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Adverse effects of cell phone radiation on Purkinje cells of the cerebellum of pregnant Balb/c mice

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Original Article

ABSTRACT

Background: Pregnant women often use mobile phones for communication and health-related information. The World Health Organization (WHO) recommends minimizing mobile phone use during pregnancy to reduce maternal and child health risks. Electromagnetic waves (EMW) from cell phones can be absorbed by the brain and may affect neuronal function.
Objective: This study aimed to evaluate the size, histology, and number of Purkinje cells in the cerebellum of pregnant mice exposed to cell phone EMW compared to unexposed controls.
Methods: Fourteen pregnant Balb/c mice were randomly divided into exposed (EG) and control (CG) groups. The EG was exposed to an electromagnetic wave with a specific absorption rate (SAR) of 1.74 W/kg for 24 hours per day from gestation day 0 to 19. On day 20, cerebellar tissues were harvested and processed with hematoxylin–eosin (HE) staining for histological assessment.
Results: The mean Purkinje cell size in the EG (0.069 ± 0.009 mm) was slightly smaller than in the CG (0.070 ± 0.007 mm), but this difference was not statistically significant ($p = 0.741$). However, the Purkinje cell count was significantly lower in the EG (6.160 ± 0.746) than in the CG (7.800 ± 0.764; $p < 0.001$), indicating that EMW exposure reduced Purkinje cell numbers.
Conclusion: Exposure to cell phone EMW during pregnancy led to a significant reduction in Purkinje cell numbers in the cerebellum, while cell size remained unchanged. These findings suggest that prenatal exposure to EMW may negatively affect fetal cerebellar development. This underscores the need for pregnant women to be cautious with mobile phone use and highlights the importance of further research to clarify the risks to maternal and fetal health.

INTRODUCTION

In today's era, humans rely heavily on cell phones for communication and information. According to a previous survey, participants' average daily time spent on gadgets rose from 4.75 hours before lockdown to 11.36 hours during lockdown.[1] Currently, 95% of the global population of seven billion lives in areas covered by mobile networks.[2] Mobile phones have completely changed people's lifestyles, with facilities ranging from receiving and making calls, taking photos, and storing data to accessing email and the Internet.[3]

Modern science extensively uses mobile phone technology to support learning, research, and information dissemination. With the Global System for Mobile Communications (GSM), mobile phones are the most commonly used technology, operating in the 900 MHz and 1800 MHz frequency bands.[4] The future of public health is also likely to become increasingly digital.[5] Pregnant women often use mobile phones to make daily chores easier and find information about health and pregnancy. Pregnant women widely use mobile phones and apps to access health

information, monitor fetal development, and manage their health. These tools provide personalized information, which can help prevent pregnancy-related complications and improve maternal health outcomes.[6] Mobile phone-based messaging treatments might enhance the utilization of maternal health care by addressing issues such as restricted access, financial constraints, discourteous treatment, lack of procedural privacy, and negative attitudes.[7]

There are established limits for cell phone use to minimize potential adverse health effects. Several organizations, such as the European Committee for Electrotechnical Standardization (CENELEC) in 1995 and the International Commission on Non-Ionizing Radiation Protection (ICNIRP) in 2020, provide guidelines and recommendations on exposure limits, primarily assessing thermal and non-thermal effects.[8] According to Cleveland and Ulcek, non-thermal effects occur when the electromagnetic wave (EMW) radiation intensity is sufficiently low. The energy involved does not significantly increase the temperature of cells, tissues, or organisms but may induce subtle physical or biochemical changes.[9]

The WHO advises limiting mobile phone use in pregnant women to emergencies due to potential health risks. EMW can irregularly affect ion channels in cell membranes, altering intracellular ion and Ca^{2+} concentrations.[10,11] Non-thermal microwave radiation may trigger harmful changes in cellular metabolism. In vivo and in vitro studies report notable increases in ROS (reactive oxygen species) and nitrogen oxide in cells exposed to non-thermal intensity levels. Both in vivo and in vitro studies have observed substantial increases in reactive oxygen species and nitrogen oxide production in cells exposed to non-thermal intensity levels.[12,13] Therefore, the various findings strongly support the idea that EMW directly influence the formation of free radical molecules. This includes the production of excessive reactive oxygen and nitrogen species, as well as a decrease in the body's ability to counteract them with antioxidants .[10, 12, 13.]

European and American organizations establish maximum Specific Absorption Rate (SAR) limits for each nation's cell phone releases. Absorption at frequencies over 300 MHz impacts body regions proximal to the emitting device. Consequently, the highest local SAR limit is regarded as essential. The SAR is typically described as the mean value within a tissue volume. However, the absorbed power by the entire human body, split by body mass, is derived solely from a whole-body average (SARwba).[14] According to ICNIRP guidelines, the safe limit for SAR values for phone cell devices is a maximum of 2 W/kg, the same as the limit the Council of the European Union set.[9] Meanwhile, the SAR limit from US Federal Communications Commission (FCC) is 1.6W/kg.[15]

Not all mobile phone models comply with the SAR limit of 1.6 W/kg or less, raising concerns about the health implications of radiation. The WHO has recognized EMW as one of the most widespread, and rapidly proliferating ecological hazards.[16] Microwave radiation is a type of EMW with frequencies between 300 MHz and 300 GHz. It is widely used across diverse sectors, including communications, industry, healthcare, and military applications. Prior research has demonstrated that microwave radiation can harm the structure and function of the nervous, heart, reproductive, and endocrine systems, jeopardizing human health.[10,11,12,17] A separate investigation with pregnant mice showed heightened oxidative stress and morphological changes at the embryo implantation site in the placenta following exposure to 2.45 GHz EMW at a specific absorption rate of 0.023 W/kg for 2 hours over 45 days. The outcome was reproductive dysfunction, indicated by implantation failure or embryo resorption, potentially attributable to elevated ROS production.[18] Some studies have suggested maternal complications due to cell phone use, although more research is needed to confirm the causal relationship. Maternal EMW exposure has been linked to pregnancy disorders, including miscarriage, stillbirth, premature labor, alterations in sex ratio, and congenital anomalies. Previous study among 1063 pregnant women in San Francisco mentioned that EMW exposure caused significant increase of abortion rates with elevated levels of maximal magnetic field exposure during regular activities.[19] Other studies have reported that prenatal exposure to higher EMW can reduce birth weight, head circumference, upper-arm circumference, abdominal circumference, and skinfold thickness measurements of the dorsal region, triceps, and abdominal area.[14] However, Wilson et al. reported that exposure to these waves was not associated with conception and pregnancy outcomes. Therefore, it remains unclear whether exposure to these waves during pregnancy is

destructive to the fetus. The mechanism of its changes in fetal growth after exposure is also not completely understood.[20]

EMW radiation can damage brain nerve cells, potentially impairing neurological function. Cell phones emit EMW in their surroundings when in use.. These waves can be absorbed by the skull and brain, directly influencing neuronal discharges. The research findings concerning the impact of EMW on neuronal discharges are inconsistent. Some research indicates that EMW may adversely impact the brain's electrical activity, while others propose no correlation. Others provide evidence that cell phones influence the permeability of the blood-brain barrier.[21]

The cerebellum contains Purkinje cells, a neuronal population highly vulnerable to cell death due to inherent genetic disorders or extrinsic toxic, hypoxic, ischemic, and traumatic injuries. Purkinje cell degeneration results in cerebellar dysfunction, as evidenced by gait and postural problems. Programmed cell death contributes to the pathophysiology of neurodegenerative disorders characterized by Purkinje cell degeneration.[22] This study is vital because pregnancy complications can have a detrimental impact, such as problems in the mother, which can affect not only the mother herself but also the unborn child.

This study focuses on Purkinje cell disorders in the cerebellum of pregnant mice exposed to cell phone EMW and aims to determine the number and size of Purkinje cells in these mice compared with untreated controls, which were rarely studied. Unlike most previous study about behavioral or biochemical effects, this study highlights direct structural changes at the cellular level in the brain because of cell phones EMW.

METHODS

Animal experimental

Fourteen pregnant Balb/c mice were randomly allocated into two groups (Fig. 1: a control group (n=7) and a treatment group (n=7)). Before the experiment, 21 Balb/c mice (19 females and two males; body weight 25 ± 5 g) were obtained for breeding. The animals were maintained under controlled laboratory conditions at 24°C ± 2°C, 50–60% relative humidity, and a 12 h light/dark cycle. A standard pellet diet and water were provided ad libitum. During the breeding phase, one male and two females were co-housed overnight. The presence of a vaginal plug the following morning indicated gestation day 0 (GD0), after which male mice were separated. Successful fertilization was further confirmed by a gradual maternal weight gain of 3–4 g between GD10 and GD13. Conversely, an abrupt decline in body weight was interpreted as an indication of pregnancy failure.[23]

Fourteen confirmed pregnant mice were housed in individual polypropylene cages (45 × 45 × 25 cm) lined with dry wood shavings, which were replaced every three days. The control group (CG) was not exposed to any EMW. In contrast, the experimental group (EG) was exposed to EMW emitted by a standard cell phone with a specific absorption rate (SAR) of 1.74 W/kg for 24 h daily from GD0 to GD19. On GD20, all animals were sacrificed, and the fetal cerebella were carefully dissected. Brain tissues were processed using standard paraffin embedding protocols.

All procedures were conducted in accordance with the ethical standards approved by the Medical and Health Research Ethics Commission, Faculty of Medicine, Universitas Diponegoro (Approval No. 116/EC-H/KEPK/FK-UNDIP/IX/2023)

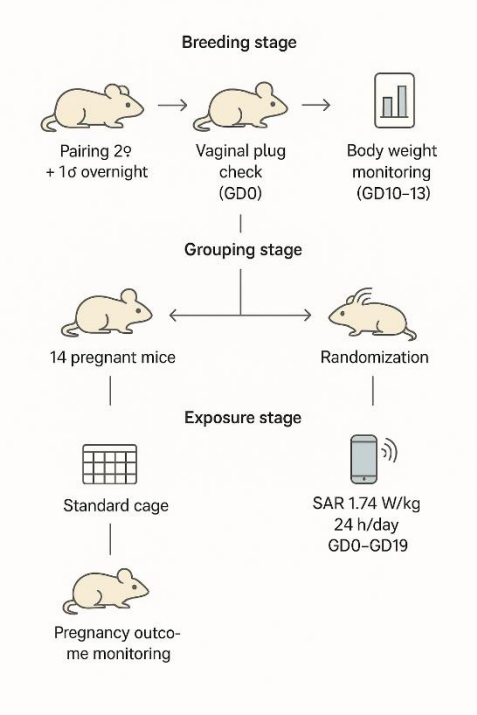


Figure 1. Animal experimental workflow

Research sample

Mice were euthanized via ketamine injection on the 20th day of gestation. The cerebellum was immediately excised.

Histological examination

The cerebellum was preserved in 4% paraformaldehyde for histological analysis, dehydrated through graded alcohols, cleared in xylene, and embedded in paraffin using established protocols. Serial coronal sections (5 µm thickness) were prepared using a rotary microtome and stained with HE. Histological evaluation focused on the morphology and density of Purkinje cells. Microscopic observations were performed using an Olympus CX23 microscope (Olympus, Japan) under 400× magnification. Quantitative measurements included Purkinje cell size (µm) and cell counts (number per field of view), assessed in randomly selected regions of the cerebellar cortex. Purkinje cell size was evaluated using an Olympus EP50 digital microscope and its associated software.

Statistical analysis

Statistical analysis was performed using the non-parametric Kruskal–Wallis test followed by Mann–Whitney post hoc comparisons, as the data did not meet normality assumptions. A p-value of <0.05 was considered statistically significant. Data was analyzed using SPSS version 26.0.

RESULTS

Cerebellum Purkinje cell count and size

Statistical analysis was performed using non-parametric tests because the data did not follow a normal distribution (Shapiro–Wilk, $p < 0.05$). Intergroup comparisons were therefore conducted using the Mann–Whitney U test. The results are presented as mean ± standard deviation (SD) for descriptive purposes, while the hypothesis testing was based on median distribution.

The average Purkinje cell size in the EG was slightly smaller than that of the CG; however, this difference was not statistically significant ($p = 0.741$). In contrast, the Purkinje cell count was significantly lower in the EG than in the CG ($p < 0.001$). These findings indicate that EMW

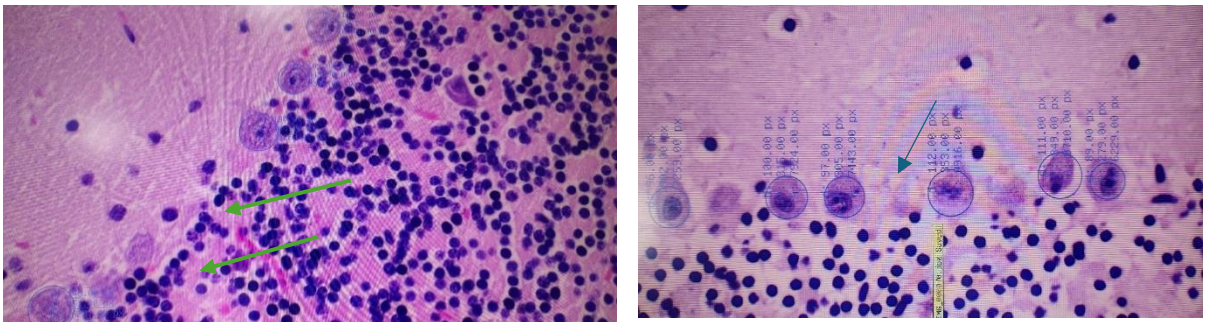
exposure did not alter Purkinje cell morphology, but significantly decreased the number of Purkinje cells (Table 1).

Table 1. Histological analysis of cerebellum

Group	Purkinje Cell Size (mm) Mean ± SD	p-value	Purkinje Cell Count (number) Mean ± SD	p-value
EG	0.069 ± 0.009	0.741	6.160 ± 0.746	0.000*
CG	0.070 ± 0.007		7.800 ± 0.764	

*p < 0.05 considered statistically significant
EG = Exposed group; CG = Control group; SD = Standard deviation

Figure 1 shows the differences in the histology of Purkinje cells between the control and treatment groups in the mouse cerebellum. In the treatment group, Purkinje cells disappear, as evidenced by the significant distance between their rows. On the other hand, the Purkinje cells are not far apart in the control group.



(a) (b)

Figure 1. Histology of the mouse cerebellum, Haematoxylin-eosin staining. (a) Low magnification (100x) view showing the distribution of Purkinje cells in EG (b) High magnification (400×) illustrating the method used to measure Purkinje cell size. Green arrows indicate missing Purkinje cells, while blue arrows demonstrate the measurement approach.

DISCUSSION

This study demonstrated a significant reduction in the number of Purkinje cells in the cerebellum due to EMW exposure at an absorption rate of 1.74 W/kg during pregnancy. However, no significant difference in cell size was observed. These findings suggest that prenatal EMW exposure may impair neurodevelopment by affecting neuronal survival rather than cell morphology.

The significant rise in cell phone use has generated substantial apprehension about its potential detrimental effects, including maternal complications .[24] As indicated by prior research, cerebellar interference in pregnant female mice may reduce the number or size of Purkinje cells. The biological impacts of this exposure on brain development and the associated mechanisms are inadequately clarified. Exposure to EMW before and after birth has been documented to induce several disruptions in the physiological, morphological, and behavioral characteristics of animal species. [25]. Previous studies found that the number of Purkinje cells in the female rat cerebellum has decreased after 28 days of 900 MHz radiation.[26,27] Another study has suggested that cell phone EMW induces increased neurodegeneration in Purkinje cells within the cerebellum of rats.[28] Research has shown that cerebellar disorders can reduce Purkinje cell size. Apoptosis, or type I programmed cell death, can be triggered by abnormal increases in intracellular calcium concentration [Ca2+].[29] Stress markedly reduces the volume of the granule layer, accompanied by a decline in numerical density and the overall quantity of granule cells in the cerebellum.[30] The abnormalities were reduced Purkinje cells, vacuolization of neurons and glial cells, and interstitial edema in the cerebellum.[31] Exposure to 900 MHz EMW has provided evidence of causation of

blood-brain barrier damage, induction of glial reactivity, and initiation of biochemical modifications in the rat brain.[32]

Xu et al. showed that exposure to RF electromagnetic fields at 1,800 MHz induced oxidative damage to mitochondrial DNA and altered neuronal activity.[33] Following exposure to EMW, Dindic et al. observed biochemical and histological alterations in the rat brain.[34] Multiple studies reported EMW from mobile phones negatively impacts tissue and enzyme activity, indicating that 12 weeks of EMW exposure could disrupt myelin in the cerebral cortices of C57BL/6[35,36] mice. EMW exposure for 5 hours a day resulted in hyperactivity, myelin damage in cortical neurons, and activation of autophagy in cortical cell bodies. This exposure specifically led to marked increases in both autophagy transcriptional and protein levels, as well as autophagosome accumulation. The investigation by Saikhedkar et al. revealed that radio waves induced neurodegeneration by increasing ROS, a consequence of the depletion of enzymatic and non-enzymatic antioxidants and increased lipid peroxidation.[37]

The EMW has been shown to induce oxidative stress in cerebellar Purkinje cells, leading to cellular damage. Oxidative stress arises from an imbalance between ROS production and the organism's capacity to neutralize them. This imbalance may lead to cellular damage, encompassing DNA damage, protein oxidation, and lipid peroxidation. Numerous studies have shown that exposure to EMW, especially in the radiofrequency range, can elevate the synthesis of reactive oxygen species in the brain. A survey of neonatal mice exposed to mobile phone radiation revealed significant elevations in oxidative stress markers, including malondialdehyde (MDA), alongside reductions in antioxidant enzymes, such as superoxide dismutase (SOD) and glutathione (GSH), in the cerebellum.[26,38] A study in rats exposed to 900 MHz EMW revealed increased levels of thiobarbituric acid-reactive substances (TBARS) and reduced total antioxidant capacity (TA) in the cerebellum, indicating oxidative stress.[26]

The precise processes via which EMW generate oxidative stress remain incompletely elucidated. However, multiple paths have been suggested. A potential mechanism involves impairment of voltage-gated ion channels (VGICs) in the cell membrane, leading to an influx of calcium ions (Ca^{2+}) into the cell. The elevation of intracellular Ca^{2+} can stimulate nitric oxide synthases (NOS), resulting in the synthesis of nitric oxide (NO•), which may react with superoxide anion ($O_2•^-$) to generate peroxynitrite ($ONOO^-$), a potent oxidant.[39] Another suggested mechanism involves activation of NADPH oxidase, a principal source of ROS in cells. Exposure to EMW has been shown to enhance NADPH oxidase activity, leading to the generation of superoxide anions ($O_2•^-$).[39] Furthermore, EMWs can interfere with the mitochondrial electron transport chain, resulting in electron leakage and the generation of ROS.[40]

The oxidative stress induced by EMWs can have several consequences for cerebellar Purkinje cells. ROS can damage cellular components, including DNA, proteins, and lipids, leading to cellular dysfunction and death. For example, ROS can cause DNA strand breaks, leading to genetic mutations and apoptosis if not appropriately repaired.[41] Additionally, it can oxidize proteins to form protein carbonyls, which are markers of oxidative stress.[41]

Apoptosis, or programmed cell death, is another mechanism by which EMW can damage cerebellar Purkinje cells. Apoptosis is a meticulously regulated process that includes a sequence of molecular events, including the activation of caspases, a family of proteases that cleave specific cellular substrates, resulting in cell death. Numerous investigations have shown that EMW exposure can trigger apoptosis in cerebellar Purkinje cells. A study on rats exposed to 900 MHz EMW revealed heightened expression of caspase-3, a principal executioner caspase, in Purkinje cells.[42] A further investigation involving neonatal mice subjected to mobile phone radiation revealed increased DNA fragmentation and apoptotic cell death within the cerebellum.[38]

Apoptosis triggered by EMW in Purkinje cells is believed to be facilitated by the mitochondria-dependent caspase-3 pathway. This process entails the release of cytochrome c from the mitochondria into the cytosol, which associates with Apaf-1 and caspase-9, resulting in the formation of the apoptosome, which then activates caspase-3.[42,43] Exposure to EMW has been demonstrated to induce mitochondrial malfunction, characterized by the depletion of mitochondrial membrane potential and the liberation of cytochrome c, resulting in the activation

of caspases and subsequent apoptosis.[42,44] Besides the mitochondria-dependent process, EMWs can trigger apoptosis via alternative mechanisms, including activating the p53 tumor suppressor protein. p53 is a transcription factor that modulates the expression of pro-apoptotic genes, such as Bax, and anti-apoptotic genes, such as Bcl-2. Exposure to EMW has been shown to elevate p53 and Bax expression while diminishing Bcl-2 expression, thereby shifting the equilibrium toward apoptosis.[43] The death of cerebellar Purkinje cells can lead to substantial functional repercussions, as these cells are essential for motor coordination and learning. The degeneration of Purkinje cells may lead to cerebellar ataxia, characterized by impaired coordination and balance.[25,45]

Besides oxidative damage and apoptosis, EMW can also impair synaptic function in cerebellar Purkinje cells. Synaptic disruption may compromise neuronal communication, adversely affecting motor coordination and cognitive function. One route by which EMW might impair synaptic function is by altering the electrophysiological properties of Purkinje cells. A rats exposed to 900 MHz EMW revealed alterations in the membrane current-voltage (I/V) characteristics of Purkinje cells, including decreased spontaneous firing and a shift in the I/V curve.[21] These alterations can influence the capacity of Purkinje cells to convey signals to neighboring neurons, resulting in compromised synaptic function.

EMW might also impair synapse function by altering the expression and activity of ion channels. A study of rat cerebellar granule cells exposed to EMW revealed increased sodium currents (I_Na) and altered voltage-gated sodium channel (VGSC) activity.[46] These alterations can influence neuronal excitability and synaptic signal transmission. EMW can potentially interfere with synapse function by modifying neurotransmitter release. A study on rats exposed to 2450 MHz EMW revealed diminished acetylcholine (ACh) levels and heightened acetylcholinesterase (AChE) activity, the enzyme responsible for ACh degradation, in the hippocampus.[44] Reductions in ACh levels can hinder cholinergic neurotransmission, which is essential for cognitive function and motor coordination.

Interference of EMW with synapse activity can result in substantial functional repercussions, including compromised motor coordination, learning, and memory. Research on rats exposed to 2450 MHz EMW revealed cognitive impairments, including diminished performance in the Y-maze paradigm, a spatial memory and learning test.[44]

This study has several limitations that should be acknowledged. First, the sample size was relatively small, which may restrict the generalizability of the findings. Second, Second, although nonparametric statistical methods were used to address data distribution, only mean ± SD values were presented for descriptive purposes; future studies should also report median and interquartile range to enhance statistical accuracy. Third, the experiment was limited to histological assessment of Purkinje cell number and size, without evaluating molecular or biochemical markers such as oxidative stress parameters, apoptotic signaling pathways, or calcium homeostasis, which could provide mechanistic insights. Fourth, exposure was restricted to a single frequency and specific absorption rate (SAR); therefore, the results cannot be extrapolated to other EMW frequencies or exposure durations. Finally, behavioral outcomes were not assessed, which may have provided a more comprehensive understanding of the functional impact of prenatal EMW exposure on cerebellar development.

The implications of our findings extend beyond animal models and are highly relevant to maternal health. Mobile phone use has become ubiquitous, with pregnant women frequently exposed to EMW daily. Although the safety of such exposure remains controversial, the reduction in Purkinje cell numbers observed in this study suggests that prenatal EMW exposure could pose a potential risk to fetal neurodevelopment. The cerebellum plays a critical role in motor coordination and cognitive processing; therefore, disturbances in cerebellar integrity during gestation may have long-term consequences for neurobehavioral outcomes in offspring.

Epidemiological studies in humans remain limited, but preliminary evidence has suggested associations between maternal EMW exposure and adverse pregnancy outcomes, including increased risks of miscarriage, low birth weight, and neurobehavioral alterations in children. While direct extrapolation from animal data to humans should be approached with caution, our

results provide experimental support for the hypothesis that prenatal EMW exposure may constitute an environmental risk factor during Pregnancy.

Thus, this study underscores the importance of continued investigation into the potential neurological consequences of EMW exposure in pregnant women. A better understanding of this relationship may inform public health recommendations and contribute to strategies to minimize prenatal exposure to potentially harmful EMW.

CONCLUSION

Prenatal exposure to EMW significantly reduced the number of Purkinje cells in the cerebellum, although cell size was unaffected. These findings suggest that EMW exposure during pregnancy may impair cerebellar development and potentially influence neurological function. Given the widespread use of mobile phones, precautionary measures for pregnant women may be warranted. Further studies, particularly in human populations, must clarify the underlying mechanisms and assess the potential risks to maternal and fetal health.

CONFLICT OF INTEREST

The authors declare that the research was conducted without any commercial or financial relationships that could be construed as a potential conflict of interest.

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DATA AVAILABILITY

This published article and its supplementary information files include all data generated or analyzed during this study.

SUPPLEMENTAL DATA

All data supporting the findings of this study are fully available within the article and its supplementary materials. No additional data are required or withheld.

AUTHOR CONTRIBUTIONS

DA conceived the study, formulated the experimental protocol, executed the animal experiments, gathered and analysed the data, and composed the initial publication. NS oversaw the entire research process, authenticated the study design, and rigorously reviewed the work for intellectual substance. HS aided in developing the scientific framework, offered specialised insights on biomedical matters, and evaluated the paper for scientific precision. S oversaw laboratory procedures, directed the analysis of histological and biochemical data, and offered methodological assistance during the experimental phase. All authors have reviewed and endorsed the final version of the text.

DECLARATION OF USING AI IN THE WRITING PROCESS

The authors used AI-assisted tools to support specific tasks in preparing this manuscript. SPSS software was utilized for statistical data analysis. QuillBot and DeepL were employed for paraphrasing and translation purposes to ensure clarity and consistency in language. Grammarly was used to check sentence structure and grammar accuracy. No generative AI tools were used to write or interpret the manuscript's scientific content.

LIST OF ABBREVIATIONS

CENELEC: European Committee for Electrotechnical Standardization; Ca^{2+}: Calcium ion; EMF: Electromagnetic Field; EMW: Electromagnetic Wave; FCC: Federal Communications Commission, GSM: Global System for Mobile Communications; HE:Hematoxylin-Eosin; ICNIRP: International Commission on Nonionizing Radiation Protection; mHealth: Mobile Health. NO: Nitric Oxide; ROS: Reactive Oxygen Species,

SAR: Specific Absorption Rate; WHO: World Health Organization;

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