

Review

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Radiofrequency radiation-induced gene expression

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Abstract: Genes are differentially expressed in cells in response to changes in the internal or external environment. The response is generally an adaptive mechanism to the environmental challenge to restore cell functions to homeostasis. There are numerous studies reporting changes in gene expression in cells exposed to radiofrequency radiation (RFR), the type of energy emitted by wireless communication devices. The major genes affected are those involved in: repair of damaged proteins, response to stress, oxidative changes, apoptosis, DNA damage detection and repair, and changes in neural functions. Gene expression data supports the notion that RFR is a stressor that causes oxidative changes and DNA and protein damage in cells under different exposure conditions, in many biological systems. Changes in all these significant gene expression effects are supported by results of other biological studies of RFR exposure in the literature. They should be considered in the setting of RFR-exposure guidelines.

Keywords: gene expression; radiofrequency radiation; stress; DNA damage; apoptosis; oxidative changes

Introduction

As of July 2025, there are more than 500 papers published on genetic effects of radiofrequency radiation (RFR). Most of the studies are on genotoxicity (e.g., DNA damage, chromatin conformation changes, and micronucleus formation, among

others) and gene expression (see ‘RFR-genetic effects’ file in references [1] and [2]).

Genetic effects of EMF can depend on various factors, including field parameters and characteristics (such as frequency, intensity, modulation, and orientation of the field); cell type; and exposure duration. There are many reports showing genetic effects in cells and animals after exposure to RFR at intensities like those now found in ambient, public, and occupational environments. Particularly, there are many reports that showed genetic effects at intensities below the SAR limits of <0.4 W/kg (the dose rate considered to be safe by most international RFR exposure guidelines) (see ‘RFR-low-intensity effects’ file in reference [1, 3]).

As a singular entity, the mechanism by which RFR causes genetic effects is not well understood but appears due to multiple inter-dependent mechanisms. One possible mechanism with ample research data involves free radical formation. There are many reports of free radicals and oxidative processes involvement with RFR-exposure, including both reactive oxygen and reactive nitrogen species (see ‘RFR oxidative effects’ file in reference [1, 4]). It is conceivable that increases in free radicals in cells could cause macromolecular damage, including that to DNA.

Effects of RFR on gene expression

Many studies have examined the effects of exposure to RFR on gene expression in cells. Changes in gene expression are a reflection on how cells “perceive” the disturbance of the radiation and their response to restore normal functions. These studies include *in vitro*, *in vivo*, and acute- and chronic/repeated-exposure experiments. In most studies, RFR radiation was generated by waveguides or antennas, and in some studies, wireless devices such as cell phones and Wi-Fi routers provided the radiation. In the latter instance, the radiation is less uniform, and the specific absorption rate (SAR) is generally not available. However, these latter studies provide a more realistic exposure situation.

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Changes in expression of genes of various biological functions have been reported (Appendix 1 in Supplementary Material, literature can be retrieved from the PubMed). This paper presents the findings of several major gene-expression topics: 1. repair and removal of damaged proteins; 2. DNA damage; 3. oxidative processes; 4. stress responses; 5. apoptosis; and 6. brain functions. The majority of the studies included are on RFR between 800 and 2,500 MHz. SAR was provided in most studies and power density in several studies. The SAR values varied from several $\mu\text{W/kg}$ to 20 W/kg. However, power density provides little information on the amount of energy absorbed in tissues, particularly when they are not given as incident power densities.

Genes involved in repair and removal of damaged proteins

Of all the genes that have been shown to be affected by RFR, heat shock protein (Hsp) genes stand out as a major target. Studies that showed effects of RFR exposure on Hsp are listed in Table 1. (References of studies cited in the tables can be found in the Appendix at the end of the text.) Up-regulation is generally reported. Note that the effect has been observed in different biological systems and exposure conditions. One would argue that the responses are caused by the thermal effect of RFR. However, in several instances, upregulation of Hsp was observed at low-intensity RFR, defined as at a SAR of $< 0.4 \text{ W/kg}$ (see studies marked 'L' in Table 1) when heating was unlikely. In one study [5], Hsp upregulation was observed after exposure to a GSM-modulated field and not to a continuous-wave field of the same SAR, which further argues against a thermal effect. Results observed at low intensity are more relevant to environmental RFR exposure from far-field radiation emitted by transmitters (e.g., radar, radio, and TV) and base stations, whereas higher intensity (thus, higher localized SAR) studies are related to cell phone use where body parts, particularly the brain, are exposed at a close distance.

In addition to heat stress, various forms of Hsp are activated by different stressors such as cold, oxidative stress, and tissue damage. The major function of Hsp is to stabilize and repair denatured proteins. It also is responsible for the removal of damaged protein via the lysosome process when that protein is beyond repair. This prevents damaged protein aggregation that can affect cell functions and is a leading cause of certain neurodegenerative diseases such as Alzheimer's disease. On the

other hand, Hsp has anti-apoptosis properties that, in cancer cells, can play a role in the initiation and progression of cancer.

Previous reports have suggested that RFR could change protein conformation [6–8] which can lead to the expression of Hsp genes that facilitate repair. But how does RFR affect protein structure? RFR does not have enough quantum energy to directly break covalent bonds. Can RFR affect weak molecular interactions, e.g., London dispersion forces? Further research will be needed to understand the mechanism.

Genes involved in DNA damage

Another important consideration is the expression of genes related to DNA biology reported in the publications listed in Table 2. Genes affected are mostly those in response to DNA damage or related to molecular mechanisms of DNA damage repair. These data also indicate that RFR at low intensities can damage DNA (see LI studies in Table 2). DNA damage after exposure to RFR has been reported in numerous studies [2].

Genes involved in oxidative processes

A consistently reported bioeffect of RFR is changes in oxidative processes. More than 350 papers have been published reporting effects (see 'RFR oxidative effects' file in reference [1]). Numerous gene-expression studies have shown up-regulation of oxidative process-related genes (Table 3). That different related genes are expressed to counteract oxidative stress (e.g., anti-oxidation enzymes) after exposure to RFR support the notions that RFR affects oxidative processes in cells. Hsp70 seems to play an important role. It activates different biochemical antioxidant mechanisms to bring the cells to normal homeostasis [9]. Protein damage could also be caused by RFR-induced free radicals leading to the expression of genes related to repair or removal of the damaged proteins (as discussed above).

Genes involved in stress response

It has been proposed by various researchers that RFR is a stressor (e.g., references [3] and [10]) that causes "cellular

Table 1: Repair and removal of damaged proteins.

References (see Appendix)	Exposure conditions	Results	Comments
Balakrishnan et al. (2014) ^a	Serum of cellular phone frequent and infrequent users.	Increased Hsp70 gene expression with phone use.	Hsp70 stabilizes partially synthesized protein and prevents aggregation, protects cells from thermal and oxidative stress, disposal of damaged proteins, and inhibits apoptosis.
Bourdineaud et al. (2017) (LI)	<i>Eisenia fetida</i> (earthworm) exposed to 900 MHz for 2 h; SAR 0.00013–0.00933 W/kg.	Gene expressions up regulated for Hsp70.	Hsp70 stabilizes partially synthesized protein and prevents aggregation, protects cells from thermal and oxidative stress, disposal of damaged proteins, and inhibits apoptosis.
Cappucci et al. (2022) (LI)	Head, ovary, and testis of <i>Drosophila melanogaster</i> exposed to 2,437 MHz RFR (Wi-Fi) from embryo to adult stage at 0.0608 W/kg.	Increased gene expression of Hsp70; induced gene instability.	
Czyz et al. (2004)	Pluripotent embryonic stem (ES) cells (wild-type and deficient for the tumor suppressor p53 exposed to pulsed 1710 MHz RFR at GSM-217 (1.5 W/kg) or GSM-talk (0.4 W/kg) mode for 6–48 h.	Upregulation of mRNA levels of Hsp70.	
Franzellitti et al. (2008)	Human trophoblasts 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.	Intracellularly localized Hsp70s are an important part of the cell's machinery for protein folding, performing chaperoning functions, and helping to protect cells from the adverse effects of physiological stresses. They inhibit apoptosis.
Koohstaniidehaghi et al. (2023) ^a	Mouse preimplantation embryos exposed to 900–1,800 MHz RFR from a cellular phone or 30 min; 0.636–1.8 W/kg.	Increased expression of Hsp70 gene.	
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 mRNA and protein expression of Hsp70.	
Migdal et al. (2023) (LI)	Bees exposed to 900 MHz RFR for 0.25, 1, or 3 h at 0.05, 0.3, or 1.4 W/kg.	Increased expression of Hsp70 and Hsp90 genes (at 0.05 W/kg for 3 h).	Hsp90 assists proper protein folding, stabilizes proteins against heat stress, and aids in protein degradation. It also stabilizes a number of proteins required for tumor growth.
Valbonesi et al. (2014)	Rat PC12 cells exposed to continuous-wave 1.8 GHz RFR or GSM-217 Hz and GSM-Talk signals for 4, 6, or 24 h, SAR 2 W/kg.	After PC12 cells exposure to the GSM-217 Hz signal for 16 or 24 h, Hsp70 mRNA transcription significantly increased, whereas no effect was observed in cells exposed to the CW or GSM-Talk signals.	
Wang et al. (2022) (LI)	<i>Drosophila melanogaster</i> exposed to 3.5 GHz RFR for 2–3 days at 0.0026 W/kg, 0.026 W/kg, or 0.26 W/kg.	In third-instar larvae, increased expression levels of the heat shock protein genes Hsp22, Hsp26 and Hsp70.	Translocation of both Hsp22 and INOS to the mitochondria is necessary for Hsp22-mediated stimulation of oxidative phosphorylation. Hsp26 binds and prevents unfolded proteins from forming aggregates.

Table 1: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Yang et al. (2012)	Hippocampus of rats exposed to 2,450 MHz RFR for 20 min at 6 W/kg.	Upregulated Hsp27 and Hsp70 genes expression.	The main function of Hsp27 is to provide thermotolerance <i>in vivo</i> , cytoprotection, and support of cell survival under stress conditions, inhibiting protein aggregation and by stabilizing partially denatured proteins. It has anti-apoptotic properties and roles in cell growth (termination) and inflammatory and stress responses.
Zalata et al. (2015) ^a	Human semen samples exposed to 850-MHz RFR from a cellular phone for 1 h; SAR 1.46 W/kg at 10 cm.	Significant increase in sperm DNA fragmentation percent, clusterin gene expression and clusterin protein (associated with clearance of cellular debris and apoptosis) levels in the exposed semen samples.	Clusterin is an extracellular molecular chaperone which binds to misfolded proteins in body fluids to neutralize their toxicity and mediate their cellular uptake by receptor-mediated endocytosis and transfer into lysosomes for degradation.

^aStudies that involved exposure to radiation from wireless devices, such as cell phones. (LI), Low-intensity study (SAR<0.4 W/kg). (Number of studies: acute exposure/*in vivo*=4; repeated (chronic) exposure/*in vivo*=3; acute exposure/*in vitro*=5; repeated (chronic) exposure/*in vitro*=0).

stress response” and general systematic stress. In addition to upregulation of genes of Hsp that regulate oxidative and other stress, many other stress-related genes have also been reported to be activated by RFR (Table 4). This data supports the notion that RFR is a “stressor”. Persistent stress can adversely affect biological functions and health.

Genes involved in apoptosis

There are many studies that indicated RFR exposure affected genes involved in apoptosis (Table 5). Both pro- and anti-apoptosis gene effects have been reported. It is not readily known what factors/conditions differentially trigger the pro-apoptotic or anti-apoptotic effects. This may be due to the unstable oscillation of the feedback control physiological system involved. Understanding this could have important implications in the application of low-intensity RFR for cancer treatment. There are 11 studies on cancer cells listed in Table 5. Up-regulation of pro-apoptotic genes or down regulation of anti-apoptotic genes is reported in eight studies: U118-MG human glioma cells [11]; SH SY-5Y human neuroblastoma cells [12–15]; HL-60 human leukemia cells [16, 17] and Rat PC- 12 pheochromocytoma cells [18]; and the opposite effects (i.e., anti-apoptosis) in three studies: SK-Mel-3 human melanoma cells [19]; rat PC-12 cells [5]; and human T-lymphoblastoid leukemia cells [20]. Thus, conceivably, RFR could impede or promote cancer growth depending on exposure parameters. The frequency (800–2,450 MHz) (except the Cheon et al. study [19] that used a pulsed 1.6-THz field) and exposure duration (<48 h) are similar. The SARs of these studies varied from 0.0035-10 W/kg. There is not enough data to make any speculation regarding exposure parameters and apoptosis outcomes.

Genes involved in brain functions

When using a cell phone, the brain is exposed to RFR at a relatively constant spatial pattern [21]. How do genes in brain cells respond to RFR? Table 6 lists some reports. RFR generally affects genes involved in brain functions and structure. Several papers reported changes in neurotransmitter genes, including those of acetylcholine, N-methyl-D-aspartate, serotonin, norepinephrine, and dopamine [22–25]. Changes in expression of these genes could lead to behavioral changes and neurological/psychiatric disorders [26]. Of note are the several studies by Dasdag et al. [27–30] reporting changes in expression of genes of miRNA involved in cancer processes in brain cells. These effects could lead to tumor

Table 2: Genes related to DNA damage.

References (see Appendix)	Exposure conditions	Results	Comments
Cantu et al. (2023) (LI)	Human keratinocytes exposed to 900 MHz RFR for 1 h at 0.00155 W/kg.	Six common DNA targets were both differentially methylated and differentially expressed.	Six genes related to cell survival and responses to damage were affected (SPDYA, BBS5, CCS, DOK3, DD12, and MGA). Up-regulation of DNA-damage induced 1 homolog 2 (DD12) increases cell survival following stress.
Czyz et al. (2004)	Pluripotent embryonic stem (ES) cells (wild-type and deficient for the tumor suppressor p53 exposed to pulsed 1710 MHz RFR at GSM-217 (1.5 W/kg) or GSM-talk (0.4 W/kg) mode for 6–48 h.	Upregulation of p21 gene in p53-deficient, but not in wild-type cells.	p21 represents a major target of p53 activity and is associated with linking DNA damage to cell cycle arrest.
Ghatei et al. (2017) ^a	Mice exposed pre- and postnatally to radiation from a cellular phone jammer (900 and 1800 MHz).	Gene expression level of p21 was increased.	
He et al. (2016) (LI)	Mouse bone marrow stromal cells exposed to 900 MHz RFR for 3 h/day for 5 days at peak and average SARs of 4.1×10^{-4} and 2.5×10^{-4} W/kg.	Increased mRNA of poly(ADP-ribose) polymerase-1, a nuclear enzyme which plays an important role in the repair of damaged DNA.	Poly(ADP-ribose) polymerase 1 is involved in recovery from DNA damage (repair of single strand DNA breaks).
He et al. (2017) (LI)	Same as above and included cells exposed to gamma radiation.	Increased mRNA of poly(ADP-ribose) polymerase-1, cells treated with RFR and gamma radiation showed less genetic damage and faster kinetics of repair.	
Jin et al. (2021)	Murine melanoma cell line B16 and the human keratinocyte cell line HaCaT exposed to EMF-long term evolution radiation (LTE, 1.762 GHz) for 4 h at 8 W/kg.	p53 gene up-regulated in cells.	p53 plays a role in the protective effect of EMF-LTE against DNA double strand breaks.
Kim JH et al. (2021)	SH-SY5Y neuronal cells exposed to 1760 MHz RFR 4 h/day for 4 days; 4 W/kg.	Transcriptional activation of p53.	p53 activates DNA repair proteins in response to DNA damage, causes cell cycle arrest at the G1/S point, initiates apoptosis when DNA damage is irreparable.
Kucukbagciak et al. (2022) (LI)	Liver and blood of mice exposed to 900 MHz GSM RFR; 4 h/day for 7 days; 0.339 W/kg.	Increased DNA repair mechanism genes expression (p53 and oxoguanine DNA glycosylase (OGG-1)).	8-Oxoguanine glycosylase (OGG1) is involved in base excision repair.
Nikolova et al. (2005)	Mouse embryonic neural progenitor stem cells exposed to 1,710-MHz GSM RFR for 6 or 48 h; SAR 1.5 W/kg.	Exposure for 6 h, but not for 48 h, up-regulation of GADD45 gene expression.	GADD45 gene product can enable more efficient recognition and repair of spontaneous DNA damages generated by physiological processes and environmental stressors. It is induced by treatment with DNA-damaging agents, mediating cell cycle arrest at G2/M phase and inducing apoptosis.
Üstündağ et al. (2020)	Zebrafish embryos exposed to 3,000 MHz RFR. (No dosimetry data).	Decreased TP53 and increased Casp3a gene expression.	TP53 gene encodes proteins that bind to DNA and regulate gene expression to prevent mutations of the genome.
Vafaei et al. (2020) (LI) ^a	Placenta of pregnant mice exposed to 2.4 GHz RFR from a D-link Wi-Fi router; 2 or 4 h/day at 30 or 60 cm from router from 5 days after mating to 1 day before expected delivery, 0.09 W/kg at 30 cm.	Increased gene expression of CDKN1A and GADD45a (DNA repair enzymes).	CDKN1A functions as a regulator of cell cycle progression at G1. CDKN1A plays a critical role in the cellular response to DNA damage.

^aStudies that involved exposure to radiation from wireless devices, such as cell phones. (LI). Low-intensity study (SAR<0.4 W/kg). (Number of studies: acute exposure/*in vivo*=0; repeated (chronic) exposure/*in vivo*=4; acute exposure/*in vitro*=5; repeated (chronic) exposure/*in vitro*=2).

Table 3: Genes related to oxidative processes.

References (see Appendix)	Exposure conditions	Results	Comments
Balakrishnan et al. (2014) ^a Bertuccio et al. (2024)	Serum of cellular phone frequent and infrequent users. Human neuronal-like SH-SY5Y and peripheral blood mononuclear cells to 2,450 MHz RFR for 24 h, no dosimetry data.	Increased Hsp70 gene expression with phone use. Decreased Nrf2 (transcription factor for stress response) and SOD2 gene expression in both cell types.	Hsp70 protects cells from thermal and oxidative stress. Nuclear factor erythroid 2-related factor 2 (NRF2) is involved in the cellular defense against toxic and oxidative insults through the expression of oxidative stress response and drug detoxification genes. SOD2 is an anti-oxidant enzyme.
Bourdineaud et al. (2017) (LI)	<i>Eisenia fetida</i> (earthworm) exposed to 900 MHz for 2 h; SAR 0.00013–0.00933 W/kg.	Gene expressions up regulated for Hsp70, MEK1 (signal transduction); oxidative stress; and chemical and immune defenses.	Hsp70 protects cells from thermal and oxidative stress. MEK1 suppresses oxidative stress-induced apoptosis.
Cappucci et al. (2022) (LI)	Head, ovary, and testis of <i>Drosophila melanogaster</i> exposed to 2,437 MHz RFR (Wi-Fi) from embryo to adult stage at 0.0608 W/kg.	Increased gene expression of Hsp70.	Hsp70 protects cells from thermal and oxidative stress.
Chow et al. (2024)	Human umbilical vein endothelial cells exposed 6.78 MHz RFR at 10 μ T for 24 h.	Increased expression of genes involved in promotion of cell proliferation and reduction of oxidative stress (carbonic anhydrase 8 (CA8), aldo-keto reductase family members including AKR1C1 and AKR1C3 and malic enzyme).	
Czyz et al. (2004)	Pluripotent embryonic stem (ES) cells (wild-type and deficient for the tumor suppressor p53 exposed to pulsed 1710 MHz RFR at GSM-217 (1.5 W/kg) or GSM-talk (0.4 W/kg) mode for 6–48 h.	Upregulation of mRNA levels of Hsp70.	Hsp70 protects cells from thermal and oxidative stress.
Dahon et al. (2025)	Human embryonic kidney HEK293 cells exposed to 1800 MHz RFR at 11×10^{-4} mW/cm ² - 0.66×10^{-4} μ W/cm ² for 15 min.	Increased expression of oxidative processes related genes (KIAA, RPS, GPX-1, SOD2). (Dose response nonlinear.)	KIAA gene promotes degradation of SLC43A2 mRNA. Downregulation of SLC43A2 reduces oxidative stress. The RPS gene product reduces oxidative stress induced cellular damage via a ribosomal-related process. GPX and SOD are antioxidant enzymes.
Ding et al. (2022)	Mouse embryo Balb/c-3T3 cells exposed to 1800 MHz RFR; 4 h/day for 40 or 60 days at 8 W/kg.	Induction of miRNA.	Expression of 13 miRNAs, including miRNA-124 that is involved in the microglial response to oxidative stress.
Koohestanidehaghi et al. (2023) ^a	Mouse preimplantation embryos exposed to 900–1,800 MHz RFR from a cellular phone or 30 min; 0.636–1.8 W/kg.	Increased Hsp70 and decreased superoxide dismutase (SOD) gene expressions.	Hsp70 protects cells from thermal and oxidative stress. SOD is an antioxidant enzyme.
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 mRNA and protein expression of Hsp70.	Hsp70 protects cells from thermal and oxidative stress.
Piccinetti et al. (2018) (LI)	Zebrafish embryos exposed to 100 MHz RFR from immediately after fertilization to 72 h post-fertilization, SAR about three orders of magnitude lower than ICNIRP basic restrictions of 0.08 W/kg.	At the 48 h post-fertilization stage- an increased transcription of oxidative stress genes and reduced growth.	

Table 3: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Porcher et al. (2023) (LI)	Leaves of <i>Arabidopsis thaliana</i> exposed to 2,450 MHz RFR for 30 min at 0.21 W/kg.	Increased transcripts of genes involved in ROS metabolism (Zat12 and APX1).	Zat12 plays a central role in reactive oxygen and abiotic stress signaling in Arabidopsis. APX1 has the role of scavenging reactive oxygen species for protection against oxidative damage and maintaining normal plant growth and development.
Valbonesi et al. (2014)	Rat PC12 cells exposed to continuous-wave 1.8 GHz RFR or GSM-217 Hz and GSM-Talk signals for 4, 6, or 24 h, SAR 2 W/kg.	After PC12 cells exposure to the GSM-217 Hz signal for 16 or 24 h, Hsp70 mRNA transcription significantly increased.	Hsp70 protects cells from thermal and oxidative stress.
Wang et al. (2022) (LI)	<i>Drosophila melanogaster</i> exposed to 3.5 GHz RFR for 2–3 days at 0.0026 W/kg, 0.026 W/kg, or 0.26 W/kg.	In third-instar larvae, increased expression levels Hsp70.	Hsp70 protects cells from thermal and oxidative stress.
Yang et al. (2012)	Hippocampus of rats exposed to 2,450 MHz RFR for 20 min at 6 W/kg.	23 upregulated and 18 downregulated genes were identified including Hsp70 genes.	Hsp70 protects cells from thermal and oxidative stress.
Yao et al. (2025)	Testis of rats exposed to 2,856 MHz RFR, 15 min/day, 5 day/week for 6 weeks; whole body SAR 9 W/kg; testis 19.8 W/kg; body temperature increase 0.83 °C.	Up-regulation of Gpx5 gene expression.	Gpx5 is related to oxidative stress

^aStudies that involved exposure to radiation from wireless devices, such as cell phones. (LI), Low-intensity study (SAR<0.4 W/kg). (Number of studies: acute exposure/*in vivo*=5; repeated (chronic) exposure/*in vivo*=4; acute exposure/*in vitro*=6; repeated (chronic) exposure/*in vitro*=1).

Table 4: Genes related to stress response.

References (see Appendix)	Exposure conditions	Results	Comments
Arslan et al. (2024) (LI)	Brain of rats exposed to 189 MHz RFR (217-Hz modulation); 2 h/day for 8 weeks at 0.06 W/kg.	p38MAPK gene expression up-regulated.	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.
Beaubois et al. (2007)	Tomato plants (Lycopersicon esculentum) exposed 90 MHz RFR at 5 V/m for 10 min.	Increased wound-inducible transcript bZIP.	Basic leucine zipper (bZIP) gene is one of the transcription factor families in plants and plays important roles in abiotic and biotic stress responses.
Bertuccio et al. (2024)	Human neuronal-like SH-SY5Y and peripheral blood mononuclear cells to 2,450 MHz RFR for 24 h, no dosimetry data.	Decreased Nrf2 (a transcription factor) gene expression in both cell types.	Nuclear factor erythroid 2-related factor 2 (NRF2) is involved in the cellular defense against toxic and oxidative insults through the expression of oxidative stress response and drug detoxification genes.
Bourdineaud et al. (2017) (LI)	<i>Eisenia fetida</i> (earthworm) exposed to 900 MHz for 2 h; SAR 0.00013–0.00933 W/kg.	Gene expression up regulated for MEK1.	MEK1 suppresses oxidative stress-induced apoptosis.
Buttiglione et al. (2007)	Human SH-SY5Y neuroblastoma cells exposed to modulated 900 MHz RFR for 24 h; SAR 1 W/kg.	Increased gene expression SAPK/JNK.	Stress-activated protein kinases (SAPK)/Jun N-terminal kinases (JNK) are activated by a variety of environmental stressors.
Cantu et al. (2023) (LI)	Human keratinocytes exposed to 900 MHz RFR for 1 h at 0.00155 W/kg.	Six common DNA targets were both differentially methylated and differentially expressed.	Up-regulation of DNA-damage induced 1 homolog 2 (DD12) increases cell survival following stress.

Table 4: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Chen G et al. (2012)	<i>Saccharomyces cerevisiae</i> yeast cells exposed to 1,800 MHz RFR for 6 h; SAR 4.7 W/kg.	Expressions of aquaporin 2 (AQY2 (m), whereas three other genes: halotolerance protein 9 (HAL9), another kinase 1 (YAK1) and one function-unknown gene (open reading frame: YJL171 C), showed opposite changes in expression.	The genes are involved in salt stress and growth of the bacteria.
Chow et al. (2024)	Human umbilical vein endothelial cells exposed 6.78 MHz RFR at 10 μ T for 24 h.	Increased expression of genes involved in promotion of cell proliferation and reduction of oxidative stress (carbonic anhydrase 8 (CA8), aldo-keto reductase family members including AKR1C1 and AKR1C3 and malic enzyme).	
Dahon et al. (2025)	Human embryonic kidney HEK293 cells exposed to 1800 MHz RFR at 11×10^{-4} mW/cm ² - 0.66×10^{-4} μ W/cm ² for 15 min.	Increased expression of oxidative processes related genes (KIAA and RPS). (Dose response nonlinear.)	KIAA gene promotes degradation of SLC43A2 mRNA. Downregulation of SLC43A2 reduces oxidative stress. The RPS gene product reduces oxidative stress induced cellular damage via a ribosomal-related process. Expression of 13 miRNA, including miRNA-124 involved in the microglial response to oxidative stress.
Ding et al. (2022)	Mouse embryo Balb/c-3T3 cells exposed to 1800 MHz RFR; 4 h/day for 40 or 60 days at 8 W/kg.	Changes in gene expression, and induction of miRNAs.	NF- κ B is involved in cellular responses to stimuli such as stress, cytokines, free radicals, heavy metals.
Ding et al. (2024)	RANKL-induced osteoclast differentiation in RAW264.7 cells exposed to 900 MHz CW RFR, 4 h/day for 5 days at 50, 150, or 450 μ W/cm ² (Osteoclasts involved in breakdown of bone tissue, and bone remodeling).	RFR decreased RANKL-induced NF- κ B activation, and suppressed the expression of downstream NF- κ B target genes, such as NFATc1 and TRACP. Effect most effective at 150 μ W/cm ² (0.150 mW/cm ²).	
El-Kafoury et al. (2023)	Hippocampus of rats exposed to three GSM phones (850–1,900 MHz) 2 h/day, 6 days/week, for 12 weeks (50 missed calls/h, 35 sec durations separated by 15 sec (No dosimetry data).	Increased expression of Sirt1 gene.	Sirt1 gene encodes enzymes involved in cellular regulation, e.g., stressors, longevity.
Hamann et al. (2006)	Rat sciatic nerve in mid-thigh, or to the L4 anterior primary ramus just distal to the intervertebral foramen exposed to 20 ms 500 KHz pulses at 2 Hz.	Up-regulation of transcription factor 3 (ATF3), an indicator of cellular “stress”. (Effect appeared to be selective on neurons whose axons are the small diameter C and A delta nociceptive fibers.)	Cyclic AMP-dependent transcription factor ATF-3 is a transcription factor regulating gene expression.
Lameth et al. (2020)	Healthy rats undergoing acute neuroinflammation triggered by a lipopolysaccharide (LPS) treatment, and transgenic hSOD1 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 1.21 or 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. At 1.21 W/kg, downregulation of Parg, Akna, Fam107a, and Crif1 genes were observed in the LPS-treated rats. No effect on healthy animals.	Fam107a is a stress-inducible actin-binding protein that plays a role in synaptic and cognitive functions.
Lee K-S et al. (2008)	<i>Drosophila</i> exposed to 833 MHz RFR for 12 or 18 h at 1.6 or 4 W/kg.	1.6 W/kg activated an anti-apoptotic gene, whereas SAR 4.0 W/kg activated expression of JNK gene.	JNK1 is involved in response to reactive oxygen species, and a variety of stress stimuli can activate JNK.
Manta et al. (2017) (L1) ^a	Four days-old adult female flies (<i>Drosophila melanogaster</i>) exposed to GSM-1800 talk mode RFR emitted by a cellular phone for 30 min; SAR 0.15 W/kg.	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.	

Table 4: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Nikolova et al. (2005)	Mouse embryonic neural progenitor stem cells exposed to 1,710-MHz GSM RFR for 6 or 48 h; SAR 1.5 W/kg.	Up-regulation of GADD45 gene expression.	GADD45 genes are stress sensors that modulate the response of mammalian cells to genotoxic/physiological stress.
Ohtani et al. (2016)	Sprague-Dawley rats exposed to wideband code division multiple access 2,140 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. At the 48 h post-fertilization stage- an increased transcription of oxidative stress genes and reduced growth.	Heat-shock transcription factors regulate the expression of heat shock proteins. Its functions to overcome the proteotoxic effects of thermal stress.
Piccinetti et al. (2018) (LI)	Zebrafish embryos exposed to 100 MHz RFR from immediately after fertilization to 72 h post-fertilization, SAR about three orders of magnitude lower than ICNIRP basic restrictions of 0.08 W/kg.		
Porcher et al. (2023) (LI)	Leaves of <i>Arabidopsis thaliana</i> exposed to 2,450 MHz RFR for 30 min at 0.21 W/kg.	Increased transcripts of stress-related ZAT12 transcription factor).	Zat12 plays a central role in reactive oxygen and abiotic stress signaling in Arabidopsis.
Roux et al. (2006)	Tomato plants exposed to a 900-MHz RFR, 2–10 min, 5–40 V/m.	Stress-related transcripts (calmodulin, protease inhibitor and chloroplast mRNA-binding protein) increased 15 min after the end of exposure (10 min), 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.	
Suzuki et al. (2025)	<i>Arabidopsis thaliana</i> exposed to 2,450 MHz RFR for 1 h (No dosimetry data).	Changed expression of genes involved in altered circadian clock as well as hormonal response especially in auxin and gibberellin, which promoted plant growth by inducing amino acid biosynthesis and stress tolerance.	
Tran et al. (2023) ^a	Lettuce plants (<i>Lactuca sativa</i>) exposed to 1,800–1,900 MHz (0.0008 mW/cm ²) and 2,450 and 5,000 MHz RFR (0.0002 mW/cm ²) from DECT base station and phones for 2–6 weeks.	Down-regulation of two stress-related genes of violaxanthin de-epoxidase and zeaxanthin epoxidase.	
Vafaei et al. (2020) (LI) ^a	Placenta of pregnant mice exposed to 2.4 GHz RFR from a D-link Wi-Fi router; 2 or 4 h/day at 30 or 60 cm from router from 5 days after mating to 1 day before expected delivery, 0.09 W/kg at 30 cm.	Increased gene expression of GADD45a gene.	The GADD45 genes transcript levels are increased following stressful growth arrest conditions.
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).	The bZIP gene family plays important roles in stress responses in plants.
Wang et al. (2022) (LI)	<i>Drosophila melanogaster</i> exposed to 3.5 GHz RFR for 2–3 days at 0.0026 W/kg, 0.026 W/kg, or 0.26 W/kg.	In third-instar larvae, expression of TotA gene was significantly increased.	The <i>Drosophila</i> Turandot A (TotA) gene encodes a stress-induced humoral factor which gives increased resistance to the lethal effects of high temperature.
Zhao TY. et al. (2007) ^a	Primary cultured neurons and astrocytes exposed to a GSM 1,900 MHz cellular phone for 2 h.	Up-regulation of caspase-2 gene. Neurons appeared to be more sensitive to this effect than astrocytes.	In addition to apoptotic role, caspase-2 also contributes to maintaining genomic stability and responding to cellular stress.

^aStudies that involved exposure to radiation from wireless devices, such as cell phones. (LI), Low-intensity study (SAR<0.4 W/kg). (Number of studies: acute exposure/*in vivo*=11; repeated (chronic) exposure/*in vivo*=6; acute exposure/*in vitro*=10; repeated (chronic) exposure/*in vitro*=1).

Table 5: Genes involved in apoptosis.

References (see Appendix)	Exposure conditions	Results	Comments
Almášiová et al. (2025)	Mesonephros (temporary urinary excretory system in developing animals) of chick embryos (in eggs) exposed to 2,450 MHz RFR for 9 days; 0.00002–0.00005 mW/cm ² .	Up-regulation of caspase-1 gene expression (increased apoptosis and cell proliferation).	Caspase-1 initiates apoptosis in response to certain stressors such as pathogen infection.
Arslan et al. (2024) (LI)	Brain of rats exposed to 189 MHz RFR (217 Hz modulation); 2 h/day for 8 weeks at 0.06 W/kg.	p38MAPK gene expression up-regulated.	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.
Azimipour et al. (2020) ^a	Pre-antral ovary follicles of mouse exposed to radiation from a Sony Ericsson K800 cellular phone for 1 h (No dosimetry data available).	Increased the matrix metalloproteinases MMP-2 gene and decreased MMP-9 gene expression.	Matrix metalloproteinases (MMP) are proteases capable of degrading all kinds of extracellular matrix proteins. They cause release of apoptotic ligands and are involved in cell proliferation, migration (adhesion/dispersion), differentiation, angiogenesis, apoptosis, and host defense.
Balakrishnan et al. (2014) ^a Balci et al. (2025)	Serum of cellular phone frequent and infrequent users. Kidney of rats with acute kidney injury caused by lipopolysaccharide (LPS) exposed to 27.2 MHz RFR at 10 v/m for 3 h.	Increased Hsp70 gene expression with phone use. RFR reversed effects of LPS on gene expression of Bcl-2.	Hsp70 inhibits apoptosis. Bcl-2 blocks apoptosis.
Bertuccio et al. (2024)	Human neuronal-like SH-SY5Y and peripheral blood mononuclear cells to 2,450 MHz RFR for 24 h (No dosimetry data).	Increased BAX/BCL2 ratio (apoptosis-related), gene expression in SH-SY5Y only (with increased in caspase level).	BAX is a pro-apoptosis protein that affects the voltage-gated anion channels on mitochondrial membrane. BCL2 blocks apoptosis.
Bourdineaud et al. (2017) (LI)	<i>Eisenia fetida</i> (earthworm) 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, MEK1 (signal transduction); oxidative stress; and chemical and immune defenses.	MEK1 suppresses oxidative stress-induced apoptosis.
Burtigione et al. (2007)	Human SH-SY5Y neuroblastoma cells exposed to modulated 900 MHz RFR for 24 h; SAR 1 W/kg.	Decrease in mRNA of Bcl-2.	Bcl-2 blocks apoptosis.
Cantu et al. (2023) (LI)	Human Keratinocytes exposed to 900 MHz RFR for 1 h at 0.00155 W/kg.	Six common DNA targets were both differentially methylated and differentially expressed.	Six genes related to cell survival and responses to damage were affected (SPDYA, BBS5, CCS, DOK3, DD12, and MGA). Particularly, down-regulation of docking protein-3 (DPK3) could negatively regulate C-Jun N-terminal kinase (JNK) leading to apoptosis.
Cappucci et al. (2022) (LI)	Head, ovary, and testis of <i>Drosophila melanogaster</i> exposed to 2,437 MHz RFR (Wi-Fi) from embryo to adult stage at 0.0608 W/kg.	Increased gene expression of Hsp70, induced gene instability, and increased DNA damage.	Hsp70 inhibits apoptosis.
Cheon et al. (2023)	SK-MEL-3 melanoma cells exposed to pulsed 1.6 THz RFR (1 kHz repetition) for 1–48 h at 14.7 mW/cm ² .	Downregulated genes involved in cancer and apoptosis pathways (FOS, JUN, and CXCL8), DNA demethylation involved.	c-jun is an oncogenic transcription factor. It is required for progression through the G1 phase of the cell cycle. It has anti-apoptotic activity and is required for tumor cell survival between the initiation and progression stages.

Table 5: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Czyz et al. (2004)	Pluripotent embryonic stem (ES) cells (wild-type and deficient for the tumor suppressor p53 exposed to pulsed 1,710 MHz RFR at GSM-217 (1.5 W/kg) or GSM-talk (0.4 W/kg) mode for 6–48 h.	Upregulation of mRNA levels of the heat shock protein, Hsp70 and a low and transient increase of c-jun, c-myc, and p21 levels in p53-deficient, but not in wild-type cells.	Hsp70 inhibits apoptosis. c-jun has anti-apoptotic activity and is required for tumor cell survival between the initiation and progression stages.
Dasdag et al. (2015b) (LI)	Brain of rats exposed to 2,450-MHz RFR 24 h/day for 1 year; 0.004 W/kg.	Decreased expression of microRNA miR-106b-5p and miR-107.	c-myc gene is a transcription factor that regulates cell cycle progress, proliferation, growth, adhesion, differentiation, apoptosis, and metabolism. miR-106b-5p acts as an oncomiR or tumor suppressor via regulating almost all cancer cell biological processes, including cell cycle, proliferation, apoptosis, differentiation, invasion, angiogenesis, drug resistance and metastasis.
Deena et al. (2025) ^a	Human SH-SY5Y neuroblastoma cells; 2,400 MHz RFR from cellular phone for 4 h (No dosimetry data).	Increased BAX gene expression, no effect on Bcl2 expression (increased apoptosis).	BAX is a pro-apoptosis protein. BCL2 blocks apoptosis.
Ding et al. (2024)	RANKL-induced osteoclast differentiation in RAW264.7 cells exposed to 900 MHz CW RFR, 4 h/day for 5 days at 50, 150, or 450 $\mu\text{W}/\text{cm}^2$ (Osteoclasts involved in breakdown of bone tissue, and bone remodeling).	RFR decreased RANKL-induced NF- κB activation and suppressed the expression of downstream NF- κB target genes, such as NFATc1 and TRACP. Effect most effective at 150 $\mu\text{W}/\text{cm}^2$ (0.150 mW/cm ²).	NFATc1 and TRACP are involved in osteoclast differentiation, increase osteoclast apoptosis and decreased osteoclast differentiation.
Eker et al. (2018) (LI)	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.	The CASP3 protein is a member of the cysteine-aspartic acid protease (caspase) family. Activated caspases plays a central role in the execution-phase of cell apoptosis. p38 mitogen-activated protein kinases are a class of mitogen-activated protein kinases (MAPKs) that are responsive to stress stimuli, such as heat shock, and osmotic shock, and are involved in cell differentiation, apoptosis, and autophagy.
Franzellitti et al. (2008)	Human trophoblasts HTR-8/SVneo exposed to 1,800 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.	Intracellularly localized Hsp70s inhibit apoptosis.
Ghatei et al. (2017) ^a	Mice exposed pre- and postnatally to radiation from a cellular phone jammer (900 and 1,800 MHz).	At 8–10 weeks old, gene expression level of <i>bax</i> was decreased and gene expression level of <i>p21</i> was increased.	BAX is a pro-apoptosis protein that affects the voltage-gated anion channels on mitochondrial membrane.
Harvey and French (1999)	Humen mast cell HMC-1 exposed to 864.3 MHz RFR for three 20 min/day for 7 days at 7 W/kg.	Up-regulation of the apoptosis-associated gene DAD-1, and some stress response genes.	DAD1, the defender against apoptotic cell death, is a negative regulator of apoptosis.
Islam et al. (2023) ^a	Liver, brain and caecal tonsil samples taken from embryos in chick eggs exposed to 2,100 MHz RFR from cellular phones for 60 min/day for 14 days.	Vascular gene (VEGF-A) mRNA expression was higher in the exposed group.	Vascular endothelial growth factor (VEGF) acts on endothelial cells and increased vascular permeability, inducing angiogenesis, vasculogenesis, and endothelial cell growth, promoting cell migration, and inhibiting apoptosis.

Table 5: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Ivaschuk et al. (1997)	Rat PC12 pheochromocytoma cells treated with nerve growth factor exposed to 836.55 MHz TDMA signal for 20, 40, or 60 min at 0.00026, 0.0026 or 0.026 W/kg.	Decreased in c-jun gene expression at 0.026 W/kg after 20 min exposure (no effect at longer exposure duration); no effect on c-fos gene expression.	c-jun is an oncogenic transcription factor. It is required for progression through the G1 phase of the cell cycle. It has anti-apoptotic activity and is required for tumor cell survival between the initiation and progression stages.
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.	Signal transducer and activator of transcription 3 (STAT3) mediates the expression of a variety of genes in response to cell stimuli, and plays a key role in many cellular processes such as cell growth and apoptosis.
Kim JH et al. (2021)	SH-SY5Y neuronal cells exposed to 1,760 MHz RFR 4 h/day for 4 days; 4 W/kg.	Transcriptional activation of p53.	P53 initiates apoptosis when DNA damage is irreparable.
^a Koohestanidehaghghi et al. (2023)	Mouse preimplantation embryos exposed to 900–1,800 MHz RFR from a cellular phone or 30 min; 0.636–1.8 W/kg.	Increased Hsp70 gene.	Hsp70 inhibits apoptosis.
Kucukbagciak et al. (2022) (LI)	Liver and blood of mice exposed to 900 MHz GSM RFR; 4 h/day for 7 days; 0.339 W/kg.	Increased expression of p53 gene.	P53 activates DNA repair proteins in response to DNA damage, causes cell cycle arrest at the G1/S point, initiates apoptosis when DNA damage is irreparable.
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 were found to be potentially modulated. PCR confirmed 5 genes (CRIP2, PLXND1, PTX3, SERPINF1, and TRPV2) were differentially expressed after 6 h of exposure.	SERPINF1 has antiangiogenic, antitumorigenic, and neurotrophic properties. Inhibit cancer cell proliferation and increase apoptosis.
Lee K-S et al. (2008)	Drosophila exposed to 833 MHz RFR for 12 or 18 h, 1.6 or 4 W/kg.	1.6 W/kg activated an anti-apoptotic gene, whereas SAR 4.0 W/kg activated expression of apoptotic genes (JNK and ERK).	JNK1 is involved in apoptosis, neurodegeneration, cell differentiation and proliferation. Inflammation, reactive oxygen species, and a variety of stress stimuli can activate JNK.
Lee S et al. (2005)	HL-60 cells exposed to a pulsed 2,450 MHz RFR (155 μ s, duty cycle 7.5 %) for 2 or 6 h; 10 W/kg.	Number of gene expressions depended on exposure duration.	ERK (and related pathways) is involved in cell proliferation, differentiation, motility, and survival. ERK1/2 kinases also have pro-apoptotic functions and enhanced ERK1/2 signaling can cause tumor cell death.
Lixia et al. (2006)	Human lens epithelial cells exposed to GSM-1800 MHz RFR for 2 h, SAR 1, 2, 3 W/kg.	Increased mRNA and protein expression of Hsp70.	Apoptosis-related genes were among the upregulated ones and the cell cycle genes among the downregulated ones.
Marinelli et al. (2004) (LI)	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 of genes involved in pro-survival signaling (Bcl-2, Ras, Akt1).	Hsp70 inhibits apoptosis. Bcl-2 blocks apoptosis. Ras-regulated signal pathways is involved in actin cytoskeletal integrity, cell proliferation, cell differentiation, cell adhesion, apoptosis, and cell migration. Akt1 is a mediator of growth factor-induced neuronal survival and inhibitor of apoptosis.

Table 5: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Martin et al. (2020)	Four types of human keratinocytes exposed to 60 GHz RFR for 3 h at 594 W/kg.	With four keratinocyte cell types, three different genes (ADAMTS6, IL7R, NOG) expression patterns (down-regulation, upregulation, and no effect) were observed, despite their exposure having been the same in all regards.	Interleukin-7 receptor (IL7R) plays a critical role in the development of lymphocytes by blocking apoptosis.
Nikolova et al. (2005)	Mouse embryonic neural progenitor stem cells exposed to 1,710-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 down-regulation of neural-specific Nurr1 and in up-regulation of Bax and GADD45 gene expression.	Bax is a pro-apoptosis protein.
Ozden et al. (2025)	Blood from terminal ileum of rats subjected to mesenteric artery ischemia and exposed to 27.12 MHz RFR before or after ischemia; 30 min at 10 V/m.	Ischemia-induced increased gene expression of VEGF, BAX, and HIF1α and decreased gene expression of BCL-2 and eNOS attenuated by RFR.	Vascular endothelial growth factor (VEGF) acts on endothelial cells and increased vascular permeability, inducing angiogenesis, vasculogenesis, and endothelial cell growth, promoting cell migration, and inhibiting apoptosis. Bax is a pro-apoptosis protein. BCL2 blocks 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 3, G2/mitotic-specific cyclin G1), cell growth inhibitors (e.g., transforming growth factor-beta), and genes controlling apoptosis (e.g., Bax).	BCL2 blocks apoptosis.
Saka et al. (2023) ^a	Primary cortical neurons from neonatal rat cerebral cortex exposed to 2,100 MHz RFR for 2 h at 1.6 W/kg.	Increased expression of Bax and p53 genes, decreased expression of Bcl 2 gene.	Bax is a pro-apoptosis protein. Bcl2 blocks apoptosis.
Son et al. (2023)	Hippocampus of 5xFAD mice (mouse model of Alzheimer's disease) exposed to 1950 MHz RFR, 2 h/day, 5 days/week for 6 months at 5 W/kg.	Decreased expression of genes related to microgliosis and microglial functions, and interleukin-1β.	Interleukin-1β is a cytokine that mediates inflammatory response, and is involved in a variety of cellular activities, including cell proliferation, differentiation, and apoptosis.
Sueiro-Benavides et al. (2023)	HL-60 cells exposed to 2,450 MHz RFR for 8, 24, or 48 h, 0.406 W/kg.	Increased FAS-R gene (related to apoptosis) expression after 24 and 48 h exposure. Interacted with black carbon particles.	The FAS receptor is on the surface of cells that leads to apoptosis when it binds to its ligand.
Tarsaei et al. (2022)	Hippocampus of rats exposed to 2,450 MHz RFR for 7 or 30 days, at 4 mW/cm ² .	Increased Bax (Bcl2-associated) and reduced Bcl-2 (B-cell lymphoma 2) genes expression. (Genes are related to apoptosis.)	Increase apoptosis.
Tohidi et al. (2021) ^a	Hippocampus of mice exposed to RFR from a mobile phone jammer with 4 bands (900 MHz, 1,800 MHz, CDMA and GSM) for 0.5, 1, 2, or 4 h twice a day for 30 days, or 4 h/day for 30 days.	Expression of both Bax and Bcl2 genes (related to apoptosis) were upregulated in mice exposed for one and 2 h, and downregulated in animals with longer exposure.	Bax is a pro-apoptosis protein. Bcl2 blocks apoptosis.
Tuysuz et al. (2025)	Astrocyte-derived U118-MG cells exposed to 2,100 MHz RFR for 1, 24, or 48 h at 1.12 W/kg.	Increased CASP3, CASP8, and CASP9 mRNA after 24 or 48 h exposure. (Apoptosis after 48 h exposure with an increase in BAX/BCL-2 ratio, that initiates apoptosis.)	Caspase-3 is a caspase protein that plays an important role in apoptosis. It interacts with caspase-8 and caspase-9.

Table 5: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Vafaei et al. (2020) (LI) ^a	Placenta of pregnant mice exposed to 2,400 MHz RFR from a D-link Wi-Fi router; 2 or 4 h/day at 30 or 60 cm from router from 5 days after mating to 1 day before expected delivery, 0.09 W/kg at 30 cm.	Increased gene expression of SOD, and GSKN1A, and GADD45a (DNA repair enzymes).	CDKN1A functions as a regulator of cell cycle progression at G1. CDKN1A plays a critical role in the cellular response to DNA damage. The GADD45 genes transcript levels are increased following stressful growth arrest conditions and treatment with DNA-damaging agents, mediating cell cycle arrest at G2/M phase and inducing apoptosis. Hsp70 inhibits apoptosis.
Valbonesi et al. (2014)	Rat PC12 cells exposed to continuous-wave 1800 MHz RFR or GSM-217 Hz and GSM-Talk signals for 4, 6, or 24 h, SAR 2 W/kg.	After PC12 cells exposure to the GSM-217 Hz signal for 16 or 24 h, Hsp70 mRNA transcription significantly increased, whereas no effect was observed in cells exposed to the CW or GSM-Talk signals. In third-instar larvae, expression levels of the heat shock protein genes Hsp22, Hsp26 and Hsp70 and humoral immune system genes AttC, TotC and TotA were all significantly increased.	Hsp70 inhibits apoptosis.
Wang Y et al. (2022) (LI)	<i>Drosophila melanogaster</i> exposed to 3,500 MHz RFR for 2–3 days at 0.0026 W/kg, 0.026 W/kg, or 0.26 W/kg.		
Wu H et al. (2012)	At Sertoli cells exposed to s-band microwaves for 4 min at 100 mW/cm ² .	Upregulation of Bax/Bcl-2 and caspase-3 genes.	Increase apoptosis
Yang et al. (2012)	Hippocampus of rats exposed to 2,450 MHz RFR for 20 min at 6 W/kg.	23 upregulated and 18 downregulated genes were identified including Hsp27 and Hsp70 genes.	The main function of Hsp27 is to provide thermotolerance <i>in vivo</i> , cyto-protection, and support of cell survival under stress conditions. Inhibiting protein aggregation and by stabilizing partially denatured proteins. It has anti-apoptotic properties and roles in cell growth (termination) and inflammatory and stress responses. Hsp70 inhibits apoptosis. Increase apoptosis.
Yavas et al. (2024) (LI)	Brain and testis of exposed rats; 2,100 MHz RFR; 5 h/day for 14 days; 10 g and 1 g SAR (W/kg)- brain 0.292 and 0.578; testes 0.163 and 0.316.	Brain- decreased Bax (pro-apoptosis), increased Bcl-2 (apoptosis blocker), no effect on p52 gene expressions; increased double strand DNA breaks. Testes- decreased Bax gene expression.	
Zalata et al. (2015) ^a	Human semen samples exposed to 850-MHz RFR from a cellular phone for 1 h; SAR 1.46 W/kg at 10 cm.	Significant increase in sperm DNA fragmentation percent, clusterin gene expression and clusterin protein (associated with clearance of cellular debris and apoptosis) levels in the exposed semen samples.	Clusterin is an extracellular molecular chaperone which binds to misfolded proteins in body fluids to neutralize their toxicity and mediate their cellular uptake by receptor-mediated endocytosis and transfer into lysosomes for degradation.
Zhao TY. et al. (2007) ^a	Primary cultured neurons and astrocytes exposed to a GSM 1900 MHz cellular 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.	Caspase-6 plays a central role in the execution-phase of cell apoptosis. Asc is involved in the inflammatory and apoptotic signaling pathways via the activation of caspase.

^aStudies that involved exposure to radiation from wireless devices, such as cell phones. (LI), Low-intensity study (SAR<0.4 W/kg). (Number of studies: acute exposure/*in vivo*=7; repeated (chronic) exposure/*in vivo*=15; acute exposure/*in vitro*=22; repeated (chronic) exposure/*in vitro*=4).

Table 6: Genes related to brain functions.

References (see Appendix)	Exposure conditions	Results	Comments
An et al. (2023)	Mouse embryonic neural stem cells exposed to 1950 Hz RFR for 48 h at 2 W/kg.	Altered gene expression of Bbs1, Slc38a3, and Vps18 genes.	Bbs1, Slc38a3, and Vps18 are involved in neuro-development and brain functions.
Arslan et al. (2024) (LI)	Brain of rats exposed to 189 MHz RFR (217 Hz modulation); 2 h/day for 8 weeks at 0.06 W/kg.	p38MAPK gene expression up-regulated.	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.
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.	Early growth response protein-1 (Egr-1) is a transcriptional regulator in the brain. It is a tumor suppressor gene and has a role in neuronal plasticity.
Chen et al. (2021)	Neural stem cells-derived neurons and retinoic acid-induced Neuro-2A cells exposed to 1800 MHz GSM-talk mode signal (5 min ON/10 min OFF) for 48 h at 4 W/kg.	Gene expression of Eph receptors 5 (EPHA5) was inhibited.	EPHA5 is required for neurite outgrowth.
Dasdag et al. (2015a) (LI)	Brain of rats exposed to 900 MHz RFR 3 h/day. 7 day/week for 12 months; 0.0369 W/kg.	Decreased rno-miR107.	Involved in cancer progression
Dasdag et al. (2015b) (LI)	Brain of rats exposed to 2450-MHz RFR 24 h/day for 1 year; 0.004 W/kg.	Decreased expression of miR-106b-5p and miR-107.	miR-106b-5p acts as a tumor suppressor via regulating almost all cancer cell biological processes, including cell cycle, proliferation, apoptosis, differentiation, invasion, angiogenesis, drug resistance and metastasis.
Dasdag et al. (2019) (LI)	Brain of rats exposed to 900 MHz FR 3 h/day (7 days a week) for 12 months, 0.198 W/kg.	Increased expression of rno-miR-145-5p.	A tumor suppressor miRNA in diverse types of cancers, including bladder cancer, breast cancer, cervical cancer, cholangiocarcinoma, renal cancer, and gastrointestinal cancers.
Dasdag et al. (2022) (LI)	Brain of rats exposed to 2,400 MHz RFR emitted from a Wi-Fi generator feed to an antenna for 24 h/day for one year; 0.000328 W/kg.	Increased rno-miR-181a-5p and membrane lipids.	MIR-181a-5p regulates angiogenesis and inflammatory response. It exerts regulatory influence on cancer development and progression.
El-Kafoury et al. (2023)	Hippocampus of rats exposed to three GSM phones (850–1,900 MHz) 2h/day, 6 days/week, for 12 weeks (50 missed calls/h, 35 sec durations separated by 15 sec (No dosimetry data).	Increased expression of Sirt1 gene and Atg-7 gene. (Effects reversed by lipoic acid, an antioxidant).	Sirt1 gene encodes enzymes involved in cellular regulation, e.g., stressors, longevity. Atg-7 gene is involved in cell degeneration and recycling (autophagy).
Gökçek-Saraç et al. (2021)	Hippocampus of rats exposed to UMTS 2,100 MHz signal, 2 h/day for 7 days; 0.41 or 1.3 W/kg.	Decreased mRNA expressions of acetylcholinesterase, choline acetyltransferase and vesicular acetyl-choline transporter.	
Huang et al. (2023)	Rat oligodendroglial exposed to CW or pulsed (50-Hz) 2,400 MHz RFR for 6 or 48 h at SAR 0.23–0.8 W/kg (in medium of culture disk).	Change in expression of CCAAT/Enhancer-Binding Protein β (C/EBP β) genes after pulsed RFR exposure (no effect with CW and on three other types of brain cells).	CCAAT/Enhancer-Binding Protein β (C/EBP β) genes play a significant role in the development and function of nerve cells.
Jeong et al. (2020)	Hippocampus of two groups of C57BL/6 mice, aged 2 and 12 months, exposed to 1,950-MHz RFR 2 h/day, 5 days/week for 8 months: 5 W/kg.	15 genes involved in neurogenesis, were altered in both groups: increased gene expression of EphA8 in the hippocampi of the older group.	EphA8 plays a role in short-range contact-mediated axonal guidance during development of the mammalian nervous system.

Table 6: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
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.	Decreased expression of the STAT3 genes.	Signal transducer and activator of transcription 3 (STAT3) mediates the expression of a variety of genes in response to cell stimuli, and plays a key role in many cellular processes such as cell growth and apoptosis.
Lai et al. (2023)	Rats exposed to 1,500 MHz RFR (10.57 W/kg, 15 min) or EMP (400 pulses, 1 Hz, peak 11.65 kV/m) or combination of the two fields.	Increased mRNA expression of PTGS2 gene in hippocampus.	PTGS2 (COX-2) converts arachidonic acid (AA) to prostaglandin endoperoxide H ₂ .
Lameth et al. (2020)	Healthy rats undergoing acute neuroinflammation triggered by a lipopolysaccharide (LPS) treatment, and transgenic hSOD1 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 1.21 or 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. At 1.21 W/kg, downregulation of Fam107a, and Crif1 genes were observed in the LPS-treated rats. No effect on healthy animals.	Fam107a is a stress-inducible actin-binding protein that plays a role in synaptic and cognitive functions. Crif1 gene encodes a member of the cytokine type I receptor family. The protein forms a complex with other cytokine factors that can promote the survival of neurons.
Lameth et al. (2025)	Rats with head exposed to 3.5 GHz RFR, 1 h/day (5 days/week) for 6 weeks. Whole brain SAR 0.19 W/kg (antenna on right side; right cerebral hemisphere 0.43 W/kg, left hemisphere 0.14 W/kg).	Cells in entorhinal and piriform cortex showed up-regulation of genes related to glutamatergic synapses, and genes of the mitochondrial genome related to oxidative phosphorylation system. (More genes were activated in the left than the right cerebral hemisphere).	
Li et al. (2020)	Pregnant rats exposed to 1800 + 2,400 MHz or 2,400 MHz RFR beginning on the 21st day of pregnancy; hippocampus of offspring examined at 7 weeks postnatal.	Altered mRNA expression of NMDA receptors. (Down and up-regulation dependent on the subtypes of receptor.)	NMDA receptors are involved in synaptic plasticity and learning and memory functions
Li et al. (2022)	PC12 cells and 293T cells exposed to 2,856 MHz RFR, 5 min 3X at intervals of 5 min, 19 W/kg.	Transcriptional activity of 5-HT _{1A} receptor promoter containing rs198585630 C allele was higher than that of 5-HT _{1A} receptor promoter containing T allele (polymorphism). <i>In vivo</i> experiments with rats showed cognitive deficits and inhibition of brain electrical activity.	
Megha et al. (2015) (LI)	Fischer rats exposed to 900 and 1,800 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 down-regulation of mRNA of tyrosine hydroxylase and tryptophan hydroxylase (synthesizing enzymes for the transmitters) in the hippocampus.	
Park et al. (2018)	HT22 mouse hippocampal neuronal cells exposed to 1950 MHz RFR 2 h/day for 3 days at 6 W/kg.	mRNA levels of APP, BACE1, ADAM10 and PSEN1 were decreased in HT22 cells.	Amyloid-beta precursor protein (APP) is an integral membrane protein expressed in the synapses of neurons. It is involved in synapse formation and neural plasticity; it is the precursor molecule of amyloid beta (Aβ). BACE1, ADAM10 and PSEN1 are secretase enzymes that process APP and involved in the formation of Aβ.

Table 6: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
Son et al. (2023)	Hippocampus of 5xFAD mice (mouse model of Alzheimer's disease) exposed to 1950 MHz RFR, 2 h/day, 5 days/week for 6 months at 5 W/kg.	Decreased expression of genes related to microgliosis and microglial functions, and interleukin-1 β .	Interleukin-1 β is a cytokine that mediates inflammatory response, and is involved in a variety of cellular activities, including cell proliferation, differentiation, and apoptosis.
Sun L. et al. (2025)	Blood of rats exposed to 2,800 and/or 9,300 MHz RFR, 6 min at 10 mW/cm ² .	Increased Htt and Bdnf gene expression. (Huntingtin (Htt) is the protein coded in humans by the HTT gene. Mutated HTT is the cause of Huntington's disease. It also is involved in long-term memory storage. BDNF (Brain-derived neurotrophic factor) acts on certain neurons of the central and peripheral nervous system, helping to support survival of existing neurons, and encouraging growth and differentiation of new neurons and synapses.)	
Tarsaei et al. (2022)	Hippocampus of rats exposed to 2,450 MHz RFR for 7 or 30 days, at 4 mW/cm ² .	Increased Bax (Bcl2-associated) and reduced Bcl-2 (B-cell lymphoma 2) genes expression. (Genes are related to apoptosis.)	Increase apoptosis.
Tohidi et al. (2021) ^a	Hippocampus of mice exposed to RFR from a cellular phone jammer with 4 bands (900 MHz, 1,800 MHz, CDMA and GSM) for 0.5, 1, 2, or 4 h twice a day for 30 days, or 4 h/day for 30 days.	Expression of both <i>Bax</i> and <i>Bcl2</i> genes (related to apoptosis) were upregulated in mice exposed for one and 2 h, and downregulated in animals with longer exposure.	
Xue et al. (2024)	Brain (hippocampus, thalamus, and cerebrum) of rats exposed to dual frequency 2,650/800 MHz RFR (each frequency for 2 h for 21 days); SAR 4 W/kg.	A decrease in the expression of <i>Cnr1</i> , encoding cannabinoid receptor 1 Type (CB1R) gene, only in the cerebrum.	CB1-receptors are brain development, learning and memory, motor behavior, regulation of appetite, body temperature, pain perception, and inflammation.
Yang et al. (2012)	Hippocampus of rats exposed to 2,450 MHz RFR for 20 min at 6 W/kg.	23 upregulated and 18 downregulated genes were identified including Hsp27 and Hsp70 genes.	The main function of Hsp27 is to provide thermotolerance <i>in vivo</i> . cyto-protection, and support of cell survival under stress conditions. Inhibiting protein aggregation and by stabilizing partially denatured proteins. It has anti-apoptotic properties and roles in cell growth (termination) and inflammatory and stress responses.
Yavas et al. (2024) (LI)	Brain of exposed rats to 2,100 MHz RFR; 5 h/day for 14 days; 10 g and 1 g SAR (W/kg)- brain 0.292 and 0.578; testes 0.163 and 0.316.	Decreased Bax (pro-apoptosis), increased Bcl-2 (apoptosis blocker).	Hsp70 stabilizes partially synthesized protein and prevents aggregation, protects cells from thermal and oxidative stress, disposal of damaged proteins, and inhibits apoptosis. Increase apoptosis.

^a Studies that involved exposure to radiation from wireless devices, such as cell phones. (LI). Low-intensity study (SAR<0.4 W/kg). (Number of studies: acute exposure/*in vivo*=4; repeated (chronic) exposure/*in vivo*=16; acute exposure/*in vitro*=5; repeated (chronic) exposure/*in vitro*=2).

suppression or progression since these genes are involved in cancer progression suppression, and angiogenesis. There are other studies in the list reporting that RFR activates genes that promote apoptosis and autophagy (see the section on ‘apoptosis’ above). This would suggest that RFR could affect the risk of cancer development in the brain. For cancer to develop, there must be some other processes triggered by RFR that promote tumor initiation and growth. With the interplay of activation of pro- and anti-apoptosis genes in brain cells (see the section on ‘apoptosis’ above), it is possible to observe either a decrease or an increase in brain cancer risk after RFR exposure [31, 32]. On the other hand, apoptotic cell death in the nervous system could lead to neurodegenerative diseases, such as Alzheimer’s and Parkinson diseases [3].

Discussion

Gene expression provides a strong argument that RFR affects cellular functions. It is a direct response to the exogenous disturbance and a homeostatic process to restore normalcy. In addition to the genes of the different functions described above, the effects of RFR on genes regarding other biological functions (e.g., wound healing and cellular structure) have also been reported (see Appendix 1 in Supplementary Material). It must be pointed out that why specific genes were chosen for various studies was not necessarily guided by a hypothesis or speculation but rather by the expertise of the investigators.

Gene expression indicates the type of effects that RFR exerts on cells. For example, it is irrational to argue that DNA damage-repair genes are turned on without DNA damage in the cells that initiated the process to begin with.

Gene expressions are under feedback control in response to external challenges with numerous variables. RFR has a complex interaction with living organisms depending on various parameters of the field. With these considerations in mind, response oscillation and non-linear dose-response could occur. But gene expression is not expected under all exposure conditions and biological systems studied. Thus, inevitably, in some studies, no significant effects would be expected. There are studies that reported no

significant effect of RFR on gene expression related to protein damage and removal, DNA, oxidative processes, stress, and brain functions (Table 7).

Forty gene expression effects (marked as low intensity (LI) in Tables 1–6) were observed at a SAR less than 0.4 W/kg. This is relevant to the effects of exposure to RFR in the environment. 0.4 W/kg is the dose rate assumed to be safe by international radiofrequency radiation guidelines (e.g., the International Commission on Non-Ionizing Radiation Protection (ICNIRP) and the International Electronics and Electrical Engineers (IEEE)). This is important to understand regarding the validity of those organization’s high-intensity standards-setting models. (For readers who are interested in ‘low-intensity’ RFR effects, there are more than 200 studies that reported biological effects at SAR <0.4 W/kg [see the ‘RFR-low-intensity studies’ file in reference [1]]).

Lastly, one may wonder whether up-regulation of gene expression would lead to translation into functional proteins. Several RFR studies support increased translation (e.g., [33–36]). It is an important complement to the gene-expression studies to look at translation levels of proteins as these are the functional units that promote/sustain both health and illness. Proteomic studies are an attractive next step in understanding the biological effects of RFR exposure. This was suggested more than a decade ago [37]. Changes in transcription and protein functions point to a need to investigate molecular signaling pathways after RFR exposure [38]. In addition, the data on gene-expression suggests two areas of mechanism research that are worth pursuing. The first area is the involvement of stress in RFR-induced biological effects. Involvements of ‘cellular stress responses’ and the hypothalamic-pituitary-adrenal axis and, particularly, effects on the limbic system should be further investigated [3]. The second area is the cellular oxidative processes. Particularly, the mechanism of induction of free radicals by RFR should be systematically studied. Present data indicates that low-frequency modulations in RFR may play a role [39]. It is, however, not known how much these low-frequency components contribute to the overall effect. Similar effects on gene expression have been reported after exposure to extremely-low frequency electromagnetic fields (see ‘static and ELF EMF genetic effects’ file in reference [1]).

Table 7: Studies that show no significant effect of RFR on expression of genes related to protein damage, DNA damage, oxidative processes, stress, apoptosis, and brain functions.

References (see Appendix)	Exposure conditions	Results	Comments
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.
Chauhan et al. (2006)	Human lymphoblastoma cells (TK6) exposed to pulsed-modulated, intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 6 h; SAR 1 or 6 W/kg.	No evidence of a general stress response with proto-oncogene and heat-shock protein gene transcriptions.	
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) 1,900-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. (Hsp 40, 27, and 105 were studied).	Hsp 40 (chaperone DnaI) associates with unfolded polypeptide chains and prevents their aggregation Hsp 27 is a chaperone of the sHsp (small heat shock protein) is involved in thermotolerance, inhibition of apoptosis, regulation of cell development, cell differentiation, and support of cell survival under stress conditions. The cytoprotective properties of Hsp 27 result from its ability to modulate reactive oxygen species and to raise glutathione levels. Hsp 105 regulates biogenesis and control of Misfolded proteins.
Dawe et al. (2008)	Transgenic Hsp16-1::lacZ strain of <i>Caenorhabditis elegans</i> exposed to 1,800 MHz RFR (continuous-wave or talk-pulsed) for 2.5 h at 1.8 W/kg.	No effect on Hsp16-1 heat-shock gene expression.	Hsp-16.1 mediate cyto-protection from hyperthermia.
Gurisik et al. (2006)	Human cell lines (one of neuronal (SK-N-SH) and the other of monocytoid (U937) origin) exposed to a 900 MHz (217 Hz pulses) RFR for 2 h at 0.2 W/kg.	No effect on gene expression including Hsp70.	
Hirose et al. (2007)	Human glioblastoma A172 cells 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.	No significant induction of phosphorylation of Hsp27 or expression of Hsp gene family.	The main function of Hsp27 is to provide thermotolerance <i>in vivo</i> , cyto-protection, and support of cell survival under stress conditions. Inhibiting protein aggregation and by stabilizing partially denatured proteins. It has anti-apoptotic properties and roles in cell growth (termination) and inflammatory and stress responses.
Huang et al. (2008a)	Jurkat human T lymphoma cells exposed for 24 h to 1,763 MHz RFR; SAR 10 W/kg.	Alterations in cell proliferation, cell cycle progression, DNA integrity or global gene expression were not detected.	
Huang et al. (2008b)	HELOC1 immortalized mouse auditory hair cells exposed to 1763 MHz (CDMA) RFR for 24 or 48 h; SAR 20 W/kg.	No significant effects on cell cycle distribution, DNA damage, stress response, and gene expression.	
Kuribayashi et al. (2005)	Brain of rats exposed to 1,439 MHz RFR, 90 min/day for 1–2 weeks, 2 or 6 W/kg.	No effect on expression of three blood-brain barrier-related genes (p-glycoprotein, claudin-5, and aquaporin-4). Increase in the mRNA expression of aquaporin-4 was observed in the young rats exposed at 6 W/kg for 1 week.	P-Glycoprotein is an ATP-dependent drug transport protein that is predominantly found in the membranes of a number of epithelial cell types. Absence of functional P-glycoprotein in the blood-brain barrier leads to highly increased permeability. At the blood-brain barrier, claudin-5 is the most enriched tight junction protein that limits diffusion of material between blood and brain. Aquaporin-4 in the blood-brain barrier is involved in its development and integrity.

Table 7: (continued)

References (see Appendix)	Exposure conditions	Results	Comments
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.	
Paparin 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.	
Porcher et al. (2024) (LI)	<i>Arabidopsis thaliana</i> (thale cress weed) grew on petri dish to approx. 1 cm; exposed to 30,000 EMF pulses (50 Hz, (237 kV m ⁻¹ , 280 ps rise-time, duration of 500 ps) carrier frequency < 2000 MHz; SAR 10 ⁻¹³ to 10 ⁻¹⁶ W/kg.	No effect on mRNA levels of calmodulin, Zinc-Finger protein ZAT12, NADPH oxidase/respiratory burst oxidase homolog (RBOH) isoforms D and F, Catalase (CAT2), glutamate-cysteine ligase (GSH1), glutathione synthetase (GSH2), Sucrose non-fermenting-related Kinase 1 (SnRK1) and Target of rapamycin (TOR).	
Sannino et al. (2024)	SH-SY5Y human neuroblastoma cells to 1950 MHz, UMTS signal; 20, 10, 3 or 1 h/day for 3 days, 0.3 or 1.5 W/kg; followed by menadione injection.	RFR had no effect on DNA single strand breaks, but attenuated breaks induced by menadione. RFR had no effect on thioredoxin-1, heat shock transcription factor 1, heat shock protein 70, and poly [ADP-ribose] polymerase 1 expression (molecules involved on cellular stress response). No effects on gene expressions of BAX, BCL-2, SOD2, CAT, collagen III, and TGF β-1.	
Savchenko et al. (2023)	Mice exposed to 915 MHz RFR 9 h/day for 14 days at 40 W/kg.	No significant change in either Hsp70 or Hsc70 protein or gene expression, or DNA single strand breaks.	Hsc70 plays a role in protein folding, signal transduction, apoptosis, protein homeostasis, and cell growth and differentiation. It is involved in chaperone-mediated autophagy by aiding the unfolding and translocation of substrate proteins across the membrane into the lysosomal lumen. Unlike Hsp70, Hsc70 is constitutively expressed and performs functions related to normal cellular processes, such as protein degradation.
Valbonesi et al. (2008)	Human trophoblast cell line HTR-8/SVneo exposed to pulsed 1,817 MHz RFR for 1 h; SAR 2 W/kg.		
Valbonesi et al. (2016)	Rat PC12 cells exposed to 1.8 GHz 217-GSM signal for 24 h. SAR 2 W/kg.	AChE transcriptional or translational pathways were not affected, whereas AChE enzymatic activity increased.	Fos is involved in cell proliferation, differentiation and survival, associated with hypoxia and angiogenesis.
Whitehead et al. (2005)	Murine embryonic fibroblasts exposed to 835.62 MHz FMCW, 847.74 MHz CDMA, or 836.55 MHz TDMA RFR for 24 h at 5 or 10 W/kg.	No effect on Fos proto-oncogene expression.	
Whitehead et al. (2006)	Murine embryonic fibroblasts exposed to 835.62 MHz FMCW, 847.74 MHz CDMA RFR for 24 h at 5 W/kg.	No statistically significant effect on gene expression (Gapd, Fos, Jun, Rasa3, Hsp).	GAPDH acts as a reversible metabolic switch to glycolysis under oxidative stress. Fos is involved in cell proliferation, differentiation and survival and associated with hypoxia and angiogenesis. Jun regulates gene expressions involved in cell growth, differentiation, and neoplastic transformation. Rasa3 is a member of the GAP1 family of GTPase-activating proteins involved in the control of cellular proliferation and differentiation.

(LI), Low-intensity study (SAR < 0.4 W/kg). (Number of studies: acute exposure/*in vivo*=3; repeated (chronic) exposure/*in vivo*=3; acute exposure/*in vitro*=1; repeated (chronic) exposure/*in vitro*=1).

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