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Psychophysiological Mechanisms of Stress Impact on Hair Loss Processes

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Abstract: ***The article presents a theoretical and analytical review of current data on the neuroendocrine, neurogenic, molecular, and cellular mechanisms of stress-induced hair loss and their practical significance for the hairdressing industry. The study is based on an interdisciplinary approach combining dermatology, trichology, neurobiology, and psychophysiology, allowing stress to be viewed as a complex factor acting through hormonal cascades, neural regulation, microinflammation, and oxidative damage to follicular cells. Particular attention is paid to the analysis of pathogenetic links, including the influence of CRH, ACTH, substance P, norepinephrine, cortisol, and sympathetic nervous system responses, as well as the role of DNA damage and pigmentation disorders in altering hair growth cycles. Therapeutic intervention points already used in medicine (NK-1R inhibitors, JAK inhibitors, antioxidants, local norepinephrine administration) are presented, and possibilities for their integration with aesthetic care methods are outlined. It is shown that a hairdresser-stylist, possessing knowledge of the pathogenesis of stress-induced alopecia, can act as the first link in the prevention system, ensuring early detection of pathological signs, informing clients about possible medical approaches, and facilitating collaboration with physicians. The article will be useful to professionals in hairdressing, trichology, dermatology, and researchers studying the psychophysiological aspects of hair health.***

Keywords: stress-induced alopecia, neuroendocrine regulation, substance P, cortisol, norepinephrine, oxidative stress, hair loss, therapeutic interventions, hairdressing, trichology.

1. Introduction

Contemporary hairdressing and trichology practice is marked by a rising number of clients reporting hair loss associated with psychoemotional and physiological overload. In the context of urbanization, high living pace, and social instability, stress has become one of the most common triggers of alopecias of diverse origin, including androgenetic, alopecia areata, and telogen effluvium. The importance of the stress factor is supported by both clinical observations and experimental data showing that stress-associated mediators can alter hair-follicle function at the molecular, cellular, and tissue levels [4].

Hair is a salient component of outward appearance and social identity; its loss exerts a pronounced effect on self-esteem, emotional state, and social adaptation [1]. For beauty-industry professionals, understanding the biological mechanisms of stress-related hair loss enables accurate client communication and timely referral to a physician, thereby improving service quality and trust. Stress-induced hair loss represents a complex cascade that includes activation of the follicle's local HPA-like axis, neurogenic mediators such as Substance P, and the sympathetic nervous system [5].

One of the key challenges for hairdressing specialists is integrating knowledge of hair physiology into daily practice. This does not imply conducting medical diagnostics, but rather developing competence in primary assessment of hair and scalp condition, identifying signs of stress-induced changes, and communicating appropriately with clients—especially in light of findings by Wang E. [9], which demonstrate direct sympathetic regulation of hair regeneration.

The aim of this study is to analyze the psychophysiological mechanisms of stress-induced hair loss, identify key pathogenetic links, and determine the practical implications of these data for the hairdresser-stylist's work.

2. Materials and Methods

The methodological framework draws on trichology, neurophysiology, and immunology. Particular attention is paid to how stress affects hair-follicle function through hormonal, neural, and inflammatory mechanisms [6]. Lee J. H. [7] shows that the state of follicular stem cells is linked to signaling pathways and immune defense. Liang A. [8] reports that chronic stress, hormonal shifts, and oxidative imbalance accelerate follicular aging.

The analysis is based on scientific sources. Fischer T. W. [4] used a model of male follicles to study the effects of CRH, ACTH, and neuropeptides with the involvement of caffeine. Cheng Y. [2] examined the association between psychological stress and neurotrophin levels in patients with androgenetic alopecia. Kim C. [5] considered the role of Substance P and its receptor NK-1R in inflammation in alopecia areata. Ahn D. [1] described autoimmune and apoptotic processes in stress-induced hair loss. Du F. [3] systematized oxidative-stress pathways and options for their correction. Zhang J. [9] investigated the influence of the sympathetic nervous system and norepinephrine on hair regeneration and graying.

The study method is a comparative analysis with five focal areas: the CRH/ACTH hormonal axis and its role in the follicle [4]; neurogenic regulation via Substance P/NK-1R [5]; immune niches and regulation of the hair cycle [6]; oxidative stress and its signaling pathways [3]; sympathetic regulation and nerve–follicle interactions [9]. Clinical data on links between stress markers and alopecia progression were also considered [2].

3. Results

Stress affects hair via hormonal and neural signals. Corticotropin-releasing hormone (CRH) and adrenocorticotropic hormone (ACTH) activate a local hypothalamic–pituitary–adrenal (HPA) -like axis in follicular tissues. This amplifies the production of inflammatory mediators and disrupts the function of cells responsible for hair growth [4]. Fischer T. W. [4] showed that activation of CRH-R1/2 receptors elevates intracellular calcium and initiates inflammatory processes. The follicle's growth zone weakens, anagen is interrupted, and the hair shifts into catagen.

Substance P is a neuropeptide released from nerve endings under stress. Kim C. [5] found that Substance P binds the NK-1R receptor on outer root sheath cells and recruits immune cells, creating a microinflammatory focus. Reactive oxygen species and enzymes damage matrix cells, precipitating hair shedding. The sympathetic nervous system also participates in growth regulation. Zhang J. [9] showed that norepinephrine activates follicular stem cells and stimulates growth; however, in chronic stress, excess norepinephrine depletes the stem-cell pool, impairing proper regrowth. For hair professionals, these mechanisms are important because they confirm stress-related hair loss as a biologically grounded process. If clients exhibit signs of such loss, a referral to a trichologist is advisable. Table 1 summarizes the principal neuroendocrine and neurogenic pathways regulating hair under stress.

Table 1: Neuroendocrine and neurogenic stress-related pathways in hair follicle regulation (Compiled by the author based on sources: [4, 5, 9])

Mechanism	Key mediator	Cellular target in hair follicle	Main effect on hair cycle	Potential risk in chronic stress
Hypothalamic–pituitary–adrenal (HPA) -like axis activation	CRH, ACTH	Dermal papilla cells, outer root sheath keratinocytes	Induces catagen, inhibits anagen	Persistent growth suppression
Neurogenic inflammation	Substance P	Outer root sheath keratinocytes, peribulbar immune cells	Attracts immune cells, damages matrix cells	Chronic perifollicular inflammation
Sympathetic nervous system signaling	Norepinephrine	Hair follicle stem cells	Activates regeneration	Stem cell depletion and impaired regrowth

As Table 1 shows, CRH and ACTH initiate a hormonal cascade that suppresses hair growth; Substance P provokes inflammation around the follicle; and under stress, norepinephrine shifts from a useful regulator to a risk factor. These processes act in concert and intensify hair loss.

Chronic stress is accompanied by persistently elevated cortisol. This hormone modifies signaling pathways in follicular cells, pushing them toward quiescence. Kiselev A. [6] demonstrated that cortisol disturbs the balance within the follicle's immune niches, reducing the activity of cells responsible for hair regeneration. With prolonged exposure, a "dormant" follicle state forms, which does not return to active growth without external intervention.

Acute sympathetic activation during severe emotional stress can lead to a rapid loss of hair pigmentation. Zhang J. [9] established that excessive norepinephrine release activates and subsequently exhausts melanocyte stem cells. The follicle then fails to produce melanin, which clinically presents as sudden graying. Oxidative stress is another key factor damaging follicular cells. Du F. [3] reported that accumulation of reactive oxygen species causes DNA strand breaks and mitochondrial damage in matrix cells. These early, often subclinical injuries slow growth and thin the hair. Natural follicular antioxidant systems—such as superoxide dismutase and catalase—become depleted under prolonged stress.

For the hairdresser-stylist, understanding these processes clarifies why cosmetic products cannot fully reverse the consequences of chronic stress. When clients present with a combination of shedding, thinning, and color change, the practitioner can recommend supportive care and refer the client to a medical specialist for comprehensive evaluation. Table 2 systematizes key molecular and cellular changes in the follicle under stress.

Table 2: Molecular and cellular consequences of stress in hair follicles (Compiled by the author based on sources: [3, 6, 9])

Mechanism	Key mediator	Cellular target	Main pathological effect	Clinical manifestation
Chronic cortisol exposure	Cortisol	Dermal papilla cells, follicular immune niches	Suppressed stem cell activation, prolonged telogen phase	Thinning hair, reduced regrowth
Acute sympathetic activation	Norepinephrine	Melanocyte stem cells in hair bulb	Stem cell depletion, melanin production arrest	Sudden graying
Oxidative stress	Reactive oxygen species (ROS)	Matrix keratinocytes, mitochondrial structures	DNA strand breaks, mitochondrial dysfunction	Slowed hair growth, fragility

As Table 2 indicates, different stress mechanisms target distinct follicular structures. Cortisol acts on regulatory cells to maintain quiescence; norepinephrine damages pigmentary stem cells; reactive oxygen species affect the matrix and mitochondria, reducing growth rate. Together, these processes create conditions for long-term disruption of the hair cycle.

Collectively, the findings indicate that stress activates interlinked neuroendocrine and cell–molecular mechanisms that disturb the hair-growth cycle. Chronic cortisol elevation

maintains follicles in a resting state; acute sympathetic activation accelerates pigment loss; and oxidative stress damages matrix cells and mitochondria. Acting together, these factors drive accelerated thinning, weakening, and shedding, underscoring the need for a comprehensive approach to diagnosing and preventing stress-induced alopecia.

4. Discussion

The analysis shows that stress does not act on the hair follicle in isolation but through a network of interconnected neuroendocrine, neurogenic, immune, and cell–molecular pathways. These mechanisms operate synchronously, forming a cascade that begins with hormonal activation and culminates in structural damage to matrix cells and the stem-cell compartment. At the neuroendocrine level, stress initiates the follicle's local HPA axis, where corticotropin-releasing hormone and adrenocorticotropic hormone induce the transition from anagen to catagen [8]. This effect is amplified by neuropeptides such as Substance P, which provoke local microinflammation and immune-cell recruitment to the hair bulb [5].

The immune reaction is complemented by sympathetic input. While norepinephrine normally activates stem cells, chronic hyperactivation depletes their regenerative capacity and impedes rapid regrowth [9]. At the cellular level, prolonged cortisol elevation maintains follicles in telogen, lowering dermal papilla activity [2]. Added to this is oxidative stress, arising from imbalance between reactive oxygen species and antioxidant defense. Du F. [3] showed that free radicals damage DNA and mitochondria in matrix cells, slowing hair growth and impairing structure. In acute stress, sympathetic activation can trigger abrupt pigment loss via melanocyte depletion, clinically manifesting as rapidly graying hair [9].

Practical experience confirms that addressing only surface manifestations—for example, with cosmetic masks, ampoules, or massage techniques—may yield temporary improvement but does not remove the root cause. As long as stress persists, follicles remain in an adverse biological milieu, predisposing to recurrent shedding. For hair professionals, it is important to differentiate physiological, "everyday" shedding from pathological, stress-related loss. The former falls within a norm of up to 100 hairs per day and is not accompanied by changes in structure or pigment. The latter features more pronounced shedding, alterations in fiber thickness and color, and focal thinning. A comprehensive approach should combine supportive cosmetic care with referral to a trichologist to investigate systemic drivers, assess stress levels, and, when necessary, initiate therapeutic measures.

The analysis points to several therapeutic directions for stress-induced hair loss, each targeting a specific link in the pathogenetic cascade. Contemporary medical practice employs pharmacologic and biotechnological methods aimed at neutralizing neuropeptides, suppressing pathological immune activity, reducing oxidative stress, and restoring follicular stem-cell function. One promising avenue is the use of neurokinin-1 receptor (NK-1R) antagonists, which block Substance P binding to target cells, preventing microinflammation and degradation of the dermal papilla [5]. These agents have been shown to reduce inflammatory infiltration around the follicle and stabilize anagen.

Another class comprises JAK inhibitors (Janus kinase inhibitors), which suppress aberrant signaling in immune cells attacking the follicle [1]. Their efficacy has been confirmed in clinical trials for alopecia areata, including stress-associated cases. To reduce oxidative stress, antioxidants are used both systemically and topically. Du F. [3] demonstrated that natural antioxidants can reestablish balance between free radicals and protective enzymes, improving conditions for hair growth.

A separate direction is local administration of norepinephrine (NE) in microdoses to activate follicular stem cells and accelerate regeneration [10]. However, exceeding a therapeutic dose risks exhausting regenerative potential, necessitating strict medical supervision. For stylists, awareness of these approaches is important not for independent application but for accurate client guidance and collaboration with clinicians. The practitioner can play a key role in early recognition of stress-induced alopecia, referral to the appropriate specialist, and provision of supportive care—scalp massage, microcirculation stimulation, gentle care products, and minimizing chemical and thermal damage. Table 3 outlines major therapeutic intervention points, their targets, and the hair professional's role in an integrated recovery strategy.

Table 3: Therapeutic intervention points in stress-induced hair loss and the role of the hair professional (Compiled by the author based on sources: [3, 4, 10])

Therapeutic Method	Target Mechanism	Clinical Effect	Role of Hair Professional
NK-1R inhibitors	Block substance P binding to NK-1 receptors	Reduce perifollicular inflammation, stabilize anagen	Inform client, monitor hair condition
JAK inhibitors	Inhibit autoimmune signaling (JAK-STAT)	Prevent immune-mediated follicle damage	Explain medical option, refer to specialist
Antioxidants	Neutralize reactive oxygen species (ROS)	Reduce oxidative stress, improve follicle health	Recommend supportive antioxidant-rich care
Local norepinephrine injection	Stimulate follicular stem cells	Promote hair regeneration	Avoid direct use, explain medical control

Integrating these approaches indicates that successful therapy requires combining medical treatment with cosmetic support. Informed practitioners become a bridge between client and physician, facilitating timely diagnosis, improving adherence to treatment, and fostering conditions for restoration of a normal hair-growth cycle.

In summary, stress-induced hair loss arises from several interconnected pathogenetic mechanisms. Neuroendocrine

factors such as CRH and ACTH trigger inflammatory cascades and alter the follicular microenvironment, while neuropeptides including Substance P intensify microinflammation and hasten entry into shedding [5]. Concurrently, chronic cortisol elevation maintains follicular quiescence, and acute sympathetic activation can cause rapid pigment loss and premature graying [9]. Oxidative stress exacerbates cellular and DNA damage, further slowing regeneration [7].

The complexity of these processes explains the limits of cosmetic methods that address only external signs. Durable therapeutic effects are achievable only when internal mechanisms of pathogenesis are targeted—modulating immune responses, neuropeptide signaling, and oxidative balance. Therefore, integrating medical and hairdressing approaches through interdisciplinary collaboration is essential for effective prevention and correction of stress-induced alopecia.

5. Conclusion

This study identified the complex neuroendocrine, neurogenic, molecular, and cellular mechanisms by which stress initiates hair loss and alters regeneration. Stress hormones and neuropeptides activate inflammatory cascades, disrupt the follicular microenvironment, and precipitate premature transition into the shedding phase. A chronically elevated cortisol background sustains long-term follicular quiescence, while acute sympathetic responses can provoke rapid graying. Oxidative stress and DNA damage decelerate reparative processes, establishing conditions for persistent disturbances in hair growth.

The analysis demonstrates that stress operates on multiple levels and cannot be effectively addressed with topical cosmetics alone. A durable result is achievable only by combining medical and aesthetic approaches that target both external care and the internal drivers of pathology.

The hairdresser-stylist assumes a pivotal role as a professional able to recognize signs of stress-induced alopecia early and recommend comprehensive intervention. Combining pathogenetic insight from the scientific literature with practical client work enables the practitioner to serve as a vital link in an interdisciplinary chain for preventing and correcting hair-growth disorders.

Accordingly, integrating aesthetic, educational, and supportive functions into salon practice can enhance assistance for clients with signs of stress-related hair loss. Future research should focus on developing structured protocols for collaboration between hair professionals and medical specialists and on evaluating the effectiveness of such protocols in preventing and correcting stress-induced hair-growth disturbances.

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