

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.

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.*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

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

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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 ($\frac{s}{m}$); 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

$\delta=2.26$ s/m, $\rho=1040$ ($\frac{kg}{m^3}$), regarding the distance of pregnant rat

from the modem; SAR was computed minimally 0.15 ($\frac{W}{kg}$), 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 ± 0.32 mm, 104.59 ± 5.20 mm, 181.10 ± 3.87 mm; 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 ± 0.11 gr; 35.55 ± 6.33 gr; 206.55 ± 27.14 gr, respectively) lower in comparison to that found in the control group (6.32 ± 0.19 gr; 36.16 ± 5.37 gr; 229.73 ± 4.83 gr) 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 ± 0.64 mm vs. 45.58 ± 0.32 mm, $t(10) = -18.43$, $P = 0.0001$. 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 ± 3.87 mm 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 ± 0.03 mm) in delivery time was significantly higher than the control group (17.17 ± 0.05 mm), $t(10) = -96.69$, $P = 0.0001$. Conversely, at 21 PND, the control group's tail length was significantly longer than that of the EMF group 67.31 ± 4.75 mm vs. 46.33 ± 7.02 mm; and at 56 PND 156.23 ± 6.44 mm 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

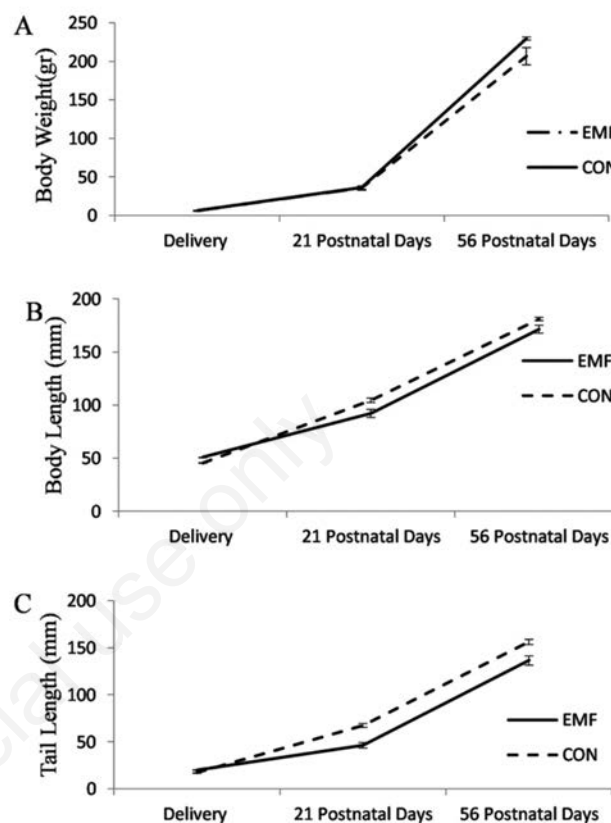


Figure 1. Comparison average of the body weight (A), body length (B), and tail length (C) within groups at delivery, at 21 postnatal days, and at 56 postnatal days; each value represents the mean \pm SD for six rats at electromagnetic fields and control groups.

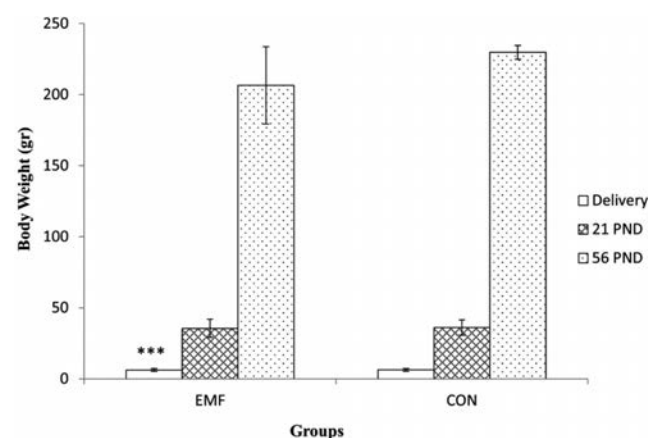


Figure 2. Assessing the average body weight between groups; any values indicate the mean \pm SD for six rats at electromagnetic fields and control groups. Significant differences from the electromagnetic fields group with $P \leq 0.05$; *** illustrates $P \leq 0.001$.

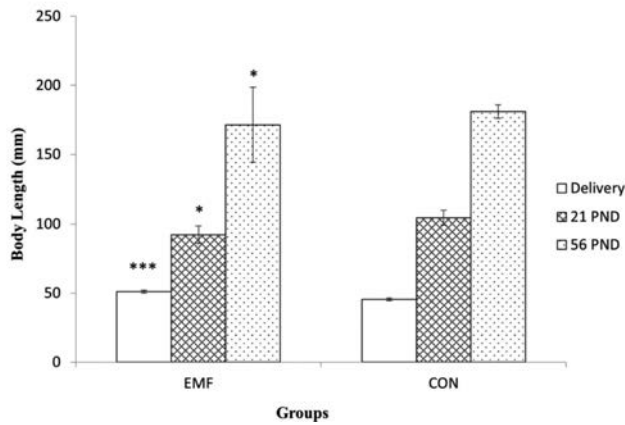


Figure 3. Assessing the average body length between groups; any values indicate the mean±SD for six rats at electromagnetic fields and control groups. Significant differences from the electromagnetic fields group with $P \leq 0.05$; ** illustrates $P \leq 0.01$ *** shows $P \leq 0.001$.

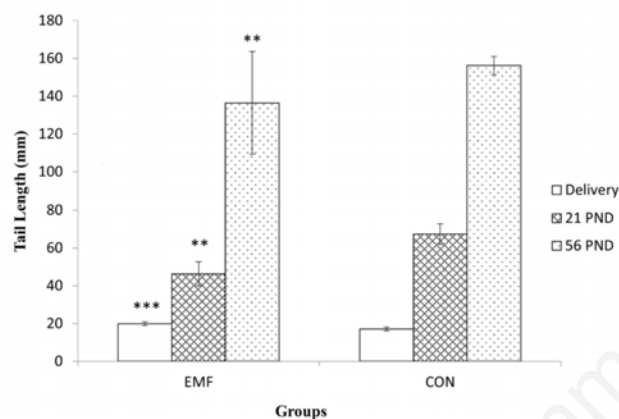


Figure 4. Assessing the average tail length between groups; any values indicate the mean±SD for six rats at electromagnetic fields and control groups. Significant differences from the electromagnetic fields group with $P \leq 0.05$; ** illustrates $P \leq 0.01$ *** shows $P \leq 0.001$.

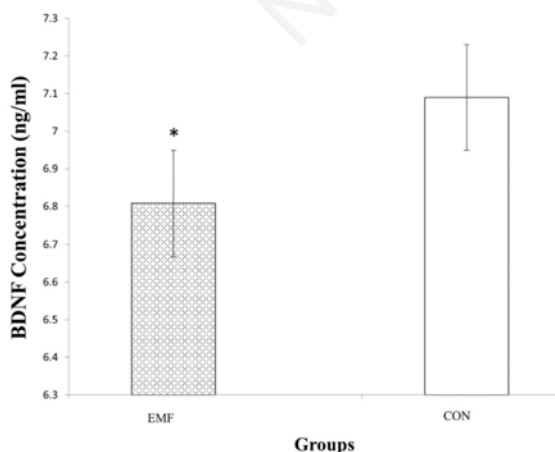


Figure 5. Assessing the brain-derived neurotrophic factor concentration (ng/ml) for subjects at electromagnetic fields, and control groups.

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 chloride and activate calcium-dependent mechanisms⁵¹. 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

References

1. Dasdag S, Akdag MMZ, Erdal ME, et al. Effects of 2.4 GHz radiofrequency radiation emitted from Wi-Fi equipment on microRNA expression in brain tissue. *Int J Radiat Biol* 2015;91:555-61.

2. Li ZQ, Zhang Y, Wan YM, et al. Testing of behavioral and cognitive development in rats after prenatal exposure to 1800 and 2400 MHz radiofrequency fields. *J Radiat Res* 2020;61:197-206.
3. Takahashi S, Imai N, Nabae K, et al. Lack of adverse effects of whole-body exposure to a mobile telecommunication electromagnetic field on the rat fetus. *J Radiat Res* 2010;173:362-72.
4. Lane RH. Fetal programming, epigenetics, and adult onset disease. *Clin Perinatol* 2014;41:815-31.
5. Abdelmelek H, Molnar A, Servais S, et al. Skeletal muscle HSP72 and norepinephrine response to static magnetic field in rat. *J Neural Transm* 2006;113:821-7.
6. Zecca L, Mantegazza C, Margonato V, et al. Biological effects of prolonged exposure to ELF electromagnetic fields in rats: III. 50 Hz electromagnetic fields. *Bioelectromagnetics* 1998;19:57-66.
7. Banaceur S, Banasr S, Sakly M, Abdelmelek H. 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* 2013;240:197-201.
8. Jadidi M, Safari M, Baghian A. Effects of extremely low frequency electromagnetic fields on cell proliferation. *Koomesh* 2013;15:1-10.
9. DastAmooz S, Tahmasebi Boroujeni S, Shahbazi M, Vali Y. Physical activity as an option to reduce adverse effect of EMF exposure during pregnancy. *Int J Dev Neurosci* 2018;71.
10. Jirsa VK, Haken H. Field Theory of Electromagnetic Brain Activity. *Phys Rev Lett* 1996;77:960-3.
11. Li DK, Chen H, Ferber JR, et al. Association Between Maternal Exposure to Magnetic Field Nonionizing Radiation During Pregnancy and Risk of Attention-Deficit/Hyperactivity Disorder in Offspring in a Longitudinal Birth Cohort. *JAMA Netw open* 2020;3:e201417.
12. Balassa T, Szemerszky R, Bárdos GY. Effect of short-term 50 Hz electromagnetic field exposure on the behavior of rats. *Acta Physiol Hung* 2009;96:437-48.
13. Balind SR, Selaković V, Radenović L, et al. Extremely low frequency magnetic field (50 Hz, 0.5 mT) reduces oxidative stress in the brain of gerbils submitted to global cerebral ischemia. Arumugam T V., ed. *PLoS One* 2014;9:e88921.
14. Haarala C, Ek M, Björnberg L, et al. 902 MHz mobile phone does not affect short term memory in humans. *Bioelectromagnetics* 2004;25:452-6.
15. Tapia-Arancibia L, Rage F, Givalois L, Arancibia S. Physiology of BDNF: Focus on hypothalamic function. *Front Neuroendocrinol* 2004;25:77-107.
16. Sasi M, Vignoli B, Canossa M, Blum R. Neurobiology of local and intercellular BDNF signaling. *Pflugers Arch* 2017;469:593-610.
17. Rosas-Vargas H, Martínez-Ezquerro JD, Bienvenu T. Brain-derived neurotrophic factor, food intake regulation, and obesity. *Elsevier* 2011;42:482-94.
18. Gray J, Yeo GSH, Cox JJ, et al. Hyperphagia, severe obesity, impaired cognitive function, and hyperactivity associated with functional loss of one copy of the brain-derived neurotrophic factor (BDNF) gene. *Diabetes* 2006;55:3366-71.
19. Schlotz W, Phillips DIW. Fetal origins of mental health: Evidence and mechanisms. *Brain Behav Immun* 2009;23:905-16.
20. Briana DD, Papastavrou M, Boutsikou M, et al. Differential expression of cord blood neurotrophins in gestational diabetes: the impact of fetal growth abnormalities. *J Matern Neonatal Med* 2018;31:278-83.
21. Mayeur S, Silhol M, Moitrot E, et al. Placental BDNF/TrkB signaling system is modulated by fetal growth disturbances in rat and human. *Placenta* 2010;31:785-91.
22. Glud M, Christiansen T, Larsen LH, et al. Changes in Circulating BDNF in relation to Sex, Diet, and Exercise: A 12-Week Randomized Controlled Study in Overweight and Obese Participants. *J Obes* 2019;2019:1-7.
23. Sandrini L, Di Minno A, Amadio P, et al. Association between obesity and circulating brain-derived neurotrophic factor (BDNF) levels: Systematic review of literature and meta-analysis. *Int J Mol Sci* 2018;19.
24. Kawamura K, Kawamura N, Fukuda J, et al. Regulation of preimplantation embryo development by brain-derived neurotrophic factor. *Dev Biol* 2007;311:147-58.
25. Huber R, Schuderer J, Graf T, et al. Radio Frequency Electromagnetic Field Exposure in Humans: Estimation of SAR Distribution in the Brain, Effects on Sleep and Heart Rate. *Bioelectromagnetics* 2003;24:262-76.
26. Popii V, Baumann G. Laboratory measurement of growth hormone. *Clin Chim Acta* 2004;350:1-16.
27. Goodway J, Ozmun J, Gallahue D. Understanding Motor Development: Infants, Children, Adolescents, Adults 2019.
28. Cruciani S, Campi T, Feliziani M, Maradei F. Optimum coil configuration of wireless power transfer system in presence of shields. *IEEE Int Symp Electromagn Compat* 2015:720-25.
29. DastAmooz S, Tahmasebi Boroujeni S, Shahbazi M, Vali Y. Physical activity as an option to reduce adverse effect of EMF exposure during pregnancy. *Int J Dev Neurosci* 2018;71:10-17.
30. Alchalabi ASH, Aklilu E, Aziz AR, et al. Different periods of intrauterine exposure to electromagnetic field: Influence on female rats' fertility, prenatal and postnatal development. *Asian Pacific J Reprod* 2016;5:14-23.
31. Hand JWW, Li Y, Thomas ELL, et al. Prediction of specific absorption rate in mother and fetus associated with MRI examinations during pregnancy. *Magn Reson Med* 2006;55:883-93.
32. Kikuchi S, Saito K, Takahashi M, et al. SAR computation inside fetus by RF coil during MR imaging employing realistic numerical pregnant woman model. *IEICE Trans Commun* 2009;E92-B:431-9.
33. Shahbazi M, Naghdi N, Tahmasebi S, et al. The effect of iron and zinc dietary restriction of pregnant rats on physical growth of litters. *Biol Trace Elem Res* 2009;128:232-8.
34. Pillai A, Bruno D, Sarreal AS, et al. Plasma BDNF levels vary in relation to body weight in females. Zhang XY, ed. *PLoS One* 2012;7:e39358.
35. Centre AR. Rat and Mice Weights. *Animal Resources C*.
36. Milham S. Evidence that dirty electricity is causing the worldwide epidemics of obesity and diabetes. *Electromagn Biol Med* 2014;33:75-8.
37. Pelleymounter MA, Cullen MJ, Wellman CL. Characteristics of BDNF-induced weight loss. *Exp Neurol* 1995;131:229-38.
38. An JJ, Kinney CE, Tan JW, et al. TrkB-expressing paraventricular hypothalamic neurons suppress appetite through multiple neurocircuits. *Nat Commun* 2020;11:1-16.
39. Barbacid M. The Trk family of neurotrophin receptors. *J Neurobiol* 1994;25:1386-403.
40. Elmquist JK, Maratos-Flier E, Saper CB, Flier JS. Unraveling the central nervous system pathways underlying responses to leptin. *Nat Neurosci* 1998;1:445-50.
41. Vanevski F, Xu B. Molecular and neural bases underlying roles of BDNF in the control of body weight. *Front Neurosci* 2013;7:37.
42. Komori T, Morikawa Y, Nanjo K, Senba E. Induction of brain-derived neurotrophic factor by leptin in the ventromedial hypothalamus. *Neurosci* 2006;139:1107-15.

43. Bariohay B, Lebrun B, Moyses E, Jean A. Brain-derived neurotrophic factor plays a role as an anorexigenic factor in the dorsal vagal complex. *Endocrinol* 2005;146:5612-20.
44. Liao GY, An JJ, Gharami K, et al. Dendritically targeted Bdnf mRNA is essential for energy balance and response to leptin. *Nat Med* 2012;18:564-71.
45. Baydyuk M, Nguyen MT, Xu B. Chronic deprivation of TrkB signaling leads to selective late-onset nigrostriatal dopaminergic degeneration. *Exp Neurol* 2011;228:118-25.
46. Abramson MJ, Benke GP, Dimitriadis C, et al. Mobile telephone use is associated with changes in cognitive function in young adolescents. *Bioelectromagnetics* 2009;30:678-86.
47. Marková E, Malmgren LOGG, Belyaev IY. Microwaves from mobile phones inhibit 53BP1 focus formation in human stem cells more strongly than in differentiated cells: Possible mechanistic link to cancer risk. *Environ Health Perspect* 2010;118:394-9.
48. Akbarnejad Z, Esmailpour K, Shabani M, et al. Spatial memory recovery in Alzheimer's rat model by electromagnetic field exposure. *Int J Neurosci* 2018;128:691-6.
49. Kamal A, Biessels GJ, Duis SEJ, Gispen WH. Learning and hippocampal synaptic plasticity in streptozotocin-diabetic rats: Interaction of diabetes and ageing. *Diabetol* 2000;43:500-6.
50. Jerusalinsky D, Kornisiuk E, Izquierdo I. Cholinergic neurotransmission and synaptic plasticity concerning memory processing. *Neurochem Res* 1997;22:507-15.
51. Teyler TJ, Cavus I, Coussens C, et al. Multideterminant role of calcium in hippocampal synaptic plasticity. *Hippocampus* 1994;4:623-34.
52. Lisi A, Ciotti MT, Ledda M, et al. Exposure to 50 Hz electromagnetic radiation promote early maturation and differentiation in newborn rat cerebellar granule neurons. *J Cell Physiol* 2005;204:532-8.
53. Jelenković A, Janać B, Pešić V, et al. Effects of extremely low-frequency magnetic field in the brain of rats. *Brain Res Bull* 2006;68:355-60.

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