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Chronic Continuous Whole-Body 2.45 GHz Wi-Fi Exposure (Estimated SAR ~1.4 W/kg) Reduces Serum Thyroid Hormones and Alters Thyroid Histology in Adult Male Wistar Rats

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Abstract

Background & Objectives: This study aimed to evaluate serum thyroid hormone levels and thyroid histopathology following chronic whole-body exposure to 2.45 GHz Wi-Fi radiation in adult male Wistar rats using an estimated whole-body specific absorption rate (SAR).

Materials & Methods: Twenty adult male Wistar rats (150 to 200 g) were randomly allocated to either the control group (n = 10) or the Wi-Fi exposure group (n = 10). The exposure group underwent continuous whole-body irradiation with a 2.45 GHz Wi-Fi signal for 24 h/day over a 30 day period. The estimated mean whole-body SAR in the exposed group was 1.4 W/kg (range: 1.2 to 1.6 W/kg). Serum concentrations of triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) were measured using radioimmunoassay. Thyroid tissues were subsequently processed for histopathological examination using hematoxylin and eosin staining. Statistical analyses were conducted using appropriate parametric or nonparametric tests, with statistical significance defined as $p < 0.05$.

Results: Compared with the control group, rats exposed to Wi-Fi radiation exhibited significantly lower serum concentrations of T3 (1.73 ± 0.16 vs. 2.14 ± 0.17 ng/mL; $p < 0.001$), T4 (6.88 ± 0.19 vs. 7.38 ± 0.30 µg/dL; $p = 0.003$), and TSH (0.03 ± 0.01 vs. 0.05 ± 0.02 mIU/L; $p = 0.014$). Histopathological evaluation demonstrated reduced follicular diameter, manifested by the appearance of microfollicles, diminished colloid content, and disorganization of follicular epithelial cells in exposed animals relative to controls.

Conclusion: Continuous exposure to 2.45 GHz Wi-Fi radiation at an estimated whole-body SAR of approximately 1.4 W/kg (range: 1.2 to 1.6 W/kg) for 30 days was associated with reduced serum T3, T4, and TSH concentrations, together with structural alterations in thyroid tissue in adult male rats.

Keywords: Electromagnetic fields, Wi-Fi, Thyroid hormones, Hypothalamic–pituitary–thyroid axis

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Introduction

In an era marked by the rapid expansion of communication technologies, exposure to electromagnetic fields (EMFs) emitted by Wi-Fi systems operating at 2.45 GHz has become an integral part of everyday life (1,2). Although these technologies provide substantial practical benefits, growing concern has emerged regarding their potential biological effects, particularly because several reports suggest that exposure levels may exceed recommended safety thresholds in certain environments (3,4).

A number of experimental investigations have suggested that radiofrequency (RF) radiation may function as a biological stressor capable of influencing cellular redox balance and inflammatory processes (5,6). These observations are primarily derived from evidence demonstrating alterations in oxidative homeostasis following RF exposure in specific biological systems. Nevertheless, the precise biological mechanisms underlying these effects remain insufficiently understood and were not directly investigated in the present study.

The thyroid gland may be especially vulnerable to environmental stressors because of its rich vascular supply and the substantial metabolic demands required for hormone synthesis. Previous studies have indicated that disturbances in cellular homeostasis may interfere with thyroid hormone production and disrupt normal follicular architecture (7,8). However, the mechanisms responsible for these alterations remain largely speculative and were not experimentally examined in the current investigation. Although several studies have independently evaluated the effects of EMFs on thyroid hormone levels or thyroid tissue morphology (9,10), comprehensive investigations integrating both hormonal assessment and detailed histopathological evaluation remain limited. Furthermore, studies simultaneously examining thyroid structure and function in the context of Wi-Fi exposure are scarce.

Accordingly, the present study was designed to evaluate the effects of chronic exposure to 2.45 GHz Wi-Fi radiation on thyroid hormone levels, including thyroid-stimulating hormone (TSH), triiodothyronine (T3) and thyroxine (T4), as well as on thyroid histopathological structure in adult male rats.

Materials and Methods

Study Design and Animals

Twenty adult male Wistar rats weighing 150 to 200 g were obtained from the animal facility of Zabol University of Medical Sciences. The animals were housed in standard nonmetallic polypropylene cages with internal dimensions of [40 × 25 × 20 cm] and a uniform wall thickness of [2 to 3 mm], with two to three rats accommodated per cage. Housing conditions were maintained under controlled environmental parameters, including a temperature of 23 ± 2 °C, relative humidity of 55 ± 5%, and a 12 h light/12 h dark cycle.

Prior to the initiation of the experiment, the rats were allowed to acclimatize for 10 days and were provided ad libitum access to standard laboratory chow and water throughout the study period. All animals remained freely mobile within their cages during the experiment. All experimental procedures were conducted in accordance with institutional ethical guidelines and were approved by the Institutional Animal Care and Use Committee (IACUC) of Zabol University of Medical Sciences.

SAR Calculation

SAR was estimated using the classical equation: $SAR = (\sigma \times E^2) / \rho$

Where:

σ = tissue conductivity (S/m) for rat body at 2.45 GHz (0.9–1.1 S/m)

E = estimated electric field strength inside the cage

ρ = tissue density (1.03 g/cm^3)

The electric field strength was approximated

on the basis of router output power (17 to 20 dBm; 50 to 100 mW), antenna gain (2 to 3 dBi), and free-space propagation at a distance of 30 to 50 cm from the cage. Published rodent studies employing comparable exposure geometries have reported SAR values ranging from 1.0 to 1.6 W/kg under similar experimental conditions (11).

Assumptions in SAR Estimation

- Rat posture, movement, and orientation vary, affecting absorption.
- Field distribution inside the exposure cage is non-uniform and may differ from free-space estimates.

Router transmission was assumed to be continuous, although real Wi-Fi signals fluctuate with data traffic.

Tissue dielectric properties were assumed constant and derived from standard reference tables.

Estimated SAR Range

Based on these parameters and comparisons with previously validated exposure models, the approximate whole-body SAR for rats in the present study was estimated to be 1.4 W/kg, with a range of 1.2 to 1.6 W/kg.

Experimental Groups and Randomization

The rats were randomly assigned to two groups (n = 10 per group) using the block randomization method. Group A served as the control group and underwent sham exposure, whereas Group B was subjected to continuous whole-body irradiation with 2.45 GHz Wi-Fi radiation. Investigators responsible for sample collection and histopathological assessment were blinded to group allocation. To prevent incidental exposure of control animals to the 2.45 GHz signal, control and Wi-Fi exposed rats were housed in separate yet otherwise identical rooms.

Wi-Fi Exposure Protocol

A commercially available TP-LINK modem operating at 2.45 GHz and equipped with a 5 dBi external omnidirectional antenna was used to generate the Wi-Fi signal. The modem was positioned 50 cm directly above the center of the animal cage, with the antenna maintained in a vertical orientation relative to the cage. The modem output power ranged from 17 to 20 dBm, corresponding to approximately 50 to 100 mW. The emitted signal consisted of a standard modulated Wi-Fi signal compliant with IEEE 802.11b/g/n protocols rather than a continuous wave signal.

During exposure, the animals were housed in standard polycarbonate cages. On the basis of the specified output power and exposure distance, the average whole-body SAR for rats weighing 150 to 200 g was estimated to be approximately 1.4 W/kg. Although direct field probe mapping was not performed, the exposure configuration, including distance, power, and antenna characteristics, was designed and calibrated to reproduce the exposure conditions and SAR values reported in previous foundational studies investigating the biological effects of Wi-Fi radiation (9).

Rats assigned to the exposure group were continuously exposed to 2.45 GHz Wi-Fi radiation for 24 h/day over 30 consecutive days. Control animals were handled identically; however, the signal generator remained switched off throughout the study period. Food and water availability were identical for both groups.

Environmental Monitoring

Ambient temperature within the exposure room was continuously monitored and maintained at 23 ± 2 °C. The 50 cm distance between the modem and the animal cage was specifically selected to minimize any potential thermal effects. Under these experimental conditions, any increase in temperature attributable solely to Wi-Fi exposure was confirmed to be negligible (≤ 0.5 °C).

Biochemical Analysis

At the conclusion of the exposure period, the animals were anesthetized with ketamine (87 mg/kg) and xylazine (13 mg/kg), after which blood samples were collected via cardiac

puncture. Serum samples were separated by centrifugation at 3000 rpm for 10 min and stored at −20 °C until analysis. Serum concentrations of T3, T4, and TSH were measured using commercially available radioimmunoassay kits (Pars Azmoon, Iran) according to the manufacturer's instructions. The intra-assay and inter-assay coefficients of variation were <5% and <8%, respectively.

Note: No external citation is required for standard anesthesia or manufacturer-directed assay procedures.

Histological Assessment

Thyroid tissues were fixed in formalin, sectioned, and stained with hematoxylin and eosin (H&E) for histopathological evaluation.

Statistical Analysis

All statistical analyses were performed using SPSS software version 26. The Shapiro-Wilk test was used to assess the normality of continuous variables. For normally distributed data, independent sample t-tests were applied, whereas the Mann-Whitney U test was used for nonnormally distributed variables. Statistical significance was defined as $p < 0.05$.

Results

All animals completed the study protocol successfully. Body weight measurements obtained at baseline and on day 30 did not differ significantly between the two groups (baseline: control, 163 ± 2.3 g vs. exposure, 165 ± 3.1 g; day 30: control, 163 ± 3.2 g vs. exposure, 162 ± 2.1 g). Effect size analysis demonstrated a moderate difference at baseline (Cohen's d = 0.73) and a small difference on day 30 (Cohen's d = 0.37); however, neither difference reached statistical or biological significance.

Serum thyroid hormones

Serum concentrations of T3, T4, and TSH were significantly lower in the Wi-Fi exposure group than in the control group (Table 1). Specifically, T3 concentrations decreased from 2.14 ± 0.17 ng/mL in control animals to 1.73 ± 0.16 ng/mL in exposed rats ($p < 0.001$). Similarly, T4 concentrations declined from 7.38 ± 0.30 µg/dL to 6.88 ± 0.19 µg/dL ($p = 0.003$), whereas TSH concentrations decreased from 0.05 ± 0.02 mIU/L to 0.03 ± 0.01 mIU/L ($p = 0.014$).

To further evaluate the strength of the association between Wi-Fi exposure and thyroid hormone concentrations, point-biserial correlation analysis was performed. The findings demonstrated statistically significant inverse correlations between exposure to Wi-Fi radiation and reductions in all measured thyroid hormones. Specifically, a strong negative correlation was observed for T3 ($p < 0.001$, $r = -0.804$), followed by T4 ($p = 0.003$, $r = -0.728$) and TSH ($p = 0.014$, $r = -0.638$).

Histopathology

Light microscopic examination of H&E stained thyroid sections obtained from control animals revealed normal glandular architecture (Figure 1). Thyroid follicles were uniform in size, filled with abundant colloid material, and lined by flattened to low cuboidal epithelial cells arranged in a regular and orderly pattern, with no evidence of structural abnormalities.

In contrast, thyroid sections derived from rats exposed to 2.45 GHz Wi-Fi radiation exhibited distinct morphological alterations (Figure 2). These changes included the presence of a microfollicular pattern characterized by reduced follicular diameter, depletion of colloid content, and disorganization of the follicular epithelial lining.

Table 1. Serum thyroid hormones in control and Wi-Fi exposed rats (mean ± SD).

Groups	No	T4 (µg/dL)	T3 (ng/mL)	TSH (mIU/L)
Control	10	7.38±0.30	2.14±0.17	0.05±0.02
Wi-Fi	10	6.88±0.19	1.73±0.16	0.03±0.01
p-value		0.003	<0.001	0.014

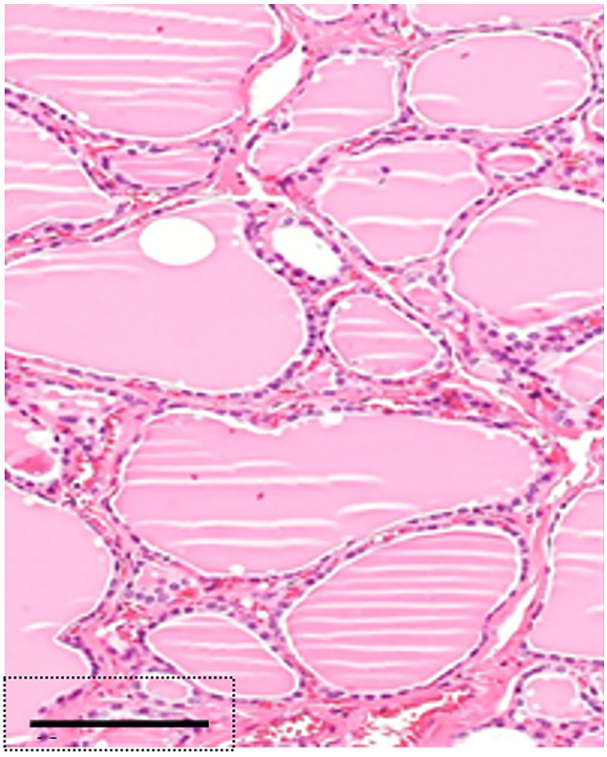


Figure 1. Representative microscopic image of thyroid tissue from the control group showing normal follicular architecture. Follicles contain abundant colloid and are lined by uniformly arranged flattened to low-cuboidal epithelial cells, with no evidence of structural abnormalities. (H&E, ×400).

Furthermore, the epithelial cells appeared irregular and demonstrated loss of their characteristic uniform arrangement. Quantitative morphometric analysis was not performed in the present study.

Discussion

The present study investigated the effects of chronic, continuous exposure to 2.45 GHz Wi-Fi radiation on thyroid function and histological structure in adult male rats. The findings demonstrated a concurrent reduction in serum T3, T4, and TSH concentrations together with distinct histopathological alterations, including microfollicle formation, colloid depletion, and disruption of the follicular epithelium. By integrating both hormonal and histopathological assessments, this study provides a more comprehensive evaluation of Wi-Fi induced thyroid alterations than previous investigations in which these parameters were examined independently (12,13). Particularly noteworthy was the observed hormonal profile. The reduction in circulating T3 and T4 concentrations, indicative of a hypothyroid-

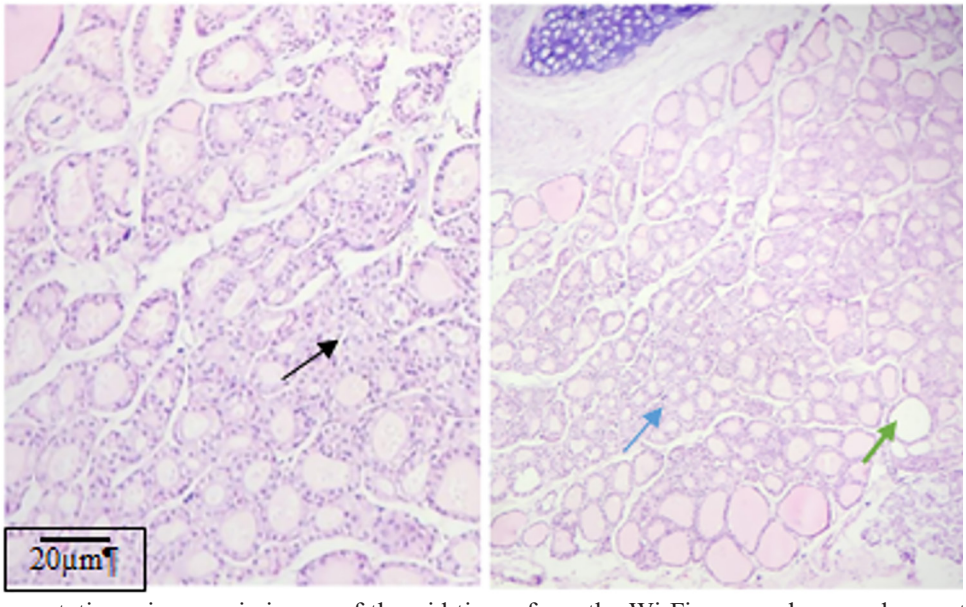


Figure 2. Representative microscopic image of thyroid tissue from the Wi-Fi–exposed group demonstrating marked histological alterations, including reduced follicle size with a microfollicular pattern (blue arrow), disorganization of the follicular epithelial lining (black arrow), and decreased colloid content with smaller follicular lumens (green arrow). (H&E, ×400)

like state, was accompanied by decreased TSH levels. This pattern may suggest the involvement of a central regulatory component affecting the hypothalamic-pituitary-thyroid (HPT) axis rather than isolated primary thyroid dysfunction, in which compensatory elevation of TSH would ordinarily be expected. Nevertheless, because hypothalamic and pituitary functions were not directly assessed, this interpretation remains speculative. Similar patterns characterized by reduced thyroid hormone concentrations accompanied by inappropriately low or normal TSH levels have previously been reported in conditions associated with impaired central regulation of the HPT axis; however, confirmation of such mechanisms requires targeted neuroendocrine investigation (9,10).

Although the present findings are consistent with several experimental studies reporting adverse effects of RF-EMF exposure on thyroid hormone levels and thyroid histology (9,13-15), the overall body of evidence remains inconsistent. A number of well-designed animal studies have reported no significant alterations in T3, T4, or TSH concentrations following RF-EMF exposure (10,16-18). For instance, Mortazavi et al. (2009) observed no significant changes in thyroid hormone levels in rats exposed to mobile phone radiation, and several other carefully controlled rodent studies have similarly yielded null findings (10,16). Furthermore, a systematic review conducted by Baliatsas et al. (2024) concluded that evidence linking RF-EMF exposure to thyroid dysfunction remains limited and inconsistent, with no clearly established dose-response relationship (19). Similarly, the World Health Organization systematic review published in 2022 emphasized the substantial heterogeneity among experimental protocols, exposure parameters, and outcome measures, thereby limiting the ability to draw definitive conclusions regarding thyroid related effects in either animals or humans (17).

The discrepancies observed across studies are likely attributable to variations in exposure duration, signal modulation, SAR values, animal strains, and the timing of hormonal assessment relative to exposure. Within this context, the present study contributes additional evidence by demonstrating thyroid related alterations under conditions of chronic, continuous exposure at a moderate SAR level. Nevertheless, caution should be exercised when extrapolating these findings to humans, whose exposure patterns are generally intermittent and considerably more variable.

Two nonmutually exclusive mechanisms may account for the observed findings: direct thyroidal injury occurring concurrently with impaired pituitary TSH secretion, or primary central suppression of the HPT axis leading secondarily to thyroid atrophy. Variability among previous reports, including studies demonstrating increased hormone concentrations or compensatory elevation of TSH levels (15), likely reflects differences in exposure parameters as well as the temporal stage of thyroid dysfunction, thereby underscoring the complexity of EMF-endocrine interactions.

The histopathological findings observed in the present study were consistent with the accompanying functional alterations. The predominance of microfollicles together with reduced colloid content is characteristic of impaired thyroid hormone synthesis and storage and corresponds directly to the hormonal deficiencies identified in exposed animals. Comparable structural alterations have been reported in previous investigations (13,14), suggesting that the thyroid gland may represent a relatively consistent target of EMF exposure across diverse experimental conditions.

Although the mechanistic pathways underlying these alterations were not directly investigated, several biologically plausible explanations may be proposed. One of the most

widely discussed mechanisms involves RF induced oxidative stress, which may increase the production of reactive oxygen species (ROS) and disrupt intracellular redox homeostasis (5,6). Elevated ROS levels may impair thyroid hormone synthesis by inhibiting key enzymes involved in hormone production, including thyroid peroxidase, thereby interfering with iodide oxidation and thyroglobulin iodination (7,14). In addition, oxidative stress may activate redox sensitive transcription factors such as nuclear factor kappa B (NF-κB), subsequently promoting the expression of proinflammatory cytokines, including interleukins and tumor necrosis factor α. Chronic low grade inflammation mediated through NF-κB activation may therefore contribute not only to functional suppression of thyroid hormone synthesis but also to structural disruption of thyroid follicles (7-9).

Moreover, inflammatory cytokines and oxidative stress may influence hypothalamic and pituitary regulation of TSH secretion, potentially contributing to the observed reduction in HPT axis activity. Sustained oxidative and inflammatory stress may also promote mitochondrial dysfunction, membrane lipid peroxidation, and DNA damage, ultimately triggering apoptotic pathways within thyroid tissue and contributing to follicular disorganization and colloid depletion. Nevertheless, these proposed mechanisms remain speculative within the context of the present investigation and are inferred from previous experimental and clinical evidence rather than from direct mechanistic measurements.

Finally, several additional considerations warrant attention. The present study was conducted exclusively in adult male rats and did not include evaluation of stress related parameters, such as circulating corticosterone concentrations or behavioral stress assessments. Sex specific differences in HPT axis regulation, together with the potential influence of chronic stress, may therefore have acted as confounding variables. Future investigations should include both sexes and incorporate physiological and behavioral markers of stress to better distinguish the direct biological effects of EMF exposure from endocrine alterations secondary to stress related responses.

Conclusion

Herein, the chronic Wi-Fi exposure was associated with both hormonal suppression and structural alterations within the thyroid gland. Continuous whole-body exposure of adult male Wistar rats to 2.45 GHz EMFs at an average SAR of 1.4 W/kg for 30 days resulted in significant reductions in serum T3, T4, and TSH concentrations, accompanied by disruption of normal thyroid architecture. The concurrent reduction in TSH together with decreased T3 and T4 concentrations may suggest involvement of central regulatory mechanisms within the HPT axis; however, this possibility cannot be confirmed in the absence of direct assessment of hypothalamic or pituitary function.

Although oxidative stress and inflammatory processes represent biologically plausible mechanisms underlying the observed effects, these pathways were not directly evaluated in the present study. Future investigations should therefore incorporate direct assessment of oxidative stress biomarkers, inflammatory cytokines, and NF-κB activation in order to clarify the underlying mechanisms, establish potential dose-response relationships, and more accurately determine the relevance of these findings to human exposure scenarios.

Limitations and Suggestions

Several limitations of the present study should be acknowledged. First, although the continuous 24 h/day exposure paradigm was effective for identifying potential biological effects, it does not accurately reflect the intermittent nature of typical human Wi-Fi

exposure. Second, despite being sufficient to detect statistically significant differences, the sample size remained relatively modest. Third, the histopathological evaluation was qualitative rather than quantitative in nature; consequently, future studies incorporating detailed morphometric analyses would provide a more robust assessment of structural alterations.

In addition, the reported SAR represented a whole-body average, whereas more comprehensive dosimetric characterization, including spatial mapping of absorbed energy distribution, would improve both reproducibility and translational relevance. The absence of circulating corticosterone measurements and behavioral stress assessments also constitutes an important limitation. Finally, because the study was conducted exclusively in adult male Wistar rats, potential sex specific differences in HPT axis regulation and susceptibility to RF-EMF exposure were not evaluated. Future studies should therefore include both male and female animals to determine whether the observed effects are sex dependent.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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Code of Ethices

The research protocol was reviewed and approved by the Ethics Committee of Zabol University of Medical Sciences (Approval Code: IR.ZBMU.REC.1400.143).

Authors' Contribution

Conceptualization: J P, and Kh S; Data Curation: J P; Formal analysis: J P, L Sh-M; Funding acquisition: J P, and L Sh-M; Investigation: J P, and Kh S; Methodology: J P, and Kh S; Project administration: J P, and L Sh-M; Resources: J P, and Kh S; Supervision: J P; Validation: J P, and L Sh-M; Visualization: J P; Writing–original draft: J P; Writing–review & editing: J P, M M and M Gh.

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