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REVIEW



Radiofrequency radiation and Alzheimer's disease: harmful and therapeutic implications

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ABSTRACT

Purpose: Alzheimer's disease (AD) is a neurodegenerative disorder characterized by memory loss and cognitive decline. The relationship between AD and radiofrequency (RF) radiation emitted by wireless devices remains under investigation. The aim of this review is to comprehensively explore the effects of RF radiation on AD by evaluating existing literature. This review used Web of Science, Scopus, and PubMed to find relevant studies on AD and RF radiation. This review evaluates a total of 81 studies, including animal models, human studies, and in vitro experiments, with results summarized in tables for clarity.

Conclusion: Some studies suggest RF aggravates AD by increasing oxidative stress, impairing blood-brain barrier integrity, and promoting amyloid-beta deposition. Conversely, other studies indicate RF may have protective benefits, such as enhancing brain mitochondrial functions and reducing amyloid-beta levels. Understanding the RF-AD relationship, including parameters like frequency and exposure time, is crucial for therapeutic strategies. The studies reviewed highlight RF radiation's dual effects on AD, underscoring the need for a detailed approach. Further studies are required to clarify these effects and inform preventive and therapeutic measures.

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Alzheimer's disease (AD); radiofrequency radiation exposure (RF); oxidative stress; amyloid-beta; neurodegeneration; therapy and harmful effects

Introduction

Alzheimer's disease (AD) is a neurodegenerative disorder associated with aging and is a growing public health concern worldwide. The disease is characterized by progressive memory loss, cognitive impairments, and severe neurological symptoms that ultimately affect an individual's daily life. The pathogenesis of AD is complex and not yet fully understood. However, mechanisms such as oxidative stress, neuroinflammation, protein aggregation, and neuronal cell death are implicated in the development of the disease (Firdous et al. 2024).

In recent years, the potential effects of radiofrequency (RF) radiation on human health have been the focus of increasing attention. RF radiation is emitted by widely used technological devices such as wireless communication devices, mobile phones, Wi-Fi networks, and base stations. With the widespread use of these technologies, the potential effects of exposure to RF radiation on the risk of neurodegenerative diseases such as AD are increasingly being investigated (Shirbandi et al. 2023).

The purpose of this review is to evaluate relation between RF exposures on AD. At the First step, basic information about the pathophysiology of AD and sources of RF radiation will be defined. Secondly, the study results included in this article will be evaluated from an objective perspective

and the relation between RF radiations on AD will be summarized.

Therefore, aim of this review is to evaluate the findings from current research and provide a framework to better understand the potential effects of RF radiation on AD pathogenesis. The role of RF exposure in the prevention or treatment of neurodegenerative diseases such as AD will be revealed in this article and it will serve as a guide for future researches.

Materials and methods

In this study, we systematically reviewed the relationship between RF radiation and AD by searching databases such as Web of Science, Scopus, and PubMed. The search was conducted using the keywords 'Alzheimer's Disease', 'AD', 'Radiofrequency radiation', 'RF exposure', 'RF', 'therapy', and 'harmful effects'. The inclusion criteria were carefully defined to ensure the relevance and quality of the selected studies. Specifically, studies published between 1994 and 2024 were included to encompass both foundational and contemporary findings in the field. The review focused on human and animal models investigating the effects of RF radiation on AD or relevant biomarkers such as amyloid-beta accumulation, tau phosphorylation, oxidative stress, and neuroinflammation.

To ensure the reliability of the findings, only studies that reported specific RF exposure parameters—including frequency, SAR (Specific Absorption Rate), and exposure duration—were included. Additionally, selected publications were required to demonstrate robust methodologies, appropriate sample sizes, and transparent statistical reporting, including the use of control groups and clear descriptions of statistical analyses. Only English-language articles available in full-text format were considered.

A total of 81 studies meeting these criteria were evaluated in this review. The results of the included studies were summarized in tables to enhance clarity and facilitate comparison. This systematic approach aimed to provide a balanced and impartial analysis of the therapeutic and harmful effects of RF radiation on AD. Moreover, our previous review in 2020 examined the broader impacts of electromagnetic radiations, including ELF-MF and ionizing radiation, on AD (Dasdag et al. 2020). Building on those findings, this review narrows the focus specifically to RF radiation, addressing the need for a more detailed understanding of its potential effects on AD.

Results

Overview of RF radiation and Alzheimer's disease pathogenesis

RF radiation, emitted by wireless communication devices, is a low-frequency component of the electromagnetic spectrum and has become an integral part of modern life (Lin 2022). AD is a neurodegenerative disorder characterized by amyloid beta ($A\beta$) plaques and neurofibrillary tangles, leading to progressive neuronal loss and cognitive decline (Scheltens et al. 2021). Its pathogenesis involves a complex interplay of genetic predisposition, aging, oxidative stress, neuroinflammation, and environmental factors.

$A\beta$ Plaques & Neurofibrillary Tangles: $A\beta$ plaques are abnormal peptide accumulations impairing neuronal communication, while tau protein misfolding leads to intracellular tangles, disrupting cellular function (Moloney et al. 2021; Scheltens et al. 2021).

Genetic Factors & Aging: Variants in genes such as APOE $\epsilon 4$, APP, PSEN1, and PSEN2 significantly influence AD risk (Ibanez et al. 2021; Li et al. 2023), while aging alters neuronal function and increases disease susceptibility (Yap et al. 2024).

Oxidative stress & Neuroinflammation: Excess reactive oxygen species (ROS) contribute to lipid peroxidation, protein/DNA damage, and neuronal dysfunction (Bai et al. 2022). Additionally, chronic inflammation accelerates neurodegeneration (Sharma et al. 2022).

Environmental and Lifestyle Factors: Diet, sleep, physical activity, and cognitive engagement influence AD risk, underlining the multifactorial nature of the disease (Guo et al. 2020).

The complex interaction of these factors underlies AD pathogenesis, necessitating further research to understand its precise mechanisms.

Potential mechanisms of RF radiation impact on Alzheimer's disease pathogenesis

While the effects of RF radiation on AD pathogenesis remain inconclusive, several mechanisms have been proposed:

Increase in Oxidative Stress: RF radiation may increase free radical formation and weaken antioxidant defenses, contributing to neuronal oxidative damage, a key factor in AD progression (Bouji et al. 2020; Singh et al. 2020).

Blood-Brain Barrier (BBB) Damage: RF exposure is suggested to impair BBB integrity, potentially facilitating neurodegenerative processes (Tang et al. 2015; Narayanan et al. 2019).

Cellular Function Impairment: Disruptions in mitochondrial activity and cellular signaling due to RF exposure may negatively affect neuronal survival (Tsoy et al. 2019; Von Niederhäusern et al. 2019).

$A\beta$ Deposition & Tau Protein Aggregation: Some studies indicate RF radiation may influence $A\beta$ accumulation and tau misfolding, both of which are hallmark features of AD (Shirbandi et al. 2023).

Inflammation & Immune Response Modulation: RF exposure has been linked to chronic neuroinflammation, which may accelerate neurodegeneration by promoting $A\beta$ plaque accumulation and synaptic dysfunction (Lameth et al. 2017).

Although these mechanisms are actively studied, most findings are based on in vitro and animal studies, with limited evidence from human research. Further clinical and epidemiological studies are needed to determine the true impact of RF radiation on AD development.

Current literature and study classification

Existing studies report conflicting results regarding RF radiation's impact on AD:

- **Neuroprotective Effects:** Some studies suggest RF exposure may mitigate AD pathology by reducing $A\beta$ accumulation and improving neuronal function.
- **Neurodegenerative Effects:** Others report that RF radiation exacerbates oxidative stress, neuroinflammation, and cognitive decline.
- **No Significant Effect:** Certain studies find no statistically significant correlation between RF exposure and AD progression.

To systematically evaluate these findings, this review categorizes studies into positive (Table 1), negative (Table 2), and neutral (Table 3) effects. This classification aims to synthesize existing knowledge and guide future research directions in understanding RF radiation's role in AD pathology.

Discussion

The effects of RF exposure on AD remain inconsistent across the literature. To better understand these variations, this review categorized studies into positive effects, negative effects, and no observed effects. However, while studies

Table 1. Positive effects of RF radiation on Alzheimer's disease.

Reference	Species	Frequency	SAR	Duration of exposure	Main outcomes
Arendash et al. (2010)	Transgenic mice	918 MHz (GSM)	0.25 W/kg	1 h/d for 8.5 months	Reduced A β accumulation, enhanced clearance, transient brain temperature increase.
Arendash et al. (2012)	Transgenic mice	918 MHz (GSM)	Between 0.25 and 1.05 W/kg	2 h/d for 2 months	Improved memory, reduced A β deposition, stable brain temperature, decreased cerebral blood flow.
Arendash et al. (2019)	Human (Alzheimer patients)	915 MHz	1.6 W/kg	Twice-daily, 1h/d, for 2 months	Improved ADAS-cog and Rey AVLT scores, increased soluble CSF A β 1-40/A β 1-42, reduced CSF p-tau/A β 1-42 and plasma oligomeric A β , stable-to-increased brain glucose utilization, enhanced cingulate cortex connectivity.
Arendash et al. (2022)	Human (Alzheimer patients)	915 MHz	1.6 W/kg	Twice-daily, 1h/d, intermittent TEMT application over periods of 2 months, 4 months, and 12 months, totaling 2.5 years.	No adverse effects, stable cognitive function (ADAS-cog13, Rey AVLT, MMSE, ADL), reduced CSF C-reactive protein, p-tau217, A β 1-40/A β 1-42, and balanced plasma tau levels.
Banaceur et al. (2013)	Wildtype mice and triple transgenic mice	2.40 GHz	1.60 W/kg	2h for 1 month	Enhanced cognitive behavior in 3xTg-AD mice.
Bayat et al. (2023)	Sprague-Dawley rat	2.45 GHz	From 0.0346 to 0.0060 W/Kg	2h/d for 45 days	Enhanced learning-memory performance, LTP induction, increased hippocampal CA1 neuronal density, partial neurotransmitter recovery, reduced GABA transmission, no effect on BST.
Cao et al. (2022)	Human (Alzheimer patients)	915 MHz	1.6 W/kg	Twice-daily, 1h/d, for 2 months	Balanced 11 of 12 cytokine levels in blood/brain, improved cognition, reduced plasma CRP levels.
Dragicevic et al. (2011)	Transgenic (Tg) and non-transgenic (NT) mice	918 MHz (GSM)	0.25–1.05 W/kg	1 h/d for 1 month	Enhanced mitochondrial function in cognitively relevant brain regions of Tg and NT mice, increased soluble A β 1-40 in mitochondria.
Falone et al. (2018)	SH-SY5Y neuroblastoma cells	1950 MHz UMTS	0.3 or 1.25 W/kg	Continuously for 20h	Reduced menadione-induced DNA damage.
Ji Jeong et al. (2015)	Tg-5xFAD mice, wild type mice	1950 MHz	5 W/kg	2h/d, 5 d/week for 8 months	Reduced A β plaques in Tg-5xFAD mice (notably in hippocampus and entorhinal cortex), decreased APP and its carboxyl-terminal fragments, lowered A β 42/A β 40 ratio in hippocampus, inhibition of β -secretase and neuroinflammation, improved memory, altered expression of five A β -related genes.
Kim et al. (2018)	C57BL/6 mice	835 MHz	4.0 W/kg	5h/d for 4 weeks	Reduced pan-calcium channel expression in hippocampal neurons, autolysosome accumulation, increased autophagy-related genes and proteins (e.g. LC3B-II).
Kumlin et al. (2007)	Wistar rat	900 MHz	0.3 or 3.0 W/kg	2h/d, 5d/weeks for 5 weeks	No neurodegenerative changes, neuronal loss, or blood-brain barrier leakage; no differences in open field, plus maze, or acoustic response tests; improved learning and memory.
Mori and Arendash (2011)	Transgenic (Tg) and non-transgenic (NT) mice	918 MHz (GSM)	0.25–1.05 W/kg	1 h/d for 2 month	Enhanced neuronal activity in the entorhinal cortex of Tg and NT mice, improved cognitive function.
Park et al. (2018)	HT22 mouse hippocampal neuronal cells and SH-SY5Y human neuroblastoma cells	1950 MHz	6 W/kg	2h/d for 3 d	Reduced mRNA levels of APP, BACE1, ADAM10, and PSEN1 in HT22 cells; unchanged APP mRNA in SH-SY5Y cells; no differences in APP/BACE1 protein expression or secreted A β peptide.
Perez et al. (2021)	Primary human brain cultures	64, 100, 918 MHz	0.4, 0.6, and 0.9, W/kg	1 h/d during 4, 8, 14 or 21 days	Reduced secreted A β 40/A β 42 levels, no cellular toxicity or changes in secreted A β precursor protein- α .
Dasdag et al. (2009)	Wistar Albino	900 MHz	0.17–0.58W/kg	2h/d, 7 days a week, over a 10-month period	Low apoptosis score, stable p53 expression, oxidative stress index, and total oxidant status; high total antioxidant capacity and catalase levels.
Sannino et al. (2022)	Human SH-SY5Y neuroblastoma cells	1950 MHz (UMTS)	0.3 W/kg	20h	Reduced menadione-induced DNA damage; no DNA damage reduction in SH-SY5Y cells inhibited by bafilomycin A1 and E64d; no adaptive response in CRISPR SH-SY5Y cells lacking ATG7 or ATG5.
Son et al. (2018)	5xFAD mice	1950 MHz	5.0 W/kg	2h/d and 5 d/week for 8 months	Improved cognitive deficits, reduced hyperactivity and anxiety-like behaviors, increased glucose metabolism in the hippocampus and amygdala.
Son et al. (2023)	5xFAD mice	1950 MHz	5.0 W/kg	2h/d and 5 d/week for 6 months	Improved cognitive deficits and reduced amyloid- β deposition; decreased Iba1 and CSF1R expression in the hippocampus; reduced Csf1r, CD68, and Ccl6 levels; downregulated Trem2, Fcgr1a, Ctss, and Spi1 gene expression.
Sun et al. (2022)	5 $\times$ FAD mice	1500 MHz	2 or 4 W/kg	2h/d, 5 d/week for 5 months	Improved learning and memory, reduced A β 42 and microglia in the hippocampus, downregulated BACE1 expression, decreased anxiety-like behaviors and IL-6 mRNA levels, increased IL-4 and IL-10.
Zeni et al. (2021)	SH-SY5Y neuroblastoma cells	1950 MHz UMTS	0.3 W/kg	Continuously for 20h	Increased HSP70 in culture medium, no change in intracellular HSP70 levels, RF protection against menadione-induced DNA damage.

d: day, min: minute, h: hour.

Table 2. Negative effects of RF radiation on Alzheimer's disease.

Reference	Species	Frequency	SAR	Duration of exposure	Main outcomes
Akefe et al. (2023)	C57BL/6 mice	900 MHz	1.25 W/kg	1 h/d, for gestation period and from days 7–28 of neonatal development	Impaired short-term memory, increased SOD, CAT, and GPx levels, reduced TNF- α , improved neurotransmitter homeostasis (glutamate, serotonin, dopamine, GABA) in Myrtenal-treated mice.
Sharma et al. (2017)	Swiss albino mice	10 GHz	0.1790 W/kg	2 h/d for 15 days	Reduced body and brain weight, increased lipid peroxidation, decreased glutathione, catalase, superoxide dismutase, and protein levels, impaired spatial learning and memory.
B. Tan et al. (2022)		2450 MHz (CW)		12 h/d, one month before fertilization, until the 18th day of the gestation period	Increased pERK, pTau, pJNK, and pP38 levels in the brain; hemorrhagic areas, irregular cellular localization, and vascular abnormalities in fetal and adult female rat brains across all generations.
Bas et al. (2009)	Female Wistar albino rats	900 MHz	2 W/kg	For 60 min/d between the first and last days of gestation.	Reduced total pyramidal cell number in the hippocampus.
Bouji et al. (2016)	Wistar rat	900 MHz (GSM)	6 W/kg	45 min/d for 1 month	No differences in emotional memory, spatial memory, locomotor activity, cerebral IL-1b and IL-6 levels, or GFAP expression in the cortex and hippocampus; reduced anxiety-related behaviors in the elevated plus maze.
Bouji et al. (2020)	Samaritan rat model	900 MHz (GSM)	1.5 W/kg, 6 W/kg	15 min/d, and 45 min /d for 1 month.	No memory changes in AD or control rats; increased in hippocampal heme oxygenase-1 (HO1) and reduced corticosterone in AD rats.
Buttiglione et al. (2007)	Human SH-SY5Y neuroblastoma cells	900 MHz (GSM)	1 W/kg	5, 15 and 30 min, 6 and 24 h	Anti-proliferative activity; increased Egr-1 mRNA levels and MAPK subtype activation; decreased Bcl-2 and survivin mRNA levels; G2-M arrest.
Chaturvedi et al. (2011)	Mice (Parkes Strain)	2.45 GHz continuous	0.03561 W/Kg	2 h/d for 30 days	Increased erythrocytes and leucocytes, significant DNA single-strand breakage in brain cells, and impaired spatial memory in mice.
Daniels et al. (2009)	Sprague-Dawley rat	840 MHz	NC	3 h/d for 13 days	No change in learning ability, reduced locomotor activity, increased grooming and freezing behavior; tendency for higher corticosterone levels.
Dasdag et al. (2012)	Wistar Albino rat	900 MHz	0.17–0.58 W/kg	2 h/d 10 months	Increased beta amyloid protein, protein carbonyl, and malondialdehyde levels.
Dasdag et al. (2015)	Wistar Albino rat	2.4 GHz	141.4 μ W/kg	24 hours/d for 12 months	3.65- and 3.3-fold decrease in miR-106b-5p and miR-107 levels, respectively; unchanged miR-9-5p, miR-29a-3p, and miR-125a-3p levels.
Deshmukh et al. (2013)	Fischer rat	900 MHz	8.5×10^{-5} W/kg	2 h/d for 30 days	Cognitive impairment, increased MDA and protein carbonyl levels, unchanged GSH.
Deshmukh et al. (2015)	Fischer rat	900, 1800 and 2450 MHz	5.953×10^{-4} W/kg, 5.835×10^{-4} W/kg, 6.672×10^{-4} W/kg	2 h/d for 180 days	Reduced cognitive function, increased HSP70 levels and DNA damage.
Elamin et al. (2020)	Wistar albino rats	900 MHz	NC	1 h/d, for 28 days	Reduced total hippocampal pyramidal neurons, increased SOD and CAT activity
El-Kafoury et al. (2023)	Wistar rats	850 and 1900 MHz	NC	2 h/d for 3 weeks	Shorter latency to reach the platform, reduced spatial long-term memory consolidation, decreased Ach, glutamate, and MDA levels, increased Sirt1 and Atg7 gene expression in the hippocampus.
Ertilav et al. (2018)	Female Wistar rats	900, 1800 MHz	0.1 W/kg	1 h/d for 1 year (5 d/week)	Increased mitochondrial oxidative stress, programmed cell death, and Ca ²⁺ entry via TRPV1 activation in hippocampal and dorsal root ganglion neurons.
Garvanova et al. (2020)	Human	GSM	0.36 W/kg	15–20 min/d	Altered brainwave frequency.
Gupta et al. (2018)	Charles–Foster albino male rats	900, 1800, 2450 MHz	0.0227, 0.030 and 0.0616 W/kg	1 h/d for 28 consecutive days	Cognitive deficit with mitochondrial dysfunction, impaired cholinergic function, and increased amyloidogenesis.
Li et al. (2015)	Wistar rat	2.856 GHz	NC	For 6 min three times a week up to 6 weeks	Impaired spatial learning and memory, inhibited brain electrical activity, hippocampal neuronal degeneration, altered amino acid levels, increased 5-HT, and upregulated 5-HT1AR and 5-HT2CR expression.
Hasan et al. (2022)	Swiss albino mice	2400 MHz	2.7 W/kg	40 and 60 minutes/d for 60 days	Increased anxiety-like behavior, hippocampal degeneration, and reduced pyramidal and granule neurons.
Ikinci et al. (2013)	Female Sprague Dawley rat	900 MHz	NA	1 h/d, from the 13th to 21st days of pregnancy	Histopathological changes in the hippocampus and impaired learning behavior.
Jeong et al. (2020)	C57BL/6 mice	1950 MHz	5.0 W/kg	2 h/d and 5 d/week for 8 months	In 20-month-old RF-exposed mice: reduced time spent in the central region (open field test), impaired memory (Y-maze and novel object recognition test). No significant behavioral or memory changes in the 10-month RF exposure group. Chronic RF exposure altered 15 neurogenesis-related genes in the hippocampus, with increased EphA8 and Wnt6 expression but no significant changes in the other 13 genes.

(Continued)

Table 2. Continued.

Reference	Species	Frequency	SAR	Duration of exposure	Main outcomes
Singh et al. (2023)	Wistar rat	2115 MHz	1.51 W/kg	8 h	Increased lipid peroxidation and carbon-centered lipid radicals in the brain, single-stranded DNA damage, reduced BrdU-positive cells in the dentate gyrus, neuronal loss and degeneration in DG, CA3, CA1, and cerebral cortex, with no change in Pro-caspase3 activity.
Keleş et al. (2018)	Male Sprague Dawley rat	900 MHz	0.01 W/kg	1 h/d for 25 days	Impaired pyramidal and granular cell structures, with no changes in learning, memory or locomotor behavior.
Kim et al. (2021)	Human SH-SY5Y neuroblastoma cells	1760 MHz	4 W/kg	4 h/d for 4 days	Reduced growth rate and proliferation, delayed G0/G1 phase, increased Akt and mTOR phosphorylation, elevated total and phosphorylated p53 levels.
Lai et al. (1994)	Sprague–Dawley rat	2450 MHz, (pulsed)	0.6 W/kg	45 min/d for 10 days	Impaired spatial working memory.
Li et al. (2008)	Wistar rats	2.45 GHz (pulsed)	0.7 W/kg	3 h/d for 30 days	Significant deficits in spatial learning and memory, increased plasma corticosterone, GR nuclear translocation, and hippocampal apoptosis. Reduced cognitive impairment and neuronal loss with GR antagonist RU486.
Lu et al. (2012)	Male Wistar rats	2.45 GHz- (pulsed)	0.7 W/kg	3 h/d, for 30 days	Impaired spatial memory.
Luukkonen et al. (2009)	Human SH-SY5Y neuroblastoma cells	872 MHz (GSM and CW)	5 W/kg	1 h	CW RF increased ROS production by menadione, while GSM signal had no effect on ROS and DNA damage.
Marjanovic Cermak et al. (2018)	Human SH-SY5Y neuroblastoma cells	1800 MHz	1.6 W/kg	10, 30 and 60 minutes	Increased ROS, lipid and protein damage.
Odaci et al. (2008)	Female Wistar albino rats	900 MHz	2 W/kg	For 60 min/d between the first and last days of gestation.	Reduced granule cell count in the dentate gyrus.
Shahin et al. (2015)	Swiss strain male mice	2.45 GHz (continuous)	0.0146 W/kg	2 h/d for 15, 30, and 60 days	Increased oxidative/nitrosative stress and apoptosis in hippocampal neurons and non-neuronal cells. Learning and spatial memory deficits worsened with RF exposure duration (15 < 30 < 60 days), linked to reduced hippocampal neuronal arborization and dendritic spines.
Shahin et al. (2018)	Swiss albino mice	2.45 GHz (continuous)	0.0146 W/Kg	2 h/d for 15, 30, and 60 days	Slow learning, increased working and reference memory errors, elevated serum corticosterone, and upregulated CRH, CRH-R1 and i-NOS. Downregulated iGluRs, n-NOS, PSD-95, PKC ϵ , PKA, ERK-p-ERK, CREB and p-CREB.
Sharma et al. (2020)	Wistar rats	900 MHz	0.231 W/kg	1, 2, and 4 h for 90 days	Increased MDA, decreased SOD, CAT and redox enzymes (GSH, GPX, GR, GST, G-6PDH). Reduced AChE, altered neurotransmission, hippocampal degeneration, and impaired working memory.
Sharma et al. (2022)	Wistar rat	2100 MHz	0.453 W/kg	4 h/d, 5 d/week for 3 months	Increased serum triglycerides and cholesterol, disrupted glutathione homeostasis, elevated oxidative stress, altered pro- and anti-inflammatory cytokines, and hippocampal degeneration.
Spandole-Dinu et al. (2023)	BALB/c mice	2.45 GHz	17.86 mW/kg	16 weeks	Increased locomotor activity with no structural or morphological changes. Reduced global DNA methylation.
Stefi et al. (2019)	SH-SY5Y human neuroblastoma cells	1800 MHz	0.23 W/kg	3 times, for 10 min, for 2 days	Increased ROS levels, α -syn monomers, and APP amyloidogenic fragment formation.
Tan et al. (2017)	Wistar Rat	2.856 and 1.5 GHz	1.7 and 1.8 W/kg; 3.3 and 3.7 W/kg	6 min	Impaired spatial learning and memory, fluctuating brain electrical activity, hippocampal morphological changes, reduced neuronal Nissl content, disrupted cholinergic metabolism, cytokine imbalance, altered energy metabolism, and oxidative stress-related protein dysregulation in the hippocampus.
Tang et al. 2015	Sprague-Dawley rat	900 MHz	0.016 (whole body) and 2 W/kg (locally in the head)	3 h/d for 14 or 28 days	Reduced platform crossings and target quadrant time, cellular edema, neuronal organelle degeneration, blood-brain barrier disruption with albumin and HO-1 extravasation in the hippocampus and cortex, increased mmp-1 expression, and ERK dephosphorylation.
Joubert et al. (2008)	Primary cortical neurons	900 MHz (CW)	2 W/kg	24 h	Apoptotic pathway activation via AIF, independent of caspase.
Wang et al. (2013)	Wistar rat	2.856 GHz (pulsed)	3.5, 7 and 35 W/kg	6 min	Impaired spatial learning and memory, reduced population spike amplitudes, hippocampal neuron degeneration, decreased synaptic vesicles, and blurred synaptic intervals.
Wang et al. (2015)	Wistar rat	2.856 GHz (pulsed)	35 W/kg	6 min	Impaired spatial learning and memory, reduced amino acid neurotransmitters, hippocampal neuronal degeneration, increased postsynaptic density, blurred synaptic gaps, and decreased NR1 and NR2B expression.
Wang et al. (2017)	Wistar rat	2.856 GHz	1.75, 3.5 and 7 W/kg	6 min/d, 5 d/week and up to 6 weeks	Impaired spatial learning and memory abilities and EEG disturbances, structural and ultrastructural hippocampal changes, reduced NR2A, NR2B, and p-NR2B expression.
Zhang et al. (2017)	C57BL/6 mice	1800 MHz	2.2 W/kg	6 h/day for 4 weeks,	No changes in depression-like behavior, spatial memory, or brain histology; increased anxiety and reduced GABA and Asp levels.

(Continued)

Table 2. Continued.

Reference	Species	Frequency	SAR	Duration of exposure	Main outcomes
Li et al. (2020)	Pregnant Wistar rats	1800 and 2400 MHz	NC	12 h/d for 21 days	Earlier eye opening, shorter total path length, delayed stopping time, tendency for increased path length, duration, entries and stopping time in the central area. Down-regulated NR2A and NR2B; upregulated NR2D, NR3A, NR3B, NR1, and NR2C in the WiFi group.
Zheng et al. (2023)	Male C57BL/6 N mice	2650 MHz	2.06 W/kg	4 h/d for 28 days	No morphological changes in hippocampal cells; increased serum corticosterone; reduced hippocampal BDNF and GR; no changes in GABA, spatial memory, or depression-like behavior; associated with anxiety like behavior.
Zielinski et al. (2020)	Murine N9 microglial and human SH-SY5Y neuroblastoma cells	935 MHz	4 W/kg	2 and 24 h	Time-dependent increase in ATG5; no changes in apoptosis, LC3B-I/II, pERK, hydrogen peroxide and cytochrome c.

NC: Not calculated.

CW: continuous wave.

d: day, min: minute, h: hour.

Table 3. No effect of RF radiation on Alzheimer's disease.

Reference	Species	Frequency	SAR	Duration of exposure	Main outcomes
Ammari et al. (2008)	Male Sprague–Dawley rat	900 MHz (GSM)	1.5 or 6 W/kg	15 min/d or 45 min/d for 8 or 24 weeks	No changes in spatial memory.
Cassel et al. (2004)	Sprague–Dawley rat	2450 MHz, (pulsed)	0.6 W/kg	45 min/d for 10 days	No impairment in spatial working memory.
Cosquer et al. (2005)	Sprague–Dawley rat	2450 MHz (pulsed)	0.6 W/kg	45 min/d for 10 days	No impairment in spatial working memory.
Cosquer et al. (2005)	Sprague–Dawley rat	2450 MHz (pulsed)	0.6 W/kg	45 min/d for 10 days	No effect on anxiety responses.
Dubreuil et al. (2002)	Male Sprague–Dawley rat	900 MHz-(GSM)	1 and 3.5 W/kg	45 min/d, for 10 and 14 days	No changes in spatial learning.
Dubreuil et al. (2003)	Male Sprague–Dawley rat	900 MHz-(GSM)	1 and 3.5 W/kg	45 min/d, for 10 and 14 days	No changes in spatial and non-spatial memory.
Kang et al. (2014)	U87, PC12 and SH-SY5Y	837 MHz (CDMA) and 1950 MHz (WCDMA)	2 W/kg	2 h	No change in intracellular ROS.
Klose et al. (2014)	Wistar rat	900 MHz (GSM-modulated)	0.7, 2.5 and 10 W/kg.	2 h/d, 5 d/week, from an age of 14 days to 19 months	No harmful effects on development, learning or behavior.
Merola et al. (2006)	Human neuroblastoma cell line LAN-5	900 MHz (GSM)	1 W/kg	24, 48, and 72 h	No change in proliferation, differentiation, or apoptosis.
Shirai et al. (2014)	Sprague–Dawley rat	2.14 GHz (WCDMA)	high (<0.2 4 W/kg), low (<0.08 W/kg)	20 h/d	No abnormalities in biological parameters, including neurological function.
Sienkiewicz et al. (2000)	C57BL/6J mice	900 MHz	0.05 W/kg	45 min/d for 10 days	No effect on spatial learning and working memory.
Joubert et al. (2007)	Primary cortical neurons	900 MHz (GSM)	0.25 W/kg	24 h	No change in Caspase-3 activity or apoptosis.
Von Niederhäusern et al. (2019)	SH-SY5Y neuroblastoma cells	935 MHz	4 W/kg	24 h	No changes in neuronal phenotypes, MAPK, p-Erk1/2, Akt, GSK3 β , or Wnt/ β -catenin signaling.

d: day, min: minute, h: hour.

often report different outcomes, no clear independent relationship between SAR levels or frequency and AD-related effects has been identified. Instead, the findings suggest that RF exposure outcomes are influenced by multiple interacting factors, including modulation type, exposure duration, and biological conditions.

Although attempts have been made to analyze SAR and frequency separately, the available data does not support a simple dose-response relationship or a linear correlation between these parameters and AD progression. Instead, some studies with similar SAR values report opposite effects, while others with different frequency exposures yield comparable results. This highlights the importance of experimental conditions, biological variability, and methodological differences in shaping RF-induced effects on neurodegeneration.

To provide a more structured evaluation, the following sections discuss the studies grouped by their reported effects: those suggesting positive outcomes, those indicating negative consequences, and those showing no significant impact.

Studies demonstrating positive effects

Research investigating the effects of RF radiation on AD has yielded promising results, suggesting potential therapeutic benefits. Several studies suggest that 'RF radiation may exert neuroprotective effects in AD, particularly through mechanisms such as autophagy activation, mitochondrial enhancement, inflammation modulation, and oxidative stress reduction.' Research by Kim et al. (2018) demonstrated that 'RF exposure promotes autophagy in hippocampal neurons,

leading to decreased A β deposition and lower apoptotic activity' (Kim et al. 2018). This aligns with findings from Arendash et al. (2010, 2012) who observed that 'long-term RF exposure reduced A β accumulation in AD transgenic mice, improved cognitive function, and increased neuronal activity, with benefits achieved independently of hyperthermia' (Arendash et al. 2010; 2012). Similarly, Dragicevic et al. (2011) and Mori and Arendash (2011) reported that 'RF exposure enhances mitochondrial function and memory performance, suggesting its role in counteracting early neuronal hypofunction in AD' (Dragicevic et al. 2011; Mori and Arendash 2011).

Beyond its effects on amyloid pathology, RF exposure has been linked to cognitive improvements and neuroinflammation modulation. Sun et al. (2022) found that 'RF exposure led to a significant reduction in A β 42 levels, downregulation of BACE1 expression, and improved learning and memory in AD mice' (Sun et al. 2022). Meanwhile, Son et al. (2018, 2023) reported that 'long-term RF exposure increased glucose metabolism in the hippocampus and amygdala while reducing microgliosis and inflammatory markers such as IL-1 β , Csf1r, CD68, and Ccl6, indicating a potential anti-inflammatory effect' (Son et al. 2018, 2023). Supporting this, Banaceur et al. (2013) observed that 'RF exposure improved cognitive behavior in Alzheimer-like transgenic mice' (Banaceur et al. 2013), while Bayat et al. (2023) demonstrated enhanced memory and synaptic function in vascular dementia models following chronic Wi-Fi exposure (Bayat et al. 2023).

In addition to its effects on cognitive function and neuroinflammation, RF exposure may also play a role in A β processing at the molecular level. Park et al. (2018) investigated the effects of 1950MHz RF exposure on key genes involved in A β metabolism in mouse hippocampal and human neuroblastoma cells. Their findings suggest that 'RF exposure decreased mRNA levels of APP, BACE1, ADAM10, and PSEN1 in hippocampal neurons, although protein levels remained unchanged, highlighting the need for further research into its molecular mechanisms' (Park et al. 2018). Similarly, Ji Jeong et al. (2015) found that 'chronic RF exposure (1950MHz, 5W/kg, 8months) significantly altered A β pathology in AD transgenic mice, but had no effect on normal brains, suggesting a selective impact on disease-related processes' (Ji Jeong et al. 2015).

RF radiation has also been implicated in reducing oxidative stress and protecting against DNA damage. Dasdag et al. (2009) showed that 'RF exposure increased catalase activity and total antioxidant capacity while reducing apoptosis in brain glial cells, suggesting a protective role in oxidative stress regulation' (Dasdag et al. 2009). Additionally, studies by Zeni et al. (2021), Falone et al. (2018), and Sannino et al. (2022) highlighted that 'RF exposure attenuates menadione-induced DNA damage in SH-SY5Y neuroblastoma cells, potentially through heat shock protein 70 (Hsp70) induction and autophagy activation' (Falone et al. 2018; Zeni et al. 2021; Sannino et al. 2022). Furthermore, Kumlin et al. (2007) reported that 'young male rats exposed to 900MHz RF radiation for five weeks showed no neurodegenerative changes but exhibited significantly improved learning and memory abilities in maze tests, suggesting potential cognitive benefits even in the absence of direct neuroprotective effects' (Kumlin et al. 2007).

Transcranial electromagnetic treatment (TEMT)

Transcranial Electromagnetic Treatment (TEMT) has emerged as a potential neuromodulatory approach for combating AD. Unlike traditional electromagnetic stimulation techniques, TEMT utilizes RF waves to target pathological features of AD, including A β and tau oligomers (Cao et al. 2022). Preclinical studies in AD transgenic mice suggest that RF radiation can dissipate intraneuronal A β and tau aggregates, potentially through the weakening of hydrogen bonds and dipole-dipole interactions between oligomeric proteins (Arendash et al. 2010; Dragicevic et al. 2011; Mori and Arendash 2011; Arendash et al. 2012). Additionally, TEMT has been associated with mitochondrial enhancement and neuroinflammation modulation, contributing to its proposed neuroprotective effects (Arendash et al. 2022).

Clinical applications of TEMT have been explored using MemorEM, a noninvasive device allowing patients to undergo RF treatment at home. A study by Arendash et al. (2019) demonstrated that twice-daily TEMT for two months in mild-to-moderate AD patients led to significant improvements in ADAS-cog and Rey AVLT scores, as well as increases in soluble A β 1-40 and A β 1-42 in cerebrospinal fluid (CSF), reduced p-tau/A β 1-42 ratios, and altered plasma oligomeric A β levels. Furthermore, functional brain imaging showed increased connectivity in the cingulate cortex, a key region for cognition (Arendash et al. 2019). Long-term safety assessments indicate that TEMT is well tolerated with no adverse effects over a 2.5-year period (Arendash et al. 2022). Extended TEMT exposure has been linked to reduced CSF levels of p-tau217, A β 1-40, and A β 1-42, as well as plasma tau regulation. Notably, TEMT has also been associated with a reduction in C-reactive protein (CRP), a marker of systemic inflammation, suggesting its broader anti-inflammatory potential. The exact mechanisms underlying these effects remain unclear, but hypotheses suggest that TEMT may influence cytokine secretion from microglia and astrocytes, modulating neuroinflammation (Gary and Chuanhai 2023). A related study investigated Repeated Electromagnetic Field Stimulation (REMFS) in human brain cultures, revealing that REMFS significantly reduced A β 40 and A β 42 peptide levels without inducing cellular damage or affecting secreted A β precursor protein- α (sAPP α) levels (Perez et al. 2021). These findings highlight the potential for electromagnetic-based therapies in mitigating AD pathology through noninvasive, biologically targeted mechanisms.

Overall, TEMT represents a promising therapeutic avenue for AD, demonstrating potential benefits in reducing A β burden, modulating inflammation, and enhancing brain connectivity. However, further clinical trials and mechanistic studies are needed to validate its long-term efficacy and elucidate its precise biological effects.

Studies demonstrating negative effects

Various studies have highlighted the potential neurodegenerative effects of RF exposure on AD. The hippocampus, one of the first brain regions affected in AD, plays a crucial role in memory and learning. RF exposure has been linked to oxidative stress, neuronal apoptosis, mitochondrial dysfunction, and

neurotransmission disturbances, potentially exacerbating AD symptoms.

Ertilav et al. (2018) demonstrated that '900 and 1800 MHz RF exposure elevated TRPV1 currents, induced mitochondrial depolarization, increased oxidative stress, and triggered apoptosis in hippocampal and dorsal root ganglion neurons, along with caspase-3 and caspase-9 activation' (Ertilav et al. 2018). Similarly, Gupta et al. (2018) found that '2450 MHz RF exposure led to cognitive impairment, disrupted mitochondrial function, increased amyloid-beta levels, cytochrome-c release, apoptotic factor activation, acetylcholine depletion, and acetylcholinesterase overactivity' (Gupta et al. 2018). Sharma and Shukla (2020) linked RF exposure to heightened oxidative stress, reduced redox enzyme levels, neurotransmission alterations, hippocampal degeneration, and working memory deficits (Sharma and Shukla 2020). Prolonged exposure to 2100 MHz RF caused hippocampal myelin loss through oxidative stress, compromising neural communication (Sharma et al. 2022).

Histopathological analyses revealed hippocampal neuronal damage despite unchanged cognitive functions (Keleş et al. 2018). Long-term RF exposure led to increased locomotor activity but reduced global DNA methylation without morphological changes in the brain (Spandole-Dinu et al. 2023). Chaturvedi et al. (2011) reported that '2.45 GHz RF exposure in Parkes mice induced DNA strand breaks, elevated erythrocyte and leukocyte counts, and caused spatial memory deficits' (Chaturvedi et al. 2011). Garvanova et al. (2020) analyzed EEG alterations following RF exposure and found that 'holding a mobile phone close to the ear increased left-side brain-wave frequencies to 15 Hz'. This suggests RF radiation may modulate cortical excitability and neural oscillations (Garvanova et al. 2020). Wang et al. (2017) further demonstrated that 'continuous RF exposure impaired spatial memory and induced EEG abnormalities alongside hippocampal structural damage in a dose-dependent manner, implicating NMDAR subunit reductions and NR2B phosphorylation in RF-induced cognitive deficits' (Wang et al. 2017).

Exposure to 2400 MHz RF for 60 days resulted in reduced pyramidal neuron count in the cornu ammonis region, increased anxiety-like behaviors, and hippocampal neuronal degeneration (Hasan et al. 2022). Similarly, 900 MHz RF exposure led to structural alterations and hippocampal pyramidal neuron depletion, while *Garcinia kola* and curcumin provided neuroprotection against these effects (Elamin et al. 2022). Lu et al. (2012) and Lai et al. (1994) demonstrated that '2.45 GHz RF exposure impaired spatial memory, delayed learning, and involved cholinergic and opioid neurotransmitter systems in cognitive decline' (Lai et al. 1994; Lu et al. 2012). Zhang et al. (2017) found that '1.8 GHz RF exposure increased anxiety-like behavior and reduced GABA and aspartic acid levels, though spatial memory and depression-related behaviors remained unaffected' (Zhang et al. 2017).

El-Kafoury et al. (2023) examined the role of lipoic acid (LA) in counteracting RF effects in juvenile rats and found that 'RF-exposed rats initially showed enhanced learning but exhibited long-term deficits in memory consolidation, hippocampal acetylcholine and glutamate imbalances, and upregulation of *Sirt1* and *Atg7* genes, suggesting RF-induced

cognitive alterations may be modulated by LA' (El-Kafoury et al. 2023).

Sharma et al. (2017) investigated the effects of 10 GHz RF radiation on brain development and reported that 'RF exposure led to significant reductions in body and brain weight, increased lipid peroxidation, decreased antioxidant enzyme levels, and spatial learning deficits within 15 days of exposure' (Sharma et al. 2017). Singh et al. (2023) further demonstrated that 'adolescent Wistar rats exposed to 2115 MHz RF for 8 hours exhibited enhanced carbon-centered lipid radical formation, nuclear DNA damage, and impaired neurogenesis, contributing to hippocampal neuronal degeneration' (Singh et al. 2023).

Chronic stress-induced cortisol elevation may accelerate AD onset and symptom progression (Do et al. 2024). Zheng et al. (2023) found that 'RF exposure raised serum cortisol levels, decreased hippocampal BDNF and glucocorticoid receptor expression, and induced anxiety-like behavior' (Zheng et al. 2023). Shahin et al. (2018) reported learning impairments, increased memory errors, elevated corticosterone levels, and upregulated CRH, CRH-R1, and iNOS expression, suppressing hippocampal memory mechanisms (Shahin et al. 2018). Similarly, Li et al. (2008) suggested that 'low-intensity RF exposure increased plasma corticosterone levels and altered hippocampal glucocorticoid receptor function, contributing to cognitive decline' (Li et al. 2008).

Research on aging and AD progression suggests that RF exposure may exacerbate neurodegeneration. Bouji et al. (2016) found that '900 MHz RF exposure did not worsen age-related neurobiological deficits, though older rats displayed spatial memory impairment, anxiety, and increased hippocampal inflammatory markers' (Bouji et al. 2016). In another study, Bouji et al. (2020) reported that 'RF exposure increased hippocampal oxidative stress while reducing corticosterone levels in AD-model rats, suggesting enhanced susceptibility to neurodegeneration' (Bouji et al. 2020).

Deshmukh et al. (2013, 2015) found that '900, 1800, and 2450 MHz RF exposure led to cognitive impairment, oxidative stress, HSP70 upregulation, and DNA damage' (Deshmukh et al. 2013, 2015). Tang et al. (2015) demonstrated that 'RF exposure disrupted blood-brain barrier integrity, impaired spatial memory, and activated the mKp-1/ERK pathway' (Tang et al. 2015). Shahin et al. (2015) reported that 'prolonged RF exposure resulted in learning deficits, hippocampal neuronal degeneration, oxidative stress, and p53-mediated apoptosis' (Shahin et al. 2015). Daniels et al. (2009) found that 'early RF exposure altered behavioral patterns in rat pups and increased corticosterone levels' (Daniels et al. 2009), while prenatal 2.45 GHz RF exposure disrupted MAPK pathways associated with cognitive processes (Tan et al. 2022).

Oxidative stress resulting from RF exposure led to lipid and protein damage, contributing to neurodegenerative changes (Marjanovic Cermak et al. 2018). RF exposure enhanced ROS production and DNA damage in neuroblastoma cells (Luukkonen et al. 2009). Chronic RF exposure activated the Akt/mTOR and p53 pathways, inducing cellular senescence (Kim et al. 2021). Similarly, cell cycle disruptions associated with AD were observed following RF-induced

alterations in MAPK, Bcl-2, and survivin expression, leading to apoptosis and G2-M arrest (Buttiglione et al. 2007).

Long-term studies indicate that 'RF exposure may influence AD-related molecular mechanisms'. Jeong et al. (2020) found that '20-month RF exposure altered neurogenesis-related gene expression and impaired memory in mice' (Jeong et al. 2020). Chronic 2.4 GHz RF exposure reduced miR-106b-5p and miR-107 levels, contributing to neurodegenerative changes (Dasdag et al. 2015). Long-term 900 MHz RF exposure elevated beta-amyloid levels, oxidative stress, and protein damage in the brain (Dasdag et al. 2012).

Tan et al. (2017) reported that 'RF-induced cognitive impairment was power-dependent rather than frequency-dependent' (Tan et al. 2017). Wang et al. (2013, 2015) observed persistent spatial memory deficits, hippocampal synaptic degeneration, neurotransmitter imbalances, and NMDAR subunit downregulation following RF exposure (Wang et al. 2013, 2015). Li et al. (2015) linked chronic RF exposure to serotonin system disruption, potentially contributing to cognitive impairment (Li et al. 2015).

Prenatal and early-life RF exposure studies suggest developmental risks. Maternal 900 MHz RF exposure impaired hippocampal neuronal development (Odaci et al. 2008; Bas et al. 2009). Li et al. (2020) found that 'Wi-Fi and RF exposure altered hippocampal NMDAR expression in offspring, affecting cognitive and behavioral outcomes' (Li et al. 2020). Similarly, prenatal 900 MHz RF exposure impaired learning and memory (Ikinci et al. 2013), while myrtenal showed protective effects against RF-induced neurodevelopmental damage (Akefe et al. 2023).

RF exposure has been linked to neurodegenerative pathways, including beta-amyloid accumulation, oxidative stress, and apoptosis. Stefi et al. (2019) found that '1800 MHz RF exposure increased amyloidogenic activity and oxidative stress in neuroblastoma cells' (Stefi et al. 2019). RF exposure induced autophagy but not apoptosis in neuronal cells (Zielinski et al. 2020), while continuous wave RF activated caspase-independent apoptosis pathways (Joubert et al. 2008).

Collectively, these studies demonstrate that 'RF exposure induces oxidative stress, apoptosis, neurotransmitter imbalances, structural damage, and cognitive decline, particularly targeting hippocampal neurons'. While further research is required to define safe exposure levels, these findings suggest that RF radiation may contribute to AD progression and neurodegenerative mechanisms.

Studies showing no effect

Several studies indicate that RF exposure does not significantly impact mechanisms associated with AD, suggesting that RF radiation is not a contributing factor to its onset or progression.

Oxidative stress is a key driver in AD pathology. However, exposure to 837 MHz CDMA and 1950 MHz WCDMA RF radiation (SAR: 2 W/kg, 2 hours) did not alter intracellular ROS levels in neuronal cells (U87, PC12, and SH-SY5Y), either alone or in combination with oxidative stress inducers (Kang et al. 2014). Similarly, 900 MHz RF exposure for

24 hours did not affect apoptosis rates in primary cultured neurons from embryonic Wistar rats, with no significant changes observed immediately post-exposure or 24 hours later (Joubert et al. 2007). Likewise, neuroblastoma cells exposed to 900 MHz RF radiation for up to 72 hours showed no alterations in proliferation, differentiation, or apoptosis (Merola et al. 2006).

Behavioral studies also reported no significant cognitive effects. Rats exposed to 2.45 GHz RF radiation for a shorter duration at a lower SAR level showed no anxiety-related changes in the elevated plus maze test (Cosquer, Galani, et al. 2005). Similarly, 900 MHz RF radiation had no effect on spatial learning or working memory in mice performing food-motivated tasks (Sienkiewicz et al. 2000). Other studies confirmed that RF exposure did not impair spatial working memory in rats (Cassel et al. 2004; Cobb et al. 2004; Cosquer, Kuster, et al. 2005). Furthermore, head-only exposure to 900 MHz RF for 45 minutes per day did not affect spatial learning or non-spatial memory in rats (Dubreuil et al. 2002, 2003). Long-term exposure to GSM 900 MHz RF (45 min or 15 min, SAR: 1.5 W/kg or 6 W/kg) did not cause spatial memory deficits in the radial-arm maze (Ammari et al. 2008).

Lifespan studies also support the absence of RF-induced cognitive impairment. In a study tracking rats across juvenile, adult, and presenile stages, RF exposure at high SAR values showed no detrimental effects on development, learning skills, or behavior (Klose et al. 2014). Similarly, three-generation exposure (F0, F1, F2 mothers and F1, F2, F3 offspring) to 2.14 GHz W-CDMA signals (20 hours/day) did not induce neurological or biological abnormalities (Shirai et al. 2014).

Mitochondrial dysfunction is a critical factor in AD progression. However, SH-SY5Y cells exposed to 935 MHz RF radiation showed no alterations in signaling pathways related to neuronal differentiation, neurodegeneration, or mitochondrial function (Von Niederhäusern et al. 2019). While RF exposure under glucose deprivation conditions impaired mitochondrial respiration, glutathione levels and mitochondrial fission/fusion markers remained unchanged, suggesting that RF effects may only emerge under conditions of extreme metabolic stress.

Overall, these findings indicate that 'RF radiation does not significantly impact spatial learning, memory, apoptosis, neuronal proliferation, differentiation, oxidative stress, or mitochondrial dysfunction in AD models'. While further research on cumulative and stress-enhanced RF exposure is warranted, current evidence does not support a causal link between RF radiation and AD pathology.

Conclusion

The studies examining the impacts of RF radiation on the pathogenesis of AD present diverse and sometimes conflicting findings. Some research suggests that RF exposure may have adverse effects on AD development, potentially exacerbating the condition through mechanisms such as oxidative stress, blood-brain barrier damage, impairment of cellular

functions, and aberrant build-up of tau and amyloid beta proteins. Conversely, several studies indicate that RF radiation might offer protective or therapeutic benefits, such as improving brain mitochondrial function and enhancing cognitive performance, thereby positively impacting AD pathology.

The variability in these findings underscores the intricate nature of RF radiation's influence on neurodegenerative diseases. Further clinical research and epidemiological studies are essential to comprehensively comprehend the long-term effects of RF radiation on health outcomes, particularly in relation to AD. Understanding the mechanisms of these impacts can pave the way for innovative strategies to mitigate and manage such diseases.

This review synthesizes current findings and aims to provide a comprehensive framework for future research. By emphasizing the complex interactions between RF radiation and AD, we hope to guide future studies toward more conclusive evidence regarding the role of RF exposure in the pathogenesis of AD.

Continued investigation into the mechanisms underlying these effects, including oxidative stress, blood-brain barrier integrity, and cellular function, will be crucial. As scientific work in this area progresses, a clearer understanding of RF radiation's impact on AD can be developed, potentially leading to improved prevention and treatment strategies for neurodegenerative disorders.

Finally, it can be suggested that evaluating both the harmful and therapeutic effects of radiofrequencies together is a more useful approach. Because, when the articles discussed in this review are evaluated in general, they indicate that parameters such as duration, frequency, amplitude, exposure time, modulation, etc. have important roles in the harm or benefit duo.

Author contributions statement

Hava Bektas and Suleyman Dasdag contributed equally to the conceptualization and design of this review. Hava Bektas conducted the literature search, data analysis, and drafting of the manuscript. Suleyman Dasdag provided critical revisions and contributed to the interpretation of the results. Both authors reviewed and approved the final manuscript.

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References

- Akefe IO, Nyan ES, Adegoke VA, Lamidi IY, Ameh MP, Chidiebere UC, Ubah SA, Ajayi IE. 2023. Myrtenal improves memory deficits in mice exposed to radiofrequency-electromagnetic radiation during gestational and neonatal development via enhancing oxido-inflammatory, and neurotransmitter functions. *Heliyon*. 9(4):e15321. doi:10.1016/j.heliyon.2023.e15321.
- Ammari M, Jacquet A, Lecomte A, Sakly M, Abdelmelek H, De Seze R. 2008. Effect of head-only sub-chronic and chronic exposure to 900-MHz GSM electromagnetic fields on spatial memory in rats. *Brain Inj*. 22(13-14):1021–1029. doi:10.1080/02699050802530599.
- Arendash G, Abulaban H, Steen S, Andel R, Wang Y, Bai Y, Baranowski R, McGarity J, Scritsmier L, Lin X, et al. 2022. Transcranial electromagnetic treatment stops Alzheimer's disease cognitive decline over a 2½-year period: a pilot study. *Medicines*. 9(8):42. doi:10.3390/medicines9080042.
- Arendash G, Cao C, Abulaban H, Baranowski R, Wisniewski G, Becerra L, Andel R, Lin X, Zhang X, Wittwer D, et al. 2019. A clinical trial of transcranial electromagnetic treatment in Alzheimer's disease: cognitive enhancement and associated changes in cerebrospinal fluid, blood, and brain imaging. *J Alzheimers Dis*. 71(1):57–82. doi:10.3233/JAD-190367.
- Arendash GW, Mori T, Dorsey M, Gonzalez R, Tajiri N, Borlongan C. 2012. Electromagnetic treatment to old Alzheimer's mice reverses β -amyloid deposition, modifies cerebral blood flow, and provides selected cognitive benefit. *PLoS One*. 7(4):e35751. doi:10.1371/journal.pone.0035751.
- Arendash GW, Sanchez-Ramos J, Mori T, Mamcarz M, Lin X, Runfeldt M, Wang L, Zhang G, Sava V, Tan J, et al. 2010. Electromagnetic field treatment protects against and reverses cognitive impairment in Alzheimer's disease mice. *J Alzheimers Dis*. 19(1):191–210. doi:10.3233/JAD-2010-1228.
- Bai R, Guo J, Ye X-Y, Xie Y, Xie T. 2022. Oxidative stress: The core pathogenesis and mechanism of Alzheimer's disease. *Ageing Res Rev*. 77:101619. doi:10.1016/j.arr.2022.101619.
- Banaceur S, Banasr S, Sakly M, Abdelmelek H. 2013. Whole body exposure to 2.4GHz WIFI signals: effects on cognitive impairment in adult triple transgenic mouse models of Alzheimer's disease (3xTg-AD). *Behav Brain Res*. 240:197–201. doi:10.1016/j.bbr.2012.11.021.
- Bas O, Odaci E, Kaplan S, Acer N, Uçok K, Colakoglu S. 2009. 900MHz electromagnetic field exposure affects qualitative and quantitative features of hippocampal pyramidal cells in the adult female rat. *Brain Res*. 1265:178–185. doi:10.1016/j.brainres.2009.02.011.
- Bayat M, Karimi N, Karami M, Haghighi AB, Bayat K, Akbari S, Haghighi M. 2023. Chronic exposure to 2.45GHz microwave radiation improves cognition and synaptic plasticity impairment in vascular dementia model. *Int J Neurosci*. 133(2):111–122. doi:10.1080/0207454.2021.1896502.
- Bouji M, Lecomte A, Gamez C, Blazy K, Villégier A-S. 2016. Neurobiological effects of repeated radiofrequency exposures in male senescent rats. *Biogerontology*. 17(5-6):841–857. doi:10.1007/s10522-016-9654-8.
- Bouji M, Lecomte A, Gamez C, Blazy K, Villégier A-S. 2020. Impact of cerebral radiofrequency exposures on oxidative stress and corticosterone in a rat model of Alzheimer's disease. *J Alzheimers Dis*. 73(2):467–476. doi:10.3233/JAD-190593.
- Buttiglione M, Roca L, Montemurno E, Vitiello F, Capozzi V, Cibelli G. 2007. Radiofrequency radiation (900 MHz) induces Egr-1 gene ex-

- pression and affects cell-cycle control in human neuroblastoma cells. *J Cell Physiol.* 213(3):759–767. doi:10.1002/jcp.21146.
- Cao C, Abulaban H, Baranowski R, Wang Y, Bai Y, Lin X, Shen N, Zhang X, Arendash GW. 2022. Transcranial electromagnetic treatment “rebalances” blood and brain cytokine levels in Alzheimer’s patients: a new mechanism for reversal of their cognitive impairment. *Front Aging Neurosci.* 14:829049. doi:10.3389/fnagi.2022.829049.
- Cassel J-C, Cosquer B, Galani R, Kuster N. 2004. Whole-body exposure to 2.45 GHz electromagnetic fields does not alter radial-maze performance in rats. *Behav Brain Res.* 155(1):37–43. doi:10.1016/j.bbr.2004.03.031.
- Chaturvedi CM, Singh VP, Singh P, Basu P, Singaravel M, Shukla RK, Dhawan A, Pati AK, Gangwar RK, Singh S. 2011. 2.45 GHz (CW) microwave irradiation alters circadian organization, spatial memory, DNA structure in the brain cells and blood cell counts of male mice, *mus musculus*. *Pier B.* 29:23–42. doi:10.2528/PIERB11011205.
- Cobb BL, Jauchem JR, Adair ER. 2004. Radial arm maze performance of rats following repeated low level microwave radiation exposure. *Bioelectromagnetics.* 25(1):49–57. doi:10.1002/bem.10148.
- Cosquer B, Galani R, Kuster N, Cassel J-C. 2005. Whole-body exposure to 2.45 GHz electromagnetic fields does not alter anxiety responses in rats: a plus-maze study including test validation. *Behav Brain Res.* 156(1):65–74. doi:10.1016/j.bbr.2004.05.007.
- Cosquer B, Kuster N, Cassel J-C. 2005. Whole-body exposure to 2.45 GHz electromagnetic fields does not alter 12-arm radial-maze with reduced access to spatial cues in rats. *Behav Brain Res.* 161(2): 331–334. doi:10.1016/j.bbr.2005.02.026.
- Daniels WM, Pitout IL, Afullo TJ, Mabandla MV. 2009. The effect of electromagnetic radiation in the mobile phone range on the behaviour of the rat. *Metab Brain Dis.* 24(4):629–641. doi:10.1007/s11011-009-9164-3.
- Dasdag O, Adalier N, Dasdag S. 2020. Electromagnetic radiation and Alzheimer’s disease. *Biotechnol Biotechnol Equip.* 34(1):1087–1094. doi:10.1080/13102818.2020.1820378.
- Dasdag S, Akdag MZ, Erdal ME, Erdal N, Ay OI, Ay ME, Yilmaz SG, Tasdelen B, Yegin K. 2015. Effects of 2.4 GHz radiofrequency radiation emitted from Wi-Fi equipment on microRNA expression in brain tissue. *Int J Radiat Biol.* 91(7):555–561. doi:10.3109/09553002.2015.1028599.
- Dasdag S, Akdag MZ, Kizil G, Kizil M, Cakir DU, Yukus B. 2012. Effect of 900 MHz radio frequency radiation on beta amyloid protein, protein carbonyl, and malondialdehyde in the brain. *Electromagn Biol Med.* 31(1):67–74. doi:10.3109/15368378.2011.624654.
- Dasdag S, Akdag MZ, Ulukaya E, Uzunlar AK, Ocak AR. 2009. Effect of mobile phone exposure on apoptotic glial cells and status of oxidative stress in rat brain. *Electromagn Biol Med.* 28(4):342–354. doi:10.3109/15368370903206556.
- Deshmukh PS, Banerjee BD, Abegaonkar MP, Megha K, Ahmed RS, Tripathi AK, Mediratta PK. 2013. Effect of low level microwave radiation exposure on cognitive function and oxidative stress in rats. *Deshmukh PS, Nasare N, Megha K, Banerjee BD, Ahmed RS, Singh D, Abegaonkar MP, Tripathi AK, Mediratta PK. 2015. Cognitive impairment and neurogenotoxic effects in rats exposed to low-intensity microwave radiation. Int J Toxicol.* 34(3):284–290. doi:10.1177/1091581815574348.
- Do BK, Jang J-H, Park GH. 2024. Effects of corticosterone on beta-amyloid-induced cell death in SH-SY5Y cells. *Biomol Ther (Seoul).* 32(1):77–83. doi:10.4062/biomolther.2023.133.
- Dragicevic N, Bradshaw P, Mamcarz M, Lin X, Wang L, Cao C, Arendash G. 2011. Long-term electromagnetic field treatment enhances brain mitochondrial function of both Alzheimer’s transgenic mice and normal mice: a mechanism for electromagnetic field-induced cognitive benefit? *Neuroscience.* 185:135–149. doi:10.1016/j.neuroscience.2011.04.012.
- Dubreuil D, Jay T, Edeline J-M. 2002. Does head-only exposure to GSM-900 electromagnetic fields affect the performance of rats in spatial learning tasks? *Behav Brain Res.* 129(1-2):203–210. doi:10.1016/s0166-4328(01)00344-8.
- Dubreuil D, Jay T, Edeline J-M. 2003. Head-only exposure to GSM 900-MHz electromagnetic fields does not alter rat’s memory in spatial and non-spatial tasks. *Behav Brain Res.* 145(1-2):51–61. doi:10.1016/s0166-4328(03)00100-1.
- El-Kafoury BMA, Abdel-Hady EA, El Bakly W, Elayat WM, Hamam GG, Abd El Rahman SMM, Lasheen NN. 2023. Lipoic acid inhibits cognitive impairment induced by multiple cell phones in young male rats: role of Sirt1 and Atg7 pathway. *Sci Rep.* 13(1):18486. doi:10.1038/s41598-023-44134-2.
- Elamin AAE, Deniz OG, Kaplan S. 2022. The effects of Gum Arabic, curcumin (*Curcuma longa*) and *Garcinia kola* on the rat hippocampus after electromagnetic field exposure: a stereological and histological study. *J Chem Neuroanat.* 120:102060. doi:10.1016/j.jchemneu.2021.102060.
- Ertilav K, Uslusoy F, Ataizi S, Nazıroğlu M. 2018. Long term exposure to cell phone frequencies (900 and 1800 MHz) induces apoptosis, mitochondrial oxidative stress and TRPV1 channel activation in the hippocampus and dorsal root ganglion of rats. *Metab Brain Dis.* 33(3):753–763. doi:10.1007/s11011-017-0180-4.
- Falzone S, Sannino A, Romeo S, Zeni O, Santini SJ, Rispoli R, Amicarelli F, Scarfi MR. 2018. Protective effect of 1950 MHz electromagnetic field in human neuroblastoma cells challenged with menadione. *Sci Rep.* 8(1):13234. doi:10.1038/s41598-018-31636-7.
- Firdous SM, Khan SA, Maity A. 2024. Oxidative stress-mediated neuroinflammation in Alzheimer’s disease. *Naunyn Schmiedeberg Arch Pharmacol.* 397(11):8189–8209. doi:10.1007/s00210-024-03188-3.
- Garvanova M, Garvanov I, Borissova D. 2020. The influence of electromagnetic fields on human brain. Proceedings of the 21st International Symposium on Electrical Apparatus & Technologies (SIELA), Bourgas, Bulgaria, 03–06 June 2020. IEEE. doi:10.1109/SIELA49118.2020.9167099.
- Gary A, Chuanhai C. 2023. Transcranial electromagnetic wave treatment: a fountain of healthy longevity? *Int J Mol Sci.* 24(11):9652.
- Guo T, Zhang D, Zeng Y, Huang TY, Xu H, Zhao Y. 2020. Molecular and cellular mechanisms underlying the pathogenesis of Alzheimer’s disease. *Mol Neurodegener.* 15(1):40. doi:10.1186/s13024-020-00391-7.
- Gupta SK, Mesharam MK, Krishnamurthy S. 2018. Electromagnetic radiation 2450 MHz exposure causes cognition deficit with mitochondrial dysfunction and activation of intrinsic pathway of apoptosis in rats. *J Biosci.* 43(2):263–276. doi:10.1007/s12038-018-9744-7.
- Hasan I, Jahan MR, Islam MN, Islam MR. 2022. Effect of 2400 MHz mobile phone radiation exposure on the behavior and hippocampus morphology in Swiss mouse model. *Saudi J Biol Sci.* 29(1):102–110. doi:10.1016/j.sjbs.2021.08.063.
- Ibanez L, Cruchaga C, Fernández MV. 2021. Advances in genetic and molecular understanding of Alzheimer’s disease. *Genes (Basel).* 12(8):1247. doi:10.3390/genes12081247.
- Ikinci A, Odaci E, Yildirim M, Kaya H, Akça M, Hanci H, Aslan A, Sönmez OF, Bas O. 2013. The effects of prenatal exposure to a 900 megahertz electromagnetic field on hippocampus morphology and learning behavior in rat pups. *NeuroQuantology.* 30(3):582–590.
- Jeong YJ, Son Y, Choi HD, Kim N, Lee YS, Ko YG, Lee HJ. 2020. Behavioral changes and gene profile alterations after chronic 1,950-MHz radiofrequency exposure: An observation in C57BL/6 mice. *Brain Behav.* 10(11):e01815. doi:10.1002/brb3.1815.
- Ji Jeong Y, Kang G-Y, Hwa Kwon J, Choi H-D, Pack J-K, Kim N, Lee Y-S, Lee H-J. 2015. 1950 MHz electromagnetic fields ameliorate A β pathology in Alzheimer’s disease mice. *Curr Alzheimer Res.* 12(5):481–492. doi:10.2174/156720501205150526114448.
- Joubert V, Bourthoumieu S, Leveque P, Yardin C. 2008. Apoptosis is induced by radiofrequency fields through the caspase-independent mitochondrial pathway in cortical neurons. *Radiat Res.* 169(1):38–45. doi:10.1667/RR1077.1.
- Joubert V, Leveque P, Cueille M, Bourthoumieu S, Yardin C. 2007. No apoptosis is induced in rat cortical neurons exposed to GSM phone fields. *Bioelectromagnetics.* 28(2):115–121. doi:10.1002/bem.20274.
- Kang KA, Lee HC, Lee J-J, Hong M-N, Park M-J, Lee Y-S, Choi H-D, Kim N, Ko Y-G, Lee J-S. 2014. Effects of combined radiofrequency radiation exposure on levels of reactive oxygen species in neuronal cells. *J Radiat Res.* 55(2):265–276. doi:10.1093/jrr/rrt116.
- Keleş Aİ, Yıldırım M, Gedikli Ö, Çolakoğlu S, Kaya H, Baş O, Sönmez OF, Odacı E. 2018. The effects of a continuous 1-ha day 900-MHz electromagnetic field applied throughout early and mid-adolescence on hippocampus morphology and learning behavior in late adoles-

- cent male rats. *J Chem Neuroanat.* 94:46–53. doi:10.1016/j.jchemneu.2018.08.006.
- Kim JH, Jeon S, Choi H-D, Lee J-H, Bae J-S, Kim N, Kim H-G, Kim K-B, Kim HR. 2021. Exposure to long-term evolution radiofrequency electromagnetic fields decreases neuroblastoma cell proliferation via Akt/mTOR-mediated cellular senescence. *J Toxicol Environ Health A.* 84(20):846–857. doi:10.1080/15287394.2021.1944944.
- Kim JH, Yu D-H, Kim H-J, Huh YH, Cho S-W, Lee J-K, Kim H-G, Kim HR. 2018. Exposure to 835 MHz radiofrequency electromagnetic field induces autophagy in hippocampus but not in brain stem of mice. *Toxicol Ind Health.* 34(1):23–35. doi:10.1177/0748233717740066.
- Klose M, Grote K, Spathmann O, Streckert J, Clemens M, Hansen VW, Lerchl A. 2014. Effects of early-onset radiofrequency electromagnetic field exposure (GSM 900 MHz) on behavior and memory in rats. *Radiat Res.* 182(4):435–447. doi:10.1667/RR13695.1.
- Kumlin T, Iivonen H, Miettinen P, Juvonen A, van Groen T, Puranen L, Pitkääho R, Juutilainen J, Tanila H. 2007. Mobile phone radiation and the developing brain: behavioral and morphological effects in juvenile rats. *Radiat Res.* 168(4):471–479. doi:10.1667/RR1002.1.
- Lai H, Horita A, Guy AW. 1994. Microwave irradiation affects radial-arm maze performance in the rat. *Bioelectromagnetics.* 15(2):95–104. doi:10.1002/bem.2250150202.
- Lameth J, Gervais A, Colin C, Lévêque P, Jay TM, Edeline J-M, Mallat M. 2017. Acute neuroinflammation promotes cell responses to 1800 MHz GSM electromagnetic fields in the rat cerebral cortex. *Neurotox Res.* 32(3):444–459. doi:10.1007/s12640-017-9756-3.
- Li H-J, Peng R-Y, Wang C-Z, Qiao S-M, Yong Z, Gao Y-B, Xu X-P, Wang S-X, Dong J, Zuo H-Y, et al. 2015. Alterations of cognitive function and 5-HT system in rats after long term microwave exposure. *Physiol Behav.* 140:236–246. doi:10.1016/j.physbeh.2014.12.039.
- Li M, Wang Y, Zhang Y, Zhou Z, Yu Z. 2008. Elevation of plasma corticosterone levels and hippocampal glucocorticoid receptor translocation in rats: a potential mechanism for cognition impairment following chronic low-power-density microwave exposure. *J Radiat Res.* 49(2):163–170. doi:10.1269/jrr.07063.
- Li Y, Chang J, Chen X, Liu J, Zhao L. 2023. Advances in the study of APOE and innate immunity in Alzheimer's disease. *J Alzheimers Dis.* 93(4):1195–1210. doi:10.3233/JAD-230179.
- Li ZQ, Zhang Y, Wan YM, Zhou Q, Liu C, Wu HX, Mu YZ, He YF, Rauniyar R, Wu XN. 2020. Testing of behavioral and cognitive development in rats after prenatal exposure to 1800 and 2400 MHz radiofrequency fields. *J Radiat Res.* 61(2):197–206. doi:10.1093/jrr/rz097.
- Lin JC. 2022. Carcinogenesis from chronic exposure to radio-frequency radiation. *Front Public Health.* 10:1042478. doi:10.3389/fpubh.2022.1042478.
- Lu Y, Xu S, He M, Chen C, Zhang L, Liu C, Chu F, Yu Z, Zhou Z, Zhong M. 2012. Glucose administration attenuates spatial memory deficits induced by chronic low-power-density microwave exposure. *Physiol Behav.* 106(5):631–637. doi:10.1016/j.physbeh.2012.04.019.
- Luukkonen J, Hakulinen P, Mäki-Paakkanen J, Juutilainen J, Naarala J. 2009. Enhancement of chemically induced reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells by 872 MHz radiofrequency radiation. *Mutat Res.* 662(1-2):54–58. doi:10.1016/j.mrfmmm.2008.12.005.
- Marjanovic Cermak AM, Pavicic I, Trosic I. 2018. 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.* 53(2):132–138. doi:10.1080/10934529.2017.1383124.
- Merola P, Marino C, Lovisolo G, Pinto R, Laconi C, Negroni A. 2006. Proliferation and apoptosis in a neuroblastoma cell line exposed to 900 MHz modulated radiofrequency field. *Bioelectromagnetics.* 27(3):164–171. doi:10.1002/bem.20201.
- Moloney CM, Lowe VJ, Murray ME. 2021. Visualization of neurofibrillary tangle maturity in Alzheimer's disease: a clinicopathologic perspective for biomarker research. *Alzheimer's Dement.* 17(9):1554–1574. doi:10.1002/alz.12321.
- Mori T, Arendash G. 2011. Long-term electromagnetic field treatment increases brain neuronal activity: linkage to cognitive benefit and therapeutic implications for Alzheimer's disease. *J Alzheimer's Dis Parkinsonism.* 1(2):2–5.
- Narayanan SN, Jetti R, Kesari KK, Kumar RS, Nayak SB, Bhat PG. 2019. Radiofrequency electromagnetic radiation-induced behavioral changes and their possible basis. *Environ Sci Pollut Res Int.* 26(30):30693–30710. doi:10.1007/s11356-019-06278-5.
- Odaci E, Bas O, Kaplan S. 2008. Effects of prenatal exposure to a 900 MHz electromagnetic field on the dentate gyrus of rats: a stereological and histopathological study. *Brain Res.* 1238:224–229. doi:10.1016/j.brainres.2008.08.013.
- Park J, Kwon JH, Kim N, Song K. 2018. Effects of 1950 MHz radiofrequency electromagnetic fields on Aβ processing in human neuroblastoma and mouse hippocampal neuronal cells. *J Radiat Res.* 59(1):18–26. doi:10.1093/jrr/rxx045.
- Perez FP, Maloney B, Chopra N, Morisaki JJ, Lahiri DK. 2021. Repeated electromagnetic field stimulation lowers amyloid-beta peptide levels in primary human mixed brain tissue cultures. *Sci Rep.* 11(1):621. doi:10.1038/s41598-020-77808-2.
- Sannino A, Scarfi MR, Dufossée M, Romeo S, Poeta L, Prouzet-Mauléon V, Priault M, Zeni O. 2022. Inhibition of autophagy negates radiofrequency-induced adaptive response in SH-SY5Y neuroblastoma cells. *Int J Mol Sci.* 23(15):8414. doi:10.3390/ijms23158414.
- Scheltens P, De Strooper B, Kivipelto M, Holstege H, Chételet G, Teunissen CE, Cummings J, van der Flier WM. 2021. Alzheimer's disease. *The Lancet.* 397(10284):1577–1590. doi:10.1016/S0140-6736(20)32205-4.
- Shahin S, Banerjee S, Singh SP, Chaturvedi CM. 2015. 2.45 GHz microwave radiation impairs learning and spatial memory via oxidative/nitrosative stress induced p53-dependent/independent hippocampal apoptosis: molecular basis and underlying mechanism. *Toxicol Sci.* 148(2):380–399. doi:10.1093/toxsci/kfv205.
- Shahin S, Banerjee S, Swarup V, Singh SP, Chaturvedi CM. 2018. From the cover: 2.45-GHz microwave radiation impairs hippocampal learning and spatial memory: involvement of local stress mechanism-induced suppression of iGluR/ERK/CREB signaling. *Toxicol Sci.* 161(2):349–374. doi:10.1093/toxsci/kfx221.
- Sharma A, Kesari KK, Saxena VK, Sisodia R. 2017. Ten gigahertz microwave radiation impairs spatial memory, enzymes activity, and histopathology of developing mice brain. *Mol Cell Biochem.* 435(1-2):1–13. doi:10.1007/s11010-017-3051-8.
- Sharma A, Shrivastava S, Singh A, Gupte SS, Rathour A, Reshi MS, Shukla S. 2022. Evidences of the radiofrequency exposure on the antioxidant status, potentially contributing to the inflammatory response and demyelination in rat brain. *Environ Toxicol Pharmacol.* 94:103903. doi:10.1016/j.etap.2022.103903.
- Sharma S, Shukla S. 2020. Effect of electromagnetic radiation on redox status, acetylcholine esterase activity and cellular damage contributing to the diminution of the brain working memory in rats. *J Chem Neuroanat.* 106:101784. doi:10.1016/j.jchemneu.2020.101784.
- Shirai T, Imai N, Wang J, Takahashi S, Kawabe M, Wake K, Kawai H, Watanabe S, Furukawa F, Fujiwara O. 2014. Multigenerational effects of whole body exposure to 2.14 GHz W-CDMA cellular phone signals on brain function in rats. *Bioelectromagnetics.* 35(7):497–511. doi:10.1002/bem.21871.
- Shirbandi K, Khalafi M, Bevelacqua JJ, Sadeghian N, Adiban S, Zarandi FB, Mortazavi SA, Mortazavi SH, Mortazavi SMJ, Welsh JS. 2023. Exposure to low levels of radiofrequency electromagnetic fields emitted from cell-phones as a promising treatment of Alzheimer's Disease: a scoping review study. *J Biomed Phys Eng.* 13(1):3–16. doi:10.31661/jbpe.v0i0.2109-1398.
- Sienkiewicz ZJ, Blackwell RP, Haylock RG, Saunders RD, Cobb BL. 2000. Low-level exposure to pulsed 900 MHz microwave radiation does not cause deficits in the performance of a spatial learning task in mice. *Bioelectromagnetics: Journal of the Bioelectromagnetics Society, The Society for Physical Regulation in Biology and Medicine, The European Bioelectromagnetics Association.* 21(3):151–158. doi:10.1002/(SICI)1521-186X(200004)21:3<151::AID-BEM1>3.0.CO;2-Q.
- Singh KV, Gautam R, Meena R, Nirala JP, Jha SK, Rajamani P. 2020. Effect of mobile phone radiation on oxidative stress, inflammatory response, and contextual fear memory in Wistar rat. *Environ Sci Pollut Res Int.* 27(16):19340–19351. doi:10.1007/s11356-020-07916-z.
- Singh KV, Prakash C, Nirala JP, Nanda RK, Rajamani P. 2023. Acute radiofrequency electromagnetic radiation exposure impairs neuro-

- genesis and causes neuronal DNA damage in the young rat brain. *Neurotoxicology*. 94:46–58. doi:[10.1016/j.neuro.2022.11.001](https://doi.org/10.1016/j.neuro.2022.11.001).
- Son Y, Kim JS, Jeong YJ, Jeong YK, Kwon JH, Choi HD, Pack JK, Kim N, Lee YS, Lee HJ. 2018. Long-term RF exposure on behavior and cerebral glucose metabolism in 5xFAD mice. *Neurosci Lett*. 666:64–69. doi:[10.1016/j.neulet.2017.12.042](https://doi.org/10.1016/j.neulet.2017.12.042).
- Son Y, Park HJ, Jeong YJ, Choi HD, Kim N, Lee HJ. 2023. Long-term radiofrequency electromagnetic fields exposure attenuates cognitive dysfunction in 5xFAD mice by regulating microglial function. *Neural Regen Res*. 18(11):2497–2503. doi:[10.4103/1673-5374.371379](https://doi.org/10.4103/1673-5374.371379).
- Spandole-Dinu S, Catrina A-M, Voinea OC, Andone A, Radu S, Haidoiu C, Călborean O, Popescu DM, Săhăianu V, Baltag O, et al. 2023. Pilot study of the long-term effects of radiofrequency electromagnetic radiation exposure on the mouse brain. *Int J Environ Res Public Health*. 20(4):3025. doi:[10.3390/ijerph20043025](https://doi.org/10.3390/ijerph20043025).
- Stefi AL, Margaritis LH, Skouroliaou AS, Vassilacopoulou D. 2019. Mobile phone electromagnetic radiation affects amyloid precursor protein and α -synuclein metabolism in SH-SY5Y cells. *Pathophysiology*. 26(3-4):203–212. doi:[10.1016/j.pathophys.2019.02.004](https://doi.org/10.1016/j.pathophys.2019.02.004).
- Sun Q, Li S, Zhu P, Yin J, Zhou H, Zhao X, Liu Q, Dong G, Cheng J, Tian Z. 2022. Beneficial effects of 1500 MHz microwave on old 5x FAD mice.
- Tan B, Canturk Tan F, Yalcin B, Dasdag S, Yegin K, Yay AH. 2022. Changes in the histopathology and in the proteins related to the MAPK pathway in the brains of rats exposed to pre and postnatal radiofrequency radiation over four generations. *J Chem Neuroanat*. 126:102187. doi:[10.1016/j.jchemneu.2022.102187](https://doi.org/10.1016/j.jchemneu.2022.102187).
- Tan S, Wang H, Xu X, Zhao L, Zhang J, Dong J, Yao B, Wang H, Zhou H, Gao Y, et al. 2017. Study on dose-dependent, frequency-dependent, and accumulative effects of 1.5 GHz and 2.856 GHz microwave on cognitive functions in Wistar rats. *Sci Rep*. 7(1):10781. doi:[10.1038/s41598-017-11420-9](https://doi.org/10.1038/s41598-017-11420-9).
- Tang J, Zhang Y, Yang L, Chen Q, Tan L, Zuo S, Feng H, Chen Z, Zhu G. 2015. Exposure to 900 MHz electromagnetic fields activates the mep-1/ERK pathway and causes blood-brain barrier damage and cognitive impairment in rats. *Brain Res*. 1601:92–101. doi:[10.1016/j.brainres.2015.01.019](https://doi.org/10.1016/j.brainres.2015.01.019).
- Tsoly A, Saliev T, Abzhanova E, Turgambayeva A, Kaiyrykyzy A, Akishev M, Saparbayev S, Umbayev B, Askarova S. 2019. The effects of mobile phone radiofrequency electromagnetic fields on β -amyloid-induced oxidative stress in human and rat primary astrocytes. *Neuroscience*. 408:46–57. doi:[10.1016/j.neuroscience.2019.03.058](https://doi.org/10.1016/j.neuroscience.2019.03.058).
- Von Niederhäusern N, Ducray A, Zielinski J, Murbach M, Mevissen M. 2019. Effects of radiofrequency electromagnetic field exposure on neuronal differentiation and mitochondrial function in SH-SY5Y cells. *Toxicol in Vitro*. 61:104609. doi:[10.1016/j.tiv.2019.104609](https://doi.org/10.1016/j.tiv.2019.104609).
- Wang H, Peng R, Zhou H, Wang S, Gao Y, Wang L, Yong Z, Zuo H, Zhao L, Dong J, et al. 2013. Impairment of long-term potentiation induction is essential for the disruption of spatial memory after microwave exposure. *Int J Radiat Biol*. 89(12):1100–1107. doi:[10.3109/09553002.2013.817701](https://doi.org/10.3109/09553002.2013.817701).
- Wang Q, Zhu G-h, Xie D-h, Wu W-j, Hu P. 2015. Taurine enhances excitability of mouse cochlear neural stem cells by selectively promoting differentiation of glutamatergic neurons over GABAergic neurons. *Neurochem Res*. 40(5):924–931. doi:[10.1007/s11064-015-1546-9](https://doi.org/10.1007/s11064-015-1546-9).
- Wang S, Wang S, Asgar J, Joseph J, Ro JY, Wei F, Campbell JN, Chung M-K. 2017. Ca^{2+} and calpain mediate capsaicin-induced ablation of axonal terminals expressing transient receptor potential vanilloid 1. *J Biol Chem*. 292(20):8291–8303. doi:[10.1074/jbc.M117.778290](https://doi.org/10.1074/jbc.M117.778290).
- Yap LE, Hunt JE, Turner RS. 2024. Aging as a target for the prevention and treatment of Alzheimer's disease. *Front Neurol*. 15:1376104. doi:[10.3389/fneur.2024.1376104](https://doi.org/10.3389/fneur.2024.1376104).
- Zeni O, Romeo S, Sannino A, Palumbo R, Scarfi MR. 2021. Evidence of bystander effect induced by radiofrequency radiation in a human neuroblastoma cell line. *Environ Res*. 196:110935. doi:[10.1016/j.envres.2021.110935](https://doi.org/10.1016/j.envres.2021.110935).
- Zhang JP, Zhang KY, Guo L, Chen QL, Gao P, Wang T, Li J, Guo GZ, Ding GR. 2017. Effects of 1.8 GHz radiofrequency fields on the emotional behavior and spatial memory of adolescent mice. *Int J Environ Res Public Health*. 14(11):1344. doi:[10.3390/ijerph14111344](https://doi.org/10.3390/ijerph14111344).
- Zheng R, Zhang X, Gao Y, Gao D, Gong W, Zhang C, Dong G, Li Z. 2023. Biological effects of exposure to 2650 MHz electromagnetic radiation on the behavior, learning, and memory of mice. *Brain Behav*. 13(6):e3004. doi:[10.1002/brb3.3004](https://doi.org/10.1002/brb3.3004).
- Zielinski J, Ducray AD, Moeller AM, Murbach M, Kuster N, Mevissen M. 2020. Effects of pulse-modulated radiofrequency magnetic field (RF-EMF) exposure on apoptosis, autophagy, oxidative stress and electron chain transport function in human neuroblastoma and murine microglial cells. *Toxicol In Vitro*. 68:104963. doi:[10.1016/j.tiv.2020.104963](https://doi.org/10.1016/j.tiv.2020.104963).