Evaluation of 900 and 1800 Mhz Radiofrequency Radiation Emitted from Mobile Phones on Pregnant Women

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Abstract

The purpose of the present study was to investigate effects of radiofrequency radiation (RFR) emitted from mobile phones (MOBPHs) on pregnant women and fetuses (PW&F). Correlations between effects occurring and MOBPH usage duration (MOBPH-UD) and frequency of the RFR emitted were evaluated. The study comprised pregnant women divided into 4 groups: (control) non-MOBPH users; 1) MOBPH-UD: 2–15 min/day; 2) MOBPH-UD: 15–60 min/day; and 3) MOBPH-UD: >60 min/day.

We investigated were the effects of RFR exposure on protein carbonyl (PCO), malondialdehyde (MDA), total oxidant and antioxidant status (TOS, TAS), 8-hydroxy-2- deoxyguanosine (8-OHDG), and DNA single-strand breaks (SSBs) in the cord blood and placenta of pregnant women who used MOBPHs for different durations. A positive correlation existed between the MOBPH-UD and harmful effects examined. Additionally, the alkaline comet assay for determination of DNA SBs showed that using MOBPHs caused DNA SSBs.

The results indicated that 900 and 1800 MHz RFR had great potential to affect PW&F. However, a correlation existed between the MOBPH-UD and harmful effects. In conclusion, the results suggested that exposure to 900 and 1800 MHz RFR from MOBPHs usage could potentially affect PW&F.

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Introduction

In recent years, with technological advancements, MOBPHs have become indispensable in daily life. Radiofrequency radiation (RFR) is used to transmit data (voice, image, etc.) to/from MOBPHs. Public concern about potential adverse health effects of RFR, which spreads worldwide via these devices, on the environment increases daily. Mobile phones (MOBPHs) produce relatively low-intensity RFR; therefore, researchers mostly focus on non-thermal effects. However, some studies indicated that RFR could degrade structures of biomolecules like protein, lipid, and DNA, through oxidative stress.[1,2] RFR causes oxidative stress by increasing free radicals or decreasing antioxidants.[2] However, a contradiction exists among some studies performed in this area. The number of human oxidative stress studies on this subject is limited. RFR was reported to cause pronounced adverse effects on pregnant women and fetuses (PW&F), which may be due to increased water content in the organism,[3–5] because higher water content affects dielectric values, which influence RFR energy absorption.[4] Exposure to RFR emitted from Global System for Mobile Communications (GSM) MOBPHs (900 MHz, 217 Hz modulation frequency, pulse width: 577 µs) throughout pregnancy causes a decrease in birth weight.[6] Studies on the relation between the fetus, mother, and RFR exposure are limited and have contradictive results. Moreover, most were animal studies and their experimental setups, techniques, and specific absorption rates (SARs) differed. Therefore, the World Health

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Organization (WHO) stated that reliable human studies are necessary, because animal study data are inadequate and inappropriate. Hence, this study will contribute to the elimination of uncertainties in this area. The purpose herein was to investigate the effects of RFR exposure emitted from MOBPHs on PW&F. Also investigated was the role of the MOBPH usage duration (MOBPH-UD) and frequency of MOBPH RFR. Investigated was the effect of RFR emitted from MOBPHs on cellular oxidative reactions, including protein carbonyl (PCO), malondialdehyde (MDA), total oxidant status (TOS), total antioxidant status (TAS), 8-hydroxy-2 -deoxyguanosine (8-OHdG), and DNA single-strand breaks (SSBs) in cord blood and placenta of pregnant women with varying MOBPH-UD. This study will provide a comprehensive source to stimulate future studies.

Materials and methods

The study protocol was approved by the Ethics Committee of the Medical Faculty of Istanbul Medeniyet University and followed the Declaration of Helsinki guidelines.

Determination and grouping of pregnant women

First, appropriate pregnant women were identified, excluding those who had medical treatment, chronic systemic disease, or multiple pregnancies. Only women with natural pregnancy were included. Volunteer pregnant women, aged 18–40 years, provided signed written consent. They were divided into 4 groups depending on their daily MOBPH usage: Control: non-MOBPH users (n: 37). Group-1: MOBPH-UD: 2–15 min/day (n: 39). Group-2: MOBPH-UD: 15–60 min/day (n: 37). Group-3: MOBPH-UD: >60 min/day (n: 36). The Control consisted of non-MOBPH users during pregnancy to avoid the effects of RFR, and some who never used MOBPHs. Daily MOBPH-UDs during pregnancy were determined via the bills obtained from their GSM providers. The RFs of the MOBPHs used were determined by their subscribed GSM providers. A questionnaire containing all confounding factors that could affect the results herein was administered. Newborns were clinically examined by a pediatrician just after delivery. Cord blood and placenta tissues were collected immediately as well.

Placental tissue isolation and analysis

A scalpel was used to collect 1 × 1-cm tissue samples from the central section of the maternal placenta surface, which were weighed, minced, and homogenized in phosphate buffered saline (PBS; pH: 7.4; 9 mL of PBS/1 g of tissue) with a glass homogenizer on ice. A protease inhibitor (aprotinin) was added to the PBS solution. Further disintegration of the cells entailed sonification of the suspension with an ultrasonic cell disrupter (MP fast prep-24 tissue and cell homogenizer, USA). The homogenates were centrifuged for 15 min at 15,000 g to obtain supernatants. The total tissue protein content was measured via the Bradford method, spectrophotometrically.

Isolation and Analysis of Umbilical Blood Samples

When birth occurred, the umbilical cord was clamped and 1–2 mL of blood was collected from the umbilical artery using a previously marked injector. These samples were transferred to biochemical tubes and centrifuged at 3000xg for 10 min. Serum samples were placed into Eppendorf tubes and stored at –80 °C.

Additionally, full blood samples were collected from the umbilical cord using heparinized tubes after delivery. Immediately after blood sample collection, lymphocyte separation was performed.

8-0HdG, MDA, PCO analysis in cord blood and placental tissue samples

Serum and tissue homogenate MDA, PCO, and 8-OHdG (Eastbiopharm, Catalogue numbers: CK-E10376, CK-E11583, and CK-E11652, respectively) levels were measured using commercial ELISA kits. The kit detection ranges were MDA: 2–64 nmol/mL, PCO: 10–640 ng/mL, and 8-0HDG: 10–128 ng/mL.

TAS and TOS analysis in cord blood and placental tissue samples

TAS levels were measured spectrophotometrically with a commercial kit (Rel Assay, Turkey) using a Perkin Elmer, 1420 Victor 3 instrument (Waltham, MA, USA). The TOS levels were measured spectrophotometrically using a commercial kit (Rel Assay, Turkey), using a Perkin Elmer, 1420 Victor 3 instrument. Measured TAS and TOS values were used in the oxidative stress index (OSI) calculation: OSI = TOS/TAS×100.

Alkaline Comet assay analysis

(single-cell gel electrophoresis)

Alkaline comet assay (ACA) was used herein. All steps were conducted under dimmed light. First, each microscope slide was precoated with a 0.5% normal melting agarose layer in distilled water and dried thoroughly at room temperature. Next, 10 µL cell suspension was mixed with 85 µL low-melting agarose (0.7% in PBS solution, pH: 7.4; 37 °C) and dripped onto the first layer. Slides were allowed to solidify for 5 min/4 °C in a moist box. Coverslips were gently placed on the slides and left in a humid chamber for 7–10 min/4 °C. After, coverslips were delicately removed and the slides were placed in lysis solution (146.1 g $NaCl_2$, 1.2 g Trisma Base, 37.2 g EDTA, 1% Triton-X, pH: 10.0). Then, the slides were removed, drained, and placed in a horizontal electrophoresis unit filled with fresh alkaline electrophoresis solution (TBE), containing 54 g Tris, 27.5 g boric acid, 20 mL EDTA (pH: 8.4) for 20 min to allow for DNA unwinding. Electrophoresis was performed for 18 min/room temperature/25 V and was adjusted to 300 mA. Observations were made at 400x using a Leica DM 1000 Led fluorescent microscope (Wetzlar, Germany). Images of 100 randomly chosen nuclei were analyzed via ACA software IV (Perceptive Instruments, Suffolk, UK).

Statistical Analyses

All statistical analyses were performed with the STATA/MP11 program. Data were expressed as number and percentage or mean ± SD. Normal distribution data were analyzed by ANOVA and post-hoc Bonferroni test, while those without normal distribution were analyzed by Kruskal–Wallis test and post-hoc Dunn test. $P < 0.05$ was considered statistically significant.

Results

Analysis of daily MOBPH-UD and DNA SSBs in cord blood lymphocytes

Results of cord blood lymphocyte ACA [tail intensity (TI), tail moment (TM)] are shown in Table 1. A significant increase was observed in TI and TM values in all of the MOBPH usage groups (MOBPH-UGs) compared to the control ($P < 0.001$). ACA results also indicated that TI and TM increased as the daily MOBPH-UD increased. Moreover, the DNA damage was graded from 0 to 4 based on the appearance of the lymphocytes (Figure 1).

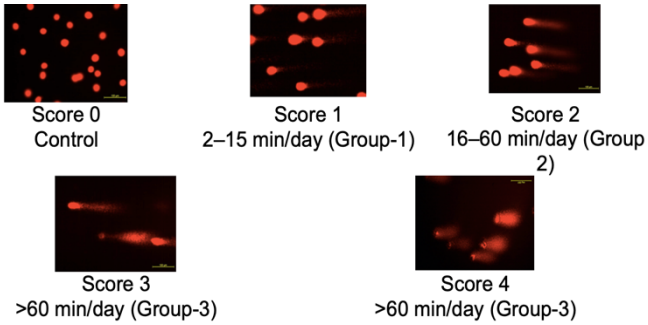

Figure 1. ACA images of the cord blood lymphocytes according to the daily MOBPH-UD.

Oxidative stress parameters findings in cord blood based on MOBPH emission frequency and MOBPH-UD

No significant difference was observed between the 900- and 1800-MHz MOBPH users in terms of the oxidative stress parameters (8-OHdG, MDA, PCO, TAS, TOS, and OSI) in the cord blood. Hence, the frequency did not play an important role. Detailed results are shown in Table 1. However, a correlation between the MOBPH-UD and oxidative stress parameters was found in the cord blood. The 8-OHdG and MDA levels of the cord blood with exposure to 900 and 1800 MHz RFR emitted from MOBPHs increased in Groups 2 and 3 compared to other groups ($P < 0.001$). A significant increase was observed in the PCO levels of the 900- and 1800-MHz MOBPH users (Groups 1–3) compared to the control ($P < 0.05$). The TAS level was quite low in Groups 2 and 3 compared to the control and Group-1, only in women using 900-MHz MOBPHs ($P < 0.05$). TOS was also found to be higher in Groups 2 and 3 compared to the control and Group-1, only in women using 900-MHz MOBPHs ($P < 0.05$). The OSI value was higher in Groups 2 and 3 than in the control and Group-1, only in women using 900-MHz MOBPHs ($P < 0.05$).

Oxidative stress parameters findings in placenta with respect to MOBPH emission frequency and MOBPH talking duration

No significant difference was observed between the 900 and 1800 MHz MOBPH users in terms of the oxidative stress parameters (8-OHdG, MDA, PCO, TAS, TOS, and OSI) in the placenta, indicating that frequency had no important role in the effects. A correlation existed between the MOBPH-UD and oxidative stress parameters in the placenta. The 8-OHdG, MDA, and PCO levels in Group-3 were significantly

higher than in the other groups (P < 0.001) with exposure to 900- and 1800-MHz of RFR. The PCO results of the MOBPH-UGs were also increased compared to the control (P < 0.05). The lowest TAS in the MOBPH-UGs was observed in Group-3. The TOS was higher in Groups 2 and 3 compared to the control and Group-1, only in women using 900-MHz MOBPHs (P < 0.05). The OSI value was higher in Group-3 compared to the other groups (P < 0.001) Detailed comparison results for the groups are shown in Table 2.

Confounding Factors

The confounding factors, comprising paternal age, maternal weight gain during pregnancy, infant weight, and phone SAR values, are shown in Table 3. No statistical difference was found in these parameters among the research groups.

Statistical Analysis

Descriptive statistics were presented as the mean and SD. Two-way ANOVA was performed to analyze the characteristics. Following ANOVA, the Duncan multiple comparison test was used to identify different means. Statistical significance was accepted as 5%. IBM SPSS Statistics for Windows 21.0 (IBM Corp., Armonk, NY, USA) was used for all statistical computations.

Discussion

Living organisms developed complex antioxidant defense mechanisms to protect from free radical attacks. Oxidative stress, which leads to tissue damage, results from an imbalance between antioxidant defense and the amount of free radicals.[7] Some recent studies reported that RFR emitted from MOBPHs caused oxidative stress during pregnancy.[7] It was reported that pre- and postnatal exposure to 900, 1800, and 2450 MHz RFR could cause decreased TAS and GSH, and increased MDA in the kidneys of offspring.[8] Another study indicated that prenatal RFR exposure (900 MHz, SAR 0.027 W/kg) increased MDA and SOD levels, and decreased glutathione in the liver of newborn rats.[9] It was stated that RFR exposure (2.45 GHz, continuous wave, SAR: 0.023 W/kg) during mating and throughout pregnancy caused DNA damage in the brain and decreased antioxidant enzyme activity in the liver, kidney, and ovary.[10] Herein, it was similarly found that RFR exposure during pregnancy increased the TOS and decreased the TAS in cord blood. The TAS in Groups 2 and 3 was significantly lower than in the Control group and Group-1, only in women using 900-MHz MOBPHs (P < 0.05). An inverse relationship existed between the cord blood TAS and daily MOBPH-UD in all of the MOBPH-UGs. The TOS in Groups 2 and 3 were higher than in Group-1 and the Control. Moreover, the OSI in Groups 2 and 3 were higher than in Group-1 and the Control, only in women using 900-MHz MOBPHs (P < 0.05).

It was found that RFR exposure (1800 MHz GSM, 15 min/day, between days 15 and 22 of pregnancy) led to oxidative damage in lipid and DNA molecules caused by the release of free radicals in the brain of pregnant rabbits, whereas no significant difference was observed in the newborns. However, under the same experimental conditions, an increase was observed in the lipid peroxidation and 8-OHdG in the liver of newborn rabbits.[11] However, RFR exposure (analogue MOBPH, 834 MHz, 8.5 h/day during pregnancy) did not caused a significant difference in the blood and liver oxidative stress parameters of rat offspring, except for an increase in micronucleus formation.[12] RFR exposure (15 min/day, for 1 week, GSM 1800 MHz) was also reported to increase MDA in non-pregnant and pregnant rabbits. No significant change was observed in the liver MDA of newborn rabbits; however, the 8-OHdG was increased in the liver.[13]

Most of the animal studies discussed above indicated that RFR has the potential to cause an increase of MDA in the liver, brain, and blood. Herein, the cord blood MDA levels of the MOBPH-UGs were higher than in the controls (P < 0.001). A positive correlation existed between the MOBPH-UD and cord blood MDA. Therefore, the results of previous animal studies supported these results.[8,14] The PCO levels in the MOBPH-UGs were higher than in the controls, especially in women using 900-MHz MOBPHs (P < 0.001). Moreover, a positive relationship existed between MOBPH-UD and cord blood PCO. PCO studies in pregnancy are rare; however, one study was found,[12] the results of which did not support the results herein. The reason for the contradiction may be because different MOBPH systems were studied. They investigated the bio-effects of analogue, while the digital MOBPH system was

studied herein.

In the present study, the cord blood 8-OHdG (a marker of oxidative DNA damage) in Groups 2 and 3 was higher than in the control. Similarly a significant increase was observed in the TI and TM values in all of the MOBPH-UGs compared to the control ($P < 0.001$). Comet results also indicated that TI and TM increased as daily MOBPH-UD increased. In this study, an agreement was found between the results of 8-OHDG and DNA SSBs. Therefore, this was the most important data of this study, which pointed to the possible damage potential to biomolecules by the RFR emitted from MOBPHs. As a result, it can be speculated that RFR may have the potential to cause fetal DNA damage; thus, pregnant MOBPH users need to protect themselves, their embryos, and fetuses. These results were supported by animal studies.[8,9,11–14] Another important result of this study was the positive correlation between MOBPH-UD and the parameters investigated in the cord blood.

The second step of the present study was to investigate the effects of MOBPH-UD on the same parameters discussed above in the placenta. A correlation existed between MOBPH-UD and the oxidative stress parameters in placenta. It was reported that, in the early period of pregnancy, exposure to RFR emitted from MOBPHs (GSM 900 MHz, SAR 1.46 W/kg) may alter the profile of chorionic tissue proteins, and cause adverse effects on cell proliferation and prevent the development of the nervous system.[15] It was also indicated that RFR (915 MHz, for 90 min) had the potential to increase utero-placental blood flow and estradiol in pregnant rats (16).

The lowest TAS in all of the MOBPH-UGs was observed in Group-3. TOS was also found to be higher in Groups 2 and 3 according to the control and the Group-1, only in women using 900-MHz MOBPHs ($P < 0.05$). On the other hand, the OSI value was also higher in Group-3 compared to the other groups ($P < 0.001$).

In this study, the placental 8-OHdG, MDA, and PCO levels in Group-3 were higher than in the other groups ($P < 0.001$) with exposure to both 900 and 1800 MHz RFR-emitting MOBPHs. The 8-OHdG and PCO results of all of the MOBPH-UGs were also increased significantly compared to the control ($P < 0.05$). However, the increase of MDA between Group-1 and the control was not statistically significant. Placental TOS and OSI in Groups 2 and 3 were also higher than in the control and Group-1 ($P < 0.001$), whereas placental TAS in Group-3 was lower than in the control ($P < 0.001$). In these cases, no significant difference was found between Group-1 and the control. It is possible that the exposure in Group-1 was too brief to cause effects. Thus, the results indicated that RFR exposure emitted from MOBPHs has the potential to enhance oxidative stress in the placenta.

No statistically significance difference was found between the RFR frequencies on the parameters investigated in this study ($P > 0.05$). This indicated that frequency was not an important factor of effects, at least for 900 and 1800 MHz. These results were supported by some previous studies.[8,17–21]

Numerous studies found that exposure to RFR under safety limits caused adverse health effects in living organisms. Recently, some animal studies have indicated that exposure to RFR in pregnancy has induced some adverse effects in infants.[22–24] However, human studies of RFR exposure during pregnancy and embryogenesis are almost nonexistent. Moreover, in a previously conducted human study, it was found that exposure to RFR during pregnancy caused significant changes in the biochemical parameters of cord blood.[25] The results of this study also indicated that RFR exposure was not as benign as is known during pregnancy. This work was the first detailed human study carried out on the effects of MOBPH radiation exposure on the bio-molecular parameters in placenta and cord blood.

Conclusions

The results of the present study indicated that exposure to 900 and 1800 MHz RFR emitted from MOBPHs could potentially have adverse health effects on PW&F. Moreover, a positive correlation was found between daily MOBPH-UD and the detected harmful effects in this study. Therefore, it is suggested that women should protect themselves from unnecessary RFR exposure during pregnancy. More detailed human bio-molecular studies are needed to further understand the topic and set up safe exposure guidelines.

Declaration of Interest

The authors report no conflict of interest.

	Serum	900 MHz (n: 80)			1800 MHz (n: 32)			P-value
	Usage duration	Mean	SD±	Median	Mean	SD±	Median	(900–1800 MHz)
Tail Intensity	Control	24.28 d	3.63	23.91	24.28 d	3.63	23.91	-
	2–15 min/day	38.11 c	8.82	35.78	37.97 c	9.00	35.73	0.966
	15–60 min/day	45.01 b	8.46	42.37	44.66 b	9.79	42.84	0.913
	>60 min/day	57.56 a	7.12	57.96	57.42 a	5.61	56.93	0.955
	P-value	0.001			0.001			
Tail Moment	Control	39.86 d	9.16	37.33	39.86 d	9.16	37.33	-
	2–15 min/day	46.96 c	9.20	43.09	42.58 c	5.16	41.44	0.164
	15–60 min/day	113.65 b	13.76	114.37	117.33 b	17.60	119.21	0.499
	>60 min/day	217.83 a	64.70	218.26	177.01 a	85.77	165.09	0.124
	P-value	0.001			0.001			
8OHdG (ng/mL)	Control	81.09 b	10.49	82.44	81.09 b	10.49	82.44	-
	2–15 min/day	89.20 b	8.42	89.70	84.29 b	11.84	86.43	0.161
	15–60 min/day	95.95 a	9.25	96.84	94.36 a	13.25	97.45	0.325
	>60 min/day	112.85 a	10.98	113.99	110.53 a	13.82	114.09	0.874
	P-value	0.001			0.001			
MDA (nmol/mL)	Control	11.05 c	7.66	9.15	11.05 c	7.66	9.15	-
	2–15 min/day	14.44 c	7.33	12.71	17.38 c	8.26	16.12	0.297
	15–60 min/day	24.10 b	8.87	22.34	24.31 b	6.25	22.64	0.678
	>60 min/day	41.13 a	7.36	38.70	43.36 a	9.95	42.09	0.458
	P-value	0.001			0.001			
PCO (ng/ml)	Control	65.27 c	14.57	62.78	65.27 b	14.57	62.78	-
	2–15 min/day	78.22 b	14.40	76.79	76.77 b	15.38	81.29	0.790
	15–60 min/day	83.10 b	12.26	81.23	91.55 a	15.60	92.98	0.086
	>60 min/day	91.29 a	13.03	89.62	95.63 a	14.00	95.95	0.374
	P-value	0.002			0.019			
TAS (mmol/L)	Control	1.20 a	0.47	0.95	1.20	0.47	0.95	-
	2–15 min/day	1.13 a	0.49	0.89	0.97	0.37	0.82	0.351
	15–60 min/day	0.78 b	0.42	0.61	0.83	0.48	0.57	0.724
	>60 min/day	0.84 b	0.13	0.88	0.80	0.13	0.81	0.393
	P-value	0.002			0.540			
TOS (µmol/L)	Control	9.59 b	1.96	8.99	9.59 b	1.96	8.99	-
	2–15 min/day	10.10 ab	2.11	9.78	10.80 ab	1.87	10.81	0.364
	15–60 min/day	11.13 a	1.89	10.82	11.14 a	2.35	10.13	0.989
	>60 min/day	12.14 a	1.94	11.55	11.63 a	2.25	10.78	0.492
	P-value	0.002			0.681			
OCI	Control	9.29 b	3.93	10.06	9.29 b	3.93	10.06	-
	2–15 min/day	10.92 b	5.51	11.77	12.50 ab	4.59	13.23	0.423
	15–60 min/day	18.29 a	8.82	18.89	17.04 a	8.21	16.90	0.691
	>60 min/day	14.77 ab	3.34	15.75	14.82 ab	3.73	13.91	0.968
	P-value	0.001			0.226			

Table 1. Comparison of serum parameters depend on frequency and MOBPH talking duration (min/day).

	Placenta	900 MHz (n: 80)			1800 MHz (n: 32)			P-value (900–1800 MHz)
	Usage duration	Mean	SD±	Median	Mean	SD±	Median	
8OHdG (ng/mL)	Control	68.28 c	10.54	69.44	68.28 b	10.54	69.44	-
	2–15 min/day	83.69 b	8.37	83.72	78.91 b	11.87	81.45	0.171
	15–60 min/day	91.14 b	9.68	92.88	91.25 a	11.07	91.80	0.976
	>60 min/day	106.52 a	11.39	108.19	104.16 a	14.01	107.29	0.597
	P-value	0.001			0.001			
MDA (nmol/mL)	Control	14.80 c	7.73	12.70	14.80 c	7.73	12.70	-
	2–15 min/day	17.51 c	7.31	16.07	20.85 c	8.46	19.35	0.238
	15–60 min/day	34.69 b	8.57	33.47	34.50 b	6.31	32.96	0.948
	>60 min/day	46.47 a	8.64	43.23	50.21 a	11.26	50.78	0.284
	P-value	0.001			0.001			
PCO (ng/mL)	Control	59.53 c	14.40	57.86	59.53 c	14.40	57.86	-
	2–15 min/day	72.33 b	14.63	70.24	71.69 b	14.52	75.74	0.905
	15–60 min/day	76.91 b	13.96	75.33	81.51 b	8.33	82.03	0.318
	>60 min/day	87.14 a	12.92	86.32	87.61 a	10.73	88.62	0.916
	P-value	0.001			0.012			
TAS (mmol/L)	Control	1.23 a	0.46	1.00	1.23 a	0.46	1.23 a	-
	2–15 min/day	1.01 a	0.39	0.91	1.33 a	0.41	1.33 a	0.054
	15–60 min/day	1.00 a	0.44	0.80	0.81ab	0.46	0.81ab	0.237
	>60 min/day	0.62 b	0.10	0.63	0.58 b	0.09	0.58 b	0.365
	P-value	0.001			0.001			
TOS (µmol/L)	Control	10.33 b	2.21	9.92	10.33 b	2.21	9.92	-
	2–15 min/day	10.31 b	1.98	9.67	10.52 b	2.37	9.49	0.783
	15–60 min/day	11.58 a	2.05	10.90	11.78 a	2.65	12.33	0.802
	>60 min/day	12.48 a	2.08	12.37	12.44 a	1.41	12.03	0.957
	P-value	0.001			0.147			
OCI	Control	9.55 b	3.94	9.35	9.55 b	3.94	9.35	-
	2–15 min/day	11.44 b	4.17	11.17	8.60 b	2.91	8.35	0.054
	15–60 min/day	13.53 b	5.51	12.58	18.08 a	8.98	20.59	0.067
	>60 min/day	20.81a	5.10	19.25	21.72 a	3.46	21.07	0.593
	P-value	0.001			0.001			

Table 2. Comparison of placental parameters depending on frequency and MOBPH-UD (min/day).

	Confounding factors	900 MHz			1800 MHz			P-value
	Usage duration	Mean	SD±	Median	Mean	SD±	Median	(900–1800 MHz)
Maternal age	Control	27.97	5.23	28.00	27.97	5.23	28.00	-
	2–15 min/day	29.41	5.49	28.00	30.20	6.09	29.00	0.706
	15–60 min/day	27.88	3.66	28.00	26.55	3.64	26.00	0.315
	>60 min/day	29.08	6.30	30.00	29.64	4.03	29.00	0.790
	P-value	0.538			0.165			
Paternal age	Control	31.19	5.08	32.00	31.19	5.08	32.00	-
	2–15 min/day	32.62	4.70	33.00	32.90	6.10	33.00	0.882
	15–60 min/day	31.65	4.65	32.00	30.18	4.19	30.00	0.372
	>60 min/day	33.04	6.43	33.00	33.45	4.70	33.00	0.849
	P-value	0.629			0.281			
during pregnancy (maternal)	Control	10.92	5.33	10.00	10.92	5.33	10.00	-
	2–15 min/day	9.16	3.65	8.00	12.30	5.87	10.00	0.053
	15–60 min/day	11.27	4.36	10.00	9.45	3.05	8.00	0.219
	>60 min/day	12.24	4.74	11.00	11.55	6.50	9.00	0.720
	P-value	0.057			0.453			
Infant weight (gr)	Control	3147.57	475.92	3250.00	3147.57	475.92	3250.00	-
	2–15 min/day	3168.62	307.34	3150.00	3314.00	697.83	3025.00	0.369
	15–60 min/day	3028.46	479.57	3000.00	3070.00	356.12	3000.00	0.798
	>60 min/day	2979.20	521.70	2900.00	2960.00	394.94	2850.00	0.914
	P-value	0.261			0.270			
SAR (W/kg)	Control	0.51	0.31	0.56	0.51	0.31	0.56	-
	2–15 min/day	0.58	0.25	0.58	0.55	0.23	0.57	0.759
	15–60 min/day	0.64	0.24	0.57	0.69	0.19	0.58	0.556
	>60 min/day	0.56	0.15	0.57	0.59	0.25	0.58	0.657
	P-value	0.415			0.374			

Table 3. Comparison of confounding factors that affected the results according to frequency and MOBPH-UD (min/day).
Different lower cases in each column represent statistically significant differences.

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