UNIVERSAL JOURNAL OF MEDICAL AND NATURAL SCIENCES
ISSN 2992-8826
https://sr-journals.org/index.php/UJMANS



Current research on the effects of hypothyroidism and hypokinesia on hair follicles: a review of experimental findings

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Abstract: Hair follicles are highly dynamic mini-organs whose growth and regeneration are regulated by endocrine, metabolic, and environmental factors. Among these factors, thyroid hormones play a crucial role in maintaining normal follicular activity, while physical activity influences tissue metabolism and microcirculation. Experimental hypothyroidism and hypokinesia have been associated with structural and functional alterations in skin appendages, including hair follicles. This review summarizes current experimental findings regarding the individual and combined effects of hypothyroidism and hypokinesia on hair follicle morphology and function. The available evidence suggests that both conditions disrupt the normal hair growth cycle, reduce follicular activity, and induce degenerative histological changes. Understanding these mechanisms may contribute to the development of preventive and therapeutic strategies for hair disorders associated with endocrine and sedentary conditions.

Keywords: Hypothyroidism; Hypokinesia; Hair Follicles; Morphology; Histology; Experimental Animals; Skin Biology; Follicular Degeneration.

Introduction. Hair follicles are complex epithelial structures that undergo continuous cycles of growth (anagen), regression (catagen), and rest (telogen). The regulation of these cycles depends on a coordinated interaction between genetic, hormonal, nutritional, and environmental factors. Thyroid hormones, particularly triiodothyronine (T3) and thyroxine (T4), are recognized as essential regulators of epidermal proliferation, keratin synthesis, and follicular metabolism.

Hypothyroidism is characterized by a deficiency of thyroid hormones, resulting in reduced metabolic activity and impaired cellular regeneration. Clinical and experimental studies have consistently demonstrated that hypothyroidism contributes to hair thinning, delayed follicular growth, and increased hair shedding. In addition, modern lifestyles are increasingly associated with hypokinesia, a condition characterized by reduced physical activity. Hypokinesia adversely affects blood circulation, tissue oxygenation, and cellular metabolism, potentially influencing the structural integrity of skin and hair follicles.

Although numerous studies have independently investigated hypothyroidism and hypokinesia, limited information exists regarding their combined effects on hair follicle morphology. Therefore, a comprehensive evaluation of current experimental evidence is necessary to better understand the pathophysiological mechanisms underlying these alterations.

Methods

A narrative review approach was employed to analyze published experimental studies focusing on thyroid dysfunction, reduced motor activity, and hair follicle morphology. Relevant articles were selected based on their investigation of histological, morphometric, and ultrastructural changes in skin and hair follicles under conditions of experimentally induced hypothyroidism, hypokinesia, or both.

Studies involving laboratory animals, including rats and mice, were prioritized due to their relevance in modeling endocrine and metabolic disorders. Data concerning follicular density,

follicular diameter, epidermal thickness, cellular proliferation, and vascular changes were extracted and synthesized to identify common morphological patterns.

Results

Analysis of the reviewed literature revealed consistent evidence that hypothyroidism significantly affects hair follicle structure and function. Experimental models demonstrated a reduction in follicular size, decreased proliferative activity of matrix cells, and prolonged telogen phases. Histological examination frequently revealed follicular atrophy, thinning of the hair shaft, and diminished keratinization.

Similarly, hypokinesia was associated with reduced microcirculatory efficiency and impaired trophic support of skin tissues. Several studies reported decreased dermal vascularization, reduced oxygen supply, and alterations in connective tissue architecture. These changes were accompanied by lower follicular activity and delayed hair regeneration.

Studies evaluating the combined effects of hypothyroidism and hypokinesia indicated a synergistic influence on follicular degeneration. Animals exposed to both conditions exhibited more pronounced morphological abnormalities, including reduced follicular density, increased dystrophic changes, disruption of follicular epithelial organization, and signs of apoptotic cell death. Morphometric analyses demonstrated statistically significant reductions in follicular dimensions compared with control groups.

Discussion. The observed alterations can be explained by the fundamental role of thyroid hormones in regulating cellular metabolism and tissue homeostasis. Thyroid hormone deficiency reduces mitochondrial activity, protein synthesis, and cellular proliferation within follicular structures. Consequently, hair follicles lose their capacity to maintain normal growth cycles and structural integrity. Hypokinesia further exacerbates these effects by impairing peripheral circulation and reducing nutrient delivery to actively proliferating tissues. The resulting hypoxic and metabolic stress conditions promote degenerative changes within follicular cells and surrounding dermal components. The combined presence of endocrine insufficiency and reduced physical activity creates a pathological environment that accelerates follicular involution and disrupts normal hair growth dynamics. Current evidence suggests that the interaction between hormonal imbalance and physical inactivity may involve oxidative stress, inflammatory mediators, and altered expression of growth-regulating genes. However, the precise molecular pathways remain insufficiently investigated. Future studies employing immunohistochemical and molecular biological techniques may provide deeper insight into these mechanisms.

Conclusion. Current experimental research demonstrates that both hypothyroidism and hypokinesia exert significant adverse effects on hair follicle morphology. These conditions independently induce structural and functional impairments, while their combined action produces more severe degenerative changes. The findings highlight the importance of thyroid hormone homeostasis and physical activity in maintaining normal follicular physiology. Further investigations are required to elucidate the molecular mechanisms underlying these alterations and to develop effective therapeutic interventions for hair follicle disorders associated with endocrine and sedentary conditions.

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