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Biochemical and Hormonal Alterations in the Rat Thyroid Gland Following Chronic Ultraviolet-A Exposure: A Duration-Dependent Study

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ABSTRACT

Ultraviolet-A (UVA) radiation, the most prevalent component of solar UV reaching the earth's surface, is known to induce oxidative stress and cellular damage in various tissues. However, its specific effects on endocrine organs, particularly the thyroid gland, remain inadequately characterized. This study aimed to evaluate the biochemical and hormonal changes in the thyroid gland of rats following chronic exposure to UVA radiation for different durations. Twenty adult male albino rats (200-250 g) were divided into four groups (n=5 each): a control group (no exposure) and three experimental groups exposed to UVA radiation (8 hours/day) for 10, 15, and 25 days, respectively. Thyroid tissue levels of triiodothyronine (T3), thyroxine (T4), thyroid-stimulating hormone (TSH), and the antioxidant enzyme reduced glutathione (GSH) were measured. Chronic UVA exposure significantly altered thyroid hormone profiles. T3 levels initially increased at 10 days (3.65±0.92 vs. control 2.6±0.25) and 15 days (3.62±0.29), then declined towards near-normal levels by 25 days (2.9±0.53). T4 showed a non-significant fluctuation, while TSH exhibited a dramatic and sustained reduction from 0.8±0.14 (control) to near-undetectable levels (0.016±0 to 0.01±0) in all exposed groups. GSH levels varied, showing a non-significant decrease at 10 days (54.63±1.31), followed by an increase at 15 and 25 days (59.2±3.78 and 58.01±2.28, respectively). Chronic UVA radiation exposure induces duration-dependent disruption of thyroid hormonal homeostasis, characterized by marked TSH suppression and complex alterations in T3 and T4, accompanied by changes in antioxidant enzyme levels. These biochemical findings complement previously reported histological damage (Qabeeli et al., 2026) and suggest that UVA radiation may impair the hypothalamic-pituitary-thyroid axis.

Introduction

Ultraviolet-A (UVA) radiation (315–400 nm) constitutes approximately 95% of solar ultraviolet radiation reaching the Earth's surface and penetrates biological tissues more deeply than UVB or UVC [1]. Due to increasing environmental and occupational exposure from natural sunlight, tanning beds, industrial equipment, and medical devices, UVA radiation is recognized as a significant environmental stressor with potential systemic health effects beyond the skin [2, 7]. The biological effects of UVA are primarily mediated through the generation of reactive oxygen species (ROS), leading to oxidative stress, lipid peroxidation, DNA damage, and cellular dysfunction [7]. While the cutaneous effects of UV radiation are well documented, the impact on internal endocrine organs, particularly the thyroid gland, remains inadequately characterized.

The thyroid gland is a critical regulator of metabolism, growth, and physiological homeostasis. It is highly sensitive to environmental stressors and radiation due to its high metabolic activity and rich vascularity [3]. Thyroid function is maintained by the hypothalamic-pituitary-thyroid (HPT) axis, where thyroid-stimulating hormone (TSH) regulates the synthesis and secretion of triiodothyronine (T3) and thyroxine (T4). Disruption of this axis can have profound metabolic consequences.

Recent histological evidence has demonstrated that chronic UVA exposure induces progressive structural damage in rat thyroid tissue, including follicular disorganization, epithelial cell crowding, vascular congestion, colloid depletion, and stromal expansion, with severity increasing with exposure duration [6]. However, histopathological alterations alone do not fully elucidate the functional consequences of UVA exposure on thyroid hormone regulation.

Emerging evidence from UVB studies has shown that UV exposure can induce thyroid toxicity characterized by altered thyroid hormone profiles (elevated T3 and T4, decreased TSH), increased oxidative stress markers (decreased GSH, increased lipid peroxidation), and histopathological follicular damage [4, 5, 11]. UV filters used in sunscreens have also been shown to disrupt HPT axis function [8, 9].

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Given the increasing prevalence of UVA exposure and limited understanding of its effects on thyroid endocrine function, this study aimed to: (1) investigate biochemical and hormonal correlates of UVA-induced thyroid injury by measuring T3, T4, TSH, and GSH following chronic UVA exposure for 10, 15, and 25 days; (2) characterize duration-dependent patterns of thyroid hormone disruption; and (3) integrate these findings with existing histopathological evidence.

Methods

Experimental Animals

Twenty adult male albino rats (Wistar strain), weighing 200-250 g, were used in this study. Animals were housed under standard conditions (20±2°C, 12-hour light/dark cycle) with free access to food and water. Dorsal hair was shaved to ensure uniform UVA exposure. All procedures followed institutional animal ethics guidelines.

Experimental Design and Grouping

The rats were randomly divided into four equal groups of five animals each. The first group served as the control group and was not exposed to any UVA radiation. The remaining three groups were designated as experimental groups and were exposed to UVA radiation for 8 hours daily over different durations. Specifically, the second group (UVA-10) was exposed for 10 consecutive days, the third group (UVA-15) was exposed for 15 consecutive days, and the fourth group (UVA-25) was exposed for 25 consecutive days.

UVA Exposure System

UVA radiation was generated using a fluorescent UVA lamp (Sylvania, 40 W) with a peak wavelength of 368 nm. The lamp was positioned 30 cm above cages, delivering irradiance of 3.5 mW/cm², resulting in a cumulative daily radiation dose of 100.8 J/cm² over the 8-hour exposure period. All exposure parameters were kept constant throughout the study.

Sample Collection and Biochemical Assays

At the end of each exposure period (10, 15, or 25 days), animals were anesthetized by ether inhalation, and thyroid tissues were carefully excised. Tissue homogenates were prepared in ice-cold phosphate-buffered saline. Levels of triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) were measured using enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's protocols. Reduced glutathione (GSH) levels were determined using a colorimetric method based on the reaction with 5,5'-dithiobis (2-nitrobenzoic acid) (DTNB). All assays were performed in duplicate.

Statistical Analysis

Data were analyzed using SPSS version 28.0 (IBM Corp., Armonk, NY, USA) and are presented as mean ± standard deviation (S.D.). One-way analysis of variance (ANOVA) followed by Tukey's post-hoc test was used to compare differences among the four groups. A p-value of less than 0.05 was considered statistically significant.

Results

As shown in Table 1 and Figure 1, UVA exposure induced a biphasic T3 response. Compared to control (2.6±0.25), T3 increased markedly at 10 days (3.65±0.92, +40.4%) and 15 days (3.62±0.29, +39.2%), then declined towards control levels by 25 days (2.9±0.53, +11.5%), as shown in Table 1.

Table 1: T3 Hormone Levels in Thyroid Tissue

Parameter	Control	Exposed 10 days	Exposed 15 days	Exposed 25 days
Mean ± S.D	2.6 ± 0.25	3.65 ± 0.92	3.62 ± 0.29	2.9 ± 0.53
Min - Max	2.33 - 2.93	2.19 - 4.63	3.41 - 4.12	2.21 - 3.38

T4 levels (Table 2, Figure 2) showed non-significant fluctuations: control (2.4±0.32), 10 days (2.9±0.45, +20.8%), 15 days (2.1±0.25, -12.5%), and 25 days (2.5±0.53, +4.2%) as demonstrated in Table 2.

Table 2: T4 Hormone Levels in Thyroid Tissue

Parameter	Control	Exposed 10 days	Exposed for 15 days	Exposed 25 days
Mean ± S.D	2.4 ± 0.32	2.9 ± 0.45	2.1 ± 0.25	2.5 ± 0.53
Min - Max	1.91 - 2.71	2.44 - 3.56	1.69 - 2.31	1.99 - 3.36

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The most dramatic finding was profound TSH suppression (Table 3, Figure 3). Control TSH was 0.8±0.14. Following UVA exposure, TSH dropped to near-undetectable levels: 0.016±0 (10 days), 0.01±0.013 (15 days), and 0.01±0 (25 days), representing a 98-99% reduction as mentioned in Table 3.

Table 3: TSH Levels in Thyroid Tissue

Parameter	Control	Exposed 10 days	Exposed for 15 days	Exposed 25 days
Mean ± S.D	0.8 ± 0.14	0.016 ± 0	0.01 ± 0.013	0.01 ± 0
Min - Max	0.01 - 0.34	0.01 - 0.01	0.01 - 0.04	0.01 - 0.01

GSH levels (Table 4, Figure 4) showed non-significant changes: control (55.56±0.71), 10 days (54.63±1.31, -1.7%, p=0.101), 15 days (59.2±3.78, +6.5%, p=0.489), and 25 days (58.01±2.28, +4.4%, p=0.205) as shown in Table 4.

Table 4: GSH Levels in Thyroid Tissue

Parameter	Control	Exposed 10 days	Exposed for 15 days	Exposed 25 days
Mean ± S.D	55.56 ± 0.71	54.63 ± 1.31	59.2 ± 3.78	58.01 ± 2.28
P-value (vs. Control)	-	0.101	0.489	0.205

Discussion

This study provides the first comprehensive biochemical characterization of thyroid responses to chronic UVA exposure, complementing recent histopathological evidence [6]. The results reveal that chronic UVA exposure induces significant, duration-dependent disruption of thyroid hormone homeostasis. The most striking finding is the dramatic and sustained TSH suppression (98-99%) from the earliest exposure time point (10 days) through 25 days, with no sign of recovery. This suggests severe disruption of the HPT axis.

TSH, secreted by pituitary thyrotroph cells, is the primary regulator of thyroid function [3]. The near-complete suppression observed could result from: (1) direct oxidative damage to pituitary thyrotroph cells by UVA-induced systemic ROS [7]; (2) impaired hypothalamic TRH secretion; or (3) disruption of feedback mechanisms. The persistence of suppression despite normalized T3 levels at 25 days suggests a primary defect at the pituitary or hypothalamic level rather than simple feedback inhibition. This pattern is consistent with UVB studies reporting decreased TSH following UV exposure [4, 5]. UV-filter compounds have also been shown to disrupt HPT axis function [8, 9].

UVA exposure induced a biphasic T3 pattern: initial increase at 10 and 15 days, followed by decline towards normal at 25 days. The early T3 elevation may represent a compensatory hypermetabolic response to UVA-induced oxidative stress, potentially enhancing cellular repair mechanisms or increasing antioxidant enzyme production. Alternatively, it could result from increased peripheral conversion of T4 to T3 via upregulated deiodinase activity. The decline towards normal levels by 25 days may indicate either exhaustion of this compensatory mechanism or the onset of more profound thyroid dysfunction. This pattern differs from UVB studies where T3 remained elevated [4], possibly reflecting different tissue penetration depths or biological effects of UVA versus UVB. The lack of significant T4 changes, despite marked T3 and TSH alterations, is intriguing. This pattern—normal T4 with elevated then normal T3 and suppressed TSH could indicate impaired 5'-deiodinase activity or altered thyroid hormone binding. Similar discordances are observed in non-thyroidal illness syndrome (euthyroid sick syndrome), where systemic stress alters thyroid hormone metabolism without changing TSH or T4.

GSH showed a non-significant trend: slight decrease at 10 days (possibly indicating initial oxidative depletion), followed by elevations at 15 and 25 days (suggesting adaptive upregulation). The lack of statistical significance may reflect insufficient sample size or that other antioxidant systems (SOD, catalase) are more prominently involved. Rai and Mahobiya (2022) reported decreased GSH and increased lipid peroxidation following UVB exposure [4, 5]. Our finding of elevated GSH at later time points differs from those reports, possibly representing a species-specific or UVA-specific adaptive response. UVB has higher energy per photon than UVA and may cause more direct cellular damage, while UVA effects are more mediated through indirect oxidative stress mechanisms [7].

The biochemical alterations observed correlate well with histological changes reported by Qabeeli et al. (2026) [6]. The early T3 elevation at 10 days coincides with initial histopathological changes (follicular size reduction, epithelial cell crowding). The profound TSH suppression at all time points parallels progressive tissue damage. The most severe histological damage at 25 days (edema, stromal expansion, vascular congestion, extensive follicular disruption) was associated with near-normal T3/T4 but persistently suppressed TSH, suggesting that structural integrity may be maintained at the expense of functional reserve.

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Our findings align with and extend previous research on ultraviolet radiation and thyroid function. Rai and Mahobiya (2022) reported that UVB exposure in female Wistar rats induced a hyperthyroid state characterized by increased T3 and T4 levels, decreased TSH, reduced glutathione (GSH), and elevated lipid peroxidation (LPO) [4]. Similarly, the histological study by Qabeeli et al. (2026) demonstrated that chronic UVA exposure causes progressive structural damage in rat thyroid tissue, including follicular disorganization, vascular congestion, epithelial cell crowding, and colloid depletion [6]. These histological alterations correlate well with the biochemical changes observed in the present study, particularly the profound TSH suppression and biphasic T3 response.

Further supporting evidence comes from studies on UV-filter compounds, which are commonly used in sunscreens and share some biological effects with UV radiation itself. Klammer et al. (2007) demonstrated that exposure to the UV-filter octyl-methoxycinnamate (OMC) disrupts hypothalamic-pituitary-thyroid (HPT) axis function in rats, leading to alterations in T3, T4, and TSH levels [8]. More recently, Greco et al. (2025) showed that organic UV-filters can interfere with thyroid hormone regulation through interactions with transthyretin (TTR) and disruption of thyroid receptor signaling pathways [9].

The profound TSH suppression observed in our study a 98-99% reduction from control levels suggests that chronic UVA exposure may be particularly disruptive to the HPT axis compared to UVB or UV-filter exposure alone. This heightened effect may be attributed to UVA's deeper tissue penetration capacity and its ability to generate systemic oxidative stress through the production of reactive oxygen species (ROS), as previously described by Osipov et al. (2022) [7]. Unlike UVB, which is largely absorbed by the superficial skin layers, UVA reaches deeper vascular and connective tissues, potentially allowing for more widespread systemic effects, including direct or indirect damage to the pituitary gland and hypothalamus. Taken together, these comparisons suggest that UVA radiation poses a significant and under-recognized risk to thyroid endocrine function, warranting further investigation and public health attention.

These findings have significant implications for human health. Outdoor workers, individuals using tanning beds, and populations in high-UV environments may be at risk for UVA-induced thyroid dysfunction. The persistence of TSH suppression even after 25 days of exposure raises concerns about potential long-term or permanent HPT axis damage.

Conclusion

Chronic UVA exposure induces profound TSH suppression (98-99%) and biphasic T3 alterations, demonstrating significant disruption of the hypothalamic-pituitary-thyroid axis at multiple levels. These findings highlight the need to minimize unnecessary chronic UVA exposure, particularly in occupational settings, and warrant future investigation into the molecular mechanisms of UVA-induced HPT axis disruption.

Conflict of interest. Nil

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