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Original Article

Prenatal Exposure to 3.5 GHz Radiofrequency Radiation and Long-Term Skin Histomorphometry: An 18-Month Experimental Rat Study

3.5 GHz Radyofrekans Radyasyonuna Prenatal Maruziyet ve Uzun Dönem Deri Histomorfometrisi: 18 Aylık Deneysel Rat Çalışması

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ABSTRACT

Objective: This study aimed to evaluate long-term skin histomorphometry at 18 months postpartum in rats exposed in utero to 3.5 GHz radiofrequency radiation (RFR).

Methods: Pregnant Wistar Hannover rats were exposed to Global System for Mobile Communications-modulated 3.5 GHz RFR for 2 h/day throughout gestation, while the sham group underwent mock exposure. Offspring (n=5 per group) were not exposed to any further RFR until 18 months after birth. Dorsal skin samples were stained with hematoxylin–eosin and Masson's trichrome, and dermal thickness, adipose tissue area, dermal area, adipose/dermis ratio, and fat percentage were quantified. Specific absorption rate (SAR) was calculated using CST Studio Suite. Data were analyzed using the Student's t-test or the Mann-Whitney U test. Statistical significance was defined as $p<0.05$.

Results: The peak spatial SAR (psSAR) values were 0.06622 mW/g (for 1 g) and 0.03825 mW/g (for 10 g). No statistically significant differences were observed between RFR-exposed and sham groups in dermal thickness (655.32±87.46 µm vs 544.42±135.01 µm); fat area percentage (0.73±0.29% vs 0.66±0.22%); dermal area (1.05±0.17 vs 0.88±0.22); adipose/dermis ratio (1.78 ± 0.24 vs 1.54±0.28); or fat percentage (40.04±11.78% vs 42.96±11.60%) (all $p>0.05$).

Conclusions: Prenatal exposure to 3.5 GHz RFR did not cause significant skin histomorphometric alterations in the dermis of aged female rats. The skin's barrier properties, regenerative capacity, and repair mechanisms may mitigate long-term structural effects of such exposures.

Keywords: Radiofrequency radiation, prenatal exposure, skin histomorphometry, 3.5 GHz

ÖZ

Amaç: Bu çalışma, intrauterin dönemde 3,5 GHz radyofrekans radyasyonuna (RFR) maruz kalan ratlarda doğum sonrası 18. ayda uzun dönem deri histomorfometrisini değerlendirmeyi amaçlamaktadır.

Yöntemler: Gebelik süresince Wistar Hannover ratları, deney grubunda günde 2 saat Global System for Mobile Communications modülasyonlu 3.5 GHz RFR'ye maruz bırakılırken, yalancı (sham) gruba simüle maruziyet uygulanmıştır. Doğan yavrular (her grupta n=5), 18 aylık olana kadar ek RFR maruziyeti olmaksızın yetiştirilmiştir. Sırt bölgesinden alınan deri örnekleri hematoksilen–eozin ve Masson trikrom ile boyanmış; dermis kalınlığı, yağ dokusu alanı, dermal alan, yağ/dermis oranı ve yağ yüzdesi nicel olarak değerlendirilmiştir. Spesifik soğurma oranı (SAR) CST Studio Suite kullanılarak hesaplanmıştır. Veriler Student's t-testi veya Mann-Whitney U testi ile analiz edilmiş; istatistiksel anlamlılık $p<0,05$ olarak kabul edilmiştir.

Bulgular: Tepe uzaysal SAR (psSAR) değerleri 1 g için 0,06622 mW/g ve 10 g için 0,03825 mW/g olarak bulunmuştur. RFR'ye maruz kalan ve sham grupları arasında dermis kalınlığı (655,32±87,46 µm vs 544,42±135,01 µm), yağ alanı yüzdesi (0,73±0,29% vs 0,66±0,22%), dermal alan (1,05±0,17 vs 0,88±0,22), yağ/dermis oranı (1,78±0,24 vs 1,54±0,28) veya yağ yüzdesi (40,04±11,78% vs 42,96±11,60%) açısından istatistiksel olarak anlamlı fark saptanmamıştır (tüm $p>0,05$).

Sonuçlar: Prenatal 3.5 GHz RFR maruziyeti, yaşlı dişi sıçanlarda dermiste anlamlı histomorfometrik değişikliklere yol açmamıştır. Derinin bariyer özellikleri, yenilenme kapasitesi ve onarım mekanizmaları, bu tür maruziyetlerin uzun dönem yapısal etkilerine karşı korunma sağlayabilir.

Anahtar kelimeler: Radyofrekans radyasyonu, prenatal maruziyet, deri histomorfometrisi, 3,5 GHz

INTRODUCTION

In recent years, there has been remarkable progress in information and communication technologies, resulting in the widespread integration into daily life of various electronic communication devices and technologies, such as mobile phones and Wi-Fi, which operate via

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electromagnetic fields (EMFs). Radiofrequency radiation (RFRs), defined as electromagnetic radiation in the frequency range 100 kHz to 300 GHz, is predominantly used in communication technologies, particularly in devices such as mobile phones, base stations, laptops, and other wireless systems[1]. 2G, 3G, 4G, and 5G mobile communication technologies utilize radiofrequency (RF) bands such as 900 MHz, 1800 MHz, 2100 MHz, 2400 MHz, 2600 MHz, and 3500 MHz; 5G also employs higher frequencies. 5G technology, which has recently been adopted in numerous countries, primarily operates in the 3.5-GHz frequency band[2].

The biological effects of RF radiation are generally classified as either thermal or non-thermal. Given the low energy levels of RF radiation used in wireless communications, scientific investigations have largely centered on non-thermal effects[3]. Prior studies suggest that non-thermal RF exposure may induce protein conformational alterations and misfolding[4], activate heat shock proteins (HSPs)[5], and cause oxidative stress[6-8].

Fetuses are considered more susceptible than adults to the potential adverse effects of prolonged radiofrequency electromagnetic field (RF-EMF) exposure because rapidly proliferating tissues, such as the central nervous system, endocrine glands, and skin, are sensitive during development. In a study by Sharma et al.[9], prenatal exposure of mice to microwave radiation led to significant alterations in the cytoarchitecture of the hippocampus and cerebellum, including a reduction in Purkinje cell numbers. Similarly, Jensh[10] reported statistically significant growth retardation in rat fetuses exposed to irradiation. Additionally, Odaci et al.[11] demonstrated that 0.9 GHz EMF exposure during development impaired granule cell formation in the dentate gyrus of the rat hippocampus, potentially resulting in cell loss due to disrupted neurogenesis. In contrast, a study by Shirai et al.[12] found that simultaneous exposure to multiple communication-signal EMFs, ranging from 0.8 GHz to 5.2 GHz, had no adverse effects on pregnancy or development in rats.

The skin, as the largest organ of the human body, serves as a crucial barrier against environmental insults. In addition to RF-EMF, other physical environmental agents, most notably ultraviolet radiation (UV-R), are known to affect the skin. While limited exposure to UV-R is beneficial to health by promoting vitamin D synthesis, excessive exposure is closely associated with DNA mutagenesis, apoptosis, and skin cancers such as cutaneous melanoma[13]. Modern wireless communication systems, including 3G, 4G, and Wi-Fi, operate at higher frequencies than earlier GSM 900 MHz systems. As frequency increases, the penetration depth of RF waves into tissues decreases; consequently, the majority of the RF-specific absorption rate (SAR) is concentrated in the skin. Because of its high water content, the skin absorbs more RF or microwave energy than do organs with lower water content[14,15]. However, RF exposure is not ionizing and is therefore not considered to be directly genotoxic on its own[16]. Fetal skin development involves complex processes such as epidermal proliferation, dermal stratification, and connective tissue organization. Any disruption of these processes by external factors can affect the integrity and function of the skin after birth.

Prior studies have reported that RF exposure associated with mobile phone use leads to increased skin temperature[17,18], induces thermally related stress and injury to the skin, and triggers repair processes involving inflammation and tissue matrix remodeling[19]. Furthermore, RF exposure has been linked to a vasodilatory effect on cutaneous blood flow[20].

In the present study, female rats were exposed to RF radiation at a frequency of 3.5 GHz throughout all trimesters of pregnancy. Their offspring were raised to 18 months of age, at which point skin tissue samples were collected and examined histologically and histomorphometrically. The objective was to investigate the long-term effects of prenatal RF radiation exposure on mature skin architecture. It is anticipated that the findings of this study will help to address the current gaps in the literature on the long-term impacts of intrauterine environmental RF exposure on tissue integrity and will enhance our understanding of potential health risks to humans.

MATERIALS AND METHODS

Animal Experiments

The study involved 10 adult female Wistar Hannover rats (18 months old, weighing 300-350 g), comprising groups with and without intrauterine RFR exposure. The reason for selecting 18-month-old rats in our study was to investigate the long-term effects of prenatal exposure to 3.5 GHz RFR that may manifest during the aging process. The number of animals was determined based on an a priori power analysis and in accordance with the 3R principles (Replacement, Reduction, Refinement). All procedures were designed and performed in accordance with the guidelines of the Animal Experiments Local Ethics Committee of Istanbul Bagcilar Training and Research Hospital with decision no: 2024-53, date: 26.11.2024).

The rats were divided into two groups: a sham group and an experimental group. To obtain offspring for the experimental group, pregnant rats were exposed within a Faraday cage to 3.5 GHz RFR for 2 hours per day from the first day of pregnancy until delivery. Rats in the sham group were maintained under the same experimental conditions, except that the signal generator was turned off. Pregnancy was confirmed by vaginal smear cytology and observation of a copulatory plug. Female rats were housed overnight with males, and the presence of spermatozoa in the vaginal smear the following morning or the detection of a copulatory plug was designated as gestation day 0. From each group, five female newborns were randomly selected following parturition, resulting in a total of 10 offspring, which were then monitored under standard laboratory conditions (room temperature 24±1 °C, 12-hour light/12-hour dark cycle, and ad libitum access to water and rodent pellet feed) for 18 months without additional irradiation. To reduce sex-related variability in dermal and adipose tissue parameters and to ensure a more homogeneous sample, only female offspring were included in the study. At the end of the 18-month period, the animals were deeply anesthetized by intraperitoneal injection of ketamine (80 mg/kg) and xylazine (10 mg/kg). Adequate anesthetic depth was verified by loss of pedal reflex. Subsequently, euthanasia was performed via intracardiac administration of a high dose of pentobarbital. Skin tissue samples were collected from the dorsal region of each rat.

Exposure and Field Measurements

In this study, pregnant rats were exposed to 3.5-GHz GSM-modulated RFR generated by a 1-W signal generator (Model 3500 PM10, Everest Comp., Türkiye) for 2 hours per day throughout pregnancy. The power output of the signal generator was maintained at 1 W during the exposure. Exposure was conducted in specially designed, metal-free Plexiglas cages that were placed inside separate Faraday cages. The generator's antenna, similar to a mobile-phone antenna, was positioned 50 cm above the animals at the center of the cage and fitted with a metal reflector above it. Shams were kept under identical conditions with the generator turned off. After birth, female offspring were not exposed to RFR until 18 months of age, thereby ensuring only prenatal exposure. Electric field strengths measured with an EMR 300 device (NARDA, Pfullingen, Germany) ranged from 24 V/m to 27 V/m at the cage corners and were 28 V/m at the center.

SAR Analysis

SAR in the rat uterus was simulated using CST Studio Suite (CST AG, Germany) with a detailed voxel-based rat model derived from CT scans. The simulation replicated the experimental setup, including the antenna, reflector, Plexiglas carousel, and rat positioning. The finite integration technique (FIT) was applied to perform precise electromagnetic and thermal calculations. Electric field strengths, measured with a calibrated EMR-300 device (Narda, Germany) at the four cage corners and at the center, ranged from 24-27 V/m at the corners and 28 V/m at the center, confirming a uniform field distribution. Peak spatial SAR (psSAR) simulations were based on the average 28 V/m field at 3.5 GHz. The exposure plane remained fixed, with no metallic surfaces present, and no body temperature changes were detected during exposure.

Histomorphometric Studies

Skin tissue samples obtained from the dorsal region of the rats were fixed in 10% neutral buffered formalin, processed through routine tissue preparation procedures, and embedded in paraffin. 5-µm sections were cut on a Leica RM2245 microtome and mounted on standard glass slides. For histological evaluation, the slides were stained with hematoxylin and eosin (H&E) to assess dermal thickness, dermal adipose tissue area, and total dermal area (dermis and adipose tissue), and with Masson's trichrome to evaluate the general structural features of the skin and the organization of collagen fibers.

All sections were examined using a light microscope (Olympus BX53) for histological and histomorphometric analyses. A camera and imaging system (Olympus DP21) attached to the microscope was used for measurements and photodocumentation. Dermal thickness was measured in eight fields per section at 10× magnification. Areas of dermal adipose tissue, dermis, and total dermal area were measured at 4× magnification using a guiding line approximately 1.5 mm long. The proportion of dermal adipose tissue was calculated as the adipose tissue area divided by the total dermal area.

Statistical Analysis

SPSS (Statistical Package for Social Sciences) (for Windows, version 22.0) (SPSS Inc., Chicago, IL, USA) software package was used for the statistical analysis of the obtained data. The data distribution was evaluated using the Shapiro-Wilk test. Differences between groups in terms of dermis thickness, dermal adipose tissue area, dermal adipose tissue ratio, and dermis area were evaluated using the Student's t-test. Differences in total dermal area between groups were analyzed using the Mann-Whitney U test. Significant p-values were denoted as $p<0.05$ (*), $p<0.01$ (**), and $p<0.001$ (***). All data were reported as mean ± standard error of the mean.

RESULTS

In this study, the effects of prenatal exposure to RFR during all trimesters on the skin morphology of female rats at 18 months of age were investigated. Histological analyses were performed using H&E staining, and several parameters including dermal thickness, dermis area, dermal adipose tissue area, total dermal area, and dermal adipose tissue ratio were quantitatively measured. Representative histological images illustrating the skin tissue architecture in both groups are shown in Figure 1.

The SAR calculations were conducted using the Institute of Electrical and Electronics Engineers/ International Electrotechnical Commission 62704-1 standard to assess the distribution of SAR values. The PsSAR in the uterus was 0.06622 mW/g for 1 g and 0.03825 mW/g for 10 g.

No statistically significant differences were observed between the sham and RFR-exposed groups in any of the measured parameters. Mean dermal thickness values were 544.42±60.38 µm in the sham group and 655.32±39.11 µm in the RFR-exposed group (p=0.162). Similarly, dermal adipose tissue areas showed no significant difference, with means of 0.66±0.10 in the sham group and 0.73±0.13 in the experimental group (p=0.717).

The dermis area was slightly increased in the RFR group (1.05±0.08) compared with the sham group (0.88±0.10), although this difference was not statistically significant (p=0.198). The total dermal area did not differ significantly between groups (sham: 1.54±0.13; RFR: 1.78±0.11; p=0.175). Finally, dermal adipose tissue ratio did not differ between groups (sham: 42.96±5.19%; RFR: 40.04±5.27%; p=0.704).

These findings suggest that chronic intrauterine exposure to RFR does not cause significant morphological changes in the dermal structure of 18-month-old female rats. The comparative statistical results for dermal

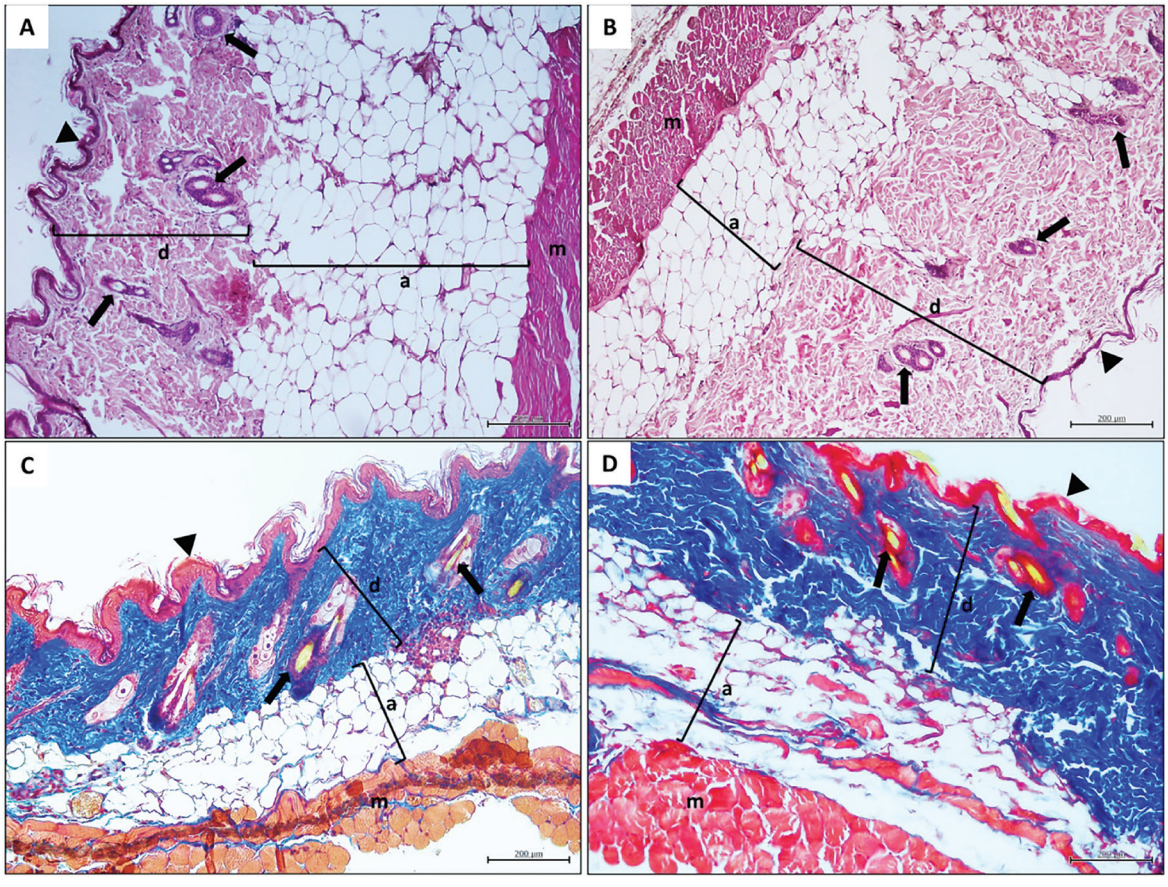

Figure 1. Representative hematoxylin&eosin (H&E) and Masson's Trichrome stained sections of skin tissue from female offspring rats at 18 months of age. Panels A and C: Sham group; Panels B and D: Prenatal RFR exposure group. Arrowheads indicate the epidermal layer; black arrows indicate hair follicles; d = dermis layer; a = adipose tissue layer; m = muscle layer. Scale bars: 200 µm.

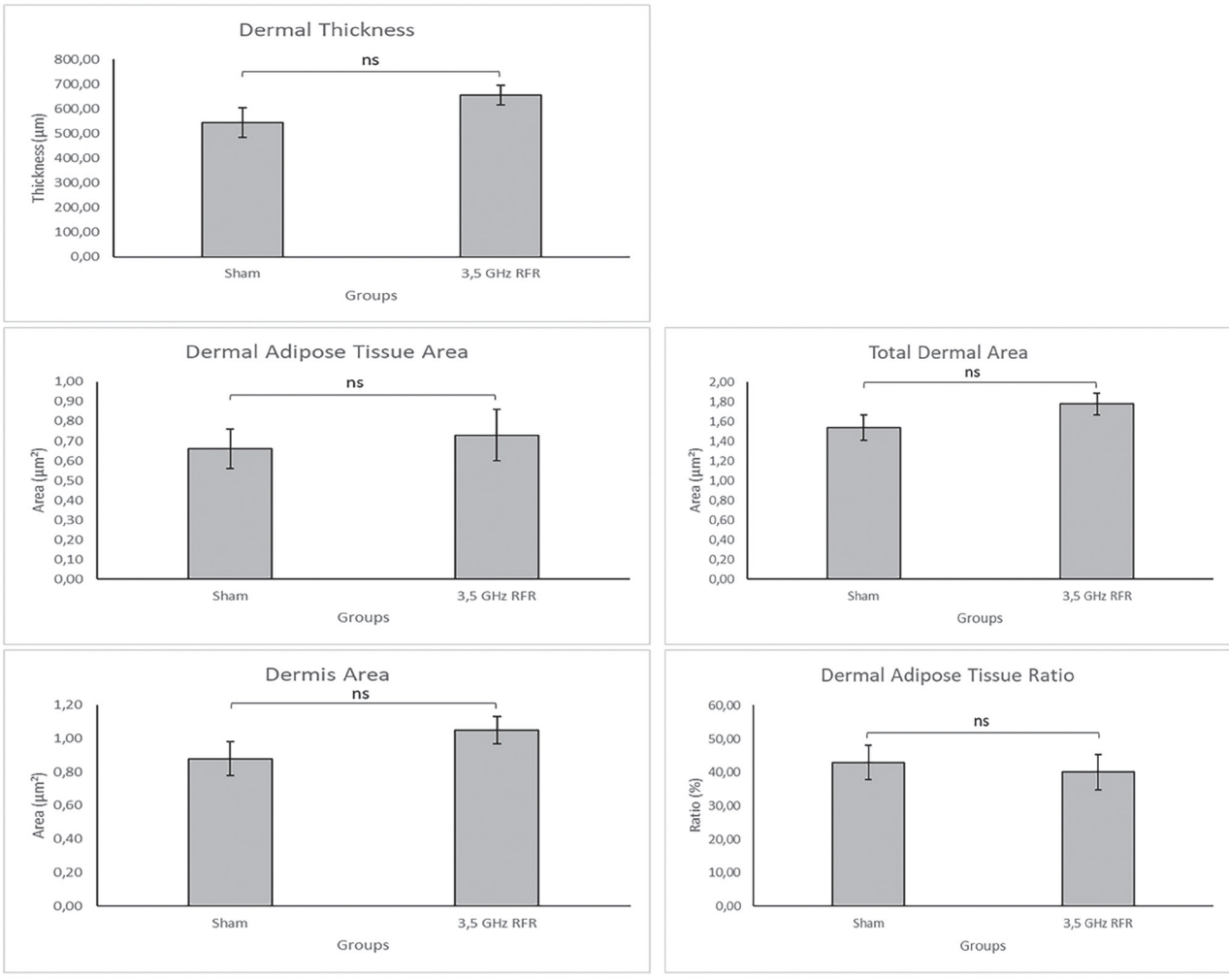

Figure 2. Comparison of histomorphometric parameters in skin tissue of female offspring rats with and without prenatal RFR exposure at 18 months of age. Bar graphs represent mean ± standard error (SE) for dermal thickness, dermal adipose tissue area, dermis area, total dermal area, and dermal adipose tissue ratio. "ns" indicates that the difference between groups was not statistically significant.

RFR: Radiofrequency radiation

thickness, dermis area, dermal adipose tissue area, total dermal area, and dermal adipose tissue ratio are illustrated in Figure 2.

DISCUSSION

In this study, histomorphometric measurements of skin tissue at 18 months of age showed no statistically significant differences between female rats prenatally exposed to RFR throughout all trimesters of the intrauterine period and unexposed shams. Parameters—dermal thickness, dermis area, dermal adipose tissue area, total dermal area, and dermal adipose tissue ratio (p=0.162, 0.717, 0.198, 0.175, and 0.704, respectively). These findings indicate that prenatal RFR exposure produces changes in skin morphology similar to those associated with aging in female rats.

Skin cells, including keratinocytes and fibroblasts, are constantly exposed to various environmental stressors. To counteract these effects, they have evolved numerous protective mechanisms that enable adaptive responses to stimuli such as UV-R, temperature fluctuations, and mechanical stress. Key protective systems include HSPs; antioxidant enzymes, such as superoxide dismutase, catalase, and glutathione peroxidase; and DNA repair

mechanisms[21]. Mild cellular stress caused by low-energy, non-ionizing stimuli such as RFR may not exceed the threshold required to elicit these adaptive responses, or may be rapidly compensated for.

This claim is further substantiated by the findings of Joushomme et al.[22], who conducted a study of the effects of 5G RF-EMF exposure at 3.5 GHz in human fibroblast and keratinocyte cell lines. Their research assessed the activation of stress response pathways, including *HSF1*, RAS, ERK, and *PML*, under both continuous and intermittent exposure conditions. Consistent with our findings, the observed effects were limited and inconsistent: specifically, a reduction in *HSF1* activity and a slight decrease in *PML* SUMOylation were observed in fibroblasts exposed to low SAR levels, but were absent in keratinocytes and not reproducible across different exposure regimens. They concluded that was no convincing evidence that 5G RF-EMF, either alone or in combination with a chemical stressor, induced a significant and consistent cellular stress response[22]. While our study focused on *in vivo* long-term morphological effects, this referenced work explored in vitro short-term exposure and its impact on cellular stress signaling pathways. Together, these complementary approaches suggest that the biological effects of RFR on the skin may be limited at both the molecular and morphological levels.

Similarly, exposure to 1800 MHz RF-EMF at low doses has been shown to cause single- and double-strand breaks in DNA, but it also that, over time, reduce DNA damage below control levels, thereby eliciting a hormesis-like effect[23]. This hormetic response facilitates tissue integrity by activating adaptive mechanisms against low-level environmental stresses. Consequently, chronic and systemic RFR exposure during the prenatal period may activate such adaptation and repair pathways in developing tissues, preventing deterioration of skin morphology later in life.

The skin, particularly the stratum corneum, functions as a robust barrier that limits the penetration of RF-EMF energy into deeper tissues due to its low water content and high keratin density. The absorption of electromagnetic waves depends on the dielectric properties of the tissue; thus, the stratum corneum predominantly reflects or attenuates RFR at the skin surface. Consistent with this, Habauzit et al.[24] (2020) investigated the biological effects of chronic exposure to 94-GHz millimeter waves on skin gene expression in young and adult hairless rats. Their experimental design involved exposure of 3 hours per day, 3 days per week, over 5 months at 10 mW/cm^2—twice the International Commission on Non-Ionizing Radiation Protection occupational exposure limit—without inducing thermal elevation. Using microarray analysis, they found no statistically significant changes in skin gene expression in either age group[24]. The absence of detectable molecular-level alterations supports our morphological findings and suggests that skin tissue may represent a highly resistant target to RF-EMF exposure.

Moreover, the epidermis is a continuously renewing tissue; basal layer cells migrate to the surface and are shed approximately every 28 days. This rapid turnover may prevent the accumulation of sublethal damage induced by RFR. Even under long-term exposure, tissue integrity can be maintained through the elimination of damaged cells[25]. Supporting this, Xu et al.[26] exposed six cell lines to short-term (1 h) and long-term (24 h) intermittent 1800 MHz GSM RF-EMF exposures at a SAR of 3.0 W/kg, assessing DNA damage via γH2AX foci formation. Results showed cell-type-dependent induction of γH2AX foci—a marker of DNA double-strand breaks—with a significant increase in human skin fibroblasts (HSF) and Chinese hamster lung cells. However, the increase in γH2AX in HSF cells did not correspond with sustained functional impairments, as measured by comet assay parameters, Terminal deoxynucleotidyl transferase dUTP Nick End Labeling assay, cell cycle progression, proliferation, or viability. Thus, there was limited evidence that DNA damage translated into cellular dysfunction[26]. Our finding of absent histomorphometric alterations in chronic-phase skin following prenatal RFR exposure supports the hypothesis that potential DNA-level damage does not necessarily lead to functional impairments. Furthermore, it suggests that the epidermis's regenerative capacity during the chronic phase effectively prevents the accumulation of cellular damage over time.

Considering evidence from our study and related *in vitro* and molecular research, it appears that prenatal exposure to RFR under the tested conditions does not significantly impair skin morphology or function over the long term. This resilience likely stems from the skin's robust barrier properties, its dynamic cellular renewal, and effective adaptive and repair mechanisms that mitigate potential damage. Nonetheless, given the variability in exposure parameters and biological responses, further investigations exploring different frequencies, intensities, and combined environmental factors—especially during critical developmental windows—are essential to comprehensively understand the potential risks and safety profile of RFR exposure.

Study Limitations

This study has several limitations that should be acknowledged. First, the investigation was limited to a specific RFR frequency and exposure regimen, which may not represent the full spectrum of environmental exposures. Secondly, only female rats were included, and potential sex-specific differences were not assessed. Thirdly, the focus was restricted to skin morphology without direct in vivo evaluation of functional or molecular endpoints, which could have provided deeper insight into subtle biological effects. Lastly, the sample size may limit the detection of minor effects. Future studies addressing these limitations, including multi-frequency exposures, both sexes, and integrated morphological, molecular, and functional analyses, would strengthen the understanding of RFR's impact on developing tissues.

CONCLUSION

In conclusion, our long-term in vivo study indicated that prenatal 3.5 GHz RFR exposure did not result in significant histomorphometric alterations in the skin of aged (18-month-old) female rats. This lack of morphological change is likely attributable to the skin's inherent protective barriers, efficient cellular repair mechanisms, and continuous regenerative capacity. In conjunction with existing *in vitro* and molecular data, these findings suggest that, under the exposure conditions examined, RFR poses minimal risk to skin structural integrity and function. We believe that further studies in this area will make valuable contributions to the field.

Ethics

Ethics Committee Approval: All procedures were designed and performed in accordance with the guidelines of the Animal Experiments Local Ethics Committee of Istanbul Bagcilar Training and Research Hospital with decision no: 2024-53, and date: 26.11.2024).

Informed Consent: Not appreciable.

Footnotes

Author Contributions

Surgical and Medical Practices: E.G.D., T.M., R.N.K., Concept: E.G.D., U.U., S.D., Design: E.G.D., U.U., S.D., Data Collection and/or Processing: E.G.D., T.M., R.N.K., U.U., Analysis or Interpretation: E.G.D., T.M., S.D., Literature Search: E.G.D., T.M., S.D., Writing: E.G.D., T.M., R.N.K., U.U., S.D.

Conflict of Interest: The authors have no conflict of interest to declare.

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