Alopecia: A Comprehensive Review of Pathophysiology, Diagnosis, Conventional Management, Nutraceuticals, and Emerging Therapeutic Approaches

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Abstract
Alopecia, commonly referred to as hair loss, is a prevalent dermatological condition that affects individuals of different ages and may have considerable physical, psychological, and social consequences. It is a multifactorial disorder associated with genetic, hormonal, immune-mediated, inflammatory, nutritional, systemic, and environmental factors. Depending on the underlying pathology, alopecia may present as nonscarring or scarring hair loss, with clinical patterns ranging from diffuse shedding and progressive follicular miniaturization to localized patches and permanent follicular destruction. This review provides a comprehensive overview of alopecia, including the normal hair-growth cycle, classification, pathophysiology, clinical manifestations, diagnostic approaches, complications, and current management strategies. Conventional treatments such as topical minoxidil, antiandrogenic therapies, corticosteroids, immunomodulatory agents, and hair transplantation remain important therapeutic options. Recent advances have expanded the treatment landscape through targeted therapies, particularly Janus kinase inhibitors for alopecia areata, as well as emerging approaches such as platelet-rich plasma, low-level light therapy, microneedling, follicular stem-cell activation, stem-cell-based therapies, exosomes, extracellular vesicles, and novel drug-delivery systems. Nutraceuticals and micronutrient supplementation may provide adjunctive benefits, particularly in patients with documented nutritional deficiencies, although evidence supporting routine supplementation remains variable. In addition to clinical manifestations, alopecia can significantly impair self-esteem, body image, psychological well-being, social functioning, and overall quality of life. Therefore, early diagnosis and individualized, multidisciplinary management are essential. Continued research into follicular biology, immune pathways, androgen signalling, regenerative medicine, and personalized therapeutic strategies is expected to improve treatment outcomes and provide safer and more effective long-term approaches for individuals affected by alopecia.

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Introduction

Hair is an important appendage of the skin and its appearance is influenced by the continuous cycling activity of the hair follicle. Individual hair follicles undergo repeated phases of growth (anagen), regression (catagen), and resting/shedding (telogen and exogen). Disturbances in the normal hair cycle, follicular structure, or scalp environment can result in excessive shedding or

progressive hair loss. Therefore, alopecia is not a single disease entity but a clinical manifestation with diverse genetic, hormonal, immune-mediated, nutritional, systemic, and environmental causes. Hair loss is broadly classified into nonscarring and scarring alopecias. Nonscarring alopecias include diffuse, patterned, and focal forms, whereas scarring alopecias are characterized by permanent destruction of hair follicles and may result in irreversible hair loss. [1]

Androgenetic alopecia (AGA) is the most common form of patterned hair loss in both men and women. It is a progressive disorder influenced by genetic susceptibility and androgen-dependent follicular miniaturization. In men, hair loss typically begins with recession of the frontal and temporal hairlines and may progress to involve the vertex. In women, the characteristic presentation is progressive thinning over the central scalp with relative preservation of the frontal hairline. Although the clinical pattern is often sufficient for diagnosis, several other disorders can produce similar presentations; therefore, appropriate clinical assessment is essential for accurate diagnosis and management. [2]

Hair loss may also develop acutely or diffusely following physiological or pathological stress. Telogen effluvium (TE) is a common cause of diffuse nonscarring hair shedding and occurs when an increased proportion of hair follicles prematurely enters the telogen phase. Common precipitating factors include acute systemic illness, fever, major physical or psychological stress, childbirth, substantial weight loss, nutritional deficiencies, and certain medications. In many cases, shedding is self-limiting and improves after identification and correction of the underlying trigger. However, persistent or recurrent hair shedding warrants further clinical evaluation to exclude nutritional, endocrine, systemic, or dermatological causes. [3]

Alopecia areata (AA) is an immune-mediated nonscarring alopecia characterized by sudden or progressive hair loss in well-defined patches or more extensive patterns. Although the scalp is most frequently affected, the disease may also involve the eyebrows, eyelashes, beard, and other hair-bearing areas. The clinical course is highly variable, ranging from spontaneous hair regrowth to persistent, recurrent, or extensive disease. Disease severity and its impact on quality of life are important considerations in clinical management. Advances in understanding the immunopathogenesis of alopecia areata have contributed to the development of targeted therapies, particularly treatments directed toward the Janus kinase (JAK) signaling pathway. [4]

In addition to AGA, TE, and AA, hair loss may result from nutritional deficiencies, endocrine and metabolic disorders, infections, autoimmune diseases, medications, mechanical or chemical injury, and primary disorders of the hair follicle. Scarring alopecias represent a particularly important group because prolonged follicular inflammation can lead to permanent follicular destruction. Consequently, early recognition of the clinical pattern, identification of the underlying cause, and appropriate treatment are essential to prevent progression and optimize hair regrowth.

Types and Classification of Alopecia

Hair loss can initially be classified according to the structural integrity of the hair follicle into nonscarring and scarring alopecia. In nonscarring alopecia, the hair follicle is generally preserved, allowing the potential for hair regrowth when the underlying cause is identified and appropriately managed. In contrast, scarring alopecia is associated with inflammatory or destructive processes that damage the follicular unit and replace it with fibrotic scar tissue, resulting in permanent hair loss. Early recognition and treatment of scarring alopecias are therefore particularly important. Nonscarring alopecia can be further categorized into diffuse, patterned, and focal forms based on the distribution of hair loss. [5]

The major forms of nonscarring alopecia include androgenetic alopecia (pattern hair loss), telogen effluvium, anagen effluvium, alopecia areata, and traction alopecia. Pattern hair loss is characterized by progressive follicular miniaturization in a characteristic distribution and includes male and female clinical patterns. Telogen effluvium generally presents as diffuse shedding, whereas anagen effluvium is characterized by more rapid hair loss following disruption of actively growing anagen follicles, commonly after exposure to cytotoxic agents or other severe insults. Alopecia areata typically produces focal, well-demarcated patches of nonscarring hair loss.

Scarring alopecias, also referred to as cicatricial alopecias, comprise a heterogeneous group of disorders in which inflammation and destruction of the hair follicle can result in permanent hair loss. Important examples include lichen planopilaris, frontal fibrosing alopecia, central centrifugal cicatricial alopecia, discoid lupus erythematosus, and other primary cicatricial alopecias. These disorders require early diagnosis because hair follicles that have been irreversibly destroyed generally cannot regenerate. [6]

A practical clinical classification is also based on the distribution of hair loss, which may be diffuse, patterned, or focal. Diffuse alopecia involves widespread reduction in hair density over the scalp, whereas patterned alopecia follows a characteristic anatomical distribution. Focal alopecia produces localized areas of hair loss and may be associated with conditions such as alopecia areata, infections, or localized inflammatory disorders. The distribution, onset, progression, and associated scalp findings can provide important clues for establishing the differential diagnosis before specialized investigations are undertaken. [6]

Traction alopecia represents a mechanically induced form of hair loss caused by repeated or prolonged tension on the hair follicles. Tight hairstyles, braids, ponytails, hair extensions, and other practices that exert persistent traction can contribute to its development. Early traction alopecia is usually nonscarring and may improve after removal of the mechanical stress. However, prolonged or severe traction can cause follicular inflammation, miniaturization, and eventual permanent follicular destruction. Clinical examination, dermoscopic findings, and, when necessary, histopathological evaluation can assist in distinguishing traction alopecia from other causes of hair loss. [7]

Pathophysiology

Normal hair growth is a dynamic cyclic process involving four major phases: anagen (growth), catagen (regression), telogen (resting), and exogen (shedding). During anagen, matrix keratinocytes actively proliferate and differentiate to produce the hair shaft. Catagen is characterized by controlled involution of the lower follicle, followed by the relatively quiescent telogen phase. Exogen represents the release of the hair shaft from the follicle. The duration and synchronization of these phases determine scalp hair density. Alterations in the duration or transition between phases can therefore result in excessive shedding, reduced hair density, or progressive

follicular miniaturization. [8]

In telogen effluvium, an increased proportion of hair follicles prematurely enters the telogen phase following a physiological or pathological trigger. Because the affected follicles remain in the resting phase before the hair shaft is shed, hair loss generally becomes clinically apparent only after a delay following the triggering event. Common precipitating factors include acute systemic illness, fever, major psychological or physical stress, childbirth, nutritional deficiencies, substantial weight loss, and certain medications. Restoration of the normal hair cycle generally occurs after the underlying trigger is eliminated, although chronic or recurrent telogen effluvium may require further investigation. [8]

Androgenetic alopecia (AGA) is characterized by progressive follicular miniaturization in genetically susceptible individuals. In affected follicles, the anagen phase becomes progressively shorter and terminal hairs gradually decrease in diameter and length, eventually producing finer, vellus-like hairs. Dihydrotestosterone (DHT), formed from testosterone by the enzyme 5α-reductase, plays an important role in androgen-sensitive follicles. However, the development and progression of AGA depend on a complex interaction between genetic susceptibility, androgen signaling, follicular sensitivity, and local molecular pathways. Trichoscopic features, particularly increased hair-diameter variability and a higher proportion of thin hairs, provide clinical evidence of follicular miniaturization. [9]

Female pattern hair loss (FPHL) also involves progressive follicular miniaturization but typically produces diffuse thinning over the central and parietal scalp with relative preservation of the frontal hairline. Although androgen signaling may contribute to disease development in some women, the pathophysiology of FPHL is heterogeneous and cannot be attributed solely to circulating androgen concentrations. Genetic susceptibility, individual follicular sensitivity to hormones, age-related changes, and local follicular signaling mechanisms may all contribute to disease expression and progression. [10]

In alopecia areata (AA), disruption of the normal immune privilege of the hair follicle permits immune-mediated attack against follicular structures. The disease is believed to result from interactions between genetic susceptibility and environmental or physiological triggers. $CD8^+$ cytotoxic T lymphocytes play an important role in the inflammatory response, while cytokine signaling involving interferon-γ (IFN-γ) and interleukin-15 (IL-15) contributes to amplification and maintenance of the immune response. Activation of the JAK–STAT signaling pathway has emerged as an important component of AA pathogenesis and has provided a mechanistic basis for the development of targeted JAK inhibitor therapies. [11]

Scarring (cicatricial) alopecias, including lichen planopilaris and frontal fibrosing alopecia, involve inflammatory destruction of the hair follicle with subsequent fibrosis. In several primary cicatricial alopecias, the bulge region, which contains important epithelial hair-follicle stem cells, is particularly vulnerable to inflammatory injury. Damage to follicular epithelial structures and depletion or dysfunction of follicular stem cells can impair follicular regeneration and lead to permanent replacement of the follicular unit by fibrotic tissue. Consequently, early recognition and suppression of active inflammation are essential therapeutic goals to prevent further irreversible follicular destruction. [12]

Overall, the pathophysiology of alopecia involves diverse mechanisms, including abnormal hair-cycle regulation, follicular miniaturization, androgen-dependent signaling, immune dysregulation, inflammation, and follicular fibrosis. Understanding these mechanisms has contributed to the development of increasingly targeted therapeutic approaches and provides an important foundation for evaluating conventional and emerging treatments for hair loss.

Clinical Features

The clinical presentation of hair loss varies considerably according to its underlying cause, duration, and severity. Patients may report increased hair shedding on the pillow, comb, or during bathing, generalized reduction in hair density, widening of the central part, recession of the hairline, or clearly demarcated areas of baldness. Associated scalp symptoms, including itching, burning, tenderness, pain, erythema, scaling, or other inflammatory changes, may provide important diagnostic clues and should be specifically assessed during clinical evaluation.

Androgenetic alopecia (AGA) generally develops gradually and follows a characteristic distribution. In men, progressive recession of the frontal and temporal hairline and thinning of the vertex are common, whereas women typically develop diffuse thinning over the central scalp with relative preservation of the frontal hairline. The rate and extent of progression vary among individuals. Early recognition is important because treatment may help slow progression and preserve existing hair before extensive follicular miniaturization occurs. [14]

Telogen effluvium (TE) usually presents with diffuse, excessive hair shedding rather than sharply defined areas of hair loss. Patients may report the onset of noticeable shedding several weeks to months after an acute illness, fever, major psychological or physical stress, substantial dietary change, childbirth, or exposure to certain medications. The scalp generally appears clinically normal without significant inflammation or scarring. Identification and correction of the precipitating factor are important components of assessment and management. [15]

Alopecia areata (AA) commonly presents as one or more smooth, well-circumscribed patches of nonscarring hair loss. Depending on disease severity, hair loss may progress to involve the entire scalp (alopecia totalis) or all body hair (alopecia universalis). Eyebrows, eyelashes, beard, and other hair-bearing sites may also be affected. Nail abnormalities, including pitting and other dystrophic changes, may occur in some patients. Trichoscopy can demonstrate characteristic findings and may provide additional support for the diagnosis.

Scarring alopecias may present with progressive hair loss accompanied by clinical evidence of inflammation, including perifollicular erythema, scaling, tenderness, burning, or pruritus. An important diagnostic feature is the reduction or complete absence of visible follicular openings in areas of established scarring. Frontal fibrosing alopecia (FFA) typically presents with progressive frontotemporal hairline recession and may be associated with eyebrow loss. Early inflammatory changes may be subtle; therefore, careful scalp examination and trichoscopy are valuable in suspected cases. [16]

Diagnosis

Diagnosis of alopecia begins with a detailed clinical history and physical examination. The history should establish the onset, duration, progression, and distribution of hair loss and should include recent systemic illness, psychological or physical stress, dietary changes, weight loss, medications, hormonal factors, pregnancy or childbirth, family history, and hair-care or hairstyling practices. Associated symptoms such as itching, pain, burning, scaling, or erythema should also be documented. Examination should include the scalp and, when clinically indicated, the eyebrows, eyelashes, nails, beard, and other hair-bearing areas. The distribution and morphology of hair loss help distinguish diffuse, patterned, focal, and scarring disorders. [19]

The hair-pull test is a simple bedside examination that can provide supportive evidence of active shedding. Examination of shed or broken hairs may help differentiate increased follicular shedding from hair-shaft breakage. Trichoscopy (dermoscopy of the hair and scalp) provides non-invasive magnified visualization of follicular openings, hair shafts, scalp skin, and vascular or inflammatory changes. It is particularly useful in differentiating clinically similar forms of alopecia. In AGA, findings such as increased hair-diameter variability, a greater proportion of thin hairs, and follicular miniaturization may support the diagnosis. [17]

Laboratory investigations should be guided by the clinical presentation rather than performed routinely in every patient. Depending on the history and examination, investigations may include assessment for iron deficiency, thyroid dysfunction, nutritional abnormalities, hormonal disturbances, or systemic disease. A scalp biopsy may be indicated when the diagnosis is uncertain, particularly when a scarring alopecia or another inflammatory scalp disorder is suspected. [18]

Histopathological examination is especially valuable in suspected cicatricial alopecia, where it can demonstrate follicular inflammation, follicular destruction, fibrosis, and other characteristic structural changes. In disorders such as lichen planopilaris and frontal fibrosing alopecia, biopsy findings can complement clinical and trichoscopic observations when these are insufficient for a definitive diagnosis. Early evaluation may be particularly valuable because advanced scarring can obscure the original clinical pattern. [19]

Complications

Although most forms of hair loss are not life-threatening, alopecia can produce significant psychological and psychosocial consequences. Individuals may experience reduced self-confidence, embarrassment, altered body image, anxiety, or social discomfort, particularly when hair loss is prominent or progressive. The psychological impact varies considerably among individuals and should therefore be assessed on a patient-specific basis. [20]

Scarring alopecia has an additional important complication: permanent destruction of hair follicles. Once the follicular unit has been replaced by scar tissue, spontaneous regrowth from the destroyed follicle is generally unlikely. Delayed diagnosis may therefore reduce the opportunity to suppress active inflammation and preserve unaffected or partially affected follicles. [21]

Inappropriate self-treatment may also complicate the clinical course. Excessive use of cosmetic products, repeated chemical treatments, aggressive hairstyling, unnecessary supplementation, or frequent changes between unproven hair-growth products may contribute to hair-shaft damage or scalp irritation and may delay appropriate diagnosis. Persistent, rapidly progressive, painful, or inflammatory hair loss should therefore undergo appropriate clinical evaluation rather than being managed solely with cosmetic products. [22]

Precautions

Appropriate hair-care practices can help minimize avoidable hair-shaft and follicular damage. Hair should be handled gently, while prolonged or repeated use of tight hairstyles, excessive heat, and harsh chemical treatments should be minimized, particularly in individuals with existing hair breakage or traction-related hair loss. Adequate nutrition and avoidance of extreme dieting are also important because inadequate energy or nutrient intake may contribute to diffuse hair shedding in susceptible individuals.

Patients should also be provided with realistic expectations regarding treatment. Most established hair-growth therapies require consistent use for several months before their effectiveness can be adequately assessed. Treatment should be selected according to the specific type and cause of alopecia. Self-medication should be avoided when the diagnosis is uncertain, particularly because some conditions require specific anti-inflammatory, immunomodulatory, antimicrobial, hormonal, or other medical interventions rather than cosmetic treatment alone. [23]

Management

Management of alopecia should begin with establishing the underlying diagnosis and addressing reversible contributing factors. In telogen effluvium, management primarily involves identifying and correcting the precipitating factor, such as nutritional deficiency, systemic illness, major physiological stress, or medication-related causes. Once the trigger is appropriately addressed, the normal hair cycle may gradually recover, although visible improvement can require several months.

For androgenetic alopecia and female pattern hair loss, topical minoxidil is an established therapeutic option. Additional pharmacological or procedural approaches may be considered according to the patient's age, sex, disease severity, reproductive considerations, contraindications, and treatment response. Because pattern hair loss is generally chronic and progressive, long-term management and appropriate follow-up are often necessary. [24]

Treatment of alopecia areata is individualized according to disease extent, duration, age, site involvement, disease activity, and patient preferences. Topical or intralesional corticosteroids may be appropriate for selected patients with localized disease. For patients with more extensive or clinically significant disease, systemic therapies, including targeted Janus kinase (JAK) inhibitors, have expanded the therapeutic options available. Treatment selection should consider efficacy, safety, comorbidities, monitoring requirements, and regulatory indications.

The management of scarring alopecias primarily aims to suppress active inflammation and prevent further follicular destruction. Anti-inflammatory and immunomodulatory therapies may be selected according to the specific type of cicatricial alopecia and its clinical activity. Because established follicular scarring is generally irreversible, early diagnosis, prompt treatment, and regular monitoring are

particularly important. Overall, management should be individualized following appropriate clinical assessment and confirmation of the underlying type of alopecia.

Recent Advances and Novel Therapeutic Approaches in Alopecia

The therapeutic landscape of alopecia has expanded considerably with improved understanding of the molecular mechanisms regulating hair-follicle cycling, androgen signaling, immune privilege, inflammation, and follicular stem-cell activity. Conventional therapies such as minoxidil, finasteride, corticosteroids, and hair transplantation remain important; however, recent research has increasingly focused on targeted immunomodulation, androgen-receptor inhibition, regenerative medicine, cellular therapies, novel drug-delivery systems, and follicular stem-cell activation. The most substantial therapeutic advances have occurred in alopecia areata, while several promising approaches for androgenetic alopecia and scarring alopecia remain under clinical investigation.

1. Janus Kinase Inhibitors in Alopecia Areata

One of the most important advances in alopecia treatment has been the development of Janus kinase (JAK) inhibitors for alopecia areata (AA). Alopecia areata involves immune-mediated disruption of the hair follicle's immune privilege, with activation of cytokine pathways including interferon-γ and interleukin-15. These cytokines signal through the JAK–STAT pathway, making JAK inhibition a rational targeted therapeutic strategy.

Baricitinib, a selective JAK1/JAK2 inhibitor, became the first systemic therapy approved by the US FDA specifically for severe alopecia areata in adults in 2022.

Ritlecitinib, which inhibits JAK3 and the TEC-family kinase pathway, was approved in 2023 for severe alopecia areata in adults and adolescents aged 12 years and older. Its approval was supported by a randomized clinical trial involving 718 patients with severe AA.

Another important development was deuruxolitinib, a JAK1/JAK2 inhibitor approved by the FDA in July 2024 for severe alopecia areata. In two phase III trials, treatment with deuruxolitinib 8 mg twice daily resulted in approximately 29–32% of patients achieving a SALT (Severity of Alopecia Tool) score ≤20 at week 24, compared with approximately 1% in placebo groups.

These therapies represent a major shift from nonspecific immunosuppression toward pathway-directed treatment. However, JAK inhibitors require appropriate patient selection and monitoring because systemic immunomodulation may be associated with infections, laboratory abnormalities, and other class-related safety concerns. [25]

2. Novel Androgen-Targeted Therapy for Androgenetic Alopecia

Androgenetic alopecia (AGA) remains challenging because existing treatments primarily slow progression or stimulate follicles rather than directly reversing the underlying follicular miniaturization.

Clascoterone is a topical androgen-receptor inhibitor being investigated as a potential treatment for male AGA. Unlike systemic antiandrogens, clascoterone is designed to act locally by competing with androgens at the androgen receptor. A large phase III trial involving 703 participants evaluated 5% clascoterone solution applied twice daily to affected scalp areas, with the study completed in January 2026. It remains an investigational approach rather than an established approved treatment for AGA.

This approach is particularly interesting because local androgen-receptor blockade may provide a means of targeting androgen-sensitive follicles while potentially reducing systemic exposure. [26]

3. RNA-Based and Gene-Targeted Approaches

The increasing understanding of molecular signaling in AGA has also led to investigation of RNA interference (RNAi), small interfering RNA (siRNA), antisense approaches, and other gene-targeted technologies.

One potential strategy is selective modification of androgen-receptor signaling or other molecular pathways involved in follicular miniaturization. These approaches could theoretically provide greater molecular specificity than conventional antiandrogenic therapy. However, most remain in the experimental or early clinical-development stage. Recent reviews identify androgen-receptor-targeting RNA-based approaches as an emerging area of AGA research. [27]

4. Platelet-Rich Plasma Therapy

Platelet-rich plasma (PRP) is an autologous regenerative treatment that involves concentrating platelets from the patient's blood and injecting the preparation into the scalp.

Platelets release multiple growth factors, including platelet-derived growth factor, vascular endothelial growth factor, transforming growth factor-β, and epidermal growth factor. These mediators may influence dermal papilla cells, angiogenesis, follicular proliferation, and the hair-growth cycle.

Recent systematic evidence supports PRP as a potentially useful treatment for AGA, although substantial variation exists in platelet concentration, preparation techniques, injection schedules, and treatment protocols. Consequently, standardization remains an important limitation. [28]

5. Low-Level Light Therapy

Low-level light therapy (LLLT), also called photobiomodulation, has emerged as a non-invasive adjunctive treatment for AGA. Devices generally use low-intensity red or near-infrared light to influence cellular signaling and mitochondrial activity within follicular cells.

Proposed mechanisms include stimulation of cellular metabolism, modulation of inflammatory pathways, and effects on the anagen phase of the hair cycle. A systematic review of AGA therapies identified low-level light treatment among interventions associated with hair-growth benefits, although treatment protocols and device characteristics vary substantially.

LLLT may therefore have a role as an adjunctive therapy, particularly for patients seeking non-pharmacological approaches. [29]

6. Microneedling and Combination Regimens

Microneedling involves producing controlled microscopic injuries in the scalp using needles. The resulting wound-healing response may stimulate growth-factor release, dermal remodelling, and follicular activity.

Increasing evidence suggests that microneedling may be particularly useful when combined with topical minoxidil or

other treatments, rather than being used as a stand-alone therapy. Its potential advantages include improved local drug penetration and stimulation of regenerative pathways.

The growing evidence for multimodal treatment reflects the multifactorial pathogenesis of AGA. Combining approaches that influence different mechanisms - for example, minoxidil with antiandrogenic treatment, PRP, LLLT, or microneedling - may provide greater benefit than relying on a single modality in selected patients. [30]

7. Stem-Cell-Based and Cell-Based Therapies

Regenerative medicine is investigating whether mesenchymal stem/stromal cells, adipose-derived cells, dermal papilla cells, and follicular stem cells can promote hair regeneration.

Potential mechanisms include secretion of growth factors, modulation of inflammation, stimulation of angiogenesis, and activation of endogenous follicular stem cells. Cell-based approaches are particularly attractive because they attempt to restore follicular function rather than merely prolonging the existing hair-growth cycle.

However, clinical translation remains limited by challenges involving cell survival, delivery, reproducibility, treatment standardization, long-term safety, and regulatory oversight. [31]

8. Exosome-Based and Extracellular Vesicle Therapies

Exosomes and extracellular vehicles (EVs) are being investigated as regenerative approaches because they can carry proteins, lipids, messenger RNA, and microRNA capable of influencing cellular communication.

In hair-follicle research, exosome-based preparations derived from stem or stromal cells have been investigated for their potential effects on dermal papilla cells, angiogenesis, inflammation, and follicular growth.

Although preliminary studies have generated considerable interest, important limitations remain, including differences in source cells, preparation methods, purification, dosage, route of administration, and characterization of the vesicles. Therefore, exosome-based hair treatments should currently be regarded as experimental rather than established standard therapy. [32]

9. Novel Drug-Delivery Systems

Another important area of research is improving the delivery of existing and emerging therapies directly to the hair follicle. Investigational approaches include:

- Microneedle-based delivery
- Nanoparticle and liposomal formulations
- Follicle-targeted topical systems
- Controlled-release formulations
- Iontophoretic delivery
- Drug-loaded biomaterials

The objective is to increase drug concentration at the follicular target while reducing systemic exposure and improving patient adherence. [33]

10. Personalized and Precision Treatment

The future management of alopecia is increasingly moving toward personalized treatment. Instead of treating all patients with the same regimen, treatment selection can incorporate:

- Clinical subtype and severity
- Disease duration
- Trichoscopic findings
- Genetic susceptibility
- Hormonal status
- Inflammatory activity
- Treatment history
- Patient preferences and risk factors

Digital photography, trichoscopy, quantitative hair-density measurements, and standardized severity scores such as SALT (Severity of Alopecia Tool) can also improve treatment monitoring and clinical-trial assessment.

11. Hair-Follicle Stem-Cell Activation and Regenerative Therapy:

A major emerging concept is to move beyond simply preventing hair loss and instead reactivate dormant hair-follicle stem cells.

PP405 is an investigational topical therapy designed to reactivate dormant hair-follicle stem cells. Its mechanism involves modulation of the mitochondrial pyruvate carrier (MPC) and cellular metabolism, with the aim of stimulating regenerative activity within the follicle. Early clinical development has demonstrated follicular target engagement, and a phase IIa study in AGA reported encouraging preliminary efficacy and tolerability. The developer has reported plans to advance the program into phase III studies.

Importantly, PP405 should currently be described as an investigational regenerative therapy, not as an approved treatment or established cure for baldness. [34]

12. Biologics and Targeted Immunotherapy for Scarring Alopecia

Scarring alopecias such as lichen planopilaris, frontal fibrosing alopecia, folliculitis decalvans, central centrifugal cicatricial alopecia, and dissecting cellulitis remain difficult to treat because established follicular destruction is irreversible.

Recent research has therefore focused on targeting specific inflammatory pathways. Investigational approaches include JAK inhibitors and biologics directed against TNF-α, IL-17, IL-23, and interferon-related pathways. A 2025 review identified TNF-α, IL-17, and JAK inhibition as among the more promising emerging approaches, although much of the evidence remains based on small studies, case series, and off-label use.

The major therapeutic objective in scarring alopecia remains disease stabilization and preservation of viable follicles, rather than regeneration of follicles that have already been destroyed. [35]

- **Future Perspectives**

The next generation of alopecia therapies is moving from general stimulation of hair growth toward mechanism-specific follicular intervention. JAK inhibitors have demonstrated that targeting a defined immune pathway can produce clinically meaningful hair regrowth in severe alopecia areata. In AGA, investigational therapies such as androgen-receptor blockade and follicular stem-cell activation aim to address mechanisms that are not directly targeted by conventional treatments. Regenerative medicine involving PRP, extracellular vesicles, stem cells, and tissue-engineering approaches may further expand therapeutic

possibilities.
Nevertheless, many regenerative, biologic, and molecular therapies remain investigational, and differences in trial design, patient populations, treatment protocols, and outcome measures make direct comparison difficult. Long-term randomized controlled trials are required to establish their efficacy, safety, durability, and cost-effectiveness before widespread clinical adoption.

- **Role of Nutraceuticals and Emerging Therapies In Alopecia**

Nutritional status and metabolic health can influence hair-follicle function because the rapidly proliferating cells of the hair matrix require adequate energy, protein, vitamins, minerals, and other micronutrients. Nutraceuticals have therefore attracted considerable interest as adjunctive treatments for alopecia, particularly androgenetic alopecia (AGA) and diffuse hair shedding. However, the therapeutic role of nutritional supplementation depends strongly on the presence of an underlying deficiency. Current evidence does not support indiscriminate supplementation of all patients with hair loss, and nutraceuticals should generally be considered adjunctive rather than replacement therapies for established medical treatment.

1. Vitamins and Micronutrients

Several micronutrients have been investigated because of their roles in keratin synthesis, cellular proliferation, oxidative stress, and hair-follicle cycling. Iron, vitamin D, zinc, selenium, and B vitamins are among the nutrients most frequently studied in relation to hair loss. A 2024 systematic review identified vitamin B, vitamin D, iron, and zinc as potentially important nutritional factors in AGA, although the findings across individual studies were not completely consistent.
Iron deficiency is particularly relevant in patients with diffuse hair shedding, especially when dietary insufficiency, blood loss, or other risk factors are present. Similarly, vitamin D deficiency has been associated with several forms of alopecia, although association does not necessarily establish that supplementation will improve hair growth in patients without deficiency. Nutritional assessment should therefore be individualized rather than based solely on the presence of hair loss.

2. Biotin

Biotin (vitamin B7) is frequently marketed as a hair-growth supplement because of its role in fatty-acid synthesis and keratin-related metabolism. However, clinically significant biotin deficiency is uncommon, and evidence supporting biotin supplementation in individuals without deficiency is limited. Therefore, routine high-dose biotin supplementation should not be considered an evidence-based treatment for AGA.
An additional concern is that high-dose biotin can interfere with certain laboratory immunoassays, potentially producing misleading results. Consequently, unnecessary high-dose supplementation should be avoided.

3. Vitamin D

Vitamin D has received substantial research attention because vitamin D receptors are expressed in hair-follicle cells and are involved in normal follicular cycling. Observational studies have reported associations between low vitamin D status and several alopecias. However, evidence that vitamin D supplementation independently improves hair growth remains less definitive.
Thus, vitamin D supplementation is most rational when documented deficiency or inadequate intake is present, rather than as routine treatment for every patient with alopecia.

4. Zinc and Other Trace Elements

Zinc participates in protein synthesis, cellular proliferation, DNA metabolism, and tissue repair, making adequate zinc availability important for normal hair growth. Zinc deficiency can be associated with hair loss, but supplementation in individuals with adequate zinc status has not consistently demonstrated benefit.
Similarly, selenium, copper, and other trace elements have been investigated. Both deficiency and excessive intake may be harmful; therefore, indiscriminate use of multiple mineral-containing supplements should be discouraged.

5. Plant-Derived Nutraceuticals

Several botanical ingredients have been investigated as potential adjunctive therapies for AGA. These include:

- Saw palmetto (Serenoa repens)
- Pumpkin seed oil (Cucurbita pepo)
- Tocotrienols
- Apple polyphenol extracts
- Curcumin-containing preparations
- Capsaicin/isoflavone combinations
- Omega-3 and omega-6 fatty acids

Their proposed mechanisms include modulation of androgen metabolism, antioxidant effects, reduction of inflammation, and improvement of the follicular microenvironment.
A recent network meta-analysis of 19 randomized controlled trials involving 1,658 patients with AGA found positive signals for several supplements, including standardized plant extracts, pumpkin seed oil, tocotrienols, apple extract, and selected multicomponent formulations. However, substantial differences existed among products, doses, treatment durations, and study designs, so these findings should not be interpreted as evidence that all nutraceuticals are equally effective.

6. Pumpkin Seed Oil

Pumpkin seed oil (PSO) has attracted interest because of its potential effects on androgen metabolism and follicular growth. Clinical studies have reported improvements in hair-related outcomes, and the recent network meta-analysis identified PSO as one of the supplements with favourable results in some outcome measures. Nevertheless, additional large, independent trials are required before it can be considered equivalent to established pharmacological treatments.

7. Marine Protein and Collagen-Containing Supplements

Marine protein-based supplements containing combinations of marine proteins, vitamins, minerals, and other ingredients have been evaluated in randomized clinical studies. Some trials have reported reductions in hair shedding and increase in hair density or hair-fibre diameter.
However, these findings should be interpreted cautiously because many commercial formulations contain multiple active ingredients, making it difficult to determine which

individual component is responsible for the observed effect. They may therefore be considered adjunctive nutritional approaches rather than established first-line treatments.

8. Amino Acids and Protein Supplementation

Hair shafts are primarily composed of keratin, and adequate dietary protein and amino-acid availability are essential for normal hair production. Nutritional insufficiency, severe caloric restriction, or rapid weight loss can precipitate diffuse telogen shedding.

Some nutraceutical formulations contain L-cystine, methionine, marine proteins, collagen, or other amino acids. A recent randomized study evaluating a combination containing L-cystine, Serenoa repens, pumpkin seed, and Pygeum africanum reported improvements in hair-related outcomes in participants with AGA or chronic telogen effluvium.

Nevertheless, supplementation should primarily address inadequate dietary intake or documented nutritional insufficiency, rather than replacing a balanced protein-containing diet. [36, 37]

Complications and Psychosocial Impact of Alopecia

Alopecia is not only a cosmetic concern but can significantly affect an individual's physical, emotional, and social well-being. The severity of the impact varies according to the type, extent, duration, age of onset, and visibility of hair loss. Although alopecia is generally not life-threatening, persistent or severe hair loss may lead to substantial psychological distress and reduced quality of life.

1. Physical Complications

The physical complications associated with alopecia depend largely on its underlying cause.

- Permanent hair loss: In scarring alopecias, chronic inflammation damages the hair follicle and can result in irreversible follicular destruction and permanent hair loss.
- Progressive follicular miniaturization: Androgenetic alopecia may progressively reduce hair density and increase the proportion of fine, vellus-like hairs.
- Scalp symptoms: Some inflammatory and scarring alopecias may be associated with itching, burning, tenderness, erythema, scaling, or pain.
- Nail abnormalities: Patients with alopecia areata may develop nail pitting, ridging, brittleness, or other nail changes, particularly in extensive disease.
- Eyebrow and eyelash loss: Alopecia areata and frontal fibrosing alopecia may involve the eyebrows and eyelashes, producing functional and cosmetic consequences.
- Risk of inappropriate treatment: Unsupervised use of topical or oral medications, excessive supplementation, or unproven cosmetic treatments may cause adverse effects and delay appropriate diagnosis.

2. Psychological Impact

Hair is closely associated with physical appearance, identity, and self-esteem. Consequently, visible hair loss may produce psychological distress even when the underlying condition is medically benign.

Common psychological consequences include:

- Reduced self-esteem and self-confidence
- Body-image dissatisfaction
- Anxiety and excessive concern about appearance
- Depressive symptoms
- Social withdrawal and avoidance of social activities
- Embarrassment or feelings of unattractiveness
- Fear of being judged or stigmatized
- Difficulty forming or maintaining social and interpersonal relationships

The psychological burden can be particularly pronounced in individuals with extensive alopecia areata, visible patterned hair loss, or eyebrow/eyelash involvement.

3. Social and Occupational Effects:

Alopecia may influence social interactions and everyday functioning. Individuals may avoid photographs, public appearances, social gatherings, or activities where their hair loss is highly visible. Some may frequently use hairstyles, wigs, hair fibers, caps, or cosmetic products to conceal hair loss.

In severe cases, concerns regarding appearance may affect academic performance, occupational confidence, interpersonal relationships, and participation in social activities. Young adults and individuals whose occupations involve frequent public interaction may experience greater perceived psychosocial burden.

4. Impact on Quality of Life

The impact of alopecia extends beyond the visible loss of hair. Patients may experience persistent worry about disease progression, treatment effectiveness, and the possibility of permanent hair loss. Repeated treatment attempts and the uncertainty associated with conditions such as alopecia areata can further increase emotional distress.

Therefore, assessment of alopecia should consider not only hair density and disease severity, but also quality of life, psychological well-being, and social functioning.

5. Importance of Psychological Support

Management of alopecia should adopt a patient-centred and multidisciplinary approach. Along with appropriate dermatological treatment, patients experiencing significant psychological distress may benefit from:

- Patient education and counselling
- Reassurance regarding the expected disease course
- Cosmetic camouflage and hair prostheses when appropriate
- Psychological or behavioural counselling
- Support groups and peer support
- Screening for significant anxiety or depressive symptoms
- Regular follow-up to assess both clinical response and quality of life. [38, 39]

Conclusion

Alopecia is a multifactorial disorder characterized by excessive or progressive hair loss that can arise from genetic, hormonal, immune-mediated, inflammatory, nutritional, systemic, and environmental factors. Although hair loss is rarely life-threatening, its effects can extend considerably beyond the scalp, affecting physical appearance, self-esteem, psychological well-being, social interaction, and overall quality of life. Understanding the normal hair-growth cycle

and the underlying pathophysiological mechanisms is essential for accurate classification, diagnosis, and selection of appropriate treatment.

The clinical management of alopecia has traditionally focused on identifying and correcting underlying causes, modifying contributing factors, and promoting hair regrowth. Established therapies such as topical minoxidil, antiandrogenic approaches, corticosteroids, and hair transplantation remain important components of treatment. However, therapeutic responses vary considerably among individuals, and long-term treatment may be required to maintain clinical benefits. Early diagnosis is particularly important in scarring alopecias, where irreversible follicular destruction can occur.

Recent advances have substantially expanded the therapeutic landscape of alopecia. Targeted therapies, particularly Janus kinase (JAK) inhibitors, have demonstrated significant clinical benefits in alopecia areata, while newer approaches targeting androgen signalling, follicular stem-cell activity, and intracellular metabolic pathways are being investigated for patterned hair loss. Regenerative approaches, including platelet-rich plasma, low-level light therapy, microneedling, stem-cell-derived products, extracellular vesicles, and exosome-based therapies, represent promising areas of research. Nevertheless, differences in study design, treatment protocols, patient populations, and outcome measures indicate that several of these approaches require further well-designed clinical trials before they can be considered established therapies.

Nutraceuticals and micronutrient supplementation may have an adjunctive role, particularly in individuals with documented nutritional deficiencies. Evidence regarding biotin, vitamin D, iron, zinc, selenium, plant-derived supplements, and combination formulations remains variable, and routine supplementation without an established deficiency should be approached cautiously. Thus, nutraceuticals should complement rather than replace evidence-based medical treatment.

Importantly, the management of alopecia should not be limited to achieving hair regrowth. Assessment of psychological distress, body-image concerns, anxiety, depression, social withdrawal, and quality-of-life impairment should form an integral part of patient care. A personalized and multidisciplinary approach combining accurate diagnosis, appropriate pharmacological or procedural therapy, nutritional optimization, psychological support, and long-term monitoring is therefore essential.

Overall, the future of alopecia treatment is moving toward precision, targeted, regenerative, and patient-centred therapies. Continued research into follicular biology, immune pathways, androgen signalling, stem-cell activation, extracellular vesicles, novel drug-delivery systems, and individualized treatment strategies may lead to more effective and durable therapeutic options. However, robust randomized clinical trials and long-term safety studies are still required to establish the efficacy and clinical relevance of many emerging interventions. An integrated approach that combines established treatments with scientifically validated novel therapies offers the greatest potential for improving hair growth, preventing irreversible follicular damage, and ultimately enhancing the quality of life of individuals affected by alopecia.

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