A primary study on rat fetal development and brain-derived neurotrophic factor levels under the control of electromagnetic fields

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Abstract

Background. In previous researches, electromagnetic fields have been shown to adversely affect the behavior and biology of humans and animals; however, body growth and brain-derived neurotrophic factor levels were not evaluated.

Objective. The original investigation aimed to examine whether Electromagnetic Fields (EMF) exposure had adverse effects on spatial learning and motor function in rats and if physical activity could diminish the damaging effects of EMF exposure. In this study, we measured anthropometric measurements and brain-derived neurotrophic factor (BDNF) levels in pregnant rats' offspring to determine if Wi-Fi EMF also affected their growth. These data we report for the first time in this publication.

Methods. Twenty Albino-Wistar pregnant rats were divided randomly into EMF and control (CON) groups, and after delivery, 12 male fetuses were randomly selected. For assessing the body growth change of offspring beginning at delivery, then at 21 post-natal days, and finally at 56 post-natal days, the crown-rump length of the body was assessed using a digital caliper. Examining BDNF factor levels, an Enzyme-linked immunosorbent assay ELISA kit was taken. Bodyweight was recorded by digital scale.

Results. Outcomes of the anthropometric measurements demonstrated that EMF blocked body growth in rats exposed to EMF. The results of the BDNF test illustrated that the BDNF in the EMF liter group was remarkably decreased compared to the CON group. The results indicate that EMF exposure could affect BDNF levels and harm body growth in pregnant rats' offspring.

Conclusions. The results suggest that EMF exposure could affect BDNF levels and impair body growth in pregnant rats' offspring.

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Key words: electromagnetic fields, BDNF factor, anthropometric measurements, male offspring, crown-rump length.

Acknowledgments: the authors are very grateful to Dr. Peter Ernest Wigand for reading and editing our article. Moreover, they would like to thank the Faculty of Veterinary Medicine, University of Tehran, for providing them with their laboratory facilities.

Conflict of interest: the authors declare no potential conflict of interest.

Ethical approval and consent to participate: this project is ethically approved by the Research Institute of Physical Education and Sports Sciences ID: IR.SSRI.REC.1399.919.

Funding: none.

Availability of data and materials: data and materials are available by the authors.

Received for publication: 12 October 2022.
Accepted for publication: 22 December 2022.

Journal of Public Health in Africa 2023; 14:2347
doi:10.4081/jphia.2023.2347

Introduction

The various frequencies of Electromagnetic Fields (EMF) are the fastest-growing, invisible, global environmental pollutants.[1,2] Some natural sources also produce EMF, *e.g.*, the earth and the sun.[3] Though EMF exposure may endanger an individual's health throughout his lifespan, disclosing the human fetus to EMF during pregnancy may cause significant health issues. The vast majority of investigations reveal a link between suboptimal gestational status, causing fetal growth disorders such as metabolic and psychiatric disease, fetal macrosomia, and intrauterine growth restriction.[4] EMF exposure by the fetus during pregnancy has focused the attention of scientists on the examination of the bio-effects of EMF on post-natal cognitive behaviors.[5-7] Reconfiguration of neural mechanisms and consequently cognitive and non-cognitive behaviors in mammals during EMF exposures are proved by previous studies.[8] For instance, prior investigations have claimed that EMF has caused some issues: reproductive disorders, memory deficits, cancer, hyperactivity, *etc*.[1,9-11] In contrast, other studies have often discounted the withering effects of EMF exposure.[12-14] However, none of them have considered the effect of EMF exposure on human growth, nor have they examined anthropometric measurements to document such effects.

The brain-derived neurotrophic factor (BDNF) that is found in the central nervous system and the peripheral nervous system plays an essential role in synaptic connection, neuronal repair, differentiation, and neuronal outgrowth.[15,16] Moreover, diseases such as depression and obesity occur because of the lack of function mutations in high-affinity tropomyosin-related kinase B as BDNF receptor.[17,18] Prior investigations have shown a direct relationship between fetal growth and brain health.[19] According to these studies, brain-derived neurotrophic factor modulates patterns of fetal development,[20,21] particularly regarding weight.[22,23] This is particularly evidenced in the development and maturation of peripheral and central fetal organs containing the placenta.[24] EMF exposure has been known as a risk factor in the endangerment of brain health.[25] No study has been conducted regarding EMF exposure on the BDNF levels in pregnant rat fetuses.

Weight change is an indication of growth in rodents.[26] According to the dynamic system theory, one branch of the developmental milestone theory, development, is a process that is dependent on three elements: individual, task, and environment.[27] EMF exposure is an environmental factor that affects the embryo during pregnancy.[9]

The present study is aimed to examine the impacts of 2.4 GHz EMF Wi-Fi exposure on body measurements and hippocampus BDNF Factor levels of the fetus. This hypothesis is addressed using electrical coils and the Rat BDNF ELISA kit.

Materials and Methods

This project is ethically approved by the Research Institute of Physical Education and Sports Sciences ID: IR.SSRI.REC.1399.919.

Subjects

Twenty first-day pregnant Albino-Wistar rats purchased Pasteur Institute of Iran, were employed in the present investigation (weight: 180-220 g). The advent of the vaginal plug after mating was computed as the first day of gestational day. The pregnant rats were kept in two separate temperature-controlled rooms (23±2°C): i) a standard unshielded room (for pregnant rats in the EMF group); ii) a shielded room, which was insulated against EMF using aluminum foil (for pregnant rats in control (CON) group).[28] The rats were independently housed in standard Plexiglas cages. They had access to unlimited food and water sources. Each group was exposed to a cycle of 12 hours of light and 12 hours of dark.[29] After delivery, 12 male neonates from EMF and CON maternal groups were randomly selected for the experiment using a simple randomization approach. Helsinki ethics were applied in this research.

Intervention

Electromagnetic fields exposure

A 2.4 GHz Wi-Fi Modem, which is habitually used in homes, was placed on a table at a one-meter distance from the EMF group cages. The effect of space was negated by the daily exchange of the location of the cages. From the first day to the day of delivery, between 8 a.m. and 2 p.m., pregnant rats were exposed to EMF waves, 2.4 GHz, during the download or upload of various files.[29] As previously mentioned, during radiation, the control group was kept in the shielded room. The specific absorption rate (SAR) was determined using the mathematical Finite Difference Time Domain formula:

$$SAR = \left(\frac{W}{kg}\right) = \left(\frac{d}{dt}\right)\left(\frac{dW}{dm}\right) = \left(\frac{d}{dt}\right)\left(\frac{dW}{\rho dV}\right) = \left(\frac{\delta E^2}{\rho}\right)$$

where δ is the particular tissue conductivity $\left(\frac{s}{m}\right)$; E is associated with the generated electric field, and at the end, ρ the tissue density $\left(\frac{kg}{m^3}\right)$.[30-32]

The conductivity and density of embryo tissue, respectively, are δ=2.26 s/m, ρ=1040 $\left(\frac{kg}{m^3}\right)$, regarding the distance of pregnant rat from the modem; SAR was computed minimally 0.15 $\left(\frac{W}{kg}\right)$, and maximal 0.31.

Tests

Anthropometry measurements

The neonate's physical growth measurements, including; the length of the body, length of tail, and body weight, were performed three times; at birth, at 21 post-natal days, and at 56 post-natal days.[33] Crown-rump length of the body was assessed using a digital caliper (Guanglu Measuring Instrument Co. Ltd, Guilin, China). The accuracy of the digital caliper and digital balance was 0.01mm, and 0.0001 gr, respectively.

The brain-derived neurotrophic factor

At 56 post-natal days, the subjects were deeply anesthetized using Medetomidine hydrochloride, 1mg/ml (Dorbene Vet brand). Then the brain tissues of the rats were removed and stored in liquid nitrogen at -80°C. Hippocampal samples were centrifuged using a German Hectic centrifuge for 5 min at around 16,000 rpm. The protein content of the hippocampus in the brain tissue was then analyzed by a Rat BDNF ELISA kit (Zellbio, Germany) using the manufacturer's instructions to an accuracy of 0.2. The results were evaluated using an ELISA reader (Stat Fax, US), and the absorbance of the samples was read at 450 nm.

Data analysis

The descriptive statistics that were evaluated in this study included the mean and standard deviation. Firstly, the normal data distribution was tested using a Shapiro-Wilk, and Leven statistic to test the homogeneity of variances. Anthropometric measurements, and body measurements in each group were assessed using ANOVAs with repeated measures at three stages: after delivery, 21 post-natal days, and 56 post-natal days. One-way ANOVA was used in Bonferroni post-hoc manner. Assigning differences between groups, the independence T-test was applied. Total outcomes were depicted as Mean±SD in all statistical differentiation. The scale for statistical significance was $P \leq 0.05$.

Results

Intragroup anthropometrics results

In Figure 1A-C, the comparison of the weight, body length, and tail length changes among the CON and EMF groups are displayed.

Statistical analysis revealed a significant rise in body weight, body length, and tail length during the three measurement periods. In the EMF group at A) delivery: $F(1.01, 10) = 345.85$, $P=0.0001$; B) 21 PND: $F(2, 10) = 319.011$, $P=0.0001$; C) 56 PND: $F(2, 10) = 358.93$, $P=0.0001$; and in the CON group at A) delivery: $F(1.46, 10) = 4554.76$, $P=0.0001$; B) 21 PND: $F(2, 10) = 1971.29$, $P=0.0001$; C) 56 PND: $F(2, 10) = 2111.99$, $P=0.0001$).

The Bonferroni post-hoc tests in the EMF group discovered consistent changes in body weight, body length, and tail length from delivery day to 56 PND (body weight: 6.21±0.11 gr, 35.55±6.33 gr, 206.55±27.14 gr; body length: 51.02±0.64 mm, 92.21±9.25 mm, 171.38±9.40 mm; tail length: 19.95±0.03 mm, 46.33±7.02 mm, 136.45±9.89 mm), and also in the CON group (body weight: 6.32±0.19 gr, 36.16±5.37 gr, body length: 229.73±4.83 gr; 45.58±0.32mm, 104.59±5.20mm, 181.10±3.87mm; tail length: 17.17±0.05 mm, 67.31±4.75 mm, 156.23±6.44 mm).

Intergroup anthropometrics results

As illustrated in Figure 2, this study found that body weight in delivery, 21 postnatal days, and 56 postnatal days in neonates who were exposed to EMF (6.21±0.11gr; 35.55±6.33gr; 206.55±27.14gr, respectively) lower in comparison to that found in the control group (6.32±0.19 gr; 36.16±5.37gr; 229.73±4.83gr) however, these rates were not significant (*t* (10) = 1.20, P=0.257; *t* (10) = 0.182, P=0.859; *t* (10) = 2.06, P=0.066, respectively).

In contrast, the body length of the EMF group at delivery time was significantly higher than the control group: 51.02±0.64mm *vs*. 45.58±0.32mm, t (10) = -18.43, P=0.0001). However, the control group's body length at 21 PND: 104.59±5.20 mm *vs*. 92.21±9.25 mm, correspondingly was significantly higher than that of the EMF group and at 56 PND: 181.10±3.87mm *vs*. 171.38±9.20 mm), *t* (10) = 2.85, P=0.017; t (10) = 2.34, P=0.041 (Figure 3).

Moreover, according to the Statistical analysis outcomes, the tail length of EMF group (19.95±0.03mm) in delivery time was significantly higher than the control group (17.17±0.05mm), *t* (10) = -96.69, P=0.0001. Conversely, at 21 PND, the control group's tail length was significantly longer than that of the EMF group 67.31±4.75mm *vs*. 46.33±7.02mm; and at 56 PND 156.23±6.44mm *vs*. 136.45±9.89 mm, *t* (10) = 6.06, P=0.001, and *t* (10) = 4.100, P=0.002, respectively (Figure 4).

Brain-derived neurotrophic factor measurement

The BDNF factor concentration comparisons between groups were assessed by the independent t-test, and the outcomes are illustrated in radar Figure 5.

This study found that neonates who had been affected by EMF exposure had significantly lower, statistically BDNF concentrations (6.8±0.2 ng/ml) at 56 post-natal days compared to the control group (7.08±0.16 ng/ml), *t* (10) = 2.59, P=0.027.

Discussion and Conclusions

Anthropometry measurements

Examining the effects of 2.4 GHz EMF Exposure on anthropometric measurements, BDNF concentrations in the fetuses of exposed pregnant rats were the primary study's purpose. The results were: i) 2.4 GHz EMF exposure in the pregnancy period leads to the reduced physical growth of the fetuses after birth; ii) 2.4 GHz EMF exposure during pregnancy results in lower BDNF concentrations in male rats.

As mentioned previously, the fetus's body weight in the EMF exposure group was significantly lower compared to the CON group. Moreover, body length and tail length were significantly lower in the EMF group than in the CON group.

In zoological texts, the index of rat growth is assessed by body weight,[26] and the BDNF levels are manipulated by body weight.[34] According to the animal resource center report, the expected average weight for male Albino-Wistar rats is 305 gr.[35] As our results illustrate, both groups are lower than the normal range. On the other hand, the CON group at 56 PND is heavier than the EMF group. Therefore, the control group had better physical development. Moreover, prior studies have indicated that both obesity and anorexia have a direct and inverse relationship with BDNF.[22,23,34] These findings are not in line with previous investigations that suggest that EMF exposure causes obesity.[36] Many investigations have shown that BDNF levels can affect appetite.[34,37,38] BDNF is a component of the neurotrophins of concealing signaling molecules that involve nerve growth factor, neurotrophin-3, and neurotrophin-4/5.[39] The major molecule involved in controlling body weight is leptin, which is the protein product of the obese (ob) gene and is formed in and secreted by adipose tissue and acts as an indicator of fat mass.[40] Adjusting the formation, preservation, and action of neuronal connections is the way BDNF manipulates energy homeostasis.[41] Various investigations have revealed that BDNF

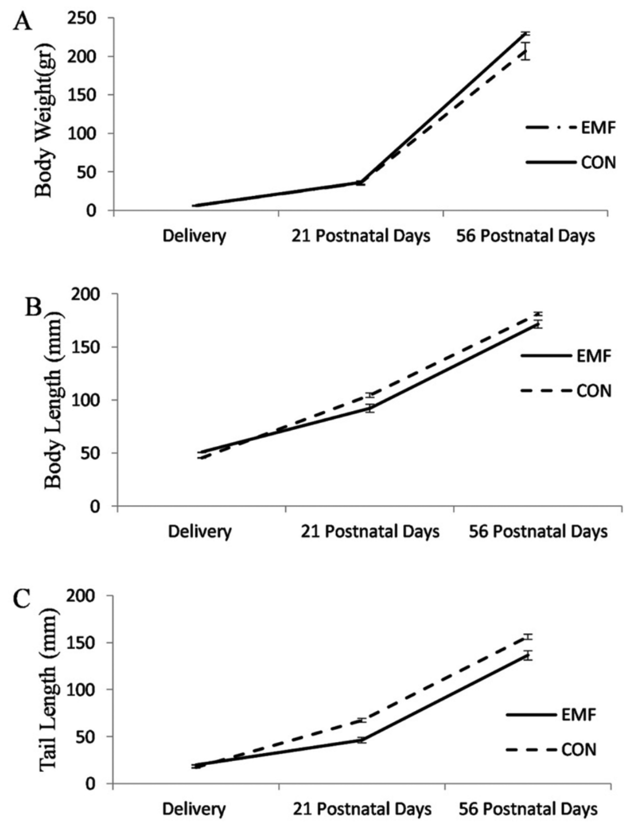

Figure 1. Comparison average of the body weight (A), body length (B), and tail length (C) within groups at delivery, at 21 postnatal days, and at 56 postnatal days; each value represents the mean±SD for six rats at electromagnetic fields and control groups.

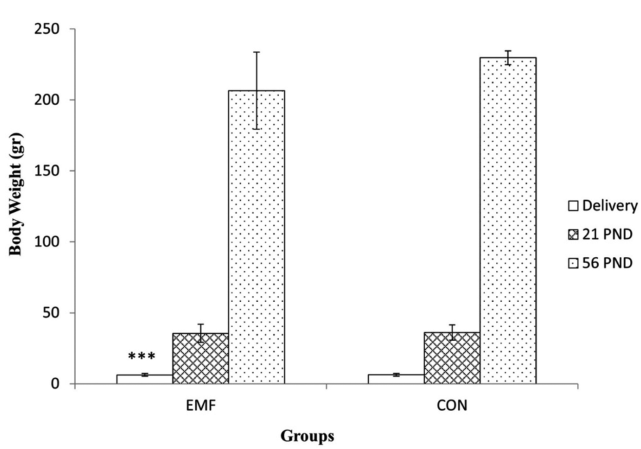

Figure 2. Assessing the average body weight between groups; any values indicate the mean±SD for six rats at electromagnetic fields and control groups. Significant differences from the electromagnetic fields group with P≤0.05; * illustrates P≤0.001.**

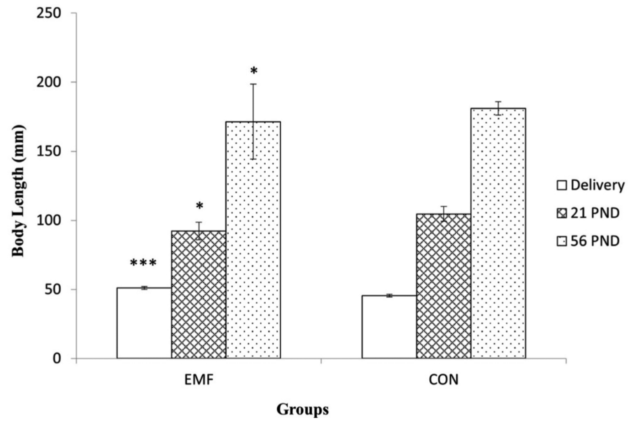

Figure 3. Assessing the average body length between groups; any values indicate the mean±SD for six rats at electromagnetic fields and control groups. Significant differences from the electromagnetic fields group with P≤0.05; ** illustrates P≤0.01 * shows P≤0.001.**

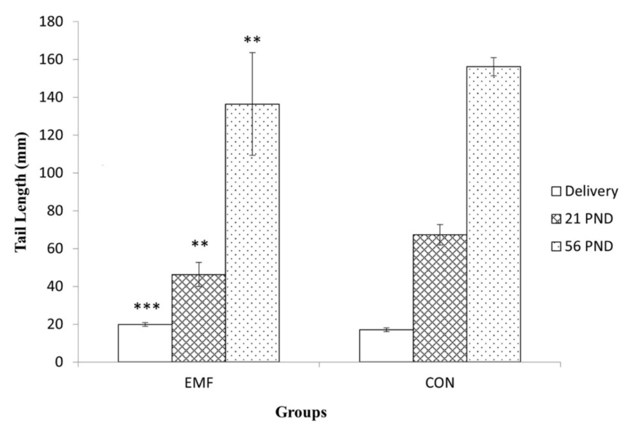

Figure 4. Assessing the average tail length between groups; any values indicate the mean±SD for six rats at electromagnetic fields and control groups. Significant differences from the electromagnetic fields group with P≤0.05; ** illustrates P≤0.01 * shows P≤0.001.**

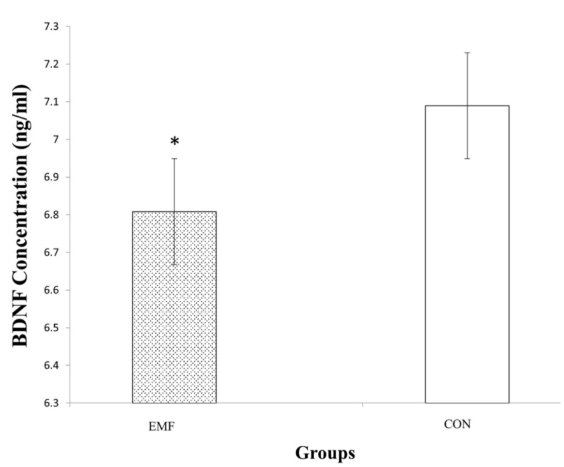

Figure 5. Assessing the brain-derived neurotrophic factor concentration (ng/ml) for subjects at electromagnetic fields, and control groups.

affects other anorexigenic factors, such as leptin and cholecystokinin, to adjust food intake and body weight.[41] Introducing peripheral leptin was found to improve the BDNF mRNA level in the ventromedial hypothalamus,[42] and the BDNF factor rate in the dorsal vagal complex.[43] Improving BDNF mRNA in dendrites results from these effects, while leptin grew dendritic BDNF mRNA translation in hypothalamic neurons.[44] Initiating BDNF-expressing neurons through a polysynaptic neural circuit is the way that leptin obliquely begins synthesis of BDNF mRNA and BDNF Factor.[41] BDNF stimulates the growth and preservation of neural circuits.[45] So, obesity syndrome associated with BDNF inadequacy could result from structural impairment in neural circuits, controlling energy homeostasis, or reduced anorexigenic actions of BDNF.[18]

The brain-derived neurotrophic factor

As indicated previously, significant differences between BDNF factor levels in the EMF group compared to the CON group were observed. It is in line with prior investigations.[46,47] In contrast, some preliminary studies have failed to find the adverse effects of EMFs.[7] Moreover, some of these studies have even claimed that EMFs would act as an obstacle to the progression of Alzheimer's disease in rats.[48]

Since there is a correlation between the level of hippocampal BDNF, memory, and learning abilities, it is clear that the decline in the level of neonatal BDNF, when exposed to electromagnetic waves, is due to the changes in the synaptic plasticity of the hippocampus.[49] This synaptic activity is regulated by a cholinergic system that acts synergistically with glutamatergic transmission.[50] In general, the cholinergic system plays a vital role in learning abilities and memory associated with the hippocampus. Hippocampal cells have long and constant synaptic plasticity. All forms of synaptic plasticity are stimulated by the activation of the afferent, and are involved in the absorption of calcium, and can inhibit calcium chlorateand activate calcium-dependent mechanisms [51]. The possible basis of the inhibitory effects of magnetic fields can be due to an increase in intracellular. This increase in the concentration of intracellular calcium ions is due to the excessive or continuous activation of glutamate ion channels, resulting in neurodegeneration.[52] High calcium ion levels lead to a decline in cholinergic activity in the frontal cortex and hippocampus of rats acutely exposed to magnetic radiations.[53] These effects may be attributed to the impact of the electric and magnetic parts of EMF exposure.

Strength and limitations

It is the first study to evaluate the effects of EMF exposure on physical development and BDNF levels in fetuses. This study was limited by the small sample size, the lack of measurement of growth hormone levels, the short exposure period to electromagnetic fields, and the lack of measurement of BDNF levels at delivery. It is therefore suggested that extremely low frequencies ELF might be used as a stimulator in future studies and that a larger sample size might be needed to confirm these findings. Additionally, it is proposed to take blood samples to test the level of growth hormone and BDNF at the time of delivery.[26]

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