

Taylor & Francis
Taylor & Francis Group

Electromagnetic Biology and Medicine

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/iebm20

Genetic effects of non-ionizing electromagnetic fields

Henry Lai

To cite this article: Henry Lai (2021): Genetic effects of non-ionizing electromagnetic fields, Electromagnetic Biology and Medicine

To link to this article: https://doi.org/10.1080/15368378.2021.1881866

View supplementary material

Published online: 04 Feb 2021.

Submit your article to this journal

View related articles

View Crossmark data

Full Terms & Conditions of access and use can be found at
https://www.tandfonline.com/action/journalInformation?journalCode=iebm20

ELECTROMAGNETIC BIOLOGY AND MEDICINE
https://doi.org/10.1080/15368378.2021.1881866

Taylor & Francis
Taylor & Francis Group

REVIEW

Check for updates

Genetic effects of non-ionizing electromagnetic fields

Henry Lai

Department of Bioengineering, University of Washington, Seattle, WA, USA

ABSTRACT

This is a review of the research on the genetic effects of non-ionizing electromagnetic field (EMF), mainly on radiofrequency radiation (RFR) and static and extremely low frequency EMF (ELF-EMF). The majority of the studies are on genotoxicity (e.g., DNA damage, chromatin conformation changes, etc.) and gene expression. Genetic effects of EMF depend on various factors, including field parameters and characteristics (frequency, intensity, wave-shape), cell type, and exposure duration. The types of gene expression affected (e.g., genes involved in cell cycle arrest, apoptosis and stress responses, heat-shock proteins) are consistent with the findings that EMF causes genetic damages. Many studies reported effects in cells and animals after exposure to EMF at intensities similar to those in the public and occupational environments. The mechanisms by which effects are induced by EMF are basically unknown. Involvement of free radicals is a likely possibility. EMF also interacts synergistically with different entities on genetic functions. Interactions, particularly with chemotherapeutic compounds, raise the possibility of using EMF as an adjuvant for cancer treatment to increase the efficacy and decrease side effects of traditional chemotherapeutic drugs. Other data, such as adaptive effects and mitotic spindle aberrations after EMF exposure, further support the notion that EMF causes genetic effects in living organisms.

ARTICLE HISTORY
Received 22 September 2020
Accepted 13 December 2020

KEYWORDS
Radiofrequency radiation; static/extremely low frequency EMF; genetic effects; genotoxicity; gene expression

Introduction

This is a review on studies on the genetic effects of non-ionizing electromagnetic fields (EMF). We will concentrate on two parts of the EMF spectrum which are common in our environment: static and extremely low-frequency electromagnetic fields (ELF-EMF) and radiofrequency radiation (RFR).

Studies are summarized in Supplements 1 (RFR) and 2 (static/ELF-EMF). Basically, there are two types of studies: genetic damages and gene expression. The research covers a wide area of biological systems: both in vitro and in vivo involving many animal and cell models, and various exposure conditions. First, a few words have to be said on the exposure set-ups used in these studies. It is relatively easy to set up a reliable exposure system for static and ELF-EMF. Most exposure systems used these studies are generally satisfactory. However, it is difficult to set up good exposure systems for RFR studies. In my opinion, most set-ups are relatively satisfactory, considering that there is no perfect guideline on what is a good system. However, preferably, incident power density and specific absorption rate should be provided in each study. These are generally lacking when telecommunication devices, such as cellular phones, are used in a study. It becomes difficult to compare the results of these studies with other studies using exposure systems. It is not totally without merit to use these devices for studies. If properly set up, these devices provide more realistic exposure parameters. A general problem is that some researchers generally showed ignorance on the independent variable, i.e., EMF, that they worked on.

Regarding biological measurements, with few exceptions, the researcher are generally knowledgeable in the methodology used. However, there are studies that showed that the researchers are not familiar with the methodology that they used in their studies. An example is the use of the "Comet assay" to determine DNA strand breaks. 31% of the studies listed in Supplements 1 and 2 used the "Comet assay". A few words have to be said on it. Different versions of the assay have been developed. These versions have different detection sensitivities and can be used to measure different aspects of DNA strand breaks. A comparison of data from experiments using different versions of the assay may be misleading. Another concern is that most of the 'comet assay' studies were carried out by experimenters who had no prior experience on the assay. My experience with the 'Comet assay' is that it is a very sensitive assay and requires great care in performing. Thus, different detection sensitivities could result from different

CONTACT Henry Lai hlai@uw.edu Department of Bioengineering,University of Washington, Seattle, WA 98195, USA
Supplemental data for this article can be accessed on the publisher's website.
© 2021 Taylor & Francis Group, LLC

experimenters, even following the same procedures. One way to solve this experimental variation problem is for each researcher or laboratory to report their sensitivity of the 'Comet assay', e.g., threshold of detecting strand breaks in human lymphocytes exposed to x-rays. This information is generally not available from the EMF-genotoxicity studies. However, in one incidence, an incredibly high sensitivity was even reported (Malyapa et al., 1998), suggesting the inexperience of the researchers on the assay.

Supplements 1 and 2 show that the majority of studies reported genetic effects of EMF (66% for RFR and 79% for static/ELF-EMF). Thus, it is safe to conclude that genotoxic effects of EMF have been reported. The most common effects found are: DNA strand breaks, micronucleus formation, and chromosomal structural changes. There are not many studies on mutation. Thus, it is not known whether these genotoxic effects transform into mutation and involved in carcinogenesis. Interestingly, available data do not suggest mutagenic effect after RFR exposure (Chang et al., 2005; Meltz et al., 1990; Ono et al., 2004; Takahashi et al., 2002); whereas most static/ELF-EMF studies (Chahal et al., 1993; Mairs et al., 2007; Miyakoshi, 1997; Miyakoshi et al., 1998, 1996; Potenza et al., 2004; Wilson et al., 2015) suggested some mutagenic effects. Another interesting speculation is that ELF EMF acts as a promoter of cancer in the presence of an initiator by modulation of signaling pathways involved free radicals and apoptosis (Lacy-Hulbert et al., 1998). Such a possibility has not been well investigated.

There are similarly many studies that showed changes in gene expression after EMF exposure (Supplement 3). Changes in expression of many different genes have been reported. Studies in gene expression by static/ELF-EMF are far more diversified that those of RFR. The most interesting results are the expression of genes related to stress response both in vitro and in vivo in plans and animals. Another important finding is the expression of heat shock proteins, particularly HSP70, which is an important protein involving in protein misfolding and protecting cells from environmental stress.

The data point to four areas of interest: involvement of free radicals, effects at low-intensity of exposure, contributions of exposure parameters and biological system being studied, and interaction with other entities. Let us look at each of these four topics.

Involvement of free radicals (Citations of references in italic in this section are in Supplements 1 and 2)

Effects of EMF on cellular free radical processes have been reported in many experiments (cf. Lai, 2019; Yakymenko et al., 2016). It is conceivable that an increase in free radicals in cells could cause macromolecular damages including DNA. There are many reports on involvements of free radicals in genetic processes, including both reactive oxygen species and reactive nitrogen species: **RFR** – *Agarwal et al., 2009; Alkis et al., 2019a, b, 2021; Bektas et al., 2020; Bourdineaud et al., 2017; Burlaka et al., 2013; De Iuliis et al., 2009; Duan et al., 2015; Gajski and Garaj-Vrhovac 2009; Garaj-Vrhovac et al., 2009, 2011; Guler et al., 2010; Gürler et al., 2014; Houston et al., 2019; Kesari et al., 2011, 2014; Khalil et al., 2012; Kumar et al., 2010; Lai and Singh, 1997; Li et al. 2018: Liu et al., 2013a, b; Luukkonen et al., 2009; Manta et al. 2017; Magha et al., 2015b; Meena et al. 2014; Millenbaugh et al., 2008; Odacı et al., 2016; Pandey et al., 2017; Pandey and Giri, 2018; Qin et al., 2019; Sahin et al., 2016; Shahin et al., 2013, 2019; Sharma and Shukla, 2020; Sokolovic et al., 2015; Sun et al. 2017; Tkalec et al., 2013; Vafaei et al. 2020; Varghese et al., 2018; Veerachari and Vasan, 2012; Vilić et al., 2017; Wang et al., 2015; Wu et al., 2008; Xu et al., 2010; Yakymenko et al., 2018; Yao et al., 2008; Zong et al. 2015; Zothansiama et al., 2017;* **Static and ELF EMF** – *Alcaraz et al., 2014; Amara et al., 2007b; Ashta et al., 2020; Hosseinabadi et al., 2020; Bertea et al., 2015; Bułdak et al., 2012; Consales et al., 2018; Dong et al. 2019; Jajte et al., 2001; Jouni et al., 2012; Kimsa-Dudek et al. 2018; Kindzelskii and Petty, 2000; Lai and Singh, 1997b, 2004; Li et al., 2001; Luukkonen et al., 2014; Rageh et al., 2012; Shokrollahi et al., 2018; Solek et al., 2017; Wang et al., 2020; Wolf et al., 2005; Yin et al., 2016; Yokus et al., 2008; Yuan et al., 2020; Zhang et al., 2016*. Brief descriptions of these reports are in Supplements 1 and 2. However, changes in cellular free radical and genetic processes do not imply a cause–effect relationship. A convincing argument on direct involvement of free radicals on EMF-induced genetic changes comes from data showing that the effects could be blocked by free radical scavengers (e.g., antioxidants) e.g., see Lai and Singh (*1997; 2004*). The free radicals involved probably include both reactive oxidative species (ROS) and reactive nitrogen species (RNS) (*Lai and Singh, 2004*). RNS (e.g., nitric oxide) have longer mean free path than ROS (e.g., hydroxyl radical) and could cause more widespread cellular molecular damages. Nitric oxide can further enhance iron-mediated free radical formation via its effects on iron metabolism and release of iron from ferritin (Reif and Simmons 1990; Richardson and Ponka 1997) that generates ROS via the Fenton reaction. Nitric oxide can either be mutagenic or cytotoxic. It is mutagenic when the intracellular level of reduced glutathione is low, but cytotoxic (leading to

apoptosis and inhibition of tumor growth) in a thiol-rich environment that favors the formations of toxic nitrosothiols (Felley-Bosco 1998). These situations could occur under EMF exposure.

The mechanisms on how EMF affects free radicals in cells are not known. There are various speculations. Readers may be interested to take a look at these publications: Barnes and Greenebaum (2015); Binhi and Prato (2017); Davila et al. (2005); Dodson et al. (2013); Hore (2019); Hore and Mouritsen (2016); Kirschvink et al. (2001); Landler and Keays (2018); Sheppard et al. (2017); Sherrard et al. (2018); and Sisakht et al. (2020).

Furthermore, it has to be pointed out that EMF-induced genetic effects have been observed without free radical changes *(Alcaraz et al., 2014; Ferreira et al., 2006; Furtado-Filho et al., 2014)* and free radical changes without genetic effects *(Frahm et al., 2006; Senturk et al., 2019; Tiwari et al., 2015; Tomruk et al., 2010)* have also been reported. This may imply that mechanisms other than free radicals are involved,

Effects at low exposure intensities

There are many reports of genetic effects induced by low intensities of EMF. The studies are listed in Supplement 4. This is an important topic to consider since living organisms are being constantly exposed to low levels of EMF in the occupational and public environments. This is particularly true for ELF-EMF, since intensities of ELF-EMF in the environment are in microtesla (μT) levels, even exposure to fields from electrical appliances rarely exceed 10 microtesla (i.e., 0.01 mT). However, most laboratory cell and animal studies in ELF-EMF used fields in the millitesla (mT) level.

A survey of level of RFR in the environment of various countries (Amoako et al., 2009; Aris et al., 2020; Bhatt et al., 2016; Dhami, 2012; Dode et al., 2011; Estenberg and Augustsson, 2014; Firlarer et al., 2003; Frei et al., 2009; Hardell et al., 2016, 2017; Henderson and Bangay, 2006; Joseph et al., 2008, 2010; Kim and Park, 2010; Kurnaz and Aygun, 2020; Lahham and Hammash, 2012; Lahham et al., 2015, 2017; Sagar et al., 2018; Tell and Kavet, 2014; Thuroczy et al., 2006; Urbinello et al., 2014; Viel et al., 2009; Waldmann-Selsam et al., 2016) gave a mean power density level of 0.00259 mW/cm^2 and median of 0.000545 mW/cm^2. Reports (Abuasbi et al. 2018; Al-Badi, 2012; AL-rajhi, 2014; Eskelinen et al., 2002; Ilonen et al., 2008; Lindgren et al., 2001; Röösli et al., 2011) on the levels of magnetic fields in the human environment came up with a mean level of 0.0036 mT and median level of 0.00062 mT. Much higher exposure levels could be found in occupational situations. Operators and technicians in a power plant could be exposed to 0.0126 mT, whereas the magnetic field level in the vicinity of a power transmission line could be as high as 0.0482 mT (Hosseinsbadi et al., 2020).

Besides genetic effects, other physiological processes have also been reported to be affected by low-intensity EMFs, e.g., **RFR**: retarded development of frog (Balmori, 2010; 88.5–1873.6 MHz cell phone base station emission; 0.00859–0.00325 mW/cm^2); slowing of circadian rhythm in cockroach (Bartos et al., 2019; broadband RF noise; 0.000429 mT); changes in electrical activities in rat sciatic nerve (Comelekoglu et al., 2018, 1800-MHz RFR; 0.00421 W/kg); delayed growth in rose (Grémiaux et al., 2016; 900 MHz RFR; 0.00072 W/kg); retarded memory in rat (Nittby et al., 2008; 900 MHz GSM signal; 0.0006 W/kg); adrenal gland stimulation in rat (Perov et al., 2019; 171 MHz RFR; 0.0006 W/kg); human blood mononucleus cells showed higher immunological activates (Szymanski et al., 2020; 0.024 W/kg) (see also the Table in Lai, 2018 on low-intensity effect on neurological functions); **static and ELF-EMF**: decreased number of living and quality of movement of sperms of mouse (de Bruyn and de Jager, 2010; 50-Hz MF 0.0005–0.077 mT) and free radicals (see Table 1: „Free radical effects observed at low intensities of static and ELF-EMF" in Lai, 2019, effects have been observed with exposure to a 50 Hz MF of 0.0005 mT). In addition, mechanisms have evolved for organisms to detect very low levels of static EMF, e.g., 26 nT (i.e., 0.000026 mT) in honey bees (Kirschvink et al., 1992); 20 microV/cm in platypus (Manger and Pettigrew, 1996); and 2–3 nT in songbird (Pakhomov et al., 2017). These capabilities of detecting very low-intensity static/ELF EMF fields is actually not surprising because they are results of evolution over millions of years to enable the survival of the species. On the other hand, these functions are much vulnerable to disturbance from recent man-made EMF. However, it is a little surprising that RFR at very low intensity could also cause biological effect. The RFR studied are mostly man-made and have only existed in the environment in the last several decades. This points to a possibility that EMFs (RFR and static/ELF EMF), in general, act on some common unknown basic biological mechanisms.

Interaction effects (citations of references in italic in this section are in supplements 1 and 2)

Another important observation of the studies is that EMF can interact with other entities and synergistically cause genetic effects. These entities include:

RFR: Chemical mutagens (*Baohong et al., 2005*); ultraviolet ray (*Baohong et al., 2007*); 17-β-estradiol (*Cervellati et al., 2013*); bee venum (*Gajski and Garaj-Vrhovac, 2009*);

garlic (*Gurler et al., 2014*); γ-radiation (*He et al., 2017; Ji et al., 2016; Jiang et al., 2013*); clastogens (*Kim et al., 2008);* incoherent electromagnetic noise (*Lai and Singh, 2005; Wu et al., 2008; Yao et al., 2008*); lipopolysaccharide (*Lameth et al., 2020; Zuo et al., 2015*); mitomycin C (*Maes et al., 1996; Sannino et al., 2011,2017; Zeni et al., 2012a; Zhang et al., 2002*); x-rays (*Manti et al., 2008; Gapeyev et al., 2014;* Sannino et al., 2014); aphidicolin (*Tiwari et al., 2008);* picrotoxin (*López-Martín et al., 2009*); bleomycin (*Koyama et al., 2003;* Zong et al., 2015) and doxorubicin (*Zhijian et al., 2010*).

Static – and ELF-EMF: Zinc (*Amara et al., 2007*); Tremozolomide (*Ashta et al., 2020*); Cisplastin (*Buldak et al., 2012; El-Bialy et al., 2013; Chen et al., 2010; Mahmoudinasab and Saadat, 2018a; Sanie-Jahromi and Saadat, 2017; Sanie-Jahromi et al., 2016*); Bleomycin (*Cho et al., 2007; Sanie-Jahromi and Saadat, 2017*); Gadolinium (*Cho et al., 2014*); alkaline-ph (*Fan et al., 2018*); natural radioactivity in soil (*Jouni et al., 2012*); sodium fluoride (*Kimsa-Dudek et al., 2018, 2020);* gamma radiation (*Arruda-Neto et al., 2009; Kubinyl et al., 2010; Lagroye and Poney, 1997; Mairs et al., 2007*); hydrogen peroxide and methyl methane sulfonate *(Koyama et al., 2008*); menadione (*Luukkonan et al., 2011, 2014, 2017; Markkanen et al., 2008*), morphine (*Mahmoudinasab and Saadat, 2018b*); X-ray (Miyakoshi et al., 1996*b; 1999, 2000; Teodori et al., 2014; Udroiu et al., 2015*); Xenobiotics (*Moretti et al., 2005*); lipopolysaccardide (*Nakayama et al., 2016*); heat (*Robison et al., 2002);* N-methyl-N'-nitro -N-nitrosoguanidine, 4-nitroquinoline N-oxide, benzene, 1,4-benzenediol, 1,2,4-benzenetriol (*Scassellati Sforzolini et al., 2004; Villarini et al., 2000);* mineral oil (*Skyberg et al., 2001*); Paclitaxel (*Sun et al., 2012*); IR (*Yoon et al., 2014*); $FeCl_2$ (*Zmyslony et al., 2000*); UV (*Zmyslony et al., 2000*).

Most of the compounds that have been shown to interact with EMF are mutagens. This is important because in real-life situations, a person is usually exposed simultaneously to EMF and many different environmental factors, including mutagens. On the other hand, some of these entities are drugs used in cancer chemotherapy. EMF can possibly be used as an adjuvant in chemotherapy to enhance the anticancer efficacy of these drugs and decrease their side-effects. Thus, synergism of these entities with EMF should be further studied.

However, it is important to point out that are reports (listed below) that showed no significant interaction effects.

RFR: Mitomycin C (*Hansteen et al., 2009; Kerbacher et al., 1990; Maes et al., 1997, 2000, 2001, 2006; Zhijian et al., 2009*); Adrimycin (*Kerbacher et al., 1990*); x-ray (*Maes et al., 2000; Stronati et al., 2006);* proflavin (*Meltz et al.,* 1990); 3-Chloro-4-(dichloromethyl)-5-Hydroxy-2 (5 H)-furanone (an environmental mutagen) (*Sannino et al., 2009; Verschaeve et al., 2006*).

Static – and ELF-EMF: Methylmethane sulfonate, chromate (*Cantoni et al., 1996*); UV (*Cantoni et al., 1996; Mizuno et al., 2014*); ionizing radiation, H_2O_2, mitomycin C (*Jin et al., 20112, 2014*); IR and H_2O_2 (*Jin et al., 2015; Yoon et al., 2014*); chemical mutagens (*Verschaeve et al., 2011*); heat (*Williams et al., 2006*).

Effects of waveform

Two other important findings of recent studies are that the effects of EMF are waveform specific and cell-type specific (Supplement 5). These findings underscore the complicity of interaction of EMF with biological tissues and may partially explain why effects were observed in some studies and not others. It is essential to understand why and how certain wave-characteristics of an EMF are more effective than other characteristics in causing biological effects, and why certain types of cells are more susceptible to the effect of EMF? The fact that "there are different biological effects elicited by different EMF wave-characteristics" is a critical proof for the existence of non-thermal effects.

Wave-from dependency is one of the major puzzlements of Bioeletcromagnetics research. In the 1970s, research in the laboratories of Ross Adey (Bawin et al.,1975; 1978) and Carl Blackman (Blackman et al., 1979) showed the importance of modulations on the EMF-carrier frequency on calcium efflux from cells. Other biological effects of EMF also showed wave-form dependency, e.g., see discussion in Lai (2018) on neurological effects of RFR. And, research presented here also showed similar dependency in EMF-induced genetic effects. So far, there has not been a credible unifying explanation for the "wave-form dependency effect".

Regarding cell-type specificity, one can speculate that: 1. Cells that are metabolic active are more susceptible to EMF effects with an increase in generation of free radical in the mitochondria; 2. Cells that have higher antioxidative activities are less susceptible; 3. Transitional elements, e.g., iron, may play a role in the effect via the Fenton reaction (see Lai, 2019). Brain cells contain a relatively high concentration of free iron, particularly intercalated in the DNA molecules, and are more susceptible; 4. Cell cycle arrests are common in cells exposed to EMF. It may be a response to repair genetic damages caused by EMF. If damage could not be repaired, cell death occurs, particularly via apoptosis, which is a common outcome after EMF exposure. These effects are consistent with the gene expression

studies, showing activation of genes involved in both cell death and repair. 5. If genetic damaged cells are allowed to survive, cancer may occur. However, if they die, the risk of cancer would actually be reduced. But, other detrimental health outcomes may occur, e.g., death of brain cells could lead to neurodegenerative diseases. Increased incidences of degenerative diseases (including Alzheimer's disease, amyotrophic lateral sclerosis, dementia, and motor dysfunctions) after EMF exposure, particularly under occupational conditions, have been reported (Gervasi et al. 2019; Gunnarsson and Bodin 2018, 2019; Huss et al. 2018; Koeman et al. 2017; Jalilian et al. 2018; Pedersen et al. 2017; Sorahan and Mohammed 2014).

Discussion

The main question is whether EMF exposure could cause genetic effects? It is pertinent here to quote a recent statement made by two prominent bioelectromagnetic researchers (Barnes and Greenebaum, 2020): "The evidence that weak radiofrequency (RF) and low-frequency fields can modify human health is still less strong, but the experiments supporting both conclusions are too numerous to be uniformly written off as a group due to poor technique, poor dosimetry, or lack of blinding in some cases, or other good laboratory practices." All in all, in the studies reviewed in Supplements 1 and 2, approximately 70% of them showed effects. One could say that EMF exposure can lead to genetic changes. Some genetic damages could eventually lead to detrimental health effects. However, the mechanisms remain to be uncovered. But, knowing the mechanism is not necessary to accept that the data are valid. It is also a general criticism that most EMF studies cannot be replicated. I think it is a conceptual and factual misstatement. Replication is also not a necessary and sufficient condition to believe that certain data are true. Scientific studies are hardly replicated. Rational funders do not generally fund replications. All scientists should know that it is very difficult to replicate exactly an experiment carried out by another lab. This is particularly true when the effects of EMF depend on many unknown factors. By the way, not many replication experiments have been carried out in EMF genetic-effect research to justify the statement that "data from EMF are not replicable". In some cases, the experimenters deliberately changed the procedures of an experiment that they were supposed to be replicating and claimed that their experiment was a replication, for example, compare the experimental procedures of Lai and Singh (1995) and Malyapa et al. (1998).

To prove an effect, one should look for consistency in data. Genetic damage studies have shown similar effects with different set-up and in various biological systems. And, the gene expression results (Supplement 3) also support the studies on genetic damages. Expression of genes related to cell differentiation and growth, apoptosis, free radical activity, DNA repair, and heat-shock proteins have been reported. These changes could be consequences of EMF-induced genetic damages. In addition, other effects of EMF, such as mitotic-spindle disruption (De Amicis et al., 2015; Hintzsche et al., 2011; Li et al., 2013; Schrader et al., 2011, 2008; Tkalec et al., 2009) and "adaptive" effects, i.e., the ability of concomitant exposure of RFR to decrease the genotoxic effects of other agents, such as ionizing radiation (He et al., 2017; Ji et al., 2016; Jiang et al., 2012, 2013; Sannino et al., 2014, 2017, 2011; Sun et al., 2016; Zeni et al., 2012; Zong et al., 2015) also support the notion that EMF exposure could affect genetic processes in cells. In conclusion, there are enough reasons to believe that genetic effects of EMF are real and possible.

During cell phone use, a relatively constant mass of tissue in the brain is exposed to the radiation at relatively high intensity (peak specific absorption rate (SAR) of 4–8 W/kg). Many papers have reported genetic effect/DNA damage at much lower SAR (or power density) (see Supplement 4). This questions the wisdom of the several exposure standard-setting organizations in using the obsolete data of 4 W/kg (whole-body averaged SAR) as the threshold for exposure-standard setting. Furthermore, since critical genetic mutations in one single cell are sufficient to lead to cancer and there are millions of cells in a gram of tissue, it is inconceivable that some standards have changed the SAR from averaged over 1 gm to 10 gm of tissue. (The limit of localized tissue exposure has been changed from 1.6 W/kg averaged over 1 gm of tissue to 2 W/kg over 10 gm of tissue. Since distribution of radiofrequency energy is non-homogenous inside tissues, this change allows a higher peak level of exposure.) What actually needed is a better refinement of SAR calculation to identify 'peak values' of SAR inside the brain.

Any effect of EMF has to depend on the energy absorbed by a biological entity and on how the energy is delivered in space and time. Aside from influences that are not directly related to experimentation (Huss et al., 2007), many factors could influence the outcome of an experiment in bioelectromagnetics research. Frequency, intensity, exposure duration, and the number of exposure episodes can affect the response, and

these factors can interact with each other to produce different effects. In addition, in order to understand the biological consequences of EMF exposure, one must know whether the effect is cumulative, whether compensatory responses result, and when homeostasis will break down. A drawback in the interpretation and understanding of experimental data from bioelectromagnetic research is that there is no general accepted mechanism on how EMF affects biological systems. Since the energy level is not sufficient to cause direct breakage of chemical bonds within molecules, the effects are probably indirect and secondary to other induced chemical changes in the cell. The mechanisms by which EMF causes genetic effects are unknown. This author suspects that biological effects of EMF exposure are caused by multiple inter-dependent biological mechanisms.

Disclosure of Interest

The author declares no conflict of interest

References

Abuasbi, F., A. Lahham, and I. R. Abdel-Raziq. 2018. Residential exposure to extremely low frequency electric and magnetic fields in the city of pamallah-palestine. *Rad. Prot. Dosimetry*. 179:49–57. doi:10.1093/rpd/ncx209.

Al-Badi, A. H. 2012. Measurement and analysis of extremely low frequency electromagnetic field exposure in oman. *J. Electromagn. Analy. App.* 4:333–39.

AL-rajhi, M. A. 2014. Assessment of ambient magnetic field exposure emitted from underground transmission lines in some areas of riyadh. *Internat. J. Phys.* 2:76–77. doi:10.12691/ijp-2-3-1.

Amoako, J. K., J. J. Fletcher, and E. O. Darko. 2009. Measurement and analysis of radiofrequency radiations from some mobile phone base stations in ghana. *Radiat. Prot. Dosimetry*. 135:256–60. doi:10.1093/rpd/ncp115.

Aris, A., K. Yiannis, S. Vasiliki, S., . K. Constantin, K. Charilaos, and T. Kiki. 2020. RF-EMF exposure levels in sensitive land use in greece: educational units census in the municipality of korydallos. *Radiat. Prot. Dosimetry* online ahead of print. doi:10.1093/rpd/ncaa090.

Balmori, A. 2010. Mobile phone mast effects on common frog (rana temporaria) tadpoles: the city turned into a laboratory. *Electromagn. Biol. Med.* 29:31–35. doi:10.3109/15368371003685363.

Barnes, F., and B. Greenebaum. 2020. Setting guidelines for electromagnetic exposures and research needs. *Bioelectromagnetics* 41:392–97. doi:10.1002/bem.22267.

Barnes, F. S., and B. Greenebaum. 2015. The effects of weak magnetic fields on radical pairs. *Bioelectromagnetics* 36:45–54. doi:10.1002/bem.21883.

Bartos, P., R. Netusil, P. Slaby, D. Dolezel, T. Ritz, and M. Vacha. 2019. Weak radiofrequency fields affect the insect circadian clock. *J. R. Soc. Interface.* 16:20190285. doi:10.1098/rsif.2019.0285.

Bawin, S. M., L. K. Kaczmarek, and W. R. Adey. 1975. Effects of modulated VHF fields on the central nervous system. *Annals of the New York Academy of Sciences* 247:74–81. doi:10.1111/j.1749-6632.1975.tb35984.x.

Bawin, S. M., W. R. Adey, and I. M. Sabbot. 1978. Ionic factors in release of <sup>45Ca2+ from chicken cerebral tissue by electromagnetic fields. . *Proceedings of the National Academy of Sciences* 75:6314–18. doi:10.1073/pnas.75.12.6314.

Bhatt, C. R., A. Thielens, B. Billah, M. Redmayne, M. J. Abramson, M. R. Sim, R. Vermeulen, L. Martens, W. Joseph, and G. Benke. 2016. Assessment of personal exposure from radiofrequency-electromagnetic fields in australia and belgium using on-body calibrated exposimeters. *Environmental Research* 151:547–63. doi:10.1016/j.envres.2016.08.022.

Binhi, V. N., and F. S. Prato. 2017. A physical mechanism of magnetoreception: extension and analysis. *Bioelectromagnetics* 38:41–52. doi:10.1002/bem.22011.

Blackman, C. F., J. A. Elder, C. M. Weil, S. G. Benane, D. C. Eichinger, and D. E. House. 1979. Induction of calcium-ion efflux from brain tissue by radio-frequency radiation: Effects of modulation frequency and field strength. *Radio Science* 14:93–98. doi:10.1029/RS014i06Sp00093.

Chahal, R., D. Q. Craig, and R. J. Pinney. 1993. Investigation of potential genotoxic effects of low frequency electromagnetic fields on *escherichia coli*. *J. Pharm. Pharmacol.* 45:30–33. doi:10.1111/j.2042-7158.1993.tb03674.x.

Chang, S.-K., J.-S. Choi, H.-W. Gil, J.-O. Yang, E.-Y. Lee, Y.-S. Jeon, Z.-W. Lee, M. Lee, M.-Y. Hong, T.-H. Son, et al. 2005. Genotoxicity evaluation of electromagnetic fields generated by 835-MHz mobile phone frequency band. *Eur. J. Cancer Prev.* 14:175–79. doi:10.1097/00008469-200504000-00014.

Comelekoglu, U., S. Aktas, B. Demirbag, M. I. Karagul, S. Yalin, M. Yildirim, A. Akar, B. K. Engiz, F. Sogut, and E. Ozbay. 2018. Effect of low-level 1800 MHz radiofrequency radiation on the rat sciatic nerve and the protective role of paricalcitol. *Bioelectromagnetics* 39:631–43. doi:10.1002/bem.22149.

Davila, A. F., M. Winklhofer, V. P. Shcherbakov, and N. Petersen. 2005. Magnetic pulse affects a putative magnetoreceptor mechanism. *Biophysical Journal* 89:56–63. doi:10.1529/biophysj.104.049346.

De Amicis, A., S. De Sanctis, S. Di Cristofaro, V. Franchini, F. Lista, E. Regalbuto, E. Giovenale, G. P. Gallerano, P. Nenzi, R. Bei, et al. 2015. Biological effects of in vitro THz radiation exposure in human foetal fibroblasts. *Mutat. Res.* 793:150–60. doi:10.1016/j.mrgentox.2015.06.003.

de Bruyn, L., and L. de Jager. 2010. Effect of long-term exposure to a randomly varied 50 hz power frequency magnetic field on the fertility of the mouse. *Electromagnetic Biology and Medicine* 29:52–61. doi:10.3109/15368371003776659.

Dhami, A. K. 2012. Study of electromagnetic radiation pollution in an Indian city. . *Environmental Monitoring and Assessment* 184:8597–8512. doi:10.1007/s10661-011-2436-5.

Dode, A. C., M. M. D. M. M. Leão, A. Tejo Fde, A. C. R. A. C. Gomes, D. C. Dode, M. C. Dode, C. W. Moreira, V. A. Condessa, C. Albinatti, and W. T. Caiaffa. 2011. Mortality by neoplasia and cellular

telephone base stations in the belo horizonte municipality, minas gerais state, brazil. *Sci. Total. Environ.* 409:3649–65. doi:10.1016/j.scitotenv.2011.05.051.

Dodson, C. A., P. J. Hore, and M. I. Wallace. 2013. A radical sense of direction: Signalling and mechanism in cryptochrome magnetoreception. *Trends in Biochemical Sciences* 38:435–46. doi:10.1016/j.tibs.2013.07.002.

Eskelinen, T., J. Keinänen, H. Salonen, and J. Juutilainen. 2002. Use of spot measurements for assessing residential ELF magnetic field exposure: A validity study. *Bioelectromagnetics* 23:173–76. doi:10.1002/bem.110.

Estenberg, J., and T. Augustsson. 2014. Extensive frequency selective measurements of radiofrequency fields in outdoor environments performed with a novel mobile monitoring system. *Bioelectromagnetics* 35:227–30. doi:10.1002/bem.21830.

Felley-Bosco, E. 1998. Role of nitric oxide in genotoxicity: Implication for carcinogenesis. *Cancer and Metastasis Reviews* 17:25–37. doi:10.1023/A:1005948420548.

Firlarer, A., R. Hamid, M. Cetintas, H. Karacadag, A. Gedik, M. Yogun, and M. Celik (2003). Measurement of electromagnetic radiation from GSM base stations. Proceedings of the IEEE International Symposium of Electromagnetic Compatibility, in Istanbul, Turkey, pp.1211–14.

Frei, P., E. Mohler, G. Neubauer, G. Theis, A. Bürgi, J. Fröhlich, C. Braun-Fahrländer, J. Bolte, M. Egger, and M. Röösli. 2009. Temporal and spatial variability of personal exposure to radio frequency electromagnetic fields. *Environmental Research* 109:779–85. doi:10.1016/j.envres.2009.04.015.

Gervasi, F., R. Murtas, A. Decarli, and A. G. Russo. 2019. Residential distance from high-voltage overhead power lines and risk of alzheimer's dementia and parkinson's disease: A population-based case-control study in a metropolitan area of northern italy. *International Journal of Epidemiology* 48:1949–57. doi:10.1093/ije/dyz139.

Grémiaux, A., S. Girard, V. Guérin, J. Lothier, F. Baluška, E. Davies, P. Bonnet, and A. Vian. 2016. Low-amplitude, high-frequency electromagnetic field exposure causes delayed and reduced growth in rosa . *Hybrida. Journal of Plant Physiology* 190:44–53. doi:10.1016/j.jplph.2015.11.004.

Gunnarsson, L. G., and L. Bodin. 2018. Amyotrophic lateral sclerosis and occupational exposures: A systematic literature review and meta-analyses int. *J. Environ. Res. Public Health*. 2018 15 .pii. : E2371. . doi: 10.3390/ijerph15112371.

Gunnarsson, L.-G. L.-G., and L. Bodin. 2019. Occupational exposures and neurodegenerative Diseases—A systematic literature review and meta-analyses. . *International Journal of Environmental Research and Public Health* 16:337. doi:10.3390/ijerph16030337.

Hardell, L., M. Carlberg, T. Koppel, and L. Hedendahl. 2017. High radiofrequency radiation at stockholm old town: an exposimeter study including the royal castle, supreme court, three major squares and the swedish parliament. *Mol. Clin. Oncol.* 6:462–76. doi:10.3892/mco.2017.1180.

Hardell, L., T. Koppel, M. Carlberg, M. Ahonen, and L. Hedendahl. 2016. Radiofrequency radiation at stockholm central railway station in sweden and some medical aspects on public exposure to RF fields. *Int. J. Oncol.* 49:1315–24. doi:10.3892/ijo.2016.3657.

He, Q., L. Zong, Y. Sun, V. P. J. Vijayalaxmi, J. Prihoda, J. Tong, and Y. Y. Cao. 2017. Adaptive response in mouse bone marrow stromal cells exposed to 900 MHz radiofrequency fields: impact of poly (ADP-ribose) polymerase (PARP). *Mutat. Res.* 820:19–25. doi:10.1016/j.mrgentox.2017.05.007.

Henderson, S. I., and M. J. Bangay. 2006. Survey of RF exposure levels from mobile telephone base stations in australia. *Bioelectromagnetics* 27:73–76. doi:10.1002/bem.20174.

Hintzsche, H., C. Jastrow, T. Kleine-Ostmann, H. Stopper, E. Schmid, and T. Schrader. 2011. Terahertz radiation induces spindle disturbances in human-hamster hybrid cells. *Radiat. Res.* 175:569–74. doi:10.1667/RR2406.1.

Hore, P. J. (2019). Upper bound on the biological effects of 50/60 Hz magnetic fields mediated by radical pairs. *Elife*. 8: e44179.

Hore, P. J., and H. Mouritsen. 2016. The radical-pair mechanism of magnetoreception. *Annual Review of Biophysics* 45:299–344. doi:10.1146/annurev-biophys-032116-094545.

Hosseinabadi, M. B., N. Khanjani, M. H. Ebrahimi, and J. Bigane. 2020. Estimation of thermal power plant workers exposure to magnetic fields and simulation of hazard zone. radiat. prot. *Dosimet* 1–8. doi:10.1093/rpd/ncaa101.

Huss, A., M. Egger, K. Hug, K. Huwiler-Müntener, and M. Röösli. 2007. Source of funding and results of studies of health effects of mobile phone use: Systematic review of experimental studies. *Environ. Health Perspect.* 115:1–4. doi:10.1289/ehp.9149.

Huss, A., S. Peters, and R. Vermeulen. 2018. Occupational exposure to extremely low-frequency magnetic fields and the risk of ALS: A systematic review and meta-analysis. *Bioelectromagnetics* 39:156–63. doi:10.1002/bem.22104.

Ilonen, K., A. Markkanen, G. Mezei, and J. Juutilainen. 2008. Indoor transformer stations as predictors of residential ELF magnetic field exposure. *Bioelectromagnetics* 29:213–18. doi:10.1002/bem.20385.

Jalilian, H., S. H. Teshnizi, M. Röösli, and M. Neghab. 2018. Occupational exposure to extremely low frequency magnetic fields and risk of Alzheimer disease: A systematic review and meta-analysis. *NeuroToxicology* 69:242–52. doi:10.1016/j.neuro.2017.12.005.

Ji, Y., Q. He, Y. Sun, J. Tong, and Y. Cao. 2016. Adaptive response in mouse bone-marrow stromal cells exposed to 900-MHz radiofrequency fields: Gamma-radiation-induced DNA strand breaks and repair. *Journal of Toxicology and Environmental Health, Part A* 79:419–26. doi:10.1080/15287394.2016.1176618.

Jiang, B., C. Zong, H. Zhao, Y. Ji, J. Tong, and Y. Cao. 2013. Induction of adaptive response in mice exposed to 900MHz radiofrequency fields: Application of micronucleus assay. *Mutat. Res.* 751:127–29. doi:10.1016/j.mrgentox.2012.12.003.

Jiang, B., J. Nie, Z. Zhou, J. Zhang, J. Tong, Y. Cao, and Y. Bai. 2012. Adaptive response in mice exposed to 900 MHz

radiofrequency fields: Primary DNA damage. *PLoS One* 7: e32040. doi:10.1371/journal.pone.0032040.

Joseph, W., G. Vermeeren, L. Verloock, M. M. Heredia, and L. Martens. 2008. Characterization of personal RF electromagnetic field exposure and actual absorption for the general public. *Health Physics* 95:317–30. doi:10.1097/01.HP.0000318880.16023.61.

Joseph, W., P. Frei, M. Roösli, G. Thuróczy, P. Gajsek, T. Trcek, J. Bolte, G. Vermeeren, E. Mohler, P. Juhász, et al. 2010. Comparison of personal radio frequency electromagnetic field exposure in different urban areas across Europe. *Environmental Research* 110:658–63. doi:10.1016/j.envres.2010.06.009.

Kim, B. C., and S.-O. S.-O. Park. 2010. Evaluation of RF electromagnetic field exposure levels from cellular base stations in korea.. *Bioelectromagnetics* 31:495–98. doi:10.1002/bem.20585.

Kirschvink, J. L., T. Kuwajima, and S. Ueno. 1992. Discrimination of low-frequency magnetic fields by honeybees: Biophysics and experimental tests. In *Sensory transduction, society of general physiologists, 45th annual symposium" in woods hole, MA, USA*, ed. D. P. Corey and S. D. Roper, 225–40. New York, NY: Rockefeller University Press.

Kirschvink, J. L., Walker, M.M., and Diebel, C.E. 2001. Magnetite-based magnetoreception. *Current Opinion in Neurobiology* 11:462–67. doi:10.1016/S0959-4388(00)00235-X.

Koeman, T., P. Slottje, L. J. Schouten, S. Peters, A. Huss, J. H. Veldink, H. Kromhout, P. A. van den Brandt, and R. Vermeulen. 2017. Occupational exposure and amyotrophic lateral sclerosis in a prospective cohort. *Occup. Environ. Med.* 74:578–85. doi:10.1136/oemed-2016-103780.

Kurnaz, C., and T. Aygun. 2020. Exposure assessment of radio frequency electromagnetic field levels in hospitals of Samsun Province, Turkey. *Environmental Science and Pollution Research* 27:34005–17. doi:10.1007/s11356-020-09669-1.

Lacy-Hulbert, A., J. C. Metcalfe, and R. Hesketh. 1998. Biological responses to electromagnetic fields 1. *The FASEB Journal* 12:395–420. doi:10.1096/fasebj.12.6.395.

Lahham, A., and A. Hammash. 2012. Outdoor radiofrequency radiation levels in the West . *Bank–Palestine. Radiation Protection Dosimetry* 149:399–402. doi:10.1093/rpd/ncr354.

Lahham, A., A. Sharabati, and H. Al Masri. 2015. Public exposure from indoor radiofrequency radiation in the city of hebron, west Bank—palestine. *Health Physics* 109:117–21. doi:10.1097/HP.0000000000000296.

Lahham, A., A. Sharabati, and H. Al Masri. 2017. ASSESSMENT OF PUBLIC EXPOSURE FORM WLANS IN THE WEST . *BANK—PALESTINE. Radiation Protection Dosimetry* 176:434–38. doi:10.1093/rpd/ncx028.

Lai, H. 2018. A summary of recent literature (2007-2017) on neurobiological effects of radiofrequency radiation. In *Mobile communications and public health*, ed. M. Markov, 187–222. Boca Raton, FL: CRC Press.

Lai, H. 2019. Exposure to static and extremely-low frequency electromagnetic fields and cellular free radicals. *Electromagnetic Biology and Medicine* 38:231–48. doi:10.1080/15368378.2019.1656645.

Lai, H., and N. P. Singh. 1995. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. *Bioelectromagnetics* 16:207–10. doi:10.1002/bem.2250160309.

Landler, L., and D. A. Keays. 2018. Cryptochrome: The magnetosensor with a sinister side? *PLOS Biology* 16:e3000018. doi:10.1371/journal.pbio.3000018.

Li, -S.-S.-S.-S., Z.-Y. Z.-Y. Zhang, C.-J. C.-J. Yang, H.-Y. H.-Y. Lian, and P. Cai. 2013. Gene expression and reproductive abilities of male drosophila melanogaster subjected to ELF–EMF exposure. *Mutat. Res.* 758:95–103. doi:10.1016/j.mrgentox.2013.10.004.

Lindgren, M., M. Gustavsson, Y. Hamnerius, and S. Galt. 2001. ELF magnetic fields in a city environment. *Bioelectromagnetics* 22:87–90. doi:10.1002/1521-186X(200102)22:2<87::AID-BEM1010>3.0.CO;2-W.

Mairs, R., K. J. Hughes, S. K. Fitzsimmons, K. M. S. Prise, A. K. M. Livingstone, L. A. Wilson, N. L. Baig, A. M. N. Clark, A. M. Timpson, G. A. Patel, et al. 2007. Microsatellite analysis for determination of the mutagenicity of extremely low-frequency electromagnetic fields and ionising radiation in vitro. . *Mutation Research/Genetic Toxicology and Environmental Mutagenesis* 626:34–41. doi:10.1016/j.mrgentox.2006.08.005.

Malyapa, R. S., E. W. Ahern, C. Bi, W. L. Straube, M. LaRegina, W. F. Pickard, and J. L. R. J. L. Roti Roti. 1998. DNA damage in rat brain cells after in vivo exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia. *Radiat. Res.* 149:637–45. doi:10.2307/3579911.

Manger, P. R., and J. D. Pettigrew. 1996. Ultrastructure, number, distribution and innervation of electroreceptors and mechanoreceptors in the bill skin of the platypus, *ornithorhynchus anatinus*. *Brain, Behavior and Evolution* 48:27–54. doi:10.1159/000113185.

Meltz, M. L., P. Eagan, and D. N. Erwin. 1990. Proflavin and microwave radiation: Absence of a mutagenic interaction. *Bioelectromagnetics* 11:1990. doi:10.1002/bem.2250110206.

Miyakoshi, J. 1997. Mutation induction by high-density, 50-Hz magnetic fields in human meWo cells exposed in the DNA synthesis phase. *Int. J. Radiat. Biol.* 71:75–79. doi:10.1080/095530097144445.

Miyakoshi, J., N. Yamagishi, S. Ohtsu, K. Mohri, and H. Takebe. 1996. Increase in hypoxanthine-guanine phosphoribosyl transferase gene mutations by exposure to high-density 50-Hz magnetic fields. *Mutat. Res.* 349:109–14. doi:10.1016/0027-5107(95)00166-2.

Miyakoshi, J., Y. Mori, N. Yamagishi, K. Yagi, and H. Takebe. 1998. Suppression of high-density magnetic field (400 mT at 50 Hz)-induced mutations by wild-type p53 expression in human osteosarcoma cells. *Biochem. Biophys. Res. Commun.* 243:579–84. doi:10.1006/bbrc.1997.8140.

Nittby, H., G. Grafström, D. P. Tian, L. Malmgren, A. Brun, B. R. R. B. R. R. Persson, L. G. Salford, and J. Eberhardt. 2008. Cognitive impairment in rats after long-term exposure to GSM-900 mobile phone radiation. *Bioelectromagnetics* 29:219–32. doi:10.1002/bem.20386.

Ono, T., Y. Saito, J. Komura, H. Ikehata, Y. Tarusawa, T. Nojima, K. Goukon, Y. Ohba, J. Wang, O. Fujiwara, et al. 2004. Absence of mutagenic effects of 2.45 GHz radiofrequency exposure in spleen, liver, brain, and testis of lacZ-transgenic mouse exposed in utero. *Tohoku J. Exp. Med.* 202:93–103. doi:10.1620/tjem.202.93.

Pakhomov, A., J. Bojarinova, R. Cherbunin, R. Chetverikova, P. S. Grigoryev, K. Kavokin, D. Kobylkov, R. Lubkovskaja, and N. Chernetsov. 2017. Very weak oscillating magnetic

field disrupts the magnetic compass of songbird migrants. *J. R. Soc. Interface*. 14:20170364. doi:10.1098/rsif.2017.0364.

Pedersen, C., A. H. Poulsen, N. H. Rod, P. Frei, J. Hansen, K. Grell, O. Raaschou-Nielsen, J. Schüz, and C. Johansen. 2017. Occupational exposure to extremely low-frequency magnetic fields and risk for central nervous system disease: An update of a Danish cohort study among utility workers. *International Archives of Occupational and Environmental Health* 90:619–28. doi:10.1007/s00420-017-1224-0.

Perov, S., N. Rubtsova, and Q. Balzano. 2019. Effects of 171 MHz low-intensity electromagnetic field on glucocorticoid and mineral corticoid activity of the adrenal glands of rats. *Bioelectromagnetics* 40:578–87. doi:10.1002/bem.22226.

Potenza, L., L. Cucchiarini, E. Piatti, U. Angelini, and M. Dachà. 2004. Effects of high static magnetic field exposure on different DNAs. *Bioelectromagnetics* 25:352–55. doi:10.1002/bem.10206.

Reif, D. W., and R. D. Simmons. 1990. Nitric oxide mediates iron release from ferritin. *Archives of Biochemistry and Biophysics* 283:537–41. doi:10.1016/0003-9861(90)90680-W.

Richardson, D. R., and P. Ponka. 1997. The molecular mechanisms of the metabolism and transport of iron in normal and neoplastic cells. *Biochim. Biophys. Acta* 1331:1–40.

Röösli, M., D. Jenni, L. Kheifets, and G. Mezei. 2011. Extremely low frequency magnetic field measurements in buildings with transformer stations in Switzerland. *Sci. Total. Environ.* 409:3364–69. doi:10.1016/j.scitotenv.2011.05.041.

Sagar, S., S. M. Adem, B. Struchen, S. P. Loughran, M. E. Brunjes, L. Arangua, M. A. Dalvie, R. J. Croft, M. Jerrett, J. M. Moskowitz, et al. 2018. Comparison of radiofrequency electromagnetic field exposure levels in different everyday microenvironments in an international context. *Environ. Int.* 114:297–306. doi:10.1016/j.envint.2018.02.036.

Sannino, A., O. Zeni, M. Sarti, S. Romeo, S. B. Reddy, M. A. Belisario, T. J. Prihoda, V. Vijayalaxmi, Scarfi, and M. R. ScarfiM.R. 2011. Induction of adaptive response in human blood lymphocytes exposed to 900 MHz radiofrequency fields: Influence of cell cycle. *Int. J. Radiat. Biol.* 87:993–99. doi:10.3109/09553002.2011.574779.

Sannino, A., O. Zeni, S. Romeo, R. Massa, G. Gialanella, G. Grossi, L. Manti, V. S. Vijayalaxmi, ScarfiM.R, and M. R. Scarfi. 2014. Adaptive response in human blood lymphocytes exposed to non-ionizing radiofrequency fields: Resistance to ionizing radiation-induced damage. *J. Radiat. Res.* 55:210–17. doi:10.1093/jrr/rrt106.

Sannino, A., O. Zeni, S. Romeo, R. Massa, and M. R. Scarfi. 2017. Adverse and beneficial effects in Chinese hamster lung fibroblast cells following radiofrequency exposure. *Bioelectromagnetics* 38:245–54. doi:10.1002/bem.22034.

Schrader, T., K. Münter, T. Kleine-Ostmann, and E. Schmid. 2008. Spindle disturbances in human-hamster hybrid (A L) cells induced by mobile communication frequency range signals. *Bioelectromagnetics* 29:626–39. doi:10.1002/bem.20428.

Schrader, T., T. Kleine-Ostmann, K. Münter, C. Jastrow, and E. Schmid. 2011. Spindle disturbances in human-hamster hybrid (AL) cells induced by the electrical component of the mobile communication frequency range signal. *Bioelectromagnetics* 32:291–301. doi:10.1002/bem.20634.

Sheppard, D. M. W. D. M., J. Li, K. B. Henbest, S. R. T. S. R. T. Neil, K. Maeda, J. Storey, E. Schleicher, T. Biskup, R. Rodriguez, S. Weber, et al. 2017. Millitesla magnetic field effects on the photocycle of an animal cryptochrome. *Scientific Reports* 7:42228. doi:10.1038/srep42228.

Sherrard, R. M., N. Morellini, N. Jourdan, M. El-Esawi, L.-D. Arthaut, C. Niessner, F. Rouyer, A. Klarsfeld, M. Doulazmi, J. Witczak, et al. 2018. Low-intensity electromagnetic fields induce human cryptochrome to modulate intracellular reactive oxygen species. *PLoS Biol.* 16: e2006229. doi:10.1371/journal.pbio.2006229.

Sisakht, M., M. Darabian, A. Mahmoodzadeh, A. Bazi, S. M. Shafiee, P. Mokarram, and Z. Khoshdel. 2020. The role of radiation induced oxidative stress as a regulator of radio-adaptive responses. *Int. J. Radiat. Biol.* 96:561–76. doi:10.1080/09553002.2020.1721597.

Sorahan, T., and N. Mohammed. 2014. Neurodegenerative disease and magnetic field exposure in UK electricity supply workers. *Occupational Medicine* 64:454–60. doi:10.1093/occmed/kqu105.

Sun, C., X. Wei, Y. Fei, L. Su, X. Zhao, G. Chen, and Z. Xu. 2016. Mobile phone signal exposure triggers a hormesis-like effect in Atm+/+ and Atm−/− mouse embryonic fibroblasts. *Sci. Rep.* 6:37423. doi:10.1038/srep37423.

Szymański, Ł., E. Sobiczewska, A. Cios, P. Szymanski, M. Ciepielak, and W. Stankiewicz. 2020. Immunotropic effects in cultured human blood mononuclear cells exposed to a 900 MHz pulse-modulated microwave field. *J. Radiat. Res.* 61:27–33. doi:10.1093/jrr/rrz085.

Takahashi, S., S. Inaguma, Y.-M. Cho, K. Imaida, J. Wang, O. Fujiwara, and T. Shirai. 2002. Lack of mutation induction with exposure to 1.5 GHz electromagnetic near fields used for cellular phones in brains of big blue mice. *Cancer Res.* 62:2002.

Tell, R. A., and R. Kavet. 2014. A survey of the urban radiofrequency (RF) environment. *Radiation Protection Dosimetry* 162:499–507. doi:10.1093/rpd/ncu021.

Thuroczy, G., F. Molnár, J. Szabó, G. Janossy, N. Nagy, G. Kubinyi, and J. Bakos (2006). Public exposure to RF from installed sources: Site measurements and personal exposimetry. Proceedings of the European Conference on Antennas and Propagation: EuCAP 2006 (ESA SP-626). 6-10 November 2006 in Nice, France, H. Lacoste, and L. Ouwehand (eds.) Published on CDROM, p.51.

Tkalec, M., K. Malarić, M. Pavlica, B. Pevalek-Kozlina, and Z. Vidaković-Cifrek. 2009. Effects of radiofrequency electromagnetic fields on seed germination and root meristematic cells of allium cepa L. *Mutat. Res.* 672:76–81. doi:10.1016/j.mrgentox.2008.09.022.

Urbinello, D., A. Huss, J. Beekhuizen, R. Vermeulen, and M. Röösli. 2014. Use of portable exposure meters for comparing mobile phone base station radiation in different types of areas in the cities of basel and amsterdam. *Sci. Total Environ.* 468-469:1028–33. 2014. doi:10.1016/j.scitotenv.2013.09.012.

Viel, J. F., S. Clerc, C. Barrera, R. Rymzhanova, M. Moissonnier, M. Hours, and E. Cardis. 2009. Residential exposure to radiofrequency fields from mobile phone base stations, and broadcast transmitters:

A population-based survey with personal meter. *Occup. Environ. Med.* 66:550–56. doi:10.1136/oem.2008.044180.

Waldmann-Selsam, C., A. B.-D. la Puente, H. A. Breunig, and A. H. Balmori. 2016. Radiofrequency radiation injures trees around mobile phone base stations. *Sci. Total Environ.* 572:554–69. doi:10.1016/j.scitotenv.2016.08.045.

Wilson, J. W., J. Haines, Z. Sienkiewicz, and Y. E. Dubrova. 2015. The effects of extremely low frequency magnetic fields on mutation induction in mice. *Mutat. Res.* 773:22–26. doi:10.1016/j.mrfmmm.2015.01.014.

Yakymenko, I., O. Tsybulin, E. Sidorik, D. Henshel, O. Kyrylenko, and S. Kyrylenko. 2016. Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation. *Electromagnetic Biology and Medicine* 35:186–202. doi:10.3109/15368378.2015.1043557.

Zeni, O., A. Sannino, S. Romeo, R. Massa, M. Sarti, A. B. Reddy, T. J. Prihoda, V. Vijayalaxmi, Scarfì, and M. R. ScarfìM.R. 2012. Induction of an adaptive response in human blood lymphocytes exposed to radiofrequency fields: Influence of the universal mobile telecommunication system (UMTS) signal and the specific absorption rate. *Mutat. Res.* 747:29–35. doi:10.1016/j.mrgentox.2012.03.013.

Zong, C., Y. Ji, Q. He, S. Zhu, F. Qin, J. Tong, and Y. Cao. 2015. Adaptive response in mice exposed to 900 MHz radiofrequency fields: Bleomycin-induced DNA and oxidative damage/repair. *Int. J. Radiat. Biol.* 91:270–76. doi:10.3109/09553002.2014.980465.

Supplement 1

Genetic effects of radiofrequency electromagnetic radiation (*study with no effect observed) Study reported effect =237 (66%); study reported no effect = 124 (34%) (Literature up to January 2021).

	Exposure conditions	Results
*Agarwal et al. (2009)	Human semen sample to cell phone radiation in talk mode for 1 h	No significant DNA damage, increase in reactive oxygen species; decrease in sperm motility and viability.
Aitken et al. (2005)	Mice to 900-MHz RFR for 7 days at 12 h/day; SAR 0.09 W/kg	Significant damage to Mitochondrial genome and nuclear β-globin locus in epididymal spermatozoa.
Akdag et al. (2016)	Male Wistar-Albino rats to 2400 MHz RFR from a Wi-Fi signal generator for a year; SAR 0.000141 (min)- 0.007127 (max) W/kg	No significant change in DNA single strand breaks (Comet assay) in brain, kidney, liver, and skin tissues, increased in testes.
Akdag et al. (2018)	Men who used cell phone for different durations per day; peak head SAR 0.45-0.79 W/kg	Increased DNA single strand breaks (Comet assay) in ear canal hair follicle cells; a dose-response relationship was observed.
Akhavan-Sigari et al. (2014)	Resected Glioblastoma multiforme (GBM) brain tumors from human patients	Increased mutant type of p53 expression in the peripheral zone of GBM in patient who use cell phone form ≥3h/day; the increase was significantly correlated with shorter overall survival time.
Alkis et al. (2019a)	Rats exposed to 900 MHz (brain SAR 0.0845 W/kg), 1800 MHz (0.04563 W/kg), and 2100 MHz (0.03957 W/kg) RFR 2 h/day for 6 months	Increased DNA single strand break (Comet assay), oxidative DNA damage, and oxidative stress in brain frontal lobe.
Alkis et al. (2019b)	Rats exposed to 900 MHz, 1800 MHz, and 2100 MHz RFR 2 h/day for 6 months; maximum SAR over the rat 0.017 W/kg	Increased DNA single strand beak (Comet assay), oxidative DNA damage and oxidative stress in testicular tissue.
Alkis et al. (2021)	Rats exposed to 1800	Significant increases in liver in 8-hydroxydeoxyguanosine, DNA single strand

	MHz (SAR 0.62 W/kg), 1800 MHz (0.04563 W/kg), or 2100 MHz (0.2 W/kg) RFR 2 h/day for 7 months	breaks (Comet assay), malondialdehyde, total oxidant status, oxidative stress index,
*Al-Serori et al. (2017)	Human U87 (wild-type) and U251 (mutated) glioblastoma cells exposed to intermittent (5 mi ON/10 min OF) UMTS 1750 MHz signal for 16 h, SAR 0.25, 0.5, and 1 W/kg	No effect on micronucleus frequency. Apoptosis was induced in U231 cells.
Al-Serori et al. (2018)	Ten human cell types exposed to intermittent (5 mi ON/10 min OF) UMTS 1750 MHz signal for 16 h, SAR 0.25, 0.5, and 1 W/kg	Increased in single strand breaks (Comet assay) in U87 p52- proficient glioblastoma cells grew under serum free condition; no effect on double strand breaks (γH2AX foci); nucleotide excision repair induced.
*Antonopoulos et al. (1997)	Human blood samples exposed to 380 MHz (17.65 Hz modulation, 0.08 W/kg); 900 MHZ (217 Hz modulation, 0.208 W/kg); or 1700 MHz (217 Hz modulation, 1.7 W/kg) for 48-68 h	No significant effect on cell cycle progression and frequency of sister-chromatin exchange in lymphocytes.
Atasoy et al. (2013)	Male Wister rats exposed to 2437 MHz (Wi-Fi) RFR; 24 h/day for 20 weeks; maximum SAR 0.091 W/kg	Increased oxidative DNA damage and decreased catalase and glutathione activities in blood and testes.
Atlı Şekeroğlu et al (2013)	Immature (whole body SAR 0.38-0.78 W/kg) and mature (0.31-0.52 W/kg) rats exposed to 900 MHz RFR 2 h/day for 45 days	Increased bone marrow cell chromosome aberration, micronucleus frequency, mitotic index and ratio of polychromatic erythrocytes. Cytogenetic damages in immature rats were significantly higher than in the mature rats. No recovery on day 15 post-exposure.
Balode (1996)	Blood samples from female Latvian Brown cows lived close to and in front of the Skrundra	Significantly higher micronucleus concentration was found in the erythrocytes of the exposed cows.

	Radar and from a control area	
Banerjee et al. (2016)	Buccal mucosal cells from subjects who used their cellular phone less than five years and less than three hours a week (low), and those who used more than five years and more than 10 hours a week comprised of the second group.	Micronucleated frequency in buccal mucosal cells was found to be significantly increased in longer cellular phone users.
Baohong et al. (2005)	Human lymphocytes exposed in vitro to 1800 MHz RFR (SAR 3 W/kg) for two hours and also co-treated with various mutagens	DNA strand break assayed (Comet assay) at 0 and 21 h after treatment. No effect when cells were exposed to RFR alone. But, RFR co-exposure enhanced the DMA damage induced by mitomycin C and 4-nitroquinoline-1-oxide.
Baohong et al. (2007)	Human lymphocytes exposed in vitro to 1800 MHz RFR (SAR 3 W/kg) for 0. 1.5, and 4 h. Cells were also co-treated with ultraviolet ray C	DNA damage as assayed by the Comet assay showed no significant effect with RFR alone. But, RFR co-exposure reduced DNA damage induced by ultraviolet C.
Beaubois et al. (2007)	Tomato plant leaves exposed to a 900-MHz RFR or 10 min at 0.066 mW/cm^2	Evoked rapid and substantial accumulation of basic leucine-zipper transcription factor (bZIP) mRNA in the terminal leaf with kinetics very similar to that seen in response to wounding. (Effect attenuated by calcium antagonist.)
Bektas et al (2020)	Pregnant women who used cell phone and Wi-Fi; placenta and cord blood samples were analyzed	Samples from cell phone users showed increased oxidative DNA damage and oxidative stress; Wi-Fi users showed increased oxidative DNA damage but no oxidative stress; more DNA single strand breaks (Comet assay) in cell phone users than in control (did not use cell phone nor Wi-FI) and Wi-Fi users; Wi-Fi and cell phone uses were synergistic.
Belyaev et al. (1992)	X-irradiated E. coli cells exposed to 51.62-51.84 GHz and 41.25-41.50 GHz millimeter-wave	Power density of 1 $\mu W/cm^2$ was sufficient to suppress X-radiation-induced repair of genome conformational state.

	RFR	
Belyaev et al. (2005)	Lymphocytes from human subjects exposed to GSM 915 MHz RFR for 2 h ; SAR 0.037 W/kg;	Increased condensation of chromatin; no significant difference between responses of blood samples of healthy and electro-hypersensitive subjects.
Belyaev et al. (2006)	Rats exposed to GSM 915 MHz RFR for 2 h, SAR 0.4 W/kg	Affected gene expression in brain cells; no significant effect on chromatin conformation and double strand DNA breaks.
Belyaev et al. (2009)	Human lymphocytes exposed to UMTS cell phone signal(1947.4 MHz, 5 MHz band width) for 1 h; SAR 0.04 W/kg	Chromatin affected and inhibition of DNA double-strand break co-localizing 53BPI/gamma-H2AX DNA repair foci; lymphocytes from electro-hypersensitive subjects responded differently to UMTS and GSM signals in the formation of DNA repair foci than in healthy subjects.
*Bisht et al. (2002)	Mouse embryo sarcoma fibroblast C3H 10T½ cells exposed to FDMA (835.62 MHz; SAR 3.2 or 5.1 W/kg) and CDMA (847.74 MHz; SAR 3.2 or 4.8 W/kg) RFR for 3, 8, 16 or 24h	No significant effect on micronucleus formation.
Bourdineaud et al. (2017)	earthworms (Eisenia fetida) exposed to 900 MHz for 2 h; SAR 0.00013-0.00933 W/kg	DNA genotoxic effect persisted for at least 24 h; gene expressions up regulated for HSP70 (heat shock protein), MEKKI (signal transduction); oxidative stress; and chemical and immune defenses.
*Bourthoumieu et al. (2010)	Human amniotic cells exposed to GSM-900 MHz RFR for 24 h; SAR 0.25 W/kg	No significant genotoxic effect was observed at 0 and 24 h after exposure by visual examination of chromosomal rearrangement.
*Bourthoumieu et al. (2011)	Human amniotic cells exposed to GSM-900 MHz RFR for 24 h; SAR 0.25, 1,2, and 4 W/kg	No significant change in the rate of aneuploidy of chromosomes 11 and 17 was found.
*Bourthoumieu et al. (2013)	Human amniotic cells exposed to GSM-900 MHz RFR for 24 h; SAR 0.25, 1,2, and 4 W/kg	No significant change in the expression and activation of the p53 protein was found. (p53 can cause cell cycle arrest and allow time for DNA repair or apoptosis.)
Burlaka et al. (2013)	Male Wister rats exposed to 245 MHz RFR for 2 h a day. 7 days a week for 2, 8, 15, or 30 days at 5-	Increased micronucleus formation was found in bone marrow erythropoietic cells after 15- day exposure; erythrocyte count, haemoglobin and haematocrit were

	10 mW/cm^2.	increased in peripheral blood after 8 and 15 days of exposure.
Buttiglione et al. (2007)	Human SH-SY5Y neuroblastoma cells exposed to modulated 900 MHz RFR for 24 h; SAR 1 W/kg	Increased Egr-1 gene expression paralleled with activation of the MAPK subtypes ERK1/2 and SAPK/JNK, and decrease in mRNA of Bcl-2 and surviving genes. RFR has anti-proliferative effect and causes cell cycle arrest at G2-M.
Cam and Seyhan (2012)	Hair root cells of human subjects after 15-30 min use of a 900-MHz GSM cell phone	Increased in DNA single strand breaks (Comet assay) was observed; more damages resulted after 30 min than after 15 min use.
Campisi et al. (2010)	Rat neocortical astroglial to 50 Hz-modulated or CW 900 MHz RFR for 5, 10, or 20 min; incident power density 0.0265 mW/cm^2	Significant increases in DNA fragmentation and reactive oxygen species were observed at 20 min only after exposure to the modulated RFR.
Cervellati et al. (2013)	Human placenta trophoblast-derived HTR-8/SVneo cells exposed to 1.8 GHz GSM RFR amplitude modulated by rectangular pulses of 217 Hz for 1 h; SAR 2 W/kg	Increased connexin Cx40 and Cx43 mRNA expression; decreased Integrin alpha1 and β 1 mRNA levels but enhanced Int alpha5 mRNA expression.
Chandel et al. (2019a)	Onion roots (Allium cepa L.) were exposed to 2350 MHz RFR for 1, 2, or 4 h, SAR 0.313 W/kg	Increased in mitotic index and chromosomal aberration; significant increase in DNA single strand break (Comet assay) at 2 and 4 h.
Chandel et al. (2019b)	Onion roots (Allium cepa L.) were exposed to 2100 MHz RFR for 1 or 4 h, SAR 0.282 W/kg	Increased mitotic index, chromosomal aberration, and DNA single-strand breaks (Comet assay) after 4 h of exposure.
*Chang et al. (2005)	Escherichia coli and Salmonella typhimurium exposed to 835 MHz RFR for 48h; SAR 4W/kg	835-MHz RFR neither affected the reverse mutation frequency nor accelerated DNA degradation in vitro. (Some interaction effects with mutagens were observed.)
Chaturvedi et al. (2011)	Male mice exposed to 2450 MHz RFR, 2 h/day for 30 days; SAR 0.03561 W/kg	Increased DNA single strand breaks (Comet assay) in brain cells.
*Chauhan et al. (2006a)	Human lymphoblastoma cells (TK6) exposed to pulsed-modulated,	No evidence of a general stress response with proto-oncogene and heat-shock protein gene transcriptions.

	intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 6 h; SAR 1 or 6 W/kg	
*Chauhan et al. (2006b)	Human –derived immune cell-lines HL-60 and MM6 cells exposed to pulsed-modulated, intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 6 h; SAR 1 or 10 W/kg	No evidence of detectable change in stress-related gene expression.
*Chauhan et al. (2007)	Human glioblastoma-derived cell-line (U87MG) and human monocyte-derived cell-line (MM6) exposed to pulsed-modulated, intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 24 and 6 h; SAR 0.1-10 W/kg	No evidence that the RFR exposure altered late onset gene expression in either cultured cell-lines.
Chavdoula et al. (2010)	Drosophila melanogaster flies exposed to GSM-900 MHz and DCS-1800 MHz cell phone radiation; 6 min per day for 5 days	Decreased insect's reproductive capacity with fragmented DNA (apoptosis) in the egg chamber.
*Chemeris et al. (2004)	Frog (Xenopus laevis) erythrocytes exposed to high peak power pulsed RFR (8.8 GHz, 180 ns pulse width, peak power 65 kW, repetition rate 50 Hz) for 40 min; SAR 1.6 kW/kg (peak SAR 300 MW/kg)	Increased DNA single strand breaks (Comet assay) caused by temperature rise.
*Chemeris et al. (2006)	Human whole blood leukocytes and isolated lymphocytes exposed to pulsed 8.8 Hz RFR (180 ns pulse width, peak power 65 kW, pulse repetition frequency 50 Hz) for 40 min: average SAR 1.6 kW/kg (peak	No change in DNA single strand breaks (Comet assay)

	300 mW/kg)	
Chen et al. (2012)	Saccharomyces cerevisiae yeast cells exposed to 1800 MHz RFR for 6 h; SAR 4.7 W/kg	Expression of several genes.
*Choi et al. (2020)	Human adipose tissue-derived stem cells (ASCs), Huh7 and Hep3B liver cancer stem cells (CSCs), HeLa and SH-SY5Y cancer cells, and normal fibroblast IMR-90 cells exposed to WCDMA-signal 1.7-GHz RFR for 72 h, SAR 1 and 2 W/kg	No significant effect on double strand breaks; increased intracellular reactive oxygen species and deceased proliferation.
*Ciaravino et al. (1991)	Chinese hamster ovary cells exposed to 2450-MHz pulsed RFR (SAR 33.8 W/kg) simultaneously with adriamycin for 2 h	RFR did not affect changes in cell progression and number of sister chromatid exchanges induced by adriamycin.
d'Ambrosio et al. (1995)	Human blood exposed to 9 GHz RFR (continuous-wave or 50-Hz amplitude modulated) for 10 min; SAR 90 W/kg	Increased in micronucleus frequency in lymphocytes after exposure to the amplitude modulated RFR.
d'Ambrosio et al. (2002)	Human blood cultures exposed to 1748 MHz RFR (continuous –wave or phase modulated (GMSK)) for 15 min: SAR ~5 W/kg	Micronucleus frequency in lymphocytes was increased only after exposure to phase-modulated RFR.
Danese et al. (2017)	Human whole blood exposed to 900 MHz RFR from a cell phone for 30 min	No change in frequency of γ-H2AX foci (double strand DNA breaks) in lymphocytes.
De Amicis et al. (2015)	Human fetal fibroblasts exposed to THz radiation (0.1-0.15 THz) for 20 min; SAR 15-20 W/kg	Increased total number of micronuclei and centromere positive micronuclei that could lead to chromosome loss. No significant effect on DNA strand breaks (Comet assay), phosphorylation of H2AX histone and apoptosis.
De Iuliis et al. (2009)	Human spermatozoa exposed to 1800-MHz	Increased oxidative DNA damage and fragmentation (apoptosis) and reactive

	RFR; SAR 0.4 – 27.5 W/kg for 16 h	oxygen species; sperm motility and vitality were reduced.
*de Oliveira et al. (2017)	Human buccal cells from cell phone users; Averaged years of use 11.4 yrs; mean duration of daily use 2.8 min	Cells ipsilateral to cell phone use did not have a statistically significantly higher micronucleus frequency, compared to cells contralateral to exposure.
Del Re et al. (2019)	Human HeLa, BE2C and SH-SY5Y cells exposed to 900 MHz 217-Hz pulse-modulated RFR for 48 h; SAR 1 W/kg	Increased transcription of repetitive DNA, type of transcription depended on cell type. (Alteration of repetitive DNA transcription can be induced by environmental stress conditions, causing human pathological effects.)
Del Vecchio et al. (2009)	Murine SN56 cholinergic cell line (48 and 72 h) and rat primary cortical neurons (24, 72, 120 h) exposed to GSM-modulate 900 MHz RFR; SAR 1 W/kg	Increased expression of beta-thymosin (cytoskeleton regulating factor) m-RNA, and reduced neurite generation.
Demsia et al. (2004)	Rats exposed to 910-MHz RFR 2 h/day for 30 days; SAR 0.42 W/kg.	Increased of micronuclei in polychromatic polymorphonuclear cells in bone marrow smears. Effects less in female rats.
Deshmukh et al. (2013)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 30 days.	Increased DNA single strand breaks (Comet assay) in brain tissues.
Deshmukh et al. (2015)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 180 days.	Increased DNA single strand breaks (Comet assay) in brain tissues; elevated heat-shock protein-70 level.
Deshmukh et al. (2016)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week	Increased DNA single strand breaks (Comet assay) in brain tissues; elevated heat-shock protein-70 level.

	for 90 days.	
Diem et al.(2005)	Human diploid fibroblasts and cultured rat granulosa cells exposed to 1800 MHz intermittent (5 min On/10 min Off) or continuous –wave; SAR 1.2 or 2 W/kg	Increased in DNA single and double strand breaks (Comet assay) in both cell types after 16 h exposure. Intermittent wave showed a higher effect than continuous wave.
Duan et al (2015)	Mouse spermatocyte-derived GC-2 cells exposed to intermittent (5 min On/10 min Off) 1800 MHz RFR (from a GSM cell phone in talk mode) for 24 h; SAR 1. 2 , or 4 W/kg	Increased oxidative DNA damage a 4 W/kg; no significant with Comet assay.
*Durdik et al. (2019)	Umbilical cord blood (UCB) cells exposed to a GSM900 (1-17 h, 0.004 or 0.04 W/kg) or UMTS-1947.4 MHz (3 h, 0.04 /kg) cell phone signals fed to a TEM cell	No changes in DNA single and double strand breaks (Comet assay), and apoptosis; increased reactive oxygen species was observed.
Eker et al. (2018)	Female Wistar-albino rats exposed to 1800-MHz RFR for 2h/day for 8 weeks; SAR 0.06 W/kg	Caspase-3 and p38MAPK gene expressions increased in eye tissues.
Engelmann et al. (2008)	Cell suspension cultures of Arabidopsis thaliana exposed to 1900 MHz UMTS-modulated RFR for 24 h; SAR peak 2 W/kg, average 0.75 W/kg	Significant changes in transcription of 10 genes.
Esmekaya et al. (2011)	Human peripheral blood lymphocytes exposed to 1800 MHz GSM- (217 HZ) modulated RFR for 6, 8, 24, or 48 h; SAR 0.21 W/kg	Chromatin changes and increase in sister chromatin exchange.
*Falzone et al. (2010)	Human spermatozoa exposed to pulse-modulated 900-MHz RFR for 1 h; SAR: 2.0 and 5.7 W/kg	No significant effects on DNA fragmentation, reactive oxygen species, and capase-3 activity.

Ferreira et al. (2006)	Pregnant rats exposed to a cell phone at 834 MHz for 8.5 h/day from conception to birth; SAR 0.55-1.23 W/kg	Increased erythrocyte micronucleus frequency but no significant effects in oxidative parameters in blood and liver of newborn pups.
Figueiredo et al. (2004)	Human whole blood exposed to 2.5 GHz RFR (from a microwave oven) for 40 sec (SAR 626.67 W/kg) or 10.5 GHz RFR for 5 min (SAR 0.25 W/kg)	No chromosomal aberrations observed in lymphocytes; no alteration in radiosensitivity to gamma radiation; cell mortality increased markedly after RFR exposure.
*Finnie et al. (2006)	Pregnant mice exposed to 900-MHz RFR (modulated at 217 Hz with pulse-width of 0.6 ms) for 60 min per day from day 1-19 of gestation; SAR 4 W/kg	No significant effect on c-fos expression in brain of offspring.
Fragopoulou et al. (2018)	C57BL/6 adult male mice exposed to 2 hr to GSM 1800-MHz RFR (from a phone) for 2 h at an average power density of 0.0049-0.081 mW/cm^2	In the hippocampus, the expression of 178 genes changed significantly, revealing an impact on genes involved in critical biological processes, such as cell cycle, DNA replication and repair, cell death, cell signaling, nervous system development and function, immune system response, lipid metabolism, and carcinogenesis.
Franchini et al. (2018a)	Human fetal and adult fibroblasts exposed to 25 GHz RFR for 20 min; SAR 20W/kg	Increased total number of micronuclei and centromere positive micronuclei in exposed samples. No significant effect on DNA single strand break (Comet assay).
Franchini et al. (2018b)	Human adult fibroblasts exposed to 0.15 THz (150 GHz) RFR (4 μs pulses at 25 Hz) for 20 min; SAR 15-20 W/kg	Increased centromere-positive micronuclei frequencies and chromosomal nondisjunction events, indicating induction of aneuploidy and not by DNA breakage.
Franzellitti et al. (2008)	Human trohoblastes HTR-8/SVneo exposed to 1800 MHz continuous-wave, GSM-217-Hz, and GSM-Talk signals for 4-24 h, time averaged SAR 2 W/kg	Levels of the inducible HSP70C transcript were significantly enhanced after 24 h exposure to GSM-217Hz signals and reduced after 4 and 16 h exposure to GSM-Talk signals. No effect on inducible HSP70A, HSP70B and the constitutive HSC70 transcripts.
Franzellitti et al. (2010)	Human trophoblast HTR-8/SVneo cells exposed to1800 MHz	GSM signals increased DNA single strand breaks (Comet assay) after 16 and 24 h exposure; recovered within 2 h post-

	continuous –wave. GSM (217 Hz modulated) and GSM intermittent (5 min on/10 min off) RFR for 4. 16, or 24 h: SAR 2 W/kg	exposure; continuous-wave RFR was without effect.
*Fritze et al. (1997)	Rats expose to GSM 90 MHz RFR for 4 h, brain average SAR 0.3- 1.5 W/kg	No effect on C-jun and GFAP expression in brain.
Fucic et al. (1992)	Lymphocytes from humans occupationally exposed to RFR; 1250-1350 MHz, 10 $\mu W/cm^2$-20 mW/cm^2	Showed preferentially clastogenic effect measured by micronucleus. Effect on genetic material similar to both of a chemical agent and of ionizing radiation.
Furtado-Filho et al. (2014)	Rats of different ages (0-30 days) exposed 950 MHz RFR for 0.5 h/day for 51 days (21 days of gestation and 6-30 days old): SAR pregnant rat 0.01-0.03 W/kg; neonate 0.88 W/kg, 6-day old 0.51 W/kg, 15-day old 0.18 W/kg, 30-day old 0.06 W/kg.	Decreased DNA single strand breaks (Comet assay) in liver of 15-day old and increased breaks in 30-day old rats, no oxidative stress detected.
*Furtado-Filho et al. (2015)	At exposed to 950 MHz RFR. 0.5 h/day to 27 days (throughout pregnancy and 6 days postnatal); SAR 0.44-0.35 W/kg, neonatal rat 1.32 W/kg, 6-day old 1.14 W/kg	Right cerebral cortex showed an increase in DNA single strand breaks (Comet assay), but no significant effect in the left cerebral cortex in RFR-exposed 6-day old rats. No oxidative effects observed.
Gadhia et al. (2003)	Blood samples of cell phone and non-cell phone users	Increased dicentric chromosomes and sister chromatid exchange in lymphocytes of cell phone users.
Gajski and Garaj-Vrhovac (2009)	Blood samples from Wistar rats exposed to GSM-modulated 915 MHz RFR for 30 min, SAR 0.6 W/kg	Increased basal (single strand) and oxidative DNA damage (Comet assay) in lymphocytes.
Gandhi and Anita (2005)	Blood from cell phone users (most for 2-5 yrs)	Increased DNA single strand breaks (Comet assay) and micronucleus found in cell phone users.
Gandhi and Singh	Blood and buccal cells	Increased micronucleated buccal cells and

(2005)	from cell phone users (3-4,5 yrs); controls never used cell phone	chromosomal aberration in peripheral lymphocytes.
Gandhi et al. (2015)	People lived within 300 m of a cell phone base station (average power density= 1.149 mW/cm^2) for an average of 7.45 yrs, controls average power density = 0.0045 mW/cm^2.	Increased DNA single strand breaks (Comet assay) in peripheral blood leukocytes. Daily cell phone usage, location of residence, and power density are significant predictor of DNA damage.
Gapeyev et al. (2014)	Mouse blood samples exposed to 1-Hz pulse-modulated 42.2 GHz RFR for 20 min, SAR 1.5 W/kg; and x-rays	Pre-exposure to pulse-modulated RFR (not continuous-wave) reduced x-ray-induced DNA single strand breaks (Comet assay) in lymphocytes Effect may be related induction of reactive oxygen species by RFR.
Garaj-Vrhovac et al. (1990)	V79 Chinese hamster cells exposed to 7.7 GHz RFR for 15, 30, or 60 min; power density 30 mW/cm^2	Inhibited [^{3}H]thymidine into DNA with stoppage of cell cycle at S phase; chromosome aberration observed.
Garaj-Vrhovac et al. (1991)	V79 Chinese hamster fibroblast cells exposed to 7.7 GHz RFR for 15, 30, or 60 min; power density 0.5 mW/cm^2	Increased chromosome aberration (dicentric and ring chromosomes) and micronucleus.
Garaj-Vrhovac et al. (1992)	Human whole blood samples exposed to 7.7 GHz RFR for 10, 30, or 60 min; power density 0.5, 10, or 30 mW/cm^2	Increased chromosome aberration (dicentric and ring chromosomes) and micronucleus in lymphocytes.
Garaj-Vrhovac and Fucic (1993)	Air traffic controllers who did repair on radar devices two days ago and exposed to 1250-1350 MHz RFR of unknown intensity (pulse power 100 kW). (presumably higher than normal exposure of 10 $\mu W/cm^2$-20 mW/cm^2)	Lymphocytes showed increased number of chromosome breaks, acentric fragments, dicentric and polycentric chromosomes with accompanying fragments, ring chromosomes and chromatid interchange. Most aberrations returned to normal after 30 weeks, except dicentrics and ring chromosomes.
Garaj-Vrhovac. (1999)	Peripheral blood lymphocytes of workers on radar equipment and antenna system service, 1250-1350 MHz; power	Exposed subjects shows an increase in the number of micronucleus and number of micronucleus per cell; disturbance of cells in the cell cycle.

	density 10 μW/cm^2 -20 mW/cm^2; average employment duration 13.3 yrs	
Garaj-Vrhovac and Orescanin (2009)	Peripheral blood lymphocytes of workers on radar equipment and antenna system service, 1250-1350 MHz; power density 10 μW/cm^2 -20 mW/cm^2; average employment duration 13.3 yrs	Increased DNA single strand breaks (Comet assay) and bleomycin-induced chromatid breakage.
Garaj-Vrhovac et al. (2009)	Wistar rats exposed to 915 MHz RFR 1 h/day for two weeks, SAR 0.6 W/kg	Increased basal DNA single strand break and oxidative DNA damages (Comet assay) in blood leukocytes.
Garaj-Vrhovac et al. (2011)	Workers occupationally exposed to marine radar pulsed RFR (3, 5.5, and 9.4 GHz)	Increased DNA single strand break (Comet assay) and micronucleus in blood lymphocytes; increased oxidative stress.
*Garson et al. (1991)	Blood samples of radio-linemen occupationally exposed to 400 kHz – 20 GHz	No increase in chromosomal damage in lymphocytes.
Ghatei et al. (2017)	Mice exposed pre- and post-natally to radiation from a cellular phone jammer (900 and 1800 MHz)	At 8-10 weeks old, in the cerebellum, no effect on expression level of bcl-2 and p53 genes, but gene expression level of *bax* was decreased and gene expression level of *p21* was increased.
*Glaser et al. (2016)	Human hematopoietic stem cells and leukemia HL-60 cells exposed to GSM (900 MHz), UMTS (1,950 MHz) and LTE (2,535 MHz) for 4, 20 or 66 h;SAR 0-4 W/kg	No effect on apoptosis, oxidative stress, cell cycle, DNA damage (DNA single strand breaks (Comet assay)) and DNA repair. A significant decrease in DNA breaks was found in hematopoietic stem cells exposed for 4 h to GSM signal.
Gökçek-Saraç et al. (2020)	Rats exposed to UMTS 2100 MHz RFR 2h/day for 7 days; whole body average SAR 0.47or 2.17 W/kg	Decreased RNA expressions of acetylcholinesterase (AChE), choline acetyltransferase (ChAT), and vesicular acetylcholine transporter (VAChT) in the hippocampus; deficit in object location and Y-maze tests.
*Görlitz et al. (2005)	B6C3F1 mice exposed to GSM900 or DCS 1800 signals for 2 h/day for 1	No effect on micronucleus frequency in erythrocytes of the bone marrow or peripheral blood, in keratinocytes, or in

	week (SAR 0-33.2 W/kg) or 6 weeks (SAR 0-24.9 W/kg)	spleen lymphocytes.
Gorpinchenko et al. (2014)	Human sperms exposed to a cell phone in stand-by/talk mode for 5 h	Increased DNA fragmentation (apoptosis) and decreased motility in spermatozoa.
Gulati et al. (2016)	Blood and buccal cells of people lived close (<400 meters) to a cell tower; 1800 MHz, Maximum power density (at 150 meters) 1.22 $\mu W/cm^2$, some subjects lived in the area for more than 9 yrs	Inceased DNA single strand breaks (Comet assay) in lymphocytes and micronucleus in buccal cells. Female subjects had significantly higher effects than males.
Gulati et al. (2018)	Blood samples from subjects lived 400 m from cell towers for 8-9 years, power density 0.037-12.20 mW/cm^2	A significant association of genetic polymorphism of antioxidant genes (for MnSOD and CAT) with oxidative damage has been observed in human population exposed to radiations emitted from mobile towers. Decreased MnSOD and CAT activities and increased lipid peroxidaton observed in blood serum.
Gulati et al. (2020)	Human lymphocytes exposed to UMTS signals at 1923, 1947.47, and 1977 MHz for 1 or 3 hr; SAR 40 mW/kg	Observed DNA damage (Comet assay) depending on UMTS frequency wth maximal effect at 1977 MHz; no effects on ROS, apoptosis, preleukemic fusion genes, and mutations in TP53 gene.
Guler et al (2010)	Pregnant and non-pregnant New Zealand white rabbit exposed to GSM 1800-MHz RFR for 15 min/day for 7 days (15^{th} to 22^{nd} days of gestation); power density 0.052 mW/cm^2	Increased oxidative DNA damage and lipid peroxidation in brain tissues in adult rabbits, no significant effect in newborn rats
Guler et al. (2012)	New Zealand white rabbits exposed to GSM 180-MHz RFR for 15 min/day in utero between 15^{th} to 22^{nd} days of gestation and at 1-month old 15 min/day 7 days for female and 14 days for male; SAR 1.8 W/kg	Increased DNA oxidative damage in liver of female rabbits (not in male) and increased lipid peroxidation in liver of both male and female rabbits.
*Gurbuz et al. (2010)	Female Wistar rats	No significant effect on micronucleus

	exposed to GSM 1800-MHz RFR 20 min/day, 5 days/week for 1 month; power density 0.0054 mW/cm^2	frequency in bladder cells.
*Gurbuz et al. (2014)	Male Wistar rats exposed to 1800- or 2100-MHz RFR 30 min/day, 6 days/week for 1 or 2 months; SAR 0.23 W/kg	No significant effect on micronucleus frequency in bladder cells.
*Gurbuz et al. (2015)	Normal and diabetic rats exposed to a 2100-MHz RFR 30 min/day, 5 days/week for 1 month; SAR 0.24 W/kg	No effect on micronucleus frequency in exfoliated bladder cells in both normal rats and rats with chronic disorder.
*Gurisik et al. (2006)	Two human cell lines (neuronal SK-N-SH) and monocytoid U937) exposed to a GSM 900-MHz RFR for 2 h; SAR 0.2W/kg	No significant effects on gene expression, heat shock protein level, and cell cycle distribution in SK-N-SH cells; and no effects on cell viability and cell cycle in U937 cells.
Gürler (2014)	Wistar rats exposed to 2450 MHz RFR 1 h/day for 30 consecutive days; power density 0.0036 mW/cm^2	Increased oxidative DNA damage in brain and blood, and oxidative protein products in blood.
Gustavino et al. (2016)	Secondary roots of Vicia faba (broad bean) seedlings exposed to continuous-wave 915-MHz RFR for 2 h; SAR 0.4-1.5 W/kg	Increased micronucleus frequency up to 7-fold.
Habauzit et al. (2014)	Human keratinocytes exposed to 60.4 GHz RFR for 3 hr, incident power density of 20 mW/cm^2: SAR 594 W/kg (average), 1233 M/kg (peak)	7 gene expressions showed specific electromagnetic effect under hyperthermia condition (i.e., not mimicked by heat-shock controls).
* Habauzit et al. (2020)	Male hairless rats exposed to 94 GHz RFR 3 h/day, 3 days/week for 5 months, incident power density 10 mW/cm2	No significant modification of gene expression in skin cells.
Haider et al. (1994)	Plant cutting bearing young flower buds	Increased micronucleus was found in all conditions (compared to lab controls).

	exposed for 30 h to short-wave 10-21 MHz RFR on both sides of a slewable curtain antenna (0.424-7.67 mW/cm^2), at 15 m (2.15 mW/cm^2) and 30 m (1.3 mW/cm^2) from a cage antenna; and 200 m from a broadcasting station (0.00027-0.0024 mW/cm^2)	
Hanci et al. (2013)	Pregnant rats exposed 1 h/day on days 13-21 of pregnancy to 900-MHz RFR at power density 0.0265 mW/cm^2.	Testicular tissue of 21-day old offspring showed increased DNA oxidative damage, apoptotic index, and lipid peroxidation.
*Hansteen et al. (2009a)	Human lymphocytes exposed to18 GHz or pulsed 16.5 GHz RFR for 53 h	No significant effect on chromosomal aberration frequency.
*Hansteen et al. (2009b)	Human lymphocytes exposed to 2.3 GHz continuous-wave or pulsed (200 Hz, 50% duty cycle) RFR	No significant effect on chromosomal aberration frequency.
Hao et al. (2010)	Murine N9 microglial cells were exposed to pulsed 2450-MHz RFR for 20 min, SAR 6.2 W/kg	Significant induced phosphorylation of STAT3, increased transcription levels of the inflammation-associated genes, iNOS and TNF-alpha, which are reported to contain STAT-binding elements in their promoter region. (STAT3 is a transcription activator that mediates the expression of a variety of genes in response to cell stimuli, and thus plays a key role in many cellular processes such as cell growth and apoptosis.)
He et al. (2016)	Mouse bone marrow stromal cells exposed to a 900 MHz RFR 3 h/day for 5 days; peak and average SAR 4.1 x 10^{-4} and 2.5 x 10^{-4} W/kg	Increased expression of PARP-1 mRNA. (PARP-1 involved in DNA repair, genomic stability and apoptosis and is activated by DNA single strand breaks.)
He et al. (2017)	Mouse bone marrow stromal cells exposed to a 900 MHz RFR 3 h/day for 5 days; peak and average SAR 4.1 x 10^{-4}	Induced PARP-1. Cells exposed to RFR and gamma ray showed significantly decreased genetic damage (DNA single strand break (Comet assay)) as well as faster kinetics of repair compared with those exposed to GR

	and 2.5 x 10^{-4} W/kg, some cells were challenged with one dose of gamma ray.	alone.
Hekmat et al. (2013)	Calf thymus exposed to 940 MHz RFR for 45 min; SAR 0.04 W/kg	Altered DNA structure at 0 and 2 h after exposure; conformational changes and disaggregation caused by increment in surface charge and size of DNA.
*Hintzsche and Stopper (2010)	Oral cavity mucosa cells from human subjects who used cell phones for different durations weekly (0, <3 h, and > 3h)	No significant change in micronucleus frequency in mucosa cells with cell phone use.
*Hintzsche et al. (2012a)	Human HaCaT cells and A(L) human-hamster hybrid cells exposed to continuous-wave or GSM-modulated 900 MHz RFR for 30 min or 22 h; power density 0.0066-2.15 mW/cm^2	No significant effect on micronucleus frequency.
*Hintzsche et al. (2012b)	Human keratinocytes (HaCaT) and human dermal fibroblasts (HDF) exposed to 0.106 THz (106 GHz) RFR for 2, 8, 24 h; 0.88 -2 mw/cm^2 (2mw/cm^2 gave a SAR of 13.34 W/kg)	No effect on micronucleus frequency and DNA single strand breaks (Comet assay).
*Hirose et al. (2006)	Human glioblastoma A172 cells exposed to 2.1425 GHz W-CDMA radiation at SARs of 0.08, 0.25, and 0.8 W/kg, and continuous-wave radiation at 0.08 W/kg for 24 or 48 h; and human IMR-90 fibroblasts from fetal lungs exposed to both W-CDMA and continuous-wave RFR at a SAR of 0.08 W/kg for 28 h	No significant changes in induction of p53-dependent apoptosis, DNA damage, or other stress response
*Hirose et al. (2007)	Human glioblastoma	No significant induction of phosphorylation

	A172 cells were exposed to W-CDMA radiation at SARs of 0.08 and 0.8 W/kg for 2-48 h, and continuous-wave 2.1425 GHz RFR at 0.08 W/kg for 24 h, and human IMR-90 fibroblasts from fetal lungs were exposed to W-CDMA at 0.08 and 0.8 W/kg for 2 or 28 h, and continuous-wave at 0.08 mW/kg for 28 h.	of hsp27 or expression of heat shock protein gene family.
*Hook et al. (2004)	Human Molt-4 T lymphoblastoid cells exposed to 847.74 MHz code-division multiple-access (CDMA) (SAR 3.2 W/kg), 835.62 MHz frequency-division multiple-access (FDMA) (3.2 W/kg), 813.56 MHz iDEN(R) (iDEN) (0.0024 or 0.024 W/KG), and 836.55 MHz time-division multiple-access (TDMA) (0.0026 or 0.026 W/kg) for up to 24 h	No significant changes in DNA single strand breaks (Comet assay) and apoptosis.
*Hou et al. (2015)	Mouse embryonic fibroblasts (NIH/3T3) exposed to intermittent (5 min on/10 min off) 1800-MHz GSM-talk mode RFR from 0.5 to 8 h; SAR 2 W/kg.	No effect on γH2AX foci frequency (Increased reactive oxygen species and late apoptotic cells).
Houston et al. (2019)	Male mice exposed to 906 MHz RFR for 12 h/day for 1, 3, or 5 weeks; SAR 2.2 W/kg	Increased DNA oxidative and fragmentation (Comet assay) in spermatozoa across all exposure periods, increased mitochondrial reactive oxygen species.
*Huang et al. (2008a)	Jurkat human T lymphoma cells exposed for 24 h to 1763 MHz RFR; SAR 10 W/kg	Alterations in cell proliferation, cell cycle progression, DNA integrity (Comet assay) or global gene expression were not detected.
*Huang et al. (2008b)	HEI-OC1 immortalized mouse auditory hair cells	No significant effects on cycle distribution, DNA damage (Comet assay), stress response

	exposed to 1763 MHz (CDMA) RFR for 24 or 48 h; SAR 20 W/kg	and gene expression.
*Jeong et al. (2018)	14-month old C57BL/6 mice exposed to 1950 MHz RFR for 2 h/day, 5 day/wk, 8 months; SAR 5 W/kg	No significant effects on levels of oxidative stress, oxidative DNA damage, apoptosis, astrocyte, or microglia markers in brain tissues.
Jeong et al. (2020)	2 and 12-month old C57BL/6 mice exposed to 1950-MHz RFR 2h/day, 5 day/wk for 8 months; SAR 5 W/kg	Increased expression of Epha8 and Wnt6 genes in the hippocampi at 20 months after exposure, although 13 additional genes showed no significant changes. Cognitive enhancement detected in 1-month mice after exposure may be associated with increases in neurogenesis-related signals.
Ji et al (2004)	Human subjects used cell phones for 4 h.	DNA single strand breaks (Comet assay) increased in peripheral blood cells (T-cells, B-cells, granulocytes).
Ji et al. (2016)	Mouse bone-marrow stromal cells (BMSC) exposed to 900-MHz RFR for 4 h/day for 5 days; power density 0.12 mW/cm^2; some cells were also irradiated with 1.5 Gy γ-radiation after RFR exposure	RFR followed by γ-radiation exposure significantly decreased number of DNA strand breaks (Comet assay) and resulted in faster kinetics of repair of DNA strand breaks compared to γ-radiation alone. Thus, data suggest that RFR preexposure protected cells from damage induced by γ-radiation.
Jiang et al. (2012)	Mice were pre-exposed to a 900-MHz RFR for 4 h/day for 1, 3, 5, 7, and 14 days; power density 0.12 mW/cm2 and then subjected to an acute dose of 3 Gy γ-radiation	DNA single strand breaks (Comet assay) in blood leukocytes from mice pre-exposed to RFR for 3, 5, 7, and 14 days showed progressively decreased damage and was significantly different from those exposed to γ-radiation alone.
Jiang et al. (2013)	Mice exposed to a 900-MHz RFR 4/day for 7 days, SAR 0.548 W/kg and also γ-radiation	Pre-exposure to RFR decreased micronucleus frequency induced by γ-radiation in immature erythrocytes in peripheral blood and bone marrow.
*Juutilainen et al. (2007)	Female CBA/S mice were exposed for 78 weeks (1.5 h/day, 5 day/week) to either a continuous 902.5-MHz signal similar to that emitted by analog NMT (Nordic Mobile	No significant effects of RFR on micronucleus frequency in polychromatic or normochromatic erythrocytes.

	Telephone) phones at a whole-body SAR of 1.5 W/kg, or to a pulsed 902.4-MHz signal similar to that of digital GSM phones at 0.35 W/kg and also 4 Gy of X-ray on the first three weeks; female transgenic mice (line K2) and their nontransgenic littermates were exposed for 52 weeks (1.5 h/day, 5 day/week) to two digital mobile phone signals, GSM and DAMPS at SAR 0.5 W/kg, and repeated ultraviolet radiation	
Karaca et al. (2012)	Mouse brain cells exposed to a 10.715 GHz RFR for 6 h/day for three days, SAR 0.725 W/kg	Increased micronucleus apoptosis and necrosis, and decreased expression of the STAT3 genes.
*Kerbacher et sl. (1990)	Chinese Hamster Ovary cells exposed for 2 h to pulsed 2450 MHz RFR; SAR 33.8 W/kg	No significant effect on chromosome aberration; no interactions with Mitomycin C and Adriamycin.
Kesari and Behari (2009)	Male Wistar rats exposed to 50-GHz RFR 2 h/day for 45 days; SAR 0.0008 W/kg	Increased in brain tissue DNA double strand breaks (Comet assay); decreased antioxidant enzymes superoxides dismutase and glutathione peroxidase, and increased catalase activity.
Kesari et al. (2010)	Male Wistar rats exposed to 2.45-GHz RFR 2 h/day for 35 days; SAR 0.11 W/kg	Increased in brain tissue DNA double strand breaks (Comet assay); decreased antioxidant enzymes superoxides dismutase and glutathione peroxidase, and increased catalase activity.
Kesari et al. (2011)	Male Wistar rats exposed to 900 MHz-GSM signal 2 h/day for 35 days; SAR 0.9 W/kg	Decreased micronucleus frequency, change in cell cycle and increased oxidative stress in sperm cells.
Kesari et al. (2014)	Male Wistar rats exposed to a 3D cell phone. 2h/day for 60 days; SAR 0.26 W/kg	Increased DNA double strand breaks (comet assay), micronuclei, Caspase 3 and apoptosis in brain cells; activation of hsp27/p38MAPK stress pathway.
*Khalil et al (2011)	Mice exposed to 900	No effects on plasma, brain, and spleen 8-

	MHz-GSM signal 30 min/day for 30 days; SAR 1 W/kg	oxo-7, 8-dihydro-2'- deoxyguanosine and oxidative stress.
Khalil et al. (2012)	Male Sprague-Dawley rats exposed for 2 h to 1800-MHz GSM signal, SAR 1 W/kg	Urine samples collected 0.5, 1, 2, and 4 h from the beginning of exposure showed elevated 8-oxo-7, 8-dihydro-2'-deoxyguanosine (from repair of oxidative DNA damage) level.
*Khalil et al. (2014)	Saliva of cellular phone users collected before as well as after 15 and 30 min use of phones.	No change in 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-Oxo-dG). There was no relationship between cell phone use and changes in the salivary oxidant/antioxidant profile.
Kim et al. (2008)	Mouse lymphoma cells and Chinese hamster lung cells exposed to 835-MHz RFR for 48 h; SAR 4W/kg	RFR increased clastogens-induced DNA single strand breaks (Comet assay).
*Komatsubara et al. (2005)	Mouse m5S cells exposed for 2 h to 2450 MHz CW RFR (SAR 5,10, 20, 50 and 100 W/kg) or pulsed RFR (SAR mean 100W/kg, peak 900 W/kg)	No chromosomal aberration observed.
Korenstein-Ilan et al (2008)	Human dividing lymphocytes exposed to 0.1 THz RFR (0.031 mW/cm^2) for 1, 2, or 24 h	Change in chromosomes number in chromosoms11 and 17 were most vulnerable (about 30% increase in aneuploidy after 2 and 24 h of exposure), while chromosomes 1 and 10 were not affected, and in the asynchronous mode of replication of centromeres 11, 17 and 1 (by 40%) after 2 h of exposure. 0.1 THz radiation induces genomic instability. It is speculated that these effects are caused by radiation-induced low-frequency collective vibration modes of proteins and DNA.
Koyama et al. (2003)	Chinese hamster ovary (CHO)-K1 cells exposed to 2450 MHz RFR for 18 h:SAR 13-100 W/kg	Higher micronucleus frequency after exposure at 78 W/kg and higher. Synergistic with bleomycin in microbnucleus formation.
Koyama et al. (2004)	Chinese hamster ovary K1 cells exposed to 2450 MHz RFR for 2h; SAR5-200 W/kg	Increased micronucleus formation above 50 W/kg (May be related to temperature rise).

*Koyama et al. (2016a)	Human corneal epithelial (HCE-T) cells exposed to 0.12 THz radiation at 5 mW/cm² for 24 h	No effect on micronucleus formation, morphological change and hest shock protein expression (Hsp27, Hsp70, and Hsp90α).
*Koyama et al. (2016b)	Human corneal epithelial (HCE-T) and human lens epithelial (SRA01/04) cells exposed to 60 gigahertz (GHz) RFR for 24 h; 1 mW/cm2	No effect on micronucleus formation DNA single strand breaks (Comet assay) and heat shock protein expression.
Kumar A. et al. (2020)	Allim cepa (onion) root meristematic cells exposed to 900- (0.0902 W/kg) and 1800-MHz (0.169 W/kg) RFR for 0.5, 1, 2, and 4 h	Increased chromosomal aberrations and increased DNA single strand breaks (Comet assay).
*Kumar G. et al. (2011)	Long bone (femur and tibia) of male Sprague – Dawley rats exposed to 900-MHz continuous-wave RFR for 30 min; SAR 2 W/kg	No significant effect on DNA single-strand breaks (Comet assay) in bone marrow lymphocytes.(Assayed at 72 h after exposure.)
*Kumar G. et al. (2015)	Long bone (femur and tibia) of male Sprague – Dawley rats exposed to 900 and 1800 MHz continuous-wave and pulsed RFR; 900-MHz CW at 2 and 10 W/kg for 90 min and 1800-MHz CW and PW at 2.5 and 12.4 W/kg for 120 min	No significant effect on DNA single-strand breaks (Comet assay) in bone marrow lymphoblasts. (Assayed at 1 h after exposure.)
Kumar R. et al. (2020)	male Wistar rats exposed to 900 MHz, 1800 MHz and 2450 MHz RFR at a specific absorption rate (SAR) of 5.84×10^{4} W/kg, 5.94×10^{-4} W/kg and 6.4×10^{-4} W/kg, respectively for 2 h per day for 1-month, 3-month and 6-month periods.	RFR exposure caused significant epigenetic modulations (DNA and histone methylation) which alter gene expression in the hippocampus.
Kumar S. et al. (2010)	Male Wistar rats exposed to 10-GHz RFR 2 h a day for 45 days, SAR	Increased micronucleus and reactive oxygen species in blood cells.

	0.014 W/kg	
Kumar S. et al. (2013)	Male Wistar rats exposed to a 10 GHz RFR 2h/day for 45 days; SAR 0.014 W/kg	Increased micronucleus frequency in blood lymphocytes and increased single strand breaks (Comet assay) in spermatozoa. Decreased testosterone and testicular size.
Kumar S. et al. (2014)	Male Wistar rats exposed to 1910.6 MHz RFR from a cell phone in "talk mode" for 60 days (2 h/day, 6 days a week); SAR 0.28 (Max.) and 0.0226 (Min.)	Increased DNA single strand breaks (Comet assay) an lipid peroxidation in spermatozoa,
*Lagroye et al. (2004a)	Sprague-Dawley rats exposed to pulsed 2450-MHz RFR for 2 h; SAR 1.2 W/kg	No significant change in DNA single strand breaks (Comet assay) (with or without proteinase-k treatment of samples-for detection of DNA-protein crosslinks) in brain cells.
*Lagroye et al. (2004b)	Clonal mouse embryo C3H 10T(1/2) cells exposed 2450-MHz continuous-wave RFR for 2 h; SAR 1.9 W/kg	No significant change in DNA single strand breaks (Comet assay) (with or without proteinase-k treatment of samples.)
Lai and Singh (1995)	Male Sprague-Dawley rats exposed to pulsed or continuous-wave 2450-MHz RFR for 2 h; SAR 0.6 and 1.2 W/kg	Increased DNA single strand breaks (Comet assay) in brain cells was observed at 4 h after exposure to pulsed RFR and at 0 and 4 h after continuous-wave exposure.
Lai and Singh (1996)	Male Sprague-Dawley rats exposed to pulsed or continuous-wave 2450-MHz RFR for 2 h; SAR 1.2 W/kg	Increased DNA single- and double-strand breaks (Comet assay) in brain cells was observed at 4 h after exposure to pulsed or continuous-wave RFR.
Lai and Singh (1997)	Male Sprague-Dawley rats exposed to pulsed 2450-MHz RFR for 2 h; SAR 1.2 W/kg	Increased DNA single- and double-strand breaks (Comet assay) in brain cells at 4 h after exposure. Effects blocked by melatonin or the spin-trap compound N-tert-butyl-alpha-phenylnitrone. (Free radicals are involved in the effects).
Lai and Singh (2005)	Male Sprague-Dawley rats exposed to continuous-wave 2450-MHz RFR for 2 h; SAR 0.6 W/kg	Increased DNA single- and double-strand breaks (Comet assay) in brain cells at 4 h after exposure. Effects blocked by a temporally incoherent magnetic field.
Lai et al. (1997)	Male Sprague-Dawley rats exposed to pulsed 2450-MHz RFR for 2 h;	Increased DNA double-strand breaks (Comet assay) in brain cells at 4 h after exposure. Effect blocked by naltrexone. (Involvement

	SAR 1.2 W/kg	of endogenous opioids in the effects).
Lakshmi et al. (2010)	Human subjects professionally using VDTs	No effect on DNA single strand break (comet assay) and micronucleus frequency in blood cells of subjects exposed for 2 years; increased in long-term (>10 years) users.
Lameth et al. (2020)	Healthy rats, rats undergoing an acute neuroinflammation triggered by a lipopolysaccharide (LPS) treatment, and transgenic hSOD1^{G93A} rats that modeled a presymptomatic phase of human amyotrophic lateral sclerosis (ALS) exposed head only to a GSM-1800 MHz RFR for 2 h, SAR 3.22 W/kg.	Cortical cell gene modulations triggered by GSM-RFR in the course of an acute neuroinflammation and indicate that GSM-induced gene responses can differ according to pathologies affecting the CNS.
*Lamkowski et al. (2018)	Human peripheral blood cells exposed to 900 MHz RFR for 30, 60, and 90 min; SAR 9.3 W/kg	No significant effect on gene expression.
Le Quément et al. (2012)	Primary human skin cells exposed to a 60.4-GHz RFR for 1, 6, or 24 h, SAR 42.4 W/kg.	Expression of 130 transcripts was found to be potentially modulated. PCR confirmed 5 genes as differentially expressed after 6 h of exposure.
*Lerchl et al. (2020)	Pregnant mice exposed to UMTS ~1960 MHz RFR from day 7 post-conception (p.c.) at SAR 0.04 and 0.4 W/kg (24 h/day, 7 days/week);at day 14 p.c., injected with ethylnitrosoures(ENU)	No DNA adenyl adduct formation was observed in the brain of fetuses at 24, 36, and 72 h after ENU inection.
Lee et al. (2005)	Human HL-60 cells exposed to a pulsed 2450 MHz RFR for 2 or 6 h; SAR 10 W/kg	Many genes apoptosis-related genes were affected. Apoptosis- related genes were among the upregulated ones and the cell cycle genes among the downregulated ones.
*Li et al. (2001)	Murine C3H 10T(1/2) fibroblasts exposed to 847.74 MHz code-division multiple access (CDMA) and 835.62 frequency-division	No significant effect on DNA single strand breaks (Comet assay).

	multiple access (FDMA) RFR for 2, 4, or 24 h; SAR 3.2 - 5.1 W/kg	
Li et al. (2018)	Mouse spermatocyte-derived cells (GC-2) were exposed to 1800-MHz RFR for 24 h, SAR 1, 2 or 4 W/kg	No effect on DNA double strand streak, increased DNA single strand breaks (Comet assay); free radicals involved.
Li et al. (2020)	Pregnant female rats exposed to 1800 (1 mW/cm^2) and 2400 (0.1 mW/cm^2) MHz RFR during the 21st day of pregnancy (8 pm- 8 am). Offspring tested from 3-9 weeks postnatal	Up- and down-regulation expressions of different forms (NR1, NR2A, NR2B, NR2C, NR2D, NR3A, NR3B) of methyl-D-aspartate receptors (NMDARs) in the hippocampus were obsersed; animals showed behavioral and cognitive development effects which may be associated with altered mRNA expression of NMDARs.
Lin et al. (2016)	Budding yeast exposed to 2-GHz RFR for 96 h, SAR 0.12 W/kg	Upregulation of the expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle.
Liu et al. (2013a)	Mouse spermatocyte-derived GC-2 cell line exposed to 1800-MHz Global System for Mobile Communication (GSM) signals (5 min on and 10 min off) for 24 h; SAR 1, 2, or 4 W/kg	Increased DNA single strand breaks (comet assay) and DNA adduct 8-oxoguanine at SAR of 4 W/kg; increased reactive oxygen species generation.
Liu et al. (2013b)	Mouse spermatocyte-derived GC-2 cell line was exposed to a commercial mobile phone handset once every 20 minutes in standby, listen, dialed or dialing modes for 24 h; power density 0.0059-0.0122 mW/cm^2	Increased DNA single strand breaks (Comet assay) (attenuated by melatonin).
Lixia et al. (2006)	Human lens epithelial cells exposed to GSM-1.8 GHz RFR for 2 h, SAR 1, 2, 3 W/kg	Increased DNA single strand breaks (comet assay) at 3 W/kg at o and 30 min post-exposure; Increased mRNA and protein expression of Hsp70.
López-Martín et al. (2009)	Picrotoxin-pretreated male Sprague-Dawley rats exposed to 900-MHz GSM-modulated or unmodulated RFR for 2	Increased c-fos expression in brain areas.

	h, SAR modulated RFR 0.03 W/kg average– peak 0.14 W/kg in brain; unmodulated RFR average 0.26 W/kg- peak 1.4 w/kg in brain	
Luukkonen et al. (2009)	Human SH-SY5Y neuroblastoma cells exposed to 872-MHz (CW and GSM) RFR for 1 h; SAR 5 W/kg	CW RFR increased DNA single strand breaks (Comet assay) and reactive oxygen species in cells treated with menadione (a chemical that induces intracellular ROS production and DNA damage) compared to cells treated with menadione alone. GSM-modulated RFR had no significant effect.
*Luukkonen et al. (2010)	Human SH-SY5Y neuroblastoma cells exposed to 872-MHz (CW and GSM) RFR for 3 h (DNA damage) and 1 h (reactive oxygen species) ; SAR 5 W/kg	CW and modulated RFR had no significant effect on DNA single strand breaks (Comet assay) and reactive oxygen species production in cells treated with ferrous chloride,
Maes et al (1993)	Human peripheral blood lymphocytes exposed to pulsed2450-MHz RFR for 30 or 120 min, SAR 75 W/kg	Increase in the frequency of chromosome aberrations (including dicentric chromosomes and acentric fragments) and micronuclei.
Maes et al (1996)	Human whole blood samples exposed to GSM 954- MHz emitting antenna for 2 h, SAR 1.5 W/kg, some samples also incubated with mitomycin C after exposure	Synergistic effect between RFR and mitomycin C was observed the frequencies of sister chromatid exchanges in metaphase figures.
Maes et al. (1995)	Human whole blood cells exposed to 954 MHz RFR from an antenna for 2 h; SAR 1.5 W/kg. Blood from maintenance workers of transmission antenna (450, 900 MHz) exposed at least 1 h/day for a year.	Increased chromosome aberration (dicenric chromosome) in lymphocytes. No effect found in blood of antenna maintenance workers.
*Maes et al. (1997)	Human whole blood cells exposed to 935.2 MHz RFR alone and in	No significant effects of RFR on chromosome aberration, sister chromatid exchange, and DNA single strand breaks

	combination with mitomycin C for 2 h; SAR 0.3-0.4 W/kg	(comet assay). No synergistic effect with mitomycin C.
*Maes et al (2000)	Human lymphocytes exposed to 455.7 MHz RFR from antenna of a car phone for 2 h; SAR 6.5 W/kg	No significant effects of RFR on chromosome aberration and sister chromatid exchange. No synergistic effect with mitomycin C.
*Maes et al (2001)	Human lymphocytes exposed to 900-MHz RFR for 2 h, SAR 0-10 W/kg	No significant effects of RFR on chromosome aberration and sister chromatid exchange. No synergistic effect with mitomycin C.
*Maes et al (2006)	Peripheral blood lymphocytes from subjects who were professionally exposed to cell phone RFR	No evidence of RFR-induced genetic effects: DNA single strand breaks (Comet assay), chromosome aberration, and sister chromatid exchange.
*Malini (2017)	Blood and semen samples from subjects who used cellular phones for 1-5. 6-10, and >10h/day.	No DNA damages (ladder assay) and oxidative changes observed.
*Malyapa et al. (1997a)	U87MG and C3H 10T1/2 cells exposed to 2450-MHz continuous-wave RFR for 2 h; SAR 0.7 and 1.9 W/kg	No significant effects on DNA single strand breaks (Comet assay).
*Malyapa et al. (1997b)	Mouse C3H 10T1/2 fibroblasts and human glioblastoma U87MG cells exposed to 835.62 MHz (FMCW) and 847.74 MHz (CDMA) RFR up to 24 h; SAR 0.6 W/kg	No significant effects on DNA single strand breaks (Comet assay).
*Malyapa et al. (1998)	Male Sprague-Dawley rats exposed to 2450 MHz continuous-wave (CW) RFR for 2 h; SAR 1.2 W/kg	No significant effects on DNA single strand breaks (Comet assay) in cerebral cortex or hippocampus.
Manti et al. (2017)	Four days-old adult female flies (Drosophila melanogaster) exposed to GSM-1800 talk mode RFR emitted by a commercial cellular	168 genes were differentially expressed associated with multiple and critical biological processes, such as basic metabolism and cellular subroutines related to stress response and apoptotic death. Free radicals may be involved.

	phone for 30 min; SAR 0.15 W/kg	
Manti et al. (2008)	Human peripheral blood lymphocytes exposed a UMTS 1.95 GHz signal for 24 h; SAR 0.5 and 2.0 W/kg; some samples also exposed to x-ray	X-ray induced chromosome exchange per cell was increased by RFR exposure. (RFR may either influence the repair of X-ray-induced DNA breaks or alter the cell death pathways of the damage response.)
Marinelli et al. (2004)	acute T-lymphoblastoid leukemia cells exposed to 900 MHz RFR for 2-48 h, SAR 0.0035 W/kg	Increased DNA damage (DNA ladder) and activation genes involved in pro-survival signaling.
Markova et al. (2005)	Human lymphocytes exposed to 905 and 915 MHz GSM signals for 1 h. SAR 0.037 W/kg	RFR from GSM cell phone affected chromatin conformation and 53BP1/gamma-H2AX foci similar to heat shock. No significant difference between lymphocytes from healthy and electro-hypersensitive subjects.
Markova et al. (2010)	Human diploid VH-10 fibroblasts and human adipose-tissue derived mesenchymal stem cells exposed to GSM (905 MHz or 915 MHz) or UMTS (1947.4 MHz, middle channel) RFR for 1, 2, or 3 hr; SAR 0.037-0.039 W/kg	915 MHz and 1947.4 MHz signals inhibited tumor suppressor TP53 binding protein 1 (53BP1) foci that are typically formed at the sites of DNA double strand break location in both cell types. 905 MHz RFR did not inhibit 53BP1 foci in differentiated cells but in stem cells. (Inability to form DNA repair foci has been correlated to radiosensitivity, genomic instability, and other repair deficits.)
Martin et al. (2020)	Human neonatal foreskin keratinocytes (HEK-3N, HEK-1N, and NHEK-3N) and human skin keratinocyets HeCAT exposed to a 60-GHZ RFR for 3 h, Average SAR 513 W/kg and peak SAR 1233 W/kg	Different cell types showed different patterns of expreson of ADAMTS6, IL7R, and NOG genes.
Mashevich et al. (2003)	Human peripheral blood lymphocytes exposed to830 MHz RFR for 72 hr, SAR 1.6-8.8 W/kg	A linear increase in chromosome 17 aneuploidy (loss and gain of chromosome) and abnormal chromosome-17 replication were observed as a function of the SAR value, demonstrating that this radiation has a genotoxic effect.
Mazor et al. (2008)	Human lymphocytes exposed to continuous-wave800 MHz for 72 hr;	Increased levels of aneuploidy depending on the chromosome studied as well as on the level of exposure. In chromosomes 1 and 10,

	SAR 2,9 and 4,1 W/kg	there was increased aneuploidy at the higher SAR, while for chromosomes 11 and 17, the increases were observed only for the lower SAR.
*McNamee et al. (2002a)	Human blood cultures exposed to continuous-wave 1900 MHz RFR for 2 h; SAR 0-10 W/kg	No effect on DNA single strand breaks (Comet assay) in leukocytes.
*McNamee et al. (2002b)	Human blood cultures exposed to pulsed 1900 MHz RFR for 2 h; SAR 0-10 W/kg	No effect on DNA single strand breaks (Comet assay) and micronucleus formation in leukocytes.
*McNamee et al. (2003)	Human blood cultures exposed to continuous-wave or pulsed 1900 MHz RFR for 24 h; SAR 0-10 W/kg	No effect on DNA single strand breaks (Comet assay) and micronucleus formation in leukocytes.
*McNamee et al. (2016)	Male C57BL/6 mice exposed to pulse-modulated or continuous-wave 1900 MHz RFR for 4 h/day for 5 consecutive days; whole body average SAR ~0.2 W/kg and ~1.4 W/kg.	No differentially expressed gene expressions were identified in various regions of the brain.
Meena et al. (2014)	Wistar rats exposed to 2.45 MHz RFR 2 h/day for 45 days; SAR 0.14 W/kg. Rats also treated with melatonin.	Increased in DNA single strand breaks (Comet assay) and oxidative stress in testicular tissue. Effects attenuated by melatonin.
Megha et al. (2015a)	Fischer rats exposed to 900 and 1800 MHz RFR for 30 days (2 h/day, 5 days/week); SAR 0.00059 and 0.00058 W/kg	Reduced levels of neurotransmitters dopamine, norepinephrine, epinephrine, and serotonin, and downregulation of mRNA of tyrosine hydroxylase and tryptophan hydroxylase (synthesizing enzymes for the transmitters) in the hippocampus.
Megha et al. (2015b)	Fischer rats exposed to 900, 1800, and 2450 MHz RFR for 60 days (2 h/day, 5 days/week); SAR 0.00059, 0.00058, and 0.00066 W/kg	Increased DNA single-strand breaks (Comet assay) in hippocampus, increased oxidative stress and pro-inflammatory cytokines (IL-2, IL-6, TNF-α, and IFN-γ)
*Meltz et al. (1990)	Mouse leukemic cells exposed to pulsed 2450	No evidence in any mutagenic action by the RFR exposure alone or interaction with

	MHz RFR for 4 h, SAR 40 W/kg	proflavin, a DNA-intercalating drug.
Mildažienė et al. (2019)	Sunflower seeds exposed to 5.28 MHz RFR for 5, 10, 15 min, 12.7 kV/m	RFR exposure induced a long-term effect on gene expression in leaves, mostly stimulating expression of proteins involved in photosynthetic processes and their regulation.
Millenbaugh et al. (2008)	Rats exposed to 35 GHz RFR at 75 mW/cm^2 untik colonic temperature reached 41-41^oC, skin was assayed	Changes were detected in 56 genes at 6 h and 58 genes at 24 h post-exposure. Genes associated with regulation of transcription, protein folding, oxidative stress, immune response, and tissue matrix turnover were affected at both times. At 24 h, more genes related to extracellular matrix structure and chemokine activity were altered.
*Miyakoshi et al. (2002)	Human brain tumor derived M)54 cells exposed to 2450 MHz RFR for 2 h; SAR 50 or 100 W/kg	No effect on DNA single strand breaks (Comet assay) observed.
*Mizuno et al. (2015)	WI38VA13 subcloned 2RA human fibroblast cells exposed to wireless power transfer (WPT) 12.5 MHz resonant frequency for 48, 96, or 144 h; SAR 21 W/kg	No effects on cell growth, cell cycle distribution, DNA single strand breaks (Comet assay), micronucleus formation, and hypoxanthine-guanine phosphoribosyltransferase (HPRT) gene mutation.
*Nakatani-Enomoto et al. (2016)	Human spermatozoa exposed to to 1950 MHz Wideband Code Division Multiple Access (W-CDMA)-like RFR for 1 h; SAR 2.0 or 6.0 W/kg	No effect on percentage of 8-hydroxy-2'-deoxyguanosine positive spermatozoa.
Narasimhan and Huh (1991)	Lambdaphage DNA exposed to short pulses of RFR	Observed conformational anomolies in DNA probably resulting from single strand breaks and localized strand separations induced by RFR.
Nikolova et al. (2005)	Mouse embryonic neural progenitor stem cells exposed to 1710-MHz GSM RFR for 6 or 48 h; SAR 1.5 W/kg	Exposure for 6 h, but not for 48 h, resulted in a low and transient increase of DNA double-strand breaks and the transcript level of genes related to apoptosis and cell cycle control..
Nittby et al. (2008)	Fischer 344 rats exposed to 1800 MHz GSM RFR	Expression in cortex and hippocampus of genes connected with membrane functions.

	for 6 h; SAR whole body average 0.013 W/kg, head 0.03 W/kg	
Nylund and Leszczynski (2006)	Human endothelial cell line: EA.hy926 and EA.hy926v1exposed to 900-MHz GSM RFR for 1 h; SAR 2.8 W/kg	Gene and protein expression were altered dependent on the cell type.
Odaci et al. (2016)	Pregnant Sprague - Dawley rats exposed to 900 MHz RFR 1 h each day during days 13 - 21 of pregnancy; SAR whole body average 0.024 W/kg	Testis and epididymis of offspring showed higher DNA oxidation and lipid peroxidation at 60 days postnatal.
Ohtani et al. (2016)	Sprague-Dawley rats exposed to wideband code division multiple access 2140 MHz RFR for 6 h or 3 or 6 h/day for 4 days, SAR 4 or 0.4 W/kg	Exposure at 4 W/kg (at 6 h/day) increased core temperature and upregulation of some stress markers, heat-shock proteins and heat-shock transcription factors family, in the cerebral cortex and cerebellum.
*Ohtani et al. (2019)	Mice exposed to 85 kHz (for charging electrical vehicles) EMF at 25.3 mT, 1 h/day for 10 days	No significant change in gene transcriptional expression in brain and liver.
*Ono et al. (2004)	Pregnant lacZ-transgenic mice exposed intermittently (10 sec On, 50 sec OFF) 16 h/day to 2450-MHz RFR from embryonic days of 0 to 15; SAR whole body average 0.71 W/kg	No significant effects on mutation frequencies at the lacZ gene in spleen, liver, brain, and testis in offspring. The RFR is not mutagenic in utero.
Ozgur et al. (2014)	Hepatocarcinoma cells exposed to intermittent (15 min ON, 15 min OFF) GSM 900- and 1800-MHz RFR for 1, 2, 3, or 4 h; SAR 2 W/kg	Cells showed irregular nuclei pattern and DNA damage (apoptosis).
Pacini et al. (2002)	Human skin fibroblasts exposed to GSM 904.2-MHz RFR for 1 h (from a cell phone); SAR 0.6 W/kg	Increased the expression of mitogenic signal transduction genes (e.g., MAP kinase kinase 3, G2/mitotic-specific cyclin G1), cell growth inhibitors (e.g., transforming growth factor-beta), and genes controlling apoptosis (e.g., bax).

Panagopoulos et al. (2007)	Flies (Drosophila melanogaster) exposed to either GSM 900-MHz or DCS 1800-MHz signals from a digital cell phone, for few minutes per day during the first 6 days of their adult life.	Degeneration of large numbers of egg chambers after DNA fragmentation (apoptosis) of their constituent cells, induced by both types of mobile telephony radiation.
Panagopoulos (2019)	Human peripheral blood lymphocytes exposed to UMTS signal (1900-2200 MHz) using a cell phone for 15 min	Chromatid-type aberrations (gaps and breaks) observed.
Panagopoulos (2020)	Human lymphocytes (in G2/M phase) exposed to UMTS (3G) 1920-1960 MHz RFR entted from a smart phone on talk mode for 15 min; peak power density 92 $\pm$27 $\mu W/cm^2$; averaged over 6 min 29 $\pm$ 14 $\mu W/cm^2$	Chromatid-type aberrations were observed. Effect synergistic with caffeine.
Pandey et al. (2017)	Swiss albino mice exposed to 900-MHz RFR for 4 or 8 h per day for 35 days; SAR 0.0054-0.0516 W/kg	RFR exposure-induced oxidative stress causes DNA single-strand breaks (Comet assay) in germ cells, with altered cell cycle progression leading to low sperm count in mice (depolarization of mitochondrial membranes resulting in destabilized cellular redox homeostasis). Larger effect with longer exposure time, and recovery at 35 days post-exposure.
Pandey and Giri (2018)	Swiss albino mice exposed to GSM 900-MHz RFR 3h twice/day for 35 days, SAR 0.0516-0.0054W/kg	Increased DNA single strand breaks (Comet assay) and free radicals in testis and germ cells, effects attenuated by melatonin.
*Paparini et al. (2008)	Mice exposed to GSM 1800-MHz signal for 1 h; SAR whole body average 1.1 W/kg, brain 0.2 W/kg	No significant modulation in gene expression in whole brain.
Paulraj and Behari (2006)	35-day old male Wistar rats exposed 2 h/day for 35 days to 2450 MHz or 16.6 GHz RFR; SAR 1.0 and 2.01 W/kg,	Increased in DNA single strand breaks (Comet assay) in brain cells for both frequencies.

	respectively.	
Pesnya and Romanovsky (2013)	Onion (Allium cepa) exposed to GSM 900-MHz RFR from a cell phone for 1 h/day or 9 h/day for 3 days; incident power density 0.05 $\mu W/cm^2$	Increased the mitotic index, the frequency of mitotic and chromosome abnormalities, and the micronucleus frequency in an exposure-duration manner.
Phillips et al. (1998)	Human Molt-4 T-lymphoblastoid cells exposed to pulsed signals at cellular telephone frequencies of 813.5625 MHz (iDEN signal) and 836.55 MHz (TDMA signal) for 2or 21 h. SAR 0.0024 and 0.024 W/Kg for iDEN and 0.0026 and 0.026 W/kg for TDMA)	Changes in DNA single strand breaks (increase and decrease depending on exposure parameters) (Comet assay) were observed.
*Port et al. (2003)	Human leukaemia cells (HL-60) exposed to pulsed (1 Hz) 400 MHz RFR for 6 min;50 kV/m-25 times higher than the ICNIRP reference levels for occupational exposure	No significant effects on apoptosis, micronucleation, abnormal morphologies and gene expression assayed at 9, 24, 48, and 72 h post-exposure.
Qin et al. (2018)	Male mice exposed to 1800-MHz RFR 2 h/day for 32 days, SAR 0.0553 W/kg	Inhibition of testosterone synthesis might be mediated through CaMKI/RORα signaling pathway.
Qin et al. (2019)	Mouse Leydig cells exposed to a 1800-MHz RFR for 1, 2 or 4 h, SAR 0.116 W/kg	Cells showed downregulated of testosterone synthase genes (*Star, Cyp11a1*, and *Hsd-3β*) and clock genes (*Clock, Bmal1*, and *Rorα*), also reduced level of testosterone and increased oxidative stress.
*Qutob et al. (2006)	Human U87MG glioblastoma cells exposed to pulse-modulated 1900 MHz RFR for 4 h; SAR 0.1, 1.0, and10 W/kg	No significant effect on gene expression.
Racuciu (2009)	Zea mays root tips exposed to continuous-	Increased mitotic index and chromosomal aberration frequency linear with increased

	wave 900 MHz RFR for 1 – 36 h; SAR < 1 W/kg)	exposure time.
Rago et al. (2013)	Human subjects with different daily durations of cell phone use (no use, < 2 h, 2-4 h, > 4 h) and "trouser users" and "shirt users"	>4 h daily use and "trouser users" had higher sperm DNA fragmentations.
Rammal et al. (2014)	Lycospersicon esculentum (tomato) exposed to 1250 MHz RFR for 10 days at 0.0095 mW/cm^2	Increased expression of proteinase inhibitor (Pin II) and Lycospersicon esculentum basic leucine Zipper1 (lebZIP1), two wound-plants genes.
*Regalbuto et al. (2020)	Human fibroblasts exposed to 2450 MHz continuous-wave or pulsed (1 ms square oulses, 50% duty cycle) RFR; SAR 0.7W/kg	No significant effect on γ-H2AX/53BP1 foci, differential gene expression, micronucleus formation, and cell cycle.
Remondini et al. (2006)	Six human cell types exposed to 900 and 1800 MHz RFR; three exposure systems were used, exposure time 1, 24, or 44 h, SAR 1 - 2.5 W/kg (Details in Table 1 of paper.)	Some but not all human cells reacted to RFR with an increase in expression of genes encoding ribosomal proteins and therefore up-regulating the cellular metabolism.
Romano-Spica et al. (2000)	Human hemopoietic and testicular cell types exposed to 50 MHz RFR modulated (80%) with a 16-Hz frequency for 0.5-24 h; the exposure system generates a 0.2 microT magnetic field parallel to the ground and a 60 V/m electric field orthogonal to the earth's magnetic field.	Overexpression of the proto-oncogene ets1 mRNA in Jurkat T-lymphoblastoid and Leydig TM3 cell lines only in the presence of the 16-Hz modulation.
*Ros-Lior et al. (2012)	Cells collected from cheeks of human subjects	Comparing control area with the side cell phone was placed; no significant genotoxic effect was found (DNA damage and cytokinetic defects, proliferative potential, and cell death).

*Roti-Roti et al (2001)	C3H 10T(1/2) cells exposed to 835.62 MHz FDMA or 847.74 MHz CDMA for 7 days and then one-dose X-ray followed by RFR for 42 days; SAR 0.6 W/kg	No significant effect of RFR on neoplastic transformation (induced by X-ray) was observed.
Roux et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 2-10 min at 0.0066 mW/cm^2	Increased stress-related transcripts (calmodulin, protease inhibitor and chloroplast mRNA-binding protein) in leaves. (Increased at 15 min after the end of electromagnetic stimulation, dropped to close to initial levels by 30 min, and then increased again at 60 min.)
Roux et al. (2008)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Induction of stress gene expression; similar to wound responses suggesting that the radiation is perceived by plants as an injurious stimulus.
Sagripanti and Swicord (1986)	Purified DNA solution exposed to 2.55-GHz RFR for 20min; SAR_{min} and SAR_{max} ranges: 0, 2-8-5 and 21-85 W/kg,	Structural changes in DNA suggested that exposure to RFR can cause single as well as double-strand breaks in DNA in solution.
Sagripanti et al. (1987)	Purified plasmid DNA exposed to RFR in the frequency range from 2.00 to 8.75 GHz for 20 min; SAR 0, 8.5, or 85 W/kg	Induced dose- and exposure-duration-dependent DNA single and double strand beaks depends on the presence of small amounts of cuprous ions.
Sahin et al. (2016)	Rats exposed to 3-G 2100 MH RFR 6 h/day for 10 or 40 days	Oxidative DNA damage (8-hydroxy-2'deoxyguanosine) in brain increased after 10-day exposure but decreased after 40 day exposure.
Said-Salman et al. (2019)	Escherichia coli K-12 DH5α exposed to 2.4 GHz RFR for 5 h	Expression of 101 genes was differentially affects (up- and down-regulation).
*Sakuma et al. (2006)	Human glioblastoma A172 cells exposed to W-CDMA 2.1426 GHz radiation at SARs of 80, 250, and 800 mW/kg and CW radiation at 0.08 W/kg for 2 and 24 h; normal human IMR-90 fibroblasts from fetal lungs exposed to W-	No significant effect on DNA single strand breaks (Comet assay).

	CDMA and CW radiations at a SAR of 0.08 W/kg for 2 and 24 h.	
*Sakurai et al. (2011)	Human glial cell line, SVGp12, exposed to continuous-wave 2450 MHz RFR for 1, 4, and 24 h; SAR 1, 5, and 10 W/kg	No evidence of effect on gene expression.
*Salmen et al. (2018)	*S. aureus*, *S. epidermidis*, and *P. aeruginosa*. Exposed to exposed to 900 and 1800 MHz RFR for 2 h using a cell phone	No significant effects on DNA, growth rate and antibiotic susceptibility.
*Sannino et al. (2006)	Human blood leukocytes exposed to UMTS-1950 MHz signal for 24 h; SAR 0.5 or 2 W/kg	No effect on DNA single strand breaks (Comet assay) and cell viability.
*Sannino et al. (2009a)	Human dermal fibroblasts from a healthy subject and from a subject affected by Turner's syndrome exposed to GSM 900 MHz.RFR for 24 h; SAR 1 W/kg	No significant effect on DNA single strand breaks (Comet assay)
*Sannino et al. (2009b)	Human dermal fibroblasts from one subject exposed to 900 MHz RFR for 24 h; SAR 1 W/kg	No significant effect on DNA single strand breaks (Comet assay) and micronucleus frequency.
Sannino et al. (2011)	Phytohemagglutinin activated human blood lymphocytes exposed to a 900-MHz RFR for 20 h; SAR 1.25 W/kg, and then to mitomycin C	RFR attenuated micronucleus induced by mitomycin c at S-phase, and not at G(0)- and G(1)-phases of the cell cycle. (Adaptive response)
Sannino et al. (2014)	Phytohemagglutinin activated human blood lymphocytes exposed to a 900-MHz RFR for 20 h; SAR 0.3 W/kg, and then to x-ray	RFR attenuated micronucleus induced by x-ray.
Sannino et al. (2017)	Chinese hamster lung fibroblasts exposed to 1950 MHz, Universal	Increased micronucleus frequency at 0.15 and 0.3 W/kg, no effect at 0.6 and 1.25 W/kg; attenuated micronucleus induced by

	Mobile Telecommunication System signal for 20 h; SAR 0.15 – 1.25 W/kg	mitomycin-C at 1.25 W/kg.
Sarimov et al. (2004)	Human lymphocytes exposed to GSM 895-915 MHz signals for 30 min; SAR 0.0054 W/kg	Condensation of chromatin was observed. (Stronger effect at 1 h exposure.)
Sarkar et al. (1994)	Mice exposed to 2450 MHz RFR 2 h/day for 120, 150, and 200 days; SAR 1.18 W/kg	Rearrangements of DNA segments were observed in brain and testis.
Scarfi et al (1996)	Bovine lymphocytes exposed to 9 GHz RFR for 10 min, SAR 70 W/kg	Increased micronucleus frequency.
*Scarfi et al. (2003)	Human peripheral blood lymphocytes exposed to pulsed 120-130 GHz (pulse rate 2 Hz, pulsed duration 4 μs) field for 20 min; delivered energy 1.2 and 0.72 J for the two frequencies, respectively.	No effect on micronucleus frequency and cell proliferation.
*Scarfi et al (2006)	Human lymphocytes exposed to GSM 900 MHz RFR for 24 h, SAR 1, 5, and 10 W/kg).	The results provided no evidence for the existence of genotoxic (micronucleus) or cytotoxic effects
* Schuermann et al. (2020)	Human MRC-5 lung fibroblasts, human osteosarcoma cells, HTR-8/SVneo human trophoblasts, and GFP-tagged XRcc1 cells exposed to intermittent (5/10 min ON/FF) or continuous 1950 MHz, 2450 MHz (GSM or unmodulated) RFR for 1-24 h; SAR 0.5-4.9 W/kg.	No significant effect on DNA single strand breaks (Comet assay).
Schwarz et al. (2008)	Human fibroblasts and lymphocytes exposed to UMTS 1950 MHz RFR for 4-48 h; SAR 0.05 to 2.0 W/kg	Increased DNA single strand breaks (comet assay) and micronuleus were observed in fibroblasts but not in lymphocytes either unstimulated or stimulated with phytohemegglutinin.

Sekeroğlu et al. (2012)	Immature (2 week old) and mature (10 weeks old) Wistar rats exposed to continuous–wave 1800 MHz RFR for 2 h/days for 45 days; SAR 0.38-0.78 W/kg (immature rats), 0.31-0.52 W/kg (mature rats)	Bone marrow cells showed chromosome aberrations, micronucleus frequency, mitotic index and ratio of polychromatic erythrocytes (PCEs) in all exposed groups. Immature group showed more effect and less recovery at day 15 post-exposure. The cytogenotoxic damage in immature rats was statistically higher than the mature rats.
Sekeroglu et al. (2013)	Immature and mature rats exposed to 900 MHz RFR for 2 h/days for 45 days; SAR immature rats, 0.38-0.78 W/kg; mature rats 0.31-0.52 W/kg	Bone marrow cells showed chromosome aberrations, increases in micronucleus frequency, mitotic index, and ratio of polychromatic erythrocytes. Effects persisted for 15 days after exposure.
*Sekijima et al. (2010)	Human A172 (glioblastoma), H4 (neuroglioma), and IMR-90 (fibroblasts from normal fetal lung) cells exposed to continuous-wave and W-CDMA 2.1425 GHz RFR up to 96 h; SAR 0.08, 0.25, 0.8 W/kg	No significant effects on gene expression and cell proliferation.
Semin et al. (1995)	DNA in glycine and formaldehyde exposed to 10 different 4 to 8 GHz RFR 25 ms pulses, 1-6-Hz repetition rate, 0.4 to 0.7 mW/cm^2 peak power density	3 or 4 Hz pulses and 0.6 mW/cm^2 peak power increased the accumulated damage to the DNA secondary structure. However, 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 had no effect ("window effect").
*Senturk et al. (2019)	Lymphocytes from patients received radiofrequency treatment on inferior turbinate as they were diagnosed with inferior turbinate hypertrophy	No significant effect on DNA single strand breaks (Comet assay) on Day 15 post-treatment. Increase in oxidative stress was observed.
Shah et al. (2015)	Human blood samples exposed to 916-MHz RFR at two power densities and 1-8 hr using an antenna	Chromosomal damage observed in lymphocytes at higher power density and longer exposure duration.

Shahin et al. (2013)	Female mice (*Mus musculus*) exposed to continuous-wave 2.45 GHz RFR 2 h/day for 45v days; SAR 0.023 W/kg	Increased DNA strand breaks (Comet assay) observed in the brain. Changes in oxidative mechanisms and oxidative stress were observed in liver, kidney and ovary. Increased embryo implantation/resorption and abnormal pregnancy were observed.
Shahin et al. (2019)	Male Wistar rats exposed to 900 MHz RFR for 2 h/day for 8 weeks, SAR 1.075 W/kg	Increased DNA single strand breaks (Comet assay) in testis and increased oxidative stress.
Sharma ad Shukla (2020)	Male Wistar rats exposed to 900 MHz RFR for 1, 2, or 4 h/day for 90 days; SAR brain 0.231 W/kg	Increased DNA single strand breaks (Comet assay) and increased oxidative stress in brain.
Shckorbatov et al. (2009)	Human buccal epithelium cells exposed to 35 GHz RFR for 10 sec; SAR 0.75 W/kg	Caused condensation of chromatin. Left circularly polarised radiation induced less effect than linearly polarised radiation. Cell membrane damage observed.
Shckorbatov et al. (2010)	Human fibroblasts exposed to 36.65 GHz RFR at incident power densities of 1, 10, 30 and 100 microW/cm^2 for 10 sec	Chromosome condensation observed at 10 and 100 μW/cm^2 exposure. Right-handed elliptically polarized radiation was more biological activity than the left-handed polarized one.
*Shi et al (2014)	Cultured human lens epithelial cells (HLECs) exposed to 90 kHz magnetic field for 2 and 4 h; 93.36 μT	No significant effects on DNA single strand break (comet assay) and double strand breaks.
*Silva et al. (2016)	Human primary thyroid cells exposed to 895 and 900 MHz RFR for 3-65 h, SAR 0.082-0.170 W/kg	No effect on expressions of Ki-67 (involved in cell proliferation) p53 (tumor suppression) HSP-70 (stress biomarker), and reactive oxygen species.
Smith-Roe et al. (2020)	Male and female Hsd:Sprague Dawley rats and B6C3F1/N mice exposed from Gestation day 5 or Postnatal day 35, respectively, to code division multiple access (CDMA) or global system for mobile modulations over 18 hr/day, at 10-min	Significant increases in DNA single strand breaks (Comet assay) observed in the frontal cortex of male mice (both modulations), leukocytes of female mice (CDMA only), and hippocampus of male rats (CDMA only). No significant increases in micronucleated red blood cells were observed in rats or mice.

	intervals for 19 (rats) or 14 (mice) weeks; SAR 1.5, 3, or 6 W/kg (rats, 900 MHz) or 2.5, 5, or 10 W/kg (mice, 1,900 MHz).	
Sokolovic et al. (2015)	Wistar rats exposed to RFR (4 h/day, for 20, 40, and 60 days) from a Nokia 3110 cell phone:SAR 0.043-0.135 W/kg; some rats treated with melatonin (2 mg/kg, ip)	Melatonin reduced DNA fragmentation in testicular tissues also reversed oxidative changes caused by RFR (malondialdehyde, xanthine oxidase, and acid-DNase)
Soubere Mahamoud et al. (2016)	Human keratinocyte exposed to a 60.4-GHz RFR at an incident power density of 20 mW/cm^2 for 3 hours	No keratinocyte transcriptome modifications were observed. Co-treatment with a glycolysis inhibitor slightly alter the transcriptome of 6 genes encoding transcription factors or inhibitors of cytokine pathways. Thus, the RFR exposure may affect metabolically stressed cells
Souza et al. (2014)	Exfoliated cells from the oral epithelium from human subjects who spent different time using cell phones (group I, $t > 5$ h; group II, $t > 1$ h and ≤ 5 h; and group III, $t \leq 1$ h).	Structures that may be associated with gene amplification were significantly greater in the individuals in group I. No significant effects on micronucleus frequency and apoptosis and necrosis were observed.
*Speit et al. (2007)	Human fibroblasts (ES1 cells) and Chinese hamster cells (V79) exposed to intermittent (5 min ON/10 min OFF)1800-MHz for 1, 4, 24 h; RFR; SAR 2 W/kg	No significant effects on DNA single strand break (Comet assay) and micronucleus frequency.
*Speit et al. (2013)	Human HL-60 exposed to intermittent (5 min ON/10 min OFF) 1800 MHz RFR for 24 r; SAR 1.3 W.kg	No significant effects on DNA single strand break (Comet assay) and micronucleus frequency.
*Stronati et al. (2006)	Human blood samples exposed to GSM 935-MHz signal for 24h; SAR 1 and 2 W/kg	Lymphocytes showed no changes in DNA single strand breaks (Comet assay), chromosomal aberrations, sister chromatid exchanges, micronuclei frequency and cell cycle. No significant interaction with x-ray.
*Su et al (2017)	Neurogenic A172, U251, and SH-SY5Y cells	No significant DNA damage (γH2AX foci)

	exposed to an intermittently (5 min ON/10 min OFF) 1800 MHz RFR at SAR of 4.0 W/kg for 1, 6, or 24 h.	
*Su et al. (2018)	Primary cultured astrocytes, microglia and cortical neurons were exposed to intermittent (5 min ON/10 min OFF) GSM 1800 MHz RFR for 1, 6 or 24 h; SAR 4.0 W/kg.	The RFR did not elicit DNA double strand breaks (γH2AX foci) but inhibited the phagocytic ability of microglia and the axon branch length and branch number of cortical neurons.
Sun C. et al. (2016)	Mouse embryonic fibroblasts (MEFs) with proficient ($Atm^{+/+}$) or deficient ($Atm^{-/-}$) ataxia telangiectasia mutated, which is critical to initiation of DNA repair, to GSM 1800-MHz RFR for 1, 12, 24, or 36 h; SAR 4 W/kg.	Increased DNA single-strand breaks (SSBs) (Comet assay) and activated the SSB repair mechanism. This effect reduced the DNA damage to less than that of the background level after 36 hours of exposure. In the $Atm^{-/-}$ MEFs, the same RF-EMF exposure for 12 h induced both DNA single and double-strand breaks (Comet assay) and activated the two repair processes, which also reduced the DNA damage to less than the control level after prolonged exposure. (compensatory effects) (Conclusion from interpretation f different results from ($Atm^{+/+}$) and ($Atm^{-/-}$) cells.
Sun, LX et al. (2006a)	Human lens epithelial cells exposed to 217 Hz-modulated 1800 MHz RFR for 2 h; SAR 1, 2, 3, 4 W/kg	No or repairable DNA single strand breaks (Comet assay) was observed after 2 hour irradiation of 1.8 GHz microwave on LECs when SAR </= 3 W/kg. The DNA damages caused by 4 W/kg irradiation were irreversible.
Sun, LX et al. (2006b)	Human lens epithelial cells exposed to 217 Hz-modulated 1800 MHz RFR for 2 h; SAR 1, 2, 3, 4 W/kg	No DNA single strand breaks (comet assay) was induced using comet assay after 2 hours irradiation of 1. 8 GHz microwave on hLECs at the dose SAR < or = 3.0 W/kg. 4.0 W/kg irradiation caused significantly DNA damage and inhibition of hLECs proliferation.
Sun Y. et al. (2017)	HL-60 cells from human leukemia exposed to a 900-MHz RFR for 4 h/day for 5 days, Peak and average SAR 4.1x	Increased oxidative DNA damage, decreased mitochondrial transcription, and increased oxidative stress.

	10^{-4} and 2.5 x 10^{-4} W/kg	
Sykes et al. (2001)	pKZ1 mice exposed daily for 30 min to 217-Hz modulated 900 MHz RFR 1, 5, or 25 days; SAR 4 W/kg	After 25 days of exposure, RFR could lead to a perturbation in recombination frequency which may have implications for recombination repair of DNA.
*Takahashi et al. (2002)	Male Big Blue mice (BBM) exposed to 1.5 GHz RFR in the head region for 90 min/day, 5 days/week, for 4 weeks; SAR 0.67 and 2 W/kg	There was no significant variation in the frequency of independent mutations of the lacItrans gene and deletion mutation in the brain.
Tice et al. (2002)	Human blood leukocytes and lymphocytes exposed to voice modulated 837 MHz produced by an analog signal generator or by a time division multiple access (TDMA) cellular telephone, 837 MHz generated by a code division multiple access (CDMA) cellular telephone (not voice modulated), and voice modulated 1909.8 MHz generated by a global system of mobile communication (GSM)-type personal communication systems (PCS) cellular telephone for 3 or 24 h, SAR 1-10 W/kg	No significant effect on DNA single strand break (Comet assay). Exposure to each of the four RF signal technologies for 24 h at an average SAR of 5.0 or 10.0 W/kg resulted in a significant and reproducible increase in the frequency of micronucleated lymphocytes.
Tiwari et al. (2008)	Blood samples from male human subjects exposed to a CDMA cell phone for 1 h	In vitro exposure to RFR induces reversible DNA single strand breaks (Comet assay) in synergism with aphidicolin, a DNA repair inhibitor,
Tkalec et al. (2009)	Allium cepa L root meristematic cells from seeds exposed to 400 and 900 MHz RFR for 2 h, power density 10, 23, 41 and 120 V/m).	Lagging chromosomes, vagrants, disturbed anaphases and chromosome stickiness were observed.

Tkalec et al. (2013)	Earthworm (Eisenia fetida) exposed to continuous-wave and AM-modulated 900-MHz RFR for 2 - 4 h; SAR 0.00013, 0.00035, 0.0011, and 0.00933 W/kg	Increased DNA single strand breaks (Comet assay) in earthworms coelomocytes and oxidative stress (lipid and protein oxidation)
Tohidi et al. (2020)	Male BALB/c mice exposed to RFR from a cell phone jammer that emits 900- and 1800 MHz CDMA and GSM signals) for 0.5, 1, 2, or 4 h twice a day for 30 days.	Apoptotic genes Bax and Bc12 expression in the hippocampus were upregulated for 1- and 2-h exposures and down-regulated with longer exposure.
*Tomruk et al. (2010)	Nonpregnant and pregnant New Zealand White rabbits exposed to GSM 1800 MHz RFR 15 min/day for a week	No oxidative damage in liver of exposed adult and offspring, increased lipid peroxidation.
Trivino Pardo et al (2012)	T-lymphoblastoid leukemia cells exposed to 900 MHz RFR for 2 or 48 h; SAR 9.0035 W/kg	Changes in gene expressions (e.g., an early activation of genes involved in DNA double- and single-strand breaks repair).
Trosic (2001)	Rats exposed to 2450 MHz RFR for 2, 8, 13 and 22 irradiation treatments of two hours each; power density 5-15 mW/cm^2, SAR 20 W/kg	Increased multinucleated alveolar macrophages- the elevation of the number of nuclei per cell was exposure time- and dose-dependent.
Trosic and Busljeta (2005)	Wistar rats exposed to continuous-wave 2450 MHz RFR 2 h/day 7 days /week for a total of 4, 16, 30, and 60 h. power density 5-10 mW/cm^2 SAR 1-2 W/kg	The frequency of micronucleated bone marrow erythrocytes was significantly increased after 15 irradiation treatments. No effect after 2, 8, and 30 exposure treatments.
Trosic and Busljeta (2006)	Rats exposed to 2450 MHz RFR 2 h/day, 7 days/week; SAR 1.24 W/kg	Bone marrow cell micronucleus frequency increased on experimental day 15, and micronucleated polychromatic erythrocytes in peripheral blood increased on day 8.
Trosic et al. (2002)	Male Wistar rats exposed for 2 h/day, 7 days a week for up to 30 days to continuous-wave 2450	Increased micronuclei in peripheral blood polychromatic erythrocytes on the 2nd, 8^{th}, and 15^{th} day of exposure. It is likely that an adaptive mechanism, both in

	MHz RFR; power density 5-10 mW/cm^2 SAR 1-2 W/kg	erythrocytopoiesis and genotoxicity occurred.
Trosic et al. (2004)	Male Wistar rats exposed for 2 h/day, 7 days/week for 4, 16, 30, and 60 h to continuous-wave 2450 MHz RFR; power density 5-10 mW/cm^2 SAR 1.25 W/kg	The frequency of micronucleated polychromatic erythrocytes in bone marrow was significantly increased on experimental day 15, but not on 2, 8, and 30 days.
Trosic et al. (2011)	Male Wistar rats exposed to GSM 915 MHz RFR for 1 h /day 7 days/week for 2 weeks; SAR 0.6 W/kg	Increased DNA single strand breaks (Comet assay) in brain, renal, and liver cells.
Tsybulin et al. (2013)	Japanese Quail embryos exposed in ovo to GSM 900 MHz signal from a cell phone intermittently (48 sec ON/12 sec OFF) during initial 38 h of brooding or for 158 h (120 h before brooding plus initial 38 h of brooding): SAR 0.000003 W/kg	The lower duration of exposure led to a significant decrease in DNA single strand breaks (Comet assay) in cells of 38-h embryos, while the higher duration of exposure resulted in a significant increase in DNA damage.
Usikalu et al., (2013)	Sprague-Dawley rats exposed to 2450 MHz RFR for 10 min: SAR 0-4.3 W/kg	Increased DNA single strand breaks (Comet assay) found in ovary and testis.
Vafaei et al. (2020)	Pregnant mice exposed to 2400 MHz RFR from a D-link Wi-Fi router from 5 days after mating to 1 day before delivery for 2-4 h/day, head SAR at 30 cm from router 0.09 W/kg	Placenta tissue showed increased superoxide dismutase mRNA, CDKN1A, and Gadd 45a expression. (CDKN1A, and Gadd 45a are involved in DNA repair, cell cycle arrest, apoptosis, and cellular responses to environmental stressors.) Also, increased BAX mRNA and decreased Bcl-2 mRNA leads to apoptosis.
*Valbonesi et al. (2008)	Human trophoblast cell line HTR-8/SVneo exposed to pulsed 1817 MHz RFR or 1 h; SAR 2 W/kg	No significant change in either HSP70 or HSC70 protein or gene expression, or DNA single strand breaks (Comet assay).
Valbonesi et al. (2014)	Rat PC12 cells exposed to continuous-wave 1.8 GHz RFR or GSM-	After PC12 cells exposure to the GSM-217 Hz signal for 16 or 24 h, HSP70 mRNA transcription significantly increased, whereas

	217Hz and GSM-Talk signals for 4, 6, or24 h, SAR 2W/kg	no effect was observed in cells exposed to the CW or GSM-Talk signals.
*Valbonesi et al. (2016)	Rat PC12 cells exposed to 1.8 GHz 217-GSM signal for 24 h. SAR 2 W/kg	Acetylcholine esterase transcriptional or translational pathways not affected, whereas acetylcholine esterase enzymatic activity increased.
Vanishree et al. (2018)	Buccal cells from low and high cellular phone users	There was a significant increase in micronucleus counts in subject who use the phone longer. There was highly significant difference in the mean micronucleus count of participants using (code division multiple access) CDMA than (global system for mobiles) GSM cellular phones.
Varghese et al. (2018)	Female Sprague-Dawley rats exposure 2450 MHz RFR, 4/day. For 45 days; SAR 0.23W/kg	Increased caspase-3 gene expression in brain tissues; decreased antioxidant enzymes and increased lipid preoxidation. Rat showed lowering of learning and memory and expression of anxiety behavior.
Veerachari and Vasan (2012)	Human elected semen exposed to a 900-GSM cellular phone in talk mode for 1 h; power density 1-40 $\mu W/cm^2$ at 2.5 cm from antenna.	Increased DNA fragmentation index and reactive oxygen species, and decreased sperm motility and viability.
*Verschaeve et al. (2006)	Female rats exposed to RF fields for 2 h per day, 5 days per week for 2 years; SAR 0.3 or 0.9 W/kg. the mutagen and carcinogen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) was given in the drinking water. at a concentration of 19 mug/ml.	No significant genotoxic activity of MX in blood and liver cells measured by micronucleus and DNA single strand breaks (comet assay). However, MX induced DNA damage in rat brain. Co-exposures to MX and RF radiation did not significantly increase the response of blood, liver and brain cells. (no data on RFR alone.)
Vian et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Induction of mRNA encoding the stress-related bZIP transcription factor.(3.5 folds at 5-15 min post-exposure)
Vijayalaxmi et al. (1997a)	C3H/HeJ mice exposed to for 20 h/day, 7 day to continuous-wave 2450 MHz RFR MHz for 20 h/day. 7 days/week, over	Significant increases in micronucleus formation in peripheral blood and bone marrow cells were observed.

	18 months: SAR 1.0 W/kg	
Vijayalaxmi et al. (1997b)	Human peripheral blood exposed to 2450 MHz RFR either continuously for 90 min or intermittently (30 min on and 30 min off, repeated three times); SAR 12.46 W/kg	No effect on several genotoxic indexes including chromosome damage, exchange aberrations, and micronucleus frequency.
*Vijayalaxmi et al. (1999)	CF-1 male mice exposed to ultra-wideband electromagnetic radiation (UWBR) for 15 min; SAR 0.037 W/kg	No significant effects on micronucleus frequency and polychromatic erythrocytes in peripheral blood and bone marrow cells at 16 and 24 h post-exposure.
*Vijayalaxmi et al. (2000)	3 human peripheral blood samples exposed to pulsed 2450-MHz RFR for 2 h; SAR 2.135 W/kg	No significant effect on DNA single strand breaks (Comet assay) was observed in lymphocytes immediately and at 4 h post-exposure.
*Vijayalaxmi et al. (2001a)	4 human peripheral blood samples exposed to835.62 MHz (FDMA) RFR for 24 h, SAR 4.4 or 5.0 W/kg	Lymphocytes were stimulated with a mitogen, phytohemagglutinin. No significant effects at 48 and 72 h post=exposure in mitotic indices, incidence of exchange aberrations, excess fragments, binucleate cells, and micronucleus frequency.
*Vijayalaxmi et al. (2001b)	Male Sprague-Dawley rats exposed to continuous-wave 2450 MHz RFR for 24 h; SAR 12 W/kg	Peripheral blood and bone marrow smears showed no effects on frequency of micronuclei in polychromatic erythrocytes at 24 h post-exposure. .
*Vijayalaxmi et al. (2001c)	4 human peripheral blood samples exposed to continuous-wave 847.74 MHz (CDMA) RFR for 24 h; SAR 4.9 or 5.5 W/kg	No significant effects on mitotic indices, frequencies of exchange aberrations, excess fragments, binucleate cells, and micronuclei in lymphocytes at 48 and 72 h post-exposure.
*Vijayalaxmi et al. (2003)	Timed-pregnant Fischer 344 rats (from nineteenth day of gestation) and their nursing offspring (until weaning) exposed to a far-field 1.6 GHz Iridium wireless communication signal for 2 h/day, 7 days/week	No significant effects on micronuclei in polychromatic erythrocytes in bone marrow.

	for 2 years; SAR 0.036 to 0.077 W/kg	
*Vijayalaxmi et al. (2004)	Mice exposed to 42.2 GHz RFR applied to the nasal region 30 min/day for 3 days; peak SAR 622 W/kg	No effect on micronucleus frequency in polychromatic erythrocytes of peripheral blood and bone marrow cells collected 24 h after exposure.
*Vijayalaxmi et al. (2006)	Human peripheral blood samples exposed to 2.45 GHz or 8.2 GHz pulsed-wave RFR for 2 h; SAR 2.13 W/kg (245 MHz) or 20.71 W/kg (8.2 GHz),	No significant effects on chromosomal aberrations and micronuclei in lymphocytes.
Vilic et al. (2017)	Honey bee (Apis mellifera) larvae exposed to 900 MHz at field levels of 10, 23, 41 and 120 V m^{-1} for 2 h. At a field level of 23 V m^{-1} the effect of 80% AM 1 kHz sinusoidal and 217 Hz modulation was investigated as well.	DNA single strand break (Comet assay) increased significantly in honey bee larvae exposed to modulated (80% AM 1 kHz sinus) field at 23 V m^{-1}. Oxidative changes also observed. Modulated RF-EMF produced more negative effects than the corresponding unmodulated field.
*Waldmann et al. (2013)	Human peripheral blood samples exposed to GSM 1800 MHz RFR for 28 h; SAR 0.2, 2, and 10 W/kg	No significant effects on lymphocytes on chromosome aberration, micronucleus frequency, sister chromatid exchange and DNA single strand break (comet assay).
Wang et al. (2015)	Neuro-2a (mouse neuroblastoma) cells exposed to GSM 900 MHz RFR for 24 h; SAR 0.5, 1 or 2 W/kg	Increased DNA oxidative damage (comet assay) and reactive oxygen species. OGG1(a base excision DNA repair enzyme) may be involved.
Wu et al. (2008)	Human lens epithelial cells exposed to 1800 MHz mobile phone radiation for 24 h; SAR 4 W/kg	Increased DNA single strand breaks (Comet assay) and reactive oxygen species.
Xu et al. (2010)	Sprague-Dawley rat primary cultured cortical neurons exposed to intermittent (5 min ON/10 min OFF) 217-Hz pulsed 1800 MHz RFR for 24 h; SAR 2 W/kg	Increased in the levels of 8-hydroxyguanine, a common biomarker of DNA oxidative damage, in the mitochondria of neurons, levels of mitochondrial RNA (mtRNA) transcripts showed a reduction.
Xu et al. (2013)	Six different types of cells intermittently (5	RFR induced DNA damage (γH2AX foci and alkaline and neutral comet assay) in a

	min ON/10 min OFF) exposed to pulsed GSM 1800 MHz RFR for 1 or 24 h: SAR 3.0 W/kg	cell type-dependent manner.
Yadav and Shama (2008)	Buccal-mucosa cells from 85 regular cell phone users (exposed) and 24 non-users (controls)	A positive correlation between 0-1, 1-2, 2-3 and 3-4 years of exposure and the frequency of micronucleated cells and total micronuclei.
Yakymenko et al. (2018)	Quail embryos exposed to GSM 1800 GHz signal from a smart phone (48 s ON/12 s OFF) for5 days before and 14 days during incubation , power density 0.00032 mW/cm^2	Increased DNA single sand breaks (comet assay), oxidative DNA damage, reactive oxygen species, and mortality.
Yan et al. (2008)	Adult Sprague-Dawley rats exposed to a cell phones 1.9 GHz (PCE CDMA) for 6 h per day for 126 days (18 weeks).	Significant mRNA up-regulation of injury-related proteins in the brain of rats exposed to cell phone radiation
Yao et al. (2004)	Rabbit lens epithelial cells exposed to continuous-wave 2450-MHz RFR for 8 h, power densities 0.10, 0.25, 0.50, 1.00, and 2.00 mW/cm^2	The RFR higher than 0.50 mW/cm^2 can inhibit lens epithelial cell proliferation, and increase the expression of P27Kip1.
Yao et al. (2008)	Human lens epithelial cells intermittently (5 min ON/10 min OFF) exposed to GSM 1.8 GHz RFR for 2 h; SAR 1, 2, 3, and 4 W/kg	Increased DNA single strand breaks (Comet assay), no change in double strand breaks (γH2AX foci), and increased reactive oxygen species.
Ye et al. (2016)	Chicken embryos exposed to GSM 900 MHz RFR from cell phones 3 h/day from day 2 to day 21 of incubation	Increased DNA single strand breaks (Comet assay) from blood cells and mortality.
*Yildirim et al. (2010)	People who lived around cell phone base stations and healthy controls	There was no significant difference in micronucleus frequency and chromosomal aberrations in blood lymphocytes between the two study groups
Zalata et al. (2015)	Human semen samples exposed to 850-MHz	Significant increase in sperm DNA fragmentation percent, clusterin gene

	RFR from a cell phone for 1 h; SAR 1.46 W/kg at 10 cm	expression and clusterin protein (associated with clearance of cellular debris and apoptosis) levels in the exposed semen samples.
*Zeni et al. (2003)	Human peripheral blood exposed to continuous wave 925 MHz RFR or GSM 925 MHz (6 min ON/ 3 h OFF for 44h (SAR 1.6 W/kg); or GSM signal 1 h/day for 3 days (SAR 0.2 W/kg).	No statistically significant differences were detected in micronucleus frequency in lymphocytes.
*Zeni et al. (2005)	Human peripheral blood lymphocytes exposed to GSM 900 MHz signal for 2 h; SAR 0.3 and 1 W/kg	No significant effects on DNA single strand breaks (Comet assay), chromosome aberration, or sister chromatid exchange.
*Zeni et al. (2007)	Human whole blood samples exposed to 120 GHz (SAR 0.4 W/kg) and 130 GHz (SAR 0.24, 1.4, or 2 W/kg) RFR for 20 min.	No effects in leukocytes on micronucleus frequency and DNA single strand breaks (comet assay).
*Zeni et al. (2008)	Human peripheral blood exposed intermittently (6 min ON/2 h OFF) to 1945 MHz RFR for 24 – 68 h; SAR 2.2 W/kg	No significant effects on DNA single strand breaks (Comet assay) and micronucleus frequency in leukocytes.
Zeni et al. (2012a)	Human peripheral blood lymphocytes exposed to 1950-MHz RFR UMTS (universal mobile telecommunication system) signal for 20 h; SAR 1.25, 0.6, 0.3, or 0.15 W/kg. and then tomitomycin C	Cells pre-exposed to RFR at 0.3W/kg (less consistent at the other SARs) and then treated with MMC showed a significant reduction in the frequency of micronucleus, compared with the cells treated with MMC alone
*Zeni et al. (2012b)	Rat neuron-like pheochromocytoma (PC12) cells exposed to 1950-MHz 3G Universal Mobile Telecommunications System (UMTS) signal for 24 h; SAR 10 W/kg	No effect on DNA single strand break (Comet assay), cell viability, and apoptosis.

Zhang et al. (2006)	Chinese hamster lung cells exposed intermittently (5 min ON/10 min OFF) to GSM 1800 MHz RFR for 1 or 24 h; SAR 3 W/kg	Cells exposed for 24 h showed increased DNA double strand breaks (γH2AX foci).
Zhang et al. (2002)	Human whole blood exposed to 2450 MHz RFR for 2 h; Power density 5 mW/cm^2	2450-MHz RFR cannot induce DNA and chromosome damage, but can increase DNA single strand breaks (Comet assay) induced by mitomycin C .
Zhang et al. (2008)	Primary culture of rat neurons exposed to a 1.8 GHz RFR for 24 h; SAR 2 W/kg.	Changes (up- and down-regulation) of many genes transcription (involving cytoskeleton, signal transduction pathway, metabolism, etc.) were observed.
Zhao J. et al. (2020)	Escherichia coli exposed to 3.1 THz RFR for 8 h at 33 mW/cm2 and 10 Hz repetition frequency	Plasmid copy number, protein expression and fluorescence intensity of bacteria from the irradiated area were 3.8-, 2.7-, and 3.3 times higher than in bacteria from the un-irradiated area, respectively.
Zhao R. et al. (2007)	Rat neurons exposed to pulsed 217-Hz modulated 1800 MHz RFR for 24 h; SAR 2 W/kg	up- and down-regulation of genes transcriptions were observed.
Zhao TY. et al. (2007)	Primary cultured neurons and astrocytes exposed to a GSM 1900 MHz cell phone for 2 h;	Up-regulation of caspase-2, caspase-6 and Asc (apoptosis associated speck-like protein containing a card) gene expression in neurons and astrocytes. Additionally, astrocytes showed up-regulation of the Bax gene. Neurons appeared to be more sensitive to this effect than astrocytes.
*Zhijian et al. (2009)	Leukocytes from four young healthy donors exposed intermittent (5 min ON/10 min OFF) to 1800 MHz RFR for 24 h; SAR 2 W/kg; Cell also exposed x-ray	No significant effect on DNA single strand breaks (Comet assay) and no synergistic effect with x-ray.
*Zhijian et al. (2010)	Human B-cell lymphoblastoid cells exposed to 1800 GHz RFR for 2 h; SAR 2 W/kg	RFR did not directly induce DNA single strand breaks (Comet assay)
*Ziemann et al. (2009)	Peripheral blood erythrocytes of B6C3F1	No significant effect on micronucleus frequency.

	mice exposed to GSM 900 or DCS 1747 MHz RFR 2 h/day, 5 days /week for 2 years; SAR 0.4, 1.3 and 4 W/kg	
Zong et al. (2015)	Mice exposed to 900 MHz RFR 4 h/day for 7 days; SAR 0.05 W/kg	RFR alone had no effect on DNA single strand breaks (Comet assay) and oxidative damage in blood leukocytes. It attenuated bleomycin-induced DNA breaks and repair, and oxidative damage.
Zothansiama et al. (2017)	Blood samples from people lived closed to cell phone base station	The exposed group, residing within a perimeter of 80 m of mobile base stations, showed significantly higher frequency of micronuclei in lymphocytes when compared to the control group, residing 300 m away from the mobile base stations.
Zotti-Martelli et al. (2000)	Human peripheral blood lymphocytes exposed to 2.45 and 7.7 GHz RFR for 15, 30, or 60 min; power density 10, 20, or 30 mW/cm^2	Increased micronucleus frequency at a power density of 30mW/cm^2 and after an exposure of 30 and 60 min.
Zotti-Martelli et al. (2005)	Human whole blood samples exposed to continuous-wave 1800 MHz RFR for 60, 120 and 180 min; power density 5, 10, or 20 mW/cm^2	A statistically significant increase of micronucleus was observed in lymphocytes dependent on exposure time and applied power density.
*Zuo et al. (2015)	Sprague-Dawley rat spiral ganglion neurons exposed intermittently (5 min ON/10 min OFF) to GSM 1800 MHz RFR for 24 h; SAR 2 and 4 W/kg	The RFR could not directly induce DNA single strand breaks (Comet assay) in normal spiral ganglion neurons, but it could cause the changes of cellular ultrastructure at SAR 4.0 W/kg when cells are in fragile or micro-damaged condition.

References

Agarwal, A., Desai, N.R., Makker, K., et al. (2009). Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study. Fertil. Steril. 92: 1318-1325.

Aitken, R.J., Bennetts, L.E., Sawyer, D., et al. (2005). Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline. Inter. J. Androl. 28:171-179.

Akdag, M.Z., Dasdag, S., Canturk, F., et al. (2016). Does prolonged radiofrequency radiation emitted from Wi-Fi devices induce DNA damage in various tissues of rats? J. Chem. Neuroanat. 75(Pt B):116-122.

Akdag, M., Dasdag, S., Canturk, F., et al (2018). Exposure to non-ionizing electromagnetic fields emitted from mobile phones induced DNA damage in human ear canal hair follicle cells. Electromagn. Biol. Med. 37:66-75.

Akhavan-Sigari, R., Baf, M.M., Ariabod, V., et al. (2014). Connection between cell phone use, p53 gene expression in different zones of glioblastoma multiforme and survival prognoses. Rare Tumors. 6:5350.

Alkis, M.E., Bilgin, H.M., Akpolat, V., et al. (2019a). Effect of 900-1800-, and 2100-MHz radiofrequency radiation on DNA and oxidative stress in brain. Electromagn. Biol. Med. 38:32-47.

Alkis, M.S., Akdag, M.Z., Dasdag, S., et al. (2019b). Single-strand DNA breaks and oxidative changes in rat testes exposed to radiofrequency radiation emitted from cellular phones. Biotech. Biotechnological Equipment 33: 1733-1740.

Alkis, M.E., Akdag, M.Z., Dasdag, S. (2021) Effects of low-intensity microwave radiation on oxidant-antioxidant parameters and DNA damage in the liver of rats. Bioelectromagnetics 42:76-85.

Al-Serori, H., Kundi, M., Ferk, F., Mišík M, et al. (2017). Evaluation of the potential of mobile phone specific electromagnetic fields (UMTS) to produce micronuclei in human glioblastoma cell lines. Toxicol. In Vitro. 40:264-271.

Al-Serori, H., Ferk, F., Kundi, M., et al. (2018). Mobile phone specific electromagnetic fields induce transient DNA damage and nucleotide excision repair in serum-deprived human glioblastoma cells. PLoS One. 13:e0193677.

Antonopoulos, A., Eisenbrandt, H., Obe, G. (1997). Effects of high-frequency electromagnetic fields on human lymphocytes in vitro. Mutat. Res. 395: 209-214.

Atasoy, H.I., Gunal, M.Y., Atasoy, P. (2013). Immunohistopathologic demonstration of deleterious effects on growing rat testes of radiofrequency waves emitted from conventional Wi-Fi devices. J. Pediatr. Urol. 9:223-229.

Atlı Şekeroğlu, Z., Akar, A., Sekeroğlu, V. (2013). Evaluation of the cytogenotoxic damage in immature and mature rats exposed to 900 MHz radio frequency electromagnetic fields. Int. J. Radiat. Biol. 89:985-992.

Avendaño, C., Mata, A., Sanchez Sarmiento, C.A., et al. (2012) Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation. Fertil. Steril. 97:39-45.

Balode, Z. (1996). Assessment of radio-frequency electromagnetic radiation by the micronucleus test in bovine peripheral erythrocytes. Sci. Total Environ. 180:81-85.

Banerjee, S., Singh, N.N., Sreedhar, G., et al. (2016). Analysis of the genotoxic effects of mobile phone radiation using buccal micronucleus assay: a comparative evaluation. J. Clin. Diagn. Res. 10:ZC82-85.

Baohong, W., Jiliang, H., Lifen, J., et al.(2005). Studying the synergistic damage effects induced by 1.8 GHz radiofrequency field radiation (RFR) with four chemical mutagens on human lymphocyte DNA using comet assay in vitro. Mutat. Res. 578:149-157.

Baohong, W., Lifen, J., Lanjuan, L., et al. (2007). Evaluating the combinative effects on human lymphocyte DNA damage induced by ultraviolet ray C plus 1.8 GHz microwaves using comet assay in vitro. Toxicol. 232:311-316.

Beaubois, E., E., Girard, S., Lallechere. S., et al. (2007). Intercellular communication in plants: evidence for two rapidly transmitted systemic signals generated in response to electromagnetic field stimulation in tomato. Plant Cell. Environ. 30:834-844.

Bektas, H., Dasdag, S., Bektas, M.S. (2020). Comparison of effects of 2.4 GHz Wi-Fi and mobile phone exposure on human placenta and cord blood. Biotechn. Biotechnological Equipment, 34: 154-162, 2020.

Belyaev, I.Ya., Alipov, Y.D., Shcheglov, V.S., et al. (1992). Resonance effect of microwaves on the genome conformational state of E. coli cells. Z. Naturforsch. [C] 47:621-627.

Belyaev, I.Y., Hillert, L., Protopopova, M., et al. (2005). 915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons. Bioelectromagnetics 26:173-184.

Belyaev, I.Y., Koch, C.B., Terenius, O., et al. (2006). 915 MHz GSM microwaves induces changes in gene expression but not double stranded DNA breaks or effects on chromatin conformation. Bioelectromagnetics 27:295-306.

Belyaev, I.Y., Markovà, E., Hillert, L., et al. (2009). Microwaves from UMTS/GSM mobile phones induce long-lasting inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes. Bioelectromagnetics 30:129-141.

Bisht, K.S., Moros, E.G., Straube, W.L., et al. (2002). The Effect of 835.62 MHz FDMA or 847.74 MHz CDMA modulated radiofrequency radiation on the induction of micronuclei in C3H 10T½ cells. Radiat. Res. 157: 506–515.

Bourdineaud, J.P., Šrut, M., Štambuk, A., et al. (2017). Electromagnetic fields at a mobile phone frequency (900 MHz) trigger the onset of general stress response along with DNA modifications in Eisenia fetida earthworms. Arh. Hig. Rada. Toksikol. 68:142-152.

Bourthoumieu, S., Joubert, V., Marin, B., et al. (2010). Cytogenetic studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using R-banded karyotyping. Radiat. Res. 174:712-718.

Bourthoumieu, S., Terro, F., Leveque, P., et al. (2011). Aneuploidy studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using FISH. Int. J. Radiat. Biol. 87:400-408.

Bourthoumieu, S., Magnaudeix, A., Terro, F., et al. (2013). Study of p53 expression and post-transcriptional modifications after GSM-900 radiofrequency exposure of human amniotic cells. Bioelectromagnetics. 3:52-60.

Burlaka, A., Tsybulin, O., Sidorik, E., et al. (2013). Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation. Exp. Oncol. 35:219-225.

Busljeta, I., Trosic, I., Milkovic-Kraus, S. (2004). Erythropoietic changes in rats after 2.45 GJz nonthermal irradiation. Int. J. Hyg. Environ. Health. 207:549-554.

Buttiglione, M., Roca, L., Montemurno, E., et al. (2007). Radiofrequency radiation (900 MHz) induces Egr-1 gene expression and affects cell-cycle control in human neuroblastoma cells. J. Cell Physiol. 213:759-767.

Cam, S.T., Seyhan, N. (2012). Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation. Int. J. Radiat. Biol. 88:420-424.

Campisi, A., Gulino, M., Acquaviva, R., et al. (2010). Reactive oxygen species levels and DNA fragmentation on astrocytes in primary culture after acute exposure to low intensity microwave electromagnetic field. Neurosci. Lett. 473:52-55.

Cervellati, F., Valacchi, G., Lunghi, L., et al. (2013). 17-β-estradiol counteracts the effects of high frequency electromagnetic fields on trophoblastic connexins and integrins. Oxid. Med. Cell Longev. 2013:280850.

Chandel, S., Kaur, S., Issa, M., et al. (2019a) Exposure to mobile phone radiations at 2350 MHz incites cyto- and genotoxic effects in root meristems of *Allium cepa*. J. Environ. Health Sci. Eng. 17:97-104.

Chandel, S., Kaur, S., Issa, M., et al. (2019b) Appraisal of immediate and late effects of mobile phone radiations at 2100 MHz on mitotic activity and DNA integrity in root meristems of Allium cepa. Protoplasma. 256:1399-1407.

Chang, S.K., Choi, J.S., Gil, H.W., et al. (2005). Genotoxicity evaluation of electromagnetic fields generated by 835-MHz mobile phone frequency band. Eur. J. Cancer Prev. 14:175-179.

Chaturvedi, C.M., Singh, V.P., Singh, P., et al. (2011). 2.45 GHz (CW) microwave irradiation alters circadian organization, spatial memory, DNA structure in the brain cells and blood cell counts of male mice, Mus musculus. Prog. Electromagn. Res. B, 29, 23-42.

Chauhan, V., Mariampillai, A., Bellier, P.V., et al. (2006a). Gene expression analysis of a human lymphoblastoma cell line exposed in vitro to an intermittent 1.9 GHz pulse-modulated radiofrequency field. Radiat. Res. 165:424-429.

Chauhan, V., Mariampillai, A., Gajda, G.B., et al. (2006b). Analysis of proto-oncogene and heat-shock protein gene expression in human derived cell-lines exposed in vitro to an intermittent 1.9 GHz pulse-modulated radiofrequency field. Int. J. Radiat. Biol. 82:347-354.

Chauhan, V., Qutob, S.S., Lui, S., et al. (2007) Analysis of gene expression in two human-derived cell lines exposed in vitro to a 1.9 GHz pulse-modulated radiofrequency field. Proteomics. 7:3896-3905.

Chavdoula, E.D., Panagopoulos, D.J., Margaritis, L.H. (2010). Comparison of biological effects between continuous and intermittent exposure to GSM-900-MHz mobile phone radiation: detection of apoptotic cell-death features. Mutat. Res. 700:51-61.

Chemeris, N.K., Gapeyev, A.B., Sirota, N.P., et al. (2004). DNA damage in frog erythrocytes after in vitro exposure to a high peak-power pulsed electromagnetic field. Mutat. Res. 558: 27-34.

Chemeris, N.K., Gapeyev, A.B., Sirota, N.P., et al. (2006). Lack of direct DNA damage in human blood leukocytes and lymphocytes after in vitro exposure to high power microwave pulses. Bioelectromagnetics 27:197-203.

Chen, G., Lu, D., Chiang, H., et al. (2012). Using model organism Saccharomyces cerevisiae to evaluate the effects of ELF-MF and RF-EMF exposure on global gene expression. Bioelectromagnetics. 33:550-560.

Choi, J., Min, K., Jeon, S., et al. (2020). Continuous exposure to 1.7 GHz LTE electromagnetic fields increases intracellular reactive oxygen species to decrease human cell proliferation and induce senescence. Sci. Rep. 10(1):9238.

Ciaravino, V., Meltz, M.L., Erwin, D.N. (1991). Absence of a synergistic effect between moderate power radio-frequency electromagnetic radiation and adriamycin on cell-cycle progression and sister-chromatid exchange. Bioelectromagnetics 12:289-298.

D'ambrosio, G., Lioi, M.B., Massa, R., et al. (1995). Genotoxic Effects of amplitude-modulated microwaves on human lymphocytes exposed in vitro under controlled conditions. Electro-Magnetiobiol. 14:157-164.

d'Ambrosio, G., Massa, R., Scarfi, M.R., et al. (2002). Cytogenetic damage in human lymphocytes following GMSK phase modulated microwave exposure. Bioelectromagnetics 23:7-13.

Danese, E., Lippi, G., Buonocore, R., et al. (2017). Mobile phone radiofrequency exposure has no effect on DNA double strand breaks (DSB) in human lymphocytes. Ann. Transl. Med. 5:272.

De Amicis, A., Sanctis, S.D., Cristofaro, S.D., et al. (2015). Biological effects of in vitro THz radiation exposure in human foetal fibroblasts. Mutat. Res. Genet. Toxicol. Environ. Mutagen. 793:150-160.

De Iuliis, G.N., Newey, R.J., King, B.V., et al. (2009) Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro. PLoS One 4:e6446, 2009.

de Oliveira, F.M., Carmona, A.M, Ladeira, C. (2017). Is mobile phone radiation genotoxic? An analysis of micronucleus frequency in exfoliated buccal cells. Mutat. Res. 822:41-46.

Del Re, B., Bersani, F., Giorgi, G. (2019). Effect of electromagnetic field exposure on the transcription of repetitive DNA elements in human cells. Electromagn. Biol. Med. 38:262-270,

Del Vecchio, G., Giuliani, A., Fernandez, M., et al. (2009). Continuous exposure to 900MHz GSM-modulated EMF alters morphological maturation of neural cells. Neurosci. Lett. 455:173-177.

Demsia, G., Vlastos, D., Matthopoulos, D.P. (2004). Effect of 910-MHz electromagnetic field on rat bone marrow. ScientificWorld Journal 4 Suppl 2:48-54.

Deshmukh, P.S., Megha, K., Banerjee, B.D., et al. (2013). Detection of low level microwave radiation induced deoxyribonucleic acid damage vis-à-vis genotoxicity in brain of Fischer rats. Toxicol. Int. 20:19-24.

Deshmukh, P.S., Nasare, N., Megha, K., et al. (2015). Cognitive impairment and neurogenotoxic effects in rats exposed to low-intensity microwave radiation. Int. J. Toxicol. 34:284-290.

Deshmukh, P.S., Megha, K., Nasare, N., et al. (2016). Effect of low level subchronic microwave radiation on rat brain. Biomed. Environ. Sci. 29:858-867.

Diem, E., Schwarz, C., Adlkofer, F., et al. (2005). Non-thermal DNA breakage by mobile-phone radiation (1800MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro. Mutat. Res. 583:178-183.

Duan, W., Liu, C., Zhang, L., et al. (2015). Comparison of the genotoxic effects induced by 50 Hz extremely low-frequency electromagnetic fields and 1800 MHz radiofrequency electromagnetic fields in GC-2 cells. Radiat. Res. 183:305-314.

Durdik, M., Kosik, P., Markova, E., et al. (2019). Microwaves from mobile phone induce reactive oxygen species but not DNA damage, preleukemic fusion genes and apoptosis in hematopoietic stem/progenitor cells. Sci. Rep. 9:16182.

Eker, E.D., Arslan, B., Yildirim, M., et al. (2018). The effect of exposure to 1800 MHz radiofrequency radiation on epidermal growth factor, caspase-3, Hsp27 and p38MAPK gene expressions in the rat eye. Bratisl. Lek. Listy. 119:588-592.

Engelmann, J.C., Deeken, R., Müller, T., et al. (2008). Is gene activity in plant cells affected by UMTS-irradiation? A whole genome approach. Adv. Appl. Bioinform Chem. 1:71-83.

Esmekaya, M.A., Aytekin, E., Ozgur, E., et al. (2011). Mutagenic and morphologic impacts of 1.8GHz radiofrequency radiation on human peripheral blood lymphocytes (hPBLs) and possible protective role of pre-treatment with Ginkgo biloba (EGb 761). Sci. Total Environ. 410-411:59-64.

Falzone, N., Huyser, C., Franken, D.R., et al. (2010). Mobile phone radiation does not induce pro-apoptosis effects in human spermatozoa. Radiat. Res. 174:169-176.

Ferreira, A.R., Knakievicz, T., de Bittencourt Pasquali, M.A., et al. (2006). Ultra high frequency-electromagnetic field irradiation during pregnancy leads to an increase in erythrocytes micronuclei incidence in rat offspring. Life Sci 80: 43-50.

Figueiredo, A.B.S., Alves, R.N., Ramalho, A.T. (2004). Cytogenetic analysis of the effects of 2.5 and 10.5 GHz microwaves on human lymphocytes. Genet. Mol. Biol. 27: 460-466.

Finnie, J.W., Cai, Z., Blumbergs, P.C., et al. (2006). Expression of the immediate early gene, c-fos, in fetal brain after whole of gestation exposure of pregnant mice to global system for mobile communication microwaves. Pathology. 38:333-335.

Fragopoulou, A.F., Polyzos, A., Papadopoulou, M.D., et al. (2018). Hippocampal lipidome and transcriptome profile alterations triggered by acute exposure of mice to GSM 1800 MHz mobile phone radiation: An exploratory study. Brain Behav. 8(6):e01001.

Franchini, V., Regalbuto, E., De Amicis, A., et al. (2018a). Genotoxic effects in human fibroblasts exposed to microwave radiation. Health Phys. 115:126-139.

Franchini, V., De Sanctis, S., Marinaccio, J., et al (2018b). Study of the effects of 0.15 terahertz radiation on genome integrity of adult fibroblasts. Environ. Mol. Mutagen. 59:476-487.

Franzellitti, S., Valbonesi, P., Contin, A., et al. (2008). HSP70 expression in human trophoblast cells exposed to different 1.8 GHz mobile phone signals. Radiat. Res. 170:488-497.

Franzellitti, S., Valbonesi, P., Ciancaglini, N., et al. (2010). Transient DNA damage induced by high-frequency electromagnetic fields (GSM 1.8 GHz) in the human trophoblast HTR-8/SVneo cell line evaluated with the alkaline comet assay. Mutat. Res. 683:35-42.

Fritze, K., Wiessner, C., Kuster, N., et al. (1997). Effect of global system for mobile communication microwave exposure on the genomic response of the rat brain. Neurosci. 81:627-639.

Fucic, A., Garaj-Vrhovac, V., Skara, M., et al. (1992). X-rays, microwaves and vinyl

chloride monomer: their clastogenic and aneugenic activity, using the micronucleus assay on human lymphocytes. Mutat. Res. 282:265-271.

Furtado-Filho, O.V., Borba, J.B., Dallegrave, A., et al. (2014). Effect of 950 MHz UHF electromagnetic radiation on biomarkers of oxidative damage, metabolism of UFA and antioxidants in the livers of young rats of different ages. Int. J. Radiat. Biol. 90:159-168.

Furtado-Filho, O.V., Borba, J.B., Maraschin, T., et al. (2015). Effects of chronic exposure to 950 MHz ultra-high-frequency electromagnetic radiation on reactive oxygen species metabolism in the right and left cerebral cortex of young rats of different ages. Int. J. Radiat. Biol. 91:891-897.

Gadhia, P.K., Shah, T., Mistry, A. (2003). A preliminary study to assess possible chromosomal damage among users of digital mobile phones. Electromag. Biol. Med. 22:149-159.

Gajski, G., Garaj-Vrhovac, V. (2009). Radioprotective effects of honeybee venom (Apismellifera) against 915-MHz microwave radiation-induced DNA damage in wistar rat lymphocytes: in vitro study. Int. J. Toxicol. 28:88-98.

Gandhi, G., Anita. (2005). Genetic damage in mobile phone users: some preliminary findings. Ind. J. Hum. Genet. 11:99-104.

Gandhi, G., Singh, P. (2005). Cytogenetic damage in mobile phone users: preliminary data. Int. J. Hum. Genet. 5:259-265.

Gandhi, G., Kaur, G., Nisar, U. (2015). A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station. Electromagn. Biol. Med. 34:344-354.

Gapeyev, A.B., Lukyanova, N.A., Gudkov, S.V. (2014). Hydrogen peroxide induced by modulated electromagnetic radiation protects the cells from DNA damage. Cent. Europ. J. Biol. 9, 915–921.

Garaj-Vrhovac, V., Horvat, D., Koren, Z. (1990). The effect of microwave radiation on the cell genome. Mutat. Res. 243:87-93.

Garaj-Vrhovac, V., Horvat, D., Koren, Z. (1991). The relationship between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation. Mutat. Res. 263:143-149.

Garaj-Vrhovac, V., Fucic, A., Horvat, D. (1992). The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation in vitro. Mutat. Res. 281:181-186.

Garaj-Vrhovac, V., Fucic, A. (1993). The rate of elimination of chromosomal aberrations after accidental exposure to microwave radiation. Bioelectrochem. Bioenerg. 30: 319-325.

Garaj-Vrhovac, V. (1999). Micronucleus assay and lymphocyte mitotic activity in risk

assessment of occupational exposure to microwave radiation. Chemosphere 39:2301-2312.

Garaj-Vrhovac, V., Orescanin, V. (2009). Assessment of DNA sensitivity in peripheral blood leukocytes after occupational exposure to microwave radiation: the alkaline comet assay and chromatid breakage assay. Cell, Biol, Toxicol. 25:33-43.

Garaj-Vrhovac, V., Gajski, G., Trosić, I., et al. (2009). Evaluation of basal DNA damage and oxidative stress in Wistar rat leukocytes after exposure to microwave radiation. Toxicology. 259:107-112.

Garaj-Vrhovac, V., Gajski, G., Pažanin, S., et al. (2011). Assessment of cytogenetic damage and oxidative stress in personnel occupationally exposed to the pulsed microwave radiation of marine radar equipment. Int. J. Hyg. Environ. Health. 4:59-65.

Garson, O.M., McRobert, T.L., Campbell, L.J., et al. (1991). A chromosomal study of workers with long-term exposure to radio-frequency radiation. Med. J. Aust. 155:289-292.

Ghatei, N., Nabavi, A.S., Toosi, M.H.B., et al. (2017). Evaluation of *bax*, *bcl-2*, *p21* and *p53* genes expression variations on cerebellum of BALB/c mice before and after birth under mobile phone radiation exposure. Iran J. Basic Med. Sci. 201037-1043.

Glaser, K., Rohland, M., Keine-Ostmann, T., et al. (2016). Effect of radiofrequency radiation on human hematopoietic stem cells. Radiat. Res. 186: 455-465.

Gökçek-Saraç, C., Akçay, G., Karakurt, S., et al. (2020) Possible effects of different doses of 2.1 GHz electromagnetic radiation on learning, and hippocampal levels of cholinergic biomarkers in Wistar rats. Electromagn. Biol. Med. doi: 10.1080/15368378.2020.1851251. Online ahead of print.

Görlitz, B-D., Müller, M., Ebert, S., et al. (2005). Effects of 1-week and 6-week exposure to GSM/DCS radiofrequency radiation on micronucleus formation in B6C3F1 mice. Radiat. Res. 164(4 Pt 1):431-439.

Gorpinchenko, I., Nikitin, O., Banyra, O., et al (2014). The influence of direct mobile phone radiation on sperm quality. Cent. Europ. J. Urol. 6765-71.

Gulati, S., Yadav, A., Kumar, N., et al. (2016). Effect of GSTM1 and GSTT1 polymorphisms on genetic damage in humans populations exposed to radiation from mobile towers. Arch. Environ. Contam. Toxicol.70615-625.

Gulati, S., Yadav, A., Kumar, N., et al. (2018). Phenotypic and genotypic characterization of antioxidant enzyme system in human population exposed to radiation from mobile towers. Mol. Cell. Biochem. 440:1-9.

Gulati, S., Kosik, P., Durdik, M., et al. (2020). Effects of different mobile phone UMTS signals on DNA, apoptosis and oxidative stress in human lymphocytes. Environ Pollut. 267:115632.

Guler, G., Tomruk, A., Ozgur, E., et al. (2010). The effect of radiofrequency radiation on DNA and lipid damage in non-pregnant and pregnant rabbits and their newborns. Gen. Physiol. Biophys. 29:59-66.

Güler, G., Tomruk, A., Ozgur, E., et al. (2012). The effect of radiofrequency radiation on DNA and lipid damage in female and male infant rabbits. Int. J. Radiat. Biol. 88:367-373.

Gurbuz, N., Sirav, B., Yuvaci, H.U., et al. (2010). Is there any possible genotoxic effect in exfoliated bladder cells of rat under the exposure of 1800 MHz GSM-like modulated radio frequency radiation (RFR)? Electromagn. Biol. Med. 29:98-104.

Gurbuz, N., Sirav, B., Colbay, M., et al. (2014). No genotoxic effect in exfoliated bladder cells of rat under the exposure of 1800 and 2100-MHz radio frequency radiation. Electromagn. Biol. Med. 33:296-301.

Gurisik, E., Warton, K., Martin, D.K., et al. (2006). An in vitro study of the effects of exposure to a GSM signal in two human cell lines: monocytic U937 and neuroblastoma SK-N-SH. Cell Biol. Int. 30:793-759.

Gürler, H.S., Bilgici, B., Akar, A.K., et al. (2014). Increased DNA oxidation (8-OHdG) and protein oxidation (AOPP) by low level electromagnetic field (2.45 GHz) in rat brain and protective effect of garlic. Int. J. Radiat. Biol. 90:892-896.

Gustavino, B., Carboni, G., Petrillo, R., et al. (2016). Exposure to 915 MHz radiation induces micronuclei in Vicia faba root tips. Mutagenesis. 31:187-192.

Habauzit, D., Le Quément, C., Zhadobov, M., et al. (2014). Transcriptome analysis reveals the contribution of thermal and the specific effects in cellular response to millimeter wave exposure. PLoS One. 9:e109435.

Habauzit, D., Nugue, G., Bourbon, F., et al. (2020). Evaluation of the effect of chronic 94 GHz exposure on gene expression in the skin of hairless rats *In Vivo*. Radiat. Res. 193:351-358.

Haider, T., Knasmueller, S., Kundi, M., et al. (1994). Clastogenic effects of radiofrequency radiations on chromosomes of Tradescantia. Mutat. Res. 324:65-68.

Hancı, H., Odacı, E., Kaya, H., et al. (2013). The effect of prenatal exposure to 900-MHz electromagnetic field on the 21-old-day rat testicle. Reprod. Toxicol. 42:203-209.

Hansteen, I.L., Lågeide, L., Clausen, K. O., et al. (2009a). Cytogenetic effects of 18.0 and 16.5 GHz microwave radiation on human lymphocytes in vitro. Anticancer Res. 29:2885-2892.

Hansteen, I.L., Clausen, K.O., Haugan, V., et al. (2009b). Cytogenetic effects of exposure to 2.3 GHz radiofrequency radiation on human lymphocytes in vitro. Anticancer Res. 29:4323-4330.

Hao, Y., Yang, X., Chen, C., et al. (2010). STAT3 signalling pathway is involved in the activation of microglia induced by 2.45 GHz electromagnetic fields. Int. J. Radiat. Biol. 86:27-36.

He, Q., Sun, Y., Zong, L., et al. (2016). Induction of poly(ADP-ribose) polymerase in mouse bone marrow stromal cells exposed to 900 MHz radiofrequency fields: preliminary observations. Biomed. Res. Int. 2016:4918691.

He, Q., Zong, L., Sun, Y., et al. (2017). Adaptive response in mouse bone marrow stromal cells exposed to 900 MHz radiofrequency fields: Impact of poly (ADP-ribose) polymerase (PARP). Mutat. Res. 820:19-25.

Hekmat, A., Saboury, A.A., Moosavi-Movahedi, A.A. (2013). The toxic effects of mobile phone radiofrequency (940MHz) on the structure of calf thymus DNA. Ecotoxicol. Environ. Saf. 88:35-41.

Hintzsche, H., Stopper, H. (2010). Micronucleus frequency in buccal mucosa cells of mobile phone users. Toxicol. Lett. 193;124-130.

Hintzsche, H., Jastrow, C., Kleine-Ostmann, T., et al. (2012a). 900 MHz radiation does not induce micronucleus formation in different cell types. Mutagenesis. 27:477-483.

Hintzsche, H., Jastrow, C., Kleine-Ostmann, T., et al. (2012b) Terahertz electromagnetic fields (0.106 THz) do not induce manifest genomic damage in vitro. PLoS One 7(9):e46397.

Hirose, H., Sakuma, N., Kaji, N., et al. (2006). Phosphorylation and gene expression of p53 are not affected in human cells exposed to 2.1425 GHz band CW or W-CDMA modulated radiation allocated to mobile radio base stations. Bioelectromagnetics 27:494-504.

Hirose, H., Sakuma, N., Kaji, N., et al. (2007). Mobile phone base station-emitted radiation does not induce phosphorylation of Hsp27. Bioelectromagnetics 28:99-108.

Hook, G.J., Zhang, P., Lagroye, I., et al. (2004). Measurement of DNA damage and apoptosis in molt-4 cells after in vitro exposure to radiofrequency radiation. Radiat. Res. 161:193-200.

Hou, Q., Wang, M., Wu, S., et al. (2015). Oxidative changes and apoptosis induced by 1800-MHz electromagnetic radiation in NIH/3T3 cells. Electromagn. Biol. Med. 34:85-92.

Houston, B.J., Nixon, B., McEwan, K.E., et al. (2019). Whole-body exposures to radiofrequency-electromagnetic energy can cause DNA damage in mouse spermatozoa via an oxidative mechanism. Sci. Rep. 9:17478.

Huang, T.Q., Lee, M.S., Oh, E., et al. (2008a). Molecular responses of Jurkat T-cells to 1763 MHz radiofrequency radiation. Int. J. Radiat. Biol. 84:734-741.

Huang, T.Q., Lee, M.S., Oh, E.H., et al. (2008b). Characterization of biological effect of 1763 MHz radiofrequency exposure on auditory hair cells. Int. J. Radiat. Biol. 84:909-915.

Jeong, Y.J., Son, Y., Han, N.K., et al. (2018). Impact of long-term RF-EMF on oxidative stress and neuroinflammation in aging brains of C57BL/6 mice. Int. J. Mol. Sci. 19: 2103.

Jeong, Y.J., Son,Y., Choi, H-D., et al. (2020). Behavioral changes and gene profile alterations after chronic 1,950-MHz radiofrequency exposure: An observation in C57BL/6 mice. Brain Behav. 10(11):e01815.

Ji, S., Oh, E., Sul, D., et al. (2004). DNA Damage of lymphocytes in volunteers after 4 hours use of mobile phone. J. Prev. Med. Public Health. 37:373-380.

Ji, Y., He, Q., Sun, Y., et al. (2016). Adaptive response in mouse bone-marrow stromal cells exposed to 900-MHz radiofrequency fields: Gamma-radiation-induced DNA strand breaks and repair. J. Toxicol. Environ. Health A. 79:419-426.

Jiang, B., Nie, J., Zhou, Z., et al. (2012). Adaptive response in mice exposed to 900 MHz radiofrequency fields: primary DNA damage. PLoS One. 7(2):e32040.

Jiang, B., Zong, C., Zhao, H., et al. (2013). Induction of adaptive response in mice exposed to 900MHz radiofrequency fields: application of micronucleus assay. Mutat. Res. 751:127-129.

Juutilainen, J., Heikkinen, P., Soikkeli, H., et al. (2007). Micronucleus frequency in erythrocytes of mice after long-term exposure to radiofrequency radiation. Int. J. Radiat. Biol. 83:213-220.

Karaca, E., Durmaz, B., Altug, H., et al. (2012). The genotoxic effect of radiofrequency waves on mouse brain. J, Neurooncol, 106:53-58.

Kerbacher, J.J., Meltz, M.L., Erwin, D.N. (1990). Influence of radiofrequency radiation on chromosome aberrations in CHO cells and its interaction with DNA-damaging agents. Radiat. Res. 123:311-319.

Kesari, K.K., Behari, J. (2009). Fifty-gigahertz microwave exposure effect of radiations on rat brain. Appl. Biochem. Biotechnol. 158:126-139.

Kesari, K.K., Behari, J., Kumar, S. (2010). Mutagenic response of 2.45 GHz radiation exposure on rat brain. Int. J. Radiat. Biol. 86:334-343.

Kesari, K.K., Kumar, S., Behari, J. (2011). Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats. Appl. Biochem. Biotechnol. 164:546-559.

Kesari, K.K., Meena, R., Nirala, J., et al. (2014). Effect of 3G cell phone exposure with computer controlled 2-D stepper motor on non-thermal activation of the hsp27/p38MAPK stress pathway in rat brain. Cell. Biochem. Biophys. 68:347-358.

Khalil, A.M., Ashamali, A.M., Gagaa, M. (2011). Detection of oxidative stress induced by mobile phone radiation in tissues of mice using 8-0xo-7, 8-dihydro-2'-deoxyguanosine as a biomarker. Int. J. Biol. Biomol. Agricul. Food Biotech. Eng. 5: 240-245.

Khalil, A.M., Gagaa, M., Alshamali, A. (2012). 8-Oxo-7, 8-dihydro-2'-deoxyguanosine as a biomarker of DNA damage by mobile phone radiation. Hum. Exp.Toxicol. 31:734-740.

Khalil, A.M., Khadra, K.M.A., Aljaberi, A.M., et al. (2014). Assessment of oxidant/antioxidant status in saliva of cell phone users. Electromagn. Biol. Med. 33:92-97.

Kim, J.Y., Hong, S.Y., Lee, Y.M., (2008). In vitro assessment of clastogenicity of mobile-phone radiation (835 MHz) using the alkaline comet assay and chromosomal aberration test. Environ. Toxicol. 23:319-327.

Komatsubara, Y., Hirose, H., Sakurai, T., et al. (2005). Effect of high-frequency electromagnetic fields with a wide range of SARs on chromosomal aberrations in murine m5S cells. Mutat. Res. 587:114-119.

Korenstein-Ilan, A., Barbul, A., Hasin, P., et al. (2008). Terahertz radiation increases genomic instability in human lymphocytes. Radiat. Res. 170:224-234.

Koyama, S., Nakahara, T., Wake, K., et al. (2003). Effects of high frequency electromagnetic fields on micronucleus formation in CHO-K1 cells. Mutat. Res. 541: 81-89.

Koyama, S., Isozumi, Y., Suzuki, Y., et al. (2004). Effects of 2.45-GHz electromagnetic fields with a wide range of SARs on micronucleus formation in CHO-K1 cells. ScientificWorld J. 4: Suppl 2:29-40.

Koyama, S., Narita, E., Shimizu, Y., et al. (2016a). Twenty four-hour exposure to a 0.12 THz electromagnetic field does not affect the genotoxicity, morphological changes, or expression of heat shock protein in HCE-T cells. Int. J. Envrion. Health Res. Pub. Health. 13:793, 1-9.

Koyama, S., Narita, E., Shimizu, Y., et al. (2016b) Effects of long-term exposure to 60 GHz millileter-wavelength radiation on the genotoxicity and heat shock protein (Hsp) expression of cells derived from human eye. Int. J. Envrion. Health Res. Pub. Health. 13: 802, 1-9.

Kumar, G., Wood, A.W., Anderson, V., et al. (2011). Evaluation of hematopoietic system effectsa in vitro radiofrequency radiation exposure in rats. Int. J. Radiat. Biol. 87:231-240.

Kumar, G., McIntosh, R.L., Anderson, V., et al. (2015). A genotoxic analysis of the hematopoietic system after mobile phone type radiation exposure in rats. Int. J. Radiat. Biol. 91:664-672.

Kumar R, Deshmukh P.S,, Sharma, S., et al. (2021) Effect of mobile phone signal radiation on epigenetic modulation in the hippocampus of Wistar rat. Environ. Res. 192:110297.

Kumar, S., Kesari, K.K., Behari, J. (2010). Evaluation of genotoxic effects in male Wistar rats following microwave exposure. Indian J. Exp. Biol. 48:586-592.

Kumar, S., Behari, J., Sisodia, R. (2013). Influence of electromagnetic fields on reproductive system of male rats. Int. J. Radiat. Biol. 89:147-154.

Kumar, S., Nirala, J.P., Behari, J., et al. (2014). Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario. Indian J. Exp. Biol. 52890-897.

Kumar, A., Kaur, S., Chandel, S., et al. (2020). Comparative cyto- and genotoxicity of 900 MHz and 1800 MHz electromagnetic field radiations in root meristems of Allium cepa. Ecotoxicol. Environ. Saf. 188:109786.

Lagroye, I., Anane, R., Wettring, B.A., et al. (2004a). Measurement of DNA damage after acute exposure to pulsed-wave 2450 MHz microwaves in rat brain cells by two alkaline comet assay methods. Int. J. Radiat. Biol. 80:11-20.

Lagroye, I., Hook, G.J., Wettring, B.A., et al. (2004b). Measurements of alkali-labile DNA damage and protein-DNA crosslinks after 2450 MHz microwave and low-dose gamma irradiation in vitro. Radiat. Res. 161: 201-214.

Lai, H., Singh, N.P. (1995). Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics 16:207-210.

Lai, H., Singh, N.P. (1996). Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int. J. Radiat. Biol. 69:513-521.

Lai, H., Singh, N.P. (1997). Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells. Bioelectromagnetics 18:446-454.

Lai, H., Singh, N.P. (2005). Interaction of microwaves and a temporally incoherent magnetic field on single and double DNA strand breaks in rat brain cells. Electromag. Biol. Med. 24:23-29.

Lai, H., Carino, M.A., Singh, N.P. (1997). Naltrexone blocks RFR-induced DNA double strand breaks in rat brain cells. Wireless Networks 3:471-476.

Lakshmi, N.K., Tiwari, R., Bhargava, S.C., et al. (2010). Investigations on DNA damage and frequency of micronuclei in occupational exposure to electromagnetic fields (EMFs) emitted from video display terminals (VDTs). Gen. Mol. Biol. 33:154-158.

Lameth, J., Arnaud-Cormos, D., Lévêque, P., et al. (2020). Effects of a single head exposure to GSM-1800 MHz signals on the transcriptome profile in the rat cerebral cortex: enhanced gene responses under proinflammatory conditions. Neurotox. Res., doi: 10.1007/s12640-020-00191-3. [Epub ahead of print]

Lamkowski, A., Kreitlow, M., Radunz, J., et al. (2018). Gene expression analysis in human peripheral blood cells after 900 MHz RF-EMF short-term exposure. Radiat. Res. 189:529-540.

Le Quément, C., Nicolas Nicolaz, C., Zhadobov, M., et al. (2012). Whole-genome expression analysis in primary human keratinocyte cell cultures exposed to 60 GHz radiation. Bioelectromagnetics 33:147-158.

Lee, S., Johnson, D., Dunbar, K., et al. (2005). 2.45 GHz radiofrequency fields alter gene expression in cultured human cells. FEBS Lett. 579:4829-4836.

Lerchl, A., Klose, M., Drees, K. (2020) No increased DNA damage observed in the brain, liver, and lung of fetal mice treated with ethylnitrosourea and sxposed to UMTS radiofrequency electromagnetic fields. Bioelectromagnetics doi: 10.1002/bem.22301. Online ahead of print.

Li, L., Bisht, K.S., LaGroye, I., et al. (2001). Measurement of DNA damage in mammalian cells exposed in vitro to radiofrequency fields at sars of 3-5 w/kg. Radiat. Res. 156:328-332.

Li, R., Ma, M., Li, L., et al. (2018). The protective effect of autophagy on DNA damage in mouse spermatocyte-derived cells exposed to 1800 MHz radiofrequency electromagnetic fields. Cell. Physiol. Biochem. 48:29-41.

Li, Z.Q., Zhang, Y., Wan, Y.M., et al. (2020). Testing of behavioral and cognitive development in rats after prenatal exposure to 1800 and 2400 MHz radiofrequency fields. J Radiat Res. 61:197-206.

Lin, K.W., Yang, C.J., Lian, H.Y., et al. (2016). Exposure of ELF-EMF and RF-EMF increase the rate of glucose transport and TCA cycle in budding yeast. Front. Microbiol. 7:1378.

Liu, C., Duan, W., Xu, S., et al. (2013a). Exposure to 1800 MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line. Toxicol. Lett. 218: 2-9.

Liu, C., Gao, P., Xu, S.C., et al. (2013b). Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin. Int. J. Radiat. Biol. 89:993-1001.

Lixia, S., Yao, K., Kaijun, W., et al. (2006). Effects of 1.8 GHz radiofrequency field on DNA damage and expression of heat shock protein 70 in human lens epithelial cells. Mutat. Res. 602:135-142.

López-Martín, E., Bregains, J., Relova-Quinteiro, J.L., et al. (2009). The action of pulse-modulated GSM radiation increases regional changes in brain activity and c-Fos expression in cortical and subcortical areas in a rat model of picrotoxin-induced seizure proneness. J. Neurosci. Res. 871484-1499.

Luukkonen, J., Hakulinen, P., Mäki-Paakkanen, J., et al. (2009). Enhancement of chemically induced reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells by 872 MHz radiofrequency radiation. Mutat. Res. 662:54-58.

Luukkonen, J., Juutilainen, J., Naarala, J. (2010). Combined effects of 872 MHz radiofrequency radiation and ferrous chloride on reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells. Bioelectromagnetics 31:417-424.

Maes, A., Verschaeve, L., Arroyo, A., et al. (1993). In vitro cytogenetic effects of 2450 MHz waves on human peripheral blood lymphocytes. Bioelectromagnetics 14:495-501.

Maes, A., Collier, M., Slaets, D., et al. (1995). Cytogenetic effects of microwaves from Mobile communication frequencies (954 MHz). Electro- MagnetoBiol. 14: 91-98.

Maes, A., Collier, M., Slaets, D., et al. (1996). 954 MHz microwaves enhance the mutagenic properties of mitomycin C. Environ. Mol. Mutagen 28:26-30.

Maes, A., Collier, M., Van Gorp, U., et al. (1997). Cytogenetic effects of 935.2-MHz (GSM) microwaves alone and in combination with mitomycin C. Mutat. Res. 393:151-156.

Maes, A., Collier, M., Verschaeve, L. (2000). Cytogenetic investigations on microwaves emitted by a 455.7 MHz car phone. Folia Biol. (Praha) 46:175-180.

Maes, A., Collier, M., Verschaeve, L. (2001). Cytogenetic effects of 900 MHz (GSM) microwaves on human lymphocytes. Bioelectromagnetics 22:91-96.

Maes, A., Van Gorp, U., Verschaeve, L.(2006). Cytogenetic investigation of subjects professionally exposed to radiofrequency radiation. Mutagenesis 21:139-142.

Malini, V. (2017). Resolving the enigma of effect of mobile phone usage on spermatogenetsis in humans in south Indian population. Asian J. Pharmaceut. Clin. Res. 10: 233-237.

Malyapa, R.S., Ahern, E.W., Straube, W.L., et al. (1997a). Measurement of DNA damage after exposure to 2450 MHz electromagnetic radiation. Radiat. Res. 148:608-617.

Malyapa, R.S., Ahern, E.W., Straube, W.L., et al. (1997b). Measurement of DNA damage after exposure to electromagnetic radiation in the cellular phone communication frequency band (835.62 and 847.74 MHz). Radiat. Res. 148:618-627.

Malyapa, R.S,, Ahern, E.W., Bi, C., et al. (1998). DNA damage in rat brain cells after in vivo exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia. Radiat. Res. 149:637-645.

Manta, A.K., Papadopoulou, D., Polyzos, A.P., et al. (2017). Mobile-phone radiation-induced perturbation of gene-expression profiling, redox equilibrium and sporadic-apoptosis control in the ovary of Drosophila melanogaster. Fly (Austin). 11:75-95.

Manti, L., Braselmann, H., Calabrese, M.L., et al. (2008). Effects of modulated microwave radiation at cellular telephone frequency (1.95 GHz) on X-ray-induced chromosome aberrations in human lymphocytes in vitro. Radiat. Res. 169:575-583.

Marinelli, F., La Sala, D., Cicciotti, G., et al. (2004). Exposure to 900 MHz electromagnetic field induces an unbalance between pro-apoptotic and pro-survival signals in T-lymphoblastoid leukemia CCRF-CEM cells. J. Cell. Physiol. 198:324-332.

Markova, E., Hillert, L., Malmgren, L., et al. (2005). Microwaves from GSM

mobile telephones affect 53BP1 and gamma-H2AX foci in human lymphocytes from hypersensitive and healthy persons. Environ. Health Perspect. 113:1172-1177.

Markovà, E., Malmgren, L.O., Belyaev, I.Y. (2010). Microwaves from mobile phones inhibit 53BP1 focus formation in human stem cells more strongly than in differentiated cells: possible mechanistic link to cancer risk. Environ. Health Perspect. 118:394-399.

Martin, C., Percevault, F., Ryder, K., et al. (2020). Effects of radiofrequency radiation on gene expression: a study of gene expressions of human keratinocytes from different origins. Bioelectromagnetics.41:552-557.

Mashevich, M., Folkman, D., Kesar, A., et al. (2003). Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability. Bioelectromagnetics 24:82-90.

Mazor, R., Korenstein-Ilan, A., Barbul, A., et al. (2008). Increased levels of numerical chromosome aberrations after in vitro exposure of human peripheral blood lymphocytes to radiofrequency electromagnetic fields for 72 hours. Radiat. Res. 169:28-37.

McNamee, J.P., Bellier, P.V., Gajda, G.B., et al. (2002a). DNA damage and micronucleus induction in human leukocytes after acute in vitro exposure to a 1.9 GHz continuous-wave radiofrequency field. Radiat. Res. 158:523-533.

McNamee, J.P., Bellier, P.V., Gajda, G.B., et al. (2002b). DNA Damage in human leukocytes after acute in vitro exposure to a 1.9 GHz pulse-modulated radiofrequency field. Radiat. Res. 158:534-537.

McNamee, J. P., Bellier, P. V., Gajda, G. B., et al. (2003). No evidence for genotoxic effects from 24 h exposure of human leukocytes to 1.9 GHz radiofrequency fields. Radiat. Res. 159:693-697.

McNamee, J.P., Bellier, P.V., Konkle, A.T., et al. (2016). Analysis of gene expression in mouse brain regions after exposure to 1.9 GHz radiofrequency fields. Int. J. Radiat. Biol. 92:338-350.

Meena, R., Kumari, K., Kumar, J., et al. (2014). Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats. Electromag. Biol. Med. 33: 81-91

Megha, K., Deshmukh, P.S., Ravi, A.K., et al. (2015a). Effect of low-intensity microwave radiation on monoamine neurotransmitters and their key regulating enzymes in rat brain. Cell. Biochem. Biophys. 73:93-100.

Megha, K., Deshmukh, P.S., Banerjee, B.D., et al. (2015b) Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain. NeuroToxicol. 51: 158-165.

Meltz, M.L., Eagan, P., Erwin, D.N. (1990). Proflavin and microwave radiation: absence of a mutagenic interaction. Bioelectromagnetics 11:149-157.

Mildažienė, V., Aleknavičiūtė, V., Žūkienė, R., et al. (2019). Treatment of common sunflower (Helianthus annus L.) seeds with radio-frequency electromagnetic field and cold plasma induces changes in seed phytohormone balance, seedling development and leaf protein expression. Sci. Rep. 9:6437.

Millenbaugh, N.J. , Roth, C., Sypniewska, R., et al. (2008) Gene expression changes in the skin of rats induced by prolonged 35 GHz millimeter-wave exposure. Radiat. Res. 169:288-300.

Miyakoshi, J., Yoshida, M., Tarusawa, Y., et al. (2002). Effects of high- frequency electromagnetic fields on DNA strand breaks using comet assay method. Electrical Eng. Japan. 141: 9–15.

Nakatani-Enomoto, S., Okutsu, M., Suzuki, S., et al. (2016). Effects of 1950MHz W-CDMA-Like signal on human spermatozoa. Bioelectromagentics. 37: 373- 381.

Narasimhan, V., Huh, W.K. (1991). Altered restriction patterns of microwave irradiated lambdaphage DNA. Biochem. Int. 25:363-370.

Nikolova, T., Czyz, J., Rolletschek, A., et al. (2005). Electromagnetic fields affect transcript levels of apoptosis-related genes in embryonic stem cell-derived neural progenitor cells. FASEB J. 19:1686-1688.

Nittby, H., Widegren, B., Krogh, M., et al. (2008). Exposure to radiation from global system for mobile communications at 1,800 MHz significantly changes gene expression in rat hippocampus and cortex. Environmentalist 28: 458-465.

Nylund, R., Leszczynski, D. (2006). Mobile phone radiation causes changes in gene and protein expression in human endothelial cell lines and the response seems to be genome- and proteome-dependent. Proteomics 6:4769-4780.

Odacı, E., Hancı, H., Yuluğ, E., et al. (2016). Effects of prenatal exposure to a 900 MHz electromagnetic field on 60-day-old rat testis and epididymal sperm quality. Biotech. Histochem. 91:9-19.

Ohtani, S., Ushiyama, A., Maeda, M., et al. (2016). Exposure time-dependent thermal effects of radiofrequency electromagnetic field exposure on the whole body of rats. J. Toxicol. Sci. 41:655-666.

Ohtani, S., Ushiyama, A., Maeda, M., et al. (2019). Global analysis of transcriptional expression in mice exposed to intermediate frequency magnetic fields utilized for wireless power transfer systems. Int. J Environ. Res. Public Health 16:1851.

Ono, T., Saito, Y., Komura, J., et al. (2004). Absence of mutagenic effects of 2.45 GHz radiofrequency exposure in spleen, liver, brain, and testis of lacZ-transgenic mouse exposed in utero. Tohoku J. Exp. Med. 202:93-103.

Ozgur, E., Guler, G., Kismali, G., et al. (2014). Mobile phone radiation alters proliferation of hepatocarcinoma cells. Cell. Biochem. Biophys. 70:983-991.

Pacini, S., Ruggiero, M., Sardi, I., et al. (2002). Exposure to global system for mobile communication (GSM) cellular phone radiofrequency alters gene expression, proliferation, and morphology of human skin fibroblasts. Oncol. Res. 13:19-24.

Panagopoulos, D.J., Chavdoula, E.D., Nezis, I.P., et al. (2007). Cell death induced by GSM 900-MHz and DCS 1800-MHz mobile telephony radiation. Mutat. Res. 626:69-78.

Panagopoulos, D.J. (2019). Chromosome damage in human cells induced by UMTS mobile telephony radiation. Gen. Physiol. Biophys. 38:445-454.

Panagopoulos, D.J. (2020). Comparing chromosome damage induced by mobile telephony radiation and a high caffeine dose: Effect of combination and exposure duration. Gen. Physiol. Biophys. 39:531-544.

Pandey, N., Giri , S., Das, S., et al. (2017). Radiofrequency radiation (900 MHz)-induced DNA damage and cell cycle arrest in testicular germ cells in swiss albino mice. Toxicol. Ind. Health. 33:373-384.

Paparini, A., Rossi, P., Gianfranceschi, G., et al. (2008). No evidence of major transcriptional changes in the brain of mice exposed to 1800 MHz GSM signal. Bioelectromagnetics. 29:312-323.

Paulraj, R., Behari, J. (2006). Single strand DNA breaks in rat brain cells exposed to microwave radiation. Mutat. Res. 596:76-80.

Pesnya, D.S., Romanovsky, A.V. (2013). Comparison of cytotoxic and genotoxic effects of plutonium-239 alpha particles and mobile phone GSM 900 radiation in the Allium cepa test. Mutat. Res. 750:27-33.

Phillips, J.L., Ivaschuk, O., Ishida-Jones, T., et al. (1998). DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro. Bioelectrochem. Bioenerg. 45:103-110.

Port, M., Abend, M., Romer, B., et al. (2003). Influence of high-frequency electromagnetic fields on different modes of cell death and gene expression. Int. J. Radiat. Biol. 79:701-708.

Qin, F., Cao, H., Yuan, H., et al. (2018). 1800 MHz radiofrequency fields inhibits testosterone production via CaMKI /RORα pathway. Reprod. Toxicol. 81:229-236.

Qin, F., Shen, T., Cao, H., et al. (2019). CeO_2 NPs relieve radiofrequency radiation, improve testosterone synthesis, and clock gene expression in Leydig cells by enhancing antioxidation. Int. J. Nanomed. 14:4601-4611.

Qutob, S.S., Chauhan, V., Bellier, P.V., et al. (2006). Microarray gene expression profiling of a human glioblastoma cell line exposed in vitro to a 1.9 GHz pulse-modulated radiofrequency field. Radiat. Res. 165:636-644.

Racuciu, M. (2009). Effects of radiofrequency radiation on root tip cells of Zea mays. Roumanian Biotechnol. Lett. 14:4365-4369.

Rago, R., Salacone, P., Caponecchia, L., et al. (2013). The semen quality of the mobile phone users. J. Endocrinol. Invest. 36:970-974.

Rammal, M., Jebai, F.. Rammal, H., et al. (2014). Effects of long-term exposure to RF/MW radiations on the expression of mRNA of stress proteins in *Lycospersicon esculentum.* WSEAS Transect Biol Biomed. 11:10-14.

Regalbuto, E., Anselmo, A., De Sanctis, S., et al. (2020) Human fibroblasts in vitro exposed to 2.45 GHz continuous and pulsed wave signals: evaluation of biological effects with a multimethodological approach. Int. J. Mol. Sci. 21:E7069.

Remondini, D., Nylund, R., Reivinen, J., et al. (2006). Gene expression changes in human cells after exposure to mobile phone microwaves. Proteomics 6:4745-4754.

Romano-Spica, V., Mucci, N., Ursini, C.L, et al. (2000). Ets1 oncogene induction by ELF-modulated 50 MHz radiofrequency electromagnetic field. Bioelectromagnetics 21(1):8-18.

Ros-Llor, I., Sanchez-Siles, M., Camacho-Alonso, F., et al. (2012). Effect of mobile phones on micronucleus frequency in human exfoliated oral mucosal cells. Oral Dis. 18:786-792.

Roti Roti, J.L. , Malyapa, R.S, Bisht, K.S., et al. (2001). Neoplastic Transformation in C3H 10T(1/2) cells after exposure to 835.62 MHz FDMA and 847.74 MHz CDMA radiations. Radiat. Res. 155(1):239-247.

Roux, D., Vian, A., Girard, S., et al. (2006). Electromagnetic fields (900 MHz) evoke consistent molecular responses in tomato plants. Physiologia Plantarum 128: 283–288.

Roux, D., Vian, A., Girard, S., et al. (2008). High frequency (900 MHz) low amplitude (5 V m-1) electromagnetic field: a genuine environmental stimulus that affects transcription, translation, calcium and energy charge in tomato. Planta. 227(4):883-891.

Sagripanti, J.L, Swicord, M.L. (1986). DNA structural changes caused by microwave radiation. Int. J. Radiat. Biol. Relat. Stud. Phys. Chem. Med. 50:47-50.

Sagripanti, J.L., Swicord, M.L., Davis, C.C. (1987). Microwave effects on plasmid DNA. Radiat. Res. 110:219-221.

Sahin, D., Ozgur, E., Guler, G., et al. (2016). The 2100 MHz radiofrequency radiation of a 3G-mobile phone and the DNA oxidative damage in brain. J. Chem. Neuroanat. 75: 94-98.

Said-Salman, I.H., Jebaii, F.A., Yusef, H.H., et al. (2019).. Global gene expression analysis of Escherichia coli K-12 DH5α after exposure to 2.4 GHz wireless fidelity radiation. Sci. Rep. 9:14425.

Sakuma, N., Komatsubara, Y., Takeda, H., et al. (2006). DNA strand breaks are not induced in human cells exposed to 2.1425 GHz band CW and W-CDMA modulated radiofrequency fields allocated to mobile radio base stations. Bioelectromagnetics 27:51-57.

Sakurai, T., Kiyokawa, T., Narita, E., et al. (2011). Analysis of gene expression in a human-derived glial cell line exposed to 2.45 GHz continuous radiofrequency electromagnetic fields. J. Radiat. Res. 52:185-192.

Salmen, S.H., Alharbi, S.A., Faden, A.A., et al. (2018). Evaluation of effect of high frequency electromagnetic field on growth and antibiotic sensitivity of bacteria. Saudi J. Biol. Sci. 25:105-110.

Sannino, A., Calabrese, M.L., d'Ambrosio, G., et al. (2006). Evaluation of cytotoxic and genotoxic effects in human peripheral blood leukocytes following exposure to 1950- MHz modulated signal. IEEE Trans. Plasma Sci. 34: 1441-1448.

Sannino, A., Di Costanzo, G., Brescia, F., et al. (2009a). Human fibroblasts and 900 MHz radiofrequency radiation: evaluation of DNA damage after exposure and co-exposure to 3-Chloro-4-(dichloromethyl)-5-Hydroxy-2(5h)-furanone (MX). Radiat. Res 171:743-751.

Sannino, A., Costanzo, G.D., Brescia, F., et al. (2009b). Human fibroblasts and 900 MHz radiofrequency radiation: Evaluation of DNA damage after exposure and co-exposure to 3-chloro-4-(dichromomethyl)-5-hydroxy-2(5H)-furanone. Radiat. Res. 171: 743-751.

Sannino, A., Zeni, O., Sarti, M., et al. (2011). Induction of adaptive response in human blood lymphocytes exposed to 900 MHz radiofrequency fields: influence of cell cycle. Int. J. Radiat. Biol. 87: 993–999.

Sannino, A., Zeni, O., Romeo, S., et al. (2014). Adaptive response in human blood lymphocytes exposed to non-ionizing radiofrequency fields: resistance to ionizing radiation-induced damage. J. Radiat. Res. 55: 210-217.

Sannino, A., Zeni, O., Romeo, S., et al. (2017). Adverse and beneficial effect in Chinese hamster lung fibroblast cells following radiofrequency exposure. Bioelectromagnetics. 38: 245-254.

Sarimov, R., Malmgren, L.O.G., Markova, E., et al. (2004). Nonthermal GSM microwaves affect chromatin conformation in human lymphocytes similar to heat shock. IEEE Trans. Plasma Sci. 32:1600-1608.

Sarkar, S., Ali, S., Behari, J. (1994). Effect of low power microwave on the mouse genome: a direct DNA analysis. Mutat. Res, 320:141-147.

Scarfi, M.R., Lioi, M.B., d'Ambrosio, G., et al. (1996). Genotoxic effects of mitomycn-C and microwave radiation on bovine lymphocytes. ElectroMagneto Biol. 15, 99-107.

Scarfi, M.R., Romano, M., Di Pietro, R., et al. (2003). THz exposure of whole blood for the study of biological effects on human lymphocytes. J. Biol. Phy. 29: 171-177.

Scarfi, M.R., Fresegna, A.M., Villani, P., et al. (2006). Exposure to radiofrequency radiation (900 MHz, GSM signal) does not affect micronucleus frequency and cell proliferation in human peripheral blood lymphocytes: an interlaboratory study. Radiat. Res. 165:655-663.

Schuermann, D., Ziemann, C., Barekati, Z., et al. (2020). Assessment of genotoxicity in human cells exposed to modulated electromagnetic fields of wireless communication devices. Genes (Basel). 11(4). pii: E347.

Schwarz, C., Kratochvil, E., Pilger, A., et al. (2008). Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes. Int. Arch. Occup. Environ. Health 81:755-767.

Sekeroğlu, V., Akar, A., Sekeroğlu, Z.A. (2012). Cytotoxic and genotoxic effects of high-frequency electromagnetic fields (GSM 1800 MHz) on immature and mature rats. Ecotoxicol. Environ. Saf. 80:140-144.

Sekeroglu, Z.A., Akar, A., Sekeroglu, V. (2013). Evaluation of the cytogenetic damage in immature and mature rats exposed to 900 MHz radiofrequency electromagnetic fields. Int. J. Radiat. Biol. 89:985-892.

Sekijima, M., Takeda, H., Yasunaga, K., et al. (2010). 2-GHz band CW and W-CDMA modulated radiofrequency fields have no significant effect on cell proliferation and gene expression profile in human cells. J. Radiat. Res. 51:277-284.

Semin, IuA, Shvartsburg, L.K., Dubovik, B.V. (1995). [Changes in the secondary structure of DNA under the influence of external low-intensity electromagnetic field] Radiats. Biol. Radioecol. 35:36-41.

Senturk, E., Tugrul, S., Doğan, R., et al. (2019). Effect of radiofrequency on DNA damage and oxidative status in patients with turbinate uypertrophy. Indian J. Otolaryngol. Head Neck Surg. 71(Suppl 3):1810-1815.

Shah, C., Nair, A., Naik, M, et al (2015). Cell phone radiation and genomic damage: In vitro exposure and assessment. Int. J. Innnov. Res. Sci. Eng. Technol. 4: 401-405.

Shahin, S., Singh, V.P., Shukla, R.K., et al. (2013). 2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus. Appl. Biochem. Biotechnol. 169:1727-1751.

Shahin, N.N., El-Nabarawy, N.A., Gouda, A.S., et al. (2019). The protective role of spermine against male reproductive aberrations induced by exposure to electromagnetic field - An experimental investigation in the rat. Toxicol. Appl. Pharmacol. 370:117-130.

Sharma, S., Shukla, S. (2020). Effect of electromagnetic radiation on redox status, acetylcholine esterase activity and cellular damage contributing to the diminution of the brain working memory in rats. J. Chem. Neuroanat. 2020 Mar 20:101784.

Shckorbatov, Y.G., Pasiuga, V.N., Kolchigin, N.N., et al. (2009). The influence of differently polarized microwave radiation on chromatin in human cells. Int. J. Radiat. Biol. 85: 322-329.

Shckorbatov, Y.G., Pasiuga, V.N., Goncharuk, E.I., et al. (2010). Effects of differently polarized microwave radiation on the microscopic structure of the nuclei in human fibroblasts. J. Zhejiang Univ. Sci. B. 11: 801-805.

Shi, D., Zhu, C., Lu, R., et al. (2014). Intermediate frequency magnetic field generated by a wireless power transmission device does not cause genotoxicity in vitro. Bioelectromagnetics. 35:512-518.

Silva, V., Hilly. O., Strenov, Y., et al. (2016). Effect of cell phone-like electromagnetic radiation on primary human thyroid cells. Int. J. Radiat. Biol. 92: 107-115.

Smith-Roe, S.L., Wyde, M.E., Stout, M.D., et al. (2020). Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure. Environ. Mol. Mutagen. 61:276-290.

Sokolovic, D., Djordjevic, B., Kocic, G., et al. (2015) The effects of melatonin on oxidative stress parameters and DNA fragmentation in testicular tissue of rats exposed to microwave radiation. Adv. Clin. Exp. Med. 24:429-436**.**

Soubere Mahamoud, Y., Aite, M., Martin, C., et al. (2016). Additive effects of millimeter waves and 2-deoxyglucose co-exposure on the human keratinocyte transcriptome. PLoS One. 11:e0160810.

Souza, L.D., Cerqueira, E.D., Meireles, J.R. (2014). Assessment of nuclear abnormalities in exfoliated cells from the oral epithelium of mobile phone users. Electromagn. Biol. Med. 33:98-102.

Speit, G., Schütz, P., Hoffmann, H. (2007). Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in cultured mammalian cells are not independently reproducible. Mutat. Res. 626:42-47.

Speit, G., Gminski, R., Tauber, R. (2013). Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in HL-60 cells are not reproducible. Mutat. Res. 755:163-166.

Stronati, L., Testa, A., Moquet, J., et al. (2006). 935 MHz cellular phone radiation. An in vitro study of genotoxicity in human lymphocytes. Int. J. Radiat. Biol. 82:339-346.

Su, L., Yimaer, A., Xu, Z., et al. (2018). Effects of 1800 MHz RF-EMF exposure on DNA damage and cellular functions in primary cultured neurogenic cells. Int. J. Radiat. Biol. 94:295-305.

Sun, C., Wei, X., Fei, Y., et al. (2016). Mobile phone signal exposure triggers a hormesis-like effect in Atm+/+ and Atm-/- mouse embryonic fibroblasts. Sci. Rep. 6:37423.

Sun, L.X., Yao, K., He, J.L., et al. (2006a) [Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro.] Zhonghua Lao Dong Wei Sheng Zhi Ye Bing ZaZhi. 24:465-467. [Article in Chinese]

Sun, L.X., Yao, K., Jiang, H., et al. (2006b). [DNA damage and repair induced by acute exposure of microwave from mobile phone on cultured human lens epithelial cells] Zhonghua Yan Ke Za Zhi. 42:1084-1088. [Article in Chinese]

Sun, Y., Zong, L., Gao, Z., et al. (2017). Mitochondrial DNA damage and oxidative damage in HL-60 cellsexposed to 900 MHz radiofrequency fields. Mutat. Res. 797: 7-14.

Sykes, P.J., McCallum, B.D., Bangay, M.J., et al. (2001). Effect of exposure to 900 MHz radiofrequency radiation on intrachromosomal recombination in pKZ1 mice. Radiat. Res. 156:495-502.

Takahashi, S., Inaguma, S., Cho, Y-M., et al. (2002). Lack of mutation induction with exposure to 1.5 GHz electromagnetic near fields used for cellular phones in brains of big blue mice. Cancer Res. 62:1956-1960.

Tice, R.R., Hook, G.G., Donner, M., et al. (2002). Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells. Bioelectromagnetics 23:113-126.

Tiwari, R., Lakshmi, N.K., Surender, V., et al. (2008). Combinative exposure effect of radio frequency signals from CDMA mobile phones and aphidicolin on DNA integrity. Electromagn. Biol. Med. 27:418-425.

Tkalec, M., Malarić, K., Pavlica, M., et al. (2009). Effects of radiofrequency electromagnetic fields on seed germination and root meristematic cells of Allium cepa L. Mutat. Res. 672:76-81.

Tkalec, M., Stambuk, A., Srut, M., et al. (2013). Oxidative and genotoxic effects of 900 MHz electromagnetic fields in the earthworm Eisenia fetida. Ecotoxicol. Environ. Saf. 90:7-12.

Tohidi, F., Sadr-Nabavi, A., Haghir, H., et al.(2020). Long-term exposure to electromagnetic radiation from mobile phones can cause considerable changes in the balance of Bax/Bcl2 mRNA expression in the hippocampus of mice. Electromagn Biol Med 2020 Oct 20;1-7. doi: 10.1080/15368378.2020.1830793. Online ahead of print.

Tomruk, A., Guler, G., Dincel, A.S. (2010). The influence of 1800 MHz GSM-like signals on hepatic oxidative DNA and lipid damage in nonpregnant, pregnant, and newly born rabbits. Cell. Biochem. Biophys. 56:39-47.

Trivino Pardo, J.C., Grimaldi, S., Taranta, M., et al. (2012). Microwave electromagnetic field regulates gene expression in T-lymphoblastoid leukemia CCRF-CEM cell line exposed to 900 MHz. Electromagn. Biol. Med. 31:1-18.

Trosic, I. (2001). Multinucleated giant cell appearance after whole body microwave irradiation of rats. Int. J. Hyg. Environ. Health. 204:133-138.

Trosic, I., Busljeta, I. (2005). Frequency of micronucleated erythrocytes in rat bone marrow exposure to 2.45 GHz radiation. Physica Scripta. T118: 168-170.

Trosic, I., Busljeta, I. (2006). Erythropoietic dynamic equilibrium in rats maintained after microwave irradiation. Exp. Toxicol. Pathol. 57:247-251.

Trosic, I., Busljeta, I., Kasuba, V., et al. (2002). Micronucleus induction after whole-body microwave irradiation of rats. Mutat. Res. 521:73-79.

Trosic, I., Busljeta, I., Modlic, B. (2004). Investigation of the genotoxic effect of microwave irradiation in rat bone marrow cells: in vivo exposure. Mutagenesis. 19:361-364.

Trosić, I., Pavicić, I., Milković-Kraus, S., et al. (2011). Effect of electromagnetic radiofrequency radiation on the rats' brain, liver and kidney cells measured by comet assay. Coll. Antropol. 35:1259-1264.

Tsybulin, O., Sidorik, E., Brieieva, O., et al. (2013). GSM 900 MHz cellular phone radiation can either stimulate or depress early embryogenesis in Japanese quails depending on the duration of exposure. Int. J. Radiat. Biol. 89:756-763.

Usikalu, M.R., Obembe, O.O., Akinyemi, M.L., et al. (2013). Short-duration exposure to 2.45 GHz microwave radiation induces DNA damage in Sprague Dawley rat's reproductive systems. African J. Biotechnol. 12:115-122.

Vafaei, H., Kavari, G., Izadi, H.R., et al. (2020). Wi-Fi (2.4 GHz) affects anti-oxidant capacity, DNA repair genes expression and, apoptosis in pregnant mouse placenta. Iran J. Basic Med. Sci. 23:833-840.

Valbonesi, P., Franzellitti, S., Piano, A., et al. (2008). Evaluation of HSP70 expression and DNA damage in cells of a human trophoblast cell line exposed to 1.8 GHz amplitude-modulated radiofrequency fields. Radiat. Res. 169:270-279.

Valbonesi, P., Franzellitti, S., Bersani, F., et al. (2014). Effects of the exposure to intermittent 1.8 GHz radio frequency electromagnetic fields on HSP70 expression and MAPK signaling pathways in PC12 cells. Int. J. Radiat. Biol. 90:382-391.

Valbonesi, P., Franzellitti, S., Bersani, F., et al. (2016). Activity and expression of acetylcholinesterase in PC12 cells exposed to intermittent 1.8 GHz 217-GSM mobile phone signal. Int. J. Radiat. Biol. 92:1-10.

Vanishree, M., Manvikar, V., Rudraraju, A., et al. (2018). Significance of micronuclei in buccal smears of mobile phone users: A comparative study. J. Oral Maxillofacial Pathol. 22:448–452.

Varghese, R., Majumdar, A., Kumar, G., et al. (2018). Rats exposed to 2.45GHz of non-ionizing radiation exhibit behavioral changes with increased brain expression of apoptotic caspase 3. Pathophysiology. 25:19-30.

Veerachari, S.B., Vasan, S.S. (2012). Mobile phone electromagnetic waves and its effect on human ejaculated semen: an in vitro study. Int. J. Infertility Fetal Med. 3:15-21.

Verschaeve, L., Heikkinen, P., Verheyen, G., et al. (2006). Investigation of co-genotoxic effects of radiofrequency electromagnetic fields in vivo. Radiat. Res. 165:598-607.

Vian, A., Roux, D., Girard, S., et al. (2006). Microwave irradiation affects gene expression in plants. Plant Signal Behav. 1:67-70.

Vijayalaxmi, Frei, M.R., Dusch, S.J., et al. (1997a). Frequency of micronuclei in the peripheral blood and bone marrow of cancer-prone mice chronically exposed to 2450 MHz radiofrequency radiation. Radiat. Res. 147:495-500. "See also *Radiat. Res.* 149:308, 1998."

Vijayalaxmi, Mohan, N., Meltz, M.L., et al. (1997b). Wittler MA. Proliferation and cytogenetic studies in human blood lymphocytes exposed in vitro to 2450 MHz radiofrequency radiation. Int. J. Radiat. Biol. 72:751-757.

Vijayalaxmi, Seaman, R.L., Belt, M.L., et al. (1999). Frequency of micronuclei in the blood and bone marrow cells of mice exposed to ultra-wideband electromagnetic radiation. Int. J. Radiat. Biol. 75:115-120.

Vijayalaxmi, Leal, B.Z., Szilagyi, M., et al. (2000). Primary DNA damage in human blood lymphocytes exposed In vitro to 2450 MHz radiofrequency radiation. Radiat. Res. 153:479-486.

Vijayalaxmi , Leal, B.Z., Meltz, M.L., et al (2001a). Cytogenetic studies in human blood lymphocytes exposed in vitro to radiofrequency radiation at a cellular telephone frequency (835.62 MHz, FDMA). Radiat. Res. 155:113-121.

Vijayalaxmi, Pickard ,W.F,, Bisht, K.S., et al. (2001b). Micronuclei in the peripheral blood and bone marrow cells of rats exposed to 2450 MHz radiofrequency radiation. Int. J. Radiat. Biol. 771109-1115.

Vijayalaxmi, Bisht, K.S., Pickard, W.F., et al (2001c). Chromosome damage and micronucleus formation in human blood lymphocytes exposed in vitro to radiofrequency radiation at a cellular telephone frequency (847.74 MHz, CDMA). Radiat. Res. 156.430-432.

Vijayalaxmi, Sasser, L.B., Morris. J.E., et al. (2003). Genotoxic potential of 1.6 GHz wireless communication signal: in vivo two-year bioassay. Radiat. Res. 159:558-564.

Vijayalaxmi, Logani, M.K., Bhanushali, A., et al. (2004). Micronuclei in peripheral blood and bone marrow cells of mice exposed to 42 GHz electromagnetic millimeter waves. Radiat. Res. 161: 341–345.

Vijayalaxmi. (2006). Cytogenetic studies in human blood lymphocytes exposed in vitro to 2.45 GHz or 8.2 GHz radiofrequency radiation. Radiat. Res. 166:532–538.

Vilić, M., Tlak Gajger, I., Tucak, P., et al. (2017). Effects of short-term exposure to mobile phone radiofrequency (900 MHz) on the oxidative response and genotoxicity in honey bee larvae. J. Apic. Res. 56:430–438.

Waldmann, P., Bohnenberger, S., Greinert, R., et al. (2013). Influence of GSM signals on human peripheral lymphocytes: study of genotoxicity. Radiat. Res. 179:243-253.

Wang, X., Liu, C., Ma, Q., et al. (2015). 8-oxoG DNA glycosylase-1 inhibition sensitizes Neuro-2a cells to oxidative DNA base damage induced by 900 MHz radiofrequency electromagnetic radiation. Cell. Physiol. Biochem. 37:1075-1088.

Wu, W., Yao, K., Wang, K.J., et al. (2008). [Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields.] Zhejiang Da XueXueBao Yi Xue Ban 37:34-38. [Article in Chinese]

Xu, S., Zhong, M., Zhang, L., et al. (2010). Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons. Brain Res. 1311:189-196.

Xu, S., Chen, G., Chen, C., et al. (2013). Cell type-dependent induction of DNA damage by 1800 MHz radiofrequency electromagnetic fields does not result in significant cellular dysfunctions. PLoS One. 8:e54906.

Yadav, A.S., Sharma, M.K. (2008). Increased frequency of micronucleated exfoliated cells among humans exposed in vivo to mobile telephone radiations. Mutat. Res.650175-180.
.

Yakymenko, I., Burlaka, A., Tsybulin, I., et al. (2018). Oxidative and mutagenic effects of low intensity GSM 1800 MHz microwave radiation. Exp. Oncol. 40:282-287.

Yan, J.G., Agresti, M., Zhang, L.L., et al. (2008). Upregulation of specific mRNA levels in rat brain after cell phone exposure. Electromagn. Biol. Med. 27:147-154.

Yao, K., Wang, K.J., Sun, Z.H., et al. (2004). Low power microwave radiation inhibits the proliferation of rabbit lens epithelial cells by upregulating P27Kip1 expression. Mol. Vis. 10:138-143.

Yao, K., Wu, W., Wang, K., et al. (2008). Electromagnetic noise inhibits radiofrequency radiation-induced DNA damage and reactive oxygen species increase in human lens epithelial cells. Mol. Vis. 14:964-969.

Ye, W., Wang, F., Zhang, W., et al. (2016). Effect of mobile phone radiation on cardiovascular development of chick embryo. Anat. Histol. Embryol. 45:197-208.

Yildirim, M.S., Yildirim, A., Zamani, A.G., et al. (2010). Effect of mobile phone station on micronucleus frequency and chromosomal aberrations in human blood cells. Genet. Couns. 21:243-251.

Zalata, A., El-Samanoudy, A. Z., Shaalan, D., et al. (2015). In vitro effect of cell phone radiation on motility, DNA fragmentation and clusterin gene expression of sperm. Int. J. Fertil. Steril. 9:129-136.

Zeni, O., Schiavoni, A. S., Sannino, A., et al. (2003). Lack of genotoxic effects (micronucleus induction) in human lymphocytes exposed jn vitro to 900 MHz electromagnetic fields. Radiat. Res. 160:152-158.

Zeni, O., Romano, M., Perrotta, A., et al. (2005). Evaluation of genotoxic effects in human peripheral blood leukocytes following an acute in vitro exposure to 900 MHz radiofrequency fields. Bioelectromagnetics. 26:258-265.

Zeni, O., Gallaerano, G.P., Perrotta, A., et al. (2007). Cytogenetic observations in human peripheral blood leukocytes following in vitro exposure to THz radiation: A pilot study. Health Phy. 92: 349-357.

Zeni, O., Schiavoni, A., Perrotta, A., et al. (2008). Evaluation of genotoxic effects in human leukocytes after in vitro exposure to 1950 MHz UMTS radiofrequency field. Bioelectromagnetics 29:177-184.

Zeni, O., Sannino, A., Romeo, S., et al. (2012a). Induction of an adaptive response in human blood lymphocytes exposed to radiofrequency fields: Influence of the universal mobile telecommunication system (UMTS) signal and the specific absorption rate. Mutat. Res. 747: 29-35.

Zeni, O., Sannino, A., Sarti, M., et al. (2012b). Radiofrequency radiation at 1950 MHz (UMTS) does not affect key cellular endpoints in neuron-like PC12 Cells. Bioelectromagnetics. 33:497-507.

Zhang, D.Y., Xu , Z.P., Chiang, H., et al. (2006). [Effects of GSM 1800 MHz radiofrequency electromagnetic fields on DNA damage in Chinese hamster lung cells.] Zhonghua Yu Fang Yi XueZaZhi 40:149-152. [Article in Chinese]

Zhang, M.B., He, J.L., Jin, L.F., et al. (2002). Study of low-intensity 2450-MHz microwave exposure enhancing the genotoxic effects of mitomycin C using micronucleus test and comet assay in vitro. Biomed. Environ. Sci. 15:283-290.

Zhang, S.Z., Yao, G.D., Lu, D.Q., et al. (2008). [Effect of 1.8 GHz radiofrequency electromagnetic fields on gene expression of rat neurons]. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 26:449-452. [Article in Chinese]

Zhao, J., Hu, E., Shang, S., et al. (2020). Study of the effects of 3.1 THz radiation on the expression of recombinant red fluorescent protein (RFP) in E. coli. Biomed Opt Express. 11(7):3890-3899.

Zhao, R., Zhang, S., Xu, Z., et al. (2007). Studying gene expression profile of rat neuron exposed to 1800 MHz radiofrequency electromagnetic fields with cDNA microassay. Toxicology 235:167-175.

Zhao, T.Y., Zou, S.P., Knapp, P.E. (2007). Exposure to cell phone radiation up-regulates apoptosis genes in primary cultures of neurons and astrocytes. Neurosci. Lett. 412:34-38.

Zhijian, C., Xiaoxue, L., Yezhen, L., et al. (2009). Influence of 1.8-GHz (GSM) radiofrequency radiation (RFR) on DNA damage and repair induced by X-rays in human leukocytes in vitro. Mutat. Res. 677:100-104.

Zhijian, C., Xiaoxue, L., Yezhen, L., et al. (2010). Impact of 1.8-GHz radiofrequency radiation (RFR) on DNA damage and repair induced by doxorubicin in human B-cell lymphoblastoid cells. Mutat. Res. 695:16-21.

Ziemann, C., Brockmeyer, H., Reddy, S.B., et al. (2009). Absence of genotoxic potential of 902 MHz (GSM) and 1747 MHz (DCS) wireless communication signals: In vivo two-year bioassay in B6C3F1 mice. Int. J. Radiat. Biol. 85:454-464.

Zong, C., Ji, Y., He, Q., Zhu, S., et al. (2015). Adaptive response in mice exposed to 900 MHz radiofrequency fields: Bleomycin-induced DNA and oxidative damage/repair. Int. J. Radiat. Biol. 91: 270-276.

Zothansiama, Zosangzuali, M., Lalramdinpuii, M., et al. (2017). Impact of radiofrequency radiation on DNA damage and antioxidants in peripheral blood lymphocytes of humans residing in the vicinity of mobile phone base stations. Electromagn. Biol. Med. 36:295-305.

Zotti-Martelli, L., Peccatori, M., Scarpato, R., et al. (2000). Induction of micronuclei in human lymphocytes exposed in vitro to microwave radiation. Mutat. Res. 472:51-58.

Zotti-Martelli, L., Peccatori, M., Maggini, V., et al. (2005). Individual responsiveness to induction of micronuclei in human lymphocytes after exposure in vitro to 1800-MHz microwave radiation. Mutat. Res. 582:42-52.

Zuo, W.Q,, Hu, Y.J., Yang, Y., et al. (2015). Sensitivity of spiral ganglion neurons to damage caused by mobile phone electromagnetic radiation will increase in lipopolysaccharide-induced inflammation in vitro model. J. Neuroinflammation. 12(1):105.

Supplement 2

Genetic effects of static and ELF EMF (*study with no effect observed) Study reported effect =168 (79%); study reported no effect = 45 (21%). (literature up to January 2021)

	Exposure conditions	Results
Agliassa et al. (2018)	Arabidopsis thaliana (thale cress) exposed to 0.00004 mT static magnetic field for 38 days after sowing.	Changes in gene expression in leaf and floral meristem (cryptochrome-related gene involved); delayed flowering time and a significant reduction of leaf area index and flowering stem length, with respect to controls under geomagnetic field.
Ahuja et al. (1999)	Human peripheral blood samples exposed to 50 Hz EMF at 2, 3, 5, 7, or 10 mT	Increased DNA single strand breaks (Comet assay) in lymphocytes.(Damage levels higher in female than in male subjects.)
*Albert et al. (2009)	Human subjects exposed to exposed to 60-Hz magnetic field at 0.2 mT for 4 h	No significant effect on DNA single strand breaks (Comet assay) and micronucleus frequency in lymphocytes.
Alcaraz et al. (2013)	Swiss mice exposed to 50-Hz magnetic field at 0.2 mT for 7, 14, 21, or 28 days	Increased micronucleus frequency in bone marrow. Effect not affected by antioxidants.
Al-Huqail and Abdelhaliem (2015)	Maize seedlings exposed to 50-Hz electric field at 6 kV/m for 1, 3, or 5 days	Increased DNA single strand breaks (comet assay)
Amara et al. (2006)	Male rats exposed to a static magnetic field at 128 mT, I h/day for 30 days	Increased 8-oxo-dG concentration and oxidative damage in testis.
Amara et al. (2007a)	Human monocytic leukemia THP-1 cells exposed to static magnetic field at 250 mT for 1, 2, or 3 h	Lower level of DNA single strand breaks (Comet assay) at 3 h of exposure, no effect on oxidative damages and enzymes and oxidative DNA damage.
Amara et al. (2007b)	Rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days	Increased 8-oxo-7,8-dihydro-2'-desoxyguanosine in kidney but not in liver. Also decreased anti-oxidative enzymes and increased lipid peroxidation. Zinc supplementation attenuated DNA oxidation induced by static magnetic field in kidney to the control level.

*Amara et al. (2009)	Rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days	No significant effect on 8-oxo-7,8-dihydro-2'-deoxyguanosine in frontal cortex and oxidative stress induced. However, there was an increase in metallothioneins level which might have protected DNA from oxidative damage.
*Amara et al. (2011)	Rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days, also treated with cadmium (Cd)	Magnetic field had no interaction on Cd-induced increase in 8-oxo-7,8-dihydro-2-desoxyguanosine in the frontal cortex and hippocampus. However, static magnetic field enhanced Cd-induced increase in oxidative damage in the rat brain.
Arruda-Neto et al. (2009)	*Microcystis panniformis*, the eukaryote *Candida albicans* and human MRC5 lung cells exposed to gamma radiation and then to static electric field for 2-20 h at 20- 1250 V/cm	Static electric field caused suppression of DNA repair in *C. albicans*. It decreased cell growth in *M. panniformis* when compared with gamma radiation alone. The electric field increased number of nuclei with γ-H2AX foci in the irradiated MRC5 cells. Electric field interferes mostly in the DNA repair mechanisms.
Ashta et al. (2020)	Human glioblastoma cells (A172) exposed to 10 Hz or static magnetic field at 5 mT, up to 96 h	Increased p52 gene expression, cytotoxicty and free radical formation; effects enhanced by Temozolomide.
Back et al. (2019)	Mouse embryonic stem cells exposed to hypomagnetic field (<0.005 mT) up to 12 days	Induced abnormal DNA methylation through the dysregulation of DNA methyltransferase3b (Dnmt3b) expression, eventually resulting in incomplete DNA methylation during differentiation.
Bagheri Hosseinabadi et al. (2019)	Blood samples from 102 thermal power plant workers as the exposure group and 136 subjects as the unexposed group.	Increased DNA single strand breaks (Comet assay) in lymphocytes of exposed subjects.
Bagheri Hosseinabadi et al. (2020)	Blood samples from thermal power plant workers; mean levels of exposure to ELF magnetic and electric fields were .0165 mT (±6.46) and 22.5 V/m (±5.38), respectively,	DNA single strand breaks (Comet assay) in lymphocytes decreased by antioxidants.

Balamuralikrishnan et al. (2012)	Blood from electrical workers exposed to ELF EMF occupationally	Increased chromosome aberrations and micronucleus in lymphocytes.
Baraúna et al. (2015)	Chromobacterium violaceum bacteria cultures exposed to ELF-EMFfor 7 h at 0.00066 mT	Five differentially expressed proteins detected including the DNA-binding stress protein, which may help to prevent physical damage to DNA.
Belyaev et al. (2005)	Human lymphocytes exposed to 50 Hz magnetic field at 0.015 mT (peak) for 2 h (measurements made at 24 and 48 h after exposure).	Induced chromatin conformation changes and decreased background 53BP1 (protein co-localized with DNA double strand breaks and involves in DNA damage signaling pathway.)
Bertea et al. (2015)	Arabidopsis thaliana (thale cress) exposed to artificially reversed geomagnetic field conditions for 10 days at .0419 mT	Significant effects on plant growth and gene expression observed. This supports the hypothesis that GMF reversal contributes to inducing changes in plant development that might justify a higher selective pressure, eventually leading to plant evolution.
Borhani et al. (2011)	Female NMRI mice exposed to a 50-Hz EMF at 0.5 mT for 4 h/day, 6 days/week for 2 weeks. Mated on day 8 after exposure, on day 4, blastocysts were obtained by flushing the uterus horns.	DNA fragmentation index increased and decrease in blastocytes in exposed group.
*Brix t al. (2020)	Young volunteers allocated to three study arms were exposed to [^{18}F] fluoro-D-glucose alone, to a 3-T SMF alone or to both combined over 60 min at a PET/CT or a PET/MRI system.	No significant change in lymphocyte DNA double strand breaks (γH2AX) to static magnetic field or interaction with [^{18}F] fluoro-D-glucose.
Buddak et al. (2012)	Murine AT478 carcinoma cells cultured with cisplatin exposed to 50-Hz EMF for 16 min at 1 mT	Exposure to ELF-EMF alone resulted in an increase in DNA single strand breaks (Comet assay) compared to control cells. ELF-EMF lessened the effects of oxidative stress and DNA damage that were induced by cisplatin;

		however, ELF-EMF alone was a mild oxidative stressor and DNA damage inducer. The addition of ELF-EMF exposure to cisplatin treatment resulted in decreased ROS levels and antioxidant enzyme activity.
Burgos-Molina et al (2020)	DNA double strand breaks were induced in Saccharomyces cerevisiae yeast and exposed to a 50-Hz magnetic field for 21 days at 2.45 mT	Long-term magnetic field exposure increased the DNA repair activity.
Calabro et al. (2011)	Human neuronal-like cells exposed to static (2 mT) and 50 Hz (1 mT) for 3 h.	Fourier self deconvolution spectroscopic analysis showed alteration in DNA/RNA and increased beta-sheet.
Calabro et al. (2020)	Human Neuronal-like cells and roots of Allium sativum and Vicia faba exposed to a static and 50 Hz magnetic fields at intensities ranging from 1 mT to 0.8 T	Exposure to both low- and high-intensity magnetic fields in typical human and plant cells induces uncoiling and unpackaging of chromatin constituents, followed by chromosome alignment towards the direction of applied magnetic field, providing further demonstration that magnetic fields can induce the orientation of organic macromolecules even at low-intensity values.
*Cantoni et al.(1996)	Cultured mammalian cells exposed to 50 Hz electric (0.2 - 20 kV/m), magnetic (0.0002-0.2 mT), or combined electric and magnetic fields.	Repair of DNA single strand breaks (Comet assay) induced by the carcinogens methylmethane sulphonate (MMS), chromate, and 254 U.V. radiation not affected by ELF EMF exposure.
Celikler et al. (2009)	Workers from transfrormer and distribution line stations. The electric field was in the range from 130–8310 V/m and from 300–15,000 V/m, the magnetic field was between 0.5 and 1.7 A/m and 0.25–17 A/m around and inside transformer buildings. Average time of exposure was 19 years.	Increased chromosomal aberrations and micronucleus in peripheral lymphocytes. The frequency of chromosomal aberration in exposed groups correlated with the years of exposure.

*Cellini et al. (2008)	Escherichia coli ATCC 700926 exposed to 50-Hz EMF (0.1, 0.5, 1.0 mT); 20-120 min	No changes among DNA finger-printings. Other measurements indicates 50 Hz EMF acts as a stressing factor on bacteria
*Chahal et al. (1993)	Escherichia coli strain AB1157 exposed to a frequency of 1 Hz with field strengths of 1 or 3 kV m-1	Low frequency electromagnetic fields do not increase spontaneous mutation, induce DNA repair or increase the mutagenic effects of UV or mitomycin C.
Chen GD et al. (2008)	Human MCF-7 breast cancer cells exposed to a 50-Hz magnetic fields for 24 h at 0.4 mT	Identified three 50 Hz MF responsive genes in MCF-7 cells.
*Chen G et al. (2012)	*Saccharomyces cerevisiae* yeast cells exposed to a 50-Hz magnetic field at 0.4 mT for 6 h	Yeast cells did not alter gene expression in response to 50 Hz magnetic field.
Chan J. et al. (2020)	Human choriocarcinoma cells exposed to DC electric field (150 mV/mm) for 8 h	Increased gene expressions of ErbB and HIF-1 signaling pathways involved in cell migration/motility, cell cycle progression and proliferation.
Chen WF et al. (2010)	Human myelogenous leukemia K562 cells exposed to static magnetic field at 8.8 mT with or without cisplatin	Static magnetic field exposure induced DNA to become thicker than controls, and enhanced DNA breakage (Comet assay) induced by cisplatin.
Cho S et al. (2014)	Human lymphocytes exposed to 60-Hz EMF at 0.8 mT for 12-72 h with or without gadolinium.	ELF-EMF increased cell death, micronucleus frequency, DNA single strand break (Comet assay), and apoptosis induced by gadolinium.
Cho YH et al. (2007)	Human fibroblasts exposed to 60-HZ EMF at 0.8 mT plus bleomycin for 28, 88, and 240 h	The co-exposure of cells to bleomycin and EMF led to a significant increase in the frequencies of micronucleus and aneuploidy compared to the cells treated with bleomycin alone.
Chow and Tung (2000a)	Escherichia coli strain XL-1 Blue exposed a 50-HZ magnetic field at 0.1-1.2 mT for 1 h	This result was indicative that the efficiency of DNA repair had been improved. The improvement was found to be mediated by the induced overproduction of heat shock proteins DnaK/J (Hsp70/40).
Chow and Tung (2000b)	Escherichia coli strain XL-1 Blue (transformed by plasmid pUC8 that had been mutagenized by	Improved efficiency of DNA repair mediated by the induced overproduction of heat shock proteins DnaK/J (Hsp70/40).

	hydroxylamine exposed a 50-HZ magnetic field at 0.1-1.2 mT for 1 h	
Collard et al. (2013)	Epidermis cultures harvested from human abdominoplasty exposed to ELF electric fields (a biphasic, asymmetric, charge-balanced current stimuli, with a repetition frequency of4 0 Hz modulated by a fundamental frequency of 0.125 Hz.The exposure was repeated during 4 s followed by a 4 s break for 40 min/day for 11 days	Observed a significant change in genes expression after 4 days and change in expression in another group of genes at day 4 and 7. Genes are involved in cell proliferation or differentiation, mitosis, cell cycle or in the DNA replication transcription and translation.
Consales et al. (2018)	Human SH-SY5Y neuroblastoma cells and mouse primary cortical neurons exposed to a 50-Hz magnetic field at 1 mT for 4-72 h	Expressions of microRNA miR-34b/c that caused mitochondrial oxidative stress, also altered α-synuclein expression involved in synaptic functions. These effects may be related to neuro-degeneration.
Cuccurazzu et al. (2010)	Mice exposed to 50 Hz EMF at 1 mT for 1-7 h/day for 7 days	Induced increases in the transcription of pro-neuronal genes (Mash1, NeuroD2, Hes1) and genes encoding Ca(v)1.2 channel α(1C) subunits in the hippocampus. Generation of new granule cells in the dentate gyrus.
Del Re et al. (2006)	Escherichia coli exposed to sinusoidal or pulsed square wave 50-Hz magnetic field at 1 mT for 40 min	Sinusoidal magnetic field exposure induced a significantly higher level of DnaK and GroEL, whereas a lower level was observed after pulsed magnetic field exposure. When bacterial cells were exposed to heat shock (HS) after ELF-magnetic field exposure: again sinusoidal and pulsed fields resulted in an increase and in a reduction of HSP amount.
Delimaris et al. (2006)	Human lymphocytes exposed to 50-Hz pulsed electric fields (10-Hz carrier frequency) at 4 x 10^5 V/m for 120 min	Increased in DNA single strand breaks (Comet assay).
Di Campli et al. (2010)	Helicobacter pylori biofilm exposed to 50-	No changes in DNA patterns were recorded, whereas a modulation in amiA gene

	Hz EMF at 1 mT for 2 days	expression was detected; phenotypic changes induced.
Dominici et al. (2011)	Lymphocytes from welders (average magnetic field exposure from personal dosimeters 0.00781 mT (general environmental level 0.00003 mT)	Higher micronucleus frequency correlated with EMF exposure levels; decreased in sister chromatid exchange frequency.
Dong et al. (2019)	Human pre-osteoclast RAW264.7 cells exposed to a 16 T static magnetic field for 2-4 days	HiSMF markedly blocked the expression of osteoclast-associated transcription factors and osteoclast marker genes and inhibited iron absorption and iron storage-related protein expression. Mitochondrial concentration and oxidative stress levels in osteoclasts were decreased under magnetic field exposure.
Du et al. (2008)	Cultured human lens epithelial cells exposed 50-Hz magnetic field at 0.4 mT for 2 h, 6 h, 12 h, 24 h and 48 h	Increased DNA doubled strand breaks (γ–H2AX foci) after 24 h exposure.
Duan et al. (2015)	A mouse spermatocyte-derived GC-2 cell line intermittently (5 min on and 10 min off) exposed to a 50 Hz EMF at 1, 2 or 3 mT for 24 h	Increased DNA strand breaks (Comet assay and γ–H2AX foci) at 3 mT exposure.
El-Bialy and Rageh (2013)	Mice with Ehrlich tumors exposed to a 50-Hz magnetic field 1 h/day for 2 weeks at 10 mT	Exposure cause DNA single strand breaks (Comet assay) in tumor cells and increased micronucleus frequency in bone marrow cells. ELF-MF enhanced the effects of cisplatin.
Erdal et al. (2007)	Wistar rats exposed to 50 Hz magnetic field at 1 mT for 4 h or 4h/day for 45 days	Micronucleus frequency higher in bone marrow cells of long-term exposed rat. Mitotic index decreased in both exposed groups.
*Fairbairn and O'Neill (1994)	Human cells exposed to ELF-EMF	No significant effect on DNA single strand breaks (Comet assay)
Fan et al. (2005)	Rat bone marrow derived-mesenchymal stem cells exposed to a 50-Hz EMF at 1 mT for 4 h/day for 3 days	Increased cell viability, DNA synthesis and proportion of cells in S phase and up-regulated the expressions of hematopoietic growth factors.

Fan et al. (2018)	*Enterococcus faecalis* (isolated from dental infection) exposed to a static magnetic field at 170 mT for 24 or 72 h.	Static magnetic field up-regulated the expression of stress gene (dnaK) and virulence genes (efaA and ace). Synergistic with alkaline pH induced by calcium hydroxide (a major dental antimicrobial) in antimicrobial action and up-regulation of stress and virulence genes.
Fatigoni et al. (2005)	Tradescantia (a perennial wildflower) exposed to a 50-Hz magnetic field at 1 mT for 6 or 24 h	Caused a time-dependent increase in micronucleus frequency.
Fedrowitz and Loscher (2012)	Female F344 and Lewis rats exposed to a 50-Hz magnetic field at 0.1 mT 24 h/day for two weeks	F344 breast tissue showed alterations in gene expression, which were absent in Lewis rats, particularly, α-amylase, a stress marker.
*Fiorani et al. (1992)	Human immortalized myelogenous leukemia K562 cells exposed to50-Hz electric (0.2-20 kV/m) or magnetic (0.0002-.2 mT) or combination of electric and magnetic fields, for 24 h	No detectable DNA lesions (measured by filter elution technique).
Focke et al. (2010)	Human fibroblasts exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 15 h	Increased DNA single strand breaks (Comet assay) caused by magnetic and not electric field, No oxidative DNA damage. Could be caused by minor disturbances in S-phase processes and occasional triggering of apoptosis rather than by the generation of DNA damage.
*Frahm et al. (2006)	Mouse macrophages exposed to a 50-Hz magnetic field for 45 min, 12, 24, or 48 h; 0.05 – 1 mT	No genotoxic effect (micronucleus formation); increased phagocytic activity, free radicals, and IL-1 beta production.
*Frazier et al. (1990)	Human lymphocytes induced with DNA damage with ionizing radiation were exposed to 60-Hz magnetic field at 1 mT, electric field at 1 or 20V/m, or combinations of magnetic and electric	EMF exposure did not affect repair of DNA single strand breaks (Comet assay).

	fields (0.2 V/m and 0.05 mT, 6 V/m and 0.6 mT, or 20 V/m and 1 mT) up to 180 min	
Frisch et al. (2013)	Transfected rat primary fibroblast (RAT1) cells exposed to 10 Hz electric fields at 20-500 V/m for 2 h	Induced HSP70 heat shock expression, with peak responses obtained at 8 h following exposure.
Giorgi et al. (2011)	Two Escherichia coli model systems were exposed to sinusoidal or pulsed-square wave magnetic fields of various frequencies (20, 50, 75 Hz) and for different exposure times (15 and 90 min). at 1 mT	ELF-MF exposure affected transposition activity (transposon (Tn) mobility) and the effects critically depended on the wave shape of the field, but not on the frequency and the exposure time.
*Giorgi et al. (2014)	Human neuroblastoma BE(2)C cells treated with hydrogen peroxide exposed to 50-Hz pulsed magnetic field at 1 mT for 1-72 h	Pulsed magnetic field exposure did not interfere with genotoxicity (DNA double strand breaks measured by γ-H2AX foci) and cytotoxicity induced by oxidative stress.
Giorgi et al. (2017)	Human neural cells (BE(2)C) exposed to pulsed 50-Hz magnetic field at 1 mT for 24 and 48 h in combination with oxidative stress (hydrogen peroxide)	Pulsed magnetic field and oxidative stress induced weak decreases and increases of DNA methylation levels; combined exposure led to significant transient decrease of DNA methylation levels at different genome loci.
Heredia-Rojas et al. (2010)	Human non-small cell lung cancer cells (INER-37) and mouse lymphoma cells (RMA E7) (transfected with a plasmid with hsp70 expression when exposed to magnetic field and contains the reporter for the luciferases gene) exposed to a 60-Hz magnetic field at 0.008 and 0.00008 mT for 20 min.	An increased in luciferase gene expression was observed in INER-37 cells exposed to magnetic field, but similar exposure had no effect on the RMA E7 cell line.

Hong et al. (2005)	Mice exposed to a 50-Hz EMF at 0.2 or 6.4 mT for 4 weeks	EMF induced DNA single strand breaks (Comet assay) in testicular cells and chromatin condensation in spermatozoa.
*Huwiter et al. (2012)	Escherichia coli K-12 MG1655 exposed at 50-Hz magnetic fields generated by three signal types (sinusoidal continuous, sinusoidal intermittent, and power line intermittent) at 1 mT for 8 min, 2.5 h, or 15 h	No effect on transcription of 4358 gene studied.
Ivancsits et al. (2002)	Human diploid fibroblasts exposed to continuous or intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 24 h	Intermittent exposure induced DNA single and double strand breaks (Comet assay).
Ivancsits et al. (2003a)	Human diploid fibroblasts exposed to intermittent (5 min ON/10 min OFF)50-Hz EMF at 0.02- 1 mT for 1-24 h	DNA Single and double strand breaks (Comet assay) observed at 0.035 mT at 15 h; recovered within 9 h.
Ivancsits et al.(2003b)	Fibroblasts from human subjects of different ages exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 1-24 h	Increased DNA Single and double strand breaks (Comet assay) at 15 h; more pronounced in cells from older donors
Ivancsits et al. (2005)	Various cell types exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 1-24 h	Effects on DNA Single and double strand breaks (Comet assay) showed three responder (human fibroblasts, human melanocytes, rat granulosa cells) and three non-responder cell types (human lymphocytes, human monocytes, human skeletal muscle cells).
Jajte et al. (2001)	Rat peripheral blood lymphocytes exposed to a 50-Hz magnetic field at 7 mT for 3 h	Increased DNA single strand breaks (Comet assay) in cells treated with ferrous chloride; melatonin attenuated the effect.
*Jin H. et al. (2015)	Non-tumorigenic human lung epithelial L132 cells exposed to a 60-Hz magnetic field at 1 or 2 mT for 9 h	No G2/M arrest or aneuploidy nor interaction with gamma radiation and H_2O_2

*Jin et al, (2012)	Mouse embryonic fibroblast NIH3T3 cells and human lung fibroblast WI-38 cells exposed to a 60 Hz magnetic field at 1 mT for 4 h	No significant effect on micronucleus frequency and interaction with ionizing radiation, H_2O_2, or c-Myc activation.
*Jin et al, (2014)	NIH3T3 mouse fibroblast cells, WI-38 human lung fibroblast cells, L132 human lung epithelial cells, and MCF10A human mammary gland epithelial cells exposed to a 60-Hz magnetic field at 1 mT for 4 or 16 h	No significant effect on DMA single strand breaks (Comet assay), and interaction with ionizing radiation, H_2O_2, or c-Myc activation.
Jin et al. (2019)	Arabidopsis young seedlings exposed to a static magnetic field at 600 mT	Increased auxin (a plant growth hormone) from expression of PIN3 and AUX1 genes in root tips; cryptochromes (cry1 and cry 2) are also involved. Root growth enhanced. Effects occurred when static magnetic field was parallel and perpendicular not opposite, to geomagnetic field.
Jouni et al. (2012)	*Vicia faba* (broad bean) culture in soil with high background radioactivity and exposed to static magnetic field at 15 mT for 8h/day for 8 days	Increased chromosomal aberration and DNA damage in root tip cells with lowering of antioxidant defense; soil radioactivity enhanced the effects.
Kesari et al. (2015)	Human neuroblastoma SH-SY5Y cells exposed to a 50-Hz 100 μT magnetic field for 24 h.	Micronucleus formation was observed at 15 and 30 days postexposure. Effect not related to oxidative changes.
Kesari et al. (2016)	Human glioblastoma SH-SY5Y and rat glioma C6 cells exposed to a 50-Hz magnetic field at 0.01 and 0.03 mT for 24 h with menadione as a cofactor	Micronuclei were significantly increased in SH-SY5Y cells at 0.03 mT Increased cytosolic and mitochondrial superoxide levels were observed in C6 cells. The results indicate that the threshold for biological effects of ELF magnetic field is 0.01 mT or less.
Khalil and Qassem (1991)	Human lymphocytes exposed to a pulsing 50-Hz EMF at 1.05 mT for 24, 48 and 72 h	Suppression of mitotic activity and a higher incidence of chromosomal aberrations. Delay in cell proliferation index and an increase in the baseline frequency of sister-chromatid

		exchanges occurred only after 72 h f exposure.
Ki et al. (2020)	Human hair follicle dermal papilla cells, a type of cells involved in hair growth, exposed to a 70 Hz EMF at intensities ranging from 0.5 to 10 mT over four days	Increased the expression of anagen-related molecules, including collagen IV, laminin, ALP, and versican, and increased β-catenin and Wnt3α expression and GSK-3β/ERK/Akt phosphorylation. Cell proliferation enhanced.
Kim HJ. et al. (2013)	Bone marrow derived mesenchymal stem cells (BM-MSCs) were subjected to a 50-Hz EMF	Increased levels of neuronal differentiation marker (MAP2), while early neuronal marker (Nestin) was down-regulated; increased differentially expression of 8 proteins; notably, a significantly increased expression of the ferritin light chain.
Kim J. et al. (2010)	IMR90 (human lung fibroblast) primary cells and HeLa (human cervical carcinoma) cells exposed to a time-varying (rotating) 60-Hz magnetic field at 6 mT for 60 min or 30 min/day for 3 days	Repeated exposure showed DNA double strand breaks (γ-H2AX foci) and decreased cell viability and increased apoptosis through p38 activation.
Kim J. et al. (2012)	Human primary fibroblast and cervical cancer cells exposed to a time-varying 60-Hz magnetic field at 7 mT for 10-60 min	DNA double strand breaks (γ-H2AX foci and Comet assay) detected (intracellular reactive oxygen species not affected).
Kimsa-Dudek et al. (2018)	Normal human dermal fibroblasts exposed to static magnetic field at 0.65 T for 24 h and sodium fluoride	Static magnetic field attenuated expression of antioxidant defense genes (SOD1, PLK3, CLN8, XPA, HAO1) induced by sodium fluoride.
Kimsa-Dudek et al. (2020)	Normal human dermal fibroblasts exposed to static magnetic field at 0.45, 0.55 and 0.5 T for 24 h and sodium fluoride	The field reduced fluoride-induced apoptosis and affected apoptosis gene expression; reduced fluoride-induced increases in reactive oxygen species and lipid peroxidation and decrease in antioxidant enzymes.
Kimura et al. (2008)	*Caenorhabditis elegans* exposed to 2, 3, or 5 T static magnetic field for 4-24 h	Genes involved in motor activity, actin binding, cell adhesion, and cuticles are transiently and specifically induced; also hsp (heat shock protein) 12 and 16 family genes.

Kindzelskii and Petty (2000)	Human neutrophils exposed to pulsed square-wave (20 msec) DC electric field at 0.2 V/m for 30, 45, 60 min	Increased DNA single strand breaks (Comet assay).
*Kirschenlohr et al. (2012)	Male human subjects exposed to 50-Hz EMF at 0.062 mT for 2 h (Exposure repeated two more times.)	No genes or gene sets in blood samples showed consistent response profiles to repeated ELF-EMF exposures (including immediate early genes, stress response, cell proliferation and apoptotic genes).
Koyama et al. (2008)	Human glioma A172 cells exposed to a 60-Hz magnetic field at 5 mT for2, 4, 8, 16, 24 h	The number of apurinic/apyrimidinic sites induced by gentoxic agents methyl methane sulfonate and H_2O_2 was enhanced by exposure to ELF magnetic fields. (Apurinic/apyrimidinic sites are common DNA lesions arise from spontaneous depurination or by base excision repair of oxidized, deaminated or alkylated bases.)
Kubinyi et al. (2010)	Human lymphocytes exposed to an inhomogeneous static magnetic field with a lateral magnetic flux density gradient of 47.7, 1.2, or 0.3 T/m by 10 mm lateral periodicity, or a homogeneous SMF of 159.2 mT magnetic flux density for a time period of 0.5 min, 1, 2, 4, 6, 18, 20, or 24 h.	Increased DNA single strand breaks (Comet assay); affected DNA repair induced by gamma ray when exposure occurred after ionizing radiation treatment.
Kumari et al. (2017)	Mice exposed continuously for 5 weeks to 7.5 KHz MF at 120 μT	Expression of the pro-inflammatory cytokine tumor necrosis factor alpha mRNA was significantly increased in the hippocampal region; impairment of memory observed.
*Lacy-Hulbert et al. (1995)	Human leukemic cells (HL60) exposed to a 60-Hz EMF for 20 min at 0.00057, 0.0057, or 0.057 mT	No change in MYC and beta-actin gene expression observed.
Lagroye and Poncy (1997)	Rat tracheal epithelial cell lines were first exposed to gamma rays and then cultured in a 50-Hz magnetic field at 0.1 mT for 24 h.	Increased binucleated cells with micronuclei in cells exposed to gamma rays and magnetic field, compared with gamma irradiation alone. Magnetic field alone had no significant effect on micronucleus frequency.

Lai and Singh (1997a)	Male Sprague-Dawley rats exposed to a 60-Hz magnetic field at 0.1, 0.25, or 0.5 mT for 2 h	Increased DNA single and double strand break (Comet assay) in brain cells.
Lai and Singh (1997b)	Male Sprague-Dawley rats exposed to a 60-Hz magnetic field at 0.5 mT for 2 h	Increased DNA single and double strand break (Comet assay) in brain cells. Effects blocked by melatonin and a spin-trap compound.
Lai and Singh (2004)	Male Sprague-Dawley rats exposed to a 60-Hz magnetic field at 0.01 mT for 24 or 48 h	Increased DNA single and double strand break (Comet assay) in brain cells. More effect with 48-h than 24-h exposure. Effects blocked by Trolox (a vitamin E analog) and 7-nitroindazole (a nitric oxide synthase inhibitor).
Laramee et al. (2014)	Transfected rat primary fibroblast (RAT1) cells exposed to static magnetic fields of 1 to 440 mT for 16, 24, or 48 h starting at 24 and 48 h post transfection	Induction of heat shock protein (HSP70) expression showed a dependency on flux density, exposure duration, and start time post transfection.
Lee et al. (2010)	*Caenorhabditis elegans* exposed to exposed to a static magnetic field at 200 mT	Expression of genes involved in development and aging. Accelerated development and shorten lifespan.
Lee et al. (2016)	MCF10A, MCF7, Jurkat, and NIH3T3 cells exposed to a 60 Hz magnetic field at 1 mT for 4 or 16 h	MCF10A and MCF7 cells showed consistent and significant decreases in cell number, cell viability, and DNA synthesis rates (cell cycle delay), whereas Jurkat and NIH3T3 cells showed no effect. MCF7 cells (2 mT for 16 h) showed up-regulation of PMAIP1 gene (involved in apoptosis).
Lee et al. (2011)	Human lymphocytes exposed to EMF generated during MRI scanning (clinical routine brain examination protocols: three-channel head coil) for 22, 45, 67, and 89 min	Significant increases in DNA single-strand breaks (Comet assay), and frequencies of both chromosome aberrations and micronuclei in a time-dependent manner.
Leone et al. (2014)	Neural stem cells isolated from hippocampi of newborn mice exposed to a 50-Hz EMF at 1 mT for 10 days	Histone acetylation-related chromatin remodeling leading to enhanced proliferation and neuronal differentiation.

Li and Chow (2001)	E. coli XL-1 Blue transformed with plasmid pUC18 and DNA samples exposed to a 50-Hz magnetic field at 1.2 mT for 1-5 h, with heat shock response suppressed	Without the protection of the heat shock response, magnetic field exposure induced DNA degradation, which could be attenuated by the presence of an antioxidant,
*Li L. et al (2015)	Workers from a power supply bureau (inspection workers vs. logistic staff); The average time-weighted average was 0.0073 mT (0.00156-0.02633 mT) and the subjects were subgrouped by cumulative ELF-magnetic field exposure dose: low (<0.0156 mT), middle (0.0156-0.073 mT) and high (> 0.073 mT)	No significant effect on the frequency of micronucleus lymphocytes or micronuclei frequency; no changes in antioxidant enzymes and cellular oxidative damage.
Li SS et al. (2013)	Male *Drosophila melanogaster* fruit flies exposed to a 50-HZ EMF at 3 mT for 72 or 312 h	Different sets of genes were up- and down-regulated after short- or long-term exposure. Short-term exposure may decrease the reproductive ability of males, whereas long-term exposures had no effect on reproductive ability.
Li Y. et al. (2014)	Fertilized embryos of zebra fish (*Danio rerio*) exposed to a 50-Hz magnetic field at 0.1 - 0.8 mT for 96 h	The transcription of apoptosis-related genes (caspase-3, caspase-9) was significantly up-regulated in exposed embryos. Delayed hatching and apoptosis observed.
Li, Y. et al. (2015)	Rat oligodendrocyte precursor cells exposed to DC electric field at 50, 100. Or 200 mV/mm for 1.5 h	Mitogen-activated protein kinase pathway that signals cell migration was significantly upregulated in cells treated with an EF of 200 mV/mm compared with control cells and downregulation of differentially expressed genes in chemotaxis.
Li Y. et al. (2019)	Dementia rats induced by streptozotocin (STZ) intracerebroventricular injection exposed to a 10 mT 20-Hz pulsed EMF,	Pulsed EMF increased expression of insulin growth factor 2 (IFG-2) in the hippocampus and improved the ability of learning and memory in STZ-treated rats.

	2 h/day, 10 days	
Lin et al. (2016)	Budding yeast exposed to a 50-Hz EMF at 6 mT for 96 h	The transcription levels of 28 genes were upregulated and those of four genes were downregulated. Exposure can upregulate the expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle, but not the glycolysis pathway.
Liu et al. (2015)	Mouse spermatocyte-derived GC-2 cell line exposed to an intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1, 2, or 3 mT for 72 h	Exposure decreased genome-wide methylation at 1 mT, but global methylation was higher at 3 mT. Expression of DNMT1 and DNMT3b (DNA methyltransferases) was decreased at 1 mT, and increased at 3 mT.
*Lopucki et al. (2005)	Cotyledons dissected from placentas obtained immediately after physiological labors exposed to a 50-Hz magnetic field at 2 or 5 mT for 3 h	No significant effect on level of 8-hydroxy-2'-deoxyguanosine in DNA (oxidative DNA damage).
Lourencini da Silva et al. (2000)	SnCl2-treated pBR322 plasmids exposed to a 3400Hz square-wave EMF with peak power of 4V for 2 h	An EMF-dependent potentiation of DNA scission (i.e. the appearance of relaxed plasmids) was observed. The results indicate that the EMF, in the presence of a transition metal, is capable of causing DNA damage.
*Luceri et al. (2005)	Human peripheral blood lymphocytes and DBY747 *Saccharomyces cerevisiae* exposed to a50-Hz magnetic field at 0.001, 0.01or 0.1 mT for 18 h	No significant effects on DNA single strand breaks (Comet assay), oxidated DNA base, and gene expression.
Lupke et al (2006)	Human umbilical cord blood-derived monocytes exposed to a 50-Hz magnetic field at 1 mT for 45 min	Alteration of 986 genes involved in metabolism, cellular physiological processes, signal transduction and immune response.
Luukkonen et al. (2011)	Human SH-SY5Y neuroblastoma cells. Exposed to a 50-Hz magnetic field at 0.1 mT for 24 hours, followed by chemical (menadione) exposure for 3 h	Magnetic field enhanced menadione-induced DNA damage, DNA repair rate, and micronucleus formation. No effects were observed after magnetic field exposure alone.

Luukkonen et al. (2014)	Human SH-SY5Y neuroblastoma cells. Exposed to a 50-Hz magnetic field at 0.1 mT for 24 hours, followed by menadione exposure for 3 h	Persistently elevated levels of micronuclei were found in the progeny of magnetic field (alone)-exposed cells at 8 and 15 days after exposure, indicating induction of genomic instability. (No magnetic field x menadione interaction effect). Magnetic field disturbed oxidative balance immediately after the exposure, which might explain the previous findings on MF altered cellular responses to menadione-induced DNA damage.
Luukkonen et al. (2017)	Human SH-SY5Y neuroblastoma cells. Exposed to a 50-Hz magnetic field at 0.1 mT for 24 hours, followed by menadione exposure for 1 or 3 h	Decreased p21 protein (a DNA damage response-related proteins) level after 1-h menadione treatment, as well as increased proportion of cells in the G1 phase and decreased proportion of S phase cells after 3-h menadione treatment. Magnetic field exposure decreased DNA single strand breaks (Comet assay) caused by I h treatment with menadione.
Ma et al. (2014)	Mouse embryonic neural stem cells exposed to a 50-Hz EMF at 2 mT for 3 days	Expression of genes regulating neuronal differentiation was altered.
Mahaki et al. (2019)	Rats exposed to a 50-Hz EMF at 0.001-2 mT for 2 h/day for 60 days	In the spleen, gene expression levels of RORα (retinoid-related orphan receptor alpha) and c-Maf (transcription factor Maf) were significantly down-regulated at 0.001 and 0.1 mT, while the expression of STAT6 (signal transducer and activator of transcription 6) was only significantly decreased at the density of 0.1 mT. No effect on thymus.
Mahmoudinasab and Saadat (2016)	Human MCF-7 cells exposed to a 50-Hz magnetic field at 0.25 and 0.5 mT (5 min ON/5 min OFF, 15 min ON/15 min OFF, or 30 min field-on continuously) for 30 min	Alterations in the *NQO1* and *NQO2* (NAD(P)H: quinone oxidoreductase) mRNA levels seen at the "5 min ON/5 min OFF" condition.
Mahmoudinasab and Saadat (2018a)	MCF-7 and SH-SY5Y cells exposed to 50-Hz EMF at 0.5 mT (15 min ON/ 15 min OFF), and treated with morphine and cisplatin.	EMF exposure could protect SH-SY5Y cells from the cytotoxicity of cisplatin and morphine, whereas it has no significant change in MCF-7 cells. Expression patterns of antioxidant genes are different in both cell lines.

Mahmoudinasab and Saadat (2018b)	SH-SY5Y cells exposed to 50-Hz EMF at 0.5 mT ("15 min ON/ 15 min OFF" and "30 min ON") for 30 min, and treated with morphine and beta-lapachone	NQO1 mRNA level decreased in the "15 min field-on/15 min field-off" condition, the expression level of NQO2 was increased. Morphine and EMF reduced the cytotoxicity of beta-lapachone.
Mahmoudinasab et al. (2016)	Human MCF-7 cells exposed to a 50-Hz magnetic field at 0.25 and 0.5 mT (5 min ON/5 min OFF, 15 min ON/15 min OFF, or 30 min field-on continuously) for 30 min	Significant changes in mRNA levels of seven antioxidant genes for "the 15 min field-on/15 min field-off condition".
Mairs et al. (2007)	UVW human glioma cells to a 50-Hz EMF at 1 mT for 12 h	Induced 0.011 mutations/locus/cell, which was equivalent to a 3.75-fold increase in mutation induction compared with unexposed controls. The field also potentiated the mutagenic capacity of gamma-irradiation.
Manzella et al. (2015)	Human dermal fibroblasts exposed to a 50 Hz magnetic field at 0.1 mT for 1 h	Changes in expression of clock genes.
Mariucci et al. (2010)	CD1 mice exposed to a 50-Hz magnetic field at 1 mT for 1 or 7 days (15 h/day)	Increased DNA single strand breaks (Comet assay) in brain areas detected immediately after 7-day exposure. No effect on HSP-70 expression.
Markkanen et al. (2008)	Murine L929 fibroblasts exposed to a 50-Hz magnetic field at 0.1 or 0.3 mT for 24 h, with or without ultraviolet B (UVB, wavelength 280-320 nm) radiation or menadione (MQ)	Pre-exposure to magnetic field can alter cellular responses to other agents, and indicate that magnetic field as low as 0.1 mT has measurable impacts on cancer-relevant cellular processes such as DNA-damage.
Mastrodonato et al. (2018)	Mice exposed to a 50 Hz, 1 mT EMF 3.5 h/day for 12 days	Increased Wnt3 (neurogenesis gene) mRNA expression and nuclear localization of its downstream target β-catenin in subventricular zone of the lateral ventricle. Mice showed enhanced olfactory memory at 30 days post-exposure.

*McNamee et al. (2002)	10-day-old mice exposed to a 60-Hz magnetic field at 1 mT for 2 h, cerebellum assayed at 0. 2, 4, and 24 h after exposure	DNA single strand breaks (Comet assay): "While increased DNA damage was detected by tail ratio at 2h after MF exposure, no supporting evidence of increased DNA damage was detected by the other parameters." "Taken together, these results do not support the hypothesis that acute MF exposure causes DNA damage in the cerebellums of immature mice." No change in apoptosis.
*McNamee et al. (2005)	Rodents (adult rats, adult mice, and immature mice) exposed to a 60-Hz magnetic field at 0.1, 1 or 2 mT for 2 h. Assayed at 0, 2 and 4 h after exposure	This study provided no evidence of magnetic-field-induced DNA single strand breaks (Comet assay) in the brain.
Mercado-Sáenz et al. (2019)	*Saccharomyces cerevisiae* wild type strain (WS8105-1C) exposed to sinusoidal magnetic field (2.45 mT, 50 Hz, continuous) or pulsed magnetic field (1.5 mT, 25 Hz, 8 h/day). Chronological aging was evaluated during 40 days	Decreased spontaneous frequency of mitochondrial mutation during aging was observed in pulsed magnetic field-treated samples.
*Miyakoshi et al. (1996a)	Chinese hamster ovary (CHO) cells exposed to a 60-Hz magnetic field at 5 mT for 130 h	No significant effect on c-myc expression and cell growth rate.
Miyakoshi et al. (1996b)	Human melanoma MeWo cells exposed to a 50-Hz magnetic field at 400 mT up to 20 h	Induced mutations in the hypoxanthine-guanine phosphoribosyl transferase gene, synergistic with X-ray. No significant increase in mutant frequency occurred when DNA replication was inhibited during magnetic field exposure. DNA replication error is suspected of causing the mutations produced by ELFMF exposure.
Miyakoshi et al. (1997)	Human melanoma MeWo cells exposed to a 50-Hz magnetic field at 400 mT for 2 h	Induced mutations in the hypoxanthine-guanine phosphoribosyl transferase gene, DNA replication errors and/or disturbance of the mismatch repair systems caused by exposure to ELF-MF may be involved in the

		mutagenic effect.
Miyakoshi et al. (1998)	Human osteosarcoma cells (Saos-LP-12), with deleted 53 gene, exposed to a 50-Hz magnetic field at 400 mT for 4 h	Induced mutations in the hypoxanthine-guanine phosphoribosyl transferase gene. Introduction of the wild-type (wt) p53 expression plasmid (pOPRSVp53) suppressed the magnetic induced mutation. The findings suggest that wt p53 has a function in suppression of DNA replication errors and/or in maintenance of genomic stability after high-density magnetic field exposure.
Miyakoshi et al. (1999)	Chinese hamster ovary K1 (CHO-K1) cells exposed to a 60-Hz magnetic field at 5 mT for up to 6 weeks	No effect on mutant frequency of the hypoxanthine-guanine phosphoribosyl transferase but enhanced the effect of x-ray.
Miyakoshi et al. (2000)	Human glioma MO54 cells exposed to a 50-Hz magnetic field at 55, 50, or 400 mT at 4^0C or on ice, for 30 min	Exposure to magnetic field at more than 50 mT potentiated X-ray-induced DNA single strand breaks (Comet assay).
*Mizuno et al. (2014)	Human fibroblast WI38VA13 subcloned 2RA and XP2OS(SV) cells exposed to a 60-Hz magnetic field at 5 mT for 24 h	Magnetic field exposure did not have modification effect on cell survival after UV-B irradiation and on repair process of DNA damage induced by UV-B irradiation.
Moraveli et al. (2016)	dermal papilla mesenchymal cells exposed to 50-Hz EMF at 1 mT for 5-14 days	Increased expression of MAP gene with decreased cell proliferation (cell differentiation occurred.) (MAP2 protein involves in neuritogenesis to stabilize microtubules.)
Moretti et al. (2005)	Jurkat cells exposed to a 50-Hz magnetic field at 1 mT for 1 h with added xenobiotics	Magnetic field exposure enhanced genotoxic effects (DNA single strand breaks (Comet assay)) of xenobiotics.
Mouhoub et al. (2017)	*Salmonella hadar* grown under static magnetic field of 200 mT for 3, 6, or 9 h	Increased expression of gene involved in the production of acdiolipin and phosphatidylethanolamine (both components of bacteria cell membrane).
Nakayama et al. (2016)	Macrophages stimulated with the bacterial endotoxin, lipopolysaccharide and	Increased DNA single strand breaks (Comet assay) and decreased viability.

	posed to a 50-Hz magnetic field at 0.5 mT for 24 h	
Nasrabadi et al. (2018)	Neonatal human retinal pigment epithelial cells exposed to pulsed 50-Hz EMF at 1 mT for 8 h daily for 3 days	Both gene and protein expressions of retinal progenitor cell markers were reduced.
Nikolova et al. (2005)	Mouse embryonic stem (ES) cells exposed to an intermittent (5 min ON/30 min OFF) 50-Hz EMF at 2 mT for 6 or 48 h	Significantly affected transcript levels of the apoptosis-related bcl-2, bax, and cell cycle regulatory "growth arrest DNA damage inducible" GADD45 genes, No effect on DNA single and double strand breaks (Comet assay).
*Okudan et al. (2010)	Swiss mice exposed to a 50-Hz EMF at 0.001 - 0.005 mT for 40 days	The results suggest that </=0.005 mT intensities of 50 Hz EMFs did not cause genotoxic effect in the mouse.(However, The number of micronucleus per peripheral blood lymphocytes in the 0.004 and 0.005 mT-exposure groups were significantly higher than those of the lower intensity exposure groups. The males in 0.004 mT-exposure group displayed the highest micronucleus number per lymphocyte).
Panagopoulos et al. (2013)	Newly eclosed *Drosophila melanogaster* exposed to 50-Hz magnetic field (0.1, 1.1, and 2.1 mT) continuously during the first 5 days of their adult lives	Severe DNA damage (DNA fragmentation by TUNEL assay) and consequent cell death induction in the reproductive cells.
Pesqueira et al. (2017)	Human tendon-derived cells exposed to a 2 Hz magnetic field at 350 mT for 4 or 8 h, or 8 h every 24 or 48 h up to 14 days	8-h exposure significantly upregulated the expression of tendon-associated genes SCX, COL1A1, TNC and DCN. 8 h every 24 h exposure significantly upregulated COL1A1, COL3A1 and TNC at day 14.
Pilger et al. (2004)	Human fibroblasts exposed to an intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 15 h	Exposure resulted in an increase in DNA single strand breaks (Comet assay) unlikely to be caused by intracellular changes that affect intracellular [Ca2+] or mitochondrial membrane potential.
Potenza et al. (2004a)	*E. coli* XL-1Blue exposed to static	Increased cell proliferation and changes in gene expression observed. The field

	magnetic field at 300 mT up to 50 h	magnetic field may stimulate transposition activity.
Potenza et al. (2004b)	Escherichia coli DNA, plasmid, and amplification products of different lengths exposed to static magnetic field at 200-150 mT for 5 h	The in vitro assays displayed interactions between the magnetic field and DNA, revealing principally that magnetic field exposure induces DNA alterations in terms of point mutations.. This genotoxic effect of the magnetic field, however, is minimized in living organisms due to the presence of protective cellular responses.
Rageh et al. (2012)	Newborn rats (10 days after delivery) exposed continuously to a 50 Hz magnetic field at 0.5 mT for 30 days	Increased DNA single strand breaks (Comet assay) in brain cells and micronucleus frequency in bone cells. Changes in anti-oxidative enzymes and increased lipid peroxidation.
*Reese et al. (1998)	Chinese hamster ovary (CHO) cells exposed to 60-Hz magnetic fields (0.1 or 2 mT), electric fields (1 or 38 V/m), or combined magnetic and electric fields (2 mT and 38 V/m, respectively) for 1 h	No significant effect on DNA single strand breaks (Comet assay) from exposures.
Reyes-Guerrero et al. (2010)	Adult male and female Wistar rats exposed to a 60-Hz magnetic field at 1 mT for 2 h/day for 9 days	ELF EMF modulates estrogen receptor- beta gene expression in the olfactory bulb of female adult rats but not in males.
Robison et al. (2002)	HL-60, HL-60R, and Raji cell lines exposed to a 60-Hz EMG at 0.15 mT for 24 h	EMF exposure offers significant protection from apoptosis (DNA double strand breaks (Comet assay)) and significantly decreased DNA repair rates in HL-60 and HL-60R cell lines but not in the Raji cell line.
*Ross et al. (2018)	Human mesenchymal stromal cell exposed to a 5-Hz EMF at 0.4 mT for 20 min/day, 3 times a week for 2 weeks	No chromosome breaks, viability and proliferation rate detected.
*Ruiz-Gómez et al. (2010)	Wild type (wt) and radiation sensitive mutant yeast strains (Saccharomyces cerevisiae) exposed to a	The exposure did not induce alterations in cell cycle and cause DNA damage.

	50 Hz magnetic field at 2.45 mT for 96 h	
Sadri et al. (2017)	Human mesenchymal stem cells derived from human newborn cords exposed to a static magnetic field of 12, 18, or 24 mT for 2 h	Induced differentiation and decreased expression of Sox-2, Nanong, and Oct-4 genes (These genes are involved in embryonic orgen development, maintenance of multipentency and self renewal of undifferentiated embryonic stem cell.)
Sanie-Jahromi et al. (2016)	Human breast adenocarcinoma MCF-7 and neuroblastoma SH-SY5Y cells exposed to 50-Hz EMF at 0.25 and 0.5 mT (5 min ON/5min OFF; 15 min ON/15min OFF, or 30 ON continuously) for 30 min	mRNA levels of seven genes involved in DNA repair pathways down regulated in MCF-7 cells. Synergistic with cisplatin in MCF-7 and SH-SY5Y cells.
Sanie-Jahromi and Saadat (2017)	MCF-7 and SH-SY5Y cells exposed to an intermittent (15 min OF/15-min OFF) 50-Hz EMF at 0.5 mT for 30 min. Cells were also treated with cisplastin and bleomycin	EMF exposed MCF-7 cells treated with cisplastin and bleomycin showed more effects on some DNA repair gene expression compared with “cisplastin and bleomycin” treatment alone, while SH-SY5Y susceptibility was not changed between the two treatments.
Sanie-Jahromi and Saadat (2018)	MCF-7 and SH-SY5Y cells were treated with 5.0 μM morphine and exposed to an intermittent (15 min ON/15 min OFF) 50-Hz EMF at 0.50 mT for 30 min	Morphine treatment showed significant down-regulation of expression of genes involved in DNA repair pathways, while in "Morphine + EMF" treatment, the genes were not significantly changed.
Sarimov et al. (2011)	Human lymphocytes exposed to 50-Hz magnetic field at 0.005-0.02 mT for 15-180 min	Magnetic field condensed relaxed chromatin and relaxed condensed chromatin.
*Scarfi et al (2005)	Human diploid fibroblasts exposed to an intermittent (5 min ON/10 min OFF) 50-Hz EMF or a 50-Hz field plus its harmonics for 24 h (1,2,4-BT) also studied	No significant effects on DNA single strand breaks (Comet assay) and micronucleus frequency.

Scassellati Sforzolini et al. (2004)	Cells exposed to a 50-Hz magnetic field at 5 mT; co-genotoxic effects with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), 4-nitroquinoline N-oxide (4NQO), benzene, 1,4-benzenediol (1,4-BD), or 1,2,4-benzenetriol	Magnetic field showed genotoxic (micronucleus test) and co-genotoxic (comet assay) capabilities.
Schmitz et al. (2004)	Male adult mice exposed to a 50-Hz magnetic field at 1.5 mT for 8 weeks	A significant increase in both unscheduled DNA synthesis and in situ nick translation was only found for epithelial cells of the choroid plexus. Mitochondrial DNA synthesis was exclusively increased in renal epithelial cells of distal convoluted tubules.
Seong et al. (2014)	Human bone marrow-mesenchymal stem cells exposed to a 50 Hz EMF at 1 mT for 8 days	Increased expression of early growth response protein 1 (Egr1).
*Shen et al. (2016)	Chinese Hamster Lung cells exposed to a 50-Hz EMF at 0.4mT for 30 min or 24 h	Increase in LC3-II expression and increased autophagosome formation; no significant effect on γH2AX foci.(EMF-induced autophagy may balance the cellular homeostasis to protect the cells from severe adverse biological consequences.)
Shokrollahi et al. (2018)	Soybean plants exposed to static magnetic field at 20 and 30 mT for 5 h/day for 5 days	Exposure to 20 mT decreased gene expression of Fe transporter, ferrous and H_2O_2 contents and gene expression, content and activity of ferritin and catalase. Opposite responses were observed at 30 mT exposure. Tertiary structures of ferritin, apoferritin and catalase altered by static magnetic field.
Singh and Lai (1998)	Rats exposed to a 60-Hz magnetic field at 0.5 mT for 2 h	Data suggested that both DNA-protein and DNA-DNA crosslinks (Comet assay) were formed in brain cells.
Skyberg et al. (2001)	Blood samples from high voltage laboratory workers exposed to electromagnetic fields and mineral oil	In inhibited (hydroxyurea-inhibits DNA synthese, and caffeine-inhibits DNA repair) lymphocyte cultures, there were indications that electromagnetic fields in combination with mineral oil exposure may produce chromosomal aberrations. No effect on un-inhibited cells.

Solek et al. (2017)	Mouse spermatogenic cell lines (GC-1 spg and GC-2 spd) exposed to pulsed (1sec on/off) or continuous-wave 2, 50, 120 Hz EMF at 2.5- 8 mT for 2 h	EMF activated oxidative and nitrosative stress-mediated DNA damage pathways, resulting in p53/p21-dependent cell cycle arrest and apoptosis
*Song et al. (2018)	HeLa and primary IMR-90 fibroblasts exposed to a 60-Hz EMF at 1, 3, 6, or 10 or mT continuously for up to 168 h or 30 min every 24h for 3 days	No effect on DNA damage (gamma-H2AX foci).; promoted cell proliferation (probably due to decreased reactive oxygen species).
Stankevičiūtė et al. (2019)	Rainbow trout (*Oncorhynchus mykiss*) exposed to a 50-Hz EMF at 1 mT for 40days; and the common ragworm (*Hediste diversicolor*) and the Baltic clam (*Limecola balthica*) for 12 days	Trout and ragworm erythrocytes and clam gill cells showed elevated micronucleus frequency, nuclear buds, nuclear buds on filament cells, and cells with blebbed nuclei.
*Stronati et al. (2004)	Human whole blood exposed to a 50-Hz magnetic field at 1 mT for 2 h	No significant effects on DNA single strand breaks (Comet assay), sister chromatid exchanges, chromosome aberrations, and micronucleus frequency in lymphocytes. A slight decrease in cell proliferation observed.
*Sun C et al. (2018)	ATM-proficient ($Atm^{+/+}$) and ATM-deficient ($Atm^{-/-}$) mouse embryonic fibroblasts exposed to a 50-Hz magnetic field at 2 mT for 15 min.(Ataxia telangiectasia mutated (ATM) plays a central role in DNA damage repair.)	No effect on γ-H2AX foci in both types of cells.
Sun L et al. (2019)	Irpex lacteus, a white-rot fungus, exposed to a 50-Hz magnetic field ay 3.5 mT for 3 h/day for 4 days	Global gene expression changes were observed.
Sun RG et al.(2012)	K562 human leukemia cells exposed to	The potency of the combination of SMF and paclitaxel was greater than that of SMF or

	paclitaxel in the presence or absence of 8.8 mT static magnetic field for 24 h	paclitaxel alone on K562 cells, and these effects were correlated with DNA single strand breaks (Comet assay).
Suzuki et al. (2001)	Mouse exposed to high intensity static magnetic fields (3.0 T for 48 and 72 h and 4.7 T for 24, 48 and 72 h).	Increased micronucleus frequency in bone marrow cells.
Svedenstal et al. (1999)	Brain cells of CBA mice exposed to a 50 Hz magnetic field at 0.5 mT 2 h, 5 days or 14 days	DNA single strand breaks (Comet assay) increased after 14 days of exposure,
*Szerencsi et al. (2013)	Peripheral blood samples from men exposed to EMF produced by 3T magnetic resonance imaging equipment for 0, 22, 45, 67, and 89 min during the scanning procedure	No significant effect on DNA single strand breaks (Comet assay) and DNA integrity in lymphocytes.
Teodori et al. (2014)	Human glioblastoma cells exposed to static magnetic field at 80 mT for 6,12, or 24 h, also in combination with X-ray	Increased in DNA single strand breaks (Comet assay) after 24 h of exposure; x-ray induced DNA strand breaks significantly reduced by post-irradiation exposure to static magnetic field. Further data suggested that static magnetic field modulated DNA damage and/or repair, possibly through a mechanism that affects mitochondria.
*Testa et al. (2004)	Human blood samples exposed to a 50-Hz magnetic field at 1 mT for 48 h	No significant effect on micronucleus frequency and proliferation of lymphocytes. No interaction with x-ray.
*Tiwari et al. (2015)	Blood samples of human subjects occupationally exposed to 132 kV high-voltage substations (mean duration on job 9.27 years, range 2-30 years).	No significant effect on DNA single strand breaks (Comet assay) in lymphocytes, increased oxidative stress observed.
Udroiu et al. (2006)	Liver and peripheral blood sampled from newborn mice exposed to a 50-Hz magnetic field of 0.65 mT during the	Data obtained in newborn mice showed a significant increase in micronuclei frequencies. No significant effect was recorded on exposed adults.

	whole intra-uterine life (21 days), and on bone marrow and peripheral blood from adult mice exposed to the same magnetic field for the same period	
Udroiu et al. (2015)	Mice exposed to 50-Hz, 0.065 mT magnetic field, 24 hours/day, for a total of 30 days, starting from 12 days post-conception	Magnetic field induced a slight genotoxic damage (micronucleus formation) and no interaction with x ray in erythrocytes, but modulate the response of male germ cells to X-rays with an impact on proliferation/differentiation processes. Magnetic field exposure decreased DNA single and double strand breaks (Comet assay) in germ cells at 42 days after birth.
*Verschaeve et al. (2011)	*Salmonella typhimurium* exposed to a 50-Hz magnetic field at 0.1 or 0.5 mT for 1 or 2 h	The magnetic field did not induce mutagenicity in S. typhimurium bacteria and did not show any synergetic effect when combined with chemical mutagens.
*Verschaeve et al. (2016)	*Salmonella typhimurium* exposed to50 Hz magnetic field at 0.1 mT for 1 h	The magnetic field did not damage DNA and had no influence on the DNA damaging capacity of several mutagens.
Villarini et al. (2006)	Human leukocytes exposed to a 50-Hz magnetic field at 3 mT for 30, 60, or 120 min and treated with mutagens	Magnetic field exposure increased N-methyl-N'-nitro-N-nitrosoguanidine and decreased 4-nitroquinoline N-oxide-induced DNA single strand breaks (Comet assay).
Villarini et al. (2013)	Male CD1 mice exposed to a 50-Hz magnetic field at 0.1, 0.2, 1 or 2 mT for 7 days (15 hours/day) and sacrificed either at the end of exposure or after 24 h	Magnetic field exposure induced DNA single strand breaks (Comet assay) and did not affect hsp70 expression in the brain.
Villarini et al. (2015)	Blood leukocytes from electric arc welders presumably exposed to 50-Hz EMF(mean 0.0078 mT; range: 0.00003-0.171 mT)	Decreased DNA single strand beaks (Comet assay), may be caused by DNA-protein crosslinks by metal exposure.
*Villarini et al. (2017)	SH-SY5Y and SK-N-BE-2 human neuroblastoma cells	or $AlCl_3$ alone induced DNA single strand breaks (Comet assay), changes in GSH/GSSG ratio or variations in Hsp70

	exposed to a 50-Hz magnetic field at 0.01. 0.1, or 1 mT for 1 h continuously or 5 h intermittently (15 min ON/15 min OFF), and also aluminum	expression. Co-exposure to ELF-MF and $AlCl_3$ did not have any synergic toxic effects.
Wahab et al. (2007)	Human peripheral blood lymphocytes exposed to 50 Hz sinusoidal (continuous or pulsed) or square (continuous or pulsed) magnetic fields at 0.001 or 1 mT for 72 h	A significant increase in the number of sister chromatid exchange /cell observed.
*Wang Y et al. (2019)	Human ventricular cardiomyocytes exposed to a 50-Hz magnetic field at 0.1 mT for 1 h continuously or 75 min intermittently (15 min ON/15 min OFF). Sprague-Dawley rats exposed to 50 Hz magnetic field at 0.1 mT for 15 h/day for 7 days	Magnetic field exposure did not cause DNA single strand breaks (Comet assay) in heart cells in both in vitro and in vivo experiments.
Wang Y. et al. (2020)	*Caenorhabditis elegans* exposed to 50-Hz, 3 mT EMF for 15 generations	Expression levels of the *r53.4, hpo-18, atp-5*, and *atp-3* genes encoding ATPase and *sod-1, sod-2,* and *sod-3* genes encoding superoxide dismutase (SOD) were significantly upregulated.
Wang Z et al. (2009)	Human embryoid body derived (hEBD) LVEC cell line exposed to 0.23-0.28 T static magnetic field for 24 h	Gene expression in cells showed nine signaling networks responded to static magnetic field
*Williams et al. (2006)	Salmonella bacteria cultures exposed to a 60-Hz intermittent magnetic field (5 min ON/10 min OFF) at 14.6 mT for 4 h	No significant increase in recombination events and DNA single and double strand breaks (assayed using a recombination event counter).However, magnetic field exposure induced protection from heat stress.
Wilson et al. (2015)	BALB/c×CBA/Ca F1 hybrid males exposed to 50Hz magnetic fields at 0.01, 0.1 or 0.3 mT for 2 or 15 h	There was a marginally significant increase in a non-dose-dependent mutation frequency in sperm, and not in blood cells.

Winker et al. (2005)	Human fibroblasts exposed to a 50-Hz intermittent (5 min ON/10 min OFF) EMF at 1 mT for 2-24 h	Increased micronucleus frequency and chromosomal aberration.
Wolf et sl. (2005)	HL-60 leukemia cells, Rat-1 fibroblasts, and WI-38 diploid fibroblasts exposed to a 50-Hz EMF at 0.5-1 mT for 24-72 h	Dose-dependent increases in DNA single strand breaks (Comet assay) and formation of 8-hydroxy-2'-deoxyguanosine adducts were observed in all cell lines. There were increases in cell proliferation and reactive oxygen species.
Yagci and Kesim (2016)	Human gingival fibroblasts exposed in vitro to static magnetic fields produced by dental magnetic attachments for 10-12 days. (The maximum magnetic flux densities measured at the magnet centers of 4 types of attachment were 95.6-148.1 mT and became almost zero at 10 mm away)	Increased micronucleus frequency.
Yaguchi et al. (1999)	Mouse embryonic skin m55 cells exposed to a 60-Hz magnetic field at 5, 50, or 400 mT for 42 h	Increase in sister chromatid exchanges after 400 mT exposure.
Yaguchi et al. (2000)	Mouse embryonic skin m55 cells exposed to 60-Hz (5 or 50 mT) or 50-Hz (400 mT) magnetic fields for 40 h. Some cells also treated with mitomycin C or X-ray	Increased chromosomal aberration, synergistic with mitomycin C and X-ray.
Yao et al. (2015)	Rat Schwann cells exposed to DC electric field for 36-72 h at 50, 100, or 200 mV/mm	Differentially expression of genes participate in multiple cellular signaling pathways involved in the regulation of cell migration, including pathways of regulation of actin cytoskeleton, focal adhesion, and PI3K-Akt cell cycle regulation).
Yin et al. (2016)	Primary cultured rat hippocampal neurons exposed to a 50-HZ EMF at mT for 90 min	Increase in DNA single strand breaks (Comet assay); free radicals involved.

Yokus et al. (2005)	Female Wistar rats exposed to a 50-Hz magnetic field at 0.97 mT for 3 h/day for 50 and 100 days	Increased 8-hydroxy-2'-deoxyguanosine in blood cells.
Yokus et al. (2008)	Male Sprague-Dawley rats exposed to a 50-Hz magnetic field at 0.1 or 0.5 mT for 2 h/day for 10 months	Increased DNA base modifications in leucocytes [8-hydroxyguanine (8-OH-Gua), 2,6-diamino-4-hydroxy-5-formamidopyrimidine (FapyGua), and 4,6-diamino-5-formamidopyrimidine (FapyAde)]
Yoon et al. (2014)	Human lung fibroblast WI38 cells and human lung epithelial L132 cells exposed to a 60-Hz magnetic field at 2 mT for 6 h	2 mT field induced increased γ-H2AX expression, as well as γ-H2AX foci production. Interacted with gamma radiation but not H_2O_2.
Yuan et al. (2020)	Tumor cell lines including lung cancer, gastric cancer, pancreatic cancer and nephroblastoma exposed to a 50-Hz EMF modulated by static MF with time-average intensity of 5.1 mT, for 2 h/day for 3 days	Induced DNA single strand breaks (Comet assay), gamma-H2AX and activation of DNA repair pathways, increased reactive oxygen species and ferroptosis, and decreased proliferation.
Zendehdel et al. (2019)	Peripheral blood cells of male power line workers in a power plant. The median value of the magnetic field at the working sites was 0.00085 mT	Increased in DNA single strand breaks (Comet assay).
Zhang H et al. (2016)	ICR mice exposed to a 50-Hz EMF at 8 mT for 4 h/day for 28 days	Declined DNA content and increased expression of apoptosis genes in spleen. Free radical may be involved.
Zhang Y et al. (2016)	Workers with or without exposure to ELF-EMF (50 Hz) of 110-420kV power lines	Significant increased urinary 8-isoprostane and 8-OHdG were observed in workers with EMF exposure. Free radical may be involved.
Zheng et al. (2018)	dental pulp stem cells exposed to a static magnetic field of 1,2, 4 mT for 15 min, 30 min, 1 h or 24 h	Increased expression of several growth factors (FGF-2, TGF-β, and VEGF), migration genes (MMP-1 and MMP-2), and upregulated the two YAP/TAZ-regulated genes, CTGF and ANKRD1. (YAP/TAZ are transcriptional activators particularly

		involved in cancer cell proliferation, therapy resistance and metastasis. Increased cell proliferation, osteo/odontogenesis and mineralization observed in the stem cells.
*Zhu et al. (2016)	Human lens epithelial cells exposed to a 50-Hz magnetic field at 0.4 mT for 2, 6, 12, 24, or 48 h	No effect on DNA single strand breaks (Comet assay) and gamma-H2AX foci.
Zmyslony et al. (2000)	Rat exposed to a static or 50-Hz magnetic field at 7 mT for 3 h	In combination with $FeCl_2$, increases in DNA single strand breaks (Comet assay) observed for both static and 50-Hz field exposure in lymphocytes.
Zmyslony et al. (2004)	Rat lymphocytes exposed first to ultraviolet radiation and then to a 50-Hz magnetic field at 0.04 mT for 5 or 60 min	60-min magnetic field exposure (plus UVA) caused an increase in DNA single strand breaks (Comet assay). MF may affect the radical pairs generated during the oxidative or enzymatic processes of DNA repair.

References

Agliassa, C., Narayana, R., Bertea, C.M., et al. (2018). Reduction of the geomagnetic field delays Arabidopsis thaliana flowering time through downregulation of flowering-related genes. Bioelectromagnetics. 39:361-374.

Ahuja, Y.R., Vijayashree, B., Saran, R., et al. (1999). In vitro effects of low-level, low-frequency electromagnetic fields on DNA damage in human leucocytes by comet assay. Indian J. Biochem. Biophys. 36:318-322.

Albert, G.C., McNamee, J.P., Marro, L., et al. (2009). Assessment of genetic damage in peripheral blood of human volunteers exposed (whole-body) to a 200 muT, 60 Hz magnetic field. Int. J. Radiat. Biol. 85:144-152.

Alcaraz, M., Olmos, E., Alcaraz-Saura, M., et al. (2014). Effect of long-term 50 Hz magnetic field exposure on the micronucleated polychromatic erythrocytes of mice. Electromagn. Biol. Med. 33:51-57.

Al-Huqail, A.A., Abdelhaliem, E. (2015). Evaluation of genetic variations in maize seedlings exposed to electric field based on protein and DNA markers. Biomed. Res. Int. 2015:874906.

Amara, S., Abdelmelek, H., Garrel, C., et al. (2006). Effects of subchronic exposure to static magnetic field on testicular function in rats. Arch. Med. Res. 37:947-952.

Amara, S., Douki, T., Ravana, J.L., et al. (2007a). Influence of a static magnetic field (250 mT) on the antioxidant response and DNA integrity in THP1 cells. Phys. Med. Biol. 52:889-898.

Amara, S., Abdelmelek, H., Garrel, C., et al. (2007b) Zinc supplementation ameliorates static magnetic field-induced oxidative stress in rat tissues. Environ. Toxicol. Pharmacol. 23:193-197.

Amara, S., Douki, T., Garel, C., et al. (2009). Effects of static magnetic field exposure on antioxidative enzymes activity and DNA in rat brain. Gen. Physiol. Biophys. 28:260-265.

Amara, S., Douki, T., Garrel, C., et al. (2011). Effects of static magnetic field and cadmium on oxidative stress and DNA damage in rat cortex brain and hippocampus. Toxicol. Ind. Health. 27:99-106.

Arruda-Neto, J.D.T., Friedberg, E.C., Bittencourt-Oliveira, M.C., et al. (2009). Static electric fields interfere in the viability of cells exposed to ionising radiation. Int. J. Rad. Biol. 85:314-321.

Ashta, A., Motalleb, G., Ahmadi-Zeidabadi, M. (2020). Evaluation of frequency magnetic field, static field, and Temozolomide on viability, free radical production and gene expression (p53) in the human glioblastoma cell line (A172) Electromagn. Biol. Med. doi: 10.1080/15368378.2020.1793171. (Online ahead of print).

Baek, S., Choi, H., Park, H., et al. (2019). Effects of a hypomagnetic field on DNA methylation during the differentiation of embryonic stem cells. Sci. Rep. 9:1333.

Bagheri Hosseinabadi, M., Khanjani, N., Mirzaii, M., et al. (2019). DNA damage from long-term occupational exposure to extremely low frequency electromagnetic fields among power plant workers. Mutat. Res. 846:403079.

Bagheri Hosseinabadi, M., Khanjani, N., Atashi, A., et al. (2020). The effect of vitamin E and C on comet assay indices and apoptosis in power plant workers: A double blind randomized controlled clinical trial. Mutat. Res. 850-851:503150.

Balamuralikrishnan, B., Balachandar, V., Kumar, S.S., et al. (2012). Evaluation of chromosomal alteration in electrical workers occupationally exposed to low frequency of electromagnetic field (EMFs) in coimbatore population, India. Asian Pac. J. Cancer Prev. 13:2961-2966.

Baraúna, R.A., Santos, A.V., Graças, D.A., et al. (2015). Exposure to an extremely low-frequency electromagnetic field only slightly modifies the proteome of *Chromobacterium violaceum* ATCC 12472. Genet. Mol. Biol. 38:227-230.

Belyaev, I.Y., Hillert, L., Protopopova, M., et al (2005). 915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons. Bioelectromagnetics 26:173-184.

Bertea, C.M., Narayana, R., Agliassa, C., et al. (2015). Geomagnetic field (Gmf) and plant evolution: investigating the effects of Gmf reversal on *Arabidopsis thaliana* development and gene expression. J. Vis. Exp. (105), e53286.

Borhani, N., Rajaei, F., Salehi, Z., et al. (2011). Analysis of DNA fragmentation in mouse embryos exposed to an extremely low-frequency electromagnetic field. Electromagn. Biol. Med. 30:246-252.

Brix, G., Günther, E., Rössler, U., et al. (2020). Double-strand breaks in lymphocyte DNA of humans exposed to [^{18}F]fluorodeoxyglucose and the static magnetic field in PET/MRI. EJNMMI Res. 10(1):43, 2020.

Bułdak, R.J., Polaniak, R., Bułdak, L., et al. (2012). Short-term exposure to 50 Hz ELF-EMF alters the cisplatin-induced oxidative response in AT478 murine squamous cell carcinoma cells. Bioelectromagnetics. 33:641-651.

Burgos-Molina, A.M., Mercado-Sáenz, S., Sendra-Portero, F., et al. (2020). Effect of low frequency magnetic field on efficiency of chromosome break repair. Electromagn. Bio.l Med. 39:30-37.

Calabrò, E., Condello, S., Magazù, S., et al. (2011). Static and 50 Hz electromagnetic fields effects on human neuronal-like cells vibration bands in the mid-infrared region. J. Electromagn. Analy. Appl. 3: 69-78.

Calabrò, E., Goswami, H.K., Salvatore Magazù, S.(2020). Chromosome aberration in typical biological systems under exposure to low- and high-intensity magnetic fields. Electromagn. Biol. Med. (March 5, 2020)

Cantoni, O., Sestili, P., Fiorani, M., et al. (1996). Effect of 50 Hz sinusoidal electric and/or magnetic fields on the rate of repair of DNA single strand breaks in cultured mammalian cells exposed to three different carcinogens: methylmethane sulphonate, chromate and 254 nm U.V. radiation. Biochem. Mol. Biol. Int. 38:527-533.

Celikler, S., Aydemir, N., Vatan, O., et al. (2009). A biomonitoring study of genotoxic risk to workers of transformers and distribution line stations. Int. J. Environ. Health Res. 19421-430.

Cellini, L., Grande, R., Di Campli, E., et al. (2008). Bacterial response to the exposure of 50 Hz electromagnetic fields. Bioelectromagnetics. 29:302-311.

Chahal, R., Craig, D.Q., Pinney, R.J. (1993). Investigation of potential genotoxic effects of low frequency electromagnetic fields on Escherichia coli. J. Pharm. Pharmacol. 45:30-33.

Chen, G.D., Lu, D.Q., Jiang, H., et al. (2008) [Effects of 50 Hz magnetic fields on gene expression in MCF-7 cells]. Zhejiang Da Xue Xue Bao Yi Xue Ban. 37:15-22. [Article in Chinese]

Chen, G., Lu, D., Chiang, H., et al. (2012). Using model organism Saccharomyces cerevisiae to evaluate the effects of ELF-MF and RF-EMF exposure on global gene expression. Bioelectromagnetics. 33:550-560.

Chen, J., Guan, L., Fan, P., et al. (2020). In vitro study of the effects of DC electric fields on cell activities and gene expression in human choriocarcinoma cells. Electromagn. Biol. Med. doi: 10.1080/15368378.2020.1846555. (Online ahead of print.)

Chen, W.F., Qi, H., Sun, R.G., et al. (2010). Static magnetic fields enhanced the potency of cisplatin on k562 cells. Cancer Biother. Radiopharm. 25:401-408.

Cho, S., Lee, Y., Lee, S., et al. (2014). Enhanced cytotoxic and genotoxic effects of gadolinium following ELF-EMF irradiation in human lymphocytes. Drug Chem. Toxicol. 37:440-447.

Cho, Y.H., Jeon, H.K., Chung, H.W. (2007) Effects of extremely low-frequency electromagnetic fields on delayed chromosomal instability induced by bleomycin in normal human fibroblast cells. J. Toxicol. Environ. Health A. 70:1252-1258.

Chow, K., Tung, W.L (2000a) Magnetic field exposure enhances DNA repair through the induction of DnaK/J synthesis. FEBS Lett. 478:133-136.

Chow, K.C., Tung, W.L. (2000b) Magnetic field exposure stimulates transposition through the induction of DnaK/J synthesis. Biochem. Biophys. Res. Commun. 270:745-748.

Collard, J.F., Lazar, C., Nowé, A., et al. (2013). Statistical validation of the acceleration of the differentiation at the expense of the proliferation in human epidermal cells exposed to extremely low frequency electric fields. Prog. Biophys. Mol. Biol. 111:37-45.

Consales, C., Cirotti, C., Filomeni, G., et al. (2018). Fifty-hertz magnetic field affects the epigenetic modulation of the miR-34b/c in neuronal cells. Mol. Neurobiol. 55:5698-5714.

Cuccurazzu, B., Leone, L., Podda, M.V., et al. (2010). Exposure to extremely low-frequency (50 Hz) electromagnetic fields enhances adult hippocampal neurogenesis in C57BL/6 mice. Exp. Neurol. 226:173-182.

Del Re, B., Bersani, F., Mesirca, P., et al. (2006). Synthesis of DnaK and GroEL in *Escherichia coli* cells exposed to different magnetic field signals. Bioelectrochemistry. 69:99-103.

Delimaris, J., Tsilimigaki, S., Messini-Nicolaki, N., et al. (2006). Effects of pulsed electric fields on DNA of human lymphocytes. Cell. Biol. Toxicol. 22:409-415.

Di Campli, E., Di Bartolomeo, S., Grande, R., et al. (2010). Effects of extremely low-frequency electromagnetic fields on Helicobacter pylori biofilm. Curr. Microbiol. 60:412-418.

Dominici, L., Villarini, M., Fatigoni, C., et al. (2011). Genotoxic hazard evaluation in welders occupationally exposed to extremely low-frequency magnetic fields (ELF-MF). Int. J. Hyg. Environ. Health. 215:68-75.

Dong, D., Yang, J., Zhang, G., et al. (2019). 16 T high static magnetic field inhibits receptor activator of nuclear factor kappa-B ligand-induced osteoclast differentiation by regulating iron metabolism in Raw264.7 cells. J. Tissue Eng. Regen. Med. 13:2181-2190.

Du, X.G., Xu, S.S., Chen, Q., et al. (2008). [Effects of 50 Hz magnetic fields on DNA double-strand breaks in human lens epithelial cells]. Zhejiang Da Xue Xue Bao Yi Xue Ban. 37:9-14. [Article in Chinese]

Duan, W., Liu, C., Zhang, L., et al. (2015). Comparison of the genotoxic effects induced by 50 Hz extremely low-frequency electromagnetic fields and 1800 MHz radiofrequency electromagnetic fields in GC-2 cells. Radiat. Res. 183:305-314.

El-Bialy, N.S., Rageh, M.M. (2013). Extremely low-frequency magnetic field enhances the therapeutic efficacy of low-dose cisplatin in the treatment of Ehrlich carcinoma. Biomed. Res. Int. 2013:189352.

Erdal, N., Gürgül, S., Celik, A. (2007). Cytogenetic effects of extremely low frequency magnetic field on Wistar rat bone marrow. Mutat. Res. 630:69-77.

Fairbairn, D.W., O'Neill, K.L. (1994). The effect of electromagnetic field exposure on the formation of DNA single strand breaks in human cells. Cell. Mol. Biol. (Noisy-legrand). 40:561-567.

Fan, W., Qian, F., Ma, Q., et al. (2015). 50 Hz electromagnetic field exposure promotes proliferation and cytokine production of bone marrow mesenchymal stem cells. Int. J. Clin. Exp. Med. 8:7394-7404.

Fan, W., Huang, Z., Fan, B. (2018). Effects of prolonged exposure to moderate static magnetic field and its synergistic effects with alkaline pH on *Enterococcus faecalis.* Microb. Pathog. 115:117-122.

Fatigoni, C., Dominici, L., Moretti, M., et al. (2005). Genotoxic effects of extremely low frequency (ELF) magnetic fields (MF) evaluated by the Tradescantia-micronucleus assay Environ. Toxicol. 20:585-591.

Fedrowitz, M., Löscher, W. (2012). Gene expression in the mammary gland tissue of female Fischer 344 and Lewis rats after magnetic field exposure (50 Hz, 100 μT) for 2 weeks. Int. J. Radiat. Biol. 88:425-429. (See also: Fedrowitz, M., Hass, R., Löscher, W. (2012). Effects of 50 Hz magnetic field exposure on the stress marker α-amylase in the rat mammary gland. Int. J. Radiat. Biol. 88:556-564.)

Fiorani, M., Cantoni, O., Sestili, P., et al. (1992). Electric and/or magnetic field effects on DNA structure and function in cultured human cells. Mutat. Res. 282:25-29.

Focke, F., Schuermann, D., Kuster, N., et al. (2010). DNA fragmentation in human fibroblasts under extremely low frequency electromagnetic field exposure. Mutat. Res. 683:74-83.

Frahm, J., Lantow, M., Lupke, M., et al. (2006). Alteration in cellular functions in mouse macrophages after exposure to 50 Hz magnetic fields. J. Cell. Biochem. 99:168-177.

Frazier, M.E., Reese, J.A., Morris, J.E., et al. (1990). Exposure of mammalian cells to 60-Hz magnetic or electric fields: analysis of DNA repair of induced, single strand breaks. Bioelectromagnetics. 11:229-234.

Frisch, P., Li, G.C., McLeod, K., et al. (2013). Induction of heat shock gene expression in RAT1 primary fibroblast cells by ELF electric fields. Bioelectromagnetics. 34:405-413.

Giorgi, G., Marcantonio, P., Bersani, F., et al. (2011). Effect of extremely low frequency magnetic field exposure on DNA transposition in relation to frequency, wave shape and exposure time. Int. J. Radiat. Biol. 87:601-608.

Giorgi, G., Lecciso, M., Capri, M., et al. (2014). An evaluation of genotoxicity in human neuronal-type cells subjected to oxidative stress under an extremely low frequency pulsed magnetic field. Mutat. Res. Genet. Toxicol. Environ. Mutagen. 775-776:31-37.

Giorgi, G., Pirazzini, C., Bacalini, M.G., et al. (2017). Assessing the combined effect of extremely low-frequency magnetic field exposure and oxidative stress on LINE-1 promoter methylation in human neural cells. Radiat. Environ. Biophys. 56:193-200.

Heredia-Rojas, J.A., Rodríguez de la Fuente, A.O., Alcocer González, J.M., et al. (2010). Effect of 60 Hz magnetic fields on the activation of hsp70 promoter in cultured INER-37 and RMA E7 cells. *In Vitro* Cell. Dev. Biol. Anim. 46:758-763.

Hong, R., Zhang, Y., Liu, Y., et al. (2005). [Effects of extremely low frequency electromagnetic fields on DNA of testicular cells and sperm chromatin structure in mice] Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 23:414-417. [Article in Chinese]

Huwiler, S.G., Beyer, C., Fröhlich, J., et al. (2012). Genome-wide transcription analysis of Escherichia coli in response to extremely low-frequency magnetic fields. Bioelectromagnetics. 33:488-496.

Ivancsits, S., Diem, E., Pilger, A., et al. (2002). Induction of DNA strand breaks by intermittent exposure to extremely-low-frequency electromagnetic fields in human diploid fibroblasts. Mutat. Res. 519:1-13.

Ivancsits, S., Diem, E., Jahn, O., et al. (2003a). Intermittent extremely low frequency electromagnetic fields cause DNA damage in a dose-dependent way. Int. Arch. Occup. Environ. Health. 76:431-436.

Ivancsits, S., Diem, E., Jahn, O., et al. (2003b). Age-related effects on induction of DNA strand breaks by intermittent exposure to electromagnetic fields. Mech. Ageing Dev.

124:847-850.

Ivancsits, S., Pilger, A., Diem, E., et al. (2005). Cell type-specific genotoxic effects of intermittent extremely low-frequency electromagnetic fields. Mutat. Res. 583:184-188.

Jajte, J., Zmyslony, M., Palus, J., et al. (2001). Protective effect of melatonin against in vitro iron ions and 7 mT 50 Hz magnetic field-induced DNA damage in rat lymphocytes. Mutat. Res. 483:57-64.

Jin, H., Yoon, H.E., Lee, J.S., et al. (2015). Effects on g2/m phase cell cycle distribution and aneuploidy formation of exposure to a 60 Hz electromagnetic field in combination with ionizing radiation or hydrogen peroxide in l132 nontumorigenic human lung epithelial cells. Korean J. Physiol. Pharmacol. 19:119-124.

Jin, Y.B., Kang, G.Y., Lee, J.S., et al. (2012). Effects on micronuclei formation of 60-Hz electromagnetic field exposure with ionizing radiation, hydrogen peroxide, or c-Myc overexpression. Int. J. Radiat. Biol. 88:374-380.

Jin, Y.B., Choi, S.H., Lee, J.S., et al. (2014). Absence of DNA damage after 60-Hz electromagnetic field exposure combined with ionizing radiation, hydrogen peroxide, or c-Myc overexpression. Radiat. Environ. Biophys. 53:93-101.

Jin, Y., Guo, W., Hu, X., et al. (2019). Static magnetic field regulates Arabidopsis root growth via auxin signaling. Sci. Rep. 9:14384.

Jouni, F.J., Abdolmaleki, P., Ghanati, F. (2012). Oxidative stress in broad bean (*Vicia faba L.)* induced by static magnetic field under natural radioactivity. Mutat. Res. 741:116-121.

Kesari, K.K., Luukkonen, J., Juutilainen, J., et al. (2015). Genomic instability induced by 50 Hz magnetic fields is a dynamically evolving process not blocked by antioxidant treatment. Mutat. Res. Genet. Toxicol. Environ. Mutagen 794:46-51.

Kesari, K.K., Juutilainen, J., Luukkonen, J., et al. (2016). Induction of micronuclei and superoxide production in neuroblastoma and glioma cell lines exposed to weak 50 Hz magnetic fields. J. R. Soc. Interface. 13:20150995.

Khalil, A.M., Qassem, W. (1991). Cytogenetic effects of pulsing electromagnetic fields on human lymphocytes in vitro: chromosome aberrations, sister-chromatid exchanges and cell kinetics. Mutat. Res. 247:141–146.

Ki, G.E., Kim, Y.M., Lim. H.M., et al. (2020). Extremely low-frequency electromagnetic fields increase the expression of anagen-related molecules in human dermal papilla cells via GSK-3β/ERK/Akt signaling pathway. Int. J. Mol. Sci. 21:784.

Kim, H.J., Jung, J., Park, J.H., et al. (2013). Extremely low-frequency electromagnetic fields induce neural differentiation in bone marrow derived mesenchymal stem cells. Exp. Biol. Med. (Maywood). 238:923-931.

Kim, J., Ha, C.S., Lee, H.J., et al. (2010). Repetitive exposure to a 60-Hz time-varying magnetic field induces DNA double-strand breaks and apoptosis in human cells. Biochem. Biophys. Res. Commun. 400:739-744.

Kim, J., Yoon, Y., Yun, S., et al. (2012). Time-varying magnetic fields of 60 Hz at 7 mT induce DNA double-strand breaks and activate DNA damage checkpoints without apoptosis. Bioelectromagnetics. 33:383-393.

Kimsa-Dudek, M., Synowiec-Wojtarowicz, A., Derewniuk, M., et al. (2018). Impact of fluoride and a static magnetic field on the gene expression that is associated with the antioxidant defense system of human fibroblasts. Chem. Biol. Interact. 287:13-19.

Kimsa-Dudek, M., Synowiec-Wojtarowicz, A., Krawczyk, A., et al. (2020). Anti-apoptotic effect of a static magnetic field in human cells that had been treated with sodium fluoride. J. Environ. Sci. Health A Tox. Hazard. Subst. Environ. Eng. 2020 Jun 25:1-8.

Kimura, T., Takahashi, K., Suzuki, Y., et al. (2008). The effect of high strength static magnetic fields and ionizing radiation on gene expression and DNA damage in *Caenorhabditis elegans* Bioelectromagnetics. 29:605-614.

Kindzelskii, A.L., Petty, H.R. (2000). Extremely low frequency pulsed DC electric fields promote neutrophil extension, metabolic resonance and DNA damage when phase matched with metabolic oscillators. Biochim. Biophys. Acta. 1495:90-111.

Kirschenlohr, H., Ellis, P., Hesketh, R., et al. (2012). Gene expression profiles in white blood cells of volunteers exposed to a 50 Hz electromagnetic field. Radiat. Res. 178:138-149.

Koyama, S., Sakurai, T., Nakahara, T., et al. (2008). Extremely low frequency (ELF) magnetic fields enhance chemically induced formation of apurinic/apyrimidinic (AP) sites in A172 cells. Int. J. Radiat. Biol. 84:53-59.

Kubinyi, G., Zeitler, Z., Thuróczy, G., et al. (2010). Effects of homogeneous and inhomogeneous static magnetic fields combined with gamma radiation on DNA and DNA repair. Bioelectromagnetics. 31:488-494.

Kumari, K., Koivisto, H., Viluksela, M., et al. (2017). Behavioral testing of mice exposed to intermediate frequency magnetic fields indicates mild memory impairment. PLoS One. 12(12):e0188880.

Lacy-Hulbert, A, Wilkins, R.C., Hesketh, T.R., et al. (1995).No effect of 60 Hz electromagnetic fields on MYC or beta-actin expression in human leukemic cells. Rad. Res. 144:9-17.

Lagroye, I., Poncy, J.L. (1997). The effect of 50 Hz electromagnetic fields on the formation of micronuclei in rodent cell lines exposed to gamma radiation. Int. J. Radiat. Biol. 72:249-254.

Lai, H., Singh, N.P. (1997a). Acute exposure to a 60 Hz magnetic field increases DNA strand breaks in rat brain cells. Bioelectromagnetics. 18:156-165.

Lai, H., Singh, N.P. (1997b). Melatonin and N-tert-butyl-alpha-phenylnitrone block 60-Hz magnetic field-induced DNA single and double strand breaks in rat brain cells. J. Pineal Res. 22:152-162.

Lai, H., Singh, N.P. (2004). Magnetic-field-induced DNA strand breaks in brain cells of the rat. Environ. Health Perspect. 112:687-694.

Laramee, C.B., Frisch, P., McLeod, K., et al. (2014). Elevation of heat shock gene expression from static magnetic field exposure in vitro. Bioelectromagnetics. 35:406-413.

Lee, C-H., Hung, Y-C., Huang, G.S. (2010). Static magnetic field accelerates aging and development in nematode. Commun. Integr. Biol. 3:528-529.

Lee, H.C., Hong, M.N., Jung, S.H., et al. (2016). Effect of extremely low frequency magnetic fields on cell proliferation and gene expression. Bioeletcromagnetics 36:506-516.

Lee, J.W., Kim, M.S., Kim, Y.J., et al. (2011). Genotoxic effects of 3 T magnetic resonance imaging in cultured human lymphocytes. Bioelectromagnetics. 32:535-542.

Leone, L., Fusco, S., Mastrodonato, A., et al. (2014). Epigenetic modulation of adult hippocampal neurogenesis by extremely low-frequency electromagnetic fields. Mol. Neurobiol. 49:1472-1486.

Li, S.H,, Chow, K.C. (2001). Magnetic field exposure induces DNA degradation. Biochem. Biophys. Res. Commun. 280:1385-1388.

Li, L., Xiong, D.F., Liu, J.W., et al. (2015). A cross-sectional study on oxidative stress in workers exposed to extremely low frequency electromagnetic fields. Int. J. Radiat. Biol. 91:420-425.

Li, S.S., Zhang, Z.Y., Yang, C.J., et al. (2013). Gene expression and reproductive abilities of male Drosophila melanogaster subjected to ELF-EMF exposure. Mutat. Res. 758:95-103.

Li, Y., Liu, X., Liu, K., et al. (2014). Extremely low-frequency magnetic fields induce developmental toxicity and apoptosis in zebrafish (Danio rerio) embryos. Biol. Trace Elem. Res. 162:324-332.

Li, Y., Wang, X., Yao, L. (2015). Directional migration and transcriptional analysis of oligodendrocyte precursors subjected to stimulation of electrical signal. Am. J. Physiol. Cell. Physiol. 309:C532-540.

Li, Y., Zhang, Y., Wang, W., et al. (2019) Effects of pulsed electromagnetic fields on learning and memory abilities of STZ-induced dementia rats. Electromagn. Biol. Med. 38:123-130.

Lin, K.W., Yang, C.J., Lian, H.Y., et al. (2016). Exposure of ELF-EMF and RF-EMF increase the rate of glucose transport and TCA cycle in budding yeast. Front. Microbiol. 7:1378.

Liu, Y., Liu, W.B., Liu, K.J., et al. (2015). Effect of 50 Hz extremely low-frequency electromagnetic fields on the DNA methylation and DNA methyltransferases in mouse spermatocyte-derived cell line GC-2. Biomed. Res. Int. 2015:237183.

Lopucki, M., Schmerold, I., Dadak, A., et al. (2005). Low dose magnetic fields do not cause oxidative DNA damage in human placental cotyledons in vitro. Virchows Arch. 446:634-639.

Lourencini da Silva, R., Albano, F., Lopes dos Santos, L.R., et al. (2000). The effect of electromagnetic field exposure on the formation of DNA lesions. Redox Rep. 5:299-301.

Luceri, C., De Filippo, C., Giovannelli, L., et al. (2005). Extremely low-frequency electromagnetic fields do not affect DNA damage and gene expression profiles of yeast and human lymphocytes. Radiat. Res. 164:277-285.

Lupke, M., Frahm, J., Lantow, M., et al. (2006). Gene expression analysis of ELF-MF exposed human monocytes indicating the involvement of the alternative activation pathway. Biochim. Biophys. Acta. 1763:402-412.

Luukkonen, J., Liimatainen, A., Höytö, A., et al. (2011). Pre-exposure to 50 Hz magnetic fields modifies menadione-induced genotoxic effects in human SH-SY5Y neuroblastoma cells. PLoS One. 6:e18021.

Luukkonen, J., Liimatainen, A., Juutilainen, J., et al. (2014). Induction of genomic instability, oxidative processes, and mitochondrial activity by 50 Hz magnetic fields in human SH-SY5Y neuroblastoma cells. Mutat. Res. 760:33-41.

Luukkonen, J., Höytö, A., Sokka, M., et al. (2017). Modification of p21 level and cell cycle distribution by 50 Hz magnetic fields in human SH-SY5Y neuroblastoma cells. Int. J. Radiat. Biol. 93:240-248.

Ma, Q., Deng, P., Zhu, G., et al. (2014). Extremely low-frequency electromagnetic fields affect transcript levels of neuronal differentiation-related genes in embryonic neural stem cells. PLoS One. 9:e90041.

Mahaki, H., Jabarivasal, N., Sardarian, K., et al. (2019). The effects of extremely low-frequency electromagnetic fields on c-Maf, STAT6, and RORα expressions in spleen and thymus of rat. Electromagn. Biol. Med. 38:177-183.

Mahmoudinasab, H., Saadat, M. (2016). Short-term Exposure to 50-Hz Electromagnetic field and alterations in *NQO1* and *NQO2* expression in MCF-7 Cells. Open Access Maced. J. Med. Sci. 4:548-550.

Mahmoudinasab, H., Sanie-Jahromi, F., Saadat, M. (2016). Effects of extremely low-frequency electromagnetic field on expression levels of some antioxidant genes in human MCF-7 cells. Mol. Biol. Res. Commun. 5:77-85.

Mahmoudinasab, H., Saadat, M. (2018a). Electromagnetic field could protect SH-SY5Y cells against cisplatin cytotoxicity, but not MCF-7 Cells. DNA Cell. Biol. 37:330-335.

Mahmoudinasab, H., Saadat, M. (2018b). Expressions of some antioxidant genes in SH-SY5Y cells treated with β-lapachone, morphine and electromagnetic field. Mol. Biol. Rep. 45:379-387.

Mairs, R.J., Hughes, K., Fitzsimmons, S., et al. (2007). Microsatellite analysis for determination of the mutagenicity of extremely low-frequency electromagnetic fields and ionising radiation in vitro. Mutat. Res. 626:34-41.

Manzella, N., Bracci, M., Ciarapica, V., et al. (2015). Circadian gene expression and extremely low frequency magnetic fields: an in vitro study. Bioelectromagnetics. 36:294-301.

Mariucci, G., Villarini, M., Moretti, M., et al. (2010). Brain DNA damage and 70-kDa heat shock protein expression in CD1 mice exposed to extremely low frequency magnetic fields. Int. J. Radiat. Biol. 86:701-710.

Markkanen, A., Juutilainen, J., Naarala, J. (2008). Pre-exposure to 50 Hz magnetic fields modifies menadione-induced DNA damage response in murine L929 cells. Int. J. Radiat. Biol. 84:742-751.

Mastrodonato, A., Barbati, S.A., Leone, L., et al. (2018). Olfactory memory is enhanced in mice exposed to extremely low-frequency electromagnetic fields via Wnt/β-catenin dependent modulation of subventricular zone neurogenesis. Sci. Rep. 8:262.

McNamee, J.P., Bellier, P.V., McLean, J.R., et al. (2002). DNA damage and apoptosis in the immature mouse cerebellum after acute exposure to a 1 mT, 60 Hz magnetic field. Mutat. Res. 513:121-133.

McNamee, J.P., Bellier, P.V., Chauhan, V., et al. (2005). Evaluating DNA damage in rodent brain after acute 60 Hz magnetic-field exposure. Radiat. Res. 164:791-797.

Mercado-Sáenz, S., Burgos-Molina, A.M., López-Díaz, B., et al. (2019). Effect of sinusoidal and pulsed magnetic field exposure on the chronological aging and cellular stability of S. cerevisiae. Int. J. Radiat. Biol. 95:1588-1596.

Miyakoshi, J., Ohtsu, S., Shibata, T., et al. (1996a). Exposure to magnetic field (5 mT at 60 Hz) does not affect cell growth and c-myc gene expression. J. Radiat. Res. 37:185-191.

Miyakoshi, J., Yamagishi, N., Ohtsu, S., et al. (1996b). Increase in hypoxanthine-guanine phosphoribosyl transferase gene mutations by exposure to high-density 50-Hz magnetic fields. Mutat. Res. 349:109-114.

Miyakoshi, J., Kitagawa, K., Takebe, H. (1997). Mutation induction by high-density, 50-Hz magnetic fields in human MeWo cells exposed in the DNA synthesis phase. Int. J. Radiat. Biol. 71:75-79, 1997.

Miyakoshi, J, Mori, Y., Yamagishi, N., et al. (1998). Suppression of high-density magnetic field (400 mT at 50 Hz)-induced mutations by wild-type p53 expression in human osteosarcoma cells. Biochem. Biophys. Res. Commun. 243:579-584.

Miyakoshi, J., Koji, Y., Wakasa, T., et al. (1999). Long-term exposure to a magnetic field (5 mT at 60 Hz) increases X-ray-induced mutations. J. Radiat. Res. 40:13-21.

Miyakoshi, J., Yoshida, M., Shibuya, K., et al. (2000). Exposure to strong magnetic fields at power frequency potentiates X-ray-induced DNA strand breaks. J. Radiat. Res. (Tokyo). 41:293-302.

Mizuno, K., Narita, E., Yamada, M., et al. (2014). ELF magnetic fields do not affect cell survival and DNA damage induced by ultraviolet B. Bioelectromagnetics. 35:108-115.

Moraveji, M., Haghighipour, N., Keshvari, H., et al. (2016).Nourizadeh Abbariki T, Shokrgozar MA, Amanzadeh A. Effect of extremely low frequency electromagnetic field on MAP2 and Nestin gene expression of hair follicle dermal papilla cells. Int. J. Artif. Organs. 39:294-299.

Moretti, M., Villarini, M., Simonucci, S., et al. (2005). Effects of co-exposure to extremely low frequency (ELF) magnetic fields and benzene or benzene metabolites determined in vitro by the alkaline comet assay. Toxicol. Lett. 157:119-128.

Mouhoub, R.B., Mansouri, A., Aliliche, K., et al. (2017). Unraveling the expression of genes involved in the biosynthesis pathway of cardiolipin and phosphatidylethanolamine in Salmonella Hadar grown under static magnetic field 200 mT. Microb. Pathog. 111:414-421.

Nakayama, M., Nakamura, A., Hondou, T., et al. (2016). Evaluation of cell viability, DNA single-strand breaks, and nitric oxide production in LPS-stimulated macrophage RAW264 exposed to a 50-Hz magnetic field. Int. J. Radiat. Biol. 92:583-589.

Nasrabadi, N., Soheili, Z.S., Bagheri, A., et al. (2018). The effects of electromagnetic fields on cultured human retinal pigment epithelial cells. Bioelectromagnetics. 39:585-594.

Nikolova, T., Czyz, J., Rolletschek, A., et al. (2005). Electromagnetic fields affect transcript levels of apoptosis-related genes in embryonic stem cell-derived neural progenitor cells. FASEB J. 19:1686-1688.

Okudan, N., Celik, I., Salbacak, A., et al. (2010). Effects of long-term 50 Hz magnetic field exposure on the micro nucleated polychromatic erythrocyte and blood lymphocyte frequency and argyrophilic nucleolar organizer regions in lymphocytes of mice. NeuroEndocrinol. Lett. 31:208-214.

Panagopoulos, D.J., Karabarbounis, A., Lioliousis, C. (2013). ELF alternating magnetic field decreases reproduction by DNA damage induction. Cell. Biochem. Biophys. 67:703-716.

Pesqueira, T., Costa-Almeida, R., Gomes, M.E. (2017). Uncovering the effect of low-frequency static magnetic field on tendon-derived cells: from mechanosensing to tenogenesis. Sci. Rep. 7:10948.

Pilger, A., Ivancsits, S., Diem, E., et al. (2004). No effects of intermittent 50 Hz EMF on cytoplasmic free calcium and on the mitochondrial membrane potential in human diploid fibroblasts. Radiat. Environ. Biophys. 43:203-207.

Potenza, L., Ubaldi, L., De Sanctis, R., et al. (2004a). Effects of a static magnetic field on cell growth and gene expression in Escherichia coli. Mutat. Res. 561:53-62.

Potenza, L., Cucchiarini, L., Piatti, E., et al. (2004b). Effects of high static magnetic field exposure on different DNAs. Bioelectromagnetics. 25:352-355.

Rageh, M.M., El-Gebaly, R.H., El-Bialy, N.S. (2012). Assessment of genotoxic and cytotoxic hazards in brain and bone marrow cells of newborn rats exposed to extremely low-frequency magnetic field. J. Biomed. Biotechnol. 2012:716023.

Rammal, M., Jebai, F., Rammal, H., et al. (2014). Effects of long-term exposure to RF/MW radiations on the expression of mRNA of stress proteins in *Lycospersicon esculentum.* WSEAS Transect Biol Biomed. 11:10-14.

Reese, J.A., Jostes, R.F., Frazier, M.E. (1998). Exposure of mammalian cells to 60-Hz magnetic or electric fields: analysis for DNA single-strand breaks. Bioelectromagnetics. 9:237-247.

Reyes-Guerrero, G., Guzmán, C., García, D.E., et al. (2010). Extremely low-frequency electromagnetic fields differentially regulate estrogen receptor-alpha and -beta expression in the rat olfactory bulb. Neurosci. Lett. 471:109-113.

Robison, J.G., Pendleton, A.R., Monson, K.O., et al. (2002). Decreased DNA repair rates and protection from heat induced apoptosis mediated by electromagnetic field exposure. Bioelectromagnetics. 23:106-112.

Ross, C.L., Pettenati, M.J., Procita, J., et al. (2018). Evaluation of cytotoxic and genotoxic effects of extremely low-frequency electromagnetic field on mesenchymal stromal cells. Glob. Adv. Health Med. 7:2164956118777472.

Ruiz-Gómez, M.J., Sendra-Portero, F., Martínez-Morillo, M. (2010) Effect of 2.45 mT sinusoidal 50 Hz magnetic field on Saccharomyces cerevisiae strains deficient in DNA strand breaks repair. Int. J. Radiat. Biol. 86:602-611.

Sadri, M., Abdolmaleki, P., Abrun, S., et al. (2017). Static magnetic field effect on cell alignment, growth, and differentiation in human cord-derived mesenchymal stem cells. Cell. Mol. Bioeng. 10:249-262.

Sanie-Jahromi, F., Saadat, I., Saadat, M. (2016). Effects of extremely low frequency electromagnetic field and cisplatin on mRNA levels of some DNA repair genes. Life Sci. 166:41-45.

Sanie-Jahromi, F., Saadat, M. (2017). Different profiles of the mRNA levels of DNA repair genes in MCF-7 and SH-SY5Y cells after treatment with combination of cisplatin, 50-Hz electromagnetic field and bleomycin. Biomed. Pharmacother. 94:564-568.

Sanie-Jahromi, F., Saadat, M. (2018). Effects of electromagnetic field, cisplatin and morphine on cytotoxicity and expression levels of DNA repair genes. Mol. Biol. Rep. 45:807-814.

Sarimov, R., Alipov, E.D., Belyaev, I.Y. (2011). Fifty hertz magnetic fields individually affect chromatin conformation in human lymphocytes: dependence on amplitude, temperature, and initial chromatin state. Bioelectromagnetics. 32:570-579.

Scarfi, M.R., Sannino, A., Perrotta, A., et al. (2005). Evaluation of genotoxic effects in human fibroblasts after intermittent exposure to 50 Hz electromagnetic fields: a confirmatory study. Radiat. Res. 164:270-276.

Scassellati Sforzolini, G., Moretti, M., Villarini, M.,et al. (2004). [Evaluation of genotoxic and/or co-genotoxic effects in cells exposed in vitro to extremely-low frequency electromagnetic fields]. Ann Ig. 16(1-2):221-240. [Article in Italian]

Schmitz, C., Keller, E., Freuding, T., et al. (2004). 50-Hz magnetic field exposure influences DNA repair and mitochondrial DNA synthesis of distinct cell types in brain and kidney of adult mice. Acta Neuropathol. (Berl). 107:257-264.

Seong, Y., Moon, J., Kim, J. (2014). Egr1 mediated the neuronal differentiation induced by extremely low-frequency electromagnetic fields. Life Sci. 102:16-27.

Shen, Y., Xia, R., Jiang, H., et al. (2016). Exposure to 50 Hz-sinusoidal electromagnetic field induces DNA damage-independent autophagy. Int. J. Biochem. Cell. Biol. 77(Pt A):72-79.

Shokrollahi, S., Ghanati, F., Sajedi, R.H., et al. (2018). Possible role of iron containing proteins in physiological responses of soybean to static magnetic field. J. Plant Physiol. 226:163-171.

Singh, N., Lai, H. (1998). 60 Hz magnetic field exposure induces DNA crosslinks in rat brain cells. Mutat. Res. 400:313-320.

Skyberg, K., Hansteen, I.L., Vistnes, A.I. (2001). Chromosomal aberrations in lymphocytes of employees in transformer and generator production exposed to electromagnetic fields and mineral oil. Bioelectromagnetics. 22:150-160.

Solek, P., Majchrowicz, L., Bloniarz, D., et al. (2017). Pulsed or continuous electromagnetic field induce p53/p21-mediated apoptotic signaling pathway in mouse spermatogenic cells in vitro and thus may affect male fertility. Toxicology. 382:84-92.

Song, K., Im, S.H., Yoon, Y.J., et al. (2018). A 60 Hz uniform electromagnetic field promotes human cell proliferation by decreasing intracellular reactive oxygen species levels. PLoS One. 13:e0199753.

Stankevičiūtė, M., Jakubowska, M., Pažusienė, J., et al. (2019). Genotoxic and cytotoxic effects of 50 Hz 1 mT electromagnetic field on larval rainbow trout (*Oncorhynchus mykiss*), Baltic clam (*Limecola balthica*) and common ragworm (*Hediste diversicolor*). Aquat. Toxicol. 208:109-117.

Stronati, L., Testa, A., Villani, P., et al. (2004). Absence of genotoxicity in human blood cells exposed to 50 Hz magnetic fields as assessed by comet assay, chromosome aberration, micronucleus, and sister chromatid exchange analyses. Bioelectromagnetics. 25:41-48.

Sun, C., Wei, X., Yimaer, A., et al. (2018). Ataxia telangiectasia mutated deficiency does not result in genetic susceptibility to 50 Hz magnetic fields exposure in mouse embryonic fibroblasts. Bioelectromagnetics. 39:476-484.

Sun, L., Li, X., Ma, H., et al. (2019). Global gene expression changes reflecting pleiotropic effects of *Irpex lacteus* induced by low-intensity electromagnetic field. Bioelectromagnetics. 40:104-117.

Sun, R.G., Chen, W.F., Qi, H., et al. (2012). Biologic effects of SMF and paclitaxel on K562 human leukemia cells. Gen. Physiol. Biophys. 31:1-10.

Suzuki, Y., Ikehata, M., Nakamura, K., et al. (2001). Induction of micronuclei in mice exposed to static magnetic fields. Mutagenesis16:499-501.

Svedenstal, B.M., Johanson, K.J., Mild, K.H. (1999). DNA damage induced in brain cells of CBA mice exposed to magnetic fields. In Vivo. 13:551-552..

Szerencsi, Á., Kubinyi, G., Váliczkó, É., et al. (2013). DNA integrity of human leukocytes after magnetic resonance imaging. Int. J. Radiat. Biol. 89:870-876.

Teodori, L., Giovanetti, A., Albertini, M.C., et al. (2014). Static magnetic fields modulate X-ray-induced DNA damage in human glioblastoma primary cells. J. Radiat. Res. 55:218-227.

Testa, A., Cordelli, E., Stronati, L., et al. (2004). Evaluation of genotoxic effect of low level 50 Hz magnetic fields on human blood cells using different cytogenetic assays. Bioelectromagnetics. 25:613-619.

Tiwari, R., Lakshmi, N.K., Bhargava, S.C., et al. (2015). Epinephrine, DNA integrity and oxidative stress in workers exposed to extremely low-frequency electromagnetic fields (ELF-EMFs) at 132 kV substations. Electromagn. Biol. Med. 34:56-62.

Udroiu, I., Cristaldi, M., Ieradi, L.A., et al. (2006). Clastogenicity and aneuploidy in newborn and adult mice exposed to 50 Hz magnetic fields. Int. J. Radiat. Biol. 82:561-567.

Udroiu, I., Antoccia, A., Tanzarella, C., et al. (2015). Genotoxicity induced by foetal and infant exposure to magnetic fields and modulation of ionising radiation effects. PLoS One. 10:e0142259.

Verschaeve, L., Anthonissen, R., Grudniewska, M., et al. (2011). Genotoxicity investigation of ELF-magnetic fields in Salmonella typhimurium with the sensitive SOS-based VITOTOX test. Bioelectromagnetics. 32:580-584.

Verschaeve, L., Wambacq, S., Anthonissen, R., et al. (2016). Co-exposure of ELF-magnetic fields and chemical mutagens: An investigation of genotoxicity with the SOS-based VITOTOX test in *Salmonella typhimurium*. Mutat. Res. 795:31-35.

Villarini, M., Moretti, M., Scassellati-Sforzolini, G., et al. (2006). Effects of co-exposure to extremely low frequency (50 Hz) magnetic fields and xenobiotics determined in vitro by the alkaline comet assay. Sci. Total Environ. 361:208-219.

Villarini, M., Ambrosini, M.V., Moretti, M., et al. (2013). Brain hsp70 expression and DNA damage in mice exposed to extremely low frequency magnetic fields: a dose-response study. Int. J. Radiat. Biol. 89:562-570.

Villarini, M., Dominici, L., Fatigoni, C., et al. (2015). Primary DNA damage in welders occupationally exposed to extremely-low-frequency magnetic fields (ELF-MF). Ann. Ig. 27:511-519.

Villarini, M., Gambelunghe, A., Giustarini, D., et al. (2017). No evidence of DNA damage by co-exposure to extremely low frequency magnetic fields and aluminum on neuroblastoma cell lines. Mutat. Res. 823:11-21.

Wahab, M.A., Podd, J.V., Rapley, B.I., et al. (2007). Elevated sister chromatid exchange frequencies in dividing human peripheral blood lymphocytes exposed to 50 Hz magnetic fields. Bioelectromagnetics. 28:281-288.

Wang, Y., Liu, X., Zhang, Y., et al. (2019). Exposure to a 50 Hz magnetic field at 100 μT exerts no DNA damage in cardiomyocytes. Biol. Open. 8 pii: bio041293.

Wang, Y., Sun, Y., Zhang, Z., et al. (2020). Enhancement in the ATP level and antioxidant capacity of *Caenorhabditis elegans* under continuous exposure to extremely low-frequency electromagnetic field for multiple generations. Int. J. Radiat. Biol. doi: 10.1080/09553002.2020.1828657. (Online ahead of print).

Wang, Z., Sarje, A., Che, P.L., et al. (2009). Moderate strength (0.23-0.28 T) static magnetic fields (SMF) modulate signaling and differentiation in human embryonic cells. BMC Genomics. 10:356.

Williams, P.A., Ingebretsen, R.J., Dawson, R.J. (2006). 14.6 mT ELF magnetic field exposure yields no DNA breaks in model system Salmonella, but provides evidence of heat stress protection. Bioelectromagnetics. 27:445-450.

Wilson, J.W., Haines, J., Sienkiewicz, Z., et al. (2015). The effects of extremely low frequency magnetic fields on mutation induction in mice. Mutat. Res. 773:22-26.

Winker, R., Ivancsits, S., Pilger, A., et al. (2005). Chromosomal damage in human diploid fibroblasts by intermittent exposure to extremely low-frequency electromagnetic fields. Mutat. Res. 585:43-49.

Wolf, F.I., Torsello, A., Tedesco, B., et al. (2005). 50-Hz extremely low frequency electromagnetic fields enhance cell proliferation and DNA damage: possible involvement of a redox mechanism. Biochim. Biophys. Acta. 1743:120-129.

Yagci, F., Kesim, B. (2016). Cytotoxic and genotoxic effects on gingival fibroblasts from static magnetic fields produced by dental magnetic attachments. Gerodontology. 33:421-427.

Yaguchi, H., Yoshida, M., Ejima, Y., et al. (1999). Effect of high-density extremely low frequency magnetic field on sister chromatid exchanges in mouse m5S cells. Mutat. Res. 440:189-194.

Yaguchi, H., Yoshida, M., Ding, G.R., et al. (2000). Increased chromatid-type chromosomal aberrations in mouse m5S cells exposed to power-line frequency magnetic fields. Int. J. Radiat. Biol. 76:1677-1684.

Yao, L., Li, Y., Knapp, J., et al. (2015). Exploration of molecular pathways mediating electric field-directed Schwann cell migration by RNA-seq. J Cell Physiol. 230:1515-1524.

Yin, C., Luo, X., Duan, Y., et al. (2016). Neuroprotective effects of lotus seedpod procyanidins on extremely low frequency electromagnetic field-induced neurotoxicity in primary cultured hippocampal neurons. Biomed. Pharmacother. 82:628-639.

Yokus, B., Cakir, D.U., Akdag, M.Z., et al. (2005). Oxidative DNA damage in rats exposed to extremely low frequency electro magnetic fields. Free Radic. Res. 39:317-323.

Yokus, B., Akdag, M.Z., Dasdag, S., et al. (2008). Extremely low frequency magnetic fields cause oxidative DNA damage in rats. Int. J. Radiat. Biol. 84:789-795.

Yoon, H.E., Lee, J.S., Myung, S.H., et al. (2014). Increased γ-H2AX by exposure to a 60-Hz magnetic fields combined with ionizing radiation, but not hydrogen peroxide, in non-tumorigenic human cell lines. Int. J. Radiat. Biol. 90:291-298.

Yuan, L.Q., Wang, C., Lu. D.F., et al. (2020). Induction of apoptosis and ferroptosis by a tumor suppressing magnetic field through ROS-mediated DNA damage. Aging (Albany NY). doi: 10.18632/aging.102836. [Epub ahead of print]

Zendehdel, R., Yu, I.J., Hajipour-Verdom, B., et al. (2019). DNA effects of low level occupational exposure to extremely low frequency electromagnetic fields (50/60 Hz). Toxicol. Ind. Health. 35:424-430.

Zhang, H., Cheng, Y., Luo, X., et al. (2016). Protective effect of procyanidins extracted from the lotus seedpod on immune function injury induced by extremely low frequency electromagnetic field. Biomed. Pharmacother. 82:364-372.

Zhang, Y., Zhang, D., Zhu, B., et al. (2016). Effects of dietary green tea polyphenol supplementation on the health of workers exposed to high-voltage power lines. Environ. Toxicol. Pharmacol. 46:183-187.

Zheng, L., Zhang, L., Chen, L., et al. (2018). Static magnetic field regulates proliferation, migration, differentiation, and YAP/TAZ activation of human dental pulp stem cells. J. Tissue Eng. Regen. Med. 12:2029-2040.

Zhu, K., Lv, Y., Cheng, Q., et al. (2016). Extremely low frequency magnetic fields do not induce DNA damage in human lens epithelial cells in vitro. Anat. Rec. (Hoboken). 299:688-697.

Zmyslony, M., Palus, J., Jajte, J., et al. (2000). DNA damage in rat lymphocytes treated in vitro with iron cations and exposed to 7 mT magnetic fields (static or 50 Hz). Mutat. Res. 453:89-96.

Zmyslony, M., Palus, J., Dziubaltowska, E., et al. (2004). Effects of in vitro exposure to power frequency magnetic fields on UV-induced DNA damage of rat lymphocytes. Bioelectromagnetics. 25:560-562.

Supplement 3

Gene expressions after RFR and static/ELF EMF exposure (literature up to January 2021)

RFR	***Exposure effects***
Akhavan-Sigari et al. (2014)	Increased risk for the mutant type of p53 gene expression in the peripheral zone of the glioblastoma, and that this increase was significantly correlated with shorter overall survival time.
Beaubois et al. (2007)	Accumulation of basic leucine-zipper transcription factor (bZIP) mRNA in the exposed terminal leaf of tomato plant.
Belyaev et al. (2006)	Expression of genes encode proteins with diverse functions including neurotransmitter regulation, blood-brain barrier (BBB), and melatonin production in rat brain.
Buttiglione et al. (2007)	Affected both Egr-1 gene expression and cell regulatory functions, involving apoptosis inhibitors like Bcl-2 and surviving in human neuroblastoma cells.
Cervellati et al. (2013)	Induced 17-β-estradiol modulates connexins and Integrins as well as estrogen recptor (ER-β) expression in trophoblast cells, suggesting an influence on cell differentiation and migration.
Chen et al. (2012)	Expression was limited to only a very small number of genes in yeast. (Expressions of structural maintenance of chromosomes 3 (SMC3) and aquaporin 2 (AQY2 (m)) while halotolerance protein 9 (HAL9), a kinase 1 (YAK1) and one function-unknown gene showed opposite changes in expression.
Del Vecchio et al. (2009)	Increased expression of beta-thymosin gene, a cytoskeleton regulating factor in murine cortical neurons, correlated to reduced number of neurites generated.
Deshmukh et al. (2015)	Increased heat shock protein 70 (HSP70) in rat brain.
Eker et al. (2018)	Caspase-3 and p38 mitogen-activated protein kinase (p38MAPK) (a kinase responsive to stress stimuli, and involved in cell differentiation, apoptosis and autophage) gene expressions were significantly up-regulated in the ocular tissues of rat.
Engelmann et al. (2008)	Significant changes in transcription of 10 genes in Arabidopsis thaliana (thale cress) cells.
Fragopoulou et al. (2018)	Expression of 178 genes changed significantly mouse hippocampus
Franzellitti et al. (2008)	Levels of the inducible HSP70C transcript were significantly enhanced after 24 h exposure to GSM-217Hz signals and reduced after 4 and 16 h exposure to GSM-Talk signals in human trophoblasts.
Ghatei et al. (2017)	No effect on expression level of bcl-2 and p53 genes, but gene expression level of *bax* decreased and gene expression level of *p21* increased in cerebellum of mice exposed pre-and postnatally to RFR.
Gökçek-Saraç et al. (2020)	Decreased RNA expressions of acetylcholinesterase (AChE), choline acetyltransferase (ChAT), and vesicular acetylcholine transporter (VAChT)

	in the hippocampus of rats exposed to 2100 MHz RFR.
Gulati et al. (2018)	A significant association of genetic polymorphism of antioxidant genes (for MnSOD and CAT) with oxidative damage has been observed in human population exposed to radiations emitted from mobile towers.
Habauzit et al. (2014)	7 genes were differentially expressed in human keratinocytes, associated to the cellular response to hyperthermia.
Hao et al. (2010)	Significant induced phosphorylation of STAT3, increased transcription levels of the inflammation-associated genes, iNOS and TNF-alpha murine N9 microglial cells. (Signal transducer and activator of transcription 3 (STAT3) is a transcription activator that mediates the expression of a variety of genes in response to cell stimuli, and thus plays a key role in many cellular processes such as cell growth and apoptosis.)
He et al. (2016)	Mouse bone marrow stromal cells showed increased PARP-1 mRNA expression.(PARP-1 involves in differentiation, tumor transformation and DNA repair.)
He et al. (2017)	Mouse bone marrow stromal cells showed increased PARP-1 mRNA expression. Gamma radiation decreased RFR-induced PARP-1 expression.
Karaca et al. (2012)	Decreased STAT3 expression in moue brain. (STAT3 acts as transcription activator).
Kumari et al. (2017)	Increased expression of the pro-inflammatory cytokine tumor necrosis factor alpha mRNA in the hippocampal region.
Kumar, R. (2020)	Altered expression of DNA (epigenetic) methylating enzymes, DNA methyltransferase1 (DNMT1) and histone methylating enzymes euchromatic histone methylthransferase1 (EHMT1) in hippocampus.
Jeong et al. (2020)	Increased expression of Epha8 and Wnt6 genes in the hippocampi of mice. (Both genes are involved in development, particularly, Epha8 coded protein mediates developmental events in the nervous system in axonal guidance).
Lameth et al. (2020)	Altered gene expressions in rat cerebral cortex in an acute neuroinflammation. Gene responses to RFR can differ according to pathologies affecting the CNS.
Le Quément et al.(2012)	Human skin cells showed differential expression of genes involved in functions such as cardiovascular development, facilitate pathogen recognition by macrophages, inhibition of angiogenesis, nonspecific ion channels, etc.
Lee et al. (2005)	Many genes were affected in human HL60 cells. Apoptosis-related genes were among the upregulated ones and the cell cycle genes among the downregulated ones.
Li et al. (2020)	Offspring of pregnant female rats exposed to RFR showed differential expression of methyl-D-aspartate receptors

	(NMDARs) genes in the hippocampus.
Lin et al. (2016)	Upregulated the expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle, but not the glycolysis pathway. Transcription levels of 29 genes were upregulated and 24 genes were downregulated.
López-Martín et al. (2009)	c-Fos expressions in brain of picrotoxin-treated and untreated rats.
Manta et al. (2017)	168 genes differentially expressed in the house fly Drosophila melanogaster), associated with multiple and critical biological processes, such as basic metabolism and cellular subroutines related to stress response and apoptotic death.
Martin et al. (2020)	Four different types of human keratinocytes showed different patterns of expression of ADAMTS6, IL7R, and NOG genes
Megha et al. (2015)	Downregulation in mRNA expression of enzymes involved in monoamine transmitter synthesis in rat hippocampus.
Mildažienė et al. (2019)	Leaves from exposed common sunflower (Helianthus annus L.) seeds showed gene expression mostly of proteins involved in photosynthetic processes and their regulation.
Millenbaugh et al. (2008)	Genes associated with regulation of transcription, protein folding, oxidative stress, immune response, and tissue matrix turnover were affected in rat skin.
Nittby et al. (2008)	Altered gene expression in both cortex and hippocampus of the rat: extracellular region, signal transducer activity, intrinsic to membrane, and integral to membrane.
Nylund and Leszczynski (2006)	Gene and protein expressions altered differentially in two human endothelial cell lines.
Ohtani et al. (2016)	Heat-shock proteins (Hsp) and heat-shock transcription factors (Hsf) gene expression levels were significantly upregulated in the cerebral cortex and cerebellum of the rat.
Ohtani et al. (2019)	No change in transcription gene expression in brain and liver of mice exposed to a 85-kHZ field.
Pacini et al. (2002)	Human skin fibroblasts showed increased expression of mitogenic signal transduction genes (e.g., MAP kinase kinase 3, G2/mitotic-specific cyclin G1), cell growth inhibitors (e.g., transforming growth factor-beta), and genes controlling apoptosis (e.g., bax).
Qin et al. (2018)	Altered the expression of genes involved in testosterone synthesis (*Star, P450scc, P450c17 and 3β-Hsd*) in mouse testicular tissue.
Qin et al. (2019)	Exposed Leydig cells showed downregulated of testosterone synthase genes (*Star, Cyp11a1*, and *Hsd-3β*) and clock genes

	(*Clock, Bmal1*, and *Rorα*),
Rammal et al. (2014)	Increased expression of two wound-plant gene in tomato.
Remondini et al. (2006)	Different human cell types responded differently in gene expression. Affected gene families did not point towards a stress response, but suggested upreglulating of cellular metabolism.
Romano-Spica et al. (2000)	Overexpression of the proto-oncogene ets1 mRNA in Jurkat T-lymphoblastoid and Leydig TM3 cell lines
Roux et al. (2006)	Leaves of tomato plants showed increased stress-related transcripts (calmodulin, protease inhibitor and chloroplast mRNA-binding protein).
Roux et al. (2008)	Tomato plant showed increase in stress-related mRNA (calmodulin, calcium-dependent protein kinase and proteinase inhibitor), similar to wound responses.
Said-Salman et al. (2019)	101 genes were differentially expressed in Escherichia coli. Up-regulated genes are involved in metabolic pathways, transposition, response to stimuli, motility, chemotaxis, and cell adhesion, while the down-regulated genes are associated with metabolic pathways and localization of ions and organic molecules.
Silva et al. (2016)	No effect on expressions of Ki-67 (involved in cell proliferation) p53 (tumor suppression) HSP-70 (stress biomarker), and reactive oxygen species in human thyroid cells.
Soubere Mahamoud et al. (2016)	Exposed human keratinocytes treated with the glycolysis inhibitor, 2-deoxyglucose showed changes in genes encode transcription factors or inhibitors of cytokine pathways,
Souza et al. (2014)	Cells from oral mucosa of individual used cellular phones more than 5 h/week high number of broken egg which may be associated with gene amplification.
Sun Y. et al. (2017)	Decreased gene expression in mitochondria of HL-60 human leukemia cells. Free radicals involved.
Tohidi et al. (2020)	Apoptotic genes Bax and Bc12 expression in the hippocampus were upregulated in mice exposed to RFR from a cell phone jammer for 1, 2, twice a day for 30 days and down-regulated with longer exposure schedule.
Trivino Pardo et al. (2012)	Gene expression affected in acute T-lymphoblastoid leukemia cells. Genes which act as sensors of DNA damage (*ATM, RAD17,RAD50, and PRKDC*) are activated. This over-expression could produce a signal cascade that causes the activation of the main DNA repair signaling. Some of the genes that were defined as essentials in double-strands repair

	(*BRCA1, LIG4, XRCC2*) and single-chain DNA repair process (*XPC, MSH5*) were found to over-express. More cells in S-phase.
Vafaei et al. (2020)	Increased superoxide dismutase, CDKN1A, GADD45a, Bax mRNA, and decreased Bcl-2 mRNA. (CDKN1A and GADD45a are involved in DNA repair and cellular responses to stressors.)
Valbonesi et al. (2014)	HSP70 transcription was significantly increased in rat neuronal-like PC12 cells.
Varghese et al. (2018)	Increased caspase-3 gene expression in brain tissues of rats exposed to 2450 MHz RFR
Vian et al. (2006)	Rapid induction of mRNA encoding the stress-related bZIP transcription factor in plants.
Yan et al. (2008)	Brain of exposed rat showed mRNA up-regulation of several injury-associated proteins. RFR exposure may result in cumulative injuries that could eventually lead to clinically significant neurological damage.
Yao et al. (2004)	Rabbit lens epithelial cells showed increased expression of P27kip1 protein, also G/G1 cell cycle arrest. (p27kip1 is a cyclin-dependent kinase inhibitor which binds to cyclinE/cdk2, blocking the G1/S transition.)
Zhang et al. (2008)	Primary culture neurons showed gene up- and down-regulation. Genes are associated with multiple cellular functions (cytoskeleton, signal transduction pathway, metabolism, etc.)
Zhao et al. (2007)	Up-regulation of caspase-2, caspase-6 genes occurred in both GSM 1900-MHz "on" and "stand-by" modes in neurons, but only in "on" mode in astrocytes. Additionally, astrocytes showed up-regulation of the Bax gene.
Static/extremely-low frequency EMF	
Agliassa et al. (2018)	Near-null MF condition (i.e., <100 nT) delayed transition to flowering in Arabidopsis thaliana and changes in expression of several genes in leaf and floral meristem.
Ashta et al. (2020)	Temozolomide (TMZ) with static MF or ELF MF (10 Hz) together increased p53 protein expression in the human glioblastoma cell line (A172) and increased cytotoxicity.
Baraúna et al. (2015)	The bacteria Chromobacterium violaceum, exposed to ELF MF, showed differential expression of 5 proteins. Expression of the protein, DNA-biding stress protein, may help to prevent DNA damage.
Bertea et al. (2015)	Exposing *Arabidopsis thaliana* to artificially reversed

	geomagnetic field conditions induced gene expressions.
Chen et al. (2008)	Human breast cancer MCF-7 cells exposed to a 50-Hz MF induced expression of thee responsive genes.
Chen et al. (2020)	Human choriocarcinoma cells exposed to DC electric field showed increased gene expressions of ErbB and HIF-1 signaling pathways
Collard et al. (2013)	Epidermis cultures harvested from human abdominoplasty exposed to ELF electric fields induced expression of various genes. Some genes are involved in cell proliferation or differentiation, mitosis, cell cycle, or in the DNA replication transcription and translation.
Consales et al. (2018)	Exposure to a 50-Hz magnetic field in vitro. We demonstrate that ELF-MFs drive an early reduction of the expression level of miR-34b and miR-34c in SH-SY5Y human neuroblastoma cells, as well as in mouse primary cortical neurons, by affecting the transcription of the common pri-miR-34. Data also indicate epigenetic control of gene expression in vitro and shed light on the possible mechanism(s) producing detrimental effects and predisposing neurons to degeneration.
Cuccurazzu et al. (2010)	Exposure to a 50-Hz MF in vivo induced increases in the transcription of pro-neuronal genes (Mash1, NeuroD2, Hes1) and genes encoding Ca(v)1.2 channel α(1C) subunits in the hippocampus of the mouse. Hippocampal neurogenesis also observed.
Del Re et al. (2006)	ELF-MF influenced the synthesis of heat shock proteins in E. coli in a way that critically depends on the signal characteristics (static or pulsed MF).
Di Campli et al. (2010)	*Helicobacter pylori* biofilm expose to a 50-Hz EMF showed amiA gene expression and decreased cell adhesion. (AmiA protein is responsible for transition of H. pylori from bacillary to coccoid forms. These coccoid forms can escape detection by the immune system and therefore could participate in the persistence of *H. pylori* infection during the lifetime of its human host.)
Dong et al. (2019)	16 T static magnetic field markedly blocked the expression of osteoclast-associated transcription factors and osteoclast marker genes and inhibited iron absorption and iron storage-related protein expression.
Fan et al. (2015)	Rat bone marrow derived-mesenchymal stem cells and mesenchymal stem cells exposed to a 50-Hz EMF induced expressions of various genes. Expressions of hematopoietic growth factors increase proliferation and migration of macrophagocytes.
Fan et al. (2018)	Static magnetic field up-regulated the expression of stress gene (dnaK) and virulence genes (efaA and ace).
Fedrowitz and Loscher	50-Hz MF-exposed F344 rat breast tissue showed alterations in

(2012)	gene expression, which were absent in Lewis rats.
Frisch et al. (2013)	Rat primary fibroblasts exposed to a 10-Hz electric fields induced HSP70 expression.
Heredia-Rojas et al. (2010)	"Electromagnetic field" plasmid transfected into INER-37 and RMA E7 cell lines exposed to a 60-Hz MF. An increased luciferase gene expression was observed in INER-37 cells but had no effect on the RMA E7 cell line.
Jin et al. (2019)	Arabidopsis seedling exposed to static magnetic field showed increased auxin (a plant growth hormone) from expression of PIN3 and AUX1 genes in root tips; cryptochromes (cry1 and cry 2 genes) are also involved.
Ki et al. (2020)	Human hair follicle dermal papilla cells exposed to a 70-Hz EMF enhance cell activation and proliferation via the GSK-3β/ERK/Akt signaling pathway. Various genes were activated.
Kim et al. (2010)	Human normal and cancer cells exposed repeatedly to a 60-Hz MF showed p38 gene expression and induction of checkpoint kinase 2 critical to the DNA damage checkpoint pathway.(P38 mitogen-activated protein kinases are a class of mitogen-activated protein kinases (MAPKs) that are responsive to stress stimuli, and are involved in cell differentiation, apoptosis and autophagy.)
Kimsa-Dudek et al. (2018)	Static magnetic field attenuated expression of antioxidant defense genes (SOD1, PLK3, CLN8, XPA, HAO1) induced by sodium fluoride.
Kimsa-Dudek et al. (2020)	Exposure of human fibroblast cultures that had been co-treated with fluoride ions to a static MF caused specific genes expression that were involved in apoptosis.
Kimura et a. (2008)	Caenorhabditis elegans exposed to high intensity (2, 3, 5 T) static magnetic fields showed induction of genes involved in motor activity, actin binding, cell adhesion, and cuticles; also upregulation of hsp (heat shock protein) 12 and 16 family genes.
Lacy-Hulbert et al. (1995)	No effect on MYC and beta-actin gene expression in human leukemic cells.
Laramee et al. (2014)	Transfected rat primary cells in monolayer were exposed to a static MF caused HSP expression.
Lee et al. (2010)	C. elegans exposed to a 200 mT static magnetic field showed up-regulation of genes involved in development and aging (clk-1,unc-3, age-1,daf-2, lim-7).
Lee et al. (2016)	MCF7 cells showed up-regulation of PMAIP1 gene (gene involved in apoptosis) after 60-Hz magnetic field exposure.
Leone et al. (2014)	ELF-EMF enhanced proliferation and neuronal differentiation of hippocampal neural stem cells by regulation of epigenetic mechanisms leading to pro-neuronal gene expression.
Li et al. (2013)	Male Drosophila melanogaster exposed to ELF-EMF showed changes in gene expression. Differentially expressed genes following short-term exposures were involved in metabolic processes, cytoskeletal organization, mitotic spindle organization,

	cell death, protein modification and proteolysis. Long-term exposure let to changes in expression of genes involved in metabolic processes, response to stress, mitotic spindle organization, aging, cell death, and cellular respiration.
Li et al. (2014)	Zebra fish embryos exposed to a 50-Hz MF showed transcription of apoptosis-related genes (caspase-3, caspase-9) was significantly upregulation.
Li et al. (2015)	Rat oligodendrocyte precursor cells exposed to DC electric field showed upregulated mitogen-activated protein kinase pathway that signals cell migration and downregulation of differentially expressed genes in chemotaxis.
Li et al. (2019)	Pulsed EMF (20 Hz) increased expression of insulin growth factor 2 (IFG-2) in the hippocampus of streptozotocin-induced dementia rats.
Lin et al. (2016)	Budding yeast exposed to a 50-Hz EMF caused upregulation expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle, but not the glycolysis pathway. (A response to environmental stress.)
Lupke et al. (2006)	Human umbilical cord blood-derived monocytes exposed to ELF-MF caused expression of 5 genes.
Ma et al. (2014)	Mouse embryonic neural stem cells exposed to a 50-HZ EMF induced expression of genes regulating neuronal differentiation although cell proliferation and the percentages of neurons and astrocytes differentiated from eNSCs were not affected which might be compensation by post-transcriptional mechanisms to support cellular homeostasis.
Mahaki et al. (2019)	A 50-Hz EMF reduced the expression levels of c-Maf, STAT6, and RORα genes in the spleen of rats.
Mahmoudinasab and Saadat (2016)	Human MCF-7 breast cancer cells exposed to a 50-Hz EMF showed decreased NQO1 and increased NQO2 gene expression. (NQO1 and NQO2 are detoxification enzymes).
Mahmoudinasab and Saadat (2018a)	Patterns of up-regulation of antioxidant genes are different between MCF-7 and SH-SY5Y cells exposed to an intermittent 50-Hz EMF.
Mahmoudinasab and Saadat (2018b)	SH-ST5Y cells exposed to a 50-Hz EMF. NQO1 mRNA level decreased in the "15 min field-on/15 min field-off" condition, the expression level of NQO2 was increased.
Mahmoudinasab et al. (2016)	Human MCF-7 breast cancer cells exposed to a 50-Hz EMF showed up and down regulations of 7 antioxidant genes.
Manzella et al. (2015)	50-Hz magnetic field affected in human dermal fibroblasts expression of clock genes: BMAL1, PER2, PER3, CRY1, and CRY2.
Mastrodonato et al. (2018)	50-HZ EMF exposure increased Wnt3 (neurogenesis gene) mRNA expression in subventricular zone of the lateral ventricle of mice.
Moraveji et al. (2016)	50-Hz EMF activated MAP2 gene in dermal papilla mesenchymal

	cells.
Mouhoub et al. (2017)	Static magnetic field enhanced expression of gene involved in the production of acdiolipin and phosphatidylethanolamine in Salmonella hadar.
Nasrabadi et al. (2018)	In neonatal human retinal pigment epithelial cells exposed to pulsed 50-Hz EMF, gene expressions of NES, RPE65, and PAX6 were decreased. (NES gene encodes nestin involved in radial growth of neurons. The RPE65 gene provides instructions for making a protein that is essential for normal vision**.** PAX6 acts as a "master control" gene for the development of eyes and other sensory organs.)
Nikolova et al. (2005)	Mouse embryonic stem cells exposed to a 50-Hz EMF changed transcript levels of the apoptosis-related bcl-2, bax, and cell cycle regulatory "growth arrest DNA damage inducible" GADD45 genes.
Pesqueira et al. (2017)	Short term exposure (8 h) upregulated the expression of tendon-associated genes SCX, COL1A1, TNC and DCN. Long-term exposure (8 h every 24 h up to 14 days) significantly upregulated COL1A1, COL3A1 and TNC.
Potenza et al. (2004a)	Escherichia coli exposed to static magnetic field showed three cDNAs to be expressed only in the exposed cells, whereas one cDNA was more expressed in the controls.
Reyes-Guerrero et al. (2010)	ELF EMF exerted a biphasic effect on female olfactory bulb estrogen receptor-beta mRNA gene expression, which increased during diestrous and decreased during estrous. No effect on estrogen receptor-alpha gene expression and in male rats.
Sadri et al. (2017)	Static magnetic field decreased expression of Sox-2, Nanong, and Oct-4 genes in human mesenchymal stem cells derived from newborn umbilical cords.
Sanie-Jahromi et al. (2016)	Human MCF-7 breast cancer cells and neuroblastoma SH-SY5Y cells exposed to a 50-Hz EMF had mostly down regulation of 7 DNA repair genes in MCF-7 cells. Co-treatment with cisplatin and EMF can enhance down-regulation of the genes involved in non-homologous end-joining pathway in both cell types.
Sanie-Jahromi et al. (2017)	ELF-EMF enhanced the effects of cisplatin + bleomycin on viability of MCF-7 cells, while SH-SY5Y cells were not affected. MCF-7 and SH-SY5Y cells showed non-random disagreement in DNA repair gene expression in these conditions.
Sanie-Jahromi and Saadat (2018)	MCF-7 and SH-SY5Y cells were treated with morphine and then exposed to a 50-Hz EMF. Non-homologous end joining (NHEJ) related genes were significantly decreased in co-treatment of cisplatin and "morphine + EMF".
Seong et al. (2014)	Human bone marrow-mesenchymal stem cells were exposed to a 50-Hz EMF. Analysis of neurons derived from these cells showed that early growth response protein 1 (Egr1) is one of the key transcription factors in ELF-EMF-induced neuronal

	differentiation.
Shokrollahi et al. (2018)	Soybean plants exposed to static magnetic field had decreased gene expression of Fe transporter at 20 mT. Opposite response observed at 30 mT. The results suggest that SMF triggered a signaling pathway that is mediated by iron.
Wang et al. (2009)	Human embryonic cells exposed to static magnetic field showed a short-term (<24 h) activation of IL-6 involved the coordinate up-regulation of toll-like receptor-4 (TLR4) with complementary changes to NEU3 and ST3GAL5 that reduced ganglioside GM3 and augmented the activation of TLR4 and IL-6. Loss of GM3 also provided a plausible mechanism for the attenuation of cellular responses to SMF that occurred over longer exposure periods.
Wang et al. (2020)	Caenorhabditis elegans exposed to 50-Hz, 3 mT EMF for 15 generations showed enhanced up-regulations of genes encoding ATPase and superoxde dismutase.
Yao et al. (2015)	Rat Schwann cells exposed to DC electric field showed expression of genes participate in multiple cellular signaling pathways involved in the regulation of cell migration, including pathways of regulation of actin cytoskeleton, focal adhesion, and PI3K-Akt.
Zhang H et al. (2016)	Mice exposed to a 50-Hz EMF showed a significant suppression in Bcl-2 expression and increase in Bax, Caspase-3 and Caspase-9 expression in splenic cells. G0/G1 cycle arrest observed.
Zhao et al. (2020)	Escherichia coli exposed to 3.1 THz RFR for 8 h showed increased plasmid copy number and protein expression.
Zheng et al. (2018)	Static magnetic field increased expression of several growth factors, migration genes, and upregulated the two YAP/TAZ-regulated genes in human dental pulp mesenchymal stem cells.

Supplement 4

Genetic effects at low intensity exposure to RFR and static/ELF EMF (literature up to January 2021)

	Power density/SAR (<0.1 W/Kg) or magnetic flux density	**Effects observed**
RFR studies		
Aitken et al. (2005)	Mice to 900-MHz RFR for 7 days at 12 h/day; SAR 0.09 W/kg	Mitochondrial genome damage in epididymal spermatozoa.
Akdag et al. (2016)	Male Wistar-Albino rats to 2400 MHz RFR from a Wi-Fi signal generator for a year; SAR 0.000141 (min)-0.007127 (max) W/kg	DNA damage in testes.
Alkis et al. (2019a)	Rats exposed to 900 MHz (brain SAR 0.0845 W/kg), 1800 MHz (0.04563 W/kg), and 2100 MHz (0.03957 W/kg) RFR 2 h/day for 6 months	Increased DNA strand breaks and oxidative DNA damage in brain.
Alkis et al. (2019b)	Rats exposed to 900 MHz, 1800 MHz, and 2100 MHz RFR 2 h/day for 6 months; maximum SAR over the rat 0.017 W/kg	
Atasoy et al. (2013)	Male Wister rats exposed to 2437 MHz (Wi-Fi) RFR; 24 h/day for 20 weeks; maximum SAR 0.091 W/kg	Oxidative DNA damage in blood and testes.
Beaubois et al. (2007)	Leaves of tomato plant exposed to 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Increased expression of leucine-zipper transcription factor (bZIP) gene.
Belyaev et al. (2005)	Lymphocytes from human subjects exposed to GSM 915 MHz RFR for 2 h ; SAR 0.037 W/kg;	Increased condensation of chromatin.

Belyaev et al. (2009)	Human lymphocytes exposed to UMTS cell phone signal (1947.4 MHz, 5 MHz band width) for 1 h; SAR 0.04 W/kg	Chromatin affected and inhibition of DNA double-strand break.
Bourdineaud et al. (2017)	Eisenia fetida earthworms exposed to 900 MHz for 2 h; SAR 0.00013-0.00933 W/kg	DNA genotoxic effect and HSP70 gene expressions up regulated.
Campisi et al. (2010)	Rat neocortical astroglial to CW 900 MHz RFR for 5, 10, or 20 min; incident power density 0.0265 mW/cm^2	Significant increases in DNA fragmentation.
Chaturvedi et al. (2011)	Male mice exposed to 2450 MHz RFR, 2 h/day for 30 days; SAR 0.03561 W/kg	Increased DNA strand breaks in brain cells.
Deshmukh et al. (2013)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 30 days.	Increased DNA strand breaks in brain tissues.
Deshmukh et al. (2015)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 180 days.	Increased DNA strand breaks in brain tissues.
Deshmukh et al. (2016)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 90 days.	Increased DNA strand breaks in brain tissues.

Eker et al. (2018)	Female Wistar albino rats exposed to 1800-MHz RFR for 2 h/day for 8 weeks; SAR 0.06 W/kg	Caspase-3 and p38MAPK gene expressions increased in eye tissues.
Furtado-Filho et al. (2014)	Rats of different ages (0-30 days) exposed to 950 MHz RFR for 0.5 h/day for 51 days (21 days of gestation and 6-30 days old): SAR pregnant rat 0.01-0.03 W/kg; neonate 0.88 W/kg, 6-day old 0.51 W/kg, 15-day old 0.18 W/kg, 30-day old 0.06 W/kg.	Decreased DNA strand breaks in liver of 15-day old and increased breaks in 30-day old rats.
Gulati et al. (2016)	Blood and buccal cells of people lived close (<400 meters) to a cell tower; 1800 MHz, Maximum power density (at 150 meters) 0.00122 mW/cm^2, some subjects lived in the area for more than 9 yrs	Increased DNA strand breaks in lymphocytes and micronucleus in buccal cells.
Gürler (2014)	Wistar rats exposed to 2450 MHz RFR 1 h/day for 30 consecutive days; power density 0.0036 mW/cm^2	Increased oxidative DNA damage in brain and blood.
Hanci et al. (2013)	Pregnant rats exposed 1 h/day on days 13-21 of pregnancy to 900-MHz RFR at power density 0.0265 mW/cm^2.	Testicular tissue of 21-day old offspring showed increased DNA oxidative damage.
He et al. (2016)	Mouse bone marrow stromal cells exposed to 900 MHz RFR 3 h/day for 5 days; SAR 4.1×10^{-4} W/kg (peak), 2.5×10^{-4} W/kg (average)	Increased expression of PARP-1 mRNA

Hekmat et al. (2013)	Calf thymus exposed to 940 MHz RFR for 45 min; SAR 0.04 W/kg	Altered DNA structure at 0 and 2 h after exposure.
Kesari and Behari (2009)	Male Wistar rats exposed to 50 GHz RFR for 2 h/day for 45 days; SAR 0.0008 W/kg	Increased in brain tissue DNA strand.
Kumar R. et al. (2021)	Male Wistar rats exposed to 900 MHz, 1800 MHz and 2450 MHz RFR at a specific absorption rate (SAR) of 5.84×10^{-4} W/kg, 5.94×10^{-4} W/kg and 6.4×10^{-4} W/kg, respectively for 2 h per day for 1-month, 3-month and 6-month periods.	Epigenetic modifications in the hippocampus, bigger effects with increasing frequency and duration of exposure.
Kumar S. et al. (2010)	Male Wistar rats exposed to 10-GHz RFR for 2 h a day for 45 days, SAR 0.014 W/kg	Increased micronucleus in blood cells.
Kumar S. et al. (2013)	Male Wistar rats exposed to 10 GHz RFR for 2 h a day for 45 days; SAR 0.014 W/kg	Increased micronucleus in blood cells and DNA strand breaks in spermatozoa.
Marinelli et al. (2004)	Acute T-lymphoblastoid leukemia cells exposed to 900 MHz RFR for 2-48 h, SAR 0.0035 W/kg	Increased DNA damage and activation of genes involved in pro-survival signaling.
Markova et al. (2005)	Human lymphocytes exposed to 905 and 915 MHz GSM signals for 1 h; SAR 0.037 W/kg	Affected chromatin conformation and 53BP1/gamma-H2AX foci
Markova et al. (2010)	Human diploid VH-10 fibroblasts and human adipose-tissue derived mesenchymal stem	Inhibited tumor suppressor TP53 binding protein 1 (53BP1) foci that are typically formed at the sites of DNA double strand break

	cells exposed to GSM (905 MHz or 915 MHz) or UMTS (1947.4 MHz, middle channel) RFR for 1, 2, or 3 hr; SAR 0.037-0.039 W/kg	location.
Megha et al. (2015a)	Fischer rats exposed to 900 and 1800 MHz RFR for 30 days (2 h/day, 5 days/week), SAR 0.00059 and 0.00058 W/kg	Reduced levels of neurotransmitters dopamine, norepinephrine, epinephrine, and serotonin, and downregulation of mRNA of tyrosine hydroxylase and tryptophan hydroxylase (synthesizing enzymes for the transmitters) in the hippocampus.
Megha et al. (2015b)	Fischer rats exposed to 900, 1800, and 2450 MHz RFR for 60 days (2 h/day, 5 days/week); SAR 0.00059, 0.00058, and 0.00066 W/kg	Increased DNA damage in the hippocampus
Nittby et al. (2008)	Fischer 344 rats exposed to 1800 MHz GSM RFR for 6 h; SAR whole body average 0.013 W/kg, head 0.03 W/kg	Expression in cortex and hippocampus of genes connected with membrane functions.
Odaci et al. (2016)	Pregnant Sprague - Dawley rats exposed to 900 MHz RFR 1 h each day during days 13 - 21 of pregnancy; whole body average SAR 0.024 W/kg	Testis and epididymis of offspring showed higher DNA oxidation.
Pandey et al. (2017)	Swiss albino mice exposed to 900-MHz RFR for 4 or 8 h per day for 35 days; SAR 0.0054-0.0516 W/kg	DNA strand breaks in germ cells.
Pesnya and Romanovsky (2013)	Onion (Allium cepa) exposed to GSM 900-MHz RFR from a cell phone for 1 h/day or 9 h/day for 3 days;	Increased the mitotic index, the frequency of mitotic and chromosome abnormalities, and the micronucleus frequency in an exposure-duration manner.

	incident power density 0.0005 mW/cm^2	
Phillips et al. (1998)	Human Molt-4 T-lymphoblastoid cells exposed to pulsed signals at cellular telephone frequencies of 813.5625 MHz (iDEN signal) and 836.55 MHz (TDMA signal) for 2or 21 h. SAR 0.0024 and 0.024 W/Kg for iDEN and 0.0026 and 0.026 W/kg for TDMA)	Changes in DNA strand breaks
Qin et al. (2018)	Male mice exposed to 1800-MHz RFR 2 h/day for 32 days, SAR 0.0553 W/kg	Might be mediated through CaMKI/RORα signaling pathway.
Rammal et al. (2014)	Tomato exposed to a 1250-MHz RFR for 10 days at 0.0095 mW/cm^2	Increased expression of two wound-plant genes.
Roux et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 2-10 min at 0.0066 mW/cm^2	Induction of stress gene expression.
Roux et al. (2008)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Induction of stress gene expression.
Sarimov et al. (2004)	Human lymphocytes exposed to GSM 895-915 MHz signals for 30 min; SAR 0.0054 W/kg	Condensation of chromatin was observed.
Shahin et al. (2013)	Female mice (Mus musculus) exposed to continuous-wave 2.45 GHz RFR 2 h/day for 45v days; SAR 0.023 W/kg	Increased DNA strand breaks in the brain.
Sokolovic et al. (2015)	Wistar rats exposed to RFR (4 h/day, for 20, 40, and 60 days) from a Nokia 3110 cell	DNA fragmentation and oxidative changes in testicular tissues.

	phone; SAR 0.043-0.135 W/kg.	
Sun Y. et al. (2017)	Human HL-60 cells exposed to 900 Hz RFR 5 h/day for 5 days; peak and average SAR 4.1 x 10^{-4} and 2.5 x 10^{-4} W/kg	Increased oxidative DNA damage and decreased mitochondrial gene expression.
Tkalec et al. (2013)	Earthworm (Eisenia fetida) exposed to continuous-wave and AM-modulated 900-MHz RFR for 2 - 4 h; SAR 0.00013, 0.00035, 0.0011, and 0.00933 W/kg	Increased DNA strand breaks.
Tsybulin et al. (2013)	Japanese Quail embryos exposed in ovo to GSM 900 MHz signal from a cell phone intermittently (48 sec ON/12 sec OFF) during initial 38 h of brooding or for 158 h (120 h before brooding plus initial 38 h of brooding): SAR 0.000003 W/kg	The lower duration of exposure decreased DNA strand breaks, whereas higher duration resulted in a significant increase in DNA damage.
Vian et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Induction of mRNA encoding the stress-related bZIP transcription factor.
Yakymenko et al. (2018)	Quail embryos exposed to GSM 1800 GHz signal from a smart phone (48 s ON/12 s OFF) for5 days before and 14 days during incubation, power density 0.00032 mW/cm^2	Increased DNA strand breaks and oxidative DNA damage.
Zong et al. (2015)	Mice exposed to 900 MHz RFR 4 h/day for 7 days; SAR 0.05 W/kg	Attenuated bleomycin-induced DNA breaks and repair.

Static and ELF EMF Studies		
Agliassa et al. (2018)	Arabidopsis thaliana (thale cress) exposed to 0.00004 mT static magnetic field for 38 days after sowing	Changes in gene expression in leaf and floral meristem.
Back et al. (2019)	Mouse embryonic stem cells exposed to hypomagnetic field (<0.005 mT) up to 12 days	Induced abnormal DNA methylation.
Bagheri Hosseinabadi et al. (2020)	Blood samples from thermal power plant workers; mean levels of exposure to ELF magnetic and electric fields were 0.0165 mT (±6.46) and 22.5 V/m (±5.38), respectively.	DNA strand breaks .in lymphocytes.
Baraúna et al. (2015)	Chromobacterium violaceum bacteria cultures exposed to ELF-EMF for 7 h at 0.00066 mT	Five differentially expressed proteins detected including the DNA-binding stress protein.
Belyaev et al. (2005)	Human lymphocytes exposed to 50 Hz magnetic field at 0.015 mT (peak) for 2 h (measurements made at 24 and 48 h after exposure).	Induced chromatin conformation changes.
Dominici et al. (2011)	Lymphocytes from welders (average magnetic field exposure from personal dosimeters 0.00781 mT (general environmental level 0.00003 mT)	Higher micronucleus frequency correlated with EMF exposure levels; decreased in sister chromatid exchange frequency.
Heredia-Rojas et al. (2010)	Human non-small cell lung cancer cells (INER-37) and mouse lymphoma cells (RMA E7) (transfected with a	An increased in luciferase gene expression was observed in INER-37 cells.

	plasmid with hsp70 expression when exposed to magnetic field and contains the reporter for the luciferases gene) exposed to a 60-Hz magnetic field at 0.008 and 0.00008 mT for 20 min.	
Sarimov et al. (2011)	Human lymphocytes exposed to 50-Hz magnetic field at 0.005-0.02 mT for 15-180 min	Magnetic field condensed relaxed chromatin and relaxed condensed chromatin.
Villarini et al. (2015)	Blood leukocytes from electric arc welders presumably exposed to 50-Hz EMF (mean 0.0078 mT; range: 0.00003-0.171 mT)	Decreased DNA strand beaks.
Wahab et al. (2007)	Human peripheral blood lymphocytes exposed to 50 Hz sinusoidal (continuous or pulsed) or square (continuous or pulsed) magnetic fields at 0.001 or 1 mT for 72 h.	Increase in the number of sister chromatid exchange/cell
Zendehdel et al. (2019)	Peripheral blood cells of male power line workers in a power plant. The median value of the magnetic field at the working sites was 0.00085 mT.	Increased in DNA strand breaks.

Supplement 5

Effects of EMF wave-form and cell types (*in italic*) studied (Literature up to January 2021)

RFR	
Belyaev et al. (2009)	UMTS different from GSM signal on DNA repair foci in human lymphocytes.
Campisi et al. (2010)	Increased DNA fragmentation in rat neocortical astroglial by 50-Hz modulated 900-MHz RFR, but no effect from continuous wave field.
D'Ambrosia et al. (1995)	Micronucleus frequency in human lymphocytes affected by pulsed but not CW 9 GHz RFR.
D'Ambrosia et al. (2002)	Micronucleus frequency in human lymphocytes affected by pulsed but not CW 1748-MHz RFR.
Del Re et al. (2019)	*Changes in repetitive–DNA in human cell exposed to GSM 900-MHz RFR depended on cell type studied (HeLa, BE(2)C, and SH SY5Y).*
Franzellitti et al. (2008)	HSP70C gene expression enhanced after 24 h exposure to GSM-217Hz signals and reduced after 4 and 16 h exposure to GSM-Talk signals In human trophoblasts.
Franzellitti et al. (2010)	DNA damage in human trophoblasts induced by GSM 1800 MHz RFR, but not by continuous-wave field.
Gapeyev et al. (2014)	Protective effect to x-ray induced DNA strand break in mouse lymphocytes with pulse-modulated and not continuous-wave RFR.
Heredia-Rojas et al. (2010)	*"Electromagnetic field" plasmid transfected into INER-37 and RMA E7 cell lines exposed to a 60-Hz MF. An increased luciferase gene expression was observed in INER-37 cells but had no effect on the RMA E7 cell line.*
Kumar et al. (2020)	1800-MHz more effective than 900-MHz RFR on inducing DNA damage in onion.
Lopaz-Martin et al. (2009)	Unmodulated RFR caused higher neuronal c-fos expression than pulsed modulated 900-MHz GSM field.
Luukkonen et al. (2009)	872-MHz continuous-wave RFR increased DNA strand breaks in SH-SY5Y human neuroblastoma cells, but no effect from GSM –modulated field.
Markova et al. (2005)	GSM-915 MHz RFR induced more consistent

	effect on human lymphocyte chromatin conformation than GSM-905 MHz RFR.
Martin et al. (2020)	*Four different types of human keratinocytes showed different patterns (Up- and down-regulation or no change) of expression of ADAMTS6, IL7R, and NOG genes exposed to a 60-GHZ RFR.*
Ozgur et al. (2014)	1800-MHz RFR more potent than 900-MHz RFR on inducing DNA fragmentation (apoptosis) in hepatocarcinoma cells.
Nylund and Leszczynski (2006)	*Gene and protein expressions in response to GSM 900-MHz RFR depended on the type of human endothelial cell line (EA.hy926 and EA.hy926v1).*
Remondini et al. (2006)	*Gene expressions after exposure to 900 and 1800 –MHz RFR- NB69 neuroblastoma cells, T lymphocytes, and CHME5 microglial cells did not show significant changes, whereas EA.hy926 endothelial cells, U937 lymphoblastoma cells, and HL-60 leukemia cells showed up- or down-regulated genes.*
Romano-Spica et al. (2000)	Oncogene expression only occurred when exposed to 16-Hz modulated 50MHz RFR
Sarimov et al. (2004)	Different potencies between 915 MHz and 905-MHz RFR on chromatin conformation in human lymphocytes.
Schwartz et al. (2008)	*UMTS 1950-MHz RFR increased DNA breaks and micronucleus frequency in human fibroblasts, but not in lymphocytes.*
Semin et al. (1994)	4000-8000 MHz RFR, 1-6 Hz modulated RFR showed narrow "window" peak intensity and modulation frequency effects on DNA secondary structure.
Shckorbatov et al. (2009)	35-GHz RFR caused condensation of chromatin in human buccal epithelium cells-left circularly polarized radiation induced less effect than linearly polarized radiation.
Shckorbatov et al. (2010)	36.65-GHz RFR caused chromosome condensation in human fibroblasts –right-handed elliptically polarized radiation was more biological activity than the left-handed polarized one.
Tkalec et al. (2013)	AM-modulated 900- MHz RFR more potent than continuous-wave field in inducing DNA damage in earthworms coelomocytes.
Valbonesi et al. (2014)	GSM 1800-MHz signal, but not continuous-

	wave field, induced HSP-70 gene expression in rat PC-12 cells.
Vilic et al. (2017)	DNA damage in honey bee larvae- AM-modulated 900-MHz RFR more potent than continuous-wave field.
Xu et al. (2013)	*Gamma-H2AX foci after exposure to GSM 1800-MHz RFR induced in in Chinese hamster lung cells and Human skin fibroblasts (HSFs), but not in rat astrocytes, human amniotic epithelial cells, human lens epithelial cells, and human umbilical vein endothelial cells.*
Zhang et al. (2008)	Intermittent 1800-MHz RFR more potent than continuous exposure on gene expression in rat neurons.
Zhao et al. (2007)	*Capase-2and Capase-6 expressions up-regulated in neuron, but not in astrocytes.*
Static/ELF EMF	
Del Re et al. (2006)	50-Hz sinusoidal MF increased where as pulse square wave decreased heat-shock protein induction in E. coli.
Focke et al (2010)	Increased DNA fragmentation by intermittent 50-Hz MF, but no effect by continuous exposure.
Giorgi et al. (2011)	E. coli gene expression decreased by sinusoidal MF and increased by pulsed square-wave MF- not frequency dependent (25, 50, 75 Hz)
Heredia-Rojas et al. (2010)	*60-Hz MF induced luciferase gene expression in INER-37 cells, but not in RMA E7 cells.*
Ivancsits et al. (2002)	Intermittent more potent than continuous exposure of a 50-Hz MF on DNA damage in human fibroblasts.
Lee et al. (2016)	*60-Hz MF induced delay of cell cycle progression in MCF7 and MCF10A cells, but not in Jurkat and NIH3T3 cells.*
Mahmoudinasab and Saadat (2018a)	*Patterns of up-regulation of antioxidant genes are different between MCF-7 and SH-SY5Y cells exposed to an intermittent 50-Hz EMF.*
Mahmondinasab et al. (2016)	Different schedules of intermittent exposure to a 50-Hz MF had different effect on gene expression in human MCF-7 breast cancer cells
Mercado-Saenz et al. (2019)	Decreased spontaneous mitochondrial mutation in yeast by pulsed MF (25-Hz), no effect by sinusoidal field.

Robison et al. (2002)	*60-Hz MF exposure decreased DNA repair rate in HL-60 and HL-60R cells, but not in Raji cells.*
Sanie-Jahromi and Saadat (2018)	*co-treatment of "cisplatin +morphine + EMF" made bleomycin more cytotoxic in SH-SY5Y cells, but not in MCF-7cells.*
Sanie-Jahromi et al. (2016)	Significant differences in DNA-repair gene expression in MCF-7 cell exposed under 3 different patterns of 50-Hz EMF (5 min field-on/5 min field-off (30 min), 15 min field-on/15 min field-off (30 min), 30 min field-on continuously.)
Sanie-Jahromi et al. (2017)	*50-Hz MF exposure synergistic with cisplatin and bleomycin on DNA-repair gene expression and cell viability in MCF-7 cells, but not in SH-SY5Y cells.*
Udroiu et al. (2015)	*50-Hz MF exposure affected genotoxic effect of x-ray in mouse male germ cells, but not in peripheral blood erythrocytes.*
Wahab et al. (2007)	Sister chromatid exchange in human lymphocytes exposed to a 50-Hz MF (continuous or pulsed sinusoidal or continuous or pulsed square-wave). Square continuous-wave MF was the most potent.