



The impact of electromagnetic radiation on human health: a narrative review of in vitro and in vivo evidence

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Abstract

Purpose The pervasive use of electromagnetic radiation (EMR) emitting devices has raised public health concerns; however, a comprehensive synthesis of in vitro and in vivo evidence is lacking. This narrative review aims to critically evaluate the health impacts of EMR exposure, identify knowledge gaps, and provide a foundation for future research.

Methods A literature search of Science Direct, Google Scholar, and Web of Science (2000–2026) identified 17,240 records. After duplicate removal and title/abstract screening, 640 articles underwent full-text assessment. Following exclusion of 525 articles (lack of sham/control, solely thermal effects, inaccessibility, non-mammalian models), 115 studies were included. The relevant literature was reviewed and narratively synthesized.

Results EMR exposure consistently induces oxidative stress, DNA damage, and apoptosis, with the nervous and reproductive systems being most vulnerable. Neurobehavioral impairments (anxiety, memory deficits, hippocampal damage) and reproductive toxicity (reduced sperm quality, testicular injury, hormonal dysregulation) were frequently reported. Outcomes are highly dependent on exposure parameters frequency, power density, duration, and modulation. Carcinogenicity evidence remains mixed: some animal studies report increased tumor incidence, while others show no effects.

Conclusion EMR exposure, particularly at mobile communication frequencies, can induce biological effects primarily through oxidative stress, inflammation, and apoptosis. Future research should prioritize long-term prospective human studies and real-world multi-frequency exposure scenarios.

Keywords Carcinogenic effect · Electromagnetic field · Health risk · Nervous system Oxidative stress · Reproductive systems

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Introduction

Electromagnetic pollution is one of the most significant challenges of modern technology. While technological innovations offer many benefits that simplify daily life, they also introduce factors that can degrade our quality of life through adverse effects. The electromagnetic frequencies emitted by myriad devices used daily, such as computers, mobile phones, WiFi, Bluetooth, and radio/TV transmitters, have the potential to disrupt physiological processes [105]. Electromagnetic radiation (EMR) is broadly divided into two main categories based on frequency: static/low-frequency fields from sources like power lines and electrical appliances, and high-frequency (radiofrequency) fields produced by telecommunications equipment, radar, and security devices [106].

To date, numerous studies have demonstrated that exposure to EMR can disrupt fundamental biological processes. The impact of EMR on ecosystem constituents is influenced by the physical and electrical characteristics of individual organisms, as well as their interactions with the environment. Furthermore, variations in environmental parameters induced by EMR exposure can lead to biological disturbance in living creatures [44, 73, 131].

In vitro studies have been indispensable for elucidating the fundamental mechanisms of EMR interaction at the cellular level. These controlled experiments offer significant advantages, including precise variable control and accurate dosimetry [26]. Research using this approach has identified a wide range of sensitive endpoints, such as alterations in DNA and protein synthesis, cell cycle regulation, immune responses, and hormone-related signal transduction. For example, studies on human sperm samples have demonstrated a correlation between EMR exposure and alterations in key reproductive parameters [3]. Complementing these molecular insights, in vivo studies using animal models have consistently linked EMR exposure to systemic dysfunction and organ damage. These investigations have demonstrated disruptions in endocrine function, reproductive health, and fetal development [48]. For instance, exposure to 2.45 GHz radiation altered thyroid hormone levels and caused glandular damage in rats [108], and also induced oxidative stress in brain and kidney tissues [63]. Similarly, prolonged exposure damaged female reproductive tissues [110], while prenatal exposure caused oxidative stress and structural damage in the developing heart [127].

Despite the growing body of evidence, a comprehensive synthesis that integrates in vitro and in vivo findings, highlights inconsistencies, and identifies key knowledge gaps is currently lacking. This gap is particularly important given that increasing exposure to EMR has the potential to exacerbate adverse health effects. Therefore, to address this need

and inform future risk assessment, this review aims to: (1) critically assess the evidence from both in vitro and in vivo studies on the health impacts of EMR exposure; (2) identify the most critical knowledge gaps; and (3) provide a consolidated foundation for guiding future research and public awareness campaigns.

Methodology

Search methodology

A literature search was conducted in Science Direct, Google Scholar and Web of Science from January 2000 to July 2026. The literature search was conducted to provide a comprehensive overview of the topic, rather than a strict systematic analysis. The search strategy combined keywords related to exposure ('electromagnetic radiation' OR 'electromagnetic field' OR 'OR 'RF-EMF' OR 'radiofrequency') AND health outcomes ('oxidative stress' OR 'DNA damage' OR 'apoptosis' OR 'neurobehavioral' OR 'fertility' OR 'cancer') AND study type ('in vitro' OR 'in vivo' OR 'animal model'). The references of the identified manuscripts were also checked to identify any relevant studies that may have been missed.

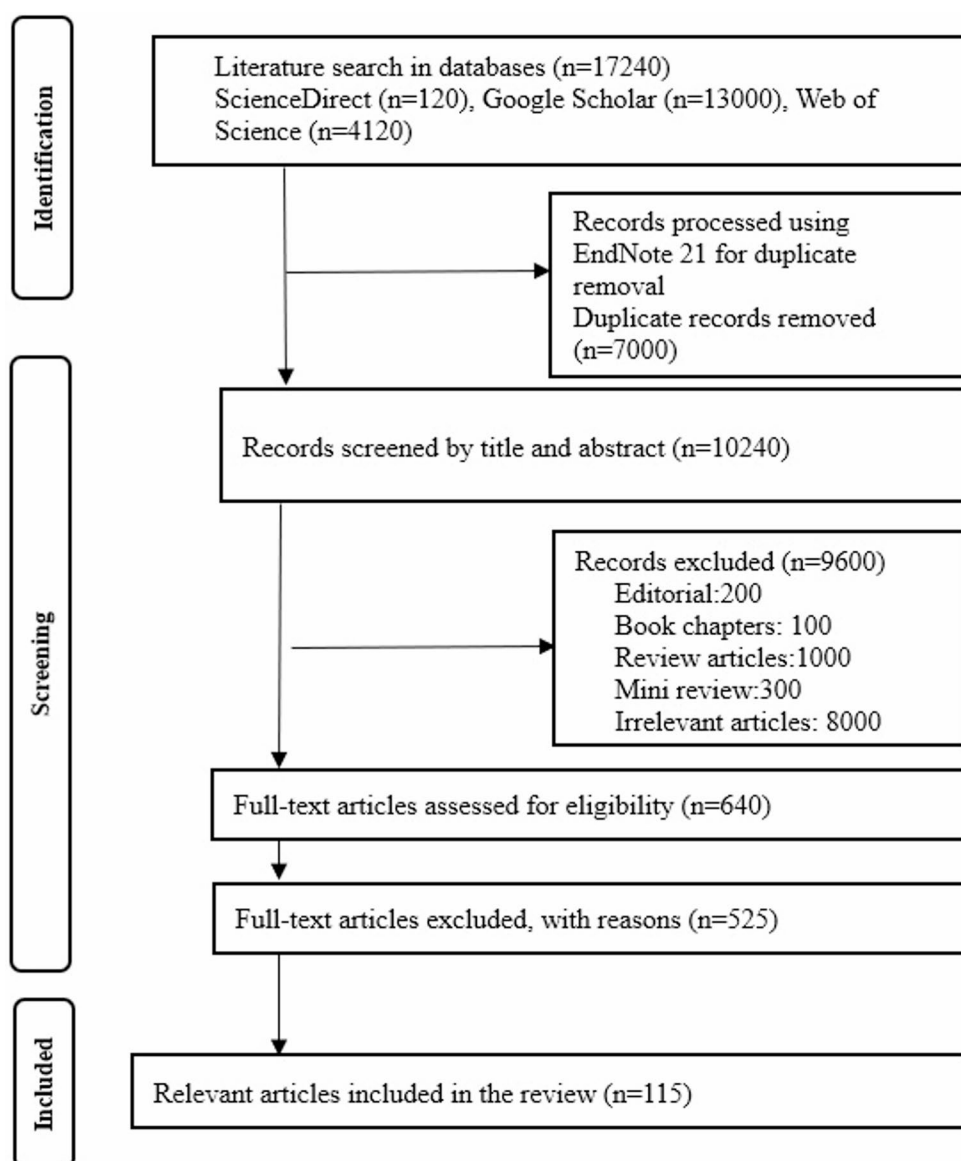
Inclusion and exclusion criteria

The following criteria were used to identify relevant studies. Inclusion criteria were: (1) the papers were published in English journals; (2) studies involving in vitro experimental investigations, studies involving in vivo experimental investigations and human studies examining health-related outcomes of electromagnetic radiation/electromagnetic fields (EMR/EMF) exposure. (3) studies reporting at least one biological endpoint related to EMR/EMF exposure. Studies were excluded if they were: (1) review articles, letters, short reports, book chapters, and conference abstracts; (2) studies examining solely thermal effects without measuring any molecular or cellular endpoint; (3) studies using non-mammalian models such as plants, bacteria, fungi, or invertebrates; and (4) articles for which the full text was not accessible.

Study selection process

The literature identification and study selection process are illustrated in Fig. 1. After the initial search, all retrieved records were imported into EndNote X21 for duplicate removal. The titles and abstracts of the remaining records were screened and any disagreements were resolved through discussion. Articles that passed this initial screening

Fig. 1 Flow diagram of the literature identification and study selection process



underwent a full-text assessment against the predefined selection criteria (Sect. 2.2). The reasons for exclusion at each stage were recorded and are presented in the literature selection flow diagram (Fig. 1). Ultimately, 115 relevant studies were included in this narrative review.

Results

Types of EMR

Non-ionizing radiation (NIR)

Non-ionizing radiation (NIR) encompasses EMR and fields with photon energies below 10 eV. This corresponds

to frequencies under 3 PHz (3×10^{15} Hz) and wavelengths greater than 100 nm. The NIR spectrum is subdivided into distinct bands: ultraviolet (UV) radiation (100–400 nm), visible light (400–780 nm), infrared radiation (780 nm–1 mm), radio-frequency fields (100 kHz–300 GHz), low-frequency fields (1 Hz–100 kHz), and static electric and magnetic fields (0 Hz) [153]. This form of radiation is characterized by energy levels insufficient to initiate ionization processes in matter, lacking the energy required to displace electrons from atomic orbitals. While it can excite atoms and electrons, it cannot eject them from their orbits [93, 138]. Technological advancements have led to a significant increase in population-wide exposure to electromagnetic fields of non-ionizing radiation (EMF-NIR), raising considerable public concern regarding their potential health and environmental impacts [101, 102, 112].

Ionizing radiation (X-rays/Gamma Rays)

Ionizing radiation consists of high-frequency and short-wavelength energy. It carries sufficient energy to break the molecular bonds and displace or remove electrons from the atoms. This results in the formation of charged particles (ions), which can cause severe damage to living cells in plants, animals and humans. Ionizing radiation is very useful and has a variety of applications in life. For example, it is used in smoke detectors, cancer treatment, and sterilization of medical equipment. Similarly, X-rays are typically used to provide static images of body parts (such as teeth and bones) and are also used in industry to find defects in welds. However, ionizing radiations can be potentially harmful if not used correctly, as these include alpha particles, beta particles, gamma rays, X-rays, and neutrons, which are emitted from naturally occurring radioactive materials (such as uranium, thorium, and radium) as well as man-made radioactive elements; therefore, it must be handled with maximum care [18, 32, 82].

Health effects of EMR

In vitro studies

Oxidative stress and DNA damage Biochemical processes essential for life are grounded in oxidation-reduction (redox) chemistry. Molecular oxygen plays a pivotal role in energy generation, both from sunlight and via mitochondrial respiration. Critical to cellular function is the maintenance of a balance between reductants and oxidants, a state referred to as redox equilibrium. This balance is actively preserved by cellular defense mechanisms and signaling pathways. A disruption favoring oxidative reactions leads to oxidative stress [116].

Research has shown that exposure to EMR may induce oxidative stress, cause DNA damage, and impair repair processes [111, 142]; these effects are summarized in Table 1. Based on the studies, exposure to EMR can induce inflammation across various tissues and organs. For instance, studies conducted on male rats have demonstrated that EMF exposure triggers inflammatory responses in hepatic and pancreatic tissues [80, 84]. A reciprocal relationship exists between oxidative stress and inflammation, with “ROS-induced inflammation” playing a pivotal role in the progression of various disease pathologies. Pathophysiological levels of reactive oxygen species (ROS) can initiate signaling cascades that lead to the onset and advancement of inflammatory diseases by modulating the structure of transcription factors and pro-inflammatory genes [23].

Moreover, inflammation acts as both a precipitating factor and a consequence of oxidative stress [80].

Research has demonstrated that microwave radiation can initiate stress responses at the cellular level. This process involves protein impairment and the subsequent activation of heat shock proteins [56]. These findings imply that microwave exposure may induce cellular stress mechanisms, with possible implications for cellular function and overall health. Chronic exposure to radiofrequency (RF) radiation emitted by devices such as mobile phones and communication towers may result in localized tissue heating. This thermal effect can interfere with normal cellular activities and has the potential to cause damage to cellular DNA [74]. This potential is corroborated by findings of DNA damage in specific human cell types, such as the epithelial cells of the lens and human hair follicles, following exposure to RF radiation [21, 144]. It is important to note, however, that the genotoxic impact of RF exposure appears to be highly dependent on specific exposure parameters, including flux densities, frequencies, and exposure patterns (e.g., continuous vs. pulsed) [77]. In addition to these effects, a study by Małgorzata Lewicka et al., demonstrated that exposure to 1 kHz EMR from LCD monitors significantly disrupted oxygen metabolism in human blood platelets. The researchers concluded that such EMR exposure can induce oxidative stress in blood platelets, potentially leading to physiological dysfunctions in the organism [69]. This effect was confirmed by a significant increase in malondialdehyde (MDA) and nitric oxide (NO) levels, along with decreased activity of antioxidant enzymes including Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px). Alterations in the xanthine oxidoreductase system and mineral imbalances (elevated iron and copper, reduced zinc and manganese) were also observed.

Nervous system and psychological disorders Table 2 presents studies on the effects of EMR on the nervous system and physiological disorders. Studies have demonstrated that EMR exerts significant neurological effects on the central nervous system, influencing cognitive and behavioral functions through both thermal and non-thermal mechanisms. Research indicates that exposure to these fields can alter blood-brain barrier permeability, neurotransmitter levels, calcium channel regulation, and myelin protein structure, while also inducing oxidative stress and changes in metabolic processes. In addition, research has indicated that exposure to EMR can elevate ROS formation, impair cognitive function, and reduce antioxidant levels through second messengers that enhance plasma membrane NADH oxidase activity and active protein kinase C. Prolonged EMR exposure may also induce DNA damage, potentially accelerating neuronal cell death [92]. Although some evidence suggests

Table 1 Effects of EMR exposure on Oxidative stress and DNA damage: In vitro

References	Intervention description	Model	Main effect
Luukkonen et al., [74]	872 MHz RF radiation (CW & GSM signals) at 5 W/kg SAR for 1 h	Human SH-SY5Y neuroblastoma cells (cell line)	CW RF (not GSM) enhanced menadione-induced ROS and DNA damage.
Çam and Seyhan [21],	900 MHz GSM mobile phone RFR for 15–30 min	Human subjects (hair root cells).	Significant increase in single-strand DNA breaks; damage was greater after 30 min vs. 15 min.
Yao et al., [144]	1.8 GHz GSM RF at 1–4 W/kg SAR for 2 h	Cultured human lens epithelial cells	RF (2–4 W/kg) increased ROS and DNA SSBs; co-exposure with electromagnetic noise blocked these effects.
Lewicka et al., [69]	1 kHz EMR at 150–220 V/m intensity for 30–60 min.	Human blood platelets	Increased ROS and MDA, decreased SOD-1 and Catalase activity; effects were dose (intensity/duration) dependent.
Yin et al., [145]	ELF-EMF exposure to primary cultured neurons	Primary cultured rat hippocampal neurons	ELF-EMF induced neurotoxicity (apoptosis, oxidative stress, DNA damage); LSPCs provided protection by inhibiting oxidative stress and the mitochondrial apoptotic pathway.
Koohestanidehaghi et al., [66]	Exposure of zygotes to 900–1800 MHz mobile phone RF-EMR for 30 min immediately post-fertilization.	Mouse preimplantation embryos (zygotes) generated via in vitro fertilization IVF.	RF-EMR exposure caused oxidative stress (increased ROS), leading to developmental delays, reduced embryo viability, and disrupted expression of oxidative stress-related genes in mouse embryos.
Tsoy et al., [129]	Exposure to 918 MHz RF-EMF (mobile phone frequency) in combination with A β 42 or H ₂ O ₂ treatment.	Primary astrocytes (human and rat cell cultures).	RF-EMF exerted protective effects against A β 42- and H ₂ O ₂ -induced oxidative stress by reducing ROS formation, restoring mitochondrial membrane potential, inhibiting NADPH-oxidase activation, and suppressing H ₂ O ₂ -induced p38MAPK/ERK1/2 phosphorylation.
Marjanovic Cermak et al., [79]	Short-term RF exposure (1800 MHz, SAR 1.6 W/kg) for 10, 30, or 60 min in a GTEM cell.	SH-SY5Y human neuroblastoma cell line.	RF exposure induced time-dependent oxidative stress: increased ROS production, elevated lipid (MDA) and protein (carbonyl) oxidation after 60 min, and transient rise in Glutathione antioxidant levels after 10 min, without affecting cell viability.

RF: Radiofrequency; **CW:** Continuous Wave; **GSM:** Global System for Mobile Communications; **SAR:** Specific Absorption Rate; **ROS:** Reactive Oxygen Species; **SSBs:** Single-Strand Breaks; **EMR:** Electromagnetic Radiation; **MDA:** Malondialdehyde; **SOD-1:** Superoxide Dismutase 1; **ELF-EMF:** Extremely Low-Frequency Electromagnetic Field; **LSPCs:** Limbus-derived Stromal/Progenitor Cells; **A β 42:** Amyloid Beta 42; **NADPH-oxidase:** Nicotinamide Adenine Dinucleotide Phosphate Oxidase; **p38MAPK:** p38 Mitogen-Activated Protein Kinase; **ERK1/2:** Extracellular Signal-Regulated Kinase 1/2; **GTEM cell:** Gigahertz Transverse Electromagnetic Cell

potential adverse outcomes such as anxiety and cellular damage, other studies indicate that specific frequencies, particularly extremely low frequencies (ELF), may enhance memory retention, learning abilities, and offer potential therapeutic benefits for neurodegenerative diseases [38].

Studies have shown that the effects of EMFs are primarily mediated by voltage-gated calcium channels (VGCCs). This is supported by evidence that L-type or other VGCC blockers can significantly inhibit or completely block various EMF-induced effects. Moreover, the voltage-gated properties of these channels provide biophysically plausible mechanisms for the biological effects of EMF exposure [96]. In contrast to the adverse effects mediated by VGCCs, some studies have reported potential beneficial effects of EMF exposure under certain conditions. Supporting these potential therapeutic effects, an in vitro study found that intermittent exposure of mouse embryonic neural stem cells (eNSCs) to 50 Hz, 1 mT ELF-EMF for 4 h/day over 1–3

days enhanced neuronal differentiation and neurite outgrowth without affecting astrocyte differentiation or apoptosis. This effect was mediated by upregulation of TRPC1 and the proneural genes *NeuroD* and *Neurogenin 1*, leading to increased intracellular calcium. Silencing TRPC1 abolished these effects, indicating a key role for the TRPC1/*NeuroD*/*Neurogenin 1* pathway in ELF-EMF-induced embryonic neurodevelopment [75]. Another study evaluated four 1 mT pulsed electromagnetic fields (PEMF) exposure protocols (50 Hz/150 Hz for 1 h/12 h daily) in rat Schwann cells (iSCs). The 50 Hz, 1 h/day regimen was the most effective, significantly enhancing iSC proliferation and upregulating mRNA expression of the regeneration markers *S100* and brain-derived neurotrophic factor (BDNF), whereas other protocols showed no significant effects [51].

Reproductive system effects EMR is recognized as a potential environmental factor influencing human reproduction by affecting gametes, gonadal function, and embry-

Table 2 Effects of EMR exposure on Nervous system and psychological disorders: In vitro

References	Intervention description	Model	Main effect
Ma et al., [75]	Intermittent ELF-EMF (50 Hz, 1 mT) exposure, 4 h/day for 1–3 days, on cultured mouse eNSCs.	Mouse embryonic neural stem cells (eNSCs).	Promotes neuronal differentiation and neurite outgrowth via the TRPC1/NeuroD/Ngn1 pathway.
Hei et al., [51]	1 mT PEMF (50 Hz/150Hz, 1 h/12 h per day)	Immortalized rat Schwann cells (iSCs)	50 Hz, 1 h/day protocol most effectively increased iSC proliferation & expression of key regeneration genes (S100, BDNF)
Bertuccio et al., [13]	Exposure to 2.45 GHz EMR (Wi-Fi frequency) for 2, 24, or 48 h via antenna	Human cell	Significant cytotoxicity, ROS overproduction, mitochondrial depolarization, and pro-apoptotic gene expression ($\uparrow$ BAX/BCL2, $\uparrow$ LC3). Neuron-like cells more vulnerable than PBMCs, with earlier antioxidant response (SOD1, mtTFA alterations). Demonstrates oxidative stress-mediated neurotoxicity of 2.45 GHz radiation.
Zhi et al., [151]	Microwave radiation (2.856 GHz; mean power density: 30 mW/cm ² ; 300 Hz; 0.015% duty cycle; 3 $\times$ 6 min/day for 4 days) on primary hippocampal neurons, NSCs, and non-contact co-culture (Transwell)	Primary hippocampal neurons & NSCs (rat) / non-contact co-culture	Synaptic impairment: Reduced dendritic complexity and spine density in neurons. NSC dysfunction: Suppressed proliferation and differentiation. Molecular mechanism: Downregulated HNRNPA2B1, reduced TrkB m6A binding, increased TrkB degradation, decreased BDNF expression.
Lin et al., [71]	EMP radiation (600 kV/m; 1000 pulses/day for 2 weeks)	Microglial cell line	M1 polarization: Induced pro-inflammatory microglial phenotype. Inflammatory mechanism: Activated NLRP3 inflammasome/ NF- κ B signaling pathway, confirming direct neuroinflammatory effects at the cellular level.

ELF-EMF: Extremely Low-Frequency Electromagnetic Field; **eNSCs:** Embryonic Neural Stem Cells; **TRPC1:** Transient Receptor Potential Canonical 1; **NeuroD:** Neurogenic Differentiation; **Ngn1:** Neurogenin 1; **PEMF:** Pulsed Electromagnetic Field; **iSCs:** Immortalized Schwann Cells; **S100:** S100 protein; **BDNF:** Brain-Derived Neurotrophic Factor; **EMR:** Electromagnetic Radiation; **ROS:** Reactive Oxygen Species; **BAX:** Bcl-2-Associated X Protein; **BCL2:** B-Cell Lymphoma 2; **LC3:** Microtubule-Associated Protein 1 A/1B-Light Chain 3; **PBMCs:** Peripheral Blood Mononuclear Cells; **SOD1:** Superoxide Dismutase 1; **mtTFA:** Mitochondrial Transcription Factor A; **NSCs:** Neural Stem Cells; **HNRNPA2B1:** Heterogeneous Nuclear Ribonucleoprotein A2/B1; **TrkB:** Tropomyosin Receptor Kinase B; **m6A:** N6-methyladenosine; **EMP:** Electromagnetic Pulse; **M1:** Type 1 Macrophage (Pro-inflammatory Phenotype); **NLRP3:** NOD-like Receptor Pyrin Domain-Containing 3; **NF- κ B:** Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells

onic development [62, 135]. In vitro studies in Table 3 have been pivotal in elucidating the direct cellular and molecular mechanisms underlying these interactions. Research on male gametes has yielded diverse findings, highlighting the critical importance of exposure parameters. For instance, Chu et al., [25] reported that sperm motility and viability were significantly reduced upon exposure to RF-EMR from WiFi-transmitting smartphones, whereas exposure to 4G or 5G signals under identical conditions showed no adverse effect, outcomes further varying across device models. This parameter specificity is a key consideration, as demonstrated by Butković et al., [19], who found that exposure to specific 5G frequencies (2500 and 3500 MHz) directly induced DNA fragmentation and membrane damage in boar spermatozoa, with the 3500 MHz band being the most detrimental. These contrasting results underscore that the biological impact of RF-EMR is profoundly dependent on the precise frequency, modulation, and intensity of exposure.

Furthermore in vitro and animal model studies reveal significant impacts on ovarian and endometrial function in female reproductive physiology. In rats, combined prenatal and postnatal exposure to a 50-Hz, 3-mT EMF led to impaired ovarian development, characterized by hormonal

imbalance (elevated FSH, reduced estradiol), upregulated iNOS expression, and histopathological tissue damage, suggesting a pathway to compromised fertility [17]. Similarly, Ahmadi et al., [4] observed that comparable EMF exposure significantly increased follicular atresia and caused adverse oocyte morphological changes, indicating a reduction in ovarian reserve [4]. Beyond ovarian toxicity, EMF exposure may also impair uterine receptivity. A comprehensive in vitro study on porcine endometrial tissue demonstrated that even brief (2-hour) exposure to 50 Hz ELF-EMF can disrupt key epigenetic regulatory pathways, including alteration in the expression of factors involved in DNA methylation, histone modification, and microRNA biogenesis [139]. Such epigenetic alterations may subsequently disrupt gene expression patterns that are essential for successful embryo implantation and uterine homeostasis.

Carcinogenic effects Cancer is a complex disease arising from genetic damage within cells. In 2011, the World Health Organization's International Agency for Research on Cancer (IARC) classified radiofrequency radiation as a Group 2B "Possible Human Carcinogen." This classification aligns with the agency's 2001 designation of ELF-EMF in the same category [103]. The complex and often dual nature of EMF interactions at the cellular level is presented in Table 4.

Table 3 Effects of EMR exposure on Reproductive System: In vitro

References	Intervention description	Model	Main effect
Koziorowska et al., [67]	Exposure of porcine uterine tissue slices to 50–120 Hz ELF-EMF at 8 mT for 2–4 h, with/without progesterone.	Porcine endometrial and myometrial tissue slices	ELF-EMF altered estradiol-17 β synthesis/secretion in a frequency-, duration-, tissue-, and progesterone-dependent manner, without changing aromatase protein levels.
Keskin et al., [62]	900 MHz RF-EMF for 30 min–1 h	Human sperm samples from healthy normozoospermic men.	Reduced sperm motility (30 min) and induced ultrastructural defects (both durations). Also increased p-AKT signaling (1 h) as a potential molecular response.
Erogul et al., [37]	Exposure of semen samples to 900 MHz (EMR from an activated cellular phone, compared to unexposed aliquots from the same donors.	Human sperm samples	EMR exposure significantly impaired sperm motility, reducing rapid and slow progressive movement and increasing the proportion of non-motile sperm, while sperm concentration remained unchanged.
Parsanezhad et al., [97]	Exposure of semen aliquots to radiofrequency radiation emitted from a commercial mobile phone jammer, with or without prior swim-up processing.	Human sperm samples	Jammer radiation significantly reduced sperm motility and increased DNA fragmentation in human semen, indicating potential adverse effects on sperm quality and reproductive health.
Agarwal et al., [3]	Exposure of semen aliquots to cellular phone RF-EMW in talk mode for 1 h under controlled laboratory conditions.	Human semen samples (unprocessed/ neat ejaculate).	RF-EMW exposure significantly reduced sperm motility and viability, increased ROS, and lowered the ROS-TAC score (indicating oxidative stress), while TAC and sperm DNA damage remained unchanged.
Wdowiak et al., [137]	Exposure of semen aliquots to 43 kHz low-frequency electromagnetic waves in an automated incubator system.	Human sperm samples from fertile, healthy men.	43 kHz exposure significantly increased sperm progressive motility, velocity, and the proportion of hyperactivated spermatozoa, suggesting a stimulatory effect on sperm maturation and motility parameters that could potentially lead to premature capacitation in vivo.
Iuliis et al., [29]	Exposure of sperm samples to 1.8 GHz RF-EMR at varying specific absorption rates (SAR: 0.4–27.5 W/kg) in a controlled laboratory system.	Purified human spermatozoa	RF-EMR exposure dose-dependently reduced sperm motility and vitality, increased mitochondrial ROS generation, and induced oxidative DNA damage (8-OH-dG) and DNA fragmentation, indicating a direct, SAR-dependent oxidative stress-mediated injury to sperm.
Houston et al., [53]	Exposure to 1.8 GHz continuous-wave RF-EMR at two specific absorption rates (SAR: 0.15 and 1.5 W/kg) for 3–4 h in a waveguide system.	Cultured mouse spermatogonial (GC1) and spermatocyte (GC2) cell lines, and cauda epididymal spermatozoa (mouse).	RF-EMR exposure increased mitochondrial ROS generation (via Complex III of the electron transport chain) in GC1 and GC2 cells, induced DNA fragmentation and oxidative DNA damage (8-OH-dG) in spermatozoa, and reduced sperm motility, without triggering a pronounced overall oxidative stress response in the somatic cell lines.
Žaja et al., [148]	In vitro exposure of boar semen samples to RF-EMR at 2500 MHz (simulating 5G-like radiation) under controlled laboratory conditions.	Boar (pig) semen spermatozoa	RF-EMR exposure significantly reduced progressive sperm motility and decreased the proportion of spermatozoa with high fertilizing potential, indicating adverse effects on semen quality relevant to artificial insemination.
Burcu et al., [17]	50 Hz, 3 mT EMF (sinusoidal)	Pregnant rats and their female offspring (Sprague-Dawley)	Impaired ovarian structure and function in female offspring (hormonal imbalance, increased iNOS, tissue damage)
Wydorski et al., [139]	Exposure to 50 Hz ELF-EMF for 2 h.	Porcine endometrial tissue slices (days 15–16 of pregnancy).	Altered epigenetic regulation (DNA methylation, histone modification, miRNA biogenesis) in the endometrium.
Butković et al., [19]	5G RF-EMR at frequencies of 700 MHz, 2500 MHz, and 3500 MHz, with an electromagnetic field strength of 10 V/m for 2 h.	Breeding boars (Pietren and German Landrace breeds).	Significant increase in sperm DNA fragmentation (frequency-dependent, most harmful at 2500 MHz & 3500 MHz), with additional damage to cell membrane integrity and mitochondrial potential at 3500 MHz.
Chu et al., [25]	Wireless-spectrum RF-EMR (4G, 5G, WiFi) from a smartphone in talk mode during a WhatsApp voice call.	Human (semen samples from normozoospermic men)	Decreased sperm motility and viability (WiFi only; no significant effect from 4G or 5G)
Omid et al., [94]	Exposure to 900 MHz mobile phone RF-EMF (SAR 2.0 W/kg) for 1 h.	Human spermatozoa	RF-EMF exposure significantly altered sperm morphometry (reduced head area and acrosome coverage) and decreased sperm–zona binding capacity, suggesting impaired fertilizing potential, but did not affect the acrosome reaction.

ELF-EMF: Extremely Low-Frequency Electromagnetic Field; **RF-EMF:** Radiofrequency Electromagnetic Field; **EMR:** Electromagnetic Radiation; **RF EMW:** Radiofrequency Electromagnetic Waves; **SAR:** Specific Absorption Rate; **ROS:** Reactive Oxygen Species; **TAC:** Total Antioxidant Capacity; **8-OHdG:** 8-Hydroxy-2'-deoxyguanosine; **iNOS:** Inducible Nitric Oxide Synthase; **miRNA:** Micro Ribonucleic Acid; **GC1:** Mouse spermatogonial cell line; **GC2:** Mouse spermatocyte cell line; **p-AKT:** Phosphorylated Protein Kinase B

Table 4 Interaction of EMR with Cancer Cells) From Cytotoxicity to Mechanistic Insights(: In vitro

References	Intervention description	Model	Main effect
Wang et al., [133]	Pulsed ELF-EMF at frequencies (7.83, 23.49, 39.15 Hz), flux density (0.5–1 mT), and variable durations/modes (12–48 h, continuous vs. switching)	Cell lines	Selective cytotoxicity in MDA-MB-231 breast cancer cells (viability reduced to 40.1%) via Ca ²⁺ influx and apoptosis, with no damage to normal breast epithelial cells.
Filipovic et al., [42]	50 Hz ELF-EMF exposure for 24–72 h (following 24 h preincubation).	Cancer Cell lines	Induced cellular disintegration and significantly increased early apoptosis in all three cancer cell lines after exposure. Computer modeling supported the experimental data. Suggests a potential anti-cancer approach.
Kim et al., [64]	Exposure to 835-MHz RF-EMF, alone and combined with clastogens (EMS, cyclophosphamide, 4-nitroquinoline 1-oxide).	Mammalian cell cultures.	No direct cytogenetic/DNA damage effect from RF-EMF alone. Potentiation (synergistic) effect observed when RF-EMF was combined with clastogens, especially in the comet assay. Suggests RF-EMF may enhance genotoxicity of other agents.
Marinelli et al., [78]	Exposure to unmodulated 900 MHz EMF (continuous wave) for 2–48 h, generated by a TEM cell.	Human acute T-lymphoblastoid leukemia cells (CCRF-CEM)	Time-dependent dual effect: Short exposure (2–12 h) induced DNA damage and early apoptosis. Long exposure (24–48 h) switched signaling to pro-survival pathways (Bcl-2, Ras, Akt1), potentially promoting survival and proliferation of the remaining cells.
Kaszuba-Zwońska et al., [60]	Triple stimulation with low-frequency PEMF (7 Hz, 30 mT, 4 h per session, 24 h intervals).	Cultures of proliferating PBMCs from children with Acute Lymphoblastic Leukemia (ALL) (N = 20).	Selectively induced apoptosis in leukemia patient cells (increased Annexin V+/PI+ cells), involving intrinsic (Bcl-2 family) and AIF pathways. No harm to healthy donor cells. Suggests a potential therapeutic application for ALL.

ELF-EMF: Extremely Low-Frequency Electromagnetic Field; **RF-EMF:** Radiofrequency Electromagnetic Field; **EMF:** Electromagnetic Field; **PEMF:** Pulsed Electromagnetic Field; **TEM cell:** Transverse Electromagnetic Cell; **Bcl-2:** B-Cell Lymphoma 2; **Akt1:** Protein Kinase B isoform 1; **Ras:** Rat sarcoma virus (family of oncogenes); **PBMCs:** Peripheral Blood Mononuclear Cells; **ALL:** Acute Lymphoblastic Leukemia; **Annexin V+/PI+:** Annexin V-positive / Propidium Iodide-positive; **AIF:** Apoptosis-Inducing Factor; **EMS:** Ethyl Methanesulfonate

Studies have linked RF radiation to an increased risk of cancer, particularly brain tumors [125]. The complexity of assessing cancer risk is further highlighted by in vitro studies. For instance, research on 835 MHz RF-EMF demonstrated that, while exposure alone did not cause significant direct genetic damage, it potentiated the DNA-damaging effects of known chemical clastogens. This finding suggests that RF-EMF may act as a co-carcinogen by lowering the biological threshold for damage from other environmental agents, underscoring the importance of evaluating combined exposures in risk assessment [64]. Furthermore, the potential carcinogenic risk may extend beyond the effects of co-exposure to include direct interference with cellular fate. In vitro research on leukemia cells revealed a time-dependent dual response to 900 MHz EMF: short-term exposure induced DNA damage and apoptosis, but prolonged exposure suppressed these death signals and activated pro-survival pathways (e.g., Bcl-2, Ras, Akt1). These studies suggest that chronic EMF exposure could act as a tumor-promoting agent by selecting for and enhancing the survival of genetically compromised cells [78].

Conversely, emerging research suggests that specific parameters of EMF exposure may offer therapeutic benefits. For instance, one study revealed that an optimized pattern of ELF-EMF with specifications of 7.83 Hz, 1 mT, and 6-hour intermittent exposure selectively inhibits growth and induces apoptosis in breast cancer cells (MDA-MB-231), while causing no detectable damage to normal breast cells (M10). The proposed mechanism underlying this effect involves an increased influx of calcium into cancer cells. This finding demonstrates the therapeutic potential of specific ELF-EMF patterns as a selective anti-cancer approach [133]. Despite these potential therapeutic applications, concerns regarding the carcinogenic risks of environmental EMF exposure remain, particularly in vulnerable populations. This mechanistic ambiguity at the cellular level is mirrored in population-based epidemiological studies. Various studies have examined the association between non-ionizing EMR and the risk of childhood leukemia. Overall, the existing evidence is inconsistent: some studies report an increased risk, particularly with exposure to magnetic fields exceeding 0.4 µT, whereas others find no significant

association. This heterogeneity likely reflects methodological challenges, including difficulties in accurate exposure assessment [99]. Collectively, these findings underscore the complexity of EMR interactions with biological systems, spanning both carcinogenic risks and therapeutic potential.

In vivo studies

Oxidative stress and DNA damage In vivo studies presented in Table 5 examine the induction of oxidative stress after exposure to EMR. The study by Bozok et al., [16] on prenatal exposure to EMF radiation in rats demonstrated that long-term exposure, particularly at higher frequencies (2100 MHz) and longer durations (24 h), caused significant myocardial damage. This damage was evidenced by increased malondialdehyde (MDA) levels and decreased glutathione levels, indicating oxidative stress in the cardiac tissue. Furthermore, an in vivo experiment showed that exposure to a 900 MHz EMF increased oxidative stress, resulting in histopathological impairment of the endometrium in rats [46]. Another study reported that exposure to a 950 MHz EMF induced significant oxidative stress and altered key enzyme systems and mineral levels in the liver and brain tissues of male rats. The co-administration of garlic extracts effectively mitigated these adverse biochemical changes, suggesting a potential protective role against EMR-induced damage [114]. In a separate study, Kilic et al., [63] investigated male and female rats exposed to a 900 MHz EMF (similar to that of mobile phones) and found that prenatal and/or postnatal exposure significantly increased inflammatory markers (such as IL-6 and TNF- α) and oxidative stress, while reducing antioxidant capacity in brain and kidney tissues. Furthermore, this exposure led to a marked activation of the renin-angiotensin system components in these tissues. Collectively, these findings indicate that EMF exposure at this frequency can disrupt key regulatory systems in vital organs by inducing inflammation and oxidative stress.

Nervous system and psychological disorders Studies have demonstrated that exposure to EMR can impair neuroendocrine activity, potentially triggering stress, depression, learning disorders, sleep disturbances, and anxiety [15, 35, 81]. These potential effects have been extensively investigated in vivo, as summarized in Table 6. The table compiles evidence of outcomes ranging from anxiety-like behavior and memory impairment to alterations in neuroendocrine and cellular integrity. For instance, an in vivo study investigated the effects of 4G mobile phone radiation (2400 MHz) on mice exposed for 40 and 60 min daily over 60 days. The results revealed that RF-EMR exposure significantly increased anxiety-like behaviors and induced structural

damage to the hippocampus, including neuronal degeneration and a marked reduction in pyramidal and granule cell populations. These morphological alterations were closely associated with the observed behavioral changes, highlighting the potential neurotoxic effects of prolonged 4G radiation exposure [50]. In contrast, Daniels et al., [27] reported that although EMR exposure did not cause spatial memory impairment or obvious morphological changes in rat brains, it led to subtle behavioral alterations, including decreased locomotor activity and increased grooming behavior, along with a tendency toward elevated basal corticosterone levels. These findings suggest that EMR may disrupt normal brain function even in the absence of pronounced structural damage. Furthermore, several studies have demonstrated a significant rise in amyloid-beta production following EMF exposure, identifying a potential mechanistic link to Alzheimer's disease pathogenesis [118, 119].

In addition to neurodegenerative effects, EMR exposure may influence sleep patterns. Deng et al., [31] investigated how specific EMR characteristics affect wakefulness in mice and found that exposure to 2.4 GHz EMR significantly increased wakefulness. Notably, this effect depended critically on the frequency and mode of the signal modulation: pulsed modulation at 100 Hz and 1000 Hz was effective, whereas 10 Hz was not. The carrier frequency was also crucial, as exposure to 935 MHz EMR did not alter wakefulness regardless of its modulation mode. Notably, while acute exposure to 2100 MHz RFR was found to induce auditory brainstem dysfunction, oxidative stress, and ultrastructural damage in the rat brain, chronic exposure with intermittent rest days did not produce harmful effects [36]. This observation highlights the importance of recovery periods in mitigating EMR-induced neural damage and suggests that continuous exposure without adequate rest intervals may pose a greater risk than interrupted exposure.

Reproductive system effects Table 7 presents evidence on the effects of electromagnetic radiation on the reproductive system. Evidence indicates that prolonged exposure to EMF from common electronic devices adversely affects key parameters of sperm quality [2]. An in vivo study exposed rats to 1800 MHz RF-EMR from telecommunication masts for five weeks. The results demonstrated notable adverse effects, including hematological alterations, reduced sperm quality, and substantial histological damage to vital organs such as the liver, heart, kidneys, and testes. The study concluded that RF-EMR exposure induces potentially fertility-impairing morphological changes in living systems [1]. Furthermore, Sundaram et al., [124] investigated the effects of whole-body exposure to 150 kHz intermediate-frequency EMR (IF-EMR) on male fertility parameters in rats. The

Table 5 Effects of EMR exposure on Oxidative Stress and DNA Damage: In vivo

References	Intervention description	Animal species	Main effect
Bozok et al., [16]	Exposed to EMF radiation (900, 1800, and 2100 MHz) for 6, 12, or 24 h per day over 20 days.	Pregnant rats	Prenatal exposure to electromagnetic fields induces oxidative stress in the myocardial tissue of newborn rats, characterized by increased malondialdehyde and decreased glutathione levels.
Guney et al., [46]	Exposed to 900 MHz EMR for 30 min per day over 30 days	Rats	900 MHz EMR exposure caused significant oxidative damage and apoptosis in rat endometrial tissue.
Shedid et al., [114]	Exposed to a 950 MHz EMF for one hour, three times per week for seven weeks, with one group pretreated with garlic extract (500 mg/kg) before each exposure.	Male rats	Exposure to 950 MHz EMF induced oxidative stress and disrupted trace element balance in the liver and brain
Kilic et al., [63]	Exposed to 900 MHz EMF for 1 h/day during prenatal (23 days of gestation), postnatal (40 days after birth), or both periods.	Male and female rat offspring	Prenatal and/or postnatal exposure to 900 MHz EMF significantly increased oxidative stress, inflammation, and expression of RAS components in the brain and kidney tissues of both male and female rat offspring.
Mojez et al., [88]	4 weeks of aerobic exercise and/or 3 h/day of mobile phone radiation exposure.	Male Wistar rats in 4 groups	Radiation caused hippocampal cell death and oxidative stress. Exercise neutralized these effects and boosted antioxidants.
Kamali et al., [58]	Continuous exposure to 2.45 GHz RF radiation from a commercial Wi-Fi device (24 h/day for 10 weeks).	Male Wistar rats.	Impaired plasma antioxidant defense: Decreased total antioxidant capacity and key enzyme activity (CAT, GSH-Px, SOD). Increased GST activity.
Bilgici et al., [14]	3-week exposure to 900 MHz EMF (1 h/day, SAR 1.08 W/kg) with or without garlic powder supplementation (500 mg/kg/day orally).	Rats.	EMF exposure induced brain oxidative stress ($\uparrow$ MDA, $\uparrow$ AOPP) and increased serum NO. Garlic supplementation prevented brain oxidative damage but did not affect serum NO elevation.
Berköz et al., [12]	Chronic exposure to 1800 MHz RF-EMR for 2 h/day over 8 weeks.	Rats (9 per group: Control, Sham, RF-EMR exposed).	RF-EMR exposure induced significant oxidative and nitrosative damage in rat liver, kidney, and brain tissues, marked by increased MDA and nitric oxide (NO) levels alongside decreased reduced glutathione levels and suppressed SOD and catalase activities.
Meral et al., [85]	30-day exposure to 900-MHz cell phone EMF (12 h/day, SAR 0.95 W/kg).	Guinea pigs (male, 14 animals total).	Exposure to 900-MHz electromagnetic fields induced tissue-specific oxidative stress: in the brain, it increased MDA and decreased glutathione and catalase activity, while leaving vitamin A, E, and D3 levels unchanged; in contrast, in the blood, it elevated MDA and CAT activity along with vitamins A, E, and D3, but reduced Glutathione levels.
Salameh et al., [104]	Continuous 24 h/day prenatal and postnatal exposure to 900 MHz RF-EMR (GSM-modulated).	Sprague-Dawley rats (pregnant females and their offspring).	RF-EMR induced significant age-dependent oxidative stress in the liver of offspring, characterized by elevated MDA, decreased CAT activity/expression, altered SOD and GPx activity/expression, and disrupted redox-related gene expression (Akt1, Nrf-2, ICAM-1).
Dasdag et al., [28]	Long-term exposure to 900 MHz microwave radiation (2 h/day, 7 days/week for 10 months) using a carousel system.	Wistar Albino adult male rats.	Exposure led to reduced apoptosis (lower cleaved caspase-3) and increased antioxidant capacity (higher TAC and catalase) in rat brain, with no significant change in p53 expression, TOS, or oxidative stress index.
Singh et al., [117]	Chronic exposure to mobile phone RF-EMF radiation (1966.1 MHz, 0.36 W/kg SAR) for 2 h/day over 16 weeks.	Young adult male Wistar rats.	MP-RF-EMF exposure induced significant hippocampal oxidative stress, elevated circulatory pro-inflammatory cytokines (IL-1 β , IL-6, TNF- α), and deregulated the HPA axis (increased adrenal weight, ACTH, and corticosterone), but did not significantly alter contextual fear memory.
Zosangzuali et al., [154]	Exposure to 1800 MHz RF-EMR from a mobile phone base station (MPBS) for 6, 12, or 24 h/day over 45 days.	Swiss albino mice.	Prolonged exposure ($\geq$ 12 h/day) induced brain-specific oxidative stress: reduced GSH, GST, and SOD activity; increased lipid peroxidation (MDA). No significant oxidative changes were observed in heart, kidney, or liver. Hematological alterations ($\downarrow$ RBC, $\uparrow$ WBC) were also noted at higher exposure durations.
Akbari et al., [5]	Six weeks of either (1) cell phone EMR exposure (0.9–1.8 GHz, 3 h/day), (2) moderate aerobic treadmill exercise (5 days/week), or (3) both combined.	Male Wistar rats.	EMR exposure induced cardiac oxidative stress ($\downarrow$ SOD, CAT, GPx; $\uparrow$ MDA) and tissue damage (apoptosis, collagen deposition). Aerobic exercise counteracted these effects, restoring antioxidant enzyme activity, reducing oxidative damage, and improving heart tissue integrity.

Table 5 (continued)

References	Intervention description	Animal species	Main effect
Usman et al., [130]	6-week exposure to mobile phone EMF + vibration/sound.	Male albino Wistar rats.	Combined exposure to mobile phone EMF, vibration, and sound induced systemic oxidative stress, characterized by decreased antioxidant enzymes (GPx, SOD) and increased lipid peroxidation (MDA) in serum, heart, and brain, alongside elevated nitric oxide in brain and heart.
Çetin et al., [22]	Daily 60-minute exposure to 900 or 1800 MHz mobile phone EMR	Growing rats (exposed from gestation through 6 weeks of age)	Induced oxidative stress in brain & liver, evidenced by reduced antioxidant enzyme (GPx) activity and altered micronutrient levels (decreased liver Vit A/β-carotene, increased brain iron/Vit A/β-carotene, decreased brain selenium).
Megha et al., [83]	Low-intensity microwave radiation (900, 1800, 2450 MHz; 2 h/day, 5 days/week).	Male Fischer 344 rats	Frequency-dependent induction of oxidative stress (↑MDA, PC, CAT; ↓GSH, SOD), inflammatory response (↑pro-inflammatory cytokines), and DNA damage in rat hippocampus.
Asl et al., [9]	1-month daily exposure to RF radiation (915 MHz mobile phone or 2450 MHz Wi-Fi, 60 min/day) with or without pre-treatment with (RA, 20 mg/kg/day orally).	Rats	RF radiation induced significant oxidative stress in rat brain (↑MDA, NO, PC; ↓GSH, GPx, TAC, SOD, CAT). RA effectively counteracted this damage, normalizing oxidative stress markers and restoring antioxidant defenses.
Alchalabi et al., [6]	Daily exposure to 1800 MHz GSM radiation for 2 h/day over 30 or 60 days.	Female Sprague Dawley rats.	RF radiation induced significant oxidative stress and histopathological damage in ovarian and uterine tissues, including follicular degeneration, nuclear abnormalities, inflammation, and epithelial apoptosis.
Gautam et al., [43]	45-day exposure to 3G mobile phone radiation (2 h/day) in a controlled setup.	Male Wistar rats.	Induced oxidative stress (↑ROS, ↑lipid peroxidation) leading to impaired male reproductive function: reduced sperm count, altered sperm tail morphology, damaged sperm membrane, and decreased spermatogenic cells.
Moghadasi et al., [86]	10-day exposure to 900 MHz mobile phone radiation (8 h/day) with or without vitamin C supplementation (200 mg/kg).	Pregnant mice.	RF radiation induced oxidative stress (↑MDA, ↑8-OHdG, ↓TAC) in pregnant mice, which was effectively prevented by vitamin C.
Pagadala and Shankar [95],	Daily 3-hour exposure to 0.9/1.8 GHz mobile phone RF-EMR (GSM, answer mode) for 80 days, with the phone placed 15–20 cm from the animals.	Male Sprague Dawley rats.	RF-EMR induced oxidative stress (↑malondialdehyde, ↑corticosterone) and reduced feeding behavior.
Ferreira et al., [41]	Acute exposure to UHF-EMF (800–1800 MHz; specific duration/dose not detailed in summary).	Rats (frontal cortex and hippocampus examined).	No significant change in oxidative stress markers (MDA, carbonyls) or non-enzymatic antioxidant defense in frontal cortex or hippocampus after acute exposure.
Alkis et al., [7]	Long-term RFR exposure (900, 1800, or 2100 MHz; 2 h/day for 6 months).	Male Sprague Dawley rats.	RFR exposure induced dose/frequency-dependent oxidative stress and DNA damage in brain tissue: ↑MDA, ↑8-OHdG, ↑TOS, altered oxidative balance, with 2100 MHz specifically causing DNA single-strand breaks.
Ragy [100],	60-day exposure to 900 MHz mobile phone EMR (1 h/day), with an additional withdrawal group observed for 30 days post-exposure.	Male albino rats.	EMR exposure induced systemic oxidative stress (↑MDA, ↓TAC) in brain, liver, and kidney tissues, alongside elevated liver enzymes (ALT, AST), renal markers (urea, creatinine), and serum corticosterone. These effects were reversible after a 30-day withdrawal period.
Keleş et al., [61]	0.9 GHz EMF (900 MHz; 1 h/day for 25 days; postnatal days 21–45)	Rat (Male, 21 days old)	0.9 GHz EMF exposure induced oxidative stress (↑LPO, ↑GSH, ↑CAT, ↑SOD) in kidney tissue, along with increased kidney volume/weight and body weight, and severe histopathological damage including hemorrhage, fibrosis, tubular and glomerular degeneration. Mechanistically, it activated the renin-angiotensin system (RAS) in the kidney.

Table 5 (continued)

References	Intervention description	Animal species	Main effect
Xu et al., [140]	Multi-frequency RF-EMR (0.9, 1.5, 2.65 GHz; sequential; 2 h/day; SAR: 4.0 W/kg) for 3–4 weeks	Mouse (Male C57BL/6, 5–8 weeks)	Improved lipid profile ($\downarrow$ TG, $\downarrow$ LDL; $\uparrow$ HDL); upregulated UCP1 and enhanced lipid metabolism in adipose tissue; promoted adiponectin secretion and hepatic β -oxidation; reduced hepatic steatosis and fibrosis in obese mice.
Ilgaz et al., [54]	6 GHz RF-EMR (SAR: 0.065 W/kg; 4 h/day for 42 days)	Rat (Adult male)	Induced histopathological damage in liver tissue (portal inflammation, single cell necrosis, vascularity, congestion) and increased DNA damage (comet assay), although the genetic damage index (GDI) and damaged cell percentage (DCP) were not statistically significant ($p > 0.05$).

EMF: Electromagnetic Field; **EMR:** Electromagnetic Radiation; **RF-EMR:** Radiofrequency Electromagnetic Radiation; **RF-EMF:** Radiofrequency Electromagnetic Field; **RFR:** Radiofrequency Radiation; **UHF-EMF:** Ultra-High Frequency Electromagnetic Field; **GSM:** Global System for Mobile Communications; **SAR:** Specific Absorption Rate; **RAS:** Renin-Angiotensin System; **HPA axis:** Hypothalamic-Pituitary-Adrenal axis; **MDA:** Malondialdehyde; **GSH:** Glutathione; **GST:** Glutathione S-Transferase; **GPx:** Glutathione Peroxidase; **SOD:** Superoxide Dismutase; **CAT:** Catalase; **TAC:** Total Antioxidant Capacity; **TOS:** Total Oxidant Status; **AOPP:** Advanced Oxidation Protein Products; **PC:** Protein Carbonyl; **8-OHdG:** 8-Hydroxy-2'-deoxyguanosine; **RBC:** Red Blood Cell; **WBC:** White Blood Cell; **ALT:** Alanine Aminotransferase; **AST:** Aspartate Aminotransferase; **ACTH:** Adrenocorticotrophic Hormone; **IL-1 β :** Interleukin-1 beta; **IL-6:** Interleukin-6; **TNF- α :** Tumor Necrosis Factor-alpha; **Akt1:** Protein Kinase B isoform 1; **Nrf2:** Nuclear Factor Erythroid 2-Related Factor 2; **ICAM1:** Intercellular Adhesion Molecule 1; **β -carotene:** Beta-carotene; **MPBS:** Mobile Phone Base Station; **ROS:** Reactive Oxygen Species; **RA:** Rosmarinic Acid; **LPO:** Lipid Peroxidation; **TG:** Triglycerides; **LDL:** Low-Density Lipoprotein; **HDL:** High-Density Lipoprotein; **UCP1:** Uncoupling Protein; **GDI:** Genetic Damage Index; **DCP:** Damaged Cell Percentage

results showed no considerable changes in body mass, rectal temperature, or testicular histology. However, significant adverse effects were observed, including reduced testicular mass, decreased interstitial cell count, impaired sperm motility, abnormal sperm morphology, and elevated follicle-stimulating hormone (FSH) levels. These findings demonstrate that prolonged IF-EMR exposure can negatively impact key reproductive parameters in male rats, suggesting potential risks to male fertility.

A cross-sectional study of male workers in an automotive foundry indicated that occupational co-exposure to heat stress and ELF-EMF, particularly electric fields (> 0.42 V/m), was associated with lower serum testosterone levels. Although the observed trend was not statistically significant ($p > 0.05$), regression analysis identified the electric field as the most influential physical stressor. The authors noted that definitive attribution was challenging due to multiple concurrent workplace exposures, including chemical, ergonomic and psychological stressors [87].

Carcinogenic effects It is widely recognized that epidemiological studies alone cannot establish a clear causal link between EMFs and cancer. Such studies primarily assess statistical associations, which do not inherently prove causation. Establishing causality requires consistent and strong associations, a clear dose-response relationship, supportive animal data, a plausible biological mechanism, and overall consistency across studies. In EMF-cancer research, most of these criteria are typically not met. Consequently, investigations into the health effects of EMFs have largely focused on

MFs, as they are considered the component most likely to elicit biological effects [11].

However, while epidemiological evidence remains inconclusive, experimental animal studies suggest that MF exposure may act as a cancer-promoting factor. Such studies are frequently employed to assess agents suspected of being human carcinogens [109]. Table 8 provides an overview of in vivo studies on the carcinogenic and antitumor effects of electromagnetic radiation (EMR), highlighting the heterogeneity of the evidence. Research conducted by the Ramazzini Institute on over 10,000 Sprague-Dawley rats demonstrated that long-term exposure to a 50 Hz MF, simulating power-line fields, in combination with gamma radiation or formaldehyde significantly increased the incidence of malignant tumors. Additionally, exposure to 1.8 GHz GSM radiation, simulating base station emissions, alone was associated with a statistically significant increase in malignant schwannomas of the heart in male rats. These findings provide experimental evidence supporting the potential carcinogenic effects of prolonged electromagnetic field exposure [120]. Further evidence for the carcinogenic potential of high-frequency EMF comes from a study on high-power microwaves (HPM). Repeated exposure of rats to nanosecond pulses at 10 and 3.7 GHz significantly reduced survival time (by 4 months) and increased subcutaneous tumor incidence (11 vs. 2 in sham), despite sub-thermal average exposure and minimal behavioral effects. This study suggests that even non-thermal, pulsed high-frequency EMF may pose cancer risks, highlighting the need for further research on exposure thresholds [30].

In contrast, a substantial number of long-term bioassays have failed to demonstrate carcinogenic effects under

Table 6 Effects of EMR exposure on Neurological and Behavioral function: In vivo

References	Intervention description	Animal species	Main effect
Gupta et al., [47]	Long-term EMR exposure (900, 1800, 2450 MHz; 1 h/day for 28 days).	Male rats	2450 MHz EMR exposure induced anxiety-like behavior and disrupted HPA axis function ($\uparrow$ corticosterone, $\downarrow$ CRH-2 & GR in amygdala), impaired mitochondrial integrity, and triggered apoptotic/necrotic cell death in the amygdala, leading to neuronal loss.
Hasan et al., [50]	2400 MHz RF-EMR (4G radiation) for 40–60 min daily over 60 days.	Swiss albino male mice (<i>Mus musculus</i>)	Increased anxiety-like behavior and significant neuronal loss and structural damage in the hippocampus of mice.
Wdowiak et al., [136]	Occupational and lifestyle exposure to electromagnetic fields from wireless systems (mobile phones, base stations/relay towers, WLAN) and engagement in physical activity.	Human female subjects (200 women aged 25–35, comprising medical staff and shopping center employees).	Exposure to GSM 900/1800 base station EMF was associated with increased depressive disorders in medical staff, while WLAN EMF showed a protective effect. Mobile phone use for conversations reduced depression but increased trait anxiety in medical staff. Physical activity intensified depressive states in trade worker
Zhu et al., [152]	Provocation exposure to pulsed 2.4 GHz microwave radiation from a cordless phone (3-minute intervals) versus sham exposure in a double-blind design.	Human subjects (25 individuals aged 37–79, including those with self-reported electrohypersensitivity).	40% of subjects showed measurable changes in HRV attributable to microwave exposure, ranging from extreme (tachycardia) to moderate autonomic nervous system shifts. Self-reported symptoms among sensitive individuals included cognitive, neurological, and cardiovascular complaints.
Li et al., [70]	Acute high-power exposure to S-band microwave radiation (30 mW/cm ² for 35 min), simulating occupational electromagnetic radiation conditions.	Rats	Exposure to microwave radiation causes severe cardiac damage, mitochondrial dysfunction, oxidative stress, elevated stress hormones, altered heart rate, weight loss, and behavioral impairments such as anxiety and depression, mediated through activation of the JNK stress pathway in cardiac tissue.
Zheng et al., [150]	Continuous whole-body exposure to 2650 MHz electromagnetic radiation (simulating 5G) at 2.06 W/kg for 4 h/day over 28 days.	Male mice	EMR exposure induced anxiety-like behavior (reduced locomotion and exploration in OFT and EPM) but no significant depression-like behavior (TST unchanged) or spatial memory impairment (MWM unchanged). It also elevated serum corticosterone (CORT) and reduced hippocampal expression of BDNF and GR, while GABA remained unchanged.
Mahdavi et al., [76]	Short-, medium-, and long-term exposure to ELF-EMF at 1 Hz and 5 Hz.	Male Wistar rats.	Exposure to ELF-EMF at 1 and 5 Hz produced frequency- and time-dependent effects: 5 Hz increased plasma glucose, elevated ACTH, reduced noradrenaline, and enhanced exploratory behaviors, while 1 Hz decreased plasma glucose, raised ACTH, and lowered noradrenaline, with no change in body weight or food/water intake.
Daniels et al., [27]	Exposed to 840 MHz EMR for 3 h per day from postnatal day 2 to day 14 (P2–P14).	Male and female rats	Reduced locomotion, increased grooming, tendency for higher corticosterone; possible neuronal damage in hippocampus (per prior study). No spatial memory or major structural changes.
Júnior et al., [57]	Short-term exposure to RF-EMR from a GSM cell phone (1.8 GHz) for 3 days.	Male Wistar rats (60 days old).	Exposure did not induce anxiety or working memory impairment, but increased stress-related behaviors were observed, suggesting RF-EMR may act as a mild stressor without causing overt central nervous system impairment.
Zhang et al., [149]	Mouse offspring exposed in utero.	Gestational exposure to 9.417-GHz microwave radiation throughout pregnancy (days 3.5–18).	In utero microwave exposure caused gender-dependent behavioral changes: increased anxiety-like behavior in both sexes, decreased depression-like behavior in both sexes, and cognitive impairment (reduced learning and memory) only in male offspring, while females were unaffected.
Klose et al., [65]	Female Wistar rats (exposed from 14 days to 19 months of age).	Long-term, repeated head-region exposure to GSM-modulated 900 MHz RF-EMF at SARs of 0.7, 2.5, and 10 W/kg (2 h/day, 5 days/week).	No consistent or profound adverse effects on development, learning, or behavior were observed across multiple tests (rotarod, Morris water maze, 8-arm radial maze, open field) at any age or SAR level. Minor sporadic differences were not systematic and likely attributable to handling or random variation.
Wang et al., [132]	Single 30-minute exposure to 1.8 GHz RF-EMR at a high specific absorption rate (SAR > 2.2 W/kg, tested up to 3.3 W/kg).	Mice	Acute RF-EMR exposure enhanced recognition memory (increased NORT index), increased dendritic spine density and length in hippocampus and prefrontal cortex, and altered neuronal excitability (changes in resting membrane potential, action potential frequency, and kinetics).

Table 6 (continued)

References	Intervention description	Animal species	Main effect
Narayanan et al., [89]	4-week exposure to mobile phone RF-EMR (GSM 0.9/1.8 GHz) via 50 missed calls/hour per day, with the phone placed in vibratory mode inside the cage.	Healthy male albino Wistar rats	RF-EMR exposure significantly impaired passive avoidance memory (shorter latency to enter the dark compartment) and induced marked morphological changes in the hippocampal CA3 region.
Xue et al., [141]	Exposure to either single-frequency (2.65 GHz–0.8 GHz) or dual-frequency (2.65/0.8 GHz) EMR.	Mice	Dual-frequency radiation (2.65/0.8 GHz) induces anxiety in mice, while single frequencies do not. This anxiety is associated with increased stress hormones and disruption of the brain's endocannabinoid system, and can be alleviated by a cannabinoid receptor agonist (Win55-212-2).
Yang et al., [143]	Continuous 72-hour exposure to 3500 MHz RF-EMR at three SAR levels (2, 4, and 10 W/kg), compared to sham exposure (0 W/kg).	Male guinea pigs.	RF-EMR exposure did not alter hearing thresholds or anxiety-like behavior, but it induced SAR-dependent oxidative stress (increased MDA, decreased CAT, SOD, GSH-px) in the auditory cortex, leading to ultrastructural damage (swollen mitochondria, layered myelin sheaths) and mitochondria-dependent apoptosis (cytochrome-c release, caspase-9/-3 activation).
Liu et al., [73]	Exposure to 2.4 GHz EMR modulated by 100 Hz square pulses (simulating typical wireless signals).	Mice	EMR exposure significantly increased wakefulness in mice, identifying sleep disruption as a direct consequence of chronic wireless signal exposure.
Narayanan et al., [90]	Chronic exposure to 900 MHz RF-EMR (power density 146.60 $\mu\text{W}/\text{cm}^2$) from a mobile phone in silent-mode for 28 days.	Adolescent male albino Wistar rats (4 weeks old at start).	RF-EMR exposure induced structural and cellular alterations in the amygdala: reduced healthy neurons and increased apoptosis in basolateral and cortical amygdala, altered dendritic arborization in basolateral amygdala, and behavioral changes (altered place preference, hyperactivity-like behavior), without affecting caspase-3 activity.
Spandole-Dinu et al., [122]	Long-term (16-week) continuous exposure to 2.45 GHz RF radiation from both a household Wi Fi router (real life simulation) and a laboratory device, compared to sham exposure.	Mice	RF exposure increased locomotor activity, reduced global DNA methylation in the brain, but did not induce significant structural or histopathological changes.
Gökçek-Saraç [45],	Short-term exposure to 2.1 GHz RF-EMR at two electric-field intensities (45 V/m and 65 V/m) for 2 h/day over 7 days, compared to sham exposure.	Rats	Short-term exposure to 2.1 GHz RF-EMR produced dose-dependent effects: higher doses (65 V/m) impaired recognition memory, while both doses (45 & 65 V/m) reduced anxiety-like behavior. Locomotor activity remained unchanged.
Choi and Choi [24],	Chronic exposure to smart-phone-emitted electromagnetic radiation (duration: 9 weeks for males, 11 weeks for females) with a recovery period tested in some females.	Male and female mice.	Chronic radiation did not impair spatial working memory or hippocampal progenitor cell proliferation, but it increased astrocyte activation (GFAP immunoreactivity) and induced delayed hyperactivity-like behavior (increased arm entries in the Y-maze after extended exposure and recovery).
Hosseini et al., [52]	Prenatal exposure to (1) chronic maternal stress, (2) ELF-EMF (50 Hz, 100 μT) before and during pregnancy, or (3) combined stress + ELF-EMF.	Female rat offspring	Both prenatal stress and ELF-EMF independently increased anxiety-like behavior in offspring, and their combined exposure further exacerbated this effect. Stress reduced serotonin and increased corticosterone, while ELF-EMF elevated the hippocampal PNMDAr2/NMDAr2 ratio and 24(S)-hydroxycholesterol. ELF-
Sharma and Shukla [113],	Long-term (90-day) daily exposure to 900 MHz microwave radiation (mobile-phone-like) at varying durations: 1 h, 2 h, or 4 h per day.	Male Wistar rats	Prolonged mobile-phone EMR exposure induced time-dependent oxidative stress (increased MDA, decreased SOD, CAT, and key redox enzymes), reduced acetylcholinesterase activity (impaired cholinergic neurotransmission), caused cellular degeneration and DNA damage in the hippocampus, and led to working-memory deficits.

Table 6 (continued)

References	Intervention description	Animal species	Main effect
Jing et al., [55]	Whole-body exposure to multi-frequency RF-EMR (0.9, 1.5, and 2.65 GHz simultaneously) at 4 W/kg for 2 h/day over 4 weeks.	Mice	Multi-frequency RF-EMR induced anxiety-like behavior in mice by triggering a neuroinflammatory response in the cerebral cortex (via activation of the CREB-BDNF-TrkB pathway, suppression of NF- κ B, elevation of pro-inflammatory cytokines, and reduction of anti-inflammatory cytokines) and decreasing excitatory neurotransmitter levels (GABA, dopamine, serotonin).
Shehu et al., [115]	4-week daily exposure (10 min of missed calls) to a mobile phone in different modes: silent, vibration, ringtone, or vibration+ringtone, compared to a switched-off phone (control).	Male albino Wistar rats	All mobile-phone exposure modes (EMR, vibration, ringtone, or combined) significantly increased anxiety-like behavior (reduced open-arm duration). Ringtone and combined vibration+ringtone modes also reduced catalase activity, indicating oxidative stress in the brain.
Tarsaei et al., [126]	Short-term and long-term daily exposure to 2.45 GHz RF-EMR (Wi-Fi-like) for 1 h/day, compared to sham exposure	Rats	Exposure induced anxiety-like behavior (reduced open-arm exploration), elevated plasma corticosterone, and altered hippocampal glucocorticoid receptor expression, while long-term exposure did not produce significant behavioral changes.
Djordjevic et al., [33]	Short-term exposure to 50 Hz ELF-EMF for 7 days	Adult male Wistar albino rats.	ELF-EMF exposure induced anxiety-like behavior (reduced exploration in open-field and elevated plus-maze) and increased hypothalamic oxidative stress (elevated superoxide anion) along with elevated nitric oxide (NO) concentration, but did not alter peroxynitrite levels.
Karimi et al., [59]	60-day exposure (2 h/day) to ELF-EMF at four intensities (1, 100, 500, and 2000 μ T), compared to sham and control groups.	Adult male Wistar rats.	Exposure to ELF-EMF produced intensity-dependent effects: at 100–2000 μ T, memory retention was improved; at 500–2000 μ T, anxiety-like behavior increased; and at 100–500 μ T, oxidative stress (elevated malondialdehyde) was heightened.
Ebrahimi et al., [34]	Chronic exposure to 50 Hz extremely low-frequency electromagnetic field (ELF-EMF) for 3 h/day over 30 days.	Male BALB/c mice	ELF-EMF exposure decreased depression-like behavior (reduced immobility in forced swim test) but increased anxiety-like behavior (reduced open-arm exploration in elevated plus maze) and elevated thyroid hormones (T4 and TSH). No significant change in gene expression was observed.
Spandole-Dinu et al., [122]	28-day exposure to 900 MHz RF-EMR from an active GSM mobile phone (peak power density 146.60 μ W/cm ²), compared to sham-exposed (phone off) and control groups.	Adolescent male albino Wistar rats (6–8 weeks old).	RF-EMR exposure increased anxiety-like behavior (reduced open-arm exploration, rearing, and grooming; increased defecation) without altering general locomotion (no change in total distance travelled or total arm entries).
Faraji et al., [40]	2-month daily exposure (1 h/day) to 50 Hz ELF-EMF at four intensities (1, 100, 500, 2000 μ T), compared to an unexposed control group.	Male adult rats.	Exposure to 50 Hz ELF-EMF at intensities of 100–2000 μ T enhanced memory retention, increased anxiety-like behavior, and triggered microglial activation in the rat brain, without inducing amyloid-beta deposition.
Deng et al., [31]	2.4 GHz or 935 MHz EMR with varied pulse modulations (10, 100, 1000 Hz)	Mice	Increased wakefulness specifically with 2.4 GHz pulsed modulation (100 & 1000 Hz), not with 935 MHz or continuous modulation.
Zhi et al., [151]	Microwave radiation (2.856 GHz; mean power density: 30 mW/cm ² ; 300 Hz; 0.015% duty cycle; 3 $\times$ 6 min/day; total 18 min/day for 4 days)	Rat	Induced cognitive/memory deficits (behavioral tests), impaired synaptic plasticity in mature hippocampal neurons, and suppressed NSC proliferation/development in the DG.
Lin et al., [71]	EMP radiation (600 kV/m; 1000 pulses/day for 2 weeks)	Mouse	Increased oxidative markers in brain tissue. Induced microglial M1 polarization and pro-inflammatory responses. Damaged blood-brain barrier integrity via microglial activation. Activated NLRP3 inflammasome/NF- κ B signaling pathway.
Narayanan et al., [91]	900 MHz RF-EMR (mobile phone; 1 h/day for 4 weeks)	Rat (Male albino Wistar, 4 weeks old, 50–60 g)	Altered contextual fear conditioning (increased compartment transitions, decreased latency to enter dark compartment). Apoptosis in CA3 region and enlarged perivascular space. Lymphocytic infiltrates, congested sinusoids, and apoptotic-like changes in zona fasciculata (medulla unaffected). Indicated HPA axis dysregulation via non-thermal mechanisms.

Table 6 (continued)

References	Intervention description	Animal species	Main effect
Er et al., [36]	2100 MHz RFR (2 h/day, 5 days/week for 1 week (acute) or 10 weeks (chronic))	Rat (Adult)	Acute exposure prolonged ABR wave latencies, indicating auditory brainstem dysfunction, while chronic exposure showed no such effects. Acute exposure also induced brain oxidative stress ($\uparrow$ 4-HNE, $\uparrow$ TBARS, $\uparrow$ protein carbonyl; $\downarrow$ CAT, $\downarrow$ SOD) and caused ultrastructural damage (edema in neurons and astrocytes, mitochondrial damage)
Postacı Karaman et al., [98]	2100 MHz or 2450 MHz EMF (1 h/day for 30 days), with or without ginseng co-administration (150 mg/kg/day for 30 days)	Rat	EMF exposure induced apoptosis in brain tissue ($\uparrow$ BAX protein, $\downarrow$ COX-2 gene expression), indicating BBB and neuronal damage. Ginseng co-administration significantly attenuated these effects ($\downarrow$ BAX, $\uparrow$ COX-2), demonstrating a protective role against EMF-induced neurotoxicity and apoptosis.
Haroon et al., [49]	2100 MHz (4G) or 3500 MHz (5G) RF-EMR; 5 h/day for 4 weeks; ADALM-PLUTO module	Rat (Adult male Sprague-Dawley)	Both 4G and 5G exposure maintained regular cortical layering, but 5G (3500 MHz) induced significantly greater granule cell necrosis in the auditory cortex compared to 4G (2100 MHz) ($P = 0.006$).
Sun et al., [123]	Compound microwave (sequential exposure to 2.8 GHz and 9.3 GHz; average power density: 10 mW/cm ² ; specific duration in full text)	Rat	Induced cognitive impairment ($\uparrow$ escape latency), EEG abnormalities ($\uparrow$ θ/δ waves), and hippocampal damage (mitochondrial disorder, $\downarrow$ Nissl bodies) at 1–7 days, with strongest effects in compound group; all recovered by day 28. Huntingtin and BDNF identified as potential blood biomarkers.

EMR: Electromagnetic Radiation; **RF-EMR:** Radiofrequency Electromagnetic Radiation; **EMF:** Electromagnetic Field; **ELF-EMF:** Extremely Low-Frequency Electromagnetic Field; **GSM:** Global System for Mobile Communications; **WLAN:** Wireless Local Area Network; **SAR:** Specific Absorption Rate; **μ W/cm²:** Microwatt per square centimeter; **mW/cm²:** Milliwatt per square centimeter; **V/m:** Volt per meter; **μ T:** Microtesla; **HPA axis:** Hypothalamic-Pituitary-Adrenal axis; **CRH-2:** Corticotropin-Releasing Hormone Receptor 2; **GR:** Glucocorticoid Receptor; **BDNF:** Brain-Derived Neurotrophic Factor; **GABA:** Gamma-Aminobutyric Acid; **ACTH:** Adrenocorticotrophic Hormone; **CORT:** Corticosterone; **JNK:** c-Jun N-Terminal Kinase; **OFT:** Open Field Test; **EPM:** Elevated Plus Maze; **TST:** Tail Suspension Test; **MWM:** Morris Water Maze; **NORT:** Novel Object Recognition Test; **HRV:** Heart Rate Variability; **CA3:** Cornu Ammonis area 3; **CREB:** cAMP Response Element-Binding Protein; **TrkB:** Tropomyosin Receptor Kinase B; **NF- κ B:** Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells; **PNMDAR2:** Phosphorylated N-Methyl-D-Aspartate Receptor subunit 2; **NMDAR2:** N-Methyl-D-Aspartate Receptor subunit 2; **24(S)-hydroxycholesterol:** 24(S)-Hydroxycholesterol; **MDA:** Malondialdehyde; **SOD:** Superoxide Dismutase; **CAT:** Catalase; **GPx:** Glutathione Peroxidase; **GFAP:** Glial Fibrillary Acidic Protein; **Win55 212 2:** Cannabinoid receptor agonist Win55,212-2; **NSC:** Neural Stem Cell; **DG:** Dentate Gyrus; **EMP:** Electromagnetic Pulse; **M1:** Type 1 Macrophage (Pro-inflammatory Phenotype); **NLRP3:** NOD-like Receptor Pyrin Domain-Containing 3; **RFR:** Radiofrequency Radiation; **ABR:** Auditory Brainstem Response; **4-HNE:** 4-Hydroxynonenal; **TBARS:** Thiobarbituric Acid Reactive Substances; **BBB:** blood-brain barrier; **BAX:** Bcl-2-Associated X Protein; **COX-2:** Cyclooxygenase-2; **EEG:** Electroencephalogram

various exposure conditions. A two-year study on Fischer 344 rats exposed to 1.6 GHz RF radiation (Iridium signal) from gestation to adulthood found no significant differences in survival, neoplastic lesions, or reproductive outcomes between exposed and sham groups [8]. Similarly, chronic lifelong exposure of AKR/J mice to UMTS RF-EMF at 0.4 W/kg in radial waveguides did not affect lymphoma incidence, survival time, or disease severity [121]. In contrast to both tumor-promoting and null findings, a separate line of research has demonstrated that specific EMF exposure parameters can induce beneficial antitumor effects. Barati et al., [10] showed that ELF-EMF exposure triggers programmed cell death in breast cancer cells via increased intracellular calcium and reactive oxygen species (ROS), activating both necroptosis and apoptosis pathways, findings further supported by in vivo evidence of inhibited tumor growth, reduced angiogenesis, and a robust antitumor immune response. Therefore, animal evidence on EMF carcinogenicity remains mixed. While some large-scale studies report significant tumor increases, others find no effects.

This inconsistency, along with methodological limitations and the absence of a clear dose-response relationship, prevents conclusive conclusions.

Discussion

The present review consolidates evidence from a wide range of in vitro and in vivo studies, demonstrating that exposure to EMR exerts a broad spectrum of biological effects on mammalian systems. The findings indicate that EMR exposure induces oxidative stress, DNA damage, apoptosis, and inflammatory responses across multiple organs, including the brain, liver, kidney, testis, and adrenal gland. Notably, the most robust evidence supports adverse effects on the nervous system, manifesting as cognitive and behavioral impairments, hippocampal structural damage, and disruption of the blood-brain barrier (BBB). Similarly, reproductive toxicity, characterized by reduced sperm quality, testicular injury, and hormonal dysregulation was

Table 7 Effects of EMR exposure on Reproductive System: In vivo

References	Intervention description	Animal species	Main effect
Aderemi et al., [2]	RF-EMR from charging laptop computers for 2–8 h daily over 4 weeks	Male albino rats	Significant reduction in sperm motility, viability, count, and testosterone; increased sperm abnormalities.
Liu et al., [72]	Chronic exposure to 900 MHz RF-EMR (SAR 0.66 W/kg) for 2 h/day over 50 days	Male rats	RF-EMR exposure significantly increased sperm apoptosis (91% rise), elevated ROS, reduced total antioxidant capacity, and shifted apoptosis-related protein/gene expression ($\downarrow$ Bcl-2, $\uparrow$ Bax, cytochrome c, caspase-3), indicating oxidative stress-mediated sperm damage.
Adebayo et al., [1]	~ 1800 MHz RF-EMR ($\approx$ 1.40 W/cm ²) from telecom masts for 5 weeks	Rats	Histological damage in liver, heart, kidney, testis; reduced sperm quality; altered blood parameters.
Yu et al., [146]	Long-term (50, 100, or 150 days) localized scrotal exposure to 4G smartphone RF-EMR (active talk-mode) for 6 h/day (1-min call per 10-min interval).	Rats (adult males)	150 days of 4G exposure caused testicular damage and reduced sperm quality via Spock3 overexpression and blood-testis barrier disruption; inhibiting Spock3 reversed this damage. Shorter exposures (50 or 100 days) were not harmful.
Kumar et al., [68]	Daily 2-hour exposure to 10 GHz microwave radiation at 0.21 mW/cm ² (SAR 0.014 W/kg) for 45 days.	Male Wistar rats (70 days old).	Exposure caused testicular damage (shrinking seminiferous tubules, apoptotic bodies, reduced testicular size), decreased testosterone, DNA strand breaks (comet assay), micronucleus formation in lymphocytes, and chromosomal aberrations.
Yüksel et al., [147]	Daily 60-minute exposure to 900 MHz (mobile phone), 1800 MHz (mobile phone), or 2450 MHz (Wi-Fi) electromagnetic radiation during pregnancy and postnatal development.	Pregnant rats and their offspring across four generations.	EMR exposure elevated uterine oxidative stress and lipid peroxidation in both maternal and offspring rats, reduced offspring uterine glutathione peroxidase activity, and lowered maternal plasma reproductive hormones (prolactin, estrogen, progesterone).
Sundaram et al., [124]	Whole-body continuous 150 kHz IF-EMR for 8 weeks	Male Sprague-Dawley rats	Reduced testicular mass, interstitial cell count, sperm motility; increased FSH. No effect on body mass, temperature, or general testicular histology.
Cakir et al., [20]	2.45 GHz EMR (2 h/day for 1 month) with or without alpha-lipoic acid (ALA; 150 mg/kg/day)	Rat (Adult male)	EMR exposure induced testicular injury (disrupted seminiferous tubules, $\uparrow$ collagen, $\uparrow$ tubular/interstitial volume), inflammation ($\uparrow$ IL-6, $\uparrow$ TNF- α), oxidative stress ($\downarrow$ SOD, GSH, GPx; $\uparrow$ MDA), and reduced AR, ZO-1, ZO-2 expression. ALA co-administration significantly prevented these alterations, demonstrating a protective role against EMR-induced reproductive toxicity.

IF-EMR: Intermediate Frequency Electromagnetic Radiation; **RF-EMR:** Radio-frequency Electromagnetic Radiation; **MHz:** Megahertz; **GHz:** Gigahertz; **kHz:** Kilo-hertz; **SAR:** Specific Absorption Rate; **W/kg:** Watt per kilogram; **mW/cm²:** Milliwatt per square centimeter; **W/cm²:** Watt per square centimeter; **ROS:** Reactive Oxygen Species; **Bcl 2:** B-Cell Lymphoma 2; **Bax:** Bcl-2-Associated X Protein; **FSH:** Follicle-Stimulating Hormone; **Spock3:** SPARC/Osteonectin, CWCV and Kazal-like domains proteoglycan 3; **EMR:** Electromagnetic Radiation; **ALA:** Alpha-Lipoic Acid; **IL-6:** Interleukin-6; **TNF- α :** Tumor Necrosis Factor-alpha; **SOD:** Superoxide Dismutase; **GSH:** Glutathione; **GPx:** Glutathione Peroxidase; **MDA:** Malondialdehyde; **AR:** Androgen Receptor; **ZO-1:** Zonula Occludens-1; **ZO-2:** Zonula Occludens-2

frequently reported. These observations are in line with previous reviews [111, 142] and reinforce the growing concern that chronic or high-intensity EMR exposure may pose significant health risks.

However, a critical finding from our review is that the biological outcome of EMR exposure is highly dependent on exposure parameters, including frequency, power density, duration, and modulation pattern. For instance, while acute exposure to 2100 MHz RFR induced auditory brainstem dysfunction and ultrastructural damage in the rat brain, chronic exposure with intermittent rest days did not produce

such harmful effects [36]. Similarly, acute exposure to 9.375 GHz HPM did not induce anxiety, depression, or cognitive impairment; paradoxically, it enhanced antioxidant enzyme activities (SOD, CAT, GSH-Px) and reduced oxidative markers (MDA, PC) in serum and brain tissue, suggesting an adaptive antioxidant response [134]. These findings challenge the oversimplified view that all EMR exposures are consistently detrimental and highlight the importance of considering exposure patterns particularly recovery periods in risk assessment. Furthermore, the protective roles of antioxidants, such as alpha-lipoic acid (ALA) and ginseng,

Table 8 Carcinogenic and Antitumor Effects of EMR Exposure: A Heterogeneous Evidence Base: In vivo

References	Intervention description	Animal species	Main effect
Soffritti and Giuliani [120],	Lifelong exposure (prenatal to natural death) to: 1. S-50 Hz MF ± gamma rays/formaldehyde. 2. 1.8 GHz GSM ± gamma rays.	Sprague Dawley rats	1. S-50 Hz MF combined with gamma rays or formaldehyde significantly increased malignant tumour incidence. 2. 1.8 GHz GSM alone (highest dose) caused a significant increase in heart malignant schwannomas in males. Provides direct experimental evidence for co-carcinogenic and carcinogenic effects.
Barati et al., [10]	100 Hz, 1 mT ELF-EMF for 2 h/day for 5 days (in vitro) & 100 mT, 1 Hz ELF-EMF for 2 h/day for 28 days	BALB/c mice	Induction of necroptosis & apoptosis in breast cancer cells; suppressed tumor growth, enhanced immune response
Anderson et al., [8]	Long-term (gestation to 2 years) exposure to 1.6 GHz RF (Iridium signal). Phase 1: Far-field, whole body (0.16 W/kg fetal brain SAR). Phase 2: Near-field, head-first (0.16–1.6 W/kg brain SAR).	Fischer 344 rats	No significant differences in survival, clinical signs, reproductive outcomes, or incidence of neoplastic lesions between exposed and control groups. No evidence of carcinogenicity under these specific exposure conditions.
Falcioni et al., [39]	Life-span (prenatal to natural death), far-field, whole-body exposure to 1.8 GHz GSM RFR at 0, 5, 25, or 50 V/m for 19 h/day.	Sprague-Dawley rats	Significant increase in heart Schwannomas in high-dose (50 V/m) males. Non-significant increases in heart Schwann cell hyperplasia (both sexes) and malignant glial tumors (females). Provides direct experimental evidence supporting RFR carcinogenicity, consistent with NTP findings.
Seze et al., [30]	Acute or repetitive (8 weeks) exposure to high-power microwave pulses (10 GHz & 3.7 GHz; ~1 MV/m; 100 pps; ns duration).	Rats	Repetitive exposure: ↓ survival time by 4 months; ↑ subcutaneous tumors (11 vs. 2 in sham). Acute exposure: Avoidance reflex at 22 W/kg; transient ↑ GFAP. Conclusion: HPM below thermal average can increase cancer risk and reduce lifespan. Residual X-ray (0.8 Gy) present but unlikely to explain effect.
Tillmann et al., [128]	Lifelong (embryo-fetal to natural death) exposure to UMTS RF-EMF at 0, 4.8, or 48 W/m ² ; low-dose group combined with prenatal ENU (40 mg/kg).	Mice	No carcinogenic effect of UMTS alone. Significant cocarcinogenic effect at 4.8 W/m ² + ENU: increased lung tumor rate, carcinoma incidence, multiplicity, and metastasis.
Sommer et al., [121]	Chronic lifelong whole-body exposure to UMTS RF-EMF (0.4 W/kg SAR, 24/7) in radial waveguides.	Mice	No significant differences in lymphoma incidence, survival time, or disease severity between exposed and sham groups. No carcinogenic or tumor-promoting effect of chronic UMTS exposure detected.
Saran et al., [107]	Neonatal exposure (5 days, 0.5 h twice daily) to 900 MHz GSM RF radiation (SAR 0.4 W/kg).	Mice	No tumorigenic, life-shortening, or tumor-promoting effects observed. No differences in survival, cerebellar tumors, rhabdomyosarcomas, or skin preneoplastic lesions between exposed and sham-exposed groups.
Saran et al., [107]	1-year chronic simultaneous exposure to CDMA (849 MHz) + WCDMA (1.95 GHz) RF at 2.0 W/kg each (total 4.0 W/kg), 45 min/day, 5 days/week.	Sprague-Dawley rats	No increase in chronic illness, mortality, or tumor incidence. Some altered hematological and serum chemistry parameters observed, but not indicative of overall toxicity or carcinogenicity. No evidence of carcinogenic or chronic disease-promoting effects under these exposure conditions.

MF: Magnetic Field; **ELF-EMF:** Extremely Low-Frequency Electromagnetic Field; **RF:** Radiofrequency; **GSM:** Global System for Mobile Communications; **RFR:** Radiofrequency Radiation; **RF-EMF:** Radiofrequency Electromagnetic Field; **UMTS:** Universal Mobile Telecommunications System; **CDMA:** Code Division Multiple Access; **WCDMA:** Wideband Code Division Multiple Access; **MHz:** Megahertz; **GHz:** Gigahertz; **Hz:** Hertz; **mT:** Millitesla; **V/m:** Volt per meter; **W/m²:** Watt per square meter; **W/kg:** Watt per kilogram; **MV/m:** Megavolt per meter; **pps:** Pulses per second; **ns:** Nanosecond; **SAR:** Specific Absorption Rate; **ENU:** Ethylnitrosourea; **NTP:** National Toxicology Program; **HPM:** High-Power Microwave; **GFAP:** Glial Fibrillary Acidic Protein

in mitigating EMR-induced testicular and brain damage, as demonstrated in several included studies [20, 98, 151], open new ways for therapeutic intervention and underscore the critical role of oxidative stress as a central pathogenic mechanism.

EMR exposure leads to excessive generation of ROS, which overwhelms cellular antioxidant defenses, resulting in lipid peroxidation, protein carbonylation, and DNA strand breaks. This oxidative insult subsequently activates pro-inflammatory cascades, including the NLRP3 inflammasome/NF- κ B signaling pathway, as demonstrated in the context of EMP-induced neuroinflammation and BBB disruption [71]. In parallel, EMR exposure has been shown to trigger apoptosis through both intrinsic (mitochondrial) and extrinsic pathways, as evidenced by altered expression of BAX, BCL-2, and COX-2 in brain tissue [98]. Also, recent studies reveal epigenetic and epitranscriptomic mechanisms. For example, microwave radiation was found to downregulate HNRNPA2B1, leading to reduced m6A modification of TrkB mRNA, increased TrkB degradation, and decreased BDNF expression, ultimately impairing synaptic plasticity and cognitive function [151]. This evidence suggests that EMR effects extend beyond conventional oxidative stress to include complex regulatory networks at the transcriptional and post-transcriptional levels which require further investigation.

Although this review is comprehensive, there are several limitations to this study. First, the majority of included studies were conducted on animal models, and while they provide valuable mechanistic insights, their translational relevance to human health remains uncertain due to inter-species differences. Second, most studies focused on acute or sub-chronic exposure (days to weeks), whereas human exposure to EMR is typically lifelong and intermittent, highlighting a critical gap in our understanding of cumulative effects. Third, exposure protocols, including variations in frequency, SAR, duty cycle, and modulation in these studies are heterogeneous and this poses a challenge to conclusions and comparing studies. Future research should prioritize well-designed, long-term prospective studies on human populations incorporate real-world exposure. Furthermore, the potential therapeutic applications of EMR should be investigated to differentiate harmful from beneficial exposure parameters.

Conclusion

This review consolidates evidence from *in vitro* and *in vivo* studies demonstrating that EMR exposure consistently induces oxidative stress and DNA damage across multiple

tissues, with the nervous and reproductive systems being the most vulnerable targets. The severity and nature of these effects are highly dependent on exposure parameters, including frequency, power density, duration, and modulation pattern, with higher frequencies and prolonged exposure generally associated with more pronounced adverse outcomes. At the mechanistic level, oxidative stress emerges as a central mediator, triggering inflammatory cascades, apoptosis, and disruption of the blood brain barrier and testicular architecture. While the majority of studies confirm neurobehavioral impairments, reproductive dysfunction, and systemic inflammation, a subset of investigations, particularly those involving intermittent or low-dose exposure revealed adaptive responses or lack of overt toxicity, underscoring the importance of exposure protocols in risk assessment. Evidence regarding carcinogenicity remains mixed: some animal studies report increased tumor incidence, while others show no effects, and specific EMF parameters even exhibit therapeutic potential against cancer cells. These contradictions highlight critical knowledge gaps, particularly the scarcity of long-term human data and the lack of standardized exposure protocols. Future research must focus on delineating the thresholds and mechanisms that distinguish harmful effects from beneficial clinical interventions. Accordingly, future research should prioritize well-designed prospective human studies, explore multi-frequency real-world exposure scenarios, and investigate the molecular mechanisms underlying both harmful and protective effects.

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Declarations

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