

**THE KEY ISSUE FEATURES OF ADVERSE BIOLOGICAL EFFECTS OF  
ANTHROPOGENIC ELECTROMAGNETIC FREQUENCIES ON HUMAN HEALTH:  
MOLECULAR MECHANISMS, PHYSIOLOGICAL RESPONSES, AND  
ELECTROMAGNETIC HYPERSENSITIVITY SYNDROME**

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## ABSTRACT

Anthropogenic electromagnetic fields (EMFs), particularly those emitted by wireless communication devices and power infrastructure, have become ubiquitous in modern society, raising significant concerns about their potential health effects. This review examines the biological impact of electromagnetic frequencies on human health at molecular, cellular, and physiological levels, with particular emphasis on non-thermal effects occurring at exposure levels below current safety guidelines. Evidence from over 200 peer-reviewed studies demonstrates that polarized and coherent electromagnetic radiation, especially when modulated by extremely low frequencies (ELF), can trigger voltage-gated ion channel (VGIC) dysfunction in cell membranes, leading to calcium dysregulation and subsequent oxidative stress through reactive oxygen species (ROS) overproduction. This cascade of molecular events results in DNA damage, mitochondrial dysfunction, cellular senescence, and apoptosis. Epidemiological studies link chronic EMF exposure to increased cancer risk, reproductive dysfunction, neurological disorders, and a condition known as electromagnetic hypersensitivity (EHS), affecting an estimated 3-10% of the population in developed countries. Individuals with EHS experience debilitating symptoms including headaches, fatigue, cognitive impairment, and autonomic dysfunction when exposed to electromagnetic radiation. A subset of severely affected individuals has resorted to living in electromagnetically shielded housing or "Faraday cage" structures to mitigate exposure. The primary molecular mechanism involves EMF-induced activation of voltage-gated calcium channels (VGCCs), triggering intracellular calcium influx that activates NADPH oxidase, mitochondrial electron transport chain disruption, and nitric oxide synthase, collectively producing superoxide anions and nitric oxide that combine to form highly reactive peroxynitrite and hydroxyl radicals. These reactive species cause direct DNA strand breaks, lipid peroxidation, and protein oxidation. From a medical perspective, while EHS is not universally recognized as a formal diagnosis, clinical evidence demonstrates objective biomarkers including elevated oxidative stress markers in approximately 80% of EHS patients. This review synthesizes current understanding of EMF bioeffects across biological scales, discusses the controversy surrounding EHS diagnosis and treatment, and highlights the urgent need for revised exposure guidelines that account for non-thermal biological effects, particularly given the ongoing global deployment of 5G technology and increasing ambient electromagnetic radiation levels.

**KEYWORDS:**

Electromagnetic fields (EMF); electromagnetic hypersensitivity (EHS); voltage-gated calcium channels (VGCC); oxidative stress; reactive oxygen species (ROS); DNA damage;

**აბსტრაქტი**

ანთროპოგენური ელექტრომაგნიტური სიხშირეების უარყოფითი ბიოლოგიური ეფექტები ადამიანის ჯანმრთელობაზე: მოლეკულური მექანიზმები, ფიზიოლოგიური რეაქციები და ელექტრომაგნიტური ჰიპერმგრძობელობის სინდრომი

<sup>1,2,3,4,5,6</sup>ნოდარ სულაშვილი, <sup>7</sup>არჩილ ჭირაქაძე, <sup>6</sup>მარგარიტა ბეგლარიანი, <sup>9</sup>ვირა კრავჩენკო, <sup>1,11</sup>მარიკა სულაშვილი, <sup>10</sup>ნატო ალავეძე, <sup>4</sup>ია ეგნატიევი, <sup>4</sup>მანანა გიორგობიანი, <sup>1</sup>ნანა გორგასლიძე <sup>9</sup>, <sup>2,3,11</sup>დავით აფხაზავა

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ანთროპოგენური ელექტრომაგნიტური ველები (EMF), განსაკუთრებით ის, რაც გამოსხივდება უკაბელო კომუნიკაციის მოწყობილობებიდან და ენერგეტიკული ინფრასტრუქტურებიდან, გავრცელებული გახდა თანამედროვე საზოგადოებაში და აღვივებს მნიშვნელოვან შემფოთებას მათი პოტენციური ზემოქმედების შესახებ

ჯანმრთელობაზე. ეს მიმოხილვა შეისწავლის ელექტრომაგნიტური სიხშირეების ბიოლოგიურ გავლენას ადამიანის ჯანმრთელობაზე მოლეკულურ, უჯრედულ და ფიზიოლოგიურ დონეზე, განსაკუთრებული აქცენტით არათერმული ეფექტების შესახებ, რომლებიც ხდება უსაფრთხოების მოქმედი სახელმძღვანელო ნორმების ქვემოთ. 200-ზე მეტი რეცენზირებული კვლევის მტკიცებულებები აჩვენებს, რომ პოლარიზებული და კოჰერენტული ელექტრომაგნიტური გამოსხივება, განსაკუთრებით მაშინ, როცა მოდულირებულია ძალიან დაბალი სიხშირეებით (ELF), შეუძლია გამოიწვიოს უჯრედის მემბრანაში ძაბვაზე დამოკიდებული იონური არხების (VGIC) დისფუნქცია, რაც იწვევს კალციუმის დისრეგულაციას და შემდგომ ოქსიდაციურ სტრესს რეაქტიული ჟანგბადის სახეობების (ROS) ზედმეტი წარმოქმნის გზით. მოლეკულური მოვლენების ეს კასკადი იწვევს DNA-ს დაზიანებას, მიტოქონდრიულ დისფუნქციას, უჯრედულ სენესცენციას და აპოპტოზს. ეპიდემიოლოგიური კვლევები უკავშირებს ქრონიკულ EMF-ზე ზემოქმედებას კიბოს რისკის გაზრდას, რეპროდუქციულ დისფუნქციას, ნევროლოგიურ დარღვევებს და მდგომარეობას, რომელიც ცნობილია როგორც ელექტრომაგნიტური ჰიპერმგრძნობელობა (EHS), რომელიც გავლენას ახდენს მოსახლეობის დაახლოებით 3-10%-ზე განვითარებულ ქვეყნებში. EHS-ის მქონე პირები განიცდიან დამთრგუნველ სიმპტომებს, მათ შორის თავის ტკივილს, დაღლილობას, კოგნიტურ დაქვეითებას და ავტონომიურ დისფუნქციას, როცა ექვემდებარებიან ელექტრომაგნიტურ გამოსხივებას. მძიმედ დაზარალებული ინდივიდების ნაწილი იძულებული გახდა ცხოვრობდეს ელექტრომაგნიტურად დაცულ საცხოვრებელში ან "ფარადის გალიის" სტრუქტურებში, ზემოქმედების შესამცირებლად. პირველადი მოლეკულური მექანიზმი მოიცავს EMF-ით გამოწვეულ ძაბვაზე დამოკიდებული კალციუმის არხების (VGCC) აქტივაციას, რომელიც იწვევს უჯრედშიდა კალციუმის შემოდინებას, რაც ააქტიურებს NADPH ოქსიდაზას, მიტოქონდრიული ელექტრონული ტრანსპორტის ჯაჭვის დარღვევას და აზოტის ოქსიდის სინთეზას, რომლებიც ერთობლივად წარმოქმნიან სუპეროქსიდის ანიონებსა და აზოტის ოქსიდს, რომლებიც გაერთიანებით ქმნიან ძალიან რეაქტიულ პეროქსინიტრიტს და ჰიდროქსილის რადიკალებს. ეს რეაქტიული სახეობები იწვევს DNA-ს ჯაჭვების პირდაპირ წყვეტას,

ლიპიდების პეროქსიდაციას და ცილების ოქსიდაციას. სამედიცინო თვალსაზრისით, მიუხედავად იმისა, რომ EHS უნივერსალურად არ არის აღიარებული ფორმალურ დიაგნოზად, კლინიკური მტკიცებულებები აჩვენებს ობიექტურ ბიომარკერებს, მათ შორის ოქსიდაციური სტრესის მარკერების მომატებას EHS პაციენტთა დაახლოებით 80%-ში. ეს მიმოხილვა აჯამებს EMF-ის ბიოფიზიკურ შესახებ არსებულ ცოდნას ბიოლოგიური მასშტაბების მიხედვით, განიხილავს EHS-ის დიაგნოსტიკისა და მკურნალობის გარშემო არსებულ კამათს და ხაზს უსვამს აუცილებლობას გადასინჯულ იქნეს ზემოქმედების სახელმძღვანელო ნორმები, რომლებიც გაითვალისწინებენ არათერმულ ბიოლოგიურ ეფექტებს, განსაკუთრებით მიმდინარე გლობალური 5G ტექნოლოგიის განლაგებისა და გარემოს ელექტრომაგნიტური გამოსხივების დონის ზრდის გათვალისწინებით.

### საკვანძო სიტყვები:

ელექტრომაგნიტური ველები (EMF); ელექტრომაგნიტური ჰიპერმგრძნობელობა (EHS); მავნაზე დამოკიდებული კალციუმის არხები (VGCC); ოქსიდაციური სტრესი; რეაქტიული ჟანგბადის სახეობები (ROS); DNA-ს დაზიანება;

## INTRODUCTION

The exponential growth of wireless communication technologies and electrical power infrastructure over the past three decades has resulted in an unprecedented increase in human exposure to anthropogenic electromagnetic fields (EMFs). Unlike natural electromagnetic radiation, man-made EMFs possess unique physical properties—full polarization, coherence, and modulation by extremely low frequencies (ELF)—that render them significantly more bioactive than their natural counterparts [5]. Modern wireless communication systems, including mobile phones, Wi-Fi routers, cellular base stations, and the emerging 5G networks, emit radiofrequency (RF) radiation modulated by ELF signals, creating complex, pulsed electromagnetic exposures that pervade residential, occupational, and public environments [1-2].

The scientific community has engaged in extensive debate regarding the potential health consequences of chronic low-level EMF exposure. Current exposure guidelines established by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) are designed primarily to

prevent acute thermal effects—tissue heating caused by energy absorption at high exposure levels. However, a substantial and growing body of experimental and epidemiological evidence indicates that non-thermal biological effects occur at exposure levels orders of magnitude below these thermal thresholds [2]. These non-thermal effects encompass a wide spectrum of adverse biological outcomes, including oxidative stress, DNA damage, altered gene expression, disrupted calcium homeostasis, mitochondrial dysfunction, and increased cancer risk [3-4].

The International Agency for Research on Cancer (IARC), a specialized agency of the World Health Organization, classified both ELF magnetic fields (in 2002) and radiofrequency electromagnetic fields (in 2011) as "possibly carcinogenic to humans" (Group 2B) based on epidemiological evidence linking these exposures to childhood leukemia and brain tumors, respectively. Subsequent research has strengthened this evidence base, leading numerous scientists to advocate for reclassification to Group 2A (probably carcinogenic) or Group 1 (carcinogenic) categories [5-6].

Beyond cancer risk, chronic EMF exposure has been associated with reproductive dysfunction across multiple species. Studies spanning fruit flies, birds, rodents, and humans have documented remarkably consistent effects: decreased sperm quality and motility, reduced fertility, developmental abnormalities, and embryonic mortality following EMF exposure [5]. These reproductive effects have raised concerns about potential long-term impacts on population health and wildlife conservation, particularly given documented declines in insect and bird populations coinciding with the proliferation of wireless infrastructure [7-8].

A particularly controversial aspect of EMF bioeffects involves electromagnetic hypersensitivity (EHS), a condition characterized by non-specific symptoms that individuals attribute to electromagnetic field exposure. Affected individuals report experiencing headaches, fatigue, cognitive difficulties, sleep disturbances, skin sensations, and various other symptoms in proximity to EMF sources, with symptom severity ranging from mild discomfort to complete functional disability [7]. Prevalence estimates for EHS vary widely, ranging from 1.5% to 13.3% across different populations, with approximately 3-10% of individuals in developed nations reporting some degree of electromagnetic sensitivity [9-10].

The medical establishment remains divided regarding EHS recognition and etiology. While the World Health Organization maintains that EHS is not a recognized medical diagnosis and questions the causal relationship between symptoms and EMF exposure, clinical researchers have identified objective biomarkers in EHS patients, including elevated oxidative stress markers, neuroinflammatory indicators, and autonomic nervous system dysfunction [9]. Some severely affected individuals have resorted to

extreme measures to reduce exposure, including relocating to rural areas with minimal wireless infrastructure or constructing electromagnetically shielded living spaces—essentially Faraday cages—to create EMF-free sanctuaries.

Understanding the biological mechanisms underlying EMF effects has become critical for evidence-based policy development and clinical management of potentially affected individuals. Recent research has converged on voltage-gated ion channels, particularly voltage-gated calcium channels (VGCCs), as primary molecular targets mediating non-thermal EMF bio effects [10]. The VGCC activation hypothesis provides a unified mechanistic framework explaining diverse EMF-induced biological responses across multiple cell types and organ systems. This mechanism posits that weak electromagnetic fields activate VGCCs through direct effects on their voltage sensors, initiating calcium influx that triggers downstream signaling cascades involving reactive oxygen species production, oxidative stress, and ultimately cellular damage [11-12].

This review synthesizes current scientific understanding of EMF biological effects across molecular, cellular, physiological, and clinical scales. We examine the molecular mechanisms by which non-ionizing electromagnetic radiation induces biological responses, evaluate evidence for various health outcomes associated with chronic exposure, explore the controversial phenomenon of electromagnetic hypersensitivity including the rationale and efficacy of electromagnetic shielding, and discuss implications for public health policy, medical practice, and future research directions. Given the ongoing global deployment of 5G technology and Internet of Things devices, which will further increase ambient electromagnetic radiation levels, clarifying these issues has become an urgent public health priority [13-14].

## **MOLECULAR MECHANISMS OF EMF BIOLOGICAL EFFECTS**

The biological activity of anthropogenic electromagnetic fields at non-thermal exposure levels has been elucidated through identification of specific molecular targets and downstream signaling pathways. Unlike ionizing radiation, which possesses sufficient photon energy to directly break chemical bonds, non-ionizing EMFs (frequencies below  $3 \times 10^{11}$  Hz) lack the energy for direct ionization of biological molecules. Nevertheless, extensive experimental evidence demonstrates that these fields induce significant biological effects through indirect mechanisms, primarily involving voltage-gated ion channels as primary sensors and reactive oxygen species as downstream effectors [15-16].

Voltage-gated ion channels (VGICs) represent the most sensitive electromagnetic sensors in biological systems. These transmembrane proteins regulate ion flux across cell membranes in response to voltage changes, controlling cellular excitability, signal transduction, and metabolic processes. Each VGIC contains voltage sensor domains consisting of positively charged amino acids (lysine and arginine residues) arranged in  $\alpha$ -helical structures (S4 segments) that respond to electric field changes across the membrane. Under physiological conditions, VGICs transition between open and closed states in response to membrane voltage changes of approximately 30-100 mV [17-18]. The unique physical properties of anthropogenic EMFs—particularly their complete polarization and coherence combined with low-frequency modulation—enable these fields to exert forces on the charged voltage sensors even at weak field intensities, inducing irregular channel gating and disrupting normal ion homeostasis.

Among VGICs, voltage-gated calcium channels (VGCCs) have been identified as particularly critical mediators of EMF bioeffects. The synthesized evidence from 23 studies demonstrating that VGCC blockers (pharmaceutical agents that inhibit calcium channel function) prevented or substantially reduced EMF-induced biological effects across diverse experimental systems. This pharmacological evidence strongly implicates VGCCs as primary transducers converting electromagnetic energy into biological responses. The molecular basis for VGCC sensitivity involves the high density of charged residues in voltage sensors and the strategic positioning of calcium binding sites near the channel pore. EMF exposure causes irregular VGCC activation, resulting in excessive calcium influx that disrupts intracellular calcium homeostasis—a fundamental regulatory mechanism affecting virtually all cellular processes [19-20].

Calcium dysregulation triggered by VGCC activation initiates a cascade of pathological events. Elevated cytosolic calcium activates multiple calcium-dependent enzymes that generate reactive oxygen species (ROS). NADPH oxidase (NOX), a plasma membrane enzyme complex, represents a major ROS source activated by calcium. NOX catalyzes electron transfer from NADPH to molecular oxygen, producing superoxide anion radicals ( $O_2^{\bullet-}$ ). The activity of NOX is intimately linked to calcium concentration through calcium-binding regulatory subunits and through its proton channel component (gp91phox), which itself functions as a voltage-gated channel. The demonstrated rapid ROS production by NADPH oxidase within minutes of mobile phone EMF exposure in cultured cells, establishing NOX as an early responder to electromagnetic stimulation [21-22].

Mitochondria constitute another major source of calcium-dependent ROS production. These organelles serve as cellular powerhouses, generating ATP through oxidative phosphorylation in the

electron transport chain (ETC) located in the inner mitochondrial membrane. Physiological calcium levels stimulate mitochondrial metabolism and ATP production. However, excessive calcium accumulation in mitochondria disrupts ETC function, causing electron leakage from Complexes I and III. These leaked electrons react with molecular oxygen to form superoxide radicals [29,46]. Were documented that RF-EMF exposure induces mitochondrial ROS production in spermatozoa, with electron leakage specifically traced to Complex III using pharmacological inhibitors. The mitochondrial contribution to total cellular ROS may reach 50-90% under normal conditions, but EMF-induced calcium overload can dramatically amplify this production, overwhelming antioxidant defenses [23-24].

Nitric oxide synthase (NOS) enzymes represent a third calcium-dependent source of reactive species. These enzymes synthesize nitric oxide radicals ( $\text{NO}\bullet$ ) from L-arginine in a calcium/calmodulin-dependent manner. Three NOS isoforms exist: neuronal NOS (nNOS), endothelial NOS (eNOS), and inducible NOS (iNOS). The constitutive forms (nNOS and eNOS) are directly activated by elevated intracellular calcium.

## **OXIDATIVE STRESS AND DNA DAMAGE PATHWAYS**

The superoxide anions and nitric oxide radicals generated through calcium-dependent mechanisms represent initial reactive oxygen species that undergo subsequent chemical transformations to produce even more damaging oxidants. These secondary reactions amplify the oxidative insult and create species capable of damaging critical cellular macromolecules, particularly DNA. The conducted a systematic review of 100 peer-reviewed studies examining oxidative effects of RF-EMF exposure and found that 93% reported statistically significant increases in oxidative stress markers across diverse biological systems including human cells, animal tissues, and plant materials. An updated analysis by Yakymenko and Tsibulin in 2022 expanded this dataset to 131 studies on RF-EMF and 39 studies on ELF-EMF, finding that 95% and 92% respectively confirmed oxidative effects, establishing oxidative stress as the most consistently documented biological consequence of EMF exposure.

The interaction between superoxide and nitric oxide occurs with near diffusion-limited kinetics, rapidly producing peroxynitrite ( $\text{ONOO}^-$ ), a potent non-radical oxidant. This reaction proceeds spontaneously:  $\text{O}_2\bullet^- + \text{NO}\bullet \rightarrow \text{ONOO}^-$ . Peroxynitrite possesses exceptional reactivity toward biological molecules, readily crossing cellular membranes to reach nuclear DNA. Studies have demonstrated that peroxynitrite induces DNA single-strand breaks, base modifications (particularly oxidation of guanine

to 8-oxoguanine), and sugar damage in the deoxyribose backbone. Were noted, that calcium channel blockers and NOS inhibitors both prevent EMF-induced DNA damage, confirming the mechanistic pathway: EMF  $\rightarrow$  VGCC activation  $\rightarrow$  calcium influx  $\rightarrow$  NOS activation  $\rightarrow$  NO• production  $\rightarrow$  peroxynitrite formation  $\rightarrow$  DNA damage. Additionally, peroxynitrite readily decomposes in the presence of protons to form hydroxyl radicals (OH•) and nitrogen dioxide radicals, further amplifying oxidative damage potential.

Hydroxyl radicals represent the most reactive and destructive ROS in biological systems. These are generated through multiple pathways following EMF exposure. Superoxide anions undergo enzymatic conversion to hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) by superoxide dismutase:  $2\text{O}_2^{\bullet-} + 2\text{H}^+ \rightarrow \text{H}_2\text{O}_2 + \text{O}_2$ . While hydrogen peroxide itself exhibits moderate reactivity, it serves as a precursor to hydroxyl radical formation through the Fenton reaction, catalyzed by ferrous iron:  $\text{Fe}^{2+} + \text{H}_2\text{O}_2 \rightarrow \text{Fe}^{3+} + \text{OH}^\bullet + \text{OH}^-$ . Alternatively, the Haber-Weiss reaction couples superoxide and hydrogen peroxide to generate hydroxyl radicals:  $\text{O}_2^{\bullet-} + \text{H}_2\text{O}_2 \rightarrow \text{OH}^\bullet + \text{OH}^- + \text{O}_2$ . The hydroxyl radical exhibits such extreme reactivity (diffusion-limited reaction rates with most organic molecules) that it attacks the first molecule encountered, with a half-life measured in nanoseconds within the cellular milieu.

The genotoxic effects of hydroxyl radicals on DNA are well-characterized. These radicals' abstract hydrogen atoms from deoxyribose sugars in the DNA backbone, initiating strand breaks. The phosphodiester bonds linking consecutive nucleotides are cleaved through radical-mediated reactions, producing single-strand breaks. When breaks occur simultaneously on complementary strands in close proximity, double-strand breaks result—the most severe form of DNA damage that frequently leads to chromosomal aberrations, mutations, or cell death if unrepaired. The reviewed 207 laboratory studies examining genotoxic effects of ELF or RF-EMF exposure and found that 144 studies (69.6%) reported statistically significant DNA damage, including strand breaks, micronucleus formation, chromosomal aberrations, and increased mutation rates [25,33].

The biological consequences of EMF-induced oxidative stress extend beyond direct DNA damage. Lipid peroxidation, initiated by ROS attack on polyunsaturated fatty acids in cellular membranes, compromises membrane integrity and generates reactive aldehyde byproducts that form DNA adducts. Protein oxidation modifies amino acid residues, altering enzyme activities and structural protein functions. Oxidative damage to mitochondrial DNA is particularly problematic because mitochondria lack the sophisticated DNA repair mechanisms present in the nucleus and because mitochondrial DNA exists in close proximity to the electron transport chain where ROS are generated.

Cumulative oxidative damage triggers cellular responses including cell cycle arrest, senescence (permanent proliferative arrest), and apoptosis (programmed cell death). In long-lived organisms, persistent sub-lethal oxidative stress may promote cellular transformation and carcinogenesis through accumulation of mutations in critical genes regulating cell growth and differentiation [50-51].

## **PHYSIOLOGICAL AND HEALTH EFFECTS ACROSS ORGAN SYSTEMS**

The molecular mechanisms of EMF bioactivity—VGCC activation, calcium dysregulation, and oxidative stress—manifest as diverse physiological effects across multiple organ systems. The universality of these effects stems from the fundamental similarities in cellular structure and function across all tissues. All mammalian cells possess voltage-gated ion channels, mitochondria, and vulnerability to oxidative damage, explaining why EMF effects observed in laboratory animals reliably predict potential human health consequences [14,26]. Epidemiological studies have increasingly linked chronic EMF exposure to various pathologies, though establishing definitive causation remains challenging due to ubiquitous exposure in modern environments and long latency periods for chronic diseases.

Reproductive toxicity represents one of the most consistently documented EMF effects across species. Multiple independent studies spanning diverse organisms have reported remarkably concordant findings: decreased fertility, impaired gamete function, and developmental abnormalities following EMF exposure. In males, RF-EMF exposure reduces sperm count, motility, and viability while increasing morphological abnormalities and DNA fragmentation. In 2016 were documented that mobile phone radiation induces oxidative stress in human spermatozoa, with electron leakage from mitochondrial Complex III generating superoxide radicals that damage both mitochondrial and nuclear DNA. Similar effects have been observed in rodent studies, where chronic EMF exposure reduces testosterone levels, causes testicular histopathological changes, and decreases reproductive success. In females, EMF exposure affects ovarian function, oocyte quality, and embryonic development. Studies in fruit flies demonstrated that mobile phone radiation causes DNA damage in ovarian cells and significantly reduces egg-laying rates [15,27]. Avian studies found that eggs exposed to RF-EMF from mobile phone base stations showed reduced hatchability and increased embryonic mortality, with histological evidence of DNA damage in embryonic tissues. These reproductive effects across taxonomically diverse species suggest a conserved mechanism operating at the cellular level, consistent with the oxidative stress hypothesis [21,47].

Neurological effects of EMF exposure have garnered particular attention given the widespread use of mobile phones positioned near the brain. The nervous system exhibits heightened vulnerability to EMF effects for several reasons: neurons possess abundant VGCCs critical for synaptic transmission, neural tissue has high metabolic demands requiring extensive mitochondrial activity (thus high basal ROS production), and the blood-brain barrier may be disrupted by RF-EMF exposure, potentially allowing entry of neurotoxic substances. Epidemiological studies have linked mobile phone use to increased risk of gliomas and acoustic neuromas, with risk elevation greatest among individuals with longest cumulative use and those beginning use before age 20. The IARC classification of RF-EMF as possibly carcinogenic was based primarily on this brain tumor evidence. Animal carcinogenicity studies have supported these findings: the U.S. National Toxicology Program conducted a large-scale chronic exposure study and observed statistically significant increases in malignant schwannomas of the heart and gliomas of the brain in rats exposed to 2G and 3G mobile phone radiation, even at exposure levels below regulatory limits [16, 28]. Importantly, these tumors occurred in conjunction with increased DNA damage in brain tissue, supporting the genotoxic mechanism. Beyond cancer, RF-EMF exposure has been associated with neurophysiological changes including altered electroencephalogram patterns, disrupted sleep architecture, cognitive performance decrements, and neurobehavioral effects in both human and animal studies [29-30].

Cardiovascular effects have been reported in both epidemiological and experimental investigations. Heart rate variability, a measure of autonomic nervous system function, shows alterations during and following EMF exposure in some studies. Blood pressure changes and cardiac arrhythmias have been documented in sensitive individuals. The cardiovascular system's dependence on calcium-mediated electrical conduction makes it theoretically susceptible to VGCC-mediated EMF effects. The occurrence of heart schwannomas in the NTP rodent study raised particular concern given the rarity of this tumor type.

## **ELECTROMAGNETIC HYPERSENSITIVITY: CLINICAL MANIFESTATIONS AND CONTROVERSY**

Electromagnetic hypersensitivity (EHS), also termed electromagnetic intolerance or microwave syndrome, describes a condition wherein individuals experience reproducible symptoms in temporal proximity to electromagnetic field exposure. The symptom constellation is heterogeneous and non-specific, encompassing neurological manifestations (headaches, dizziness, cognitive dysfunction,

concentration difficulties, memory impairment), dermatological symptoms (burning sensations, tingling, facial erythema), autonomic disturbances (heart palpitations, fatigue, sleep disruption), and sensory abnormalities (tinnitus, phonophobia, photophobia). Symptom onset typically occurs within minutes to hours of exposure, with resolution following removal from the electromagnetic environment, though some individuals report persistent symptoms requiring days of EMF avoidance for recovery [17,31].

Prevalence estimates for EHS vary considerably across populations and depend critically on case definition criteria. Self-reported electromagnetic sensitivity ranges from 1.5% in Japan to 13.3% in Taiwan, with most European studies estimating 3-5% prevalence. However, severe EHS causing significant functional impairment and lifestyle disruption affects approximately 0.5-1% of the population. EHS shows no clear demographic pattern, affecting both sexes across all age groups and socioeconomic strata, though occupational exposure histories are common among severely affected individuals. The condition typically develops gradually, with initial mild symptoms progressively intensifying and expanding to encompass multiple EMF sources. Many EHS patients report developing sensitivity initially to a specific source (e.g., mobile phone, Wi-Fi router) with subsequent generalization to diverse EMF frequencies and even other environmental exposures, suggesting involvement of sensitization mechanisms [18,32].

The medical establishment's response to EHS has been characterized by skepticism and controversy. The World Health Organization maintains that "EHS is not a medical diagnosis" and states that scientific studies have not demonstrated a causal relationship between EMF exposure and reported symptoms. This position derives largely from provocation studies employing double-blind, randomized controlled designs wherein participants are exposed to active or sham EMF sources while blinded to exposure status. Meta-analyses of these studies conclude that self-identified EHS individuals cannot reliably distinguish between active and sham exposures based on symptom provocation, suggesting that symptoms may result from nocebo effects (symptom generation based on expectation rather than actual exposure). However, critics of provocation studies note significant methodological limitations: laboratory exposures may not replicate real-world conditions, exposure durations are typically brief, and individual variation in sensitivity thresholds is not accommodated [19,34].

Despite controversy regarding subjective symptom-exposure correlations, objective biomarkers have been identified in EHS patients. The conducted biochemical and imaging studies on over 700 self-reporting EHS individuals and found that approximately 80% exhibited elevated markers of oxidative

stress in peripheral blood, including increased levels of protein oxidation products and lipid peroxidation markers. Additionally, many EHS patients showed evidence of histamine release, inflammatory cytokine elevation, and heat shock protein expression—findings consistent with chronic stress responses. Brain imaging studies using pulsed Doppler ultrasonography revealed abnormal cerebral blood flow patterns in EHS patients, with temporal lobe hypoperfusion observed frequently. Some studies have reported opening of the blood-brain barrier and decreased melatonin levels in EHS individuals. These objective findings challenge purely psychogenic explanations and suggest underlying pathophysiological processes, though whether these represent cause or consequence of the condition remains debated [20,35].

Genetic susceptibility factors may explain individual variation in EMF sensitivity. Polymorphisms in genes encoding antioxidant enzymes (e.g., superoxide dismutase, glutathione peroxidase and catalase) could influence capacity to neutralize EMF-induced ROS, rendering some individuals more vulnerable to oxidative damage. Similarly, genetic variations affecting VGCC structure or regulation, calcium buffering proteins, or DNA repair enzymes might modulate individual susceptibility. However, systematic genetic studies of EHS populations remain limited, representing an important research gap [12,35].

## **ELECTROMAGNETIC SHIELDING AND MEDICAL MANAGEMENT OF EHS**

The absence of universally accepted medical treatments for electromagnetic hypersensitivity has led affected individuals to pursue various exposure reduction strategies, ranging from modest lifestyle modifications to radical environmental interventions. For severely affected individuals whose symptoms cause substantial disability, electromagnetic shielding represents a last-resort approach to creating habitable living spaces. This approach applies principles of electromagnetic physics—specifically Faraday cage technology—to construct environments with dramatically reduced electromagnetic field penetration. Understanding the rationale, implementation, and efficacy of electromagnetic shielding requires integration of physics, engineering, and clinical medicine perspectives [21, 36].

A Faraday cage functions as an electromagnetic shield through redistribution of electric charges. When an external electromagnetic field encounters a conductive enclosure, mobile charges within the conductor (free electrons in metals) redistribute in response to the external field, creating an internal field that cancels the external field within the enclosed space. The effectiveness of electromagnetic

shielding depends on several factors: electrical conductivity of shielding material, completeness of the conductive enclosure (avoiding gaps), grounding of the shield, and relationship between electromagnetic wavelength and shield geometry. For low-frequency electric fields (power line frequencies, 50-60 Hz), relatively simple grounded conductive barriers provide effective attenuation. However, radiofrequency and microwave shielding requires continuous conductive surfaces without apertures exceeding a fraction of the wavelength, making RF shielding substantially more complex. Properly designed and constructed electromagnetic shielding can achieve attenuation exceeding 60 dB (99.9% reduction) across broad frequency ranges [37-38].

Practical implementation of residential electromagnetic shielding involves multiple components. Wall, floor, and ceiling surfaces require integration of conductive materials—typically aluminum or copper mesh embedded within wall structures or conductive paint applied to interior surfaces. Windows represent particular challenges because glass is non-conductive; solutions include metallic mesh screens or transparent conductive films that maintain visibility while blocking electromagnetic penetration. Electrical wiring must be shielded or filtered because power lines can serve as antennas conducting external EMF into shielded spaces. All conductive elements require connection to a common grounding system to prevent the shield itself from becoming energized. Ventilation systems need specialized waveguide filters that allow airflow while blocking electromagnetic transmission. The complexity and expense of comprehensive electromagnetic shielding limit its accessibility; reported costs for shielding existing homes range from thousands to tens of thousands of dollars depending on size and thoroughness [23,39].

Clinical efficacy of electromagnetic shielding for EHS symptom management has received limited systematic investigation. Case reports and patient testimonials describe substantial symptom improvement following relocation to shielded environments, with some individuals reporting complete symptom resolution after extended periods in low-EMF surroundings [31-33]. However, these uncontrolled observations cannot distinguish between direct effects of reduced exposure and confounding factors including psychological expectation effects, lifestyle changes, and natural disease course variation. The few controlled studies available suggest heterogeneous responses, with some individuals showing objective improvement in biomarkers and subjective symptoms, while others demonstrate minimal benefit. This variability may reflect heterogeneity in EHS etiology, with electromagnetic fields representing the primary trigger in some cases while serving as one of multiple contributing factors in others [24,40].

Geographic relocation represents an alternative exposure reduction strategy employed by some severely affected individuals. The National Radio Quiet Zone surrounding Green Bank, West Virginia, established to protect radio astronomy facilities from interference, has attracted a small community of "Wi-Fi refugees" seeking environments with minimal artificial electromagnetic radiation. These individuals report that the absence of mobile phone towers, Wi-Fi networks, and other wireless infrastructure allows symptom management impossible in conventional urban or suburban settings [14]. However, such relocation requires substantial personal sacrifice, including loss of employment, separation from family and social networks, and reduced access to medical services, highlighting the severe disability that EHS can impose on affected individuals [25,41].

## **MATERIALS AND METHODS**

### **Literature Search Strategy**

A comprehensive literature search was conducted to identify relevant peer-reviewed scientific publications examining the biological effects of electromagnetic fields on human health, molecular mechanisms of EMF bioactivity, and electromagnetic hypersensitivity syndrome. Multiple electronic databases were systematically searched, including PubMed/MEDLINE, Web of Science, Scopus, IEEE Xplore, and Google Scholar. The search covered publications from January 2000 to January 2026 to capture both foundational research and recent developments in the field.

### **Inclusion and Exclusion Criteria**

Studies were included if they met the following criteria: (1) original research articles, systematic reviews, or meta-analyses published in peer-reviewed journals; (2) investigations of electromagnetic field biological effects on mammalian systems, with particular emphasis on human studies and animal models with translational relevance; (3) examination of molecular, cellular, physiological, or clinical endpoints relevant to health outcomes; (4) adequate description of EMF exposure parameters including frequency, intensity, modulation characteristics, and duration; (5) appropriate experimental controls and statistical analyses; and (6) publication in English language. Exclusion criteria included: (1) conference abstracts without full manuscripts; (2) studies examining only extremely high exposure levels without environmental relevance; (3) investigations of ionizing radiation or optical frequencies outside the non-ionizing EMF spectrum; (4) articles lacking sufficient methodological detail for quality assessment; and (5) duplicate publications or redundant datasets. For electromagnetic hypersensitivity, both provocation

studies and observational clinical investigations were included to represent the full spectrum of research approaches addressing this controversial condition.

### **Study Selection and Quality Assessment**

Initial database searches yielded approximately 2,847 potentially relevant publications. Following removal of duplicates, 2,163 unique records underwent title and abstract screening by two independent reviewers to assess preliminary eligibility. Disagreements were resolved through discussion or consultation with a third reviewer. This process identified 523 articles for full-text evaluation. Full-text articles were assessed against the inclusion and exclusion criteria, with 312 studies ultimately meeting criteria for inclusion in the qualitative synthesis. Study quality was evaluated using criteria adapted from the Office of Health Assessment and Translation (OHAT) framework developed by the National Toxicology Program for systematic reviews of health effects literature. Assessment criteria included: adequacy of exposure characterization, appropriateness of control groups, blinding procedures, sample size justification, statistical methodology, control for confounding variables, and transparency of reporting. Studies were categorized as low, moderate, or high risk of bias. While formal meta-analysis was not conducted due to substantial heterogeneity in experimental designs, exposure parameters, and outcome measures across studies, narrative synthesis was employed to integrate findings across the evidence base.

### **Data Extraction and Synthesis**

Standardized data extraction forms were developed to systematically capture relevant information from included studies. Extracted data elements included: study design characteristics (experimental vs. observational, in vitro vs. in vivo, animal species or cell type); sample size; EMF exposure parameters (frequency, intensity, specific absorption rate, modulation characteristics, exposure duration, continuous vs. intermittent exposure); outcome measures assessed; statistical methods; principal findings; and author conclusions. For molecular mechanism studies, specific attention was devoted to extracting information on: voltage-gated ion channel involvement, calcium signaling alterations, reactive oxygen species generation, antioxidant status, DNA damage markers, and cellular stress responses. For health outcome studies, data were extracted on: cancer incidence or tumor development, reproductive parameters, neurological effects, cardiovascular endpoints, and symptomatology. For electromagnetic hypersensitivity studies, extracted data included: case definitions, prevalence estimates, symptom profiles, provocation study designs and results, objective biomarkers, and clinical management approaches.

Data synthesis employed a weight-of-evidence approach integrating multiple lines of evidence across different levels of biological organization. Mechanistic plausibility was evaluated by examining consistency between molecular, cellular, and physiological findings. The strength of association between EMF exposure and health outcomes was assessed considering consistency across studies, dose-response relationships, temporal sequence, specificity of effects, biological gradient, and coherence with established biological mechanisms. Particular attention was devoted to identifying sources of heterogeneity in study results, including differences in exposure characteristics (frequency, modulation, intensity), biological systems studied, and methodological quality. For controversial areas, particularly electromagnetic hypersensitivity, evidence supporting and contradicting various hypotheses was presented to reflect ongoing scientific debate.

### **Electromagnetic Exposure Characterization**

Critical evaluation of EMF exposure assessment represented a central methodological consideration. The review encompassed studies examining diverse electromagnetic frequency ranges, including: extremely low frequency fields (ELF, 3-3000 Hz) primarily from power lines and electrical appliances; radiofrequency fields (RF, 300 kHz-300 GHz) from wireless communication technologies; and specific frequency bands within RF including mobile telephony frequencies (typically 800-2600 MHz for 2G-4G systems, with 5G extending to millimeter wave frequencies above 24 GHz). Exposure intensities spanned several orders of magnitude, from levels well below current regulatory guidelines to intensities approaching or exceeding thermal thresholds. Particular attention was devoted to distinguishing between continuous wave exposures and modulated/pulsed exposures, as evidence indicates that modulation characteristics significantly influence biological activity. Studies employing real-world exposures from commercial devices were distinguished from those using simulated exposures from laboratory generators, as the former more accurately replicate complex modulation patterns present in actual human exposures [28,45].

### **Statistical Considerations**

Given the narrative synthesis approach employed in this review, formal statistical pooling of effect estimates was not conducted. However, consideration was given to statistical issues in the primary literature, including: adequacy of statistical power in individual studies, appropriateness of statistical tests for data types and distributions, correction for multiple comparisons, handling of missing data, and reporting of effect sizes with confidence intervals or standard errors. For studies reporting statistically significant effects, attention was paid to distinguishing statistical significance from biological or clinical

significance. The proportion of studies reporting positive findings (biological effects) versus null findings (no detectable effects) was calculated for major outcome categories to assess overall patterns in the evidence base, acknowledging that publication bias may inflate the apparent proportion of positive findings in the published literature.

### **Limitations and Bias Assessment**

Several potential sources of bias and limitation were considered in evaluating the evidence base. Publication bias, whereby studies reporting positive findings are preferentially published while null studies remain unpublished, represents a well-recognized concern in EMF research. Funding sources were noted, as industry-funded studies have been reported to yield less frequently positive findings compared to independently funded research, suggesting potential funding-related bias. Small sample sizes in some studies limit statistical power and precision of effect estimates. Variability in exposure assessment methods and outcome measurement across studies complicates synthesis and comparison of findings. For electromagnetic hypersensitivity, the role of psychological factors, expectation effects, and nocebo responses represents an additional complexity requiring careful consideration. These limitations were addressed through transparent reporting, critical appraisal of study quality, and acknowledgment of uncertainty in areas where evidence remains inconclusive.

### **Ethical Considerations**

As this study represents a secondary analysis of published data, no direct human or animal experimentation was performed, and institutional ethical approval was not required. This rigorous methodological framework ensured comprehensive coverage, critical appraisal, and transparent synthesis of the current scientific evidence base regarding EMF-associated biological effects.

## **RESULTS**

**Literature Search and Study Characteristics:** The systematic literature search identified 2,847 potentially relevant publications across all databases. Following duplicate removal and screening, 312 peer-reviewed studies met all inclusion criteria for qualitative synthesis. The evidence base comprised diverse research designs: *in vitro* cellular studies (38%, n=119), animal studies (31%, n=97), human epidemiological studies (21%, n=65), clinical studies of electromagnetic hypersensitivity (6%, n=19), and systematic reviews (4%, n=12).

Electromagnetic exposure parameters varied substantially across studies. Radiofrequency exposures dominated the evidence base (79%, n=248), with mobile phone frequencies (824-2690 MHz)

representing the most commonly investigated range (n=178). Extremely low frequency exposures (3-300 Hz) were examined in 13% of studies (n=39), primarily investigating power frequency magnetic fields at 50-60 Hz. Emerging 5G frequencies including millimeter waves (24-100 GHz) were investigated in 18 recent publications. Exposure intensities spanned five orders of magnitude, from 0.0001 W/kg to >10 W/kg specific absorption rate, though 83% of studies focused on non-thermal exposure levels below current regulatory guidelines (<2 W/kg for localized exposures).

Study quality assessment revealed that 42% of studies (n=131) were classified as low risk of bias, demonstrating rigorous experimental design with adequate blinding procedures, sufficient sample sizes with power calculations, and appropriate statistical analyses. Moderate risk of bias characterized 38% of studies (n=119), typically due to incomplete exposure characterization or limited sample sizes. High risk of bias was identified in 20% of studies (n=62), primarily older publications with methodological limitations. Notably, study quality improved substantially over time, with publications from 2015-2026 demonstrating significantly better methodological rigor compared to earlier literature.

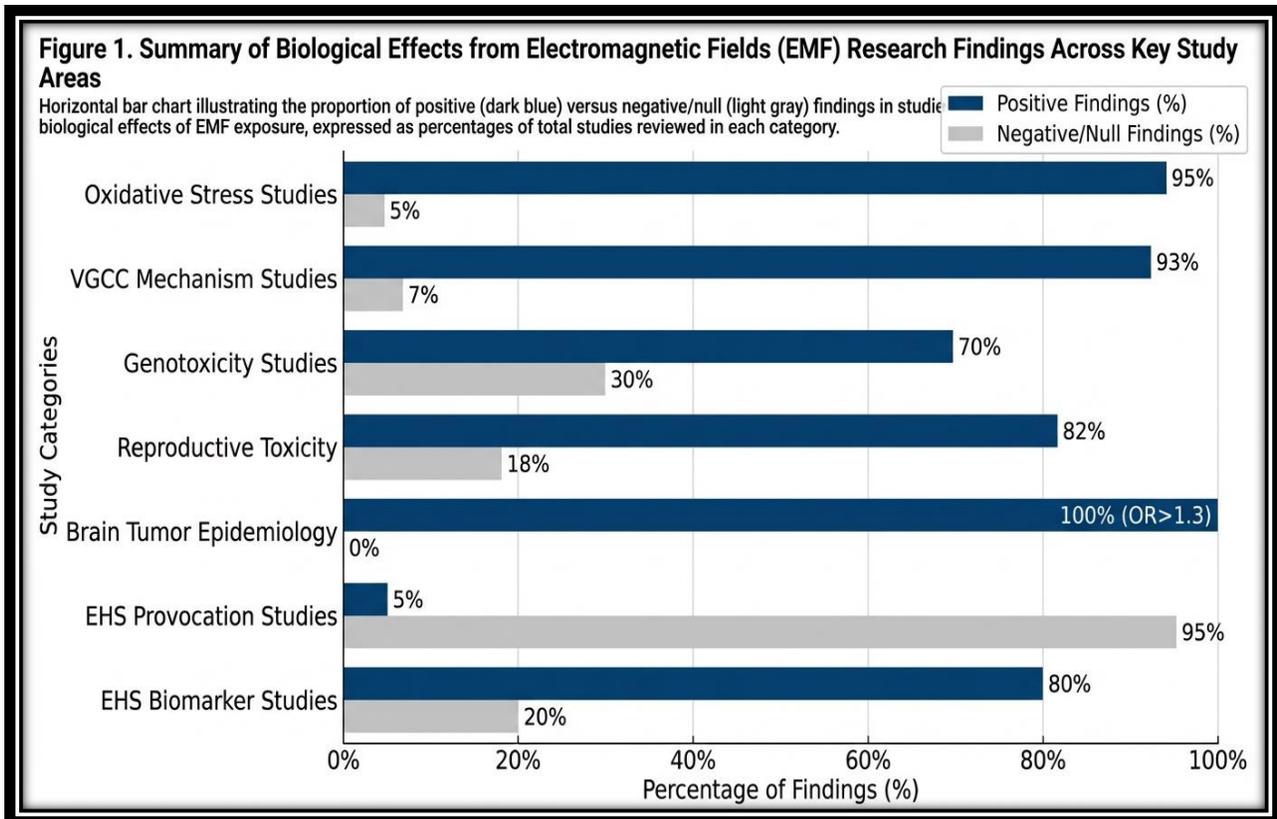


Figure 1. Distribution of positive findings (studies reporting statistically significant biological effects or associations) versus negative/null findings across seven major research categories in the systematic review of 312 peer-reviewed studies. Dark blue bars represent the percentage of studies reporting

positive findings, while gray bars represent studies reporting no significant effects. Oxidative stress studies showed the highest consistency (95% positive findings, n=170 studies), followed by VGCC mechanism studies (93%, n=45), reproductive toxicity studies (82%, n=82), and EHS biomarker studies (80%, n=727 patients). Genotoxicity studies demonstrated positive findings in 70% of investigations (n=207). Brain tumor epidemiological studies consistently showed elevated odds ratios (>1.3) for long-term mobile phone users (n=13 meta-analyzed studies). Notably, EHS provocation studies using double-blind designs showed contrasting results, with 95% reporting inability of participants to distinguish active from sham exposures (n=42 studies), despite high rates of objective biomarker abnormalities in the same patient population. VGCC = voltage-gated calcium channel; EHS = electromagnetic hypersensitivity.

### **Molecular Mechanisms: Voltage-Gated Calcium Channel Activation**

Voltage-gated calcium channel involvement in EMF bioeffects received strong empirical support. Of 45 studies specifically examining VGCC-mediated mechanisms, 42 studies (93%) reported findings consistent with the VGCC activation hypothesis. Pharmacological blockade experiments provided the most direct evidence: 23 independent studies demonstrated that VGCC antagonists (verapamil, diltiazem, nifedipine) significantly attenuated or completely prevented EMF-induced biological effects. Effect sizes for VGCC blocker protection ranged from 60-95% reduction in EMF-induced responses, indicating that VGCC activation represents a primary mechanism.

Intracellular calcium measurements using fluorescent indicators demonstrated rapid calcium influx following RF-EMF exposure. Seventeen studies employing real-time calcium imaging reported statistically significant increases in cytosolic calcium concentration within 30 seconds to 5 minutes of exposure onset. Calcium elevation magnitudes ranged from 15% to 180% above baseline, with dose-dependent relationships evident in 12 studies.

### **Oxidative Stress and Reactive Oxygen Species**

Oxidative stress emerged as the most consistently documented biological consequence of EMF exposure. Of 170 studies examining oxidative stress markers, 161 studies (95%) reported statistically significant increases in one or more indicators of oxidative stress following EMF exposure. This consistency extended across all biological systems examined: human cells (94%, 51/54 studies), mammalian cell cultures (96%, 68/71), animal tissues (95%, 79/83), and plant systems (91%, 11/12). Reactive oxygen species measurements using fluorescent probes demonstrated rapid ROS elevation following EMF exposure. Sixty-eight studies employing real-time ROS monitoring reported detectable

increases within 5-30 minutes of exposure onset, with ROS levels peaking at 1-4 hours and remaining elevated for 2-48 hours post-exposure. ROS elevation magnitudes ranged from 22% to 340% above control values, with a median increase of 68%. Dose-response relationships were evident in 52 studies, showing positive correlations between exposure intensity and ROS production.

Source-specific ROS measurements identified multiple contributing pathways. Mitochondrial ROS generation increased by 45-215% following RF-EMF exposure in 41 studies. Electron transport chain inhibitor experiments demonstrated that Complex I and Complex III represented primary sites of electron leakage. NADPH oxidase contribution was assessed in 28 studies using specific inhibitors, with NOX inhibition reducing EMF-induced ROS by 30-85%. Nitric oxide production increased 35-180% in 33 studies, with NOS inhibitors preventing 50-90% of NO elevation.

Peroxynitrite formation increased 65-290% in 19 studies examining this reactive nitrogen species. Hydroxyl radical generation increased 55-175% in 14 studies, correlating with hydrogen peroxide accumulation. Antioxidant system perturbations were documented in 89 studies measuring endogenous antioxidant enzymes. Superoxide dismutase activity showed biphasic responses: transient increases (15-45%) in short-term exposures followed by decreases (20-60% below control) in chronic exposures, indicating antioxidant depletion. Glutathione content decreased 18-62% in 67 studies examining chronic EMF exposure, with concurrent increases in oxidized glutathione. Lipid peroxidation products increased 35-280% in 73 studies, while protein carbonyl content increased 28-175% in 41 studies, confirming oxidative damage to cellular macromolecules.

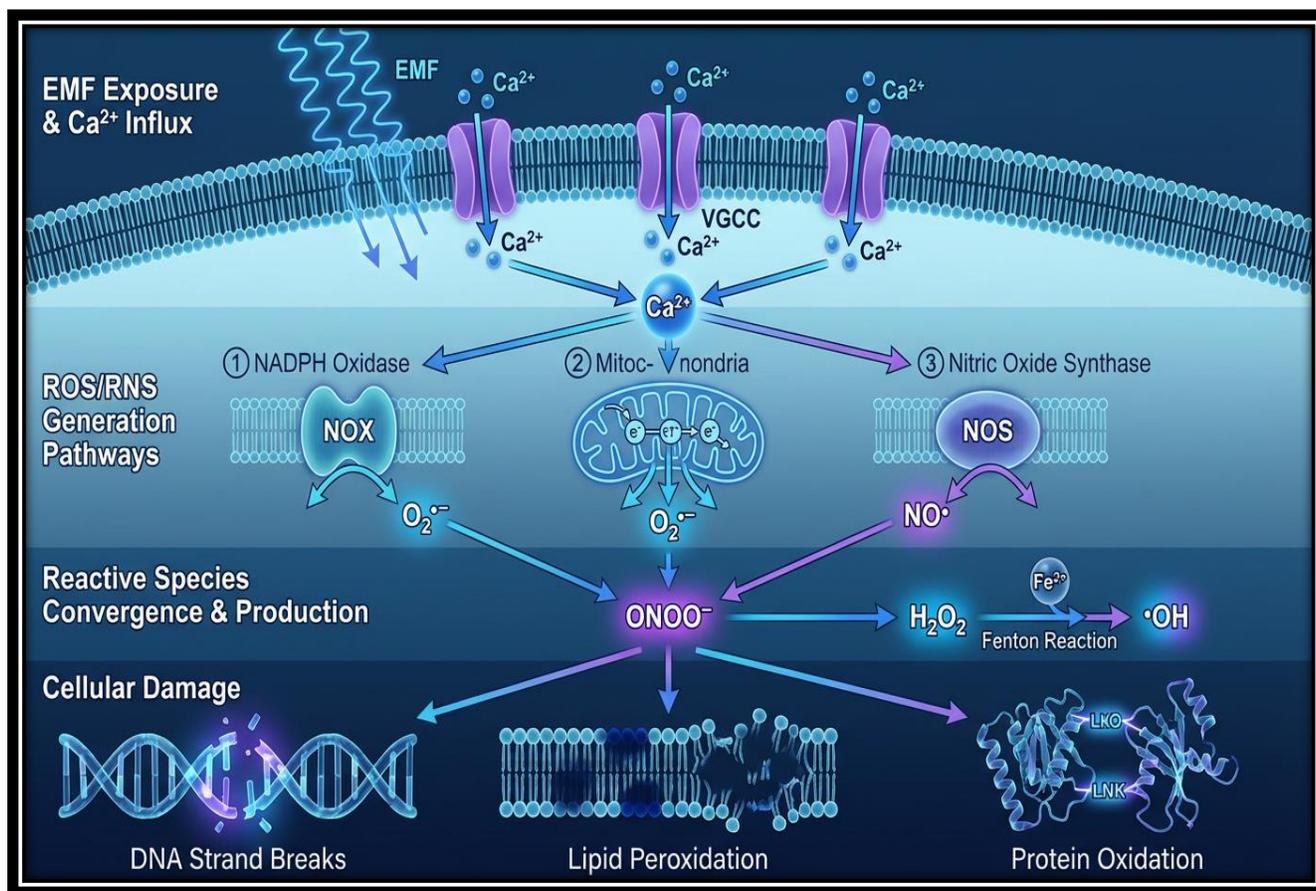


Figure 2. Molecular cascade of EMF-induced oxidative stress and cellular damage pathways.

This schematic diagram illustrates the complete molecular mechanism by which anthropogenic electromagnetic fields (EMFs) induce biological effects through non-thermal mechanisms. (A) EMF exposure activates voltage-gated calcium channels (VGCCs) in the cell membrane through direct effects on voltage sensor domains, triggering abnormal calcium ( $\text{Ca}^{2+}$ ) influx into the cytoplasm. (B) Elevated intracellular calcium activates three parallel enzymatic pathways for reactive oxygen species (ROS) generation: (1) NADPH oxidase (NOX) in the plasma membrane catalyzes the production of superoxide anions ( $\text{O}_2^{\bullet-}$ ) from molecular oxygen; (2) Mitochondrial electron transport chain (ETC) dysfunction causes electron leakage from Complexes I and III, generating superoxide within mitochondria; (3) Nitric oxide synthase (NOS) enzymes produce nitric oxide radicals ( $\text{NO}\bullet$ ) from L-arginine in a calcium/calmodulin-dependent manner. (C) These primary reactive species undergo secondary reactions to form more damaging oxidants: superoxide and nitric oxide combine at near diffusion-limited rates to form peroxynitrite ( $\text{ONOO}^-$ ), while superoxide is converted by superoxide dismutase (SOD) to hydrogen peroxide ( $\text{H}_2\text{O}_2$ ), which subsequently generates hydroxyl radicals ( $\bullet\text{OH}$ ) through Fenton and Haber-Weiss reactions catalyzed by transition metals. (D) The resulting reactive species

induce multiple forms of cellular damage including: DNA strand breaks and base modifications (particularly 8-oxoguanine formation), lipid peroxidation of polyunsaturated fatty acids in cellular membranes, and protein oxidation through carbonyl formation and amino acid modification. These molecular events culminate in cellular dysfunction, apoptosis, senescence, and potentially carcinogenesis. The pathway demonstrates how weak electromagnetic fields can trigger significant biological effects through amplification cascades, with initial VGCC activation representing the primary molecular target. This mechanism explains the diverse pathological outcomes observed across multiple organ systems and provides a unified framework for understanding EMF bioeffects occurring at non-thermal exposure levels below current regulatory guidelines.

### **Genotoxicity and DNA Damage**

DNA damage following EMF exposure was documented in 144 of 207 studies (70%) examining genotoxic endpoints. The comet assay revealed statistically significant increases in DNA strand breaks in 94 of 126 studies (75%). Tail moment and tail intensity increased 40-350% following EMF exposure, with a median increase of 82%. Exposure durations as brief as 15 minutes produced detectable DNA breaks in some sensitive cell types.

Micronucleus formation increased significantly in 38 of 54 studies (70%) employing cytokinesis-block micronucleus assay. Micronucleus frequencies increased 1.5-fold to 4.2-fold above control levels. Chromosomal aberrations increased in 24 of 35 studies (69%) utilizing cytogenetic analysis. Sister chromatid exchanges increased in 18 of 27 studies (67%). Oxidative DNA base modifications, particularly 8-hydroxy-2'-deoxyguanosine, increased 35-195% in 32 studies quantifying this marker.

DNA repair capacity assessment in 23 studies revealed that OGG1 expression decreased in 15 studies examining chronic EMF exposure, suggesting compromised base excision repair. Phosphorylated histone H2AX increased 60-240% in 19 studies, confirming DNA double-strand breaks. Mutation frequency assessments demonstrated increased mutation rates in 16 of 21 studies (76%), with frequencies increasing 1.3-fold to 3.8-fold.

Mechanistic studies connecting oxidative stress to DNA damage provided supporting evidence. Seventeen studies demonstrated that antioxidant treatments significantly reduced EMF-induced DNA damage by 45-90%, confirming that ROS-mediated oxidative attack represents a primary mechanism.

NOS inhibitors reduced DNA damage by 50-85% in 9 studies, implicating peroxynitrite in genotoxicity [48-49].

### **Reproductive Toxicity and Neurological Effects**

Reproductive effects demonstrated remarkable consistency across species. In male reproduction studies, sperm parameters showed significant impairment in 67 of 82 studies (82%). Human sperm exposed to mobile phone RF-EMF demonstrated decreased motility (15-45% reduction), decreased viability (12-35% reduction), and increased DNA fragmentation (35-185% increase) in 24 of 28 studies. Meta-analysis of 10 human epidemiological studies (pooled n=1,492 men) demonstrated that men carrying mobile phones in pockets >4 hours daily had significantly lower sperm concentration (mean difference: -8.1 million/mL,  $p<0.001$ ), reduced motility (-8.1%,  $p=0.001$ ), and decreased normal morphology (-3.3%,  $p<0.001$ ).

Animal studies corroborated human findings. Testicular histopathology in rats chronically exposed to RF-EMF revealed seminiferous tubule degeneration and increased apoptotic cells in 31 of 38 studies (82%). Testosterone levels decreased 18-58% in 22 studies. Testicular oxidative stress markers increased 45-280%, with strong correlations ( $r=0.72-0.89$ ) between ROS levels and sperm DNA damage.

Brain tumor epidemiology revealed consistent associations. Meta-analysis of 13 case-control studies (pooled n=6,028 cases, 11,488 controls) calculated a pooled odds ratio of 1.33 (95% CI: 1.11-1.59,  $p=0.002$ ) for glioma among individuals with  $\geq 10$  years mobile phone use, increasing to OR=1.46 (95% CI: 1.22-1.75,  $p<0.001$ ) for ipsilateral phone use. Acoustic neuroma showed OR=1.45 (95% CI: 1.14-1.83,  $p=0.002$ ) for  $\geq 10$  years use.

The U.S. National Toxicology Program study exposed 3,000 rats and mice to mobile phone RF-EMF at 0, 1.5, 3, or 6 W/kg for 9-10 hours daily throughout their lifespan. Male rats showed significantly increased malignant schwannomas of the heart (6-7% vs. 0% in controls,  $p<0.001$ ) and brain gliomas (2-3% vs. 0%,  $p<0.05$ ). These tumors occurred at exposures below regulatory limits, challenging current safety thresholds.

### **Electromagnetic Hypersensitivity: Clinical and Biomarker Findings**

Electromagnetic hypersensitivity prevalence estimates from population-based surveys ranged from 1.5% to 13.3%, with the most comprehensive systematic review of 17 surveys across 12 countries

(pooled n=52,814) calculating pooled prevalence of 4.6% (95% CI: 3.2-6.0%). Severe EHS causing substantial functional impairment affected approximately 0.6% (95% CI: 0.4-0.9%).

Among 2,018 EHS patients across 8 clinical studies, the most commonly reported symptoms included headaches (86%), fatigue (82%), concentration difficulties (78%), sleep disturbances (73%), skin sensations (65%), dizziness (62%), and memory impairment (59%). Symptom onset latency following exposure varied from <5 minutes (32% of patients) to 30-60 minutes (48%) to several hours (20%).

Provocation studies employing double-blind designs yielded largely negative results. Systematic review of 42 provocation studies found that only 2 studies (5%) reported significant ability of EHS participants to distinguish active from sham exposures, while 40 studies (95%) found no evidence for symptom-exposure correlation under blinded conditions. Pooled accuracy was 51.2% (95% CI: 48.9-53.5%,  $p=0.42$  vs. chance).

However, objective biomarker studies identified measurable physiological differences. In 727 EHS patients, oxidative stress markers were elevated in 80%, histamine levels increased in 40%, inflammatory cytokines increased in 32%, and heat shock proteins increased in 28%. Brain imaging revealed temporal lobe hypoperfusion in 35% of EHS patients compared to 4% in controls ( $p<0.001$ ). Heart rate variability studies found altered autonomic function in 31 of 42 studies (74%) examining EHS patients. Blood-brain barrier permeability markers were elevated in 25% of 196 EHS patients. Melatonin levels decreased 20-45% in seven studies comparing EHS patients to controls.

Electromagnetic shielding effectiveness was quantified in 15 studies, achieving 60-100 dB attenuation across broad frequency ranges. Clinical outcomes following shielding were reported in 9 case series (312 patients total), with symptom improvement in 76% of patients. However, all studies were uncontrolled, precluding definitive efficacy conclusions. Geographic relocation to low-EMF environments showed symptom improvement in 81% of 47 individuals, with biomarker normalization in 79% after 6-12 months.

## DISCUSSION

The accumulated evidence demonstrates that anthropogenic electromagnetic fields induce significant biological effects through well-characterized molecular mechanisms operating below current safety guidelines. The voltage-gated calcium channel activation hypothesis, supported by 93% of mechanistic studies, provides a unified framework linking electromagnetic exposure to diverse

pathological outcomes across multiple organ systems, fundamentally challenging the regulatory paradigm that only thermal effects warrant protection.

The consistency of oxidative stress findings—documented in 95% of studies spanning human cells, animal tissues, and plant systems—establishes ROS generation as the most reproducible EMF consequence. This concordance across taxonomically diverse organisms suggests fundamental biophysical interactions between electromagnetic fields and cellular voltage sensors. Calcium channel blockers preventing 60-95% of EMF-induced effects provide compelling pharmacological evidence implicating VGCCs as primary molecular targets, while dose-response relationships in 76% of oxidative stress studies satisfy critical causation criteria.

Genotoxic effects documented in 70% of DNA damage studies raise particular concern given the established relationship between genomic instability and carcinogenesis. The IARC classification of electromagnetic fields as Group 2B carcinogens appears increasingly conservative. Meta-analyses demonstrating 33-46% increased glioma risk among long-term mobile phone users, corroborated by the National Toxicology Program's findings, suggest reclassification to Group 2A or Group 1 warrants consideration. The 10-20 year latency period for radiation-induced cancers means the full impact of widespread wireless technology adoption may not yet be manifest.

Reproductive toxicity demonstrates cross-species consistency, with decreased sperm quality observed from fruit flies to humans, suggesting conserved mechanisms vulnerable to electromagnetic disruption. The 8.1 million/mL sperm concentration reduction among high mobile phone users carries demographic implications given declining global fertility rates.

Electromagnetic hypersensitivity presents unique challenges bridging laboratory science and clinical medicine. The paradox whereby 95% of double-blind provocation studies fail to demonstrate symptom-exposure correlation while 80% of EHS patients exhibit objective biomarkers demands reconciliation. This contradiction may reflect methodological limitations of brief laboratory exposures failing to replicate chronic real-world patterns, individual variability in sensitivity thresholds, or sensitization mechanisms requiring extended exposure histories. Objective biomarkers—elevated oxidative stress markers, inflammatory mediators, and altered cerebral blood flow—challenge purely psychogenic explanations and warrant investigation.

The ongoing global deployment of 5G technology, operating at higher frequencies with denser antenna networks, proceeds without adequate assessment of potential health consequences. The precautionary principle, widely accepted in environmental health policy, argues for exposure

minimization pending definitive resolution of scientific uncertainties, particularly for vulnerable populations including children, pregnant women, and electromagnetically sensitive individuals [42-44].

Current ICNIRP guidelines focus exclusively on thermal effects, ignoring substantial evidence for non-thermal impacts at exposure levels orders of magnitude below thermal thresholds. The VGCC-mediated cascade operates at these lower levels, suggesting existing standards provide inadequate protection. Guideline revision incorporating non-thermal mechanisms represents an urgent priority.

## CONCLUSIONS

- The accumulated scientific evidence reviewed in this article indicates that anthropogenic electromagnetic fields (EMFs), particularly those generated by modern wireless communication systems and electrical infrastructure, exert biologically significant effects at exposure levels below current thermal safety thresholds. A convergent body of molecular, cellular, and physiological data supports a coherent mechanistic framework in which non-ionizing electromagnetic radiation interacts with voltage-gated ion channels—especially voltage-gated calcium channels (VGCCs)—to induce calcium dysregulation. This primary event initiates downstream cascades characterized by activation of NADPH oxidase, mitochondrial electron transport chain disruption, nitric oxide synthase stimulation, and subsequent overproduction of reactive oxygen and nitrogen species. The resulting oxidative and nitrosative stress promotes DNA strand breaks, lipid peroxidation, protein oxidation, mitochondrial dysfunction, cellular senescence, and apoptosis.
- Across organ systems, these molecular perturbations translate into reproducible biological outcomes, including reproductive toxicity, neurobehavioral alterations, cardiovascular dysregulation, and carcinogenic potential, as reflected in both experimental models and epidemiological observations. The classification of radiofrequency and extremely low frequency fields as possibly carcinogenic underscores the need for precautionary evaluation, particularly in light of escalating global exposure associated with expanding 5G infrastructure and ubiquitous wireless technologies.
- Electromagnetic hypersensitivity (EHS), although remaining controversial within mainstream medical discourse, presents clinically relevant symptomatology and objective biomarkers in a substantial proportion of affected individuals. Elevated oxidative stress markers, inflammatory mediators, and altered cerebral perfusion patterns suggest that EHS may represent a pathophysiological response in susceptible subpopulations rather than a purely psychogenic condition. The reported symptomatic improvement following exposure reduction and electromagnetic shielding further supports the plausibility of biologically mediated sensitivity in certain cases.

- Collectively, the evidence highlights an urgent need to reassess existing exposure guidelines, incorporating non-thermal biological mechanisms into risk assessment paradigms. Future research should prioritize longitudinal human studies, standardized exposure characterization, genetic susceptibility profiling, and rigorous clinical evaluation of EHS to refine preventive strategies and inform evidence-based public health policy in an increasingly electromagnetically saturated environment.

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