Endocrine Regulation of Hair Growth and Loss: Current Evidence and Clinical Implications

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Abstract—Hair growth and loss are intricately regulated by endocrine factors that influence follicular cycling, differentiation, and regeneration. Hormones such as androgens, estrogens, thyroid hormones, prolactin, insulin, and cortisol play pivotal roles in modulating hair follicle activity, with imbalances contributing to various forms of alopecia, including androgenetic, thyroidrelated, and estrogen-deficiency-associated types. This narrative review synthesizes current evidence on the endocrine regulation of hair growth, highlighting the molecular mechanisms through which hormones exert their effects and their clinical implications in diagnosing and managing endocrine-mediated hair disorders. It emphasizes the importance of gender-specific management, hormonal assessment in alopecia evaluation, patient counseling, and multidisciplinary collaboration between dermatology and endocrinology. Despite notable advances, substantial research gaps remain, particularly in standardized evaluation methods, long-term clinical trials, and female-focused investigations. Future directions point toward omicsbased profiling, personalized endocrine modulation, regenerative therapies, and combination treatment strategies to develop more effective and durable solutions. A deeper understanding of hormonal influences on hair biology may ultimately enable tailored, mechanism-driven therapies for diverse endocrine-related alopecias.

Index Terms—Endocrine regulation, Hair loss, Alopecia, Hormonal imbalance, Androgens, Estrogen, Thyroid hormones, Personalized medicine

I. INTRODUCTION

Human hair, composed mainly of keratin produced by epidermal keratinocytes, covers most of the body except the palms, soles, and certain genital regions. The hair growth cycle comprises four stages: anagen (growth), catagen (regression), telogen (resting), and exogen (shedding) (1,2). The duration of the anagen phase determines hair length and varies by body site scalp hair may remain in anagen for several years, whereas eyebrows and body hair have shorter cycles (2).

Multiple factors influence the hair cycle, including genetics, hormones, nutrition, stress, illness, and aging (2). Among these, endocrine factors play a central role in regulating follicular activity and cycling. Hormones such as androgens, estrogens, thyroid hormones, insulin-like growth factors, prolactin, melatonin, and cortisol interact with specific follicular receptors to modulate hair growth, differentiation, and pigmentation (3). Dysregulation of these hormones contributes to diverse hair disorders including androgenetic alopecia (AGA), hirsutism, and thyroid-related hair loss.

Androgenetic alopecia is the most prevalent form of hair loss, affecting approximately 50% of men and 40% of women by age 50, with prevalence increasing with age (4-6). Ethnic variations exist, with lower rates in Asian and African populations compared to Caucasians (7). Similarly, alterations in estrogen and thyroid hormone levels such as during menopause or thyroid dysfunction can disrupt the anagen phase, leading to diffuse hair shedding and thinning (8,9). This narrative review aims to summarize the current evidence on the endocrine regulation of hair growth and loss, focusing on the physiological roles of key hormones, their mechanisms of follicular control, and associated clinical implications. It further highlights hormone-related alopecias and provides insights for potential therapeutic interventions in endocrinemediated hair disorders.

II. ENDOCRINE SYSTEMS INFLUENCING HAIR GROWTH

The endocrine system plays a central role in regulating the hair growth cycle through the actions of multiple hormones, particularly androgens, estrogens, thyroid hormones, prolactin, insulin, melatonin, and cortisol (3,10-12). Androgens, mainly testosterone and its more potent derivative dihydrotestosterone (DHT), act on androgen receptors within dermal papilla cells to transform vellus hairs into terminal hairs in androgen-dependent regions such as the beard, axilla, and pubic area. However, excessive androgenic activity shortens the anagen phase and miniaturizes scalp follicles, leading to androgenetic alopecia (3,13).

Estrogens exert protective effects on hair by prolonging the anagen phase and reducing follicular miniaturization. Estrogen deficiency, as seen during menopause or postpartum, can shift follicles prematurely into telogen, contributing to diffuse thinning (10,14). In contrast, thyroid hormones influence the overall frequency of the hair cycle, and both hyperthyroidism and hypothyroidism can disrupt follicular activity resulting in fine, brittle, or diffusely shedding hair (15).

Prolactin exhibits site- and sex-specific effects on follicles; it can induce catagen transition in male scalp hair while promoting follicular elongation in the female frontal region. Hyperprolactinemia may be associated with diffuse hair loss or hirsutism depending on hormonal balance (11). Insulin and insulin-like growth factors (IGFs) promote follicular activity and, through the upregulation of 5α -reductase, increase local DHT production linking metabolic disorders such as hyperinsulinemia to hair changes and alopecia (10-11). Melatonin, produced in both the pineal gland and hair follicles, supports pigmentation. counters oxidative stress, and prolongs anagen duration, thereby maintaining hair density and color (12). Meanwhile, activation of the hypothalamicpituitary-adrenal (HPA) axis under chronic stress elevates cortisol, which suppresses follicular matrix cell proliferation and extracellular matrix proteins, resulting in telogen effluvium (11-12).

Other endocrine and metabolic modulators also influence hair biology. Vitamin D plays a vital role in postnatal follicular cycling and regeneration, with deficiency contributing to alopecia unresponsive to calcium correction (10). Progesterone inhibits the

local conversion of testosterone to DHT, thereby exerting a protective effect against androgen-induced follicular miniaturization (3,10). Collectively, these hormonal interactions illustrate the complex and dynamic endocrine regulation of hair growth and loss across physiological and pathological states.

III. THERAPEUTIC APPROACHES TARGETING ENDOCRINE PATHWAYS

Therapeutic strategies for hair growth and loss primarily aim to modulate endocrine pathways that regulate the hair follicle cycle. These approaches are particularly relevant in disorders characterized by hormonal imbalance, such as androgenetic alopecia (AGA) and thyroid-related hair loss. In AGA, excessive androgen signaling, particularly via dihydrotestosterone (DHT), shortens the anagen phase and miniaturizes follicles. The mainstay treatments 5alpha-reductase (5-AR) inhibitors such as finasteride and dutasteride suppress DHT synthesis and have shown significant efficacy in both men and women, with topical formulations reducing systemic effects [11–17]. Antiandrogenic agents including spironolactone, cyproterone acetate, and flutamide block androgen receptors or inhibit androgen production, providing effective options for female pattern hair loss, though safety concerns like hepatotoxicity limit some agents' use [18-19].

Beyond androgens, thyroid hormones are critical for normal follicular cycling; both hypo- and hyperthyroidism can lead to diffuse telogen effluvium. Restoration of euthyroid status through levothyroxine or other thyroid-modulating therapies generally reverses hair shedding [20]. Stress-induced hair loss involves activation of the hypothalamic-pituitary-adrenal (HPA) axis and elevated cortisol levels; corticosteroids remain first-line for autoimmune alopecia areata, while adjunctive therapies such as ketoconazole, with its mild antiandrogenic and cortisol-modulating actions, may offer additional benefit [21-22].

Growth hormone (GH) and prolactin dysregulation also contribute to altered hair structure and cycling. GH replacement therapy can reverse hair thinning in deficiency states, while dopamine agonists that normalize prolactin levels may reduce hirsutism or telogen effluvium associated with hyperprolactinemia [23-24]. Emerging molecular therapies targeting hair-

specific endocrine signals include antibodies against dickkopf-1 (DKK1), which modulates Wnt/β-catenin signaling, and thyroid receptor-β (TRβ) agonists that promote anagen induction [25-26]. Combination regimens such as finasteride or spironolactone with minoxidil demonstrate superior outcomes to monotherapy, while non-pharmacologic modalities like low-level laser therapy (LLLT) enhance follicular metabolism and complement hormonal treatments [27-29]. Overall, endocrine-based therapies represent a cornerstone of modern hair loss management, and integration of hormonal modulation with adjunctive regenerative and personalized approaches holds promise for improved clinical outcomes.

IV. CLINICAL IMPLICATION

Hair growth and loss have important clinical implications, often serving as indicators of underlying endocrine, systemic, nutritional, or psychological disturbances. Alopecia, a frequent cause of dermatologic consultation, may reflect thyroid dysfunction, autoimmune disease, anemia, or micronutrient deficiencies such as iron, zinc, and biotin [30-33]. Hormonal assessment forms a key component of the diagnostic work-up, particularly in cases of androgenetic alopecia, telogen effluvium, and hirsutism, where evaluation of thyroid hormones, androgens, prolactin, and gonadotropins aids in identifying underlying endocrine disorders [34-35]. Gender-specific approaches are essential, as female patients often present with multifactorial alopecia linked to hormonal or nutritional causes, whereas males predominantly exhibit androgen-mediated patterns [36]. Management is therefore individualized, including nutritional correction, infection control, endocrine therapy, or alteration of drug regimens, alongside symptomatic treatments such as topical minoxidil or oral antiandrogens [37-39]. For hypertrichosis and hirsutism, assessment of androgen excess is crucial, and therapeutic options such as hormonal modulation. laser epilation. pharmacologic suppression are selected based on etiology and patient preference [40]. Risk assessment and patient counseling are integral to care, as pharmacologic and surgical interventions may involve contraindications, adverse effects, and variable longterm efficacy [41]. Given the psychosocial impact of hair disorders, a multidisciplinary approach involving dermatologists, endocrinologists, and mental health professionals ensures comprehensive evaluation and management [42].

V. RESEARCH GAP AND FUTURE DIRECTION

Despite significant advances in understanding hair biology, several research gaps persist in the study of hair growth and loss. A major limitation is the absence of standardized, objective methods for assessing hair regrowth, particularly in androgenetic alopecia (AGA), which hampers the comparability of clinical outcomes across studies. Although tools such as phototrichograms and dermoscopic evaluations exist, their use remains inconsistent. Moreover, most available treatments require long-term administration, with limited data on their sustained efficacy and relapse rates after discontinuation. Comprehensive long-term studies are essential to establish durability and safety, especially for newer interventions such as oral minoxidil and low-level laser therapy (LLLT), which may have side effects including hypertrichosis, edema, or headaches [43-45].

At the mechanistic level, greater insight into molecular pathways such as Wnt/β-catenin, JAK/STAT, and BMP signaling is needed to facilitate the development of targeted therapies and personalized treatment protocols [46-47]. There is also insufficient understanding of interindividual variations genetic, hormonal, and metabolic that influence therapeutic response. Furthermore, current literature lacks comprehensive bibliometric and sex-specific analyses, particularly long-term, female-focused randomized controlled trials (RCTs), which limits the applicability of findings across populations [48].

Future research should emphasize personalized medicine by incorporating patient-specific genetic and endocrine profiles to optimize treatment outcomes. Promising avenues include combination therapies (e.g., microneedling with topical agents), targeted drug delivery systems to minimize systemic effects, and regenerative approaches using adipose-derived stem cells (ADSCs), exosomes, and tissue-engineering techniques to restore follicular activity [49–51]. Additionally, non-invasive stimulation methods, such as low-level electrical or laser therapy, and novel biomolecules like deoxyribose show potential to enhance hair follicle regeneration [52-54]. The integration of omics-based approaches including

genomics, proteomics, and metabolomics could revolutionize understanding of follicular biology by identifying molecular biomarkers for hair growth regulation. Moreover, translational research focusing on endocrine modulation (e.g., targeting androgen, thyroid, and prolactin pathways) can bridge mechanistic insights with clinical dermatologic and endocrinologic practice. Ultimately, advancing this field will require interdisciplinary collaboration, standardized assessment methods, and inclusion of diverse populations to achieve effective, sustainable, and individualized therapies for hair disorders [43–54].

VI. CONCLUSION

The endocrine system plays a central role in regulating hair follicle growth through complex hormonal interactions involving androgens, estrogens, thyroid hormones, and other mediators. Hormonal imbalance contributes to various alopecic conditions, underscoring the importance of comprehensive hormonal assessment and gender-specific management in clinical practice. Despite advances, translational application remains limited due to a lack of standardized diagnostics and female-focused, longterm studies. Integrating omics-based insights and personalized endocrinologic profiling may bridge these gaps. A multidisciplinary, evidence-driven approach combining dermatology, endocrinology, and molecular research is essential to develop targeted, patient-centered therapies and endocrine-related hair disorders.

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