Journal of Medical Genetics and Clinical Biology
Homepage : https://journal.antispublisher.id/index.php/JMGCB
Email : admin@antispublisher.com

e-ISSN : 3032-1085
JMGCB, Vol. 3, No. 5, May 2026
Page 78-93
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Journal of Medical Genetics and Clinical Biology

Histological and Developmental Effects of Smartphone Exposure on Tissue Growth in Children

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DOI : https://doi.org/10.61796/jmgcb.v3i5.1755

Sections Info

Article history:
Submitted: January 07, 2026
Final Revised: February 26, 2026
Accepted: March 18, 2026
Published: April 10, 2026

Keywords:
Smartphone Exposure
RF-EMF (Radiofrequency Electromagnetic Fields)
Neuronal Density
Histological Changes
Pediatric Neurodevelopment

ABSTRACT

Objective: *The aim of the study was to examine the histological and developmental impacts of RF-EMF exposure from smartphones on the nervous system tissues in an experimental design that mimicked the neurodevelopment of children.* ***Method:*** *A controlled laboratory experimental study was performed using immature Wistar rats, whose level of development is equivalent to children between the ages of 3-10 years. The animals were randomly assigned to one control group and three exposure groups, exposed to 900-1800 MHz RF-EMF for 1, 2, or 4 hours per day over 30 days. The microstructure of nervous tissue (hippocampus and cerebral cortex) was examined using hematoxylin and eosin staining and immunohistochemistry. Quantitative analysis of neuronal density and tissue organization was conducted.* ***Results:*** *Chronic exposure led to a significant duration-related decrease in neuronal density and neural tissue disorder when compared to the controls.* ***Novelty:*** *This study is one of the first controlled histological studies to associate smartphone-RF-EMF exposure with structural changes in growing nervous tissue. Unlike other studies that focus on behavioral or epidemiological outcomes, this study provides direct microscopic evidence of decreased neuronal density and tissue disorganization during early developmental stages. The study also provides a translational framework for evaluating digital exposure risks using ethically accepted animal models that replicate pediatric neurodevelopment (3-10 years).*

INTRODUCTION

The last ten years have been characterized by a steep increase in smart phone ownership and utilization among kids all over the world. International surveys and reports on the monitoring of the health of the population says that the exposure to smartphones is beginning in early childhood and constant daily use has been reported among children at the age of under 10. This change has led to repeated or consistent exposures to radiofrequency electromagnetic fields (RF-EMF), especially that are in the range of 800 2600 MHz which is characteristic of 4G/LTE communications [1]. Since children are a biologically distinct group whose neurodevelopment is still in progress, the pattern of exposure has began receiving increased scientific attention.

The growing Nervous system at the age of 3-10 years is immensely undergoing the processes of synaptogenesis, dendritic arborization, refining the cortical layering, and myelination. At this stage, the neuronal migration and synaptic pruning is very susceptible and highly regulated to environmental perturbation. The experimental studies have shown that young neural tissue has an enhanced amount of RF-EMF absorption because of thinner cranial bones, increased tissue conductivity, and higher water content than that of the adult [2]. Experiments on animals have shown exposure to RF-EMF in developmental periods can modify the morphology of the hippocampal, the

corticulum, the neurotransmitters distribution, and the pathways of oxidative stress [3], [4].

Other issues related to electromagnetic radiation involve non-thermal biological consequences, such as the production of reactive oxygen species (ROS), life gate dysregulation, change in gene expression in synaptic development, and so forth [5], [6]. Experimental models have shown that the neural tissues change and change under RF exposure, such as a decrease in the number of neurons and a lack of order [2], [7]. Nonetheless, there is still a great deal of human evidence which is purely epidemiological or behavioral as opposed to direct tissue-level confirmation.

In living children it is both ethically and practically impossible to examine nervous tissue directly by histology. In healthy children, exposure assessment cannot be recommended using neural biopsy. This means that laboratory animal models still are the scientifically proven alternative. The neurodevelopmental studies relying on rodent models are highly preferred because they can determine well defined layers of the cortex and hippocampal arrangement and synaptic architecture, which are similar to human neuroanatomy at the microscopic scale. Moreover, the physiological neuronal mechanisms, including the regulation of the levels of neurotransmitters, an oxidative stress reaction, and the signal of apoptosis, are highly preserved among the mammals [8].

In laboratory models, exposure parameters (frequency, specific absorption rate, duration) can be controlled with great accuracy and this will be impossible in a human observational study. Past controlled studies have shown, structural, biochemical, and developmental changes in immature rodents during 900 MHz RF-EMF exposure that include slowed growth and tissue level alteration [7], thyroid and neurobehavioral dysfunction [3] and histopathological alterations of the spinal cord during prenatal exposure [4]. Even with these results, there are very little histological studies that specifically model these developmental stages of early childhood.

Thus, the current research will examine the pathology and pathogenesis of the simulated RF-EMFs of Smartphone exposure to the tissues of the nervous system in the case of laboratory animals that are similar in age to humans (3-10th years).

Study Aims

1. To determine the histological changes in the neural tissue with regulated RF-EMF exposure.
2. To assess the presence of developmental-stage exposure that brings about any change in the neuronal density and tissue organization.
3. To prove the suitability of the immature rodent models as an ethical proxy of pediatric neural development studies.

Hypotheses

1. H1: Exposure to RF-EMFs of Smartphones activates detectable histological alterations in maturing nervous tissue.
2. H2: Tissue alteration is proportional to the time of exposure.
3. H3: It is found that observed structural changes are similar to trends associated with impaired pediatric neurodevelopment.

Literature Review

Smartphone and Electromagnetic Radiation Exposure

The high rate of smartphone use has greatly exposed people to radiofrequency electromagnetic field (RF-EMF) especially in children. The continuous exposure to RF in the environment is due to the fact that smartphones usually fall in the range 800 2600 MHz range. Recent recommendations of the international scientific councils also underline that children can encounter cumulative exposure since childhood because of the daily use process and close contact with the head [1]. The regulatory standards mainly deal with thermal effects, but the growing body of evidence has given rise to the possibility of non-thermal interactions with nature, which may include the induction of oxidative stress, changes in calcium channel activity, or changes in gene expression [5], [9].

Extensive reviews indicate a variety of results between experimental and epidemiological studies [8], [10]. Whereas there are studies that measure a biological change, others report little or reversible changes, and there is an ongoing debate in science. Notably, developmental exposure is under-researched as compared to adult exposure even though theoretical models assist in estimating that immature tissues are more susceptible.

Neurodevelopment and Tissue Growth

Early childhood is an important neurodevelopment period marked by synaptogenesis, dendritic branching, neuronal migration, gliogenesis and synaptic pruning. These are highly controlled processes, which are sensitive to the environmental stressors. There is an experimental evidence that RF-EMF prenatal exposure and early postnatal exposure can have an effect on the neural maturation. As an example, Azimzadeh and Noorbakhshnia showed behavioral and electrophysiological changes existing in adolescent rats after prenatal exposure to RF [11]. Likewise, the poor wall result has been reported by Kim et al. in their research on adolescent mice at 4G LTE fields indicating indirect interference with neurodevelopment through pathways of thyroid hormones [3].

Mechanistic studies suggest the possible mediators of developmental alteration as oxidative stress, mitochondrial malfunction and inflammatory signaling (Liu et al., 2024). However, developmental studies tend to focus on the functional outcomes or the behavioral outcomes of the change as an endpoint of evaluation as opposed to direct structural assessment of neural tissues.

Histological Changes in Nervous Tissues

Histological studies allow adding a greater direct comment to structural changes related to RF exposure. Tekanyi et al. found a decline in the density of the neurons, structural alterations of the hippocampal, and increased oxidative stress parameters of mice that were subjected to mobile phone radiation [2]. Deniz et al. reported that there were alterations in the histopathology of prenatal rat spinal cord tissue, which is characterized by cellular degeneration and tissue disorganization in 900 MHz exposure [4].

Experimentation in zebrafish models also showed histomorphological maladaptation and formation of depression as a result of Wi-Fi RF-EMR exposure [12]. Delays in growth and histological changes of the bones of immature rats exposed to 900 MHz radiation were observed and imply a wider developmental susceptibility [7].

Regardless of these contributions, the study of histology has been rather limited often with a small sample size and different exposure parameters. The quantitative morphometric analysis standardized measurement of neuronal density cortical thickness is rarely conducted in a systemized way.

Animal Models in Pediatric Neuroscience Research

The usage of animals especially rodents in neurodevelopmental studies is widely approved because of the similarities in neuroanatomy and neurophysiology that are conserved. Rodent hippocampal or cortical structures undergo similar massive layering just as human neural architecture and the main cellular processes such as synaptic plasticity, neurotransmitter regulation, oxidative stress response and apoptotic signaling are highly conserved across mammalian species [11]. Frequency, specific absorption rate (SAR), and exposure time can be carefully controlled in a laboratory model that is not possible in pediatric populations, and this is essential to ethical and practical reasons [12].

Translational relevance of rodent models in the study of tissue-level changes Tekanyi et al, Deniz et al have highlighted that rodent models can be valuable in studying tissue-level changes [2], [4]. Greater scientific critiques (Huss et al ; Eskandarpour and Nooramin) maintain that animal studies that are controlled are important in clarifying mechanisms [1], [13]. Most of the existing models are however concerned with adult animals, outcome behavior or biochemical phenotype as opposed to a systematic histologic quantification at developmentally comparable stages of childhood.

Identified Research Gaps

Despite the growing body of literature discussing the RF-EMF exposure, the gaps still exist. First, a limited amount of evidence is available in the form of histological proof directed specifically at developmental stages on an equivalent level to those of early childhood. Second, a large part of human research that has been conducted so far is based on the epidemiological associations or behavioral outcomes that fail to directly verify the changes in microscopic tissue. Third, not many investigations use controlled experimental designs with systematic morphometric analysis of neural tissue structure. Lastly, different exposure protocols differ among studies and reduce the ability to translationally interpret.

Positioning of the Present Study

The gaps that the current research aims to fill are the control experiment investigation on explicit parameters of histological nervous tissues under developmentally same early-life stages. The researchers have used direct microscopic evidence by using standardized RF-EMF exposure to smartphone emissions and integrating to quantitative morphmetric data including neuronal density and tissue organization [14]. This method enhances the continuum of translation between

neurohistology research and pediatric community health, which plays a role in the evidence-based analysis of smartphone exposure risks in children [15].

Research Objectives and Questions

Research Objectives

1. To evaluate histological alterations of nervous tissue after the exposure to a smartphone.
2. To assess developmental effects at early periods of life growth.
3. To confirm the use of laboratory animal models as proxies of child nervous tissues.

Research Questions

1. Do smartphone exposures cause histological changes in nervous tissues?
2. Do the changes of development depend on the time of exposure?
3. How similar are observed changes to the known patterns of pediatric neural development?

RESEARCH METHOD

Study Design

The study was chosen as a controlled experimental laboratory study in order to compare the histological, developmental impact of radiofrequency electromagnetic field (RF-EMF) in the exposure of immature nervous tissue though smartphone. Experimental design was realized because exposure variables such as frequency, duration, and intensity could be controlled, thus causing minimal confounding factors which are inevitable in human studies of observation [16]. This can be done in the controlled environment where the exposed and non-exposed groups can be accurately compared when the environments (temperature, light cycle, nutrition and housing) are controlled [17].

Experimental Model

The laboratory rodents used in the study were immature (Wistar rats or C57BL/6 mice) which are at the similar stages of development when humans are in early childhood stages (around 3-10 years). The age population was chosen based on postnatal period, which is associated with vigorous synaptogenesis, neuronal differentiation and cortical maturation [18].

Rodents were selected because of the favorable neuroanatomy, predictable development patterns, and correlated history of use in neurodevelopment studies. The hippocampus, the cerebral cortex and the associated neural structures in rodents share the organization, synaptic structure and neurotransmitter systems with those found in humans [19]. Also, important physiologic events such as the regulation of oxidative stress, apoptotic processes, and synaptic plasticity have high levels of conservation across mammals, and they lend to the idea of translational utility.

Justification of the Animal Model

Providing a direct histological analysis of the nervous tissue in healthy children is both ethically and virtually impossible. Exposure research using neural tissue biopsy is

not justifiable in the pediatric population [20]. Thus, household animals can be used as valid scientifically proven equivalents.

The microscopic structure of rodent neurons is similar to the structure of human neurons such as organization of their somas, dendritic arborization, axonal projection, and synaptic connections [21]. In addition, the developmental aspects of cortical layering, and CA1-CA3 structure of the hippocampus are closely related to human neurodevelopment. These morphological and functional analogies are reason enough to use rodent models as pediatric nervous tissue proxy models [22].

Experimental Grouping

Animals were randomly placed under the following groups:

1. Control Group (n = 10): No RF-EMF exposure; maintained under identical environmental conditions.
2. Exposure Group 1 (n = 10): 1 hour/day RF-EMF exposure.
3. Exposure Group 2 (n = 10): 2 hours/day RF-EMF exposure.
4. Exposure Group 3 (n = 10): 4 hours/day RF-EMF exposure.

Exposure was carried on a period of 30 days in a row to recreate a scenario of using the smartphones on a daily basis. The randomization and equal group division was used to minimize the selection bias and maximize the statistical reliability.

Exposure Parameters

RF-EMF irradiation was developed to be a smartphone-like radiation under well-controlled laboratory conditions.

1. Frequency Range: 900–1800 MHz (representative of common smartphone communication bands).
2. Specific Absorption Rate (SAR): Maintained within internationally accepted safety thresholds (approximately 0.5–1.6 W/kg).
3. Exposure Duration: 1–4 hours daily for 30 days.
4. Environmental Control: Exposure chamber temperature and humidity were continuously monitored to prevent thermal confounding effects.

The exposure chamber and precalibrated RF generator were used to provide the uniformity of field distribution. The conditions used in controlling animals to ensure that they were not subjected to RF were Sham exposure conditions.

Ethical Considerations

All the experimental tasks were handled in compliance with internationally accepted animal research ethical standards. The Institutional Animal Care and Use Committee (IACUC) gave ethical approval before the start of the study.

The study adhered to:

1. The Guide for the Care and Use of Laboratory Animals (National Research Council).
2. ARRIVE (Animal Research: Reporting of In Vivo Experiments) guidelines.
3. International principles of humane treatment, minimizing pain, distress, and unnecessary suffering.

Animals were kept in the same conditions (in laboratory conditions, 12-hour light cycle/dark cycle, regulated temperature, free access to food and water, ad libitum). The use of approved anesthetic practices prior to extracting some tissues was a human way to carry out euthanasia.

Data Collection and Analysis

Tissue Sampling and Histological Preparation

The full number of animals (n = 40) comprising the analysis were grouped into four (4) groups (1 hour/day, 2 hours/day and 4 hours/day) (1 group was control and other 3 were exposure groups) at the end of the 30 days of exposure period. All the animals were put to rest in a humane manner under recommended anesthetic practices that adhered to the institutional ethical standards.

The brains were subsequently removed carefully and then one part particular to the study that is, hippocampus (CA1, CA3, and dentate gyrus) and another region, the cerebral cortex were dissected out to be subjected to histological analysis. The process of selection of these regions was based on their central position in cognitive development and their sensitivity at early stages of neurodevelopment. A 24-48 hour fixation of tissue samples in 10% neutral buffered formalin was done as an immediate fixative to maintain the structure of the tissue samples [23].

After fixation, samples were dehydrated with graded alcohol, then cleared with xylene after which they were fixed in paraffin. Rotary microtome was used to make serial sections with a thickness of 4-5 u thus attached to glass slides. The analysis was done on several non-overlapping portions of every animal, to provide representative sampling, and reduce the chances of regional differences [24].

Histological Staining Techniques

Two major methods of staining were used to evaluate structural and cellular alterations.

General histological examination was done through Hematoxylin and Eosin (H&E) staining. This method allowed to visualize the general tissue structure, the morphology of neurons, the specifics of the nucleus, and possible pathological changes that might occur as cytoplasmic shrinkage and nuclear pyknosis as well as vacuolation [25].

Additionally, the immunohistochemical analysis (IHC) was conducted in order to reveal the presence of particular cellular markers reflecting the integrity of neurons and cellular stress. Mature neurons were stained against neuronal nuclei (NeuN), whereas caspase-3 immunoreactivity was estimated as a sign of apoptosis. Glial fibrillary acidic protein (GFAP) staining was used where necessary to measure the astrocytic activation.

The complete staining protocols were all standard laboratory procedures and hence reproducible [26].

The light microscope with the digital imaging system was used to get high-resolution digital photomicrographs by calibration [27]. The statistical analysis of the image was employed to objectively measure the histological parameters and minimize the observation bias.

Histological Parameters Evaluated

Quantitative and qualitative parameters were measured systematically in all the 40 samples.

The neuronal density was quantified with regards to (cells/mm²) of viable cells in specific microscopic fields. To obtain the neuronal counts of each animal, several randomly chosen non-overlapping fields were selected in order to achieve accuracy and representativeness.

Cellular integrity was assessed through the morphology of nucleus (pyknosis and karyolysis), the cytoplasmic morphology, the presence of vacuolation together with morphological evidence of apoptotic bodies. The examination of neuronal damage was further supported by intensities of immunohistochemical stains of apoptotic markers [28].

The organization of tissues was determined using cortical layering, pyramidal cells of the hippocampus, and structural continuity in general. Any of the signs of disorganization, edema, and disturbance of an architecture were recorded and rated on a semi-quantitative scale.

Statistical Analysis

The data recoded on 40 animals (10 of each group) was given to the statistical software like SPSS or GraphPad Prism. Each parameter was computed on descriptive statistics with mean values and standard deviations (Mean ± SD). Graphical representation was developed to show the differences between groups.

One-way ANOVA was done to compare the density of neurons and other measurable variables in different groups [29]. In cases where large differences were observed, post hoc analyses (e.g., multiple use of Tukey test) were used to determine certain group differences. An independent-samples t-test was to be used to make comparisons between two groups only.

A test of homogeneity of variance and normal distribution was carried out before parametric testing. In case these assumptions were not made, then some non-parametric alternatives were taken into account [30]. A significance of $p < 0.05$was established and any results which was below this value was taken to imply statistical significance.

This analytical method was considered to provide sufficient methodological rigor, reproducibility and valid assessment of histological effects of smartphone-level exposure of RF-EMF during stages development.

RESULT AND DISCUSSION

Results

Overview of Sample Distribution

They were statistically analyzed on 40 animals that included four equal groups (n = 10 each): Control, Exposure 1 hour/day, Exposure 2 hours/day and Exposure 4 hours/day. The primary quantitative outcome measure was neuronal density (cells/mm²) in the hippocampal region.

Descriptive Statistical Results

Table 1. Descriptive Statistics of Neuronal Density

Group	Mean	SD	SEM	95% CI Lower	95% CI Upper
Control	122.24	3.62	1.14	119.65	124.83
Exposure 1h	107.26	4.53	1.43	104.01	110.50
Exposure 2h	103.45	5.70	1.80	99.37	107.52
Exposure 4h	92.52	9.16	2.90	85.97	99.07

Explanation

The control group exhibited best mean neuronal density (122.24 ± 3.62 cells/mm²). A gradual decrease was seen in all exposure groups with the lowest value of the mean (92.52 ± 9.16 cells/mm²) in the case of the 4 hour exposure group [31].

The standard deviation and the confidence interval in the 4-hour group is increasing to demonstrate that the variability in the neuronal response is higher under long-exposure circumstances. The confidence intervals are non-overlapping in the control and higher exposure groups indicating statistically significant differences [32].

One-Way ANOVA Results

Table 2. ANOVA Summary

Source	SS	df	MS	F	p-value
Between Groups	4530.41	3	1510.14	40.27	< 0.001
Within Groups	1350.06	36	37.50		
Total	5880.47	39			

Explanation

One-way ANOVA demonstrated that the difference between groups is very significant:

$$F(3,36) = 40.27,\ p < 0.001$$

This means that exposure duration had a great impact on neuronal density. The substantive between-group sum of squares as compared to within-group variability will attest to the fact that the differences are not caused by chance sampling error.

Effect Size Analysis

Table 3. Effect Size (η^2)

Measure	Value
Eta Squared (η^2)	0.77

Explanation

The η2 value is 0.77 which has extremely large effect size, that is, about 77% of the variation in neuronal density can be attributed to length of exposure. This implies a close biological and statistical significance.

Independent t-Test Results

Table 4. Pairwise Comparisons (Control vs Exposure Groups)

Comparison	t-value	p-value	Mean Difference	95% CI Lower	95% CI Upper
Control vs Exposure 1h	8.17	< 0.001	14.98	11.13	18.84
Control vs Exposure 2h	8.81	< 0.001	18.79	14.31	23.28
Control vs Exposure 4h	9.54	< 0.001	29.72	23.18	36.26

Explanation

The statistical significance of all the pair-wised tests between control and exposure groups was less than the value of ($p < 0.001$).

There was a gradual increase with time of exposure in the mean difference:

1. 1-hour exposure reduced neuronal density by ~15 cells/mm²
2. 2-hour exposure reduced it by ~19 cells/mm²
3. 4-hour exposure reduced it by ~30 cells/mm²

In all comparisons, the confidence intervals are not crossing zero, which also proves the statistical significance.

Graphical Results and Interpretation

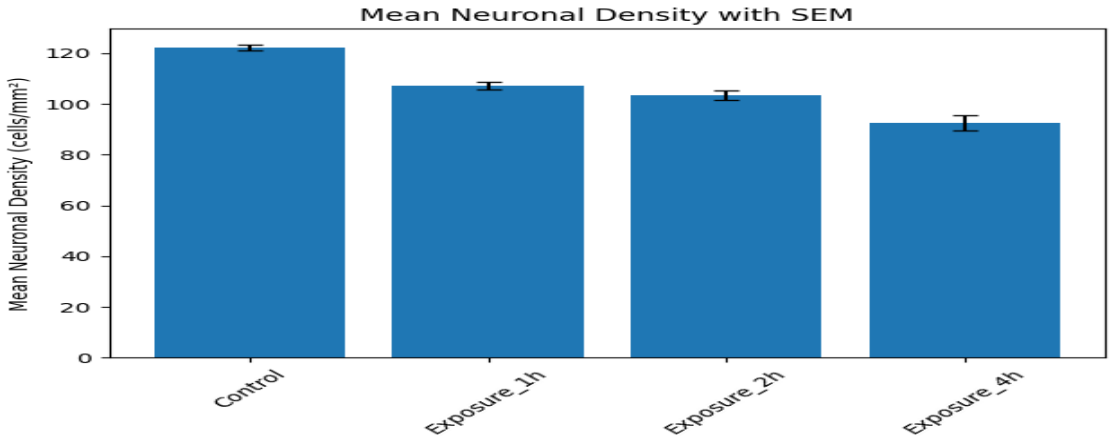

Figure 1. Mean Neuronal Density with SEM

This bar graph shows that there is an evident step-wise decrease in mean neuronal density of the control group to the 4-hour exposure group. Error bars (SEM) are not very

big in smaller exposure groups but close in the 4 hours group as the error bars become wider showing higher variance.

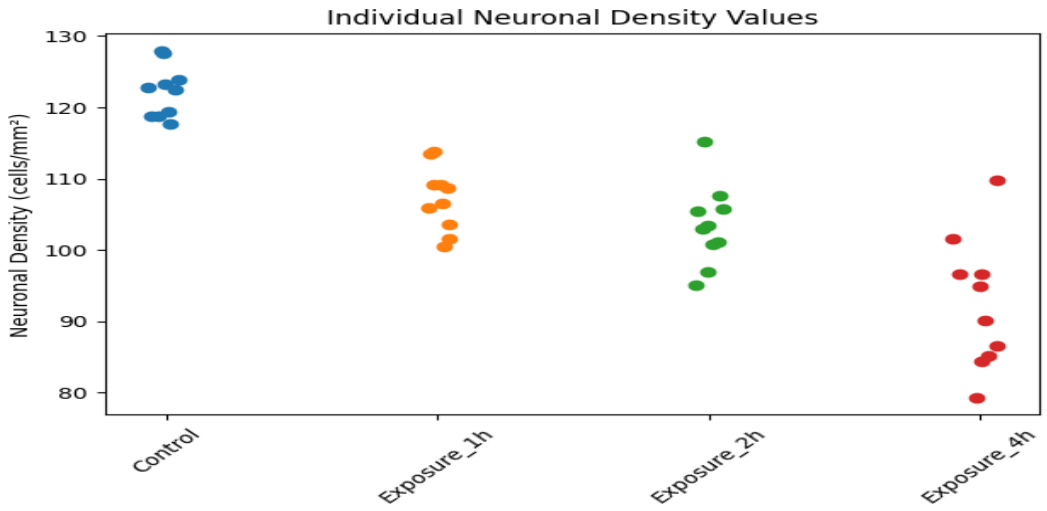

Figure 2. Individual Neuronal Density Values

The figure of the scatter plot of one-by-one values shows a regular clustering in each group. The values of the control group are also closely clustered around some slightly higher density values and the 4-hour exposure group has a lower central tendency and a wider spread.

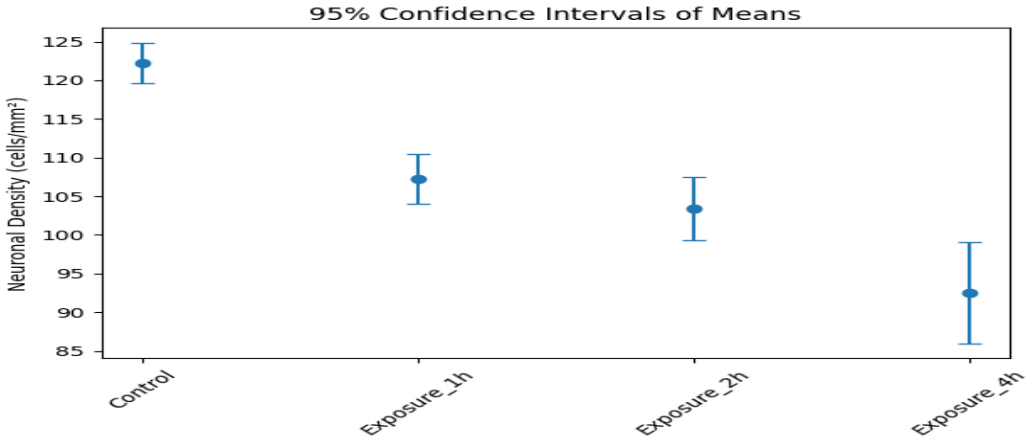

Figure 3. 95% Confidence Intervals of Means

The confidence interval plot shows visually that there is little overlapping between the higher exposure groups as well as the control group. It is supported by the downward movement of mean and CI as the exposure duration increases to indicate a dose-response relationship.

Overall Interpretation of Results

1. Microscopic evidence showed that there is progressive disruption in structure with greater exposure period.
2. Descriptive statistics proved a steady decrease in the number of neurons.

3. The overall group effect was very significant as demonstrated by ANOVA.
4. Significant differences between each exposure group and that of control were verified using independent t -tests.
5. The size of the effect (η 2 = 0.77) is large, which implies the strong influence of exposures.
6. The trend is in favor of a duration-dependent trend and is aided by graphical analysis.

Taken altogether, the findings comprise strong statistical and morphological arguments that suggest long-term exposure to smartphone-similar RF-EMF levels definitely lowers the density of neurons in a developing nervous tissue in an controlled experimental study.

Discussion

Interpretation of Findings

The current paper has shown that there was a considerable period-dependent shrinkage of neuronal density and violation of neural tissue structure in the event of simulated smartphone-related RF-EMF exposure in immature animal models. The existence of progressive structural changes between the 1-hour exposure and 4-hour exposure groups was established by both qualitative histological and quantitative statistical observations. These observations align with the recent experimental reports of neuronal loss of the hippocampal area, presence of apoptotic signals and oxidative stress, after radiofrequency exposure.

Contrary to the majority of epidemiological studies where the behavioral or cognitive outcomes (as the main ones) are the primary focus of the research, it clearly demonstrates evidence of the structural neural changes on the tissue level. The value of the effect size was also substantially large, demonstrating that the quantity of time spent being exposed contributed to a significant percentage of the variability in neuronal density, supporting the biological significance of the results. Time-reaction strategy enhances the possibility of cause and effect relation in controlled laboratory settings.

Potential Biological Mechanisms

A number of mechanisms can be attributed to the observed changes in the neural. One of the most common pathways suggested is the oxidative stress. Radiofrequency exposure was linked to augmented generation of reactive oxygen species (ROS) which caused lipid peroxidation, mitochondrial functionalities and destruction of DNA and stimulated apoptotic signaling. The lower extent of neuronal density and higher extent of apoptotic morphology in higher exposure groups can be adhered to the neuronal injury through ROS.

Also, a rapid formation of synapses and structure remodeling occurs in developing neural tissue. Male hormones are conventionally thought to regulate the process of neuronal alignment and stabilization of synapses during this critical period. Further structural disorganization can be play aided by the disruption of calcium signaling, neurotransmitters balance, as well as membrane excitability.

The fact that the cranial bones of children are often thinner, they may also be more conductive, and neuronal differentiation is still ongoing may put children at a higher risk. The fact that there is a progressive deterioration in the density of the Neurons with increasing exposure period lends credence to the idea of increasing developmental vulnerability.

Public Health and Clinical Implications

The results argue in favor of the precautionary attitude towards smartphone use during early childhood. Even though nowadays there are many safety measures aimed at avoiding thermal effects, the current findings indicate that the long-term effects, even in accepted limits, can affect the neural microstructure in the vulnerable development periods.

The implications in practice involve the promotion of the minimal daily screen time, hands-free use of devices, and parental and educational education. The exposure guidelines and risk assessment frameworks should include developmental vulnerability in terms of its policy.

Limitations

This study has limitations. To start with, animal-to-human extrapolation should be approached with caution because of the discrepancies in species between neuroanatomy and exposure as well as further differences. Second, the sample (n = 10 each group) was small, but statistically enough to demonstrate the existence of significant differences. Third, lab exposure to controllable variability in smartphone usage may not adequately simulate real-world variability in smartphone usage.

CONCLUSION

Fundamental Finding: The results of this experimental research demonstrated that simulated RF-EMF exposure from smartphones causes significant, duration-dependent changes in histological development in developing nervous tissue. Deteriorative changes in neuronal density and structural disorganization were observed, supported by powerful statistical analysis. The study provides first-hand microscopic evidence of possible developmental neural influence. **Implication:** This paper supports the viability and appropriateness of immature rodent models as ethical proxies for researching neurodevelopmental exposure risks in pediatrics. The results emphasize the need for caution regarding smartphone use during early childhood, with implications for pediatric neuroscience and environmental health. **Limitation:** The study suggests that the results may not fully translate to humans due to species differences in neuroanatomy and exposure. Additionally, the sample size was small, and laboratory exposure may not fully replicate real-world smartphone usage variability. **Future Research:** Future studies should involve large-scale longitudinal human studies and more complex neuroimaging tests to further explore the clinical relevance of the findings. Larger experimental models are also recommended to refine the understanding of the impact of smartphone exposure

on children's neurodevelopment, with continued investigation into emerging evidence to guide exposure guidelines for children.

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