

# Effects of Mobile Phone Radiofrequency Electromagnetic Fields on Protein Folding: Evidence and Mechanistic Considerations

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**Abstract**— Mobile phones and wireless devices emit radiofrequency EMFs (RF-EMFs) at 0.8-5.8 gigahertz (GHz). Concerns have been raised about the biological effects of EMF radiation on biological cells and organisms. Protein folding is the basis of most cell functions, and relies on the thermodynamic stability conferred by hydrogen bonds, hydrophobicity, and electrostatics. This review summarizes biophysical, proteomic, animal, and human evidence to assess whether RF-EMF at exposure levels within the range internationally recommended for RF-EMF could induce protein folding disruption, heat shock response and/or oxidative stress that could alter proteostasis. Taken together, the evidence does not support a causal link between RF-EMF exposure at levels that conform with international safety standards, and protein misfolding disease effects in vivo. There is evidence for stress pathways involving Hsp27 and p38 MAPK in vitro, and consistent evidence of RF-induced reactive oxygen species generation in animals and cells, but no demonstration that this would lead to protein misfolding disease in humans. More controlled studies with standardized exposure conditions are needed to resolve the remaining uncertainties in the field.

**Keywords**— Radiofrequency electromagnetic fields, protein folding, heat shock proteins, oxidative stress, mobile phone radiation, specific absorption rate (SAR).

## I. INTRODUCTION AND BACKGROUND

Modern wireless communication uses electromagnetic fields in the radio frequency (RF-EMF) portion of the EM spectrum, with frequencies from 700 MHz (4G/LTE) to 5.8 GHz (Wi-Fi 6E). RF-EMF is non-ionizing radiation, as opposed to ultraviolet radiation and X-ray radiation, and lacks energy per photon sufficient to remove electrons from atoms or break molecular bonds. The main biological effect of RF-EMF at levels close to those from personal devices is mild local heating from dielectric relaxation and ionic conduction [1–3].

In cell biology, protein folding is the process by which a nascent polypeptide folds into its three-dimensional structure in a co- and post-translational manner, driven by a lowering of the Gibbs free energy via hydrophobic collapse, hydrogen bonds, electrostatic interactions, and van der Waals interactions. Therefore, alterations of the thermal and/or ionic conditions can

affect protein conformational equilibrium, leading to protein aggregation or misfolding. The redox state of the cells, temperature, and ion concentrations can be influenced by RF-EMF, indicating that a study of the effect of RF-EMF from wireless technologies on protein folding is mechanistically sound and should be considered [4–6].

## II. REVIEW

### Search methodology

This is a narrative review. PubMed/MEDLINE, Scopus, and Web of Science Core Collection were searched from the start of each database through January 2026 using keyword searches with combinations of the following terms, combined with Boolean operators: "radiofrequency", "electromagnetic field", "RF-EMF", "mobile phone", "microwave", "specific absorption rate", "protein folding", "protein conformation", "heat shock protein", "HSF1", "proteostasis", "proteome", and "oxidative stress". ICNIRP, IARC, WHO and ARPANSA reports were hand searched, as were the reference lists of identified records. Eligible records were peer-reviewed primary studies and SRs, as well as authoritative agency reports, written in English and containing outcomes for protein structure, proteomics, chaperone, oxidative stress, or other human health outcomes following RF-EMF exposure. Studies on extremely low frequency fields, static magnetic fields, and ionizing radiation were excluded. Conference abstracts were excluded if full text could not be obtained. Selection was by consensus and no risk of bias assessment tool or statistical pooling (meta-analysis) was used. Accordingly, this synthesis is, by nature, more descriptive than systematic and must be interpreted accordingly.

## III. PHYSICAL BASIS: HOW RF-EMF INTERACTS WITH BIOLOGICAL MATTERS

The SAR in units of W/kg gives the rate of energy absorption per unit body mass. Since a 1998 publication from the International Commission on Non-Ionizing Radiation

Protection (ICNIRP) the maximum allowed exposure of members of the general public to personal devices is 2 W/kg averaged over 10 grams of tissue mass. Below this limit the temperature increases due to RF energy absorption in tissues can be dissipated without undue thermoregulatory strain [1,2,7].

At the molecular level, the RF-EMF in the GHz range interacts with water molecules mainly through the dipole rotation mechanism, converting field energy into atomic thermal kinetic energy. These interactions can be categorized into thermal effects, non-thermal stress responses, and oxidative mechanisms (Figure 1).

The situation regarding proteins is more complex. Permanent dipole moments and polar side chains experience systematic interactions with these oscillating electric fields. The conformational dynamics of proteins are influenced by them as shown for instance by molecular dynamics simulations of small proteins such as chignolin, which showed diversification in fold-state populations and transition-state rates. These effects were achieved at field intensities far greater than those encountered in humans, and the authors of the study stated that there was a large difference between their simulation and real RF exposure levels[8–11].

#### IV. DIRECT EFFECTS ON PROTEIN FOLDING KINETICS AND CONFORMATION

##### *In Vitro biophysical techniques*

Direct evidence of RF effects on protein folding came from a study of the acid induced refolding of tuna myoglobin, where a narrow distribution of tryptophan fluorescence lifetimes and slightly altered folding kinetics were found at 1.95 GHz. This was interpreted as evidence of a change in the distribution of the conformational substates involved in folding. This non-thermal transition is so named because the temperature of the bulk sample was kept constant during the experiment [12].

More supporting tests used the thermosensor protein GrpE, which is the co-chaperone of the Escherichia coli Hsp70 chaperone system. GrpE is known to undergo a well-characterized coil-to-helix transition that can be reversed around physiological temperature, making the protein a sensitive reporter of conformational changes. Using a real-time circular dichroism exposure chamber with a sensitivity of less than 1% of the total conformational change, GrpE was subjected to sinusoidal radiation at frequencies of 0.1, 1.0, and 1.9 GHz at field strengths of 0.1 to 5.0 kV/m and to GSM signals. No statistically significant changes in the equilibrium of the conformational state of GrpE were observed at any frequency or field strength[12,13].

Molecular dynamics simulations of 29 model proteins under microwave conditions likewise showed that higher rotational temperature increased intermolecular electrostatic and hydrogen bonding within protein cores, resulting in a compaction rather than an unfolding of the protein. Simulations of the amyloidogenic peptide, apoC-II(60-70), have shown that both global peptide mobility and the population of fibril inhibiting structures reach maxima around intermediate RF field strengths, raising the possibility that different RF environments may promote or stabilize proper folding rather

than destabilizing it, leading to a non-linear, protein specific relationship between RF exposure and protein folding [14,15].

##### *Hemoglobin and other blood protein studies*

In a number of spectroscopic studies of structural changes of RF-exposed human hemoglobin and other blood proteins, one of them with normal human hemoglobin exposed to mobile-phone radiofrequency, the studies found that RF-exposed hemoglobin showed minor alterations in its secondary structure elements and oxygen-binding parameters. However, these experiments used isolated proteins or blood samples during continuous RF exposure under laboratory conditions, which may not replicate normal exposure in human phone users [16,17].

#### V. HEAT SHOCK PROTEIN RESPONSES AND CELLULAR STRESS SIGNALING

##### *Induction of Hsp27 and p38 MAPK*

The heat shock response is one of the most common studies in vitro in the field. Non-thermal exposure of a human endothelial cell line (EA.hy926) to 900 MHz GSM radiation for one hour caused transient phosphorylation of Hsp27 by p38 MAPK, with no noticeable temperature change in cell cultures. The study also provided further evidence that the phosphorylation of many proteins may have been altered, activating several intracellular signal transduction pathways. The authors suggested that if Hsp27 becomes constitutively activated, apoptosis may be inhibited or actin stress fibers stabilized in endothelial cells in a long-term manner, and speculated on a potential link between this and brain tumor formation or increased blood-brain barrier permeability [18].

However, human peripheral blood leukocytes exposed to 900 MHz GSM RF signals at SARs of 0.4, 2.0, and 3.6 W/kg for up to 4 hours had no statistically meaningful difference in the number of HSP70 or HSP27- expressing cells compared with the unexposed condition. Positive controls (at 42 °C) did show temporary upregulation of both Hsp species. They concluded that mobile phone-type radiation is not a general stressor for human immune cells and that the heat shock response in endothelial cells may be cell-type specific [19,20].

One review concluded that many of the reports of non-thermal Hsp induction by RF are compromised by differences in exposure models, types of cells studied, and a lack of independent replication, and that the in vivo evidence is inconclusive [21]. Another study, that using BRET to investigate RF-induced HSF1 activation in living cells, found no evidence of activation under conditions that would elicit reliable HSF1 activation by thermal stress. This is meaningful because HSF1 is the master transcriptional regulator of heat shock proteins; therefore, if RF does not activate HSF1 in live cells, the heat shock response observed in some of the earlier biochemical studies may be an indirect or artifactual result [22,23].

#### VI. OXIDATIVE STRESS AS AN INDIRECT MECHANISM

##### *Animal and cell study evidence*

A 2021 systematic review of ten years of experimental studies in the International Journal of Molecular Sciences concluded that most animal studies and a high number of cell studies found an increased production of ROS and biomarkers of oxidative stress as a result of RF-EMF exposure. Many studies have found RF-induced oxidative stress in Wistar and Sprague-Dawley rats exposed in their brains and testes, and in mouse liver, kidneys, and ovaries across a range of SAR values, including regulatory levels [5].

Another review of RF exposure at the frequency of Wi-Fi (2.45 GHz) found upregulation of oxidative stress markers, reduced mitochondrial membrane potential, and induction of early antioxidant response pathways in neuron-like cells, with higher effects on neural than peripheral blood mononuclear cells [24]. Separately, the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) conducted a systematic review for the WHO, evaluating oxidative stress biomarkers in experimental animals and cells. Widespread evidence of RF-induced oxidative stress was found, but all results were of very low certainty due to poor blinding, dosimetry reporting, and lack of independent replication [25].

#### *Link between oxidative stress and proteostasis*

The general importance of oxidative stress in protein folding arises from the ability of ROS to oxidize proteins through direct or indirect mechanisms. Superoxide radicals and hydrogen peroxide may modify cysteine, methionine, and histidine residues, scramble disulfide bonds, carbonylate the protein, destabilize the native protein structure and cause protein aggregation [26–28]. Chronic oxidative stress can also overwhelm the chaperone and proteasomal systems, leading to an accumulation of misfolded proteins. This is a plausible explanation bridging RF-EMF, ROS and proteostasis. However, this causal chain has not yet been demonstrated in humans under normal exposure conditions [5,29,30].

### VII. PROTEOME-LEVEL CHANGES IN LIVING TISSUE

#### *Human skin data*

In vivo, human forearm skin exposed to a GSM signal for one hour had samples obtained before and after exposure. Among the 580 proteins identified in the skin, eight showed statistically significant differences in expression pre- and post-exposure [31]. The study provided evidence that a proteomic response occurs in vivo in human tissue exposed locally to RF fields. However, the biological importance of expression changes in a small number of proteins is unknown in cases where functional responses are not manifested, such as changes to barrier function, modulation of the immune response or activation of cell death [31–33].

#### *Animal brain proteomics*

Long-term (eight months) exposure of BALB/c mice to mobile phones or DECT base station caused statistically meaningful alterations in the expression levels of 143 proteins in the cerebellum, hippocampus, and frontal lobe, including cytoskeletal proteins, metabolic proteins and heat shock proteins. While alterations in the proteome may have biological relevance, the exposure duration and intensity were much

higher than those of typical of normal use of someone using a mobile phone, and the authors did not report any morphological neurodegeneration or behavioral phenotype accompanying the alterations[34].

### VIII. HUMAN HEALTH EVIDENCE

#### *Cancer risk*

The International Agency for Research on Cancer (IARC) classified RF-EMF exposure as “possibly carcinogenic to humans” (Group 2B), based on limited epidemiologic evidence for glioma and acoustic neuroma in heavy mobile phone users, in 2011. Group 2B is used where a causal link is credible but the available evidence is inconclusive on whether it does or does not exist[35,36]. Multiple studies, particularly the INTERPHONE study, have been conducted on the relationship between brain cancer and mobile telephones, and the consensus is that there is insufficient evidence of an increased risk with mobile phone use but that an increased risk from long-term heavy use cannot be ruled out [37,38].

#### *Neurodegenerative and metabolic disease*

A protein folding effect induced by RF-EMF in various tissues, including the human brain, would be expected to increase the frequency of protein-misfolding diseases of the central nervous system, such as Parkinson's disease, Alzheimer's disease or prion-like proteinopathies in heavy RF-EMF users or populations with high RF-EMF exposure. However, this has not been observed. RF is also not considered by international health authorities to be a candidate for causation of type 2 diabetes or metabolic syndrome, and current mechanistic data on RF exposure and metabolic endpoints remains, at best, speculative [20,39,40].

### IX. LIMITATIONS OF THE CURRENT EVIDENCE AND FUTURE DIRECTIONS

Common methodological issues of these studies included differences in the protocol for exposure, not reporting dosimetry parameters, a lack of statistical power to detect an effect, and lack of replication by other laboratories. However, the systematic review by the WHO concluded that despite the numerous positive observations related to oxidative stress processes, all of the health endpoints in this review had very low certainty owing to serious risk of bias in many studies [20,41].

Additionally, reproducibility across different cell types, as well as SAR reporting, may be helped by more rigorous controls (sham controls, even blinding for ideal studies). Future mechanistic studies may also exploit in vivo protein folding reporters that have been established in mammalian cells and are directly sensitive to chaperone activity, such as split-luciferase or chaperone client-based FRET. Future cohort studies of humans should be of longer duration, objective in their measurements and incorporate biologically relevant measures of RF exposure (e.g., cumulative lifetime RF exposure from high-volume mobile phone users). Additionally, intrinsically disordered proteins, which have no stable tertiary structure but may have widely divergent conformations in different

environmental conditions, are another area with high potential for these reporter strategies, albeit an untested one [41].

## X. CONCLUSION

No current evidence suggests that exposure to mobile phone or other wireless telecommunication radiation at levels in accordance with international safety recommendations, is capable of non-thermal disruption of protein folding that would be expected to produce disease. Non-thermal disruption of protein folding free-energy landscapes appears to be highly protein-specific, and detectable only under precisely controlled experimental conditions that do not resemble likely real-life exposures. Non-thermal stress responses like Hsp27 phosphorylation by p38 MAPK are found in certain in vitro cell types but are variable between models and not yet definitively linked to proteostasis failure in vivo. Oxidative stress appears to be the response that has shown the most consistent result across animal and cell-based models. This is the most plausible indirect mechanism of protein folding failure due to RF-EMF exposure but, while it cannot currently be used to infer risk of human health effects from RF-EMF exposure, the quality of evidence needs to be higher.

## XI. ADDITIONAL INFORMATION

### Author Contributions

Concept and design: Rawayh Muslim Albaghlany, Ranya M. Dawood, Raed A. Malallah

Acquisition, analysis, and interpretation of data: Rawayh Muslim Albaghlany

Drafting of the manuscript: Rawayh Muslim Albaghlany

Critical review of the manuscript for important intellectual content: Ranya M. Dawood and Raed A. Malallah

### Disclosures

Human subjects: All authors have declared that this study did not involve human participants or tissue.

Animal subjects: All authors have confirmed that this study did not involve animal subjects or tissue.

### Conflicts of interest

In accordance with the ICMJE uniform disclosure form, all authors have declared that no financial support was received from any organization for the submitted work; all authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

### Data Availability Statement

Data sharing is not applicable to this article, as no new data were created or analyzed in this review.

## XII. FIGURE LEGEND

### Proposed mechanisms of RF-EMF interaction with protein folding

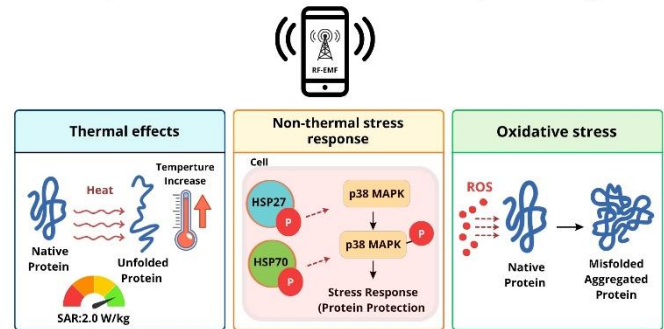


Figure 1. Proposed mechanisms of RF-EMF interaction with protein folding

There are three non-exclusive mechanisms by which RF-EMF emitted by mobile devices may affect protein folding. Thermal effects (left) where energy absorption and local heating can drive a native protein along the unfolding pathway, non-thermal stress response (center) that involves in phosphorylation of heat shock proteins via the p38 MAPK pathway and ultimately the cytoprotective stress response, and Oxidative stress (right) where the production of reactive oxygen species (ROS) may drive the conversion of a native, folded protein to misfolded, aggregated forms.

Image was created with Canva software.

**Abbreviations.** RF-EMF (radiofrequency electromagnetic field) SAR (specific absorption rate), Hsp27 (heat shock protein 27), Hsp70 (heat shock protein 70), p38 MAPK (p38 mitogen-activated protein kinase), ROS (reactive oxygen species), P (phosphorylated)

## REFERENCES

- Guidelines for Limiting Exposure to Electromagnetic Fields (100 kHz to 300 GHz). Health Phys. 2020, 118:483–524. 10.1097/HP.0000000000001210
- Foster KR, Glaser R: Thermal mechanisms of interaction of radiofrequency energy with biological systems with relevance to exposure guidelines. Health Phys. 2007, 92:609–20. 10.1097/01.HP.0000262572.64418.38
- Ontario Agency for Health Protection and Promotion (Public Health Ontario): Health Effects of Radiofrequency Electromagnetic Fields, Including 5G. King's Printer for Ontario: Toronto, ON; 2023.
- Naeem A, Fazili NA: Defective protein folding and aggregation as the basis of neurodegenerative diseases: the darker aspect of proteins. Cell Biochem Biophys. 2011, 61:237–50. 10.1007/s12013-011-9200-x
- Schuermann D, Mevissen M: Manmade Electromagnetic Fields and Oxidative Stress-Biological Effects and Consequences for Health. Int J Mol Sci. 2021, 22:3772. 10.3390/ijms22073772
- Baldwin RL: Energetics of protein folding. J Mol Biol. 2007, 371:283–301. 10.1016/j.jmb.2007.05.078
- Wainwright PR: The relationship of temperature rise to specific absorption rate and current in the human leg for exposure to electromagnetic radiation in the high frequency band. Phys Med Biol. 2003, 48:3143–55. 10.1088/0031-9155/48/19/004
- Vecchia P: Exposure to High Frequency Electromagnetic Fields, Biological Effects and Health Consequences (100 KHz - 300 GHz): Review of the Scientific Evidence on Dosimetry, Biological Effects, Epidemiological Observations, and Health Consequences Concerning Exposure to High Frequency Electromagnetic Fields (100 KHz - 300 GHz). ICNIRP; 2009.
- Zhang Q, Shao D, Xu P, Jiang Z: Effects of an Electric Field on the Conformational Transition of the Protein: Pulsed and Oscillating Electric

- Fields with Different Frequencies. *Polymers*. 2021, 14:123. 10.3390/polym14010123
10. Wu H, Ghaani MR, Futera Z, English NJ: Effects of Externally Applied Electric Fields on the Manipulation of Solvated-Chignolin Folding: Static- versus Alternating-Field Dichotomy at Play. *J Phys Chem B*. 2022, 126:376–86. 10.1021/acs.jpcc.1c06857
11. Cifra M, Pandey SK, Zakar T, et al.: The effects of external electric fields on proteins. *Chem Soc Rev*. Published Online First: 17 April 2026. 10.1039/D3CS00244F
12. Mancinelli F, Caraglia M, Abbruzzese A, d'Ambrosio G, Massa R, Bismuto E: Non-thermal effects of electromagnetic fields at mobile phone frequency on the refolding of an intracellular protein: myoglobin. *J Cell Biochem*. 2004, 93:188–96. 10.1002/jcb.20164
13. Beyer C, Christen P, Jelesarov I, Fröhlich J: Experimental system for real-time assessment of potential changes in protein conformation induced by electromagnetic fields. *Bioelectromagnetics*. 2013, 34:419–28. 10.1002/bem.21795
14. Broz M, Oostenbrink C, Bren U: The Effect of Microwaves on Protein Structure: Molecular Dynamics Approach. *J Chem Inf Model*. 2024, 64:2077–83. 10.1021/acs.jcim.3c01937
15. Todorova N, Bentvelzen A, English NJ, Yarovsky I: Electromagnetic-field effects on structure and dynamics of amyloidogenic peptides. *J Chem Phys*. 2016, 144:085101. 10.1063/1.4941108
16. Mousavy SJ, Riaz GH, Kamarei M, et al.: Effects of mobile phone radiofrequency on the structure and function of the normal human hemoglobin. *Int J Biol Macromol*. 2009, 44:278–85. 10.1016/j.ijbiomac.2009.01.001
17. Muehsam D, Lalezari P, Lekhray R, et al.: Non-Thermal Radio Frequency and Static Magnetic Fields Increase Rate of Hemoglobin Deoxygenation in a Cell-Free Preparation. *PLoS ONE*. 2013, 8:e61752. 10.1371/journal.pone.0061752
18. Leszczynski D, Joenväärä S, Reivinen J, Kuokka R: Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: molecular mechanism for cancer- and blood-brain barrier-related effects. *Differ Res Biol Divers*. 2002, 70:120–9. 10.1046/j.1432-0436.2002.700207.x
19. Puginier E, Leclercq L, Poulliet de Gannes F, et al.: Biological effects of 5G-modulated 700 MHz RF-EMF exposure on neuronal and glial cell models under isothermal conditions. *Sci Rep*. 2026, 16:10767. 10.1038/s41598-026-43960-4
20. Henschenmacher B, Bitsch A, de las Heras Gala T, et al.: The effect of radiofrequency electromagnetic fields (RF-EMF) on biomarkers of oxidative stress in vivo and in vitro: A protocol for a systematic review. *Environ Int*. 2022, 158:106932. 10.1016/j.envint.2021.106932
21. Cotgreave IA: Biological stress responses to radio frequency electromagnetic radiation: are mobile phones really so (heat) shocking? *Arch Biochem Biophys*. 2005, 435:227–40. 10.1016/j.abb.2004.12.004
22. Poque E, Ruigrok HJ, Arnaud-Cormos D, et al.: Effects of radiofrequency field exposure on proteotoxic-induced and heat-induced HSF1 response in live cells using the bioluminescence resonance energy transfer technique. *Cell Stress Chaperones*. 2021, 26:241–51. 10.1007/s12192-020-01172-3
23. Zhang B, Fan Y, Tan K: HSF1 Activation Mechanisms, Disease Roles, and Small Molecule Therapeutics. *Int J Biol Sci*. 2025, 21:3351–78. 10.7150/ijbs.110447
24. Bertuccio MP, Acri G, Ientile R, Caccamo D, Currò M: The Exposure to 2.45 GHz Electromagnetic Radiation Induced Different Cell Responses in Neuron-like Cells and Peripheral Blood Mononuclear Cells. *Biomedicines*. 2023, 11:3129. 10.3390/biomedicines11123129
25. Meyer F, Bitsch A, Forman HJ, et al.: The effects of radiofrequency electromagnetic field exposure on biomarkers of oxidative stress *in vivo* and *in vitro*: A systematic review of experimental studies. *Environ Int*. 2024, 194:108940. 10.1016/j.envint.2024.108940
26. Zhang W, Xiao S, Ahn DU: Protein oxidation: basic principles and implications for meat quality. *Crit Rev Food Sci Nutr*. 2013, 53:1191–201. 10.1080/10408398.2011.577540
27. Chahla C, Kovacic H, Ferhat L, Leloup L: Pathological Impact of Redox Post-Translational Modifications. *Antioxid Redox Signal*. 2024, 41:152–80. 10.1089/ars.2023.0252
28. Davies MJ: Methionine oxidation products as biomarkers of oxidative damage to proteins and modulators of cellular metabolism and toxicity. *Redox Biochem Chem*. 2025, 12:100052. 10.1016/j.rbc.2025.100052
29. Ulfig A, Jakob U: Cellular Oxidants and the Proteostasis Network: Balance between Activation and Destruction. *Trends Biochem Sci*. 2024, 49:761–74. 10.1016/j.tibs.2024.07.001
30. Koszla O, Solek P: Misfolding and aggregation in neurodegenerative diseases: protein quality control machinery as potential therapeutic clearance pathways. *Cell Commun Signal CCS*. 2024, 22:421. 10.1186/s12964-024-01791-8
31. Karinen A, Heinävaara S, Nylund R, Leszczynski D: Mobile phone radiation might alter protein expression in human skin. *BMC Genomics*. 2008, 9:77. 10.1186/1471-2164-9-77
32. Nylund R, Leszczynski D: Mobile phone radiation causes changes in gene and protein expression in human endothelial cell lines and the response seems to be genome- and proteome-dependent. *Proteomics*. 2006, 6:4769–80. 10.1002/pmic.200600076
33. Kuzniar A, Laffeber C, Eppink B, et al.: Semi-quantitative proteomics of mammalian cells upon short-term exposure to non-ionizing electromagnetic fields. *PLoS ONE*. 2017, 12:e0170762. 10.1371/journal.pone.0170762
34. Fragopoulou AF, Samara A, Antonelou MH, et al.: Brain proteome response following whole body exposure of mice to mobile phone or wireless DECT base radiation. *Electromagn Biol Med*. 2012, 31:250–74. 10.3109/15368378.2011.631068
35. Non-ionizing radiation, Part 2: Radiofrequency electromagnetic fields. *Iarc Monogr Eval Carcinog Risks Hum*. 2013, 102:1–460.
36. Kundi M: The Controversy about a Possible Relationship between Mobile Phone Use and Cancer. *Environ Health Perspect*. 2009, 117:316–24. 10.1289/ehp.11902
37. Swerdlow AJ, Feychting M, Green AC, Kheifets L, Savitz DA: Mobile Phones, Brain Tumors, and the Interphone Study: Where Are We Now? *Environ Health Perspect*. 2011, 119:1534–8. 10.1289/ehp.1103693
38. Humans IWG on the E of CR to: General remarks. In: *Non-Ionizing Radiation, Part 2: Radiofrequency Electromagnetic Fields*. International Agency for Research on Cancer; 2013.
39. Wyszowska J, Pritchard C: Open Questions on the Electromagnetic Field Contribution to the Risk of Neurodegenerative Diseases. *Int J Environ Res Public Health*. 2022, 19:16150. 10.3390/ijerph192316150
40. CDC: Facts About Cell Phones and Your Health. *Radiat. Your Health*. (2024). Accessed: June 16, 2026. <https://www.cdc.gov/radiation-health/data-research/facts-stats/cell-phones.html>.
41. Simkó M, Remondini D, Zeni O, Scarfi MR: Quality Matters: Systematic Analysis of Endpoints Related to “Cellular Life” in Vitro Data of Radiofrequency Electromagnetic Field Exposure. *Int J Environ Res Public Health*. 2016, 13:701. 10.3390/ijerph13070701