Sakarya Tıp Dergisi
Sakarya Medical Journal

e-ISSN : 2146-409X
Publisher : Sakarya University

Vol. 16, No. 3, 383-391, 2026
DOI: http://doi.org/10.31832/smj.1938619

Research Article

Effect of 3200 MHz Radiofrequency Fields on MCF-7 and L929 Cell Lines

Sinem Oruç Eraslan[1*], Sevilay Günay[2], Kayhan Ateş[3], Yavuz Erden[4], Bahriye Sırav Aral[2]

[1] Bandırma Onyedi Eylül University, Faculty of Medicine, Department of Biophysics, Bandırma, Türkiye
sinemoruc23@gmail.com

[2] Gazi University, Faculty of Medicine, Department of Biophysics, Ankara, Türkiye
sevilaygn7@gmail.com, bahriyes@gazi.edu.tr

[3] Akdeniz University, Faculty of Engineering, Electrical and Electronics Engineering, Antalya, Türkiye
kayhanates@akdeniz.edu.tr

[4] Bartın University, Faculty of Science, Department of Molecular Biology and Genetics, Bartın, Türkiye
yerden@bartin.edu.tr

* Corresponding Author

Received: 27.04.2026
Accepted: 01.09.2026
Published Online: 27.09.2026

Abstract

Objective: The use of technological devices emitting radio frequency fields(RFF) is increasing day by day. Nowadays, low-frequency RFF has begun to be used in cancer treatment as a non-invasive method. However, the time-dependent effects of high-frequency RFF on healthy and cancer cells are not yet clear. This study aims to examine the time-dependent effects of 3200MHz RFF on human breast cancer (MCF-7) and healthy mouse fibroblast (L929) cell lines.

Methods: MCF-7 and L929 cells were cultured with DMEM medium. RFF exposures were applied at 29.55dBm power levels (with Efield level 14V/m). MCF-7 and L929 cell lines were exposed to 3200 MHz RFF for 4 hours. Cell viability levels were determined immediately after exposure and following a 2-hour post-exposure incubation period.

Results: Immediately after 4 hours of 3200 MHz RFF exposure, cell viability decreased significantly by 12% in MCF-7 cells, with a further 15% decrease observed after a 2-hour post-exposure period. In contrast, the L929 cell line exhibited a significant increase in viability, with levels rising by 27% immediately after exposure and by 16% following the 2-hour post-exposure period($p<0.0001$).

Conclusions: The results of this study suggest that 3200MHz RFF exposure has a significant effect on the viability levels of cancer cells and that 3200MHz RFF can create the effect that low-frequency fields currently used as a non-invasive method have with a significant temperature increase, without a temperature increase. It is also effective in increasing healthy fibroblast cells, suggesting that it can be used as an alternative method to wound healing and cell renewal treatments.

Keywords: Radiofrequency field, MCF-7, L929, Cytotoxicity analysis, Cancer

1. INTRODUCTION

Over the past few decades, the widespread global use of mobile communication devices has raised public and scientific concerns regarding the biological effects of radiofrequency fields (RFF). Specifically, devices operating within the RF spectrum, including the 3200 MHz band, emit non-ionizing radiation. Although the International Commission on Non-Ionizing Radiation Protection (ICNIRP) has established strict guidelines to limit RF exposure and prevent adverse thermal effects,[1] the broader biological impacts of RFF remain a subject of ongoing investigation. Based on epidemiological studies, biological analyses, and mechanistic data, the International Agency for Research on Cancer (IARC) has classified RFF as a possible human carcinogen (Group 2B).[2] While RFF lacks the sufficient energy required for cellular ionization, it can still influence cellular organelles, potentially leading to the degradation of charged molecules and the disruption of electron transfer pathways.[3,4]

Justified by a favorable risk-benefit ratio in clinical oncology, despite its potential biological hazards, RFF has been reliably harnessed for cancer treatments since the 1990s. Its non-invasive nature and deep tissue penetration capabilities have made it a valuable tool in radical cancer therapy. For many years, radiofrequency ablation (RFA) has been a recommended modality for the treatment of malignant hepatic carcinomas.[5,6] Furthermore, recent advancements have coupled RFF with nanoparticles to induce localized hyperthermia, irreversibly destroying tumor cells [7] Today, RFA is widely utilized to treat various malignancies, including breast cancer, colorectal cancer, and hepatocellular carcinoma, particularly for metastases unsuitable for surgical resection.[8] However, it is crucial to note that the efficacy of these conventional RFF applications predominantly relies on heat generation (thermal ablation).

Cancer is fundamentally characterized by abnormal cellular proliferation and the capacity to invade or metastasize to distant organs.[9] This

Cite as: Oruç Eraslan S, Günay S, Ateş K, Erden Y, Aral Sırav B. Effect of 3200 MHz radiofrequency fields on MCF-7 and L929 cell lines. Sakarya Med J. 2026;16(3):383-91. doi:10.31832/smj.1938619.

This is an open access paper distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License.

malignant phenotype is driven by accumulated genetic and epigenetic alterations that disrupt critical cellular pathways, including DNA damage repair, cell cycle regulation, and immune evasion.[10] According to 2020 data from the International Agency for Research on Cancer (IARC), breast cancer is the most commonly diagnosed malignancy among women worldwide, exhibiting the highest incidence in 158 countries.[11,12] Breast cancer is clinically heterogeneous and classified into distinct molecular subtypes: hormone receptor-positive (expressing estrogen [ER+] or progesterone [PR+] receptors), human epidermal growth factor receptor 2-positive (HER2+), and triple-negative breast cancer (TNBC).[13,14] Treatment strategies are heavily dictated by these molecular profiles. While hormone therapy is the cornerstone for the 60-70% of patients with hormone receptor-expressing tumors,[15] other subtypes or advanced cases require alternative approaches, such as chemotherapy, targeted drug therapy, radiotherapy, and metronomic therapy.[16] Unfortunately, breast cancer frequently develops acquired resistance to these conventional therapies over time, driving disease progression and underscoring the urgent need for innovative treatment modalities. In order to accurately evaluate the biological effects of RFF and ensure experimental reproducibility, precise dosimetric evaluation is strictly required.[17] Specific Absorption Rate (SAR), expressed in watts per kilogram (W/kg), is the universal dosimetric metric used to quantify the rate of RF energy absorbed by a biological sample.[1] Calculating and modeling the SAR value is particularly crucial in in vitro studies to distinguish true frequency-dependent biological responses from heat-induced cellular damage.[18] Without accurate SAR analysis, it is impossible to confirm that the applied RFF remains below the thermal threshold.

Despite the established therapeutic potential of RFF, its specific non-thermal effects on cellular viability remain largely unexplored. To address this gap, this study investigates, for the first time, the in vitro biological responses of MCF-7 breast cancer and L929 healthy fibroblast cell lines to a 3200 MHz RFF. By employing cytotoxicity assays under strictly controlled thermal conditions, we aimed to elucidate the direct, frequency-dependent cytotoxic effects on malignant versus healthy cells, thereby isolating the biological impact of RFF from traditional heat-induced cellular damage.

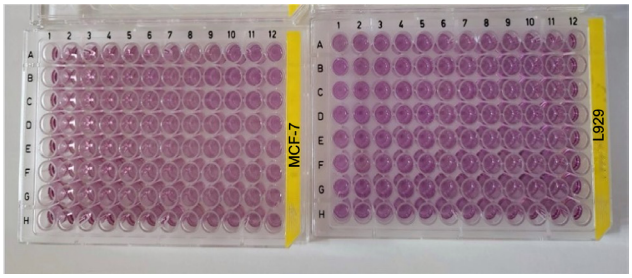
2. MATERIALS AND METHODS

2.1. Cell culture

Cells were first removed from the liquid nitrogen tank and seeded in 75 cm^2 culture flasks. High glucose Dulbecco's Modified Eagle Medium (DMEM; Catalog No: DMEM-HA; Capricorn Scientific GmbH, Germany) was used as the medium for MCF-7 cells (ATCC HTB-22). DMEM medium was used as the medium for L929 cells (ATCCCCL-1). Throughout the experimental procedures, cells were incubated at 37°C (Thermo Forma II CO_2 incubator, USA) in 5% CO_2. Cells that were confluent at the bottom of the flask were removed from the bottom of the flask using Trypsin-EDTA (Gibco, Thermo Fisher Scientific, USA; Cat. No: 25200056) solution and counted under an inverted microscope after staining with 0.4% Trypan blue (Gibco, Thermo Fisher Scientific, USA; Cat. No. 15250061). When the cell viability rate was 90% and above, the experimental steps were started and $15x10^3$ cells were seeded in each well of 96-well plates. (Figure 1).

Figure 1.

Seeding of MCF-7 and L929 cell lines in 96-well plates

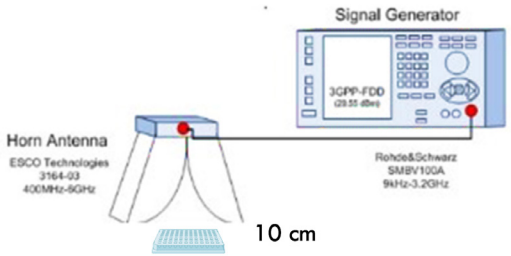


2.2. Radiofrequency field application

3200 MHz RF application was performed using a Rohde&Schwartz RF signal generator (Rohde & Schwarz, Munich, Germany) and ETS-Lindgren horn antenna (ETS Lindgren, Cedar Park, TX, USA). Cells seeded in 96-well plates were exposed to a 3200 MHz radiofrequency (RF) field for 4 hours per day. The electric field intensity was maintained at 14 V/m.The distance between the horn antenna and cell culture plates was 10 cm to ensure far field conditions (Figure 2).

Figure 2.

RFF application to MCF-7 and L929 cell lines



2.3. Temperature monitoring during RFF exposure

Temperature monitoring was conducted throughout RFF exposures to rule out thermal

contributions to the observed cellular effects. The thermometer was placed inside the control well in the 96-well plate. Temperature readings were recorded before, during and after 4 hours of RFF application. No significant temperature fluctuations were observed, with all measurements remaining within the initial incubation temperature (37°C), indicating a non-thermal exposure condition.

2.4. SAR calculations

The propagation of electromagnetic waves, as described by Maxwell's equations, was expressed below. Through this, the interaction of the wave with cell lines was modeled.[19] The following assumptions were considered in the electromagnetic exposure model.[20-22]

- The problem scenario was modeled in three dimensions.
- Cell lines were directly affected by electromagnetic waves. The outer boundary of the problem scenario was considered as an open boundary.
- The dielectric properties of cell lines were assumed to be homogeneous.

SAR simulations were performed with a finite integration technique (FIT) based commercial software called CST Microwave Studio (3DEXPERIENCE®, Dassault Systems, Hamburg). FIT operates with the integral form of Maxwell's equations, while the gridding is carried out similar to the finite difference time domain (FDTD) method.[23] In this study, RFF exposure of MCF-7 and L929 cell lines in the 3200 MHz frequency band was analyzed using the CST Studio Suite simulation program. The cells were modeled as a rectangular prism petri dish shaped material of 85.4×127.6×16 mm^3. These petri dishes were positioned 10 cm away from the horn antenna to simulate the exposure.

2.5. Cytotoxicity analysis (MTT assay)

First, MTT solution at a concentration of 0.5 mg/ml was prepared in sterile phosphate buffer. When the incubation period of the cells treated with the test compounds was completed, the medium in each well in the plates was removed and 50 µl of the prepared MTT solution was added to each well and left to incubate in an incubator with CO_2 for 3 hours. After this time, the MTT solution was removed from the wells and 100 µL of dimethyl sulfoxide (DMSO) was added to each well. The optical densities of the cells in the wells were read on an ELISA plate reader (Thermo Multiskan Go, USA) at a wavelength of 570 nm.[24] Control wells (wells containing only medium) were read and the absorbance values obtained were averaged and this value was considered as 100% viable cells.

2.6. Statistical analysis

In order to be able to perform one-way analysis of variance in the intergroup comparisons of the variables examined, conformity to normal distribution and homogeneity of variances were checked. If these assumptions were met, the difference between group averages for the relevant variables was performed with one-way analysis of variance (One Way Anova) and multiple comparisons were performed with Benferroni tests. In cases where normality assumptions were not met, Kruskal Wallis H test and Dunn test were used for multiple comparisons. Independent sample t test was used for pairwise comparisons. GraphPad Prism 9 for Macos was used in the analyses.

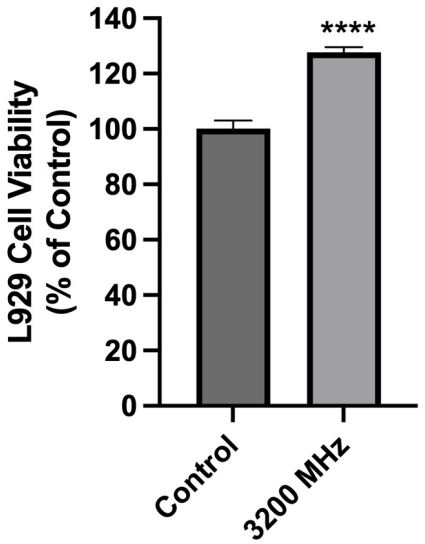
3. RESULTS

3.1. Immediate effect on MCF-7 cell viability following a 4-Hour 3200 MHz RFF exposure

Viability changes in the MCF-7 cell line immediately following a 4-hour 3200 MHz RFF exposure are presented in Figure 3. RFF exposure caused a significant decrease in viability compared to the control group (Figure 3; $p<0.0001$). Immediately after 3200 MHz RFF exposure for 4 hours, MCF-7 cell viability decreased by 12%.

Figure 3.

*Cell viability levels determined immediately after 3200 MHz RFF exposure (****p<0.0001). Group means were analyzed by Kruskal-Wallis H test. Dunn's test was used for pairwise comparisons. Data were expressed as mean±SD. *****p<0.0001 other group compared to control group*

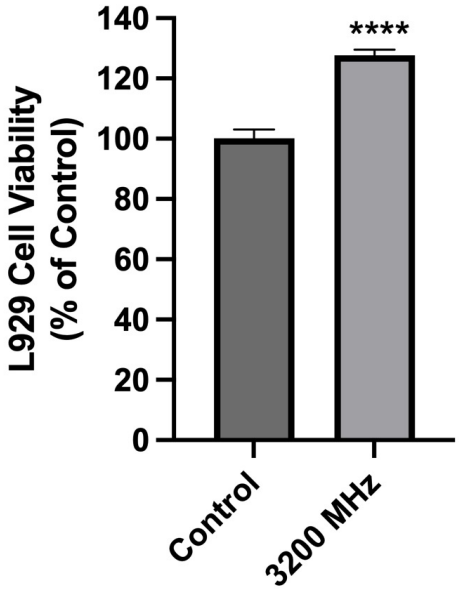


3.2. Immediate effect on L929 cell viability following a 4-Hour 3200 MHz RFF exposure

Viability changes in the L929 cell line immediately following a 4-hour 3200 MHz RFF exposure are presented in Figure 4. RFF exposure caused a significant increase in viability compared to the control group (Figure 4; p<0.0001). Immediately after 3200 MHz RFF exposure for 4 hours, we determined that the viability rate of L929 cells increased by 27%.

Figure 4.

*Cell viability levels determined immediately after 3200 MHz RFF exposure (****p<0.0001). Group means were analyzed by Kruskal-Wallis H test. Dunn's test was used for pairwise comparisons. Data were expressed as mean±SD. ****p<0.0001 other group compared to control group*

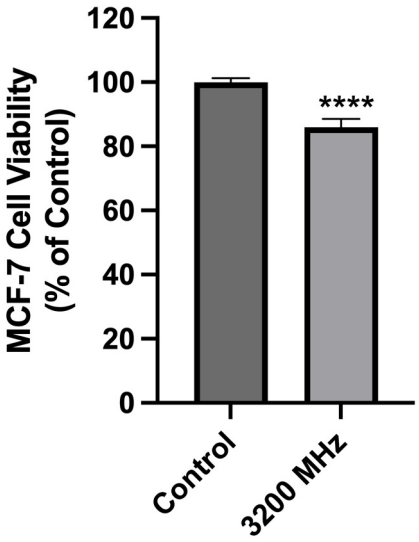


3.3. Impact of 3200 MHz exposure on MCF-7 cell viability following a 2-hour post-exposure period

Viability changes in the MCF-7 cell line, observed after a 2-hour post-exposure period following 4 hours of 3200 MHz RFF exposure, are presented in Figure 5. RFF exposure caused a significant decrease in viability compared to the control group (Figure 5; p<0.0001). MCF-7 cell viability decreased by 15 % following a 2-hour post-exposure period after 4 hours of 3200 MHz RFF application.

Figure 5.

*Cell viability levels measured after a 2-hour post-exposure period following a 4-hour 3200 MHz RFF exposure (****p<0.0001). Group means were analyzed by Kruskal-Wallis H test. Dunn's test was used for pairwise comparisons. Data were expressed as mean±SD. ****p<0.0001 other group compared to control group*

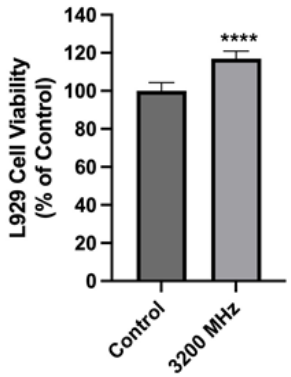


3.4. Impact of 3200 MHz exposure on L929 Cell viability following a 2-hour post-exposure period

Viability changes in the L929 cell line, observed after a 2-hour post-exposure period following 4 hours of 3200 MHz RFF exposure, are presented in Figure 6. RFF exposure caused a significant increase in viability compared to the control group (Figure 6; p<0.0001). Following a 2-hour post-treatment period after 4 hours of 3200 MHz RFF exposure, we determined that the viability rate of L929 cells increased by 16%.

Figure 6.

*Cell viability levels measured after a 2-hour post-exposure period following a 4-hour 3200 MHz RFF exposure (****p<0.0001). Group means were analyzed by Kruskal-Wallis H test. Dunn's test was used for pairwise comparisons. Data were expressed as mean±SD. ****p<0.0001 other group compared to control group*



3.5. SAR analysis

The behavior of cell lines under an incident electromagnetic wave is influenced by its dielectric properties.[25,26] A horn antenna, fed by 29.55 dBm, was designed and operated at 3200 MHz. SAR simulations were performed according to the IEEE/IEC 62704 standard. Electrical properties of cell lines at different frequencies are presented in Table.1.

Table 1.

Dielectric properties and mass density values of cell lines [27,28]

Type of Cell	ε_r	σ, S/m	Mass Density, kg/m^3
MCF-7	33.9	1.48	1300
L929	37.3	1.85	1109

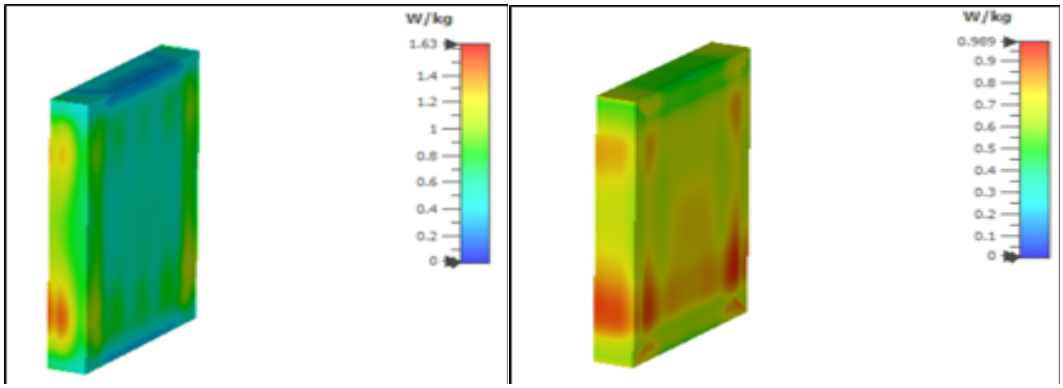
3.6. SAR analysis results for 3200 MHz exposure in MCF-7 cells

The maximum SAR analysis for 1 g of 3200 MHZ MCF-7 cells was calculated 1.63 W/kg. The maximum SAR values for 1 g and 10 g of MCF-7 cells are 1.63 W/kg and 0.989 W/kg, respectively. SAR simulation results are given in Figure 7.

Figure 7.

SAR simulation of MCF-7 cell lines:

a) 1 g average cell lines; b) 10 g average cell lines



(a) **(b)**

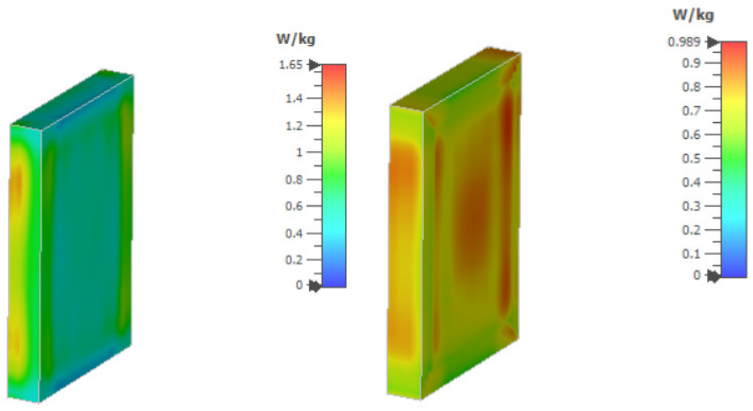
3.7. SAR analysis results for 3200 MHz exposure in L929 cells

The maximum SAR analysis result for 1 g of L929 cells at 3200 MHz was calculated as 1.65 W/kg. The maximum SAR values for 1 g and 10 g of L929 cells are 1.65 W/kg and 0.989 W/kg, respectively. SAR simulation results are depicted in Figure 8.

Figure 8.

SAR simulation results of L929 cell lines:

a) 1 g average cell lines; b) 10 g average cell lines



(a) **(b)**

4. DISCUSSION

In this study, the time-dependent effects of 3200 MHz RFF exposure on MCF-7 and L929 cell lines were examined. While RFF exposure decreased cell viability in a time-dependent manner in the MCF-7 cell line, it was found to increase viability in the L929 cell line. Our results also show that the effects of RFF in MCF-7 and L929 cell lines are markedly different.

Humans can be exposed to RFF (ranging from 30 kHz to 300 GHz) through various sources, including personal devices such as mobile phones, cordless phones, bluetooth devices, and amateur radios. Occupational exposure can arise from equipment like high-frequency dielectric and induction heaters, as well as powerful pulsed radars. Additionally, environmental sources like mobile phone base stations, broadcast antennas, and medical applications contribute to RFF exposure. For workers, most of the exposure comes from sources in close proximity, while the general public is primarily exposed to RFF from handheld devices, particularly mobile phones, which are held close to the body. Although the relationship between RFF and cancer has been studied for a long time, the answer is still not clearly known. The reason for this is the presence of many variables in RFF applications.

Studies have shown that cells may respond differently to RFF exposure. It was observed that there was no significant change in the viability of neuroblastoma cells (SH-SY5Y) exposed to 1800 MHz RFF for 10, 30 and 60 minutes.[29] Likewise, it was stated that 2.45 GHz RFF exposure applied for 1, 2, 12, 24 hours did not cause any changes in human leukemia cells.[30] In a study, they examined the effects of 1800 MHz radiofrequency application for 1, 3, 6, 12 and 24 hours on protein expression in MCF-7 cell line and investigated possible effects on normal cell physiological function. As a result, they showed that RF field-induced protein expression changes may depend on the exposure time and mode.[31] In a study, they investigated the effect of 900 MHz radiofrequency field application for 6, 21, 51 and 101 minutes/day on the growth, morphology and proliferation rate of MCF-7 and mesenchymal stem cells. As a result, they showed that both cell viability and proliferation rates of both cells could be reduced.[32] It is widely known that electromagnetic fields (EMF) are used in certain cancer treatment protocols to target and eliminate tumor cells. When cells are exposed to EMF, it induces the generation of reactive oxygen species (ROS), which disrupt antioxidant defense mechanisms and lead to lipid peroxidation.[33] The increase in ROS can result in DNA damage, leading to cell cycle arrest. However, in another study, it was stated that there was no significant change in the glutathione concentration of L929 and SH-SY5Y after 1 hour or 24 hours of exposure to 872 MHz RFF.[34] In another study, human fibroblast and prostate cancer cell lines were exposed to 2.5 GHz RFF for 24, 48, and 72 hours. It was found that RFF exposure caused a significant decrease in the viability of human fibroblasts and a notable increase in cancer cells. Morphological analysis revealed no significant changes in either cell line following RFF exposure.[35]

A critical aspect of our study, distinguishing it from conventional thermal ablation research, is the dosimetric evaluation through SAR analysis. In biological RFF studies, calculating the SAR value is not merely a procedural step; it is the fundamental parameter that defines the actual energy dose absorbed by the cellular microenvironment.[1,17] The SAR values evaluated in our experimental setup were specifically maintained at levels insufficient to induce a measurable temperature increase. By verifying this dosimetric parameter, we confidently ruled out thermal damage—which typically causes non-specific cellular necrosis—as the underlying mechanism.[18] Instead, our SAR evaluation demonstrates that the applied 3200 MHz RFF delivers a strictly non-thermal physical stimulus. This accurately quantified energy dose was capable of selectively triggering cytotoxic pathways in MCF-7 cancer cells while paradoxically promoting viability in L929 healthy fibroblasts. Thus, the SAR analysis provides the essential biophysical validation that the observed cellular responses are true radio-biological interactions driven by frequency and exposure time, completely independent of heat generation.[19]

Modern mobile phones use the 5G next-generation technology. Therefore, there is a need for more research focused on high-frequency exposures. The results of 3200 MHz RFF on the cell lines in our study are important. Frequency determines the penetration depth of RF energy; higher frequencies tend to cause less temperature increase in deeper tissues due to their reduced penetration ability. The results of this study suggest that 3200 MHz RFF exposure has a significant effect on the viability levels of cancer cells and that 3200 MHz RFF can create the effect that low-frequency fields currently used as a non-invasive method have with a significant temperature increase, without a temperature increase. It is also effective in increasing healthy fibroblast cells, suggesting that it can be used as an alternative method to wound healing and cell renewal treatments.

Article Information Form

Authors Notes

This study was presented as Oral Presentation at the 6th International 35th National Biophysics Congress on September 4-7, 2024 at Erciyes University.

Author Contributions

Conceptualization: SOE; Methodology: SOE, SG; Software: SOE, YE, KA, BSA; Validation: SOE, SG, KA, BSA, YE; Formal analysis: SOE, SG, KA, BSA, YE; Investigation: SOE, SG, YE; Resources: SOE, YE, BSA; Data curation: SOE, KA; Writing – original draft: SOE, SG; Writing – review & editing: SOE, SG, KA, BSA, YE; Visualization: SOE, KA; Project administration: SOE.

The Declaration of Conflict of Interest/ Common Interest

There is no conflict of interest or common interest among the authors.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Artificial Intelligence Statement

No artificial intelligence tools were used while writing this article.

Copyright Statement

Authors own the copyright of their work published in the journal and their work is published under the CC BY-NC 4.0 license.

REFERENCES

1. Ziegelberger G, Croft R, Feychting M, et al. Guidelines for limiting exposure to electromagnetic fields (100 kHz to 300 GHz). Health Phys. 2020;118(5):483-524. doi:10.1097/HP.0000000000001210.

2. Iarc W. IARC Classifies radiofrequency electromagnetic fields as possibly cancinogenic to humans. International Agency for Research on Cancer. 2011.

3. Marjanović A-M, Pavičić I, Trošić I. Biological indicators in response to radiofrequency/microwave exposure. Arh Hig Rada Toksikol. 2012;63(3):407-15. doi:10.2478/10004-1254-63-2012-2215.

4. Yakymenko I, Tsybulin O, Sidorik E, Henshel D, Kyrylenko O, Kyrylenko S. Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation. Electromagn Biol Med. 2016;35(2):186-202. doi:10.3109/15368378.2015.1043557.

5. McGahan JP, Browning PD, Brock JM, Tesluk H. Hepatic ablation using radiofrequency electrocautery. Invest Radiol. 1990;25(3):267-70. doi:10.1097/00004424-199003000-00011.

6. Rossi S, Fornati F, Pathies C, Buscarini L. Thermal lesions induced by 480 KHz localized current field in guinea pig and pig liver. Tumori. 1990;76(1):54-7. doi:10.1177/030089169007600114.

7. Glazer ES, Zhu C, Massey KL, Thompson CS, Kaluarachchi WD, Hamir AN, et al. Noninvasive radiofrequency field destruction of pancreatic adenocarcinoma xenografts treated with targeted gold nanoparticles. Clin Cancer Res. 2010;16(23):5712-21. doi:10.1158/1078-0432.CCR-10-2055.

8. Ripley RT, Gajdos C, Reppert AE, MacDermott T, McCarter MD, Pearlman NW. Sequential radiofrequency ablation and surgical debulking for unresectable colorectal carcinoma: Thermo-surgical ablation. J Surg Oncol. 2013;107(2):144-7. doi:10.1002/jso.23211.

9. Schwartz SM. Epidemiology of cancer. Clin Chem. 2024;70(1):140-9. doi:10.1093/clinchem/hvad202.

10. Hanahan D, Weinberg RA. Hallmarks of cancer: the next generation. Cell. 2011;144(5):646-74. doi:10.1016/j.cell.2011.02.013.

11. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: a cancer journal for clinicians. 2021;71(3):209-49. doi:10.3322/caac.21660.

12. Gadaleta E, Thorn GJ, Ross-Adams H, Jones LJ, Chelala C. Field cancerization in breast cancer. J Pathol. 2022;257(4):561-74. doi:10.1002/path.5902.

13. Liedtke C, Mazouni C, Hess KR, André F, Tordai A, Mejia JA, et al. Response to neoadjuvant therapy and long-term survival in patients with triple-negative breast cancer. J Clin Oncol. 2008;26(8):1275-81. doi:10.1200/JCO.2007.14.4147.

14. Lehmann BD, Bauer JA, Chen X, Sanders ME, Chakravarthy AB, Shyr Y, et al. Identification of human triple-negative breast cancer subtypes and preclinical models for selection of targeted therapies. J Clin Invest. 2011;121(7):2750-67. doi:10.1172/JCI45014.

15. Chen X, Xu D, Li X, Zhang J, Xu W, Hou J, et al. Latest overview of the cyclin-dependent kinases 4/6 inhibitors in breast cancer: the past, the present and the future. J Cancer. 2019;10(26):6608. doi:10.7150/jca.33079.

16. Barzaman K, Karami J, Zarei Z, Hosseinzadeh A, Kazemi MH, Moradi-Kalbolandi S, et al. Breast cancer: biology, biomarkers, and treatments. Int Immunopharmacol. 2020;84:106535. doi:10.1016/j.intimp.2020.106535.

17. Schmid G, Hirtl R, Gronau I, Meyer V, Drees K, Lerchl A. Design and dosimetric characterization of a broadband exposure facility for in vitro experiments in the frequency range 18–40.5 GHz. Bioelectromagnetics. 2022;43(1):25-39. doi:10.1002/bem.22376.

18. Paffi A, Apollonio F, Lovisolo GA, Marino C, Pinto R, Repacholi M, et al. Considerations for developing an RF exposure system: a review for in vitro biological experiments. IEEE Transactions on Microwave Theory and Techniques. 2010;58(10):2702-14. doi:10.1109/TMTT.2010.2065351.

19. Wessapan T, Keangin P, Rattanadecho P, Somsuk N. Comparative analysis of heat transfer dynamics in high-intensity focused ultrasound and microwave ablation for cancer treatment. International Journal of Thermofluids. 2025;26:101090. doi:10.1016/j.ijft.2025.101090.

20. İl N, Ateş K, Özen Ş. Electromagnetic field exposure to human head model with various metal objects at sub-6 GHz frequencies. Electromagn Biol Med. 2023;42(3):114-22. doi:10.1080/15368378.2023.2220736.

21. Gökçek-Saraç Ç, Çetin E, Ateş K, Özen Ş, Karakurt S. Different duration of exposure to a pulsed magnetic field can cause changes in mRNA expression of apoptotic genes in oleic acid-treated neuroblastoma cells. Int J Radiat Biol. 2024;100(10):1471-80. doi:10.1080/09553002.2024.2386968.

22. Gökçek-Saraç Ç, Akçay G, Karakurt S, Ateş K, Özen Ş, Derin N. Possible effects of different doses of 2.1 GHz electromagnetic radiation on learning, and hippocampal levels of cholinergic biomarkers in Wistar rats. Electromagn Biol Med. 2021;40(1):179-90. doi:10.1080/15368378.2020.1851251.

23. Weiland T. A discretization model for the solution of Maxwell's equations for six-component fields. Archiv Elektronik und Uebertragungstechnik. 1977;31:116-120.

24. Mosmann T. Rapid colorimetric assay for cellular growth and survival: application to proliferation and cytotoxicity assays. J Immunol Methods. 1983;65(1-2):55-63. doi:10.1016/0022-1759(83)90303-4.

25. Abdilla L, Sammut C, Mangion LZ. Dielectric properties of muscle and liver from 500 MHz–40 GHz. Electromagn Biol Med. 2013;32(2):244-52. doi:10.3109/15368378.2013.776436.

26. Gabriel S. The dielectric properties of biological tissues: I. Literature survey. Phys Med Biol. 1996;41:2271-93. doi:10.1088/0031-9155/41/11/001.

27. Campbell A, Land D. Dielectric properties of female human breast tissue measured in vitro at 3.2 GHz. Phys Med Biol. 1992;37(1):193-210. doi:10.1088/0031-9155/37/1/014.

28. Hasgall PA, Neufeld E, Neufeld E, Gosselin M, Klingenbock A, Kuster N, et al. IT'IS Database for thermal and electromagnetic parameters of biological tissues. Version 4.0, 2018.

29. Marjanovic Cermak AM, Pavicic I, Trosic I. Oxidative stress response in SH-SY5Y cells exposed to short-term 1800 MHz radiofrequency radiation. J Environ Sci Health A Tox Hazard Subst Environ Eng. 2018;53(2):132-8. doi:10.1080/10934529.2017.1383124.

30. Nazıroğlu M, ÇIğ B, Doğan S, Uğuz AC, Dilek S, Faouzi D. 2.45-Gz wireless devices induce oxidative stress and proliferation through cytosolic Ca2+ influx in human leukemia cancer cells. Int J Radiat Biol. 2012;88(6):449-56. doi:10.3109/09553002.2012.682192.

31. Zeng Q, Weng Y, Chen G, Lu D, Chiang H, Xu Z. Effects of global system for mobile communications 1800 MHz radiofrequency electromagnetic fields on gene and protein expression in MCF-7 cells. Proteomics. 2006;40(3):4732-8. doi:10.1002/pmic.200600234.

32. Shahbazi-Gahrouei D, Hashemi-Beni B, Moradi A, Aliakbari M, Shahbazi-Gahrouei S. Exposure to global system for mobile communication 900 mhz cellular phone radiofrequency alters growth, proliferation and morphology of michigan cancer foundation-7 cells and mesenchymal stem cells. Int J Prev Med. 2018;9:51. doi:10.4103/ijpvm.IJPVM_75_17.

33. Reuter S, Gupta SC, Chaturvedi MM, Aggarwal BB. Oxidative stress, inflammation, and cancer: how are they linked? Free Radic Biol Med. 2010;49(11):1603-16. doi:10.1016/j.freeradbiomed.2010.09.006.

34. Höytö A, Luukkonen J, Juutilainen J, Naarala J. Proliferation, oxidative stress and cell death in cells exposed to 872 MHz radiofrequency radiation and oxidants. Radiat Res. 2008;170(2):235-43. doi:10.1667/RR1322.1.

35. Górski R, Nowak-Terpiłowska A, Śledziński P, Baranowski M, Wosiński S. Morphological and cytophysiological changes in selected lines of normal and cancer human cells under the influence of a radio-frequency electromagnetic field. Ann Agric Environ Med. 2021;28(1):163-71. doi:10.26444/aaem/118260.