase activity and the lipid peroxidation level decreased after exposure to unmodulated field at 0.027 mW/cm^2. DNA damage increased after exposure to modulated (80% AM 1 kHz sinusoidal) field at 0.14 mW/cm^2. Modulated RFR produced higher effects than the corresponding unmodulated field.

There are several studies that show biological effects of CW-RFR are frequency-dependent: e.g., Belyaev et al. (2000) – chromatin conformation; Belyaev et al. (2009) – chromatin and DNA double strand breaks; Gulati et al. (2020) – DNA damage observed only in one of several carrier frequencies tested; Ioniță et al. (2021) – 950 and 1000 MHz were more effective than 720 MHz RFR in reducing recombination at immunoglobulin light chain loci in pre-B lymphocytes; Kumar et al. (2020) – 1800-MHz was more effective than 900-MHz RFR on onion root growth; Kumar et al. (2021) – rat hippocampal epigenetic changes were dependent on the carrier frequency; Lerchl et al. (2008) – hamster body weight affected differently by different carrier frequencies; Markova et al. (2005) – human lymphocyte chromatin conformational changes were dependent on carrier frequency; Nylund and Leszczynski (2006, 2010) – proteome response in human endothelial cells was carrier-frequency dependent; Ozgur et al. (2014) – proliferation of human cancer cells was carrier frequency dependent; Sarimov et al. (2004) – chromatin conformational changes depended on carrier frequency; Schneider and Stangassinger (2014) – rat social memory depended on carrier frequency; Sirav and Seyhan (2016) – blood–brain barrier permeability changes depended on carrier frequency; Tkalec et al. (2005) – duckweed growth depended on carrier frequency; Tklec et al. (2007) – oxidative stress on duckweed depended on carrier frequency; and Tkalec et al. (2009) – mitotic aberrations in Alliu cepa (onion) root cells depended on carrier frequency.

However, there are also several studies that show biological effects are independent of the carrier frequency, e.g., Behari et al. (1998) – Na^+–K^+ ATPase changes in brain; Kunjilwar and Behari (1993) – brain acetylcholine esterase changes; Sharma et al. (2021) – liver and brain oxidative and morphological changes; and Tan et al. (2017) and Zhu et al. (2021) – spatial long-term memory changes. Other studies showed effects were dependent on the type of complex modulation, e.g., Croft et al. (2010) – EEG exposed to 2G or 3G signals; Huber et al. (2005) – cerebral blood flow exposed to "base-station-like" or "hand-set-like" signals; and Valbonesi et al. (2014) – heat shock protein expression was affected by GSM-217 Hz but not by GSM-talk signals. DNA damage was also dependent on whether the exposure was intermittent or continuous (Diem et al. 2005).

Comparison studies of continuous-wave and modulated RFR

A broad analytical review shows that four basic unpredictable and contradictory research outcomes can occur with CW and modulation as described later and in Table 2.

(1) CW and modulation produce different effects.
(2) Different forms of modulation produce different effects.
(3) Modulation produces effects, but not CW.
(4) Modulation and CW have different biological effectiveness/potencies.

The advantage of some studies listed in Table 2 is that the same experimental setup and researchers were involved. This makes the research results more credible. However, it must be pointed out that in some studies, the SARs in both conditions were not exactly the same, although the differences were generally small.

In most of the relevant studies, only simple modulations were studied, i.e., frequency and amplitude modulations. But in the real world, modulations are far more complex. Furthermore, modulated EMF/RFR has also been used therapeutically to treat a wide array of illnesses but discussion of therapeutic effects is beyond the scope of this paper (see Jimenez et al. 2018; Zimmerman et al. 2013).

CW and modulation cause different effects

There are several different patterns of response within this category:

(1) CW and modulation caused opposite effects: Arber and Lin (1985) – changes in neuronal activity; Detlavs et al. (1996) – wound inflammation exudation; Sukhotina et al. (2006) – changes in melatonin secretion from the pineal gland.
(2) CW and modulation produced different patterns of effects: Grasso et al. (2020) – cell viability, apoptosis and cell marker changes; Poque et al. (2020) – induced RAS/MAPK activation in cancer cells; Somosy et al. (1991) – cell morphology changes; Sukhotina et al. (2006) – altered melatonin secretion from the pineal gland.
(3) CW caused an effect but no significant effects were seen with modulation: Kubinyi et al. (1996) – aminoacyl-tRNA synthetase in brain; López-Martín et al. (2009) – c-fos expression in brain.

In addition, there are odd reports of modulation-independent effects on cell receptor binding (Philippova et al. 1994); as well as CW and modulation producing similar effects (Tkalec et al. 2013, – DNA damage; Salford et al. 1997, – blood–brain barrier changes); and modulation was found to be more biologically active than CW (Vilić et al. 2017 – DNA damage).

Differences in responses between CW and modulated fields of the same frequency and incident power density provide strong proof that non-thermal effects occur since the two conditions should produce the same amount of heating. There are numerous examples of such responses noted in Table 2.

Different modulation frequencies cause different effects

Some studies reported that different frequencies of modulation caused different biological responses: Bachmann et al. (2006) – human EEG, higher modulation frequency caused greater effects; Barbault et al. (2009) – tumor-specific modulation frequency; Dutta et al. (1994) – different modulation frequencies had opposite effects on enolase in *E. coli*; Hinrikus et al. (2008) – human EEG was affected by certain modulation frequencies; Hung et al. (2007) – sleep onset was affected by different modulation frequencies; Kunjilwar and Behari (1993) – AChE in rat brain depended on modulation frequency; Schmid et al. (2012) – EEG activity during sleep in humans depended on modulation frequency; Schwartz et al. (1990) – calcium efflux from frog heart depended on modulation frequency; Seaman and DeHaan (1993) – beating rate of cardiac cells depended on type of modulation (pulsed or square-wave); and Veyret et al. (1991) antibody production depended on types and frequency of modulation. Since extremely-low frequency could be an important component of RFR modulation, related to this, it must be pointed out that different frequencies (e.g., Wang et al. 2021) or waveforms (e.g., Chen et al. 2021) of extremely low frequency electromagnetic fields alone could have different effects without the RFR field.

Modulated fields caused effects, but not CW

There are several reports of effects caused by modulated fields and not CW, e.g., Bolshakov and Alekseev (1992) on electrical activity of Lymnea stagnalis neurons; Campisi et al. (2010) on rat neocortex astrocytes oxidative effects; Czerska et al. (1992) on transformation of human lymphocytes; d'Ambrosio et al. (1995, 2002) on micronucleus formation in peripheral human blood; Dutta et al. (1994) on enolase in *E. coli*; Franzellitti et al. (2010) on DNA damage in human trophoblasts; Höytö et al. (2008) on oxidative stress and cell death of human neuroblastoma cells; Huber et al. (2002) on human sleep and waking EEG; Somosy et al. (1993) on pyroantimonate precipitable calcium content of mouse intestinal epithelial cells; and Valbonesi et al. (2014) on heat shock protein-70 expression in rat PC-12 cells.

There is also a study by Luukkonen et al. (2009) showing that DNA damage and oxidative stress in human neuroblastoma cells occurred after CW exposure but not with exposure to a modulated field.

Furthermore, the most established and well-studied effect that is produced by modulated but not CW fields is the microwave-hearing effect. It can only happen with a pulsed field but not CW (Lin 2021).

CW and modulation have different potencies

CW and modulated fields can cause the same effects but with different degrees of biological activity and intensity of reactions. In most instances, a modulated field was found to be more potent than CW versus only one study in which the opposite was reported (Persson et al. 1997). Modulated fields being more effective than CW has been reported by: Elekes et al. (1996) in antibody production in spleen; Panagopoulos et al. (2004) – in reproductive capacity in Drosophila melanogaster; Penafiel et al. (1997) in ornithine decarboxylase activity in L929 cells; and Sagioglou et al. (2016)in inducing apoptosis in Drosophila melanogaster. The one study that found CW more effective than modulation was Persson et al. (1997) in which CW caused more pathological changes in the rat blood–brain barrier. Salford et al. (1994), however, previously reported no significant difference between modulated and CW fields on the rat blood–brain barrier.

To add to the complexities described above, effects with modulated fields have also been shown to depend on exposure duration: Grasso et al. (2020) – cytoskeleton protein of olfactory ensheathing cells; Nikolova et al. (2005) – DNA damage at shorter exposure time; Ozgur et al. (2014) – proliferation of human cancer cells, more consistent effects with longer exposure duration; and Zhang et al. (2008) – gene expression in rat neurons. Different effects were also seen with exposure intensity: Joines and Blackman (1981) – intensity-dependent power window on calcium efflux from chick brain; Kumar et al. (2021) – intensity-dependent epigenetic modification in rat hippocampus; Phillips et al. (1998) – DNA damage in human cancer cells; Pyankov et al. (2021) – intensity-dependent effect on cancer growth; Regel et al. (2007) – effects on sleep, sleep EEG and cognitive performance; Seaman and DeHaan (1993) – effects on beating rate of frog cardiac cells; Semin et al. (1995) – an intensity-dependent power window effect on DNA secondary structure; Sukhotina et al. (2006) – effects on melatonin secretion from pineal gland; and Tkalec et al. (2005, 2007, 2009) – growth effects on duckweed and onion cells.

Furthermore, there are many studies that used intermittent exposure (e.g., 10 min ON/10 min OFF) instead of continuous exposure with the supposition that intermittent exposure is more biologically active. But not much data showed this to be true (e.g., Bortkiewicz et al. 2012; Diem et al. 2005; Zeng et al. 2006;; Zhang et al. 2008). There are not many studies that compared intermittent and continuous exposure in the same experiment. This is also puzzling since, with the same SAR, the total energy deposited in the exposed object (specific absorption) would be less with intermittent exposure. There are reports that show the same effect with intermittent and continuous exposure (Chavdoula et al. 2010; Kakita et al. 1995). Theoretically this should not happen.

This same argument also applies to pulsed RFR. There are many studies using pulsed fields, (i.e., mobile phone signals are pulsed), but there are not many studies that compared pulsed and CW field of the same SAR in the same study. However, there are reports that effects only occurred with a pulsed field but not CW (Beason and Semm 2002; Curcio et al. 2005); that pulsed was more effective in causing effects than CW field (Burlaka et al. 2014; Panagopoulos et al. 2004); that pulsed and CW fields produced the same effect (Brown et al. 1994; Dawe et al. 2008; Lai and Singh 1996; Perentos et al. 2013); that CW was more potent than a pulsed field (Persson et al. 1997), and that pulsed and CW produced different effects (Czerska et al. 1992; Halgamuge et al. 2015). Thus, it is not certain if pulsed-fields are really more potent than CW fields as is popularly believed.

The role of the exposed object

As demonstrated in Table 2, many things come into play that affected various study outcomes after exposure to CW and modulated fields, including the properties of the exposed object, e.g., several studies have shown that effects only occurred in male animals but not female (Elekes et al. 1996; Schneider and Stangassinger 2014; Sirav and Seyhan 2011), or that male and female responded differently (Sirav and Seyhan 2016). Also different organs (Kubinyi et al. 1996) or different parts of the same organ (López-Martín et al. 2009) can respond differently. Cell type-dependent effects also have been reported (An et al. 2021; Nylund et al. 2010; Nylund and Leszczynski 2006; Philippova et al. 1994). Thus, it is not surprising that there was no specific pattern to predict which outcome was more likely to occur under certain parameters of exposure.

An interesting observation is that there are many studies showing effects of RFR on the hippocampus (see Lai 2018), a brain structure involved in memory functions. Our research has shown that a rather complicated sequence of brain functions occurred after RFR

exposure (Lai 1994). The radiation activates the hypothalamic stress hormone, corticotrophin-releasing factor, which in turn activate endogenous opioids (involving μ, δ, and K receptor subtypes). These opioids affect the cholinergic system in the hippocampus leading to alterations in memory and learning functions. It is unlikely that these changes are caused by absorption of RFR at a certain brain region. Rather, they could be stress responses to a general RFR absorption. If this is true, biological responses to RFR would follow the stages of response to stressors proposed by Selye (1951) described above.

Possibility of cellular oxidative changes

Oxidative changes and stress have been reported in many papers on exposure to electromagnetic fields (Lai 2020; Yakymenko et al. 2016). These are the most consistent cellular responses to RFR exposure. Mechanisms have been proposed to account for oxidative effects that may involve the low-frequency component of modulation (e.g., see Barnes and Greenebaum 2015; Castello et al. 2021). Some studies listed in Table 2 include findings of oxidative stress (e.g., Campisi et al. 2010; Höytö et al. 2008; Luukkonen et al. 2009; Sukhotina et al. 2006; Thalec et al. 2007, 2013; Vilić et al. 2017). But there is not enough data to conclude that modulation effects are caused by oxidative processes. In fact some effects of CW exposure alone also found changes in free radical mechanisms.

The nature of modulation and significance to human exposure guidelines

Above we examined some of the individual components of simple modulation. Relevant studies mainly investigated AM, FM, and pulsed modulation. In the real world, however, man-made RFR fields are invariably composed of different and often more complex modulation processes. It is not known how these different forms interact synergistically or antagonize the effects of each other – possibly producing cascading subtle effects throughout a living system. It is important to point out as significant proof of non-thermal RFR effects that CW and modulated-waves of the same frequency and incident power density can/and do produce different effects. The bottom line is that certainty is elusive regarding precise effects in all circumstances. What is clear is that both modulation and continuous-wave RFR are biologically active and both should be considered in exposure guidelines. In situations were enough evidence exists to warrant specific caution, such as with pulsed fields used in cell phones and phased modulation with 5G, particular attention should be paid to include modulation in the guidelines beyond the suppositions of safety contained within the safety allowances. Peak exposures must also be factored in and not just the averaged values which only hide their significance.

The FCC/ICNIRP exposure guidelines only take CW into consideration and have long been criticized for not considering modulation as a separate entity with effects of its own. In 1999, the U.S. Radiofrequency Interagency Work Group (Radio Frequency Interagency Work Group (RFIAWG) 1999) – a U.S. government multi-regulatory agency group with vested interests in EMF/RF – wrote a letter to the International Electrical and Electronics Engineers (IEEE), which is responsible for writing U.S. exposure guidelines adopted by FCC for professional exposures, containing 14 issues to be addressed. Regarding intensity or frequency modulated (pulsed- or frequency-modulated) RFR, they said:

"Studies continue to be published describing biological responses to nonthermal ELF-modulated and pulse-modulated RF radiation exposures that are not produced by CW (unmodulated) RF radiation. These studies have resulted in concern that exposure guidelines based on thermal effects, and using information and concepts (time-averaged dosimetry, uncertainty factors) that mask any differences between intensity-modulated RF radiation exposure and CW exposure, do not directly address public exposures, and therefore may not adequately protect the public. The parameter used to describe dose/dose rate and used as the basis for exposure limits is time-averaged SAR; time-averaging erases the unique characteristics of an intensity-modulated RF radiation that may be responsible for producing an effect. Are the results of research reporting biological effects caused by intensity-modulated, but not CW exposure to RF radiation sufficient to influence the development of RF exposure guidelines? If so, then how could this information be used in developing those guidelines? How could intensity modulation be incorporated into the concept of dose to retain unique characteristics that may be responsible for a relationship between exposure and the resulting effects?"

The RFIAWG's concerns have gone unaddressed for over two decades, including regarding modulation, exposure duration, intensity, time averaging, and peak exposures as noted above.

Discussion

It is apparent that the biological outcome of changing the intensity and duration of RFR exposure is basically unpredictable. This is mainly due to the complex nature

of the biological system studied. Intensity and duration can interact and produce different response patterns as shown in the literature reviewed above.

It is also apparent that how RFR modulation affects biological functions is difficult to quantify. Observed effects are multi-variant and involve many factors such as intensity, carrier frequencies and modulation, the modulation waveform itself, exposure duration, and properties of the exposed object. Not enough research data are presently available to provide an explanation or prediction of modulation effects under all circumstances. It may also turn out that modulation is of little major health concern or conversely that it is the only factor that matters – evidence is thus far too contradictory regarding modulation's ability to consistently enhance the biological effects of carrier-waves. Then again, with most modulation forms the carrier-wave is completely altered. All of this awaits proper investigation with comparison studies. In the meantime, there are legitimate reasons for concern, given the contraditions in the literature.

In general, anthropogenic RFR – with highly unusual waveform characteristics and intensities that do not exist in the natural world – is new to the environment and thus has not been a factor in the evolution of species. Living organisms evolved over millions of years in the presence of static and extremely-low frequency (ELF) electromagnetic fields. These fields play critical roles in their survival, e.g., in migration, food foraging, and reproduction, etc. (see Levitt et al. 2021b). Living organisms are extremely sensitive to the presence of these environmental fields and thus, they can easily be disturbed by man-made EMF. RFR probably acts upon and modifies these primordial EMFs and affects biological functions. Interactions of static/ELF EMF and RFR are basically not well studied, not to mention the mechanisms of involvement of RFR modulations. The interactions are inevitably complex. Such interaction studies would provide answers to wildlife effects.

Regarding the perennial thermal- versus non-thermal - effects criticism inherent in human RFR exposure guidelines, it must be said that the underlying mechanisms of effects should *not* be a matter of concern in setting of exposure guidelines as is common today. *What is important is the level at which energy absorption causes an effect*. One such powerful proof – among so very many others – of non-thermal effects is evidenced in the fact that CW and modulated-waves of the same frequency and incident power density can produce different effects, as seen in the modulation section of this paper and Table 2.

Thermal effects come from absorption of thermal energy with the general result being an increase in temperature. However, thermal and RFR are two different forms of energy. There are indications that RFR energy is more biologically active than thermal energy. This renders the argument of thermal effects totally invalid. Using temperature change as a comparable stand-in comparison of SAR actually under-estimates the effectiveness of RFR on biological systems. A 1°C increase (as reported in De Lorge and Ezell 1980; De Lorge 1984) is not enough to cause behavioral effects such as work-stoppage. One degree centigrade is within the normal temperature variation of animals. The work-stoppage effect was beyond that of 1°C increase in body temperature. Also, an increase of 4°C for 90 min is needed to cause DNA breakage (Mitchel and Birnboim 1985), but data presented in Supplement 1 show that an increase of SAR of 0.014 W/kg could cause DNA strand breaks. Also, an increase in free radicals in animal cells was observed after an increase of 7°C for 20 min. (Flanagan et al. 1998) but significant oxidative changes have been shown in cells exposed to RFR at a SAR of 0.024 W/kg (from data in Supplement 1). Furthermore, thermal changes for 1°C after RFR exposure cannot explain observed memory and learning deficits (Lai 2018). In humans, an increase of 2°C has been shown not to affect memory functions (Holland et al. 1985), whereas in another study, an increase of 1°C in body temperature has been shown to enhance working memory (Wright et al. 2002). The levels of RFR that cause similar increases in human body temperature generally lead to memory and learning deficits in animals (Lai 2018).

In addition, increase in temperature of an exposed object as an explanation of biological effects cannot reconcile with the fact that effects of RFR have been observed in *in vitro* experiments in which temperature was well controlled. A thermal effect depends on the extent of increase in local temperature and the duration of temperature increase. Hot spots are formed inside a stationary exposed object that may be able to cause an effect. However, for a moving organism in a RFR field, the extent and duration of temperature increase at a certain site inside the organism cannot generally reach the level to cause an effect. Biologically effects have been reported in many *in vivo* studies of freely moving animals. In addition, it is difficult to explain why certain organs are more vulnerable to RFR exposure. The hippocampus in the brain of mammals, for instance, has been shown to be especially affected by RFR (see Lai 2018). Thus, one needs to reconsider the rationale of using temperature increase as a metric of RFR effects. Furthermore, "thermal effects" cannot account for the large amount of research on static/ELF EMF that

showed biological effects. RFR and static/ELF EMF produce very similar biological effects. Unless they have different mechanisms of action, it is difficult to explain the static/ELF EMF effects as "thermal", since the temperature change would be minimal.

When effects continue to be observed over a long period of time that go against prevailing beliefs, even when mechanisms remain imperfectly understood, the appropriate course of regulatory action is to examine the underlying basis upon which an original premise was formed. When proven incomplete or invalid by new information, the change in a regulatory course is not only justified but is imperative. Disproven or incomplete deductions of how RFR affects living cells and tissues, as well as suppositions of safety for exposed individuals and the environment are insupportable given the wealth of studies to draw from today that have filled in many gaps. We need to more responsibly address the increasing near- and far-field RFR exposures of contemporary life with an eye toward 5G technology's unique characteristics. A new conceptual framework is called for.

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