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FROM ROOTS TO RADIANCE: NUTRITIONAL SYNERGIES, INCLUDING VITAMIN E AND FOLIC ACID, FOR HEALTHY AND SHINY HAIR

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ABSTRACT

Hair health represents a complex interplay between genetic predisposition, hormonal regulation, and nutritional adequacy, with the hair follicle cycle serving as a continuously regenerating system vulnerable to both intrinsic and extrinsic stressors. Hair follicles undergo distinct phases including anagen (growth phase lasting two to seven years), catagen (transitional phase lasting two to four weeks), and telogen (resting phase lasting approximately three months), with disruptions in this cycle manifesting as various alopecias and premature graying. Genetic hair disorders including hypotrichosis, monilethrix, trichorrhexis nodosa, and ectodermal dysplasias result from mutations in genes encoding structural proteins and developmental signaling pathways. Acquired hair disorders encompass both nonscarring alopecias such as androgenetic alopecia, telogen effluvium, and alopecia areata, and scarring alopecias including discoid lupus erythematosus and lichen planopilaris. Nutritional synergies play pivotal roles in maintaining hair follicle health, with mounting evidence supporting the significance of micronutrients in hair biology. Vitamin E, particularly tocotrienol isoforms, functions as a potent antioxidant mitigating oxidative stress within hair follicles, with clinical trials demonstrating significant improvements in hair count and density following supplementation. Folic acid and vitamin B12 contribute fundamentally to nucleic acid synthesis and cellular proliferation within the highly proliferative hair follicle matrix, with deficiencies associated with premature canities and telogen effluvium. Botanical interventions including Emblica officinalis (amla), Eclipta alba (bhringraj), and Nigella sativa demonstrate hair growth-promoting activities through antioxidant, anti-inflammatory, and follicle-stimulating mechanisms validated through preclinical and clinical investigations. This comprehensive review synthesizes current understanding of hair biology, pathological conditions, and evidence-based nutritional interventions, emphasizing vitamin E and folic acid synergies in promoting healthy, radiant hair while highlighting areas requiring further investigation.

Keywords: Hair Growth, Amla, Vitamin E, Folic Acid, Trichology

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1. Introduction

A skin appendage, hair develops along the same developmental pathway as other ectodermal tissue. Hair graying is frequently interpreted as an indication of aging and diminished vigor. It takes on particular relevance among Asian races because even a few gray hairs stand out visibly among the black hair. There is little information available regarding the etiopathogenesis or clinical signs of premature graying of hair or canities, making it a mysterious condition. Regarding race and ethnicity, different reports are made regarding the age incidence. HAIR serves a variety of beneficial biological purposes, such as providing shelter from the weather and distributing sweat-gland products (such as Pheromethanes). To the untrained eye, hair seems to have a pretty homogeneous structure, with individual variations limited to color and quantity.[1] But the biology of hair, a very complicated anatomical structure, is still little understood. To make sense of the outcomes Conducting hair analysis tests with precision and comprehending the proper function of hair examination in drug misuse testing, a basic knowledge of the biology of Hair is essential. This correspondence provides a succinct overview of the anatomy and physiology of hair to comprehend how medications might enter and be held onto by hair. The terminally developed, dead keratinocytes (trichocytes) that make up hair are compressed into the hair shaft, a fiber with extraordinary tensile strength. A defining feature of animals is their hair, which has a broad variety of jobs. Physical protection, thermal protection, and dispersion of perspiration and sebum, insulation, and concealment of social relationships, as well as tactile and sensory functions. Within In human culture, hair has a significant psychological role and is linked to numerous illnesses like their loss or, less commonly, an excessive amount of hair. Based on ethnic origin, human hair can be divided into three main groups: Asian, Caucasian, and African.[2]

The follicle, though sometimes disregarded, is fascinating in a variety of ways. The ability of animals to renew themselves is nearly unique for cell and developmental biologists, recapitulating numerous embryonic stages along the way. For zoologists, loss is a feature of mammals that is important to their genetic success and survival for many mammals. Loss of fur or incorrect coloring causes one to die from the cold or predators.[3] Moreover, human follicles pose a special conundrum for endocrinologists because androgens also produce stimulation of hair growth in various regions and concurrently suppress scalp follicles, resulting in baldness. However, considering that hair loss is considered to be a medically insignificant condition, hair is not dangerous to life.

While the general characteristics of hair growth have been understood for a long time, real research into the specifics of hair growth has only just begun. A significant factor to take into account is that hair growth is cyclical with respect to the hair-producing follicle. The hair grows in the phase of the follicle and is subsequently held in the dead club form during the ensuing phase stage. Once the new hair grows during the next phase, the previous club hair perhaps lost. Often, this hair restoration or regeneration is not readily apparent by informal examination. When an animal's entire coat replaces itself every season, the coat, which typically experiences two growth cycles annually, there is a general shedding of older as the new ones grow out, club hairs appear.[4] But in mice and rats, growth cycles are more common, and in the majority of the follicles, some club hairs are kept (apart

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from the substantial protective hair follicles) in addition to the lone newly emerging hair. Typically, as many Some follicles have four or more club hairs, with the oldest ones often having their club ends in the follicle more superficially. This is not a confusing scenario. like in the chinchilla, where several follicles share a common exterior opening, with the compound follicle.

The process of embryogenesis Dermal and epidermal tissue makes up each hair follicle. An amalgam of mesenchymal cells makes up the dermal components, or dermal ma1 sheath. Dermal cells and papilla that at the onset of filamentous development, it forms directly beneath the epithelial hair germ. The hair germ on the epidermis descends to the hair peg, followed by the bulbous hair peg, which houses the anlagen of all the components of a hair follicle. Recombination of tissue research on embryonic skin has demonstrated that hair follicle numerous messages between the dermis are necessary for development as well as the skin conditions.[5] The first message tells the epidermal layer to form an appendage and is derived from the dermis. Since all vertebrate classes share this signal, the mouse dermis is capable of starting the formation of scale placodes in lizard epidermis or feather follicles in chick epidermis. After this, there is a less signal from the epidermis that is well defined but class-specific and tells the dermis to form a dermal papilla. And lastly, another dermal message causes the class-specific epidermal placode to form appendage.

2. Hair Growth Cycle

The physical accumulation that results from a coordinated process of cellular differentiation and proliferation within a hair follicle is called hair. Hair follicles are appendages derived from the epidermis that form through inductive processes involving specialized dermal fibroblasts and bipotential epithelial stem cells.[6,7]

When stem cells decide to become hair follicles, they go through a phase of intense proliferation that ends with the development of a fully grown hair follicle. The formation of patterns, stem cell augmentation, cell differentiation, epithelial–mesenchymal interactions, apoptosis (programmed cell death), cell and organ growth cycles, and coloration are all intricate aspects of the hair follicle cycle. The fact that a hair follicle is a rejuvenating system is the primary motivation for researching its cycle. Through going through all of the stages of the cycle—growth, regression, resting, shedding, and then growth again—the follicle exhibits its extraordinary capacity for self-regeneration. Anagen (growth stage), catagen (degenerative stage), telogen (resting stage), and exogen (shedding stage) are the four stages that normal hair follicles cycle through.[8]

The life cycle of scalp hairs is rather lengthy: the anagen stage lasts for two to five years, the catagen stage for a few days to a few weeks, and the telogen stage lasts for roughly three months. Disturbances in the hair cycle have a significant impact on the growth of visible hair. The affected skin area will then sport primarily catagen and/or telogen follicles, whose loosely fixed club hairs are ultimately shed (i.e. the typical anagen/telogen rate on the scalp [roughly 4:1] changes in favor of telogen). If anagen is prematurely cut short and catagen occurs too early, this must result in effluvium and alopecia. This is precisely what occurs, for instance, when anagen hair bulb proliferating cells are damaged by drugs, as in the case of drug-induced telogen effluvium or when anagen hair bulb is attacked by inflammatory cells in alopecia areata.[9]

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The three main phases of the hair growth cycle are anagen (growth phase, lasting two to seven years), catagen (exponentiation, lasting two to four weeks), and telogen (resting phase, lasting three months). primarily on the anagen (85–90.6%), telogen (10–15%), and catagen (1–2%) phases, the number of scalp hairs may vary. The anagen phase is when pigmented hair strands are created and the follicle reaches its full length and volume.[10] The lower follicle's epithelium splits through the catagen phase and expands up alongside the papilla till it lays beneath the bulge zone, where it establishes the club hair. Telogen, an inactivity period in the hair cycle, is defined by a decrease in the biological activity and proliferation of hair follicles.

3. Hair Problems

Genetic hair disorders-

In rare cases, genetic hair disorders may manifest as a multisystem syndrome in addition to causing severe alopecia in adults as well as kids. Therefore, the recognition of these genetic conditions is crucial for appropriate genetic counseling, the identification of other correlated ectodermal anomalies, and the start of appropriate therapy. A key phenotypic indicator of potential underlying genetic or metabolic syndromes is human hair. A number of genes found in the hair follicle have been identified thanks to recent developments in molecular genetics. Over the last two decades, several genes linked to different hereditary hair disorders in humans have been recognized.[11]

Hypotrichosis

A defect in hair regeneration resulting in impaired hair cycling and hair shaft anchoring in the skin causes sparse hair in hypotrichosis. Both syndromic and non-syndromic forms have been linked to mutations in various genes, including simplex hypotrichosis, hypotrichosis accompanied by HJMD, or juvenile macular dystrophy Marie Autosomal dominant and Unna hypotrichosis (MUH) Woolly hair recessive/hypotrichosis simplex.[12] Simple hereditary hypotrichoses—that is, those without any other abnormalities—are extremely rare; even rarer are those limited to one or two ectodermal defects. Hypotrichosis is a relatively prevalent feature of a variety of complex genetic syndromes only up to the scalp.[13]

The rare autosomal dominant disorder Marie Unna hypotrichosis, also known as Marie Unna hereditary hypotrichosis (MUHH), is characterized by sparse or absent scalp hair at birth, variable coarse, wiry regrowth of hair in childhood, and potential loss again at puberty resulting in non-scarring alopecia.[14,15]

Hair Shaft Disroders

Trichorrhexis Nodosa-

Trichorrhexis nodosa is the name given to the microscopic appearing of a fracture in which individual cortical cells splay out from the main shaft of the hair shaft, giving the impression that two brushes have been pushed together. Three forms of the condition can be distinguished: acquired trichorrhexis nodosa, trichorrhexis nodosa as a component of other syndromes, and primary congenital form. This review does not address an acquired form of trichorrhexis nodosa, which is caused by repeated trauma to the hair shaft.[16] Congenital trichorrhexis nodosa, the main genetic form of the disorder, inherits in an autosomal dominant manner. Normally, the hair is normal at birth and becomes fragile after a few months. With time, this condition might get better. Trichohepatoenteric syndrome, citrullinemia, and argininosuccinic aciduria are examples of Trichorrhexis Nodosa forms that are associated with syndromes.

Trichorrhexis Invaginata-

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The hair shaft invaginates multiple times along its length in trichorrhexis invaginata, commonly referred to as bamboo hair. Trichoscopy at low magnification reveals several tiny nodules distributed erratically. Trichoscopy at high magnification can be used to observe the invagination of the shaft's distal portion into its proximal portion, giving the appearance of a ball in a cup. Occasionally, the distal end may break before the proximal cupped end is visible[17]

Monilethrix-

Beaded hair, or monilethrix, is derived from the Latin monile (necklace) and Greek word thrix (hair). The most common mode of transmission is autosomal dominant; it has a high- penetrance and varying severity. Three genes (KRT81, KRT83, and KRT86) that code for the type II hair keratins Hb1, Hb3, and Hb6 have been linked to monelethrix. It has been reported that a mutation in the DSG4 gene, which codes for the desmoglein 4 protein, causes an autosomal recessive form.[18]

Ectodermal dysplasias

A broad category of diseases known as ectodermal dysplasias are typified by singular or combined anomalies that affect teeth, hair, nails, and perspiration systems. Other ectoderm-derived structures like the meibomian glands, mammary glands, branchial arch cartilages, pigment cells, anterior pituitary, thymus, and melanocytes may also exhibit abnormalities in these disorders. The most common appendage impacted by ectodermal dysplasias is the hair, which can manifest as hypotrichosis or alopecia.

Follic units with a single hair, anomalies in the pigmentation of the hair shaft, pili torti, trichoschisis, pili canaliculi, trichorrhexis nodosa, and pseudomonilethrix are common trichoscopic findings in ectodermal dysplasia.[19,20]

Hair loss (alopecia)- There are several classifications for alopecia, but the most widely used one separates nonscarring to scarring alopecia. Scarring alopecia eliminates the follicular openings, while nonscarring alopecia usually keeps the follicular ostia intact. In contrast to nonscarring alopecia, which is if not theoretically reversible, scarring alopecia results in permanent hair loss.

Different Nonscarring Alopecia Types

Androgenetic Alopecia-

Common balding, male-pattern balding, and female-pattern balding are other names for androgenetic alopecia. Instead of experiencing hair loss, the majority of people with androgenetic alopecia report thinning hair. It is advisable to get a record of baldness in grandparents and immediate relatives on each side of the family, as androgenetic alopecia is likely a multiallelic trait heritable from both mother and paternal sides.[21]

Senescent Alopecia-

Senescent alopecia is characterized by a genuinely widespread loss of hair without overtly increased shedding. This is not the case with telogen effluvium, when an elevated telogen count or the firm pull test may indicate greater shedding. If there is a disproportionate degree of thinning in the crown and vertex, this likely indicates the presence of androgenetic alopecia. There is no particular treatment for senescent alopecia.

Telogen Effluvium-

A disorder known as telogen effluvium occurs when an unusually high proportion of healthy hair from every part of the scalp enters the telogen phase, which is the resting phase of developing hair. Other disorders like trichotillomania and alopecia areata cause a large number of anagen hairs to change into telogen hairs, but the follicles that are impacted are aberrant.[22]

Alopecia Areata-

Alopecia areata typically manifest as confined patches, although it can also affect

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the entire body (alopecia universalis) or scalp (alopecia totalis). Long intervals among any such relapses and full hair regeneration in between episodes of alopecia are signs of a favorable prognosis.[23] Hair loss that is persistent or getting worse (or both) and has little to no remissions is suggestive of a poor prognosis. Additional unfavorable prognosticators include atopy history, alopecia areata in the family, onset of the disease before puberty, disease present for more than a year, extensive disease (particularly the marginal pattern or alopecia totalis) at any time, and Down syndrome. There are very rare cases of vitiligo and thyroid disorders linked to alopecia areata.

Syphilitic Alopecia-

Syphilis should always be ruled out in patients with unexplained hair loss, as a high index of skepticism is frequently needed to make the diagnosis. Patchy or diffuse alopecia accompanied by papulosquamous lesions caused by secondary syphilis on the scalp or in other locations is known as symptomatic syphilitic alopecia. Patients with essential syphilitic alopecia, which is typically linked to latent syphilis, may not exhibit any skin lesions or symptoms. Both types of syphilitic alopecia can cause hair loss, which can be either gradual and subtle or rather quick.[24]

Trichotillomania- Adolescent females are the most common patients of trichotillomania, while it can also afflict adults, children, and people of all ages and genders. While some patients readily confess to being manipulative, others argue that their hair plucking is an uncontrolled habit. While the majority of trichotillomania sufferers are experiencing some sort of emotional stress, some are downright crazy. It may become clear from a complete history—which frequently necessitates separate conversations with the patient and parents—that the hair loss was self-inflicted. In cases of trichotillomania, a trend of hair loss typically indicates the diagnosis. There is one or a few well-defined regions of hair loss, frequently shaped in an odd or geometric way. Lesions can affect a large portion of the scalp and range in size. The impacted regions greatly thin out.

Traction Alopecia-

Traction alopecia is a type of traumatic alopecia that is comparable to trichotillomania. The primary distinction is that in traction alopecia, the hairs inadvertently sustain trauma. Traction alopecia primarily affects African-American women in the United States. When hair is styled in childhood, it is twisted into tight braids that are secured with ribbons or elastic bands. This is when the trouble starts. The outer strands of the braid experience the greatest tension due to the mechanics of braiding. Alopecia gradually spreads along the scalp's edge and in the spaces between braids. Should traumatic styling start after childhood, the identical procedure takes place. The arrangement and style technique (ponytail, tight curlers, braiding) determine the sequence of hair loss.[25]

Tinea Capitis-

A common ailment called tinea capitis, or ringworm of the scalp, is brought on by dermatophytes, a class of fungi that can penetrate and inhabit keratinized surfaces. By doing this, these fungi have the ability to kill the root of hair and significantly inflame the tissues around it. Until proven differently, an African-American child's round, scaly patch of alopecia should be diagnosed as tinea capitis.

Scarring Alopecia

Scarring alopecia encompasses a heterogeneous group of diseases. The common denominator is a tendency for hair

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destruction even in early or mild disease. In nonscarring alopecia, there is the potential for hair regrowth, but in scarring alopecia hair loss is permanent. Clinical evidence of scarring alopecia is manifest by erythematous papules, pustules, or scaling centered around hair follicles and eventual obliteration of follicular orifices. An additional feature often found in scarring alopecia is polytrickia, in which multiple hair shafts exit a single enlarged orifice. The most familiar form of scarring alopecia is discoid lupus erythematosus of the scalp.[26]

Other factors can also lead to scarring alopecia. The most frequent is known as follicular degeneration syndrome in African Americans, pseudopelade in Whites, and folliculitis decalvans in cases when pustule production is present.[27]

A rare but unique type of scarring alopecia called lichen planopilaris can affect only the scalp, but it frequently coexists with lichen planus lesions somewhere else on the body. There may be sporadic patches of hair loss on the scalp that exhibit erythema and scaling as a result of perifollicular[28]

irritation. The impacted hair follicles are the focal point of the histologic alterations in lichen planus. The most prevalent locations for numerous, firm scalp nodules associated with dissecting cellulitis of the scalp are the crown, vertex, and occiput.[29] The nodules quickly grow into oval, linear, fluctuant, swampy ridges that eventually release a purulent discharge. Lesions gradually grow together to the point where pressure on one fluctuating location may cause pus to leak from holes that are several centimeters apart. The ailment can manifest singly or in conjunction with conglobate acne or hidradenitis suppurativa.[30]

4. Nutritional Synergies

Vitamin E

Micronutrients are important components of the typical cycle of hair follicles; they contribute to cellular turnover, which occurs often in the matrix cells in the hair follicle bulb which divide quickly splitting up. Aging also affects the scalp, resulting in greying, a loss in melanocyte function, and alopecia, or a reduction in hair growth. Increased oxidative stress plays a crucial part in the progressive loss of pigmentation, which is caused by a variety of factors such as diminished DNA repair, telomerase loss, antiapoptotic signals, and enzyme depletion related to melanogenesis.[31] The sensitivity of immune cells to oxidative injury is high. As a component of the immune system's defense mechanism, they also release reactive oxygen species, which can cause a lipid peroxidation process. Antioxidant supplementation fundamentally corrects immune deficiencies associated with aging, including increased T-cell subsets and total lymphocyte counts, elevated interleukin-2 levels, increased activity of natural killer cells, improved mitogen responsiveness, increased antibody response to antigen stimulation, reduced prostaglandin synthesis, and reduced lipid peroxidation.[32]

An autoimmune illness influenced by genetic predisposition, emotional stress, and environmental factors, Alopecia Areata, has been linked in clinical investigations to oxidant/antioxidant discrepancy in its patients.[33]

After reviewing these papers, the majority of reviewers found that those suffering from alopecia areata had higher levels of biomarkers associated with oxidative stress and lower levels of antioxidant enzymes that protect the skin. Engaged in the oxidant/antioxidant balance, vitamin E aids in preventing damage from free radicals.[34] When 15 individuals with alopecia areata had their serum and tissue vitamin E levels assessed, Ramadan and colleagues discovered that the vitamin E levels in the patients had dramatically decreased compared to the healthy controls.

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Vitamin E encompasses a group of eight fat-soluble compounds, comprising four tocopherols (α-, β-, γ-, and δ-tocopherol) and four tocotrienols (α-, β-, γ-, and δ-tocotrienol). Being fat-soluble, vitamin E is predominantly present in quantities ranging from 1.3 to 20.3 mg per serving in vegetable oils such as soya, corn, and sunflower. Additionally, it is found in oleaginous fruits like walnuts, hazelnuts, almonds, and whole grain germs. Applying vitamin E oil topically has been shown to delay the onset of premature aging, enhance blood flow to the scalp by widening capillaries, and hydrate hair by serving as an antioxidant. However, there is a dearth of information regarding the impact of vitamin E supplementation. Another research has demonstrated that giving patients with Alopecia areata a daily dose of a combination of tocotrienols for 4–8 weeks increased the amount of hair on their bodies.[35]

Folic acid and Vitamin B12

Hair loss has been linked to deficits in folate and vitamin B12. 52 Indian participants with premature canities (hair aging) under the age of twenty were included in a case-control study, with a matching reference for each patient. The levels of biotin, folic acid, and vitamin B12 in the two groups were measured and compared by the authors.[36] The findings revealed decreased levels of biotin in the instances without any evident biotin deficit and deficiencies in vitamin B12 and folic acid among the individuals assessed. A water-soluble B vitamin folate, which is found in food, and fully oxidized monoglutamate (folic acid), which is a naturally occurring folate. Folate is a coenzyme that is involved in the metabolism of amino acids and the production of nucleic acids. About half of the body's total concentration is found in the liver, whereas 5-methyl-tetrahydrofolate is present in the plasma. Red blood cell formation, brain function, and DNA synthesis all depend on vitamin B12.[37] Methylcobalamin and 5-deoxyadenosylcobalamin are the names of the active forms of vitamin B12. As a cofactor for methionine synthase, vitamin B12 influences the synthesis of about 100 substrates, including proteins, RNA, and DNA. For adult U.S. populations, 2.4 mcg of vitamin B12 is the recommended daily allowance. The maximum amount of vitamin B12 that can be consumed is not set because of its minimal risk of harm.[38] Given their roles in the synthesis of nucleic acids, folate, and vitamin B12 may contribute to the highly proliferative nature of hair follicles.[39,40]

Emblica officinalis

A transient tree of the Euphorbiaceae family, Phyllanthus emblica L. Is also known as Indian gooseberry. Emblic fruit may be considered a plant source of natural antioxidants, nutraceuticals, or therapeutic components because of its high content of phenolic compounds. Amla-berry improves the body's absorption of calcium, strengthening bones, teeth, nails, and hair in the process. It also helps to maintain youthful hair color, stops premature greying, and fortifies hair follicles to stop thinning as people age. The crushed fruits prevent greying hair and encourage healthy hair growth.[41]

Eclipta alba

Around the world, Eclipta alba is a well-known and widely used medicinal herb, particularly in tropical and subtropical regions. It is a member of the subfamily Asteraceae. Often referred to as Bhringraj in the Ayurvedic and Unani medical systems, it is known as the "King of Hairs".[42] the plant is widely used in India as a hair oil for long, healthy black hair. Photochemical screening reveals the presence of tannins, alkaloids, flavonoids, and carotenoid. It is abundant in calcium,

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potassium, and sodium. Petroleum ether and E. Alba Hassk's ethanol extract has been tested for stimulating hair growth activity in albino rats.[45]

Nigella sativa

The genus Nigella is a member of the Ranunculaceae family. Its essential oil components have traditionally been utilized in culinary arts, conventional medicine, and, more recently, cosmetics. Thymoquinone, the main volatile component, is a newly discovered natural medication with cytotoxic, anti-inflammatory, hepatoprotective, and antioxidant properties.[43]

A condition known as telogen effluvium is when early hair entry causes thinning or shedding of hair during the telogen phase. Thymoquinone (TQ), which has antioxidant and anti-inflammatory effects by inhibiting pro-inflammatory mediators like cyclooxygenase and prostaglandin D2, was employed in the Nigella sativa seed study. [44]

6. Clinical Trials

Table 1 summarizes the clinical trials observed

7. Patent Perspective

Table 2 summarizes the patent perpective observed

8. Future Perspective

The future of hair health management lies at the intersection of molecular biology, precision nutrition, and personalized medicine. Emerging research directions include identification of novel genetic markers associated with premature graying and various alopecias, enabling early risk stratification and targeted interventions. Advanced omics technologies including genomics, transcriptomics, and metabolomics will facilitate comprehensive profiling of individual hair follicle microenvironments, elucidating patient-specific nutritional deficiencies and oxidative stress patterns. Development of biomarker panels assessing oxidative stress markers, antioxidant enzyme activities, and micronutrient status will enable personalized supplementation regimens optimized for individual metabolic profiles. Nanotechnology-based delivery systems promise enhanced penetration of vitamins, antioxidants, and botanical extracts to hair follicles, potentially revolutionizing topical therapy efficacy. Investigation of synergistic combinations of tocotrienols, folate, cobalamin, and botanical extracts through well-designed, adequately powered clinical trials will establish evidence-based formulations for various hair disorders. Integration of artificial intelligence and machine learning algorithms analyzing clinical data, genetic profiles, and treatment responses will enable predictive modeling for therapeutic outcomes. Understanding the intricate crosstalk between gut microbiome composition, nutrient absorption, and hair health may unveil novel probiotic or prebiotic strategies. Regulatory frameworks facilitating nutraceutical product standardization and quality assurance will ensure patient safety and therapeutic consistency.

Table 1: Clinical Trials

Intervention	Treatment	Age group	Study type	NCT Identifier

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A power supplement with biotin	Hair loss; Nail ingrown	Female participants aged between 25 and 55 years old	Unicentric, exploratory, prospective	NCT05945979
Nutrafol® Men Hair Growth Nutraceutical	Hair thinning	Males between 21-55 years of age	Randomized, double-blind, placebo-controlled study	NCT05339958
Alline proMEN	Hair loss	Males between 25-55 years of age	Double Blind, Randomized, Placebo-controlled	NCT04884347
Investigational product 1: (CPMSM: containing fish collagen, methylsulphonylmethane (MSM), vitamins and zinc); Investigational product 2 (HC+: containing mixture of herbal extracts, B-vitamins and zinc)	Hair thinning; Hair loss	Caucasian volunteers of both sexes; aged 25-60 years	Double Blind, Placebo-controlled, Randomised	NCT06174441
Biotin and silicon	Facial Skin, Texture Skin Ageing; Hair Thinning	35 Years to 70 Years (Adult, Older Adult)	Randomized, Triple-blind, Placebo-controlled Study	NCT06010745
Botanical extract of standardized biotin	Hair falling, thin hair, dry hair, brittle hair	20 Years to 60 Years (Adult)	Randomized, Double-Blind, Placebo-Controlled	NCT05972512
Herbal Supplementation	Hair thinning; Hair loss	Female (18 years or older)	Randomized, parallel, Double-Blind,	NCT05019066

Table 2: Patents granted for haircare

S.No.	Title of patent	Year of grant of patent	Patent number
1.	Composition comprising Amla and Hibiscus extracts for preventing lose of hair or promoting of hair	2019	KR102040277B1
2.	Composition for preventing hair loss or stimulating hair growth, containing amla extract	2018	WO2018021847A1
3.	Hair growth oil composition	2018	US10105312B2
4.	Vitamins and mineral salts composition to reduce the hair loss and/or to promote the hair regrowth	2007	EP1172080A1
5.	Composition based on vitamins and mineral salts to reduce hair loss and / or promote hair growth	2003	FR2811550A1
6.	Composition comprising plant extracts, vitamins, organic selenium, lecithin, glycerin, chlorophyll and homeopathic agents, useful for nourishing the skin, nails and hair and reducing hair loss.	2001	DE19858670A1
7.	Hair treatment composition and method	1981	EP0008171B1

8. Conclusion

Hair serves multifaceted biological, protective, and psychosocial functions, with its health intimately connected to

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systemic wellbeing, nutritional status, and age-related physiological changes. Premature graying and hair loss significantly impact quality of life, necessitating comprehensive diagnostic approaches and evidence-based therapeutic strategies. Nutritional interventions emerge as promising adjunctive therapies, with accumulating evidence supporting critical roles for antioxidant vitamins and B-complex vitamins in maintaining follicular health. Vitamin E, particularly tocotrienol isoforms, demonstrates significant potential in combating oxidative stress-mediated hair follicle damage and promoting anagen phase prolongation, as evidenced by clinical trials showing measurable improvements in hair density and quality. Folic acid and vitamin B12, essential for DNA synthesis and cellular proliferation, address fundamental metabolic requirements of rapidly dividing hair matrix cells, with supplementation potentially reversing deficiency-associated premature graying and telogen effluvium. Traditional botanical remedies including Emblica officinalis, Eclipta alba, and Nigella sativa provide valuable complementary approaches, leveraging antioxidant, anti-inflammatory, and follicle-stimulating phytochemicals validated through preclinical and preliminary clinical investigations. However, substantial gaps in scientific evidence necessitate rigorous, well-controlled clinical trials with standardized outcome measures, adequate sample sizes, and longitudinal follow-up to definitively establish efficacy, optimal dosing, and safety profiles. Integration of nutritional assessment and targeted supplementation within comprehensive hair health management protocols, alongside conventional pharmacotherapies and lifestyle modifications, offers holistic patient-centered care. From roots to radiance, the journey toward healthy, lustrous hair traverses genetic understanding, nutritional optimization, and, promising improved outcomes for individuals experiencing hair disorders.

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